ORTHODONTICS

CURRENT PRINCIPLES AND TECHNIQUES

SIXTH EDITION

GRABER · VANARSDALL · VIG · HUANG

ELSEVIER

Expert CONSULT Built with inkling | E books

Any screen. Any time. Anywhere.

Activate the eBook version of this title at no additional charge.

Expert Consult eBooks give you the power to browse and find content, view enhanced images, share notes and highlights—both online and offline.

Unlock your eBook today.

1. Visit **expertconsult.inkling.com/redeem**
2. Scratch off your code
3. Type code into "Enter Code" box
4. Click "Redeem"
5. Log in or Sign up
6. Go to "My Library"

It's that easy!

Scan this QR code to redeem your eBook through your mobile device:

Place Peel Off Sticker Here

For technical assistance:
email expertconsult.help@elsevier.com
call 1-800-401-9962 (inside the US)
call +1-314-447-8200 (outside the US)

ELSEVIER

Use of the current edition of the electronic version of this book (eBook) is subject to the terms of the nontransferable, limited license granted on expertconsult.inkling.com. Access to the eBook is limited to the first individual who redeems the PIN, located on the inside cover of this book, at expertconsult.inkling.com and may not be transferred to another party by resale, lending, or other means.

ORTHODONTICS

CURRENT PRINCIPLES AND TECHNIQUES

SIXTH EDITION

Lee W. Graber, DDS, MS, MS, PhD

Robert L. Vanarsdall, Jr., DDS

Katherine W.L. Vig, BDS, MS, D Orth, FDS RCS

Greg J. Huang, DMD, MSD, MPH

ORTHODONTICS

CURRENT PRINCIPLES AND TECHNIQUES

SIXTH EDITION

Lee W. Graber, DDS, MS, MS, PhD
Secretary General, World Federation of Orthodontists
Past President, American Association of Orthodontists
Past President, World Federation of Orthodontists
Private Practice, Glenview and Vernon Hills, IL

Katherine W.L. Vig, BDS, MS, D Orth, FDS RCS
Othodontic Faculty, Harvard School of Dental Medicine, Boston, MA
Professor Emeritus and Former Chair of Orthodontics
The Ohio State University, College of Dentistry, OH
Past President, American Cleft Palate-Craniofacial Association

Robert L. Vanarsdall Jr., DDS
Professor and Chair Emeritus
Department of Orthodontics
School of Dental Medicine
University of Pennsylvania
Philadelphia, PA

Greg J. Huang, DMD, MSD, MPH
Professor and Chair
Department of Orthodontics
School of Dentistry
University of Washington
Seattle, WA

ELSEVIER

ELSEVIER

3251 Riverport Lane
St. Louis, Missouri 63043

ORTHODONTICS: CURRENT PRINCIPLES AND TECHNIQUES,
SIXTH EDITION ISBN: 978-0-323-37832-1

Copyright © 2017 by Elsevier, Inc. All rights reserved.

No part of this publication may be reproduced or transmitted in any form or by any means, electronic or mechanical, including photocopying, recording, or any information storage and retrieval system, without permission in writing from the publisher. Details on how to seek permission, further information about the Publisher's permissions policies and our arrangements with organizations such as the Copyright Clearance Center and the Copyright Licensing Agency, can be found at our website: www.elsevier.com/permissions.

This book and the individual contributions contained in it are protected under copyright by the Publisher (other than as may be noted herein).

Notices

Knowledge and best practice in this field are constantly changing. As new research and experience broaden our understanding, changes in research methods, professional practices, or medical treatment may become necessary.

Practitioners and researchers must always rely on their own experience and knowledge in evaluating and using any information, methods, compounds, or experiments described herein. In using such information or methods they should be mindful of their own safety and the safety of others, including parties for whom they have a professional responsibility.

With respect to any drug or pharmaceutical products identified, readers are advised to check the most current information provided (i) on procedures featured or (ii) by the manufacturer of each product to be administered, to verify the recommended dose or formula, the method and duration of administration, and contraindications. It is the responsibility of practitioners, relying on their own experience and knowledge of their patients, to make diagnoses, to determine dosages and the best treatment for each individual patient, and to take all appropriate safety precautions.

To the fullest extent of the law, neither the Publisher nor the authors, contributors, or editors, assume any liability for any injury and/or damage to persons or property as a matter of products liability, negligence or otherwise, or from any use or operation of any methods, products, instructions, or ideas contained in the material herein.

Previous editions copyrighted 2012, 2005, 2000, 1994, 1985

Library of Congress Cataloging-in-Publication Data
Names: Graber, Lee W., editor. | Vanarsdall, Robert L., editor. | Vig, Katherine W. L., editor. | Huang, Greg J., editor.
Title: Orthodontics : current principles and techniques / [edited by] Lee W. Graber , D.D.S., M.S., M.S., Ph.D., President, American Association of Orthodontists, Past President, World Federation of Orthodontists, Private Practice, Vernon Hills, IL, Robert L. Vanarsdall Jr, DDS, Professor and Chair, Department of Orthodontics, School of Dental Medicine, University of Pennsylvania, Philadelphia, Pennsylvania, Katherine W.L. Vig, BDS, MS, D Orth, FDS RCS, Professor Emeritus and Former Chair of Orthodontics, The Ohio State University, College of Dentistry, Past President, American Cleft Palate-Craniofacial Association, Greg J. Huang, DMD, MSD, MPH, Professor and Chair, Department of Orthodontics, School of Dentistry, University of Washington. University of Washington.
Description: Sixth edition. | St. Louis, Missouri : Elsevier, [2017] | Revison of: Orthodontics / Thomas M. Graber, Brainerd F. Swain. 1985. | Includes bibliographical references and index.
Identifiers: LCCN 2016027994 (print) | LCCN 2016028815 (ebook) | ISBN 9780323378321 (hardback : alk. paper) | ISBN 9780323444323 (Online)
Subjects: LCSH: Orthodontics.
Classification: LCC RK521 .G673 2017 (print) | LCC RK521 (ebook) | DDC 617.6/43–dc23
LC record available at https://lccn.loc.gov/2016027994

Content Strategist: Kathy Falk
Associate Content Development Specialist: Laura Klein
Content Development Manager: Jolynn Gower
Publishing Services Manager: Hema Rajendrababu
Project Manager: Srividhya Vidhyashankar
Design Direction: Ashley Miner

Printed in United States of America

Last digit is the print number: 9 8 7 6 5 4 3 2 1

ELSEVIER Book Aid International Working together to grow libraries in developing countries
www.elsevier.com • www.bookaid.org

DEDICATION: TO ORTHODONTIC EDUCATORS AND RESIDENTS

Every text that is conceived, written and published starts with a purpose. Although this is the sixth edition of Orthodontics: Current Principles and Techniques, the concept of this multi-authored graduate level textbook started in the late 1960's with work that culminated in the 1969 publication of *Current Orthodontic Concepts and Techniques*. Why do a graduate level textbook? The answer was very simple: The principal editor and author Tom Graber, when interviewed later in life said it best—"There was a need." "The need" was defined by a widely held perception that there was a lack of supportive materials for clinicians teaching in orthodontic specialty education programs. Indeed, many of those who served as instructors and lecturers were private practice clinicians with little or no background in professional education.

The authors sought to provide a vehicle for educators to meld the then existing scientific basis of orthodontics with clinical practices in a manner that could be universally used. Course outlines that followed the textbook chapters as well as supportive slides were made available to help good clinicians become better teachers. A secondary but no less important goal for the textbook was to provide an organized platform by which practicing orthodontists could be updated on current concepts within the specialty. Tom Graber championed the need for continuing professional education and often cited G.V. Black, the father of modern dentistry, who had stated, "The professional person has no right to be other than a continuous student." The focus of the authors and editors who participated in the 1969 orthodontic text—and those who now do so in what is the eight iteration[1] has remained the same. We want to support orthodontic educators, the residents they teach and the "continuous students" orthodontic specialists must become.

With these thoughts as a background, it is to the educators and the orthodontic residents of the past, present and future we wish to dedicate the sixth edition. They embody the purpose of this text, because it is from their efforts our supportive science and clinical practice has grown and on their shoulders that rides the future development of our dental specialty.

---

[1]This is the sixth edition of the current series, with the title of the textbook and publisher changed in 1985. The first edition of the current series was the third for the text and included a change in title and publisher.

Pictures shown are from top row left: Edward H. Angle and the Class of 1900 of the Angle School of Orthodontia, from American Journal of Orthodontics and Dentofacial Orthopedics, 148:2, cover, 2015; Charles H. Tweed (glasses and bow tie) demonstrating patient at 1940's meeting (courtesy Rolf G. Behrents); Lysle E. Johnston lecturing to residents on cephalometrics (courtesy Rolf G. Behrents); Robert L. Vanarsdall lecturing to residents on three dimensional imaging, 2016. Bottom picture is of resident attendees and faculty at the Graduate Orthodontic Residents Program outside the American Association of Orthodontists building, St. Louis, 2015 (courtesy Rolf G. Behrents).

LIST OF CONTRIBUTORS

Adrian Becker, BDS, LDS, DDO
Clinical Associate Professor Emeritus
Orthodontics
Hebrew University-Hadassah School of Dental Medicine
Jerusalem, Israel

Erika Benavides, DDS, PhD
Clinical Associate Professor
Periodontics and Oral Medicine
University of Michigan
Ann Arbor, Michigan

Philip Edward Benson, PhD FDS (Orth)
Professor of Orthodontics
Academic Unit of Oral Health and Development, School of Clinical Dentistry
University of Sheffield
Sheffield, United Kingdom

Ignacio Blasi, Jr., DDS, MS
Diplomate, American Board of Orthodontics
Private practice
Tysons Corner, Virginia

Charles J. Burstone, DMD, MS, PhD†
Professor, Emeritus
Orthodontics
University of Connecticut Health Center
Farmington, Connecticut

Peter H. Buschang, PhD
Regents Professor & Director of Orthodontic Research
Orthodontics
Texas A&M University Baylor College of Dentistry
Dallas, Texas

Tamer Büyükyilmaz, DDS, MSD, PhD
Private practice
Adana, Turkey

David S. Carlson, PhD
Regents Professor
Biomedical Sciences
Texas A&M University Baylor College of Dentistry
Dallas, Texas

Lucia Cevidanes, DDS, MS, PhD
Assistant Professor
Orthodontics and Pediatric Dentistry
University of Michigan
Ann Arbor, Michigan

Stella Chaushu, DMD, PhD
Professor and Chair
Orthodontics
Hebrew University-Hadassah School of Dental Medicine
Jerusalem, Israel

Peter D. Chemello, DDS
Private practice, Northwest Oral and Maxillofacial Surgery
Arlington Heights, Illinois

Clark D. Colville, DDS, MS
Assistant Clinical Professor
Orthodontics
University of Texas School of Dentistry
Houston, Texas
Clinical Advisory Board
Invisalign
Santa Clara, California

Hali Dale, HON.B.Sc, DDS
Diplomate, American Board of Orthodontics
Private Practice
Toronto, Ontario

Jack G. Dale†, BA, DDS
Postdoctoral Fellowship in Orthodontics
Harvard University
Cambridge, Massachusetts
Associate Professor
Faculty of Toronto
Toronto, Canada
Chairman
Charles H. Tweed Foundation
Tucson, Arizona
Private Practice
Toronto, Canada

Dwight Damon, DDS, MSD
Private practice
Spokane, Washington

M. Ali Darendeliler, BDS, PhD, Dip Orth, Certif Orth, Priv. Doc, MRACDS (Ortho), FICD
Professor and Chair
Orthodontics
University of Sydney
Sydney, NSW, AUS
Head of Department
Orthodontics
Oral Health Services and Sydney Dental Hospital, Sydney LHD
Sydney, Australia

Hakan El, DDS, PhD
Associate Professor
Orthodontics
Hacettepe University
Sihhiye, Ankara, Turkey

Theodore Eliades, DDS, MS, Dr Med Sci, PhD, FIMMM, FRSC, FInstP
Professor and Director
Clinic Orthodontics and Paediatric Dentistry, Center of Dental Medicine
University of Zurich
Zurich, Switzerland

Norah Lisa Flannigan, BDS, MDETSCI, PhD, MFDSRCPS MORTHRCS, FDS (ORTH), RCS
Senior Lecturer
Orthodontics
The University of Liverpool
Liverpool, England
Honorary Consultant
Orthodontics
Liverpool University Dental Hospital
Liverpool, England

Thomas M. Graber†, DMD, MSD, PhD, OdontDr, DSc, ScD, MD, FDSRCS (Eng)
Director, Kenilworth Dental Research Foundation
Clinical Professor, Orthodontics, University of Illinois, Chicago, Illinois
Former Professor and Chair, Section of Orthodontics, Pritzker School of Medicine, University of Chicago, Chicago, Illinois
Former Editor-in-Chief, World Journal of Orthodontics
Editor-in-Chief Emeritus, American Journal of Orthodontics and Dentofacial Orthopedics

Dan Grauer, DDS, PhD
Assistant Professor
Orthodontics
University of Southern California
Los Angeles, California
Private Practice
Santa Monica, California

† Deceased.

Nigel W.T. Harradine, BDS, MB BS, LMCC, FDS RCS (London), FDS RCS (Edin), MSc (Orth)
Senior Clinical Lecturer
Child Dental Health
University of Bristol
Bristol, United Kingdom
Consultant Orthodontist
Child Dental Health
Bristol Dental Hospital
Bristol, United Kingdom

James Kennedy Hartsfield, Jr., DMD, MS, MMSc, PhD
Professor and E. Preston Hicks Endowed Chair in Orthodontics and Oral Health Research
Oral Health Science
University of Kentucky College of Dentistry
Lexington, Kentucky
Adjunct Professor of Orthodontics and Oral Facial Genetics
Orthodontics and Oral Facial Genetics
Indiana University School of Dentistry
Indianapolis, Indiana
Adjunct Professor of Medical and Molecular Genetics
Medical and Molecular Genetics
Indiana University School of Medicine
Indianapolis, Indiana
Adjunct Clinical Professor of Orthodontics
Orthodontics
University of Illinois
Chicago, Illinois

Nan E. Hatch, DMD, PhD
Associate Professor of Dentistry
Lysle E Johnston, Jr. Collegiate Professor of Orthodontics
Chair, Orthodontics and Pediatric Dentistry
University of Michigan
Ann Arbor, Michigan

Sarandeep S. Huja, DDS, PhD
Professor and Division Chief, Orthodontics and Director of Graduate Studies, Dentistry
College of Dentistry, Division of Orthodontics
University of Kentucky
Lexington, Kentucky

Donald R. Joondeph, BS, DDS, MS
Professor Emeritus and Former Chair
Orthodontics
University of Washington
Seattle, Washington

Jung Kook Kim, DDS, MS, PhD
Adjunct Associate Professor
Department of Orthodontics
School of Dental Medicine University of Pennsylvania
Philadelphia, Pennsylvania
Clinical Professor
Department of Orthodontics
College of Dentistry Yonsei University
Seoul, South Korea

Herbert A. Klontz, DDS, BA, MS
Clinical Associate Professor
Orthodontics
College of Dentistry
University of Oklahoma
Oklahoma City, Oklahoma

Jong Suk Lee, DDS, MS, PhD
Adjunct Assistant Professor
Orthodontics
School of Dental Medicine
University of Pennsylvania
Philadelphia, Pennsylvania
Clinical Professor
Orthodontics
College of Dentistry Yonsei University
Seoul, South Korea

Robert M. Little, DDS, MSD, PhD
Professor Emeritus
Orthodontics
University of Washington
Seattle, Washington

John B. Ludlow, DDS, MS, FDS RCSEd
Adjunct Professor
Diagnostic Sciences
University of North Carolina
Chapel Hill, North Carolina

Laurie McNamara McClatchey, DDS, MS
Adjunct Clinical Assistant Professor of Dentistry
Orthodontics and Pediatric Dentistry, School of Dentistry
University of Michigan
Ann Arbor, Michigan

James A. McNamara, Jr., DDS, MS, Phd
Thomas M and Doris Graber Professor Emeritus
Orthodontics and Pediatric Dentistry, School of Dentistry
The University of Michigan
Ann Arbor, Michigan
Professor Emeritus
Cell and Developmental Biology, School of Medicine
The University of Michigan
Ann Arbor, Michigan
Research Professor Emeritus
Center for Human Growth and Development
University of Michigan
Ann Arbor, Michigan

Ann M. Mercado, DMD, MS, PhD
Clinical Assistant Professor
Orthodontics
Ohio State University
Columbus, Ohio
Member of Medical Staff
Dentistry
Nationwide Children's Hospital
Columbus, Ohio

Lorri Ann Morford, PhD
Research Assistant Professor; Supervisor, Genetics/Genomics Core of the University of Kentucky Center for the Biologic Basis of Oral/Systemic Diseases
Oral Health Science
University of Kentucky College of Dentistry
Lexington, Kentucky

David Musich, DDS, MS
Clinical Professor
Orthodontics
University of Pennsylvania School of Dental Medicine
Philadelphia, Pennsylvania

Tung Nguyen, DMD, MS
Associate Professor
Orthodontics
University of North Carolina
Chapel Hill, North Carolina

Jeffrey P. Okeson, DMD
Professor and Chief, Division of Orofacial Pain
Oral Health Science
University of Kentucky College of Dentistry
Lexington, Kentucky

Juan Martin Palomo, DDS, MSD
Professor, Director of Orthodontic Residency, Director of Craniofacial Imaging Center
Orthodontics
Case Western Reserve University
Cleveland, Ohio

Leena Bahl Palomo, DDS, MSD
Associate Professor, Director of DMD Periodontics
Periodontics
Case Western Reserve University
Cleveland, Ohio

David E. Paquette, DDS MS MSD
Faculty, Clinical Orthodontics
St. Louis University
St. Louis, Missouri
Clinical Advisory Board
Invisalign
Santa Clara, California

Young - Chel Park, DDS, PhD
Professor Emeritus
Department of Orthodontics
College of Dentistry, Yonsei University
Seoul, Korea
Director, Private Clinic
Orthodontics
Yonsei Beautiful Friend Orthodontic Center
Seoul, Korea

Dubravko Pavlin, DMD, MSD, PhD
Professor and Program Director
Orthodontics
University of Texas Health Science Center San Antonio
San Antonio, Texas

William R. Proffit, DDS, PhD
Professor Emeritus and formerly Kenan Distinguished Professor
Department of Orthodontics
University of North Carolina
School of Dentistry
Chapel Hill, North Carolina

Jorge A. Ayala Puente, DDS
FACE
Cirujano Dentista
Specialist in Orthodontics
Santiago, Chile

W. Eugene Roberts, DDS, PhD, DHC (Med)
Professor Emeritus of Orthodontics, and Adjunct Professor of Mechanical Engineering
Orthodontics and Mechanical Engineering
Indiana University and Purdue University at Indianapolis
Indianapolis, Indiana
Visiting Professor of Orthodontics
Orthodontics
Loma Linda University, School of Dentistry
Loma Linda, California

Antonio Carlos de Oliveira Ruellas, DDS, PhD
Associate Professor
Orthodontics
Federal University of Rio de Janeiro
Rio de Janeiro, Brazil
Visiting Researcher
Orthodontics and Pediatric Dentistry
University of Michigan
Ann Arbor, Michigan

Rohit ChamanLal Sachdeva, BDS, M Dent Sc
Co-Founder and Former Chief Clinical Officer
Orametrix, Inc.
Richardson, Texas

Glenn T. Sameshima, DDS PhD
Associate Professor and Chair
Orthodontics
Herman Ostrow School of Dentistry
University of Southern California
Los Angeles, California

David M. Sarver, DMD, MS
Adjunct Professor
Orthodontics
University of North Carolina
Chapel Hill, North Carolina
Adjunct Clinical Professor
Orthodontics
UAB School of Dentistry
Birmingham, Alabama

Antonino G. Secchi, DMD, MS
Diplomate, American Board of Orthodontics
Former Clinical Assistant Professor of Orthodontics
University of Pennsylvania
Private Practice
Devon, Pennsylvania

Iosif B. Sifakakis, DDS, MS, Dr Dent
Lecturer
Orthodontics
School of Dentistry, National and Kapodistrian University of Athens
Athens, Greece

Raffaele Spena
Adjunct Associate Professor
Orthodontics
Università di Ferrara
Ferrara, Italy

Kingman P. Strohl, MD
Professor of Medicine, Physiology & Biophysics, and Oncology
Case Western Reserve University School of Medicine
Cleveland, Ohio
Sleep Center Director
University Hospitals Case Medical Center
Cleveland, Ohio
Director Sleep Disorders Program
Louis Stokes Cleveland VA Medical Center
Cleveland, Ohio

Zongyang Sun, DDS, MS, PhD
Associate Professor
Orthodontics
The Ohio State University College of Dentistry
Columbus, Ohio

Birgit Thilander, Odont Dr (PhD), Med Dr (hc), Dr Odont (hc)
Professor Emeritus
Orthodontics
Institute of Odontology, Sahlgrenska Academy
University of Gothenburg

Patrick K. Turley, DDS, MSD Med
Professor Emeritus and former Chair
Orthodontics and Pediatric Dentistry
School of Dentistry
University of California at Los Angeles
Los Angeles, California
Private practice Manhattan Beach, CA

Patricia Turley, DDS
Private practice, pediatric dentistry
Manhatten Beach, California

David L. Turpin, DDS, MSD
Moore/Riedel Professor
Orthodontics
School of Dentistry
University of Washington
Seattle, Washington

Serdar Üşümez, DDS, PhD
Professor
Orthodontics
Bezmialem Vakif University, Faculty of Dentistry
Istanbul, Turkey

James L. Vaden, BA, DDS, MS
Professor
Orthodontics
University of Tennessee
Memphis, Tennessee

Timothy T. Wheeler, DMD, PhD
Professor, Former Academy 100 Eminent Scholar, Former Chair & Program Director
Orthodontics
University of Florida
Gainesville, Florida

Dirk Wiechmann, DDS, PhD
Professor
Orthodontics
Hannover Medical School
Hannover, Germany
Private Practice
Bad Essen, Germany

Leslie A. Will, DMD, MSD
Chair and Anthony A. Gianelly Professor in Orthodontics
Orthodontics and Dentofacial Orthopedics
Boston University Henry M. Goldman School of Dental Medicine
Boston, Massachusetts
Adjunct Professor
Orthodontics
University of Pennsylvania School of Dental Medicine
Philadelphia, Pennsylvania

Bjorn U. Zachrisson, DDS, MSD, PhD
Orthodontics
University of Oslo
Oslo, Norway

PREFACE

This is the eighth re-writing of a textbook that has remained the most widely used graduate orthodontic textbook in the world and has now been translated in to multiple languages. Tom Graber was the initial editor in the late 1960s. He was encouraged by colleagues within the orthodontic educational community to fill what they saw as a void within orthodontic specialty programs. They perceived that although there were many excellent graduate programs, there was significant variance in the educational assets these programs provided for their residents. In bringing together the thoughts of excellent clinicians and related scientists in a text specifically meant for the advanced study of orthodontics, concepts and techniques could be shared with all benefitting.

An additional thought, shared at the time in both academic and professional association circles, was the need for a means to provide better continuing education to those already in practice. Today we take for granted that technologies and concepts change very quickly, and we must keep ourselves current. Forty-five plus years ago, the need was not as self-evident. Thus, in developing a textbook with recognized and vetted authorities on a wide array of subjects impacting clinical practice, the editors could provide a critical resource to those who had completed their formal education and were subsequently in primarily solo practices offices. The concept was that the text would be a basis for continued learning—a foundation on which these clinicians could better judge new material seen in their journals and professional lectures. By the very nature of this concept, there was an inherent demand that the text itself be updated to reflect the current basis on which the orthodontic specialty was being practiced. This demanded that it be updated on a regular basis, reducing emphasis in some areas, adding in others as both basic science concepts and clinical techniques further matured.

The task of "keeping current" is not an easy one and has become more difficult with the explosion of orthodontic-related research and technical development. Additionally, in the early years of the text, chapters were developed primarily by North American-educated authors. Today's world of orthodontics is significantly expanded with great work being done globally, as is seen in the variety of locations listed by authors in our most prestigious journals. Tom Graber was often heard saying, "Science has no borders!" and as the initial editor of the text sought the best and the brightest to contribute, a tradition we are proud to uphold to this day. Tom was the solo editor for the first two editions published by Saunders and was joined by Brainard Swain when C.V. Mosby became the publisher of what was then the first edition of the current Mosby (now Elsevier) series. Subsequently Tom, seeking a broader expertise to aid in the development of the text, was joined in the editorial role by Robert Vanarsdall in the second edition and then Katherine Vig in the third edition followed by Lee Graber and now Greg Huang in this sixth edition. As the scope of the orthodontic specialty has grown, so has the need for added editorial background and expertise.

We write this text with the presumption that those using it will have had a basic exposure to orthodontic principles provided in dental school education both within their undergraduate dental courses and from basic orthodontic texts used in the DDS/DMD curricula. The purpose of this graduate orthodontic textbook remains the same—to provide a compendium of information from authors who are experts in specific topics that are considered important to the education of orthodontic specialists. This was the vision of Tom Graber when he wrote the first edition which met with much acclaim from educators who used it as the assigned readings for their residents. It is to these educators and the orthodontic residents of the past, present and future that we have dedicated the sixth edition.

In the writing of this text, we are acutely aware of an added need within orthodontic education. That call is to encourage current residents as well as clinicians to become part of the orthodontic specialty teaching and/or research corps for the future. This text demonstrates the broad scope of orthodontics, from basic science concepts to intricate clinical techniques as well as patient management considerations. With such breadth, the specialty provides opportunities for motivated individuals to consider becoming part- or full-time educators or research scientists (or both). We can all remember the teachers we have had who made special impact on our lives. They shared their passion for their subject, the excitement over their research and their interest in mentoring a following generation. There is much within the chapters of this text about which to have excitement and passion—as well as provide motivation for answering still vexing problems through clinical and basic science research.

What is new in the presentation of this sixth edition? The most notable changes are in how the textbook may be used. The advent of computers and advances in technology for transmitting information have resulted in transforming the methods with which we share information and teach students. These technology changes have been matched by research in education that demonstrates that there is broad variability on how individuals learn. By the fifth edition we had started to put part of the text online. This sixth edition expands that effort and the ease with which the electronic version of the text may be used by way of the Expert Consult, a feature rich eBook format for medical and dental education. The ability to have added material online also increases the opportunity for more content as discussed below. Downloading sections and chapters on to your computer, laptop or tablet—or even your phone—is now possible and widely used. Using Expert Consult, readers can access and work with the text on any platform and even communicate on material with colleagues through social media. Indeed, with the inherent convenience of an electronic format, and the opportunities for editors and authors to update material without waiting for a "new edition", the investment one makes in the purchase of the text has the potential to last long after the printed publication date. These considerations coupled with the high cost of physically printing a textbook, could result in the sixth edition being the last one offered in a printed format.

What has changed in terms of content? A lot! Fully one third of the material in this edition is completely new. In addition, chapter authors from prior editions have been joined with co-authors to further update their own material. Improvements range from the organization of the chapters and the subject material to the new chapter authors who have joined us to provide the best background for orthodontists possible within the confines of a broad-based textbook. Additional

information on the science that supports orthodontics, diagnostics and therapeutic interventions has been developed. New chapters on adjunctive treatments as well as management of the potential adverse sequelae from orthodontics have been added. We also have selected several chapters from prior editions and placed them online as "classic chapters". These represent clinical topics that had to be incorporated into other chapters to maintain the already "heavy" book in a manageable size. The material in these online-only chapters further expands the scope of the textbook, better matching the increased scope of material with which a practicing orthodontist must be knowledgeable.

Finally, the chapters within the text have been reorganized to better match the progression of subjects addressed in an orthodontic specialty residency program. Section heads provide a means by which educators and residents can better identify subject material as they move through the material. Equally important, the re-organization provides the practicing clinician with a logical grouping of subjects that can be efficiently referenced. As editors, we hope that the challenge of presenting added material to residents and practitioners is made easier by way of the improved organization of material within the text.

PART ONE

Foundations of Orthodontics

Chapter 1: Craniofacial Growth and Development: Developing a Perspective. In the opening chapter of the text, authors David Carlson and Peter Buschang provide an up-to-date discussion of craniofacial growth and development. They review the basic anatomical and functional structures within the craniofacial complex, and starting with pre-natal development, describe the complex interrelationships influencing form and function. In describing the importance of the subject material, they aptly state, "…knowledge of how the craniofacial complex develops and grows provides the foundation for understanding the etiology of the various dental and skeletal malocclusions, the best of all possible treatment approaches, and how patients might be expected to respond after treatment."

Chapter 2: Genetics and Orthodontics. James Hartsfield, joined in this edition by Lori Ann Morford, has further developed basic principles of genetics while focusing on aspects that directly affect orthodontic clinicians. Clinical concerns including aberrant facial growth and development, tooth agenesis and size variability, dental eruption problems and tissue response to orthodontic forces all are impacted by genetic factors. The interaction between environmental factors and genetic expression determine an individual's response to treatment interventions. The authors complete their chapter with a look to the future and the general movement toward "personalized" medical and dental treatment.

Chapter 3: The Biologic Basis of Orthodontics: Tissue Reactions in Orthodontics. Birgit Thilander builds on the contributions of prior authors of this chapter, Kaare Reitan and Per Rygh. There is a review of the tissue components involved in tooth movement followed by a detailed discussion of the response of those tissues to various forces. The local tissue consequence of various types of tooth movement are discussed and illustrated as well as the long-term tissue changes that take place during "retention." A review of the temporomandibular joint underscores the importance of considering tissue reactions in locations distant from orthodontic force application.

In Tooth Movement at the Cellular and Molecular Level, Zongyang Sun and Nan Hatch provide a new chapter segment that focuses on the most current understanding of the control of tooth movement, that is, cellular and molecular changes in response to physiologic as well as orthodontic forces. The means by which external forces are "seen" by cells in order to stimulate tooth movement are discussed in terms of the biologic signaling mechanisms involved. The tissue changes outlined in the first section of this chapter are further described here in terms of components of mechanobiology. There is also a discussion on how biomedicine is opening doors to future patient-specific treatment alternatives.

Chapter 4: Bone Physiology, Metabolism, and Biomechanics in Orthodontic Practice. Eugene Roberts has now been joined by Sarandeep Huja to rewrite this chapter focusing on craniofacial osteology, the dynamics of bone physiology and the impact on orthodontic treatment. In addition, there is detailed discussion on osteoblast histogenesis (bone formation), osteoclast recruitment (bone resorption), and how this interplay affects tissue response to tooth-moving forces. Bone adaptations to temporary anchorage devices are reviewed as well as those aspects that play in favor of retention of mini- screws versus early loss. Finally, the authors address how it is possible to increase the rates of tooth movement by altering the local environment.

Chapter 5: Application of Bioengineering to Clinical Orthodontics. The late Charles Burstone completed the rewrite of this chapter shortly before he died. The recognized preeminent author on orthodontic biomechanics, Dr. Burstone brings together physics, mathematics and engineering to provide the theoretical background for orthodontic appliance construction and manipulation. Specific topics include discussions of the biomechanics of tooth movement (centers of rotation, force magnitude, optimal force and stress), orthodontic appliance components, mechanical properties of wires, selection of proper wires, influence of wire size-length-cross-section, archwire design and the role of friction. No matter what appliance the orthodontic clinician decides to use, it is these shared physical principles that affect the biomechanical success and efficiency of treatment.

Chapter 6: Clinically Relevant Aspects of Dental Materials Science in Orthodontics. Theodore Eliades is joined in this edition by Iosif Sifakakis to discuss the important considerations for the everyday materials in use by the clinical orthodontist. What are the characteristics of different brackets and wire materials, and how do they influence orthodontic mechanotherapy? What are the important clinical concerns with bracket bonding materials, and what might prove adverse to the patient if not properly managed by the orthodontist? What are the considerations in longterm fixed retainer wear, and what wire should a clinician use? These questions and other practical issues are answered within this chapter.

Chapter 7: The Role of Evidence in Orthodontics. One cannot attend a dental or medical meeting without some reference to "evidence-based" practice. The early years of orthodontics were characterized by "gurus" espousing one concept or another without much call for support. "Expert opinion" determined what clinicians in private practice would use for patient care. Today's clinicians are faced with the imperative to use a balanced approach to clinical decision making. Matching findings from respected research with the one's experience and patient goals is a challenge to all practitioners. We all have hopes of providing "the best of care" and also need to respect

potential medicolegal issues as we treat patients. David Turpin and Greg Huang discuss how today's clinicians can evaluate the broad range of relevant information from multiple sources and be biased to make better decisions by way of the best evidence available.

PART TWO

Diagnosis and Treatment Planning

Chapter 8: The Decision Making Process in Orthodontics. William Proffit and Tung Nguyen have restructured this chapter to better fit with the background of entering orthodontic residents and seasoned clinicians in orthodontic specialty practice. The focus is on the process of diagnosis and treatment planning, providing a method to systematically evaluate patient information and organize it in a way that helps to avoid errors of omission. Using a problem oriented approach, the authors demonstrate how (1) excellent diagnostic records, (2) a thorough case evaluation and (3) a priority-based listing of findings form the basis of an appropriate therapeutic plan.

Chapter 9: Special Considerations in Diagnosis and Treatment Planning. We are all aware of parent and patient expectations for improved cosmetics after orthodontics. Indeed, very few patients have an appreciation for functional considerations, but they are able to point to a specific tooth out of alignment or an uneven smile. David Sarver dissects the esthetic evaluation of the face and provides a broader context for patient evaluation than what has historically been considered "orthodontic diagnosis". His protocols for the evaluation of facial proportions (macroesthetics), evaluation of the smile (miniesthetics), and evaluation of the teeth and surrounding gingiva (microesthetics) are detailed within this chapter. Societal pressures create the desire for optimal dentofacial esthetics and it falls to the orthodontist to make a thorough evaluation of these characteristics on behalf of each patient.

Chapter 10: Psychological Aspects of Diagnosis and Treatment. This chapter by Leslie Will provides information on the psychological assessment of patients and how it can be accomplished within the context of an orthodontic evaluation. It describes psychological disorders and how clinicians can best manage affected patients. Patients with craniofacial anomalies present a subset of those seen in most clinical practices, but these "special needs" patients require more than an understanding of their physical challenges. The chapter has been expanded to include important psychological considerations for orthognathic surgery patients and the effect of abrupt facial change.

Chapter 11: Orthodontic Diagnosis and Treatment Planning with Cone Beam Computed Tomography (CBCT) Imaging. This is a new chapter and reflects the increasing use of CBCT in orthodontics. CBCT has been available for quite a while, with increased utilization in routine orthodontics over the past 10 years related to improved availability and reduced patient radiation dosages. Our patients see and all of us work in a three dimensional world. One can readily understand the appeal of 3D imaging to patients and clinicians alike. Authors Lucia Cevidanes, Erika Benavides, John Ludlow and Antonia Ruellas have internationally recognized craniofacial imaging expertise. They have joined to provide an outstanding discussion on how CBCT may be used to enhance orthodontic diagnosis and treatment.

Chapter 12: Upper Airway, Cranial Morphology, and Sleep Apnea. As an additional new chapter we have added information in regards to evaluating patients for morphologic and functional traits that may predispose to disturbed sleep syndromes. Juan Martin Palomo, Hakan El, Leena Palomo, and Kingman P. Strohl provide an in-depth discussion on identifying airway problems, and a thorough review of sleep disordered breathing and the classification of associated clinical problems. They discuss how an orthodontist might influence airway patency through various treatment modalities. The authors underscore how the orthodontist fits in to a multi-disciplinary team to best manage these multifactored patients.

Chapter 13: Orthodontic Therapy and the Patient with Temporomandibular Disorder. Jeffrey Okeson is the internationally recognized authority on TMD disorders and especially how these disorders potentially impact orthodontic patients before, during and after treatment. In this updated chapter he reviews the basic concepts of orthopedic stability, how to perform a complete TMD evaluation, and how to develop a treatment plan. His discussion of a stepwise approach for TMD problems that may arise during treatment is a must read for residents and practitioners.

Chapter 14: The Orthodontist's Role in a Cleft Palate–Craniofacial Team. Katherine Vig and Ana Mercado review the important considerations for orthodontists who are treating craniofacial anomalies as part of craniofacial teams. The emphasis is on an orthodontist as a component of "the team" and the appropriate timing for various interventions for the cleft patient. New to this edition is an excellent discussion of the clinically vexing issue of how to approach the missing maxillary lateral incisor in patients with cleft palate. The authors do an excellent job of emphasizing the long term relationship with craniofacial patients and how the clinician must recognize the multi-stage needs of these patients.

PART THREE

Mixed Dentition Diagnosis and Treatment

Chapter 15: Patient Management and Motivation for the Child and Adolescent Patient. How does one create an office environment that has patients who are happy and appreciate the services provided? Patrick and Patricia Turley bring information from the pediatric dental literature as well as patient management sphere to develop this new chapter for the sixth edition. The theme of the chapter is how to best guide young patients to comply with instructions both in the office setting and at home. The Achilles heel for orthodontic treatment is most often patient cooperation. These authors present guidance on how best to communicate to patients at different ages and how to encourage compliance with home care responsibilities for hygiene as well as appliance wear. They point out that clinicians need to have a systematic approach for monitoring patient cooperation as well as routine measures to intervene when compliance is lacking.

Chapter 16: Optimizing Orthodontics and Dentofacial Orthopedics: Treatment Timing and Mixed Dentition Therapy. For an orthodontist, the opportunity to intervene and reduce the severity of a developing malocclusion needs to be recognized. Additionally, guidance that can improve the maxilla–mandibular relationships and space for erupting permanent teeth can provide significant benefit to growing patients. The questions for the clinician revolve around when and how one should intervene in a growing child. In this chapter, authors James McNamara, Laurie McNamara McClatchey, and Lee Graber provide an organized approach to mixed

dentition diagnosis and treatment. With the broad spectrum of malocclusions that present to the practitioner, there are an equally varied considerations for treatment timing and approach. There is a brief discussion of serial extraction technique within this chapter, but readers are encouraged to see Chapter 34, Interceptive Guidance of Occlusion with Emphasis on Diagnosis (online only) for further information. Chapter 16 provides information on the proper age for dentofacial orthopedic–orthodontic intervention, appliance construction and management. The appliance types and treatment protocols presented in the chapter are evidence based, but it is understood that there are many types of orthodontic appliances that can be selected as long as the underlying principles of diagnosis, appropriate timing and orthopedic–orthodontic therapy are maintained.

PART FOUR

Orthodontic Treatment

Chapter 17: Contemporary Straightwire Biomechanics. Since the straightwire appliance was introduced in the 1970s its use has grown to where it is now the most popular fixed orthodontic appliance design available today. In this chapter, Antonino Secchi and Jorge Ayala provide (1) the historical basis of the appliance, (2) the principles of precise bracket placement and (3) the three separate phases of straightwire mechanics. With excellent clinical cases and explanations, they detail the specific wire sequences, anchorage considerations, and finishing mechanics designed to optimize the benefits of the straightwire bracket system.

Chapter 18: Nonextraction Treatment. Orthodontists are faced everyday with decisions of whether or not they need to remove permanent teeth as part of a comprehensive treatment program. Our journals have documented the trends to more extraction or fewer extractions with a reduced rate of extractions currently in vogue. Realistically, these extraction decisions should be made on the basis of a solid diagnosis and evidence-based treatment planning. With this as a given, what techniques can be used to maximize the success of a non-extraction approach in a borderline patient? This chapter authored by Robert Vanarsdall and Raffaele Spena details an approach that can provide needed dental arch space in a predictable fashion and provides case examples.

Chapter 19: Standard Edgewise: Tweed-Merrifield Philosophy, Diagnosis, Treatment Planning and Force Systems. For many years, orthodontic residents and clinicians have traveled to Tuscon, Arizona to take the "Tweed Course," the oldest dental continuing education program in the world. This chapter is written by James Vaden and Herbert Klontz, co-directors of the Tweed Foundation, along with the late Jack Dale. It provides an historical perspective on the development of the Tweed technique, presents diagnostic and treatment concepts, and describes the Tweed-Merrifield edgewise appliance. The authors review the steps of the Tweed technique as currently practiced and present excellent illustrative case material.

Chapter 20: Biomechanical Considerations with Temporary Anchorage Devices (TADs). The use of temporary anchorage devices has greatly increased the ability of the clinical orthodontist to move teeth efficiently without the side effects of reciprocal undesired moments of force. "Absolute anchorage" as provided by these adjuncts provides a platform from which the clinician can better move teeth in all three planes of space. They have literally increased the range of orthodontic mechanotherapy. Authors Jong Suk Lee, Jung Kook Kim and Young-Chel Park have combined their internationally recognized expertise to provide a discussion of the biomechanical considerations when using TADs.

Chapter 21: Adult Interdisciplinary Therapy: Diagnosis and Treatment. Adult orthodontic treatment has continued to increase as a percentage of the patients seen by orthodontic specialists. There are many reasons including the ability to use "esthetic" treatment appliances, the increased interest in adults in their long term dental health, and the improved understanding within the general dental community that orthodontics can be an integral part of an overall restorative plan. Robert Vanarsdall and David Musich discuss the added considerations when treating adult patients. Treatment criteria differ, and there is concern for adverse tissue response especially where compromised tissue relationships are present before the start of orthodontics. This chapter reviews adult orthodontic diagnostic and treatment regimens, limitations in adult care, and compromises that must be made as well as the special demands for long term retention. Case reports provide excellent reference material for the subject.

Chapter 22: Periodontal-Orthodontic Interrelationships. As we see more adults entering comprehensive orthodontic treatment, we must be more attuned to the implications of periodontal issues. In this updated chapter, Robert Vanarsdall, Ignacio Blasi and Antonino Secchi review periodontal issues that impact orthodontic tooth movement. They describe periodontal "high risk" factors, mucogingival considerations, and problems with ectopic as well as ankylosed teeth. A new section on alveolar decortication and augmentation grafting has been added to address the increased use of these procedures designed to develop the alveolar housing and potentially increase the speed of tooth movement. Excellent clinical examples are pictured throughout the chapter.

Chapter 23: Orthodontic Aspects of Orthognathic Surgery. David Musich and Peter Chemello discuss the added considerations for patients who present with treatment needs that exceed the potential for orthodontic mechanotherapy alone. Diagnostic considerations and a systematic protocol are presented as a guide for orthodontists and oral and maxillofacial surgeons teaming up to provide coordinated surgical care. What are the potentials of surgical orthodontics? What are the problems and pitfalls that must be addressed? What are the timing issues when considering jaw surgery? What are the risks of treatment, and of course, how can the clinician help to stabilize results with long term retention? These questions and more are addressed within this chapter.

Chapter 24: Self-Ligating Bracket Biomechanics. The increased use of self-ligating brackets has brought forth many discussions at orthodontic meetings, articles both for and against their use, and direct-to-consumer marketing that has resulted in more questions from patients as they interact with their orthodontists. This chapter authored by Nigel Harradine provides an historical perspective on self-ligating techniques and proceeds to review the advantages and disadvantages of these appliances. Although some of the advertised claims for reduced treatment times may not have been demonstrated in systematic reviews of the technique, there do appear to be positive indications for clinicians. This chapter provides an unbiased view of current findings and makes specific recommendations for the use of these brackets.

Chapter 25: Lingual Appliance Treatment. Lingual appliance treatment has had more popularity in Europe and Asia than in North America. That may change as increasingly more clinicians adopt partial or full fixed lingual appliance treatment in their offices. This trend has been greatly aided by the integration of three dimensional modeling, the development of custom braces and/or robotically bent archwires, and increased call of "invisible" treatment by adult and adolescent patients. In addition, there is an added benefit of minimal risk of labial surface decalcification—scars seen from labial therapy— with lingual bracket treatment. This chapter has been completely re-written and is co-authored by Dirk Weichmann and Dan Grauer. Although the authors focus on their own lingual appliance type, the principles they discuss can be generalized to a number of lingual treatment systems.

Chapter 26: Clear Aligner Treatment. This chapter has been re-written by three of the most knowledgeable clinicians in the use of clear aligners, David Paquette, Clark Colville and Timothy Wheeler. The chapter focuses on the Invisalign system, the most researched and used clear aligner system in the world. The authors discuss the unique aspects of diagnosis and treatment planning for clear aligners. The continuing development of adjuncts within the aligner system has increased the type and range of tooth movement that can be accomplished. Significant research has been reported and provides a basis for discussion of what movements can be accomplished more easily—and what becomes more difficult with aligners. This comprehensive chapter provides critical considerations that distinguish treatment provided by an orthodontic specialist from the clinician who has less of a background.

Chapter 27: Bonding in Orthodontics. The premier authority on orthodontic bonding, Bjorn Zachrisson is joined in this updated chapter by Serdar Usumez and Tamer Büyükyilmaz. Adhesive dentistry is the norm for restorative clinicians, but direct and indirect bonding of brackets to enamel and restorative materials for orthodontics is equally important. This detailed chapter reviews patient preparation for bonding, techniques for the application of brackets to teeth and artificial surfaces, indirect bonding protocols, re-bonding considerations and the use of bonded lingual retainers. Additionally there is a discussion of the use of microabrasion and resin infiltration to repair decalcified enamel areas. The technical procedures are well documented with clinical pictures.

PART FIVE

Specialized Treatment Considerations

Chapter 28: Management of Impactions. This is a totally new chapter designed to aid clinicians in developing a diagnostic and therapeutic protocol for impacted teeth. Adrian Becker and Stella Chaushu are the internationally respected leaders in this important area of orthodontic diagnosis and treatment. The chapter reviews the etiology of impactions, the prevalence of impactions of specific teeth, the assessment of impactions and finally the therapeutic interventions that are required of the clinician. The authors discuss areas for potential failure in management of impactions and provide clinical suggestions to improve success. The chapter is well illustrated in support of the recommended clinical procedures.

Chapter 29: Management of Dental Luxation and Avulsion Injuries in the Permanent Dentition. Patrick Turley has provided a new chapter to cover an aspect of practice that gets little attention in orthodontics, the management of dental trauma. An orthodontists is often the first to see a youngster who has had teeth displaced by way of accident—often because this is the dental office the family has seen most often during the adolescent years. The addition of this chapter fills a prior void by addressing the sequelae of trauma to the teeth, acute management of injuries to the teeth and alveolar structures, and treatment of avulsed teeth. The author also provides information on the importance of treatment of overly procumbent teeth to reduce the risk of incisal fractures. This is information that parents in addition to clinicians need to know!

Chapter 30: Iatrogenic Effects of Orthodontic Treatment: Prevention and Management of Demineralized White Lesions. This is another new subject area addressed in an added chapter for the sixth edition. Well known clinical researchers and authors Philip Benson and Norah Flannigan review the potential for adverse effects of orthodontic appliance wear on labial surfaces that have bonded attachments. The most common adverse effect is the development of white spot lesions. The authors review the prevalence of the problem, means by which it may be diagnosed and most importantly, how these lesions can often be prevented.

In Root Resorption, Glenn Sameshima and M. Ali Darendeliler look at the problem of reduced root length during tooth movement. What are patient and mechanotherapeutic predisposing factors for root resorption? What should the clinician do when root resorption is expected or detected? What is the long term prognosis for teeth with foreshortened roots? All clinicians see these problems in daily practice, and this chapter provides an excellent summary of clinical considerations.

Chapter 31: Minimally and Noninvasive Approaches to Accelerate Tooth Movement: Micro-osteoperforations. Patients are always attracted to decreasing the time it takes to complete orthodontic treatment. Recent advances in the understating of bone biology have been applied to a number of adjunctive techniques that in certain situations may reduce the time for orthodontics. In this new chapter, Ignacio Blasi looks at the use of micro-osteoperforations and the ability of these perforations to increase bone turnover rates, providing for increased speed of tooth movement. Studies have been done that demonstrate a localized positive effect on the speed of tooth movement.

In Low-Level Mechanical Vibrations, Dubravko Pavlin presents information on the influence of low level mechanical vibration on orthodontic treatment, a technique borrowed from orthopedic colleagues. Some of the animal and human studies have reported increased rates of tooth movement and an ability to reduce pain perception when low level vibration therapy is used. Still other studies report no clinical effectiveness. Although the jury is still out on this technique, clinical and basic science research into adjuncts that can influence biologic factors, are likely to bear fruit in the future.

Chapter 32: Biodigital Orthodontics: Integrating Technology with Diagnosis, Treatment Planning and Targeted Therapeutics. Rohit Sachdeva presents a discussion of how clinical decision making should be accomplished, with enhanced integration of digital technologies. Although this chapter focuses on just one of the technology systems available to orthodontists, the thought process that underlies the use of these technologies is shared. These technologies are tools and only serve to enhance the principles of sound diagnosis and treatment planning. They can make good clinicians better, but their sophistication precludes the ability for poorly educated dental

practitioners to use them properly. Benefits to dental specialists can be significant with improved abilities at both diagnostic evaluation and treatment planning as well as, in some instances, the fabrication of appliances. These technologies gain their benefit in reducing the "error of the method" of conventional orthodontics.

PART SIX

Orthodontic Retention and Post-Treatment Changes

Chapter 33: Stability, Retention, and Relapse. Don Joondeph is joined in this updated chapter by colleagues Greg Huang and Bob Little, all respected clinicians with a wealth of research material on post-orthodontic treatment changes, developed over many years at the University of Washington. The authors outline a series of problems routinely addressed by orthodontic treatment. With evidenced-based suggestions, they provide important clinical recommendations to reduce adverse post-active treatment changes that take place over time. "Maturational change" to varying degrees is the norm, but patients often have an unrealistic expectation that their teeth will "stay perfect" after orthodontics. Perfection remains a goal, but the structural and functional environment within which the dentition exists over time makes excellent retention planning a "must" to preserve treatment accomplishments and long term patient satisfaction. The authors provide information gleaned from the UW retention studies as a basis for their clinical recommendations.

PART SEVEN

Classic Chapters (online only)

Chapter 34: Interceptive Guidance of Occlusion with Emphasis on Diagnosis. Although the general topic has been reviewed within Chapter 16, this classic chapter from Jack and Hali Dale provides more in depth background on the diagnosis of space problems in the mixed dentition and associated interceptive treatment modalities. Diagrams representing the stages of tooth eruption are coordinated with photographic case documentation of a variety of malocclusions secondary to space problems. Interception of crowding through a series of staged clinical decisions is demonstrated. Guided tooth removal is presented as an option for interceptive orthodontics, with goals for decreased time in fixed orthodontic mechanotherapy.

Chapter 35: Functional Appliances. The use of functional appliances remains high world-wide and the material herein provides background important to orthodontic specialist clinicians. This chapter was authored by the late Tom Graber, the original author and editor of the textbook. The goal of the chapter (originally published in the 4th edition) is to provide a basic understanding of how functional appliances work as well as a discuss the attributes of the most widely used designs. Although the topic is briefly addressed in Chapter 16, this specific chapter focuses more in detail on functional appliance construction and clinical management. The author reviews the mechanism of Class II correction with functional jaw orthopedics, compares fixed with removable functional appliance designs and relates how clinicians may concurrently use functional jaw orthopedics with fixed bracket treatment. The chapter is well illustrated with clinical case material and diagrammatic representations of expected treatment results. Orthodontic residents and experienced clinicians alike will gain much from this thorough review of functional jaw orthopedics.

Chapter 36: Treatment of the Face with Biocompatible Orthodontics. This "classic chapter" by Dwight Damon reviews diagnosis and treatment planning protocols used with one of the most popular fixed appliance clinical techniques in use today. Chapter 24 appropriately discusses the general topic of self-ligation brackets, but this chapter on the specific use of the Damon technique has been one of the most requested since its original publication in the fourth edition. The author uses a series of case studies to demonstrate diagnostic considerations and treatment of a variety of malocclusion types. Although the bracket design has improved since the original publication of this chapter, the principles behind the treatment remain the same and are applicable to the self-ligating bracket systems currently available.

In re-writing and presenting this sixth edition of *Orthodontics: Current Principles and Techniques* we are well aware that an undertaking of this sort requires help from many sources. We are thankful for the great work of our authors, those who have been with us before and the outstanding colleagues who have joined them. Our authors have been pressed by us as well as our publisher to succinctly provide their best material. They have done so admirably with the result that we have succeeded in giving our educators and residents a broad based textbook that provides the foundational knowledge for an orthodontic specialist. We have dedicated this sixth edition of the text to them and to the degree that we have made their teaching and learning efforts easier, we are very happy!

This text revision would never have been completed without an outstanding editorial team at Elsevier. Thanks go to Kathy Falk, Laura Klein, Anne Schook, Srividhya Vidhyashankar and all of their supporting staff. As in any large project of this type, problems arise. It is these folks who went above and beyond their normal work hours and assignments to overcome challenging issues and keep us on track. The quality of production was enhanced over prior editions in part because of improved editing and printing technologies, but most of all because of the diligence of the Elsevier personnel.

In this edition of the text, we added a new editor, Greg Huang, chair of the orthodontic department at the University of Washington. His strong contributions were important in many seen and unseen ways. All of us as editors have shared an excitement for the educational mission of the text, one that carries over from the first volume edited by Tom Graber more than 45 years ago. We are thrilled that we can share the expertise of our authors with our colleagues. We remain personally positive and passionate about the orthodontic specialty. Our hope is that the enthusiasm that we share for orthodontics comes through the pages of the text and helps to motivate a new generation of educators, residents and practitioners.

Lee W. Graber, DDS, MS, MS, PhD
Robert L. Vanarsdall, DDS
Katherine W.L. Vig, BDS, MS, D Orth, FDS, RCS
Greg J. Huang, DMD, MSD, MPH

CONTENTS

PART ONE Foundations of Orthodontics

1 **Craniofacial Growth and Development: Developing a Perspective**, 1
David S. Carlson and Peter H. Buschang
2 **Genetics and Orthodontics**, 31
James Kennedy Hartsfield, Jr., and Lorri Ann Morford
3 **The Biologic Basis of Orthodontics**, 51
Birgit Thilander, Nan Hatch, and Zongyang Sun
Tissue Reactions in Orthodontics, 51
Birgit Thilander
Tooth Movement at the Cellular and Molecular Level, 85
Nan Hatch and Zongyang Sun
4 **Bone Physiology, Metabolism, and Biomechanics in Orthodontic Practice**, 99
Wilbur Eugene Roberts and Sarandeep Singh Huja
5 **Application of Bioengineering to Clinical Orthodontics**, 154
Charles J. Burstone
6 **Clinically Relevant Aspects of Dental Materials Science in Orthodontics**, 187
Theodore Eliades and Iosif Sifakakis
7 **The Role of Evidence in Orthodontics**, 200
David L. Turpin and Greg Huang

PART TWO Diagnosis and Treatment Planning

8 **The Decision-Making Process in Orthodontics**, 208
Tung Nguyen and William Proffit
9 **Special Considerations in Diagnosis and Treatment Planning**, 245
David M. Sarver
10 **Psychological Aspects of Diagnosis and Treatment** , 289
Leslie A. Will
11 **Orthodontic Diagnosis and Treatment Planning with Cone-Beam Computed Tomography Imaging**, 302
Lucia Cevidanes, Erika Benavides, John B. Ludlow, and Antonio Carlos de Oliveira Ruellas
12 **Upper Airway, Cranial Morphology, and Sleep Apnea**, 319
Juan Martin Palomo, Hakan El, Leena Palomo, and Kingman P. Strohl
13 **Orthodontic Therapy and the Patient with Temporomandibular Disorder**, 353
Jeffery P. Okeson
14 **The Orthodontist's Role in a Cleft Palate–Craniofacial Team**, 367
Katherine W.L. Vig and Ana M. Mercado

PART THREE Mixed Dentition Diagnosis and Treatment

15 **Patient Management and Motivation for the Child and Adolescent Patient**, 395
Patrick Turley and Patricia Turley
16 **Optimizing Orthodontics and Dentofacial Orthopedics: Treatment Timing and Mixed Dentition Therapy**, 403
James A. McNamara, Laurie McNamara McClatchey, and Lee W. Graber

PART FOUR Orthodontic Treatment

17 **Contemporary Straight Wire Biomechanics**, 437
Antonino G. Secchi and Jorge Ayala Puente
18 **Nonextraction Treatment**, 455
Robert L. Vanarsdall, Jr., and Raffaele Spena
19 **Standard Edgewise: Tweed-Merrifield Philosophy, Diagnosis, Treatment Planning, and Force Systems**, 481
James L. Vaden, Herbert A. Klontz, and Jack G. Dale
20 **Biomechanical Considerations with Temporary Anchorage Devices**, 511
Jong Suk Lee, Jung Kook Kim, and Young-Chel Park
21 **Adult Interdisciplinary Therapy: Diagnosis and Treatment**, 569
Robert L. Vanarsdall, Jr., and David R. Musich
22 **Periodontal–Orthodontic Interrelationships**, 621
Robert L. Vanarsdall, Jr., Ignacio Blasi, Jr., and Antonino G. Secchi
23 **Orthodontic Aspects of Orthognathic Surgery**, 669
David R. Musich and Peter Chemello
24 **Self-Ligating Bracket Biomechanics**, 729
Nigel Harradine
25 **Lingual Appliance Treatment**, 761
Dirk Wiechmann and Dan Grauer
26 **Clear Aligner Treatment**, 778
David E. Paquette, Clark Colville, and Timothy Wheeler

PART FIVE Specialized Treatment Considerations

27 **Bonding in Orthodontics**, 812
Björn U. Zachrisson, Serdar Usumez, and Tamer Büyükyilmaz
28 **Management of Impactions**, 868
Adrian Becker and Stella Chaushu
29 **Management of Dental Luxation and Avulsion Injuries in the Permanent Dentition**, 880
Patrick Turley
30 **Iatrogenic Effects of Orthodontic Treatment**, 887
Philip Edward Benson, Norah Lisa Flannigan, Glenn Sameshima, and M. Ali Darendeliler
Prevention and Management of Demineralized White Lesions, 887
Philip Edward Benson and Norah Lisa Flannigan
Root Resorption, 896
Glenn Sameshima and M. Ali Darendeliler
31 **Minimally and Noninvasive Approaches to Accelerate Tooth Movement**, 913
Ignacio Blasi, Jr., and Dubravko Pavlin
Micro-Osteoperforations, 913
Ignacio Blasi Jr.
Low-Level Mechanical Vibrations, 918
Dubravko Pavlin
32 **Biodigital Orthodontics: Integrating Technology with Diagnosis, Treatment Planning, and Targeted Therapeutics**, 926
Rohit ChamanLal Sachdeva

PART SIX Orthodontic Retention and Posttreatment Changes

33 Stability, Retention, and Relapse, 981
Donald R. Joondeph, Greg Huang, and Robert Little

PART SEVEN Classic Chapters (Online Only)

34 Interceptive Guidance of Occlusion with Emphasis on Diagnosis, 997.e1
Jack G. Dale and Hali C. Dale
35 Functional Appliances, 997.e50
Thomas M. Graber
36 Treatment of the Face with Biocompatible Orthodontics, 997.e96
Dwight Damon

This page intentionally left blank

1

Craniofacial Growth and Development: Developing a Perspective

David S. Carlson and Peter H. Buschang

OUTLINE

Introduction, 1
Somatic Growth, 1
Differential Development and Maturation, 2
Variation in Rates of Growth during Maturation, 2
Craniofacial Complex, 3
Structural Units, 3
Desmocranium, 3
Chondrocranium, 4
Viscerocranium, 4
Dentition, 4
Functional Units, 4
Neurocranium, 4
Face, 4
Oral Apparatus, 4
Molecular Basis of Craniofacial Development and Growth, 5
Cranial Vault, 5
Development of the Cranial Vault, 5
Mechanisms of Suture Growth, 6
Postnatal Growth of the Cranial Vault, 7
Cranial Base, 8
Development of the Cranial Base, 8
Mechanism of Synchondrosal Growth, 8
Postnatal Growth of the Cranial Base, 10
Midface/Nasomaxillary Complex, 12
Development of the Midface, 12
Postnatal Growth of the Midface, 13
Mandible, 16
Development of the Mandible, 16
Growth of the Mandibular Condyle, 18
Histomorphology of the Growing Condyle, 18
Age-Related Changes in the Mandibular Condyle, 19
Mechanisms of Condylar Growth, 19
Postnatal Growth of the Mandible, 20
Arch Development, Tooth Migration, and Eruption, 23
Adult Changes in Craniofacial Form, 25
Postnatal Interrelationships during Craniofacial Growth, 25
Significance of Understanding Craniofacial Growth for Orthodontics, 27

This chapter is enhanced with the following electronic assets at www.expertconsult.com: 2 tables.

INTRODUCTION

An appreciation of the biological principles associated with growth and development, especially of the structures composing the craniofacial complex, is essential for attaining competency within the field of orthodontics. Particular emphasis for the advanced practice of orthodontics is placed on the hard tissues comprising the craniofacial regions, i.e., the skeletal structures and the teeth, because these are the primary components of the craniofacial complex that the orthodontist addresses during treatment. Development, growth, and function of other craniofacial structures and tissues, such as muscles, neural tissues, and pharyngeal structures, as well as spaces such as the airway, are also of major interest to orthodontists. However, those elements are important primarily in terms of their influence—structurally, functionally, and developmentally—on the growth, size, and form of the skeletal elements of the face and jaws.

This chapter emphasizes postnatal growth, principally of the skeletal structures of the craniofacial complex, because of its importance in orthodontic treatment. Considerable attention is also given to prenatal development of craniofacial tissues and structures because it is critical for understanding postnatal growth. The reader is referred to a number of excellent references on developmental biology and human embryology for comprehensive reviews of early craniofacial development.[1,2]

SOMATIC GROWTH

The size and form of the craniofacial complex are major components of an individual's overall body structure. Moreover, the growth and maturation of the body as a whole, referred to generally as *somatic growth*, are highly correlated with those of the craniofacial complex. Therefore, clinical evaluation of the status and potential for craniofacial growth, and thus of treatment

planning in orthodontic patients, is highly dependent on an understanding of the somatic growth process.[3]

Differential Development and Maturation

In his classic work during the 1930s, Scammon[4] drew attention to the fact that the rate and timing of postnatal maturation, measured as a proportion of total adult size, vary widely among major systems of the human body (Fig. 1-1). In what has become known as "Scammon's curve," for example, maturation of the central nervous system is shown to be completed primarily during the last trimester of gestation through age 3 to 6 years. As a result, the cranial vault, which houses the precociously developing and enlarging brain, is disproportionately large in the infant relative to the rest of the craniofacial region (Fig. 1-2). In contrast, the reproductive organs become mature a decade later, during adolescence.

The rate of general somatic growth and development, which includes the skeletal and muscular systems, is characterized by an S-shaped curve. The relative rate of growth is very high prenatally but then decreases during infancy and becomes even slower during childhood. The rate then accelerates greatly with the initiation of adolescence through the point of peak growth velocity, after which it slows once again and effectively stops altogether in adulthood. Development and growth of the craniofacial complex is intergraded between neural and somatic maturity patterns. The gradient moves from the cranium, which is the most mature, through the anterior cranial base, posterior cranial base and maxillary length, upper face height, corpus length, to ramus height, which is the least mature and most closely approximates the general S-shaped pattern of general somatic maturation.[5]

Overall somatic growth, including the onset and end of puberty, is coordinated throughout the body by sex hormones and growth factors that are expressed differentially during the first two decades of postnatal life. However, the timing, rate, and amount of secretion of endocrine factors vary significantly between males and females and within each sex relative to chronologic age.

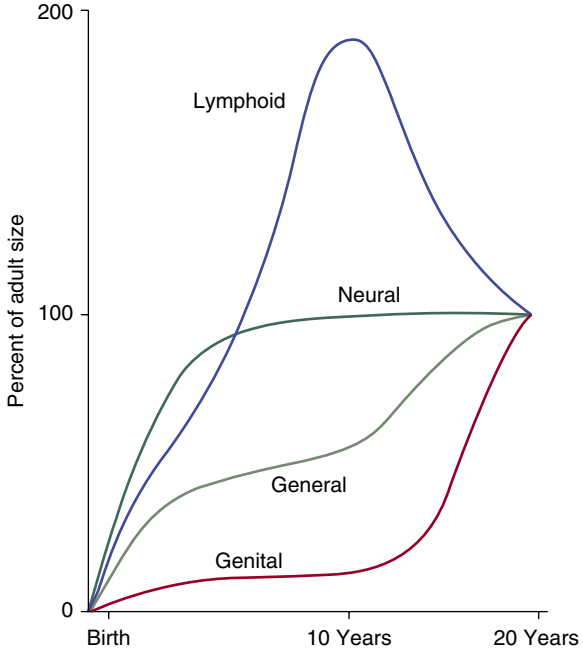


FIGURE 1-1 *Scammon's curve* illustrating the fact that different systems of the body have different rates of development and come to maturity at different ages.

Variation in Rates of Growth during Maturation

Two episodes of relatively rapid growth, or growth spurts, have been documented for both general somatic and craniofacial growth. The lesser of these, the mid-childhood spurt, takes place in approximately 50% of children between 6.5 and 8.5 years of age. The mid–growth spurt tends to occur more frequently and approximately 1 year later for boys than girls.[6] The more prominent adolescent growth spurt begins with the onset of puberty, at approximately 9 to 10 years of age in females and 11 to 12 years in males (Fig. 1-3). Female and male peak height velocities (PHV) are attained on average at 12 and 14 years of age, respectively, for North Americans and Europeans.[7] Females complete adolescence approximately 2 or more years ahead of males. The extra years of childhood growth prior to adolescence in males, as well as the slightly greater rates of adolescent growth and the slightly lengthier adolescent period, explain most of the sex differences in overall body size as well as in craniofacial dimensions.

Because growth of craniofacial structures is correlated with general somatic growth, the timing of peak height velocity (PHV), which occurs at the pinnacle of the adolescent growth spurt, is especially useful for estimating peak maxillary and mandibular growth velocity. It has been shown that maxillary growth attains its maximum rate slightly before PHV, while the maximum rate of mandibular growth occurs just after PHV.[8,9]

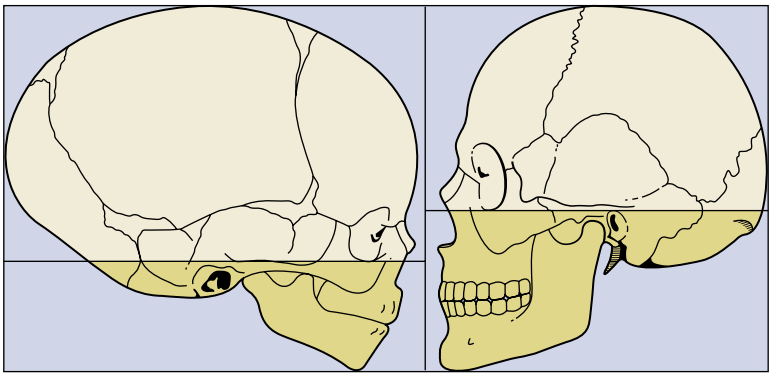


FIGURE 1-2 Disproportions of the head and face in infant and adult. The neurocranium, which houses the brain and eyes is precocious in its development and growth and therefore is proportionately larger than the face during infancy and early childhood. (Adapted from Lowry GH. *Growth and Development of Children.* 6th ed. Chicago: Year Book Medical Publishers; 1973.)

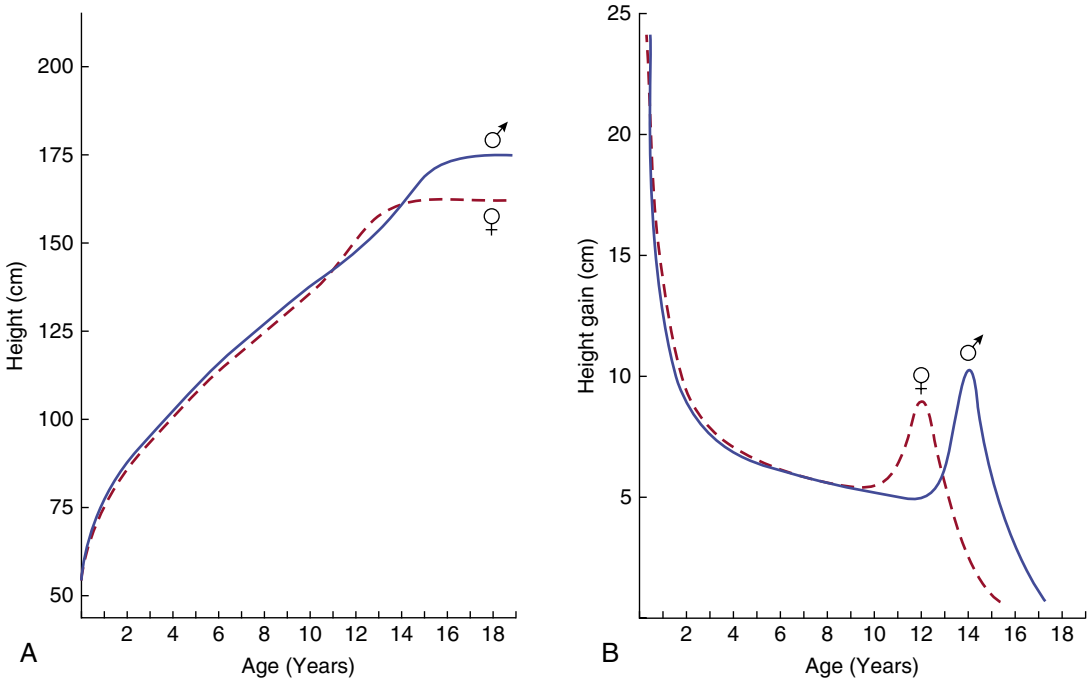


FIGURE 1-3 Growth velocity curve (growth per unit of time) for skeletal growth as general measure of human ontogeny. Velocity of growth is characterized by decrease in growth rate beginning in the last trimester of prenatal development through maturation in the adult. During adolescence, hormonally mediated growth typically occurs to bring about a spurt in skeletal growth (PHV, peak height velocity). Pubertal growth spurt is characterized by considerable variability in onset and duration among individuals and according to gender. Onset of the pubertal growth spurt typically begins about age 10 in girls and lasts approximately 2 years. Boys have later onset (12 years); the entire pubertal period can last 4 to 6 years. (Adapted from Tanner JM, Whitehouse RH, and Takaishi M: Standards from birth to maturity for height, weight, height velocity and weight velocity: British children, 1965. *Arch Dis Childh* 41:454-471, 1966.)

The timing, rate, and amount of somatic growth are best determined by changes in overall height. As such, height provides an important adjunct for cephalometric evaluations, especially during periods of rapid growth. Population-specific height percentiles make it possible to individualize craniofacial assessments. For example, if an individual's rate of somatic growth is particularly high or low, it is likely that his or her rate of craniofacial growth will be similarly high or low. Height measurements are recommended because they are noninvasive, highly accurate, and simple to obtain at multiple occasions. Reference data for height are also typically based on larger samples of defined populations than are craniofacial reference data, which makes them more precise at the extreme percentiles.[10]

Assessments of maturation also provide critical information about the likelihood that the growth of craniofacial structures will continue and for how long or that growth has been completed. This is important because patients' maturational and chronologic ages should be expected to differ, often by more than 1 to 2 years, which confounds growth assessments necessary for orthodontic diagnosis and treatment planning. For this reason, it is always better to use the patient's skeletal age based on radiologic assessments of hand/wrist ossification to determine skeletal maturity, especially for determining whether the patient has entered adolescence, attained peak velocity, is past peak growth, or is near the end of clinically meaningful growth.[11,12] Cervical vertebrae maturation provides another, albeit less precise, method to determine skeletal maturity.[13] Molecular assays are now being developed to provide more sensitive assessments to determine maturational status of skeletal growth.[14]

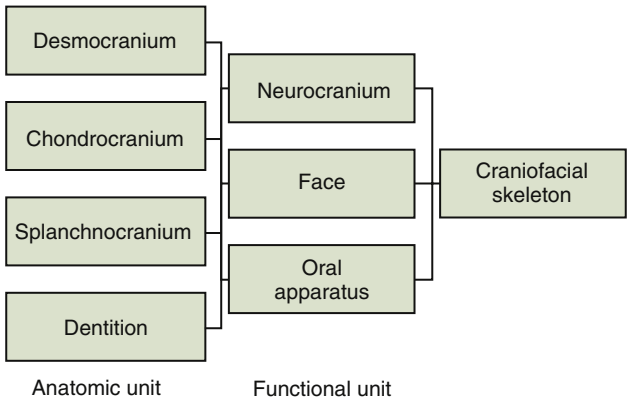


FIGURE 1-4 Schematic of organization of the craniofacial skeleton into anatomic regions and overlapping functional regions.

CRANIOFACIAL COMPLEX

The craniofacial complex is comprised of 22 separate bones that can be organized for heuristic purposes into relatively discrete anatomic and functional regions. Each of these regions has distinct mechanisms of development and growth, as well as different capacities for adaptation during growth (Fig. 1-4).

Structural Units

Desmocranium

The term *desmocranium* refers to the portion of the craniofacial skeleton that arises from a membrane of ectodermal,

mesodermal, and neural crest origin that surrounds the proximal end of the notochord very early in development. As the brain develops and expands in utero, the desmocranium develops initially as a fibrous membrane covering of the brain that eventually will give rise to the bones of the cranial vault and fibrous joints, or sutures, as well as the dura mater over the brain and the periosteum overlying the bones of the cranial vault. In fact, in the absence of a brain, as with anencephaly, the desmocranial bones will fail to develop at all. Because the skeletal derivatives of the desmocranium have exclusively a membranous precursor, initial morphogenesis and subsequent bone growth take place completely via intramembranous ossification.

Chondrocranium

The *chondrocranium* forms initially as part of the embryonic anlagen of primary cartilage that will become the cranial base, nasal septum, and nasal capsule. Like the desmocranium, the chondrocranium is also a derivative of the embryonic membrane surrounding the developing central nervous structures. However, the chondrocranium is significantly less dependent on the presence of the brain for its initial formation and subsequent development. Growth associated with the derivative bones of the cranial base occurs by means of endochondral ossification.

Viscerocranium

The *viscerocranium*, also referred to as the *splanchnocranium*, is composed of all those elements of the craniofacial complex that are derived from the first branchial arch and thus is of neural crest origin. These elements primarily include the bones of the midfacial complex and the mandible. Because the skeletal elements of the viscerocranium have no primary cartilaginous precursors, development and growth of its skeletal derivatives take place via intramembranous ossification that is also characterized by the presence of sutures and a specialized form of membrane-derived (secondary) cartilage at the mandibular condyles.

Dentition

The deciduous and permanent teeth are specialized anatomic components of the craniofacial complex that are composed of unique tissues and undergo a unique mechanism of development characterized by the interaction between ectodermal and mesenchymal tissues.

Functional Units

These four anatomic components can be combined organizationally into three overlapping and very broad functional units comprising the craniofacial complex (Fig. 1-5).

Neurocranium

The *neurocranium* houses the brain and other elements of the central nervous system, such as the olfactory apparatus and auditory apparatus. As the brain rests on the cranial base and is covered by the cranial vault, development and growth of the neurocranium are characterized by a combination of membranous (desmocranium) and cartilaginous (chondrocranium) bone growth.

Face

The upper face may be defined as the region of the orbits of the eye. The midface, comprised primarily of the maxillae and zygomatic bones, is the region between the orbits and the upper dentition. Ectocranially, the bones of the face are composed externally of the intramembranously formed bones of the viscerocranium. However, the face also receives contributions from the chondrocranium as the cartilaginous nasal capsule and nasal septum. The lower face, comprised of the mandible, develops entirely from the first branchial arch and thus is derived entirely as part of the viscerocranium. The mandible develops and grows via a specialized form of intramembranous formation of both bone and secondary cartilage.

Oral Apparatus

The oral apparatus is composed of the dentition and supporting structures within the upper and lower jaws. Thus, the oral apparatus also is characterized by a unique morphogenesis of the teeth as well as a specialized form of intramembranous bone growth of the alveolar processes of the maxilla and mandible (viscerocranium). Development and growth of the skeletal structures comprising the oral apparatus are greatly influenced by the muscles of mastication and other soft tissues associated with mastication.

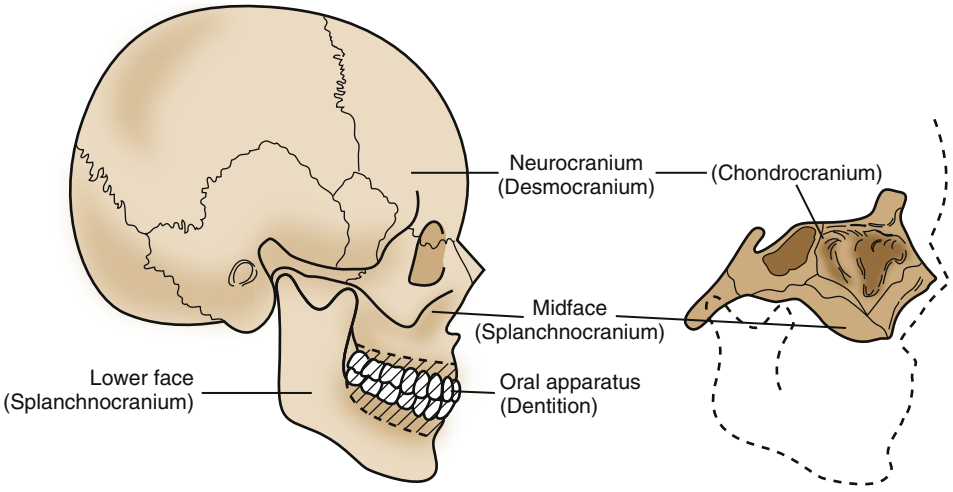


FIGURE 1-5 Major components of the craniofacial skeletal complex.

MOLECULAR BASIS OF CRANIOFACIAL DEVELOPMENT AND GROWTH

Patterning and subsequent formation of craniofacial tissues and structures have a complex, polygenic basis. For example, it has been shown that there are over 90 specific genes in which mutations will result in major disruptions of development leading to severe craniofacial malformations.[15] Moreover, variations in craniofacial development and growth, from dysmorphologies to malocclusions, are multifactorial as a result of epigenetic mechanisms.[16,17] No genes are unique to the craniofacial complex. However, certain genes, especially those associated with developmental patterning of the head region and growth of cartilage, bone, and teeth, are of particular relevance for craniofacial development and growth and thus are of special importance for orthodontics. In addition, a number of genes of interest include those responsible for specific craniofacial deformities, such as craniosynostosis and facial clefts. The reader is referred to Hartsfield and Morford (see Chapter 2) for a comprehensive review of genetic mechanisms in the craniofacial region that are most important to orthodontics. A summary of the key genes associated with the patterning, development, and growth of the craniofacial region can be found in E-Table 1-1.

The key genes associated with craniofacial development may be organized informally into two broad yet overlapping groups based on their timing and patterns of expression and also their primary target tissues. First are those highly conserved genes, such as homeobox genes and transcriptions factors, that are responsible primarily for early pattern formation and differentiation of primary embryonic tissues and structures, including neural crest cells and head mesoderm. Mutation of those genes typically has a profound role in craniofacial dysmorphogenesis. The second group is comprised of genes such as growth factors and signaling molecules that are also responsible for mediating development, growth, and maintenance of the tissues and structures associated with the craniofacial complex both during embryogenesis and throughout postnatal development. While mutations in this latter group of genes also are associated with craniofacial malformation syndromes, minor variants appear to be more common and may play a role in the development of more minor variations in growth. In addition, genes from both groups may be expressed reiteratively during development and growth, producing a highly complex matrix of interactions required for normal craniofacial morphogenesis. Adding to the complexity are the issues of wound healing, tissue regeneration, and repair—all processes important during orthodontic treatment—that can reinitiate the expression of genes required for early morphogenesis as well as postnatal growth.

Molecular research historically has focused on the role of specific genes critical for craniofacial morphogenesis during embryogenesis. The initial focus in that research typically has been on three areas: (1) naturally occurring genetic mutations associated with craniofacial dysmorphogenesis in humans; (2) development of genetically engineered animal models, typically the mouse, to produce loss of function of selected genes; and (3) mapping of gene expression in experimental animals through in situ hybridization and other biomarker approaches. More recently, significant progress has been made in the identification of gene variants (polymorphisms) that may be important for the origin of minor variations in craniofacial growth of potential relevance to orthodontic diagnosis and treatment. These genes and their variants could be significant for diagnosis and response to treatment of dentofacial deformities and minor malocclusions.[18] Significant advances in the genetic and epigenetic basis of craniofacial development, including the role of key genes in normal growth and orthodontic treatment, are expected to continue at a rapid pace.[19,20]

CRANIAL VAULT

Development of the Cranial Vault

The most prominent feature of the embryonic cephalic region at 6 to 7 weeks' gestation is the frontonasal prominence. The frontonasal prominence is a nonpaired structure that forms a dense desmocranial membrane, which covers the entire forebrain and extends laterally and inferiorly on each side of the developing head to meet the developing maxillary processes. The inner portion of the membrane contains neural crest cells and gives rise to the dura mater covering the brain. The outer portion of the desmocranial membrane, the *ectomeninx*, is comprised of surface ectoderm deep to which is the paraxial mesoderm. Patterning of the frontonasal prominence to form the cranial vault and elements of the nasal region is induced by expression of sonic hedgehog (Shh) and FGF-8.

By 8 weeks' gestation, initial blastemas of bone become apparent within the ectomeninx, first for the frontal bone and the squamous temporal bone and subsequently for the parietal bones and squamous portion of the occipital bone (Fig. 1-6).

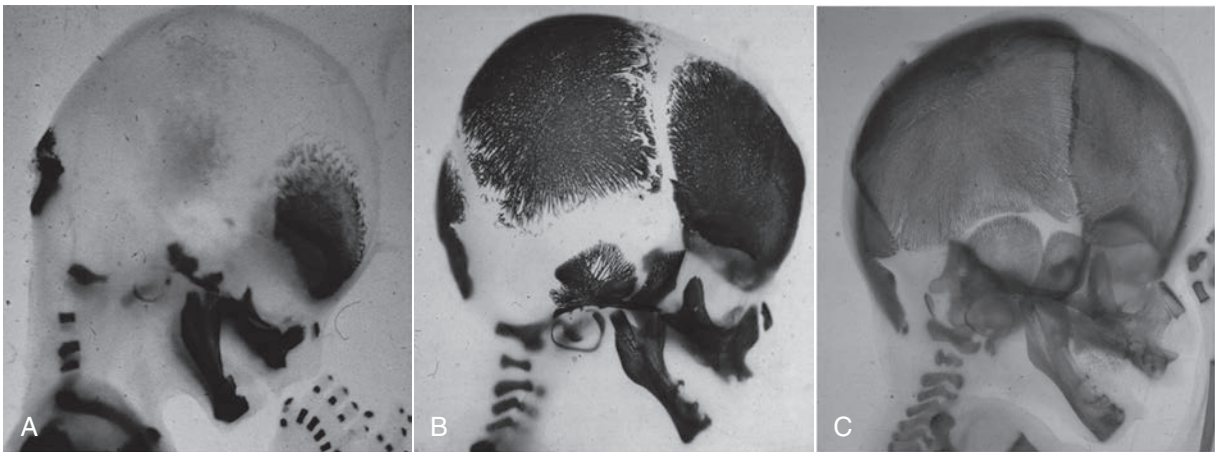


FIGURE 1-6 Cleared and stained human fetuses indicating craniofacial skeletal structures at approximately 8 weeks' gestation (A), 15 weeks' gestation (B), and 18 weeks' gestation (C).

TABLE 1-1 Summary of Key Genes Associated with the Development and Growth of the Tissues and Structures Comprising the Craniofacial Complex

Gene/Protein		General Role and Function	Significance for Craniofacial Development and Growth	References
Bmp-1 to Bmp-9	Bone morphogenetic protein 1-9	*Signaling molecule: S*keletal differentiation, growth, repair	NCC and CF mesenchyme patterning; suture development; odontogenesis; nsCL/P	1-6
Dlx-1 to Dlx-6	Distal-less 1-6	*Homeobox:* Limb development; chondrogenesis; osteogenesis	Orofacial clefting	7-9
Efnb1	Ephrin B1	*Protein coding:* Cell division, adhesion	Craniofrontonasal syndrome; candidate for role in class III malocclusion	1, 10-12
Fgf-1 to Fgf-18	Fibroblast growth factor 1-18	*Growth factors*: Differentiation and growth of multiple tissues and structures	CF ectoderm, NCC patterning; suture development; MCC growth; tooth induction; CL/P	1, 3, 4, 13-15
Fgfr-1 to Fgfr-3	Fibroblast growth factor receptor 1-3	*Transmembrane receptors*: Fgf receptor	Anterior cranial base growth; MCC growth; syndromic, nonsyndromic C-SYN; MX hypoplasia; CL/P	1, 3, 4, 15, 16, 17
Gh	Growth hormone	*Peptide hormone-mitogen*: Cell growth and tissue regeneration	Growth of multiple CF tissues, structures; variations in MD growth, dentofacial treatment	13, 18
Ghr	Growth hormone receptor	*Transmembrane receptor*: Receptor for GH	Polymorphisms associated with MD growth and MCC response to dentofacial treatment	19-21
Gli2 to Gli3	Zinc finger protein Gli2-3	*Transcription factor*: Regulates Ihh and Shh signaling	C-SYN; Greig cephalopolysyndactyly syndrome	1, 10, 22
Gsc	Goosecoid	*Transcription factor*: Dorsal–ventral patterning of NCC, head formation; rib fusion	Inner ear, cranial base, MX/MD anomalies	1, 8, 13, 23, 24
Hoxa1 to Hoxa3	Homeobox A1, A2, A3	*Homeobox*: Patterning of hindbrain rhombomeres and pharyngeal arches	Neural tube closure, 1st-2nd arch deformities	25, 26
Igf-1	Insulin-like growth factor 1	*Growth factor*: Mediator of Gh; muscle, cartilage, and bone growth	MX/MD growth; suture development/growth; mediation of MCC to dentofacial treatment	3, 8, 13, 27-30
Ihh	Indian hedgehog	*Signaling molecule*: Endochondral and intramembranous ossification	Cranial base development; mediation of MCC growth during dentofacial treatment	31-33
L-Sox5	Long-form of Sox5	*Transcription factor*: Neurogenesis; chondrogenesis; type II collagen	Mediation of MCC growth during dentofacial treatment	34
Msx1 to Msx2	Muscle segment homeobox 1-2	*Homeobox*: Limb development; ectodermal organs	NCC proliferation, migration; odontogenesis; MD development; nsCL/P; Boston-type C-SYN	1, 3, 4, 8, 10, 35
Myo1H and-Myo1C	Myosin 1H, Myosin 1C	*Protein coding*: Cell motility, phagocytosis, vesicle transport	Polymorphisms associated with MD prognathism	36, 37
Nog	Noggin	*Signaling molecule*: Patterning of the neural tube and somites	Head formation; neural tube fusion	4, 25, 26
Notch		*Transmembrane receptor*: Neuronal development; cardiac development; osteogenesis	MCC development	38
Osx	Osterix	*Transcription factor*: Osteoblast differentiation, mineralization; chondrogenesis	MCC differentiation, endochondral ossification; mediation of MCC growth during dentofacial treatment	39
Pitx1-2	Paired-like homeodomain 1-2	*Homeobox*: Left–right axis; left lateral mesoderm; skeletal development; myogenesis	MD development; role in Treacher-Collins syndrome; CL/P; odontogenesis	8, 13
Prx-1Prx-2		*Homeobox*: Epithelial development in limbs and face	NCC patterning; malformations of 1st-2nd arch structures	8, 40, 41
PTHrP	Parathyroid-related protein	*Protein coding*: Endochondral bone formation	Development/growth of cranial base, MD, dental arches	42, 43
Runx2	Runt-related transcription factor	*Transcription factor*: Osteoblast differentiation; intramembranous and endochondral bone growth	Closure of fontanelles and sutures; ossification of cranial base, MX, and MCC; cleidocranial dysplasia	32, 43-46
Shh	Sonic hedgehog	*Transcription factor*: Development of limbs, midline brain, neural tube; osteoblastic differentiation; skeletal morphogenesis	Induction of frontonasal ectoderm; cranial base; fusion of facial processes; palatogenesis; odontogenesis; holoprosencephaly	1, 9, 33
Sho2		*Signaling molecule*: Development of digits; organization of brain, CF mesenchyme	Palatogenesis; TMJ development	6, 9, 38

Continued

TABLE 1-1 Summary of Key Genes Associated with the Development and Growth of the Tissues and Structures Comprising the Craniofacial Complex—cont'd

Gene/Protein		General Role and Function	Significance for Craniofacial Development and Growth	References
Sox9		*Transcription factors*: Chondrogenesis; type II collagen; male sexual development	Cranial base; MCC growth; CL/P; Pierre-Robin sequence	38, 46-48
Spry 1-2	Sprouty	*Protein coding*: Mediates FGF signaling	MD/TMJ development	38, 48
Tcof1	Treacle	*Protein coding*: Early embryonic nucleolar-cytoplasmic transport	NCC proliferation, migration, survival; Treacher-Collins syndrome	38, 49
Tgf-ß1 to Tgf-ß3	Transforming growth factor-beta 1-3	*Growth factor*: Proliferation, differentiation, growth, function of multiple tissues	Palatogenesis; MD growth; suture development, maintenance, fusion; sCL/P	3, 24
Twist-1	Twist-related protein 1	*Transcription factor*: Skeletal development; syndactyly	MCC development; suture fusion; Saethre-Chotzen syndrome; facial asymmetry	9, 35, 38, 50, 51
Vegf	Vascular endothelial growth factor	*Growth factor*: Ingrowth of blood vessels	Chondrogenesis in cranial base, MCC	38, 45, 52
Wnt-1	Proto-oncogene protein Wnt 1	*Signaling molecule*: Cell fate, patterning during embryogenesis	MCC development/growth; MCC growth during dentofacial treatment	6, 32, 38, 53

CF, Craniofacial; *CPO*, cleft palate only; *CL/P*, cleft lip and palate; *C-SYN*, craniosynostosis; *MCC*, mandibular condylar cartilage; *MD*, mandible; *MX*, maxilla; *NCC*, neural crest cells; *nsCL/P*, non-syndromal cleft lip and palate; *sCL/P*, syndromal cleft lip and palate; *TMJ*, temporomandibular joint.

References

1. Rice DPC. Craniofacial anomalies: from development to molecular pathogenesis. *Curr Mol Med*. 2005;5:699–722.
2. Zheng L, Yamashiro T, Fukunaga T, Balam TA, Takano-Yamamoto T. Bone morphogenetic protein 3 expression pattern in rat condylar cartilage, femoral cartilage and mandibular fracture callus. *Eur J Oral Sci*. 2005;113:318–325.
3. Opperman LA, Gakunga PT, Carlson DS. Genetic factors influencing morphogenesis and growth of sutures and synchondroses in the craniofacial complex. In: Carlson DS, ed. *Control mechanisms of craniofacial development and growth. Semin Orthod*. 2005;11(4):199–208.
4. Chai Y, Maxson RE Jr. Recent advances in craniofacial morphogenesis. *Dev Dyn*. 2006;235:2353–2375.
5. Nie X, Luukko K, Kettunen P. BMP signaling in craniofacial development. *Int J Dev Biol*. 2006;50:511–521.
6. Greene RM, Pisano MM. Palate morphogenesis: current understanding and future directions. *Birth Defects Res (Part C)*. 2010;90:133–154.
7. Robledo RE, Rajan L, Li X, Lufkin T. The *Dlx5* and *Dlx6* homeobox genes are essential for craniofacial, axial, and appendicular skeletal development. *Genes Develop*. 2002;16:1089–1101.
8. Doshi RR, Patil AS. A role of genes in craniofacial growth. IIOBA. 2012;3(2):19–36.
9. Hinton RJ. Genes that regulate morphogenesis and growth of the temporomandibular joint: a review. *Devel Dyn*. 2014;243:864–874.
10. Melville H, Wang Y, Taub PJ, Jabs EW. Genetic basis of potential therapeutic strategies for craniosynostosis. *Am J Med Genet Part A*. 2010;152A:3007–3015.
11. Xue F, Wong RWK, Rabie ABM. Genes, genetics, and Class III malocclusion. *Orthod Craniofac Res*. 2010;13:69–74.
12. Xue F, Wong RWK, Rabie ABM. Identification of SNP markers on 1p36 and association analysis of *EPB41* with mandibular prognathism in a Chinese population. *Arch Oral Biol*. 2010;55:867–872.
13. Hinton RJ, Carlson DS. Regulation of growth in the mandibular condylar cartilage. In: Carlson DS, ed. *Control mechanisms of craniofacial development and growth. Semin Orthod*. 2005;11(4):209–218.
14. Hatch NE. FGF signaling in craniofacial biological control and pathological craniofacial development. *Crit Rev Eukaryot Gene Expr*. 2010;20(4):295–311.
15. Martinez-Abadias N, Heuze Y, Wang Y, Jabs EW, Aldridge K, Richtmeier J. FGF/FGFR signaling coordinates skull development by modulating magnitude of morphological integration: evidence from Apert syndrome mouse models. *PLoS One*. 2011;6(10).
16. Rice DPC, Rice R, Thesleff I. *Fgfr* mRNA isoforms in craniofacial bone development. *Bone*. 2003;33(1):14–27.
17. Heuze Y, Martinez-Abadias N, Stella JM, et al. Quantification of facial skeletal shape variation in fibroblast growth factor receptor-related craniosynostosis syndromes. *Birth Def Res (A)*. 2014;100:250–259.
18. Buschang PH, Hinton RJ. A Gradient of Potential for Modifying Craniofacial Growth. In: Carlson DS, ed. *Control mechanisms of craniofacial development and growth. Semin Orthod*. 2005;11(4):219–226.
19. Zhou J, Lu Y, Gai XH, et al. The growth hormone receptor gene is associated with mandibular height in a Chinese population. *J Dent Res*. 2005;84:1052–1056.
20. Kang EH, Yamaguchi T, Tajima A, et al. Association of the growth of the growth hormone receptor gene polymorphisms with mandibular height in a Korean population. *Arch Oral Biol*. 2009;45:556–562.
21. Sasaki Y, Satoh K, Hayasaki H, Fukumoto S, Fujiwara T, Nonaka K. The P561T polymorphism of the growth hormone receptor gene has an inhibitory effect on mandibular growth in young children. *Europ J Orthod*. 2009;31:536–541.
22. Veistinen L, Takatolo M, Tanimoto Y, Kesper D, Vortkamp A, Rice DPC. Loss-of-function of *Gli3* in mice causes abnormal frontal bone morphology and premature synostosis of the interfrontal suture. *Front Physiol*. 2012;3:1–6.
23. Sharpe PT. Homeobox genes and orofacial development. *Conn Tiss Res*. 1995;32:17–25.
24. Spears, Svoboda In: Carlson DS, ed. Growth factors and signaling proteins in craniofacial development. *Control mechanisms of craniofacial development and growth. Semin Orthod*. 2005;11(4):184–199.
25. Carlson B. *Human Embryology and Developmental Biology*. Philadelphia: Elsevier; 2014.
26. Trainor PA, Krumlauf R. Patterning the neural crest: hindbrain segmentation and *hox* gene plasticity. *Nat Rev Neuro*. 2000;1:116–124.
27. Hajjar D, Santos MF, Kimura ET. Mandibular repositioning modulates IGFBP-3, -4, -5, and -6 expression in the mandibular condyle of young rats. *Biorheology*. 2006;43(3–4):311–321.
28. Marques MR, Hajjar D, Franchini KG, Moriscot AS, Santos MF. Mandibular appliance modulates condylar growth through integrins. *J Dent Res*. 2008;87(2):153–158.
29. Patil AS, Sable RB, Kothari RM. Role of insulin-like growth factors (IGFs), their receptors and genetic regulation in the chondrogenesis and growth of the mandibular condylar cartilage. *J Cell Physiol*. 2011;227:1796–1804.
30. Frazier-Bowers S, Rincon-Rodriguez R, Zhou J, Alexander K, Lange E. Evidence of linkage in a Hispanic cohort with a class III dentofacial phenotype. *J Dent Res*. 2009;88:56–60.
31. Tang GH, Rabie ABM. Runx2 regulates endochondral ossification in condyle during mandibular advancement. *J Dent Res*. 2005;84(2):166–171.
32. Carreira AC, Lojudice FH, Halcsik E, Navarro RD, Sogayar MC, Granjeiro JM. Bone morphogenetic proteins: facts, challenges, and future perspectives. *J Dent Res*. 2014;93(4):335–345.
33. Balczerski B, Zakaria S, Tucker AS, et al. Distinct spatiotemporal roles of hedgehog signaling during chick and mouse cranial base and axial skeletal development. *Dev Biol*. 2012;371:203–214.
34. Chu FT, Tang GH, Hu Z, Qian YF, Shen G. Mandibular functional positioning only in vertical dimension contributes to condylar adaptation evidenced by concomitant expressions of L-Sox5 and type II collagen. *Arch Oral Biol*. 2008;53:567–574.
35. Bonaventure J, El-Ghouzzi V. Molecular and cellular basis of syndromic craniosynostosis. *Exp Rev Mol Med*. 2003;5(29):1–17.
36. Tassopoulou-Fishell M, Deeley K, Harvey EM, Sciote JJ, Viera AR. Genetic variation in *Myosin 1H* contributes to mandibular prognathism. *Am J Orthod Dentofac Orthoped*. 2012;141(1):51–59.
37. Desh H, Gray SL, Horton MJ, et al. Molecular motor *MYO1C*, acetyltransferase *KAT6B* and osteogenetic transcription factor *RUNX2* expression in human masseter muscle contributes to development of malocclusion. *Arch Oral Biol*. 2014;59:601–607.
38. Hinton RJ, Serrano M, So S. Differential gene expression in the perichondrium and cartilage of the neonatal mouse temporomandibular joint. *Orthod Craniofac Res*. 2009;12:168–177.

TABLE 1-1 Summary of Key Genes Associated with the Development and Growth of the Tissues and Structures Comprising the Craniofacial Complex—cont'd

39. Jing J, Hinton RJ, Jing Y, Liu Y, Zhou X, Feng JQ. Osterix couples chondrogenesis and osteogenesis in post-natal condylar growth. *J Dent Res.* 2014;93(10):1014–1021.
40. ten Berge D, Brouwer A, Korving J, et al. Prx1 and Prx2 are upstream regulators of sonic hedgehog and control cell proliferation during mandibular arch morphogenesis. *Devel.* 2001.
41. Martin JF, Bradley A, Olsen EN. The *paired*-like homeobox gene *Mhox* is required for early events of skeletogenesis in multiple lineages. *Genes Develop.* 1995;9:1237–1249.
42. Kyrkanides S, Kambylafkas P, Miller JH, Tallents RH, Puzas JE. The cranial base in craniofacial development: a gene therapy study. *J Dent Res.* 2007;86(10):956–961.
43. Hinton RJ. Genes that regulate morphogenesis and growth of the temporomandibular joint: a review. *Devel Dyn.* 2014;243:864–874.
44. Rabie ABM, Tang GH, Hägg U. Cbfa1 couples chondrocytes maturation and endochondral ossification in rat mandibular condylar cartilage. *Arch Oral Biol.* 2004;49(2):109–118.
45. Lei WY, Wong RWK, Rabie ABM. Factors regulating endochondral ossification in the spheno-occipital synchondrosis. *Angle Orthod.* 2008;78(2):215–220.
46. Nie X, Luukko K, Kvinnsland IH, Kettunen P. Developmentally regulated expression of Shh and Ihh in the developing mouse cranial base: comparison the Sox9 expression. *Anat Rec A Discov Mol Cell Evol Biol.* 2005;286(2):891–898.
47. Cendekiawan T, Wong RWK, Rabie ABM. Temporal expression of SOX9 and type II collagen in spheno-occipital synchondrosis of mice after mechanical tension stimuli. *Angle Orthod.* 2008;78(1):83–88.
48. Rabie ABM, She TT, Hägg U. Functional appliance therapy accelerates and enhances condylar growth. *Am J Orthod Dentofacial Orthop.* 2003;123(1).
49. Su P-H, Liu Y-F, Chen J-Y, Lai Y-J, Facial asymmetry and clinical manifestations in patients with novel insertion of the *TCOF1* gene. *Clin Genet.* 2012;82:460–465.
50. Coussens AK, Wilkinson CR, Hughes IP, et al. Unravelling the molecular control of calvarial suture fusion in children with craniosynostosis. *BMC Genomics.* 2007;8:458.
51. Melville H, Wang Y, Taub PJ, Jabs EW. Genetic basis of potential therapeutic strategies for craniosynostosis. *Am J Med Genet Part A.* 2010;152A:3007–3015.
52. Rabie ABM, Hägg U. Factors regulating mandibular condylar growth. *Am J Orthod Dentofacial Orthop.* 2002;122:401–409.
53. Enomoto A, Watahiki J, Nampo T, et al. Mastication markedly affects mandibular condylar growth, gene expression, and morphology. *Am J Orthod Dentfac Orthop.* 2014;146(3):365–363.

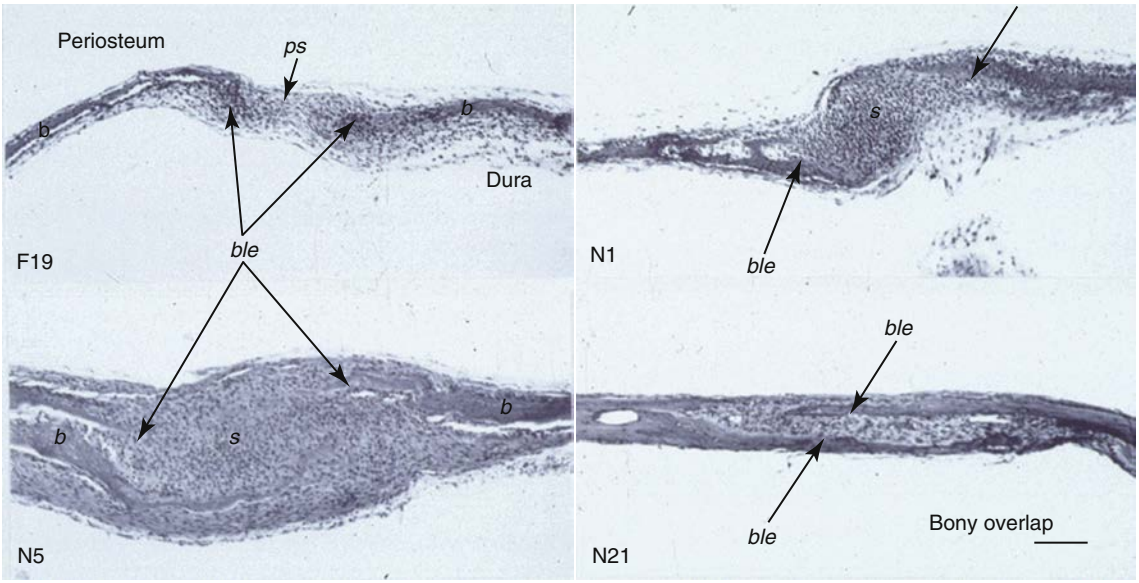


FIGURE 1-7 Photomicrographs of hematoxylin and eosin–stained histologic sections through the coronal suture of normal rats at embryonic day 19 and postnatal days 1, 5, and 21. Bone (*b*), bone leading edge (*ble*), presumptive suture mesenchyme (*ps*), and suture (*s*). (From Opperman LA, Gakunga PT, Carlson DS. Genetic factors influencing morphogenesis and growth of sutures and synchondroses in the craniofacial complex. *Semin Orthod*. 2005;11(4):199–208.)

Over the ensuing 4 weeks, these condensations of bone steadily increase in size by radial expansion of newly differentiated skeletal tissue within the ectomeninx. As the development of new bone exceeds the rate of growth of the brain, the peripheral bone fronts become located closer and closer to each other, until they approximate each other as single-thickness plates of flat bones by about 12 weeks' gestation. At this point, the intervening fibrous tissue becomes highly cellular, and fibrous articulations, or *sutures*, are formed between the individual bone elements (Fig. 1-7).

Growth of the cranial vault bones represents a specialized form of intramembranous ossification that begins prenatally as blastemas of bone tissue that arise de novo within the middle layer of the desmocranial membrane covering of the brain. Once the skeletal elements as plates of bone become located close to each other, their fibrous connections become reorganized with the periosteum and the dura mater derived from the outer and inner layers of the desmocranial membrane, respectively, extending into the sutural articulations. The sutures then continue to support growth of the cranial vault through another specialized form of intramembranous osteogenesis similar to periosteal bone formation.[21–23]

Mechanisms of Suture Growth

Sutural bone growth can best be considered as a specialized form of intramembranous periosteal bone growth. Once formed, the bones of the cranial vault are enveloped, like all bones, in a skeletogenic membrane. On the external surface, this membrane is the periosteum. On the intracranial surface, the membrane is the dura mater, which is also derived from the embryonic ectomeninx and is skeletogenic. Viewed in cross section, the outer fibrous layer of periosteum (uniting layer) spans over the cranial suture and provides structural support to the suture and its two or more skeletal elements. The inner osteogenic layers of

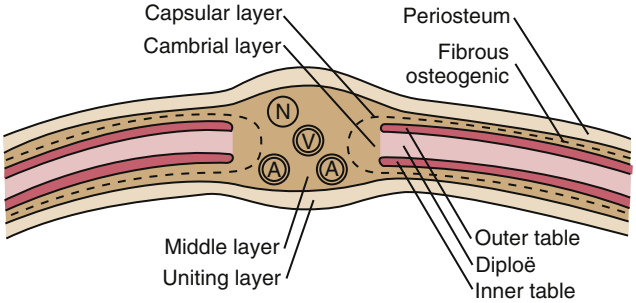


FIGURE 1-8 Schematic representation indicating the relationship between the periosteum and dura mater as a mechanism for a specialized of intramembranous growth within the sutures of cranial vault bones. (Adapted from Pritchard JJ, Scott JH, Girgis FG. The structure and development of cranial and facial sutures. *J Anat*. 1956;90:73–86.)

the periosteum and the dura reflect into the space between the two cranial vault bones and provide a source of new osteogenic cells (Fig. 1-8). As the bones of the cranial vault become separated because of expansion of the brain and intracranial contents, the osteogenic cells form skeletal tissue and thus provide a mechanism for maintaining relatively close contact through the intervening suture.

The molecular basis of the development and growth of the sutures of the cranial vault has received considerable attention, principally because of the number of naturally occurring and engineered genetic mutations characterized by craniosynostosis (see Wilkie and Morriss-Kay,[15] Rice,[24] and Chai and Maxson[25] for comprehensive reviews). Studies have shown a complex pattern of gene expression within the sutural blastema associated with the periosteal reflection as well as the intracranial dura mater. Secretion of soluble factors by the

FIGURE 1-9 Distribution of growth factors and transcription factors active during suture growth (A) and suture synostosis (B). (Adapted from Opperman LA, Gakunga PT, Carlson DS. Genetic factors influencing morphogenesis and growth of sutures and synchondroses in the craniofacial complex. *Semin Orthod.* 2005;11(4):199–208)

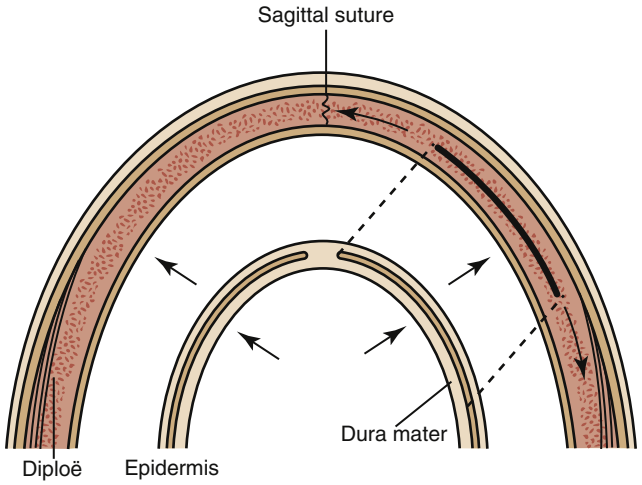


FIGURE 1-10 Schematic diagram indicating the relationship between expansile growth of the brain as a stimulus for compensatory growth of sutures of the cranial vault. (Adapted from Moss ML. The functional matrix. In: Kraus B, Reidel R, eds. *Vistas Orthod.* Philadelphia: Lea & Febiger; 1962;85–98.)

dura mater in response to growth signals from the expanding, underlying brain is essential for normal cranial suture morphogenesis as well as for the maintenance of cranial sutures as patent bone- growth sites through complex tissue interactions and feedback between dura mater, bone fronts, and sutures. Both sutures and the dura mater also contain growth factors, such as several members of the family of transforming growth factor beta (TGF-β1, TGF-β2, TGF-β3), bone morphogenetic protein (BMP2, BMP7), fibroblast growth factor 4 (FGF-4), insulin-like growth factor 1 (IGF-1), and sonic hedgehog (SHH) (Fig. 1-9).[26,27] Overexpression of transcription factors Runx2 and Msx2 and haploinsufficiency of Twist[28] and Noggin[29] are also associated with suture obliteration, while loss of function of *Gli3* results in premature synostosis.[30] Genetic analysis of naturally occurring craniosynostosis in humans has shown that mutations of genes for fibroblast growth factor receptors 1, 2, and 3 (*FGFR-1*, *FGFR-2*, and *FGFR-3*) and in *MSX2*[31] and *TWIST*[32,33] genes are also associated with premature suture fusion.

Development and growth of the cranial vault as a whole, and development and growth of bone at the sutural articulations, are primarily dependent on the expansion of the brain and other intracranial contents.[34] Furthermore, it has been clearly demonstrated that sutures are secondary, compensatory, and adaptive sites of bone growth that normally respond to biomechanical forces. As the brain expands during prenatal development and during the first decade of life postnatally, forces are created within the neurocranium that cause the bones of the cranial vault to expand outward, which tends to separate them from each other at the sutural boundaries (Fig. 1-10). Under normal conditions, the cellular and molecular substrate associated with the dura mater, the periosteum, and the suture respond to this biomechanical displacement in the same manner in which periosteum throughout the skeletal system responds—by initiating and maintaining osteogenesis within the sutures to maintain the proximity of the adjoining skeletal structures. When the biological substrate of the suture is abnormal, however, as in the case of many genetic syndromes such as Crouzon syndrome, Apert syndrome, and Jackson-Weiss syndrome, for example, each of which is associated with mutations of *FGFR-2*, premature craniosynostosis may result.[35,36] The opposite condition, reduced suture growth, and prolonged patency, as seen in cleidocranial dysostosis, may occur with abnormalities associated with growth factors, including in particular Runx2, which are necessary for normal suture fusion.

Postnatal Growth of the Cranial Vault

Due to the very precocious nature of prenatal and early postnatal human brain development, the cranial vault is disproportionately large relative to the rest of the face and body. At birth, the cranial vault is initially characterized by the presence of all of the cranial vault bones. At that time, all the major sutural fibrous articulations between the bones of the cranial vault are present, including the metopic suture between the right and left frontal bone. In addition, there typically are four larger remnants, known as *fontanels*, of the desmocranial membrane in areas where the pace of bone growth has not been sufficient to approximate the bones of the cranial vault to form a suture (Fig. 1-11).

During the first 24 months after birth, growth of the cranial vault bones proceeds rapidly enough to close the fontanels as each complex of cranial vault bones becomes organized through interlocking sutures. The metopic suture normally fuses to form a single frontal bone within the first year of life, although the suture may appear to persist for up to 8 years of age or even throughout life in a small percentage of individuals. The cranial vault will continue to enlarge primarily as a result of compensatory growth of the sutural bone fronts stimulated by expansion of the brain. By 4 years of age, the brain and the associated cranial vault will have achieved approximately 80% of adult size; by age 10, the brain and cranial vault have attained 95% of their adult size. Throughout this time of very rapid expansion, the remaining sutures of the cranial vault normally remain patent and actively growing to keep pace with the brain as it expands in size.

Osteogenesis at cranial sutural bone fronts may continue for the first two decades of life. However, by the end of the second decade of life, bone growth at cranial sutures has slowed and the potential for growth of cranial sutures has greatly diminished. Also at that time, the sutures will begin the normal process

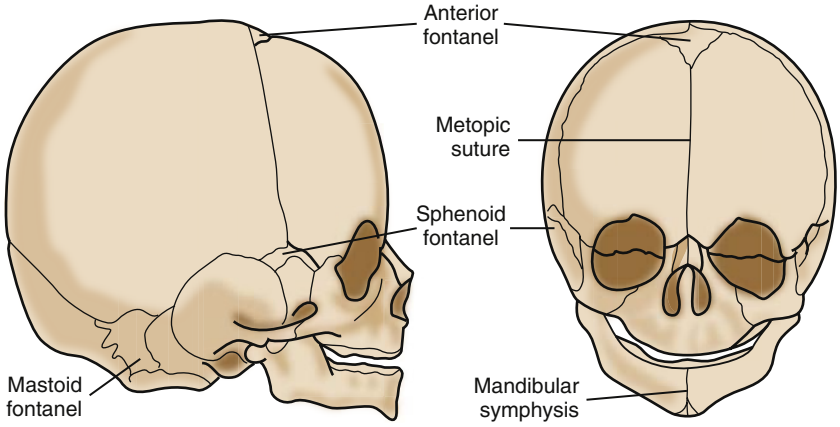


FIGURE 1-11 Lateral and frontal views of the neonate skull indicating the location of sutures and fontanels. (Adapted from Sicher H, DuBrul EL. *Oral Anatomy*. 5th ed. St. Louis: Mosby; 1970.)

of bony closure, or *synostosis*, when the potential for sutural growth ceases altogether.

The cranial sutures normally lose the capacity for growth by the end of the second decade of life, and virtually all become synostosed during the life span. Normal suture closure is initiated along the endocranial surface. Initially, this is characterized by bridging of bone across the suture and eventually through modeling of bone, leading to complete obliteration of the suture. Cessation of growth at cranial sutures typically begins around age 25 for the sagittal suture and may be extended for 2 to 3 additional years for the coronal suture.

Despite the fact that the major cranial sutures stop growing by the third decade of life, some enlargement of the cranial vault overall typically occurs throughout the lifespan as a result of periosteal deposition along the ectocranial surface. Certain specific areas of the cranial vault, such as the glabellar and nuchal regions, may exhibit slightly greater periosteal growth as a secondary sex characteristic in males.

CRANIAL BASE

Development of the Cranial Base

The ectomeningeal membrane that surrounds the developing brain in the cranial base region gives rise to a number of paired cartilaginous elements that form the embryonic chondrocranium. The first of the cartilage anlagen to form arises from neural crest cells at about 6 weeks' gestation as the parachordal cartilages, which surround the proximal end of the notochord and give rise to the anterior cranial base. The posterior component of the cranial base is derived primarily from mesoderm to form the basioccipital bone.[37] Development of the chondrocranium then progresses rostrally to the otic capsule, which will form the petrous portion of the temporal bone; the postsphenoid, presphenoid, alisphenoid, and orbitosphenoid cartilages of the sphenoid bone; and the nasal capsule and mesethmoid, which will form the ethmoid bone, inferior turbinate, and nasal septum. By 8 weeks' gestation, the separate cartilage elements have merged to form a single plate of primary hyaline cartilage, the *basal plate*, extending from the foramen magnum rostrally to the tip of the nasal cavity (Fig. 1-12).

More than 110 separate centers of ossification form in the basal plate, beginning with the parachordal cartilages and continuing rostrally through the sphenoid complex around 9 to 16 weeks, to the ethmoid region as late as 36 weeks. As

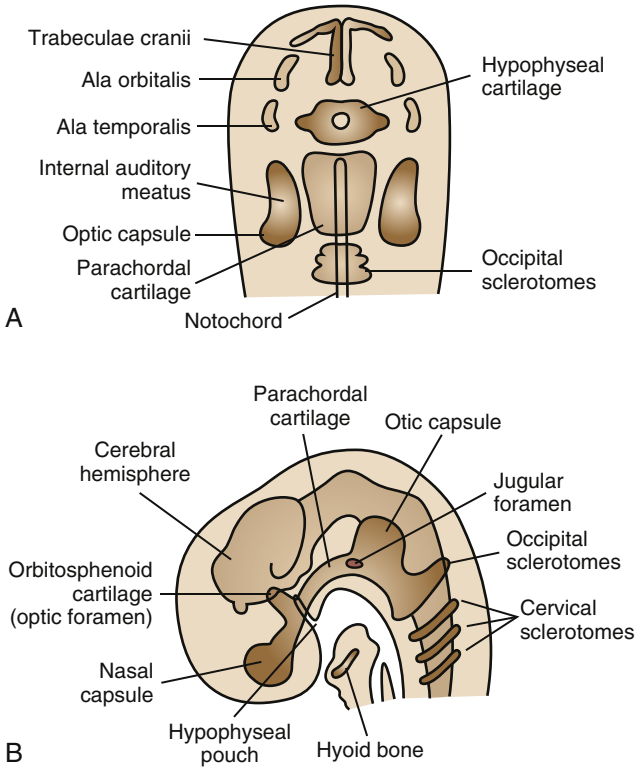


FIGURE 1-12 Schematic representation of the cartilaginous basal plate comprising the embryonic chondrocranium. A, Dorsoventral view; B, Lateral view.

these centers of ossification arise within the chondrocranium, segments of intervening cartilage form *synchondroses* (Fig. 1-13). The principal cranial base synchondroses that are most relevant for understanding craniofacial growth are the spheno-occipital synchondrosis, between the body of the sphenoid and the basioccipital bone, and the sphenoethmoidal synchondrosis, between the sphenoid and ethmoid bones. The greater wing of the sphenoid bone and the squamous portion of the occipital bone develop and grow via intramembranous ossification.

Mechanism of Synchondrosal Growth

Cranial base synchondroses are temporary cartilaginous joints located between bones of endochondral origin and growth.

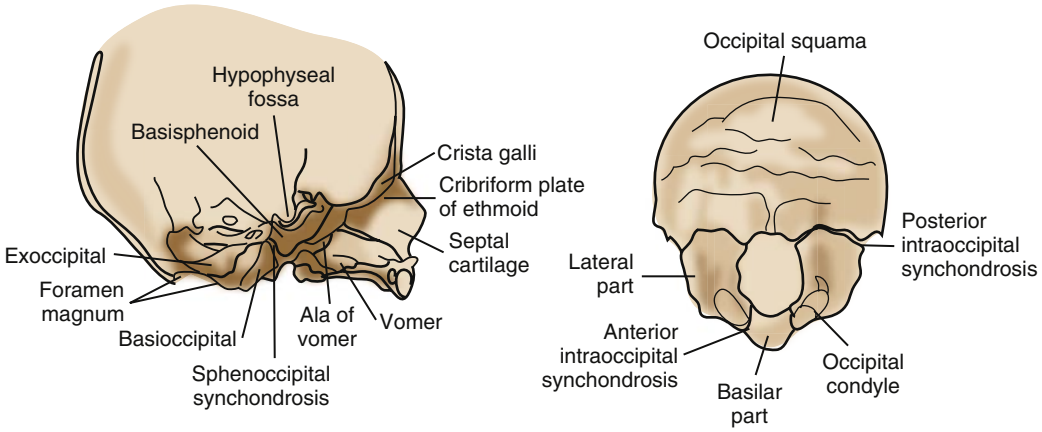


FIGURE 1-13 Drawing of sagittal and basal views of the neonatal skull indicating spheno-occipital synchondrosis and intraoccipital synchondroses. The sphenoethmoidal synchondrosis will arise between the sphenoid and ethmoid bones. (Adapted from Bosma JF. Introduction to the symposium. In: Bosma JF, ed. *Development of the Basicranium*. Bethesda, MD: US Department of Health, Education, and Welfare; 1976:3–28.)

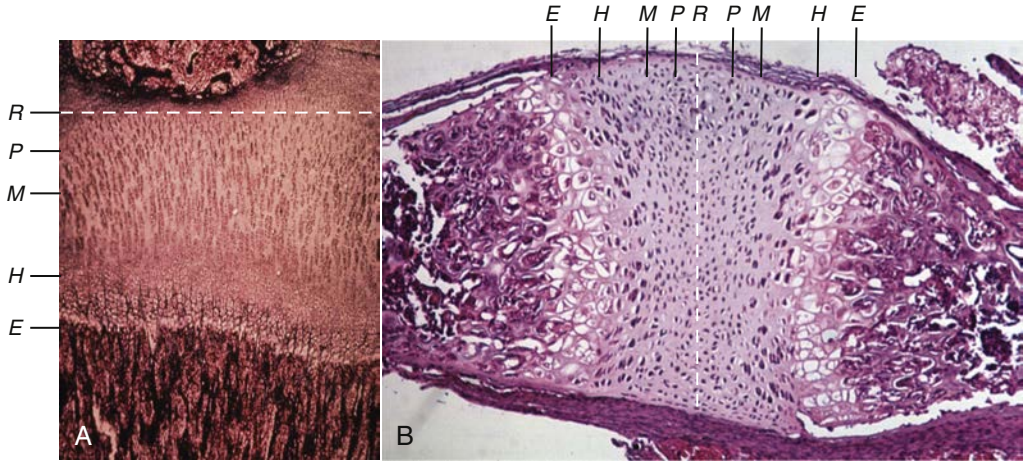


FIGURE 1-14 Histologic comparison between the cartilages within a growing epiphyseal plate (**A**) and cranial base synchondrosis (**B**) (hematoxylin and eosin–stained). *R*, Resting zone (*dashed line*); *P*, proliferating zone; *M*, maturational zone; *H*, hypertrophic zone; *E*, zone of endochondral ossification.

Synchondroses can best be considered as homologous to the epiphyseal growth plates of long bones. Functionally, both provide a mechanism for rapid endochondral growth of bone in a manner that is capable of overcoming biomechanical loads, thus exhibiting tissue-separating capabilities. Developmentally, cranial base synchondroses and epiphyseal plates of long bones synostose and become obliterated when the skeletal element achieves its mature size and shape. This typically occurs at the end of puberty for epiphyseal growth plates but varies from the end of the juvenile period through the end of puberty for the major cranial base synchondroses.

Cranial base synchondroses and epiphyseal growth plates are both derived from the primary hyaline cartilage that arises as part of the embryonic cartilaginous anlagen. Like endochondral bones and growth plates throughout the body, growth of synchondroses is controlled principally by expression of Indian hedgehog gene (Ihh) and sonic hedgehog (Shh).[38,39] The significance of *FGFR-3* for growth of the anterior cranial base is also indicated by mutations associated with achondroplasia.

Histomorphologically, both cranial base synchondroses and epiphyseal growth plates, are characterized by primary chondrocytes that are distributed into zones that are highly typical for growth plate cartilage (Fig. 1-14). However, a major difference between epiphyseal growth plates in long bones and cranial base synchondroses is that synchondroses are "bidirectional." Thus, each cranial base synchondrosis effectively has two back-to-back growth plates with a shared region of newly forming cartilage in the center and bone at each end. Growth plates are "unidirectional."

The primary hyaline cartilage of the cranial base is the same as that found throughout the embryonic cartilaginous anlage that characterizes all the other cartilaginous bones throughout the body. It is well known that growth of tissues derived from the primary embryonic cartilaginous anlagen tends to be relatively resistant to all but very extreme external influences. Growth of cartilage-derived skeletal elements throughout the body tends to be relatively resistant to environmental and other factors and instead is regulated to a large extent by intrinsic, genetically regulated growth factors and cell-signaling molecules.[40] The same is true for the cranial base synchondroses.

However, it is important to note that the growth of both epiphyses and synchondroses can be significantly affected by such epigenetic factors as disease, malnutrition, and undernutrition, as well as other conditions that affect production and expression of endocrine factors responsible for bone growth.

The cartilage cells within both epiphyseal growth plates and cranial base synchondroses are characterized by extensive amounts of extracellular matrix that are secreted by and separate the cartilage cells. This matrix makes the cartilage very dense and strong but also flexible relative to bone and thus better able to absorb mechanical forces without directly affecting the cells and potentially altering growth. Because there are no vessels within cartilage extracellular matrix, all nutrients, growth factors, and cell-signaling molecules must diffuse through the matrix to reach the chondrocytes. The matrix thus "buffers" the chondrocytes from extrinsic mechanical forces and many soluble molecules that might provide information about the external environment.[41] As a result, cartilage growth in general, and endochondral ossification from primary hyaline cartilage in particular, tend to be more rigidly programmed genetically than intramembranous bone growth associated with periosteum, such as occurs in the desmocranium and viscerocranium.

This difference in the mechanisms of growth between bone formed by means of intramembranous ossification and bone derived from endochondral ossification can be summarized through the concepts of skeletal *growth centers* versus skeletal *growth sites*.[42] Development and growth of the skeletal tissues derived from primary cartilage are significantly more intrinsically regulated and less dependent for their expression on epigenetic factors. In particular, *growth centers* have what has been described as "tissue-separating capabilities," emphasizing the capacity to grow and expand despite the presence of mechanical forces that would seem to be capable of inhibiting or restricting skeletal growth. Thus, epiphyseal and synchondrosal cartilage are referred to as growth centers. In contrast, a *growth site* is an area of skeletal growth that occurs secondarily and grows in compensatory fashion to growth and function in a separate but proximate location. Growth sites have no tissue-separating capabilities but rather respond more readily to factors extrinsic to their specific area. Periosteal bone growth associated with muscle function is one obvious example of a growth site. Sutural bone growth is another example of a class of growth sites because of its association with bones of intramembranous origin and its clear connection to periosteal bone growth.

Postnatal Growth of the Cranial Base

Late prenatal and overall postnatal growth of the cranial base is related directly to growth of the synchondroses. There are three principal growth-related cranial base synchondroses that separate the bones of the cranial base at birth. The intersphenoid synchondrosis, between the presphenoid and basisphenoid, fuses around the time of birth in humans and thus does not contribute to postnatal growth. The sphenoethmoidal synchondrosis, which lies between the sphenoid and the ethmoid bones, is most active with respect to growth of the cranial base through approximately 7 to 8 years of age in humans. At that time, the sphenoethmoidal synchondrosis loses its cartilage phenotype and becomes a suture. Once that transition occurs, growth of the anterior cranial base is essentially complete. As a result, the anterior wall of the sella turcica, which is located on the body of the sphenoid; the greater wing of the sphenoid; the cribriform plate; and the foramen cecum are commonly used after age 7 as stable reference structures for analyses of serial lateral radiographic cephalograms.

The spheno-occipital synchondrosis, between the body of the sphenoid and occipital bones, is most prominent throughout the period of active craniofacial growth and fuses shortly after puberty (Fig. 1-15). Once synostosis occurs, growth of the cranial base, especially in the anteroposterior direction, is essentially over. Subsequent changes in the form of the cranial base, such as in the angulation of the basioccipital bone relative to the anterior cranial base, for example, must come about as a result of bone modeling.

During the early postnatal years, the cranial base undergoes a dramatic shift in its growth pattern (Fig. 1-16). Anterior (nasion–sella) and posterior (sella–basion) cranial base lengths, as well as cranial base angulation (nasion–sella–basion), exhibit greater growth changes during the first 2 to 3 postnatal years than any time thereafter. For example, cranial base angulation decreases more than twice as much during the first 2 postnatal years than between 2 and 17 years of age, primarily due to differential growth of the spheno-occipital synchondrosis. Growth continues after 2 years of age, but the changes are smaller and steadier.

Between birth and 17 years of age, the anterior cranial base grows approximately 36% (males) to 53% (females) more than the posterior cranial base, with most of the differences occurring during the first few years.[43] It is important to understand that the anterior cranial base grows more and is also more mature (i.e., closer to its adult size) than the posterior cranial base throughout the postnatal growth. Longitudinal analyses have shown that the anterior cranial base has already attained approximately nearly 90% of its adult size by 4.5 years of age, while the posterior cranial base has attained only about 80% of its adult size (Fig. 1-17). The relative maturity differences between the anterior and posterior cranial base lengths are maintained throughout postnatal growth.

Anterior and posterior cranial base lengths increase because of bony deposition, as well as growth at the spheno-occipital and sphenoethmoidal synchondroses. Postnatally, the posterior cranial base becomes longer primarily due to growth at the spheno-occipital synchondrosis. Histologic studies have shown that the spheno-occipital synchondrosis fuses at approximately 16 to 17 years in females and 18 to 19 years in males.[44] Radiographically, the spheno-occipital synchondrosis shows active growth until approximately 10 to 13 years of age, at which time closure starts superiorly and continues inferiorly around 11 to 14 years in females and 13 to 16 years in males.[45,46]

Because both landmarks are commonly used to describe the growth of the anterior cranial base, it is important to distinguish the changes that occur at nasion from those that occur at foramen cecum. After fusion of the sphenoethmoidal synchondrosis, which occurs at approximately 7 to 8 years of age, increases in the distance between sella and foramen cecum are due primarily to the posterior and inferior drift of the sella turcica. The distance sella–nasion, on the other hand, continues to increase primarily due to bony apposition on the outer surface of the frontal bone associated with the development of the frontal sinus (the earliest pneumatization of the frontal sinus occurs around 2 years of age). The anterior cranial fossa continues to expand slightly, and the frontal sinus becomes more prominent. As a result, the frontal bone and root of the nose become

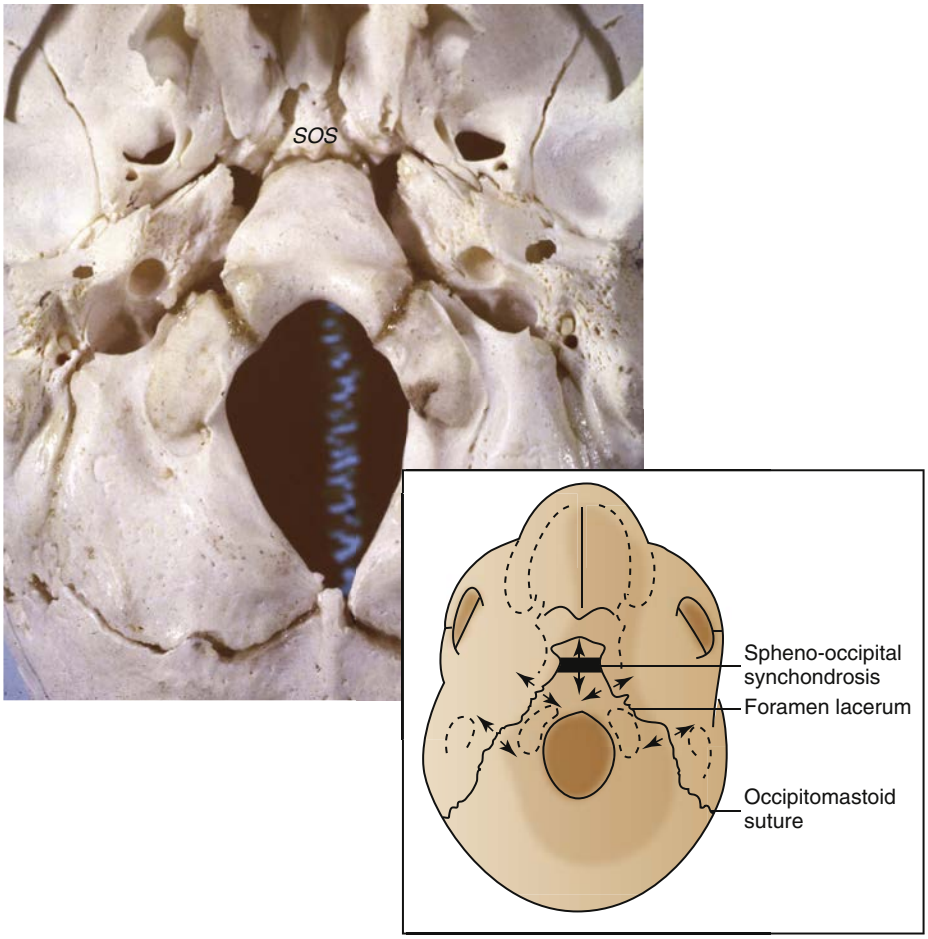


FIGURE 1-15 Basal view of a juvenile human indicating the spheno-occipital synchondrosis (*SOS*).

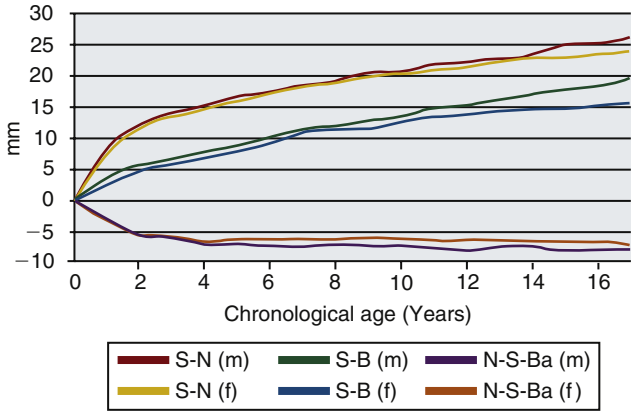


FIGURE 1-16 Male (m) and female (f) cranial base growth changes from birth through 17 years of age. (Data from Ohtsuki F, Mukherjee D, Lewis AB, etc.: A factor analysis of cranial base and vault dimensions in children, *Am J Phys Anthropol* 58(3):271-9, 1982.)

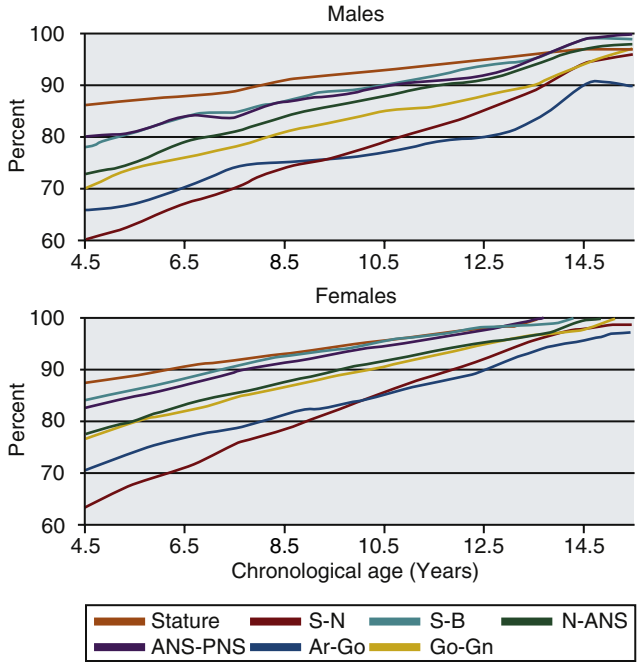


FIGURE 1-17 Craniofacial growth maturity gradient of males and females. (Adapted from Buschang PH, Baume RM, Nass GG. A craniofacial growth maturity gradient for males and females between 4 and 16 years of age. *Am J Phys Anthrop*. 1983;61:373–382.)

more anteriorly located. Ford[47] estimated that the frontal bone drifts anteriorly approximately 7 mm between the time that the sphenoethmoidal synchondrosis fuses and adulthood.

MIDFACE/NASOMAXILLARY COMPLEX

The midface, or nasomaxillary complex, is composed of the paired maxillae, nasal bones, zygomatic bones, lacrimal bones, palatine bones, and, within the nasal cavity, the turbinates and vomer. Prenatally, human fetuses also have left and right premaxillary bones; however, these normally fuse with the maxillae within 3 to 5 years after birth (Fig. 1-18).

The midface is connected to the neurocranium by a circummaxillary suture system and, toward the midline, by the cartilaginous nasal capsule, nasal septum, and vomer (Fig. 1-19). There is also an intermaxillary suture system composed of the midpalatal, transpalatal, intermaxillary, and internasal sutures. With the exception of the inferior turbinates, all the bones composing the midface are formed intramembranously from a connective tissue mass.

Development of the Midface

The midface has both viscerocranial and chondrocranial components. The chondrocranial component is comprised principally of parasagittal extensions of the cartilaginous anterior cranial base as the nasal septum and cartilaginous nasal capsule into the nasal region. The viscerocranial component is derived from two embryonic structures. The first is an inferior extension of the frontonasal prominence, which extends toward the oral opening, or stomodeum, to form nasal structures and the philtrum of the upper lip. The second is the paired maxillary processes of the first branchial arch. Differential growth of the right and left maxillary processes results in their apparent migration medially until they come into contact with the medial nasal process of the frontonasal prominence.

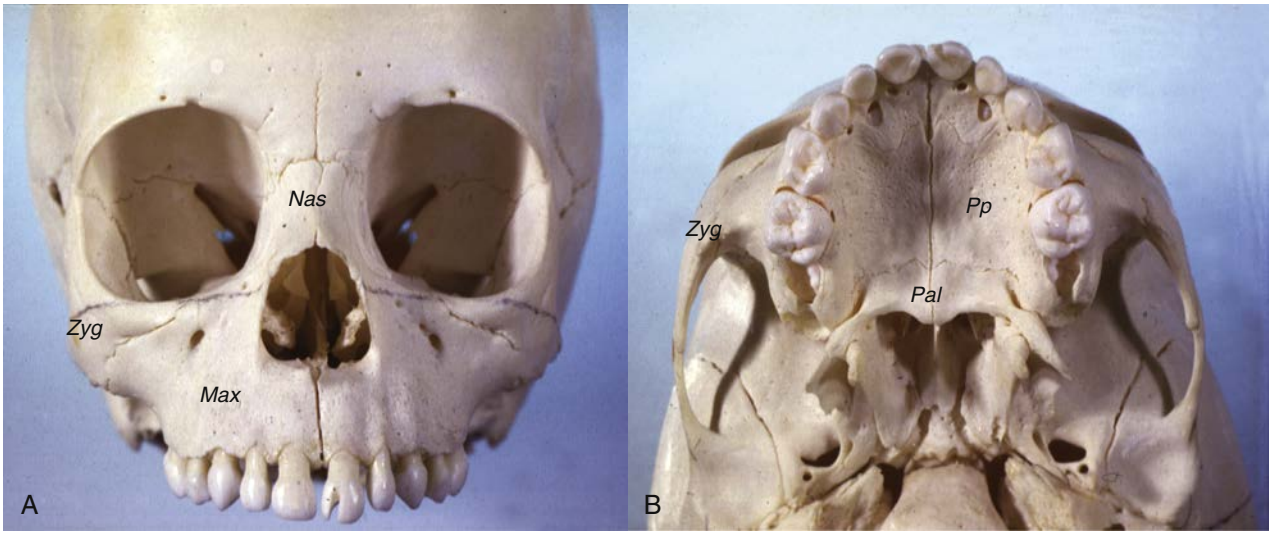


FIGURE 1-18 (**A**) Frontal and (**B**) basal views of a juvenile human indicating the bones comprising the midface. *Max*, maxilla; *Nas*, nasal bones; *Zyg*, zygomatic bones; *Pal*, palatine bones; *Pp*, palatal processes of the maxillary bones.

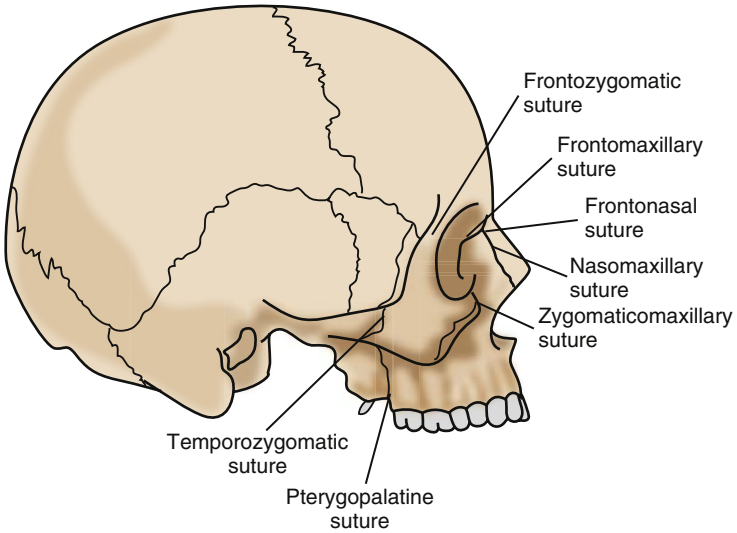


FIGURE 1-19 Location of the circummaxillary suture system articulating the midface with the neurocranium.

The skeletal elements comprising the midfacial complex arise almost exclusively from neural crest cells within the maxillary process of the first branchial arch. The primary palate, which gives rise to the four maxillary incisors, is derived from the frontonasal prominence. Only the facial ethmoid and inferior turbinate are derived from the cartilaginous component of the midface. Like the bones of the cranial vault, since the bones comprising the nasomaxillary complex have no cartilaginous precursors, they rely on intramembranous ossification for their development. However, the exact process by which initial bone formation occurs differs from that of the cranial vault bones. While the bones of the cranial vault arise within a desmocranial membrane, centers of ossification for the nasomaxillary bones develop as blastemas directly within the mesenchyme of the first branchial arch. These blastemas of bone are then surrounded by a periosteum that provides the source of new osteoblastic cells and thus for enlargement of the skeletal element. Molecular signaling mechanisms associated with the development, growth, and maintenance of the facial sutures are dependent on the presence of the nasal capsular cartilage, which appears to play a role similar to the dura mater in sutures of the cranial vault in the expression of TGF-β1, TGF-β2, TGF-β3, and Msx2.[48] It has also been shown that *Fgf8* plays a significant role in the integration and coordination of the frontonasal prominence with the nasal and optic regions.[49]

Virtually all of the major centers of ossification within the midface can be seen at approximately 7 to 8 weeks' gestation. At 6 weeks' gestation, the palatal shelves, which are mesenchymal tissue extensions of the embryonic maxillary processes of the first branchial arches, elevate within the oral cavity, where they will give rise to the hard and soft palates. The palatal shelves begin to ossify at 7 to 8 weeks' gestation, with the two bone fronts of the palatal processes each extending medially to form the secondary palate, composed of processes from the maxillary bones and from the palatine bones, as they meet in the midline where they form the midpalatal suture.

The molecular mechanisms associated with the development of the palate are among the most studied in all of craniofacial growth and development because of the obvious problem of cleft lip and palate, which is the most common craniofacial deformity (approximately 1:1000 for children of European descent).[50,51] Genes that have been identified specifically for a significant role in the genesis of cleft lip and palate now include isoforms of BMP, Dlx, Fgf-8, Msx, Pitx, Sho2, Shh, Sox9, and TGF-β, among others. It is also well documented that epigenetic factors, such as anoxia due to cigarette smoking and alcohol, have a major impact on nonsyndromal cleft lip and palate.

Development of the nasomaxillary complex proceeds laterally and anteroposteriorly with expansion of the brain and cranial cavity and expansion of the oral cavity and oronasal pharynx. Also throughout the fetal period, anterior and inferior growth of the nasal septal cartilage, which is an extension of the anterior cranial base, is most prominent. The cartilaginous nasal capsule, which envelops the nasal cavity laterally, is primarily structural and contributes little to the overall growth of the nasomaxillary complex other than possible expression of growth factors that support the facial sutures (Fig. 1-20). Thus, the primary factors influencing the growth of the nasomaxillary complex from the late embryonic period and throughout the fetal period and the juvenile period postnatally are an expansion of the brain and cranial vault and growth of the anterior cranial base, including in particular anterior and inferior growth of the nasal septum, as well as expansion of the nasal cavity and oronasal pharynx.

Postnatal Growth of the Midface

At the time of birth, the midface is well developed but diminutive relative to the neurocranium. The circummaxillary and intermaxillary sutures are all present and active as sites of bone growth. The nasal capsule and midline nasal septum are still primarily cartilaginous and continuous with the rest of the

FIGURE 1-20 Frontal histologic sections of human fetuses at approximate ages of 5 weeks' gestation (**A**) and 11 weeks' gestation (**B**) (hematoxylin and eosin–stained). *NS*, Nasal septal cartilage; *NC*, nasal capsular cartilage; *V*, vomer; *PS*, palatal shelves.

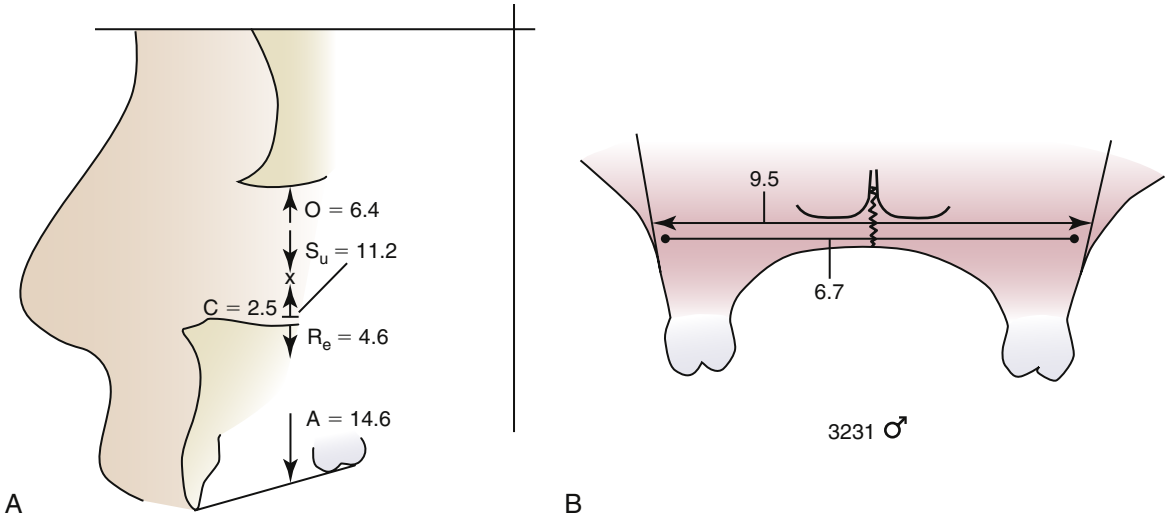


FIGURE 1-21 (A) Sutural displacement (S_u), apposition of the orbital floor (O), resorption of the nasal floor (R_e), apposition at the infrazygomatic crest (C), and dentoalveolar development (A) from 4 years of age through adulthood in nine boys. (B) Width changes (mm) of the maxilla and lateral implants between 3.9 and 17.7 years of age. (From Björk A, Skieller V: Postnatal growth and development of the maxillary complex. IN McNamara JA Jr, ed.: Factors affecting the growth of the midface, Ann Arbor, MI: Center for Human Growth and Development, Michigan Craniofacial Growth Series; 1976:61-100.)

chondrocranium from the anterior cranial base. The septum is also very actively growing by means of interstitial cartilaginous growth, leading to significant anterior and vertical growth of the midface, especially during the first 3 to 4 years of life.

With the exception of the nasal septum, postnatal development of the nasomaxillary complex occurs via intramembranous ossification. Growth at the circummaxillary and intermaxillary sutures occurs in response to midfacial displacements due principally to growth of the anterior cranial base and nasal septum. Inferior, anterior, and lateral displacements of the midface result in concomitant compensatory sutural growth to account for the majority of vertical, anteroposterior, and transverse changes that occur during both childhood and adolescence (Fig. 1-21). Along with displacements, extensive surface modeling takes place over the entire nasomaxillary complex, especially along its posterior and superior aspects.

As long as the midface undergoes displacement, sutural growth occurs, with the amounts of bony apposition being related directly to amounts of sutural separation. Growth continues until the sutures are no longer separating. The premaxillary/maxillary suture fuses at approximately 3 to 5 years of age.[52] The midpalatal and transpalatal maxillary sutures, which are the major intermaxillary growth sites associated with transverse and anteroposterior maxillary growth, have been reported to close between 15 and 18 years of age[53] and 20 to 25 years of age,[54] depending on the criteria on which closure is based. More recent studies suggest only limited amounts of sutural obliteration (i.e., the development of bony bridges, or spicules, running across the suture after growth has ceased) in adult midpalatal sutures.[55,56] The increasing complexity that characterized sutures during childhood and adolescence appears to be functionally, rather than age, related.[57] Although data are limited, it appears that closure of the circummaxillary sutures occurs somewhat later than closure of the intermaxillary sutures.

The midface undergoes a complex modeling pattern throughout childhood and adolescence (Fig. 1-22).[58] As the midface is displaced anteriorly, compensatory bony deposition occurs

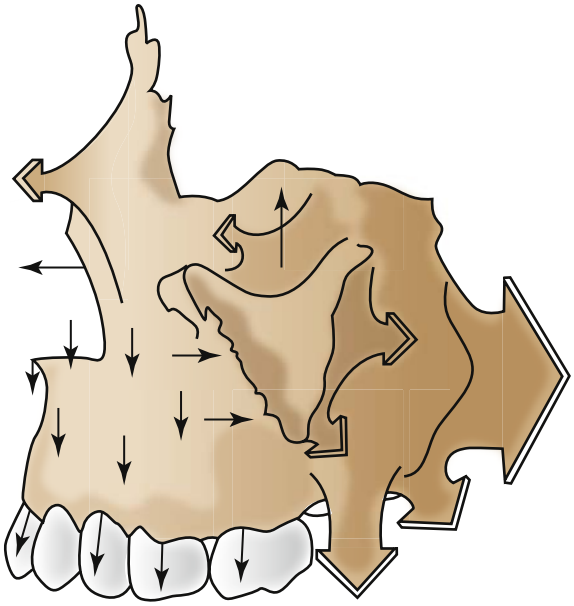


FIGURE 1-22 Maxillary remodeling, with the sizes of the arrows indicating relative amounts of change and with *dark* and *light arrows* indicating resorption and apposition, respectively. (Redrawn from Enlow DH, Bang S. Growth and remodeling of the human maxilla. *Am J Orthod.* 1965;51:446–464.)

along the posterior margin of the maxillary tuberosity, resulting in an increase in the length of the entire maxilla and of the dental arches.[59] The posterior maxilla is a major modeling site that accounts for most of the increases in maxillary length. The anterior periosteal surface of the maxilla is slightly resorptive, while the buccal surfaces undergo substantial bony deposition. From the sagittal perspective, the area of the anterior nasal spine drifts inferiorly; the A-point also drifts inferiorly and slightly posteriorly. For every 4 mm that the posterior nasal spine drifts posteriorly, it drifts approximately 3 mm inferiorly. Associated

with inferior displacement of the midfacial complex, bony resorption occurs along the floor of the nasal cavity, whereas apposition occurs on the roof of the oral cavity (i.e., palate) and orbital floor. Implant studies suggest that for every 11 mm of inferior midfacial displacement, the orbital floor drifts superiorly 6 mm and the nasal floor drifts inferiorly 5 mm.[60] Thus, midfacial height increases due to the combined effects of inferior cortical drift and inferior displacement (see Fig. 1-19). The height of the midface is further increased by continued development of the dentition and alveolar bone. The lack of naturally stable structures on the surface of the midfacial complex makes superimposition difficult.

The width of the midface at the time of birth is proportionately large due to the precocious development of the eyes, which are the central features of the neonatal midface. Growth in width during the first 2 to 3 years after birth is associated with expansion of the brain laterally and anteroposteriorly, which brings the eyes laterally with it. As this occurs, the sutures separating the two halves of the frontal bone (metopic suture), the two nasal bones (internasal suture), the two maxillae (intermaxillary suture), and the two palatine bones (midpalatal suture) are positioned to respond by secondary, compensatory bone formation. It has been estimated that the midalveolar and bijugale widths of the maxilla increase approximately 5 mm and 6 mm, respectively, between 7.6 and 16.5 years of age; rates of growth in width diminish slightly with increasing age.[61]

At the same time that the midface is increasing in width, it is increasing even more dramatically in depth (anteriorly) and height (vertically). The midface increases most in height, next in depth, and least in width. As the brain and eyes grow anteriorly relative to the middle cranial base, the orbits increase in depth and the anterior cranial base lengthens, primarily as a result of growth at the sphenoethmoidal synchondrosis. Concomitantly, the nasal septum grows vertically as the midface is displaced inferiorly relative to the anterior cranial base. The combination of these two growth processes—growth in a vertical direction associated with interstitial cartilaginous growth within the nasal septum and growth in an anterior direction associated with interstitial cartilage growth within both the nasal septum and synchondroses of the cranial base—results in the typical downward and forward growth of the entire midface relative to the anterior cranial base. Surface deposition cannot account for the downward and forward midfacial growth that occurs during childhood and adolescence.

The age of approximately 7 years is something of a benchmark for growth of the midface. Growth of the central nervous system—the brain and eyes—is essentially complete at about 7 years of age. Concomitantly, the cartilage of the sphenoethmoidal synchondrosis ossifies and a suture is formed between the sphenoid and ethmoid bones at about that time. As a result, a relatively stable anterior cranial base is established extending from the sella turcica to the foramen cecum. Also at about 7 years of age, the growth of the cartilages of the nasal capsule and nasal septum changes significantly. The cartilaginous nasal capsule becomes ossified, and the nasal septum, which remains cartilaginous throughout life in humans, decreases significantly in growth activity. Despite these important developmental changes in the growth processes of the midface, downward and forward skeletal growth continues to be significant over the next decade or so, particularly in males during adolescence.

Growth of the nasomaxillary complex continues throughout childhood and adolescence, with substantially greater vertical

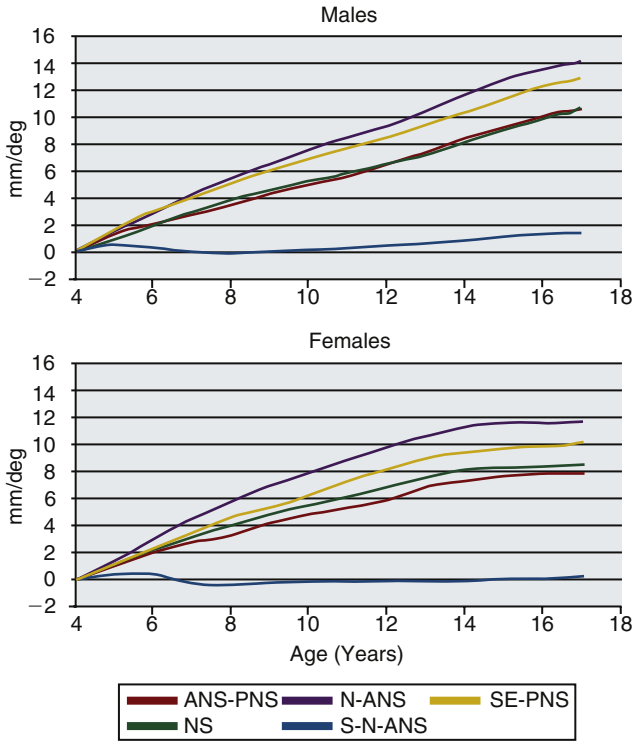
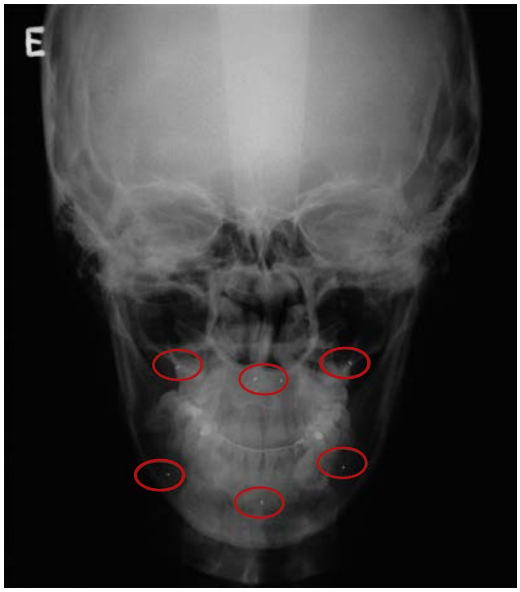


FIGURE 1-23 Maxillary growth changes between 4 and 17 years of age of males and females. (Adapted from data provided by Bhatia SN, Leighton BC. *A Manual of Facial Growth: a Computer Analysis of Longitudinal Cephalometric Growth Data.* New York: Oxford University Press; 1993.)

than anteroposterior growth potential (Fig. 1-23). By 4.5 years of age, palatal length (anterior nasal spine–posterior nasal spine) and anterior facial height (nasion–anterior nasal spine) have attained approximately 80% and 73% of their adult size, respectively (see Fig. 1-18). In terms of absolute growth, midfacial heights should be expected to increase 10 to 12 mm in females and 12 to 14 mm in males between 4 and 17 years of age. Palatal length should be expected to increase 8 to 10 mm over the same time period. Because nasion drifts anteriorly at approximately the same rate as the midface is displaced anteriorly, the sella–nasion–anterior (SNA) nasal spine angle shows little or no change during childhood or adolescence. Although vertical maxillary growth rates peak during adolescence, at approximately the same time as stature, anteroposterior maxillary growth remains more or less constant, with no distinct adolescent spurt.

Because the displacements are not parallel, the midface undergoes varying amounts of vertical and transverse true rotation. True rotation is independent of surface modeling and refers to changes that occur over time in the positions of basal bone; it is commonly assessed with metallic implants placed into the mandibles and maxillae of growing children.[62] From the sagittal perspective, most children undergo true forward or counterclockwise (subject facing to the right) rotation of the midface, due to greater inferior displacement of the posterior than anterior maxilla. The true rotation that occurs tends to be covered up or hidden by the resorption that occurs on the nasal floor. For example, true forward rotation is associated with greater resorption in the anterior than posterior aspect of the nasal floor. Due to greater transverse displacements



References	Ages (Years)	*Mx*	*Md*
Björk and Skieller, 1977	4-21	.42	N/A
Korn and Baumrind, 1990	8.5-15.5	.43	.28
Gandini and Buschang, 2000	13.9-16.7	.27	0.19
Iseri and Solow, 2000	7-12	N/A	.22
	13-18	N/A	.13

FIGURE 1-24 Transverse expansion (mm/yr) of metallic bone markers inserted into the maxillary (*Mx*) and mandibular (*Md*) basal structures.

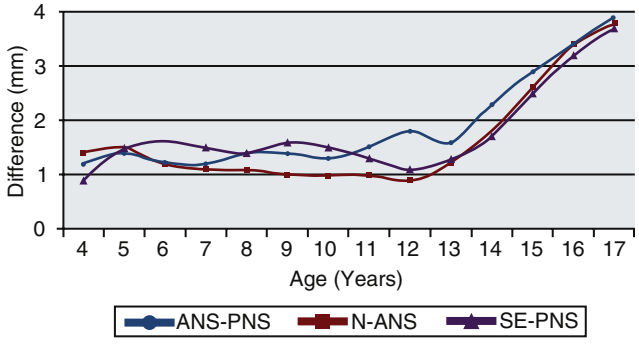


FIGURE 1-25 Sex differences (male minus female) in maxillary size. (Adapted from data provided by Bhatia SN, Leighton BC. *A Manual of Facial Growth: a Computer Analysis of Longitudinal Cephalometric Growth Data.* New York: Oxford University Press; 1993.)

posteriorly than anteriorly, the midfacial complex also exhibits transverse rotation around the midpalatal suture (Fig. 1-24). As a result, there is greater sutural growth in the posterior than anterior aspect of the midpalatal suture. Cephalometric analyses using metallic implants have shown that the posterior maxilla expands approximately 0.27 to 0.43 mm/yr, with greater expansion occurring during childhood than during adolescence.[60]

There are definite sex differences in maxillary postnatal growth (Fig. 1-25), with males being larger and growing more than females. Size differences, averaging between 1 mm and 1.5 mm, are small but consistent during infancy and childhood. Sexual dimorphism increases substantially throughout the midfacial complex during adolescence, with differences of approximately 4 mm in maxillary length (anterior nasal spine to posterior nasal spine [ANS-PNS]) and upper facial height (nasion to anterior nasal spine [N-ANS]) at 17 years of age. Males also have significantly wider midfaces than females, with differences approximating 5 to 7 mm during late adolescence.[63] The primary reason that adult males are larger than adult females is the 2 extra years of childhood growth that males have; males enter the adolescence phase of growth at approximately 12 years of age, while females enter around 10 years. Males are also larger than females because they experience a more intense adolescent spurt, but this contributes less to the sex differences observed.

MANDIBLE

Development of the Mandible

The mandible develops bilaterally within the mandibular processes of the first branchial arch. Each embryonic mandibular process contains a rod-like cartilaginous core, Meckel's cartilage, which is an extension of the chondrocranium into the viscerocranium. Throughout its course, distally Meckel's cartilage is accompanied by the mandibular division of the trigeminal nerve (CN V), as well as the inferior alveolar artery and vein. Proximally, Meckel's cartilage articulates with the cartilaginous cranial base in the petrous region of the temporal bone, where it gives rise to the malleus and incus bones of the inner ear.

By 6 weeks' gestation, a center of ossification appears in the perichondrial membrane lateral to Meckel's cartilage. It is critical to note that ossification of the mandible takes place in membrane *lateral and adjacent* to Meckel's cartilage, and *not within* Meckel's cartilage itself (Fig. 1-26). Therefore, it is clear that the mandible develops and subsequently grows by means of intramembranous ossification and not through endochondral ossification and replacement of Meckel's cartilage. The only portion of the developing lower jaw that appears to be derived from endochondral ossification of Meckel's cartilage is the mental ossicles, which are two very small sesamoid bones that are formed in the inferior aspect of the mandibular symphysis.[65] These bones are no longer present at the time of birth.

Intramembranous ossification of the body of the mandible proceeds distally toward the mental symphysis and proximally up to the region of the mandibular foramen. As it does so, Meckel's cartilage begins to degenerate and involute as the inferoalveolar neurovascular bundle becomes progressively enveloped by the intramembranously developing mandibular bone. Meckel's cartilage completely disappears by approximately 24 weeks' gestation, remaining in remnant form as the dense sphenomandibular ligament and giving rise to the malleus and incus ear ossicles.

Initial evidence of the formation of the temporomandibular joint (TMJ) is seen upon expression of the Barx-1 homeobox gene. By approximately 8 weeks' gestation, the condylar process appears as a separate carrot-shaped blastema of cartilage extending from the ramus proximal to the mandibular foramen and extending up to articulate with the squamous (membranous) portion of the developing temporal bone. Formation of

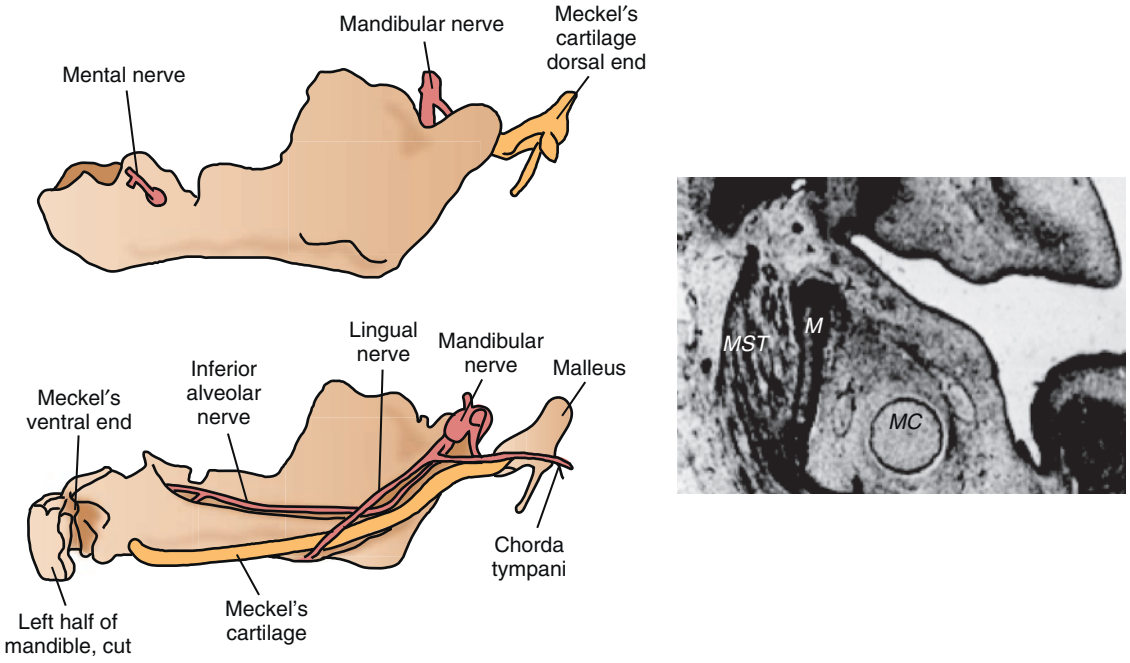


FIGURE 1-26 Drawings of a fetal mandible with lateral (*top left*) and medial (*bottom left*) views. *Right*, Photomicrograph of coronal view of human fetus indicating Meckel's cartilage medial to the mandible (*M*). *MST*, Masseter muscle. (Drawings adapted from Warwick R, Williams PL, eds. *Gray's Anatomy*. 35th ed. Philadelphia: WB Saunders; 1973.)

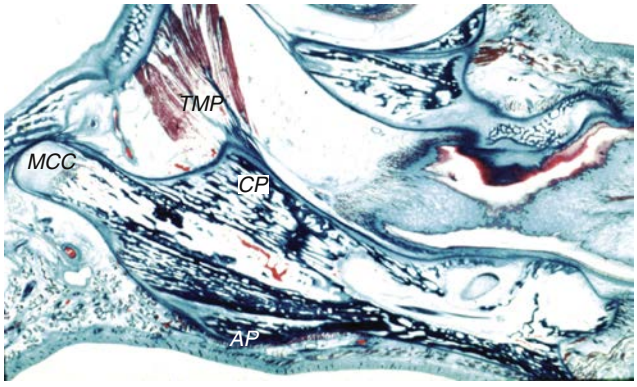


FIGURE 1-27 Parasagittal histologic section of human fetus (approximately 12 weeks' gestation) (hematoxylin and eosin–stained). *MCC*, Mandibular condylar cartilage; *CP*, coronoid process; *AP*, angular process; *TMP*, temporalis muscle.

the joint cavity between the condylar process and the squamous portion of the temporal bone is essentially completed as the TMJ by about 12 weeks' gestation (Fig. 1-27).

As the cartilage comprising the mandibular condyle arises "secondarily" within a skeletogenic membrane and apart from the primary embryonic cartilaginous anlagen, it is referred to as a *secondary cartilage* (Fig. 1-28). Secondary cartilage is a unique type of skeletal tissue that has the characteristics of both intramembranous bone and certain histologic and functional features of hyaline growth cartilage. Secondary cartilage is formed in areas of precocious stresses and strains within intramembranous bones, as well as in areas of rapid development and growth of bone.[65,66] Within the craniofacial complex, the angular and the coronoid processes of the mandible also may exhibit the presence of secondary cartilage because these are sites of very

FIGURE 1-28 Frontal histologic section of a human fetus (approximately 8 weeks' gestation) (hematoxylin and eosin–stained). The bone comprising the body and ramus of the mandible (*M*) originates in the membrane lateral to Meckel's cartilage (*MC*). The periosteal membrane enveloping the mandible gives rise secondarily to the mandibular condylar cartilage (*MCC*).

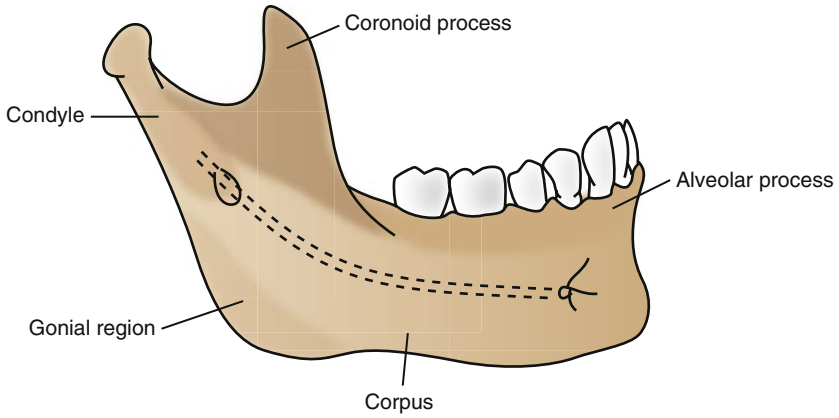


FIGURE 1-29 Major functional units of the mandible.

rapid bone growth associated with the function of the muscles of mastication. In addition, secondary cartilage may be found in areas of sutures that are characterized by rapid intramembranous bone growth and biomechanical load associated with separation and bending at the articular surfaces.

At birth, the two halves of the mandible are separated in the midline by a fibrous articulation, the mental symphysis, which will fuse by the end of the first year of life. Each half of the mandible is characterized anatomically by (1) a *condyle* and *condylar process*, which articulates with the temporal bone to make up the TMJ; (2) a *ramus*, which extends roughly vertically–inferiorly from the condylar process and provides insertions for the muscles of mastication; and (3) a *corpus*, or body, which extends roughly horizontally–anteriorly to provide a base for the mandibular dental arch and house the inferior alveolar–neurovascular bundle. Each of these anatomic structures can also be considered in terms of overlapping functional units (Fig. 1-29). The mandibular condyle and condylar processes obviously are essential for normal articular function of the TMJ and movements of the mandible, while at the same time playing a significant role in mandibular growth for most of the first two decades of life.[67] Variation in the function of the TMJ, such as might occur in association with differences in mastication, jaw movements, and jaw position, for example, is highly likely to affect its growth and form. The gonial region of the mandible, at the inferior aspect of the ramus, is related to the function of the masseter and medial pterygoid complex of muscles, while the coronoid process is primarily related to the temporalis muscle. Variation in the growth and form of each of these regions is due in large part to variation in the function of the muscles of mastication. The alveolar process of the mandible functions to provide support for the dentition. Finally, the body of the mandible, extending from the mandibular foramen to the mental process, provides support and structural connection between the various functional components of the mandible.

Growth of the Mandibular Condyle

Just as a suture can be considered to be a specialization of an osteogenic membrane (i.e., periosteum and dura mater), the condylar cartilage can also best be considered to be a specialization of periosteum. As with sutures, growth of the mandibular condyle tends to be relatively highly responsive to mechanical, functional, and hormonal stimuli both at the time of development and throughout the growth period, similar to intramembranous bone development elsewhere.

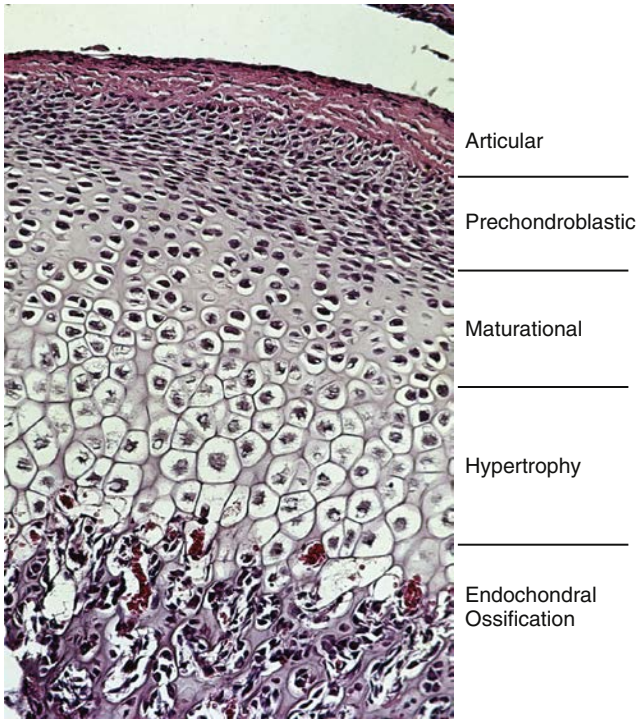


FIGURE 1-30 Histologic section indicating the various layers of the secondary cartilage in a growing mandibular condyle (hematoxylin and eosin–stained).

Histomorphology of the Growing Condyle

A number of similar but somewhat different terms have been used to describe the histomorphology of the growing mandibular condyle.[68] These are summarized according to their equivalencies in E-Table 1-2.

The secondary cartilage composing the condyle during growth can be divided into two general layers: an articular layer and a growth layer. The more superficial *articular layer* is continuous with the outer fibrous layer of the bilaminar periosteum encapsulating the condylar neck and temporal bone, respectively. Deep to the articular layer is a subarticular *growth layer*. The growth layer of the condylar cartilage is organized into an additional series of layers or zones typical of growing cartilage that blend into each other (Fig. 1-30). Each of these zones is present in the neonate and remains in the condyle through

TABLE 1-2 Comparison of Terminology Used to Describe the Histomorphology of the Condylar Cartilage

Blackwood[1]	Durkin et al.[2]	Wright and Moffett[3]	Petrovic et al.[4]	Thilander et al.[5]	Carlson et al.[6]	Luder[7]
Articular zone	Resting surface articular layer	Articular layer	Fibrous capsule	Surface articular layer	Fibrous articular tissue	*Perichondrium* Articular layer
	Transitional or proliferative layer	Proliferative layer	Prechondroblastic layer	Proliferative layer	Prechondroblastic (proliferative) layer	Polymorphic cell layer
						Flattened cell layer (1 and 2)
Intermediate zone		Zone of matrix production	Zone of maturation	Hypertrophic zone (nonmineralized)	Chondroblastic zone (maturation and hypertrophy)	Hyaline cartilage
		Zone of cell hypertrophy	Functional chondroblasts			Flattened cell layer (3)
Hypertrophic cartilage		Zone of calcification and resorption	Hypertrophic chondroblasts	Hypertrophic zone (mineralized)		Upper hypertrophic cell layer
	Hypertrophic cartilage		Zone of erosion			Lower hypertrophic cell layer
	Erosion zone		Degenerating chondroblasts			
	Subchondral bone		Zone of endochondral ossification		Zone of endochondral ossification	

References

1. Blackwood HJJ. Growth of the mandibular condyle of the rat studied with titrated thymidine. *Arch Oral Biol.* 1966;11:493–500.
2. Durkin J, Heeley J, Irving JT. The cartilage of the mandibular condyle. *Oral Sci Rev.* 1973;2:29–99.
3. Wright DM, Moffett BC. The postnatal development of the human temporomandibular joint. *Am J Anat.* 1974;141:235–250.
4. Petrovic A, Stutzmann J, Oudet C. Control processes in the postnatal growth of the condylar cartilage. In: McNamara JA Jr, ed. *Determ Mandibular Form Growth.* Ann Arbor, MI: Center for Human Growth and Development, Craniofacial Growth Series; 1975:101–154.
5. Thilander B, Carlsson GE, Ingervall B. Postnatal development of the human temporomandibular joint. I. A histological study. *Acta Odontol Scand.* 1976;34:117–126.
6. Carlson DS, McNamara Jr JA, Jaul DH. Histological analysis of the growth of the mandibular condyle in the rhesus monkey (*Macaca mulatta*). *Am J Anat.* 1978;151:103–117.
7. Luder HU. Postnatal development, aging, and degeneration of the temporomandibular joint in humans, monkeys, and rats, Ann Arbor, MI: Center for Human Growth and Development, University of Michigan, Craniofacial Growth Series; 1996:32.

maturity. However, their absolute and relative size as well as their growth-related activity may vary considerably, depending on the overall rate and amount of condylar growth and on the functional requirements placed on the condyle and TMJ.[69,70]

Articular layer. The articular layer of the joint surface of the mandibular condyle and temporal portion of the TMJ consist of an avascular dense fibroelastic connective tissue whose collagen fibers are oriented parallel to the articular surface. The articular layer varies in thickness along the condylar head and temporal joint surface, increasing in thickness in the superior aspect of the condyle and on the articular eminence of the glenoid fossa, where compressive forces associated with mastication are greatest.[71] The fibrous articular layer of the mandibular condyle and that found in the glenoid fossa and articular eminence are identical functionally to the articular cartilage found in the diarthroideal joints of the postcranial long bones, but their origin and histologic composition are completely different. Articular cartilage is derived from the primary cartilaginous anlagen at the ends of long bones; the articular tissue of the TMJ is a specialization of the fibrous layer of periosteum that covers the mandible and temporal bone.

Growth layer. The growth layer immediately deep to the articular layer is comprised of a series of cellular zones representing the various stages of chondrogenesis in secondary cartilage. The *proliferative*, or *prechondroblastic*, *zone* immediately deep to the articular layer is continuous with the osteogenic layer of the periosteal membrane along the condylar neck.[72,73] Its outer portion is composed of undifferentiated mesenchymal cells that differentiate into skeletoblastic stem cells or prechondroblasts. Morphologically, this zone appears as densely packed with spindle-shaped cells that increase in size and become increasingly separated due to production of intercellular matrix within the inner region of the proliferative zone. The newly formed cartilage cells in the proliferative zone express type I collagen, which is characteristic of bone and underscores the fact that the source of these cells is a periosteal-like membrane. Recent studies of gene expression in the proliferative zone demonstrate that the prechondroblastic layer is also characterized by high expression of FGF-13, FGF-18, TGF-β2, Igf-1, and VEGF.[74,75]

The *zone of maturation* contains larger, spherical, maturing chondrocytes arranged in an apparently random fashion. These cartilage cells undergo very few mitoses, which is atypical for cartilage cells found in a growing epiphyseal plate. In addition, there is significantly less extracellular matrix in the mandibular condylar cartilage than is found in the growth plates of developing long bones, which are comprised of primary cartilage. Cartilage cells within the zone of maturation are capable of switching their phenotype to express type II collagen, which is typically expressed by primary cartilage in growing epiphyses in response to biomechanical load.

Cartilage cells in the *zone of hypertrophy* become progressively larger through osmotic activity and absorption of water. Their nuclei become pyknotic and their cytoplasm is increasingly evacuated as the cells are about to be encroached upon by the osteoblasts from the endosteal region of the condyle. Genes for procollagen, aggrecan, Sox9, and Ihh are highly expressed in the chondroblastic layer.[74]

The *zone of endochondral ossification* is characterized by the initiation of mineralization of the intercellular matrix within the distal-most three to five layers of hypertrophying cells. This matrix is subsequently eroded away by osteoclastic activity and replaced by bone. The process of endochondral ossification associated with the condylar cartilage is identical to the process that takes place in the primary cartilage of long bone epiphyses.

Age-Related Changes in the Mandibular Condyle

Detailed histologic analysis of human autopsy specimens of the human TMJ has demonstrated progressive changes in the thickness and presumed growth activity of the condyle cartilage throughout development.[76–79] These changes appear to be coordinated with functional changes associated with occlusal development.[80,81] In general, the combined growth-related layers of the condylar cartilage begin as a relatively thick structure in the neonate (1.25 to 1.5 mm thick) but become much thinner (0.3 mm) by the mixed dentition stage. The cartilage remains generally thin but well defined and actively growing in the permanent dentition stage until, by age 20 to 30 years, the cartilage essentially disappears and the condyle is capped by a bony plate. Even in adults, however, it is not unusual to see areas of hyaline cartilage ("cartilage islands") deep to the articular layer in the condyle.

The subarticular region of the temporal component of the TMJ has the same tissue layers as the condyle; however, they are substantially less prominent. Morphologically, the temporal component of the TMJ in the neonate is essentially flat, and the articular disc interposed between the condyle and temporal bone is highly vascular. During the period of the primary dentition, at approximately 3 years of age, the temporal surface takes on its characteristic S-shaped contour, and the articular disc becomes avascular in its central region. Thereafter, the temporal surface of the TMJ grows more slowly, with the mandibular fossa becoming deeper as the articular eminence becomes steeper; this happens primarily through the process of bone deposition on the articular eminence and, to a lesser extent, by resorption of bone in the posterosuperior region of the fossa, as well as endosteal deposition in the superior aspect of the fossa. This increase in the contour of the temporal component of the TMJ normally continues until the fourth decade of life.

In summary, the mandibular condylar cartilage is a secondary cartilage that in subadult individuals serves both as a site of growth and as a place of articulation. As such, it displays functional characteristics of both a growth plate and an articular cartilage, but it differs from both in fundamental aspects of its development and structure throughout ontogeny. Its most superficial layers are not cartilaginous in phenotype but rather are perichondrial in origin. Importantly, the chondrocytes of the mandibular condylar cartilage are derived via mitosis in cells that are themselves not chondrocytes, similar to embryonic cartilage but not to the growth plate in which the cells that proliferate are chondrocytes. Finally, the prechondrogenic phenotype of these dividing cells in the mandibular condylar cartilage can be readily modulated to a preosteogenic phenotype by changes in the periarticular environment. Taken together, these features define a tissue with structural and growth characteristics that are consistent with the concept of an adaptive, compensatory growth site and set it apart from primary cartilaginous growth centers.

Mechanisms of Condylar Growth

The mandibular–condylar cartilage was initially considered to be a growth center with an intrinsic capacity for tissue-separating growth. However, it is now generally understood that growth of the mandibular–condylar cartilage is highly

adaptive and responsive to growth in adjacent regions, particularly the maxilla. Numerous experimental studies were conducted over the past several decades to assess the role that function and jaw position, in particular, might play in influencing the postnatal growth of the mandibular condyle. For example, in a number of studies involving anterior postural change of the mandible using rats[82,83] and primates[84] as experimental animals demonstrated significant increases in the overall length of the mandible. From these experiments, Petrovic and colleagues developed a "cybernetic" model of mandibular growth regulation referred to as the "servosystem hypothesis of mandibular growth" (Fig. 1-31).[85,86]

There has been a significant expansion of knowledge concerning the molecular biology and cellular dynamics associated with growth of the condylar cartilage. It has been shown, for example, that fibroblast growth factor (FGF) and insulin-like growth factor (IGF) are present in the matrix and cell surfaces of the condylar cartilage and that they vary according to their specific location, much like in sutures. Less is known of the presence or importance of transforming growth factor-beta (TGF-β) or other growth factors, and knowledge of hormonal influences on growth of the condylar cartilage is even more rudimentary and somewhat contradictory.[87–89]

Several studies have begun to explore the molecular basis for the effect of mandibular function and position on mandibular growth by using appliances that replicate the effects (e.g., increased mitotic activity, cartilage thickness) reported previously.[90–93] Fuentes and coworkers[94] used a novel incisor-borne appliance that prompted a crossbite in growing rats and produced a differential change in proliferation and cartilage thickness between the crossbite and noncrossbite sides. In animals wearing the appliance, gene expression for *IGF-1* and *FGF-2* and their receptors in condylar cartilage was altered from that in control rats. The changes in gene expression, which typically preceded the changes in mitotic activity and cartilage thickness, were in most instances opposite in direction between the crossbite and noncrossbite sides. Using a similar design, Hajjar and associates[95] found that rats fitted with an incisor-borne appliance that prompted anterior displacement of the mandible exhibited increased expression of both IGF-I and IGF-II mRNA and protein in the MCC. Rabie and colleagues[90,96,97] demonstrated that the expression of Sox9, type II collagen, and Indian hedgehog (Ihh) was increased in the condylar cartilage and glenoid fossa of rats wearing the appliance for 1 to 2 weeks.

In general, these findings parallel the findings discussed previously for development of the sutures of the cranial vault. These similarities between the condylar cartilage and sutures should not be surprising given the periosteal origin of both suture mesenchyme and the secondary cartilage of the mandibular condyle.

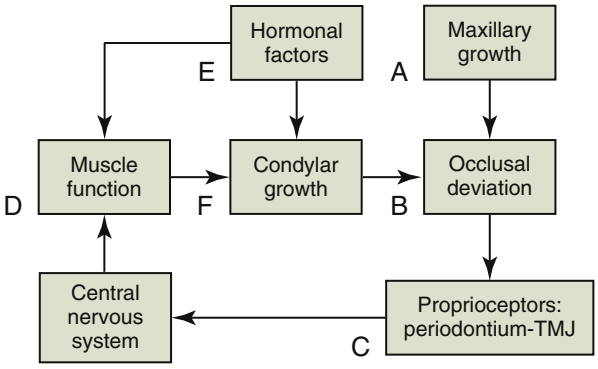


FIGURE 1-31 Simplified explanation of Petrovic's "servosystem hypothesis of mandibular growth." Independent growth of the maxilla (A) creates a minor occlusal deviation between the upper and lower dentition (B). This occlusal deviation is perceived by proprioceptors (C), which provide a signal to the muscles responsible for jaw protrusion to be tonically more active (D), which causes the mandibular condyle to become slightly more anteriorly located within the temporomandibular joint, thus stimulating condylar growth (F). Muscle function and the adaptive capacity of the condyle for growth are enhanced by expression of hormonal factors (E), and thus condylar growth may vary depending on the maturational and hormonal status of the individual. (Adapted from Carlson DS. Theories of craniofacial growth in the postgenomic era. *Semin Orthod.* 2005;11(4):172–183.)

Postnatal Growth of the Mandible

At birth, the ramus of the mandible is quite short, both in absolute terms and in proportion to the mandibular corpus. During postnatal development, the ramus becomes much more prominent, particularly in height but also in width. At the same time, the corpus increases in length, providing the necessary space for development and eruption of the mandibular dentition. Associated with these early postnatal changes in the absolute and relative sizes of the mandible are decreases in the gonial angle between the ramus and corpus and increases in the angle between the two corpora.

The mandible has the greatest postnatal growth potential of any component of the craniofacial complex. Growth changes that occur are closely associated with the functional processes that comprise the mandible, including the gonial process, coronoid process, alveolar process, and bony attachments of the suprahyoid muscles, which are all major sites of postnatal modeling. Although condylar growth is often assumed to be the mandible's primary growth site, it is important to note that the entire superior aspect of the ramus displays approximately the same amount of growth.

Viewed in its lateral projection, the posteroinferior and superior border of the ramus, including the condyle, and the posterosuperior aspect of the coronoid process are depository throughout the period of active growth. The anterior and lower borders (extending approximately to the first molars) of the ramus of the mandible are resorptive. Resorption of bone continues to occur along the anterior border of the ramus, resulting in a longer corpus and increased space for the development and eruption of the mandibular dentition (Fig. 1-32).[98] Within the corpus, the greatest growth changes are appositional growth of the alveolar bone associated with dental development and eruption. The symphysis, especially the superior aspect, becomes wider due to superior and posterior drift of its posterior aspect (Fig. 1-33).[99] There is resorption on the anterior aspect of the symphysis above the bony chin. The cortical region at or just above the chin is the only place on the entire surface of the mandible that remains stable (i.e., does not model) during postnatal growth, which is why it serves as an important site for superimposing successive radiographs. The inferior aspect of the anterior corpus tends to be depository, but the amounts of bone added are limited and variable.

Widening of the body of the mandible occurs through deposition of bone along the buccal surface and transverse rotation of the right and left corpii. The mandible also widens due to bony deposition along its posterior surface, which, due to its posterolateral orientation, produces a longer and wider body. Growth in width of the superior aspect of the ramus is somewhat more complex due to the substantial increases in height that occur. Viewed in a coronal projection, the superior aspect of the ramus and coronoid process are canted somewhat mediolaterally. As the mandibular corpus and inferior aspect of the ramus increase in width by deposition along the buccal surface, the buccal surface of bone on the superior aspect of the ramus is resorptive, while the lingual and superior surfaces of bone are depository.

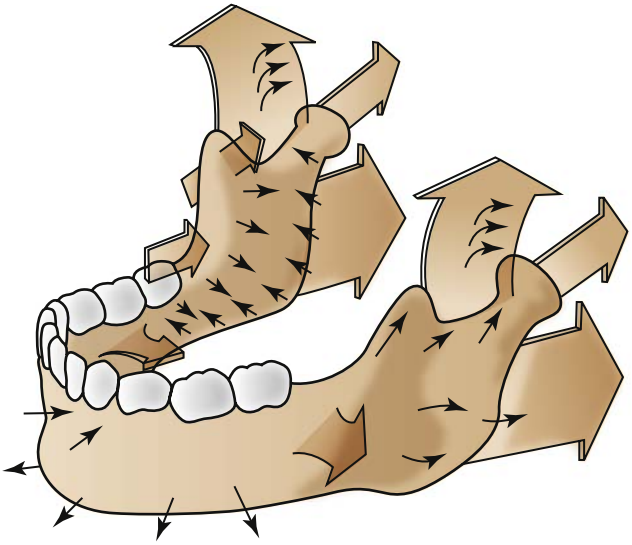


FIGURE 1-32 Mandibular remodeling, with the sizes of the arrows indicating relative amounts of change and with dark and light arrows indicating resorption and apposition, respectively. (Adapted from Enlow DH, Harris DB. A study of the postnatal growth of the human mandible. *Am J Orthod*. 1964;50:25.)

The greatest postnatal changes in mandibular growth also occur during infancy, with overall length (condylion to gnathion [Co–Gn]) increasing 15 to 18 mm during the first year, 8 to 9 mm during the second year, and then slowing down to increase approximately 5 mm during the third year. During these early years, condylar growth and modeling of the superior aspects of the ramus are directed posteriorly and superiorly, with roughly equal amounts of growth in each direction. This orientation is important because it rapidly increases corpus length to make room for the rapidly developing dentition. After the first few postnatal years, growth of the condyle and superior ramus slows down dramatically and changes orientation toward a predominant superior direction.

By 4.5 years of age, ramus height has attained approximately 64% and 70% of its adult size for males and females, respectively (see Fig. 1-18). Corpus length (Go–Gn) closely approximates the maturity pattern of midfacial height; it remains more mature than ramus height throughout postnatal growth. This supports the general principle that the vertical aspects of craniofacial growth are less mature and have greater postnatal growth potential than the anteroposterior aspects. Total mandibular length (condylion to menton [Co–Me]) undergoes the greatest increases in length (approximately 25 mm and 30 mm for females and males, respectively) between 4 and 17 years of age, followed by corpus length (gonion to pogonion [Go–Pg]; approximately 18 and 22 mm for females and males, respectively) and ramus height (condylion to gonion [Co–Go]; approximately 14 and 17 mm for females and males,

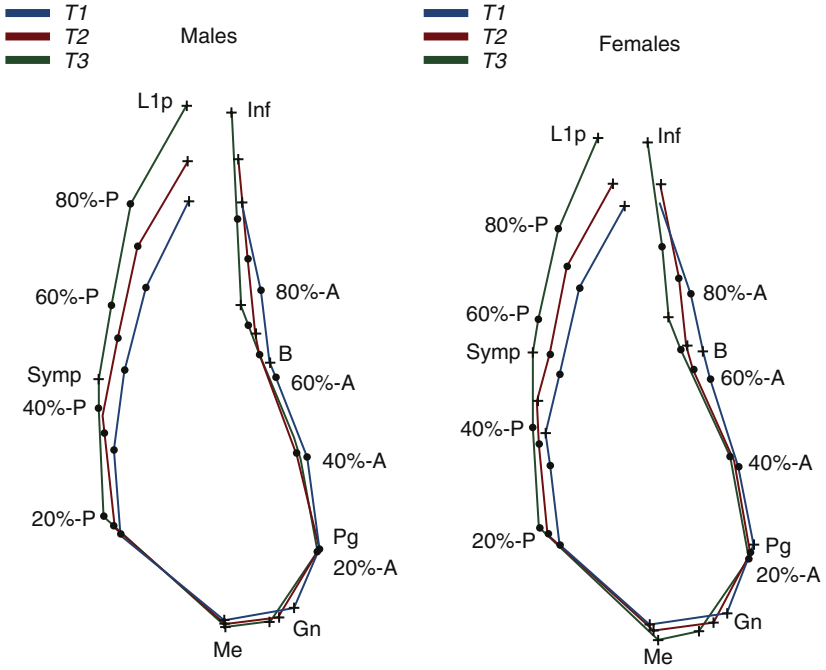


FIGURE 1-33 Remodeling changes of the symphysis between 6 (*T1*), 10 (*T2*), and 15 (*T3*) years of age. (Adapted from Buschang PH, Julien K, Sachdeva R, et al. Childhood and pubertal growth changes of the human symphysis. *Angle Orthod*. 1992;62:203–210.)

respectively) (Fig. 1-34). During later childhood and adolescence, the condyle shows substantially greater amounts of superior than posterior growth. For every 1 mm of posterior growth, there is 8 to 9 mm of superior growth. It has been estimated that the condyles of females and males grow 2 to 2.5 and 2.5 to 3.0 mm/yr, respectively, during childhood and adolescence, with the greatest rates occurring during the adolescent spurt (Fig. 1-35). The coronoid process and sigmoid notch follow similar growth patterns. Due to the resorption of bone that normally occurs in the gonial region, ramus height (measured from gonion to condylion) substantially underestimates the actual amount of growth that occurs at the condyle. There is approximately 1 mm of resorption at gonion for every 3 mm of superior condylar growth.[100] Between 7 and 15 years of age, biantegonial and bigonial widths increase approximately 10 mm and 12 mm, respectively.[61,63] Importantly, mandibular width continues to increase throughout childhood and adolescence. While an adolescent spurt in vertical mandibular growth certainly occurs, a pronounced spurt for the anteroposterior and transverse growth has not been established.

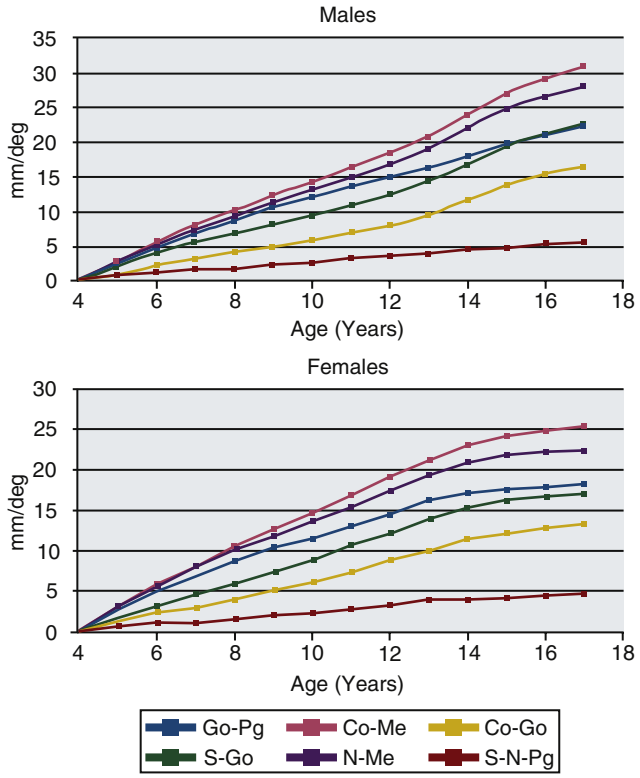


FIGURE 1-34 Mandibular growth changes between 4 and 17 years of age of males and females. (Adapted from data provided by Bhatia SN, Leighton BC. *A Manual of Facial Growth: a Computer Analysis of Longitudinal Cephalometric Growth Data.* New York: Oxford University Press; 1993.)

The mandible undergoes substantial amounts of true vertical rotation and more limited, but definite, transverse rotation. While the maxilla exhibits more transverse rotation, the mandible exhibits more vertical rotation than the maxilla. The typical pattern of vertical rotation is forward (counterclockwise with the subject facing to the right), due to greater inferior displacements of the posterior than anterior aspects of the mandible.[101] Rates of vertical mandibular rotation have been estimated to range between 0.4 and 1.3°/yr, with significantly greater rates of rotation during childhood than adolescence (Fig. 1-36). Although relatively few (<10%) children are "true" posterior rotators, up to 25% of adolescents have been reported to be posterior rotators.[80] Greater amounts of true mandibular rotation occur during the transition to the early mixed dentition than at any time thereafter.[102,103]

The mandible also rotates transversely due to greater expansion of the posterior than of the anterior aspects of the two corpii. This type of rotation has been demonstrated repeatedly in subjects with metallic implants and represents expansion of basal bone. It has also been shown that, when viewed from frontal projects, the right and left mandibular nerves are displaced laterally throughout growth. Transverse rotation is also age related, with greater amounts occurring during childhood than adolescence. The posterior aspect of the mandible expands approximately 65% to 70% as much as the posterior maxilla expands at the posterior aspect of the midpalatal suture (see Fig. 1-20).

As in the rest of the craniofacial complex, sex differences in mandibular growth are evident at the earliest ages and become pronounced during adolescence. At birth, males have significantly larger mandibles than do females. Sex differences, which are greatest for overall length, followed by corpus length and ramus height, respectively, range from 0 to 2 mm between 1 and 12 years of age, when males initiate their adolescent phase of

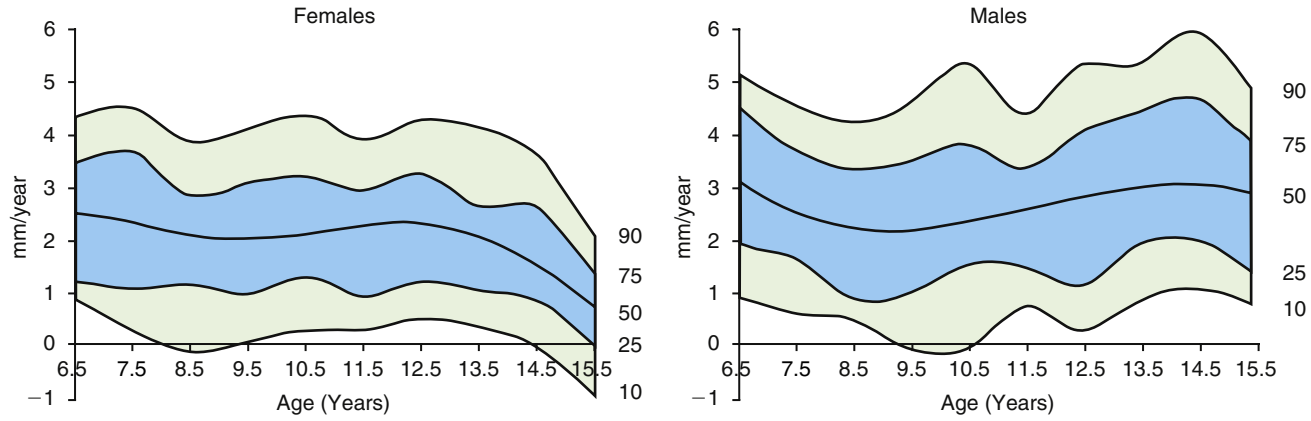
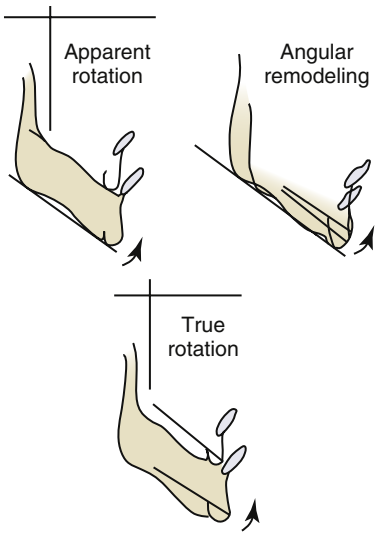


FIGURE 1-35 Percentile curves for condylar growth of females and males. (Adapted from Buschang PH, Santos Pinto A. Condylar growth and glenoid fossa displacement during childhood and adolescence. *Am J Orthod Dentofac Orthop.* 1998;113:437–442.)



References	Ages	deg/yr
Odegaard 1970	7-14	0.8
Lavergne and Gasson 1977	7-19	0.9
Skieller et al. 1984	Adolescence	1.0
Spady et al. 1992	Childhood	0.9
	Adolescence	0.4
Miller and Kerr, 1992	5-10	1.3
	10-15	0.8
Karlsen, 1995	6-12 (high angle)	0.7
	6-12 (low angle)	1.3
	12-15 (high angle)	0.7
	12-15 (low angle)	1.3
Wang et al. 2009	5.6-8.5	1.3
	8.5-15.5	0.7

FIGURE 1-36 True mandibular rotation (degrees per year) during childhood and adolescence.

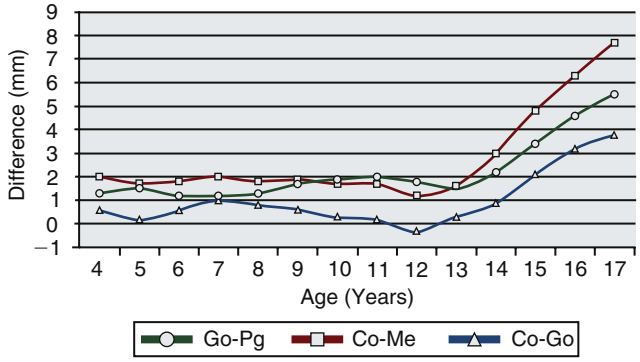


FIGURE 1-37 Sex differences (male minus female) in mandibular size. (Adapted from Bhatia SN, Leighton BC. *A Manual of Facial Growth: a Computer Analysis of Longitudinal Cephalometric Growth Data*. New York: Oxford University Press; 1993.)

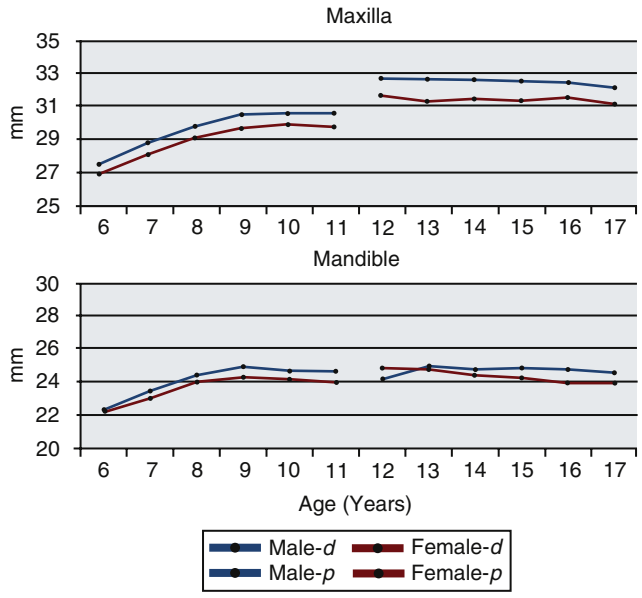


FIGURE 1-38 Maxillary and mandibular intercanine widths of males and females based on measurements taken from the deciduous (*d*) and permanent (*p*) canines. (Data from Moyers RE, van der Linden PGM, Riolo ML, et al. *Standards of Human Occlusal Development*. Ann Arbor, MI: Center for Human Growth and Development; 1976.)

growth. Mandibular dimorphism increases to 4 to 8 mm by the end of the adolescent growth phase (Fig. 1-37). There are no sex differences in vertical rotation during childhood or adolescence.

In summary, the mandible increases in size as a result of the combined processes of proliferation of secondary cartilage at the condyle and differential formation and modeling of bone along the entire surface of the mandible, particularly along its superior and posterior aspects. Growth of the mandible is expressed in a downward and forward direction relative to the cranium and cranial base. The mandible is typically displaced downward more than the maxilla, with the resulting space being taken up by the erupting dentition. Due to the geometry of the craniofacial complex, normal, coordinated growth of the jaws and a normal relationship of the associated occlusal arches require that the relative rate and amount of growth of the maxilla and mandible differ.

ARCH DEVELOPMENT, TOOTH MIGRATION, AND ERUPTION

The oral apparatus is the region of the craniofacial complex that holds the greatest potential for adaptive changes. Dental arch width and perimeter change dramatically, especially during the transitions to the early mixed and permanent dentitions.[104] Maxillary intercanine width increases approximately 3 mm during the transition to the early mixed dentition and an additional 2 mm with the emergence of permanent canines (Fig. 1-38).[105] Mandibular intercanine width increases approximately 3 mm during initial transition but shows little or no change with the eruption of the permanent canines. Intermolar widths progressively increase during childhood and adolescence, approximately 4 to 5 mm for the maxilla and 2 to 3 mm for the mandible between 6 and 16 years of age (Fig. 1-39). Maxillary arch depth (incisors to molars) decreases slightly during the transition to the early mixed dentition, increases 1 to 2 mm with the emergence of permanent incisors, and then decreases

approximately 2 mm with loss of the deciduous first and second molars. Mandibular arch depth decreases slightly during the transition to mixed dentition, maintains its dimension during most of the mixed dentition, and then decreases 2 to 3 mm with the loss of the deciduous first and second molars. Maxillary arch perimeter from first molars to first molars increases 4 to 5 mm during early mixed dentition and then decreases approximately 4 mm during late mixed dentition, resulting in only a slight overall increase between 5 and 18 years of age (Fig. 1-40). Mandibular arch perimeter, from first molar to first molar, on the other hand, increases approximately 2 mm during early mixed dentition and decreases 4 to 6 mm during late mixed dentition, resulting in overall decreases of 3.5 and 4.5 mm in males and females, respectively.

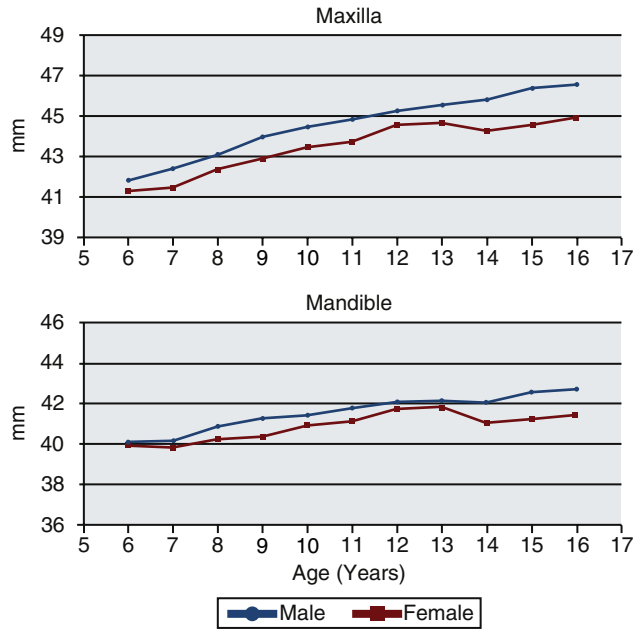


FIGURE 1-39 Maxillary intercanine width of males and females based on measurements taken from the deciduous (*d*) and permanent (*p*) canines. (Data from Moyers RE, van der Linden PGM, Riolo ML, et al. *Standards of Human Occlusal Development*. Ann Arbor, MI: Center for Human Growth and Development; 1976.)

Perhaps most important from a clinical perspective, the teeth continue to migrate and erupt throughout childhood and adolescence, even after they have attained functional occlusion. The posteruptive movements of teeth are directly related to the spaces created by growth displacements and movements of other teeth. Dentoalveolar compensation is the mechanism that coordinates their eruption and migration relative to their jaw bases; it maintains the relationships of teeth within and between the upper and lower dental arches. Dentoalveolar compensation depends on a normal eruptive system, dental equilibrium, and influences of neighboring teeth.[105] During childhood, the maxillary incisor drifts anteriorly at a greater rate than the maxillary molar (0.8 versus 0.6 mm/yr, respectively), which accounts for the arch-depth increases evident with the eruption of the incisors (Fig. 1-41). In contrast, the mandibular molars drift anteriorly at a slightly greater rate than the incisors. Between 10 and

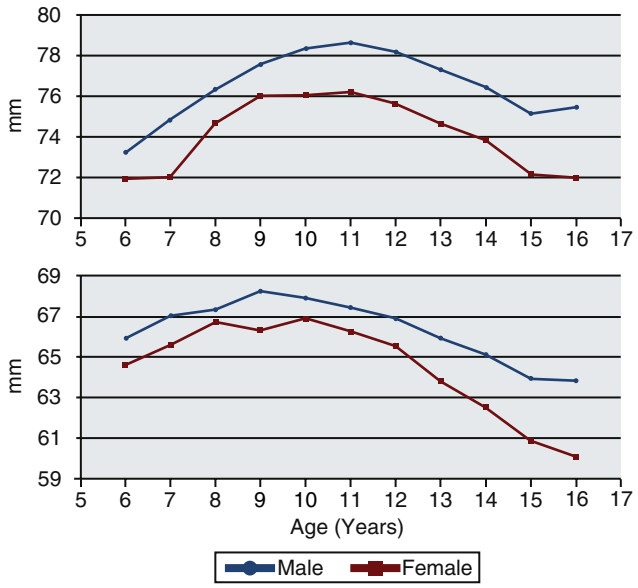


FIGURE 1-40 Maxillary and mandibular arch perimeter of males and females. (Data from Moyers RE, van der Linden PGM, Riolo ML, et al. *Standards of Human Occlusal Development*. Ann Arbor, MI: Center for Human Growth and Development; 1976.)

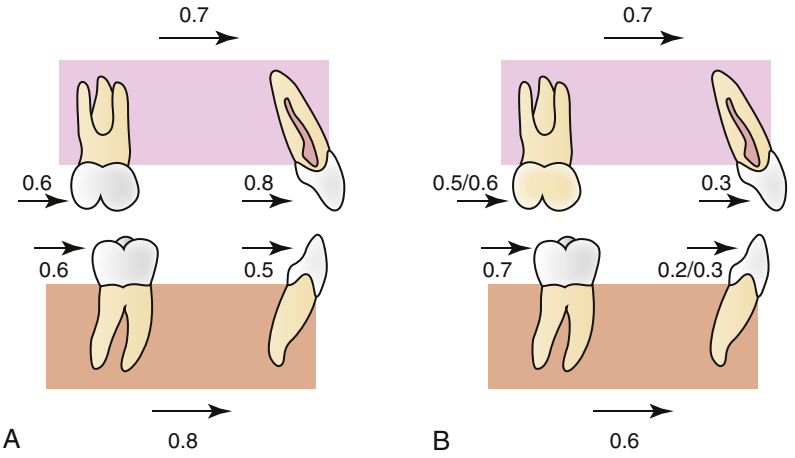


FIGURE 1-41 Approximate maxillary and mandibular AP displacements and tooth migration (mm/yr) during (A) childhood and (B) adolescence (female/male).

15 years of age, the molars (0.5 to 0.7 mm/yr) show significantly greater amounts of anterior drift than the incisors (0.3 mm/yr).

Substantial amounts of eruption occur throughout growth. During childhood, the maxillary first molars and incisors erupt at a rate of approximately 1.0 mm/yr, while their mandibular counterparts erupt at a rate of approximately 0.5 mm/yr (Fig. 1-42). During adolescence, the maxillary molars and incisors erupt at rates of 1.2 to 1.4 mm/yr and 0.9 mm/yr, respectively. The mandibular molars and incisors erupt at a rate of 0.5 to 0.9 mm/yr, with little or no differences between incisor and molar eruption. The amounts of eruption that occur are associated closely with the inferior displacements of the midface and, especially, the mandible.

During childhood, there is little or no evidence of sexual dimorphism in the migration and eruption of teeth. In contrast, there is a relatively high degree of dimorphism during adolescence in mandibular eruption, with boys showing almost twice as much eruption as girls. The maxillary teeth show only limited sex differences, pertaining primarily to the molars.

ADULT CHANGES IN CRANIOFACIAL FORM

The size and shape of the craniofacial complex continue to change throughout a considerable part of adulthood. Over 90% of the 70 cephalometric distances and 70% of the 69 angles evaluated by Behrents[106] showed changes after 17 years of age; 61% of the distances and 28% of the angles showed changes after 35 years of age. In particular, the mandibular plane angle increases in adult females and decreases in adult males, which explains why males 25 to 46 years of age exhibit greater chin projection than females, who undergo increases in NSGn.[107]

Adult soft tissues undergo the more pronounced changes than the skeletal structures. The nose grows substantially during adulthood, with the tip moving down and forward approximately 3 mm after 17 years of age. Males exhibit significantly more nasal growth than females. Upper lip length increases (approximately 2 to 3 mm) in both males and females after 17 years of age, resulting in decreases in upper incisor display over time. Lower lip length also increases, but less than upper lip length. The lips straighten and flatten during adulthood, but the most pronounced changes occur after 50 years of age. The soft tissue profile angle increases over time, with smaller increases when the nose is included than when it is excluded. Adult profile changes are limited to 2 to 3° and 4 to 6° when the nose is included and excluded, respectively.

POSTNATAL INTERRELATIONSHIPS DURING CRANIOFACIAL GROWTH

Postnatal craniofacial growth follows a gradient of relative growth that ranges between the neural and general somatic patterns. Vertical growth and modeling of the viscerocranium, as well as dental eruption, exhibit midchildhood and pubertal growth spurts. Anteroposterior growth and tooth migration, which do not exhibit midchildhood or pubertal growth spurts, change more or less regularly—except for the accelerated migration associated with the loss of teeth—throughout childhood and adolescence.

Generally, most displacements and rotations of the maxillo–mandibular complex are controlled epigenetically through growth of the chondrocranium, soft tissue growth, and expansion of the oronasal capsule. The cartilaginous growth centers play a particularly important role in the primary displacement of the chondrocranium, as well as in the secondary displacement of the viscerocranium. The anterior displacement of the midface has been associated with growth of the anterior cranial base and expansion of the anterior cranial fossa; mandibular displacements are more closely associated with growth of the posterior cranial base and middle cranial fossa. Anteroposterior length changes of the anterior cranial base, measured from sella to foramen cecum, coincide closely with expansion of the frontal lobes and growth at the sphenoethmoidal synchondrosis. Angular changes of the cranial base have been associated with growth gradients within the synchondroses, complex interactions with the growth of the brain, as well as facial growth. The cranial base angle decreases due to greater chondrogenesis in the superior than in the inferior aspects of the sphenoethmoidal and, especially, spheno-occipital synchondroses. Changes in cranial base angulation also appear to be related to changes in brain size, especially to the dramatic changes that occur during the first 2 postnatal years.

Cranial base growth influences the displacement and rotation of the viscerocranium. Growth of the posterior cranial base (i.e., spheno-occipital synchondrosis) is directly related to inferior and posterior displacements of the glenoid fossa; growth of the anterior cranial base is associated with midfacial displacement. Consequently, cranial base growth changes partially

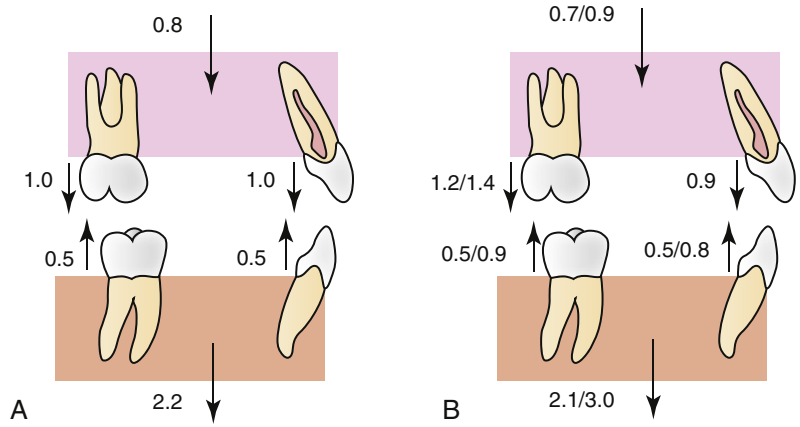


FIGURE 1-42 Approximate maxillary and mandibular vertical displacements (mm/yr) and tooth eruption during (**A**) childhood and (**B**) adolescence (female/male).

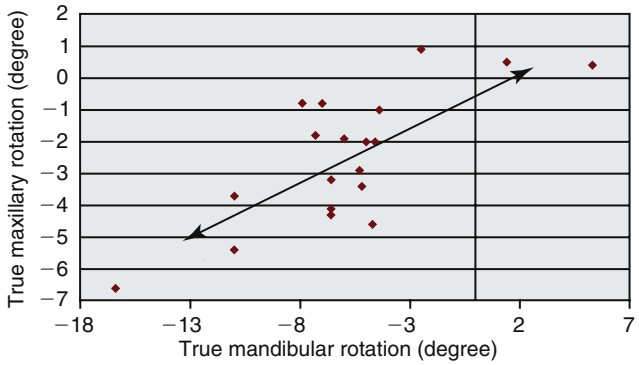


FIGURE 1-43 Relationship of true mandibular and true maxillary rotation ($r = 0.75$). (Data from Björk A, Skieller V. Facial development and tooth eruption. An implant study at the age of puberty. *Am J Orthod.* 1972;62:339–383.)

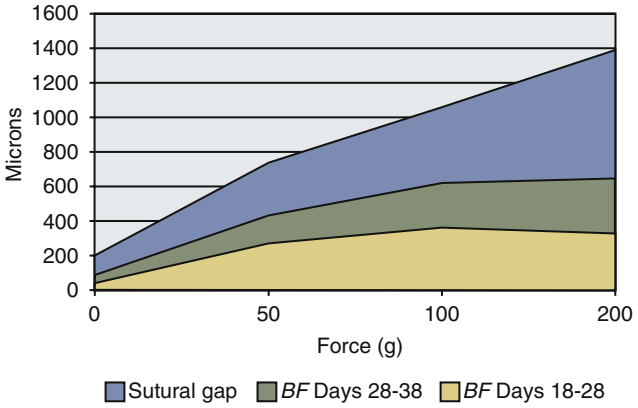


FIGURE 1-44 Relationships of bone formation (*BF*), sutural gap width, and amounts of force applied to separate sutures.

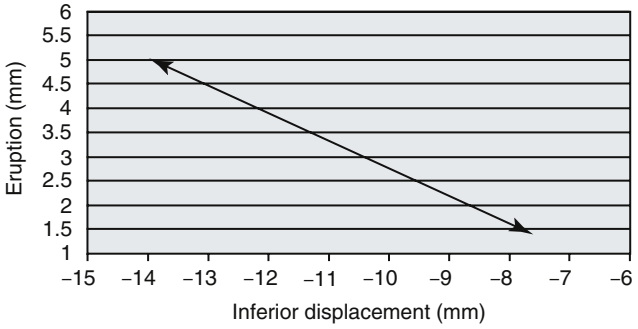


FIGURE 1-45 Relationship between the eruption of the mandibular molars and inferior displacement of the mandible.

explain individual and population differences in anteroposterior skeletal relationships. Most studies show that individuals with larger cranial base angles and/or larger anterior and posterior cranial base lengths tend to be retrognathic (i.e., Class II), whereas those with the smaller lengths and angles tend to be prognathic (i.e., Class III).

Structures within the midfacial complex also affect its displacement and rotation. Growth of the eyeball is associated with both the anterior and lateral displacements of the midface, which explains why enucleation of the eyeball results in anterior and lateral growth deficiencies of the midface.[108] The nasal septum also plays important roles in nasomaxillary growth, displacement, and rotation. However, while the anterior cranial fossa, cranial base, eyeball, and nasal septum play important roles in the early displacement and rotation of the midface, their growth potentials are limited after 7 to 8 years of age. Soft tissue growth and other factors leading to the expansion of the oronasal capsule are relatively more important in explaining the midfacial rotation and displacement during later childhood and adolescence.

In turn, mandibular displacement and rotation are greatly influenced by midfacial displacement and rotation, growth of the posterior cranial base, soft tissue growth, expansion of the oronasal capsule, and development of occlusion. Posture appears to have a profound effect on mandibular growth and remodeling. There is also a direct relationship between the true rotation of the maxilla and mandible. Both jaws usually rotate forward; individuals showing greater amount of forward rotation of the maxillary also tend to show greater forward rotation of the mandible (Fig. 1-43). Midfacial growth and the associated changes in the position of the maxillary dentition are also thought to play an important role in mandibular growth displacements. Major insults to maxillary growth can inhibit mandibular growth. Cranial growth disturbances can also influence mandibular growth indirectly through their effects on the midface, as well as on the positional changes of the glenoid fossa, especially during infancy and early childhood. For example, it has been shown that craniosynostosis, if left untreated for a sufficiently long period of time, can produce significant asymmetry of the mandible.

The anterior and, especially, inferior displacements of the maxilla and mandible have direct effects on the growth at the sutures, condylar growth, modeling patterns, dental eruption, and dental migration. Although there is an upper threshold, the amount of bony apposition that occurs at sutures is directly related to the amount of sutural separation. Larger expansion forces produce greater sutural separation, which in turn results in greater sutural bone formation (Fig. 1-44). Such growth potential is essential during periods of greater sutural separation, which require concomitantly greater bone formation. The condyle also undergoes a growth spurt that closely coincides with the increased rates of inferior displacement of the mandible that occur during adolescence.[109] Because the mandible's modeling patterns are directly related to the amounts of vertical and horizontal displacement that take place,[110] individuals with greater inferior displacement show greater superior drift of bone along the entire surface of the ramus (i.e., greater apposition superiorly and greater resorption along the lower border) than do individuals who undergo less inferior displacement. Due to the close association between mandibular displacement and rotation, individuals showing greater or lesser amounts of anterior displacement of the mandible tend to exhibit lesser or greater amounts of posterior drift of the superior aspect of the ramus, respectively. The amounts of inferior displacement of the mandible that occur are also positively related to the amount of eruption that occurs, especially of the posterior teeth (Fig. 1-45). Importantly, it is the displacement that determines the amounts of eruption that occur during growth, rather than vice versa. Displacements of the mandible also influence the anteroposterior compensations of the teeth. Individuals showing relatively greater anterior displacement of the mandible than maxilla tend to exhibit greater mesial displacement of the maxillary molars and counterclockwise rotation of the occlusal

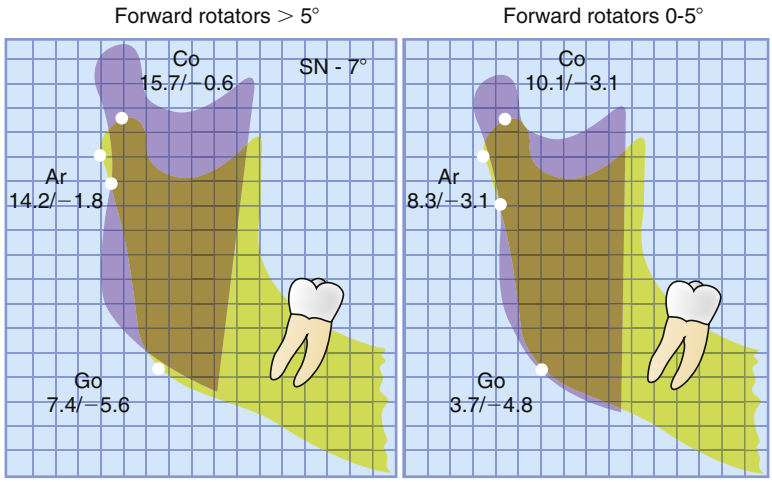


FIGURE 1-46 Vertical/horizontal (mm) remodeling changes of individuals showing greater than 5 degrees of true forward rotation, compared with those showing 0- to 5-mm true forward rotation.

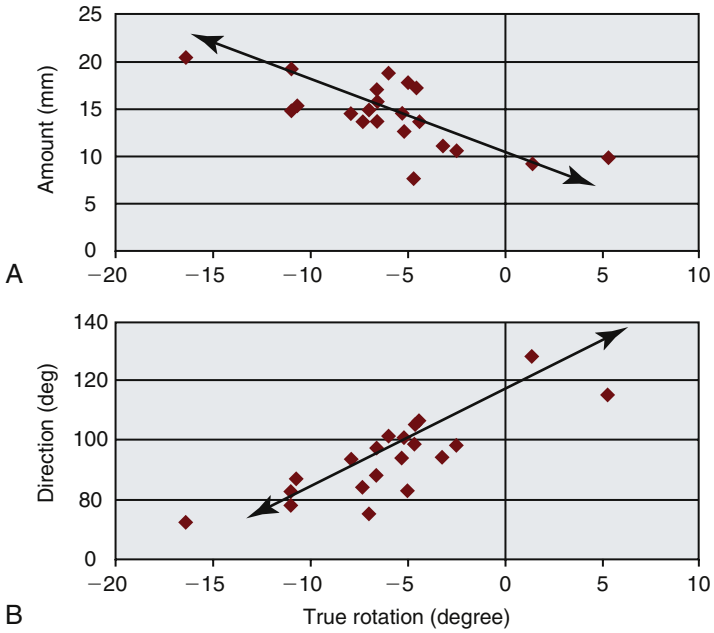


FIGURE 1-47 Relationships of true mandibular rotation and (A) the amount of condylar growth and (B) the direction of condylar growth. (Data from Björk A, Skieller V. Facial development and tooth eruption. An implant study at the age of puberty. *Am J Orthod.* 1972;62:339–383.)

plane; those who undergo relatively greater anterior maxillary displacements display greater mesial displacement of the mandibular molars and minimal mesial displacement of maxillary molars.

The morphologic correlates with true rotation are numerous and hold important clinical implications.[111] Vertical rotation has been directly related to changes in tooth position, with true forward rotators showing greater amounts of lower incisor proclination during eruption; backward rotators show retroclination of the incisors and loss of arch space. Rotation is also related to the modeling pattern that occurs on the lower mandibular border; subjects who undergo the greatest amounts of true forward rotation also exhibit the greatest amounts of posterior resorption and anterior bony deposition (Fig. 1-46). Ramus modeling in general depends on the rotational pattern of the mandible. Individuals who undergo greater amounts of true forward rotation also exhibit greater amounts of condylar growth, oriented in a more superior–anterior direction (Fig. 1-47). Perhaps the most important clinical correlate is the relationship between true rotation and chin position. True rotation of the mandible explains more of the individual variation in chin position than condylar growth or changes in glenoid fossa position.

SIGNIFICANCE OF UNDERSTANDING CRANIOFACIAL GROWTH FOR ORTHODONTICS

To be most effective as clinicians, it is essential that orthodontists understand the development, growth, and adaptive potentials of the craniofacial structures. Along with orthodontic biomechanics, knowledge of how the craniofacial complex develops and grows provides the foundation for understanding the etiology of the various dental and skeletal malocclusions, the best

of all possible treatment approaches, and how patients might be expected to respond after treatment. A thorough understanding of growth provides the basis for knowing which craniofacial components might be expected to respond to a stress and how great the response might be expected to be. Because a structure's response potential to stress is directly related to its relative growth potential, and the vertical aspects of the mandible have the greatest relative growth potential, it follows that skeletal malocclusions might be expected to relate to vertical mandibular growth. Class II malocclusion, which is the predominant form of skeletal malocclusion, pertains primarily to the mandible.[112,113] These individuals are often retrognathic due to limited true forward rotation of the mandible, which is in turn related to deficient inferior growth displacement of the posterior mandible and/or excessive inferior displacement of the anterior aspect of the mandible.

Knowledge of growth is also important because, whenever possible, orthodontists should try to mimic growth when planning treatment. An understanding of growth provides the biological limits within which treatments can be performed. As previously indicated, the viscerocranium is made up almost entirely of intramembranous bone and is predominantly under epigenetic and environmental control. It is programmed to adapt, and adaptation should be expected whenever it is stressed. The biological system cannot distinguish between stresses imposed by the orthodontist and those imposed during normal growth; it simply responds depending on its growth potential. Continuing with the previous example, individuals who exhibit good growth patterns tend to be true forward rotators with condyles that grow in a more anterior direction. Based on this knowledge, hyperdivergent retrognathnic patients would best be served by treatments that focus on rotating the mandible rather than stimulating or redirecting condylar growth in a posterior direction.[114]

Finally, an understanding of growth makes it possible to estimate morphologic changes that might be expected to occur during and after orthodontic treatment. Unless it is intentionally disrupted, an individual's growth path before treatment might be expected to continue during and after treatment. Knowing how the maxilla and mandible have been rotated and/or displaced during treatment provides an understanding of the modeling and consequent shape changes that might be expected to occur. Vertical growth after treatment might be problematic in terms of posttreatment crowding, due to its relationship with tooth eruption. It has been shown that the best predictors of crowding of the mandibular incisors, both after treatment and without treatment, are the inferior displacement of the mandible and superior eruption of the incisors.[86]

As understanding of craniofacial development, growth, and adaptation continues to improve in the future, orthodontists can look forward to even more therapeutic advances that can be used to influence growth and posttreatment stability. This understanding will facilitate greater clinical control of changes in the craniofacial complex due to the normal growth process and in compensatory adaptation of tissues after treatment. Understanding normal craniofacial growth and especially that of the complex network of underlying molecular factors responsible for craniofacial growth and treatment will also be of immeasurable benefit in assisting the orthodontist in understanding what may or may not be possible, not only with respect to diagnosing a patient's underlying abnormality but also in determining the best treatment approach for its correction.[17,18,115]

REFERENCES

1. Carlson B. *Human Embryology and Developmental Biology*. Philadelphia: Elsevier; 2014.
2. Sperber GH. *Craniofacial Development*. London: BC Decker; 2010.
3. Carlson DS, Ribbens KR. *Craniofacial Growth during Adolescence.* Craniofacial Growth Series. Ann Arbor, MI: Center for Human Growth and Development; 1987:20.
4. Scammon RE. The measurement of the body in childhood. In: Harris JA, Jackson CM, Scammon RE, eds. *The Measurement of the Body in Childhood*. Minneapolis: University of Minneapolis Press; 1930.
5. Buschang PH, Hinton RJ. A gradient of potential for modifying craniofacial growth. In: Carlson DS, ed. *Control Mechanisms of Craniofacial Development and Growth*. Semin Orthod; 2005; 11(4):219–226.
6. Buschang PH, Tanguay R, Demirjian A, LaPalme L, Turkewicz J. Mathematical models of longitudinal mandibular growth for children with normal and untreated Class II, division 1 malocclusion. *Eur J Orthod*. 1988;10:227–234.
7. Malina RM, Bouchard C, Beunen G. Human growth: selected aspects of current research on well-nourished children. *Ann Rev Anthrop*. 1988;17:187–219.
8. Lewis AB, Roche AF, Wagner B. Pubertal spurts in cranial base and mandible. Comparisons within individuals. *Angle Orthod*. 1985;55:17–30.
9. Buschang PH, Jacob HB, Demirjian A. Female adolescent craniofacial growth spurts: real or fiction? *Eur J Orthod*. 2013;35:819–825.
10. Tanner JM. Growth at adolescence: an introduction. In: Carlson DS, Ribbens KR, eds. *Craniofacial Growth during Adolescence*. Craniofacial Growth Series. Ann Arbor, MI: Center for Human Growth and Development; 1987;20:1–22.
11. Grave KC, Brown T. Skeletal ossification and the adolescent growth spurt. *Am J Orthod*. 1976;69:611–624.
12. Fishman LS. Radiographic evaluation of skeletal maturation. *Angle Orthod*. 1982;52:88–112.
13. Baccetti T, Franchi L, McNamara Jr JA. The cervical vertebral maturation (CVM) method for the assessment of optimal treatment timing in dentofacial orthopedics. *Semin Orthod*. 2005;11:119–129.
14. Massoud MI, Masoud I, Kent Jr RL, Gowharji N, Hassan AH, Cohen L. Relationship between blood-spot insulin-like growth factor 1 levels and hand-wrist assessment of skeletal maturity. *Am J Orthod Dentofac Orthop*. 2009;136(1):59–64.
15. Wilkie AOM, Morriss-Kay GM. Genetics of craniofacial development and malformation. *Nat Rev Genet*. 2001;2:458–468.
16. Hefer T, Jaochims HZ, Carlson DS, Finnell RH. Factors associated with the etiology of congenital craniofacial anomalies: an update Part I. *J Israeli Med Assoc*. 1998;135:209–213, 286–291.
17. Carlson DS. Growth modification: from molecules to mandibles. In: McNamara JA, ed. *Growth Modification: What Works, What Doesn't, and Why*. Craniofacial Growth Series. Ann Arbor, MI: Center for Human Growth and Development; 1999:17–65.
18. Carlson DS. Toward a new synthesis for craniofacial biology: a genomic-epigenomic basis for dentofacial orthopedic treatment. In: McNamara JA, ed. *The 40th Moyers Symposium. Looking Back…Looking Forward*. Craniofacial Growth Series; 50. Ann Arbor, MI: Center for Human Growth and Development; 2014:193–247.
19. D'Souza RN, Dunnvald M, Frazier-Bowers S, et al. Translational genetics: advancing fronts for craniofacial health. *J Dent Res*. 2013;92(12):1058–1064.
20. Eng G, Chen A, Vess T, Ginsburg GS. Genome technologies and personalized dental medicine. *Oral Dis*. 2012;18:223–235.
21. Pritchard JJ, Scott JH, Girgis FG. The structure and development of cranial and facial sutures. *J Anat*. 1956;90:73–86.
22. Scott JH. The growth of the human face. *Proc R Soc Med*. 1954;47:91–100.
23. Baer MJ. Patterns of growth of the skull as revealed by vital staining. *Hum Biol*. 1954;26:80–126.
24. Rice DPC. Craniofacial anomalies: from development to molecular pathogenesis. *Curr Mol Med*. 2005;5:699–722.
25. Chai Y, Maxson Jr RE. Recent advances in craniofacial morphogenesis. *Dev Dyn*. 2006;235:2353–2375.

26. Kim HJ, et al. FGF-, BMP- and Shh-mediated signalling pathways in the regulation of cranial suture morphogenesis and calvarial bone development. *Devel.* 1998;125(7):1241–1251.
27. Rice DP, Kim HJ, Thesleff I. Apoptosis in murine calvarial bone and suture development. *Eur J Oral Sci*. 1999;107(4):265–275.
28. Carver EA, Oram KF, Gridley T. Craniosynostosis in Twist heterozygous mice: a model for Saethre-Chotzen syndrome. *Anat Rec.* 2002;268(2):90–92.
29. Warren SM, et al. The BMP antagonist noggin regulates cranial suture fusion. *Nature*. 2003;422(6932):625–629.
30. Veistinen L, Takatolo M, Tanimoto Y, Kesper D, Vortkamp A, Rice DPC. Loss-of-function of *Gli3* in mice causes abnormal frontal bone morphology and premature synostosis of the interfrontal suture. *Front Physiol.* 2012;3:1–6.
31. Jabs EW, et al. A mutation in the homeodomain of the human *MSX2* gene in a family affected with autosomal dominant craniosynostosis. *Cell*. 1993;75(3):443–450.
32. el Ghouzzi V, et al. Mutations of the *TWIST* gene in the Saethre-Chotzen syndrome. *Nat Genet*. 1997;15(1):42–46.
33. Howard TD, et al. Mutations in *TWIST*, a basic helix-loop-helix transcription factor, in Saethre-Chotzen syndrome. *Nat Genet*. 1997;15(1):36–41.
34. Moss ML, Salentijn L. The primary role of functional matrices in facial growth. *Am J Orthod*. 1969;55:566–577.
35. Martinez-Abadias N, Heuze Y, Wang Y, Jabs EW, Aldridge K, Richtmeier J. FGF/FGFR signaling coordinates skull development by modulating magnitude of morphological integration: evidence from Apert syndrome mouse models. *PLoS One*. 2011;6(10).
36. Heuze Y, Martinez-Abadias N, Stella JM, et al. Quantification of facial skeletal shape variation in fibroblast growth factor receptor-related craniosynostosis syndromes. *Birth Def Res (A)*. 2014;100:250–259.
37. Di Ieva A, Bruner E, Haider T, et al. Skull base embryology: a multidisciplinary review. *Childs Ner Syst*. 2014;30:991–1000.
38. Lei WY, Wong RWK, Rabie ABM. Factors regulating endochondral ossification in the spheno-occipital synchondrosis. *Angle Orthod*. 2008;78(2):215–220.
39. Balczerski B, Zakaria S, Tucker AS, et al. Distinct spatiotemporal roles of hedgehog signaling during chick and mouse cranial base and axial skeletal development. *Dev Biol*. 2012;371:203–214.
40. Kuhn JL, Ciarelli MJ. The biomechanics of the growth plate. In: Carlson DS, Goldstein SA, eds. *Bone Biodynamics in Orthodontic and Orthopedic Treatment*. Craniofacial Growth Series. Ann Arbor, MI: Center for Human Growth and Development; 1992:93–110.
41. Opperman LA, Gakunga PT, Carlson DS. Genetic factors influencing morphogenesis and growth of sutures and synchondroses in the craniofacial complex. In: Carlson DS, ed. *Control mechanisms of craniofacial development and growth*. Semin Orthod; 2005; 11(4):199–208.
42. Baume LJ. Principles of cephalofacial development revealed by experimental biology. *Am J Orthod*. 1961;47(12):881–901.
43. Buschang PH, Baume RM, Nass GG. A craniofacial growth maturity gradient for males and females between 4 and 16 years of age. *Am J Phys Anthrop*. 1983;61:373–382.
44. Melsen B. The cranial base: the postnatal development of the cranial base studied histologically on human autopsy material. *Acta Orthod Scand Suppl*. 1974;62:1–126.
45. Latham RA. The sella point and postnatal growth of the cranial base in the human skull. *Am J Orthod*. 1972;61:156–162.
46. Powell TV, Brodie AG. Closure of the spheno-occipital synchondrosis. *Anat Rec*. 1963;147:15–23.
47. Ford HER. Growth of the human cranial base. *Am J Orthod*. 1958;44:498–506.
48. Adab K, Sayne JR, Carlson DS, Opperman LA. Tgf-β1, Tgf-β2, Tgf-β3 and Msx2 expression is elevated during frontonasal suture morphogenesis and during active postnatal facial growth. *Orthod Craniofac Res*. 2002;5(4):227–237.
49. Griffin JN, Compagnucci C, Hu D, et al. *Fgf8* dosage determines midfacial integration and polarity within the nasal and optic capsules. *Dev Biol*. 2013;374:185–197.
50. Greene RM, Pisano MM. Palate morphogenesis: current understanding and future directions. *Birth Defects Res (Part C)*. 2010;90:133–154.
51. Bush JO, Jiang R. Palatogenesis: morphological and molecular mechanisms of secondary palate development. *Devel*. 2012;139:231–243.
52. Behrents RG, Harris EF. The premaxillary-maxillary suture and orthodontic mechanotherapy. *Am J Orthod Dentofac Orthop*. 1991;99:1–6.
53. Melsen B. Palatal growth studied on human autopsy material. A histologic microradiographic study. *Am J Orthod*. 1975;68:42–54.
54. Persson M, Thilander B. Palatal suture closure in man from 15 to 35 years of age. *Am J Orthod*. 1977;72:42–52.
55. Korbmacher H, Schilling A, Puschel K, Amling M, Kahl-Nieke B. Age-dependent three-dimensional microcomputed tomography analysis of the human midpalatal suture. *J Orofac Orthop*. 2007;68:364–376.
56. Knaup B, Yildizhan F, Wehrbein H. Age-related changes in the midpalatal suture. A histomorphometric study. *J Orofac Orthop*. 2004;65:467–474.
57. Herring SW, Mucci RJ. In vivo strain in cranial sutures: the zygomatic arch. *J Morphol*. 1991;207:225–239.
58. Björk A, Skieller V. Postnatal growth and development of the maxillary complex. In: McNamara Jr JA, ed. *Factors Affecting the Growth of the Midface*. Craniofacial Growth Series. Ann Arbor, MI: Center for Human Growth and Development; 1976:61–100.
59. Enlow DH, Bang S. Growth and remodeling of the human maxilla. *Am J Orthod*. 1965;51:446–464.
60. Björk A, Skieller V. Growth of the maxilla in three dimensions as revealed radiographically by the implant method. *Br J Orthod*. 1977;4:53–64.
61. Hersby RM, Marshall SD, Dawson DV, et al. Transverse skeletal and dentoalveolar changes during growth. *Am J Orthod Dentofac Orthop*. 2006;130:721–731.
62. Solow B, Houston WJ. Mandibular rotations: concepts and terminology. *Eur J Orthod*. 1988;10:177–179.
63. Lux CJ, Conradt C, Burden D, et al. Transverse development of the craniofacial skeleton and dentition between 7–15 years of age: a longitudinal postero-anterior cephalometric study. *Eur J Orthod*. 2004;26:31–42.
64. Shibata S, Sakamoto Y, Yokohama-Tamaki T, Murakami G, Cho BH. Distribution of matrix proteins in perichondrium and periosteum during incorporation of Meckel's cartilage into ossifying mandible: an immunohistochemical study. *Anat Rec*. 2014;297:1208–1217.
65. Hall BK. Immobilization and cartilage transformation into bone in the embryonic chick. *Anat Rec*. 1972;173:391–404.
66. Vinkka H. Secondary cartilages in the facial skeleton of the rat. *Proc Finn Dent Soc*. 1982;78:36–39.
67. Copray JCVM, Dibbets JMH, Kantomaa T. The role of condylar cartilage in the development of the temporomandibular joint. *Angle Orthod*. 1988;58:369–380.
68. Carlson DS. Growth of the temporomandibular joint. In: Zarb GA, Carlsson GE, Sessle BJ, Mohl ND, eds. *Temporomandibular Joint and Masticatory Muscle Disorders*. Copenhagen: Munksgaard; 1994: 128–155.
69. Hinton RJ, Carlson DS. Regulation of growth in the mandibular condylar cartilage. In: Carlson DS, ed. *Control mechanisms of craniofacial development and growth*. Semin Orthod; 2005; 11(4):209–218.
70. Enomoto A, Watahiki J, Nampo T, Ichikawa Y, Tachikawa T, Maki K. Mastication markedly affects mandibular condylar cartilage growth, gene expression, and morphology. *Am J Orthod Dentofac Orthop*. 2014;146(3):355–363.
71. Hinton RJ, Carlson DS. Histological changes in the articular eminence and mandibular fossa during growth of the rhesus monkey (*Macaca mulatta*). *Am J Anat*. 1983;166:99–116.
72. Carlson DS, McNamara Jr JA, Jaul DH. Histological analysis of the growth of the mandibular condyle in the rhesus monkey (*Macaca mulatta*). *Am J Anat*. 1978;151:103–117.
73. Wright DM, Moffett BC. The postnatal development of the human temporomandibular joint. *Am J Anat*. 1974;141:235–250.
74. Hinton RJ, Serrano M, So S. Differential gene expression in the perichondrium and cartilage of the neonatal mouse temporomandibular joint. *Ortho Craniofac Res*. 2009;12:168–177.
75. Hinton RJ. Genes that regulate morphogenesis and growth of the temporomandibular joint: a review. *Dev Dyn*. 2014;243:864–874.

76. Öberg T, Carlsson GE, Fajers CM. The temporomandibular joint. A study on human autopsy material. *Acta Odontol Scand*. 1971;29: 349–384.
77. Carlsson GE, Öberg T. Remodeling of the temporomandibular joint. *Oral Sci Rev*. 1974;6:53–86.
78. Lubsen CC, Hansson TL, Nordstrom BB, et al. Histomorphometric analysis of cartilage and subchondral bone in mandibular condyles of young human adults at autopsy. *Arch Oral Biol*. 1985;30:129–136.
79. Lubsen CC, Hansson TL, Nordstrom BB, et al. Histomorphometry of age and sex changes in mandibular condyles of young human adults. *Arch Oral Biol*. 1987;32:729–733.
80. Dibbets JMH, Carlson DS. Implications of temporomandibular disorders for facial growth and orthodontic treatment. *Semin Orthod Temporomandibular Jt Disord: Facts Fallacies*. 1995;1(4):258–272.
81. Dechow PD, Carlson DS. Development of mandibular form: phylogeny, ontogeny and function. In: McNeill C, ed. *Science and Practice of Occlusion*. New Malden, UK: Quintessence; 1997:3–22.
82. Petrovic A, Stutzmann J, Oudet C. Control processes in the postnatal growth of the condylar cartilage. In: McNamara Jr JA, ed. *Determinants of Mandibular Form and Growth*. Craniofacial Growth Series. Ann Arbor, MI: Center for Human Growth and Development; 1975:101–154.
83. Petrovic A. Control of postnatal growth of secondary cartilages of the mandible by mechanisms regulating occlusion. Cybernetic model. *Trans Eur Orthod Soc*. 1974:69–75.
84. McNamara JA, Carlson DS. Quantitative analysis of temporomandibular joint adaptations to protrusive function. *Am J Orthod*. 1979;76:593–611.
85. Petrovic A. Experimental and cybernetic approaches to the mechanism of action of functional appliances on mandibular growth. In: McNamara Jr JA, Ribbens KA, eds. *Malocclusion and the Periodontium*. Craniofacial Growth Series. Ann Arbor, MI: Center for Human Growth and Development; 1985:213–268.
86. Carlson DS. Theories of craniofacial growth in the postgenomic era. In: Carlson DS, ed. *Control Mechanisms of Craniofacial Development and Growth*. Semin Orthod; 2005;11(4):172–183.
87. Visnapuu V, Peltomaki T, Ronning O, et al. Distribution of fibroblast growth factors (FGFR-1 and -3) and platelet-derived growth factor receptors (PDGFR) in the rat mandibular condyle during growth. *Orthod Craniofac Res*. 2002;5:147–153.
88. Ramirez-Yanez GO, Young WG, et al. Influence of growth hormone on the mandibular condylar cartilage of rats. *Arch Oral Biol*. 2004;49:585–590.
89. Visnapuu V, Peltomaki T, Ronning O, et al. Growth hormone and insulin-like growth factor-I receptors in the temporomandibular joint of the rat. *J Dent Res*. 2001;80:1903–1907.
90. Rabie ABM, She TT, Hägg U. Functional appliance therapy accelerates and enhances condylar growth. *Am J Orthod Dentofacial Orthop*. 2003;123(1):40–48.
91. Chu FT, Tang GH, Hu Z, Qian YF, Shen G. Mandibular functional positioning only in vertical dimension contributes to condylar adaptation evidenced by concomitant expressions of L-Sox5 and type II collagen. *Arch Oral Biol*. 2008;53:567–574.
92. Owtad P, Park JH, Shen G, Potres, Darendelier MA. The biology of TMJ growth modification: a review. *J Dent Res*. 2013;92(4):315–321.
93. Xue F, Wong RWK, Rabie ABM. Genes, genetics, and Class III malocclusion. *Orthod Craniofac Res*. 2010;13:69–74.
94. Fuentes MA, Opperman LA, Buschang P, et al. Lateral functional shift of the mandible: Part II. Effects on gene expression in condylar cartilage. *Am J Orthod Dentofac Orthop*. 2003;123:160–166.
95. Hajjar D, Santos MF, Kimura ET. Propulsive appliance stimulates the synthesis of insulin-like growth factors I and II in the mandibular condylar cartilage of young rats. *Arch Oral Biol*. 2003;48:635–642.
96. Rabie ABM, Hagg U. Factors regulating mandibular condylar growth. *Am J Orthod Dentofac Orthop*. 2002;122:401–409.
97. Tang GH, Rabie ABM, Hagg U. Indian hedgehog: a mechanotransduction mediator in condylar cartilage. *J Dent Res*. 2004;83:434–438.
98. Enlow DH, Harris DB. A study of the postnatal growth of the human mandible. *Am J Orthod*. 1964;50:25.
99. Buschang PH, Julien K, Sachdeva R, et al. Childhood and pubertal growth changes of the human symphysis. *Angle Orthod*. 1992;62:203–210.
100. Buschang PH, Santos Pinto A. Condylar growth and glenoid fossa displacement during childhood and adolescence. *Am J Orthod Dentofac Orthop*. 1998;113:437–442.
101. Björk A, Skieller V. Facial development and tooth eruption. An implant study at the age of puberty. *Am J Orthod*. 1972;62:339–383.
102. Spady M, Buschang PH, Demirjian A, et al. Mandibular rotation and angular remodeling during childhood and adolescence. *Am J Hum Biol*. 1992;4:683–689.
103. Wang M, Buschang PH, Behrents R. Mandibular rotation and remodeling changes during early childhood. *Angle Orthod*. 2009;79:271–275.
104. Moorrees CFA, Grøn AM, Lebret LML, et al. Growth studies of the dentition: a review. *Am J Orthod*. 1969;55:600–616.
105. Moyers RE, van der Linden PGM, Riolo ML, et al. *Standards of Human Occlusal Development*. Ann Arbor, MI: Center for Human Growth and Development; 1976.
106. Behrents RG. Growth in the Aging Craniofacial Skeleton. *Craniofacial Growth Series*. Ann Arbor, MI: Center for Human Growth and Development, University of Michigan; 1985. Monograph #17.
107. Bishara SE, Treder JE, Jakobsen JR. Facial and dental changes in adulthood. *Am J Orthod Dentofacial Orthop*. 1994;106:175–186.
108. Sarnat BG. Eye and orbital size in the young and adult. *Ophthalmologica*. 1982;185:74–89.
109. Kim YE, Nanda RS, Sinha PK. Transition of molar relationships in different skeletal growth patterns. *Am J Orthod Dentofac Orthop*. 2002;121:280–290.
110. Buschang PH, Gandini Jr LG. Mandibular skeletal growth and modeling between 10–15 years of age. *Eur J Orthod*. 2002;24:69–79.
111. Dibbets JMH. Mandibular rotation and enlargement. *Am J Orthod Dentofac Orthop*. 1990;98(1):29–32.
112. McNamara Jr JA. Components of class II malocclusion in children 8–10 years of age. *Angle Orthod*. 1981;51:177–202.
113. Buschang PH, Martins J. Childhood and adolescent changes of skeletal relationships. *Angle Orthod*. 1998;68:199–208.
114. Driscoll-Gilliland J, Buschang PH, Behrents RG. An evaluation of growth and stability in untreated and treated subjects. *Am J Orthod Dentofac Orthop*. 2001;120:588–597.
115. Carlson DS. Biological rationale for early treatment of dentofacial deformities. *Am J Orthod Dentofac Orthop*. 2002;121(6):554–558.

2

Genetics and Orthodontics

James Kennedy Hartsfield, Jr., and Lorri Ann Morford

OUTLINE

Etiology, 31
Background and Basic Definitions, 32
Types of Genetic Effects and Modes of Inheritance, 34
Monogenic Traits, 35
Autosomal Dominant Traits and Penetrance, 35
Variable Expressivity, 36
Autosomal Recessive Traits, 37
X-Linked Traits and Lyonization (X Inactivation), 37
Complex Traits, 37
Nature versus Nurture, 38
Heritability and Its Estimation, 39
Use of Family Data to Predict Growth, 40
Facial Growth, 41
Growth Differences during Puberty, 41
Mandibular Prognathism/Class III Malocclusion, 41
Class II Division 2 (II/2) Malocclusion, 42
Tooth Size and Agnesis, 42
Dental Crown Morphology, 42
Dental Agenesis, 42
Dental Eruption Problems, 43
Canine Impaction and/or Displacement, 43
Primary Failure of Eruption, 43
Environmental and Genetic Influences on Bilateral Symmetry, 44
Genetic Factors and External Apical Root Resorption, 44
Personalized-Precision Orthodontics, 45
Summary, 45

Malocclusion arises from the combined interactions of genetic and environmental factors on the developmental pathway(s) involved in the formation of the orofacial region. Orthodontists who gain a solid foundational understanding of genetics are best equipped to understand why some patients develop certain occlusions. The consideration of family history and known genetic factors in the diagnosis and treatment planning of malocclusion is essential, especially since there are genetic influences on virtually all aspects of dental/facial growth and development. Therefore, to maximize the chance of successful treatment outcomes, there are two key considerations: (1) properly identify the cause of the problem before attempting treatment, (2) identify the factors that will influence the treatment outcome, or both. The factors involved in the cause may not be the same factors that would influence the treatment outcome. Knowing whether the cause of the problem is "genetic" has been cited as a factor in eventual outcome; that is, if the problem is genetic, then orthodontists may be limited in what they can do (or change).[1-3] However, this concept has often been misapplied. For example, in the orthodontic literature, there are many inappropriate uses of heritability estimates as a proxy for evaluating whether a malocclusion or some anatomic morphology is of "genetic origin." As will be explained in this chapter, heritability estimates have no relevance to the question of the genetic influence on a specific malocclusion in a particular patient. How specific genetic factors will influence a patient's responsiveness to environmental factors (including orthodontic treatment and the long-term stability of its outcome) as determined by studies of genetic markers, or gene sequences, and their impact on the proteins that they encode or influence, should be the greatest concern for the clinician.[4,5]

ETIOLOGY

Consideration of the potential cause(s) of a malocclusion requires careful contemplation of the following:

1. Most problems in orthodontics (or any outcome of growth and development), unless acquired by trauma, are not strictly the result of only genetic or only environmental factors.[6] Growth is the result of the interaction of genetic and environmental factors over time.[7,8]
2. Many studies examining the genetics of craniofacial growth are analyses of heritability. Heritability studies estimate the proportion of the total phenotypic variation, for a quantitative trait, that can be attributed to genetic differences among individuals within the specific population being examined up to the time of the analysis. Heritability studies *do not* determine the types of genetic influences or their mode of inheritance—that is, whether the trait is a single gene (monogenic) trait or a complex trait[5] with the effects of multiple genetic and environmental factors.
3. Even if a patient's craniofacial growth is influenced heavily by one gene (i.e., monogenic in familial skeletal Class III) as opposed to multiple genetic factors, there is no guarantee

that future growth will necessarily or absolutely be predetermined. Nor does it mean that growth will proceed on a particular immutable track, although traits with a monogenic influence may be less amenable to environmental (treatment) intervention than traits influenced by multiple genes. Orthodontic treatment itself is an environmental factor that can move the teeth within the hard and soft tissue envelope, but to what extent it can influence growth is difficult to determine. This is because it is impossible to really know exactly how much growth would have happened in the individual without treatment, even if compared to an untreated identical twin.

4. A patient's biological responsiveness to a particular environmental factor (e.g., orthodontic treatment) does not necessarily depend on any prior interactions of genetic and environmental factors but rather on the individual's biological responsiveness to the orthodontic treatment. The final outcome of orthodontic treatment will be a function of the overall interactions between the gene products generated from genetic factors that are expressed (or not expressed) during the treatment time, combined with any other environmental factors present during the treatment time, against the backdrop of the developmental maturity of the individual.[9-12] The most important and practical questions regarding orthodontics and genetics, however, lay in the determination of whether different patients respond to a specific type of orthodontic treatment in dissimilar ways due to the influence of their "unique" genetic makeups.

BACKGROUND AND BASIC DEFINITIONS

Before proceeding, a few basic genetic definitions and concept descriptions are required. An organism's *genome* is defined as the complete set of genetic instructions for that organism. The Human Genome Project (HGP), completed in April of 2003, was instrumental in helping us understand more about the overall size and complexity of the human genome. We learned that the human genome is made up of a double helix of deoxyribonucleic acid (DNA) comprised of ~3.2 billion chemical nucleotide base pairs.[13] The genetic instructions, or DNA code(s), are created by the linear pattern, order, and number of adenine (A), thymine (T), cytosine (C), and guanine (G) bases along the paired double helix, where A base pairs with T in the double helical structure and C base pairs with G. This genetic information is normally organized into smaller units (ranging in length from ~50 to 250 million base pairs each) called *chromosomes*.[13] A chromosome is made up of a continuous stretch of the double helical DNA that is wrapped around proteins that are called *histones*. Histones enable the DNA units to be tightly packed into the nucleus of our cells and also play an important role in regulating when and where our cells will use portions of the genetic information contained in the genome.[14] Altogether, we each inherit a total of 46 chromosomes; 22 *homologous pairs* of chromosomes called *autosomes* that are numbered by size and other characteristics, along with one pair of *sex chromosomes* that are homologous (X,X) in females and only partly homologous (X,Y) in males (Fig. 2-1). *Homologous chromosomes* are units of genetic material that are similar in

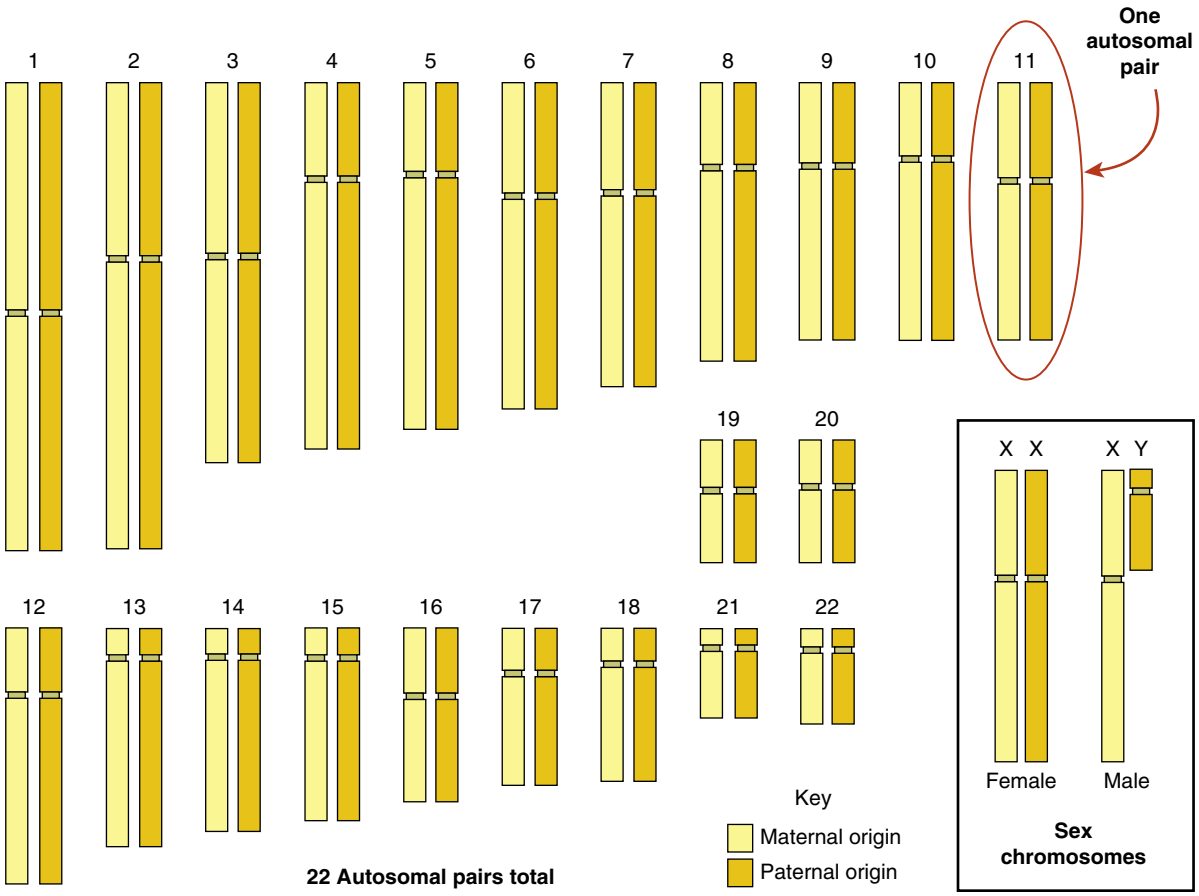


FIGURE 2-1 Diagram of human chromosomes.

size and structural features. Upon conception, a person inherits all 46 chromosomes (22 autosomal pairs total and one pair of sex chromosomes) that make them a unique individual; one chromosome for each autosomal pair is contributed by each parent, and one sex chromosome originates from each parent. Chromosomes in all subsequent cells are copies of the original maternal or paternal chromosomes.

Looking closer at the chromosomes, they are further organized into smaller units called *genes*, which represent the smallest physical and functional unit of inheritance. A gene can be defined as the complete DNA sequence that codes for the synthesis of a specific polypeptide via a messenger RNA intermediate (mRNA) (Fig. 2-2) or the synthesis of a specific RNA molecule (e.g., transfer RNA, ribosomal RNA, and noncoding regulatory RNA molecules such as microRNA or long noncoding RNA).[15] Each person normally inherits two copies of every gene within the genome: one gene copy on the autosome or sex chromosome of maternal origin and the other gene copy on the autosome or sex chromosome of paternal origin, although the X chromosome has more genes than the Y chromosome (Fig. 2-3). Based on the findings of the HGP, we have learned that (1) there are an estimated 25,000 genes in the human genome; (2) our genes only make up 2% of the whole genome; and (3) the average gene is 3000 nucleotide base pairs in length.[13]

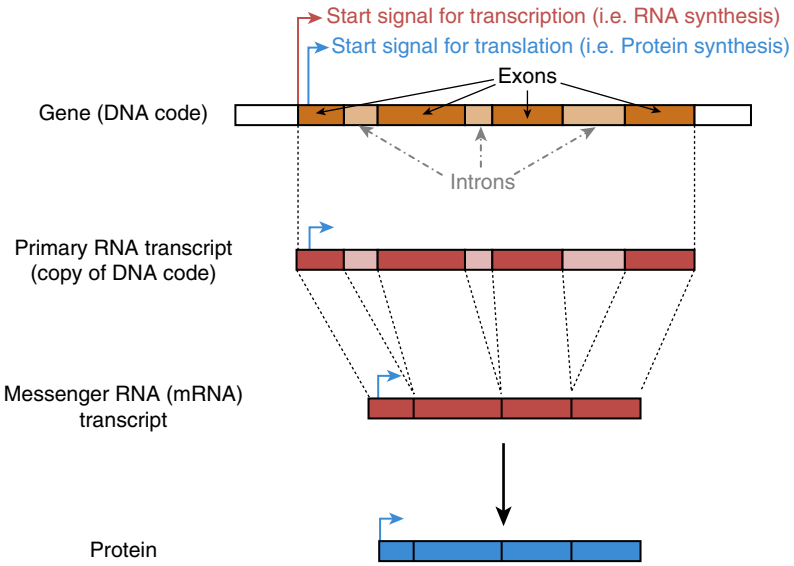


FIGURE 2-2 An illustration of how protein is synthesized from DNA. A gene contains all of the instructions in the DNA code to make a protein. Within our cells, the DNA instructions are transcribed (copied) into a primary RNA transcript by an enzyme called RNA polymerase II. The RNA transcript is processed to form a messenger RNA (mRNA) template that contains only the information that was originally coded in the genes' exon sequences (i.e., removal of the intron information). Then the code for the mRNA template is read (translated) by ribosome complexes in our cells, and protein is synthesized out of amino acids based on the information found in the mRNA.

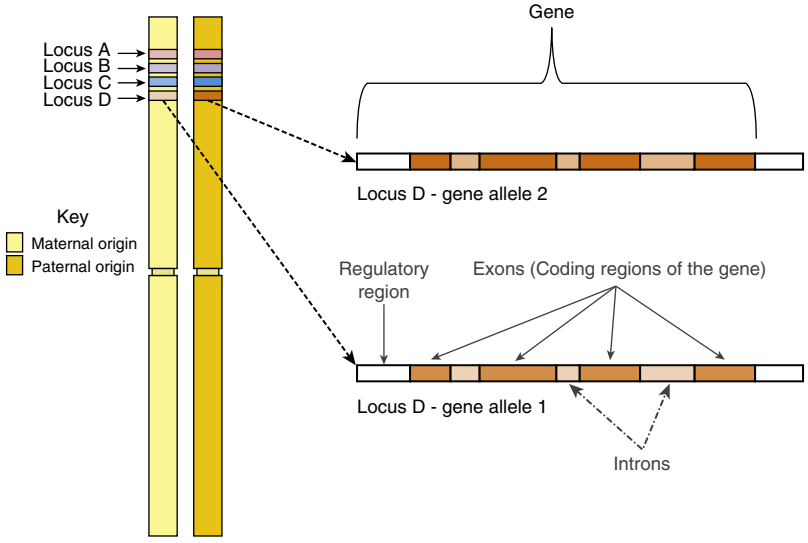


FIGURE 2-3 One autosomal pair of chromosomes illustrating the concepts of four unique gene loci contained on the autosomal pair, multiple alleles, and the general structure of a gene.

Within the human genome, every gene resides in a specific location referred to as a locus. The term *locus* is used when describing a single genetic region or location, and *loci* is plural. Genes at the same locus on a pair of homologous chromosomes are called *alleles*. One allele would be a copy of the maternal allele and the other a copy of the paternal allele. If these alleles are not identical, they can produce different polypeptide sequences and possibly diverse effects. When a pair of alleles are identical in DNA sequence (e.g., allele *A* and allele *A*), the individual is said to be *homozygous* for that locus. However, when the two alleles have one or more differences in the DNA sequence (e.g., allele *A* and allele *a*), the individual is said to be *heterozygous* for that locus. A *genotype* generally refers to the combination of alleles at a given locus within the genome (e.g., *AA*, *Aa*, or *aa*). A person's *genotype* cannot be seen with our eyes but must be determined with the use of a genetic test or analysis.

According to the information gained in the HGP, we now know that the human genome is ~99.9% identical from one person to another.[13] Thus, there is only an estimated 0.1% variation within the entire DNA code between two people that makes each individual unique. So how does this translate to the level of the gene or individual nucleotide? Homologous genes that exhibit more than one allele will vary from one another at the DNA sequence level due to either normal inherited variations or sporadic mutations. The most common inherited variation or sporadic mutation in the human genome is called a *single nucleotide polymorphism* (SNP; pronounced "snip") (Fig. 2-4). SNP describes the occasion when more than one nucleotide base (A, G, T, or C) can be inherited at a specific location in the DNA code upon comparing the DNA codes at that same position among many individuals. There are over 10 million SNPs that have been identified in the human genome to date; ~1 SNP occurs every 300 nucleotides.[13,16] Three basic categories of SNPs exist: (1) intergenic SNPs located in between genes; (2) intragenic SNPs located within the intron regions of a gene; and (3) gene coding region SNPs, which lie within an amino acid coding (exon) region of a gene. Coding region SNPs are further divided into (a) synonymous SNPs in which the variation does not lead to an amino acid change in the protein encoded by the gene and (b) nonsynonymous SNPs in which the variation does result in an amino acid change. Information pertaining to genes and such things as SNP location, the SNP minor allele frequency by ethnic group, and the biological impact of gene variations is available online at the National Center for Biotechnology Information (NCBI) Website (http://www.ncbi.nlm.nih.gov/gene/) and the 1000Genomes Browser (http://browser.1000genomes.org/index.html). Different types of sporadic or inherited variations in the DNA code can also arise from variable number tandem repeats (VNTRs; i.e., microsatellites, simple sequence repeats, short tandem repeats), gene or region duplications, insertions or deletions of a small segment of DNA sequence, inversions of the DNA sequence, translocation of a segment of the DNA sequence, or base-pair changes.

DNA variations are examined and analyzed using numerous methodologies. Large abnormalities in chromosome structure can be studied via karyotyping or genomic hybridization, which are methods that can detect insertions, deletions, translocations, and whole chromosome deletion or duplication. Smaller-scale variations can be studied: (1) within families by linkage analysis or association analysis (i.e., trios of mother, father, and child), and (2) within large populations of unrelated individuals of the same ethnic background by association analysis. These types of studies may specifically test (a) a limited number of genetic markers within a candidate gene/loci; (b) millions of genetic markers (i.e., SNPs or VNTRs) in a genome-wide association study (GWAS); or involve (c) targeted DNA sequencing, whole exome sequencing, or whole genome sequencing.[17]

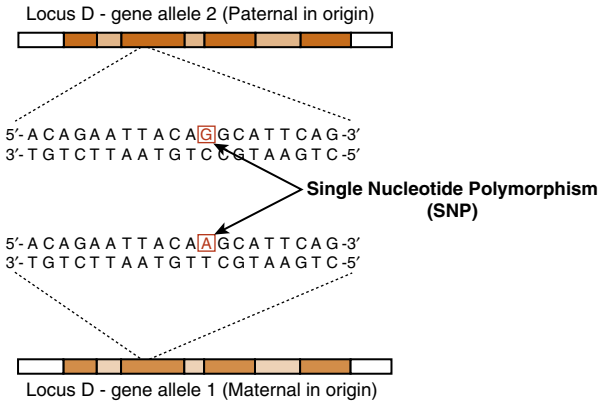


FIGURE 2-4 An example of a single nucleotide polymorphism (SNP).

In contrast to genotypes, *phenotypes* are the observable properties, measurable features, and physical characteristics of an individual.[18] A phenotype is generated by the summation of the effects arising from an individual's genotype and the environment in which the individual develops over a period of time. A *trait* is a particular aspect or characteristic of the phenotype. Examples of traits include eye color, hair color, mandibular jaw size, and stature. When considering genetic influences on traits, it is convenient to think of two types of influences: monogenic (predominantely single gene with the possibility of other smaller genetic and environmental factors), and complex (many genetic and environmental factors). Information from a numbered database/catalog of human traits, syndromes, and genetic disorders associated with monogenic influence is available at the Online Mendelian Inheritance in Man (OMIM) Website (http://omim.org/). Complex traits that are visibly expressed or not, and are not associated with a monogenic syndrome (e.g., nonsyndromic cleft lip/palate, neural tube defects such as spina bifida and anencephaly, or congenital hip dislocation), are also referred to as multifactorial traits where the combination of genetic and environmental factors have to reach a threshold for the trait to be present in that individual (Fig. 2-5).[19] For further information, the reader is referred to the reviews by Mossey,[2,20] Abass and Hartsfield,[21] Lidral et al.,[19] or Hartsfield and Bixler.[22]

TYPES OF GENETIC EFFECTS AND MODES OF INHERITANCE

It is important to understand that it is the *trait*, and *not the gene*, that influences the trait, which can be described as having a specific mode of inheritance (e.g., dominant or recessive). Why is this so? A gene is simply a set of instructions for making something. Hence, a single gene could be "turned on" (i.e., "expressed") to produce a protein that affects the development of one trait in a given tissue or area of the body. The same gene could also be "turned on" to produce the exact same protein, in a different area of the body, which affects the development of a very different trait in another tissue. Any one gene or a specific gene allele, therefore, is technically not dominant or recessive; it is simply a set of instructions. The same gene allele in

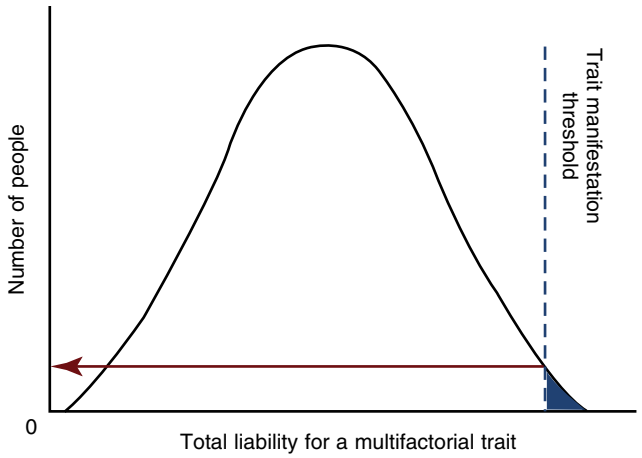


FIGURE 2-5 The liability to have a multifactorial trait is influenced by multiple genes and environmental factors that are distributed throughout a population. However, if some of the population members do not have the trait and some do, then there is a threshold on which a member of the population who has a particular susceptibility to the trait will manifest it. If the genetic liability, environmental liability, or both increase, then the liability distribution curve shifts to the right, increasing the number of persons who are affected.

an individual can influence more than one trait in that person, and each trait may have a different mode by which it is inherited. For example, the melanocortin 1 receptor gene (*MC1R*, OMIM *155555) produces a protein that is involved in the pigmentation of our skin, hair, and eyes. This gene is known to play an important role in the development of two different traits: freckles and red hair. Freckles (ephelides) are inherited as a dominant trait because a person only needs to have one causative copy of the *MC1R* gene to develop them.[23] Red hair, on the other hand, can be inherited as an autosomal recessive trait, where you have two causative copies of the *MC1R* gene to develop red hair,[23] or as a compound heterozygous trait that acts similarly to a recessive trait.[24]

Monogenic Traits

As already noted, traits that develop primarily due to the influence of a single gene locus are termed *monogenic* traits. The traits associated within the peas that Mendel described in his inheritance studies happened to be monogenic; thus, monogenic traits sometimes are called *mendelian traits*. They can have autosomal recessive or dominant, or X-linked recessive or dominant, inheritance. These types of traits also tend to be described as discrete or qualitative (dichotomous or yes/no) in occurrence. However, if they are present, these traits still may be variable and quantifiable in some cases.

Autosomal Dominant Traits and Penetrance

When a trait is present as the result of only one copy of a particular allele (e.g., *A*) in a heterozygous allele pair (e.g., *Aa*), then the trait has an *autosomal dominant* inheritance. If the trait is only present when *both* alleles at the locus are the same (e.g., *aa*; in other words, the individual is homozygous for *a*), then the trait has an *autosomal recessive* inheritance. Although it is actually the phenotype that is actually dominant or recessive and not the gene itself, the terms *dominant gene/dominant allele* and *recessive gene/recessive allele* are used commonly to describe the genes associated with these types of inherited traits in families.

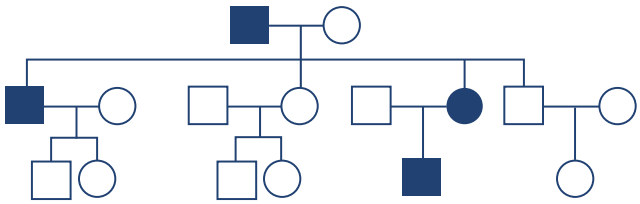


FIGURE 2-6 Three-generation pedigree of a family with an autosomal dominant trait, with the younger generations below the older generations. Square symbols are male and round symbols are female. Affected members are denoted by filling in their individual symbol.

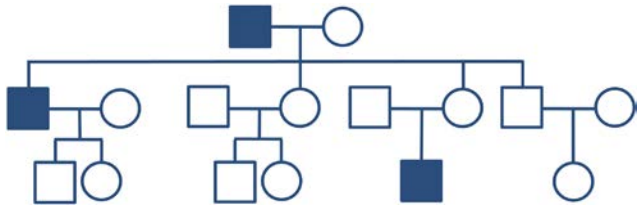


FIGURE 2-7 Three-generation pedigree of a family with an autosomal dominant trait showing incomplete penetrance. Square symbols are male and round symbols are female. Family members who are affected (the phenotype is observable) are denoted by filling in their individual symbol. An individual who does not outwardly show the trait but is able to have offspring with the trait is called a *carrier* of the genetic information that can influence trait formation.

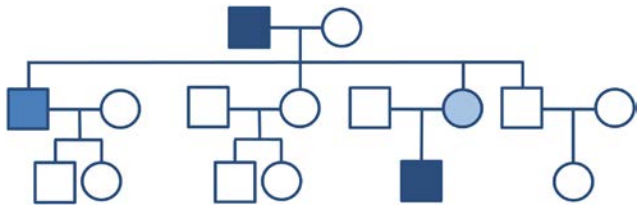


FIGURE 2-8 Three-generation pedigree of a family with an autosomal dominant trait showing variable expressivity. Square symbols are male and round symbols are female. Family members who are affected (the phenotype is observable) are denoted by filling in their individual symbol. Darker symbol filling colors are indicative of a more severe phenotype.

The nature of these family-based (familial) traits can be studied by constructing family trees called *pedigrees* in which males are denoted by squares and females by circles, noting who in the family has the trait and who does not. Constructing a pedigree as a part of the patient's medical history is indicated when more than one member of the immediate family is affected. The practitioner should solicit and record the family history in first-degree relatives of the patient (siblings and parents), second-degree relatives (half-siblings, aunts, uncles, grandparents), and third-degree relatives (first cousins). From this information, a pedigree similar to those in Figures 2-6 to 2-8, 2-10, and 2-11 may be drawn. This can be used to help understand the approximate likelihood that the patient or a sibling may also develop the same trait. This can be particularly useful for monogenic traits including Class III malocclusion, hypodontia, primary failure of eruption (PFE), and developmental dental dysplasias such as types of dentinogenesis and amelogenesis imperfecta. A family history may also be useful for complex traits such as Class II/division 2, external apical root resorption

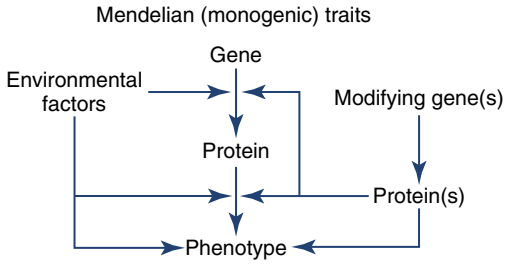


FIGURE 2-9 Mendelian (monogenic) traits or diseases result because a single gene polymorphism or mutation usually results in a recognizable phenotype. Environmental factors and other genes may modify the clinical expression of the disease or other type of trait but are not of crucial importance for its development. (From Abass SK, Hartsfield JKJ. Investigation of genetic factors affecting complex traits using external apical root resorption as a model. *Semin Orthod.* 2008;14:115–124.)

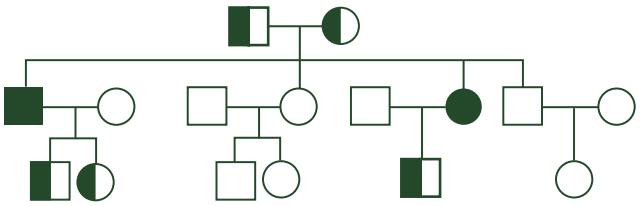


FIGURE 2-10 Three-generation pedigree of a family with an autosomal recessive trait. The symbols for presumed carriers (heterozygotes) of the autosomal recessive gene are filled in halfway. Some other family members also may be carriers but cannot be determined strictly from the pedigree.

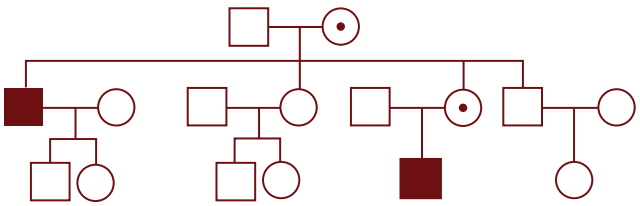


FIGURE 2-11 Three-generation pedigree of a family with an X-linked recessive trait. The symbols for presumed female carriers (heterozygotes) of the X-linked recessive gene have a dot in the middle of the circle. Some other female family members also may be carriers but cannot be determined strictly from the pedigree.

(EARR), palatally displaced canines (PDC),[25] or any trait that occurs in more than one member of the family.

The findings in the patient, combined with possible findings in other family members, may help to diagnose the presence of a syndrome or trait that may not have been previously diagnosed. For example, the presence of a single primary and permanent maxillary incisor at first may appear to be a product of dental fusion. However, if the single tooth is in the midline and symmetric with normal crown and root shape and size, then it may reflect an isolated phenotypic finding, or it could be part of the solitary median maxillary central incisor syndrome.[26] Other findings that may be syndromic in nature or of medical interest because they can be an indication for referral to a physician and/or clinical/medical geneticist include (1) dental agenesis with features of an ectodermal dysplasia, taurodontism, and/or other radiographic findings; (2) midface/malar hypoplasia, especially in the patient with any medical findings even if they seem unrelated to their craniofacial/oral findings; and (3) developmental delay. A comprehensive summary of craniofacial/oral findings along with possible syndromes that they may be a part of can be found in a resource created by Hartsfield.[27] This resource includes brief discussions and references addressing the orthodontic management of these types of cases, in addition to information on how to find and refer your patients to a clinical/medical geneticist.

If the mode of inheritance is autosomal dominant, the following characteristics may be present in a pedigree: (1) the trait occurs in successive generations (Fig. 2-6); (2) on average, 50% of the offspring of each parent who has the trait will also have the trait; (3) if an individual has the gene allele that results in the trait, each of his or her children has a 50% chance of inheriting the gene allele that leads to the expression of the trait; (4) males and females are equally likely to inherit the trait; and (5) parents who do not have the trait have offspring who do not have the trait. An exception to this occurs when the trait shows nonpenetrance in a particular offspring.

When a person inherits a gene allele or genotype that characteristically is associated with a specific trait, yet the trait is not evident in that person, then the trait is said to show *nonpenetrance* in that individual and *incomplete penetrance* (Fig. 2-7) in any group of individuals who have the genotype. The trait is present or not (nonpenetrant) in an individual. If some of the individuals do not manifest the trait in a sample of individuals with the trait-associated genotype, then the trait is said to have a penetrance of whatever percentage of the trait-associated genotype that the group actually manifests. Incomplete penetrance is a condition most commonly observed with dominant traits such as hypodontia, Class III malocclusion, and Treacher Collins syndrome, to name a few. Exceptions to this may include (1) when a new (sporadic) mutation is introduced into the DNA of the sperm or egg that will form the offspring or (2) when a germinal mosaicism arises because one of the parents was *mosaic* at the germ cell level (i.e., the sperm or eggs generated by the affected parent arise from two unique germ cell lines—one germ cell line carries a mutation and one germ cell line does not). Thus, *mosaic* is a term that describes an individual who has two of more cell populations in their body, with different genotypes, that originally came from a single fertilized egg but genetically diverged from each other during some stage of growth and development.[28] Chance usually determines which sperm cell line will be passed on. The other obvious exception is nonpaternity. Although this is not strictly a genetic problem, the illegitimacy rate in the U.S. population is high enough to make this a possible explanation for a couple without the trait to have a child with a completely penetrant dominant trait.

Variable Expressivity

Although the trait in each individual may either be present or not when discussing penetrance, if the trait is present, it may vary in its severity or degree of expression. Thus, not all individuals with the trait may have it to the same extent and they may express varying degrees of effect or severity (Fig. 2-8). Variable expressivity also may apply to the pleiotropic effect of a particular genotype; that is, the expression of the same gene may result in seemingly disparate traits in an individual. The occurrence of two or more traits appearing together, more often than what would be expected by simple chance, defines a *syndrome*. Although the term *genetic syndrome* often is used, not all syndromes necessarily have a strong genetic basis, e.g., fetal alcohol syndrome.

Variable expressivity, even in the same family with presumably the same segregating primary genetic determinant, may be observed with a large number of dominantly inherited traits, syndromes, and conditions, including Class III malocclusion,[29] hypodontia,[30] osteogenesis imperfecta involving type I collagen abnormalities,[31,32] and craniosynostosis syndromes.[33-35]

This phenomenon is presumably due to the variable interaction(s) of different proteins encoded by modifying genes plus environmental/epigenetic factors occurring in each individual (Fig. 2-9). These examples give a clear message: even an extreme phenotype that is typically associated with an autosomal dominant mutation can display some degree of variability. Simply discovering the likely causal gene mutation may indicate a future effect on craniofacial growth and development, but it will not necessarily predict the precise effect. This should be borne in mind when hoping for a precise prediction of phenotype based on genetic analysis of a complex trait, with many and not just one main genetic factor to consider.

Autosomal Recessive Traits

As previously stated, an autosomal recessive trait requires the inheritance of two causal allele copies to see an observable phenotype (i.e., homozygous *aa*). The concept of having a "gene carrier" is used regularly with autosomal recessive traits. Both parents of a child with the autosomal recessive trait are typically heterozygous for the gene allele that causes the recessive trait (i.e., they are a carrier of only one copy of the "causal" allele) and most often have a normal phenotype. Sometimes, however, the carrier status can be detected, greatly improving the precision of genetic counseling, even before a child is born with the recessive trait. The rarer the recessive gene allele, the more likely it is that the unaffected parents who have an affected child will be blood relatives—that is, a *consanguineous mating*. Still, it is highly probable that each of us carries a number of recessive-trait gene alleles, making it possible for biologically unrelated couples also to have a child with an autosomal recessive trait, condition, or syndrome. A study on inbreeding in Japan by Schull and Neel that was cited by Niswander[36] found that malocclusion occurred 6% to 23% more often (depending on the sample and the sex) in children of first cousins compared with children of nonrelated parents, indicating the potential for the effect of recessive genes when homozygous. Given that both parents who produce a child with an autosomal recessive trait are presumed to be heterozygotes, only one of the four possible gene combinations from the parents will result in the homozygous genotype associated with the autosomal recessive trait. Hence, the recurrence risk for an affected child in this case is 25% (i.e., $Aa \times A^*a^*$ would yield offspring with an approximately equal distribution of the genotypes: AA^*, Aa^*, aA^*, or aa^*, where only the aa^* genotype can manifest the recessive trait). Transmission of the phenotype in a pedigree is horizontal (typically present only in siblings) and not vertical, as with a dominant trait (Fig. 2-10).

X-Linked Traits and Lyonization (X Inactivation)

Most genes located on the X and Y chromosomes are not homologous and are unequally distributed between males and females. This inequality occurs because males inherit only one copy of the X chromosome along with one copy of the Y chromosome, compared to females who inherit two X chromosomes. Many of the unique genes found only on the Y chromosome influence the development of the male reproductive system. Since females inherit two X chromosomes, it is possible for them to be either homozygous or heterozygous at each X-linked gene locus, in a similar fashion to loci located on autosomal chromosomes. By comparison, however, males only inherit one X chromosome normally, and while some loci on the males' X chromosome do have a homologous locus on the Y chromosome, most loci on the males' X chromosome do not have a homologous locus in the males' genome. The term *hemizygous* is used to describe the fact that men inherit only half of the number of X-linked genes (one copy) that females inherit, a condition that can lead to interesting genetic phenotypes that are possible only in men. Accordingly, since a normally functioning homologous allele is not present on any other chromosome for some of the X-linked gene alleles in males, recessive genes located on the one male X chromosome express themselves phenotypically as if they were dominant genes. In females, both X-linked recessive genes must be present at a homologous locus to express the recessive phenotype. Consequently, full expression of rare X-linked recessive phenotypes is almost entirely restricted to males, e.g., in X-linked hypohidrotic ectodermal dysplasia (HED), although occasionally it is seen with variable severity in females (Fig. 2-11). Females who are heterozygous for the gene associated with the X-linked recessive phenotype may show some expression of the phenotype because most of the genes on one of the X chromosomes in the female will normally be inactivated by a process called *lyonization.*

Complex Traits

The predominant role of genetics in the clinic has focused on the study of chromosomal and monogenic phenotypes that are clearly associated with specific changes (mutations) in the genome of the individual. New knowledge and techniques, however, have enabled the study of various conditions and traits that "run in families" but do not adhere to patterns of mendelian inheritance. These conditions are referred to as *complex* or *common* diseases, phenotypes, or traits, reflecting their complex etiologic interaction among genes from more than one locus and environmental factors (Fig. 2-12), as well as their greater incidence/more common occurrence when compared with monogenic phenotypes.

The genetic influence on a complex trait takes place through many gene loci collectively asserting their influence on the trait. Historically, each gene involved in creating the trait was thought to have a minimal effect by itself but that the effect of all genes involved was additive. The associated phenotype is rarely discrete and is most commonly continuous or quantitative. Because these traits show a quantitative distribution of their phenotypes in a population, they do not show mendelian (monogenic) inheritance patterns. Environmental factors can play a variable and generally greater role in complex traits than in monogenic traits. A change in phenotype depends on the result of the genetic and environmental factors present at a given time. Thus, one may expect that compared with monogenic traits, complex traits will be more amenable to change (or a greater change) following environmental/treatment modification.

Another important aspect to consider, especially with complex traits, is the fourth dimension of development—time. Although an environmental/treatment modification may alter the development of the phenotype at a particular moment, gross structural morphology, already present, may not change readily unless the environmental modification is sufficient to alter preexisting structure or function.[12] This reinforces the possibility that understanding or even changing the genetic factors influencing the malocclusion may not be the same genetic factors that would influence the treatment outcome.

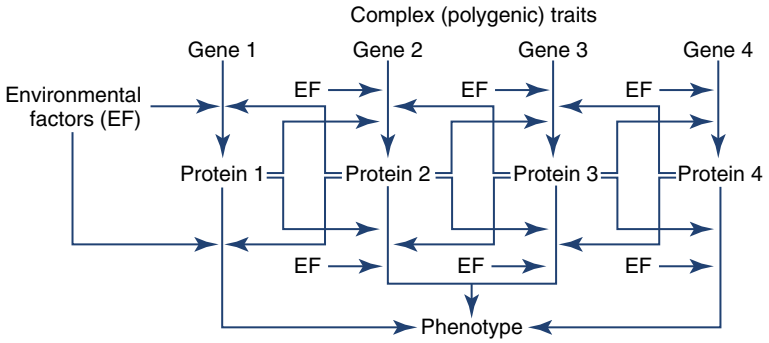


FIGURE 2-12 Unlike mendelian traits, environmental factors and multiple genes are critical to the development of complex (polygenic) traits. These types of physical traits are continuous rather than discrete (although diseases of this type can still be present or not). Such traits are referred to as quantitative traits or multifactorial because they are caused by some number of genes in combination with environmental factors. (From Abass SK, Hartsfield JKJ. Investigation of genetic factors affecting complex traits using external apical root resorption as a model. *Semin Orthod.* 2008;14:115–124.)

NATURE VERSUS NURTURE

Consideration of which factors influence, determine, or even drive growth and development has often led to discussion, if not debate, regarding the influence of nature versus nurture; as though there could only be the influence from one or the other. Growth and development, however, are not simply the results of genetic (nature) or environment (nurture) working in complete absence or independence of other. Genetic factors refer to the actual DNA code that is inherited. Environmental factors, in contrast, can include such things as diet, living conditions, stress, and learned behaviors that may influence a person's mindset, perception, and/or epigenetic landscape.

The term *epigenetic* is used to describe heritable changes to the structure of chromatin (DNA packaged around histones) that directly influence how genes are turned on and off. This type of regulation occurs in the absence of any code changes within the actual DNA sequence and can be reversible. While our DNA code provides the necessary instructions for how to make a polypeptide, a person's epigenetic landscape helps to determine what polypeptides will be made, when they will be made, and where they will be made. An *epigenetic landscape* refers to the specific pattern of modifications present on the DNA double helix backbone and/or the histone protein tails that individuals acquire from their parents and over their own lifetime. Modification of the DNA double-helix backbone by methylation, combined with various amino acid modifications on histone proteins (i.e., methylation, acetylation, phosphorylation, ubiquitination, and/or sumylation) specifically acts to "open" or "close" the regional chromosome structure to enhance or shut down gene expression. While MZ twins are identical at the level of their actual DNA code, differences in their epigenetic landscapes can still generate phenotypic differences between them. Thus, environmental factors may not only cause a change in the DNA sequence through mutations, but they may also alter gene expression (short term and long term) through epigenetic regulation. Genetic mutations, as well as epigenetic patterns located on the DNA backbone and histone protein complexes, can be inherited.[14,37]

Full siblings share, on average, half of their genes, and studies have shown that siblings can have similar occlusions. Developing similar occlusions, however, is influenced in some part by environmental factors (e.g., influenced by having dietary and respiratory factors in common) and in some part by genetic factors that influence development.[38-40] For example, malocclusion is less frequent and less severe in populations that have not been industrialized (i.e., nonurbanized) and that tend to be isolated. Typically, an increase in the occurrence of malocclusion has been noted as these populations become more "civilized" or increasingly urbanized. This has been attributed to the interbreeding of populations with different physical characteristics, presumably resulting in a synergistic disharmony of tooth and jaw relationships. This idea was supported by the crossbreeding experiments of Stockard and Anderson,[41] utilizing inbred strains of dogs that showed an increased incidence of malocclusion, typically caused by a sagittal mismatch of the jaws. The craniofacial anomalies that were produced in these experiments, however, have been attributed largely to the influence of a major gene, or collection of genes, that have been selectively bred to be part of specific canine breeds. Considering the polygenic nature of most craniofacial traits, it seems improbable that racial crossbreeding in human beings could resemble the condition of these experiments and thereby result in a synergistic increase of oral-facial malrelations.[6,36,42]

In a study of individuals from disparate ethnic groups that have interbred in Hawaii, it was discovered that children of racial crosses are at no increased risk of malocclusion beyond what would have been expected from the usual parental influence. In addition, the increase in malocclusion within populations that have moved recently into an industrialized lifestyle occurred too quickly to be attributed to genetic change caused by evolutionary fitness pressure.[42] The most likely explanation for the observed increase in the occurrence of malocclusion in "civilization" is environmental change, such as the types of foods being consumed and airway effects.[43]

Not to be lost in this discussion is the understanding that how the individual responds to environmental changes is influenced by genetic factors. Moss[8] concluded that both are necessary in a revisitation of the functional matrix hypothesis and resolving synthesis of the relative roles of genomic and epigenetic

processes and mechanisms that cause and control craniofacial growth and development. Neither genetic nor epigenetic factors alone are sufficient, and only their integrated (interactive) activities provide the necessary and sufficient causes of growth and development. Moss considered genetic factors as intrinsic, while prior causes and epigenetic causes were extrinsic and proximate. The phrase *form follows function* has been used to explain that skeletal development is secondary to muscle function, airway requirements, and other causes extrinsic to the bone. However, what about genetic and epigenetic effects on muscle that then affect skeletal development? Recent research by Sciote and collaborators has shown that variations in masseter muscle fiber type, gene expression in masseter muscle, and epigenetic changes are associated with anterior open versus deep bites, mandibular retrognathism versus prognathism, and mandibular asymmetry.[44-47]

Skeletal muscle cells produce many proteins that, when in combination, define the unique characteristics and function of the muscle fiber tissue. These proteins include myosin heavy chain (MHC) protein isoforms such as (MYH7 (I), MYH2 (IIA), and MYH7 (IIX)) and cytoskeletal muscle proteins such as α-actinin-2 and -3. While α-actinin-2 appears to be found in all types of skeletal muscle fibers, α-actinin-3 is restricted to fast-contracting type II muscle fibers[48] where it enhances muscle force.[49] The proportions of muscle fiber type may be varied during growth through interactions of α-actinin-3 and calcineurin to influence variation in muscle function. In studies of muscle composition, at least four unique muscle fiber types can be described, including type I MHC-protein containing muscle fibers (termed *type I*), type IIA and/or type IIX MHC protein containing muscle fibers (termed *type II*), hybrid type I/II muscle fibers containing a mixture of both type I and II MHC proteins, and neonatal/atrial type muscle fiber containing neonatal or α-cardiac muscle isoforms combined with some type I and II MHC isoforms. Interestingly, differences in muscle fiber composition have been noted in masseter muscle tissue obtained from patients with a mandibular asymmetry. Significant increases in type II muscle fiber area and frequency on the same side as the deviation were discovered when compared to muscle fibers on the side opposite the deviation. Moreover, no significant differences were noted when comparing the muscle composition on the right and left sides of symmetrical patients.[45] Additional studies have shown that greater human facial height (i.e., vertical dimension) is inversely related to the size and proportion of masseter muscle fast, type II muscle fibers. Accordingly, short-faced, deep bite phenotypes correlated with increased type II fiber area and frequency, while long faced, open bite phenotypes showed increased type I fiber area and frequency.[44,46,47]

In contrast to the muscle fiber variations observed in vertical dimension malocclusions, muscle fiber compositions varied to a lesser degree in malocclusions affecting the sagittal dimension, although type IIA and IIX MHC proteins were expressed in the masseter muscle of individuals with mandibular prognathism (MP).[46,50] This may help to explain the relative difficulty in maintaining an overbite correction, particularly of a deep bite. Among Class III cases, differences were observed in the average fiber area when comparing normal, open bite and deep bite cases. Class III deep bite cases showed an increased amount of type I and hybrid type I/II muscle fiber areas in the masseter muscle, compared to normal and open bite cases. Sex differences have also been observed between masseter muscle fiber type and size, along with muscle growth factor–related gene expression levels.[46] Interestingly, when the *α-actinin-3* gene (*ACTN3*) alleles were homozygous for the polypeptide to stop being made at the 577th amino acid (577Stop/Stop), the patient was more likely to have a Class II malocclusion, while this genetic variation was less likely to be found in patients with a deep bite.[49]

Epigenetic factors may also influence muscle fiber types, vertical and/or sagittal dimension variations, and malocclusion types. Masseter muscle from patients with deep bite malocclusions had a higher level of gene expression (i.e., greater mRNA production) for both histone deacetylase 4 (*HDAC4*) and lysine acetyltransferase 6B (*KAT6B;* OMIM 605880) compared to masseter muscle from patients with an open bite phenotype. Since the enzymes encoded by these two genes play an important role in regulating histone acetylation patterns, it has been suggested that masseter muscle fiber variations may be influenced via an increased acetylation of histones resulting in an opening of chromatin (DNA wrapped around histone proteins) and upregulated gene expression for fast (type II) MHC genes combined with an increased deacetylation of histones and the closing of DNA containing the slow-contracting (type I) MHC gene.

In support of this hypothesis, increased gene expression of *HDAC4* was associated with increased gene expression of the fast type IIX MHC (*MYH1*) and decreased gene expression of slow type I MHC (*MYH7*) in subjects diagnosed with Class II. Increased gene expression from the *KAT6B locus*, in contrast, correlated negatively with type IIX MHC (*MYH1*) gene expression in Class III malocclusions.[44] Overall, gene expression at both *KAT6B* and *HDAC4* loci were elevated in masseter muscle from patients with Class III malocclusions compared to individuals diagnosed with Class II.[44,51] It has been proposed that the KAT6B protein could play a potential role in MP through its ability to activate the runt-related transcription factor 2 gene (*RUNX2*), which encodes an osteogenic transcription factor.[51] Finally, the genetic variation located near the *myosin 1H gene* (*MYO1H* rs10850110; OMIM 614636), which encodes an unconventional class I myosin protein, has been associated with MP.[52] The significance of this is yet to be determined, as expression levels of *MYO1H* are extremely low in masseter muscle biopsies.[51] Clearly, this new muscle-related research field will continue to add much to the understanding of the variations that result in different malocclusions, especially since the described correlations do not explain all of the phenotypic variation observed. It is likely that additional muscle, cartilage, growth factors and their receptors, and transcription factors and other genes and proteins may influence skeletal growth.

Heritability and Its Estimation

Heritability estimates can range from 0 to 1. A trait with a heritability estimate of 1 would be expressed with complete positive correlation to genotypic factors theoretically, as measured by comparing the concordance of the phenotype to the percentage of genes in common—for example, among twins or other siblings. By comparison, a trait with a heritability of 0.5 would have half its variability of concordance (from individual to individual) positively correlated with the percentage of genes in common. One must remember some important aspects of heritability studies when reviewing them in the literature. First, hereditary estimates are just that, estimates of genetic and environmental contributions that may have been affected by not accounting

for a common environmental effect and ascertainment bias. They only include additive genetic influences and do not take into account genetic and environmental interactions.[39,53] In addition, heritability estimates refer to a specific sample and do not necessarily pertain to the situation of a given individual, even from within the sample. Thus, they do not allow one to tell to what degree a particular trait was determined by genetic or environmental factors in a single individual. Finally, heritability estimates are descriptive of variances within a sample at a given time; they are not predictive.[54]

Although the mode of inheritance (e.g., autosomal dominant or polygenic) of a trait is a fixed property in a given individual, heritability is not.[55] It is important to understand that heritability estimates can change with age. This was demonstrated in a longitudinal analysis of 30 sets of siblings, none of whom had undergone orthodontic treatment, which showed a significant increase overall in median heritability estimates between the ages of 4 and 14 years for 29 craniofacial skeletal variables. The affected variables included increases for total anterior face height, upper anterior face height, total posterior face height, and upper posterior face height. Still, despite the general increasing trend in heritability estimates for the craniofacial skeletal variables, a decrease was noted for lower posterior face height. The median estimates of heritability for craniofacial skeletal variables increased from 0.6 at age 4 years of age to 0.9 at age 14 and 20 years of age. This is in contrast to the heritability estimates of arch and occlusal variables that decreased from 0.5 at age 4 years to 0.2 and 0.1 at ages 14 and 20, respectively.[56]

Numerous studies have examined through heritability estimates how genetic variation in a sense correlates with either or both occlusal and skeletal variation among family members.[1,6,53,56-76] In most studies (particularly those that try to account for bias from the effect of shared environmental factors, unequal means, and unequal variances in monozygotic (MZ) and dizygotic (DZ) twin samples),[10] variations in cephalometric skeletal dimensions are associated in general with a moderate to high degree of genetic variation, whereas variation of occlusal relationships has generally little or no association with genetic variation.[5]

Although the heritability estimates are low, most of the studies that looked at occlusal traits found that genetic variation has more to do with phenotypic variation for arch width and arch length than for overjet, overbite, and molar relationships. Still, arch size and shape are associated more with environmental variation than with genetic variation.[61] Even so, recent preliminary GWAS data suggest relationships of the protein kinase D1 (*PRKD1*) gene with anteroposterior (AP) discrepancies between the maxillary and mandibular arch, the *Caenorhabditis elegans* homolog of the RNA-binding protein FOX1 (*RBFOX1*) gene with arch width variation, and the homolog of the drosophila dachshund 1 (*DACH1*) gene with AP discrepancies with variation in the curve of Spee.[77] Although these findings are among the first to identify variation in dental arch shape and position that may be related to variants in these genes, the mechanisms by which they affect these phenotypes are unknown.

The heritability of a trait cannot necessarily be extrapolated from one sample and set of environmental conditions to another.[9] For example, heritability estimates of lower anterior face heights change more than upper anterior face heights in a group of subjects who have a change in their breathing pattern.[78] Therefore, a high heritability cannot measure if a trait can be influenced substantially by subsequent changes in environmental/treatment conditions.[5,9] Still, confirming a certain degree of genetic influence on a trait for a particular sample in a particular environment at a particular time is a preliminary step to further specific genetic studies to determine areas of the genome that appear to be associated with the characteristics of a given trait.[54]

Use of Family Data to Predict Growth

Siblings have been noted as often showing similar types of malocclusions. Examination of parents and older siblings has been suggested as a way to gain information regarding the treatment need for a child, including early treatment of malocclusion.[36,74,79,80] Niswander[36] noted that the frequency of malocclusion is decreased among siblings of index cases with normal occlusion, whereas the siblings of index cases with malocclusion tend to have the same type of malocclusion more often. Harris and colleagues[68] showed that the craniofacial skeletal patterns of children with Class II malocclusions are heritable and that a high resemblance to the skeletal patterns occurs in their siblings with normal occlusion. From this it was concluded that the genetic basis for this resemblance was probably due to multiple genetic factors, and family skeletal patterns were used as predictors for the treatment prognosis of the child with a Class II malocclusion, although it was acknowledged that the current morphology of the patient is the primary source of information about future growth.[80]

Although each child receives half of his or her genes from each parent, they are not likely the same combination of genes in each sibling, unless the children are MZ twins. When looking at parents with a differing skeletal morphology, it is difficult to know which of the genes, in what combination from each parent are present in the child until the child's phenotype matures under the continuing influence of environmental factors. As Hunter[81] pointed out with polygenic traits, the highest phenotypic correlation that can be expected based on genes in common by inheritance from one parent to a child, or between siblings, is 0.5. Because the child's phenotype is likely to be influenced by the interaction of genes from both parents, the "mid-parent" value may increase the correlation with their children to 0.7 because of the regression to the mean of parental dimensions in their children.

Squaring the correlation between the two variables derives the amount of variation predicted for one variable in correlation with another variable. Therefore, at best, using mid-parent values, only 49% of the variability of any facial dimension in a child can be predicted by consideration of the average of the same dimension in the parents. Only 25% of the variability of any facial dimension in a child can be predicted, at best, by considering the same dimension in a sibling or one parent. Because varying effects of environmental factors interact with the multiple genetic factors, the usual correlation for facial dimensions between parents and their children is about 30%, yielding even less predictive power.[81]

Unfortunately, orthodontists usually do not have sufficient information to make precise and accurate predictions about the complex development of occlusion simply by studying the frequency of its occurrence in parents or even siblings. Still, family patterns of resemblance are frequently obvious and are ignored at our risk. The taking of a family history as already mentioned, especially for traits that have or can have a monogenic inheritance, can alert practitioners to the increased likelihood of the same trait developing in their patient.

FACIAL GROWTH

Growth Differences during Puberty

Approximate facial growth predictions based upon expected growth curves may be useful for the average patient, but a more precise and valid prediction must incorporate and account for the variation associated with individual genetic factors, including those that are highly pertinent to the pubertal growth spurt. The pubertal growth spurt response is mediated by the combination of sex steroids, growth hormone, insulin-like growth factor, and other endocrine, paracrine, and autocrine factors. The administration of low doses of testosterone in boys with delayed puberty not only accelerates their statural growth rate but their craniofacial growth rate as well.[82]

In addition to testosterone, estrogens are also a group of hormones involved in growth and development.[83] Aromatase (also known as estrogen synthetase) is a key cytochrome P450 enzyme involved in estrogen biosynthesis by catalyzing the final rate limiting step of converting testosterone and androstenedione to estradiol and estrone, respectively.[84] *CYP19A1* is the gene that encodes aromatase; therefore, regulation of this gene's transcription is critical for the testosterone/estrogen (T/E) ratio in the body. Some studies have shown that the T/E ratio is critical in the development of sex-indexed facial characteristics such as the growth of cheekbones, the mandible and chin, the prominence of eyebrow ridges, and the lengthening of the lower face.[85,86]

A significant difference in the average sagittal jaw growth was observed between the groups of Caucasian males examined who inherited different *CYP19A1* alleles/genotypes (rs2470144-T/T versus C/T or C/C, and rs2445761-T/T versus C/T or C/C); with the greatest differences in growth per year just over 1.5 mm per year during treatment for the maxilla, and 2.5 mm per year for the mandible. There was no statistical difference for the particular *CYP19A1* alleles in females. This is particularly impressive since at the beginning of treatment there was no significant difference among the males based upon the *CYP19A1* genotype. The significant difference only expressed itself over the time of treatment during the cervical vertebral stage associated with increased growth velocity.[87]

Interestingly, the same result was found in a group of Chinese males and females, strongly suggesting that this variation in the *CYP19A1* gene may be a multiethnic marker for sagittal facial growth.[88] Although the differences in average annualized sagittal mandibular and maxillary growth based upon the *CYP19A1* genotypes were significant, i.e., associated with the complex trait of sagittal jaw growth, they accounted for only part of the variation seen, and therefore by themselves have little predictive power. Further investigation of this and other genetic factors and their interactions with each other and with environmental factors will help to explain what has up to now been an unknown component of individual variations in pubertal facial growth.

Mandibular Prognathism/Class III Malocclusion

Searching for the term *malocclusion* in OMIM (http://omim.org/) reveals more than 100 entries for a variety of syndromes and traits that may include malocclusion as one of their features. Perhaps the best known example is the familial "MP" (OMIM *176700) referred to as the *Hapsburg jaw*. Although MP has been said to be a polygenic[79] or multifactorial trait (i.e., influenced by the interaction of many genes with environmental factors), in the majority of cases, there are families in which the trait (and possibly some other associated findings) appears to have autosomal dominant inheritance, such as in the European noble families. Analysis of a pedigree comprising 13 European noble families with 409 members in 23 generations determined that the MP trait was inherited in an autosomal dominant manner, with a penetrance of 0.95 (i.e., 95% of the time that someone was believed to have the gene for the MP trait in his or her pedigree, the trait itself also was expressed). Although the penetrance is high, considerable variation exists in the clinical expression of the trait, as already mentioned.[89]

Additional studies have supported the autosomal dominant mode of inheritance with variable expressivity and incomplete penetrance,[90] which may also have a major gene and multifactorial influence.[29]

Also noted was that some of the members of the European noble families had, in addition to varying degrees of MP, other facial characteristics such as a thickened lower lip, prominent nose, flat malar areas, and mildly everted lower eyelids (which may be associated with a hypoplasia of the infraorbital rims), as also were reported in three generations of a family by Thompson and Winter.[91] Apparent maxillary hypoplasia, as well as malar flattening and downward eversion of the lower eyelids, may indicate that although the trait is referred to as MP, the overall clinical effect may be at least in part due to hypoplasia of the maxilla.

Clinically, we observe a variety of anatomic changes in the cranial base, maxilla, and mandible that may be associated with "MP" or a Class III malocclusion.[92,93] Understanding the concept of phenotypic and genetic heterogeneity is critical to understanding the genetic influences on all types of phenotypes.[94] For example, although orthodontists often first classify a malocclusion as Angle Class I, II, or III, we also know that a number of different subtypes of occlusions have varying genetic and environmental influences. The concept of further delineating the Angle classification has clinically been done for the Class II division 1 and 2 phenotypes. A rationale for further subtypes is based on statistical cluster analysis of cephalometric variables that may or may not have undergone a Procrustes transformation to standardize for size, leaving differences in relative size and shape to undergo morphometric analysis. Most of the studies have sought to delineate Class III subtypes, finding either five or seven of them as separate clusters.[95] Other studies have done the same with Class II subjects.[96,97] Since they analyze different populations with sometimes different methods, it is not surprising that they do not all conclude with the same number of clusters or subtypes. Still, the clustering effect based on cephalometric morphology could have clinical importance as the subtypes may be treated differently, or have different outcomes, or retention concerns. We already do this to some degree based on observation of subtypes based on open bite versus deep bite, etc. In addition, the subtypes may be more homogeneous endpoints to explore the genetic and environmental factors that produce them and how they respond to different treatment and retention modalities.

The prevalence of Class III malocclusion varies and can show different anatomic characteristics among different ethnic groups. Considering this heterogeneity and possible epistasis (the interaction between or among gene products on gene expression), it is not surprising that genetic linkage studies to date have indicated the possible location of genetic loci influencing this trait in several chromosomal locations.[95,98] In studies of Asians, genetic linkage and association studies

have identified multiple loci and candidate genes connected to the Class III phenotype, including 1p22.3, 1q32.2, 1p35-36 (matrillin-1, *MANT1;* erythrocyte membrane protein band 4.1, *EPB4.1*; heparin sulfate proteoglycan 2, *HSPG2*; alkaline phosphatase, *ALPL*), 3q31.2, 4p16.1, 6q25, 12q13 (collagen, type II, alpha 1, *COL2A1*), 14p24.3, and 19p13.2.[99-105] By comparison, several unique loci were identified in linkage analysis of the Class III phenotype in multiple South American families including 1p22.1-22.2, 3q26.2, 7p21, 11q22.2-q22.3, 12q13.13, and 12q23.[106,107] In an association analysis of Class III malocclusion cases with a U.S.-based population compared to Class I and comprised of multiple ethnic backgrounds (European, African, Hispanic, and Asian), the SNP rs10850110 within the *MYO1H* on chromosome 12q24.11 was found to be associated with Class III, as already mentioned in the discussion of the effects of muscle variation.[52] While numerous genetic loci have been associated with Class III, so far only four causal genetic mutations have been identified in familial Class III malocclusions within the following genes: (1) dual specificity phosphatase 6 (*DUSP6*) gene (c. 3361G>A; p.Ser182Phe; rs139318648) in a family from Estonia[108], (2) Rho GTPase activating protein 21 (*ARHGAP21*) gene (c. 3361G>A; p.Gly1121Ser; rs111419738) in an Italian family:[109], (3) a mutation in the fibroblast growth factor 23 (*FGF23*) gene (c.35C>A; p. Ala12Asp; no assigned rs number) in a family from the Henan Province of China new[110], and (4) a mutation in the ADAM Metallopeptidase with Thrombospondin Type1, motif 1 (*ADAMTS1*) gene (c.2225T>C; p.Ile742Thr; rs200052788) in a Chinese family new[111].

Class II Division 2 (II/2) Malocclusion

The Class II division 2 (II/2) malocclusion is a relatively rare type of malocclusion, representing between 2.3% and 5% of all malocclusions in the western Caucasians.[110a,111a] There is evidence that Class II/2 can have a genetic component based upon a twin study in which all 20 MZ twin pairs were concordant for Class II/2, while only 10.7% of 28 DZ twin pairs were concordant.[112] The much lower concordance for DZ twins suggests that more than one genetic factor contributes to Class II/2. Further evidence for Class II/2 to have a polygenic complex etiology was found in a study of 68 Class II/2 patients, with a relative risk (RR) of first-degree relatives of the patients to have a Class II/2 of 3.3 to 7.3. The 95th percentile confidence interval was 1.1 to 10.3 if the RR was 3.3 and 1.7 to 31.6 if the RR was 7.3.[113]

There is a strong association of Class II/2 with dental developmental anomalies, more so than for other Angle malocclusion classes. Dental agenesis excluding third molars was at least three times more common in Class II/2 subjects than in the general population.[114,115] In addition, there is a statistically significant reduction in permanent maxillary incisor mesial-distal width associated with Class II/2,[116] which could influence anterior Bolton discrepancies.

TOOTH SIZE AND AGNESIS

Dental Crown Morphology

Investigation of the genetic and environmental factors that affect dental crown morphology, especially mesial-distal dimensions, is important since tooth size variation may more often play a role in skeletal Class I crowding than skeletal growth variation.[117-120] Additive genetic variation for mesial-distal and buccal-lingual crown dimensions of the permanent 28 teeth (excluding third molars) ranged from 56% to 92% of phenotypic variation, with most over 80%.[121] Estimates of heritability for a number of variables measuring overall crown size of the primary second molars and permanent first molars were moderate to high. Yet, less genetic variation was associated with distances between the cusps on each tooth, implying that phenotypic variation for overall crown size was associated more with genetic variation than was the morphology of the occlusal surfaces.[122] Based on studies of epithelial–mesenchymal interactions during tooth generation, cell proliferation in a specific spatiotemporal pattern along with *sonic hedgehog* (*SHH*) gene expression appears to have a major influence on crown width and cusp number.[123] Thus, *SHH* may be a candidate gene for Class I malocclusion with dental crowding. SNPs in the ectodysplasin-A (*EDA*; OMIM 300451) gene and the gene for its receptor, ectodysplasin A2 receptor (EDA2R/XEDAR) were found to be associated with dental crowding greater than 5 mm in a Hong Kong Chinese Class I malocclusion sample. It was thought that this may at least be due in part to variation in tooth size as the protein product of *EDA* is involved in tooth development, and mutations in *EDA* cause X-linked HED. Although these genes are located on the X chromosome, the associations remained after adjustment for sex.[120]

Dental Agenesis

Dental agenesis may occur within the context of having a family history of dental agenesis (familial) or due to a newly introduced mutation (sporadic), although it is most often familial in origin and usually observed as an "isolated" trait (i.e., nonsyndromic). Dental agenesis, however, may also occur as part of a syndrome, especially in one of the many types of ectodermal dysplasias. Genetic factors are believed to play a major role in most of these cases with autosomal dominant, autosomal recessive, X-linked, and multifactorial inheritance reported;[124] however, epigenetics and environment can also be involved in the etiology.[125]

A general trend in patients with dental agenesis is to have the mesial-distal size crowns of the teeth present to be relatively small (especially if more teeth are missing). The mesial-distal size of the permanent maxillary incisor and canine crowns tends to be large in cases with supernumerary teeth.[126] Relatives who do not have dental agenesis may still manifest teeth that are smaller than normal in size. This suggests a polygenic influence on the size and patterning of the dentition, with a multifactorial threshold for actual hypodontia in some families.

One of the most common patterns of hypodontia (excluding the third molars) involves the maxillary lateral incisors. This can be an autosomal dominant trait with incomplete penetrance and variable expressivity as evidenced by the phenotype sometimes "skipping" generations, sometimes being a peg-shaped lateral instead of agenesis, and sometimes involving one or the other or both sides.[127] A polygenic mode of inheritance also has been proposed.[128] Currently unidentified, the gene mutation that primarily influences this phenotype has been suggested, in the homozygous state, to influence agenesis of the succedaneous teeth or all or almost all of the permanent dentition.[129,130] In addition, an associated increase in the agenesis of premolars occurs,[131] as well as the appearance of PDCs.[132]

Numerous mutations in transcription factor and growth factor–related genes involved in dental development have

been shown to have roles in human dental agenesis, including paired box 9 (*PAX9*; OMIM 167416) and muscle segment homeobox 1(*MSX1*; OMIM 142983). Mutations in *PAX9* typically show a nonsyndromic autosomal dominant mode of inheritance for oligodontia with variable expressivity within families.[133-137] The characteristic pattern of dental agenesis caused by *PAX9* mutations largely affects molars in both dental arches and second premolars most often in the maxilla arch than the mandibular arch,[137,138] occasionally presenting with missing or peg-shaped mandibular central incisors and/or maxillary lateral incisors.[134-139] Agenesis of maxillary first premolars[136,138,140] and/or canines can occur with a low frequency among *PAX9* mutations,[138,140,141] while the *PAX9* Ala240Pro (c. G718C) mutation may be unique in that it leads uniquely to third molar agenesis with or without affected incisors.[142-145] *MSX1* gene mutations can lead to hypodontia or oligodontia,[138,146-155] as well as variations in the downstream signaling gene *BMP4*.[156] Mutations in the Axis inhibitor 2 gene (*AXIN2*; OMIM 604025) have also been linked to oligodontia, often exhibiting a similar pattern of affected teeth as *PAX9* mutations (i.e., molars, premolars, mandibular incisors > maxillary lateral incisors > canines).[138,157] The AXIN2 protein is a regulator of WNT and β-catenin signaling, so it is not surprising that mutations in the wingless-type MMTV integration site family, member 10A (*WNT10A*; OMIM 606268) gene have also been linked to hypodontia and oligodontia.[158-163] Other chromosomal locations and/or candidate genes thought to be involved in nonsyndromic hypodontia and/or oligodontia include,[164-171] EDA receptor–associated death domain gene (*EDARADD*),[138] transforming growth factor–alpha gene (*TNFA*),[172,173] interferon regulatory factor 6 (*IRF6*),[174,175] fibroblast growth factor receptor 1 (*FGFR1*),[174] transforming growth factor-β3 (*TGFβ3*),[156] C2H2 zinc finger transcription factor 22 (*KROX-26/ZNF22*), and 10q11.2 (OMIM 610926).[124,176,177] Many nonsyndromic EDA mutations affect incisor, canine, and premolar development.[164-171] The latent transforming growth factor beta binding protein 3 (*LTBP3*; OMIM 602090) gene, which may also influence traits of short stature and increased bone density, plays a role in autosomal recessive hypodontia.[150,178,179] An increased understanding of the various morphogenetic signaling pathways regulating tooth development should allow for induction of tooth development in areas of tooth agenesis.[180]

With the push for more personalized medicine, we are now learning more about how phenotypes and/or disease in the oral cavity can serve as a diagnostic marker or indicator of susceptibility to other health issues outside of the oral cavity. In addition to the association of dental agenesis with many syndromes, mutations in tooth development genes have been associated with other medical conditions, such as cancer. In 2004, Lammi et al.[157] reported on a Finnish family with multiple members who manifested oligodontia early in life and colon cancer later in life. These two traits segregated in an autosomal dominant pattern of inheritance with a rare nonsense mutation in the *AXIN2* DNA sequence (1966C > T) that leads to an amino acid change (p. Arg656STOP).[157] *AXIN2* mutations may also be involved with dental agenesis in combination with a variety of other phenotypes, including early onset colon and breast cancers, prostate cancer risk, mild ectodermal dysplasia, colonic polyposis, gastric polyps, and gastric cancer.[181-183] While some studies have begun to examine connections between dental tooth agenesis and epithelial ovarian cancer,[184-186] other studies have connected dental agenesis with a self-reported family history of cancer;[187] however, the causal genes are yet to be determined. Although these associations are interesting because of the possibility of the orthodontist or other dental practitioner referring an individual or family for cancer susceptibility testing or clinical screening someday, it must be emphasized that the actual risk of an increased likelihood of developing cancer associated with dental agenesis is unknown and may be very small in the general public. Further investigation is needed into this area, particularly for biomarkers that may help to determine if there is a greater risk and, if so, who has that risk.

DENTAL ERUPTION PROBLEMS

Canine Impaction and/or Displacement

Maxillary canine impaction or displacement is labial/buccal to the arch in 15% of the cases of maxillary canine impaction and often is associated with dental crowding. The canine impacted or displaced palatally occurs in 85% of the cases and typically is not associated with dental crowding.[188] PDCs frequently, but not always, are found in dentitions with various anomalies. These include small, peg-shaped, or missing maxillary lateral incisors, hypodontia involving other teeth, dentition spacing, and dentitions with delayed development.[189] Because of varying degrees of genetic influence on these anomalies, there has been some discussion about PDCs themselves also being influenced by genetic factors to some degree. In addition, the occurrence of PDCs does occur in a higher percentage within families than in the general population.[190]

A greater likelihood exists of a PDC on the same side of a missing or small maxillary lateral incisor, emphasizing a local environmental effect.[191] Also, in some cases, a canine is displaced palatally without an apparent anomaly of the maxillary lateral incisors; in some other cases, lateral incisors are missing without palatal displacement of a canine. Adding to the complexity is the heterogeneity found in studies of cases of buccally displaced canines[192] and PDCs.[189] Although the canine eruption theory of guidance by the lateral incisor root cannot explain all instances of PDCs, it does seem to play some role in some cases.[193]

With apparent genetic and environmental factors playing some variable role in these cases, the cause appears to be multifactorial.[194] The phenotype is the result of some genetic influences (directly, indirectly, or both, for example, although a primary effect on development of some or all of the rest of the dentition) interacting with environmental factors. Some of these cases may be examples of how primary genetic influences (which still interact with other genes and environmental factors) affect a phenotypic expression that is a variation in a local environment, such as the physical structure of the lateral incisor in relation to the developing canine. Candidate genes that are proposed possibly to influence the occurrence of PDCs and hypodontia in developmental fields include *MSX1* and *PAX9*.[195]

Primary Failure of Eruption

In addition to hypodontia and its primary or secondary relationship to maxillary canine eruption, there are emerging data regarding the influence of genetics on dental eruption. Presently, this is clearest in cases of PFE, in which all teeth distal to the most mesially involved tooth do not erupt or respond to

orthodontic force. The familial occurrence of this phenomenon in approximately one-quarter of cases facilitated the investigation and discovery that the parathyroid hormone 1 receptor (*PTHR1*) gene is involved.[196,197] Advancements in this area could not only help to define patients who are likely to develop or have PFE but also potentially result in the molecular manipulation of selective tooth eruption rates to enhance treatment protocols on an individual basis.[198]

ENVIRONMENTAL AND GENETIC INFLUENCES ON BILATERAL SYMMETRY

Unlike structures that have directional asymmetry when development of one side is different from that of the other during normal development, facial and dental structures lateral to the midline are essentially mirror images of each other, with the same genetic influences affecting both sides. The conditions are theoretically identical for the trait on both sides of the body because they are developing simultaneously and therefore should develop identically.

There is not one group of genes for the permanent maxillary right first molar and another group of genes for the permanent maxillary left first molar. Fluctuating asymmetry occurs randomly when a difference exists between right and left sides. This reflects the inability of the individual to develop identical, bilaterally homologous structures.[61]

Fluctuating asymmetry has been observed in the primary and permanent dentitions,[199,200] as well as in the craniofacies.[61] The greater amount of fluctuating asymmetry for the distance between cusps on each tooth than for the overall crown size of primary second molars and permanent first molars indicates that the occlusal morphology of these teeth is influenced more by environmental factors than the overall crown size.[122] In addition, an association between decreased developmental stability (evident in increased fluctuating asymmetry), arch form discrepancies, and anterior maxillary dental crowding has been reported,[201] suggesting that a variable component of occlusal variation may be the individual's relative ability to develop right and left mirror images. This clinically could be an indication for selective interproximal reduction in specific areas to maximize intercuspation.

GENETIC FACTORS AND EXTERNAL APICAL ROOT RESORPTION

Analysis of the genetic basis for variable response to treatment has been applied to the specific adverse outcome sometimes associated with orthodontic treatment called EARR. The degree and severity of EARR associated with orthodontic treatment are complex, involving host and environmental factors. An association of EARR exists in those who have not received orthodontic treatment, with missing teeth, increased periodontal probing depths, and reduced crestal bone heights.[202] Individuals with bruxism, chronic nail biting, and anterior open bites with concomitant tongue thrust also may show an increased extent of EARR before orthodontic treatment.[203]

EARR is also increased as a pathologic consequence of orthodontic mechanical loading in some patients.[204,205] The amount of orthodontic movement is positively associated with the resulting extent of EARR.[206-208] Orthodontic tooth movement, or "biomechanics," has been found to account for approximately one-tenth to one-third of the total variation in EARR.[209-211] Owman-Moll and coworkers[212] showed that individual variation overshadowed the force magnitude and the force type in defining the susceptibility to histologic root resorption associated with orthodontic force. Individual variations were considerable regarding both extension and depth of histologic root resorption within individuals, and these were not correlated to the magnitude of tooth movement achieved.[213]

There is considerable individual variation in EARR associated with orthodontic treatment, indicating an individual predisposition and multifactorial (complex) etiology.[214-219] Heritability estimates have shown that approximately half of EARR variation concurrent with orthodontia, and almost two-thirds of maxillary central incisor EARR specifically, can be attributed to genetic variation.[219,220] A retrospective twin study on EARR found evidence for both genetic and environmental factors influencing EARR.[221] In addition, studies in a panel of different inbred mice supported a genetic component involving multiple genes in histologic root resorption.[222,223]

While there is a relationship between orthodontic force and root resorption, it is against the backdrop of other treatment factors and individual susceptibility. Because mechanical forces do not adequately explain the variation seen among individual expressions of EARR, interest has increased on genetic and other factors influencing the susceptibility to EARR. The reaction to orthodontic force, including rate of tooth movement, can differ depending on the individual's genetic background.[219,220,224,225]

Since the initial investigation into genetic factors associated with EARR concurrent with orthodontics were published in 2003, there have been a number of candidate gene studies in various populations (Fig. 2-13).[226-236] Even though heritability studies, with all of their caveats, indicated the total genetic influence on EARR concurrent with orthodontics was associated with most of the variation in the phenotype, the separate contributions of the genes investigated appear to be relatively small and inconsistent, as would be expected with a complex trait.

More recently studies are including multiple treatment and genetic factors in models to explain the occurrence of EARR concurrent with orthodontics. For example, 30% of the EARR variability in one study were explained by variation in or the presence of treatment duration, a Hyrax appliance, premolar extractions, sex, and the *P2RX7* gene rs1718119 SNP, while age, overjet, tongue thrust, skeletal Class II, and other genetic polymorphisms made minor contributions.[237] Likewise in another study looking at the relative influence of multiple parameters on the occurrence of EARR including treatment duration, extraction of maxillary premolars, and numerous cephalometric measurements (pretreatment values and post-treatment change in values), as well as genotypes for multiple DNA polymorphisms, found that a longer length of treatment and specific genotypes for *P2RX7* SNP rs208294 together explained 25% of the total variation associated with EARR concurrent with orthodontics in the sample tested.[236] These studies are interesting in that they (1) emphasize the possible effect of longer treatment times on EARR concurrent with orthodontics and (2) support the involvement of a biological pathway since the P2RX7 protein is an upstream regulator of the activation of IL1B that was a focus of initial

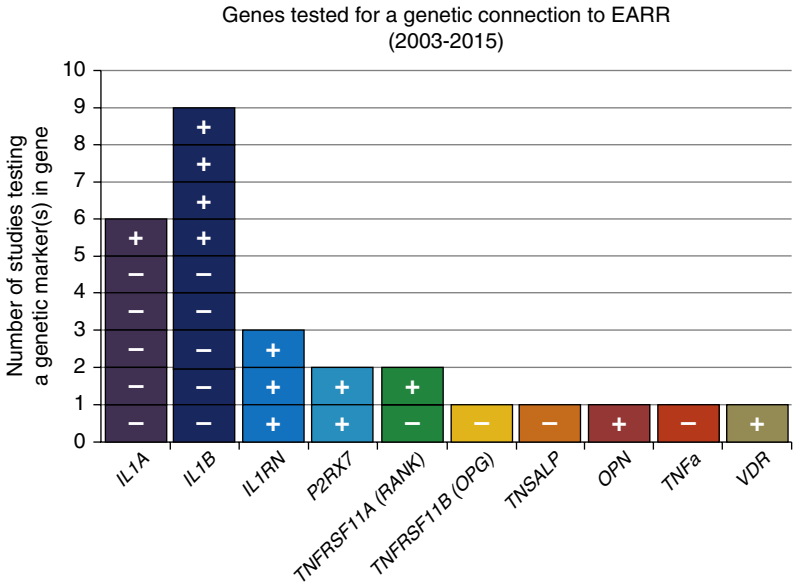


FIGURE 2-13 Several genetic variants have been investigated in association with the presence of external apical root resorption (EARR) concurrent with orthodontia, in a number of populations and using various methods of EARR assessment. Each plus sign in a box above a gene indicates that at least one genetic marker in that gene was found to be significantly associated with EARR in a published study, while a minus sign indicates that marker(s) in that gene were not significantly associated with EARR. Markers in some of the genes have been evaluated in more than one study. The references and discussion are in the text.

studies,[238] and an inbred mouse model with the mouse version of the *P2RX7* gene knocked out showed an increase in histological root resorption with orthodontic force,[239] as did a previous inbred mouse model with the mouse version of the *IL1B* gene knocked out.[240]

While these more recent studies are helping us to investigate the complexity of EARR occurring with orthodontics, they are insufficient for clinically useful prediction. The use of a nonbiased whole exome or whole genome sequencing approach when possible instead of the current candidate gene models could aid in identifying additional genetic factors that may be involved in orthodontic patient EARR. Even if all the factors were known, their number and complexity may only yield a general high, medium, or low risk of EARR concurrent with orthodontics.

PERSONALIZED-PRECISION ORTHODONTICS

The President's Council of Advisors on Science and Technology noted that personalized medicine "refers to the tailoring of medical treatment to the individual characteristics of each patient. It does not literally mean the creation of drugs or medical devices that are unique to a patient but rather the ability to classify individuals into subpopulations that differ in their susceptibility to a particular disease or their response to a specific treatment. Preventive or therapeutic interventions can then be concentrated on those who will benefit, sparing expense and side effects for those who will not."[241] Personalized medicine has garnered a tremendous amount of attention and yet has not even approached its potential in the provision of healthcare. Thus, it remains to be seen how much this will really affect daily medical practice. The same may be projected for the future of orthodontics. What would personalized orthodontics be based upon, and how would the studies be undertaken and then validated in practice? How will this be funded? The understanding of the combination and interaction of genetic and environmental (including treatment) factors (nature and nurture together) that influence the treatment response of our patients is fundamental to the evidence-based practice of orthodontics.[17]

SUMMARY

Multiple factors and processes contribute to the response to orthodontic treatment. Some patients will exhibit unusual outcomes linked to or associated with polymorphic genes. Analysis of overall treatment response requires a systems analysis using informatics for integration of all relevant information. The influence of genetic factors on treatment outcome must be studied and understood in quantitative terms. Conclusions from retrospective studies must be evaluated by prospective testing to truly evaluate their value in practice. Next Generation Sequencing and other types of genetic studies, and their careful assessment in clinical trials for their relevance to diagnosis and treatment outcomes, are necessary to further the evidence base for the practice of orthodontics. Only then will we begin to truly understand how nature (genetic factors) and nurture (environmental factors, including treatment) together affect our treatment of our patients in a truly evidence-based manner.[17]

REFERENCES

1. Manfredi C, Martina R, Grossi GB, Giuliani M. Heritability of 39 orthodontic cephalometric parameters on MZ, DZ twins and MN-paired singletons. *Am J Orthod Dentofac Orthop*. 1997;111:44–51.
2. Mossey PA. The heritability of malocclusion: Part 1: genetics, principles and terminology. *Br J Orthod*. 1999;26:103–113.
3. Vanco C, Kasai K, Sergi R, Richards LC, Townsend GC. Genetic and environmental influences on facial profile. *Aust Dent J*. 1995;40: 104–109.
4. Hartsfield Jr JK. Personalized orthodontics, the future of genetics in practice. *Semin Orthod*. 2008;14:166–171.
5. Harris EF. Interpreting heritability estimates in the orthodontic literature. *Semin Orthod*. 2008;14:125–134.
6. Proffit WR. On the aetiology of malocclusion. The Northcroft lecture, 1985 presented to the British Society for the Study of Orthodontics, Oxford, April 18, 1985. *Br J Orthod*. 1986;13:1–11.
7. Harris JE. Genetic factors in the growth of the head. Inheritance of the craniofacial complex and malocclusion. *Dent Clin North Am*. 1975;19:151–160.
8. Moss ML. The functional matrix hypothesis revisited. 4. The epigenetic antithesis and the resolving synthesis. *Am J Orthod Dentofac Orthop*. 1997;112:410–417.
9. Griffiths AJF, Gelbart WM, Miller JH. *Modern Genetic Analysis*. 7th ed. New York: W. H. Freeman; 1999.
10. Harris EF, Potter RH. Sources of bias in heritability studies. *Am J Orthod Dentofac Orthop*. 1997;112:17A–21A.
11. Vogel F, Motulsky AG. *Human Genetics: Problems and Approaches*. 2nd ed. New York: Springer-Verlag; 1986.
12. Buschang PH, Hinton RJ. A gradient of potential for modifying craniofacial growth. *Semin Orthod*. 2005;11:219–226.
13. Human Genome Program, U.S. Department of Energy, Genomics and Its Impact on Science and Society: A 2008 Primer. http://web.ornl.gov/sci/techresources/Human_Genome/publicat/primer2001/primer11.pdf.
14. Golbabapour S, Abdulla MA, Hajrezaei M. A concise review on epigenetic regulation: insight into molecular mechanisms. *Int J Mol Sci*. 2011;12: 8661–8694.
15. Everett ET, Hartsfield JKJ. *Mouse Models for Craniofacial Anomalies. Biological Mechanisms of Tooth Movement and Craniofacial Adaption Boston*. Boston: Harvard Society for the Advancement of Orthodontics; 2000.
16. National Human Genome Research Institute. *Human Genome Project Goals. 2003*; 2015.
17. Hartsfield Jr JK. Personalized orthodontics: limitations and possibilities in orthodontic practice. In: Krishnan V, Davidovitch Z, eds. *Biological Mechanisms of Tooth Movement*. 2nd ed. Oxford, UK: Wiley-Blackwell Publishing; 2015:164–172.
18. Baltimore D. Our genome unveiled. *Nature*. 2001;409:814–816.
19. Lidral AC, Moreno LM, Bullard SA. Genetic factors and orofacial clefting. *Semin Orthod*. 2008;14:103–114.
20. Mossey PA. The heritability of malocclusion: part 2. The influence of genetics in malocclusion. *Br J Orthod*. 1999;26:195–203.
21. Abass SK, Hartsfield JKJ. Investigation of genetic factors affecting complex traits using external apical root resorption as a model. *Semin Orthod*. 2008;14:115–124.
22. Hartsfield Jr JK, Bixler D. Clinical genetics for the dental practitioner. In: Dean JA, Avery DR, McDonald RE, eds. *McDonald's and Avery's Dentistry for the Child and Adolescent*. 9th ed. St. Louis, MO: Mosby/Elsevier; 2011:64–84.
23. Flanagan N, Healy E, Ray A, et al. Pleiotropic effects of the melanocortin 1 receptor (*MC1R*) gene on human pigmentation. *Hum Mol Genet*. 2000;9: 2531–2537.
24. Sturm RA, Duffy DL, Box NF, et al. Genetic association and cellular function of *MC1R* variant alleles in human pigmentation. *Ann NY Acad Sci*. 2003;994:348–358.
25. Rutledge MS, Hartsfield Jr JK. Genetic factors in the etiology of palatally displaced canines. *Semin Orthod*. 2010;16:165–171.
26. Nanni L, Ming JE, Du Y, et al. SHH mutation is associated with solitary median maxillary central incisor: a study of 13 patients and review of the literature. *Am J Med Genet*. 2001;102:1–10.
27. Hartsfield Jr JK. The benefits of obtaining the opinion of a clinical geneticist regarding orthodontic patients. In: Krishnan V, Davidovitch ZE, eds. *Integrated Clinical Orthodontics*. Oxford: John Wiley & Sons; 2012:109–131.
28. Taylor TH, Gitlin SA, Patrick JL, Crain JL, Wilson JM, Griffin DK. The origin, mechanisms, incidence and clinical consequences of chromosomal mosaicism in humans. *Hum Reprod Update*. 2014;20:571–581.
29. Cruz RM, Krieger H, Ferreira R, Mah J, Hartsfield Jr J, Oliveira S. Major gene and multifactorial inheritance of mandibular prognathism. *Am J Med Genet A*. 2008;146A:71–77.
30. McDonald RE, Avery DR, Hartsfield Jr JK. Acquired and developmental disturbances of the teeth and associated oral structures. In: Dean JA, Avery DR, McDonald RE, eds. *McDonald's and Avery's Dentistry for the Child and Adolescent*. 9th ed. St. Louis, MO: Mosby/Elsevier; 2011: 85–125.
31. Hartsfield Jr JK, Hohlt WF, Roberts WE. Orthodontic treatment and orthognathic surgery for patients with osteogenesis imperfecta. *Semin Orthod*. 2006;12:254–271.
32. Retrouvey J-M, Schwarts S, Hartsfield JK. Oral-facial aspects of osteogenesis imperfecta. In: Shapiro JR, Byers PH, Glorieux FH, Sponseller PD, eds. *Osteogenesis Imperfecta: A Translational Approach to Brittle Bone Disease*. London: Academic Press; 2014:313–327.
33. Escobar V, Bixler D. On the classification of the acrocephalosyndactyly syndromes. *Clin Genet*. 1977;12:169–178.
34. Everett ET, Britto DA, Ward RE, Hartsfield Jr JK. A novel *FGFR2* gene mutation in Crouzon syndrome associated with apparent nonpenetrance. *Cleft Palate Craniofac J*. 1999;36:533–541.
35. Mulvihill JJ. Craniofacial syndromes: no such thing as a single gene disease. *Nat Genet*. 1995;9:101–103.
36. Niswander JD. Genetics of common dental disorders. *Dent Clin North Am*. 1975;19:197–206.
37. Williams SD, Hughes TE, Adler CJ, Brook AH, Townsend GC. Epigenetics: a new frontier in dentistry. *Aust Dent J*. 2014;59(suppl 1):23–33.
38. Beecher RM, Corruccini RS, Freeman M. Craniofacial correlates of dietary consistency in a nonhuman primate. *J Craniofac Genet Dev Biol*. 1983;3:193–202.
39. Corruccini RS, Potter RH. Genetic analysis of occlusal variation in twins. *Am J Orthod*. 1980;78:140–154.
40. Garn SM, Cole PE, Bailey SM. Living together as a factor in family-line resemblances. *Hum Biol*. 1979;51:565–587.
41. Stockard CR, Anderson OD. *Genetic and Endocrine Basis for Differences in Form and Behaviour*. Philadelphia: Wistar Institute of Anatomy and Biology; 1941.
42. Chung CS, Niswander JD, Runck DW, Bilben SE, Kau MC. Genetic and epidemiologic studies of oral characteristics in Hawaii's schoolchildren. II. Malocclusion. *Am J Hum Genet*. 1971;23:471–495.
43. Corruccini RS. An epidemiologic transition in dental occlusion in world populations. *Am J Orthod*. 1984;86:419–426.
44. Huh A, Horton MJ, Cuenco KT, et al. Epigenetic influence of *KAT6B* and *HDAC4* in the development of skeletal malocclusion. *Am J Orthod Dentofac Orthop*. 2013;144:568–576.
45. Raoul G, Rowlerson A, Sciote J, et al. Masseter myosin heavy chain composition varies with mandibular asymmetry. *J Craniofac Surg*. 2011;22:1093–1098.
46. Rowlerson A, Raoul G, Daniel Y, et al. Fiber-type differences in masseter muscle associated with different facial morphologies. *Am J Orthod Dentofac Orthop*. 2005;127:37–46.
47. Sciote JJ, Raoul G, Ferri J, Close J, Horton MJ, Rowlerson A. Masseter function and skeletal malocclusion. *Rev Stomatol Chir Maxillofac Chir Orale*. 2013;114:79–85.
48. North KN, Beggs AH. Deficiency of a skeletal muscle isoform of alpha-actinin (alpha-actinin-3) in merosin-positive congenital muscular dystrophy. *Neuromuscul Disord*. 1996;6:229–235.
49. Zebrick B, Teeramongkolgul T, Nicot R, et al. *ACTN3* R577X genotypes associate with Class II and deepbite malocclusions. *Am J Orthod Dentofac Orthop*. 2014;146:603–611.
50. Sciote JJ, Horton MJ, Rowlerson AM, Ferri J, Close JM, Raoul G. Human masseter muscle fiber type properties, skeletal malocclusions, and muscle growth factor expression. *J Oral Maxillofac Surg*. 2012;70:440–448.

51. Desh H, Gray SL, Horton MJ, et al. Molecular motor *MYO1C*, acetyltransferase *KAT6B* and osteogenetic transcription factor *RUNX2* expression in human masseter muscle contributes to development of malocclusion. *Arch Oral Biol*. 2014;59:601–607.
52. Tassopoulou-Fishell M, Deeley K, Harvey EM, Sciote J, Vieira AR. Genetic variation in myosin 1H contributes to mandibular prognathism. *Am J Orthod Dentofac Orthop*. 2012;141:51–59.
53. King L, Harris EF, Tolley EA. Heritability of cephalometric and occlusal variables as assessed from siblings with overt malocclusions. *Am J Orthod Dentofac Orthop*. 1993;104:121–131.
54. LaBuda MC, Gottesman II, Pauls DL. Usefulness of twin studies for exploring the etiology of childhood and adolescent psychiatric disorders. *Am J Med Genet*. 1993;48:47–59.
55. Strachan T, Read AP. *Complex Diseases: Theories and Results*. 2nd ed. Oxford: BIOS Scientific; 1999.
56. Harris EF, Johnson MG. Heritability of craniometric and occlusal variables: a longitudinal sib analysis. *Am J Orthod Dentofac Orthop*. 1991;99:258–268.
57. Lundstrom A, McWilliam J. Comparison of some cephalometric distances and corresponding facial proportions with regard to heritability. *Eur J Orthod*. 1988;10:27–29.
58. Arya BS, Savara BS, Clarkson QD, Thomas DR. Genetic variability of craniofacial dimensions. *Angle Orthod*. 1973;43:207–215.
59. Boraas JC, Messer LB, Till MJ. A genetic contribution to dental caries, occlusion, and morphology as demonstrated by twins reared apart. *J Dent Res*. 1988;67:1150–1155.
60. Byard PJ, Poosha DV, Satyanarayana M, Rao DC. Family resemblance for components of craniofacial size and shape. *J Craniofac Genet Dev Biol*. 1985;5:229–238.
61. Cassidy KM, Harris EF, Tolley EA, Keim RG. Genetic influence on dental arch form in orthodontic patients. *Angle Orthod*. 1998;68:445–454.
62. Chung CS, Niswander JD. Genetic and epidemiologic studies of oral characteristics in Hawaii's schoolchildren: V. Sibling correlations in occlusion traits. *J Dent Res*. 1975;54:324–329.
63. Corruccini RS, Sharma K, Potter RH. Comparative genetic variance and heritability of dental occlusal variables in U.S. and Northwest Indian twins. *Am J Phys Anthropol*. 1986;70:293–299.
64. Devor EJ. Transmission of human craniofacial dimensions. *J Craniofac Genet Dev Biol*. 1987;7:95–106.
65. Gass JR, Valiathan M, Tiwari HK, Hans MG, Elston RC. Familial correlations and heritability of maxillary midline diastema. *Am J Orthod Dentofac Orthop*. 2003;123:35–39.
66. Harris EF, Smith RJ. A study of occlusion and arch widths in families. *Am J Orthod*. 1980;78:155–163.
67. Harris JE, Kowalski CJ, Watnick SS. Genetic factors in the shape of the craniofacial complex. *Angle Orthod*. 1973;43:107–111.
68. Harris JE, Kowalski CJ, Walker SJ. Intrafamilial dentofacial associations for Class II, Division 1 probands. *Am J Orthod*. 1975;67:563–570.
69. Hauspie RC, Susanne C, Defrise-Gussenhoven E. Testing for the presence of genetic variance in factors of face measurements of Belgian twins. *Ann Hum Biol*. 1985;12:429–440.
70. Horowitz SL, Osborne RH, De George FV. A cephalometric study of craniofacial variation in adult twins. *Angle Orthod*. 1960;30:1.
71. Kraus BS, Wise WJ, Frei RH. Heredity and the craniofacial complex. *Am J Orthod*. 1959;45:172.
72. Lobb WK. Craniofacial morphology and occlusal variation in monozygous and dizygous twins. *Angle Orthod*. 1987;57:219–233.
73. Nakata M, Yu PL, Nance WE. Multivariate analysis of craniofacial measurements in twin and family data. *Am J Phys Anthropol*. 1974;41:423–429.
74. Saunders SR, Popovich F, Thompson GW. A family study of craniofacial dimensions in the Burlington Growth Centre sample. *Am J Orthod*. 1980;78:394–403.
75. Susanne C, Sharma PD. Multivariate analysis of head measurements in Punjabi families. *Ann Hum Biol*. 1978;5:179–183.
76. Watnick SS. Inheritance of craniofacial morphology. *Angle Orthod*. 1972;42:339–351.
77. Miller S, Shaffer J, Marazita M, et al. Genome wide association study of dental arch form and occlusal relationships in the mixed dentition stage. *FASEB J*. 2015;29. abstract 697.4.
78. Hartsfield JKJ. Development of the vertical dimension: nature and nurture. *Semin Orthod*. 2002;8:113.
79. Litton SF, Ackermann LV, Isaacson RJ, Shapiro BL. A genetic study of Class 3 malocclusion. *Am J Orthod*. 1970;58:565–577.
80. Harris JE, Kowalski CJ. All in the family: use of familial information in orthodontic diagnosis, case assessment, and treatment planning. *Am J Orthod*. 1976;69:493–510.
81. Hunter WS. *Heredity in the Craniofacial Complex*. 3rd ed. Philadelphia: Saunders; 1990.
82. Verdonck A, Gaethofs M, Carels C, de Zegher F. Effect of low-dose testosterone treatment on craniofacial growth in boys with delayed puberty. *Eur J Orthod*. 1999;21:137–143.
83. Moss ML. *The Regulation of Skeletal Growth Regulation of Organ and Tissue Growth*. New York: Academic Press; 1972:127–142.
84. Guo Y, Xiong D-H, Yang T-L, Guo Y-F, Recker RR, Deng H-W. Polymorphisms of estrogen-biosynthesis genes *CYP17* and *CYP19* may influence age at menarche: a genetic association study in Caucasian females. *Hum Mol Genet*. 2006;15:2401–2408.
85. Schaefer K, Fink B, Mitteroecker P, Neave N, Bookstein FL. Visualizing facial shape regression upon 2nd to 4th digit ratio and testosterone. *Coll Antropol*. 2005;29:415–419.
86. Schaefer K, Fink B, Grammer K, Mitteroecker P, Gunz P, Bookstein FL. Female appearance: facial and bodily attractiveness as shape. *Psychol Sci*. 2006;48:187–204.
87. Hartsfield Jr JK, Zhou J, Chen S. The importance of analyzing specific genetic factors in facial growth for diagnosis and treatment planning. In: McNamara Jr JA, Kapila SD, eds. *Surgical Enhancement of Orthodontic Treatment*. Ann Arbor: University of Michigan; 2010:267–281 (Monograph 47, Craniofacial Growth Series, Department of Orthodontics and Pediatric Dentistry and Center for Human Growth and Development).
88. He S, Hartsfield Jr JK, Guo Y, Cao Y, Wang S, Chen S. Association between *CYP19A1* genotype and pubertal sagittal jaw growth. *Am J Orthod Dentofac Orthop*. 2012;142:662–670.
89. Wolff G, Wienker TF, Sander H. On the genetics of mandibular prognathism: analysis of large European noble families. *J Med Genet*. 1993;30:112–116.
90. El-Gheriani AA, Maher BS, El-Gheriani AS, et al. Segregation analysis of mandibular prognathism in Libya. *J Dent Res*. 2003;82:523–527.
91. Thompson EM, Winter RM. Another family with the "Habsburg jaw". *J Med Genet*. 1988;25:838–842.
92. Bui C, King T, Proffit W, Frazier-Bowers S. Phenotypic characterization of Class III patients. *Angle Orthod*. 2006;76:564–569.
93. Singh GD. Morphologic determinants in the etiology of class III malocclusions: a review. *Clin Anat*. 1999;12:382–405.
94. King RA, Rotter JI, Motulsky AG. *Approach to Genetic Basis of Common Diseases*. 2nd ed. Oxford: Oxford University Press; 2002.
95. Otero LM, Morford LA, Falcão-Alencar G, Hartsfield Jr JK. Family history and genetics of mandibular prognathism. In: Ngan PW, Deguchi T, Roberts EW, eds. *Orthodontic Treatment of Class III Malocclusion*. Bentham; 2014:3–24.
96. Moreno Uribe LM, Howe SC, Kummet C, Vela KC, Dawson DV, Southard TE. Phenotypic diversity in white adults with moderate to severe Class II malocclusion. *Am J Orthod Dentofac Orthop*. 2014;145:305–316.
97. Moyers RE, Riolo ML, Guire KE, Wainright RL, Bookstein FL. Differential diagnosis of class II malocclusions. Part 1. Facial types associated with class II malocclusions. *Am J Orthod*. 1980;78:477–494.
98. Hartsfield JK, Morford LA, Otero LM, Fardo DW. Genetics and nonsyndromic facial growth. *J Ped Genet*. 2013;2:1–12.
99. Yamaguchi T, Park SB, Narita A, Maki K, Inoue I. Genome-wide linkage analysis of mandibular prognathism in Korean and Japanese patients. *J Dent Res*. 2005;84:255–259.
100. Jang JY, Park EK, Ryoo HM, et al. Polymorphisms in the *Matrilin-1* gene and risk of mandibular prognathism in Koreans. *J Dent Res*. 2010;89:1203–1207.
101. Xue F, Wong RW, Rabie AB. Genes, genetics, and Class III malocclusion. *Orthod Craniofac Res*. 2010;13:69–74.
102. Li Q, Li X, Zhang F, Chen F. The identification of a novel locus for mandibular prognathism in the Han Chinese population. *J Dent Res*. 2011;90:53–57.

103. Li Q, Zhang F, Li X, Chen F. Genome scan for locus involved in mandibular prognathism in pedigrees from China. *PLoS One*. 2010;5.
104. Xue F, Rabie AB, Luo G. Analysis of the association of *COL2A1* and *IGF-1* with mandibular prognathism in a Chinese population. *Orthod Craniofac Res*. 2014;17:144–149.
105. Ikuno K, Kajii TS, Oka A, Inoko H, Ishikawa H, Iida J. Microsatellite genome-wide association study for mandibular prognathism. *Am J Orthod Dentofac Orthop*. 2014;145:757–762.
106. Frazier-Bowers S, Rincon-Rodriguez R, Zhou J, Alexander K, Lange E. Evidence of linkage in a Hispanic cohort with a Class III dentofacial phenotype. *J Dent Res*. 2009;88:56–60.
107. Falcão-Alencar G, Otero L, Cruz R, et al. Evidence for genetic linkage of the class III craniofacial phenotype with human chromosome 7 in 36 South American families. Paper Presented at: 60th Annual Meeting of the American Society of Human Genetics. Washington, DC; 2010.
108. Nikopensius T, Saag M, Jagomägi T, et al. Missense mutation in *DUSP6* is associated with class III malocclusion. *J Dent Res*. 2013;92:893–898.
109. Perillo L, Monsurrò A, Bonci E, Torella A, Mutarelli M, Nigro V. Genetic association of *ARHGAP21* gene variant with mandibular prognathism. *J Dent Res*. 2015;94:569–76.
110. Chen F, Li Q, Gu M, Li X, Yu J, Zhang YB. Identification of a Mutation in FGF23 Involved in Mandibular Prognathism. *Sci Rep*. 2015;5:11250. doi: 10.1038/srep11250.
110a. Ast DB, Carlos JP, Cons NC. The prevalence and characteristics of malocclusion among senior high school students in upstate New York. *Am J Orthod*. 1965;51:437–445.
111. Guan X, Song Y, Ott J, Zhang Y, Li C, Xin T, Li Z, Gan Y, Li J, Zhou S, Zhou Y. The ADAMTS1 Gene Is Associated with Familial Mandibular Prognathism. *J Dent Res*. 2015;94:1196–201.
111a. Mills LF. Epidemiologic studies of occlusion. IV. The prevalence of malocclusion in a population of 1,455 school children. *J Dent Res*. 1966;45:332–336.
112. Markovic MD. At the crossroads of oral facial genetics. *Eur J Orthod*. 1992;14:469–481.
113. Morrison MW. *Relative Risk of Class II Division 2 Malocclusion in First-Degree Relatives of Probands with Class II Division 2 Malocclusion*. Department of Orthodontics and Oral Facial Genetics; Master of Science in Dentistry; 2008.
114. Basdra EK, Kiokpasoglou M, Stellzig A. The Class II Division 2 craniofacial type is associated with numerous congenital tooth anomalies. *Eur J Orthod*. 2000;22:529–535.
115. Basdra EK, Kiokpasoglou MN, Komposch G. Congenital tooth anomalies and malocclusions: a genetic link? *Eur J Orthod*. 2001;23:145–151.
116. Peck S, Peck L, Kataja M. Class II Division 2 malocclusion: a heritable pattern of small teeth in well-developed jaws. *Angle Orthod*. 1998;68:9–20.
117. Bernabé E, Flores-Mir C. Dental morphology and crowding: a multivariate approach. *Angle Orthod*. 2006;76:20–25.
118. Hashim HA, Al-Ghamdi S. Tooth width and arch dimensions in normal and malocclusion samples: an odontometric study. *J Contemp Dent Pract*. 2005;6:36–51.
119. Poosti M, Jalali T. Tooth size and arch dimension in uncrowded versus crowded Class I malocclusions. *J Contemp Dent Pract*. 2007;8:45–52.
120. Ting TY, Wong RWK, Rabie ABM. Analysis of genetic polymorphisms in skeletal Class I crowding. *Am J Orthod Dentofac Orthop*. 2011;140:e9–e15.
121. Dempsey PJ, Townsend GC. Genetic and environmental contributions to variation in human tooth size. *Heredity*. 2001;86:685–693.
122. Townsend G, Richards L, Hughes T. Molar intercuspal dimensions: genetic input to phenotypic variation. *J Dent Res*. 2003;82:350–355.
123. Ishida K, Murofushi M, Nakao K, Morita R, Ogawa M, Tsuji T. The regulation of tooth morphogenesis is associated with epithelial cell proliferation and the expression of Sonic hedgehog through epithelial-mesenchymal interactions. *Biochem Biophys Res Commun*. 2011;405:455–461.
124. Mostowska A, Kobielak A, Trzeciak WH. Molecular basis of non-syndromic tooth agenesis: mutations of *MSX1* and *PAX9* reflect their role in patterning human dentition. *Eur J Oral Sci*. 2003;111:365–370.
125. Brook AH. Multilevel complex interactions between genetic, epigenetic and environmental factors in the aetiology of anomalies of dental development. *Arch Oral Biol*. 2009;54:s3–s17.
126. Brook AH, Elcock C, al-Sharood MH, McKeown HF, Khalaf K, Smith RN. Further studies of a model for the etiology of anomalies of tooth number and size in humans. *Connect Tissue Res*. 2002;43:289–295.
127. Woolf CM. Missing maxillary lateral incisors: a genetic study. *Am J Hum Genet*. 1971;23:289–296.
128. Peck L, Peck S, Attia Y. Maxillary canine-first premolar transposition, associated dental anomalies and genetic basis. *Angle Orthod*. 1993;63:99–109, discussion 110.
129. Hoo JJ. Anodontia of permanent teeth (OMIM # 206780) and pegged/missing maxillary lateral incisors (OMIM # 150400) in the same family. *Am J Med Genet*. 2000;90:326–327.
130. Witkop Jr CJ. Agenesis of succedaneous teeth: an expression of the homozygous state of the gene for the pegged or missing maxillary lateral incisor trait. *Am J Med Genet*. 1987;26:431–436.
131. Nieminen P, Arte S, Pirinen S, Peltonen L, Thesleff I. Gene defect in hypodontia: exclusion of *MSX1* and *MSX2* as candidate genes. *Hum Genet*. 1995;96:305–308.
132. Zilberman Y, Cohen B, Becker A. Familial trends in palatal canines, anomalous lateral incisors, and related phenomena. *Eur J Orthod*. 1990;12:135–139.
133. Kapadia H, Frazier-Bowers S, Ogawa T, D'Souza RN. Molecular characterization of a novel *PAX9* missense mutation causing posterior tooth agenesis. *Eur J Hum Genet*. 2006;14:403–409.
134. Frazier-Bowers SA, Guo DC, Cavender A, et al. A novel mutation in human *PAX9* causes molar oligodontia. *J Dent Res*. 2002;81:129–133.
135. Suda N, Ogawa T, Kojima T, Saito C, Moriyama K. Non-syndromic oligodontia with a novel mutation of *PAX9*. *J Dent Res*. 2011;90:382–386.
136. Klein ML, Nieminen P, Lammi L, Niebuhr E, Kreiborg S. Novel mutation of the initiation codon of *PAX9* causes oligodontia. *J Dent Res*. 2005;84:43–47.
137. Nieminen P, Arte S, Tanner D, et al. Identification of a nonsense mutation in the *PAX9* gene in molar oligodontia. *Eur J Hum Genet*. 2001;9:743–746.
138. Bergendal B, Klar J, Stecksen-Blicks C, Norderyd J, Dahl N. Isolated oligodontia associated with mutations in *EDARADD, AXIN2, MSX1*, and *PAX9* genes. *Am J Med Genet A*. 2011;155A:1616–1622.
139. Mitsui SN, Yasue A, Masuda K, et al. Novel *PAX9* mutations cause non-syndromic tooth agenesis. *J Dent Res*. 2014;93:245–249.
140. Zhao J, Hu Q, Chen Y, Luo S, Bao L, Xu Y. A novel missense mutation in the paired domain of human *PAX9* causes oligodontia. *Am J Med Genet A*. 2007;143A:2592–2597.
141. Liang J, Song G, Li Q, Bian Z. Novel missense mutations in *PAX9* causing oligodontia. *Arch Oral Biol*. 2012;57:784–789.
142. Wang J, Jian F, Chen J, et al. Sequence analysis of *PAX9, MSX1* and *AXIN2* genes in a Chinese oligodontia family. *Arch Oral Biol*. 2011;56:1027–1034.
143. Paixao-Cortes VR, Braga T, Salzano FM, Mundstock K, Mundstock CA, Bortolini MC. *PAX9* and *MSX1* transcription factor genes in non-syndromic dental agenesis. *Arch Oral Biol*. 2011;56:337–344.
144. Kula K, Trimmell J, Lu Y, Briscoe P, Feng JQ. Tooth agenesis in a family and homozygous *PAX9* mutation in exon 3: a case report. *World J Orthod*. 2008;9:e55–e61.
145. Pereira TV, Salzano FM, Mostowska A, et al. Natural selection and molecular evolution in primate *PAX9* gene, a major determinant of tooth development. *Proc Natl Acad Sci USA*. 2006;103:5676–5681.
146. Yamaguchi S, Machida J, Kamamoto M, et al. Characterization of novel *MSX1* mutations identified in Japanese patients with nonsyndromic tooth agenesis. *PLoS One*. 2014;9:e102944.
147. Kimura M, Machida J, Yamaguchi S, et al. Novel nonsense mutation in *MSX1* in familial nonsyndromic oligodontia: subcellular localization and role of homeodomain/MH4. *Eur J Oral Sci*. 2014;122:15–20.
148. Kim JW, Simmer JP, Lin BP, Hu JC. Novel *MSX1* frameshift causes autosomal-dominant oligodontia. *J Dent Res*. 2006;85:267–271.
149. Lidral AC, Reising BC. The role of *MSX1* in human tooth agenesis. *J Dent Res*. 2002;81:274–278.
150. Vastardis H, Karimbux N, Guthua SW, Seidman JG, Seidman CE. A human *MSX1* homeodomain missense mutation causes selective tooth agenesis. *Nat Genet*. 1996;13:417–421.

151. Mostowska A, Biedziak B, Trzeciak WH. A novel c.581C > T transition localized in a highly conserved homeobox sequence of *MSX1*: is it responsible for oligodontia? *J Appl Genet*. 2006;47:159–164.
152. Xuan K, Jin F, Liu YL, et al. Identification of a novel missense mutation of *MSX1* gene in Chinese family with autosomal-dominant oligodontia. *Arch Oral Biol*. 2008;53:773–779.
153. De Muynck S, Schollen E, Matthijs G, Verdonck A, Devriendt K, Carels C. A novel *MSX1* mutation in hypodontia. *Am J Med Genet A*. 2004;128A:401–403.
154. Chishti MS, Muhammad D, Haider M, Ahmad W. A novel missense mutation in *MSX1* underlies autosomal recessive oligodontia with associated dental anomalies in Pakistani families. *J Hum Genet*. 2006;51: 872–878.
155. Pawlowska E, Janik-Papis K, Wisniewska-Jarosinska M, Szczepanska J, Blasiak J. Mutations in the human homeobox *MSX1* gene in the congenital lack of permanent teeth. *Tohoku J Exp Med*. 2009;217:307–312.
156. Antunes LS, Küchler EC, Tannure PN, et al. *TGFB3* and *BMP4* polymorphism are associated with isolated tooth agenesis. *Acta Odontol Scand*. 2012;70:202–206.
157. Lammi L, Arte S, Somer M, et al. Mutations in *AXIN2* cause familial tooth agenesis and predispose to colorectal cancer. *Am J Hum Genet*. 2004;74:1043–1050.
158. Song S, Zhao R, He H, Zhang J, Feng H, Lin L. *WNT10A* variants are associated with non-syndromic tooth agenesis in the general population. *Hum Genet*. 2014;133:117–124.
159. Mostowska A, Biedziak B, Zadurska M, Dunin-Wilczynska I, Lianeri M, Jagodzinski PP. Nucleotide variants of genes encoding components of the Wnt signalling pathway and the risk of non-syndromic tooth agenesis. *Clin Genet*. 2013;84:429–440.
160. van den Boogaard MJ, Creton M, Bronkhorst Y, et al. Mutations in *WNT10A* are present in more than half of isolated hypodontia cases. *J Med Genet*. 2012;49:327–331.
161. Arzoo PS, Klar J, Bergendal B, Norderyd J, Dahl N. *WNT10A* mutations account for (1/4) of population-based isolated oligodontia and show phenotypic correlations. *Am J Med Genet A*. 2014;164A:353–359.
162. Mostowska A, Biedziak B, Zadurska M, Matuszewska-Trojan S, Jagodzinski PP. *WNT10A* coding variants and maxillary lateral incisor agenesis with associated dental anomalies. *Eur J Oral Sci*. 2015;123:1–8.
163. Abdalla EM, Mostowska A, Jagodziński PP, Dwidar K, Ismail SR. A novel *WNT10A* mutation causes non-syndromic hypodontia in an Egyptian family. *Arch Oral Biol*. 2014;59:722–728.
164. Sarkar T, Bansal R, Das P. Whole genome sequencing reveals novel non-synonymous mutation in ectodysplasin a (*EDA*) associated with non-syndromic x-linked dominant congenital tooth agenesis. *PLoS One*. 2014;9:e106811.
165. Tao R, Jin B, Guo SZ, et al. A novel missense mutation of the *EDA* gene in a Mongolian family with congenital hypodontia. *J Hum Genet*. 2006;51:498–502.
166. Tarpey P, Pemberton TJ, Stockton DW, et al. A novel Gln358Glu mutation in ectodysplasin A associated with X-linked dominant incisor hypodontia. *Am J Med Genet A*. 2007;143A:390–394.
167. Han D, Gong Y, Wu H, et al. Novel *EDA* mutation resulting in X-linked non-syndromic hypodontia and the pattern of *EDA*-associated isolated tooth agenesis. *Eur J Med Genet*. 2008;51:536–546.
168. Nikopensius T, Annilo T, Jagomägi T, et al. Non-syndromic tooth agenesis associated with a nonsense mutation in ectodysplasin-a (*EDA*). *J Dent Res*. 2013;92:507–511.
169. Ayub M, Ur-Rehman F, Yasinzai M, Ahmad W. A novel missense mutation in the ectodysplasin-A (*EDA*) gene underlies X-linked recessive nonsyndromic hypodontia. *Int J Dermatol*. 2010;49:1399–1402.
170. Rasool M, Schuster J, Aslam M, et al. A novel missense mutation in the *EDA* gene associated with X-linked recessive isolated hypodontia. *J Hum Genet*. 2008;53:894–898.
171. Yang Y, Luo L, Xu J, et al. Novel *EDA* p.Ile260Ser mutation linked to non-syndromic hypodontia. *J Dent Res*. 2013;92:500–506.
172. Vieira AR, Meira R, Modesto A, Murray JC. *MSX1, PAX9*, and *TGFA* contribute to tooth agenesis in humans. *J Dent Res*. 2004;83:723–727.
173. Callahan N, Modesto A, Deeley K, Meira R, Vieira AR. Transforming growth factor-alfa gene (*TGFA*), human tooth agenesis, and evidence of segmental uniparental isodisomy. *Eur J Oral Sci*. 2009;117:20–26.
174. Vieira AR, Modesto A, Meira R, Barbosa ARS, Lidral AC, Murray JC. Interferon regulatory factor 6 (*IRF6*) and fibroblast growth factor receptor 1 (*FGFR1*) contribute to human tooth agenesis. *Am J Med Genet A*. 2007;143A:538–545.
175. Vieira AR, Seymen F, Patir A, Menezes R. Evidence of linkage disequilibrium between polymorphisms at the *IRF6* locus and isolate tooth agenesis, in a Turkish population. *Arch Oral Biol*. 2008;53:780–784.
176. Gao Y, Kobayashi H, Ganss B. The human *KROX-26/ZNF22* gene is expressed at sites of tooth formation and maps to the locus for permanent tooth agenesis (He-Zhao deficiency). *J Dent Res*. 2003;82:1002–1007.
177. Liu W, Wang H, Zhao S, et al. The novel gene locus for agenesis of permanent teeth (He-Zhao deficiency) maps to chromosome 10q11.2. *J Dent Res*. 2001;80:1716–1720.
178. Stockton DW, Das P, Goldenberg M, D'Souza RN, Patel PI. Mutation of *PAX9* is associated with oligodontia. *Nat Genet*. 2000;24:18–19.
179. Noor A, Windpassinger C, Vitcu I, et al. Oligodontia is caused by mutation in *LTBP3*, the gene encoding latent TGF-beta binding protein 3. *Am J Hum Genet*. 2009;84:519–523.
180. Nuckolls GH, Shum L, Slavkin HC. Progress toward understanding craniofacial malformations. *Cleft Palate Craniofac J*. 1999;36:12–26.
181. Marvin ML, Mazzoni SM, Herron CM, Edwards S, Gruber SB, Petty EM. *AXIN2*-associated autosomal dominant ectodermal dysplasia and neoplastic syndrome. *Am J Med Genet A*. 2011;155A:898–902.
182. Pinarbasi E, Gunes EG, Pinarbasi H, Donmez G, Silig Y. *AXIN2* polymorphism and its association with prostate cancer in a Turkish population. *Med Oncol*. 2011;28:1373–1378.
183. Mazzoni SM, Fearon ER. *AXIN1* and *AXIN2* variants in gastrointestinal cancers. *Cancer Lett*. 2014;355:1–8.
184. Chalothorn LA, Beeman CS, Ebersole JL, et al. Hypodontia as a risk marker for epithelial ovarian cancer: a case-controlled study. *J Am Dent Assoc*. 2008;139:163–169.
185. Fekonja A, Cretnik A, Takac I. Hypodontia prevalence and pattern in women with epithelial ovarian cancer. *Angle Orthod*. 2014;84:810–814.
186. Fekonja A, Cretnik A, Zerdoner D, Takac I. Hypodontia phenotype in patients with epithelial ovarian cancer. *Radiol Oncol*. 2015;49:65.
187. Kuchler EC, Lips A, Tannure PN, et al. Tooth agenesis association with self-reported family history of cancer. *J Dent Res*. 2013;92:149–155.
188. McSherry PF. The ectopic maxillary canine: a review. *Br J Orthod*. 1998;25:209–216.
189. Becker A, Sharabi S, Chaushu S. Maxillary tooth size variation in dentitions with palatal canine displacement. *Eur J Orthod*. 2002;24:313–318.
190. Pirinen S, Arte S, Apajalahti S. Palatal displacement of canine is genetic and related to congenital absence of teeth. *J Dent Res*. 1996;75:1742–1746.
191. Becker A, Gillis I, Shpack N. The etiology of palatal displacement of maxillary canines. *Clin Orthod Res*. 1999;2:62–66.
192. Chaushu S, Sharabi S, Becker A. Tooth size in dentitions with buccal canine ectopia. *Eur J Orthod*. 2003;25:485–491.
193. Becker A. In defense of the guidance theory of palatal canine displacement. *Angle Orthod*. 1995;65:95–98.
194. Peck S, Peck L, Kataja M. The palatally displaced canine as a dental anomaly of genetic origin. *Angle Orthod*. 1994;64:249–256.
195. Peck S, Peck L, Kataja M. Concomitant occurrence of canine malposition and tooth agenesis: evidence of orofacial genetic fields. *Am J Orthod Dentofac Orthop*. 2002;122:657–660.
196. Proffit WR, Frazier-Bowers SA. Mechanism and control of tooth eruption: overview and clinical implications. *Orthod Craniofac Res*. 2009;12:59–66.
197. Decker E, Stellzig-Eisenhauer A, Fiebig BS, et al. *PTHR1* loss-of-function mutations in familial, nonsyndromic primary failure of tooth eruption. *Am J Hum Genet*. 2008;83:781–786.
198. Wise GE, Frazier-Bowers S, D'Souza RN. Cellular, molecular, and genetic determinants of tooth eruption. *Crit Rev Oral Biol Med*. 2002;13:323–334.
199. Black 3rd TK. Fluctuating asymmetry in the deciduous dentition. *J Dent Res*. 1980;59:725.

200. Corruccini RS, Potter RH. Developmental correlates of crown component asymmetry and occlusal discrepancy. *Am J Phys Anthropol*. 1981;55: 21–31.
201. Sprowls MW, Ward RE, Jamison PL, Hartsfield Jr JK. Dental arch asymmetry, fluctuating dental asymmetry, and dental crowding: a comparison of tooth position and tooth size between antimeres. *Semin Orthod*. 2008;14:157–165.
202. Harris EF, Robinson QC, Woods MA. An analysis of causes of apical root resorption in patients not treated orthodontically. *Quintessence Int*. 1993;24:417–428.
203. Harris EF, Butler ML. Patterns of incisor root resorption before and after orthodontic correction in cases with anterior open bites. *Am J Orthod Dentofac Orthop*. 1992;101:112–119.
204. Brezniak N, Wasserstein A. Root resorption after orthodontic treatment: Part 2. Literature review. *Am J Orthod Dentofac Orthop*. 1993;103:138–146.
205. Brezniak N, Wasserstein A. Root resorption after orthodontic treatment: Part 1. Literature review. *Am J Orthod Dentofac Orthop*. 1993;103:62–66.
206. DeShields RW. A study of root resorption in treated Class II, Division I malocclusions. *Angle Orthod*. 1969;39:231–245.
207. Sharpe W, Reed B, Subtelny JD, Polson A. Orthodontic relapse, apical root resorption, and crestal alveolar bone levels. *Am J Orthod Dentofac Orthop*. 1987;91:252–258.
208. Parker RJ, Harris EF. Directions of orthodontic tooth movements associated with external apical root resorption of the maxillary central incisor. *Am J Orthod Dentofac Orthop*. 1998;114:677–683.
209. Linge L, Linge BO. Patient characteristics and treatment variables associated with apical root resorption during orthodontic treatment. *Am J Orthod Dentofac Orthop*. 1991;99:35–43.
210. Baumrind S, Korn EL, Boyd RL. Apical root resorption in orthodontically treated adults. *Am J Orthod Dentofac Orthop*. 1996;110:311–320.
211. Horiuchi A, Hotokezaka H, Kobayashi K. Correlation between cortical plate proximity and apical root resorption. *Am J Orthod Dentofac Orthop*. 1998;114:311–318.
212. Owman-Moll P, Kurol J, Lundgren D. Continuous versus interrupted continuous orthodontic force related to early tooth movement and root resorption. *Angle Orthod*. 1995;65:395–401, discussion 401–402.
213. Kurol J, Owman-Moll P, Lundgren D. Time-related root resorption after application of a controlled continuous orthodontic force. *Am J Orthod Dentofac Orthop*. 1996;110:303–310.
214. Massler M, Malone AJ. Root resorption in human permanent teeth: a roentgenographic study. *Am J Orthod*. 1954;40:19–33.
215. Massler M, Perreault JG. Root resorption in the permament teeth of young adults. *J Dent Child*. 1954;21:158–164.
216. Reitan K. Some factors determining the evaluation of forces in orthodontics. *Am J Orthod*. 1957;43:32–45.
217. Newman WG. Possible etiologic factors in external root resorption. *Am J Orthod*. 1975;67:522–539.
218. Sameshima GT, Sinclair PM. Predicting and preventing root resorption: Part I. Diagnostic factors. *Am J Orthod Dentofac Orthop*. 2001;119:505–510.
219. Harris EF, Kineret SE, Tolley EA. A heritable component for external apical root resorption in patients treated orthodontically. *Am J Orthod Dentofac Orthop*. 1997;111:301–309.
220. Hartsfield Jr JK, Everett ET, Al-Qawasmi RA. Genetic factors in external apical root resorption and orthodontic treatment. *Crit Rev Oral Biol Med*. 2004;15:115–122.
221. Ngan DC, Kharbanda OP, Byloff FK, Darendeliler MA. The genetic contribution to orthodontic root resorption: a retrospective twin study. *Aust Orthod J*. 2004;20:1–9.
222. Al-Qawasmi RA, Hartsfield Jr JK, Everett ET, et al. Root resorption associated with orthodontic force in inbred mice: genetic contributions. *Eur J Orthod*. 2006;28:13–19.
223. Abass SK, Hartsfield Jr JK, Al-Qawasmi RA, Everett ET, Foroud TM, Roberts WE. Inheritance of susceptibility to root resorption associated with orthodontic force in mice. *Am J Orthod Dentofac Orthop*. 2008;134:742–750.
224. Iwasaki LR, Crouch LD, Nickel JC. Genetic factors and tooth movement. *Semin Orthod*. 2008;14:135–145.
225. Abass SK, Hartsfield Jr JK. Orthodontics and external apical root resorption. *Semin Orthod*. 2007;13:246–256.
226. Al-Qawasmi RA, Hartsfield Jr JK, Everett ET, et al. Genetic predisposition to external apical root resorption. *Am J Orthod Dentofac Orthop*. 2003;123: 242–252.
227. Al-Qawasmi RA, Hartsfield Jr JK, Everett ET, et al. Genetic predisposition to external apical root resorption in orthodontic patients: linkage of chromosome-18 marker. *J Dent Res*. 2003;82:356–360.
228. Fontana MLSSN, de Souza CM, Bernardino JF, et al. Association analysis of clinical aspects and vitamin D receptor gene polymorphism with external apical root resorption in orthodontic patients. *Am J Orthod Dentofac Orthop*. 2012;142:339–347.
229. Gülden N, Eggermann T, Zerres K, Beer M, Meinelt A, Diedrich P. Interleukin-1 polymorphisms in relation to external apical root resorption (EARR). *J Orofac Orthop*. 2009;70:20–38.
230. Iglesias-Linares A, Yanez-Vico R, Ballesta-Mudarra S, et al. Postorthodontic external root resorption is associated with *IL1* receptor antagonist gene variations. *Oral Dis*. 2012;18:198–205.
231. Iglesias-Linares A, Yanez-Vico RM, Ballesta S, et al. Interleukin 1 gene cluster SNPs (rs1800587, rs1143634) influences post-orthodontic root resorption in endodontic and their contralateral vital control teeth differently. *Int Endod J*. 2012;45:1018–1026.
232. Iglesias-Linares A, Yanez-Vico RM, Ballesta-Mudarra S, et al. Interleukin 1 receptor antagonist (IL1RN) genetic variations condition post-orthodontic external root resorption in endodontically-treated teeth. *Histol Histopathol*. 2013;28:767–773.
233. Iglesias-Linares A, Yanez-Vico RM, Moreno-Fernandez AM, Mendoza-Mendoza A, Orce-Romero A, Solano-Reina E. Osteopontin gene SNPs (rs9138, rs11730582) mediate susceptibility to external root resorption in orthodontic patients. *Oral Dis*. 2014;20:307–312.
234. Iglesias-Linares A, Yanez-Vico RM, Ortiz-Ariza E, et al. Postorthodontic external root resorption in root-filled teeth is influenced by interleukin-1beta polymorphism. *J Endod*. 2012;38:283–287.
235. Linhartova P, Cernochova P, Izakovicova Holla L. *IL1* gene polymorphisms in relation to external apical root resorption concurrent with orthodontia. *Oral Dis*. 2013;19:262–270.
236. Sharab LY, Morford LA, Dempsey J, et al. Genetic and treatment-related risk factors associated with external apical root resorption (EARR) concurrent with orthodontia. *Orthod Craniofac Res*. 2015;18:71–82.
237. Pereira S, Lavado N, Nogueira L, Lopez M, Abreu J, Silva H. Polymorphisms of genes encoding P2X7R, *IL-1B*, OPG and RANK in orthodontic–induced apical root resorption. *Oral Dis*. 2014;20:659–667.
238. Hartsfield Jr JK. Pathways in external apical root resorption associated with orthodontia. *Orthod Craniofac Res*. 2009;12:236–242.
239. Viecilli RF, Katona TR, Chen J, Hartsfield Jr JK, Roberts WE. Orthodontic mechanotransduction and the role of the P2X7 receptor. *Am J Orthod Dentofac Orthop*. 2009;135:694 e1–e16, discussion 694–695.
240. Al-Qawasmi RA, Hartsfield Jr JK, Everett ET, Weaver MR, Foroud TM, Roberts WE. Root resorption associated with orthodontic force in IL-1beta knockout mouse. *J Musculoskelet Neuronal Interact*. 2004;4:383–385.
241. Behrens MK. *Priorities for Personalized Medicine*. President's Council of Advisors on Science and Technology; 2008.

3

The Biologic Basis of Orthodontics

Birgit Thilander, Nan Hatch, and Zongyang Sun

OUTLINE

TISSUE REACTIONS IN ORTHODONTICS, by Birgit Thilander, 51
Tooth-Supporting Tissues, 52
Gingiva, 52
Periodontal Ligament, 52
Root Cementum, 53
Alveolar Bone, 54
Physiologic Tooth Migration, 54
Orthodontic Tooth Movements, 57
Orthodontic Forces and Tissue Reaction, 57
Tissue Response in Periodontium, 59
Initial Period of Tooth Movement, 59
Hyalinization Phase, 59
Hyalinized Zone and Root Resorption, 60
Secondary Period of Tooth Movement, 64
Types of Tooth Movements, 65
Tipping, 65
Torque, 65
Bodily Movement, 69
Rotation, 69
Extrusion, 70
Intrusion, 71
Movements in the Labial/Buccal Direction, 71
Movements into Reduced Alveolar Bone Height, 73
Transmission of Orthodontic Forces into a Cellular Reaction, 74
Tissue Response in Sutures, 74
Structure of the Suture, 76
Suture Response to Orthodontic/Orthopedic Forces, 77
Tissue Response in the Temporomandibular Joint, 78
Structure of the Temporomandibular Joint, 79
Condylar Cartilage, 79
Mandibular Neck, 79
Temporal Component, 79
Temporomandibular Joint Response to Orthopedic Forces, 79
Posttreatment Stability, 81
Tissue Reactions Seen in Orthodontic Retention and Relapse, 81
Changes during the Postretention Period, 83
Summary, 84
TOOTH MOVEMENT AT THE CELLULAR AND MOLECULAR LEVELS, by Nan Hatch and Zongyang Sun, 85
Introduction, 85
Orthodontic Forces Stimulate Biological Responses, 85
Mechanotransduction Mediates the Bone Modeling Response to Orthodontic Force, 87
Local Biological Mediators of Orthodontic Tooth Movement, 88
Neuropeptides and Orthodontic Tooth Movement, 89
RANK/RANKL/OPG System for Control of Osteoclastogenesis and Tooth Movement, 89
Biological Control of Osteogenesis Involved in Orthodontic Tooth Movement, 91
Physical Methods That Stimulate the Biology of Orthodontic Tooth Movement, 93
Injury-Facilitated Acceleration of Tooth Movement, 93
Vibration-Induced Acceleration of Tooth Movement, 93
Laser Irradiation-Induced Acceleration of Tooth Movement, 93
Translation of Biological Techniques into Orthodontic Practice: The Future of Our Profession?, 94

TISSUE REACTIONS IN ORTHODONTICS*

Orthodontic treatment today comprises a wealth of removable and fixed appliances, sometimes in combination with extraoral ones. Despite differences in design, they all involve the use and control of forces acting on the teeth and adjacent structures. The principal changes from such forces are seen within the dentoalveolar system, resulting in *tooth movements*. Other structures, such as sutures and the temporomandibular joint (TMJ) area, can also be influenced by means of *dentofacial orthopedics*. An optimal orthodontic force intends to induce a maximal cellular response and to establish stability of the tissue, whereas an unfavorable force does not result in a precise biological response and may initiate adverse tissue reactions.

Although a considerable number of studies based on human material illustrate the *quantitative* changes occurring during orthodontic treatment (e.g., cephalometry), the main evidence for the *qualitative* histologic responses to orthodontic treatment comes from experimental studies on animals. Human material has been used to study orthodontically

*Birgit Thilander

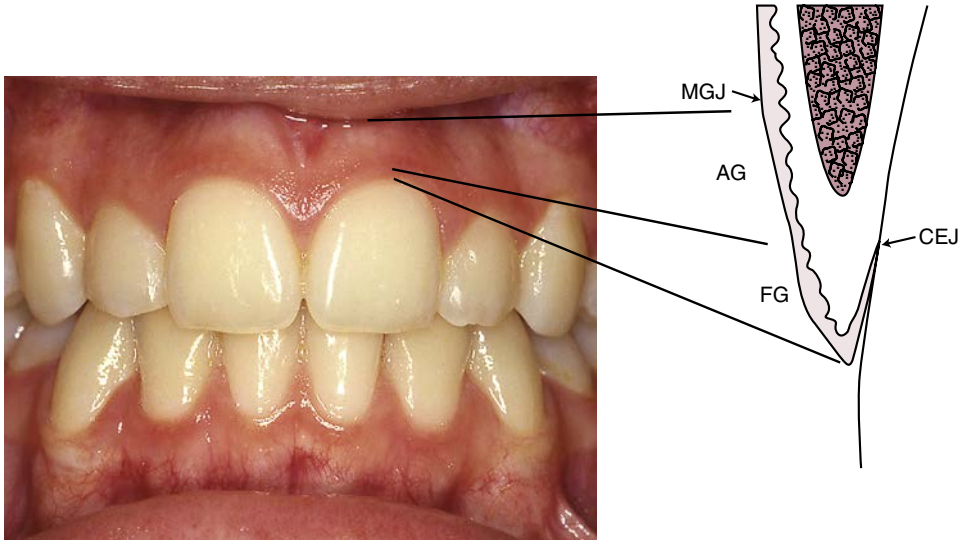


FIGURE 3-1 Macroscopic anatomy of the gingiva showing free gingiva (FG), attached gingiva (AG), mucogingival junction (MGJ), and cementoenamel junction (CEJ).

induced reactions in the supporting structures of teeth intended for extraction, but animal material must be used to study basal changes. However, conclusions based on such results should be viewed with caution because species may differ in terms of basic morphology, growth pattern, turnover rate, and speed of tissue reaction. Moreover, animal experiments involve the alteration of normal structures, whereas dentofacial orthopedic measures are aimed at correcting already existing anomalies.

The main purpose of this chapter is not to discuss appliances but instead to focus on tissue reactions in the periodontium, sutures, and the TMJ region during the active phase of treatment as well as during the retention and postretention periods.

TOOTH-SUPPORTING TISSUES

During tooth movement, changes in the periodontium occur, depending on magnitude, direction, and duration of the force applied, as well as the age of the orthodontically treated patient. Tooth movement is a complicated process, requiring changes in the gingiva, periodontal ligament, root cementum, and alveolar bone with their differences in cell population and remodeling capacity. This chapter, therefore, includes a brief description of the normal periodontium.[1]

Gingiva

The gingiva is differentiated into the free and attached gingiva (Fig. 3-1). In a clinically healthy condition, the *free gingiva* is in close contact with the enamel surface, and its margin is located 0.5 to 2 mm coronal to the cementoenamel junction after completed tooth eruption. The *attached gingiva* is firmly attached to the underlying alveolar bone and cementum by connective tissue fibers and is therefore comparatively immobile in relation to the underlying tissue.

The predominant component of the gingiva is the connective tissue, which consists of collagen fibers, fibroblasts, vessels, nerves, and matrix. The *fibroblast* is engaged in the production of various types of fibers but is also instrumental in the synthesis of the connective tissue matrix. The *collagen fibers* are bundles of collagen fibrils with a distinct orientation. They provide the resilience and tone necessary for maintaining its architectural form and the integrity of the dentogingival attachment. They are usually divided into the following groups (Fig. 3-2):

- *Circular fibers* run in the free gingiva and encircle the tooth.
- *Dentogingival fibers* are embedded in the cementum of the supraalveolar portion of the root and project from the cementum in a fanlike configuration into the free gingival tissue.
- *Dentoperiosteal fibers* are embedded in the same portion of the cementum as the dentogingival fibers but terminate in the tissue of the attached gingiva.
- *Transseptal fibers* run straight across the interdental septum and are embedded in the cementum of adjacent teeth (Fig. 3-3).

Periodontal Ligament

The periodontal ligament (PDL), about 0.25 mm wide, is the soft, richly vascular and cellular connective tissue that surrounds the roots of the teeth and joins the root cementum with the lamina dura or the alveolar bone proper. In the coronal direction, the PDL is continuous with the lamina propria of the gingiva and is separated from the gingiva by the collagen fiber bundles, which connect the alveolar bone crest with the root (the alveolar crest fibers).

The PDL and the root cementum develop from the follicle, which surrounds the tooth bud. The true periodontal fibers, *the principal fibers*, develop along with the eruption of the tooth. First, fine fibrils arise from the root cementum and the bone surface and fuse as they contact. Later on, the number and thickness of fibers increase. The orientation of the collagen fiber bundles alters continuously during tooth eruption. When the tooth has reached contact in occlusion and is functioning properly, they associate with the following well-oriented groups: *alveolar crest fibers* and *horizontal*, *oblique*, *apical*, *and interradicular fibers* (Fig. 3-4). The individual bundles have a slightly wavy course, which allows the tooth to move within its socket (physiologic mobility). The presence of a PDL makes it possible to distribute and resorb the forces elicited during mastication and is essential for movement of the teeth in orthodontic treatment.

The fibrils of the PDL are embedded in a ground substance with connective tissue polysaccharides (glycosaminoglycans),

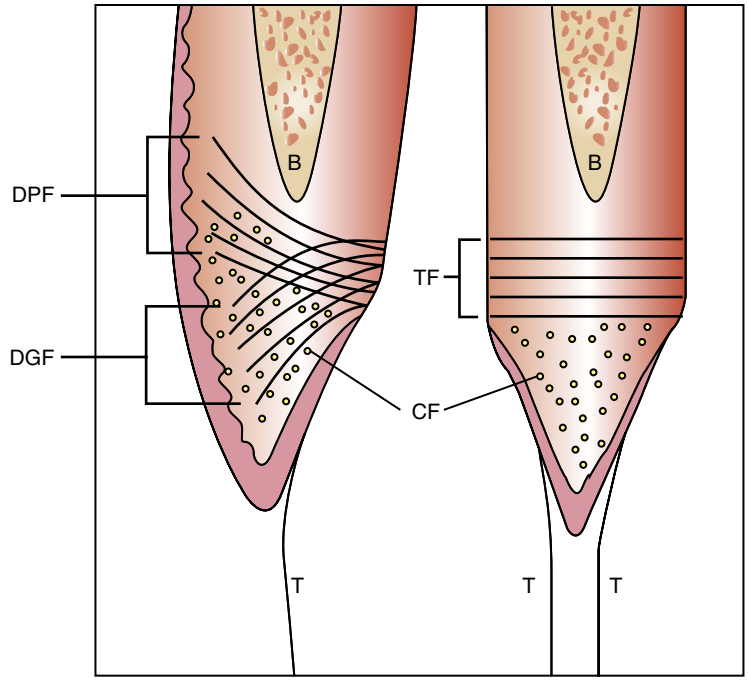


FIGURE 3-2 Drawings (vestibular and interdental aspects of the teeth) illustrating the different collagen bundles in the gingiva showing circular fibers (CF), dentogingival fibers (DGF), dentoperiosteal fibers (DPF), and transseptal fibers (TF). (Adapted from Lindhe J, Karring T. The anatomy of the periodontium. In: Lindhe J, ed. *Textbook of Clinical Periodontology*. Copenhagen: Munksgaard; 1989.)

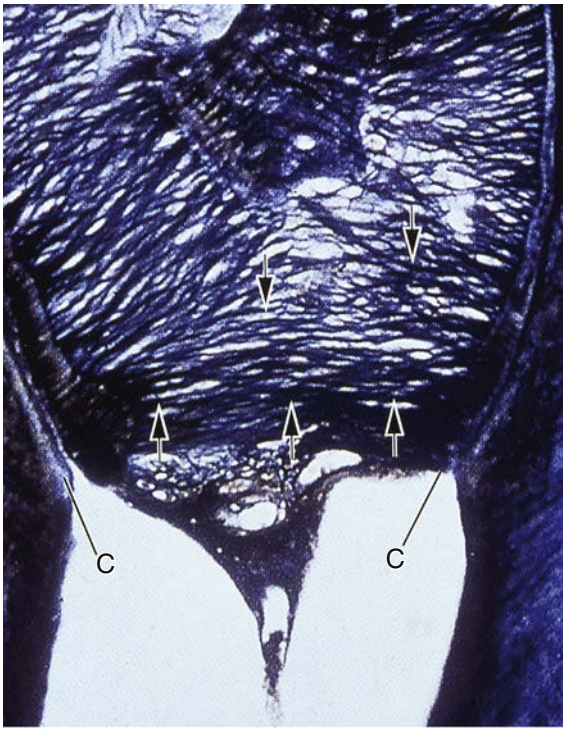


FIGURE 3-3 A histologic section illustrating the orientation of transseptal fiber bundles (*arrows*) in the supraalveolar portion of the interdental area. They also connect the cementum (*C*). (From Lindhe J, Karring T: The anatomy of the periodontium. In Lindhe J, ed. *Textbook of Clinical Periodontology*. 4th ed. Copenhagen: Blackwell Munksgaard; 2003. With permission.)

which vary with age. The tissue response to orthodontic forces, including cell mobilization and conversion of collagen fibers, is considerably slower in older individuals than in children and adolescents. The ground substance has a more rapid turnover than the collagen fibers.

During physiologic conditions, collagen turnover in the PDL is much higher than that in most other tissues (e.g., twice as high as that of the gingiva). The high turnover has been attributed to the fact that forces on the PDL are multidirectional, having vertical and horizontal components. The lower collagen turnover in the gingiva may result from the lowered functional stress as the transseptal fibers function in a manner similar to tendons, providing firm anchorage of the tooth.

Root Cementum

The root cementum is a specialized mineralized tissue covering the root surface and has many features in common with bone tissue. However, the cementum contains no blood vessels, has no innervation, does not undergo physiologic resorption or remodeling, and is characterized by continuing deposition throughout life. The cementum attaches the PDL fibers to the root and contributes to the process of repair after damage to the root surface (e.g., during orthodontic treatment).

During root formation, a *primary cementum* is formed. After tooth eruption and in response to functional demands, a *secondary cementum* is formed that, in contrast to the primary cementum, contains cells. During the continuous formation of the primary cementum, portions of the principal fibers in the PDL adjacent to the root become embedded and mineralized (Fig. 3-5). The Sharpey fibers in the cementum should be regarded as a direct continuation of the collagen fibers in the PDL.

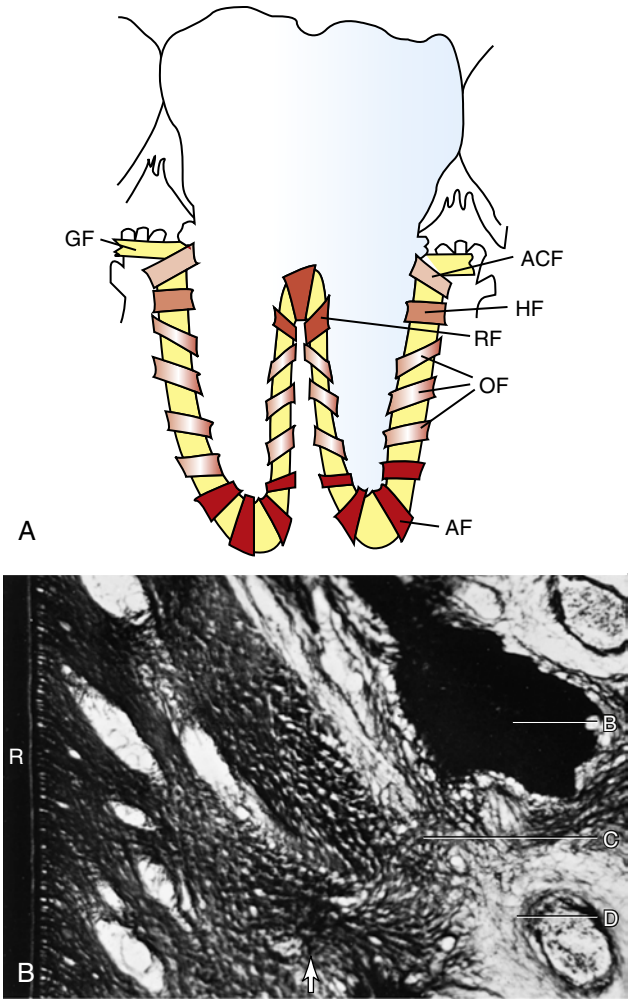


FIGURE 3-4 A, The PDL fibers: Alveolar-crest fibers (*ACF*), apical fibers (*AF*), gingival fibers (*GF*), horizontal fibers (*HF*), oblique fibers (*OF*), and interradicular fibers (*RF*). B, The periodontal fibers interlace with the fibrous matrix of the bone, forming a fibrous system. In a physiologic state this system appears relaxed. *R*, Root surface; *B*, Remaining calcified bone in which the fiber arrangement cannot be seen; *C*, Sharpey's fibers interlacing with fibers of the bone; *D*, Loose fibrous tissue around a capillary (*arrow*).

Alveolar Bone

The alveolar bone is covered with the *periosteum*, which is differentiated from the surrounding connective tissue (Fig. 3-6). The contiguous mesenchymal cells acquire the character of osteoblasts. The matrix-producing and proliferating cells in the cambium layer, as well as osteocytes inside the bone matrix, are subject to mechanical influence. Whenever the pressure exceeds a certain threshold, reducing the blood supply to osteoblasts at PDL and alveolar bone interfaces, osteogenesis ceases. However, if the periosteum is exposed to tension, it responds with bone deposition. Therefore, the periosteum continues to function as an osteogenic zone throughout life, although its regenerative capacity is extremely high in the young child.

The alveolar process forms and supports the sockets of the teeth. It consists of dense outer *cortical bone* plates with varying amounts of spongy or *cancellous bone* between them. The thickness of the cortical laminae varies in different locations (Fig. 3-7). The cancellous bone contains bone trabeculae, the architecture of which is partly genetically determined and partly the result of forces to which teeth are exposed during function or orthodontic treatment.

The alveolar bone is renewed constantly in response to functional demands. Bone-forming *osteoblasts* and *osteoclasts*, cells involved in resorption (Fig. 3-8), are responsible for this remodeling process. These cells are present on the socket walls toward the periodontal membrane, on the inside of the cortical bone toward the marrow spaces, and on the surface of the bone trabeculae in cancellous bone. The osteoblasts produce *osteoid*, consisting of collagen fibers and a matrix that contains mainly proteoglycans and glycoproteins. Osteoid is found on all bone surfaces where new bone is deposited (Fig. 3-9, *A*), and unlike calcified bone is not attacked by osteoclasts. When this bone matrix undergoes mineralization by deposition of minerals, such as calcium and phosphate, it converts into *bundle bone*. Cells and fiber bundles are incorporated in bundle bone during its life cycle. When bundle bone has reached a certain thickness and maturity, parts of it are reorganized into *lamellated bone* (Fig. 3-9, *B*).

The type of bone through which the tooth is displaced must be considered in the orthodontic treatment plan. Tooth movements in a mesial or distal direction displace the roots through the spongiosa of the alveolar bone. When a tooth is moved into the reorganizing alveolus of a newly extracted tooth, remodeling is rapid because of the many differentiating cells present and the limited amount of bone to be resorbed. On the contrary, movement of a tooth labially or lingually into the thin cortical plates should be undertaken with a high degree of caution, especially in adult patients, to avoid iatrogenic responses.

PHYSIOLOGIC TOOTH MIGRATION

Tissue reactions in the tooth-supporting tissues are connected not only with orthodontic treatment but also with eruption of the teeth and development of the occlusion. The development of the dentition is a complex process and depends on many variables, which by no means always combine harmoniously.

The *eruption of teeth* to occlusal contact is an event that covers only a short period in life. However, the teeth and their supporting tissues have a lifelong ability to adapt to functional demands and hence drift through the alveolar process, a phenomenon called *physiologic tooth migration*. Also well known clinically is that any change in the equilibrium of occlusal pressure, such as loss of a neighboring or antagonistic tooth, may induce further tooth movement.

The tissue reaction that occurs during physiologic tooth migration is a normal function of the supporting structures. This was pointed out for the first time by Stein and Weinmann,[2] who observed that the molars in adults gradually migrate in a mesial direction. When the teeth migrate, they bring the supraalveolar fiber system with them. Such movement implies remodeling of the PDL and alveolar bone. The turnover rate of the PDL is not uniform throughout the ligament, the cells being more active on the bone side than near the root cementum.

The modeling processes that occur during the physiologic migration are illustrated in Figures 3-10 and 3-11. Osteoclasts are seen in scattered lacunae associated with the *resorptive surface* along the alveolar bone wall, toward which the tooth is moving; the number of cells is higher when tooth migration is rapid. The alveolar bone wall from which the

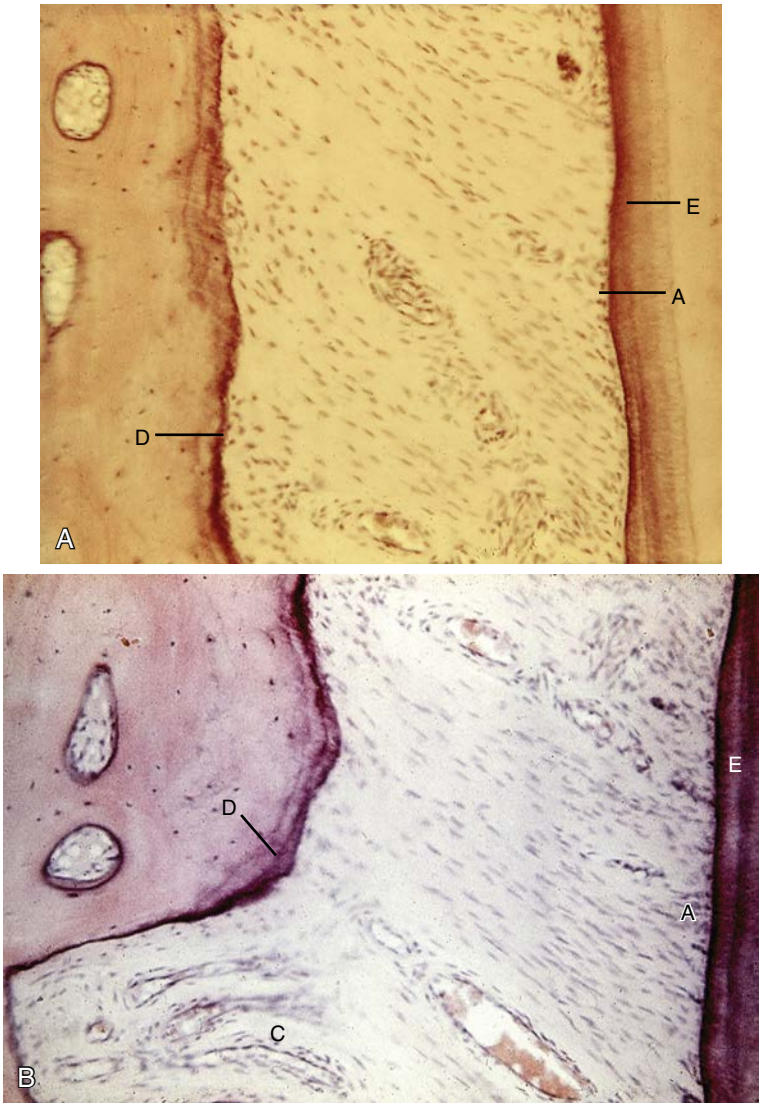


FIGURE 3-5 Area from the alveolar crest of **A**, a 39-year-old patient and, **B**, a 22-year-old patient. *A,* Chain of cementoblasts along a thick layer of cementum; *C,* Widened capillary in a cleft, where bone resorption may start during the initial stage of tooth movement; *D,* Darkly stained surface line containing connective tissue polysaccharides. Note the absence of osteoblasts along the bone surface; *E,* Embedded principal fibers.

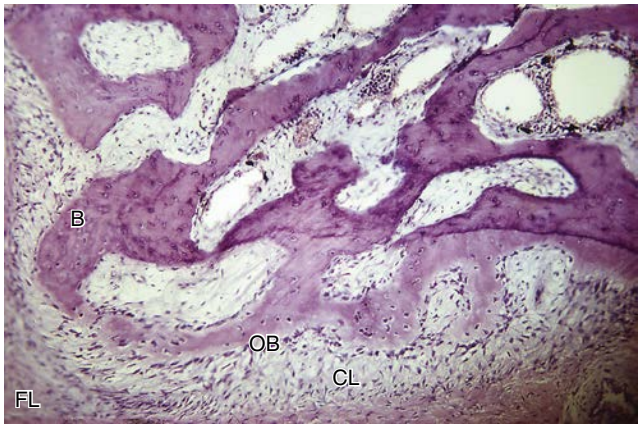


FIGURE 3-6 Photomicrograph of intramembranous bone (*B*) covered with periosteum. *OB,* Osteoblasts; *CL,* cambium layer; *FL,* fibrous layer.

tooth is moving away (*depository side*) is characterized by osteoblasts depositing nonmineralized osteoid, which later mineralizes in the deeper layer. Specific staining techniques reveal how the older fibers of the PDL are surrounded by newly deposited bone matrix and become embedded in bone. Simultaneously, new collagen fibrils are produced on the bone surface.

A slow apposition occurs on the cementum surface throughout life, a fact that is of great importance for the resorptive mechanism in the bone and cementum. The nonmineralized precementum layer has special importance as a resorption-resistant "coating" layer, thus protecting the root surface during the physiologic migration.

Because considerable changes in tooth position occur without any orthodontic invention, knowledge of periodontal remodeling during physiologic tooth migration is of utmost importance during the postretention period.

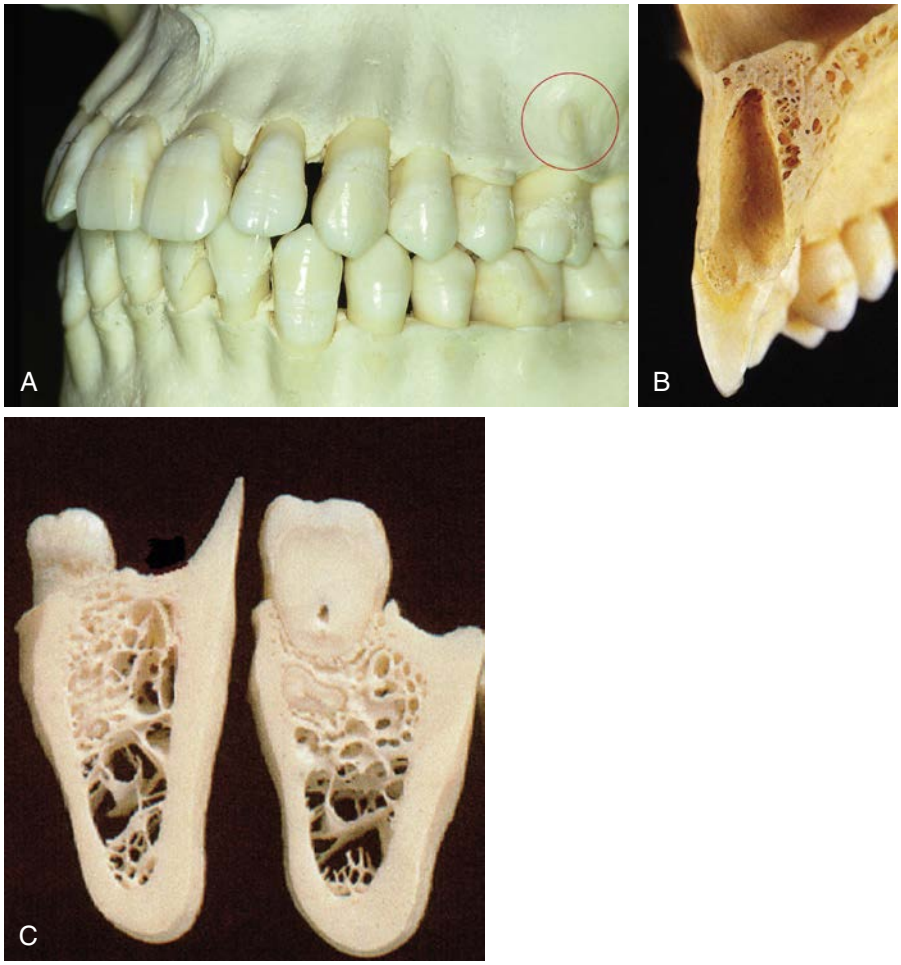


FIGURE 3-7 A, Cranium showing the various thicknesses of the cortical laminae in different locations of the alveolar processes. Note the thin bone plate at the buccal aspects of the mandibular incisor area and the teeth in the maxilla, resulting in fenestration (*circle*). B and C, Dried bone specimen from the incisor area of the maxilla and different areas of the mandible showing cancellous bone and outer cortical plate.

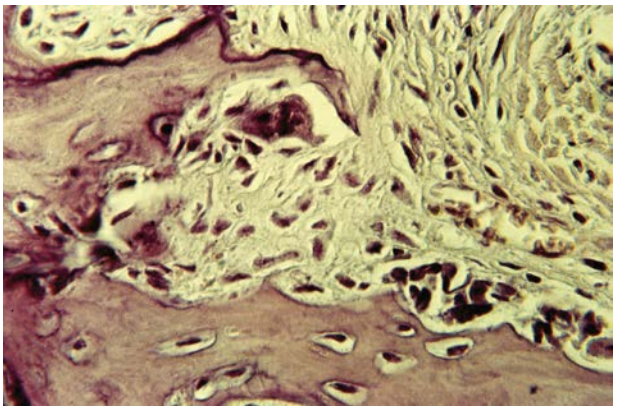


FIGURE 3-8 Photomicrograph of intramembranous bone with osteoclasts, involved in resorption.

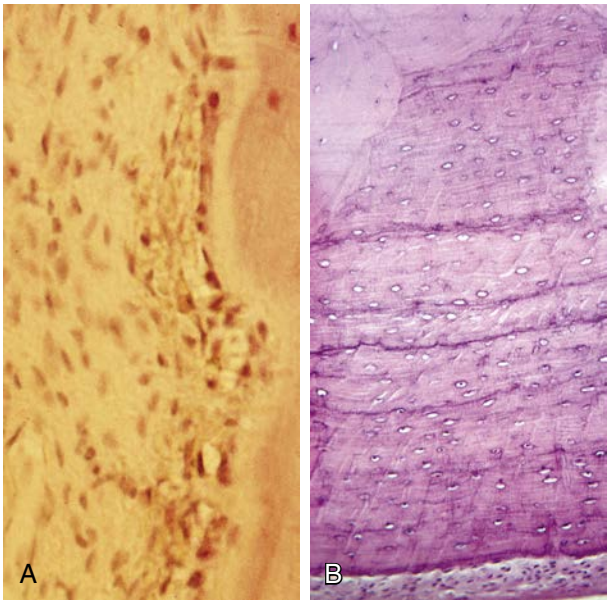


FIGURE 3-9 A, Osteoid is found on all bone surfaces, where new bone is deposited. B, Bundle bone reorganized into lamellated bone.

ORTHODONTIC TOOTH MOVEMENTS

Basically, tissue reactions observed in orthodontic tooth movement to some extent resemble that observed in physiologic tooth migration. Because the teeth are moved more rapidly during treatment, the tissue changes elicited by orthodontic forces are more significant and extensive.[3] The knowledge of the reactions of the supporting structures in orthodontic treatment is still incomplete because histologic techniques used today can provide only limited information on the behavior of the ground substance, blood, and tissue fluids. However, application of a force on the crown of the tooth leads to a biological response in its surrounding tissues, resulting in an orthodontic tooth movement, which depends on type, magnitude, and duration of the force. Thus, knowledge of several fundamental biological and mechanical concepts is necessary for a complete understanding of clinical orthodontics, and this is discussed in greater detail in Chapters 4 and 5. Hence, only a brief survey of biomechanical principles is given here, which is a factor of importance for understanding the tissue reaction in supporting tissues.

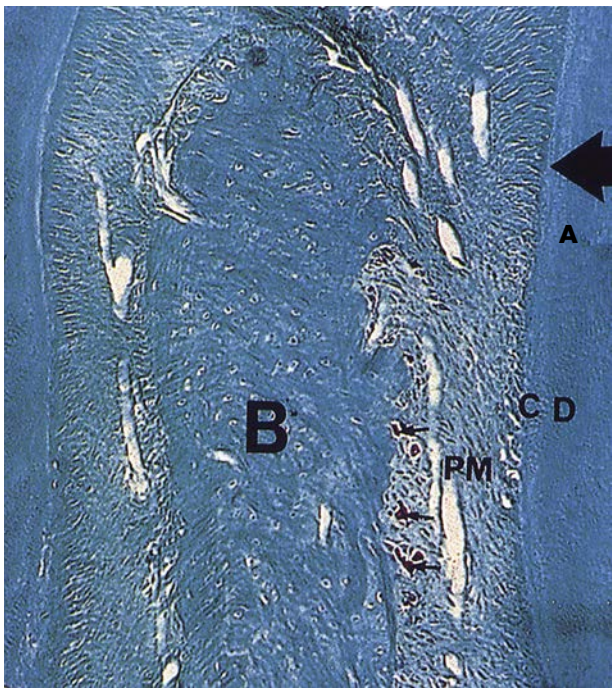


FIGURE 3-10 Physiologic migration (*PM*) of rat first molar (**A**) in direction of the arrows. Scattered osteoclasts, stained red, adjacent to the alveolar bone (**B**). *C.* Cementum *D.* Dentin (From Brudvik P, Rygh P. The initial phase of orthodontic root resorption incident to local compression of the periodontal ligament. *Eur J Orthod.* 1993;15(249).)

Orthodontic Forces and Tissue Reaction

Orthodontic forces comprise those that are meant to act on the PDL and alveolar process, whereas orthopedic forces are more powerful and act on the basal parts of the jaws. The decisive variables regarding these forces at the cellular level are application, magnitude, duration, and direction of force.[4]

Two different types of applied forces exist: *continuous* and *intermittent* (Fig. 3-12). Modern fixed appliance systems are based on light continuous forces from the archwire. However, a continuous force may be interrupted after a limited period. An example of such an *interrupted continuous* force is when it is no longer acting and hence has to be reactivated. In clinical orthodontics, an interrupted tooth movement may have certain advantages as the tissues are given ample time for reorganization, which is favorable for further tissue changes when the force is again activated. An *intermittent* force acts during a short period and is induced primarily by removable appliances, especially functional appliances.

The *magnitude* and duration of forces are important for the tissue response. Reactions in PDL to heavy, continuous loads (50 centiNewtons [cN]) in experimental tipping of first molars in rats[5] indicated that (1) up to a certain level of stress or duration, the reactions occur mainly in the periodontal membrane with increasing vascularization, cell proliferation, fiber

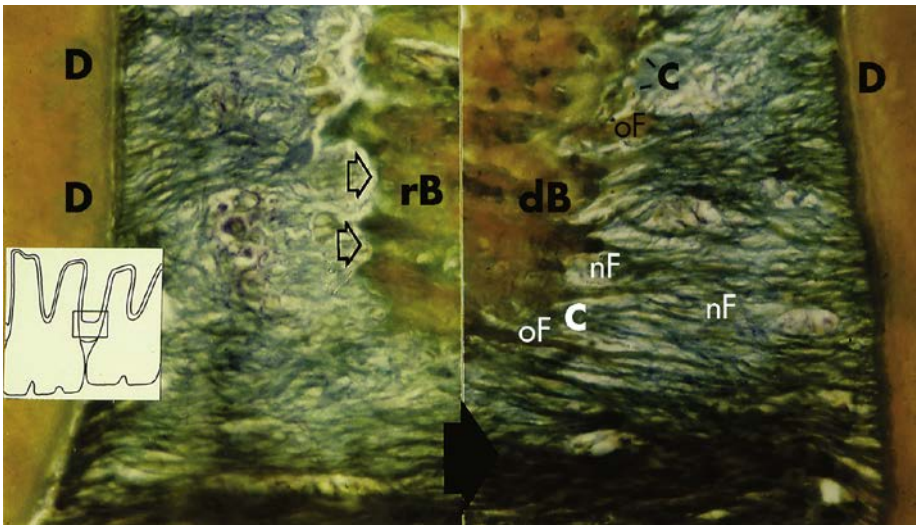


FIGURE 3-11 Physiologic migration in the rat in the interdental area in direction of *black arrow*. *rB,* Resorptive alveolar bone surface (*open arrows*); *dB,* depository alveolar bone surface; *oF,* older fibers included in the new bone formation by osteoclasts; *nF,* new fibrils near the bone surface and in the middle of the PDL; *C,* osteoblasts; *D,* dentin.

formation, and osteoid application on the bone surfaces and (2) beyond a certain level of stress or duration, decreased vascular supply in the PDL and destruction of cells between stretched fibers occur. The reactions become more significant within the alveolar bone, with removal and undermining resorption of Sharpey fibers from the rear (Fig. 3-13), subsequently permitting vascular invasion of cells into the periodontal membrane from the alveolar bone.

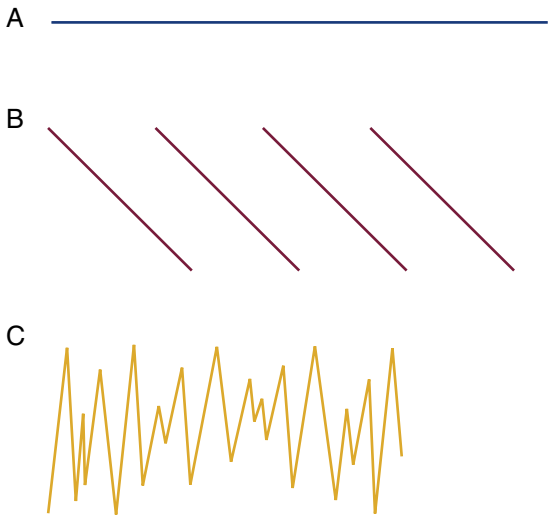


FIGURE 3-12 Orthodontic forces. **A,** Continuous. **B,** Interrupted continuous. **C,** Intermittent.

A light force over a certain distance moves a tooth more rapidly and with fewer injuries to the supporting tissues than a heavy one. The purpose of applying a light force is to increase cellular activity without causing undue tissue compression and to prepare the tissues for further changes. Another reason for applying light forces is that it results in less discomfort and pain to the patient. Unmyelinated nerve endings persist in the hyalinized tissue, and they are more or less compressed during the initial stage.

The *duration* of force, equivalent to treatment time, is considered to be a more crucial factor than the magnitude of the force regarding adverse tissue reactions, especially in connection with long treatment periods and in cases with high density of the alveolar bone.

Direction of forces will result in different kinds of tooth movements. All tooth movements can be described in terms of rotation and translation. This discussion addresses only the small, initial tooth movements that occur within the periodontal space. Clearly, the larger long-term movements are the result of a succession of such minor movements, depending on the pattern of socket remodeling. For the purposes of illustration, forces and movements are often presented in terms of *tipping*, *torquing*, *bodily*, *intrusion*, *extrusion*, *and rotation*. The different types of tooth movements will be discussed next.

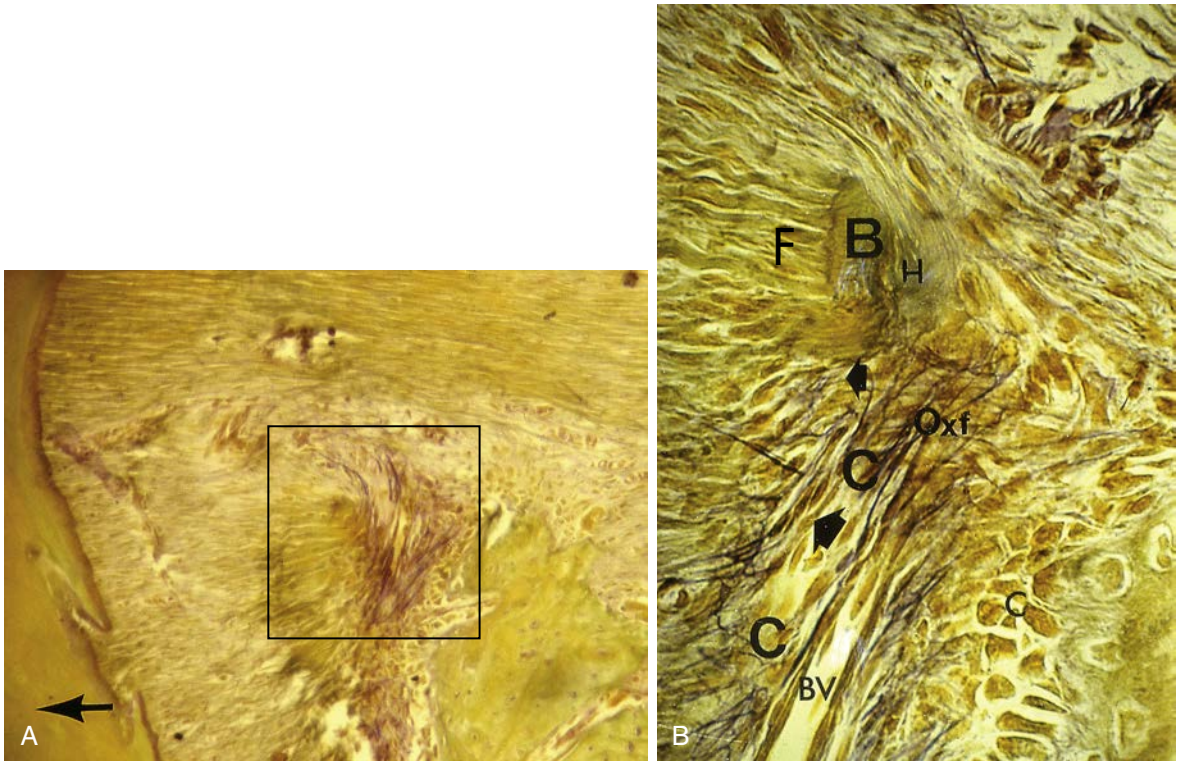


FIGURE 3-13 **A,** Area of tension of 28-day duration in the rat. Tooth moved in the direction of the arrow. Interface between Sharpey fibers (*F*) and alveolar bone (*B*) near the alveolar crest. **B,** Area corresponding to box in **A.** Sections are 6 μm apart. Note the proliferation of blood vessels (*BV*) in the alveolar bone, detaching periodontal membrane fibers from the bone surface from the rear. Blood vessels have infiltrated the alveolar bone plate (*B*) from the rear. Sharpey fibers and parts of the alveolar bone have disappeared. *C,* Undermining resorption; *H,* hyalinization; *Oxf,* oxytalan fibrils. (From Rygh P, et al. Activation of the vascular system: a main mediator of periodontal fiber remodeling in orthodontic tooth movement. *Am J Orthod.* 1986;89:453.)

TISSUE RESPONSE IN PERIODONTIUM

In 1905, Carl Sandstedt's studies[6] in dogs convincingly demonstrated that tooth movement is a process of resorption and apposition. He gave the first description of the glasslike appearance of the compressed tissue, termed *hyalinization*, which has been associated with a standstill of the tooth movement. It was not until the 1950s that tooth movements attracted wider attention with Kaare Reitan's classic study, *The initial tissue reaction incident to orthodontic tooth movement as related to the influence of function.*[7] Reitan used the dog as his experimental model but also extracted teeth from humans. Histology was his means to address the questions of clinical importance, such as the identity of the factors that control and regulate the pattern of tooth movement in different individuals. In this way, he explored age, sex, the type of alveolar bone, the kinds of forces, and the matrix of cells of the gingival and periodontal ligament. He pointed out differences in the tissue response between animals and humans. Per Rygh continued Reitan's research to explain why orthodontic tooth movements might cause damage to the tooth, possibly as a sequela of the hyalinization process.

Reitan and Rygh, dear friends and scientific colleagues, were my coauthors in "Tissue Reactions" in the third and fourth editions of *Orthodontics: Current Principles and Techniques.* They are remembered with gratitude even in this sixth edition.

Initial Period of Tooth Movement

Application of a continuous force on the crown of the tooth leads to tooth movement within the alveolus that is marked initially by narrowing of the periodontal membrane, particularly in the marginal area. After a certain period, osteoclasts differentiate along the alveolar bone wall, as occurs in young humans after 30 to 40 hours.[8] All permanent alterations depend on cellular activity. When conditions are favorable, the cells increase in number and differentiate into osteoclasts and fibroblasts. The width of the membrane is increased by osteoclastic removal of bone, and the orientation of the fibers in the periodontal membrane changes, as does the arrangement of the ground substance (Fig. 3-14). Experimental studies indicate that fibroblasts not only are capable of synthesizing fibrous tissue and ground substance but also play an important role in the breakdown of connective tissue. These processes occur simultaneously.[9]

Hyalinization Phase

During the crucial stage of the *initial application of force*, compression in limited areas of the membrane frequently impedes vascular circulation and cell differentiation, causing degradation of the cells and vascular structures rather than proliferation and differentiation. The tissue reveals a glasslike appearance in light microscopy, which is termed *hyalinization* (Fig. 3-15). It is caused partly by anatomic and partly by mechanical factors

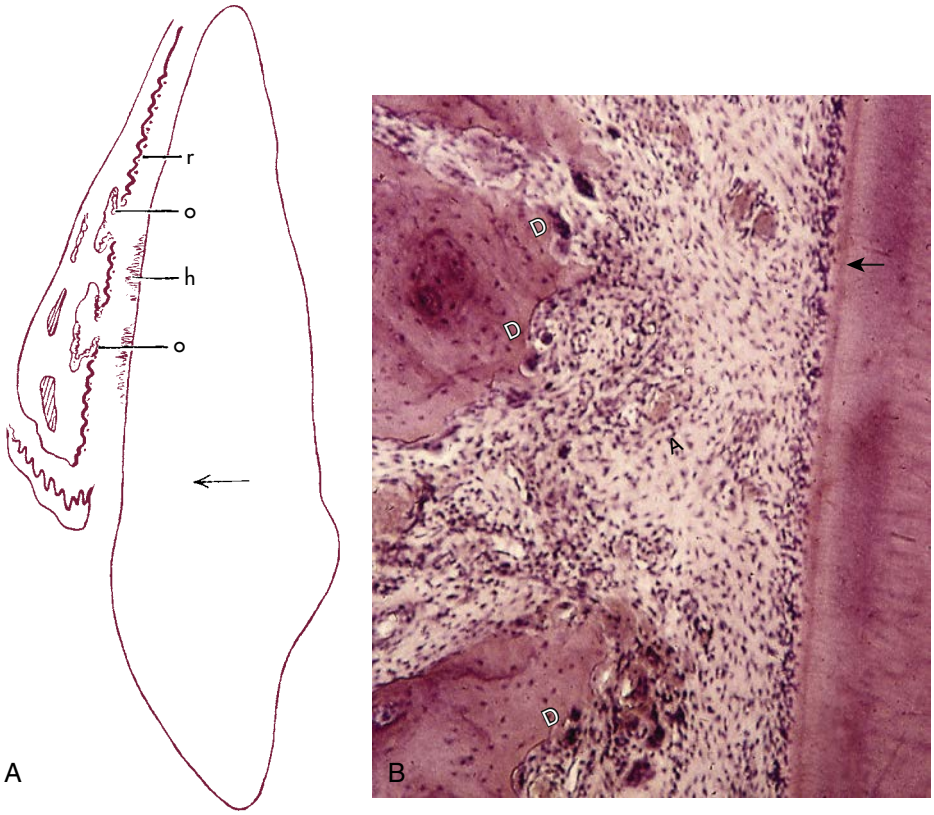


FIGURE 3-14 **A,** Location of bone resorption adjacent to the apical third of an upper canine in a 39-year-old patient. The tooth was moved continuously for 3 weeks. *O,* Compensatory formation of osteoid in open marrow spaces; *h*, Remnants of hyalinized tissue adhering to the root surface; *r,* direct bone resorption adjacent to the apical third of the root. **B,** Direct bone resorption with osteoclasts along the bone surface (*D*) (area marked *r* in A). *A,* Absence of epithelial remnants in adjacent periodontal tissue, center of the formerly cell-free zone; note the widening of the periodontal space; *D,* direct bone resorption with osteoclasts.

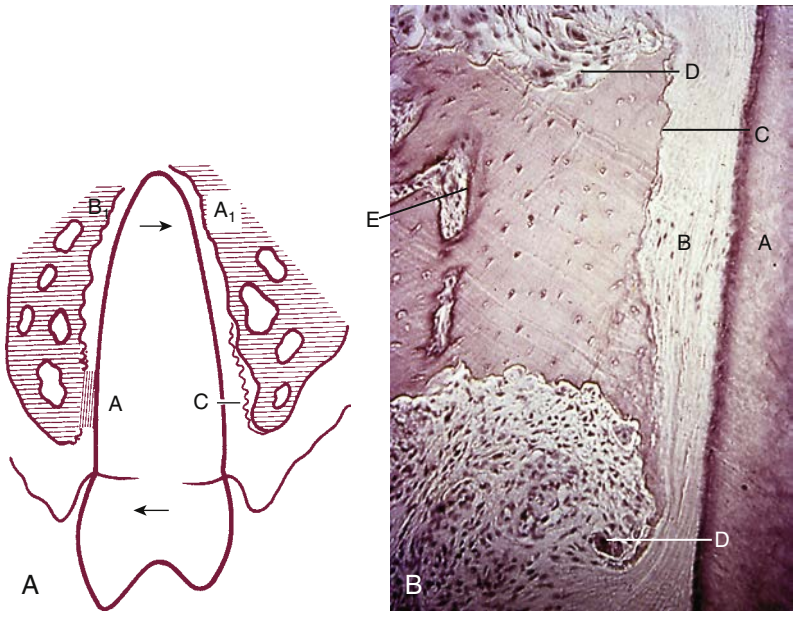


FIGURE 3-15 **A,** In most cases, tooth movement is initiated by formation of a cell-free area at *A* and new osteoid formed at *C*. A_1 and B_1 represent corresponding pressure and tension sides in the apical region. **B,** Area corresponding to *A* in **A**: the upper first premolar in a 12-year-old patient. Hyalinization of fairly long duration, here mainly caused by high bone density. No osteoclasts have been formed in the marrow spaces. *A,* Root surface; *B,* remaining pyknotic cell nuclei in hyalinized tissue; *C,* reversal line; *D,* direct bone resorption with osteoclasts; *E,* marrow space.

and is almost unavoidable in the initial period of tooth movement in clinical orthodontics. Hyalinization represents a sterile necrotic area, characterized by three main stages: degeneration, elimination of destroyed tissue, and establishment of a new tooth attachment.

Degeneration starts where the pressure is highest and the narrowing of the membrane is most pronounced, that is, around bone spicules. Degeneration may be limited to parts of the membrane or extend from the root surface to the alveolar bone. Electron microscopy[10-13] has shown that advanced cellular and vascular changes may occur within a few hours of the application of the force. Retardation of the blood flow is followed by disintegration of the vessel walls and degradation of blood elements, all occurring by mechanisms different from those seen during physiologic breakdown (Fig. 3-16). The cells undergo a series of changes, starting with a swelling of the mitochondria and the endoplasmic reticulum and continuing with rupture and dissolution of the cytoplastic membrane. This leaves only isolated nuclei between compressed fibrous elements (pyknosis) and is the first indication of hyalinization.

In hyalinized zones, no osteoclast progenitor cells can be recruited and subsequently differentiated into mature osteoclasts; thus no bone resorption can take place from the periodontal membrane. Tooth movement stops until the adjacent alveolar bone has been resorbed, the hyalinized structures are removed, and the area is repopulated by cells. A limited hyalinized area occurring during the application of light forces may be expected to persist from 2 to 4 weeks in a young patient (Fig. 3-17). When bone density is high, the duration is longer.

The peripheral areas of the hyalinized compressed tissue are *eliminated* by an invasion of cells and blood vessels from the adjacent undamaged PDL. The hyalinized materials are ingested by the phagocytic activity of macrophages and are removed completely[14] (Fig. 3-18, *A*). The adjacent alveolar bone is removed by indirect resorption by cells that have differentiated into osteoclasts on the surfaces of adjacent marrow spaces or, if the alveolar wall and the outer cortical bone are fused, on the surface of the alveolar process (Fig. 3-18, *B*).

Reestablishment of the tooth attachment in the hyalinized areas starts by synthesis of new tissue elements as soon as the adjacent bone and degenerated membrane tissue have been removed. The ligament space is now wider than before treatment started, and the membranous tissue under repair is rich in cells.[3]

Hyalinized Zone and Root Resorption

A side effect of the cellular activity during the removal of the necrotic hyalinized tissue is that the cementoid layer of the root and the bone are left with raw, unprotected surfaces in certain areas that can readily be attacked by resorptive cells (Fig. 3-19, *A*).[15,16] Root resorption then occurs around this cell-free tissue, starting at the border of the hyalinized zone (Fig. 3-19, *B*).

Some of these small resorption lacunae are visible only by the scanning microscope. According to Kvam,[17] organic tissue tends to remain in the resorbed area, which can be exposed more clearly by removing the organic components (Fig. 3-20). These initial injuries are small and insignificant.

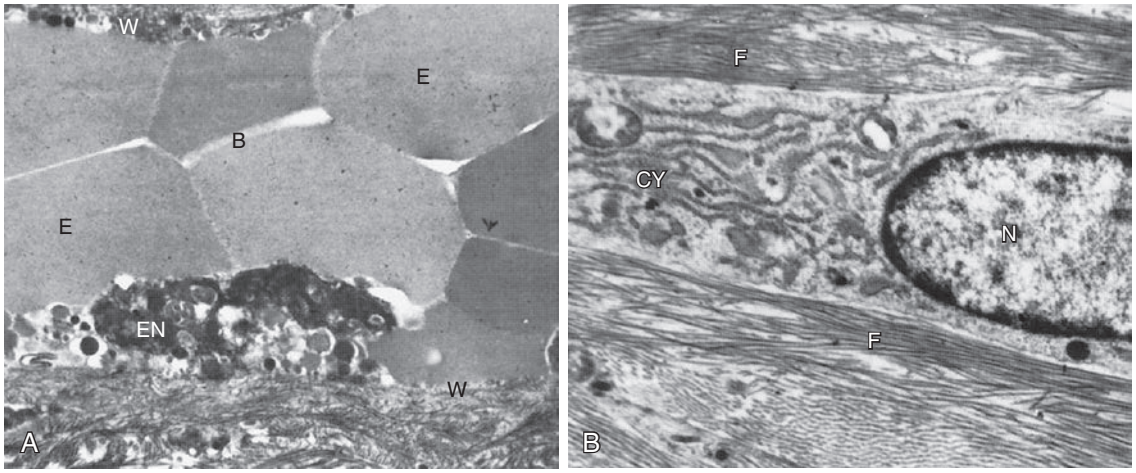


FIGURE 3-16 **A,** Early capillary changes in the hyalinized zone (human tissue) as seen with the electron microscope; findings usually not visible by light microscopy (force, 70 cN; duration, 2 days [×6000]). *E,* Stasis with packing of erythrocytes; *B,* borderline between red blood cells; *W,* disappearance of the capillary wall; *EN,* endothelial cell undergoing disintegration. **B,** Principal fibers on the tension side in the rat (force, 10 cN; duration, 28 days [×9000]). *F,* Fibrils subjected to moderate tension and located on both sides of the fibroblast; *N,* nucleus; *CY,* cytoplasm with well-developed endoplasmic reticulum. Prolonged stretching of supraalveolar fibers may cause disappearance of similarly located cellular elements as a result of compression. (From Rygh P. Ultrastructural vascular changes in pressure zones of rat molar periodontium incident to orthodontic tooth movement. *Scand J Dent Res.* 1972;80:307.)

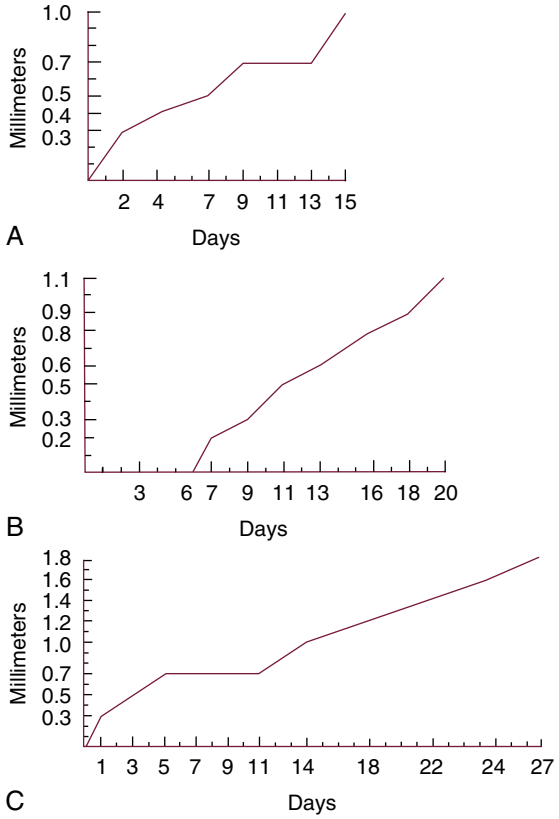


FIGURE 3-17 Movement of upper premolars with Bimler's removable appliances (force, 70 to 100 cN). Note the short hyalinization periods. **A,** A cell-free zone lasted for about 4 days. **B,** The appliance remained passive during the first 6 days. Tooth movement amounted to 1.1 mm during 14 days started after a layer of gutta-percha had been added. **C,** A cell-free area existed from the fifth to the eleventh day.

Light and transmission electron microscopy have shown that root resorption occurs near the hyalinized zone in close proximity to a rich vascular network.[10] This has been verified by Brudvik and Rygh,[18,19] who showed occurrence of small lacunae in the cementum at the coronal and apical peripheries of the hyalinized zone (Fig. 3-21). Their results indicated an association between root resorption and active removal of the hyalinized necrotic tissue. The first sign of root resorption (*initial phase*) was defined as a penetration of cells from the periphery of the necrotic tissue where mononucleated fibroblast-like cells, stained negatively by tartrate-resistant acid phosphatase (TRAP), started removing the precementum/cementum surface. Root resorption beneath the main hyalinized zone occurred in a *later phase* during which multinucleated TRAP-positive cells were involved in removing the main mass of necrotic PDL tissue and resorbing the outer layer of the root cementum. Further studies indicated that after the multinucleated TRAP-positive cells reached the subjacent contaminated and damaged root surface and removed the necrotic periodontal tissue, they continued to remove the cementum surface.

When the movement is discontinued, *repair* of the resorbed lacunae occurs, starting from the periphery (Fig. 3-22). After the force has terminated, active root resorption by TRAP-positive cells in the resorption lacunae still was observed in areas where hyalinized tissue existed (Fig. 3-23).[20,21] After the hyalinized tissue was eliminated, fibroblast-like cells invaded the active resorption site. After termination of force and in the absence of hyalinized necrotic tissue in the PDL, repair on the resorption lacunae occurred. The first sign was synthesis of collagenous fibrillar material by fibroblast- and cementoblast-like cells, followed by reestablishment of the new PDL. Further studies are needed, however, to fully clarify the factors leading to transition of an active process of resorption into one of repair.

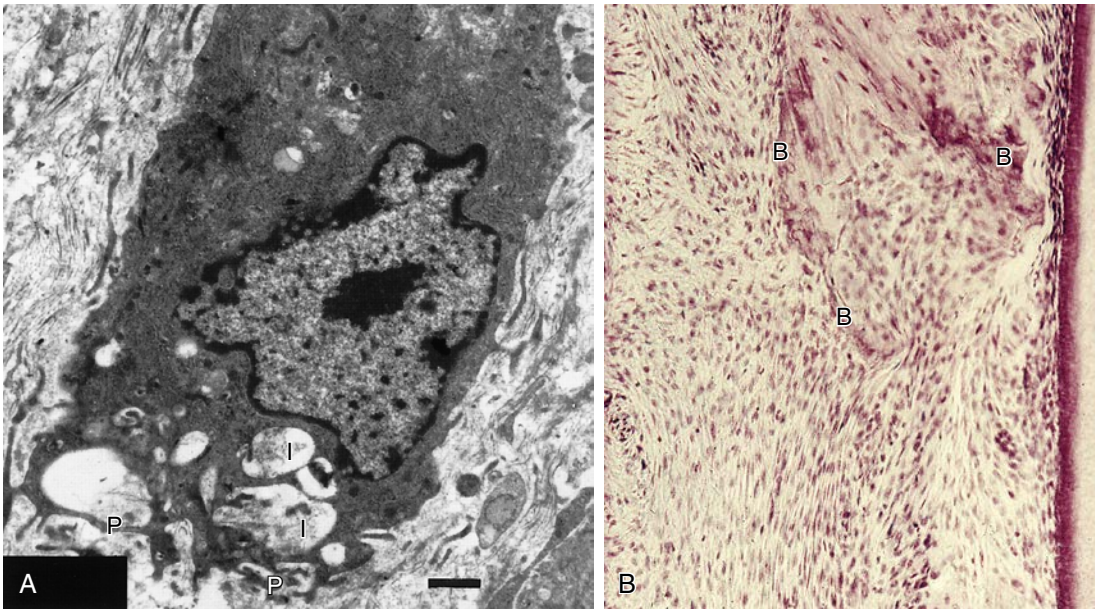


FIGURE 3-18 **A,** Pressure area in the PDL (×10,000; bar, 1 μm). Macrophage adjacent to hyalinized zone; pseudopodia (*P*) delimit inclusions (*I*) of phagocytosed material. (From Rygh P. Ultrastructural vascular changes in pressure zones of rat molar periodontium incident to orthodontic tooth movement. *Scand J Dent Res*. 1972;80:307.) **B,** Remaining bone areas (*B*) after the undermining resorption.

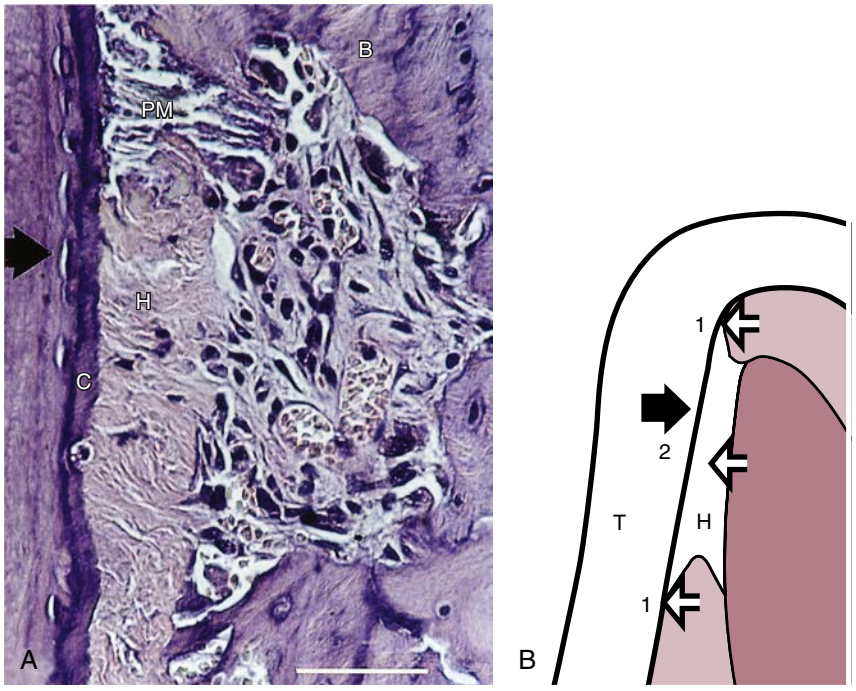


FIGURE 3-19 **A,** Migrating multinucleated cells in the middle of rat PDL close to remnants of hyalinized tissue (*H*). *B,* Bone; *PM,* periodontal membrane close to root cementum (*C*). **B,** Root resorption (*open arrows*) occurs first in the circumference of the hyalinized zone (*1*) and later in the central parts (*2*). *T*, Tooth; *H,* hyalinized zone; *filled arrow,* direction of tooth movement. (From Brudvik P, Rygh P. The initial phase of orthodontic root resorption incident to local compression of the PDL. *Eur J Orthod.* 1993;15:249.)

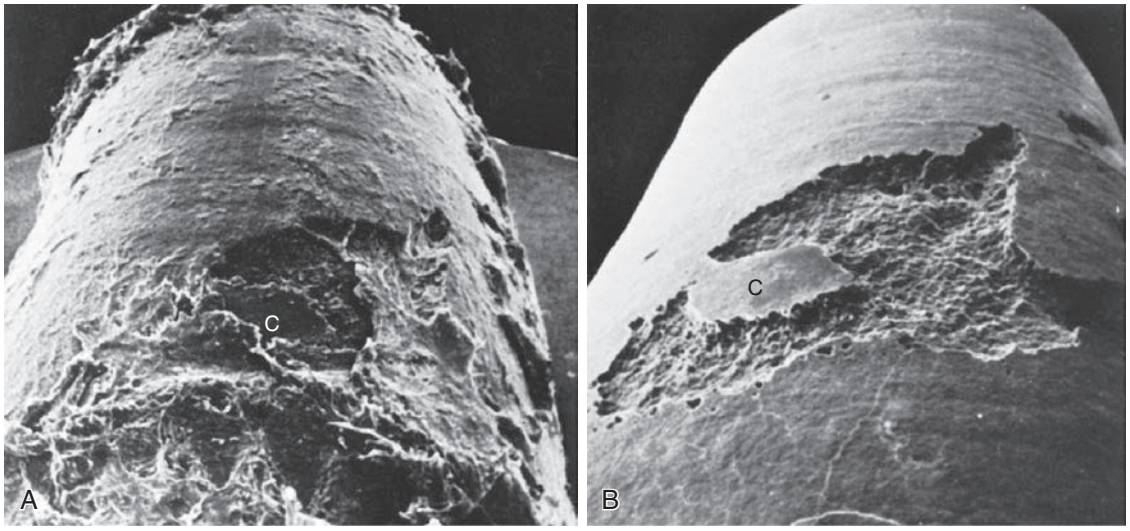


FIGURE 3-20 A, Resorbed lacunae in the middle third of a root as seen with the scanning electron microscope. *C,* Denuded root surface. Organic tissue components cover the major portion of the lacuna. B, Same root surface, previously covered by hyalinized tissue, after removal of organic tissue. (Courtesy E. Kvam.)

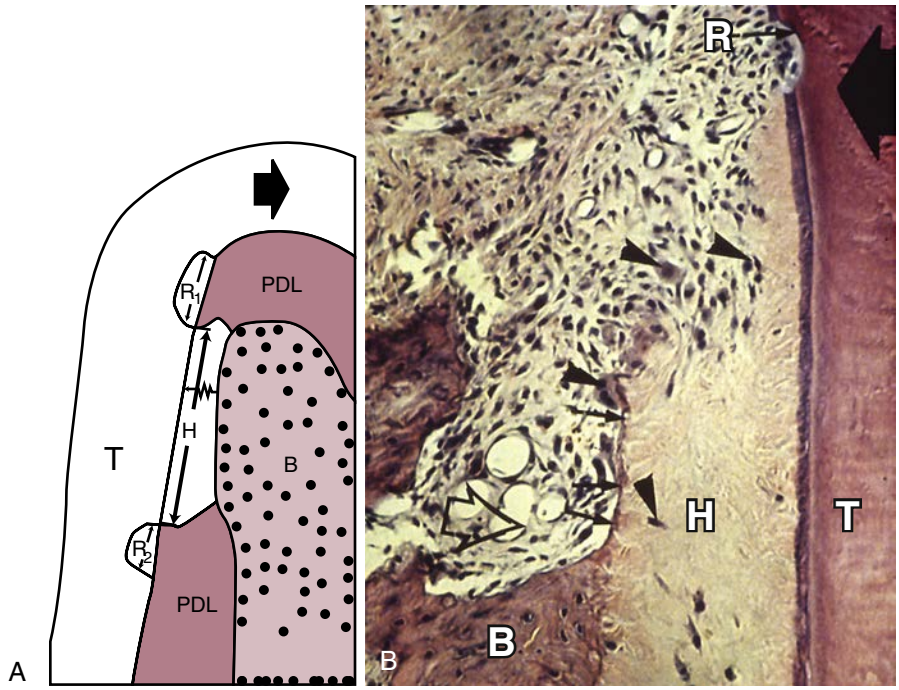


FIGURE 3-21 A, Schematic illustration and, B, photomicrograph of the hyalinized zone (*H*) between alveolar bone (*B*) and root surface (*T*). Resorption lacunae (R_1 and R_2) after tooth movement (*large arrows*) for 7 days in the mouse. Alveolar bone resorption (*open arrows*) occurs from marrow spaces; *small arrows* indicate thin line of bone between the resorbed bone and hyalinized tissue (bar is 50 μm). Resorption lacunae (*R*) with TRAP-negative cells; TRAP-positive cells (*arrowheads*) at the margin of and in the main hyalinized tissue. (Adapted from Brudvik P, Rygh P. The initial phase of orthodontic root resorption incident to local compression of the PDL. *Eur J Orthod*. 1993;15:249; Brudvik P, Rygh P. Root resorption beneath the main hyalinized zone. *Eur J Orthod.* 1994;16:249)

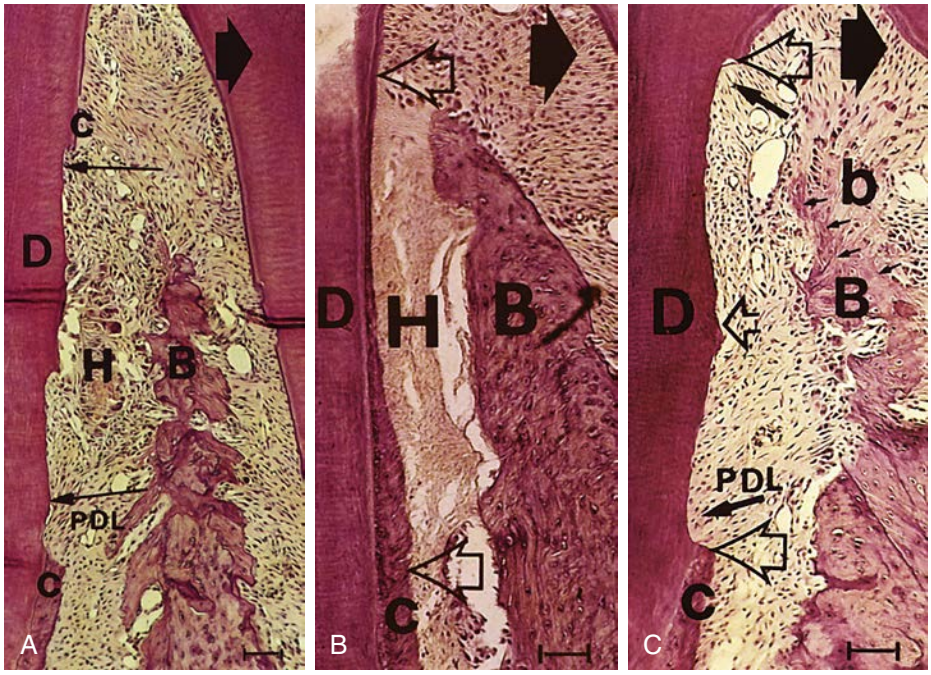


FIGURE 3-22 Photomicrographs of the root resorption and repair sequence of the compressed PDL 21 days after tooth movement in the direction of large arrows. **A,** Neighboring section with repair of peripheral parts of resorption lacunae by deposition of cementum (*arrows*). **B,** The length (between two *open arrows*) of the hyalinized zone after 3 days of compression. **C,** The length (between two *large open arrows*) of the resorbed root surface after 21 days of tooth movement. Note the regular arrangement of the PDL cells and fibers in the peripheral part of the resorption lacunae (*medium arrows*). In the central part of resorption area (*medium open arrows*) no trace of fibers is connected with the root surface. Demarcation line (*small arrows*) between old bone (*B*) and new bone (*b*) in crest area. *C,* Cementum; *D,* dentin; *H,* hyalinized zone. (Adapted from Brudvik P, Rygh P. Transition and determinants of orthodontic root resorption-repair sequence. *Eur J Orthod.* 1995;17:177.)

A reduction of root resorption is likely provided by minor trauma and can be repaired during periods of no force or possibly during periods of extremely low force application (Fig. 3-24). Chapter 30 further discusses root resorption.

Secondary Period of Tooth Movement

In this period (Fig. 3-25), the PDL is considerably widened. The osteoclasts attack the bone surface over a much wider area. As long as the force is kept within certain limits or gentle reactivation of the force is undertaken, further *bone resorption* is predominantly direct (Fig. 3-26). The fibrous attachment apparatus is reorganized by the production of new periodontal fibrils. When the application of a force is favorable, a large number of osteoclasts appear along the bone surface on the pressure side and tooth movement is rapid. Modern histologic techniques reveal that complete reorganization of the fibrous system takes place throughout the membrane.

The main feature is the *deposition of new bone* on the alveolar surface from which the tooth is moving away (tension side). Cell proliferation usually occurs after 30 to 40 hours in young human beings. The newly formed cells, osteoblasts with darkly stained nuclei, have a characteristic appearance (Fig. 3-27). Osteoblasts may be observed along stretched fiber bundles. Shortly after cell proliferation has started, osteoid tissue is deposited on the tension side. The formation of this new osteoid depends to some extent on the form and thickness of the fiber bundles. The original periodontal fibers become embedded in the new layers of prebone, or osteoid, which mineralizes in the deeper parts.[3]

New bone is deposited until the width of the membrane has returned to normal limits, and simultaneously the fibrous system is remodeled. The original stretched fibers are not broken down to the same extent as is the case on the pressure side, and the remodeling involves resorption and replacement of collagen, leading to a lengthening of the fibers, the mechanism of which is largely unknown.

Concomitantly with bone apposition on the periodontal surface on the tension side, an accompanying resorption process occurs on the spongiosa surface of the alveolar bone that tends to maintain the dimension of the supporting bone tissue (Fig. 3-28). Correspondingly, during the resorption of the alveolar bone on the pressure side, maintenance of the alveolar lamina thickness is ensured by apposition on the spongiosa surface. These processes are mediated by the cells of the endosteum, which cover all the internal bone surfaces and dental alveoli. Extensive remodeling takes place in the deeper, cell-rich layers of the periosteum, incident to the orthodontic forces, a reaction that tends to restore the thickness of the supporting bone.

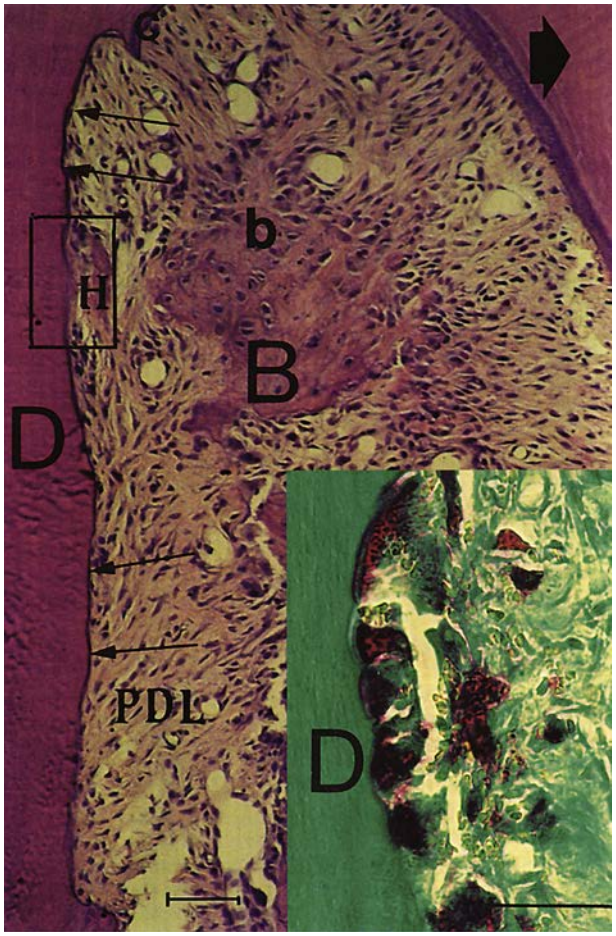


FIGURE 3-23 Repair of lacuna by cementum deposition in the periphery (*arrows*). Small amounts of hyalinized tissue (*H*) persist in boxed area, which shows active root resorption indicated by TRAP-positive cells in the lacuna (*bars* are 50 μm). The heavy arrow (*upper right*) indicates direction of force. (Adapted from Brudvik P, Rygh P. Transition and determinants of orthodontic root resorption-repair sequence. *Eur J Orthod.* 1995;17:177.)

The observation that orthodontic tooth movement involves many *inflammation-like* reactions is important in that this, in turn, has enhanced an understanding that the whole cascade of factors involved in inflammation may be part of the reactions to orthodontic forces in the tooth-supporting tissues: that is, extracellular breakdown of collagen by collagenases, produced by leukocyte–fibroblast interaction. The term *inflammation* should not be confused with the term *infection*, as is often the case in popular use. In orthodontics, inflammation is a process occurring in a local environment when a rapid response is needed for a stress that is felt transiently by the cells as being too heavy.

Types of Tooth Movements

The tissue response in different parts of the PDL is dependent on the type of tooth movement, which briefly will be exemplified.

Tipping

Tipping of a tooth leads to a concentration of pressure in limited areas of the PDL (Fig. 3-29, *A*). A fulcrum is formed, which enhances root movement in the opposite direction. A tipping movement, in general, results in the formation of a hyalinized zone slightly below the alveolar crest, particularly when the tooth has a short, undeveloped root. If the root is fully developed, the hyalinized zone is located a short distance from the alveolar crest (Fig. 3-29, *B*). Tipping of a tooth by light, continuous forces results in a greater movement within a shorter time than that obtained by any other method. In most young orthodontic patients, bone resorption resulting from a moderate tipping movement usually is followed by compensatory bone formation. The degree of such compensation varies individually and depends primarily on the presence of bone-forming osteoblasts in the periosteum. Compensatory periosteal bone apposition in the apical region is also subject to some variation, according to whether osteoblasts are present or absent in the periosteum (Fig. 3-30).

Torque

A torquing movement of a tooth involves tipping of the apex (Fig. 3-31, *A*). During the initial movement of torque, the

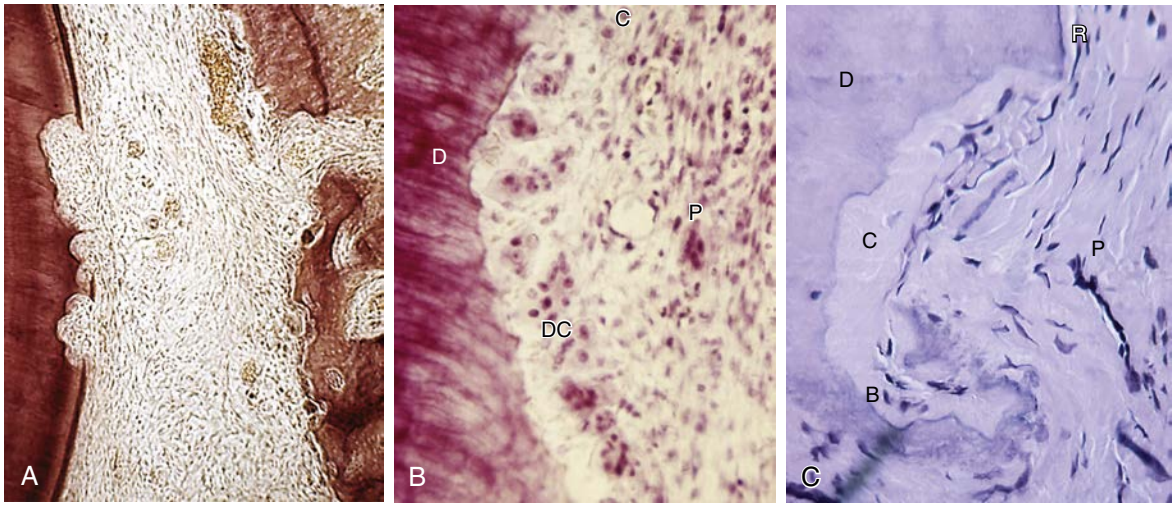


FIGURE 3-24 **A,** Superficial root resorption. **B,** Magnification of the resorption area in **A.** *D,* Dentin; *DC,* dentinoclasts. **C,** Repaired lacuna. *B,* Demarcation; *C,* secondary cementum; *D,* dentin; *P,* periodontal ligament.

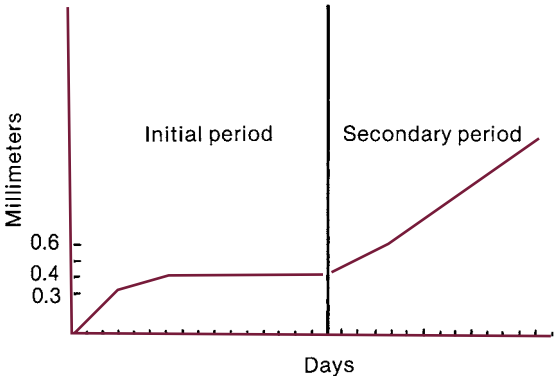


FIGURE 3-25 Degree of tooth movement before and after hyalinization. Tooth movement occurring after hyalinization is termed *secondary period.*

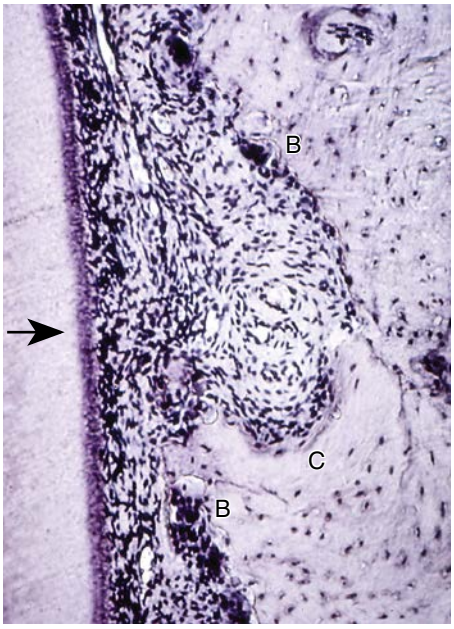


FIGURE 3-26 Effect on the pressure side of the upper premolar of a 12-year-old who wore a gradually expanded loose activator every night for 2 weeks. The root was moved as indicated by the *arrow*. The alveolar bone was bordered by an osteoid layer that persists. Extensive bone resorption has occurred in the area subjacent to this osteoid tissue. *B,* Bone surface lined with osteoclasts; *C,* persisting layer of osteoid. Note the increase in cellular elements.

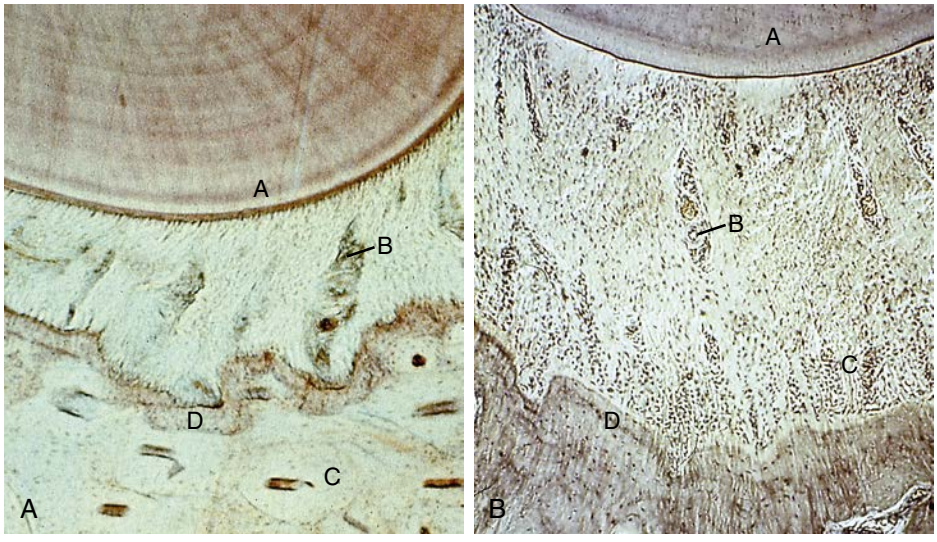


FIGURE 3-27 Animal experiment on initial movement of short duration in the upper central and lateral incisors. **A,** Control. *A,* Tooth root; *B,* interstitial space; *C,* bundle bone; *D,* undecalcified osteoid layer. **B,** Experimental second incisor, tension side (force, 45 cN; duration, 36 hours). Note the increase and spreading of new cells, particularly in areas close to the bone surface and adjacent to the stretched fiber bundles. *A,* Tooth root; *B,* interstitial space; *C,* proliferating osteoblasts between fiber bundles; *D,* increase in osteoid tissue.

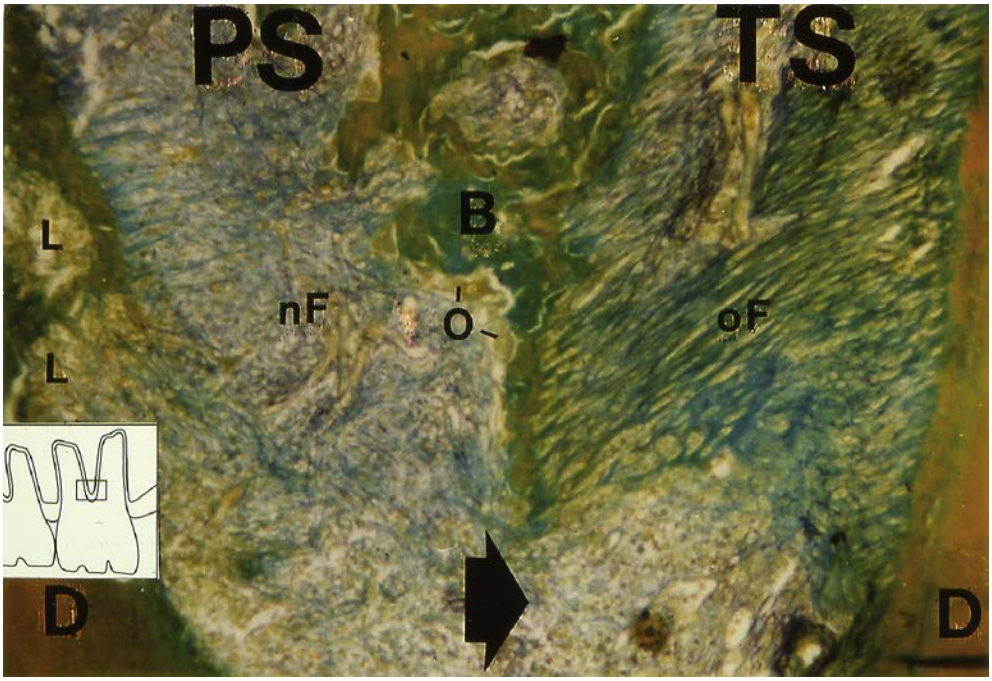


FIGURE 3-28 Experimental tooth movement (*arrow*) in the rat in the interradicular bone (*B*) and dentin (*D*). On the pressure side (*PS*), the bone is being actively resorbed by osteoclasts. Original fibers (*oF*) on the tension side (*TS*) are stretched and embedded in bone deposited on the alveolar surface. Note the complete breakdown of the original fibers and their replacement of new fibrils (*nF*) in the richly vascularized pressure side. *L,* Root resorption lacunae.

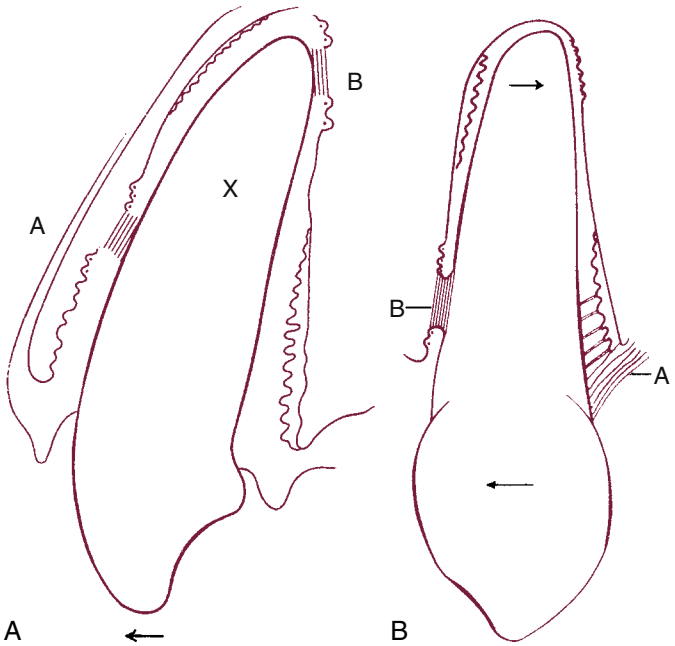


FIGURE 3-29 **A,** Location of the limited number of fiber bundles to resist movement during tipping. If the force is light, the hyalinization period will be short and the coronal portion will move quite readily. *A,* Supra-alveolar fibers; *B,* hyalinized zone on the pressure side. **B,** A prolonged tipping movement may result in formation of a secondary hyalinized zone (*A*) after the first hyalinized zone has been eliminated. Compression of the PDL is maintained in the apical region (*B*). *X,* Fulcrum.

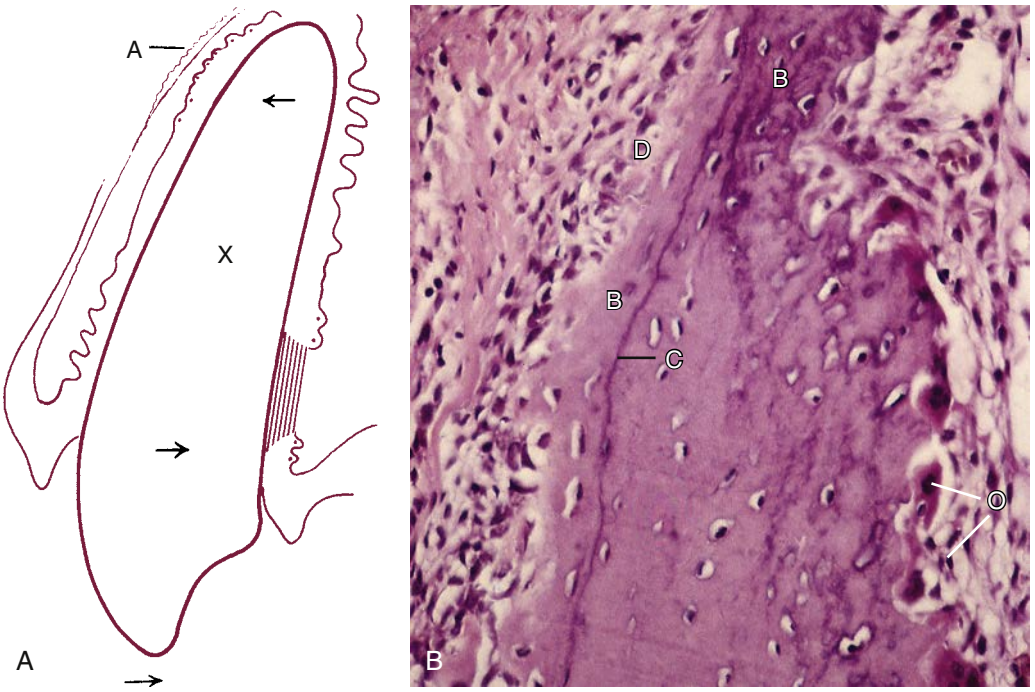


FIGURE 3-30 **A,** Compensatory bone formation on the periosteal side after tipping of an upper second incisor in the dog. *X,* Fulcrum. **B,** Corresponds to area *A*. *B,* Bone formation in formerly resorbed lacunae; *C,* resting line indicating thickness of the compensatory bone layer; *D,* osteoblast chain of the periosteum covering new partly calcified bone layer; *O,* osteoclasts on the bone surface.

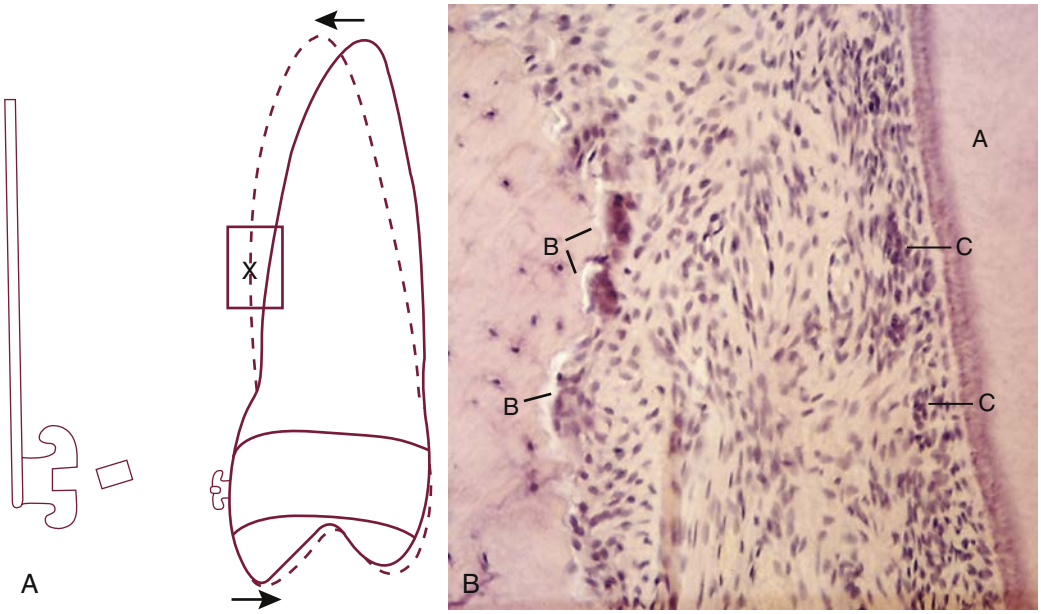


FIGURE 3-31 **A,** Torquing of an upper premolar. **B,** Pressure side in a 12-year-old patient after buccal root torquing movement performed in the apical region with a 0.021- × 0.025-inch edgewise arch (area *X* in **A**) (force, 120 to 130 cN; duration, 2 weeks). As indicated by the presence of epithelial remnants, no hyalinization of the PDL has occurred. *A,* Root surface; *B,* osteoclasts along the bone surface; *C,* epithelial remnants.

pressure area usually is located close to the middle region of the root. This occurs because the PDL is normally wider in the apical third than in the middle third. After resorption of bone areas corresponding to the middle third, the apical surface of the root gradually begins to compress adjacent periodontal fibers, and a wider pressure area is established (Fig. 3-31, *B*). However, if more torque is incorporated in the archwire, the force will increase and may result in resorption and fenestration of the buccal bone plate (Fig. 3-32).

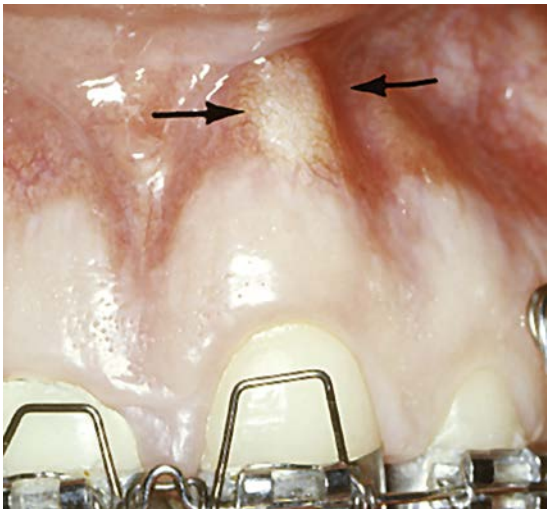


FIGURE 3-32 Bone fenestration at the left central and lateral incisors (*arrows*) after labial root torque movements.

Bodily Movement

Bodily tooth movement is obtained by establishing force couples acting along parallel lines and distributing the force over the whole alveolar bone surface. This is a favorable method of displacement, provided that the magnitude of force does not exceed a certain limit.[22]

Application of a light, continuous force on premolars in a dog resulted in small compressed areas shortly after the movement began, with a hyalinized zone of short duration usually located as seen in Figure 3-33, *A*. No bodily movement of the tooth in a mechanical sense is observed during this period; instead, a slight tipping is noticed. The degree of initial tipping varies according to the size of the arch and the width of the brackets.

The short duration of the hyalinization results from an increased bone resorption on both sides of the hyalinized tissue, especially in the apical region of the pressure side. This leads to rapid elimination of the hyalinized zone. This favorable reaction on the pressure side is caused partly by gradually increased stretching of fiber bundles on the tension side, which tends to prevent the tooth from further tipping (Fig. 3-33, *B*). New bone layers are formed on the tension side along these fiber bundles (Fig. 3-33, *C*).

Rotation

Rotation of a tooth creates two pressure sides and two tension sides (Fig. 3-34, *A*) and may cause certain variations in the type of tissue response observed on the pressure sides. Hyalinization and undermining bone resorption take place in one pressure zone, while direct bone resorption occurs in the other. These variations are caused chiefly by the anatomy of the tooth and the magnitude of the force. As in other types of tooth movements, applying a

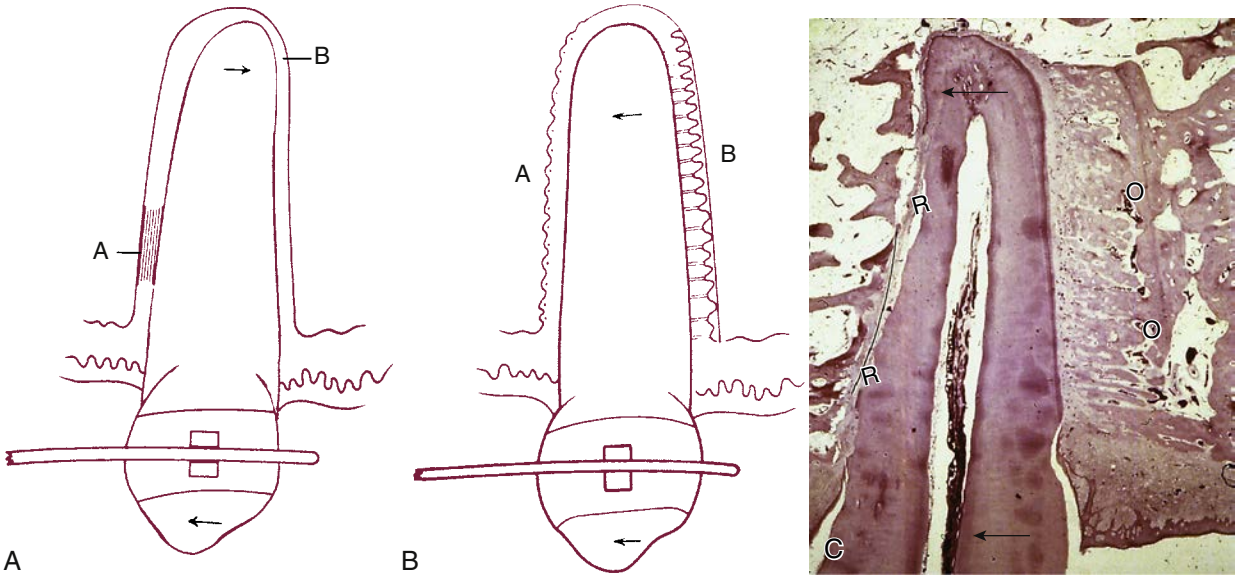


FIGURE 3-33 Two stages of bodily tooth movement. **A,** Effect observed during the initial stage of a continuous bodily tooth movement. *A,* Hyalinized tissue; *B,* slight initial compression as a result of tipping of the tooth. **B,** Undermining bone resorption terminated. Gradual upright positioning of the tooth caused increased bone resorption adjacent to the middle and apical thirds of the root. Further movement is largely controlled by stretched fiber bundles. *A,* Bone resorption on the pressure side; *B,* Bone deposition along the stretched fiber bundles. The arrows denote direction of tooth movements. **C,** Bodily movement(*arrow*) of a premolar in a dog during a period of 6 months. New bone layers on the tension side with osteoblasts (*O*); root resorption on the pressure side (*R*).

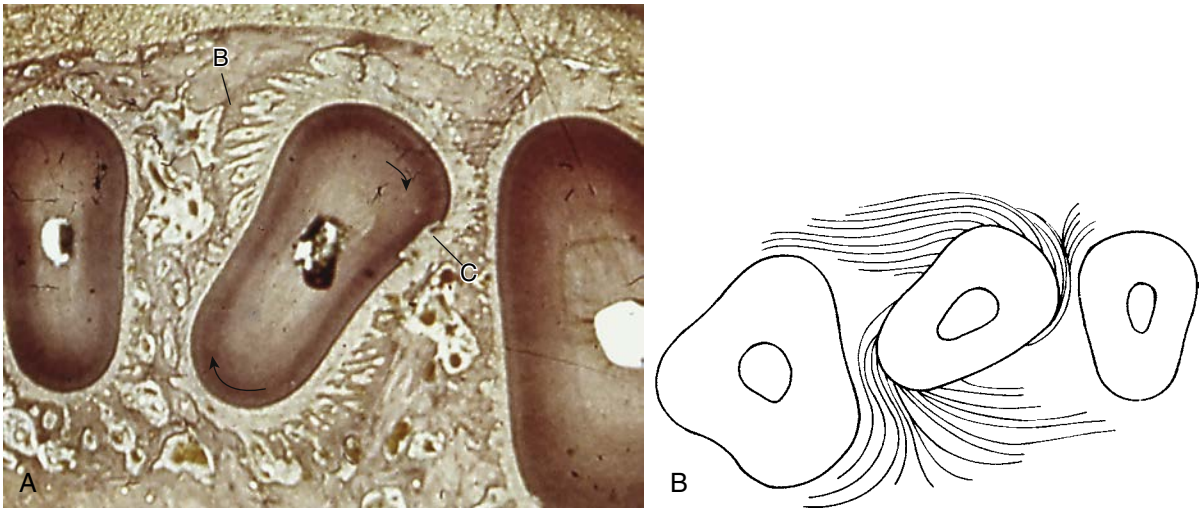


FIGURE 3-34 A, Experimental rotation of an upper second incisor in a dog. Formation of two pressure sides and two tension sides. *B,* Demarcation line between old and new bone layers; *C,* pressure side with root resorption. B, Arrangement of free gingival fibers after rotation.

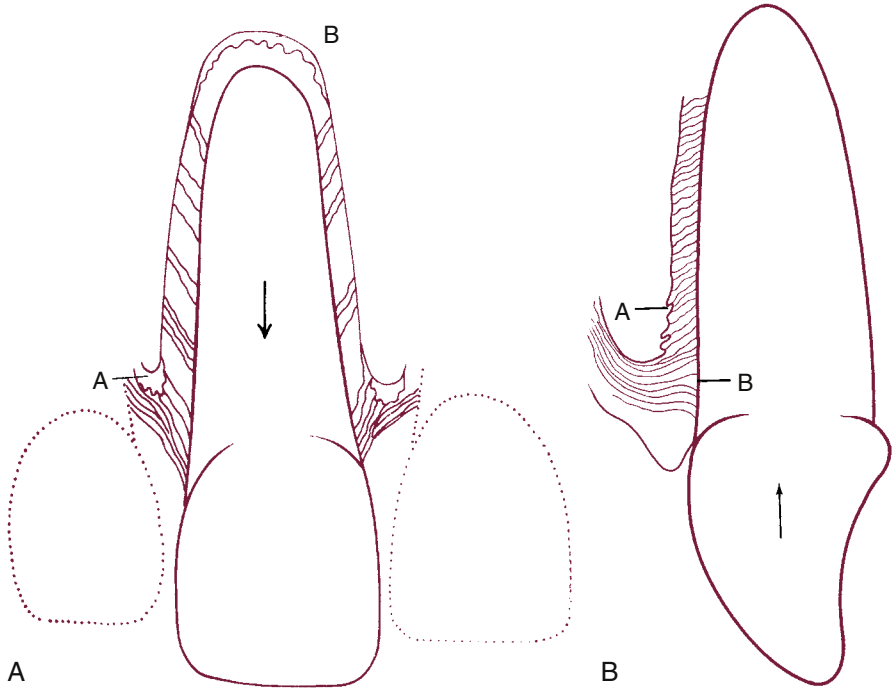


FIGURE 3-35 A, Arrangement of fiber bundles during or after *extrusion* of an upper central incisor (*arrow*). *A,* Extrusive tension results in added bone at alveolar crest. *B,* New bone layers at the alveolar fundus. B, Relaxation of the free gingival fibers during *intrusion* (*arrow*). *A,* Bone spicules laid down according to the direction of the fiber tension; *B,* relaxed supraalveolar tissue.

light force during the initial period is favorable. On the tension side, new bone spicules are formed along stretched fiber bundles arranged more or less obliquely. In the marginal region, rotation usually causes considerable displacement of fibrous structures. The free gingival fiber groups are arranged obliquely from the root surface. Because these fiber bundles interlace with the periosteal structures and the whole supraalveolar fibrous system, rotation also causes displacement of the fibrous tissue located some distance from the rotated tooth (Fig. 3-34, *B*).

Extrusion

Extrusive movements ideally produce no areas of compression within the PDL, only tension. Even if compressed areas could be avoided, heavy forces risk "extraction" of the tooth. Light forces, however, move the alveolar bone with the tooth.

Varying with the individual tissue reaction, the periodontal fiber bundles elongate and new bone is deposited in areas of alveolar crest as a result of the tension exerted by these stretched fiber bundles (Fig. 3-35, *A*). In young individuals, extrusion of

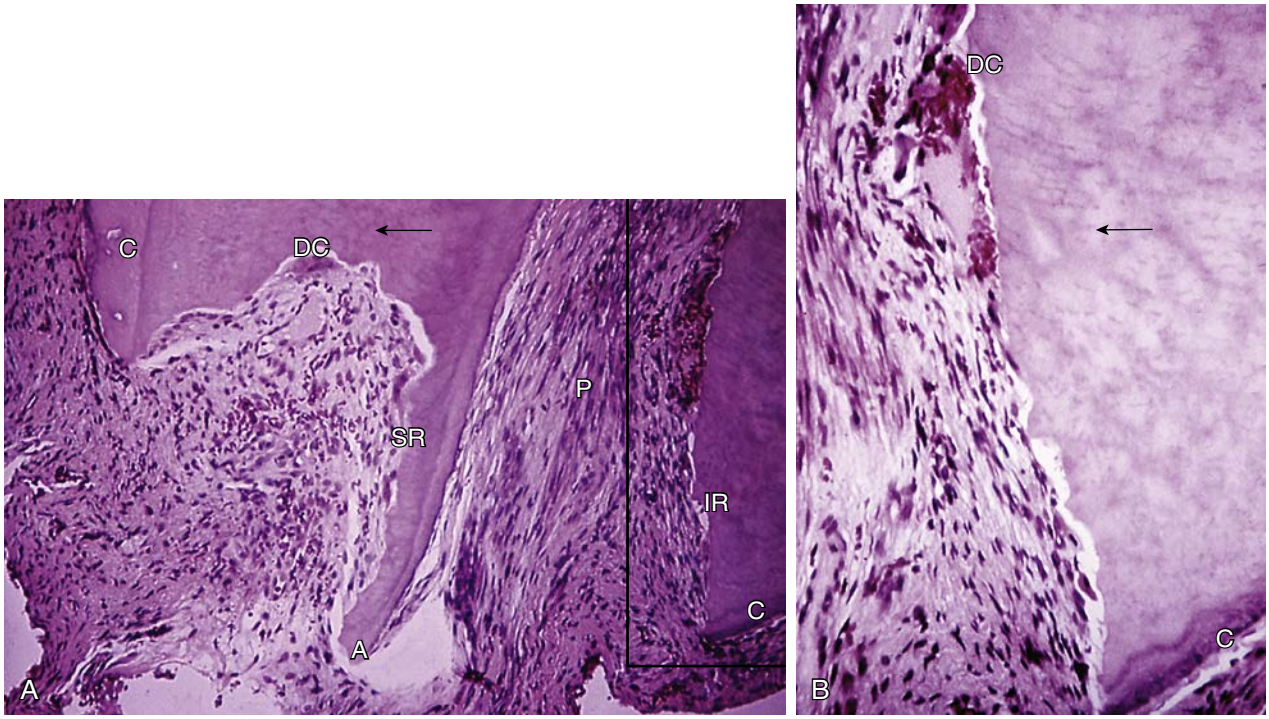


FIGURE 3-36 Apical portion of a lower first premolar tipped and intruded by a force of 100 cN for 35 days. **A,** *C,* Well-calcified cementum; *SR,* apical side resorption; *DC,* dentinoclast; *A,* remaining area of the apex, a detail of importance during reconstruction; *IR,* internal apical resorption; *P,* pulp tissue. **B,** Boxed area in **A.** *Arrows* indicate direction of applied force.

a tooth involves a more prolonged stretch and displacement of the supraalveolar fiber bundles than of the principal fibers of the middle and apical thirds. Some of the fibers may be subjected to stretch for a certain time during the tooth movement, but they will be rearranged after a fairly short retention period. Only the supraalveolar fiber bundles remain stretched for a longer time.

Intrusion

Unlike extruded teeth, intruded teeth in young patients undergo only minor positional changes after treatment. Relapse usually does not occur, partly because the free gingival fiber bundles become slightly relaxed (Fig. 3-35, *B*). Stretch is exerted primarily on the principal fibers. An intruding movement may therefore cause formation of new bone spicules in the marginal region. These new bone layers occasionally become slightly curved as a result of the tension exerted by stretched fiber bundles. Such tension also occurs in the middle third of the roots. Rearrangement of the principal fibers occurs after a retention period of a few months.

Intrusion requires careful control of force magnitude. Light force is required because the force is concentrated in a small area at the tooth apex. A light continuous force, such as that obtained in the light wire technique, has proved favorable for intrusion in young patients. In other cases the alveolar bone may be closer to the apex, increasing the risk for apical root resorption (Fig. 3-36). If the bone of the apical region is fairly compact, as it is in some adults, a light interrupted force may be preferable to provide time for cell proliferation to start, and direct bone resorption may prevail when the arch is reactivated after the rest period. Intrusion may also cause changes in the pulp tissue such as vascularization of the odontoblast and pulpal edema.[23,24]

Movements in the Labial/Buccal Direction

Heavy forces in the labial and buccal direction may result in alveolar bone dehiscence. Experiments in monkeys and beagle dogs have shown that "predisposing" alveolar bone dehiscences may be induced by uncontrolled labial expansion of teeth through the cortical plate, thereby rendering the teeth liable to the development of soft tissue recession (Fig. 3-37).[25-28] Of interest to note from those studies is that labial bone reforms in the area of dehiscence with intact epithelial junction (JE) when the tooth is retracted toward a proper positioning of the root within the alveolar process (Fig. 3-38).[27,28]

An experimental study in monkeys[29] in which teeth were moved orthodontically in a labial direction into areas with varying thickness and quality of the marginal soft tissue showed that a thin gingiva may serve as a locus minoris resistentiae to developing soft tissue defects in the presence of plaque-induced inflammation or toothbrushing trauma (Fig. 3-39).

The *clinical implication* of the results from the studies discussed is that careful examination of the dimensions of the tissues covering the facial aspect of the teeth to be moved should precede labial tooth movement. As long as the tooth can be moved within the envelope of the alveolar process, the risk of harmful side effects in the marginal tissue is minimal, regardless of the dimensions and quality of the soft tissue.

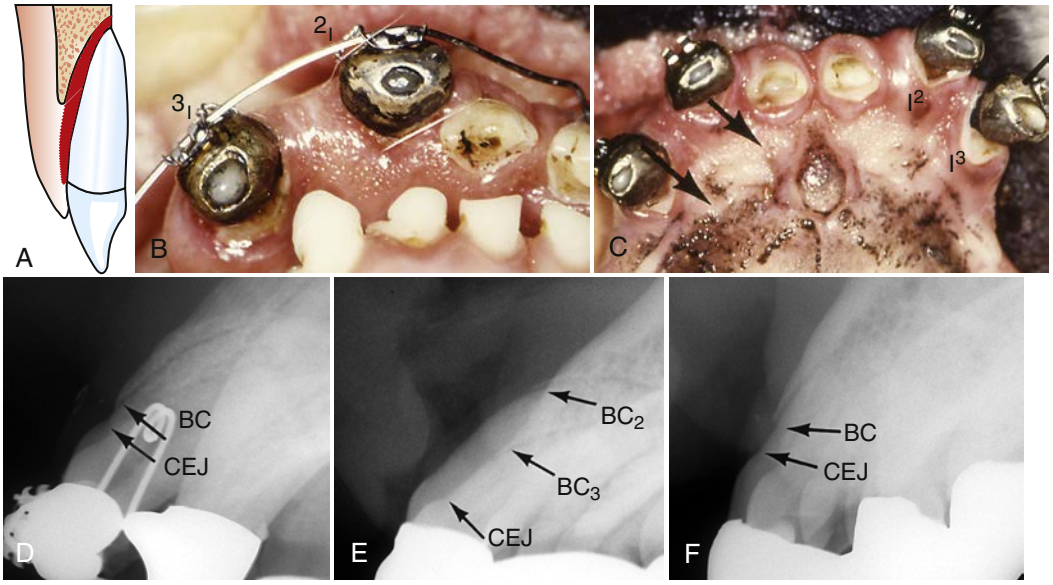


FIGURE 3-37 Bone dehiscences related to orthodontic tooth movement in a dog. **B,** The incisors 2_I and 3_I have been displaced through the alveolar bone plate to the level shown in **A**. **C,** The incisors I^2 and I^3 have been moved back to their original position; simultaneously I^2 and I^3 are displaced labially (days 150–300). Radiographs showing the buccal alveolar crest (*BC*) and the cementoenamel junction (*CEJ*) before **(D)** and after **(E)** facial displacement (day 150). BC_2 and BC_3 represent the buccal alveolar crest of I^2 and I^3. **F,** Radiograph showing reestablished bone height (*BC*) in the tooth moved back to its original position. (Adapted from Thilander B, et al. Bone regeneration in alveolar bone dehiscences related to orthodontic tooth movements. *Eur J Orthod.* 1983;5:105.)

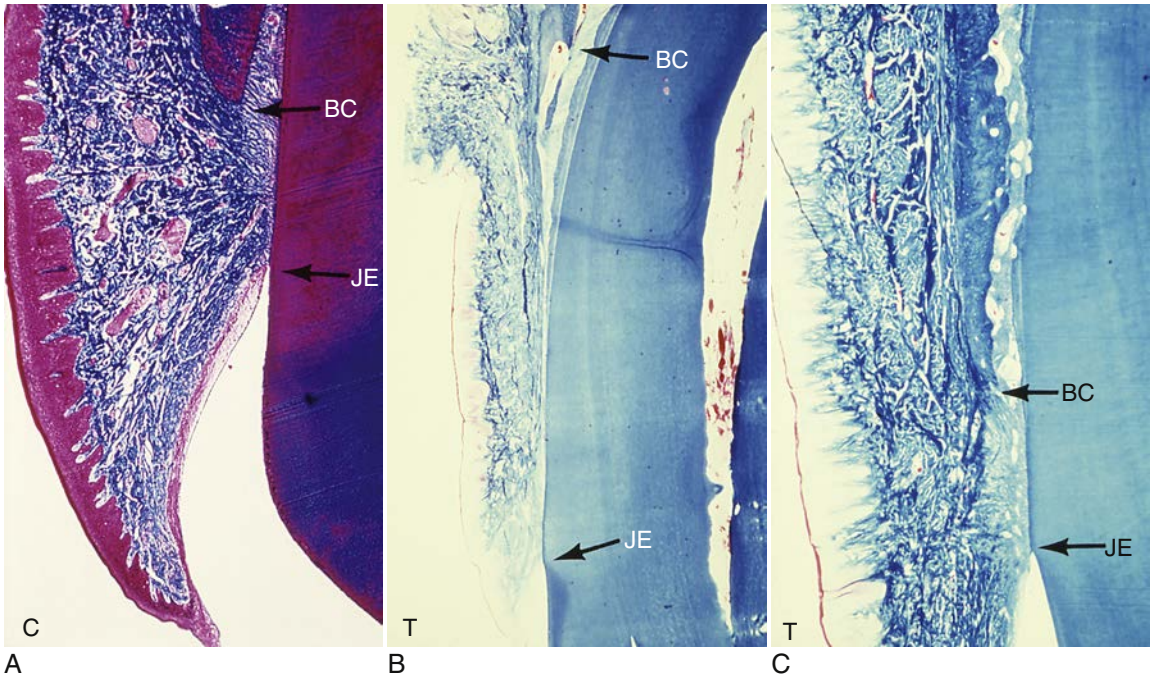


FIGURE 3-38 Histologic specimens of **(A)**, a control tooth (*C*) that was not moved and **(B** and **C)** a test tooth (*T*) first moved labially and then back again for a total of 300 days. Note the same distance between the buccal alveolar crest (*BC*) and the epithelial junction (*JE*) in the test and control teeth. (Adapted from Thilander B, et al. Bone regeneration in alveolar bone dehiscences related to orthodontic tooth movements. *Eur J Orthod.* 1983;5:105.)

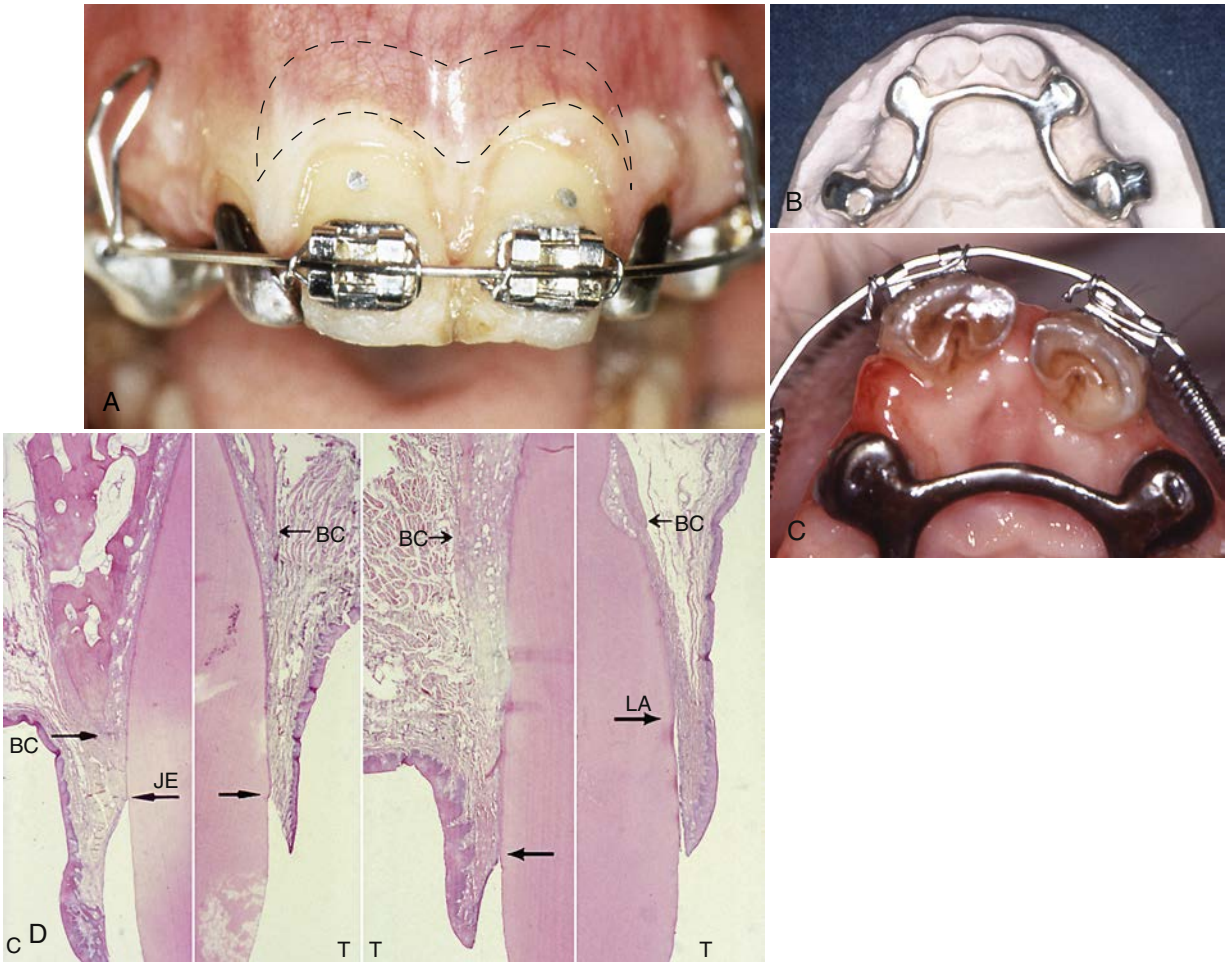


FIGURE 3-39 Incisors moved into areas with various thickness of gingiva. **A,** The volume of the keratinized gingival was surgically reduced to different degrees on left and right sides of the incisors. **B,** Orthodontic appliance used for labial displacement. **C,** The amount of labial tooth movement. **D,** Histologic specimens showing normal bone height and soft tissue margin at a control tooth (*C*) but reduced bone height and soft tissue margin at three test teeth (*T*). *JE*, Connective tissue attachment; *BC,* bone crest; *LA*, loss of attachment. (From Wennström J, et al. Some periodontal tissue reactions to orthodontic tooth movement in monkeys. *J Clin Periodontol.* 1987;14:121.)

Movements into Reduced Alveolar Bone Height

In patients with *partially edentulous dentitions* due to congenital absence or extraction of teeth, orthodontic treatment often is necessary. By positioning the teeth toward or into the edentulous area, improved aesthetics and functional results may be gained. Many of these individuals experience a *reduced alveolar bone* height.

Do tissue changes on the pressure side allow the tooth to move into such an area? An experimental study in the beagle[30] (Fig. 3-40) has shown that a tooth with normal periodontal support can be moved orthodontically into an area of reduced bone height with maintained height of the supporting apparatus. On the pressure side, supporting alveolar bone was also present, extending far coronal to the surrounding, experimentally created bone level (Fig. 3-41). The histologic picture of the bone tissue in the coronal portion of the root showed a large number of cells in contrast to the compact appearance of the more apically located bone, as in the unmoved tooth.

An interesting finding was that the newly formed bone on the pressure side showed resorption on the surface near the root and apposition on the opposite side of the thin bone plate (see Fig. 3-41, *C*), which was interpreted as a piezoelectric effect through strain-generated potentials, arising as a result of mechanically induced deformation of collagen or hydroxyapatite crystals.[31]

This experimental model has been tested in partially edentulous patients with normal periodontal tissue support located adjacent to an area with reduced bone volume.[32] Teeth have been moved orthodontically between 5 and 8 mm into such an area with maintained support and used as an abutment for a fixed prosthetic denture (Fig. 3-42). A dental implant placed in the created space showed excellent bone integration (Fig. 3-43). The results of these clinical follow-up studies are encouraging, as long as bodily tooth movements with light orthodontic forces are used. How far a tooth can be displaced into such an area can only be speculated.

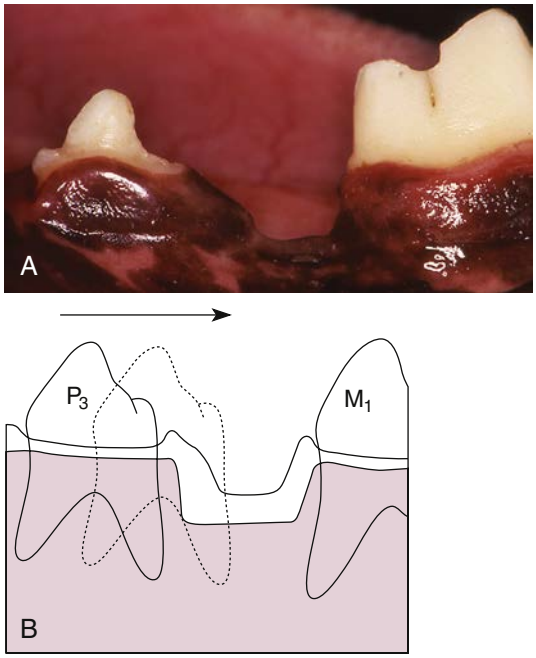


FIGURE 3-40 Clinical photograph **(A)** and schematic drawing **(B)** illustrating the bodily movement of the third premolar in the dog into an area with reduced alveolar bone height. (From Lindskog-Stokland B, et al. Orthodontic tooth movement into edentulous areas with reduced bone height. An experimental study in the dog. *Eur J Orthod.* 1993;15:89.)

Transmission of Orthodontic Forces into a Cellular Reaction

The old observation by Reitan illustrates well one of nature's wonders (Fig. 3-44).[7] The tooth is being moved in the direction of the arrow. Resorption of the alveolar bone wall has occurred. Fatty bone marrow has been changed into red bone marrow. At a little distance behind the alveolar bone wall, a new bony lamella is laid. How do the different cells know that they should react and in a special way? Tooth movement involves cell signaling leading to remodeling of the periodontal tissues,[31-39] which is described in detail in the next section of this chapter and begins on page 85.

TISSUE RESPONSE IN SUTURES

The effect of mechanical forces on the craniofacial sutures has been well described in the literature. Controlled forces generated by various types of headgear and appliances for palatal expansion have shown measurable changes of the craniofacial morphology. The changes are, however, of different degrees, and contradictory findings regarding long-term stability have been reported. One explanation of these different opinions is that the position, orientation, and even numbers of facial sutures seem to be closely oriented with respect to various forces (Fig. 3-45).

The possibility of influencing the basal parts of the nasomaxillary complex is an open suture before any bony union (synostosis) has been established. However, different opinions about the age of human suture closure are prevalent, and the mechanism of physiologic fusion of sutures is still obscure. Histologic studies

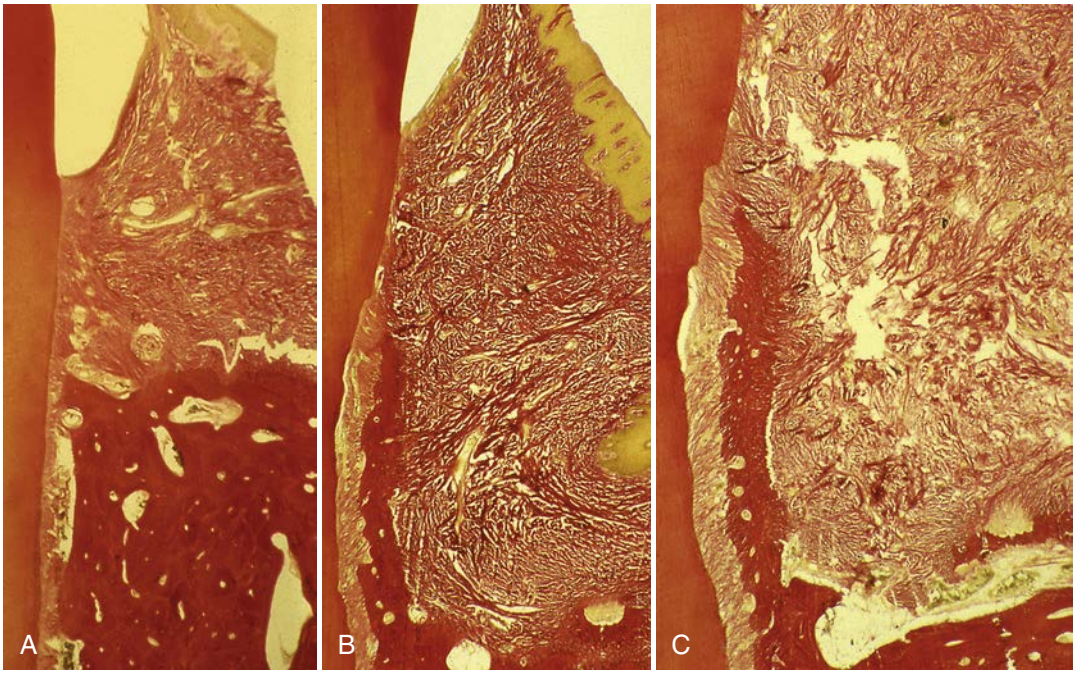


FIGURE 3-41 Specimens from a control (unmoved tooth) **(A)** and a tooth moved as shown in Figure 3-40 **(B). C,** The bone tissue in the coronal part of the root showed a large number of cells in contrast to the compact appearance of the more apically located bone. (From Lindskog-Stokland B, et al. Orthodontic tooth movement into edentulous areas with reduced bone height. An experimental study in the dog. *Eur J Orthod.* 1993;15:89.)

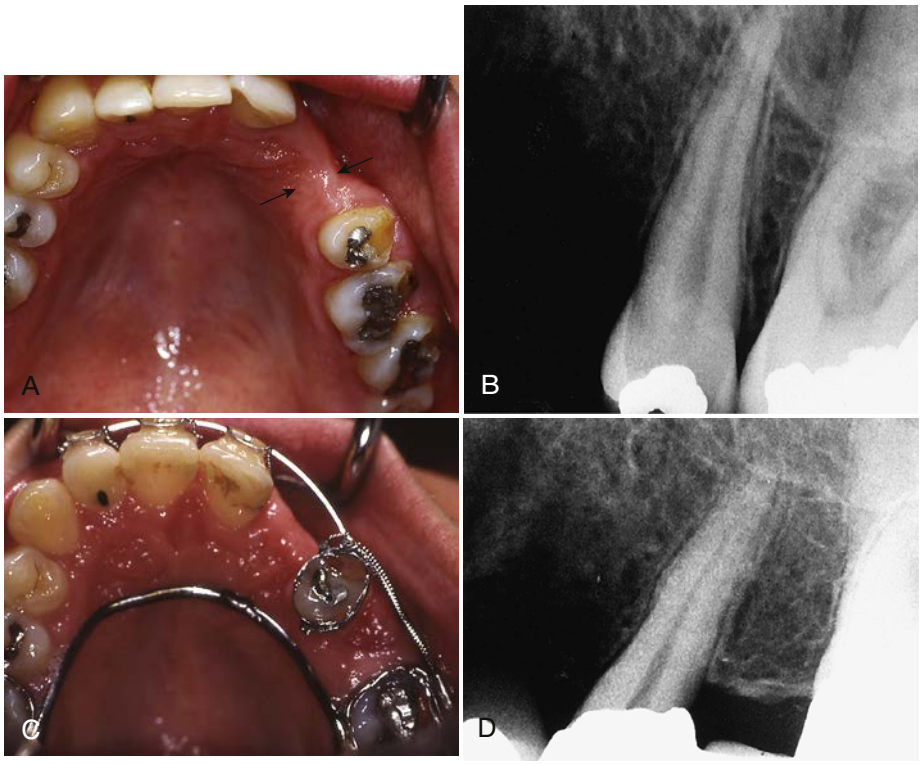


FIGURE 3-42 A 25-year-old man who is partially edentulous because of trauma. **A, B,** Marked bone loss (*arrows*) in the left maxillary alveolar process. **C,** The second premolar was moved bodily one cusp width into the area with reduced bone support. **D,** After a treatment period of 12 months, a prosthetic bridge was constructed. Note the regaining of alveolar bone. (From Thilander B. Infrabony pockets and reduced alveolar bone height in relation to orthodontic treatment, *Semin Orthod.* 1996;2:55.)

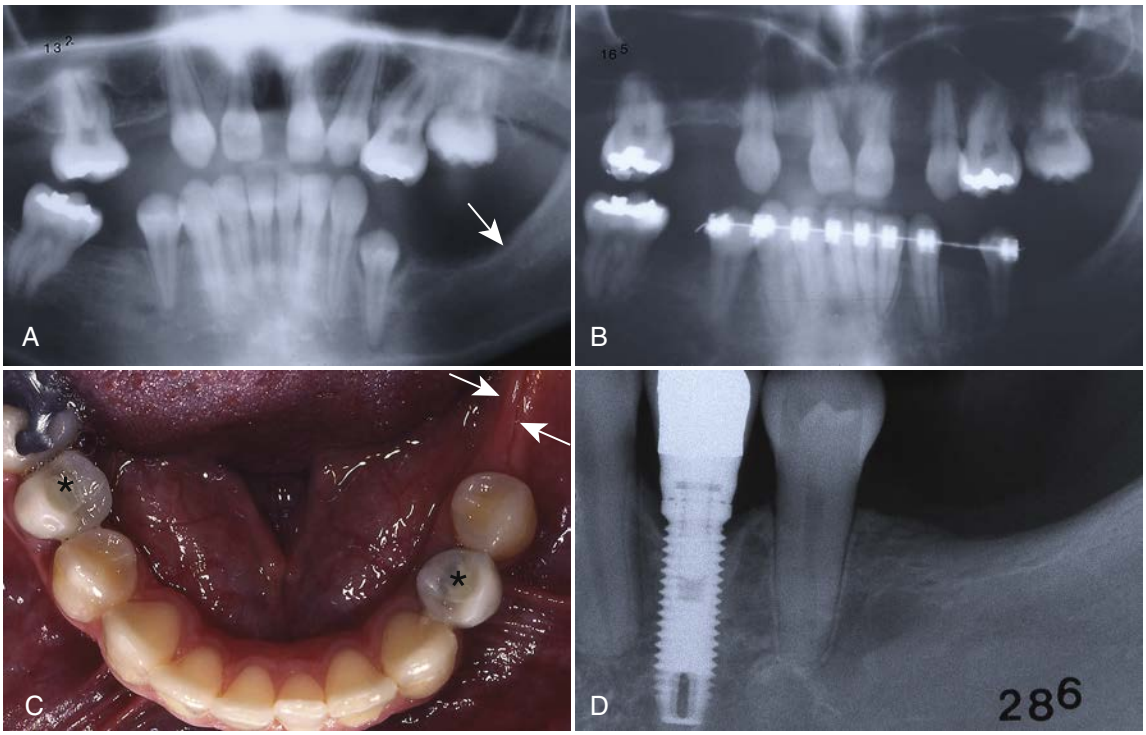


FIGURE 3-43 **A,** Radiograph of a 13-year-old girl with 12 congenitally missing teeth. In the mandible, it was decided to align the dental arch and to place an implant in the right premolar region. On the left side, implant placement was contraindicated because of thin bone volume in the buccolingual direction. **B,** Instead, the first premolar was moved one cusp width distally, and an implant was placed in the created space at age 17 years. **C,** Note the thin buccolingual bone volume in the lower left side (*arrows*) posterior to the distalized premolar. The implant-supported crowns are indicated by asterisks. **D,** At the last observation, a good result was registered with excellent bone integration of the implants. (From Thilander B, et al. Orthodontic aspects on the use of oral implants in adolescents: a 10-year follow-up study. *Eur J Orthod.* 2001;23:715.)

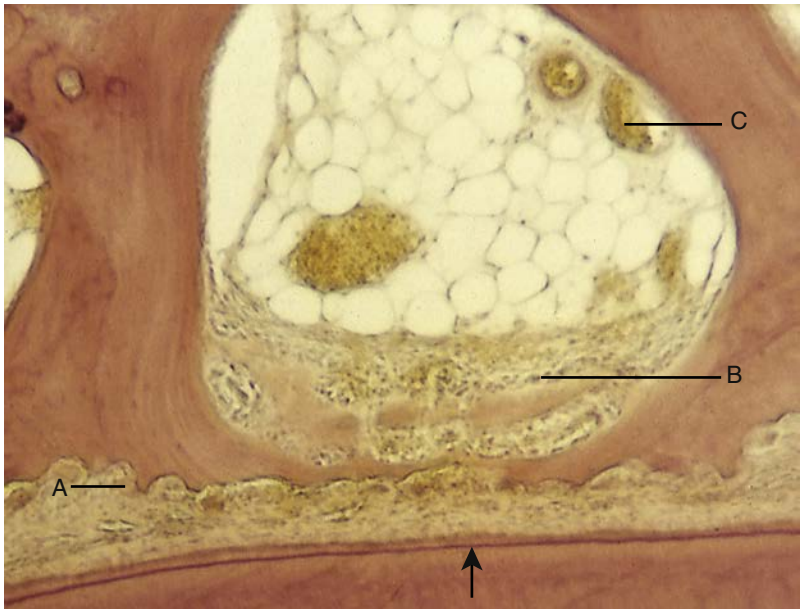


FIGURE 3-44 Bodily movement of an upper second incisor in a dog. The *arrow* indicates the tooth and direction of movement. The root was moved against a space containing fatty marrow, which was transformed into loose, fibrous tissue in advance of the movement. The periodontal space is narrower in this area because the uncalcified bone layer along the inside of the marrow space is less readily resorbed by osteoclasts. *A,* Direct bone resorption, one of the many Howship lacunae with osteoclasts; *B,* compensatory bone formation; osteoid layer bordered by osteoblasts; *C,* capillary surrounded by fatty marrow.

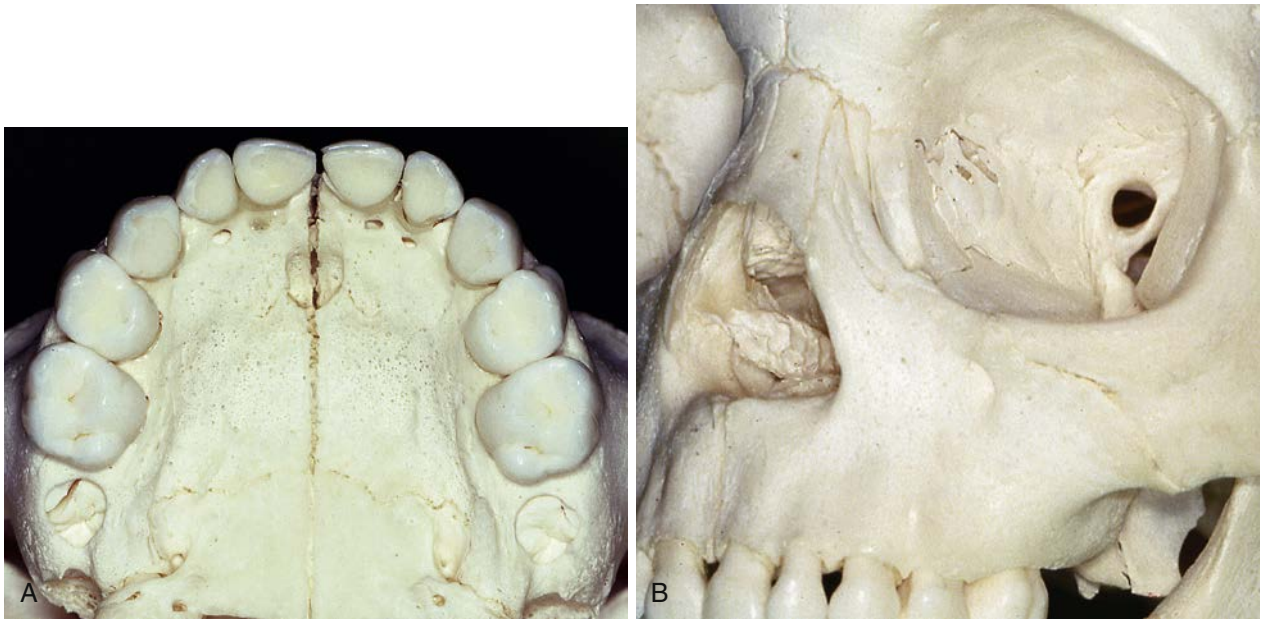


FIGURE 3-45 Cranium showing the sutural system of the maxilla.

on human maxillary sutures[40,41] have shown that palatal suture closure usually starts during the third decade of life, although with large individual variations. After adolescence, closure was observed in most specimens, indicating a rapid increase in fusion during the third decade. Based on microradiograms of the frontozygomatic suture in humans, Kokich[42] concluded that most facial sutures remain visible until the eighth decade of life.

Structure of the Suture

Sutures consist of a fibrous tissue with osteogenic layers on both surfaces (Fig. 3-46). Sutures therefore represent an extension of the periosteal layer of the bone and participate in the design of the bone by their remodeling capacity. The fiber system is mainly collagenous with a rapid synthesis and turnover. Elastic fibers are more frequent in sutures than in the PDL.

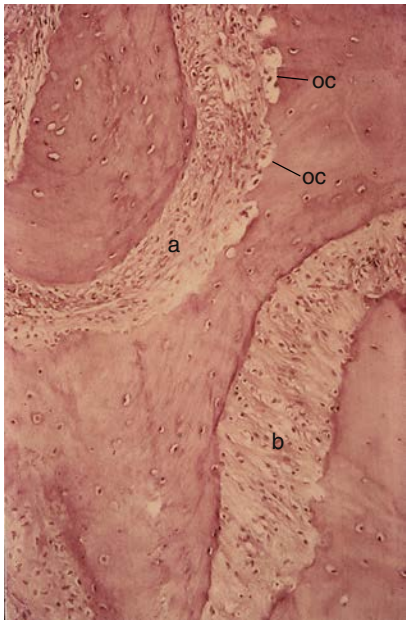


FIGURE 3-46 Photomicrograph of the intermaxillary suture of a 12-year-old boy. Variation in the fiber structure is evident; cells and fibers parallel (*a*) and perpendicular (*b*) to the bony surface. Note the osteoclasts (*oc*). (From Persson M, Magnusson B, Thilander B. Suture closure in rabbit and man: a morphological and histochemical study. *J Anat.* 1978;125:313.)

The maturing suture tissue in the growing individual demonstrates changes with age that eventually end with a bony obliteration of the sutural space (Fig. 3-47). The density and thickness of the fibrous component increase with age, and when growth ceases, bundles of fibers can be seen running transversely across the suture, increasing the mechanical strength of the joint. Osteogenesis tends to be restricted to areas of transversely densely arranged collagen bundles, a preliminary stage to bony bridgings.[43] Because of the serpentine course of the suture, areas of tension and compression develop in the tissue (Fig. 3-48).

Concomitant with a general advance of obliteration with age in a single suture, more of the palatal suture area is involved. Besides variations in the degree of closure between sutures, variations also exist between different parts of the same suture. Closure progresses more rapidly in the oral than in the nasal part of the palatal vault, and the intermaxillary suture starts to close more often in the posterior than in the anterior part.[41] Because of the increased inclusion and bone locking (interdigitation), the possibility for influencing the suture area by orthopedic treatment gradually decreases with age.

Suture Response to Orthodontic/Orthopedic Forces

The attempt to move an incisor orthodontically *through the midpalatal suture* in dogs was shown to be unsuccessful.[44] The histologic analysis revealed that the suture had been dislocated

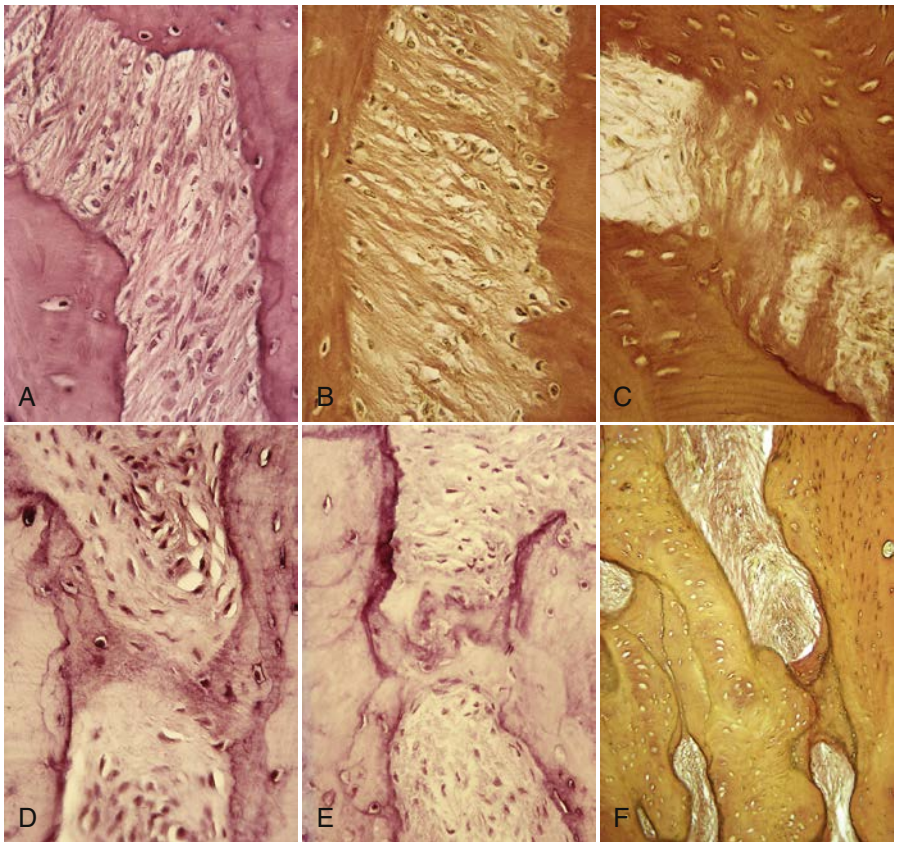


FIGURE 3-47 Photomicrographs from the human intermaxillary suture at different ages (14 to 25 years) showing various stages of fusion. The collagen fibers perpendicular to the bony surface **(A)** increase in thickness **(B)** and density **(C)** with age. A slender bridge of uncalcified tissue in the area of tightly packed fibers **(D** and **E)** is a preliminary stage to bony obliteration **(F)** of the suture. (Adapted from Persson M, Magnusson B, Thilander B. Suture closure in rabbit and man: a morphological and histochemical study. *J Anat.* 1978;125:313.)

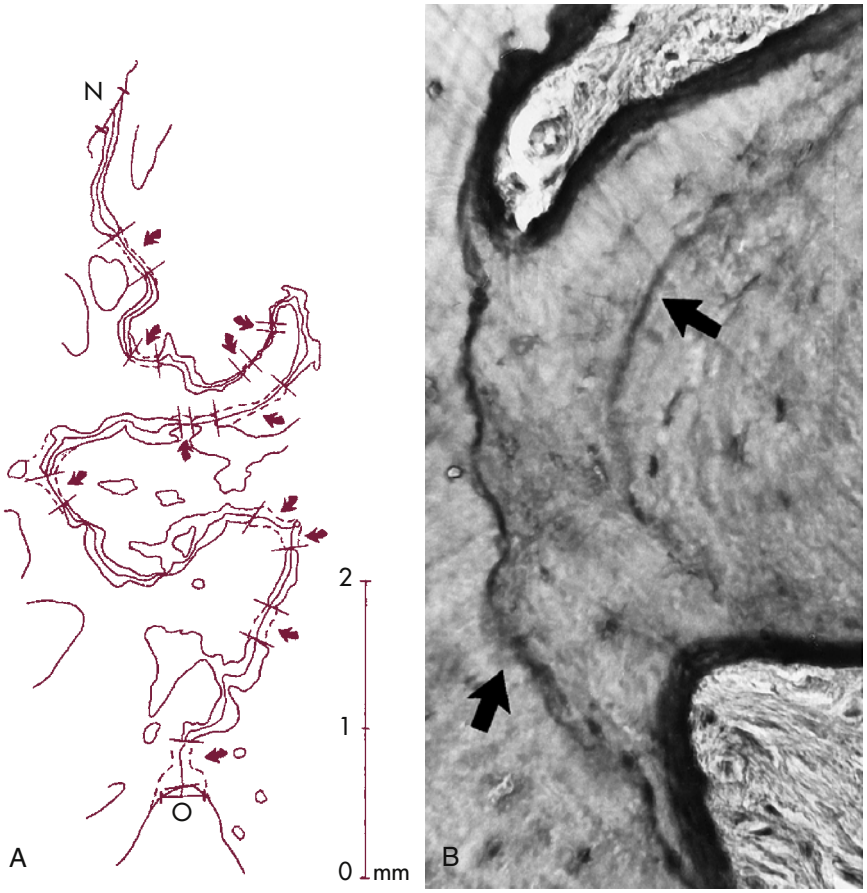


FIGURE 3-48 A, Tracing of a section in the frontal plane from the posterior part of the intermaxillary suture of a 31-year-old man. Nasal (*N*) and oral (*O*) sides are presented. Bony bridges occur at the arrows. B, An ossified part of the suture. Note the basophilic staining at the site of the earlier resting-suture margin (*arrows*). (From Persson M, Thilander B. Palatal suture closure in man from 15 to 35 years of age. *Am J Orthod.* 1977;72:42.)

in front of the orthodontically moved incisor (Fig. 3-49). Eventually, the PDL and suture tissue became merged, demonstrating an apparently close similarity between the tissues of the two joint types. However, in dogs with a closed suture, the incisor could pass the sutural area without any impediment.

Traction generated by *orthopedic forces* has long been claimed to stimulate sutural growth, and widening of the midpalatal suture is a clinically well-documented procedure in orthodontics (Fig. 3-50) (for a review, see Timms[45]). The tissue response has been reported in histologic studies in animals. The mechanical response to traction includes a widening of the suture and changes in the orientation of fiber bundles.[46] A considerable increase of osteoblasts and an osteoid zone on both sutural bone surfaces indicate bone formation (Fig. 3-51). The bone deposition accompanies traction, allowing the suture to recover a normal histologic picture. A positive correlation exists between the magnitude of the tensile force and osteogenic response.[47,48] Experiments in vitro verify that mechanical tensile forces stimulate synthesis of structural proteins.[49] *Healing* of a suture after rapid expansion may entail formation of bony bridges across the suture. Low-power laser irradiation has been shown to accelerate bone regeneration in the midpalatal suture during rapid palatal expansion in the rat.[50]

The *transduction of a mechanical force* into bone production may involve an intracellular influx of calcium and/or sodium ions, which decreases cAMP and triggers DNA synthesis and cellular proliferation.[34] Ten Cate et al.[51] used heavy forces to expand cranial sutures and showed that fibroblasts must proliferate and repair sutural connective tissue before osteogenesis and remodeling of the suture take place.

Rapid maxillary expansion is an orthopedic procedure that has been shown to influence not only the midpalatal suture but also the *circumaxillary sutural system*. In such a palatal splitting, most of the resistance to separation results from the circumaxillary structures. Because posterior and anterior *displacements of the maxilla* (*protraction*) involve more sutures than displacements caused by palatal expansion, the resistance to separation consequently increases (see Fig. 3-47).

TISSUE RESPONSE IN THE TEMPOROMANDIBULAR JOINT

The response in the TMJ region to functional and orthopedic appliances has been a subject of discussion for many years. So, it is necessary to understand the nature of the tissues that make up the TMJ, their relation to the normal growth process, and the manner in which they express themselves during maturation.

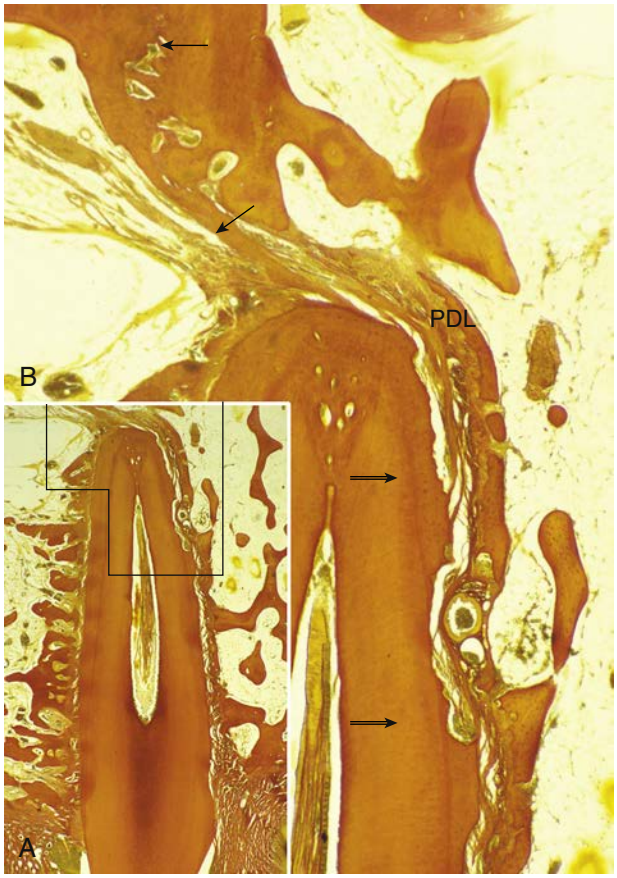


FIGURE 3-49 Photomicrographs of the intermaxillary suture of a young dog in which an attempt to orthodontically move an incisor through the suture was shown to be unsuccessful. **A,** The suture fibers are stretched around the apex and seem to continue in the periodontal ligament (*PDL*). **B,** Magnification of boxed area in **A**. *Closed arrows* indicate the intermaxillary suture; open arrows indicate the direction of movement with root resorption. (From Follin M, Ericsson I, Thilander B. Orthodontic tooth movement of maxillary incisors through the midpalatal suture area. An experimental study in dogs. *Eur J Orthod.* 1984;6:237.)

The TMJ provides the essential functional connection between the cranium and the upper and lower jaw (Fig. 3-52). However, the primary functions of the TMJ in general and the mandibular condyle in particular are not simple, and they change during development. Furthermore, striking differences exist in the extent of mineralization between the condyle and the temporal component (Fig. 3-53).

Structure of the Temporomandibular Joint

Several investigations of the anatomy and development of the TMJ in various experimental animals are available. However, there are only a few comprehensive systematic investigations of human postnatal TMJ development.[52-54]

Condylar Cartilage

The condylar cartilage varies in appearance from one part of the condyle to another, and its histomorphologic picture varies from birth to childhood, but the following four layers usually can be seen (Fig. 3-54): a fibrous connective tissue layer (surface articular zone); a highly cellular intermediate layer containing proliferating cells (proliferative zone); a cartilage layer with irregularly arranged chondrocytes (hypertrophic zone); and a zone with endochondral bone ossification (bone formative zone).

During the juvenile period, the condyle becomes progressively less vascularized, and the entire growth cartilage layer becomes significantly thinner, primarily because of reduction in the hypertrophic zone. At the age of 10 years, the mandibular condyle is characterized by a relatively thick articular tissue layer, progressive reduction in the size of the entire growth cartilage layer, and evidence of increased mineralization in the deeper portion of the hypertrophic zone. After 13 to 15 years, the cartilage layer decreases further in thickness. By the age of 19 to 22 years, only islands of cartilage cells remain in the superior and anterior regions.

Mandibular Neck

The fibrous layer of the condylar cartilage is continuous with the periosteum of the ramus, and remodeling processes are seen in all regions of the mandibular neck (Fig. 3-55). The term *condylar growth* is therefore misleading, and the term *ramus and condylar growth* is more correct, as expressed by Enlow.[55]

Temporal Component

The S-shaped curve that characterizes the temporal component of the TMJ becomes evident during the first 6 months of life. The articular surface is lined with a fibrous connective tissue resembling the surface articular zone of the condyle (Fig. 3-56, *A*), with an increase in thickness during early and mixed dentitions through puberty. The proliferating zone is seen up to the age of 17 to 18 years (Fig. 3-56, *B*). In the fossa, remodeling is observed from early childhood to adulthood[53] (Fig. 3-56, *C* and *D*).

Temporomandibular Joint Response to Orthopedic Forces

Because remodeling processes are seen in all components of the joint, most obviously in young ages, it has been speculated whether the TMJ would respond to orthodontic stimuli and influence the growth of the mandible.[56] McNamara[57] has shown that the TMJ in monkeys is capable of functional adaptation when the mandible is displaced in a *forward direction*. Hypertrophy and hyperplasia of the prechondroblastic and chondroblastic layers of the condylar cartilage were seen, particularly along the posterior border of the condyle, with rapid bone formation in the condylar head. After about 10 weeks, the generalized proliferation of the condylar cartilage was, however, no longer evident, indicating that the essential remodeling had been completed. Furthermore, an increased activity of the superior head of the lateral pterygoid muscle was recorded. A progressive modification of the neuromuscular pattern thus was observed with the skeletal adaptation to the forward displacement of the mandible. Moreover, the subperiosteal growth rate increases at the posterior border of the ramus. In addition, the bone turnover rate and mineralization in the mandible as a whole are significantly influenced.

Petrovic and coworkers have shown in a number of experiments in rats, with anterior displacement of the mandible, that a functional appliance primarily induces an amplification in skeletoblast and prechondroblast mitotic activity, an acceleration in differentiation of skeletoblasts into prechondroblasts, an increase in transformation of prechondroblasts into

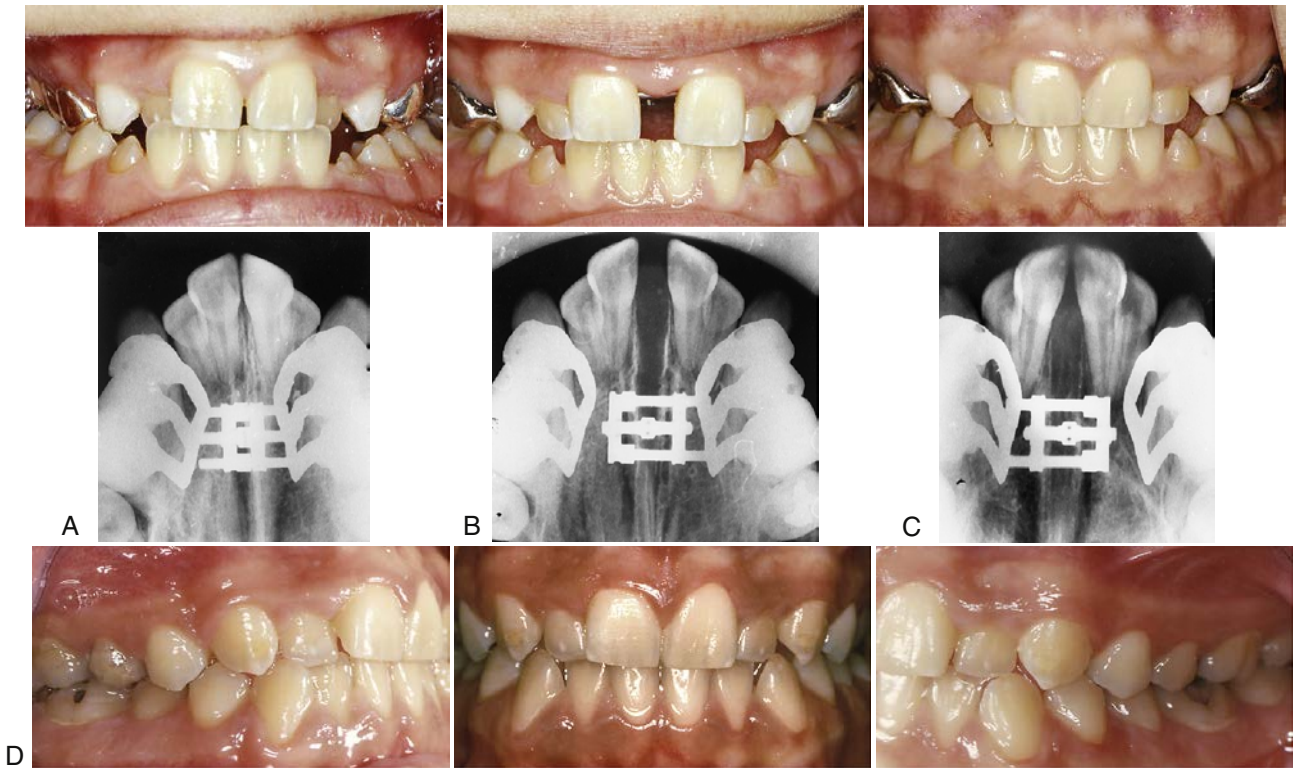


FIGURE 3-50 Widening of the midpalatal suture in a 12-year-old girl. **A,** Before maxillary expansion. **B,** After 21 days of rapid maxillary expansion. **C,** Spontaneous closure at day 60, when the appliance was removed. **D,** 5 years postretention.

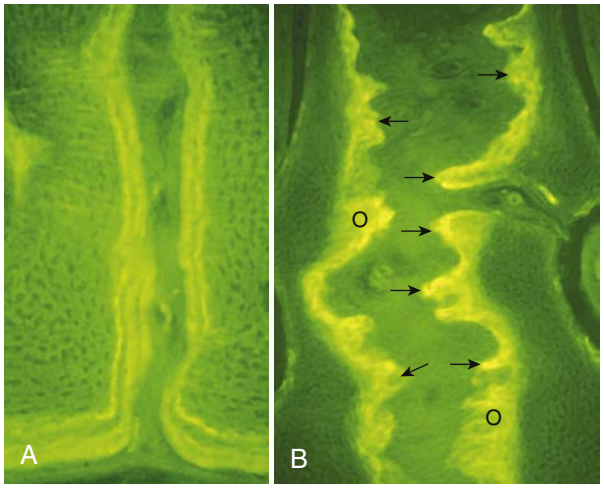


FIGURE 3-51 Photomicrographs of the intermaxillary suture in the rat. **A,** Before expansion. **B,** After sutural expansion for 7 days. Increased cellular activity is evident along the total bone surfaces (*arrows*). *O,* Osteoid layer in the yellow areas (tetracycline administration). (From Engström C, Thilander B. Premature facial synostosis: the influence of biomechanical factors in normal and hypocalcemic young rats. *Eur J Orthod.* 1985;7:35.)

functional chondroblasts, and an acceleration in chondroblast hypertrophy and endochondral bone growth (for a review, see Petrovic et al.[58]).

A *mandibular retrusion* by chin cap therapy in the rat revealed a reduced thickness of the prechondroblastic zone and a decrease in the number of dividing cells.[59] A reduced length of the mandible indicated that chin cap treatment had a retarding effect on mandibular growth, contrary to the increased length in the experiments with anterior displacement of the mandible.

How does this experimentally induced stimulation or retardation of condylar growth relate to *clinical orthodontics*? Some researchers maintain that mandibular growth in children can be altered and that this change can be detected through well-designed clinical procedures. However, several researchers consider that the therapeutic effect of a functional appliance on the lengthening of the mandible is not clinically relevant, for the individual's growth potential during the treatment period also is included in this effect.

These conflicting observations made in clinical studies over many years suggest that alterations in condylar growth in human patients are difficult to document. One reason for this is that the controlled design in animal experiments is hardly attainable in humans. Moreover, the growth rate in the rat and monkey is much faster than that in humans, factors that make appearance and quantification of the adaptive changes difficult. In addition, the clinician is aiming at changing an abnormal growth pattern into a normal one, which is different from alterations that start from normal growth pattern, as in animal experiments. Thus, the question of whether the growth of the human mandible may be altered by orthopedic and functional appliances remains partly unanswered (See also Chapter 35). However, research shows that the orthopedic change apparently is a combined change of mandibular and temporal components (i.e., condylar and remodeling processes).

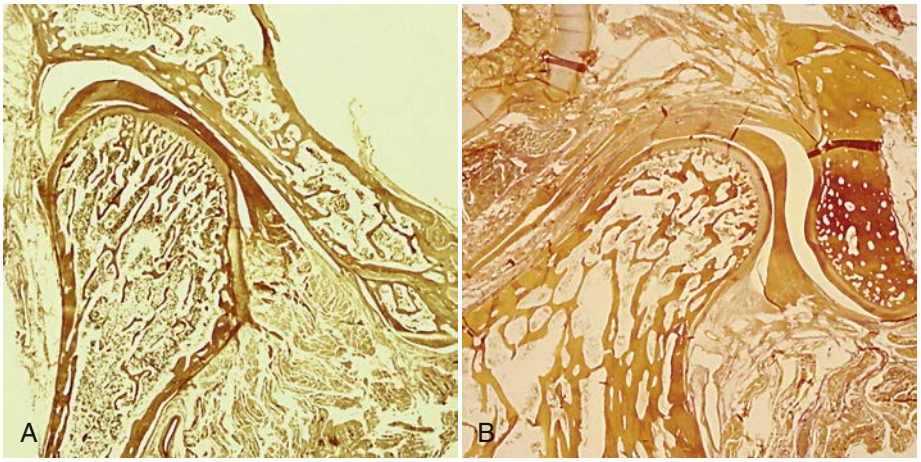


FIGURE 3-52 Photomicrographs of the human temporomandibular joint. **A,** Transverse plane. **B,** Sagittal plane. (From Thilander B, Carlsson GE, Ingervall B. Postnatal development of the human temporomandibular joint. I. A histologic study. *Acta Odont Scand.* 1976;34:117.)

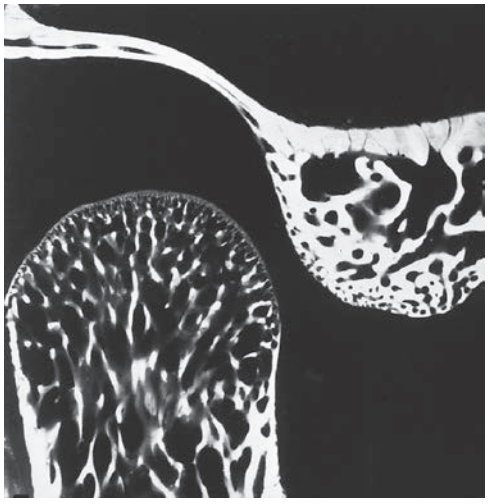


FIGURE 3-53 Microradiogram of a sagittal section of the temporomandibular joint from a 6-year-old boy. Note the difference in mineralization in the different parts of the joint. (From Ingervall B, Carlsson GE, Thilander B. Postnatal development of the human temporomandibular joint. II. A microradiographic study. *Acta Odont Scand.* 1976;34:133.)

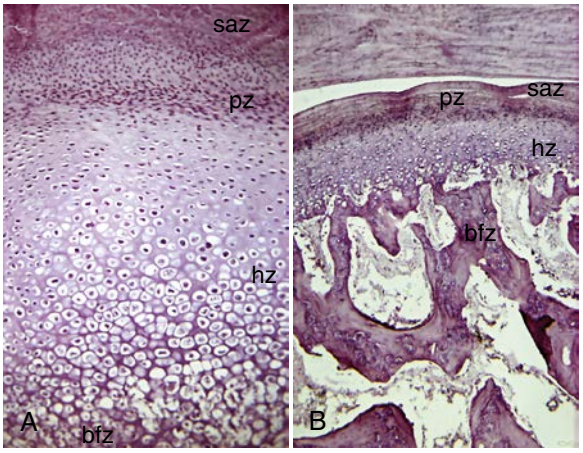


FIGURE 3-54 Photomicrographs of the condylar layer from the upper central part of the condyle (sagittal plane), showing the different morphology at various ages (×100). **A,** A 6-month-old boy. **B,** A 12-year-old boy. *saz,* Surface articular zone; *pz,* proliferation zone; *hz,* hypertrophic zone; *bfz,* bone formation zone. (Adapted from Thilander B, Carlsson GE, Ingervall B, Postnatal development of the human temporomandibular joint. I. A histologic study, *Acta Odont Scand.* 1976;34:117.)

POSTTREATMENT STABILITY

Not all orthodontically achieved changes remain stable. If an undesirable growth pattern is treated partly by influencing growth and partly by compensations in the dentoalveolar system, subsequent posttreatment growth may upset a result that looks good when the patient is young. It is well known that many patients experience changes some years after treatment, which is often described as relapse (i.e., a return toward pretreatment conditions). We have, however, to distinguish the "rapid relapse" occurring during the period of remodeling of periodontal structures from the "slow relapse" that is due to the late changes during the postretention period.

Tissue Reactions Seen in Orthodontic Retention and Relapse

Experimental studies have shown that if orthodontic movement is not followed by remodeling of the supporting tissues, the tooth will return to its former position.[59-61] The most persistent relapse tendency is caused by the structures related to the marginal third of the root, whereas little relapse tendency exists in the area adjacent to the middle and apical thirds. The effect caused by contraction of the principal and supraalveolar fibers is shown in Figure 3-57. The crown of an experimentally moved tooth in a dog was moved 2 mm by tipping over during a period of 40 days. After the tooth was released, the degree of relapse movement was recorded graphically. Some relapse was noticed to have

already occurred after 2 hours, partly caused by the tooth's regaining a more upright position within the periodontal space. Still more relapse occurred on the following days for a total of about 0.5 mm during 4 days. After this, the tooth came to a standstill. Histologic examination revealed that this was caused by a hyalinized area on the tension side.[61] Therefore, fiber contraction is strong enough to produce hyalinization. Similar hyalinized zones may be observed after tipping human teeth without subsequent retention (Fig. 3-58).

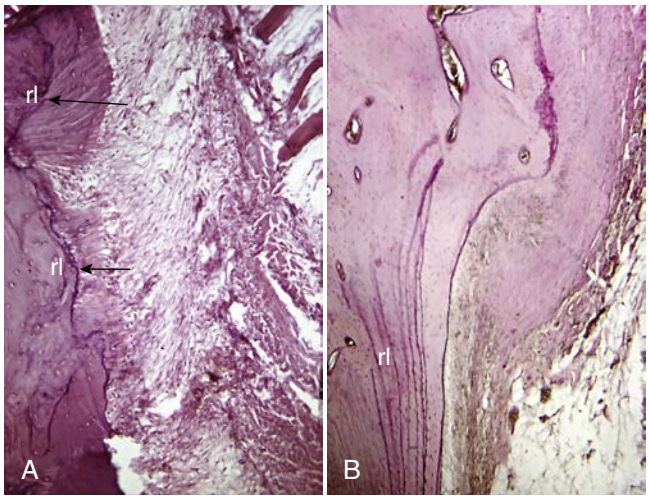


FIGURE 3-55 Photomicrographs from the mandibular neck (transversal plane) illustrating the remodeling processes (×40). **A,** A 7-year-old girl. **B,** A 23-year-old woman. *rl,* Resting lines. Apposition starts medially to *rl* (*arrows*). (Adapted from Thilander B, Carlsson GE, Ingervall B. Postnatal development of the human temporomandibular joint. I. A histologic study. *Acta Odont Scand.* 1976;34:117.)

The tissue reactions in the gingiva differ from those in the PDL and are of different importance for the stability of an acquired tooth position (see Figs. 3-2 through 3-4). The various fiber groups also respond differently to the remodeling process. Furthermore, the supporting fibrous systems, both supraalveolar and periodontal, develop during the eruption of teeth according to increasing functional demands. This explains the greater stability of teeth that are guided passively into position during the eruptive period, compared with teeth that are moved after having reached occlusal stability.

Several factors are essential for the reestablishment of an adequate supporting apparatus during and after tooth movements. The main remodeling of the PDL takes place near the alveolar bone; the remodeling of the fibrous system on the tension side is related to the direction of pull on the tooth, resulting in production of new fibers only in that direction.

Unlike the PDL, the supraalveolar fibers are not anchored in a bone wall that is readily remodeled, and therefore they have less chance of being reconstructed. Furthermore, the remodeling of gingival connective tissue is not as rapid as that of the PDL, and the slower turnover of the gingival collagen fibers[34] easily explains why such fibers are seen stretched and unremodeled as long as 232 days after experimental tooth rotation[60] (Fig. 3-59). The stretched fiber bundles on the tension side tend to become relaxed and rearranged according to the

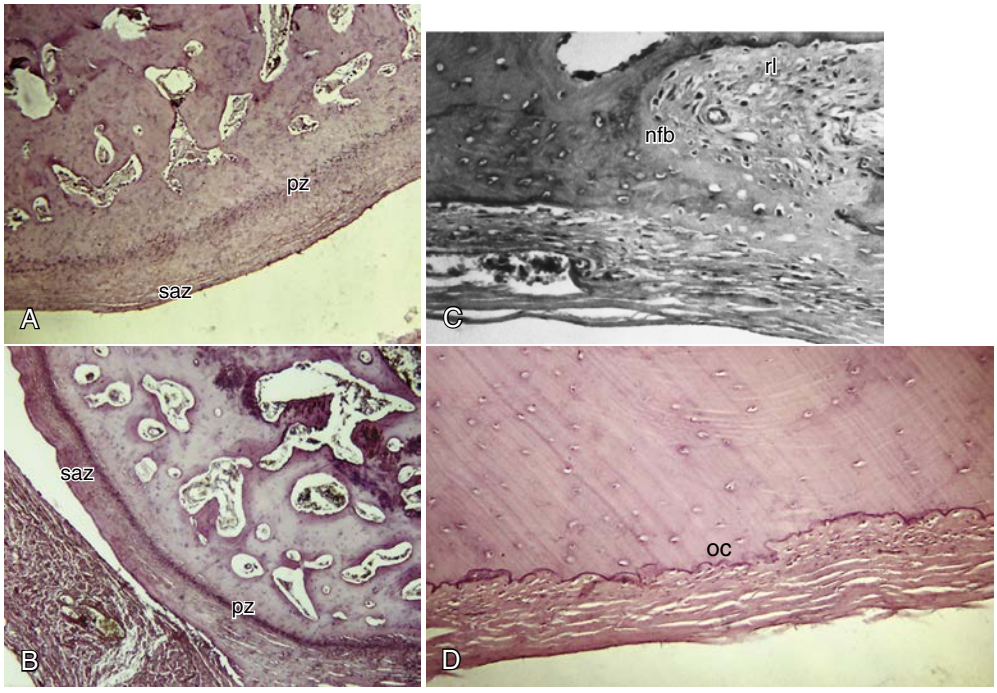


FIGURE 3-56 **A, B,** Photomicrographs from the temporal part (central section, sagittal plane) (×40) articular tubercle of **A,** 9-year-old boy and, **B,** a 14-year-old boy. *saz,* Surface articular zone; *pz,* proliferation zone. **C, D,** Articular fossa illustrating remodeling processes in **C,** a 7-year-old boy and, **D,** a 16-year-old boy. *rl,* Resting line; *nfb,* Newly formed bone; *oc,* Osteoclastic activity. (Adapted from Thilander B, Carlsson GE, Ingervall B. Postnatal development of the human temporomandibular joint. I. A histologic study. *Acta Odont Scand.* 1976;34:117.)

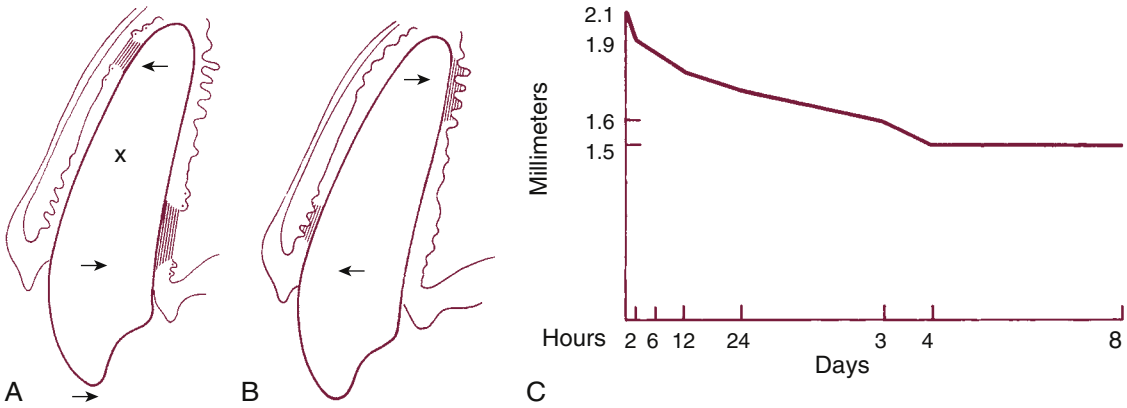


FIGURE 3-57 A, Formation of hyalinized areas during tooth movement of an upper second incisor in a dog (duration 40 days). B, Formation of hyalinized areas during the relapse period. The tooth was not retained after movement. C, Relapse movement during a period of 8 days (as seen in tooth B). Hyalinization occurred after 4 days.

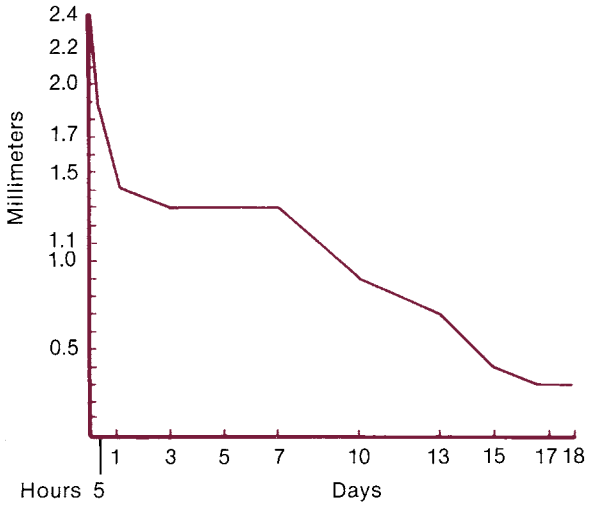


FIGURE 3-58 Degree of movement after labial tipping and the subsequent relapse movement after the tooth had released. Upper lateral incisor of a 12-year-old patient (force, 40 cN). Most of the relapse movement occurred during the first 5 hours. A hyalinized zone existed from the third to the seventh day.

physiologic movement of the tooth. During retention, new bone fills in the space between the bone spicules. This rearrangement and calcification of the new bone spicules result in a fairly dense bone tissue.[3] Therefore, to avoid relapse, a tooth should be retained until total rearrangement of the structures involved has occurred.

Slower remodeling also explains why supracrestal fiberotomy surgery prevents or reduces relapse after the experimental rotation of teeth (see Fig. 3-37, *B*). The reason for the slow remodeling of the supraalveolar tissues after experimental tooth movement probably is related to the quality of particular fiber groups, whose main function is to maintain tooth position and interproximal contact.

A clinical experience is that cases after orthodontic space closure of extraction gaps have a tendency to reopen. The orthodontic force creates a compressed gingival tissue in the extraction site (Fig. 3-60, *A*). Excision of this hyperplastic area showed a long-lasting epithelial fold (invagiation) with loss of collagen but an increased amount of glycosaminoglycans (Fig. 3-60, *B*).[62] Such "elastic" tissue together with the compression of the transseptal fibers may be responsible for relapse after closure of the extraction gap.

Changes during the Postretention Period

The length of the retention period to avoid relapse is controversial. Some recommend 2 to 5 years, whereas others have suggested a minimum of 10 years or longer. Analysis of not only orthodontically treated cases but also of nontreated cases in follow-up studies is of importance to learn more about postretention development versus relapse.

A longitudinal study of individuals, not orthodontically treated, with well-shaped dental arches and normal occlusion ("ideal cases")[63,64] clearly showed the dentofacial changes from early childhood into adolescence, young adulthood, and late adulthood, changes that are a gradual process that has to be taken into consideration in orthodontic treatment. The circumference of the dental arches increased during eruption of the incisors and canines but thereafter continuously decreased, especially in the mandible (Fig. 3-61). This will explain why 12 of the 30 subjects, examined at 31 years of age, showed incisor crowding of different degree, even in cases with congenitally missing third molars (Fig. 3-62). Width and depth of the dental arches also showed slow continuous changes of importance for postretention stability (See Chapter 33 for further discussion).

One interesting finding in that longitudinal study was the development of the palatal height. A continuous slow increase in this distance seems to indicate an important role in the tooth eruption mechanism (Fig. 3-63). This knowledge is of importance in explaining the infraposition of an implant-supported crown as a continuous eruption of its adjacent teeth[65] (Fig. 3-64).

The continuous dentoalveolar change, known as *physiologic tooth migration*, thus has to be distinguished from orthodontic relapse. The occlusion hence is to be regarded as a dynamic rather than as a static interrelation between facial structures.

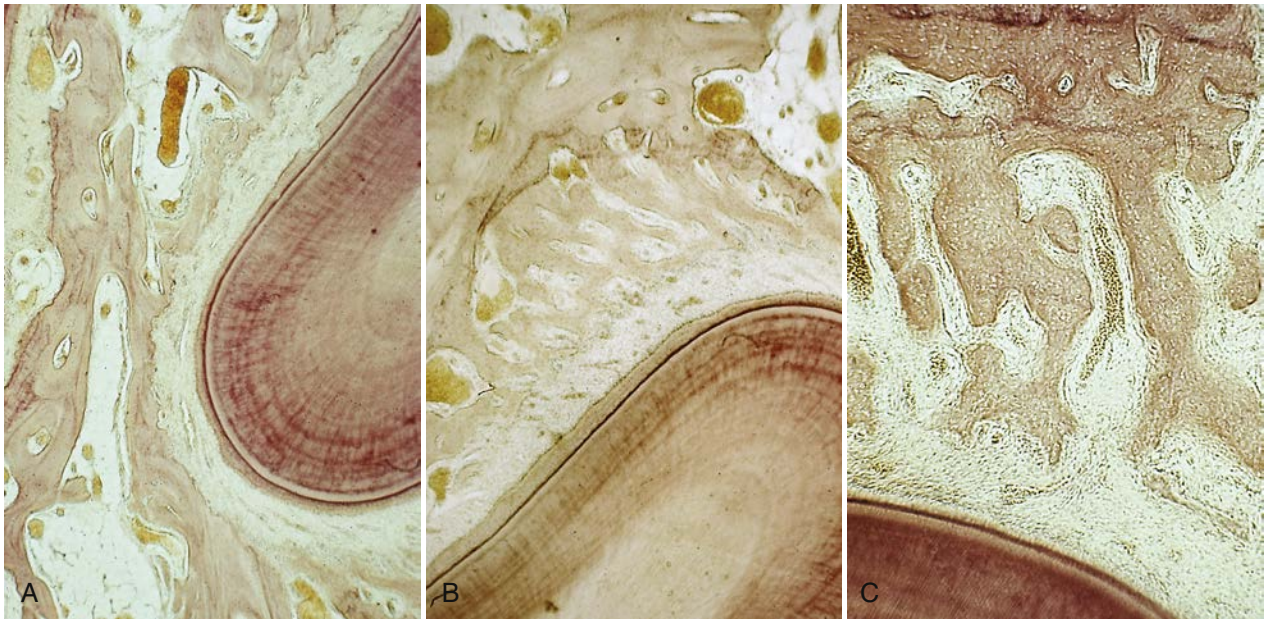


FIGURE 3-59 Tissue response after rotation of a tooth. **A,** Pressure side with direct bone resorption. **B,** Arrangement of new bone layers formed on the tension side along stretched fiber bundles after rotation. **C,** Same area after a retention period of 3 to 4 months. The bone and the principal fibers are rearranged much sooner than the displaced supraalveolar structures.

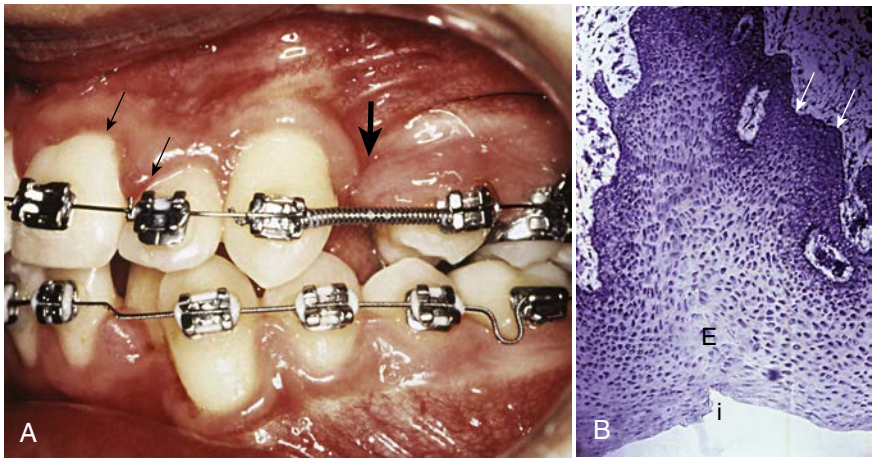


FIGURE 3-60 Fixed appliance for closure of an extraction space. **A,** With gingivitis of the incisors (*small arrows*) and invagination (*large arrow*) in the extraction area. **B,** Histologic appearance of the invagination (*i*) area. Deep proliferation of oral epithelium (*E*). High levels of oxidative enzyme activity are present in the hyperplastic basal cell layers (*arrows*). (From Rönnerman A, Thilander B, Heyden G. Gingival tissue reactions to orthodontic closure of extraction sites: histologic and histochemical studies. *Am J Orthod.* 1980;77:620.)

SUMMARY

This chapter represents a discussion of the principal tissue changes resulting from orthodontic forces acting on teeth and supporting structures, sutures, and the TMJ region. Unfavorable forces may initiate adverse tissue reactions. After orthodontic treatment, a rapid-to-slow relapse occurs during the period of remodeling of the periodontal structures. The tissue reactions in the gingiva differ from those in the periodontal ligament and are of different importance to maintain the stability of the tooth in its new position. *Late changes occurring during the postretention period generally cannot be distinguished from normal aging processes that occur regardless of whether a person has been treated orthodontically.* Even though the knowledge of tissue reaction in orthodontics is well documented by numerous experimental and clinical studies, many basic questions still are unanswered.

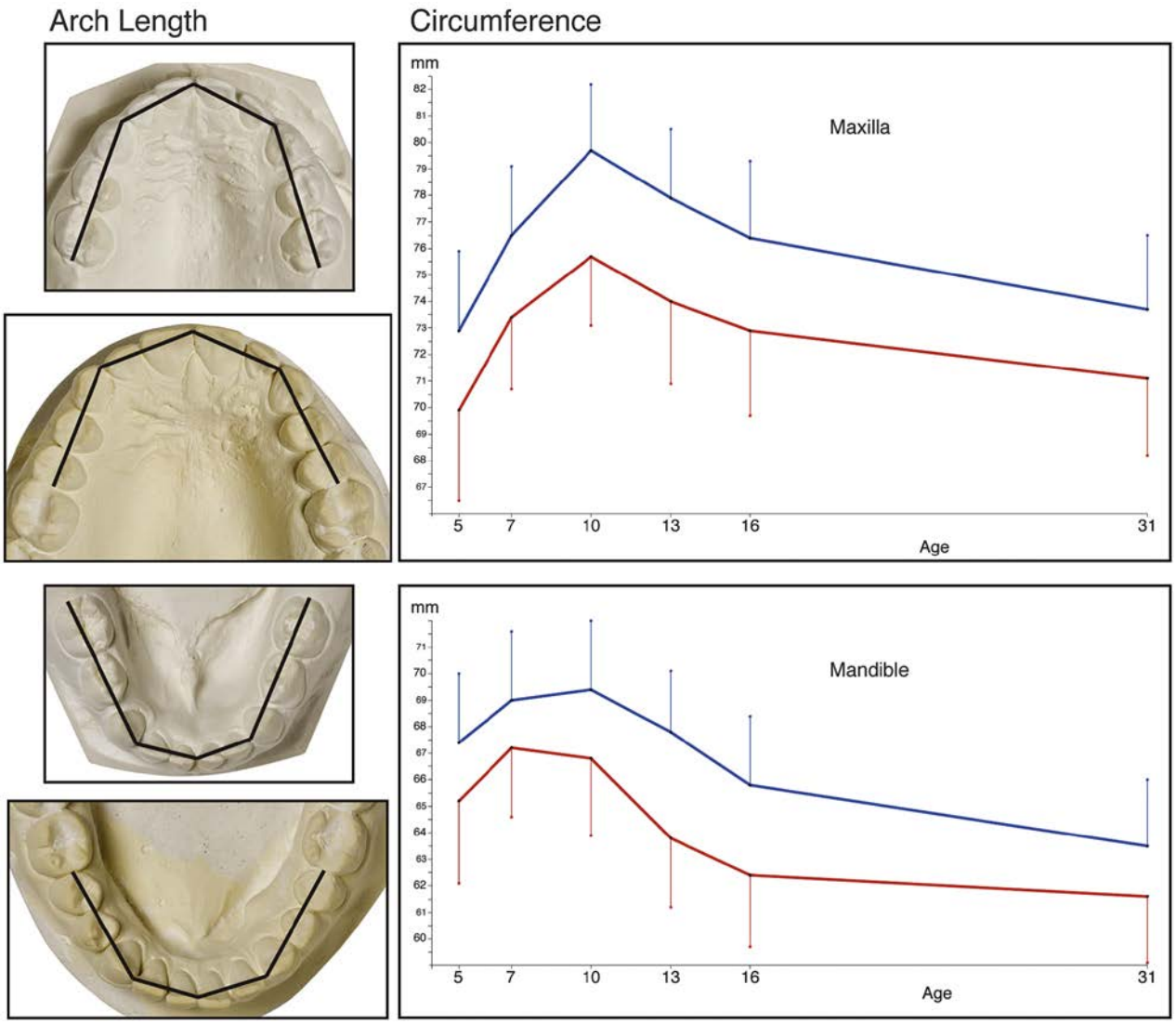


FIGURE 3-61 The dental arch circumference (mesial of the first permanent molars) in the maxilla and mandible in subjects with "ideal" occlusion. Females (red) and males (blue) followed from 5 to 31 years of age. Mean and standard deviation for each recording. (From Thilander B. Dentoalveolar development in subjects with normal occlusion: a longitudinal study between the ages of 5 and 31 years. *Eur J Orthod.* 2009;31:109.) See Chapter 33 for further discussion.

TOOTH MOVEMENT AT THE CELLULAR AND MOLECULAR LEVELS*

INTRODUCTION

Orthodontic tooth movement beyond the constraints of the original tooth socket requires the conversion of mechanical forces into biological signals by mechanosensitive cells. This mechanotransduction of signals promotes intracellular communication and allows for the coordinated cellular response of alveolar bone modeling that occurs in response to orthodontic force. Orthodontic forces are likely perceived by cells as changes in substrate strain, fluid flow shear induced stress, and/or oxygen tension. Osteoprogenitor cells, bone lining cells, and osteocytes sense orthodontic forces and initiate a number of signaling pathways through mediators such as Wnt, BMP, TNFα, IL1β, CSF-1, VEGF, and PGE2, etc. These factors subsequently lead to the recruitment, differentiation, and activation of osteoblasts and osteoclasts to conduct the bone formative and resorptive activities, respectively, needed for orthodontic tooth movement.

ORTHODONTIC FORCES STIMULATE BIOLOGICAL RESPONSES

The orthodontic profession has long been aware that changes in the supporting tissues of teeth are necessary for tooth movement beyond the constraints of the original tooth socket upon application of an orthodontic force. Theories regarding the biological response to orthodontic forces

*Nan Hatch, Zongyang Sun

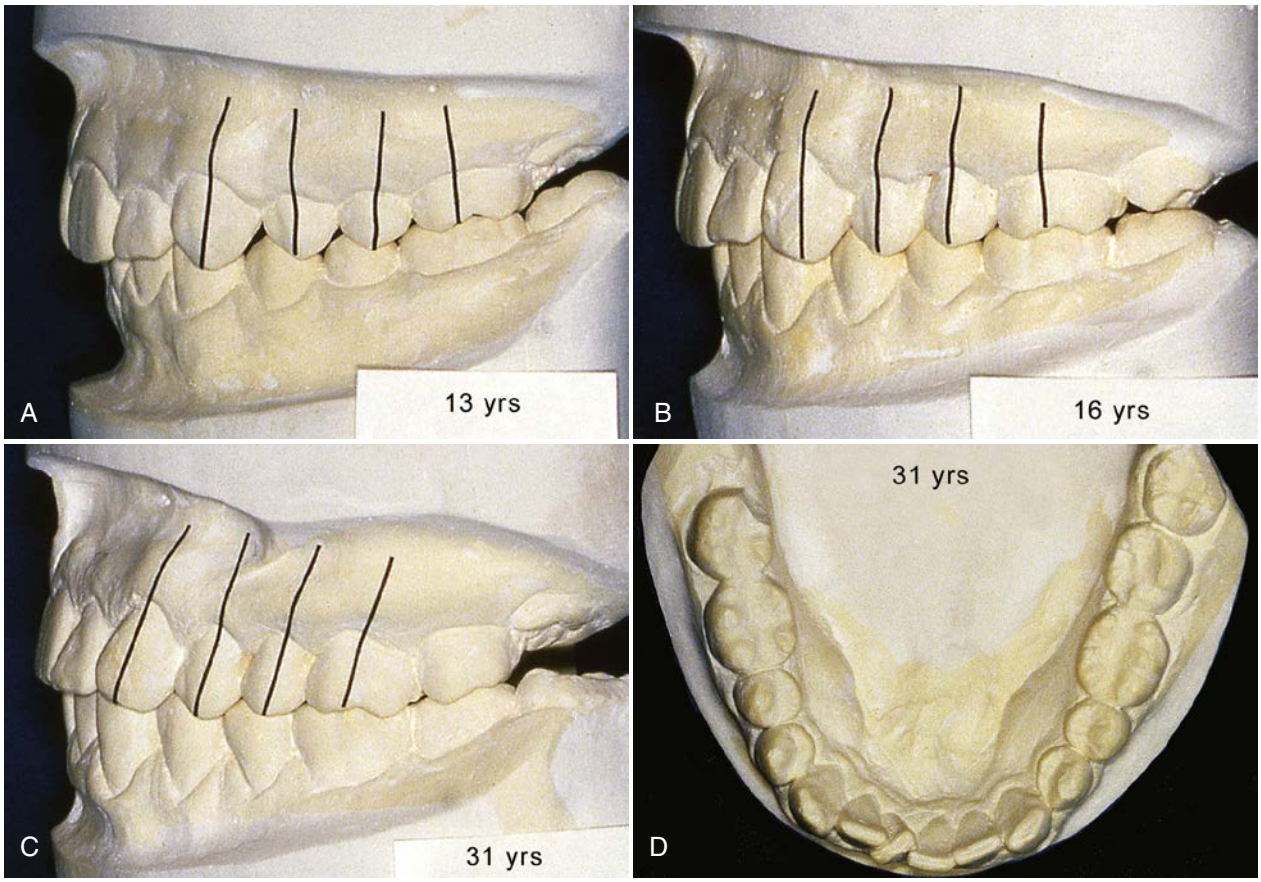


FIGURE 3-62 Study casts from a subject (male), classified as "normal" at 13 and 16 years, even at 31 years. Note the negative effect of physiologic migration, resulting in 41 in a crowded position in spite of a congenital absence of 48 in contrast to fully erupted 38.

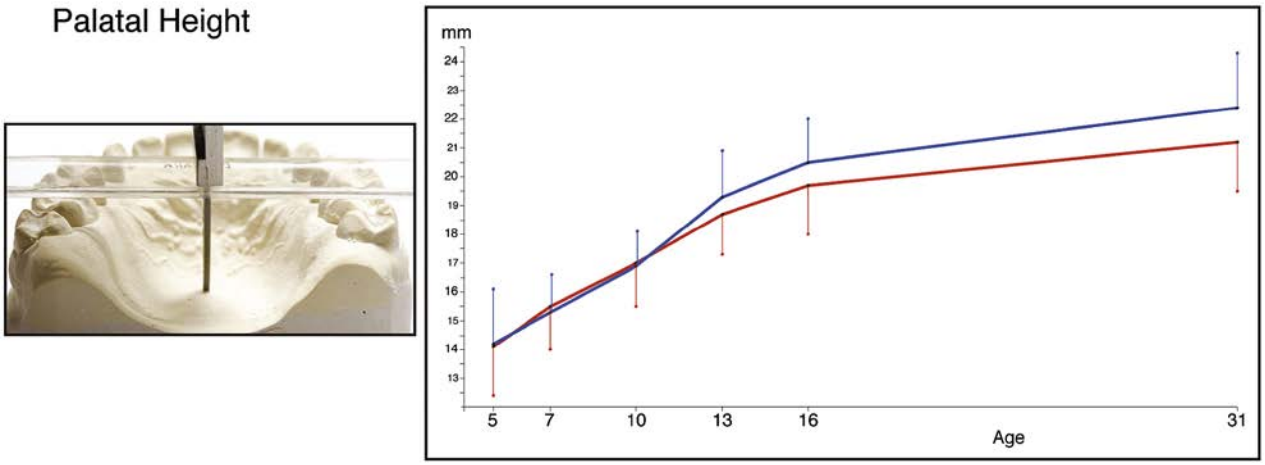


FIGURE 3-63 Palatal height (mm) in females (red) and males (blue), followed from 5 to 31 years of age; mean and standard deviation for each recording. (From Thilander B. Dentoalveolar development in subjects with normal occlusion: a longitudinal study between the ages of 5 and 31 years. *Eur J Orthod.* 2009;31:109.)

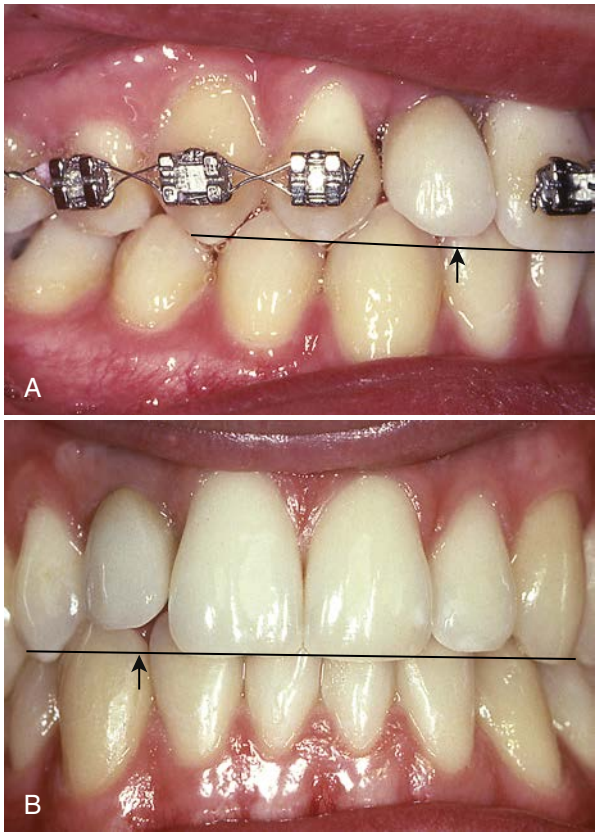


FIGURE 3-64 A, A patient with an implant-supported crown (*arrow*) replacing the congenitally missing upper right lateral incisor. B, Continuous eruption of the teeth adjacent to the implant from 16 to 24 years resulted in 1.6 mm of infraocclusion of the implant-supported crown (*arrow*). (Adapted from Thilander B, et al. Orthodontic aspects of the use of oral implants in adolescents: a 10-year follow-up study. *Eur J Orthod.* 2001;23:715.)

resulting in tooth movement were initially proposed over a century ago. Based upon their clinical observations, Kingsley and Walkhoff theorized that tooth movement depends upon the elasticity, compressibility and extensibility of bone while Schwalbe and Flouren theorized that bone resorption occurs in areas of pressure, and bone deposition occurs in areas of tension, following the application of orthodontic force.[66] The first systematic experimentation investigating local tissue responses to orthodontic force application was performed by Carl Standstedt in the early 1900s. His light microscopic studies following incisor retraction in dogs confirmed the theory of Schwalbe and Flouren and showed for the first time that bone deposition occurs in areas of tension and bone resorption occurs in areas of pressure following the application of orthodontic force.[66] Significantly, Standstedt was the first to show that lighter orthodontic compressive forces lead to rapid bone resorption along the alveolar wall while heavier compressive orthodontic forces lead to tissue necrosis within the PDL space along the alveolar wall (defined as hyalinized tissue) (see Figs. 3-15 to 3-23). He also noted that tooth movement in these hyalinized areas occurred only after bone resorption in underlying bone marrow spaces was sufficient to undermine the supporting alveolar bone (defined as undermining resorption). Schwartz extended the findings of Standstedt by correlating the tissue response to compressive orthodontic forces with PDL capillary blood pressure. He stated that lighter orthodontic forces leading to rapid alveolar bone resorption and tooth movement are those that are below the pressure of PDL blood capillaries and that heavier orthodontic forces lead to "suffocation of the peridental membrane" that leads to tissue necrosis and a delay in orthodontic tooth movement.[67] These findings, in combination with numerous other studies, suggested that orthodontic forces move teeth by stimulating a biological response involving bone modeling activity. Importantly, these results also indicated that occlusion of PDL blood vessels with resulting ischemia and necrosis is not required for bone resorption to occur along the alveolar wall on the pressure side of orthodontic force application. In other words, light compressive forces can stimulate alveolar bone resorption that allows for tooth movement beyond the original constraints of the tooth socket.

We now know that bone modeling requires the differential activity of bone-forming cells (osteoblasts) and bone-resorbing cells (osteoclasts). A study conducted in the early 1990s by King et al. confirmed that orthodontic force application induces differential osteoclastic and osteoblastic activity. Results of this study showed that orthodontic appliance activation leads to primarily osteoclastic activity along the alveolar bone in the compressed regions of the PDL space and primarily osteoblastic activity along the alveolar bone in the tensed regions of the PDL space. For osteoclastic activity to occur, osteoclast precursor cells must be recruited to the PDL space from the circulatory system and bone marrow, as these cells are hematopoietic in origin. For tooth movement to occur, recruited osteoclastic precursor cells must also then be stimulated to fuse, differentiate, and develop into multinucleated, fully functional, mature osteoclasts. Similarly to obtain bone formative activity along the alveolar wall of the tooth socket, PDL mesenchymal stem cells, osteoblast precursor cells and/or bone lining cells must be stimulated to differentiate into osteoblasts. Overall, the work of King et al.[68] therefore showed that mechanical orthodontic forces stimulate biological responses involving the recruitment and activation of osteoblasts and osteoclasts.

MECHANOTRANSDUCTION MEDIATES THE BONE MODELING RESPONSE TO ORTHODONTIC FORCE

In the years since these studies were completed, significant progress has been made in understanding how mechanical signals can initiate biological cellular responses. This process is generally known as *mechanotransduction.* Mechanotransduction requires the application of a mechanical load to tissue, conversion of that load into a mechanical signal that can be sensed at the cellular level, and cellular transformation of the mechanical signal into a biochemical signal that is then communicated to other cells to elicit a coordinated cellular response. In the context of orthodontic tooth movement, mechanotransduction very likely involves alveolar bone osteocytes, bone lining cells, and PDL mesenchymal precursor cells. Orthodontic forces move a tooth initially within the PDL space. This tooth movement likely results in mechanical strain changes in PDL fibers and in underlying alveolar bone, as well as changes in fluid flow within the lacunar–canalicular alveolar bone network and within the PDL space. Tooth movement

can also compress PDL blood capillaries resulting in localized hypoxia. Once initiated, mechanotransduction leads to the activation of downstream cell signaling pathways and cellular responses that lead to bone resorptive and formative activities (Fig. 3-65).

Recent advances in bone mechanotransduction are well detailed in several excellent reviews.[69-71] Briefly, it is now recognized that the primary bone cell type responsible for sensing mechanical load is the osteocyte rather than the osteoblast. Osteocytes are terminally differentiated osteoblasts. After osteoblasts lay down bone matrix, they either undergo apoptosis (cell death) or undergo terminal differentiation and become osteocytes embedded within the matrix. Accounting for 90% to 95% of total bone cells, osteocytes reside in a complex lacunae–canaliculi network inside the bone matrix, with numerous dendritic processes extending into the canaliculi. Through gap junctions at the end of the processes, osteocytes form direct connections with each other and with osteoblasts and bone lining cells at the bone surfaces.

When mechanical loads are applied to tissue, shear stress or strains are produced around cells. Significant evidence exists that mechanical forces applied to bone lead to interstitial fluid flow within the lacunar–canalicular network (a network of intersecting channels within the bone, in which osteocytes and their long dendritic cellular processes reside).[72] Fluid flow is sensed by osteocytes as shear stress. More specifically, a number of studies indicate that shear stress stimulates cellular responses that correspond well to *in vivo* bony responses to applied forces in terms of their frequency and magnitude. Mechanical loads applied to tissues can also be sensed by cells as strain (cell compression, stretch, or deformation of shape). While earlier studies indicated that at the cellular level, physiologic mechanical loads cannot elicit strains of a great enough magnitude to initiate a cellular response,[73,74] more recent studies have indicated that osteocytes experience significantly amplified strain upon mechanical loading of bone, due to the structural properties of bone lacunae and/or the close and regular attachment of the lengthy osteocytic cellular processes to the canalicular bone in which they reside.[75-77]

At present, the precise molecular mechanisms involved in mechanically induced osteocyte excitation are not completely clear, but abundant evidence has suggested that multiple factors and pathways are involved, such as integrins (cell to extracellular matrix adhesion molecules), cytoskeletal structural proteins, purinergic receptors, connexin 43 hemichannels, stretch-sensitive ion channels, voltage-sensitive ion channels, and/or primary cilia (microtubular structures extending from the basal body through the cell membrane into the extracellular space).[76,78-82] Soluble factors released by osteocytes may also play a role in this process known as autocrine stimuli.[83,84]

After sensing the mechanical load, osteocytes send signals to other cells to regulate osteogenesis and osteoclastogenesis. Complex molecular mechanisms are involved in these mechanotransduction processes, which researchers are just beginning to understand. Recent findings have indicated that osteocyte-mediated mechanotransduction is induced by the Wnt signaling pathway but inhibited by the sclerostin (SOST) pathway through gap-junction intercellular communications and/or extracellular cytokines,[85-87] or through soluble factors such as prostaglandins released by osteocytes in a paracrine fashion.[83,84]

In addition to the osteocyte-mediated mechanism, mechanical force can also directly stimulate osteoblasts and their progenitor cells by producing strain (deformation) within the tissues where these cells reside. As detailed in a recent review,[88] mesenchymal stem cells can sense mechanical strain through their cytoskeleton, focal adhesions, and primary cilia. Tissue strain-induced cell stretching can induce the osteoblastic differentiation of preosteoblasts via integrin/focal adhesion kinase signaling and mechanosensitive calcium channels.[89,90] A number of studies have found that cyclic tension stimulates osteogenic lineage commitment and differentiation of mesenchymal stem cells, resulting in enhanced expression of Runx2 and other matrix proteins produced by osteoblasts.[91,92] At the molecular level, mechanotransduction by mesenchymal stem cells may involve several pathways, including mitogen-activated protein kinase (MAPK), Wnt, and RhoA/Rho kinase signaling pathways.

The PDL space is also fluid filled, such that the application of orthodontic force leads to fluid-flow changes within the PDL space. Shear stress from fluid flow can stimulate mesenchymal precursor cell (such as PDL cells) Ca^{2+} signaling, which in turn promotes ATP release, the production of prostaglandin E2 (PGE2), and the proliferation of precursor cells.[72,81] Notably, studies indicate that NO synthase (the enzyme that synthesizes NO) and IL1β are also critical mediators of orthodontic tooth movement.[93-95] These mechanisms could potentially explain the differentiation of PDL preosteoblasts into osteoblasts and initial mineralization along stretched PDL fibers following orthodontic tooth movement.[66]

LOCAL BIOLOGICAL MEDIATORS OF ORTHODONTIC TOOTH MOVEMENT

Despite this gap in our knowledge of the mechanotransduction of orthodontic force, much progress has been made in identifying downstream critical biochemical mediators of orthodontic tooth movement. During quiescence, osteocytes secrete sclerostin, which inhibits Wnt cell signaling, preosteoblastic differentiation, and bone formation.[96] Upon tooth movement, PDL cells, bone lining cells, and/or alveolar bone osteocytes secrete inflammatory cytokines such as TNFα and IL1β, which function to stimulate autocrine (same cell) and paracrine (neighboring cell) cell changes, including the production of additional biological mediators (CSF-1, VEGF, and PGE2). PGE2 release is also stimulated directly by fluid flow–induced shear stress.[80,81] Each of these factors in turn elicits multiple cellular reactions. IL1β acts to propagate the opening of connexin hemichannels in response to mechanical signals.[87] In this manner IL1β may act to amplify the cellular response to mechanical load. IL1β, TNFα, and VEGF stimulate angiogenesis, which increases local vascularity. TNFα, CSF-1, and PGE2 stimulate osteoclastogenesis and bone resorption. Of note, PGE2 also stimulates osteoblastogenesis and bone formation. Together, these local biological mediators elicit changes in cell behavior resulting in increased blood vessel dilation and permeability, mononuclear osteoclastic precursor cell recruitment, and differentiation in regions of compression, as well as preosteoblastic proliferation and differentiation in regions of tension (Fig. 3-66). Evidence for the early local release of these factors following application of an orthodontic force is provided by the fact that gingival crevicular fluid levels of TNFα, IL1β, CSF-1, VEGF, and PGE2

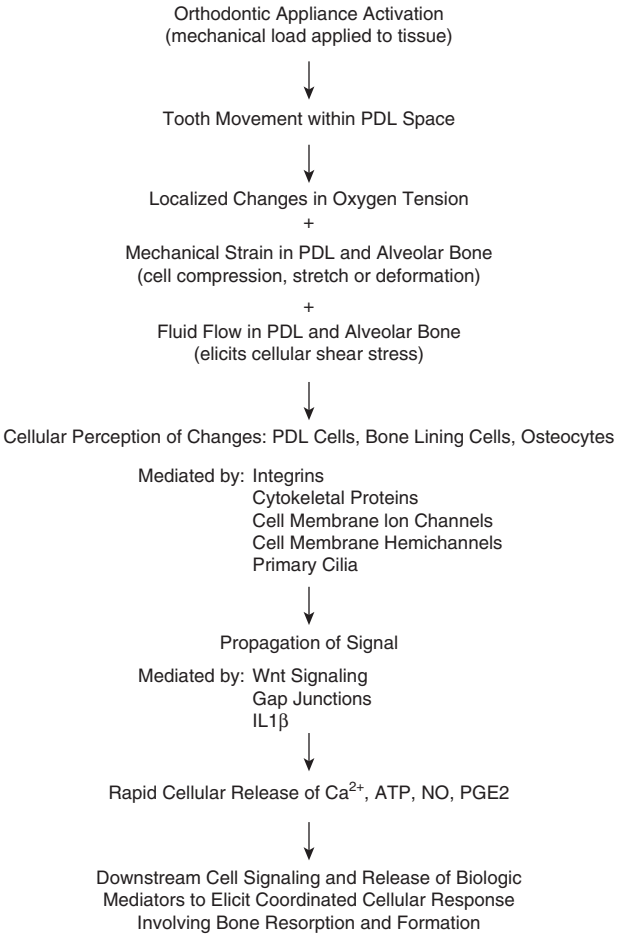


FIGURE 3-65 Mechanotransduction in orthodontic tooth movement. (Hatch NE. The biology of orthodontic tooth movement: current concepts on and applications to clinical practice. In: *Proceedings of the 37th Annual Moyers Symposium*, February, 2010, Ann Arbor, MI. Volume 48, Craniofacial Growth Series. Ann Arbor, MI: Needham Press, Inc., 2011.)

all rise significantly following orthodontic tooth movement in humans.[97-100] Each of these mediators has also previously been shown to be essential for orthodontic tooth movement (Table 3-1).

NEUROPEPTIDES AND ORTHODONTIC TOOTH MOVEMENT

PDL and pulpal nociceptors respond to orthodontic tooth movement by secreting neuropeptides such as Substance P and CGRP (calcitonin gene–related peptide).[101-105] These neuropeptides act to enhance the cellular secretion of inflammatory cytokines and to increase vasodilation and vasopermeability of blood vessels.[106,107] That sensory nerve responses are critical for orthodontic tooth movement is evidenced by studies showing that transection of the inferior alveolar nerve in rats inhibits vascular and tooth movement responses to applied loads.[108-110] While it is tempting to consider utilizing local delivery of neuropeptides to enhance orthodontic tooth movement in humans, the fact that neuropeptides also mediate pain makes this proposition less promising.

RANK/RANKL/OPG SYSTEM FOR CONTROL OF OSTEOCLASTOGENESIS AND TOOTH MOVEMENT

The regulation of osteoclastogenesis by osteoblasts is mediated in large part by the nuclear factor kappa B ligand (RANKL)/nuclear factor kappa B (RANK)/osteoprotegerin (OPG) ligand–receptor complex. RANKL is found on the surface of osteoblast lineage cells where it stimulates osteoclastogenesis by binding to receptor nuclear factor kappa B (RANK), a transmembrane protein located on osteoclast progenitors and osteoclasts. The binding of RANKL to RANK is essential for stimulating osteoclast formation and activity and for promoting osteoclast survival.[111] The interaction of RANKL with RANK is regulated by the soluble decoy receptor OPG, which is secreted by cells of the osteoblastic lineage and functions as a competitive inhibitor of RANKL.[112-114] OPG competes with RANKL for RANK and therefore acts to inhibit osteoclast differentiation, activity, and survival, hence diminishing osteoclastogenesis.

Importantly, several prior studies have demonstrated that the ratio of RANKL to OPG controls osteoclastogenesis and that inhibition of the RANKL/RANK interaction can inhibit bone resorptive activity. Transgenic overexpression of OPG in mice leads to osteopetrosis due to inadequate osteoclasts,[114-116] while a lack of OPG in mice is accompanied by decreased bone density with severely increased trabecular and cortical bone porosity.[117] Rodents administered recombinant OPG protein (OPG-Fc) show a rapid and sustained decrease in bone surface osteoclasts in combination with increased bone mineral density.[118] In humans, subcutaneous injection with OPG-Fc or a monoclonal antibody to RANKL that also functions to inhibit RANKL binding to RANK, contributes to significantly diminished serum markers of bone resorption,[119,120] reduced fracture incidence, and increased bone mineral density in postmenopausal adults.[121-125] In addition, *in vivo* evidence also supports a role for RANKL and OPG in control of mechanically induced bone resorption. For example, OPG administration in rodents inhibits bone loss resulting from mechanical unloading by reducing bone resorptive activity. Together, these studies demonstrate that RANKL and OPG are essential for regulating osteoclast activity and that inhibitors of RANKL can be used to systemically improve bone quality and reduce bone resorption induced by biological or mechanical perturbations of bone.

The RANKL/RANK/OPG system is also an essential component of orthodontic force-induced tooth movement and relapse after orthodontic appliance removal. Previous studies indicate that manipulation of this system can be utilized to control tooth movement and relapse after movement.[126] OPG expression increases in tensed regions of the PDL and alveolar bone while RANKL expression increases in compressed regions of the PDL and alveolar bone following orthodontic tooth movement.[127-132] Alveolar bone resorption is dramatically enhanced following orthodontic tooth movement in OPG null mice.[133] Delivery of OPG through gene transfer or injection of a recombinant protein to alveolar tissues inhibits osteoclastogenesis and orthodontic tooth movement while delivery of RANKL enhances osteoclastogenesis and tooth movement in rats.[134-137] Injection of recombinant OPG protein (OPG-Fc) also inhibits relapse tooth movement after appliance removal.[138] A single local injection of OPG-Fc can prevent relapse of tooth movement beyond the constraints of the tooth socket for up to 1 month after appliance removal compared to a 70% relapse in control animals, with minimal systemic effects. Additional

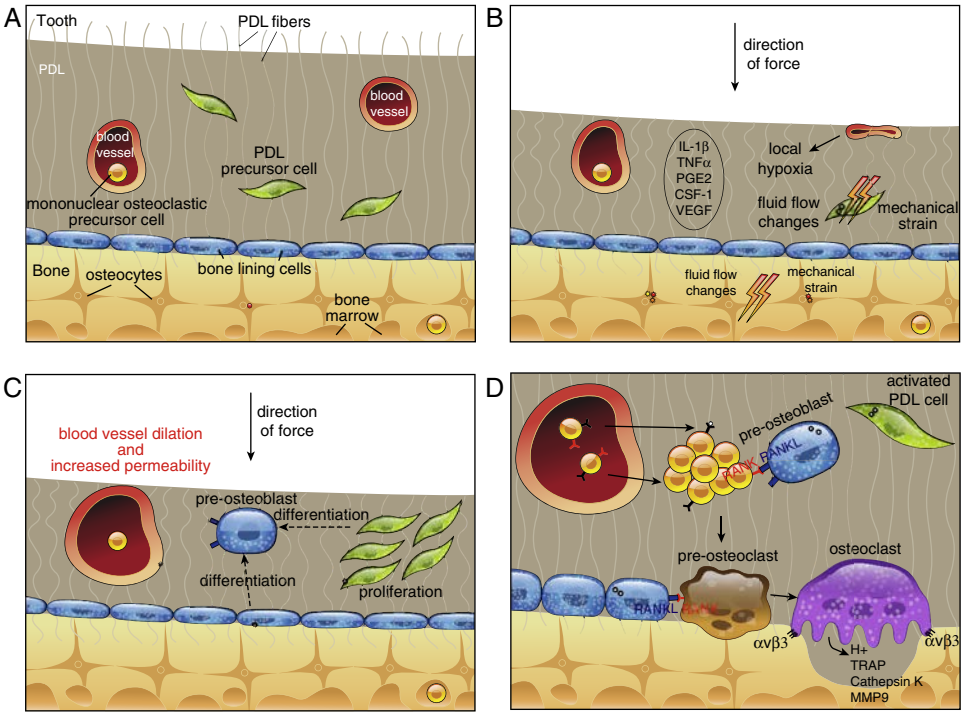


FIGURE 3-66 A, Tooth, PDL space and supporting alveolar bone environment during quiescence. Blood vessels exist in PDL space and is contiguous with bone marrow. Bone marrow exists in alveolar bone. Osteoclast precursor cells are located in bone marrow and circulating through blood vessels. Bone lining cells reside along socket wall/alveolar bone surface. PDL cells (mesenchymal precursor cells) are located within PDL space. During quiescence, osteocytes express and secrete sclerostin, which inhibits pre-osteoblastic (PDL cell and bone lining cell) activity. **B, Cellular mechanotransduction of applied orthodontic force.** Upon application of an orthodontic force, the tooth moves within the PDL space. This tooth movement results in compression of blood vessels leading to localized hypoxia (diminished oxygen tension). Tooth movement within the PDL space also results in mechanical strain (cell compression, stretch or deformation) and fluid flow changes within the PDL and underlying alveolar bone (which induces cellular shear stress). In response to perception of these changes in the physical environment, PDL cell, bone lining cells and/or alveolar bone osteocytes rapidly express and secrete local biologic mediators including IL1b, TNFa, PGE2, CSF-1 and VEGF. **C, Extracellular biologic mediators induce cellular changes.** In response to the secreted factors (small yellow, orange and red stars), cells within the PDL space undergo changes. Endothelial cells that line the blood vessels respond by proliferating and differentiating, leading to increased blood vessel dilation and increased blood vessel permeability. Mesenchymal precursor cells within the PDL space respond by proliferating and differentiating into pre-osteoblasts. Bone lining cells can also respond by proliferating and differentiating into osteoblast precursor cells. Pre-osteoblastic cells express RANK. **D, Recruitment, differentiation and activation of osteoclast precursor cells leads to alveolar bone resorption and tooth movement.** Endothelial cells, osteoblastic precursor cells, bone lining cells and/or osteocytes that have been activated by changes in oxygen tension, mechanical strain and/or shear stress produce and release biologic factors such as TNFa, CSF-1, VEGF and PGE2. Once released, these factors act to recruit mononuclear osteoclastic precursors cells (yellow/orange cells) from the adjacent bone marrow via local permeabilized blood vessels. These osteoclastic precursor cells express receptors for the secreted extracellular factors and, once recruited, are further stimulated by these factors. This stimulation leads to cellular changes including expression of the transmembrane protein, RANK. RANK binds RANKL, a transmembrane protein that is expressed on pre-osteoblasts (blue cell). Binding of RANK with RANKL, in addition to continued stimulation by the other secreted factors, leads to fusion of the mononuclear osteoclastic precursor cells into a multinucleated pre-osteoclast (brown cell). Stimulation of this pre-osteoclast via the RANK/RANKL interaction promotes differentiation of the pre-osteoclast into a mature osteoclast (purple cell). The mature osteoclast adheres tightly to bone via the integrin avb3. Once adherent, the osteoclast secretes acid and enzymes that demineralize the bone and degrade the bone matrix (bone resorption). (Hatch NE. The biology of orthodontic tooth movement: current concepts on and applications to clinical practice. In: *Proceedings of the 37th Annual Moyers Symposium*, February, 2010, Ann Arbor, MI. Volume 48, Craniofacial Growth Series. Ann Arbor, MI: Needham Press, Inc., 2011.)

TABLE 3-1 Local Biological Mediators of Orthodontic Tooth Movement

This table includes information on inflammatory cytokines, growth factors, and prostaglandins that are expressed early following orthodontic appliance application and established as essential for orthodontic tooth movement. Additional mediators of tooth movement exist and include but are not limited to neuropeptides, leukotrienes, and chemokines.

Extracellular Biological Mediator	GCF Expression	Function in Bone Modeling/Remodeling	Role in Orthodontic Tooth Movement
IL1β	Within 1-hour postappliance activation.	Propagate initial response to mechanical signal. Stimulate angiogenesis. Stimulate production/secretion of additional biological factors.	Higher IL1β and lower IL-RA (IL1 receptor antagonist) levels in GCF are associated with faster orthodontic tooth movement in humans. Homozygosity for IL1β polymorphism (A1, A1 at position +3954) is associated with faster orthodontic tooth movement in humans.
TNFα	Within 24-hour postappliance activation.	Stimulate angiogenesis. Stimulate production/secretion of additional biological factors. Stimulates osteoclastogenesis.	TNF receptor knockout mice exhibit diminished osteoclastogenesis and tooth movement.
PGE2	Within 24-hour postappliance activation.	Stimulates inflammatory cytokine expression. Bimodal function: important for bone resorption and bone formation. Stimulates RANKL expression and inhibits OPG expression by preosteoblasts and osteoblasts. Stimulates RANK expression by preosteoclasts and osteoclasts.	Prostaglandin receptor EP4 agonist enhances tooth movement in rats. PGE2 but not PGE1 enhances tooth movement in monkeys. NSAIDs (inhibit COX2 activity) inhibit osteoclastogenesis and orthodontic tooth movement in rodents.
VEGF	Within 24-hour postappliance activation.	Stimulate angiogenesis. Promote recruitment of mononuclear osteoclastic precursor cells from bone marrow. Promote differentiation of osteoclastic precursor cells. Promote osteoclast survival.	Neutralizing antibodies against VEGF inhibit osteoclastogenesis and tooth movement. Local delivery of VEGF enhances osteoclastogenesis and tooth movement.
CSF-1	Within 24-hour postappliance activation.	Stimulate angiogenesis. Promote recruitment of mononuclear osteoclastic precursor cells from bone marrow. Promote differentiation of osteoclastic precursor cells. Promote osteoclast survival.	Neutralizing antibodies against CSF-1 receptor inhibit osteoclastogenesis and tooth movement.

GCF, Gingival crevicular fluid; *IL1β*, interleukin 1 beta; *TNFα*, tumor necrosis factor alpha; *PGE2*, prostaglandin E2; *VEGF*, vascular endothelial growth factor; *CSF-1* (*mCSF*), colony stimulating factor.

studies are required to establish dose levels required for local orthodontic anchorage, inhibition of orthodontic relapse utilizing OPG-Fc in humans, as well as whether pharmacologic inhibition of orthodontic relapse requires sustained or transient inhibition of osteoclasts during a critical time-limited period after orthodontic appliance removal. From a clinical perspective, controlled local inhibition of osteoclast activity could allow for enhanced control of individual teeth during orthodontic treatment and for spatially restricted effects in the prevention of relapse after orthodontic treatment. Yet, because osteoclast activity is essential for normal bone physiology, use of osteoclast inhibitors for local control of bone resorption in otherwise healthy humans will be limited if the delivered protein yields undesirable systemic effects.

BIOLOGICAL CONTROL OF OSTEOGENESIS INVOLVED IN ORTHODONTIC TOOTH MOVEMENT

As the primary cell type in charge of osteogenesis involved in alveolar bone modeling and remodeling, most active osteoblasts at the PDL–bone interface derive from progenitor cells stimulated to differentiate and deposit bone upon the application of orthodontic force (Table 3-2; see also Fig. 4-61). Histologically, osteoblasts are cuboidal-shaped, mononucleated cells with strongly basophilic cytoplasm that appear clustered along bone surfaces. During tooth development, osteoblasts that form the primary alveolar bone largely arise from the neural crest–derived ectomesenchymal stem cells in the first branchial arch.[139] While it remains unknown whether these stem cells remain in the mature periodontium as a postnatal source of progenitor cells for osteoblasts, it is clear that even in adults, the PDL and alveolar bone still possess a steady source of progenitor cells for osteoblasts.[140]

That a main source of osteoprogenitor cells derives from perivascular stem cells (including those around PDL and alveolar bone blood vessels) is well evidenced by several findings. By administering 3H-thymidine into mouse peridontium, which labels dividing cells, McCulloch demonstrated that within 10 μm of blood vessels there is a slowly dividing population of progenitor cells.[141] Subsequently, Roberts et al. found that an osteogenic gradient radiating from blood vessels is present in

TABLE 3-2 Alveolar Bone Cells

Osteoblasts	Osteoclasts
Bone-forming cells	Bone-resorbing cells
Precursors: • mesenchymal precursor cells • PDL • bone lining cells • pericyte precursor cells	Precursors: • bone marrow-derived hematopoietic precursor cells • circulating mononuclear cells
Osteoblasts secrete factors that regulate osteoclasts	Mononuclear precursor cells fuse to form mature multinucleated osteoclasts
Regulated by growth factors (e.g., BMPs, FGFs), cytokines (e.g., IL-1), and hormones (e.g., PTH, vitamin D, estrogen)	Fusion and activation are regulated by osteoblasts (e.g., RANK/RANKL/OPG system, mCSF, prostaglandins)
Embedded osteoblasts become osteocytes	After bone resorption, osteoclasts undergo apoptosis
Express alkaline phosphatase	Express acidic phosphatase

the PDL surrounding rat molars. Less differentiated precursor cells were predominantly localized within 20 μm of the nearest major blood vessel, while cells 30 μm from the vessel wall were undergoing proliferation, differentiation, and migration to the bone surface, where they became osteoblasts.[142] Simultaneously, McCulloch et al. reported that paravascular tissues in endosteal spaces of alveolar bone are enriched with progenitor cells, whose progeny can rapidly migrate into the PDL.[143] More recently, researchers have successfully isolated a cell population from the PDL that expresses numerous cell surface markers indicative of mesenchymal stem cells, including STRO-1, CD146, CD90, CD44, CD105, etc.[144,145]

Another likely source of osteoblasts comes from bone lining cells located at the alveolar bone surface. Bone lining cells are thin, elongated cells with flat or slightly ovoid nuclei that line inactive bone surfaces. They connect with each other via gap junctions and send cell processes to canaliculi at bone surfaces,[146] where they can form connections with cytoplasmic processes of neighboring osteocytes. While it is commonly accepted that bone lining cells are quiet remnants of osteoblasts after they complete bone formation, some evidence has shown that these lining cells may differentiate into osteoblasts upon certain stimulation such as parathyroid hormone (PTH)[147] or mechanical loading.[148]

The differentiation of mesenchymal stem cells into mature osteoblasts is a multistage process, with each stage characterized by specific features of cell morphology, production of extracellular matrices, and gene expression. For a comprehensive understanding of osteoblast differentiation, the readers are referred to these review articles.[149-152] One particularly important aspect about the regulation of osteoblast differentiation is the expression of several key transcription factors. The runt-related transcription factor 2 (Runx2) is indispensable for the commitment of mesenchymal stem cells to the osteogenic lineage. Runx2 also regulates all the early stages of osteoblast differentiation and serves as an upstream regulator for another essential osteoblastic transcription factor, Osterix (Osx). Osx, through both Runx2-dependent and Runx2-independent pathways, promotes differentiation of osteoprogenitor cells into immature osteoblasts. The third important transcription factor is activating transcription factor (ATF4), which through interacting with Runx2, regulates the transcriptional activities of mature osteoblasts. Combined, these transcription factors not only serve as markers of osteoblast differentiation but also play critical roles in regulating osteoblast differentiation and function. By changing the expression of these transcription factors, a number of molecular pathways are involved in regulating osteoblast differentiation. The canonical Wnt signaling pathway, transforming growth factors (TGFs), bone morphogenic proteins (BMPs), fibroblast growth factors (FGFs), gap junction protein connexin 43 (Cx43), and calcium ion (Ca^{2+}) mediated noncanonical Wnt pathways are all likely regulators of osteoblast differentiation.[69,152]

Mature osteoblasts are very versatile cells. In addition to forming bone, they act to regulate osteoclasts and hematopoietic stem cells and also function as endocrine cells.[152] To form bone, osteoblasts first produce a highly collagenous extracellular matrix. This nonmineralized layer of tissue (osteoid) includes many structural and regulatory proteins such as type I collagen, osteopontin, osteocalcin, and bone sialoprotein. Osteoblasts then mineralize the osteoid through a tightly controlled process involving the production of matrix vesicles and a number of enzymes and proteins that function to increase local concentrations of inorganic phosphate. At the surface of osteoblasts and osteoblast-derived matrix vesicles, ectonucleotide pyrophosphatase/phosphodiesterase 1 (Enpp1) generates inorganic pyrophosphate from nucleotides (ATP), which is subsequently converted to inorganic phosphate by the enzyme tissue nonspecific alkaline phosphatase (TNAP/ALP/*Alpl*). Phospho1 (a phosphatase) and the pyrophosphate transporter known as ankylosis protein (Ank), also contribute to this process. These events lead to the precipitation of hydroxyapatite crystals (the mineral component of bone) and the mineralization of bone.

During orthodontic treatment, mechanical forces applied to teeth are transmitted to the PDL and the alveolar bone. Compared to long bones, the tooth–PDL–alveolar bone complex presents a unique environment. Mechanosensing in this environment that subsequently precipitates osteogenesis, especially on the orthodontic tension side, has been a subject of extensive discussion and review recently.[153-156] Briefly, thanks to the great advances of basic research, especially on bone mechanobiology as summarized above, it has become clear that cells in the alveolar bone and the PDL are both playing critical roles in sensing mechanical loading, and in activating osteoblast differentiation and function. Based on current understanding, a schematic drawing was made to depict major cellular events and molecular interactions involved in osteogenesis at the orthodontic tension side (Fig. 3-67). Briefly, within the PDL and surrounding alveolar bone, mesenchymal stem cells, osteoprogenitors, and bone lining cells can sense the strain of extracellular matrix caused by orthodontic tension and subsequently contribute to osteoblast differentiation and function. Both osteoprogenitors and bone lining cells can directly differentiate into osteoblasts to deposit bone along the bone surface. Inside the alveolar bone, osteocytes sense the fluid flow caused by the tensile strain (stretch of the Sharpey's fibers) and subsequently send signals to stimulate bone lining cells and osteoblasts at the surfaces.

At the molecular level, multiple factors and signaling pathways are likely involved in these processes. One of the most important of these is the Wnt pathway. In humans, mutations of the Wnt coreceptors LRP 5/6 are associated with bone loss, osteoporosis (loss-of-function),[157] or bone mass increase

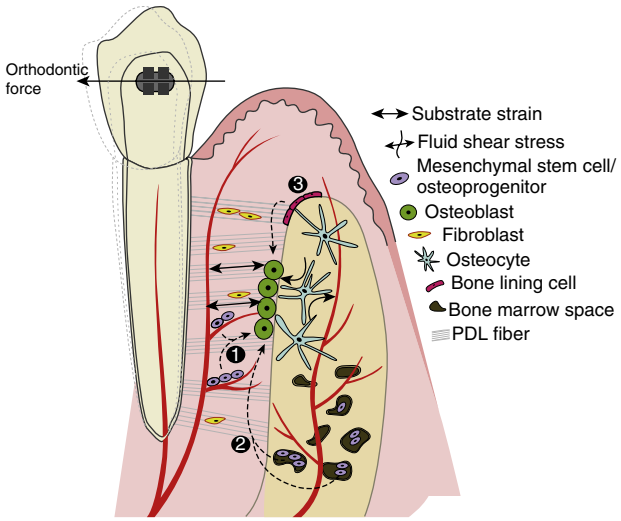


FIGURE 3-67 Osteogenesis at the tension side. Upon receiving mechanical loading, perivascular osteoprogenitor cells in the periodontal ligament and bone marrow, as well as some bone lining cells, can differentiate into osteoblasts and migrate to the bone surface to start forming new bone. Osteocytes inside the bone matrix also sense loading and subsequently regulate the differentiation and function of bone surface cells.

(gain-of-function).[158] Abundant evidence has shown that Wnt signals stimulate the differentiation of osteoblast progenitors[69,152] and periodontal cells.[159] Mechanotransduction from osteocytes to osteoblasts is also thought to be mediated, at least in part, through the Wnt pathway. More specifically, during unloading, osteocytes produce sclerostin, which binds to Wnt coreceptor LRP5/6 on osteoblasts and subsequently through a cascade of signal transduction, prevents translocation of β-catenin into the nucleus, hence inhibiting the gene expression needed for osteoblast function and osteogenesis. Upon sensing mechanical load, osteocytes produce less sclerostin, resulting in relieving the inhibition on osteoblasts and bone formation. Another important molecule is connexin 43, the principal molecular component of mechanosensitive hemichannels and gap junctions. Recent studies have demonstrated that orthodontic force leads to increased connexin 43 expression in alveolar bone osteocytes and bone lining cells,[160,161] suggesting enhanced intercellular communication resulted from orthodontic force.

PHYSICAL METHODS THAT STIMULATE THE BIOLOGY OF ORTHODONTIC TOOTH MOVEMENT

Efforts to enhance orthodontic tooth movement and decrease overall treatment time also include mechanics and/or physical insults to stimulate the cell biologic activity underlying orthodontic tooth movement.

Injury-Facilitated Acceleration of Tooth Movement

The biology underlying injury-facilitated acceleration of tooth movement is generally attributed to the regional acceleratory phenomenon (RAP), a nonspecific, dynamic healing process of bone after sustaining trauma, and this process is generally characterized by upregulated bone remodeling.[162] In support of this notion, evidence from rat studies has confirmed that decortication of the alveolar bone leads to escalated demineralization–remineralization dynamics[163] and transient upregulated expression of multiple cytokines relating bone remodeling.[164]

The concept of using local bone injuries to accelerate tooth movement was initially raised in the 1890s. In the 1950s, Kole further advanced the idea by introducing a surgical procedure that involved vertical cuts of the buccal and lingual alveolar cortical plates (corticotomy) combined with subapical horizontal cuts penetrating the entire alveolus (osteotomy).[165] Due to invasiveness, however, this technique was never popularized. A few decades later, Wilcko et al. revised this surgical procedure by adding bone grafting to the corticotomies. This procedure is now termed *periodontally accelerated osteogenic orthodontics* (PAOO).[166,167] To further reduce surgical invasiveness, cortical bone injuries without reflecting flaps characterized by small and local incisional cuts (called *corticision* or *piezocision*) were subsequently proposed.[168,169] As reviewed by Hoogeveen et al.,[170] to date, a body of clinical studies exists that has attempted to verify the effectiveness of corticotomy-facilitated orthodontic treatment. These studies indicate that surgical corticotomies are safe and possibly effective for shortening the duration of orthodontic treatment. Notably, however, well-conducted prospective clinical studies are still lacking.[170] Similarly, the effectiveness of the more recently proposed corticision or piezocision procedures also needs to be confirmed by high-quality clinical trials.

Vibration-Induced Acceleration of Tooth Movement

In recent years, appliances delivering vibrational forces, such as Acceledent® and Tooth Masseuse, have been introduced to the orthodontic profession as methods to accelerate tooth movement. The biological mechanisms and clinical effectiveness of these vibrational appliances, however, remain largely uncertain. First, data obtained from animal studies are inconsistent and preliminary. On one hand, high-frequency cyclic forces were found to stimulate bone formation and reduce osteoclast density in rabbit craniofacial sutures[171] and to upregulate rat alveolar bone osteogenesis.[172] On the other hand, high-frequency vibrations were also found to accelerate tooth movement in rats[173,174] by upregulating RANKL expression and enhancing bone resorption.[174] To date, only a few clinical studies have been conducted to investigate the effectiveness of vibrational appliances. Although a retrospective study found that an AcceleDent Type I appliance led to faster tooth movement, the comparison was against published norms rather than measurements from a control group without using vibration appliances.[175] More recently, prospective randomized clinical trials of the high-frequency Tooth Masseuse and AcceleDent devices did not accelerate tooth movement.[176,177]

Laser Irradiation-Induced Acceleration of Tooth Movement

Another physical modality that has been proposed for acceleration of tooth movement is low-energy laser irradiation. The last decade has seen a large body of animal studies that investigated the impact of low-energy radiation of orthodontic tooth movement. While many studies found stimulatory effects,[178] several others reported inhibitory effects.[179,180] Similarly, the findings from three clinical studies conducted so far are inconsistent, with acceleration of tooth movement found in two[181,182] but no acceleration of tooth movement in the third study.[183] To date, no randomized clinical trials have been conducted to address the efficacy of laser irradiation for enhancing tooth movement. Nevertheless, several animal studies have attempted to examine the potential biology underlying the

impact of low-energy laser irradiation on orthodontic tooth movement. Data from these animal studies indicate that mechanisms such as stimulation of alveolar bone remodeling,[184] upregulation of matrix metalloproteinase-9, cathepsin K and integrin expression,[178] activation of the RANK/RANKL system,[185] and stimulation of fibronectin and type I collagen expression[186] may mediate the effect of low-energy laser irradiation.

TRANSLATION OF BIOLOGICAL TECHNIQUES INTO ORTHODONTIC PRACTICE: THE FUTURE OF OUR PROFESSION?

Throughout the past century, many orthodontic academics have advocated theories that include an individualized tissue response to orthodontic force application. With more recent advances in biomedicine we may soon see an incorporation of new technologies into private practice that allow for enhanced prediction of a given patient's response to orthodontic force application. In understanding the true potential for translation of this knowledge into clinical practice, it is important to remember that each individual patient is likely to have subtle differences in expression levels and/or function of these mediators. Because the biological mediators of orthodontic tooth movement are encoded by genes and because the sequence of each gene differs among individuals (existence of polymorphisms or normal variations in the genetic code that result in subtle differences in protein expression and/or function), it is very likely that the individual variation seen upon orthodontic appliance activation is due at least in part to these differences. Gingival crevicular fluid expression of biological mediators following orthodontic appliance activation can also diminish with age.[187] Bone modeling, as mediated by osteoblastic and osteoclastic cell function, can also be influenced by hormones, medications, and diet. Significant advances in protein bone biomarkers of osteoclast and osteoblast activity have been accomplished within this past decade. Proteomic analysis of known orthodontic biological mediators and/or bone biomarkers could therefore also provide novel and relevant information for all of our patients. Given the dramatic advances that have been made in the fields of genetic testing and proteomics, it is now possible that the orthodontic records for a given patient could include genetic polymorphic testing (DNA accessed via a buccal swab) and gingival crevicular fluid proteomic analysis. With this information we could better predict tooth movement and relapse for each patient and subsequently provide individualized orthodontic treatment recommendations for each patient. While this may seem far-fetched to some, we will conclude by reminding you that recent studies demonstrate that genetic and proteomic testing for IL1β predicts the speed of orthodontic tooth movement.[95,188] Future work is required to determine if similar tests can be utilized for other known mediators of orthodontic tooth movement and to translate these tests into every orthodontic practice.

REFERENCES

References for Tissue Reactions in Orthodontics

1. Lindhe J, Karring T. In: Lindhe J, ed. *The Anatomy of the Periodontium*. Copenhagen: Munksgaard; 1989.
2. Stein G, Weinmann J. Die physiologische Wanderung der Zähne. *Z Stomatol*. 1925;23:733.
3. Reitan K. Tissue behavior during orthodontic tooth movement. *Am J Orthod*. 1960;46:881.
4. Reitan K. Effects on force magnitude and direction of tooth movement on different alveolar bone types. *Angle Orthod*. 1964;34:244.
5. Rygh P, et al. Activation of the vascular system: a main mediator of periodontal fiber remodeling in orthodontic tooth movement. *Am J Orthod*. 1986;89:453.
6. Sandstedt C. Einige Beiträge zur Theorie der Zahnregulierung. *Nord Tandl Tidskr*. 1905;6:1.
7. Reitan K. The initial tissue reaction incident to orthodontic tooth movement as related to the influence of function. *Acta Odont Scand Suppl*. 1951;6.
8. Reitan K. Tissue reaction as related to the age factor. *J Dent Res*. 1954;74:271.
9. Ten Cate A, Deporter D, Freeman E. The role of fibroblasts in the remodeling of the periodontal ligament during physiologic tooth movement. *Am J Orthod*. 1976;69:155.
10. Rygh P. Ultrastructural vascular changes in pressure zones of rat molar periodontium incident to orthodontic tooth movement. *Scand J Dent Res*. 1972;80:307.
11. Rygh P. Ultrastructural changes in pressure zones of human periodontium incident to orthodontic tooth movement. *Acta Odontal Scand*. 1973;31:109.
12. Rygh P. Ultrastructural cellular reactions in pressure zones of rat molar periodontium incident to orthodontic tooth movement. *Acta Odontol Scand*. 1973;30:575.
13. Rygh P. Ultrastructural changes in tension zones of rat molar periodontium incident to orthodontic tooth movement. *Am J Orthod*. 1974;70:269.
14. Rygh P. Elimination of hyalinized periodontal tissue associated with orthodontic tooth movement. *Scand J Dent Res*. 1974;82:57.
15. Brudvik P, Rygh P. The initial phase of orthodontic root resorption incident to local compression of the PDL. *Eur J Orthod*. 1993;15:249.
16. Brudvik P, Rygh P. Non-clastic cells start orthodontic root resorption in the periphery of hyalinized zones. *Eur J Orthod*. 1993;15:467.
17. Kvam E. Scanning electron microscopy of organic structures on the root surface of human teeth. *Scand J Dent Res*. 1972;80:297.
18. Brudvik P, Rygh P. Root resorption beneath the main hyalinized zone. *Eur J Orthod*. 1994;16:249.
19. Brudvik P, Rygh P. Multi-nucleated cells remove the main hyalinized tissue and start resorption of adjacent root surfaces. *Eur J Orthod*. 1994;16:265.
20. Brudvik P, Rygh P. Transition and determinants of orthodontic root resorption-repair sequence. *Eur J Orthod*. 1995;17:177.
21. Brudvik P, Rygh P. The repair of orthodontic root resorption. An ultrastructural study. *Eur J Orthod*. 1995;17:189.
22. Reitan K. Continuous bodily tooth movement and its histological significance. *Acta Odontol Scand*. 1947;7:115.
23. Stenvik A, Mjör I. Pulp and dentin reactions to experimental tooth intrusion: a histologic study of the initial changes. *Am J Orthod*. 1970;57:370.
24. Mostafa Y, Iskander K, El-Mangoury N. Iatrogenic pulpal reactions to orthodontic extrusion. *Am J Orthod Dentofac Orthop*. 1991;99:30.
25. Batenhorst K, Bowers G, Williams J. Tissue changes resulting from facial tipping and extrusion of incisors in monkeys. *J Periodontol*. 1974;45:660.
26. Steiner G, Pearson J, Ainamo J. Changes of the marginal periodontium as a result of labial tooth movement in monkeys. *J Periodontol*. 1981;52:314.
27. Engelking G, Zachrisson B. Effects of incisor repositioning on monkey periodontium after expansion through the cortical plate. *Am J Orthod*. 1982;82:23.
28. Thilander B, et al. Bone regeneration in alveolar bone dehiscences related to orthodontic tooth movements. *Eur J Orthod*. 1983;5:105.
29. Wennström J, et al. Some periodontal tissue reactions to orthodontic tooth movement in monkeys. *J Clin Periodontol*. 1987;14:121.
30. Lindskog-Stokland B, et al. Orthodontic tooth movement into edentulous areas with reduced bone height: an experimental study in the dog. *Eur J Orthod*. 1993;15:89.
31. Zengo A, Pawluk R, Bassett C. Stress-induced bioelectric potentials in the dentoalveolar complex. *Am J Orthod*. 1973;64:17.
32. Thilander B. Infrabony pockets and reduced alveolar bone height in relation to orthodontic treatment. *Semin Orthod*. 1996;2:55.

33. Yamasaki K, et al. Clinical application of prostaglandin E1 (PGE1) upon orthodontic tooth movement. *Am J Orthod*. 1984;85:508.
34. Roberts W, Chase D. Kinetics of cell proliferation and migration associated with orthodontically induced osteogenesis. *J Dent Res*. 1981;60:174.
35. Davidovitch Z, et al. Cellular localization of cyclicAMP in periodontal tissue during experimental tooth movement in cats. *Calcif Tiss Res*. 1976;18:316.
36. Davidovitch Z, et al. Neurotransmitters, cytokines and the control of alveolar bone remodeling in orthodontics. *Adult Orthod Dent Clin North Am*. 1988;32:411.
37. Sastry SK, Burridge K. Focal adhesions: a nexus for intracellular signaling and cytoskeletal dynamics. *Exp Cell Res*. 2000;261:25.
38. Ingber DE. Integrins as mechanochemical transducers. *Curr Opin Cell Biol*. 1991;3:841.
39. Saito M, et al. Interleukin 1 beta and prostaglandin E are involved in the response of periodontal cells to mechanical stress in vivo and in vitro. *Am J Orthod Dentofacial Orthop*. 1991;99:226.
40. Persson M. Structure and growth of facial sutures. *Odontol Rev Suppl*. 1973:6.
41. Persson M, Thilander B. Palatal suture closure in man from 15 to 35 years of age. *Am J Orthod*. 1977;72:42.
42. Kokich V. Age changes in the human frontozygomatic suture. *Am J Orthod*. 1976;69:411.
43. Persson M, Magnusson B, Thilander B. Suture closure in rabbit and man: a morphological and histochemical study. *J Anat*. 1978;125:313.
44. Follin M, Ericsson I, Thilander B. Orthodontic tooth movement of maxillary incisors through the midpalatal suture area: an experimental study in dogs. *Eur J Orthod*. 1984;6:237.
45. Timms D. *Rapid Maxillary Expansion*. Chicago: Quintessence; 1981.
46. Engström C, Thilander B. Premature facial synostosis: the influence of biomechanical factors in normal and hypocalcemic young rats. *Eur J Orthod*. 1985;7:35.
47. Miyawaki S, Forbes D. The morphologic and biochemical effects of tensile force application to the interparietal sutures of the Sprague-Dawley rat. *Am J Orthod Dentofac Orthop*. 1987;92:123.
48. Yen E, Yue C, Suga D. Effect of force on synthesis of type III and type I collagen in mouse interparietal suture. *J Dent Res*. 1989;68:1746.
49. Meikle M, et al. Molecular biology of stressed connective tissues at sutures and hard tissues *in vitro*. In: Norton LA, Burstone CJ, eds. *The Biology of Tooth Movement*. Boca Raton, FL: CRC Press; 1989:71.
50. Saito S, Shimizu N. Stimulatory effects of low-power laser irradiation on bone regeneration in midpalatal suture during expansion in the rat. *Am J Orthod Dentofac Orthop*. 1997;111:525.
51. Ten Cate A, Freeman E, Dickinson J. Sutural development: structure and its response to rapid separation. *Am J Orthod*. 1977;71:622.
52. Wright D, Moffett B. The postnatal development of the human temporomandibular joint. *Am J Anat*. 1974;141:235.
53. Thilander B, Carlsson GE, Ingervall B. Postnatal development of the human temporomandibular joint. I. A histologic study. *Acta Odontol Scand*. 1976;34:117.
54. Ingervall B, Carlsson GE, Thilander B. Postnatal development of the human temporomandibular joint. II. A microradiographic study. *Acta Odontol Scand*. 1976;34:133.
55. Enlow D. *Facial Growth*. 3rd ed. Philadelphia: WB Saunders; 1990.
56. King G, et al. The timing of treatment for Class II malocclusions in children: a literature review. *Angle Orthod*. 1990;60:87.
57. McNamara J. Neuromuscular and skeletal adaptations to altered function in the orofacial region. *Am J Orthod*. 1973;64:578.
58. Petrovic A, Stutzmann J, Lavergne J. *Mechanisms of Craniofacial Growth and Modus Operandi of Functional Appliances: A Cell-Level and Cybernetic Approach to Orthodontic Decision Making*. Craniofacial Growth Series 23. Ann Arbor: University of Michigan; 1990.
59. Ericsson I, et al. Orthodontic relapse in dentitions with reduced periodontal support. An experimental study in dogs. *Eur J Orthod*. 1980;2:511.
60. Reitan K. Tissue rearrangement during retention of orthodontically rotated teeth. *Angle Orthod*. 1959;29:105.
61. Reitan K. Principles of retention and avoidance of posttreatment relapse. *Am J Orthod*. 1969;55:776.
62. Rönnerman A, Thilander B, Heyden G. Gingival tissue reactions to orthodontic closure of extraction sites: histologic and histochemical studies. *Am J Orthod*. 1980;77:620.
63. Thilander B, Persson M, Adolfsson U. Roentgencephalometric standards for a Swedish population. A longitudinal study between the ages of 5 and 31 years. *Eur J Orthod*. 2005;27:370.
64. Thilander B. Dentoalveolar development in subjects with normal occlusion. A longitudinal study between the ages of 5 and 31 years. *Eur J Orthod*. 2009;31:109.
65. Thilander B, Ödman J, Lekholm U. Orthodontic aspects of the use of oral implants in adolescents: a 10-year follow-up study. *Eur J Orthod*. 2001;23:715.

References for Tooth Movement and the Cellular and Molecular Level

66. Stuteville OH. A summary review of tissue changes incident to tooth movement. *Angle Ortho*. 1938;8:1–20.
67. Schwarz AM. Tissue changes incident to orthodontic tooth movement. *Int J Orthod*. 1932;18:331–352.
68. King GJ, Keeling SD, Wronski TJ. Histomorphometric study of alveolar bone turnover in orthodontic tooth movement. *Bone*. 1991;12:401–409.
69. Bonewald LF, Johnson ML. Osteocytes, mechanosensing and Wnt signaling. *Bone*. 2008;42(4):606–615.
70. Robling AG, Turner CH. Mechanical signaling for bone modeling and remodeling. *Crit Rev Eukaryot Gene Expr*. 2009;19(4):319–338.
71. Govey PM, Loiselle AE, Donahue HJ. Biophysical regulation of stem cell differentiation. *Curr Osteoporos Rep*. 2013;11(2):83–91.
72. Riddle RC, Donahue HJ. From streaming-potentials to shear stress: 25 years of bone cell mechanotransduction. *J Orthop Res*. 2009;27:143–149.
73. Rubin CT, Lanyon LE. Regulation of bone formation by applied dynamic loads. *J Bone Joint Surg Am*. 1984;66:397–402.
74. You J, Yellowley CE, Donahue HJ, Zhang Y, Chen Q, Jacobs CR. Substrate deformation levels associated with routine physical activity are less stimulatory to bone cells relative to loading-induced oscillatory fluid flow. *J Biomech Eng*. 2000;122:387–393.
75. Nicolella DP, Moravits DE, Gale AM, Bonewald LF, Lankford J. Osteocyte lacunae tissue strain in cortical bone. *J Biomech*. 2006;39:1735–1743.
76. McNamara LM, Majeska RJ, Weinbaum S, Friedrich V, Schaffler MB. Attachment of osteocyte cell processes to the bone matrix. *Anat Rec*. 2009;292:355–363.
77. Wang Y, McNamara LM, Schaffler MB, Weinbaum S. Strain amplification and integrin based signaling in osteocytes. *J Musculoskelet Neuronal Interact*. 2008;8:332–334.
78. Allori AC, Sailon AM, Pan JH, Warren SM. Biological basis of bone formation, remodeling, and repair-part III: biomechanical forces. *Tissue Eng Part B Rev*. 2008;14:285–293.
79. Li J, Liu D, Ke HZ, Duncan RL, Turner CH. The P2X7 nucleotide receptor mediates skeletal mechanotransduction. *J Biol Chem*. 2005;280:42952–42959.
80. Siller-Jackson AJ, Burra S, Gu S, et al. Adaptation of connexin 43-hemichannel prostaglandin release to mechanical loading. *J Biol Chem*. 2008;283:26374–26382.
81. Genetos DC, Geist DJ, Liu D, Donahue HJ, Duncan RL. Fluid shear-induced ATP secretion mediates prostaglandin release in MC3T3-E1 osteoblasts. *J Bone Miner Res*. 2005;20:41–49.
82. Temiyasathit S, Jacobs CR. Osteocyte primary cilium and its role in bone mechanotransduction. *Ann N Y Acad Sci*. 2010;1192:422–428.
83. Ajubi NE, Klein-Nulend J, Alblas MJ, Burger EH, Nijweide PJ. Signal transduction pathways involved in fluid flow-induced PGE2 production by cultured osteocytes. *Am J Physiol*. 1999;276(1 Pt 1):E171–E178.
84. Cheng B, Kato Y, Zhao S, et al. PGE(2) is essential for gap junction-mediated intercellular communication between osteocyte-like MLO-Y4 cells in response to mechanical strain. *Endocrinology*. 2001;142(8):3464–3473.
85. Jiang JX, Siller-Jackson AJ, Burra S. Roles of gap junctions and hemichannels in bone cell functions and in signal transmission of mechanical stress. *Front Biosci*. 2007;12:1450–1462.

86. Yellowley CE, Li Z, Zhou Z, Jacobs CR, Donahue HJ. Functional gap junctions between osteocytic and osteoblastic cells. *J Bone Miner Res*. 2000;15:209–217.
87. Salter DM, Wallace WH, Robb JE, Caldwell H, Wright MO. Human bone cell hyperpolarization response to cyclical mechanical strain is mediated by an interleukin-1beta autocrine/paracrine loop. *J Bone Miner Res*. 2000;15:1746–1755.
88. Chen JC, Jacobs CR. Mechanically induced osteogenic lineage commitment of stem cells. *Stem Cell Res Ther*. 2013;4(5):107.
89. Ward Jr DF, Williams WA, Schapiro NE, et al. Focal adhesion kinase signaling controls cyclic tensile strain enhanced collagen I-induced osteogenic differentiation of human mesenchymal stem cells. *Mol Cell Biomech*. 2007;4:177–188.
90. Robinson JA, Chatterjee-Kishore M, Yaworsky PJ, et al. Wnt/beta-catenin signaling is a normal physiological response to mechanical loading in bone. *J Biol Chem*. 2006;281:31720–31728.
91. Haasper C, Jagodzinski M, Drescher M, et al. Cyclic strain induces FosB and initiates osteogenic differentiation of mesenchymal cells. *Exp Toxicol Pathol*. 2008;59(6):355–363.
92. Kearney EM, Farrell E, Prendergast PJ, Campbell VA. Tensile strain as a regulator of mesenchymal stem cell osteogenesis. *Ann Biomed Eng*. 2010;38(5):1767–1779.
93. Hayashi K, Igarashi K, Miyoshi K, Shinoda H, Mitani H. Involvement of nitric oxide in orthodontic tooth movement in rats. *Am J Orthod Dentofacial Orthop*. 2002;122:306–309.
94. Iwasaki LR, Gibson CS, Crouch LD, Marx DB, Pandey JP, Nickel JC. Speed of tooth movement is related to stress and IL-1 gene polymorphisms. *Am J Orthod Dentofacial Orthop*. 2006;130:698. e1–9.
95. Iwasaki LR, Chandler JR, Marx DB, Pandey JP, Nickel JC. IL-1 gene polymorphisms, secretion in gingival crevicular fluid, and speed of human orthodontic tooth movement. *Orthod Craniofac Res*. 2009;12:129–140.
96. Robling AG, Niziolek PJ, Baldridge LA, et al. Mechanical stimulation of bone in vivo reduces osteocyte expression of Sost/sclerostin. *J Biol Chem*. 2008;283:5866–5875.
97. Uematsu S, Mogil M, Deguchi T. Interleukin (IL)-1, IL-6, tumor necrosis factor-α, epidermal growth factor, and β2-microglobulin levels are elevated in gingival crevicular fluid during human orthodontic tooth movement. *J Dent Res*. 1996;75:562–567.
98. Grieve WG, Johnson GK, Moore RN, Reinhardt RA, DuBois LM. Prostaglandin E (PGE) and interleukin-1 beta (IL-1 beta) levels in gingival crevicular fluid during human orthodontic tooth movement. *Am J Orthod Dentofacial Orthop*. 1994;105:369–374.
99. Lee K, Park Y, Yu H, Choi S, Yoo Y. Effects of continuous and interrupted orthodontic force on interleukin-1 and prostaglandin E2 production in gingival crevicular fluid. *Am J Orthod Dentofacial Orthop*. 2004;125:168–177.
100. Kaku M, Motokawa M, Tohma Y, et al. VEGF and M-CSF levels in periodontal tissue during tooth movement. *Biomed Res*. 2008;29:181–187.
101. Kvinnsland I, Kvinnsland S. Changes in CGRP-immunoreactive nerve fibres during experimental tooth movement in rats. *Eur J Orthod*. 1990;12:320–329.
102. Nicolay OF, Davidovitch Z, Shanfeld JL, Alley K. Substance P immunoreactivity in periodontal tissues during orthodontic tooth movement. *Bone Miner*. 1990;11:19–29.
103. Norevall LI, Forsgren S, Matsson L. Expression of neuropeptides (CGRP, substance P) during and after orthodontic tooth movement in the rat. *Eur J Orthod*. 1995;17:311–325.
104. Vandevska-Radunovic V, Kvinnsland IH, Kvinnsland S, Jonsson R. Immunocompetent cells in rat periodontal ligament and their recruitment incident to experimental orthodontic tooth movement. *Eur J Oral Sci*. 1997;105:36–44.
105. Dudic A, Kiliaridis S, Mombelli A, Giannopoulou C. Composition changes in gingival crevicular fluid during orthodontic tooth movement: comparisons between tension and compression sides. *Eur J Oral Sci*. 2006;114:416–422.
106. Hall M, Masella R, Meister M. PDL neuron-associated neurotransmitters in orthodontic tooth movement: identification and proposed mechanism of action. *Today's FDA*. 2001;13:24–25.
107. Yamaguchi M, Kojima T, Kanekawa M, Aihara N, Nogimura A, Kasai K. Neuropeptides stimulate production of interleukin-1 beta, interleukin-6, and tumor necrosis factor-alpha in human dental pulp cells. *Inflamm Res*. 2004;53:199–204.
108. Vandevska-Radunovic V, Kvinnsland IH, Kvinnsland S. Effect of inferior alveolar nerve axotomy on periodontal and pulpal blood flow subsequent to experimental tooth movement in rats. *Acta Odontol Scand*. 1998;56:57–64.
109. Vandevska-Radunovic V. Neural modulation of inflammatory reactions in dental tissues incident to orthodontic tooth movement. A review of the literature. *Eur J Orthod*. 1999;21:231–247.
110. Yamashiro T, Fujiyama K, Fujiyoshi Y, Inaguma N, Takano-Yamamoto T. Inferior alveolar nerve transection inhibits increase in osteoclast appearance during experimental tooth movement. *Bone*. 2000;26:663–669.
111. Anderson DM, Maraskovsky E, Billingsley WL, et al. A homologue of the TNF receptor and its ligand enhance T-cell growth and dendritic-cell function. *Nature*. 1997;390:175–179.
112. Dougall WC, Glaccum M, Charrier K, et al. RANK is essential for osteoclast and lymph node development. *Genes Dev*. 1999;13(18):2412–2424.
113. Li J, Sarosi I, Yan XQ, et al. RANK is the intrinsic hematopoietic cell surface receptor that controls osteoclastogenesis and regulation of bone mass and calcium metabolism. *Proc Natl Acad Sci U S A*. 2000;97(4):1566–1571.
114. Simonet WS, Lacey DL, Dunstan CR, et al. Osteoprotegerin: a novel secreted protein involved in the regulation of bone density. *Cell*. 1997;89:309–319.
115. Min H, Morony S, Sarosi I, et al. Osteoprotegerin reverses osteoporosis by inhibiting endosteal osteoclasts and prevents vascular calcification by blocking a process resembling osteoclastogenesis. *J Exp Med*. 2000;192:463–474.
116. Ominsky MS, Li X, Asuncion FJ, et al. RANKL inhibition with osteoprotegerin increases bone strength by improving cortical and trabecular bone architecture in ovariectomized rats. *J Bone Miner Res*. 2008;23:672–682.
117. Bucay N, Sarosi I, Dunstan CR, et al. Osteoprotegerin-deficient mice develop early onset osteoporosis and arterial calcification. *Genes Dev*. 1998;12(9):1260–1268.
118. Capparelli C, Morony S, Warmington K, et al. Sustained antiresorptive effects after a single treatment with human recombinant osteoprotegerin (OPG): a pharmacodynamic and pharmacokinetic analysis in rats. *J Bone Miner Res*. 2003;18:852–858.
119. Bekker PJ, Holloway D, Nakanishi A, et al. The effect of a single dose of osteoprotegerin in postmenopausal women. *J Bone Miner Res*. 2001;16:348–360.
120. Bekker PJ, Holloway DL, Rasmussen AS, et al. A single-dose placebo-controlled study of AMG 162, a fully human monoclonal antibody to RANKL, in postmenopausal women. *J Bone Miner Res*. 2004;19:1059–1066.
121. McClung MR, Lewiecki EM, Cohen SB, et al. AMG 162 Bone Loss Study Group. Denosumab in postmenopausal women with low bone mineral density. *N Engl J Med*. 2006;354(8):821–831.
122. Lewiecki EM, Miller PD, McClung MR, AMG 162 Bone Loss Study Group, et al. Two-year treatment with denosumab (AMG 162) in a randomized phase 2 study of postmenopausal women with low BMD. *J Bone Miner Res*. 2007;22(12):1832–1841.
123. Cummings SR, San Martin J, McClung MR, FREEDOM Trial, et al. Denosumab for prevention of fractures in postmenopausal women with osteoporosis. *N Engl J Med*. 2009;361(8):756–765.
124. McClung MR, Lewiecki EM, Geller ML, et al. Effect of denosumab on bone mineral density and biochemical markers of bone turnover: 8-year results of a phase 2 clinical trial. *Osteoporos Int*. 2013;24(1):227–235.
125. Orwoll E, Teglbjærg CS, Langdahl BL, et al. A randomized, placebo-controlled study of the effects of denosumab for the treatment of men with low bone mineral density. *J Clin Endocrinol Metab*. 2012;97(9):3161–3169.
126. Yamaguchi M. RANK/RANKL/OPG during orthodontic tooth movement. *Orthod Craniofac Res*. 2009;12:113–119.

127. Shiotani A, Shibasaki Y, Sasaki T. Localization of receptor activator of NFkappaB ligand, RANKL, in periodontal tissues during experimental movement of rat molars. *J Electron Microsc*. 2001;50:365–369.
128. Nishijima Y, Yamaguchi M, Kojima T, Aihara N, Nakajima R, Kasai K. Levels of RANKL and OPG in gingival crevicular fluid during orthodontic tooth movement and effect of compression force on releases from periodontal ligament cells in vitro. *Orthod Craniofac Res*. 2006;9:63–70.
129. Garlet TP, Coelho U, Silva JS, Garlet GP. Cytokine expression pattern in compression and tension sides of the periodontal ligament during orthodontic tooth movement in humans. *Eur J Oral Sci*. 2007;115: 355–362.
130. Garlet TP, Coelho U, Repeke CE, Silva JS, Cunha Fde Q, Garlet GP. Differential expression of osteoblast and osteoclast chemoattractants in compression and tension sides during orthodontic movement. *Cytokine*. 2008;42:330–335.
131. Tan L, Ren Y, Wang J, et al. Osteoprotegerin and ligand of receptor activator of nuclear factor kappaB expression in ovariectomized rats during tooth movement. *Angle Orthod*. 2009;79:292–298.
132. Brooks PJ, Nilforoushan D, Manolson MF, Simmons CA, Gong SG. Molecular markers of early orthodontic tooth movement. *Angle Orthod*. 2009;79:1108–1113.
133. Oshiro T, Shiotani A, Shibasaki Y, Sasaki T. Osteoclast induction in periodontal tissue during experimental movement of incisors in osteoprotegerin-deficient mice. *Anat Rec*. 2002;266:218–225.
134. Kanzaki H, Chiba M, Takahashi I, Haruyama N, Nishimura M, Mitani H. Local OPG gene transfer to periodontal tissue inhibits orthodontic tooth movement. *J Dent Res*. 2004;83:920–925.
135. Kanzaki H, Chiba M, Arai K, et al. Local RANKL gene transfer to the periodontal tissue accelerates orthodontic tooth movement. *Gene Ther*. 2006;13:678–685.
136. Dunn MD, Park CH, Kostenuik PJ, Kapila S, Giannobile WV. Local delivery of osteoprotegerin inhibits mechanically mediated bone modeling in orthodontic tooth movement. *Bone*. September 2007;41(3):446–455. Epub 2007 May 8.
137. Smith SM. *Local Delivery of Recombinant RANKL Protein Enhances Orthodontic Tooth Movement and Root Resorption in Sprague Dawley Rats* (M.S. dissertation). University of Michigan, School of Dentistry, Department of Orthodontics and Pediatric Dentistry; 2012.
138. Hudson JB, Hatch N, Hayami T, et al. Local delivery of recombinant osteoprotegerin enhances postorthodontic tooth stability. *Calcif Tissue Int*. April 2012;90(4):330–342.
139. Ten Cate AR. The development of the periodontium–a largely ectomesenchymally derived unit. *Periodontol 2000*. 1997;13:9–19.
140. Lekic P, McCulloch CA. Periodontal ligament cell population: the central role of fibroblasts in creating a unique tissue. *Anat Rec*. 1996;245(2): 327–341.
141. McCulloch CA. Progenitor cell populations in the periodontal ligament of mice. *Anat Rec*. 1985;211(3):258–262.
142. Roberts WE, Wood HB, Chambers DW, Burk DT. Vascularly oriented differentiation gradient of osteoblast precursor cells in rat periodontal ligament: implications for osteoblast histogenesis and periodontal bone loss. *J Periodontal Res*. 1987;22(6):461–467.
143. McCulloch CA, Nemeth E, Lowenberg B, Melcher AH. Paravascular cells in endosteal spaces of alveolar bone contribute to periodontal ligament cell populations. *Anat Rec*. 1987;219(3):233–242.
144. Seo BM, Miura M, Gronthos S, et al. Investigation of multipotent postnatal stem cells from human periodontal ligament. *Lancet*. 2004;364(9429):149–155.
145. Trubiani O, Di Primio R, Traini T, et al. Morphological and cytofluorimetric analysis of adult mesenchymal stem cells expanded ex vivo from periodontal ligament. *Int J Immunopathol Pharmacol*. 2005;18(2):213–221.
146. Miller SC, de Saint-Georges L, Bowman BM, Jee WS. Bone lining cells: structure and function. *Scanning Microsc*. 1989;3(3):953–960, discussion 960–961.
147. Kim SW, Pajevic PD, Selig M, et al. Intermittent parathyroid hormone administration converts quiescent lining cells to active osteoblasts. *J Bone Miner Res*. 2012;27(10):2075–2084.
148. Chow JW, Wilson AJ, Chambers TJ, Fox SW. Mechanical loading stimulates bone formation by reactivation of bone lining cells in 13-week-old rats. *J Bone Miner Res*. 1998;13(11):1760–1767.
149. Termine JD, Robey PG. Bone matrix proteins and the mineralization process. In: Favus MJ, ed. *Primer on the Metabolic Bone Diseases and Disorders and Mineral Metabolism*. Philadelphia, PA: Lippincott-Raven; 1996.
150. Aubin JE. Advances in the osteoblast lineage. *Biochem Cell Biol*. 1998;76(6):899–910.
151. Franceschi RT, Ge C, Xiao G, Roca H, Jiang D. Transcriptional regulation of osteoblasts. *Cells Tissues Organs*. 2009;189(suppl 1-4):144–152.
152. Capulli M, Paone R, Rucci N. Osteoblast and osteocyte: games without frontiers. *Arch Biochem Biophys*. 2014;561:3–12.
153. Henneman S, Von den Hoff JW, Maltha JC. Mechanobiology of tooth movement. *Eur J Orthod*. 2008;30(3):299–306.
154. Wise GE, King GJ. Mechanisms of tooth eruption and orthodontic tooth movement. *J Dent Res*. 2008;87(5):414–434.
155. Krishnan V, Davidovitch Z. On a path to unfolding the biological mechanisms of orthodontic tooth movement. *J Dent Res*. 2009;88(7): 597–608.
156. Huang H, Williams RC, Kyrkanides S. Accelerated orthodontic tooth movement: molecular mechanisms. *Am J Orthod Dentofacial Orthop*. 2014;146(5):620–632.
157. Gong Y, Slee RB, Fukai N, Osteoporosis-Pseudoglioma Syndrome Collaborative, et al. LDL receptor-related protein 5 (LRP5) affects bone accrual and eye development. *Cell*. 2001;107(4):513–523.
158. Boyden LM, Mao J, Belsky J, et al. High bone density due to a mutation in LDL-receptor-related protein 5. *N Engl J Med*. 2002;346(20): 1513–1521.
159. Premaraj S, Souza I, Premaraj T. Mechanical loading activates beta-catenin signaling in periodontal ligament cells. *Angle Orthod*. 2011;81(4):592–599.
160. Su M, Borke JL, Donahue HJ, et al. Expression of connexin 43 in rat mandibular bone and periodontal ligament (PDL) cells during experimental tooth movement. *J Dent Res*. 1997;76:1357–1366.
161. Gluhak-Heinrich J, Gu S, Pavlin D, Jiang JX. Mechanical loading stimulates expression of connexin 43 in alveolar bone cells in the tooth movement model. *Cell Commun Adhes*. 2006;13:115–125.
162. Frost HM. The biology of fracture healing. An overview for clinicians. Part I. *Clin Orthop Relat Res*. 1989;(248):283–293.
163. Sebaoun JD, Kantarci A, Turner JW, Carvalho RS, Van Dyke TE, Ferguson DJ. Modeling of trabecular bone and lamina dura following selective alveolar decortication in rats. *J Periodontol*. 2008;79(9): 1679–1688.
164. Teixeira CC, Khoo E, Tran J, et al. Cytokine expression and accelerated tooth movement. *J Dent Res*. 2010;89(10):1135–1141.
165. Kole H. Surgical operations on the alveolar ridge to correct occlusal abnormalities. *Oral Surg Oral Med Oral Pathol*. 1959;12(5):515–529. concl.
166. Wilcko WM, Wilcko T, Bouquot JE, Ferguson DJ. Rapid orthodontics with alveolar reshaping: two case reports of decrowding. *Int J Periodontics Restorative Dent*. 2001;21(1):9–19.
167. Murphy KG, Wilcko MT, Wilcko WM, Ferguson DJ. Periodontal accelerated osteogenic orthodontics: a description of the surgical technique. *J Oral Maxillofac Surg*. 2009;67(10):2160–2166.
168. Kim SJ, Park YG, Kang SG. Effects of corticision on paradental remodeling in orthodontic tooth movement. *Angle Orthod*. 2009;79(2):284–291.
169. Keser EI, Dibart S. Sequential piezocision: a novel approach to accelerated orthodontic treatment. *Am J Orthod Dentofacial Orthop*. 2013;144(6):879–889.
170. Hoogeveen EJ, Jansma J, Ren Y. Surgically facilitated orthodontic treatment: a systematic review. *Am J Orthod Dentofacial Orthop*. 2014;145(suppl 4):S51–S64.
171. Peptan AI, Lopez A, Kopher RA, Mao JJ. Responses of intramembranous bone and sutures upon in vivo cyclic tensile and compressive loading. *Bone*. 2008;42(2):432–438.
172. Alikhani M, Khoo E, Alyami B, et al. Osteogenic effect of high-frequency acceleration on alveolar bone. *J Dent Res*. 2012;91(4):413–419.
173. Darendeliler MA, Zea A, Shen G, Zoellner H. Effects of pulsed electromagnetic field vibration on tooth movement induced by magnetic and mechanical forces: a preliminary study. *Aust Dent J*. 2007;52(4):282–287.

174. Nishimura M, Chiba M, Ohashi T, et al. Periodontal tissue activation by vibration: intermittent stimulation by resonance vibration accelerates experimental tooth movement in rats. *Am J Orthod Dentofacial Orthop*. 2008;133(4):572–583.
175. Kau CH, Nguyen JT, English JD. The clinical evaluation of a novel cyclical force generating device in orthodontics. *Orthodontic Practice US*. 2010;1(1).
176. Miles P, Smith H, Weyant R, Rinchuse DJ. The effects of a vibrational appliance on tooth movement and patient discomfort: a prospective randomised clinical trial. *Aust Orthod J*. 2012;28(2):213–218.
177. Woodhouse NR, DiBiase AT, Johnson N, Slipper C, Grant J, Alsaleh M, Donaldson AN, Cobourne MT. Supplemental vibrational force during orthodontic alignment: a randomized trial. *J Dent Res*. 2015 May;94(5):682–689.
178. Yamaguchi M, Hayashi M, Fujita S, et al. Low-energy laser irradiation facilitates the velocity of tooth movement and the expressions of matrix metalloproteinase-9, cathepsin K, and alpha(v) beta(3) integrin in rats. *Eur J Orthod*. 2010;32(2):131–139.
179. Seifi M, Shafeei HA, Daneshdoost S, Mir M. Effects of two types of low-level laser wave lengths (850 and 630 nm) on the orthodontic tooth movements in rabbits. *Lasers Med Sci*. 2007;22(4):261–264.
180. Kim SJ, Moon SU, Kang SG, Park YG. Effects of low-level laser therapy after corticision on tooth movement and paradental remodeling. *Lasers Surg Med*. 2009;41(7):524–533.
181. Cruz DR, Kohara EK, Ribeiro MS, Wetter NU. Effects of low-intensity laser therapy on the orthodontic movement velocity of human teeth: a preliminary study. *Lasers Surg Med*. 2004;35(2):117–120.
182. Youssef M, Ashkar S, Hamade E, Gutknecht N, Lampert F, Mir M. The effect of low-level laser therapy during orthodontic movement: a preliminary study. *Lasers Med Sci*. 2008;23(1):27–33.
183. Limpanichkul W, Godfrey K, Srisuk N, Rattanayatikul C. Effects of low-level laser therapy on the rate of orthodontic tooth movement. *Orthod Craniofac Res*. 2006;9(1):38–43.
184. Yoshida T, Yamaguchi M, Utsunomiya T, et al. Low-energy laser irradiation accelerates the velocity of tooth movement via stimulation of the alveolar bone remodeling. *Orthod Craniofac Res*. 2009;12(4):289–298.
185. Fujita S, Yamaguchi M, Utsunomiya T, Yamamoto H, Kasai K. Low-energy laser stimulates tooth movement velocity via expression of RANK and RANKL. *Orthod Craniofac Res*. 2008;11(3):143–155.
186. Kim YD, Kim SS, Kim SJ, Kwon DW, Jeon ES, Son WS. Low-level laser irradiation facilitates fibronectin and collagen type I turnover during tooth movement in rats. *Lasers Med Sci*. 2010;25(1):25–31.
187. Kawasaki K, Takahashi T, Yamaguchi M, Kasai K. Effects of aging on RANKL and OPG levels in gingival crevicular fluid during orthodontic tooth movement. *Orthod Craniofac Res*. 2006;9:137–142.
188. Iwasaki LR, Crouch LD, Tutor A, et al. Tooth movement and cytokines in gingival crevicular fluid and whole blood in growing and adult subjects. *Am J Orthod Dentofacial Orthop*. 2005;128:483–491.

4

Bone Physiology, Metabolism, and Biomechanics in Orthodontic Practice

Wilbur Eugene Roberts and Sarandeep Singh Huja

OUTLINE

Osteology, 100
Differential Osteology of the Maxilla and Mandible, 100
Temporomandibular Articulation, 101
Bone Physiology, 104
Specific Assessment Methodology, 104
Classification of Bone Tissue, 107
Woven Bone, 107
Lamellar Bone, 107
Composite Bone, 108
Bundle Bone, 108
Skeletal Adaptation: Remodeling and Modeling, 108
Bone Remodeling, 108
Bone Modeling, 110
Cortical Bone Growth and Maturation, 111
Cutting and Filling Cones, 113
Structural and Metabolic Fractions, 113
Bone Metabolism, 116
Calcium Conservation, 117
Endocrinology, 117
Metabolic Bone Disease, 117
Biomechanics, 118
Sutures, 119
Distraction Osteogenesis, 122
Orthodontic Tooth Movement, 123
Occlusal Trauma and Root Resorption, 124
Remodeling/Repair of Root, 125
Induction of the Tooth Movement Response, 125
Periodontal Ligament Response, 127
Osteoblast Histogenesis and Bone Formation, 130
Osteoclast Recruitment and Bone Resorption, 130
Intermittent versus Continuous Mechanics, 131
Differential Anchorage, 131
Rate of Tooth Movement, 132
Periodontitis and Orthodontics, 135
Endosseous Implants, 136
Retromolar Implant Anchorage, 138
Current Status of Miniscrew Implants, 140
Osseointegration, 141
Bone Contact, 142
Bone Remodeling, 142
Failure of Miniscrew Implants—Design or Unique Biological Constraints?, 142
Rigidity of Miniscrew Implants, 143
Animal Models for Studying Bone Adaptation, Remodeling, and Modeling, 143
Expedited Tooth Movement, 145
Regional Acceleratory Phenomena, 147
Bone Remodeling Rate, 147
Current Evidence of Expedited Tooth Movement from Experimental Studies on Rodents and Canines, 148
Summary, 149
Acknowledgments, 149

The physiologic mediators of orthodontic therapy are the facial sutures, the temporomandibular joint (TMJ), the alveolar bone, and the periodontal ligament (PDL). The PDL is the osteogenic bone–tooth interface; it is a modified periosteum with remarkable bone resorptive and formative capabilities. By means of the teeth, alveolar bone can be loaded in a noninvasive and relatively atraumatic manner. Clinical therapy is a combination of orthodontics (tooth movement) and orthopedics (relative repositioning of bones). The biomechanical response depends on the magnitude, direction, and frequency of applied load. Cell kinetic and multiple fluorochrome bone-labeling studies have helped define the fundamental mechanisms of orthodontic and dentofacial orthopedic responses. This chapter explores the determinants of craniofacial bone morphology, mechanically mediated osteogenesis, and physiologic mechanisms for therapeutic correction of malocclusion.

Bones have fascinated human beings since the dawn of time. Much of what is known about the evolution of vertebrates is based on the ordered recovery of bones and teeth from the soil. Over the millennia, these "relatively inert" structures tended to be well preserved. Compared with living bone, teeth are relatively inert structures. Accretion capability at the dentin–pulp interface (secondary dentin) is limited, and some turnover of the cementum occurs because of root resorption and cementum repair processes. The enamel is a hard and inert structure. However, bone is a dynamic structure that is adapting constantly to its environment. As a reservoir of calcium, bone remodeling (physiologic turnover) performs a critical life-support role in mineral metabolism (Fig. 4-1). In addition, the skeleton is the structural scaffold of the body. Collectively bones are essential elements for locomotion, antigravity support, and life-sustaining functions such as mastication. Mechanical adaptation of bone is the physiologic basis of orthodontics and dentofacial orthopedics. A detailed

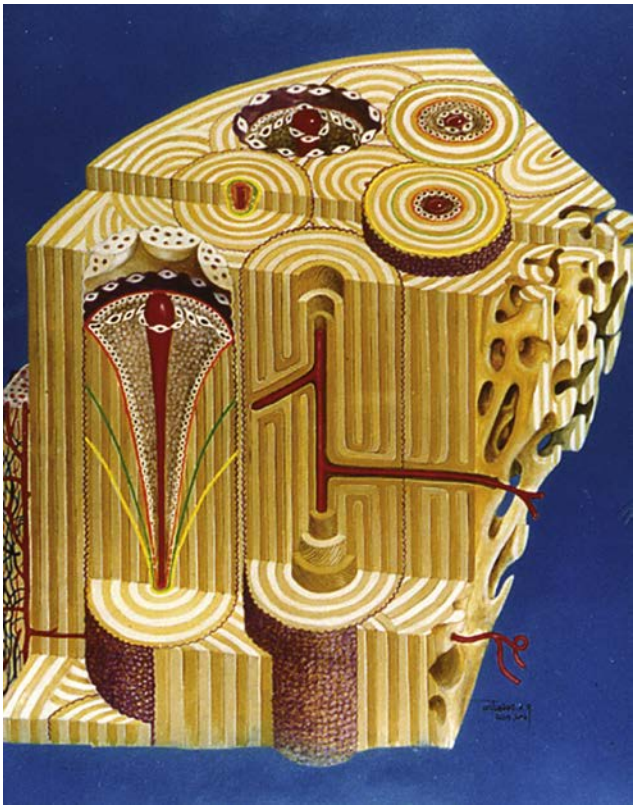


FIGURE 4-1 This artist's rendition of the dynamic principles of cortical bone remodeling was produced by the renowned dental illustrator Rolando De Castro. Remodeling is a vascularly mediated process of bone turnover that maintains the integrity of structural support and is a source of metabolic calcium. Osteoblasts are derived from preosteoblasts circulating in the blood, and perivascular mesenchymal cells give rise to osteoblasts. Note the three colored chevrons (*yellow*, *green*, and *orange*) progressively marking the mineralization front of the evolving second osteon that is moving superiorly on the left. (From Roberts WE, Arbuckle GR, Simmons KE. What are the risk factors of osteoporosis? Assessing bone health. *J Am Dent Assoc*. 1991;122(2):59–61.)

knowledge of the dynamic nature of bone physiology and biomechanics is essential for modern clinical practice.

OSTEOLOGY

In defining the physiologic basis of orthodontics, it is important to initially consider the bone morphology (osteology) of the craniofacial complex. Through systematic study of a personal collection of more than 1000 human skulls, Spencer Atkinson provided the modern basis of craniofacial osseous morphology as it relates to the biomechanics of stomatognathic function. A frontal section of an adult skull shows the bilateral symmetry of bone morphology and functional loading (Figs. 4-2 and 4-3). Because the human genome contains genes to pattern the structure of only half of the body, the contralateral side is a mirror image. Consequently, normal development of the head is symmetric; that is, unilateral structures are on the midline, and bilateral structures are equidistant from it. As shown in Figure 4-3, the vertical components of the cranium tend to be loaded in compression (negative stress), and the horizontal components are loaded in

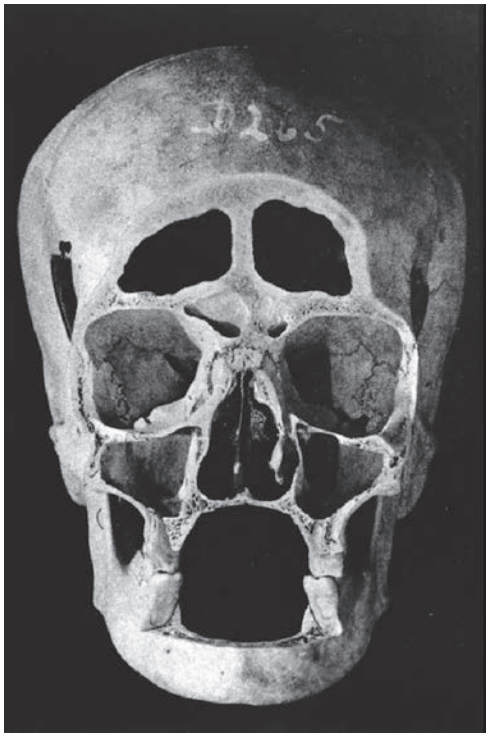


FIGURE 4-2 Frontal section of a human skull in the plane of the first molars. (From Atkinson SR. Balance: the magic word. *Am J Orthod*. 1964;50:189.)

tension (positive stress). From an engineering perspective, the internal skeletal structure of the midface is similar to that of a ladder: vertical rails loaded in compression connected by rungs loaded in tension. This is one of the most efficient structures for achieving maximal compressive strength with minimal mass in a composite material.

Note in Figure 4-3 that there is no net tension across the palate in an adult. During the prenatal and early postnatal period, the palate grows in width via the posterior palatal synchondosis (primary growth center).[1] When the first deciduous molars establish a functional occlusion, the midpalatal suture evolves into a secondary growth site that responds to occlusal loading. A soft diet decreases the rate of palatal expansion during the growing years.[2] Because growth in width of the maxilla reflects the magnitude of occlusal function,[3] inadequate functional loading may result in functional aberrations such as posterior crossbite.

Differential Osteology of the Maxilla and Mandible

Although equal and opposite functional loads are delivered to the maxilla and mandible, the maxilla transfers stress to the entire cranium, whereas the mandible must absorb the entire load. Consequently, the mandible is much stronger and stiffer than the maxilla. A midsagittal section through the incisors (Fig. 4-4) and a frontal section through the molar region (Fig. 4-5) show the distinct differences in the osseous morphology of the maxilla and mandible. The maxilla has relatively thin cortices that are interconnected by a network of trabeculae (see Figs. 4-2, 4-4, and 4-5). Because it is loaded primarily in compression, the maxilla is structurally similar to the body of a vertebra.

The mandible, however, has thick cortices and more radially oriented trabeculae (see Figs. 4-4 and 4-5). The structural array is similar to the shaft of a long bone and indicates that the mandible is loaded predominantly in bending and torsion. This

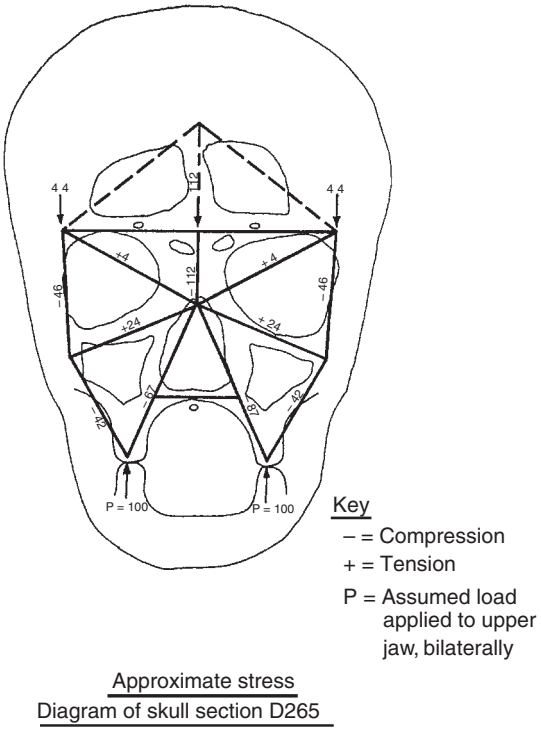


FIGURE 4-3 Two-dimensional vector analysis of stress in the frontal section of the human skull depicted in Figure 4-2. Relative to a bilateral biting force of 100 arbitrary units, the load is distributed to the vertical components of the midface as compressive (negative) stress. The horizontal structural components are loaded in tension. In a nongrowing individual, the stress across the midpalatal suture is 0. When masticating, loads increase and the midpalatal suture is subjected to a tensile load, resulting in an increase in maxillary width. (From Atkinson SR. Balance: the magic word. *Am J Orthod.* 1964;50:189.)

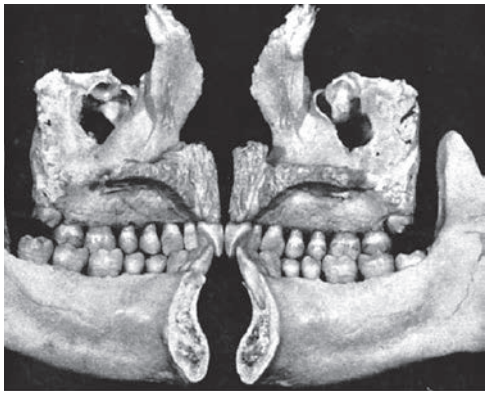


FIGURE 4-4 Midsagittal section of a human skull shows that the maxilla is composed primarily of trabecular (spongy) bone. The opposing mandible has thick cortices connected by relatively coarse trabeculae. (From Atkinson SR. Balance: the magic word. *Am J Orthod.* 1964;50:189.)

biomechanical impression based on osteology is confirmed by in vivo strain-gauge studies in monkeys. Hylander[4,5] demonstrated substantial bending and torsion in the body of the mandible associated with normal masticatory function (Fig. 4-6). A clinical correlation consistent with this pattern of surface strain is the tendency of some human beings to form tori in the areas

FIGURE 4-5 Frontal section of the maxilla and mandible in the plane of the first molars. Because it transmits masticatory loads to the entire cranium, the maxilla has thin cortices connected by relatively fine trabeculae. The mandible, however, is loaded in bending and torsion; it therefore is composed of thick cortical bone connected by coarse, oriented trabeculae. (From Atkinson SR. Balance: the magic word. *Am J Orthod.* 1964;50:189.)

of maximal bending and torsion (Fig. 4-7). The largest tori are on the side on which the individual habitually chews (preferential working side).

Temporomandibular Articulation

The TMJ is the principal adaptive center for determining the intermaxillary relationship in all three planes of space. Figure 4-8 shows optimal skeletal development consistent with normal morphology of the TMJ. Biomechanics studies have demonstrated that the TMJ eminence develops a shape that minimizes joint loads.[6] Figure 4-9 shows aberrant skeletal and dental relationships consistent with degeneration of the fossa and mandibular condyle (i.e., the enlarged mushroom shape of the condylar process, the roughened topography of the articulating surfaces, and the loss of articular cartilage and subchondral plate). Progressive degeneration or hyperplasia of one or both mandibular condyles may result in substantial intermaxillary discrepancies in the sagittal, vertical, and frontal dimensions. The growth and development of the TMJ in response to facial growth are well known. During childhood and adolescence, the face grows several centimeters anteriorly and inferiorly. Adaptation of the TMJ allows this substantial change to occur without disturbing the intermaxillary relationship of the dentition (e.g., Class I occlusion remains Class I). In the adult years, the intermaxillary relationship continues to change but at a slower rate. The face lengthens and may rotate anteriorly as much as 10 mm over the adult lifetime.[7] The mandible adapts to this change by lengthening and maintaining the intermaxillary dental relationship (Fig. 4-10). However, if the TMJs of an adult undergo bilateral degenerative change, whether or not symptomatic, the mandible can decrease in length, resulting in a shorter, more convex face (Fig. 4-11). Cone-beam computed tomography (CBCT) has revealed intraosseous changes in the metaphysis of a degenerating mandibular condyle

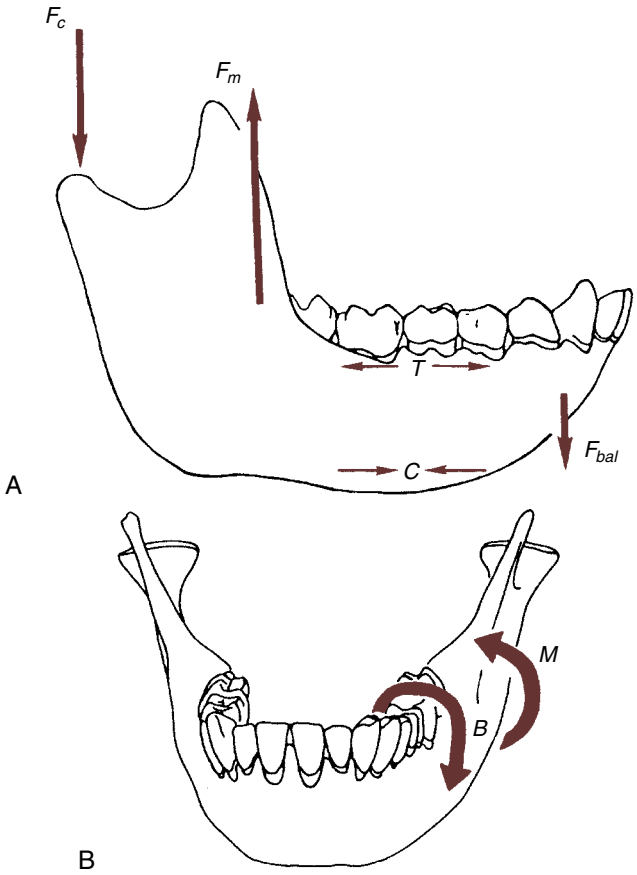


FIGURE 4-6 Stress patterns in the primate mandible during unilateral mastication. F_c and F_m are the condylar reaction and the resultant muscle forces on the balancing side, respectively. F_{bal} is the force transmitted through the symphysis from the balancing to the working side. *T* and *C* indicate the location of tensile stress and compressive stress, respectively. **A,** During the power stroke, the mandibular corpus on the balancing side is bent primarily in the sagittal plane, resulting in tensile stress along the alveolar process and compressive stress along the lower border of the mandible. **B,** On the working side, the corpus is twisted primarily about its long axis (it also experiences direct shear and is slightly bent). The muscle force on this side tends to evert the lower border of the mandible and invert the alveolar process (*curved arrow M*). The twisting movement associated with the bite force has the opposite effect (*curved arrow B*). The portion of the corpus between these two twisting movements experiences maximal twisting stress. (From Hylander WL. Patterns of stress and strain in the macaque mandible. In: Carlson DS, ed. *Craniofacial Biology*. Ann Arbor, MI: Center for Human Growth and Development; 1981.)

(Fig. 4-12). Compared with the normal left side, there are trabecular sclerosis and subcondylar osteolytic lesions on the degenerating side. The picture is consistent with a mechanical overload of the TMJ. Animal studies have described the molecular mechanism of TMJ degeneration due to mechanical overload: (1) expression of vascular endothelial growth factor (VEGF), (2) hypoxia-inducible factor-1α (Hif-1α) is activated, and (3) osteoclastogenesis via repression of osteoprotegerin (Opg) expression results in osteolytic lesions.[8]

Within physiologic limits, the TMJ has remarkable regenerative and adaptive capabilities. The TMJ can recover spontaneously

FIGURE 4-7 Occlusal view of the mandibular dentition of a male patient with extensive buccal and lingual tori. Note that the exostoses are most extensive in the area of the second premolar and first molar, the area of maximal torsion in the posterior segment of the mandible.

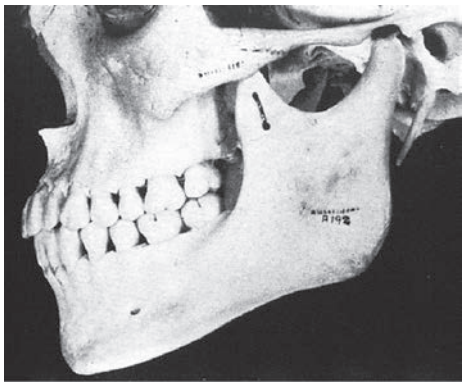


FIGURE 4-8 Adult human skull with ideal occlusion and osseous form of the maxilla and mandible. Note the ideal anatomic form of the condyle and articular fossa of the temporomandibular joint. (From Atkinson SR. Balance: the magic word. *Am J Orthod.* 1964;50:189.)

FIGURE 4-9 Adult human skull with a severe Class II malocclusion. Note the degeneration of the temporomandibular joint (i.e., the large, mushroom-shaped condyle and the enlarged, roughened articular fossa). (From Atkinson SR. Balance: the magic word. *Am J Orthod.* 1964;50:189.)

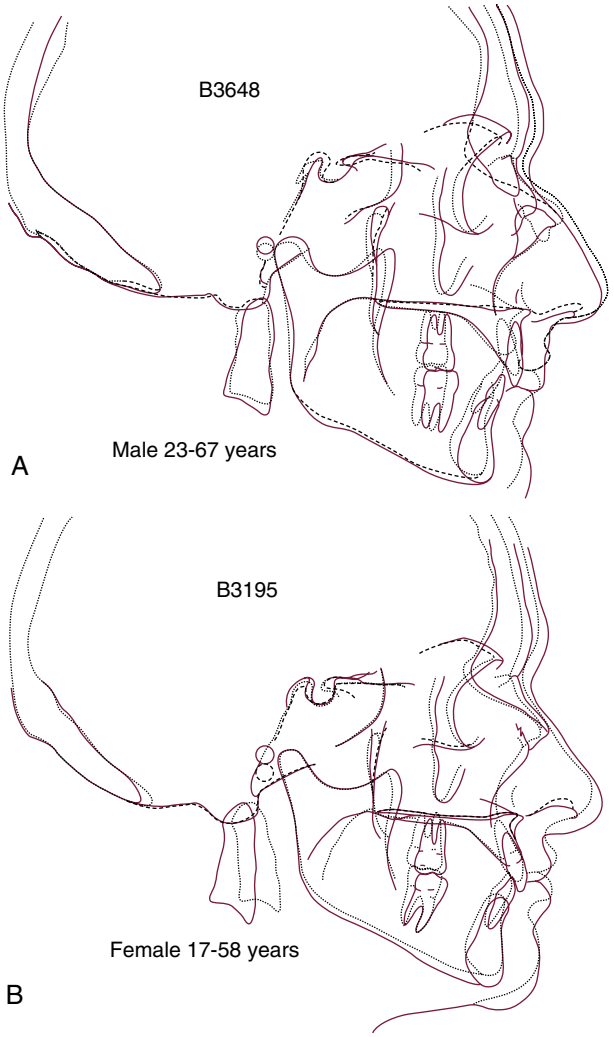


FIGURE 4-10 A, Superimposed cephalometric tracings of a male patient at ages 23 and 67 years (*black* and *red* tracings, respectively). Note the downward and forward growth of the mandible and the substantial increase in the length of the face. The nasal form was altered by a rhinoplasty in the intervening years. B, Superimposed cephalometric tracings of an adult female at ages 17 and 58 years (*black* and *red* tracings, respectively). Note the downward growth of the mandible and the substantial increase in the length of the face. (From Behrents RG. Adult facial growth. In: Enlow DH, ed. *Facial Growth*. 3rd ed., Philadelphia: WB Saunders; 1990.)

from degenerative episodes (Fig. 4-13). However, more advanced degeneration associated with abnormal occlusal loading requires distraction of the degenerating condyle with a removable orthotic, orthodontic alignment, and occlusal reconstruction to create a balance occlusal load bilaterally (Figs. 4-14 and 4-15). Unlike other joints in the body, the TMJ has a remarkable ability to adapt to altered jaw structure and function. After a subcondylar fracture, the condylar head is pulled medially by the superior pterygoid muscle and resorbs. If the interocclusal relationship is maintained, a new condyle forms and assumes normal function. Unilateral subcondylar fractures usually result in regeneration of a new functional condyle with no significant deviation of the mandible.[9] However, about one-fourth of subcondylar fractures result in a mandibular deviation toward the injured side,

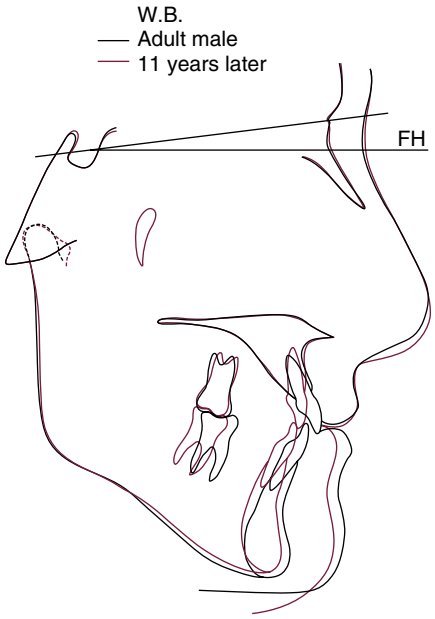


FIGURE 4-11 Superimposed cephalometric tracings of a middle-aged man over an 11-year period. Although the soft tissue changes are similar to those seen in other adult males (see Fig. 4-10, *B*), the less prominent mandible and more convex skeletal profile are consistent with a shortened mandible. This man had a history of bilateral temporomandibular internal derangement that progressed to crepitus. However, despite the substantial change in facial form and occlusal compensation, no appreciable pain was reported.

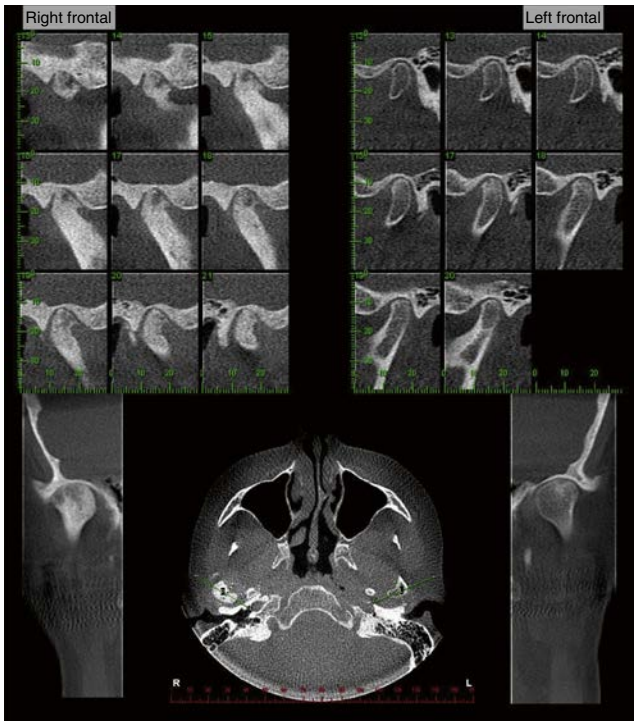


FIGURE 4-12 A CBCT image reveals osseous degeneration of the right mandibular condyle in a 65-year-old female. In both the frontal and sagittal cuts, note the lack of joint space between the condyle and the fossa, as well as the subcortical plate radiolucent erosions in the trabecular bone of the condylar metaphysis.

resulting in an asymmetric Class II malocclusion with a midline deviation.

Another sequela of mandibular trauma is internal derangement such as a unilateral closed lock (a condyle distally displaced relative to the disc). If the range of motion is reduced in a growing patient, the compromised function may inhibit mandibular growth, resulting in a cant of the occlusal plane. Progressive dysfunction and pain may ensue, particularly when associated with occlusal trauma. Reestablishing normal bilateral function allows the compromised condyle or condyles to adapt favorably.

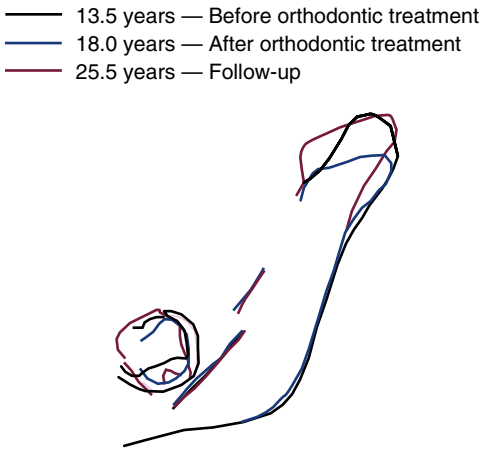


FIGURE 4-13 Superimposed tracings of a series of panoramic radiographs documenting the degeneration of a mandibular condyle between 13.5 and 18 years of age. Between the ages of 18 and 25.5 years, the degenerative condyle grew, restoring the original length of the mandible. (From Peltola JS, Kononen M, Nystrom M. A follow-up study of radiographic findings in the mandibular condyles of orthodontically treated patients and associations with TMD. *J Dent Res*. 1995;74(9):1571.)

BONE PHYSIOLOGY

The morphology of bone has been well described, but its physiology is elusive because of the technical limitations inherent in the study of mineralized tissues. Accurate assessment of the orthodontic or orthopedic response to applied loads requires time markers (bone labels) and physiologic indexes (DNA labels, histochemistry, and in situ hybridization) of bone cell function. Systematic investigation with these advanced methods has defined new concepts of clinically relevant bone physiology.

Specific Assessment Methodology

Physiologic interpretation of the response to applied loads requires the use of specially adapted methods:

- Mineralized sections are an effective means of accurately preserving structure and function relationships.[10]
- Polarized light birefringence detects the preferential orientation of collagen fibers in the bone matrix.[11]
- Fluorescent labels (e.g., tetracycline) permanently mark all sites of bone mineralization at a specific point in time (anabolic markers).[11,12]
- Microradiography assesses mineral density patterns in the same sections.[13]
- Autoradiography detects radioactively tagged precursors (e.g., nucleotides and amino acids) used to mark physiologic activity.[14-16]

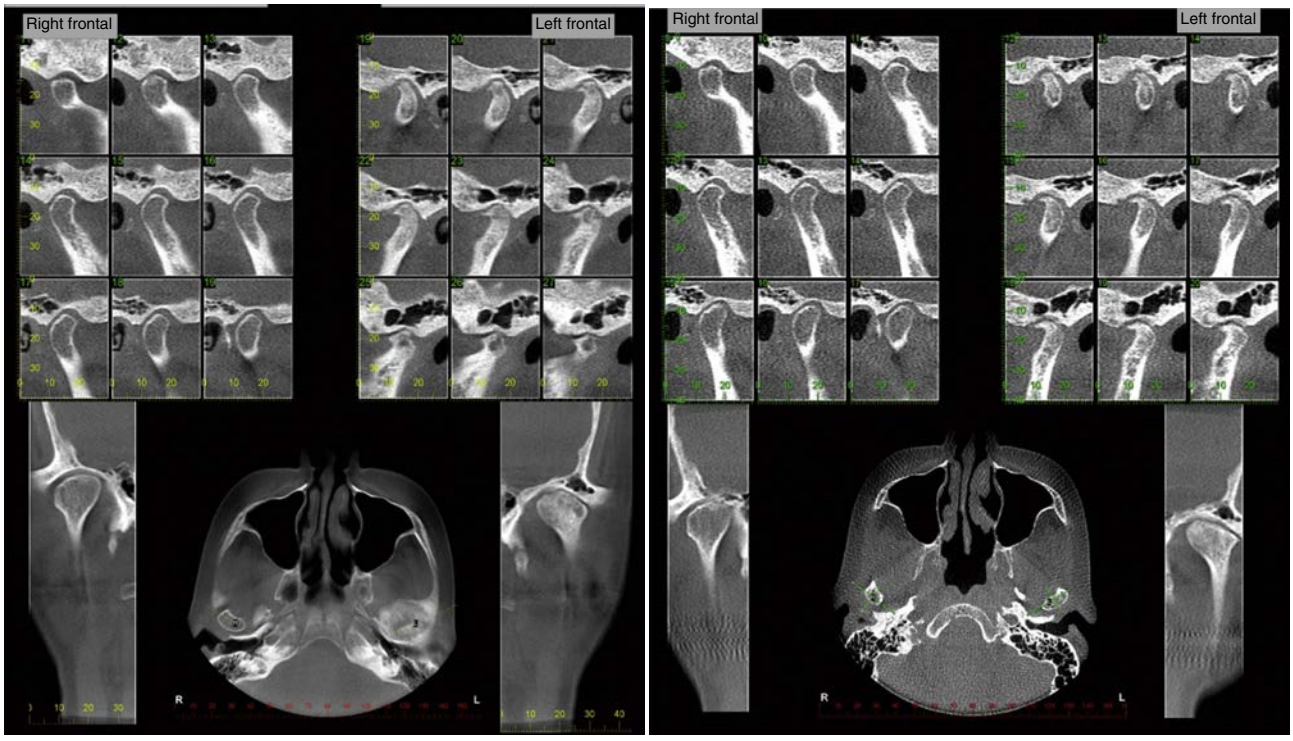


FIGURE 4-14 Pretreatment CBCT imaging of a 72-year-old female shows osseous degeneration of the left mandidular condyle, characterized by subcortical radiolucent lesions that are noted in both the frontal and sagittal cuts. A well-defined osteophyte is noted on the anterior surface of the left mandibular condyle.

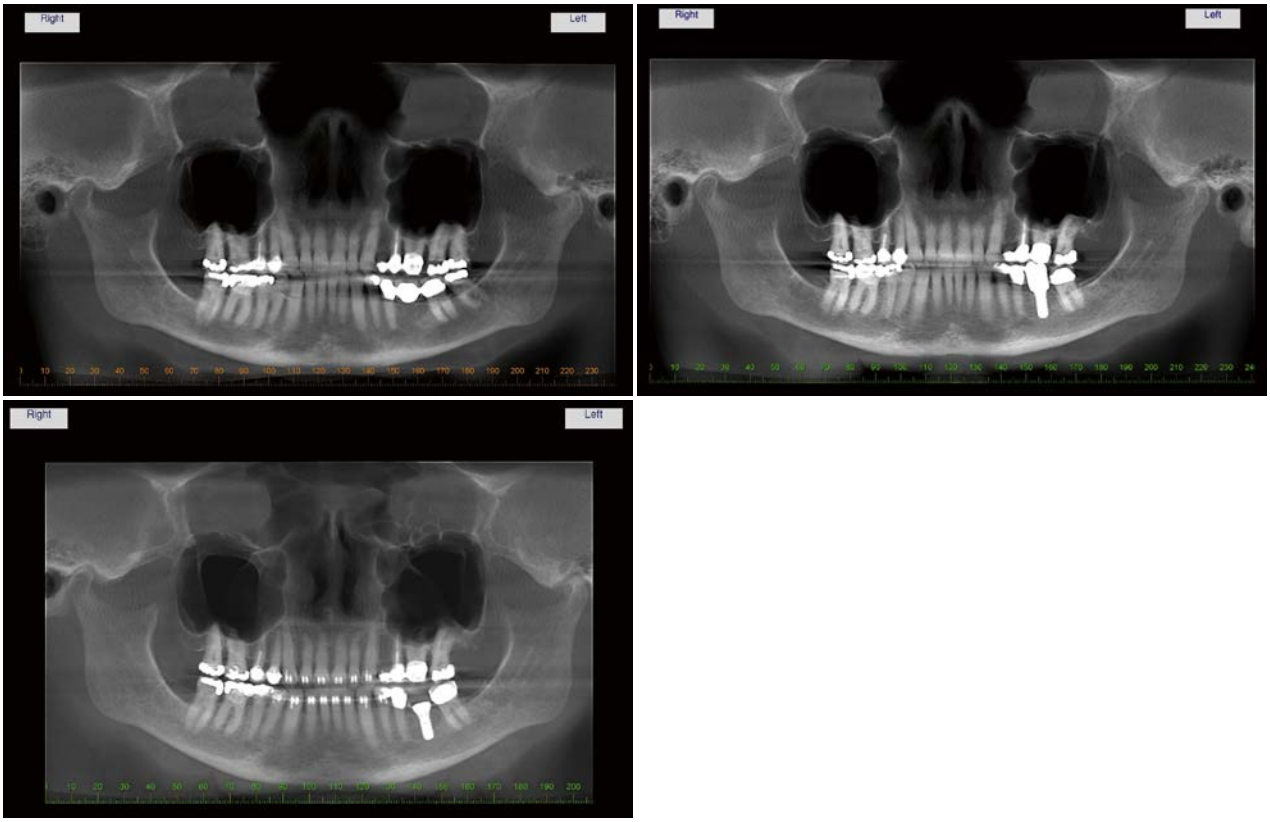


FIGURE 4-15 A posttreatment CBCT scan was performed on the patient imaged in Figure 4-14. Condylar distraction of the left mandibular condyle, producing a left-posterior open bite of about 2 mm, resulted in resolution of the subcortical osteolytic lesions, but the osteophyte on the anterior surface persisted. The patient subsequently received coordinated orthodontics and restorative treatment to correct the posterior open bite by increasing the vertical dimension of occlusion.

CASE STUDY 4-1 TEMPOROMANDIBULAR DISCREPENCIES

A 52-year-old man sought treatment for a long history of facial pain, occlusal dysfunction, and an internal derangement of the right mandibular condyle (Figs. 4-16 and 4-17). Intracapsular surgery was performed on the right TMJ to repair the internal derangement. After transient postoperative improvement, degeneration of the TMJ accelerated, the pain increased, and a progressive anterior open bite malocclusion developed. Masticatory function deteriorated, and an internal derangement of the left TMJ was noted. Bilateral intracapsular surgery was performed to restore "normal jaw function." After the second surgical procedure, the patient suffered for 10 years with chronic pain and progressive bilateral degeneration of both TMJs. Orthodontic and orthotic (splint) therapy failed to relieve the pain and functional debilitation. The patient declined further treatment and is being managed with pain medication. From a physiologic perspective, intracapsular surgery usually is contraindicated because it inhibits the natural ability of the joint to adapt to changing biomechanical demands. The TMJ is a remarkably regenerative and adaptive joint if its physiologic limits are respected.

W. Eugene Roberts

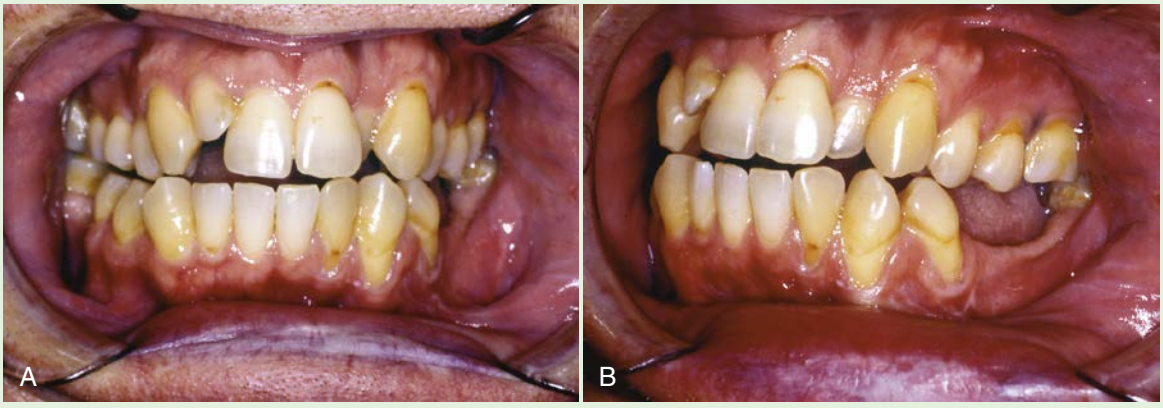


FIGURE 4-16 Frontal view (A), lateral view (B), maxillary occlusal view

CASE STUDY 4-1 TEMPOROMANDIBULAR DISCREPENCIES—cont'd

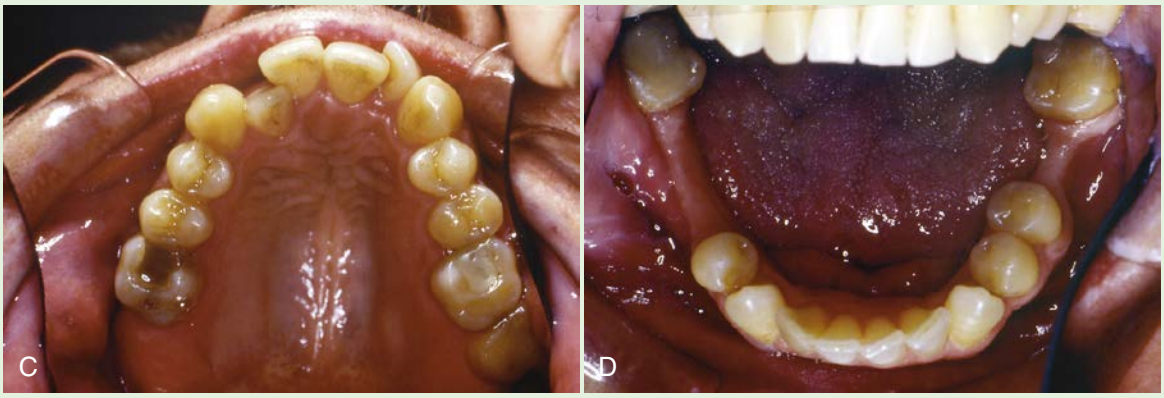


FIGURE 4-16 cont'd (C), and mandibular occlusal view (D) of the dentition of a 52-year-old man with a partly edentulous open bite malocclusion. Note the atrophic extraction sites and gingival recession. (From Roberts WE, et al. Adjunctive orthodontic therapy in adults over 50 years of age. *J Ind Dent Assoc.* 1997;76(2):33.)

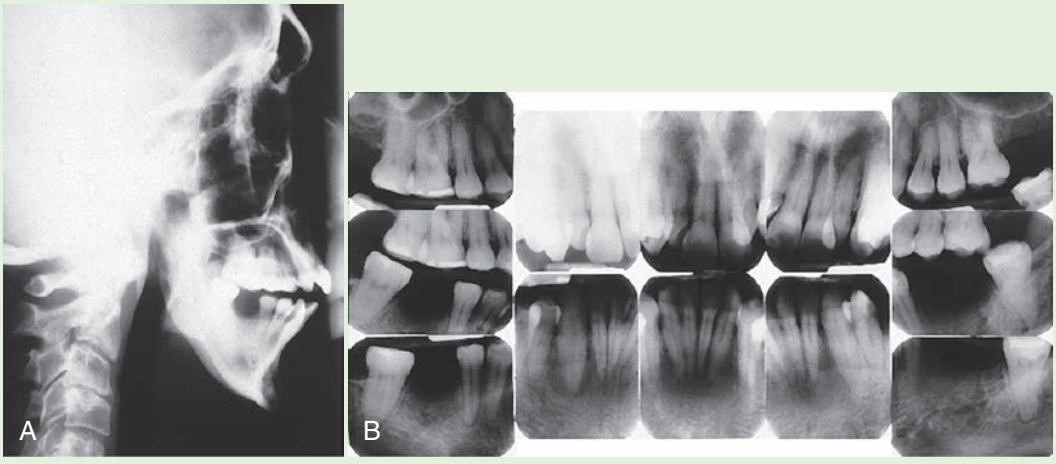


FIGURE 4-17 A, Cephalometric radiograph of the patient shown in Figure 4-16 shows a skeletal open bite with a steep mandibular plane and a relatively short ramus. The thin symphyseal cortex is consistent with a systemic osteopenia. B, A full-mouth radiographic survey shows a generalized lack of cortical bone at the alveolar crest and a pattern of indistinct lamina dura and trabeculae. This generalized ground-glass appearance of the alveolar bone is consistent with high-turnover metabolic bone disease. (From Roberts WE, et al. Adjunctive orthodontic therapy in adults over 50 years of age. *J Ind Dent Assoc.* 1997;76(2):33.)

- Nuclear volume morphometry differentially assesses osteoblast precursors in a variety of osteogenic tissues.[17]
- Cell kinetics is a quantitative analysis of cell physiology based on morphologically distinguishable events in the cell cycle (i.e., DNA synthesis [S] phase, mitosis, and differentiation-specific change in nuclear volume).[17,18]
- Finite element modeling is an engineering method of calculating stresses and strains in all materials, including living tissue.[19-22]
- Microelectrodes inserted in living tissue, such as the PDLs, can detect electrical potential changes associated with mechanical loading.[18,23]
- Backscatter emission is a variation of electron microscopy that assesses relative mineral density at the microscopic level in a block specimen.[24]
- Microcomputed tomography is an in vitro imaging method for determining the relative mineral density of osseous tissue down to a resolution of about 5 μm (about the size of an osteoblast nucleus).[25]
- Microindentation testing is a method for determining the mechanical properties of bone at the microscopic level.[26-29]

For a detailed discussion and illustration of the methods used to investigate the inherent bone physiologic mechanisms of bone, refer to the 4th edition of this text and to two issues of *Seminars in Orthodontics* (June 2004, December 2006). The first issue focuses on bone development and its inherent modeling mechanism, applied to tooth movement and dentoalveolar adaptation. The second issue is an advanced discussion of the bone remodeling mechanism, osteogenesis imperfecta, retromolar implant anchorage, and the use of stainless steel miniscrews in the zygomatic crest.

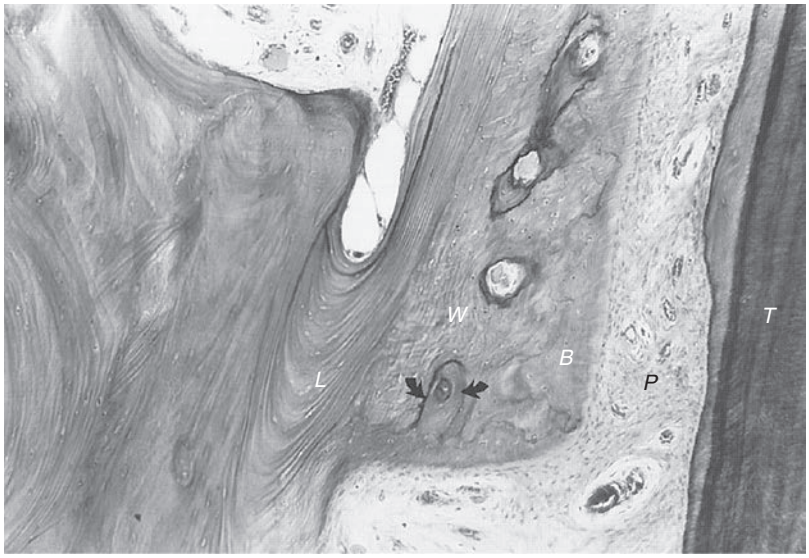


FIGURE 4-18 A section of human periodontium from the lower first molar region shows a typical histologic response to orthodontic tooth movement. With respect to the mature lamellar bone (*L*) on the left, the tooth (*T*) is being moved to the right. The first bone formed adjacent to the periodontal ligament (*P*) is of the woven type (*W*). Subsequent lamellar compaction forms primary osteons of composite bone (*arrows*). Bundle bone (*B*) is formed where ligaments such as the periodontal ligament are attached. (From Roberts WE, et al. Implants: bone physiology and metabolism. *Calif Dent Assoc J.* 1987;15(10):58.)

Classification of Bone Tissue

Orthodontic tooth movement results in rapid formation of relatively immature new bone (Fig. 4-18). During the retention period, the newly formed bone remodels and matures. To appreciate the biological mechanism of orthodontic therapy, the practitioner must have a knowledge of bone types.

Woven Bone

Woven bone varies considerably in structure. However, it is relatively weak, disorganized, and poorly mineralized. Woven bone serves a crucial role in wound healing by (1) rapidly filling osseous defects, (2) providing initial continuity for fractures and osteotomy segments, and (3) strengthening a bone weakened by surgery or trauma. The first bone formed in response to orthodontic loading usually is the woven type. Woven bone is not found in the adult skeleton under normal, steady-state conditions; rather, it is compacted to form composite bone, remodeled to lamellar bone, or rapidly resorbed if prematurely loaded.[13,30] The functional limitations of woven bone are an important aspect of orthodontic retention (Fig. 4-19) and of the healing period following orthognathic surgery.[31]

Lamellar Bone

In contrast to woven bone, lamellar bone, a strong, highly organized, well-mineralized tissue, makes up more than 99% of the adult human skeleton. When new lamellar bone is formed, a portion of the mineral component (hydroxyapatite) is deposited by osteoblasts during primary mineralization (Fig. 4-20, *D*). Secondary mineralization, which completes the mineral component, is a physical process (crystal growth) that requires many months. Within physiologic limits, the strength of bone is related directly to its mineral content.[32,33] The relative strengths of different histologic types of osseous tissues can be stated thus:

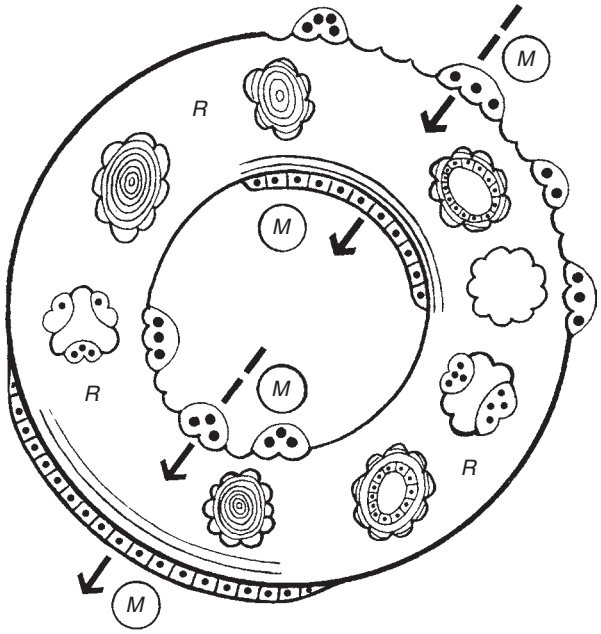


FIGURE 4-19 A schematic cross section of cortical bone shows surface modeling (*M*), which is the process of uncoupled resorption and formation. Remodeling (*R*) is the turnover of existing bone. (From Roberts WE, et al. Remodeling of devitalized bone threatens periosteal margin integrity of endosseous titanium implants. *Ind Dent Assoc J.* 1989;68:19.)

woven bone is weaker than new lamellar bone, which is weaker than mature lamellar bone.[31] Adult human bone is almost entirely of the remodeled variety: secondary osteons and spongiosa.[11,33,34] The full strength of lamellar bone that supports an orthodontically moved tooth is not achieved until about 1 year after completion of active treatment. This is an important

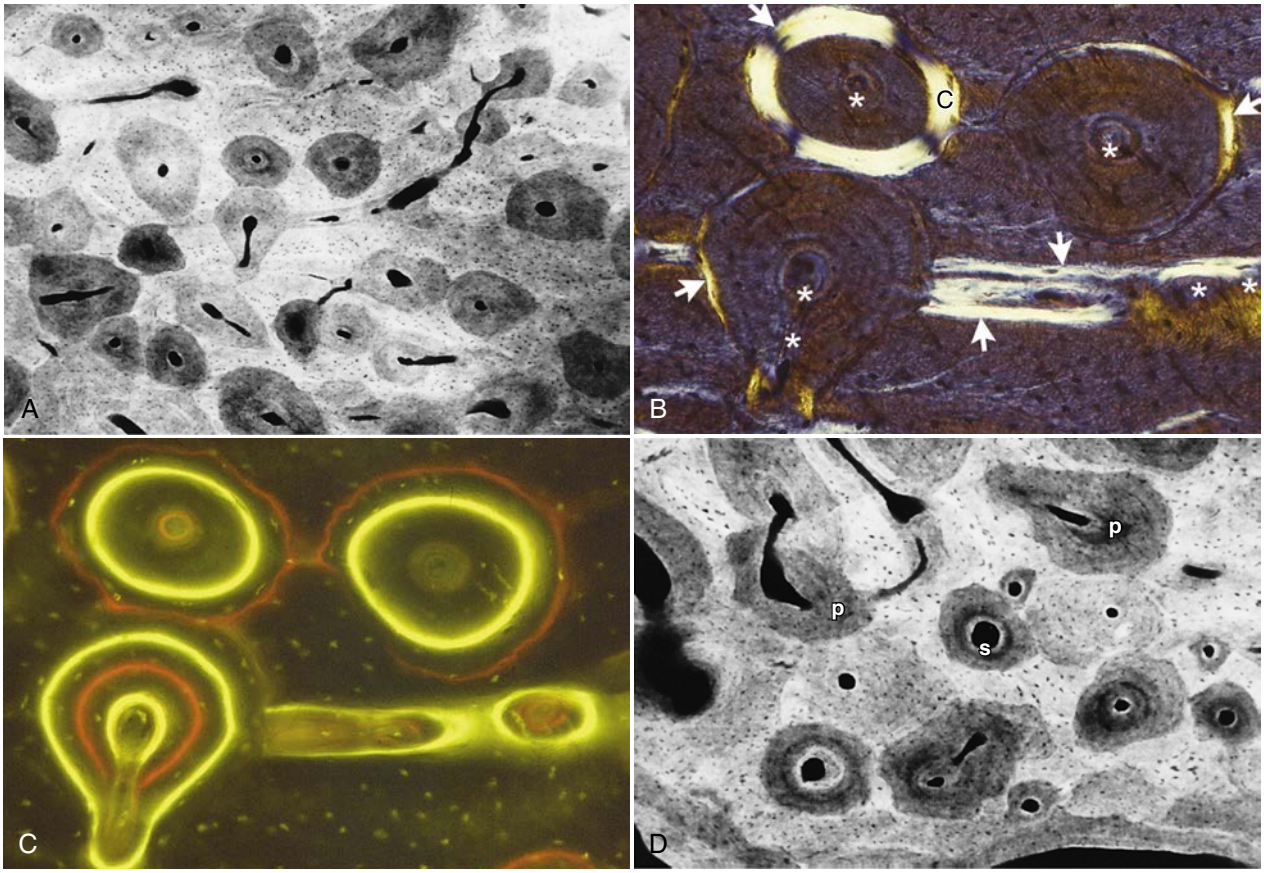


FIGURE 4-20 **A,** Microradiograph provides a physiologic index of bone turnover and relative stiffness. The more radiolucent (*dark*) osteons are the youngest, the least mineralized, and the most compliant. Radiodense (*white*) areas are the oldest, most mineralized, and rigid portions of the bone. **B,** Polarized light microscopy shows the collagen fiber orientation in bone matrix. Lamellae with a longitudinally oriented matrix (*C*) are particularly strong in tension, whereas a horizontally oriented matrix (*dark*) has preferential strength in compression (*arrows* mark resorption arrest lines, and *asterisks* mark vascular channels). **C,** Multiple fluorochrome labels administered at 2-week intervals demonstrate the incidence and rates of bone formation. **D,** This microradiograph shows an array of concentric secondary osteons (haversian systems) characteristic of rapidly remodeling cortical bone. Primary (*p*) and beginning secondary (*s*) mineralization are more radiolucent and radiodense, respectively. (D, From Roberts WE, Garotto LP, Katona TR: Principle of orthodontic biomechanics: metabolic and mechanical control mechanisms. In Carlson DS, Goldstein SA, editors: Bone biodynamics in orthodontic and orthopedic treatment, Ann Arbor, 1992, University of Michigan Press)

consideration in planning orthodontic retention (Fig. 4-21) and in the postoperative maturation period that follows orthognathic surgery.

Composite Bone

Composite bone is an osseous tissue formed by the deposition of lamellar bone within a woven bone lattice, a process called *cancellous compaction*.[10,35] This process is the quickest means of producing relatively strong bone.[36] Composite bone is an important intermediary type of bone in the physiologic response to orthodontic loading (see Fig. 4-18), and it usually is the predominant osseous tissue for stabilization during the early process of retention or postoperative healing. When the bone is formed in the fine compaction configuration, the resulting composite of woven and lamellar bone forms structures known as *primary osteons*. Although composite bone may be high-quality, load-bearing osseous tissue, it eventually is remodeled into secondary osteons.[11,31]

Bundle Bone

Bundle bone is a functional adaptation of lamellar structure to allow attachment of tendons and ligaments. Perpendicular striations, called *Sharpey fibers*, are the major distinguishing characteristics of bundle bone. Distinct layers of bundle bone usually are seen adjacent to the PDL (see Fig. 4-18) along physiologic bone-forming surfaces.[37] Bundle bone is the mechanism of ligament and tendon attachment throughout the body.

SKELETAL ADAPTATION: REMODELING AND MODELING

Bone Remodeling

Bone remodeling is a *coupled sequential* process of bone resorption followed by bone formation (Fig. 4-20, *A* to *D*). Bone remodeling occurs both in cortical (Fig. 4-20) and trabecular bone (Fig. 4-22) compartments of the skeletal system. However,

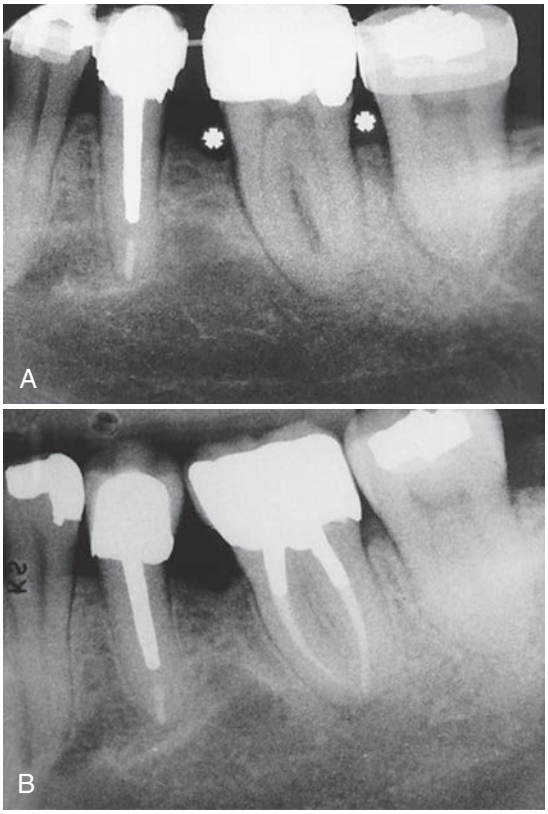


FIGURE 4-21 Periapical radiographs comparing bone maturation at the end of active orthodontic treatment and 2 years later. **A,** At the end of treatment, large amorphous areas of relatively immature bone can be seen. **B,** After retention and restorative treatment, including endodontics, distinct definition of cortices and trabeculae is evident. (From Roberts WE, Garotto LP, Katona TR: Principle of orthodontic biomechanics: metabolic and mechanical control mechanisms. In Carlson DS, Goldstein SA, editors: Bone biodynamics in orthodontic and orthopedic treatment, Ann Arbor, 1992, University of Michigan Press.)

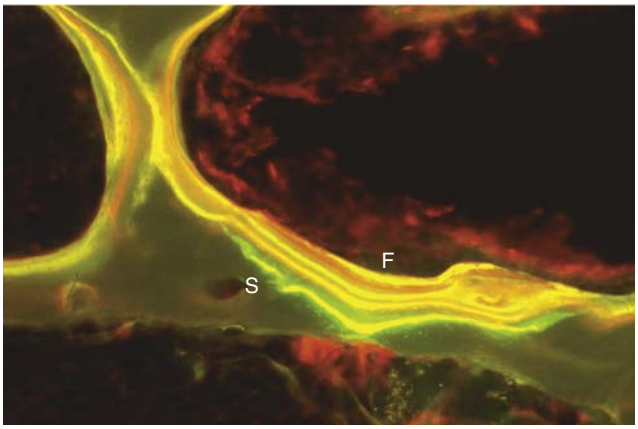


FIGURE 4-22 Trabecular bone remodeling in the vertebrae in a rat: multiple fluorochrome labels demonstrate bone formation (*F*) over a scalloped resorption arrest line (*S*). (From Roberts WE, Roberts JA, Epker BN, et al. Remodeling of mineralized tissues, part I: the Frost legacy. *Semin Orthod.* 2006;12(4):216–237.)

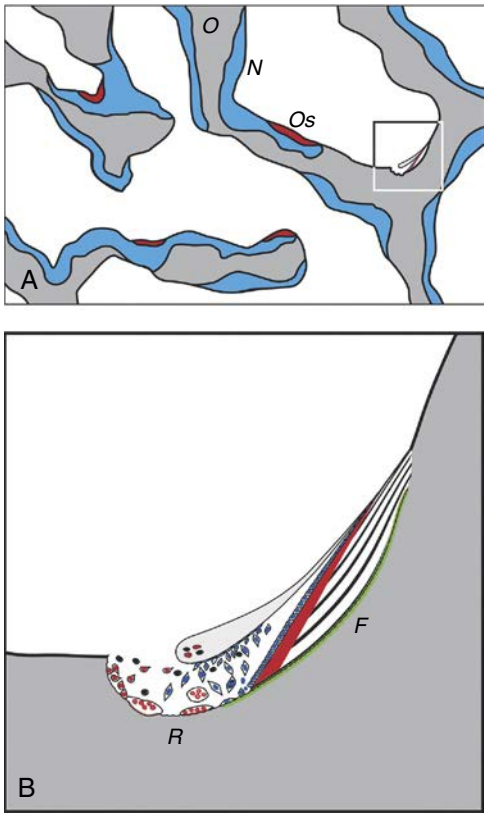


FIGURE 4-23 **A,** A schematic drawing of trabecular bone remodeling over a 1-year interval shows the pattern of new bone formation (*N*) relative to old bone (*O*) and osteoid seams (*Os*). The *box* marks an area of active trabecular resorption, which is magnified in the accompanying figure. **B,** A detailed drawing of an active remodeling site (magnified from **A**) shows a hemicutting/filling cone with a similar perivascular array of resorptive (*R*) and formative (*F*) cells as shown for cortical bone remodeling. The osteoclastic and osteoblastic cell lines are *red* and *blue*, respectively. A nonmineralized osteoid seam (*solid red line*) marks the bone-forming surface. (From Roberts WE, Roberts JA, Epker BN, et al. Remodeling of mineralized tissues, part I: the Frost legacy. *Semin Orthod.* 2006;12(4):216–237.)

there are important differences between bone remodeling in the cortical versus trabecular bone that are reflected at a tissue level and revealed by histologic studies. Histologically, when viewed in transverse sections of long bones, the end result of bone remodeling in cortical bone is the production of a new, circular (typically 200- to 300-μm diameter) shaped osteon (Fig. 4-20, *C*). This type of cortical bone remodeling can also be described as *intracortical secondary osteonal* bone remodeling. Thus, the remodeling that occurs in cortical bone occurs within the substance of the cortical bone (in the *intracortical* compartment) and away from the periosteal and endosteal surfaces. In addition, the osteons that result from the bone remodeling process result in the formation of *secondary* osteons.[38] These osteons have a reversal line and are in contrast to primary osteons,[39] which histologically resemble secondary osteons but lack the reversal line because no bone resorption occurs in the development of a primary osteon. In essence, primary osteons are produced by bone formation and thus not by a coupled process of bone resorption and bone formation. In trabecular bone, the bone tissue structure is frequently not wide enough to accommodate 200- to 300-μm sized

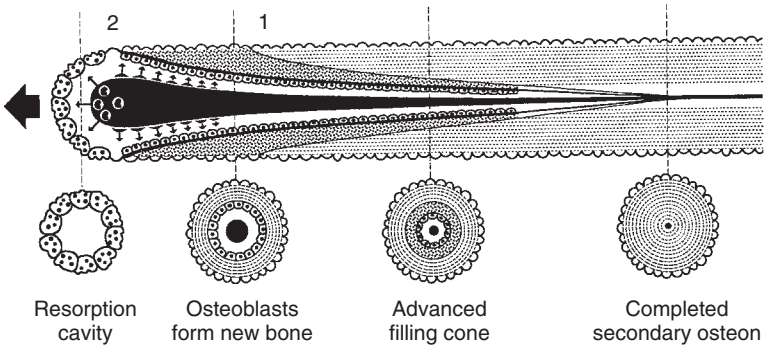


FIGURE 4-24 The cutting/filling cone has a head of osteoclasts that cut through the bone and a tail of osteoblasts that form a new secondary osteon. The velocity through bone is determined by measuring between two tetracycline labels (*1* and *2*) administered 1 week apart. (Adapted from Roberts WE, et al. Osseous adaptation to continuous loading of rigid endosseous implants. *Am J Orthod.* 1984;86:95–111.)

osteons. Thus, only "hemi-osteonal" *surface* bone remodeling (Fig. 4-23, *A* and *B*) occurs in trabecular bone.[40] However, the bone remodeling is identical to that of cortical bone as it follows the same, coupled, resorption and formation process. For a detailed discussion of calcium homeostasis and trabecular bone remodeling, refer to Roberts.[41] A schematic drawing (see Fig. 4-23, *A*) of adult trabecular bone illustrates the pattern of turnover associated with continuous remodeling to support calcium homeostasis. An individual remodeling site is shown in Figure 4-23, *B*. The A→R→F process is similar to the cutting/filling cones of cortical bone remodeling (Fig. 4-24). The trabecular bone remodeling mechanism is essentially a hemicutting/filling cone.[41] At a cellular level, though very complex interactions exist, the resorption is carried out by osteoclasts, and the formation is effected by the osteoblasts.[42] To restate, bone remodeling in both cortical and trabecular bone involves the coordinated, coupled activity of osteoclasts and osteoblasts.

Bone remodeling is a homeostatic process. It results in the rejuvenation and replacement of old bone that has served its purpose. It is unique to bone and does not occur within the substance of other mineralized tissues such as enamel, dentin and cementum. This provides a distinct advantage to bone and makes it a tissue that is capable of regeneration. The bone remodeling also underlies the immense adaptive potential of this mineralized and hard tissue, both terms otherwise potentially implying limited adaptability.[43] From a functional standpoint, bone remodeling provides for calcium and thus helps in precisely regulating calcium levels in the body.[38] From an evolutionary perspective, bone acts as a calcium reservoir, allowing life-forms to move away from the sea. Without such a reservoir and a method for mobilization of calcium stores, calcium in the immediate environment (e.g., seawater) was essential for various cellular functions.

There are two terms used to further describe the types of bone remodeling, *stochastic* and *targeted*.[44] Stochastic remodeling occurs somewhat uniformly throughout the body—that is, the continuous repair and regeneration process. There are multiple sites (e.g., ~1 million by some estimates) in both trabecular and cortical bone at which stochastic bone remodeling is occurring at any one time. These remodeling sites also provide for metabolic calcium. During calcium deprivation[45] bone remodeling is enhanced and the bone remodeling rate is increased with more "cutting/filling cones" or osteons being produced (see Fig. 4-24). Targeted remodeling occurs at a specific site of injury and not throughout the entire body. A relevant and easily understandable example for orthodontists is the bone–implant interface. In placing a miniscrew, microdamage (small linear cracks) are created within the bone by the insertion of the screw.[46] The microdamage, a manifestation of tissue injury in a mineralized tissue, is repaired by bone remodeling.[47] Thus microdamage production stimulates bone remodeling at the site of damage (e.g., close to the interface) and repairs the damaged bone. Another form of bone injury is manifestation of diffuse damage.[48] This damage is not as clearly visible in histologic sections as microdamage. Corticotomies and tissue injury of both hard and soft tissue produce a localized injury, and repair is *targeted* to that specific area. This targeted remodeling is probably important for expedited orthodontic tooth movement as most therapies (vibration, corticotomies, etc.) work at some level through the local insult and subsequent healing.

Bone Modeling

Bone modeling is a distinct and a different process from bone remodeling. These two processes are frequently confused even though they can be readily distinguished at a histologic level. Histologic sections, labeled with intravital dyes, can clearly distinguish bone remodeling and modeling.[49,50] This contrast is not trivial, and the underlying process and controls of bone remodeling and modeling are different. It is not uncommon to find bone modeling being measured in studies and being mistaken for bone remodeling. This then leads to confusion in the literature and, more unfortunately, to incorrect interpretations.

Bone modeling is a surface-specific activity and results in a change in shape and size.[38] It is an *uncoupled* process, and the bone resorption and formation are not linked or coupled in a sequential manner (Figs. 4-19 and 4-25). The bone formation and resorption mediated by the osteoblasts and osteoclasts, respectively, do not occur on the same bone surface and occur independent of each other on different bone surfaces. One example of the end result of bone modeling that can occur over a duration of years is the difference in the diameter of the dominant arm of a tennis player from the contralateral nondominant arm.[51] The bone of the dominant arm has a diameter about 1.6-fold greater than the nondominant arm. The change in size occurs over a period of years and due to modeling events on the periosteal surface (and endosteal surface) of the arm. This does not mean that bone remodeling cannot occur *within* the cortical bone (intracortical compartment) independently or simultaneously; however, they are two different processes, each having different control mechanisms.[52] There are numerous other

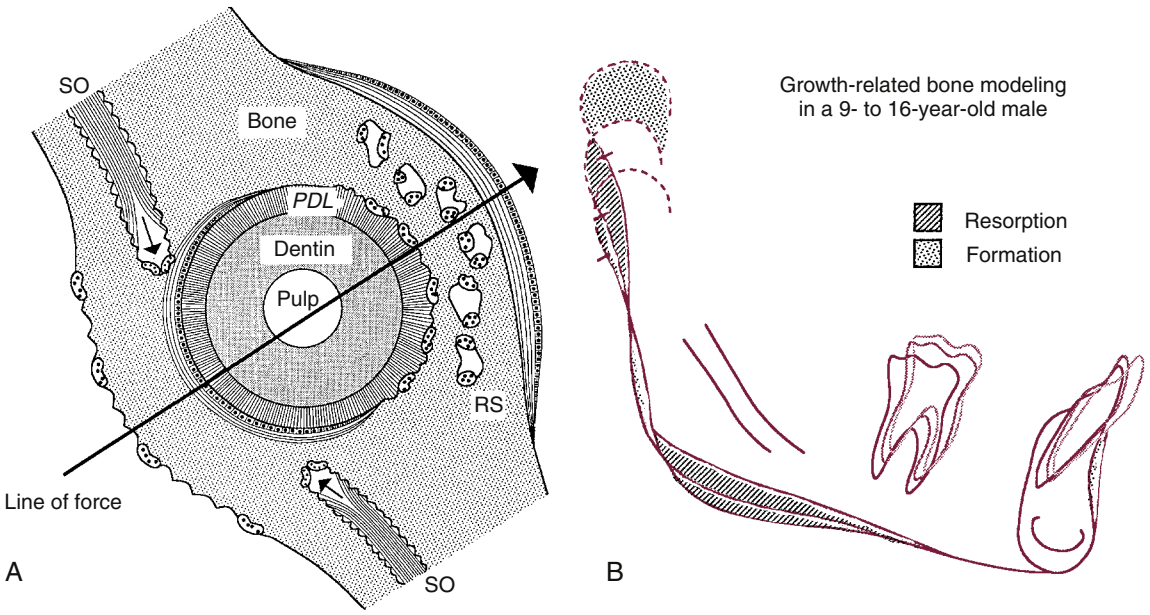


FIGURE 4-25 A, Orthodontic bone modeling, or site-specific formation and resorption, occurs along the periodontal ligament (*PDL*) and periosteal surfaces. Remodeling, or turnover, occurs within alveolar bone along the line of force on both sides of the tooth. B, Orthopedic bone modeling related to growth in an adolescent male involves several site-specific areas of bone formation and resorption. Although extensive bone remodeling (i.e., internal turnover) also is under way, it is not evident in cephalometric radiographs superimposed on stable mandibular structures.

examples of bone modeling. The formation of a callus after fracture of a bone (and insertion of an endosseous implant) and changes seen on the surfaces of bone (changes in shape and size) are readily apparent in cephalometric superimposition in a growing patient (Fig. 4-25, *B*). The surface changes are modeling; however, there is no doubt that remodeling is concurrently occurring within the bone. In fact modeling occurs primarily during growth (on periosteal and endosteal bone surfaces) and then decreases after maturity. It is activated again during healing and other pathologic biological processes (e.g., bony cyst-producing expansion).

From an orthodontic perspective, the biomechanical response to tooth movement involves an integrated array of bone modeling and remodeling events (see Fig. 4-25, *A*). Bone modeling is the dominant process of facial growth and adaptation to applied loads such as headgear, rapid palatal expansion, and functional appliances. Modeling changes can be seen on cephalometric tracings (see Fig. 4-25, *B*), but remodeling events, which usually occur at the same time, are apparent only at the tissue level. True remodeling usually is not imaged on clinical radiographs.[53] For a detailed discussion of the modeling mechanisms of tooth movement and alveolar bone adaptation, refer to Roberts et al.[43,54] The remodeling (bone turnover) aspect of tooth movement is reviewed in Roberts et al.[54] Constant remodeling (internal turnover) mobilizes and redeposits calcium by means of coupled resorption and formation: bone is resorbed and redeposited at the same site. Osteoblasts, osteoclasts, and possibly their precursors are thought to communicate by chemical messages known as *coupling factors*. Transforming growth factor β is released from bone during the resorption process; this cytokine helps stimulate subsequent bone formation to fill resorption cavities.[55] It is currently thought that growth factors released from bone mediate the coupling process via a genetic mechanism for activating and suppressing osteoclasts. Thus, *RANK*, *RANKL*, and *OPG* are gene products that control the remodeling sequence of bone resorption followed by formation. This ubiquitous genetic mechanism appears to be involved in the inflammatory induction of bone resorption and the coupling of bone formation at the same site (Fig. 4-26).[56,57]

CORTICAL BONE GROWTH AND MATURATION

Enlow[35] sectioned human skulls and histologically identified areas of surface apposition and resorption. The overall patterns of bone modeling ("external remodeling") helped define the mechanisms of facial growth. Although the method could not distinguish between active and inactive modeling sites, it was adequate for determining the overall direction of regional activity in the maxilla and mandible. This method of osseous topography was a considerable advance in the understanding of surface modeling of facial bones.

Melsen[58] used microradiographic images of mineralized sections to extend the capability of the osseous topography method. Patterns of primary and secondary mineralization (as described in Fig. 4-20) identified active appositional sites and provided a crude index of bone formation rates. Through the systematic study of autopsy specimens of 126 normal males and females from birth to 20 years of age, the most stable osseous structures in the anterior cranial base of growing children and adolescents were defined anatomically (Fig. 4-27, *A*). This research established that the three most stable osseous landmarks for superimposition of cephalometric radiographs are (1) the anterior curvature of the sella turcica, (2) the cribriform plate, and (3) the internal curvature of the frontal bone (Fig. 4-27, *B*). In effect, this research established the gold standard for reliable superimposition on the anterior cranial base.

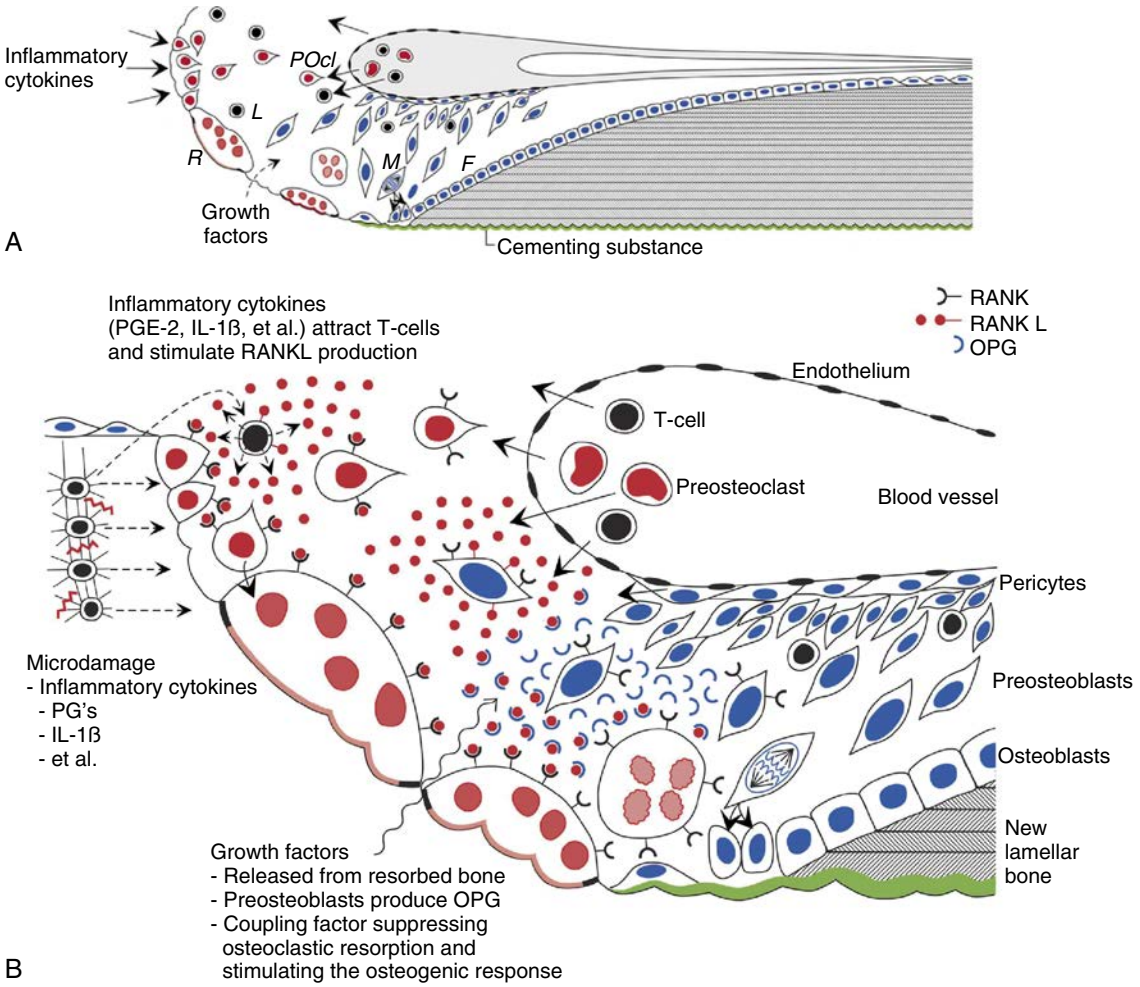


FIGURE 4-26 A, A hemisection of a cutting/filling cone moving to the left demonstrates the intravascular and perivascular mechanisms for coupling bone resorption (*R*) to formation (*F*) during the remodeling process. Lymphocytes (*L*) are attracted from the circulation by inflammatory cytokines. They help recruit preosteoclasts (*POcl*) from the circulation. See text for details. B, A magnified view of the head of a hemicutting/filling cone illustrates the proposed mechanism for coupling bone resorption to formation via the genetic *RANK/RANKL/OPG* mechanism. The cutting head is stimulated by inflammatory cytokines produced by osteocytes in damaged bone (*left*). Preosteoclasts have RANK receptors that are bound and activated by RANKL, probably produced or mediated by T cells (lymphocytes) near the resorption front. Growth factors from resorbed bone (*bottom*) stimulate production of preosteoblasts, which then produce OPG to block the RANK receptors on osteoclasts; the latter then withdraw from the scalloped surface and degenerate. Relatively flat mononuclear cells (*bottom center*) form cementing substance to form a resorption arrest line. Osteoblasts (*bottom right*) produce new lamellar bone to fill the resorption cavity. (From Roberts WE, et al. Remodeling of mineralized tissues, part II: control and pathophysiology. *Semin Orthod.* 2006;12:238–253.)

Roberts et al.[10,11,59] introduced simultaneous use of multiple fluorochrome labels and microradiography to assess modeling and remodeling patterns over extended periods of time. Noorda[60] applied these methods for a three-dimensional assessment of subcondylar growth of the mandible of adolescent rabbits. Rabbits 20 weeks of age (early adolescents) were labeled every 2 weeks with a rotating series of six different multifluorochrome labels for 18 weeks. Cross sections of the subcondylar region (Fig. 4-28, *A*) were superimposed on original, oldest labeled, and newest labeled bone according to fluorescent time markers (Fig. 4-28, *B*). Because all three sections were at the same relative level at a point in time, superimposition on original (unlabeled) bone and the oldest labeled bone (Fig. 4-28, *C*) provided an index of the relative amounts of bone resorbed and formed as the mandible grew superiorly (Fig. 4-28, *D*). This method provided the most accurate assessment to date of cortical bone drift over time. The major mechanism of the increase in interramal width during adolescent growth in rabbits is lateral drift of the entire subchondral region.

The Noorda study also produced important quantitative data on the rates of surface modeling (apposition and resorption) of primary bone (Fig. 4-29). During the last 18 weeks of growth to adult stature, the surface apposition rate decreased from more than 25 µm/day to less than 5 µm/day (Fig. 4-30, *A*). The secondary osteon census peaked at about 8 to 10 weeks

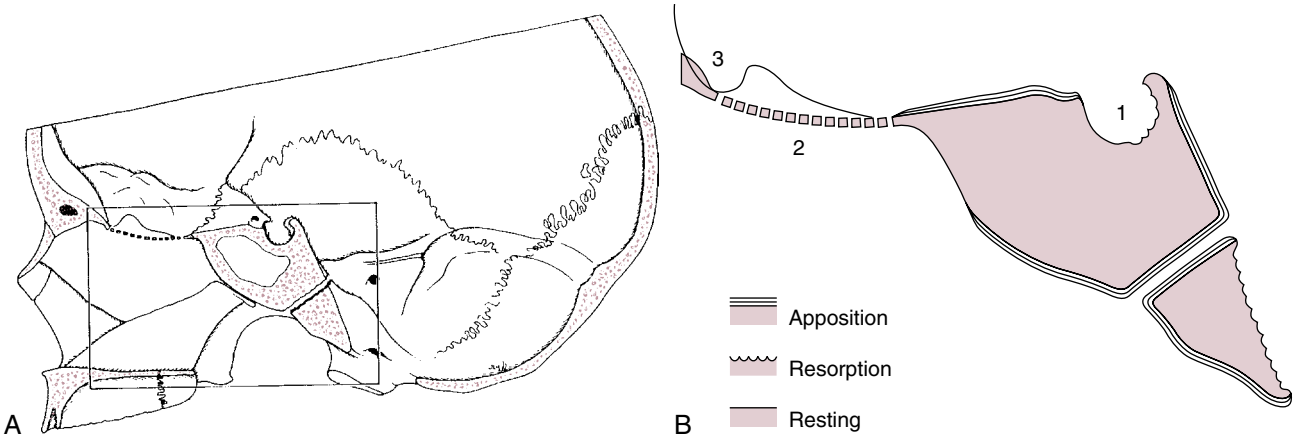


FIGURE 4-27 A, Schematic drawing of a skull showing the tissue block removed at autopsy from a series of growing children and adolescents from birth to 20 years of age. B, Diagrammatic representation of the bone modeling patterns of the cranial base in growing children. Histologic and microradiographic analysis established that the three most stable anatomic landmarks are (1) the anterior curvature of the sella turcica, (2) the cribriform plate, and (3) the internal curvature of the frontal bone. (From Melsen B. The cranial base. *Acta Odontol Scand.* 1974;32(suppl 62):103.)

(Fig. 4-30, *B*). Thus, under conditions of relatively rapid growth, primary cortical bone is remodeled to secondary osteons in about 2 months. Remodeling therefore is a time-dependent maturation of primary cortical bone.[10,11]

Cutting and Filling Cones

The rate at which cutting and filling cones progress through compact bone is an important determinant of turnover. The progression is calculated by measuring the distance between initiation of labeled bone formation sites along the resorption arrest line in longitudinal sections.[10] Using two fluorescent labels administered 2 weeks apart in adult dogs, the velocity was 27.7 ± 1.9 µm/day (mean ± SEM [standard error of the mean], $n = 4$ dogs, 10 cutting and filling cones sampled from each). At this speed, evolving secondary osteons travel about 1 mm in 36 days. Newly remodeled secondary osteons (formed within the experimental period of the dog study) contained an average of 4.5 labels (administered 2 weeks apart); the incidence of resorption cavities is about one-third the incidence of labeled osteons.[59] These data are consistent with a remodeling cycle of about 12 weeks in dogs[59] compared with 6 weeks in rabbits[10] and 17 weeks in human beings.[10,34] This relationship is useful for extrapolating animal data to human applications. More recent experimental studies have shown that new secondary osteons may continue to fix bone labels for up to 6 months, indicating that terminal filling of the lumen is slow.[61]

Traumatic or surgical wounding usually results in intense but localized modeling and remodeling responses. After an osteotomy or placement of an endosseous implant, callus formation and resorption of necrotic osseous margins are modeling processes; however, internal replacement of the devitalized cortical bone surrounding these sites is a remodeling activity. In addition, a gradient of localized remodeling disseminates through the bone adjacent to any invasive bone procedure. This process, called the *regional acceleratory phenomenon*, is an important aspect of postoperative healing.[34,62] Orthodontists can take advantage of the intense postoperative modeling and remodeling activity (1) to position a maxilla orthopedically with headgear, occlusal bite plates, or cervical support within a few weeks after a LeFort osteotomy and (2) to finish orthodontic alignment of the dentition rapidly after orthognathic surgery.[21,63]

Modeling and remodeling are controlled by an interaction of metabolic and mechanical signals. Bone modeling is largely under the integrated biomechanical control of functional applied loads. Hormones and other metabolic agents have a strong secondary influence, particularly during periods of growth and advanced aging. Paracrine and autocrine mechanisms, such as local growth factors and prostaglandins, can override the mechanical control mechanism temporarily during wound healing.[64] Remodeling responds to metabolic mediators such as PTH and estrogen primarily by varying the rate of bone turnover. Bone scans with ^{130}Te-bisphosphate, a marker of bone activity, indicate that the alveolar processes, but not the basilar mandible, have a high remodeling rate.[65,66] Uptake of the marker in alveolar bone is similar to uptake in trabecular bone of the vertebral column. The latter is known to remodel at a rate of about 20% to 30% per year compared with most cortical bone, which turns over at a rate of 2% to 10% per year.[50] Metabolic mediation of continual bone turnover provides a controllable flow of calcium to and from the skeleton.

Structural and Metabolic Fractions

The structural fraction of cortical bone is the relatively stable outer portion of the cortex; the metabolic fraction is the highly reactive inner aspect (Fig. 4-31, *A*). The primary metabolic calcium reserves of the body are found in trabecular bone and the endosteal half of the cortices, and thus these regions constitute the metabolic fraction. Analogous to orthodontic wires, the stiffness and strength of a bone are related directly to its cross-sectional area. Diaphyseal rigidity quickly is enhanced by adding a circumferential lamella at the periosteal surface. Even a thin layer of new osseous tissue at the periosteal surface greatly enhances bone stiffness because it increases the diameter of the bone. In engineering terms, cross-sectional rigidity is related to the second moment of the area. The same general relationship of round wire diameter and stiffness (strength) is well known to orthodontists. The rigidity of a wire increases as the fourth power of diameter.[67] Thus, when a relatively rigid material (bone or wire) is doubled in diameter, the stiffness increases 16 times.

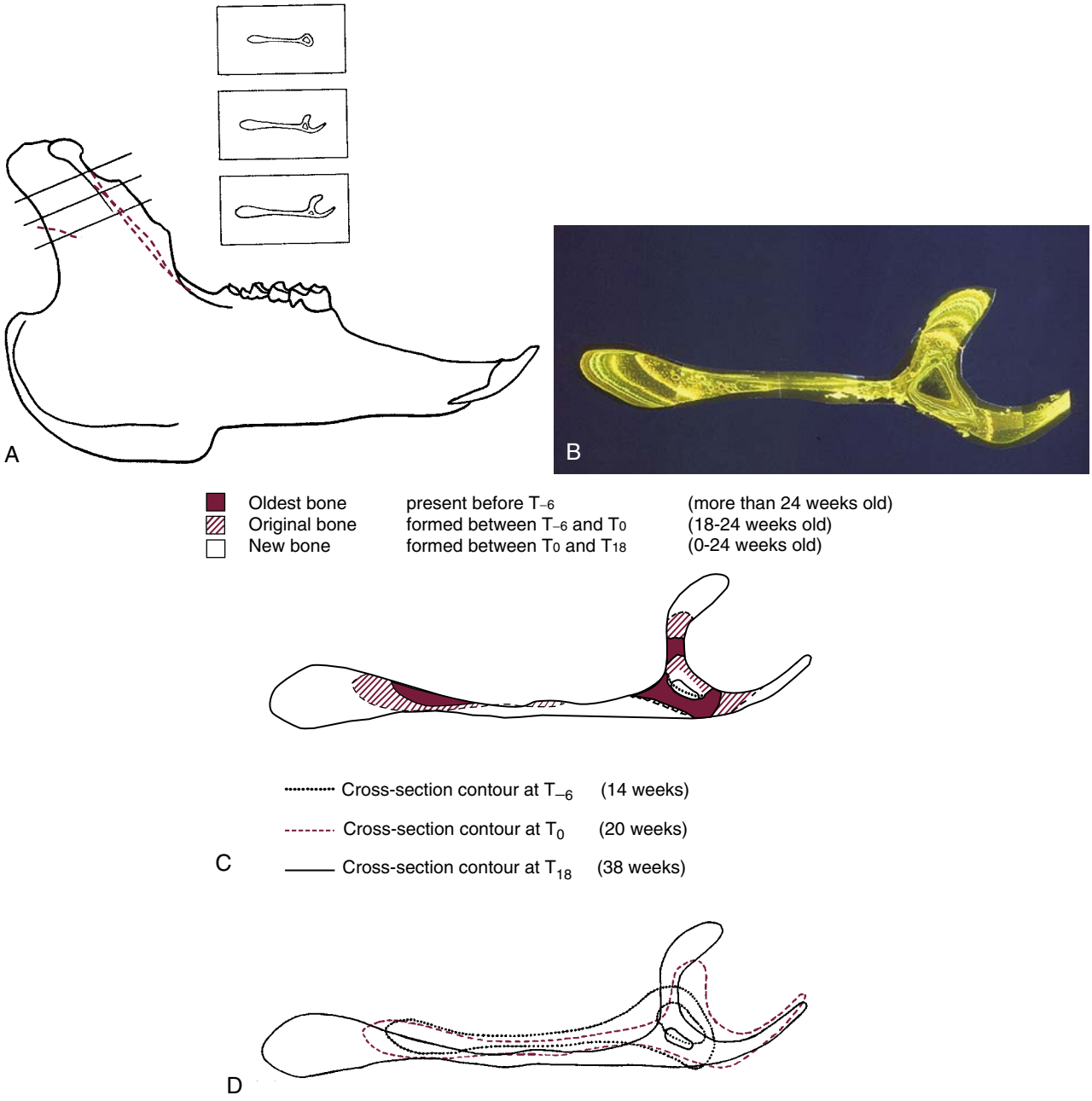


FIGURE 4-28 **A,** Schematic drawing of a rabbit mandible showing the plane of sectioning in the subcondylar region of the ramus. **B,** Fluorescent light photomicrographs of the most inferior section are arranged in a composite. The weekly deposition of bone labels over 4 months shows the patterns of bone modeling and remodeling associated with the growth and development of the subcondylar region. **C,** Based on the uptake of bone labels, the age of specific areas in a given cross section can be determined accurately. **D,** Because the subcondylar region of the ramus is growing superiorly, superimposition of the three sections on the oldest bone gives an estimation of the patterns of bone resorption (catabolic modeling) associated with growth of the mandibular ramus. (From Noorda CB. *Modeling and Remodeling in the Cortical Bone of Both Growing and Mature Rabbits*. [Master's thesis]. San Francisco: University of the Pacific; 1986.)

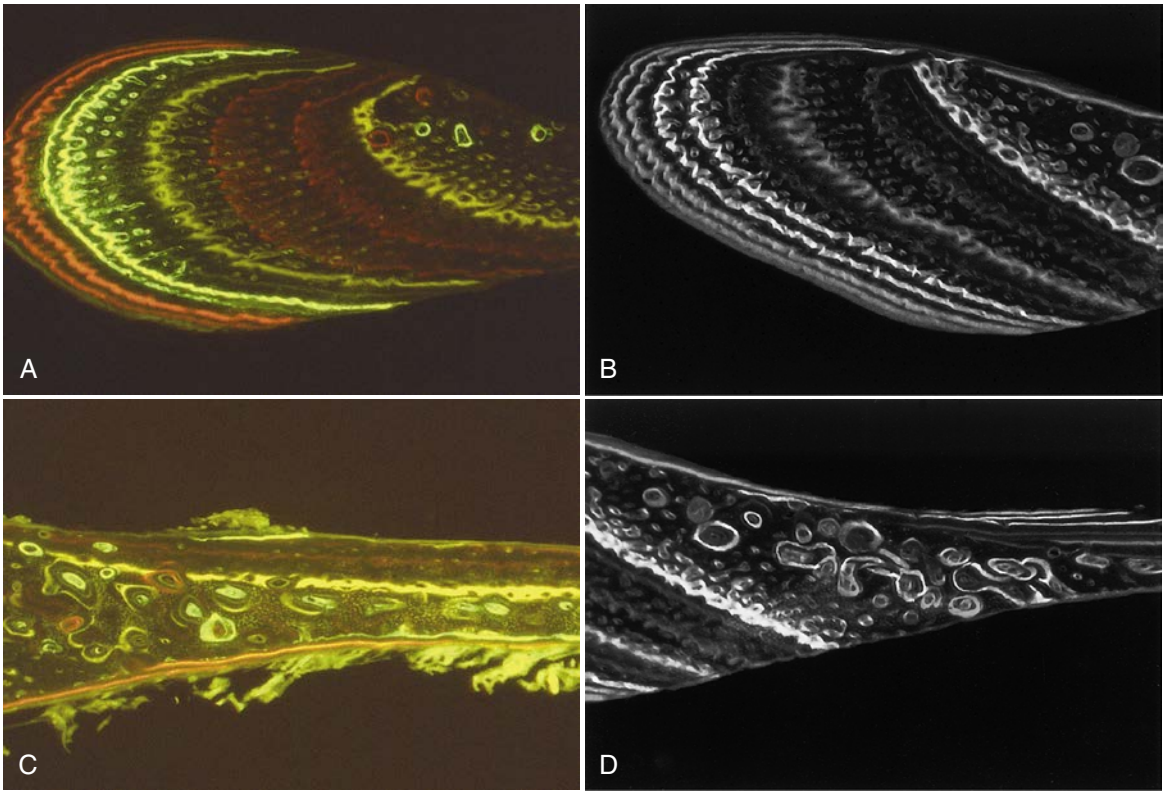


FIGURE 4-29 A, Fluorescent microscopy of weekly bone labels shows the patterns of anabolic modeling (bone apposition) in a rabbit. Note the diminishing space between the labels as growth slows and the animal achieves an adult skeletal form. B, A similar section from another rabbit in the same study shows the consistency of the growth pattern. C, In the first rabbit, the adjacent microscopic field shows several sites of bone remodeling in primary cortical bone formed about 6 to 12 weeks earlier. D, In the second rabbit, the adjacent microscopic field shows a consistent pattern of remodeling of new cortical bone at about 6 to 12 weeks after formation. (From Noorda CB. *Modeling and Remodeling in the Cortical Bone of Both Growing and Mature Rabbits*. [Master's thesis]. San Francisco: University of the Pacific; 1986.)

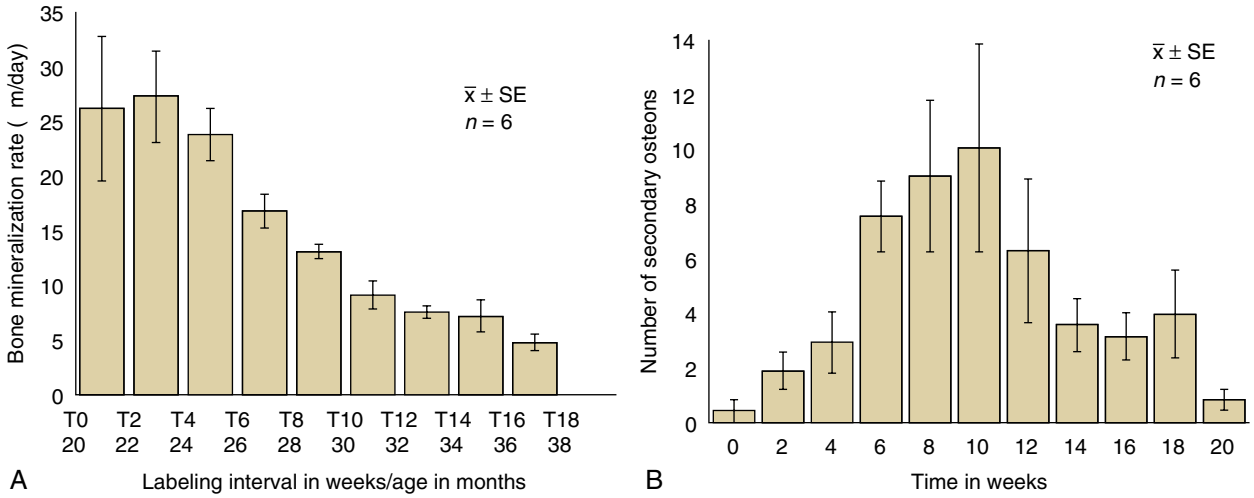


FIGURE 4-30 A, Age-related changes in the rate of periosteal apposition that occur in the posterior border of the mandibular ramus of the rabbit. Note the progressive decrease in the rate of periosteal bone apposition as the adolescent animals mature. B, Remodeling of new cortical bone. The highest incidence of remodeling to secondary osteons occurs when new cortical bone is 6 to 12 weeks old. (From Noorda CB. *Modeling and Remodeling in the Cortical Bone of Both Growing and Mature Rabbits*. [Master's thesis]. San Francisco: University of the Pacific; 1986.)

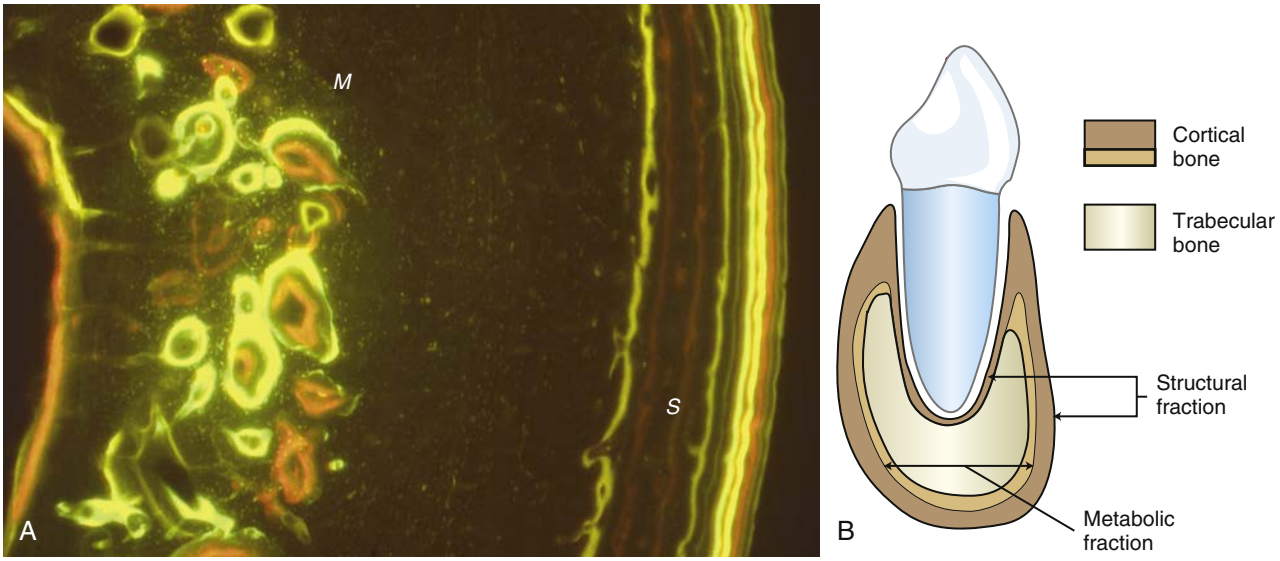


FIGURE 4-31 A, The structural (*S*) and metabolic (*M*) fractions of cortical bone are revealed by multiple fluorochrome labeling of a rabbit femur during the late growth and early adult periods. Continuing periosteal bone formation (*right*) contributes to structural strength, and high remodeling of the endosteal half of the compacta provides a continual supply of metabolic calcium. B, Structural and metabolic fractions of bone in the mandible. (Adapted from Roberts WE, et al. *Bone Dynamics in Orthodontic and Orthopedic Treatment: Craniofacial Growth Series*. Vol. 27. Ann Arbor: University of Michigan Press; 1991.)

The addition of new osseous tissue at the endosteal (inner) surface has little effect on overall bone strength. Structurally, the long bones and mandible are modified tubes—an optimal design for achieving maximal strength with minimal mass.[32] Within limits, loss of bone at the endosteal surface or within the inner third of the compacta has little effect on bone rigidity. The inner cortex can be mobilized to meet metabolic needs without severely compromising bone strength (Fig. 4-31, *B*); this is the reason patients with osteoporosis have bones with a normal diameter but thin cortices. Even under severe metabolic stress, the body follows a cardinal principle of bone physiology: maximal strength with minimal mass.[68]

BONE METABOLISM

Orthodontists and dentofacial orthopedists manipulate bone. The biomechanical response to altered function and applied loads depends on the metabolic status of the patient. Bone metabolism is an important aspect of clinical medicine that is directly applicable to orthodontics and orthopedics. This section discusses the fundamentals of bone metabolism with respect to clinical practice.

The skeletal system is composed of highly specialized mineralized tissues that have structural and metabolic functions. Structurally, lamellar, woven, composite, and bundle bone are unique types of osseous tissues adapted to specific functions. Bone modeling and remodeling are distinct physiologic responses to integrated mechanical and metabolic demands. Biomechanical manipulation of bone is the physiologic basis of orthodontics and facial orthopedics. Before addressing dentofacial considerations, an orthodontist must assess the patient's overall health status. Orthodontics is bone manipulative therapy, and favorable calcium metabolism is an important consideration. Because of the interaction of structure and metabolism, a thorough understanding of osseous structure and function is fundamental to patient selection, risk assessment, treatment planning, and retention of desired dentofacial relationships.[68,69]

Bone is the primary calcium reservoir in the body (Fig. 4-32). About 99% of the calcium in the body is stored in the skeleton. The continual flux of bone mineral responds to a complex interaction of endocrine, biomechanical, and cell-level control factors that maintain the serum calcium level at about 10 mg/dL.

Calcium homeostasis is the process by which mineral equilibrium is maintained. Maintenance of serum calcium levels at about 10 mg/d is an essential life support function. Life is thought to have evolved in the sea; calcium homeostasis is the mechanism of the body for maintaining the primordial mineral environment in which cellular processes evolved.[45] Calcium metabolism is one of the fundamental physiologic processes of life support. When substantial calcium is needed to maintain the critical serum calcium level, bone structure is sacrificed (see Fig. 4-30). The alveolar processes and basilar bone of the jaws also are subject to metabolic bone loss.[70] Even in cases of severe skeletal atrophy, the outer cortex of the alveolar process and the lamina dura around the teeth are preserved. This preservation is analogous to the thin cortices characteristic of osteoporosis.

Calcium homeostasis is supported by three temporally related mechanisms: (1) rapid (instantaneous) flux of calcium from bone fluid (which occurs in seconds); (2) short-term response by osteoclasts and osteoblasts (which extends from minutes to days); and (3) long-term control of bone turnover (over weeks to months). Precise regulation of serum calcium levels at about 10 mg/dL is essential for nerve conductivity and muscle function. A low serum calcium level can result in tetany and death. A sustained, high serum calcium level often is a manifestation of hyperthyroidism and some malignancies. Hypercalcemia may lead to kidney stones

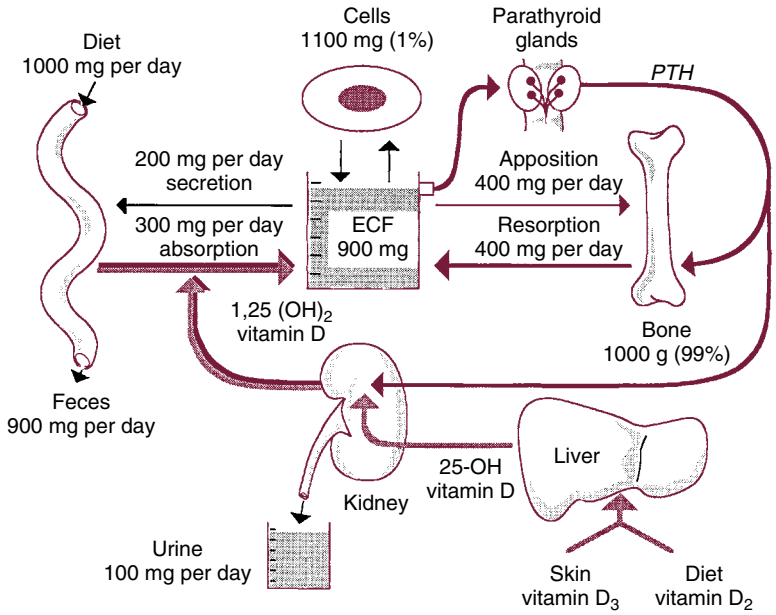


FIGURE 4-32 Calcium metabolism is a complex physiologic process. Maintaining zero calcium balance requires optimal function of the gut, parathyroid glands, bone, liver, and kidneys. Parathyroid hormone (*PTH*) and the active metabolite of vitamin D, 1,25-dihydroxycholecalciferol, are the major hormones involved. (Adapted from Roberts WE, et al. *Bone Dynamics in Orthodontic and Orthopedic Treatment: Craniofacial Growth Series*. Vol. 27. Ann Arbor: University of Michigan Press; 1991.)

and dystrophic calcification of soft tissue. Normal physiology demands precise control of the serum calcium level.[45,69,70]

CALCIUM CONSERVATION

Calcium conservation is the aspect of bone metabolism that involves preservation of skeletal mass. A failure in calcium conservation because of one problem or a combination of metabolic and biomechanical problems may leave a patient with inadequate bone mass for reconstructive dentistry, including orthodontics and orthognathic surgery. The kidney is the primary calcium conservation organ in the body (see Fig. 4-30). Positive calcium balance normally occurs during the growing period and for about 10 years thereafter. Peak skeletal mass is attained between 25 and 30 years. Zero calcium balance (see Fig. 4-30) is the ideal metabolic state for maintaining skeletal mass. Preservation of bone requires a favorable diet, endocrine balance, and adequate exercise.[68,69]

Endocrinology

Peptide hormones (e.g., PTH, growth hormone, insulin, and calcitonin) bind receptors at the cell surface and may be internalized with the receptor complex. Steroid hormones (e.g., vitamin D, androgens, and estrogens) are lipid soluble and pass through the plasma membrane to bind receptors in the nucleus.[68,69] PTH increases serum calcium by direct and indirect vitamin D–mediated effects. Clinically, a major effect of 1,25-DHCC is induction of active absorption of calcium from the gut. Sex hormones have profound effects on bone. Androgens (testosterone and other anabolic steroids) build and maintain musculoskeletal mass. The primary hypertrophic effect of androgens is to increase muscle mass. The anabolic effect on bone is a secondary biomechanical response to increase loads generated by the enhanced muscle mass. Estrogen, however, has a direct effect on bone; it conserves skeletal calcium by suppressing the activation frequency of bone remodeling.[71] At menopause, enhanced remodeling activation increases turnover.[72] Because a slightly negative calcium balance is associated with each remodeling event, a substantial increase in the turnover rate can result in rapid bone loss, leading to symptomatic osteoporosis. Even young women are susceptible to significant bone loss if the menstrual cycle (menses) stops.[36] Bone loss is a common problem in women who have low body fat and who exercise intensely (e.g., running or gymnastics), and in women who are anorexic.[73]

Estrogen replacement therapy (ERT) was widely recommended for calcium conservation and the prevention of osteoporosis in postmenopausal women.[74,75] However, the increased incidence and progression of breast cancer have greatly decreased the use of ERT.[76] The antiestrogen tamoxifen is used to treat some forms of breast cancers. Fortunately, in postmenopausal women, tamoxifen has a beneficial effect on bone similar to that of estrogen.[77] Raloxifene (Evista) has been shown to reduce the risk of osteoporosis and heart disease without increasing the risk of breast cancer. Some studies have even shown a substantial anticancer protective effect.[78] The reader is referred to the 5th edition of the textbook for additional details.

METABOLIC BONE DISEASE

Osteoporosis is a generic term for very low bone mass (osteopenia). The most important risk factor for the development of osteoporosis is age: after the third decade, osteopenia is related directly to longevity. Other high-risk factors are (1) a history of long-term glucocorticoid treatment, (2) slight stature, (3) smoking, (4) menopause or dysmenorrhea, (5) lack of or little physical activity, (6) low-calcium diet, (7) excessive consumption of alcohol, (8) vitamin D deficiency, (9) kidney failure, (10) liver disease (cirrhosis), and (11) a history of fractures. These risk factors are

effective in identifying 78% of those with the potential for osteopenia.[79,80] This is a good screening method for skeletally deficient dental patients. However, one must realize that more than 20% of individuals who eventually develop osteoporosis have a negative history for known risk factors. Any clinical signs or symptoms of low bone mass (e.g., low radiographic density of the jaws, thin cortices, or excessive bone resorption) are grounds for referral. A thorough medical workup, including a bone mineral density measurement, usually is necessary to establish the diagnosis of osteopenia. The term *osteoporosis* usually is reserved for patients with evidence of fracture or other osteoporotic symptoms. The treatment of metabolic bone diseases such as osteoporosis depends on the causative factors. Medical management of these often complex disorders is best handled by physicians specifically trained in bone metabolism.[68,69]

An increasing number of adults are seeking orthodontic treatment. Because all orthodontic and facial orthopedic therapy requires bone manipulation, orthodontists should have a detailed knowledge of bone physiology. All healthcare practitioners can play an important role in screening patients with high-risk lifestyles. Arresting the progression of metabolic bone disease is preferable to treating the condition after debilitating symptoms appear. A carefully collected history is the best screening method for determining which patients should be referred for a thorough metabolic workup. No age limit is specified for orthodontic treatment; however, clinicians must assess carefully the probability of metabolic bone disease.[68,69]

In addition to osteoporosis, orthodontists should be particularly vigilant for osteomalacia, a disease of poor bone mineralization associated with vitamin D deficiency,[50] and for renal osteodystrophy, a related condition in patients with compromised kidney function.[81]

Orthodontics usually is contraindicated in patients with active metabolic bone disease because of excessive resorption and poor rates of bone formation. If the metabolic problems (particularly negative calcium balance) are resolved with medical treatment, these patients can be treated orthodontically, assuming that sufficient skeletal structure remains. In fact, some individuals with osteoporosis retain near-normal jaw and alveolar bone, probably because they have healthy oral structures that are loaded normally. Apparently, under these circumstances, the disease preferentially attacks bone and other parts of the body with a less optimal mechanical environment.[68,69,82] A study of all adult female dental patients at Indiana University School of Dentistry showed that about 65% were at high risk for developing osteoporosis (these women were estrogen deficient or had at least two other risk factors).[83]

Osteoporosis is the most common metabolic bone disease, but orthodontic patients can be affected by many other osseous pathologies, such as renal osteodystrophy, hyperparathyroidism, hypoparathyroidism, hyperthyroidism, and osteomalacia osteogenesis imperfecta. In addition, bone can be compromised by a number of other systemic diseases. See Roberts et al.[23] for an orthodontically oriented review.

With the use of more potent bisphosphonates to treat osteoporotic patients, including Reclast, the question has arisen whether tooth movement is retarded, or even more important, will osteonecrosis of the jaw (ONJ)[84] develop after tooth extractions for orthodontics or miniscrew placement? Animal studies suggest that while potent bisphosphonates such as zoledronic acid greatly suppress bone remodeling,[85,86] they do not abolish the formation of new osteons at a site of injury. In other words, bone remodeling at a tissue level does occur in response to injury in an animal model that has received potent and high-dose bisphosphonates.

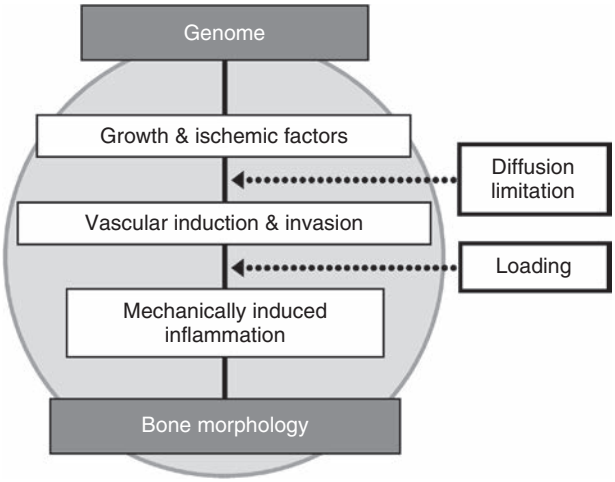


FIGURE 4-33 The genome dictates bone morphology by a sequence of three genetic mechanisms: (1) growth and ischemic factors, (2) vascular induction and invasion, and (3) mechanically induced inflammation. The latter two are influenced by two major physical influences: (1) diffusion limitation for maintaining viable osteocytes and (2) mechanical loading history. (From Roberts WE, Hartsfield JK. Bone development and function: genetic and environmental mechanisms. *Semin Orthod.* 2004;10(2):102.)

BIOMECHANICS

Mechanical loading is critical for skeletal health. An essential element of bone biomechanics is the inflammatory control of bone development, the adaptation to applied loads, and the response to pathologic challenges. The physiologic mechanism for controlling bone morphology involves inherent (genetic) and environmental (epigenetic) factors. There are three genetic mechanisms: (1) growth and ischemic factors, (2) vascular induction and invasion, and (3) mechanically induced inflammation. The latter two are influenced by two major physical influences: (1) diffusion limitation for maintaining viable osteocytes and (2) mechanical loading history (Fig. 4-33).[1]

Both bone formation and resorption are controlled at the cellular level by inflammatory mechanisms.[87] Thus, inflammation is an important factor in the mediation of bone physiology and pathology. Normal bone modeling and remodeling are controlled by inflammatory processes, both from a direct and indirect perspective. The P2X7 receptor is an important genetic mechanism for the production of inflammatory cytokines. It plays a crucial role in bone biology and inflammation, but it has no significant effect on teeth or alveolar bone morphology.[88] Thus, P2X7 knockout mice (animals with a deactivated gene) are a good model for defining the role of inflammatory cytokines in the tooth movement and alveolar adaptation to orthodontics loads. Skeletal adaptations, such as the orthodontic response, are related to the principal stress patterns in the periodontal ligament, and the P2X7 receptor plays a significant role in their mechanotransduction.[89]

Experiments in mice with deactivated *P2X7* genes have demonstrated that a principal function of the gene is the

promotion of necrotic tissue metabolism, by ensuring a normal acute-phase inflammatory response. An increased pattern of functional loading generates damage in affected musculoskeletal tissue, and there is a subsequent pain response that limits function during the initial healing stage. All forms of musculoskeletal adaptation to functional and applied loads involves an interaction of inflammatory mechanisms to stimulate bone cell activity, heal tissue damage, and limit function during the adaptive process. However, it is important to remember that the genetic mechanisms active in bone have interactive functions throughout the body. The receptor–activator system of NF-κB ligand (TNFSF11, also known as RANKL, OPGL, TRANCE, and ODF), as well as the tumor necrosis factor (TNF)-family receptor RANK, are essential regulators of bone remodeling. Recently, RANKL and RANK were found to have an essential role in the brain in initiating fever,[90] another important inflammatory mechanism. Thus, genetic inflammatory mechanisms controlling bone physiology are just beginning to be understood at the systemic level. Control of most bone modeling and some remodeling processes are related to strain history, which usually is defined in microstrain (με) (deformation per unit length $\times 10^{-6}$).[91] Repetitive loading generates a specific response, which is determined by the peak strain.[92-96] In an attempt to simplify the often conflicting data, Frost[97] proposed the mechanostat theory. Reviewing the theoretic basis of this theory, Martin and Burr[33] proposed that (1) subthreshold loading of less than 200 με results in disuse atrophy, manifested as a decrease in modeling and an increase in remodeling; (2) physiologic loading of about 200 to 2500 με is associated with normal, steady-state activities; (3) loads exceeding the minimal effective strain (about 2500 με) result in a hypertrophic increase in modeling and a concomitant decrease in remodeling; (4) after peak strains exceed about 4000 με, the structural integrity of bone is threatened, resulting in pathologic overload. Figure 4-34 is a representation of the mechanostat. Many of the concepts and microstrain levels are based on experimental data.[33,98] The strain range for each given response probably varies among species and may be site-specific in the same individual.[21,33,93-96] However, the mechanostat provides a useful clinical reference for the hierarchy of biomechanical responses to applied loads.

Normal function helps build and maintain bone mass. Suboptimally loaded bones atrophy as a result of increased remodeling frequency and inhibition of osteoblast formation.[99] Under these conditions, trabecular connections are lost and cortices are thinned from the endosteal surface. Eventually the skeleton is weakened until it cannot sustain normal function. An increasing number of adults with a history of osteopenia caused by metabolic bone disease are seeking orthodontic treatment for routine malocclusions. Assuming that the negative calcium balance is corrected and adequate bone structure remains, patients with a history of osteoporosis or other metabolic bone disease are viable candidates for routine orthodontic therapy. The crucial factor is the residual bone mass in the area of interest after the disease process has been arrested (Fig. 4-35).

When flexure (strain) exceeds the normal physiologic range, bones compensate by adding new mineralized tissue at the periosteal surface. Adding bone is an essential compensating mechanism because of the inverse relationship between load (strain magnitude) and the fatigue resistance of bone.[100] When loads are less than 2000 με, lamellar bone can withstand millions of loading cycles, more than a lifetime of normal function. However, increasing the cyclic load to 5000 με, about 20% of the ultimate strength of cortical bone, can produce fatigue failure in 1000 cycles, which is achieved easily in only a few weeks of normal activity. Repetitive overload at less than one-fifth of the ultimate strength of lamellar bone (25,000 με, or 2.5% deformation) can lead to skeletal failure, stress fractures, and shin splints.

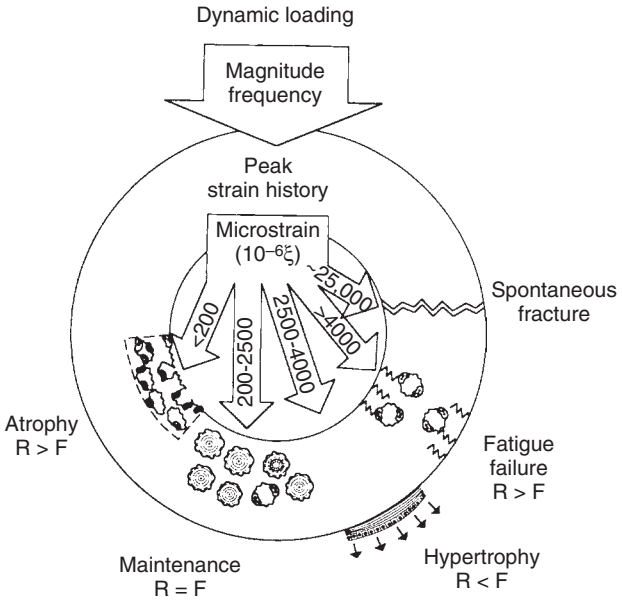


FIGURE 4-34 The mechanostat concept of Frost as defined by Martin and Burr. Bone formation (*F*) and resorption (*R*) are the modeling phenomena that change the shape or form (or both) of a bone. The peak strain history determines whether atrophy, maintenance, hypertrophy, or fatigue failure occurs. Note that the normal physiologic range of loading (Maintenance R + F) is only at less than 10% of maximal bone strength (spontaneous fracture). Fatigue damage can accumulate rapidly at greater than 4000 με.

From a dental perspective, occlusal prematurities or parafunction may lead to compromise of periodontal bone support. Localized fatigue failure may be a factor in periodontal clefting, alveolar recession, tooth oblation (cervical ditching), or TMJ arthrosis. Guarding against occlusal prematurities and excessive tooth mobility, while achieving an optimal distribution of occlusal loads, are important objectives for orthodontic treatment. The human masticatory apparatus can achieve a biting strength of more than 2200 N, or more than 500 lb of force.[101,102] Because of the high magnitude and frequency of oral loads, functional prematurities used during orthodontic treatment could contribute to isolated incidences of alveolar clefting (Fig. 4-36, *A*) and root resorption (Fig. 4-36, *B*). Excessive tooth mobility should be monitored carefully during active orthodontic treatment and retention. Prevention of occlusal prematurities is a particular concern in treating periodontally compromised teeth.

SUTURES

The facial sutures are important mediators of skeletal adaptation to craniofacial growth and biomechanical therapy.[103] Mechanical forces, both functional and therapeutic, regulate sutural growth by inducing sutural mechanical strain.[104] Expansion of the midpalatal suture often is a key objective in

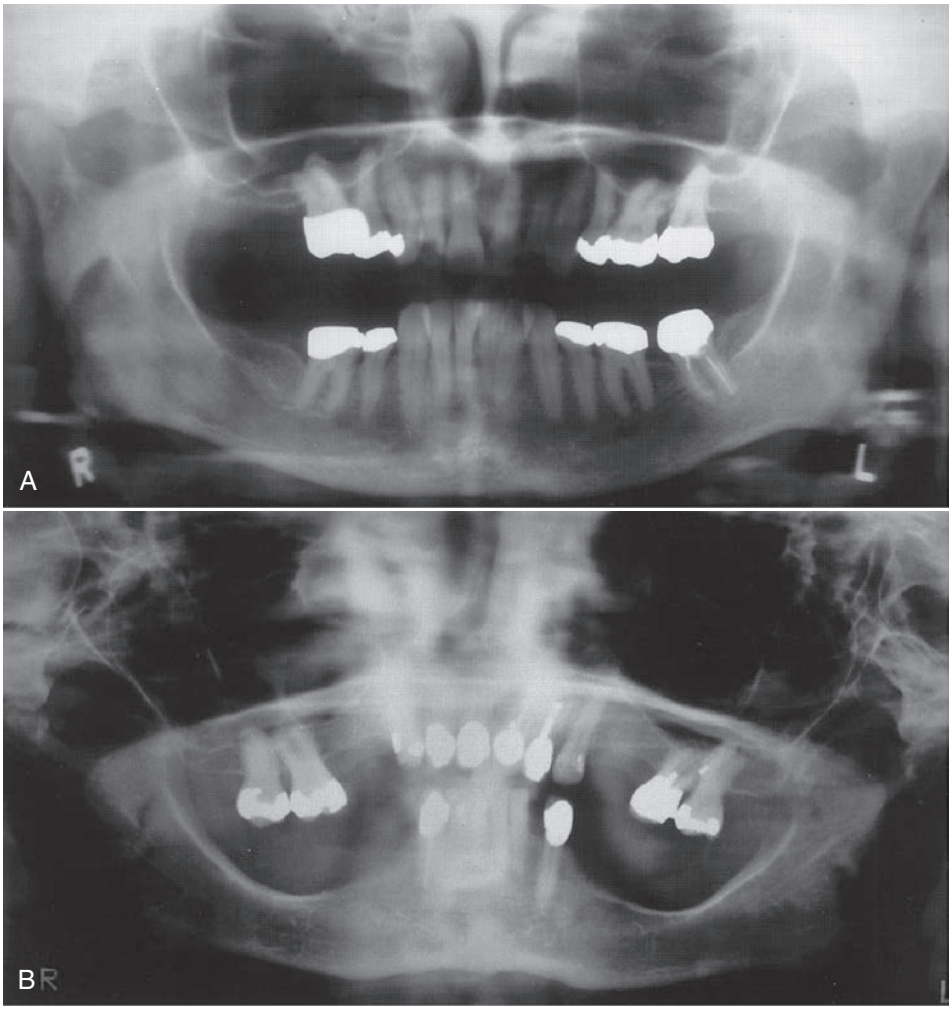


FIGURE 4-35 Two postmenopausal women with systemic osteopenia present widely varying patterns of lower posterior bone loss. **A,** Alveolar bone in the buccal segments is well preserved by functional loading of natural teeth. **B,** Severe resorption of the alveolar process and basilar mandible has occurred in the absence of adequate functional loading. (From Roberts WE. Fundamental principles of bone physiology, metabolism and loading. In: Naert I, et al., eds. *Osseointegration in Oral Rehabilitation*. London: Quintessence; 1993.)

dentofacial orthopedic treatment. Although the potential for sutural expansion has been appreciated since the middle of the nineteenth century, Andrew Haas[105] introduced the modern clinical concepts of rapid palatal expansion (RPE) in the last half of the twentieth century. RPE is very effective in growing children, but the skeletal response of the craniofacial sutures of adults is questionable. The nonsurgical RPE of young adult females was documented with a bone scintigraphy study, demonstrating a bone metabolic pattern consistent with both dental and skeletal effects.[7] Biomechanics studies using finite element models have helped explain the highly variable clinical reactions in adults, depending on if sutures are patent or fused.[106] Despite the long history of palatal expansion, little was known of the cell kinetics of osteogenesis and the bone remodeling response associated with it. Sutures and the PDL were widely assumed to have similar mechanisms of osseous adaptation. Chang et al.[107,108] compared the osteogenic reaction in the expanded midpalatal suture with orthodontically induced osteogenesis in the PDL of adjacent incisors (Figs. 4-37 to 4-40). The widened PDL resulted in direct osteogenic induction of new bone, whereas the adjacent expanded suture experienced hemorrhage, necrosis, and a wound-healing response. Vascular invasion of the blood clot in the expanded suture was a prerequisite for new bone formation. Chang et al.[108] also defined the angiogenic capillary budding process associated with the propagation of perivascular osteogenic cells (Fig. 4-41). After its vascularity had been reestablished, the expanded midpalatal suture and adjacent widened PDL produced new osteoblasts by the same mechanism. Pericytes, the osteogenic cells that are perivascular to the venules (Fig. 4-42), are the cells of origin for preosteoblasts. This vascularly mediated osteogenic mechanism for producing osteoblasts was described earlier in this chapter. (For the detailed cell-kinetic analysis that established this important mechanism, see Chang et al.)[107,108]

The role of perivascular cells in the origin of PDL osteoblasts first was reported in 1987.[109] Over the past decade, a number of investigators have reported the same mechanism for the production of osteoblasts throughout the body. Doherty

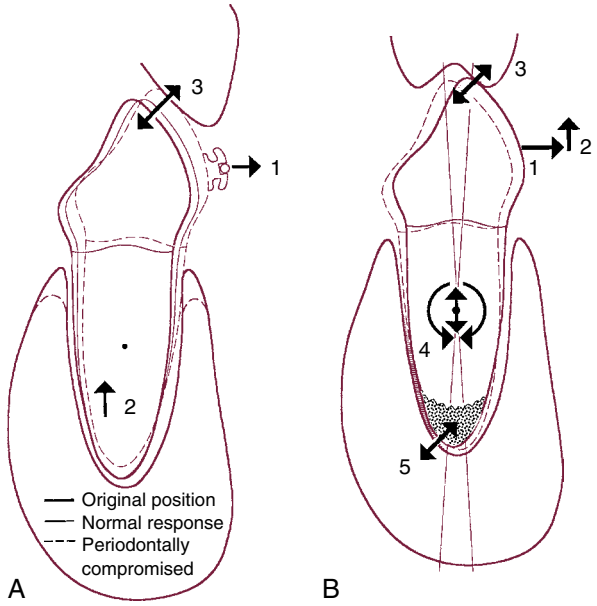


FIGURE 4-36 **A,** A moderate load in the buccal direction (1) results in tipping displacement of the crown. In the absence of vertical constraint, a normal healthy tooth would be expected to extrude slightly because of the inclined plane effect of the root engaging the tapered alveolus (2). As a result of diminished bone support and destruction of restraining collagen fibers at the alveolar crest, a periodontally compromised tooth may tip and extrude considerably more. Depending on the occlusion, this displacement may cause an occlusal prematurity (3). **B,** Orthodontic tipping (1) with an extrusive component (2) may produce an occlusal prematurity (3) and mobility (4). An individual tooth in chronic occlusal trauma is expected to fatigue the root apex continuously. This combination of physical failure in a catabolic environment may lead to progressive root resorption (5).

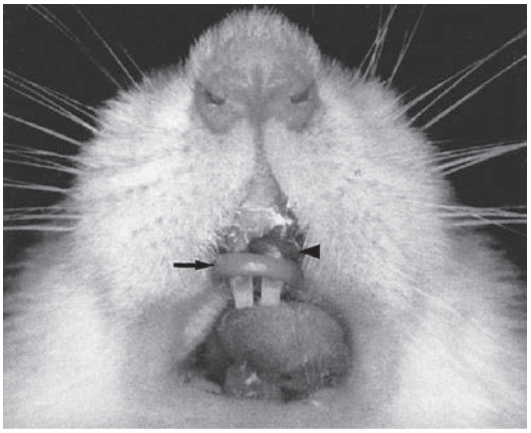


FIGURE 4-37 An expansion appliance is placed on the maxillary incisors of a rat. A 1-mm-diameter elastomeric ring (*arrowhead*) was fitted into the left incisor; a 2-mm-diameter elastomeric ring (*arrow*) encircled both incisors, 2 mm from cutting edges. The 2-mm ring constricts the incisors while the interproximal elastic elicits a parallel separation of the interpremaxillary suture. (From Chang H-N, et al. Angiogenesis and osteogenesis in an orthopedically expanded suture. *Am J Orthod Dentofac Orthop*. 1997;111(4):382-90.)

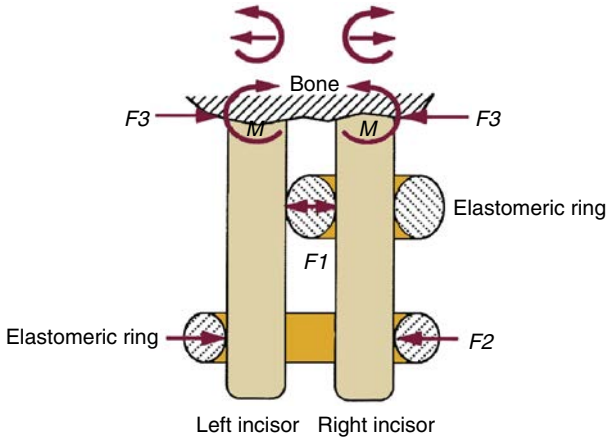


FIGURE 4-38 Forces (*F*) and moments (*M*) on a tooth. *F1* and *F2* were produced by inner and outer elastomeric rings, respectively. This illustration of the device demonstrates the formation of a couple that resulted in parallel separation of the interpremaxillary suture. As measured in a pilot study using a Dontrix tension gauge, the outer elastomeric ring exerted about 200 *g* of initial separation force (*F2*), of which 90 *g* remained at the end of day 3. This force level (90 *g*) is suitable for premaxillary expansion in rats. (From Chang H-N, et al. Angiogenesis and osteogenesis in an orthopedically expanded suture. *Am J Orthod Dentofac Orthop*. 1997:111(4):382-90.)

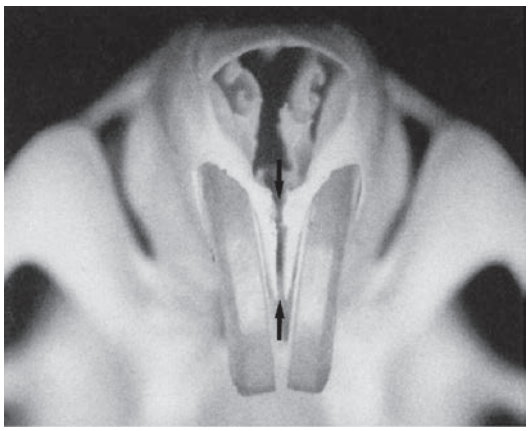


FIGURE 4-39 A dry skull expanded as illustrated in Figure 4-36 shows parallel separation of the interpremaxillary suture (*arrow*). (From Chang H-N, et al. Angiogenesis and osteogenesis in an orthopedically expanded suture. *Am J Orthod Dentofac Orthop*. 1997;111(4):382-90.)

et al.[110] recently reviewed the literature and provided evidence that vascular pericytes express osteogenic potential in vivo and in vitro. What is now clear is that perivascular osteogenesis is not a mechanism unique to the PDL and sutures, but rather it is the source of osteoblasts all over the body under a variety of osteogenic conditions.

Parr et al.[111] used an innovative endosseous implant mechanism to expand the nasal bones in young adult rabbits with forces from 1 to 3 N. Injection of multiple fluorochrome bone labels documented the bone modeling and remodeling reactions that occurred not only adjacent to the suture but also throughout the nasal bones. Expansion of a suture results in

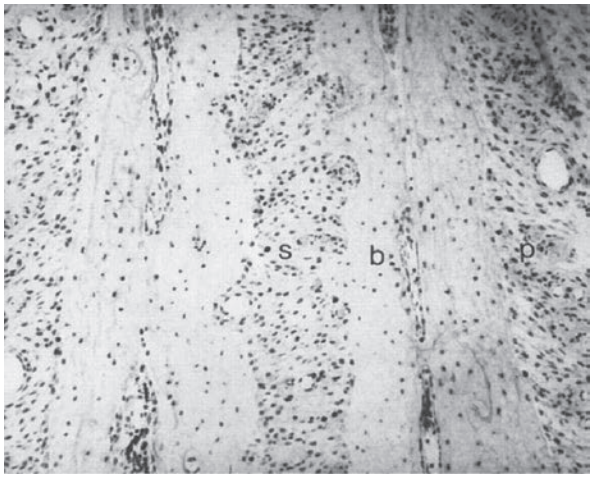


FIGURE 4-40 Photomicrograph of a sagittal section of the interpremaxillary suture, showing the relationship of expanded suture (*s*), alveolar bone (*b*), and periodontal ligament (*p*). (Stained with hematoxylin and eosin; original magnification ×40.) (From Chang H-N, et al. Angiogenesis and osteogenesis in an orthopedically expanded suture. *Am J Orthod Dentofac Orthop.* 1997;111(4):382-90.)

a regional adaptation of adjacent bones similar to the postoperative regional acceleratory phenomenon that is characteristic of bone wound healing.[33] Parr et al.[111] described the bone formation rate and mineral apposition rate for new bone formed in the suture (Figs. 4-43 to 4-45). Sutural expansion, relative to load decay, is shown for repeatedly reactivated 1- to 3-N loads (Fig. 4-46). Osseointegrated implants were excellent abutments for sutural expansion mediated by loads as large as 3 N.

Overall, expanded sutures are less efficient at initiating osteogenesis because of postactivation necrosis.[107] After a wound-healing response has occurred to reestablish sutural vitality, the vascularly mediated origin of osteoblasts is the same as for the PDL and other skeletal sites. Expansion of a suture results in a regional acceleration of bone adaptive activity, which allows for extensive adaptation of the affected bones to new biomechanical conditions. These results indicate that sutural expansion within physiologic limits is a clinically viable means of repositioning the bones of the craniofacial complex to improve aesthetics and function. With respect to fundamental bone physiology, sutural expansion is similar to surgically mediated distraction osteogenesis. Using sequential labels of ^{3}H-thymidine and bromodeoxyuridine in rabbits, Sim[112] demonstrated that the osteoblast histogenesis sequence for evolving secondary osteons was a perivascular process (Fig. 4-47) similar to that previously demonstrated for the PDL[109] and the intermaxillary suture.[107,108] The Sim data confirmed the hypothesis that the perivascular connective tissue cells proliferate and migrate along the surface of the invading capillaries or venules. Figure 4-48 is a three-dimensional perspective of a remodeling focus (cutting/filling cone) in cortical bone, which demonstrates that perivascular cells, near the head of the proliferating blood vessel, are the source of osteoblasts for the filling cone. Confirmation of a perivascular origin of osteoblasts in PDL, sutures, and cortical bone remodeling foci strongly suggests that all osteoblasts, at least in the peripheral skeleton, are derived from perivascular precursors. These data suggest that less differentiated osteogenic cells grow along the surface of bone-related blood vessels (capillaries and venules) as they invade blood clots or other connective tissue spaces in preparation for osteogenesis. From a clinical perspective, the perivascular origin of osteoblasts confirms an important surgical principle: preservation of the blood supply is essential for optimal healing of bone.

Maxillary protraction in skeletal Class III patients is particularly effective in the mixed dentition,[113] but bone biomechanics studies have failed to demonstrate the advantage of expanding the palate at the same time to enhance the sagittal response.[114] Determining bone age from hand-wrist films radiographic analysis is effective for screening patients most likely to have a positive response to maxillary protraction.[115] Recently, miniplates for anchorage of intermaxillary elastics have proven to be very effective for changing detrimental growth patterns.[116,117]

DISTRACTION OSTEOGENESIS

Distraction osteogenesis (DO) is a surgical procedure for reconstructing skeletal deformities of the skeleton, jaws, and alveolar processes. A corticotomy (osteotomy) divides the bone into segments, or if there is an intervening suture or PDL, the bone that is resisting movement is removed. A surgically assisted RPE is the most common distraction procedure used in orthodontics. Distraction of the PDL is a method for achieving rapid tooth movement,[118] but the method is too invasive for routine clinical procedures.

For DO of the maxilla, mandible, and alveolar process, the osseous segments are progressively moved apart, generating new bone in the defect. The desired position of the segments is achieved in the distraction phase. The segments are then retained, allowing the gap to fuse, and the newly generated bone remodels into a more mature structure (consolidation phase). A particular advantage of distraction osteogenesis is the simultaneous increase in length and volume of the investing soft tissues. This variation of the bone-lengthening procedure, introduced to orthopedic surgery by Ilizerov to lengthen limbs,[119] is now used extensively by reconstructive plastic and maxillofacial surgeons for the correction of micrognathia, midface deficiencies, and orbital anomalies in patients with craniofacial deformities. Although the procedure has been used effectively for some mandibular problems,[120] it has been problematic in some patients.[121] DO is widely used for maxillary deficiencies, for which favorable follow-up has been reported.[122] Surgically assisted protraction of the maxilla is an effective procedure, particularly in patients with craniofacial anomalies.[123] When all the bony attachments are cut for a structure such as the maxilla, it is free to move in any direction dictated by the applied biomechanics.[124] Much like a suture, the osseous margins can resorb if exposed to compression or become bone-forming surfaces when exposed to tension. Rigid external distraction has evolved as superior to a face mask for controlling the protraction and vertical position of the maxilla.[125] Miniimplants and miniscrews have proved to be effective for controlling osteotomy segments under some circumstances.[126] New bone can be generated in the alveolar process, and teeth can be moved into it.[127] This method can be used for rapid retraction of canines or correction of tooth size–to–arch length discrepancies.[128]

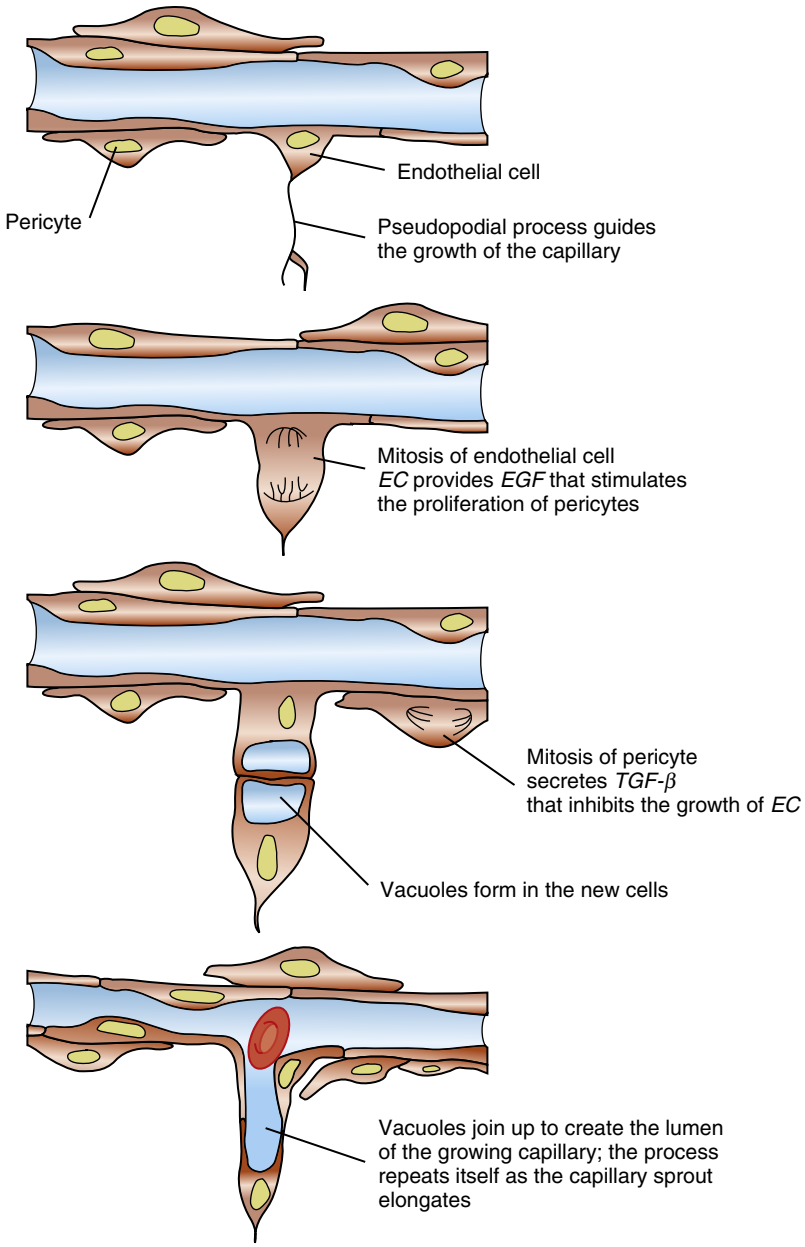


FIGURE 4-41 Angiogenesis involves a well-defined sequence of capillary budding followed by an extension of the perivascular network of pericytes, which are the source of osteoprogenitor cells. *EC*, Endothelial cell; *EGF*, epidermal growth factor; *TGF-β*, transforming growth factor β. (Redrawn from Chang H-N, et al. Angiogenic induction and cell migration in an orthopaedically expanded maxillary suture in the rat. *Arch Oral Biol*. 1996;41(10):986.)

ORTHODONTIC TOOTH MOVEMENT

Figure 4-49 illustrates a typical tooth movement response after application of a moderate, continuous load (0.2 to 0.5 N, or about 20 to 50 g). The orthodontic response is divided into three elements of tooth displacement: initial strain, lag phase, and progressive tooth movement. Initial strain of 0.4 to 0.9 mm occurs in about 1 week[21,129,130] because of PDL displacement (strain), bone strain, and extrusion (Fig. 4-50). The initial deformation response varies according to the width of the PDL, root length, anatomic configuration, force magnitude, occlusion, and periodontal health. Initial tooth displacement occurs within seconds,[131,132] but actual compression of the PDL requires 1 to 3 hours (see Fig. 4-50).

Unfortunately, no studies have used a broad range of invasive and noninvasive techniques to define completely the fluid and solid mechanics associated with initiating and sustaining tooth movement. Figure 4-47 is an attempt to unify a broad range of data. The fluid mechanics of root displacement in the PDL probably accounts for about 0.3 mm of crown movement.[133] Assuming an average anatomic configuration and bone strain of 1% (about 40% of ultimate strength) or less,[33] distortion of mineralized tissue caused by bending and creep[33,134] probably

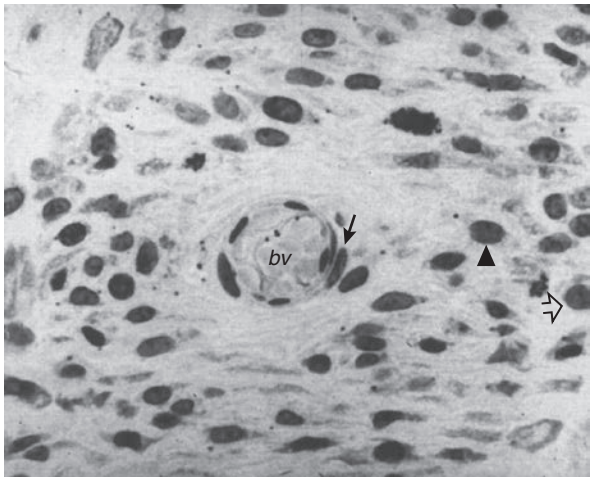


FIGURE 4-42 Photomicrograph of autoradiography of an expanded interpremaxillary suture, showing blood vessel (*bv*) and paravascular cells. Note the relationship of pericyte (*solid arrow*), fibroblast-like cells (*arrowhead*), and mature osteoblast (*open arrow*) lining the suture–bone interface. (Stained with hematoxylin and eosin; original magnification ×400.) (From Chang H-N, et al. Angiogenesis and osteogenesis in an orthopedically expanded suture. *Am J Orthod Dentofac Orthop*. 1997;111(4):382-90.)

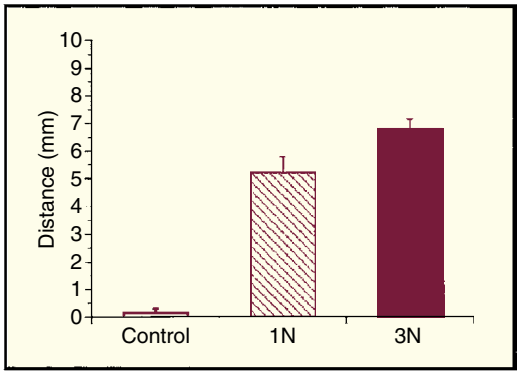


FIGURE 4-43 Expansion of the suture between the nasal bones of a rabbit is expressed as the mean difference of initial and final measurements between implants for the three loading groups (mean ± SEM, all groups significant at $p < 0.05$.). (From Parr JA, et al. Sutural expansion using rigidly integrated endosseous implants: an experimental study in rabbits. *Angle Orthod*. 1987;67(4):287.)

accounts for about another 0.3 mm of the initial movement (see Chapters 2 and 13).[133]

Occlusal Trauma and Root Resorption

Extrusion in response to a horizontal load may be a component of initial displacement,[131] depending on the direction of the force, the point of application, and the axial inclination of the root. However, varying amounts of extrusion can be expected because of the inclined-plane effect of the root apex being compressed against the alveolus (see Fig. 4-50). The tendency toward extrusion and enhanced horizontal displacement during tooth movement varies directly with force magnitude and periodontal compromise of the dentoalveolar fibers at the

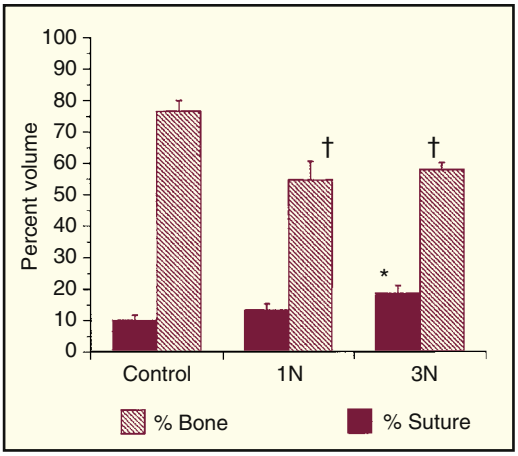


FIGURE 4-44 Volume percent of suture and bone for three loading groups (mean ± SEM); an asterisk (*) indicates significant difference in the percentage of sutural expansion from the control at $p < 0.05$; a dagger (†) indicates significant difference in the percentage of bone from the control at $p < 0.05$. (From Parr JA, et al. Sutural expansion using rigidly integrated endosseous implants: an experimental study in rabbits. *Angle Orthod*. 1987;67(4):287.)

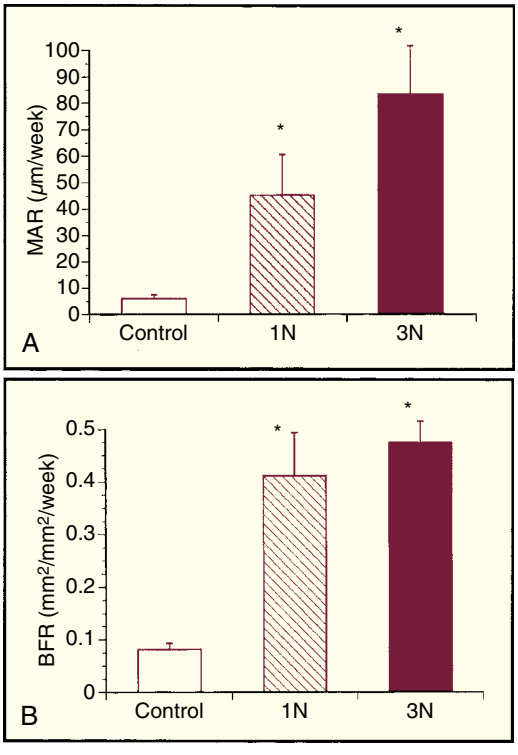


FIGURE 4-45 **A,** Mineral apposition rate (MAR). **B,** The bone formation rate (BFR) was calculated at the suture during the final 6 weeks of loading for three loading groups (mean ± SEM; an asterisk [*] indicates significant difference from the control at $p < 0.05$.). (From Parr JA, et al. Sutural expansion using rigidly integrated endosseous implants: an experimental study in rabbits. *Angle Orthod*. 1987;67(4):287.)

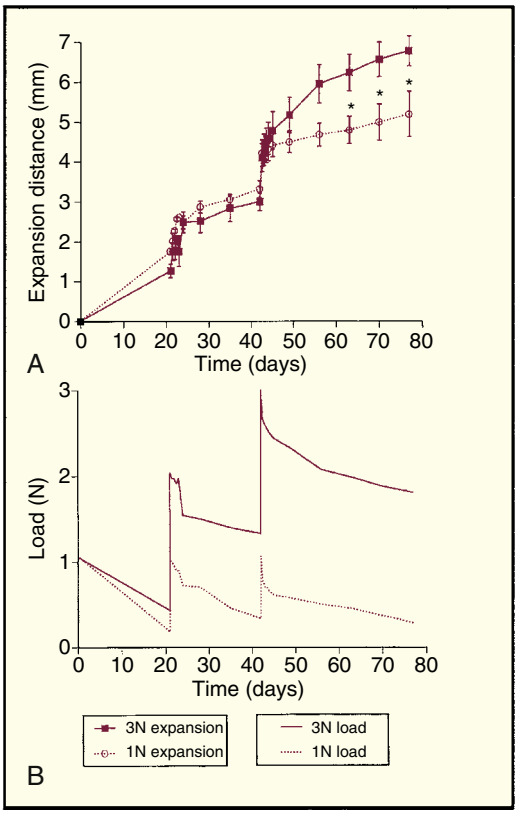


FIGURE 4-46 **A,** Sutural expansion measured as an increase in the distance between implants. The slope of this curve is the rate of sutural expansion; 3 N is significantly greater than 1 N at these time points ($p < 0.05$). **B,** Load on the suture as a function of time. Load was calculated using the formula $F = kx$, where k is the spring constant and x is the distance between implants. As sutural expansion occurs, force decays. Loads were placed at day 0 and adjusted at days 21 and 42. (From Parr JA, et al. Sutural expansion using rigidly integrated endosseous implants: an experimental study in rabbits. *Angle Orthod.* 1987;67(4):287.)

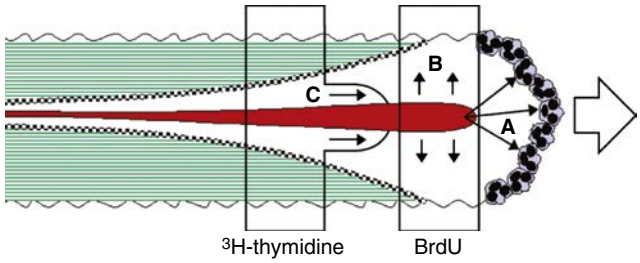


FIGURE 4-47 A cutting/filling cone in rabbit cortical bone shows the intravascular origin of osteoclasts **(A).** The perivascular proliferation and migration away from the perivascular surface **(B)** are demonstrated by bromodeoxyuridine (BrdU) labeling and nuclear volume morphometry. A sequence of ^{3}H-thymidine labels from 2 to 72 hours before sacrifice and nuclear morphometric analysis revealed migration of proliferating perivascular cells in the direction of vascular invasion **(C).** These data demonstrate the perivascular origin of osteoblasts in evolving secondary osteons. (From Sim Y. *Cell Kinetics of Osteoblast Histogenesis in Evolving Rabbit Secondary Haversian Systems Using a Double Labeling Technique with ^{3}H-Thymidine, and Bromodeoxyuridine.* [PhD thesis]. Indianapolis: Indiana University, School of Dentistry; 1995.)

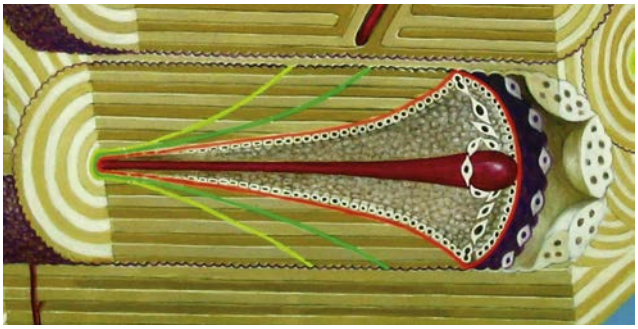


FIGURE 4-48 An evolving secondary osteon, moving to the right, shows a head of multinucleated osteoclasts (*right*), followed by a layer of mononuclear cells secreting cement substance (*blue*) to cover the scalloped resorption arrest line. The perivascular osteogenic cells proliferate and differentiate to osteoblasts, which form the new secondary osteon. Three sequential colored bone labels (*yellow*, *green*, and *orange*) allow the calculation of the velocity of the cutting/filling cone through cortical bone.

alveolar crest. Extrusion and occlusal prematurities are distinct possibilities, particularly for periodontally compromised teeth, and these conditions depend on the vertical constraint of a clinically applied load (see Fig. 4-36, *A*). If occlusal prematurity is a chronic periodontal trauma, root resorption may result because of catabolic cytokines in the PDL[135] or because of fatigue failure (see Fig. 4-36, *B*).

Remodeling/Repair of Root

Remodeling is the physiologic term for internal turnover of a mineralized tissue, without a change in its overall form. Pioneer histologic studies by Dr. Kaare Reitan (Fig. 4-51) in humans and in multiple species have demonstrated that root-resorption cavities are usually repaired (filled) with secondary cementum. In effect, this is "repair" of the root of a tooth. While cementum repair is very similar, it is not identical to remodeling of trabecular bone. The similarities between bone remodeling and root resorption are striking: Kimura and coworkers[136] concluded that the "odontoclasts" of root resorption have an intravascular origin similar to the osteoclasts of bone remodeling. Considering all available evidence, it appears that root resorption is a portion of the turnover process to replace damaged root structure. From a clinical perspective, it is important for orthodontists to consider mechanics and retention procedures that provide for adequate periods of atraumatic rest to allow resorbing sites to refill with cellular cementum.[137] Continuous forces, particularly if they are associated with traumatic occlusion, may result in permanent loss of root structure[41] if multiple sites of root resorption communicate before the initiation of their respective cementum repair phases. The reader is referred to the 5th edition of the textbook for additional details.

Induction of the Tooth Movement Response

Progressive displacement of the tooth relative to its osseous support stops in about 1 week (see Fig. 4-49), apparently because of areas of PDL necrosis (hyalinization). This lag phase varies considerably; it usually lasts 2 to 3 weeks but may be as long as 10 weeks.[129] Clinical experience and histologic studies[129,133] suggest that the duration of the lag phase is related directly to the patient's age, the density of the alveolar bone, and the extent

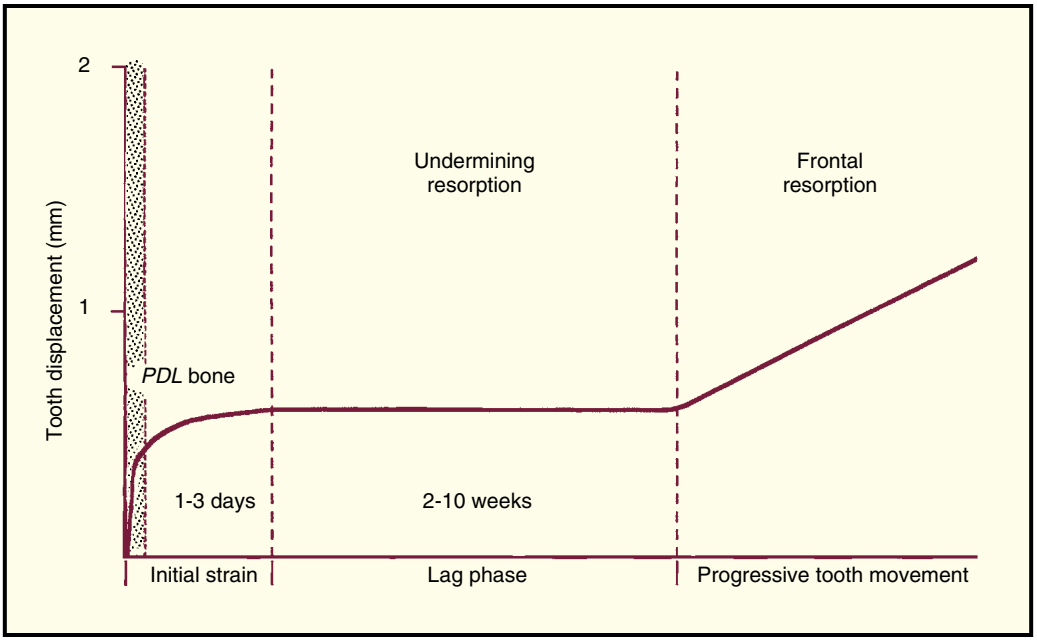


FIGURE 4-49 After application of a moderate orthodontic load (0.2 to 0.5 N, or about 20 to 50 g), tooth displacement is divided into three phases: (*1*) initial strain for 1 to 3 days in the periodontal ligament (*PDL*) and supporting bone; (*2*) a variable lag phase, in which undermining resorption removes bone adjacent to crushed areas in the PDL; and (*3*) progressive tooth movement when frontal resorption in the PDL limits the rate of orthodontic correction.

of PDL necrotic zones. After undermining resorption restores vitality to the necrotic areas of the PDL, tooth movement enters the secondary,[129,130] or progressive, tooth movement phase (see Fig. 4-49). Frontal resorption (modeling) in the PDL and initial remodeling events (resorption cavities) in cortical bone ahead of the advancing tooth (see Fig. 4-25, *A*) allow for progressive tooth movement at a relatively rapid rate. Gene expression during the initiation of tooth movement has recently been reviewed.[138] Low-energy laser irradiation enhances the initiation and velocity of tooth movement by increasing the expressions of matrix metalloproteinase-9, cathepsin K, and $\alpha_v\beta_3$ integrin.[139]

The mechanism of sustained tooth movement is a coordinated array of bone resorption and formation events (see Fig. 4-25, *A*). Both fundamental mechanisms of osseous adaptation, modeling, and remodeling, are involved.[140] A modeling response is noted in the alveolus; bone resorption occurs where the PDL is compressed (in the direction of movement), and bone formation maintains the normal width of the trailing PDL. By means of this coordinated series of surface modeling events, the alveolus drifts in the direction of tooth movement. The modeling events of tooth movement commonly are referred to as areas of compression and tension within the PDL.[129,130] For small amounts of tooth movement (less than 1 mm) over 1 or 2 months, PDL modeling probably is the predominant mechanism of tooth movement. However, when teeth are moved greater distances over longer periods, the PDL response is supplemented by alveolar bone remodeling and periosteal modeling (Figs. 4-52 and 4-53).

Remodeling of dense alveolar bone (see Fig. 4-52) may enhance the rate of tooth movement[133] and replace the less mature osseous tissue formed by rapid PDL osteogenesis (see Fig. 4-53). Resorption cavities ahead of the moving tooth reduce the density of cortical bone (see Fig. 4-52). These intraosseous cavities are the initial remodeling events that occur during the first month of the remodeling cycle (see Fig. 4-20). With progressive tooth movement, it appears that these resorption cavities are truncated remodeling events.[133]

Continuous force[141,142] or reactivation at about 1-month intervals is expected to yield the maximal rate of tooth movement through cortical bone. The remodeling-dependent concept of long-range tooth movement has important clinical implications. Efficient mechanics and regular reactivations at about 4-week intervals long have been associated with optimal rates of tooth movement.[133] However, breakage, distortion of appliances, and appointment failures substantially increase treatment time. One reason for slow tooth movement in uncooperative patients may be the tendency for resorption cavities initiated by orthodontic activation to complete the remodeling cycle by refilling with new bone if appropriate mechanics are not maintained. Repeatedly reinitiating force after periods of periodontal recovery requires one startup period after the other (see Fig. 4-47) to reestablish the modeling and remodeling mechanisms to move a tooth through dense cortical bone.

Secondary osteons are formed in new cortical bone trailing a moving tooth (Fig. 4-55). The major modeling and remodeling events associated with sustained buccal movement (controlled tipping) of a lower premolar are summarized in Figure 4-54. The efficiency of bone resorption is the rate-limiting factor in tooth movement. Bone is removed ahead of the moving tooth by two mechanisms: frontal resorption at the PDL interface and initial remodeling events (resorption cavities) in the cortical plate. In addition to PDL modeling of the alveolus (bone resorption in the area of pressure and bone formation in the area of tension), Figure 4-56 shows surface modeling on periosteal and endosteal surfaces. These coordinated modeling events

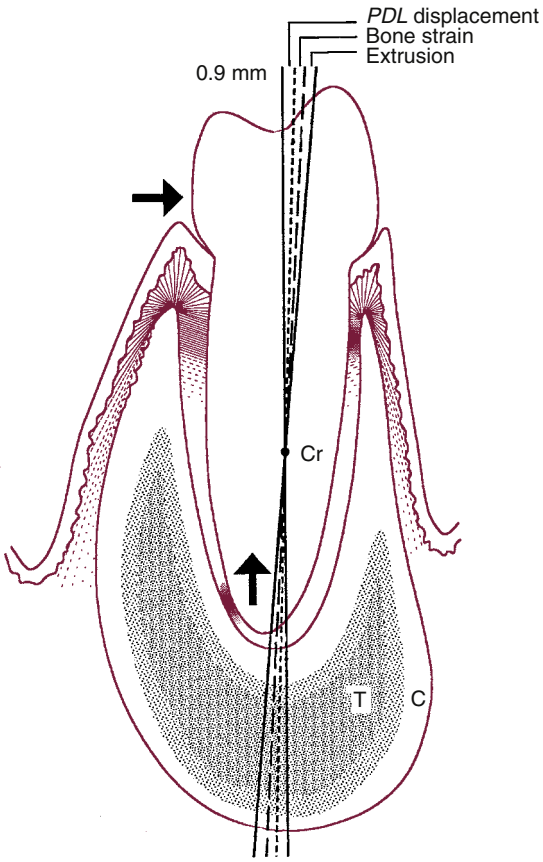


FIGURE 4-50 Initial displacement (1 to 3 days) of a tooth exposed to a tipping (horizontal) load usually is about 0.5 mm but may be as much as 0.9 mm for teeth that are slightly mobile, periodontally compromised, or heavily loaded. The three components are (1) displacement of the root in the periodontal ligament (*PDL*), (2) bone strain caused by bending and creep, and (3) extrusion caused by the inclined plane effect of the tooth root pressing against a tapered alveolus. (From Roberts WE, Garotto LP, Katona TR: Principle of orthodontic biomechanics: metabolic and mechanical control mechanisms. In Carlson DS, Goldstein SA, editors: Bone biodynamics in orthodontic and orthopedic treatment, Ann Arbor, 1992, University of Michigan Press.)

FIGURE 4-51 In 1980, Dr. Kaare Reitan is shown at the University of Oslo at the time when he shared some of his human histology slides with Dr. Roberts. (From Roberts WE, Roberts JA, Epker BN, et al. Remodeling of mineralized tissues, part I: the Frost legacy. *Semin Orthod.* 2006;12(4):216–237.)

FIGURE 4-52 Demineralized histologic section of human periodontium reveals the modeling and remodeling mechanisms of progressive tooth movement through dense cortical bone (*B*). A tooth (*T*) is moving in the direction of the *large arrow*. The rate of translation is enhanced by frontal resorption in the periodontal ligament communicating with the extensive resorption cavities (*) created by initial remodeling events (cutting cones). By this mechanism, teeth move through dense cortical bone at a rate of about 0.3 mm a month (×25). (From Roberts WE, Garotto LP, Katona TR: Principle of orthodontic biomechanics: metabolic and mechanical control mechanisms. In Carlson DS, Goldstein SA, editors: Bone biodynamics in orthodontic and orthopedic treatment, Ann Arbor, 1992, University of Michigan Press.)

maintain the structural relationship of the alveolar process as the tooth moves.[74]

PERIODONTAL LIGAMENT RESPONSE

Within 1 min after application of a continuous orthodontic load to a rat maxillary first molar, a more negative electrical potential is noted where the PDL is widened and a more positive signal where it is compressed (see Fig. 4-55). These bioelectric changes are not piezoelectric signals but are probably streaming potentials.[23] Changes in electrical potential may drive the PDL osteogenic and osteoclastic responses or may be merely physical manifestations of the intense cellular activities (ion flux across cell membranes) triggered by orthodontic stimuli.[18] Coordination of cellular reactions in the PDL to changes in electrical potential is a prime area for future investigation.

Application of orthodontic force initiates a cascade of cellular proliferation and differentiation events in the PDL. Maximal compression of the PDL occurs in 1 to 3 hours (see Fig. 4-56), a time frame consistent with most clinical

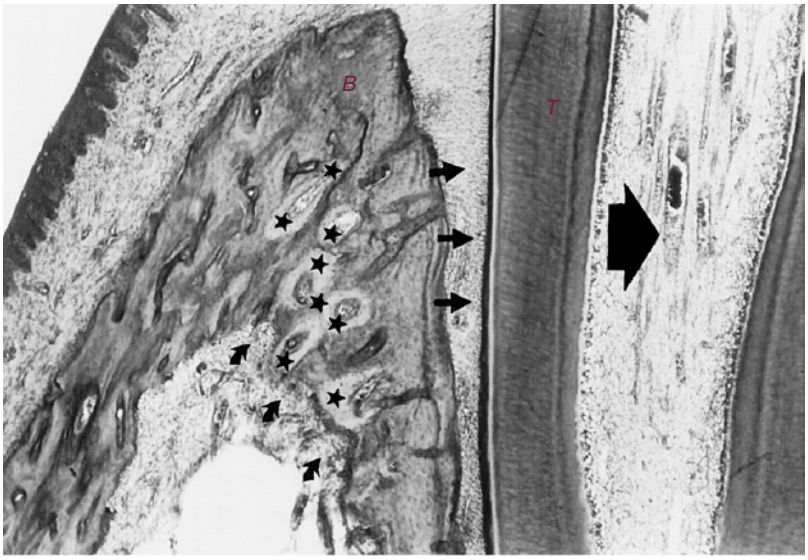


FIGURE 4-53 A demineralized histologic section of human periodontium documents the bone modeling and remodeling aspects of alveolar bone (*B*) drift in the direction of tooth (*T*) movement (*large black arrow*). Three major aspects of bone modeling are noted: (1) new bone formation in the periodontal ligament (*small horizontal arrows*), (2) periosteal resorption on the lingual alveolar bone surface (*left*), and (3) endosteal resorption (in the direction of the curved arrows) to maintain the cortical thickness of the alveolar crest as it drifts to the right. Bone remodeling replaces relatively immature bone with new secondary osteons (*stars*) (×25). (From Roberts WE, Garotto LP, Katona TR: Principle of orthodontic biomechanics: metabolic and mechanical control mechanisms. In Carlson DS, Goldstein SA, editors: Bone biodynamics in orthodontic and orthopedic treatment, Ann Arbor, 1992, University of Michigan Press.)

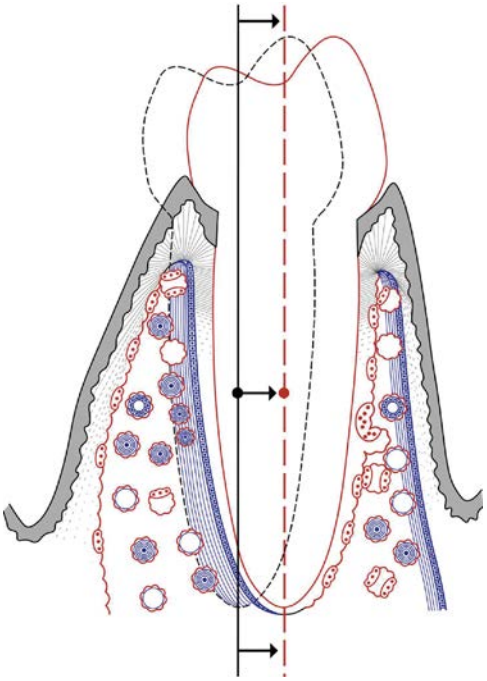


FIGURE 4-54 A schematic diagram shows the bone physiology associated with translation of a tooth. Note that there is a coordinated bone modeling and remodeling response leading and trailing the moving tooth. This mechanism allows a tooth to move relative to basilar bone while maintaining a normal functional relationship with its periodontium. Osteoclastic and osteoblastic activities are shown in *red* and *blue*, respectively. (From Roberts WE, Roberts JA, Epker BN, et al. Remodeling of mineralized tissues, part I: the Frost legacy. *Semin Orthod*. 2006;12(4):216–237.)

estimates for initial displacement after application of an orthodontic load (see Fig. 4-49). The postactivation sequence of about 6 hours for the initiation of tooth movement has been confirmed with a nitric oxide synthetase assessment in rat PDL.[143]

A variety of nervous, immune, and endocrine system responses, as well as local cytokines and intracellular messages, have been implicated in the osseous adaptive reaction as the root of the tooth is displaced.[144] These localized agents probably are mediators of the often painful transient inflammatory events noted during the initiation of the orthodontic reaction. The role of local cytokines in the sustained bone modeling and remodeling events of tooth movement are yet to be clearly outlined. However, prostaglandins are thought to be important factors in the control of mechanically mediated bone adaptation.[111,135,145,146]

Comparing control rat PDL (Fig. 4-57, *A*) with orthodontically stimulated PDL (Fig. 4-57, *B*) helps define the biological mechanisms for mechanically induced bone formation and resorption. The specificity of bone resorbing and forming sites is dictated by the physics of the applied force system.[141] Although the PDL histologic changes and cell kinetics associated with orthodontically induced osteoclasis and osteogenesis are well defined,[16,21,129,130,133,136,147-150] the cellular mechanisms controlling tooth movement are less clear. An interaction of metabolic, biomechanical, bioelectric, and biochemical factors has been implicated.[18,21,133,135,151] The challenge is to determine the sequence of cybernetic events.[133]

Minimal proliferative activity is noted in areas of the PDL where bone resorption predominates. An intense proliferative response is noted in the PDL where new bone is destined to form.[15,16,149,150,152] For the first 12 hours after initiation of

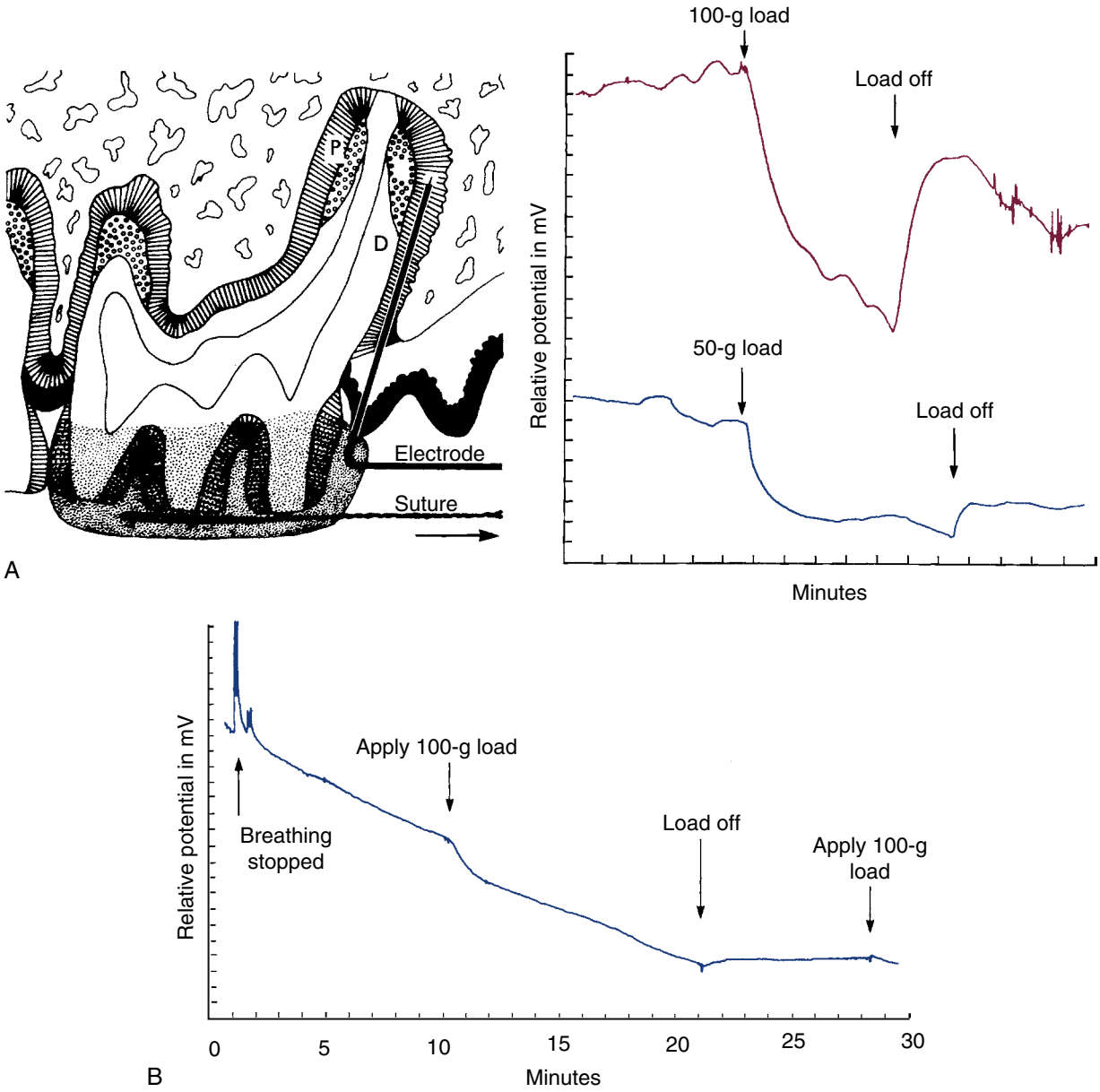


FIGURE 4-55 A, A tungsten microelectrode is inserted on the mesial aspect of a rat maxillary first molar and stabilized to the tooth with composite resin. An orthodontic load of 50 to 100 g (about 0.5-1 N) applied with a traction suture results in a more negative electrical potential in the periodontal ligament, where new bone formation occurs. B, Similar measurements of electrical potential were performed in orthodontically stimulated periodontal ligament of a rat terminated with an overdose of anesthetic. The electrical response was lost 20 min after breathing stopped. (Top left image and top right image, From Roberts WE, et al. Change in electrical potential within periodontal ligament of a tooth subjected to osteogenic loading. In: Dixon A, Sarnat B, eds. *Factors and Mechanisms Influencing Bone Growth*. New York: Allan R Liss; 1982.)

orthodontic stimulus, a modest, nonspecific increase of cells into mitotic and DNA S-phases occurs throughout the PDL (Fig. 4-58, *A*). After 16 hours, a specific osteogenic response is noted in the area of the PDL where new bone formation will begin (Fig. 4-58, *B*). More than half of the fibroblast-like PDL cells in the widened area of the PDL enter the DNA S-phase of the cell cycle[153] and subsequently divide.[133,154] Preosteoblasts then migrate from throughout the PDL to the alveolar bone surface (Fig. 4-59),[13] form osteoblasts, and initiate new bone formation in about 40 to 48 hours.[15,133]

The orthodontic response in the PDL is an effective experimental model for determining the physiologic mechanisms of mechanically induced bone modeling. Orthodontic stimulation of rat maxillary molars has been particularly useful for initiating a defined osteogenic reaction within the PDL on the mesial aspect of the mesial root (Fig. 4-59, *B*). Using the finite element modeling method, one can calculate stresses in the tissue down to the cellular level. Tissue complexity and the diverse range of mechanical properties make orthodontically stressed periodontium a relatively

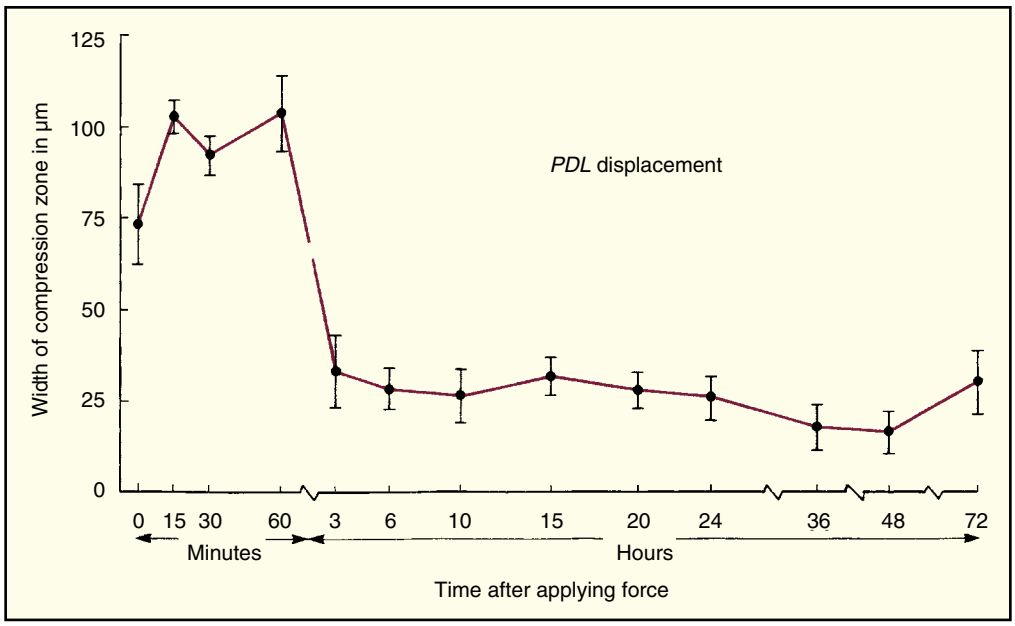


FIGURE 4-56 Initial displacement of the mesial root of rat maxillary first molars in the periodontal ligament (*PDL*) space is assessed by measuring the width of the maximal compression zone. This method is an index of periodontal ligament displacement that is relatively independent of bone deformation. Note the resistance to compression during the first hour, followed by maximal compression at 3 h, creating necrotic areas where undermining resorption will occur.

challenging application of the finite element modeling method. The model simulates the experimental tipping of a rat maxillary first molar (Fig. 4-60, *A*). Osteogenesis was concluded to occur where maximal principal stress was elevated in the PDL (Fig. 4-60, *B*). Bone resorption began where peaks in minimal principal stress, maximal shear, and strain energy density developed.[19]

OSTEOBLAST HISTOGENESIS AND BONE FORMATION

Osteoblasts are derived from paravascular connective tissue cells (Fig. 4-61). The less differentiated precursor and committed osteoprogenitor cells are associated closely with blood vessels. Their progeny (preosteoblasts) migrate away from the blood vessels. The major rate-limiting step in the histogenesis sequence occurs as the cells move through an area of low cell density about 30 µm from the nearest blood vessel.[109] The osteoblast histogenesis sequence (Fig. 4-62) was determined by in situ morphologic assessment of three distinct events in cell physiology: (1) DNA S phase, (2) mitosis, and (3) the increase in nuclear volume (A′→C shift) that accomplishes differentiation to a preosteoblast.[140]

Careful cell-kinetic analysis of orthodontically induced osteogenesis[17] demonstrated that the initial, mechanically mediated step in osteoblast histogenesis is differentiation of preosteoblasts from less differentiated precursor cells (Fig. 4-63). Subsequent studies further classified the less differentiated precursor cells into committed osteoprogenitor (A′) cells and self-perpetuating precursor (A) cells.[154] The morphologic marker for this key step in osteoblast differentiation (change in genomic expression) is an increase in nuclear volume—preosteoblasts have larger nuclei than their precursors. Orthodontically induced osteogenesis is associated with elevated stress in the PDL. The reader is referred to the 5th edition of the text-book for additional details.

OSTEOCLAST RECRUITMENT AND BONE RESORPTION

Bone resorption is the limiting factor that determines the rate of tooth movement.[18,133] The removal of osseous tissue during progressive tooth movement is related directly to (1) bone porosity, (2) the remodeling rate, (3) the resorption rate, and (4) osteoclast recruitment. Porous cortical and trabecular bone allows improved access for osteoclasts. The remodeling rate is related directly to resorption cavities and the number of osteoclasts present at any time in resisting bone. The osteoclast resorption rate largely is controlled by metabolic factors, particularly PTH.[155,156] No direct evidence exists to suggest that osteoclasts are produced in the PDL or at any other bone surface. Preosteoclasts derived from the marrow enter the PDL and adjacent bone through the blood circulation.[18,133,157]

Roberts and Ferguson[18] compared the cell kinetics of metabolic and mechanical induction of PDL resorption. As shown in Figure 4-64, the number of osteoclasts per millimeter of bone surface is maximal about 9 hours after a single injection of parathyroid extract. Mechanical stimulation produces a slow but more sustained response that requires almost 50 hours to reach the same osteoclast density. Preosteoclasts are derived from the marrow by circulating.

Because osteoclasts originate in the marrow, production of osteoclast precursor cells is under systemic (metabolic) and local (hematopoietic) control. The reservoir of circulating osteoclast precursors is controlled systemically. However, the localization

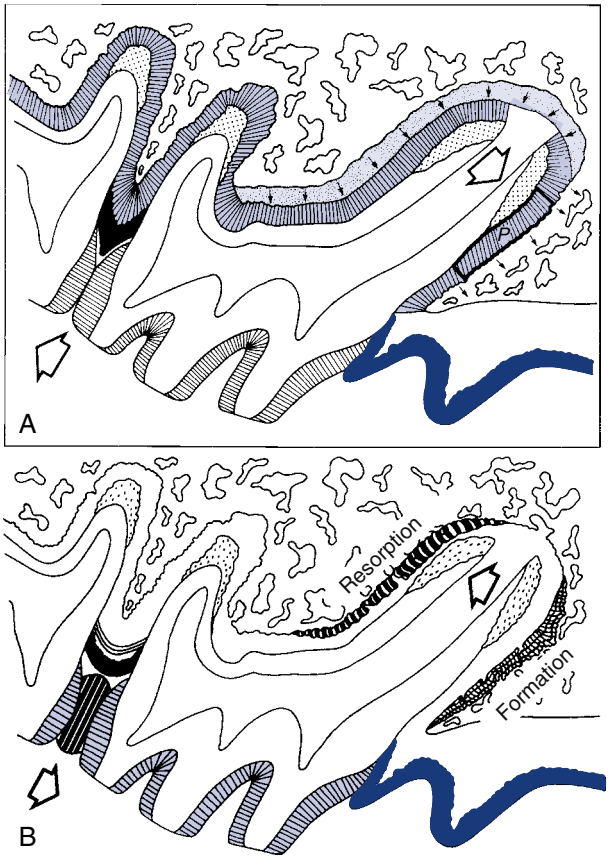


FIGURE 4-57 A, Physiologic drift for the mesial root of a rat maxillary first molar involves a relative tipping of the tooth in the direction of the large arrows. Net resorption occurs adjacent to the area of the periodontal ligament (*P*) marked with the *bold outline*. Bone formation is noted along the periodontal ligament adjacent to the stippled area. B, Inserting a Latex elastic between the first and second molars reverses the direction of tooth movement, resulting in resorption on a previously bone-forming surface (*above*) and vice versa (*below*). (A, From Roberts WE, Morey ER. Proliferation and differentiation sequence of osteoblast histogenesis under physiological conditions in rat periodontal ligament. *Am J Anat.* 1985;174:105; B, from Roberts WE. Advanced techniques for quantitative bone cell kinetics and cell population dynamics. In: Jaworski KFG, ed. *Proceedings of the First Workshop on Bone Morphometry.* Ottawa: University of Ottawa Press; 1976.)

of resorptive sites in the PDL is regulated mechanically. Metabolic stimuli such as PTH produce a relatively nonspecific resorption response along previously resorbing surfaces.[158] Mechanical induction is a specific response that occurs only in the direction of tooth movement. The current challenge is to understand the mechanical and biological components of the resorptive mechanism.

Because osteoblasts and their precursors have a more complete complement of bone-related receptors (i.e., PTH, growth hormone, and estrogen), they may play a role in controlling osteoclasts.[18,153,159] The intricate bone modeling and remodeling responses that characterize orthodontic tooth movement require close coordination of osteoblastic and osteoclastic function.[160]

Intermittent versus Continuous Mechanics

The rate of tooth movement is determined by initiating and sustaining a coordinated bone-resorptive response.[133] The kinetics of continuous mechanics are well known; the response to intermittent loads, such as headgear and removable appliances, is less clear. In general, tooth movement is related directly to the number of hours each day that force is applied. However, even when motivation and cooperation are optimal, the effectiveness of similar therapy among patients is inconsistent. This is a physiologic indication that additional variables are involved.

Reitan[129] reports that a short-duration force (12 to 14 hours per day for 15 to 27 days) delivered by a functional appliance triggers a resorptive reaction that lasts a week or more. Apparently this experience with functional appliances does not apply to cyclic intermittent mechanics such the wearing of nighttime headgear. Otherwise, headgear worn at night would be as effective as 24-hour wear. In the absence of specific experiments, interpolation from continuous-force studies appears to be the best explanation available. A 3-hour period of continuous loading is necessary to achieve maximal displacement of a tooth root in the periodontium (see Fig. 4-54). If one assumes that at least 3 hours are needed for PDL activation or recovery,[18] continuous wearing of the headgear for 12 hours per day is expected to be more effective than a longer period of wearing the headgear with frequent release of force. The recommendation is that the headgear is not removed during the day. Removing the device for meals or sports compromises the biomechanical activation of the periodontium. The complex biological response associated with irregular force application probably is the main factor in the unpredictable response to headgear or removable appliances.

Another possible variable in the response to intermittent force is the circadian rhythm of PDL proliferation and differentiation (Figs. 4-65 and 4-66). Maximal cell proliferation in the PDL occurs during the resting hours :[161,162] daytime for rats and nighttime for human beings. Differentiation to preosteoblasts, the key rate-limiting step in osteoblast differentiation, occurs during the late resting and early arousal periods.[154] These data suggest that wearing headgear or a removable appliance at night is more effective than wearing the same appliance for an equal period during the day.[162]

Differential Anchorage

The density of the alveolar bone and the cross-sectional area of the roots in the plane perpendicular to the direction of tooth movement are the primary considerations for assessing anchorage potential. The volume of osseous tissue that must be resorbed for a tooth to move a given distance is its anchorage value. If all bone offered the same resistance to tooth movement, the anchorage potential of maxillary and mandibular molars would be about the same. Clinical experience shows that maxillary molars usually have less anchorage value than mandibular molars in the same patient. A common example is space closure in a Class I four premolar extraction case; it often is necessary to use headgear on the maxillary first molars to maintain the Class I relationship. The relative resistance of mandibular molars to mesial movement is a well-known principle of differential mechanics.

Why are mandibular molars usually more difficult to move mesially than maxillary molars? At least two physiologic factors

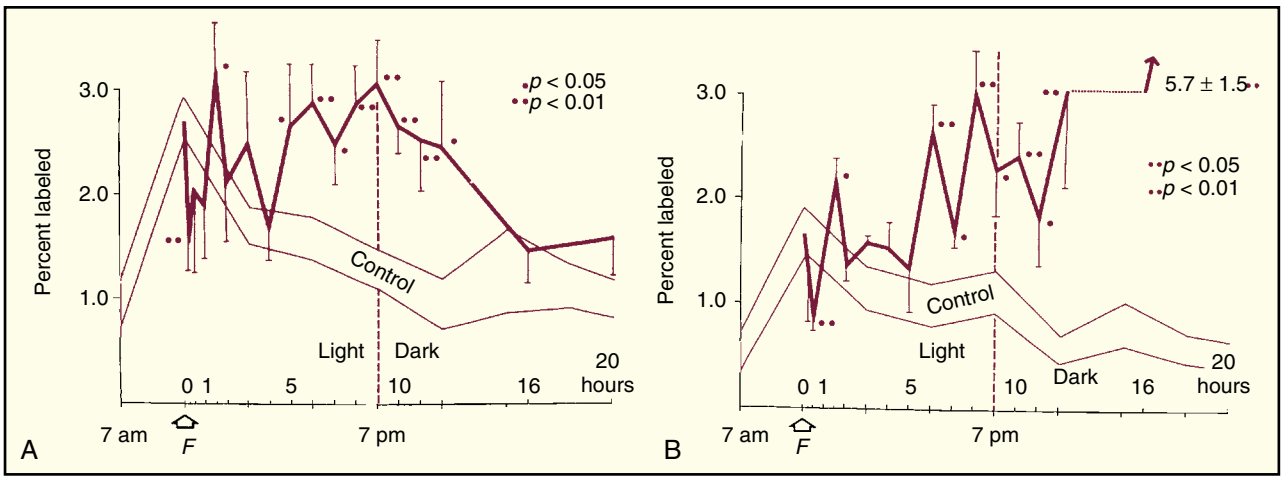


FIGURE 4-58 A, Compared with the normal circadian range of DNA S phase cells, orthodontic stimulus (*F*) results in a modest, nonspecific increase in S phase cells throughout the periodontal ligament. This is a mechanically induced release of G_1-blocked cells. These data are from the alveolar crest area, where no new bone formation occurs. B, In the midroot area, where new bone formation is induced, a similar nonspecific response is followed by a large proliferative reaction that specifically supports orthodontically induced osteogenesis. (From Smith RK, Roberts WE. Cell kinetics of the initial response to orthodontically induced osteogenesis in rat molar periodontal ligament. *Calcif Tissue Int.* 1980;30:51.)

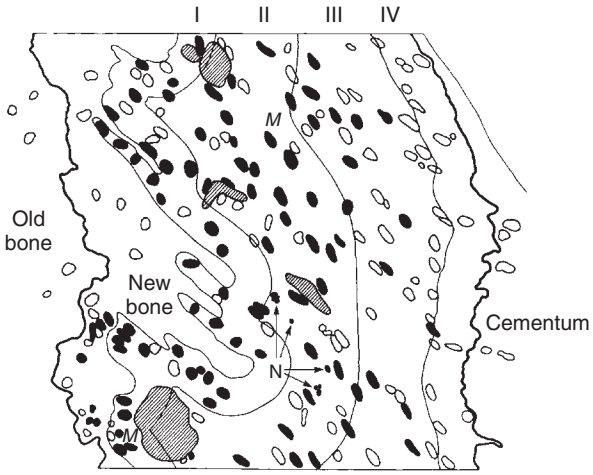


FIGURE 4-59 During the first 70 hours of orthodontically induced osteogenesis, periodontal ligament osteogenic cells migrate toward the bone surface from throughout the ligament. Cell migration patterns are tracked by repeated injection of the DNA precursor ^{3}H-thymidine. Labeled cells have black nuclei. (From Roberts WE, Chase DC. Kinetics of cell proliferation and migration associated with orthodontically induced osteogenesis. *J Dent Res.* 1981;60(2):174.)

can be considered: (1) the thin cortices and trabecular bone of the maxilla (see Figs. 4-2, 4-3, and 4-5) offer less resistance to resorption than the thick cortices and coarser trabeculae of the mandible (see Figs. 4-4 and 4-5), and (2) the leading root of mandibular molars being translated mesially forms bone that is far denser than the bone formed by translating maxillary molars mesially (Fig. 4-67).[144] The reason mandibular molars form denser bone than maxillary molars is unclear; it may be that new bone formed in the maxilla is remodeled more rapidly. In general, bones composed primarily of trabeculae remodel more rapidly than those composed primarily of cortical bone.[50,162]

Why is the alveolar process that supports the mandibular molars denser than maxillary molars? Functional loading dictates the osseous anatomy for the opposing jaws: the maxilla is predominantly trabecular bone with thin cortices, similar to a vertebral body or an epiphysis (see Figs. 4-2, 4-4, and 4-5); the mandible has thick cortices, similar to the diaphysis of a major long bone (see Figs. 4-4 and 4-5). Although the forces of occlusion are distributed equally to the maxilla and mandible, the maxilla transfers a major fraction of functional loads to the rest of the cranium.

The loads (compression, tension, and torsion) to which the maxilla and the mandible are exposed are different. The mandible is subjected to substantial torsion and flexure caused by muscle pull and masticatory function.[5,163] Thick mandibular cortices are needed to resist the torsional and bending strain (see Figs. 4-5 and 4-6). The maxilla, however, is loaded predominantly in compression, has no major muscle attachments, and transfers much of its load to the rest of the cranium. Because of its entirely different functional role, the maxilla is predominantly trabecular bone with thin cortices (see Figs. 4-2 to 4-4). This anatomic configuration is similar to that of other bones loaded primarily in compression (e.g., proximal tibia and vertebral bodies of the spine).

Rate of Tooth Movement

A histomorphometric evaluation of alveolar bone turnover between the maxilla and the mandible during experimental

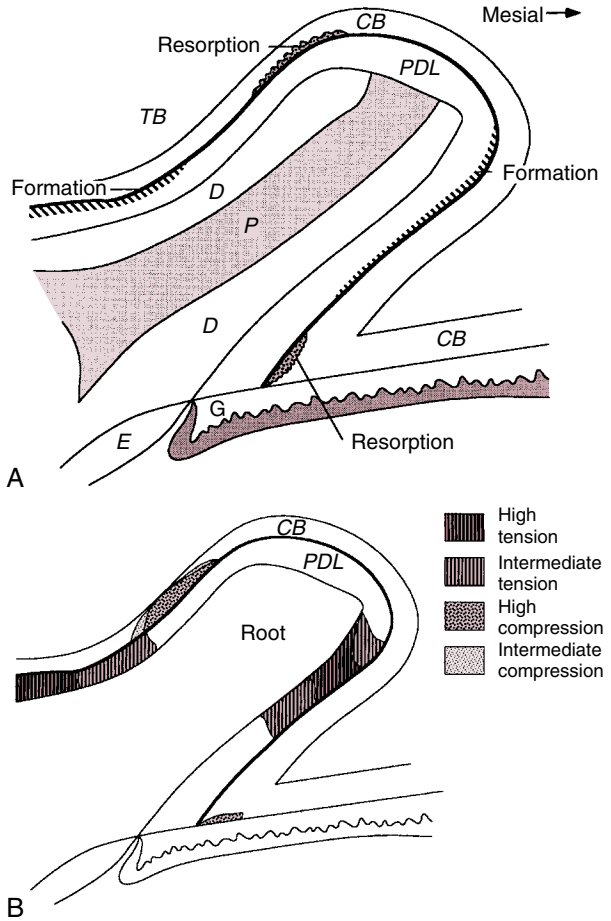


FIGURE 4-60 A, The mesial tipping of the rat maxillary first molar results in a typical pattern of bone resorption and formation that produces tooth movement. *G*, Gingiva; *E*, enamel; *D*, dentin; *TB*, trabecular bone; *CB*, cortical bone; *P*, pulp; *PDL*, periodontal ligament. B, Finite element model stress analysis of orthodontic tipping indicates that areas of elevated maximal principal stress (tension) in the periodontal ligament and minimal principal stress (compression) in the cortical bone of the lamina dura are associated with areas of bone formation and resorption, respectively. (Redrawn from Roberts WE, Garotto LP, Katona TR. Principle of orthodontic biomechanics: metabolic and mechanical control mechanisms. In: Carlson DS, Goldstein SA, eds. *Bone Biodynamics in Orthodontic and Orthopedic Treatment*. Ann Arbor: University of Michigan Press; 1992.)

tooth movement in dogs has defined the differential response of the jaws to tooth movement.[164] A similar miniscrew anchored force system of 200 to 250 *g* was applied to premolars in the maxillary and mandibular arches for 4 or 12 weeks. The tooth movement response was monitored with radiographs and fluorescent bone labels. Significantly more orthodontic tooth movement was observed for maxillary than for mandibular teeth. The primary histomorphometric analysis indicated that, after 4 weeks of tooth movement, a marginal increase in resorptive parameters. On the other hand, after 12 weeks of tooth movement, secondary histomorphometric analysis indicated an increase in the bone formation rate, resulting in increased woven bone formation, especially at the tension sites. These results indicate that tooth movement is a regional acceleratory phenomenon (RAP), manifested as increased bone turnover in the alveolar process as the teeth move through it. The RAP of resisting bone can be enhanced by corticotomies.[165]

From a clinical perspective, maxillary bone is more responsive to orthodontics because it is primarily composed of trabecular bone.[140] The rate of tooth movement is the inverse of anchorage potential; the same physiologic principles apply. Clinical studies using endosseous implants for anchorage[118] have provided excellent opportunities to assess the rate of tooth movement through dense cortical bone in the posterior mandible (see Fig. 4-73, *A*) compared with the less dense trabecular bone of the posterior maxilla (see Fig. 4-73, *B*). The enhanced anchorage value of mandibular molars is related to the high-density bone formed as the leading roots are moved mesially. After a few months of mesial translation, the trailing roots engage the high-density bone formed by the leading root and the rate of tooth movement declines (Fig. 4-68).

Overall, the maximal rate of translation of the midroot area through dense cortical bone is about 0.5 mm per month for the first few months; the rate then declines to less than 0.3 mm per month until the first molar extraction site is closed (see Fig. 4-68). A composite analysis of four similar cases of molar translation in adults showed that teeth moved out at a rate of about 0.6 mm per month for about 8 months; the rate decreased to 0.33 mm per month as the trailing (distal) root engaged the dense bone formed by the leading (mesial) root (Fig. 4-69).

When teeth are moved continuously in the same direction, the remodeling rate increases in compact bone immediately ahead of the moving tooth (see Figs. 4-52 and 4-53). This enhanced remodeling process probably is related to the regional acceleratory phenomenon commonly noted in osseous wound healing. Cutting/filling cones are the means of osteoclast access to the inner portion of dense compacta (see Fig. 4-24). This remodeling mechanism appears to be particularly important for resorbing the dense cortical bone formed by the leading root during mesial movement of lower molars (see Fig. 4-67). Note the radiolucent areas in the dense compact bone.

Figure 4-70 is a summary of the relative rates of molar translation in the upper and lower jaws of growing children and adults. A maximal rate approaching 2 mm per month is possible with space-closure mechanics or 24-h-per-day headgear wear by a rapidly growing child (Child Mx). Similar mechanics in a nongrowing adult can translate upper molars about 1 mm per month (Adult Mx). Mesial translation of lower molars in a child occurs at a rate of about 0.7 mm per month (Child Md). The slowest molar translation (0.3 mm per month) is in the lower arch of adults (Adult Md). Overall, the same teeth in growing children move about twice as fast as they move in adults. Certainly histologic factors, such as less dense alveolar bone and more cellular PDL,[30] are relevant factors; growth-related extrusion is the principal reason that space closure is almost twice as rapid in children. Figure 4-71 shows the considerably smaller volume of bone that is resorbed during space closure in a child compared with that in an adult. In general, the rate of tooth movement is inversely related to bone density and the volume of bone resorbed.

Rates for orthodontic tipping movements usually are higher but are more variable than for translation. No well-controlled studies of tipping in various intraoral sites of children and adults are available; however, some interesting theoretical considerations have been offered. When teeth are moved rapidly, immature new bone can form at a rate of 100 μm/day or more

(more than 3 mm per month). This rate of tooth movement probably is never achieved during routine treatment. Premolar and canine tipping of about 2 mm per month may be achieved with removable appliances in the maxillary arch of growing children.

Three principal variables determine the rate of tooth movement: (1) growth, (2) bone density, and (3) type of tooth movement. Alveolar bone of the maxilla is less dense than that of the mandible because it has a higher ratio of cancellous bone to cortical bone. Bone in children generally is less dense (more porous or cancellous) than bone in adults. Cancellous or trabecular bone has more surface area available for resorption, which is important because bone that impedes tooth movement can be resorbed from all sides. Cortical bone is restricted largely to frontal and undermining resorption mechanisms in the PDL. In general, children have a higher rate of bone remodeling than adults. In simple terms, more osteoclasts are present in the bone that can help with the task of removing the osseous tissue impeding tooth movement.

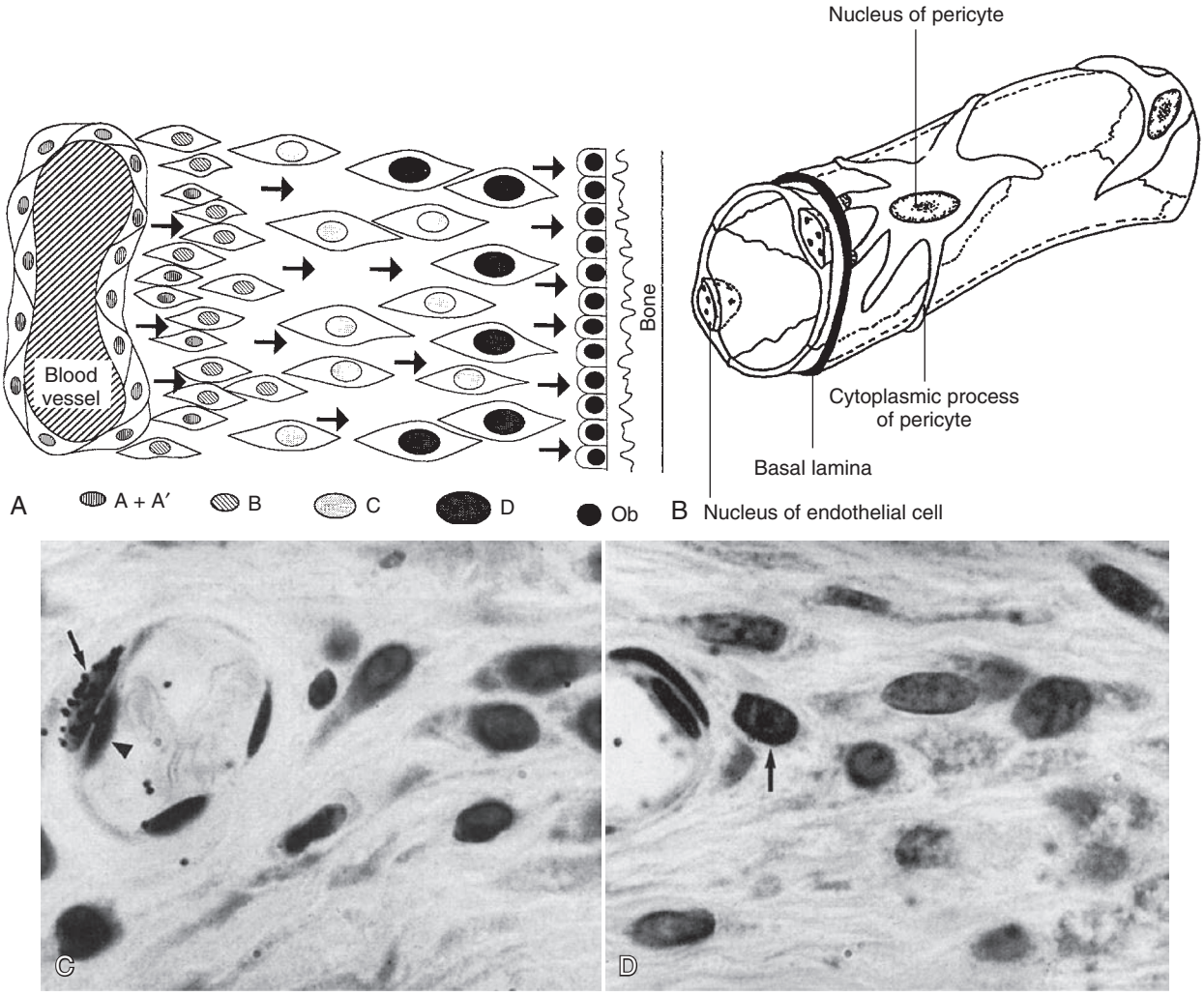


FIGURE 4-61 **A,** Less differentiated precursor cells (A cells) are a paravascular population. They differentiate to committed osteoprogenitor cells (A′ cells), which migrate away from the blood vessel. Traversing the low-cell density zone, the A′ cells differentiate to G_1-stage preosteoblasts (C cells), which synthesize DNA and become G_2-stage preosteoblasts (D cells). **B,** A pericyte, a distinct polymorphic cell of mesenchymal origin, partly encircles a microvessel and shares a common basal lamina with an endothelial cell. **C,** Photomicrograph of autoradiography of an expanded interpremaxillary suture, showing labeled pericyte (*arrow*) and unlabeled endothelial cell (*arrowhead*). (Stained with hematoxylin and eosin; original magnification ×1000 under oil immersion.) **D,** Photomicrograph of autoradiography of an expanded interpremaxillary suture, showing an activated pericyte (*arrow*) that has partly separated from the basal lamina of the blood vessel. The nuclear pattern of the activated pericyte shows that it is in the prophase of mitosis. (Stained with hematoxylin and eosin; original magnification ×1000 under oil immersion.) (From Chang H-N, et al. Angiogenesis and osteogenesis in an orthopedically expanded suture. *Am J Orthod Dentofac Orthop*. 1997;111:382.)

The teeth of growing children extrude as they move through bone. One must remember that for the basilar bone of the jaws to separate 1 to 2 cm during 2 years of orthodontic treatment is not uncommon. This means that the teeth of children move as much by differential apposition (guided eruption) as by resorption (see Fig. 4-71). Tipping the teeth requires less resorption of bone adjacent to the middle of the root. This bone, which is farthest from bone surfaces, probably is the most difficult for the osteoclasts to access. Eliminating the most difficult part of the resorptive process probably is the reason teeth move faster by tipping than by translation. Considering all the variables of age, arch, and type of tooth movement, maxillary buccal segments in children move as much as four times faster than posterior mandibular segments in adults (see Fig. 4-70).

To date almost all studies of the rate of tooth movement have used two-dimensional methods of analysis. Tooth movement is a three-dimensional phenomenon, and more advanced methods of analysis are needed.

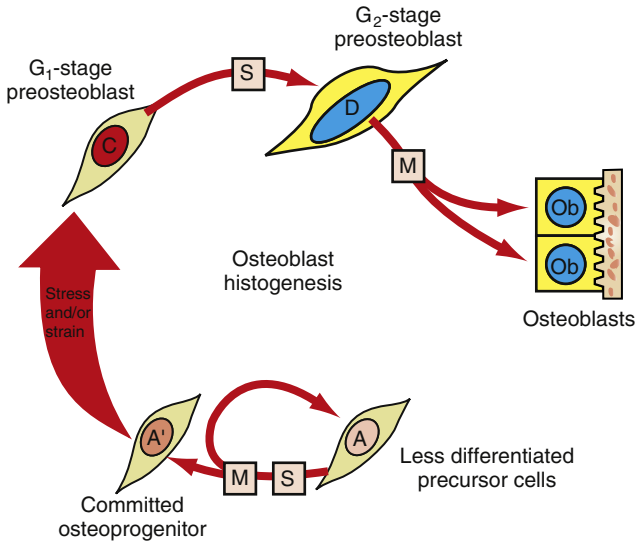


FIGURE 4-62 Frequency distribution of nuclear volume for fibroblast-like cells in unstimulated rat periodontal ligament. A, A′, C, and D cells are a morphologic classification based on peaks in the distribution curve. The osteoblast histogenesis sequence is a progression of five morphologically and kinetically distinguishable cells. The process involves two DNA S phases and two mitotic (M) events. (Redrawn from Roberts WE, Morey ER. Proliferation and differentiation sequence of osteoblast histogenesis under physiological conditions in rat periodontal ligament. *Am J Anat.* 1985;174:105.)

Periodontitis and Orthodontics

Osteoclasts are hardy cells that thrive in a pathologic environment. Osteoblasts, however, are vascularly dependent cells, and their histogenesis is disrupted easily.[18,154] Therefore, most skeletal deficits probably are errors in bone formation rather than resorption. A good example of the fragility of bone formation is suppression of osteoblast differentiation by inflammatory disease processes.[109] Orthodontics often is a useful adjunct for enhancing periodontal health, although moving teeth when progressive periodontal disease is present invites disaster.[109] Tooth movement in the alveolar process stimulates resorption and formation. Osteoclasts thrive in an inflammatory environment because they originate in the marrow, a protective site removed from the localized lesion. Preosteoclasts are attracted to the inflammatory site by cytokine mediators.[135] Vascularly mediated osteoblast histogenesis, is suppressed strongly by inflammatory disease. Therefore, when teeth are moved in the presence of active periodontal disease, resorption is normal or even enhanced and bone formation is inhibited. In a patient who has periodontitis, orthodontics may exacerbate the disease process, resulting in a rapid loss of supporting bone (Fig. 4-72). A thorough, full-mouth periodontal examination, evaluating

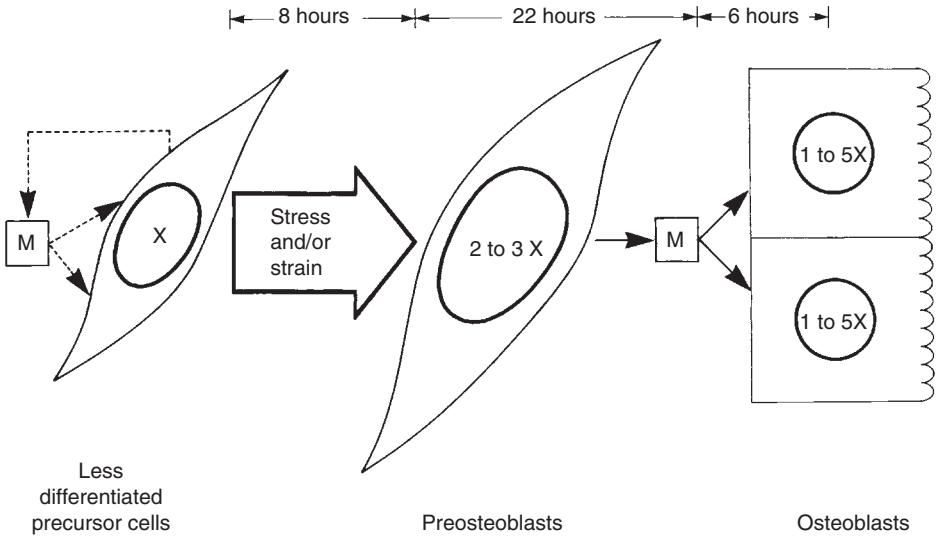


FIGURE 4-63 Differentiation of less differentiated precursor cells to preosteoblasts involves an increase in nuclear volume mediated by stress, strain, or both. The increase in nuclear size is a morphologic manifestation of change in genomic expression (differentiation). (From Roberts WE, Mozsary PG, Klingler E. Nuclear size as a cell kinetic marker for osteoblast differentiation. *Am J Anat.* 1982;165:373.)

for presence of deep periodontal pockets, recession, bleeding on probing, tooth mobility, and furcation involvement should be conducted on adult patients. For patients who demonstrate signs of periodontal disease and inflammation, the disease must be brought under control prior to commencing orthodontic treatment.

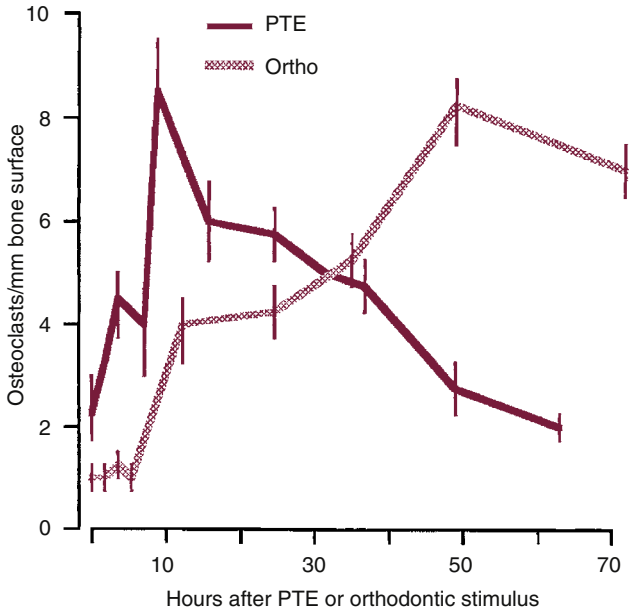


FIGURE 4-64 Metabolic (parathyroid extract [PTE]) versus mechanical (orthodontic) stimulation of periodontal ligament osteoclastic activity. (From Roberts WE, Ferguson DJ. Cell kinetics of the periodontal ligament. In: Norton L, Burstone CB, eds. *The Biology of Tooth Movement*. Boca Raton, FL: CRC Press; 1989.)

Endosseous Implants

A major problem in orthodontics and facial orthopedics is anchorage control.[31] Undesirable movement of the anchorage units is a common problem that limits the therapeutic range of biomechanics.[166] An important application of the basic principles of bone physiology is the use of rigid endosseous implants for orthodontic and orthopedic anchorage. Animal studies[59] and clinical trials of custom orthodontic devices[53] have established that rigidly integrated implants do not move in response to conventional orthodontic and orthopedic forces. These devices are opening new horizons in the management of asymmetry, mutilated dentition, severe malocclusion, and craniofacial deformity.[167]

A preclinical study in dogs tested the anchorage potential of two prosthetic-type titanium implants: (1) a prototype of an endosseous device with a cervical post, asymmetric threads, and an acid-etched surface and (2) a commercially available implant with symmetric threads (Fig. 4-73). Based on label incidence (Fig. 4-74, *A*) and the relative number of new osteons in microradiographs (Fig. 4-74, *B*), the rate of bone remodeling near the implant was higher compared with the basilar mandible only a few millimeters away.[168] Compared with titanium implants with a smooth surface, the degree of remodeling at the interface is greater for threaded implants placed in a tapped bone preparation.[31] This may be related to the increased resistance of threaded implants to torsional loads over time.[169]

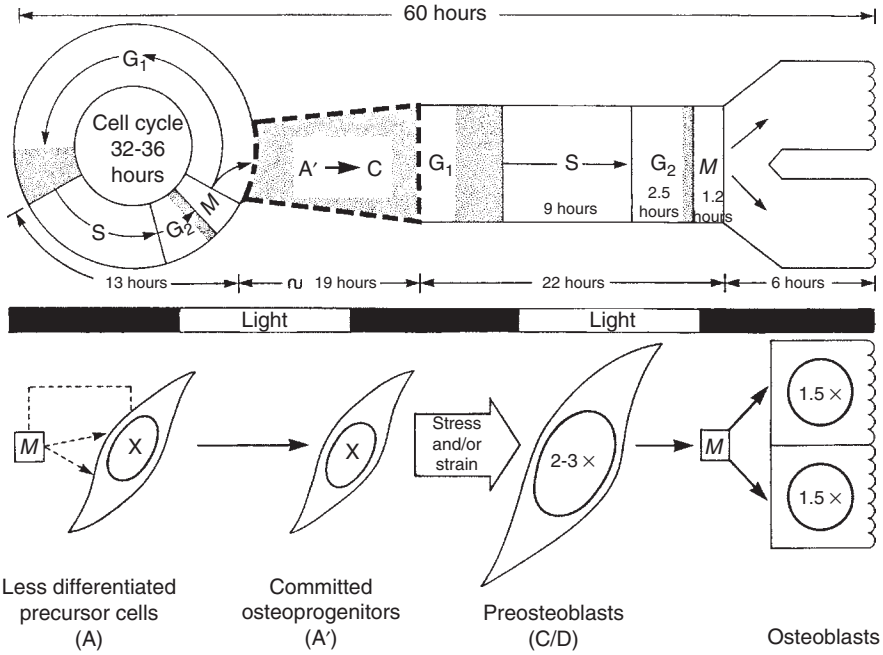


FIGURE 4-65 Photoperiod (circadian rhythm) influence on physiologic osteoblast histogenesis in rat periodontal ligament. Note that the A′→C increase in nuclear size occurs during the late resting and early arousal phases. The overall osteoblast differentiation sequence is 60 hours, five alternating dark/light cycles of 12 hours each. (From Roberts WE, Morey ER. Proliferation and differentiation sequence of osteoblast histogenesis under physiological conditions in rat periodontal ligament. *Am J Anat*. 1985;174:105.)

Direct bone apposition at the endosseous interface results in rigid fixation (osseointegration).[170] From an anchorage perspective, a rigid endosseous implant is the functional equivalent of an ankylosed tooth. Complete bony encapsulation is not necessary for an implant to serve as a rigid anchorage unit. The crucial feature is indefinite maintenance of rigidity despite continuous orthodontic loads. Over time, orthodontically loaded implants achieve a greater fraction of direct osseous interface.[161,169] From an orthodontic and orthopedic perspective, titanium implants can resist substantial continuous loads (1 to 3 N superimposed on function) indefinitely. Histologic analysis with multiple fluorochrome labels and microradiography confirm that rigidly integrated implants do not move relative to adjacent bone (see Fig. 4-74).[59] By definition, maintaining a fixed relationship with supporting bone is true osseous anchorage. Endosseous (osseointegrated) implants are well suited to many demanding orthodontic applications.[53]

Routine use of rigid implants for prosthetic or orthodontic applications requires that fixtures be placed between or near the roots of teeth. Inadvertent impingement on the PDL and the root of an adjacent tooth still may provide an acceptable result (Fig. 4-75). Cementum repair occurs where the root is cut, the PDL reorganizes, and the implant surface is integrated

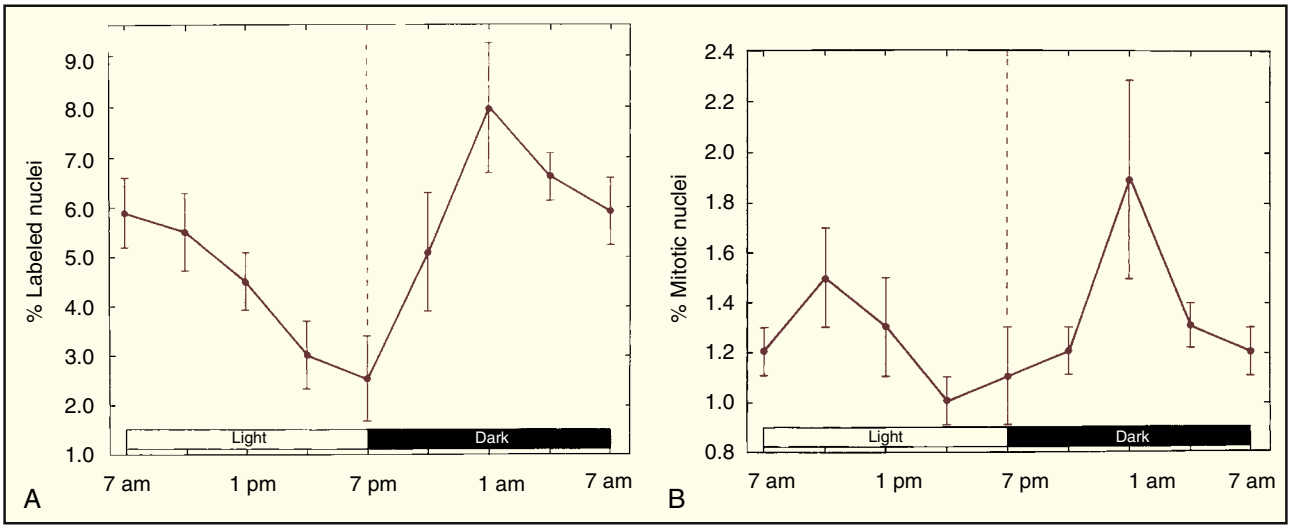


FIGURE 4-66 A, Circadian rhythm (photoperiod) of S phase cells in unstimulated rat periodontal ligament along a natural bone-forming surface. B, Circadian rhythm (photoperiod) of mitotic cells in unstimulated rat periodontal ligament along a natural bone-forming surface. (From Roberts WE, Morey ER. Proliferation and differentiation sequence of osteoblast histogenesis under physiological conditions in rat periodontal ligament. *Am J Anat*. 1985;174:105.)

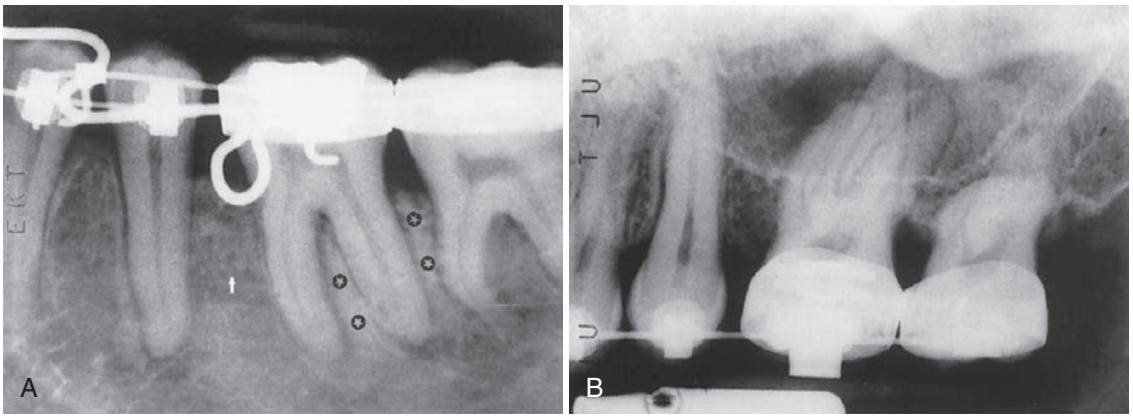


FIGURE 4-67 A, Progressive mesial translation of second and third mandibular molars generates dense cortical bone (*stars*) that is more resistant to resorption than the trabecular bone (*t*) ahead of the first molar. B, Mesial movement of the second and third molars in the maxilla of the same patient fails to demonstrate dense cortical bone distal to the moving roots. (A, From Roberts WE, Garotto LP, Katona TR. Principle of orthodontic biomechanics: metabolic and mechanical control mechanisms. In: Carlson DS, Goldstein SA, eds. *Bone Biodynamics in Orthodontic and Orthopedic Treatment*. Ann Arbor: University of Michigan Press; 1992.)

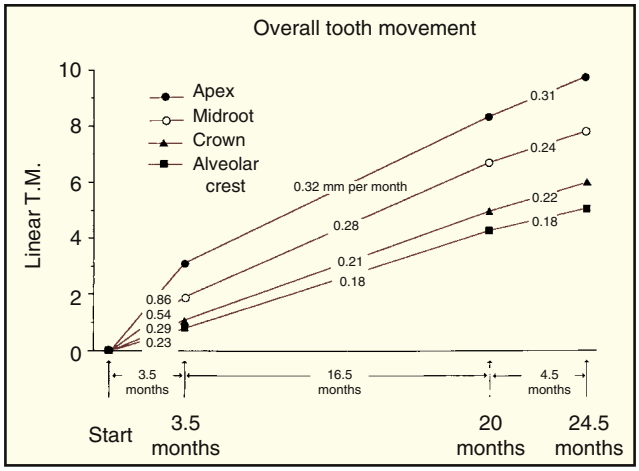


FIGURE 4-68 Overall tooth movement curve for lower second and third molars demonstrates mesial root movement and translation of 8 to 10 mm in about 2 years. Note that the rapid movement of the first 3.5 months (as much as 0.86 mm a month) slows to about 0.3 mm a month for the duration of space closure.

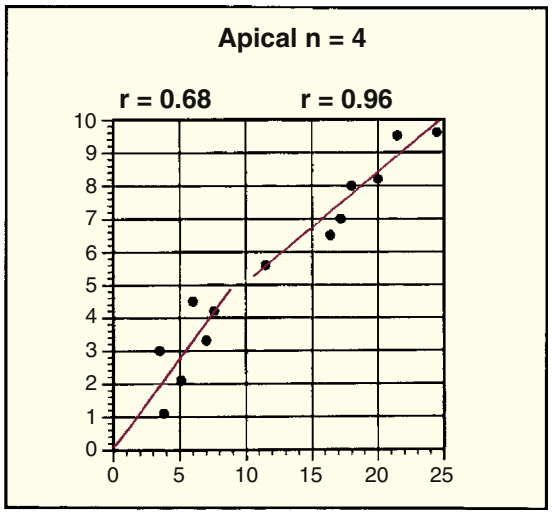


FIGURE 4-69 Separate linear regressions plotted for tooth movement during the first 8 months compared with regression seen after about 12 months in four patients in whom mesial root movement was initiated at the start of treatment. (From Roberts WE, Arbuckle GR, Analoui M. Rate of mesial translation of mandibular molars using implant-anchored mechanics. *Angle Orthod.* 1996;66(5):335.)

rigidly with osseous tissue. No evidence exists of ankylosis of the tooth.[59]

Retromolar Implant Anchorage

The isolated loss of a lower first molar with a retained third molar is a common problem. Rather than extract the third molar and replace the first molar with a three-unit bridge, mesial translation of second and third molars to close the edentulous spaces often is preferable (Fig. 4-76). The first case with long-term follow-up has been published.[53] Because of the increasing incidence of progressive bone loss and fatigue fracture associated with single tooth implants in lower first- and

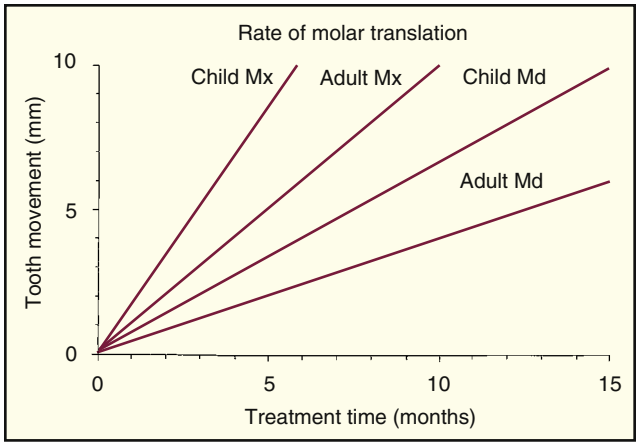


FIGURE 4-70 Relative rates of molar translation in both jaws of rapidly growing children compared with the rates in adults.

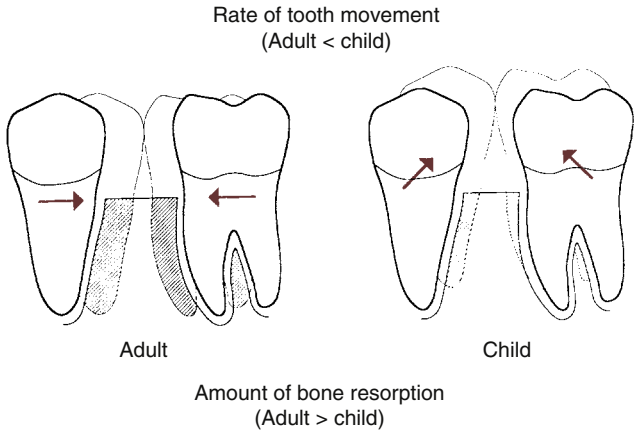


FIGURE 4-71 The rate of molar translation is related directly to the amount and density of bone that must be resorbed. Because extrusion is associated with growth, relatively less bone must be resorbed in translation of children's molars.

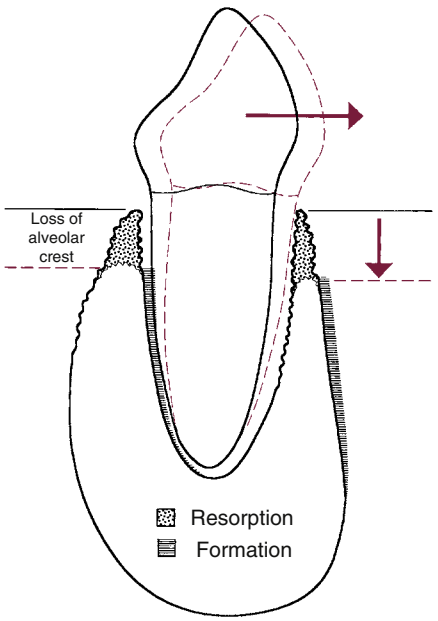


FIGURE 4-72 Because active periodontitis enhances resorption and inhibits apposition, orthodontics in patients with this condition often results in a severe loss of alveolar bone support.

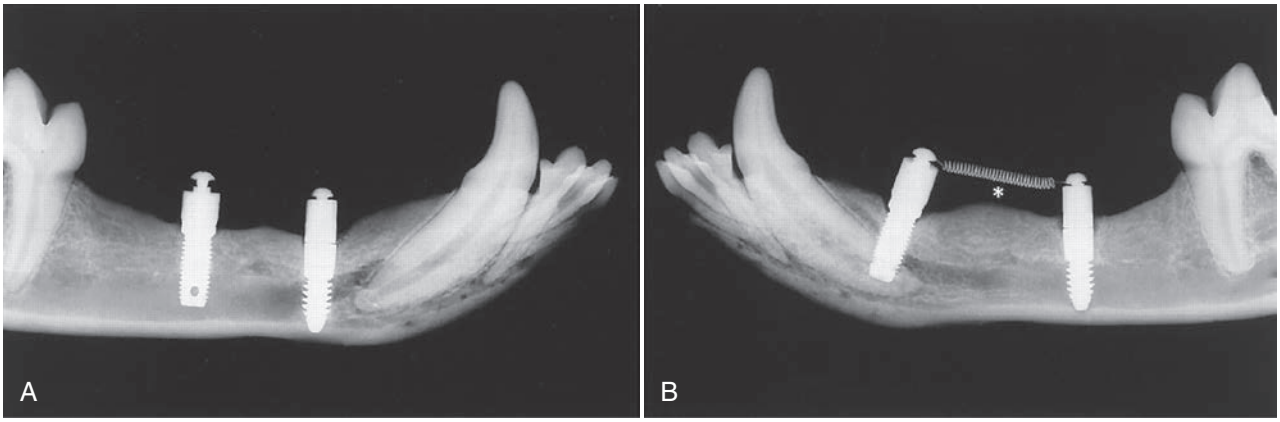


FIGURE 4-73 A, Two titanium implants of different design were placed in the partly edentulous mandibles of young adult dogs. B, After 2 months of unloaded healing, a 3-N compressive load was applied between the implants for 4 months. Increased periosteal apposition (*) was noted between the implants of some dogs. None of the rigidly integrated fixtures was loosened by the continuous load superimposed on function. (From Roberts WE, et al. Rigid endosseous implants for orthodontic and orthopedic anchorage. *Angle Orthod*. 1989;59:247.)

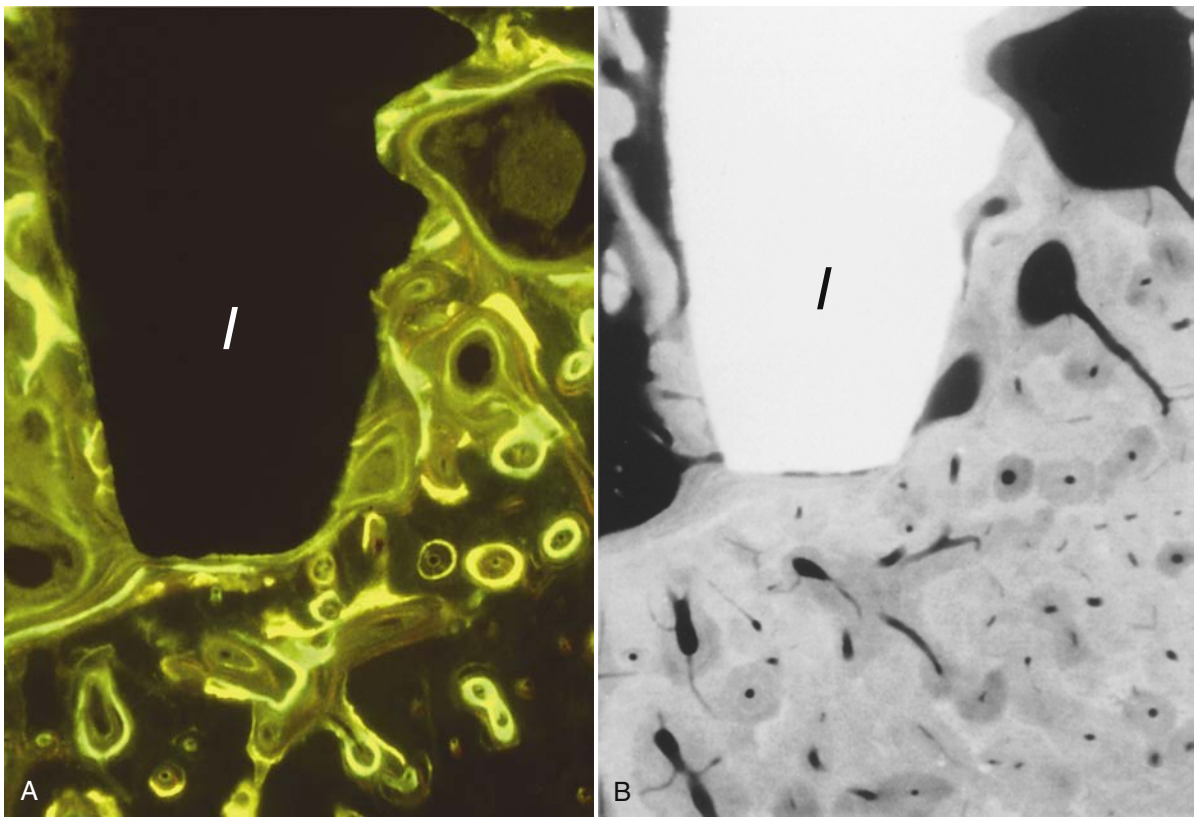


FIGURE 4-74 A, Multiple fluorochrome labels in bone adjacent to an implant (*I*) show a high rate of remodeling at the bone–implant surface. B, Microradiographic image of the same section shows direct bone contact on the surface of the implant. (From Roberts WE, Garotto LP, Katona TR. Principle of orthodontic biomechanics: metabolic and mechanical control mechanisms. In: Carlson DS, Goldstein SA, eds. *Bone Biodynamics in Orthodontic and Orthopedic Treatment*. Ann Arbor: University of Michigan Press; 1992.)

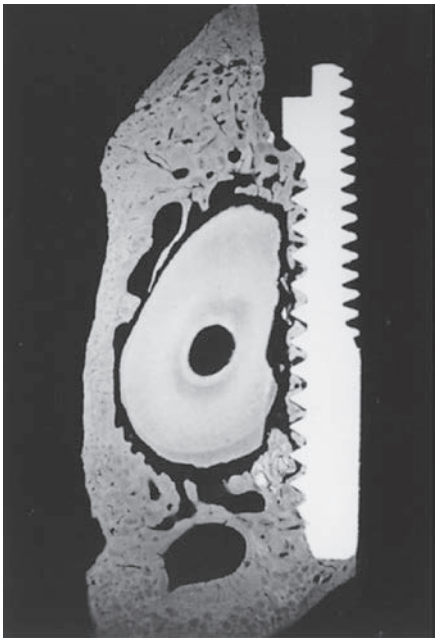


FIGURE 4-75 An endosseous implant inadvertently impinged on the root of a canine. The implant successfully integrated with bone and served as a rigid anchor for orthopedic loading. (From Roberts WE, et al. Rigid endosseous implants for orthodontic and orthopedic anchorage. *Angle Orthod*. 1989;59:247.)

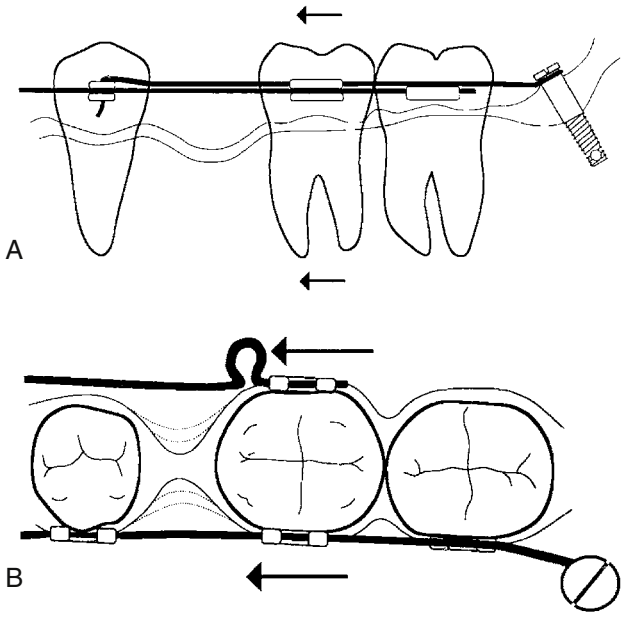


FIGURE 4-76 A, The mechanics of using a retromolar implant with an external abutment as anchorage to stabilize the premolar anterior to an extraction site. B, Using buccal and lingual mechanics to balance the load and shield the periosteum in the extraction site, the atrophic extraction site is closed without periodontal compromise of any of the adjacent teeth. (From Roberts WE, Garotto LP, Katona TR. Principle of orthodontic biomechanics: metabolic and mechanical control mechanisms. In: Carlson DS, Goldstein SA, eds. *Bone Biodynamics in Orthodontic and Orthopedic Treatment*. Ann Arbor: University of Michigan Press; 1992.)

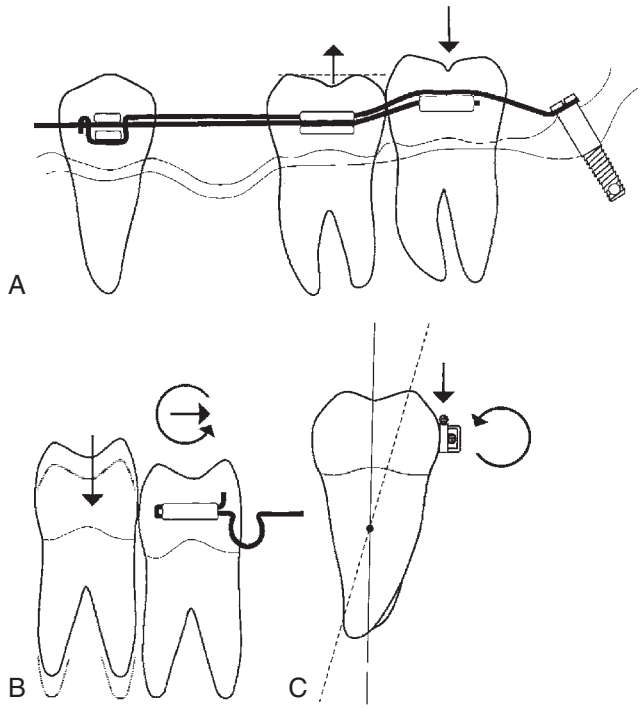


FIGURE 4-77 A, The mechanics of intruding a third molar with implant anchorage before space closure. B, A removable lingual arch prevents extrusion of the second molar. C, Because the intrusive force on the third molar is buccal to the center of resistance, the tooth tends to tip buccally. This problem is controlled by placing lingual crown torque in the rectangular wire inserted in the tube. (From Roberts WE, Garotto LP, Katona TR. Principle of orthodontic biomechanics: metabolic and mechanical control mechanisms. In: Carlson DS, Goldstein SA, eds. *Bone Biodynamics in Orthodontic and Orthopedic Treatment*. Ann Arbor: University of Michigan Press; 1992.)

second-molar areas, the orthodontic option for mesially translating the molars to close the space is increasing in popularity. The ingenious mechanics (Fig. 4-77) used with retromolar implant anchorage served to highlight anchorage requirements of molar protraction, and biomechanical resolution of forces and moments allows for translation of molars. Extraalveolar location of retromolar implants allows for great versatility and anchorage potential, and such locations are now being favored to intraradicular and intraalveolar sites at which miniscrews are placed. Clinical details have been published.[53] Rigid endosseous implants show great promise for considerably extending the therapeutic possibilities of orthodontics and dentofacial orthopedics.

CURRENT STATUS OF MINISCREW IMPLANTS

The utility of skeletal anchorage in orthodontics continues to evolve, with many new designs and emerging clinical applications. The term *temporary anchorage devices* (TAD) has gained popularity in the orthodontic literature[171] and refers to a broad group of devices, which includes, for example, miniscrew implants (MSI), palatal implants, and retromolar implants. MSI are clearly the most popular TAD in current use, and the term *MSI* is specifically used to describe small (typically 1.5 to 2 mm in diameter and 6 to 10 mm in length) machined

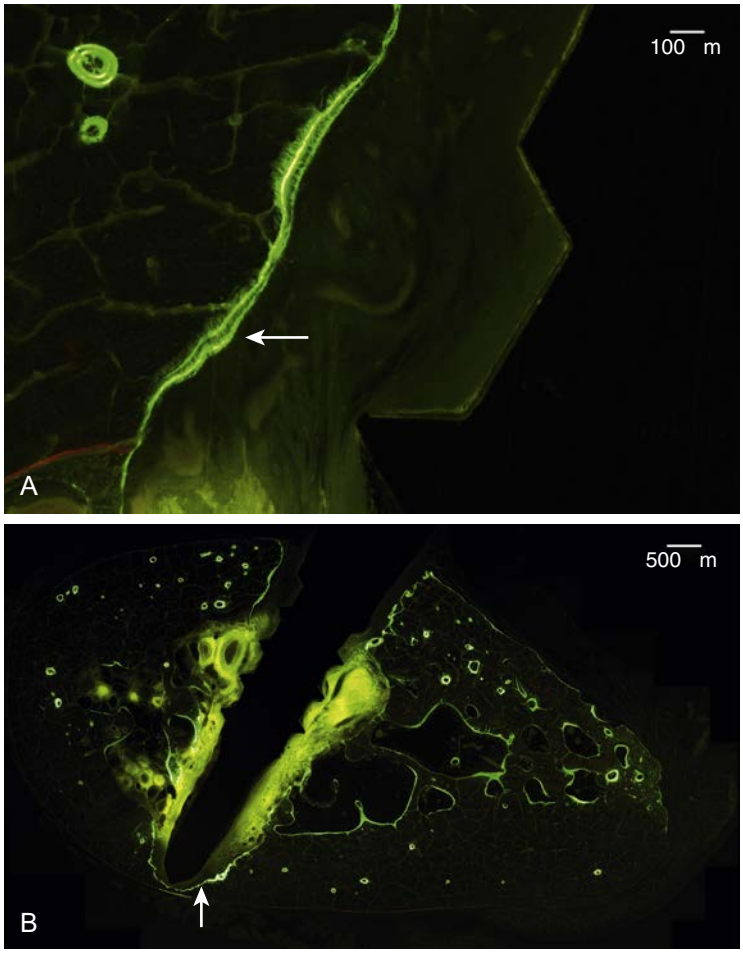


FIGURE 4-78 Failed implant with fibrous tissue interface. A, Bone is seen on left side and fibrous tissue between (arrow) the implant and toward the bone surface. B, Failed implant encapsulated with fibrous tissue in a canine model. There is no bone contact. The implant has nearly perforated the opposite cortex (*arrow*). (From Huja SS. Bone anchors – can you hitch up your wagon? *Orthod Craniofac Res*. 2015;18(suppl 1):109–16).

devices. Another less popular but possibly a more accurate collective term for these types of devices that are borne by the bone would be *skeletal anchors.* There is a large vocabulary of terms that have been introduced into the literature to describe these anchorage devices, their design, and other features associated with their use, and a description of such terms can be found in the literature.[171]

MSI are placed within the alveolar process, typically in an interradicular location. As MSI are placed in the close vicinity of the anchorage requirement, they eliminate the need for complex biomechanics and strategies that are typically seen, for example, with retromolar implants.[144] This versatility of placement is considered to be the major advantage for these anchors. The literature is replete with numerous case reports and studies on the potential and possibilities of enhancing anchorage with MSI.[172] However, one of the major issues with MSI are the persistently high failure rates.[173] Orthodontists are also interested in other skeletal anchorage options such as miniplates[174] and other extraalveolar sites for MSI placement, such as the palate.[175] The salient difference between many of these TAD is that MSI currently cannot routinely support larger forces (e.g., 10 N) over a prolonged duration (1 to 2 years) and are typically used for movement of few teeth over a period of 6 to 8 months.[172]

One of the assumptions made by researchers and clinicians alike is that MSI would serve in an identical manner to endosseous implants. Endosseous implants have been demonstrated, beyond a doubt, to be rigid and capable of withstanding high orthodontic forces and prolonged loads.[176] They osseointegrate to the bone, and no movement of implant device has been observed after load application.[10] Unfortunately, the term *absolute anchorage* has crept into the orthodontics literature and suggests that MSI are identical to endosseous implants and are entirely rigid. While it was desired that MSI would not fully osseointegrate and could be removed upon completion of their use, some of the other sequelae such as high[177] failure rate (10% to 30%) and displacement[178] were not anticipated.

Osseointegration

A major challenge for researchers in developing a more successful implant device is to determine how a "successfully osseointegrated" implant should appear on histologic examination in vivo. These histologic studies are conducted in animals or from retrieval specimens from humans. The

definition and mechanism of a successful implantation historically has been described by the term *osseointegration* at a histologic level.[179] Osseointegration is the presence of vital load-bearing bone directly in contact with the implant. The term *osseointegration* is defined at a tissue level in animals, and thus most of the implant studies examine bone sections and quantify histologicic outcome variables that are suggestive of a favorable response at the interface. For others, osseointegration may refer to the lack of ability to remove the implant and break the bone–implant interface. Percent bone to implant contact (%BIC) is frequently measured in various studies. However, there is no clear, quantifiable metrics of what constitutes a successful implant on a histologic section. In addition, one cannot evaluate a successful implant solely from a histologic section as other mechanical factors (e.g., primary and secondary stability) cannot be measured on histologic sections. Also, there is disagreement on the question of whether an MSI osseointegrates. These devices are biocompatible and can be removed easily even after 1 to 2 years after insertion. Dental implants cannot typically be removed without the aid of a trephine. Thus, the level of osseointegration or strength of the interface between the bone and implant surface must be different for an endosseous implant and MSI.

A failed or failing implant can be ascertained from histologic sections (Fig. 4-78, *A* and *B*). The presence of fibrous tissue and woven bone[180] instead of load-bearing lamellar bone at the implant interface is indicative of overload and augurs to future failure. Another major challenge in animal studies is the inability to carry out these implant studies to long durations (e.g., greater than 9 to 12 months), thus mimicking their clinical use. While many studies examine the early time points (weeks and months after implantation), longer time points after bone healing has occurred are difficult to conduct in experimental designs, and costs can be prohibitive. Finally, selection of an appropriate animal model interpretation and extrapolation of results to humans have to be attempted with caution.[181] Within the framework of implant research, in vitro studies have contributed to the understanding of the cellular and molecular responses and gene expression, which may be predictors for the success of various implant-surface modifications.[182]

Bone Contact

Bone to implant contact, frequently referred as BIC, is measured in most histologic studies. The measurement is relatively straightforward but requires an intact bone–implant interface with the implant and the bone being sectioned together, which itself is challenging. Bone contact as measured in studies is a static measurement of a dynamic variable. The presence of bone remodeling at the implant interface is evidence that bone contact is dynamic—that is, it may increase or decrease and different areas of the implant may contact bone at different times.[24] Given that the remodeling rate is elevated and high at the implant interface,[183] it is likely that bone contact does change.

A question that intrigues researchers and clinicians alike is the relationship, if any, between bone contact with implant and implant success. It is unknown if an implant with 80% of bone contact will fail, while an implant with 30% bone contact will be successful or vice versa. Bone contact alone is not a predictor for implant success. Retrieval analyses of dental implants that were in function for many years point to 50% to 60% bone contact.[184]

Bone Remodeling

Presence of viable bone is key to success at an implant interface. One method to measure the metabolic activity at an implant interface is by estimation of bone remodeling in supporting cortical and trabecular compartments. One study examined retrieval specimens from various animal species,[183] and it was observed that even after accounting for periods of time for typical bone healing, a persistent, elevated remodeling rate is observed in implant adjacent bone in the long term (e.g., 1 to 2 years after implantation). This led one group to conclude that an elevated rate of bone remodeling in the direct vicinity of the implant (0 to 1 mm from the interface) was critical to the long-term success of implants.[183] However, similar to bone implant contact, it is unclear what the magnitude of the bone turnover should be for it to be beneficial. In other words, can excessively high or low turnover rates be counterproductive at the implant interface?[185]

More recently, μCT has been used to study bone healing and adaptation. These μCT images provide 3D reconstructions of the region of interest. However, μCT still has not replaced dynamic histomorphometry but seems to have promise when measuring static histologic measurement. These subtleties may have been overlooked and confound study designs in animal experiments.[186] A major limitation of destructive examination by histology is that only a select number of 2D sections can be examined and do not reveal the true 3D nature of the implant interface.

Failure of Miniscrew Implants—Design or Unique Biological Constraints?

A major difference between MSI and endosseous implants is the need to remove the MSI after clinical use without a trephine. The ability to torque the MSI out without fracturing the implant or loss of bone in the alveolar process is critical. With this in mind, most MSI are smooth-surface machined implants. More recently, displacement of MSI, with migration of the device toward the point of force application, has been observed.[187] In addition, the MSI can be displaced (creep) within the bone without being extruded or "pulled out."[188] One key question is whether this migration of the MSI can be prevented. The nature and mechanism of MSI displacement within alveolar bone are unknown.

There is evidence to suggest that the alveolar process provides a unique milieu for implantation. It is well known that the volume of bone is small in the interradicular locations and placement of a screw close to the periodontal ligament results in increased probability that it will loosen and fail.[189] It is likely that bone within the alveolar process experiences greater strains than basal bone in the jaw and such strains may result in overloading of the implant interface or even predispose the MSI to failure in this hostile environment.[190]

Initial reports of displacement of MSI when subjected to orthodontic load were presented from 2D cephalometric data as early as 2004.[187] Subsequent reports with 3D cone beam studies suggest that MSI could be displaced by ~1 mm with a maximum value in one device of 4 mm.[191] Others indicated that MSI used in the maxilla had an average displacement of 0.78 mm; however, some of the MSI had a displacement close to 2 mm.[178] The current designs of MSI may not be able to withstand orthopedic loads. A clinical study reports that surface modification results in no difference in the survival rates of MSI used for orthodontic anchorage in the mandible and maxilla over a ~5-month period.[192] However, the CBCT data from a 9-month study of a larger C implant (SLA-coated and 1.8-mm diameter) in the maxilla for en masse canine retraction

indicates that the MSI remain stationary.[193] It is not common practice currently to use surface-modified MSI. However, it seems that an implant with surface modification of an appropriate diameter may provide more rigidity.

Rigidity of Miniscrew Implants

Initially, when skeletal anchorage was introduced to orthodontics, the anchors were used in an extraalveolar location (e.g., retromolar implants, zygomatic wires). It was only with the introduction of smaller (1.5- vs. 3.75-mm-diameter) miniimplants that interradicular placement was attempted.[194] In addition, a major advantage of MSI is the ease by which they can be placed by the orthodontist accurately at the desired site from which load could be applied.[195] This overcomes the need for patient referral, the additional cost and time for device placement by a surgeon.

One of the questions that have not been systematically addressed is the ideal diameter of MSI. The diameter has been determined primarily by the site of placement. For example, when MSI are placed between roots of maxillary molars for intrusion of maxillary posterior segment, a device with a diameter of 1.3 mm will allow for adequate bone for placement/retention and prevent impingement on adjacent roots.[196] As bone volume and vital structures are not a consideration in some extraalveolar sites, it should be possible to use wider diameter implants, should they provide greater rigidity and service. Systematic studies will be needed to address these questions. Other methods to test for a successful implant such as insertion torque, removal torque, pull-out testing, fatigue loading, and other standard tests have not been discussed but are acknowledged.

There are a large number of studies conducted in a variety of animal models. Animal models serve as one step toward translation of discovery to humans, and there are limitations and advantages to each animal model.[181] The typical histomorphometric variables that should be measured to provide relevant and useable information to the reader and for preventing repetition of studies especially on larger animal models[197] are briefly discussed below, and the reader is referred to a recent review for additional details.[198]

A novel non-anchorage related application of miniscrew implants has recently been proposed. Alveolar bone loss after extraction, loss of primary teeth or absence of permanent teeth, can compromise the vertical alveolar height for subsequent implants. An animal experiment based on clinical observations[199] suggests that vertical bone loss can be prevented by horizontal insertion of a miniscrew transcortically below the alveolar crest (Fig. 4-79). This is in contrast to insertion of a miniscrew vertically through the alveolar crest.[200] Clinical applications, such as placing a miniscrew in the palate below the alveolar crest to support a pontic for a missing lateral or at the site of second primary molars, may preserve bone for future endosseous implants.

ANIMAL MODELS FOR STUDYING BONE ADAPTATION, REMODELING, AND MODELING

Rodents (mice and rats), Lagomorpha (rabbits), canines (dogs), and porcine (pigs) serve as models for study of orthodontic tooth movement and craniofacial biology. Each of these animal models has been extensively studied. However, it is important to understand differences among these models. While selecting an animal model, it is important to ask the question or test

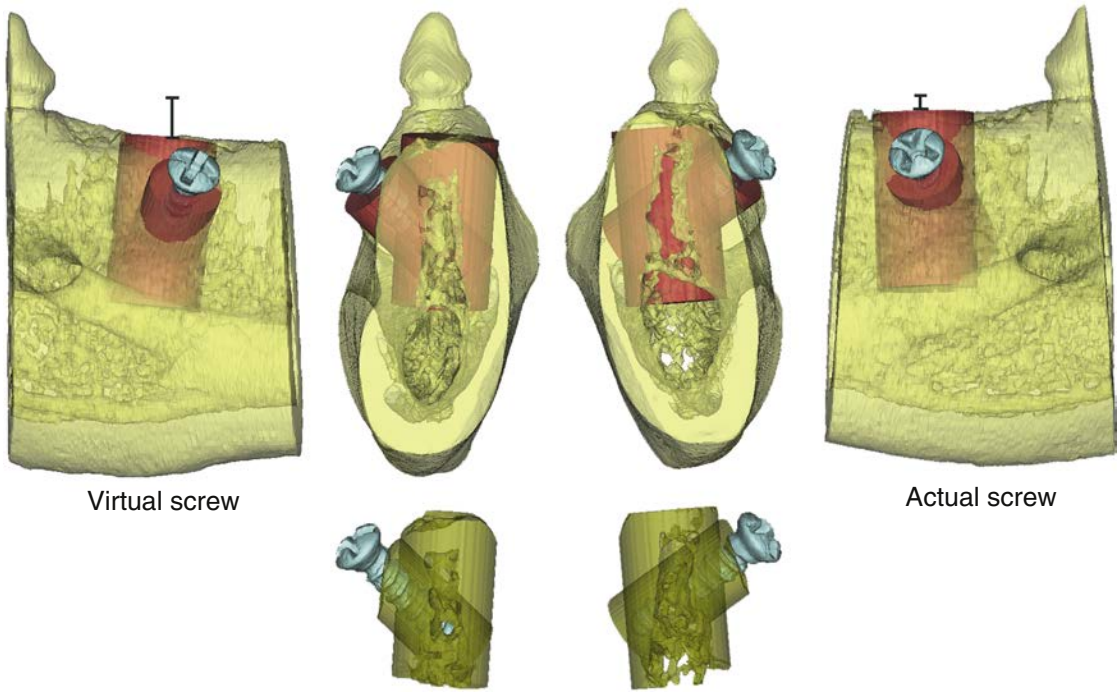


FIGURE 4-79 Surface models of a transmucosal screw placed at an extraction site versus contralateral side where no screw was placed but a virtual screw was simulated. Placement of a miniscrew below the alveolar crest prevented further bone loss as compared to the control (*virtual*) side. This bone loss will potentially allow for vertical bone height preservation. (From Melsen B, et al. Alveolar bone preservation subsequent to miniscrew implant placement in a canine model. *Orthod Craniofac Res*. 2015;18(2):77–85).

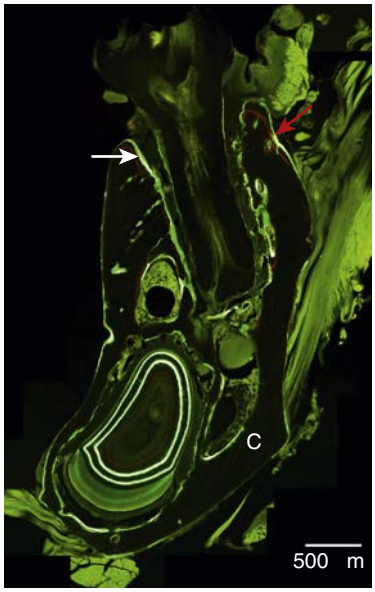


FIGURE 4-80 Epifluorescent image from an ~13-month-old rice rat mandible containing a mandibular molar and surrounding bone. Note that modeling occurs on the periosteal (*red arrow*) and periodontal surface (*white arrow*). The cortical compartment (*C*) is devoid of secondary osteons, indicating no physiologic bone remodeling.

a hypothesis that can be answered in the particular and suitable animal model. Thus, it is critical that the bone physiology of the model be understood. For example, rodents possess thin[201] cortical plates (<0.2 to 4 mm), and vascular invasion to produce an osteonal system is not required. The vascular supply from the periosteal and endosteal surfaces suffices to provide nutrients to the bone cells within the cortical plates. Thus, typically in the jaw bone of rodents only modeling is observed (Fig. 4-80) on the periodontal and periosteal surfaces of the bone. Inbred mice such as the C3H mice have thickened cortical plates (Fig. 4-81, *A* and *B*) and do demonstrate evidence of cortical bone remodeling in the femur.[201] In addition, in response to injury, ovarioectomy and possible microdamage accumulation, targeted remodeling with appearance of osteons, may occur in rodents[202] within the cortical plate. This remodeling is not physiologic but experimentally produced. When studying bone remodeling at a tissue level the rodent model will not possess the typical intracortical osteonal remodeling in the jaw bones (Fig. 4-82, *A* and *B*). However, the cell and molecular events[203] in the bone of the rodent more closely reflect the changes that are observed in larger animals. Thus, rodents possess many advantages in studying tooth movement, and many cellular, molecular pathways can be interrogated, with probes being available for a large number of molecules. In addition, transgenics offer new insights into bone biology and mechanisms of bone adaptation and can be exploited in rodent models.[204]

In contrast to the cortical bone, the trabecular bone[205] of the distal femur and proximal tibia (Fig. 4-83) are standard sites to evaluate by alterations in bone remodeling using histomorphometric methods in mice and rats, respectively. These trabecular bone sites allow researchers to examine the effects of drug interventions, experimental procedures, or transgenes on bone remodeling in a rodent model. In other words, rodents remodel in the trabecular bone but not typically in the intracortical compartment.

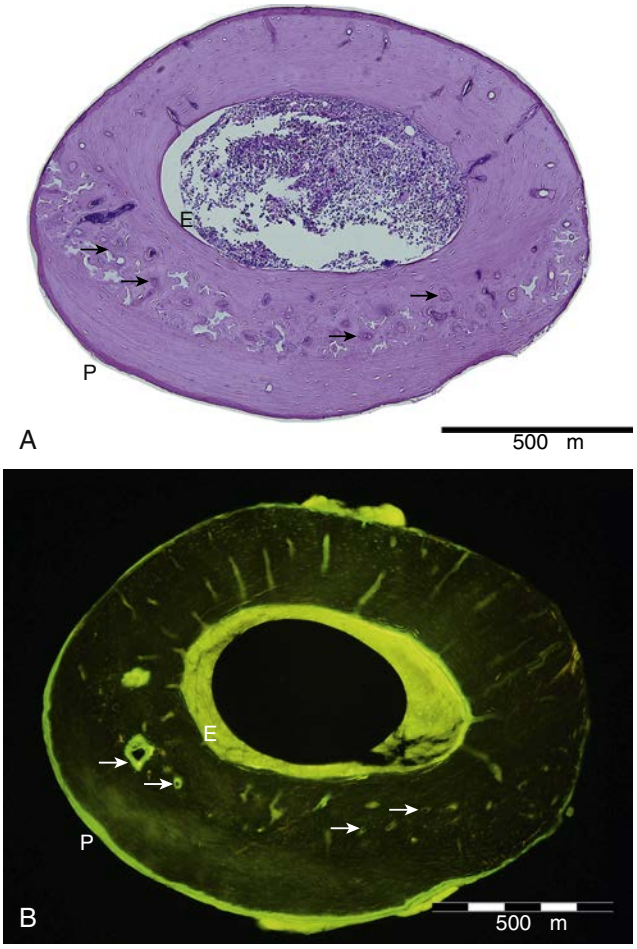


FIGURE 4-81 Hematoxylin and eosin (A) and epifluorescent (B) sections from a transverse section of a C3H mouse femur. The C3H is an inbred mouse with thick bones. When the bone dimension increases, vascular invasion with osteonal-like structures (*white and black arrows*) become apparent in the cortical compartment away from the periosteal (*P*) and endosteal (*E*) surfaces.

Rabbits are also used to study craniofacial biology such as sutural growth[111] implant biology.[206] The jaw bone of the rabbit is lamellar on the periosteal and endosteal surfaces with numerous osteons in the intracortical compartment (Fig. 4-84, *A* and *B*). Figure 4-85 shows the root structure and bone distribution in a rabbit model, which are vastly different from those in the canine model and humans. Thus studies, for example, on tooth movement have to be carefully planned and interpreted from data obtained from rabbits. The rate of osteonal bone turnover is low and is estimated to be 2%/yr in the jaw bones of 4-month-old female New Zealand rabbits (see Fig. 4-84, *A* and *B*). Thus rabbits, in a scaling order of size, are the first animal model to routinely demonstrate physiologic intracortical osteonal secondary remodeling in the jaw bone, but the remodeling remains to be at a much lower level than other larger animal models.

In the canine model, physiologic intracortical secondary osteonal remodeling (Fig. 4-86) occurs throughout the skeleton, in both the trabecular[85] and cortical bone compartments.[12,207] Animal husbandry in larger animal models can be very expensive. However, the dental structures and bone distribution are

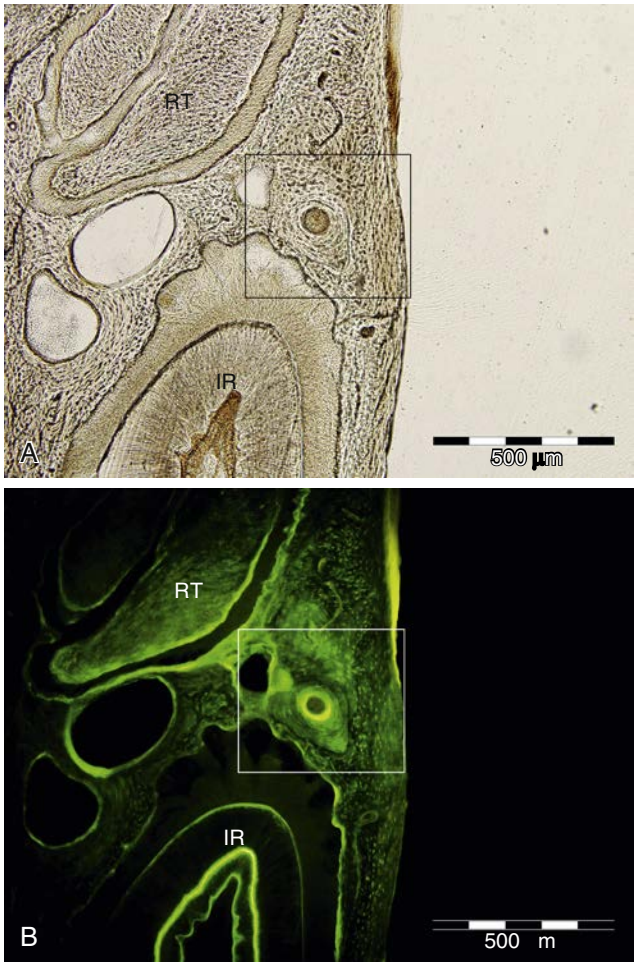


FIGURE 4-82 Brightfield (A) and epifluorescent (B) images of a buccolingual section from a B6 mouse mandible. Within the box an osteonal structure is observed just above the incisor root (*IR*) and below the root tip (*RT*). These structures are rare, indicating that mice to do have physiologic secondary intracortical osteonal bone remodeling in the mandibular bone. (From Meta IF, et al. Alveolar process anabolic activity in C3H/HeJ and C57BL/6J inbred mice. *J Periodontol*. 2008;79(7):1255–1262. Reproduced with permission from the American Academy of Periodontology).

similar to those in humans. Histomorphometric dynamics in the canine model have been studied extensively.[208,209] It is also possible to place implants and devices that are used in humans without scaling the size of the device.

Porcine models also demonstrate intracortical and trabecular bone remodeling and continuous growth (Fig. 4-87). However, the ability to obtain older animals for experimental studies remains a challenge. These animal models (such as minipigs) have been used to study distraction osteogenesis, biology of bone adaption to functional forces,[190] and occlusion. In a study of four ~2-year-old domestic female pigs the mean rate of bone turnover in the bone of the alveolar process of the mandible was 31.2%/yr and 17.9%/yr in the basal region of the mandible. The rate of bone turnover in the femur was 54%/yr.

EXPEDITED TOOTH MOVEMENT

Currently there are increasing numbers of case reports that demonstrate that substantial reductions in orthodontic

FIGURE 4-83 Epifluorescent image from an ~13-month-old rice rat proximal tibia demonstrating trabecular bone remodeling (*white arrow*) in the metaphyseal bone. Alizarin bone seeking label is seen in red (*red arrow*). This is the typical site for measuring bone remodeling activity in a rat model, in contrast to the mandibular cortical bone (see Figure 4-80) that does not demonstrate cortical bone remodeling.

treatment times are achievable. Corticotomies, vibration, laser, electric current, and controlled localized injury have been suggested to increase the bone metabolic rate that is considered to be the "mechanism" that reduces treatment time in orthodontic patients. Altering bone remodeling, a tissue-level histologic event, to expedite tooth movement has not been fully explored and substantiated by research studies. The treatment duration for comprehensive orthodontic therapy is estimated to be 24 to 30 months. Predictable reduction in treatment time to approximately 12 months would be a significant advance that would benefit orthodontic patients. Reduction in treatment time would also have the additional benefit of potentially decreasing the major sequelae of orthodontic therapy, such as root resorption and white spot lesions. Treatment duration has probably remained largely unchanged for the past century. With the synergy of rapid advances in technology and understanding the biology of tooth movement, opportunities for expediting tooth movement seem to be poised on the horizon. There are a number of clinical case reports that suggest treatment time can be reduced for orthodontic patients.[210,211] A number of hurdles remain before such claims can be substantiated and become evidence based. For example, does a reduction in treatment time during the initial phases (initial leveling/alignment or space closure) of orthodontics translate to overall reduced treatment time? Is it possible that the finishing phase of treatment would actually be prolonged? Also the cost-benefit ratio of a 4- to 6-month reduction in treatment time and the quality of the final result should be compared to cases with traditional treatment times.

In the literature a number of "mechanisms" have been suggested as a/the reason responsible for the accelerated tooth movement. These include:

1. Cortical plates are an impediment to orthodontic tooth movement[212]

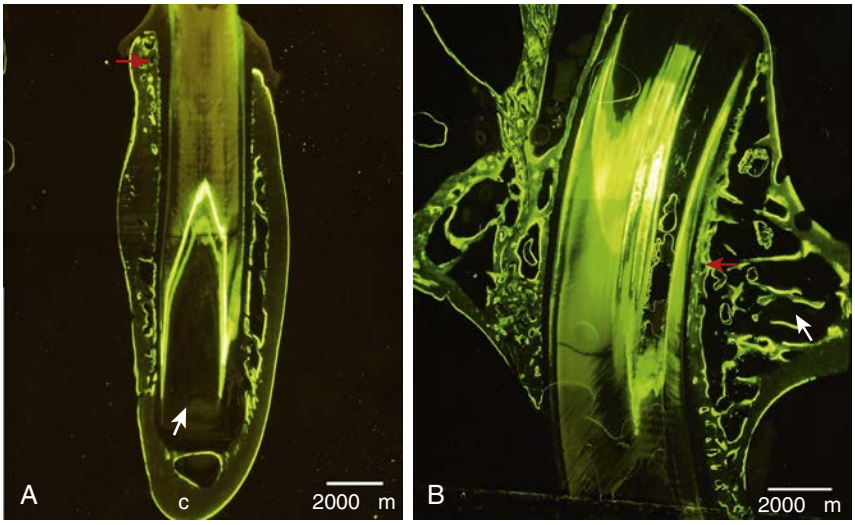


FIGURE 4-84 A, Epifluorescent buccolingual section of a rabbit mandibular molar. Note open apices of root (*white arrow*) and a thin plate of cortical bone (*C*). Only a few labeled osteons are apparent in the alveolar bone toward the crest (*red arrow*). B, Epifluorescent buccolingual section of a rabbit maxillary molar. The cortical bone supporting the tooth is very thin (*red arrow*) with large trabecular spaces (*white arrow*).

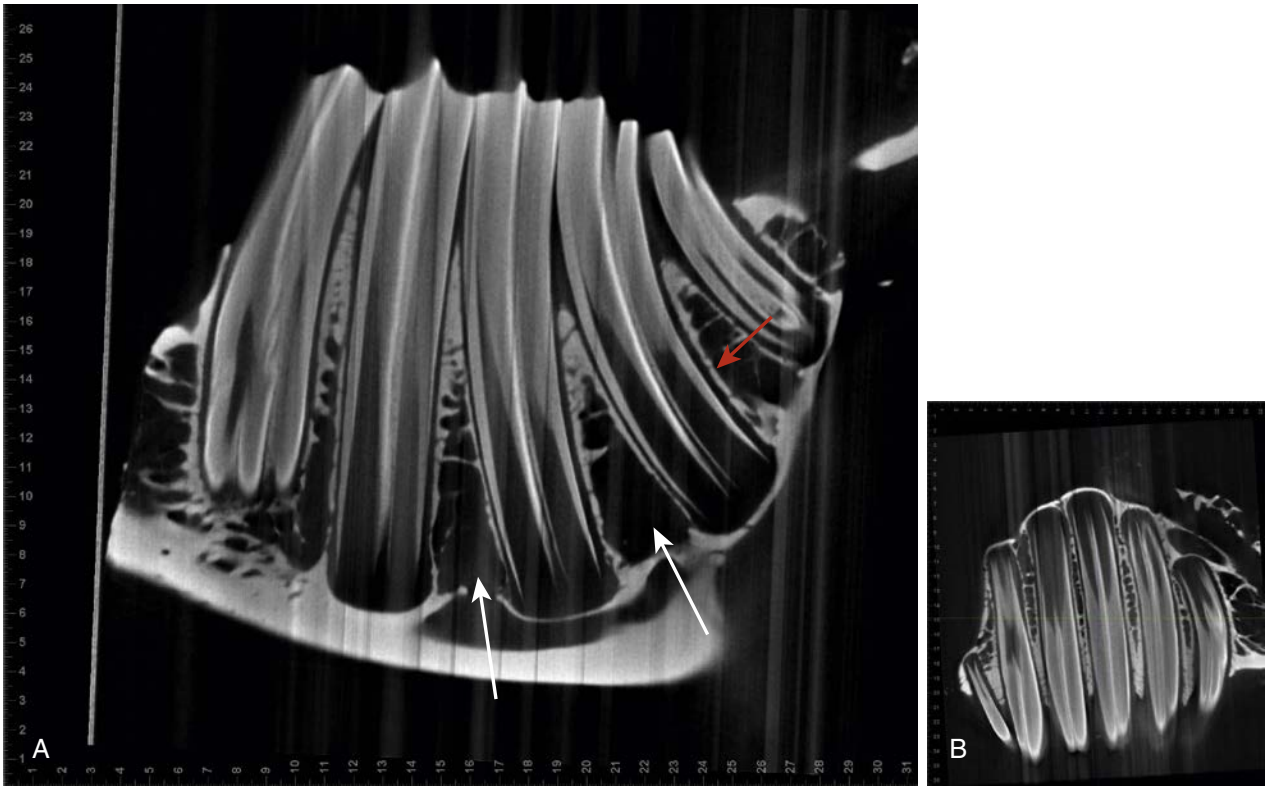


FIGURE 4-85 Sagittal view of mandibular (A) and maxillary (B) molars reconstructed from a high-resolution microCT of an ~4-month-old New Zealand female rabbits. Note the open apices of the roots of the teeth, and very thin alveolar bone (*red arrow*) with large marrow spaces (*white arrows*). The anatomy of the supporting bone is very different from a canine model and human bone.

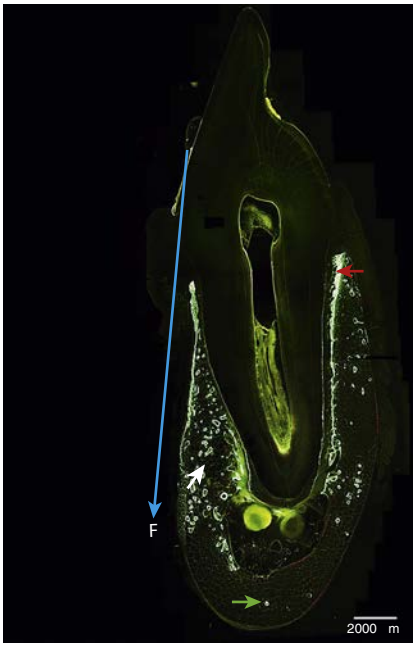


FIGURE 4-86 Epifluorescent buccolingual section from a dog molar root. An intrusive force (*F*) was applied from a miniscrew inserted between the interradicular area. Bone apposition (*red arrow*) is seen adjacent to the periodontal ligament, and secondary osteons are readily apparent and numerous in the cortical alveolar compartment (*white arrow*). The basal bone (*green arrow*) demonstrates a lower rate of bone turnover.

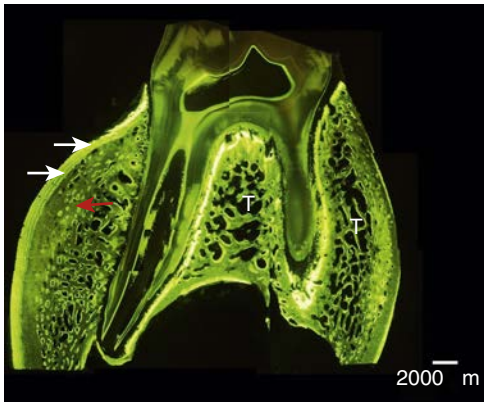


FIGURE 4-87 Epifluorescent section from a domestic pig jaw molar region. Even in this 24-month-old animal, modeling (*white arrows*) events are apparent on the periosteal surface. Below the modeling surface intracortical osteonal remodeling (*red arrow*) is observed. There is an intervening layer of trabecular bone (*T*) adjacent to the cortical bone lining the periodontal ligament.

2. Regional acceleratory phenomena and associated tissue remodeling[213]
3. High rates of bone turnover[213]
4. Demineralization/remineralization process[214]
5. Increase in cortical bone porosity, transient osteopenia, and osteopenia-facilitated rapid tooth movement[213]
6. Dramatic increase in trabecular bone turnover[213]
7. Bone matrix transportation[214]

The basis of some of these statements lies in beliefs, observations, and interpretations from clinical and animal data. All of the above listed "mechanisms" refer to tissue-level histologic events and should be explained by a thorough understanding of bone remodeling[215] and modeling[97] processes. These two processes are well described in the bone literature. However, they are frequently misunderstood and are clarified in the earlier part of this chapter.

Regional Acceleratory Phenomena

Regional acceleratory phenomena (RAP) are most exclusively and frequently cited in the literature as the basis of accelerated tooth movement. RAP was first described by Dr. Harold Frost.[62] He described RAP as a complex reaction to diverse noxious stimuli. He indicated that it is an "SOS" mechanism and acceleration of normal vital tissue processes. In humans, it lasts 4 months in bone and somewhat less in soft tissues. Importantly, RAP is a process of intermediary organization of tissues and organs *and not revealed in isolated cells*. This statement is very important as concluding from isolated cells that RAP is occurring in an experimental system is incorrect as originally defined. Initially RAP was described in cortical tissues and later in trabecular bone. RAP is *not* typically accompanied by osteopenia in the cortical[216] and trabecular[217] bone compartments as has been described in the orthodontic literature. The osteopenia associated with orthodontic tooth movement in some rodent animal models may be unique to the model in which it is being described and may not be related directly to the RAP per se. The contribution of a large rigid structure (the tooth) within the bone with and without superimposed force application may modify the response to tissue injury.

Another term that has been introduced in the orthodontic literature in terms of mechanisms of accelerated tooth movement is *demineralization* (which implies loss of mineral to some extent). New bone has less mineral content. The two phases of deposition of mineral in bone have been well described earlier in this chapter. It is not surprising that the new bone that is deposited is less rigid and more compliant and has less mineral.[29] It seems that in the orthodontic literature on expedited tooth movement, it is implied that the mineral is removed from the bone; however, the bone matrix remains intact and the tooth is transported through the matrix without the typical resistance experienced in a mineralized tissue. It is unclear how the bone mineral is exclusively removed rapidly without bone resorption by osteoclasts, which would also remove the matrix of bone. A decrease in bone volume rather than a decrease in mineral per se seems more plausible. Bone volume can be altered by osteoclastic resorption or by bone formation rather than by a mechanism for sole and rapid removal of the mineral.

Bone Remodeling Rate

In order to understand if increases in bone remodeling (bone turnover) rate are responsible for increased rate of tooth movement, it will be important to quantify the alterations (increases or decreases) in the rate of bone remodeling. It is well known that cortical bone remodels at 2 to 10%/year, and the rate of turnover in the trabecular bone is 30 to 35%/year.[40,44] Trabecular bone is metabolically active and is the source of serum calcium. Interestingly in the alveolar bone that supports the tooth, the physiologic rate of cortical bone turnover can be as high as 35%/year, this being 3- to 10-fold higher than cortical bone elsewhere (e.g., in the long bones) in the body.[12,218] In implant adjacent bone the rate of bone turnover can be as high as 100 to 500%/year, suggesting intense cortical bone remodeling in

implant adjacent bone.[219] It is likely that this elevated turnover is required to maintain a compliant zone of bone and to buffer for the modulus mismatch between the implant and bone. This elevated rate of bone turnover is seen both in miniimplant and implant adjacent bone. During tooth movement,[140] the rate of cortical bone turnover is estimate to be 100 to 200%/year—again, a 3- to 6-fold increase over the physiologic rate in the alveolar process. Currently there is no quantification of intracortical bone remodeling in the alveolar process after corticotomies. Thus, we do not know if the rate of turnover increases, for example, to 1000%/year or to any such level. Without these data from cortical and trabecular bone, it is difficult to confirm that RAP or increased bone turnover is responsible or accompanies expedited tooth movement and even the sole mechanism that allows for more rapid tooth movement.

Current Evidence of Expedited Tooth Movement from Experimental Studies on Rodents and Canines

While a multitude of animal models have been used to study orthodontic tooth movement and specifically expedited tooth movement, it behooves us to understand and interpret the results from the literature as translation to our patients must be attempted with caution.

A rat model has frequently been exploited to study the biology of expedited tooth movement in response to injury (corticotomy, piezocision, etc.) Bone changes subsequent to injury have been demonstrated[213] using a rodent/rat model. An indentation with a dental burr onto the cortical bone of the maxilla was used to represent the decortication injury common in the "Wilkodontic" procedure. Sebaoun et al.[213] demonstrated that the injury results in "disappearance" of bone in the 3-week surgery group, between the roots of the 1st molar, and at 11 weeks, the bone was restored between the roots similar to that of the 3-week control group. They conclude their histomorphometric data (intravital bone labels and TRAP staining for osteoclasts) suggests that modeling of the trabecular bone occurs and bone turnover after corticotomy *does not* involve a linear/sequential series of events. In other words, remodeling (coupled bone formation/resorption) in the rat model is not responsible for the bone response after decortication under their specific experimental conditions. In this particular study, there is no explanation of how mineralized tissue returns to its original state. Typically bone resorption by osteoclasts results in the loss of both the mineralized tissue and the organic matrix. Once bone loss occurs (e.g., in trabecular bone of the spine during osteoporosis), reversal of the bone loss and new bone formation do not occur, as no matrix exists for new bone formation to occur within. Baloul et al.[220] studied the corticotomy-facilitated tooth movement with the aid of microCT and the expression of selective osteoclast and osteoblast genes in their rodent model. They demonstrate in the split-mouth study design that the rate of tooth movement initially peaks at 7 days in the decortication side as compared to 14 days on the control side. In their mCT data, they demonstrate that apparently greater values of BV, BV/TV, BMC, and BMD occur in their corticotomy-only group compared to the tooth movement-only group or tooth movement plus corticotomy group. They also demonstrate that key osteoblastic and osteoclastic genes are elevated temporally, suggesting increased bone activity. However, due to the method of collection of the tissue sample for this gene analysis, it is not possible to analyze on which surface the bone formation or bone resorption is occurring. As Frost indicated, RAP does not occur in isolated cells but at a tissue level of organization. The field of molecular biology did not exist at the time of Dr. Frost's publication. Dibart et al.[221] used piezocision to facilitate the orthodontic tooth movement in rats. Their histologic images demonstrate aggressive/extensive bone loss that extends beyond the initial piezocision site, and the injury results in changes in both the cortical and trabecular bone. Dibart et al.[221] demonstrated reduction in percent bone from 60% to virtually 0% to 20% in all their three groups. The question remains if in a human, such reductions would result in the devastation of the bone strength and structure. Also, it is questionable if tooth movement could occur in bone that has been so severely compromised. This may be a limitation of the model and may not entirely detract from other parts of the data. Also, in the presence of such severe bone loss, it is unknown whether a matrix for regaining the bone would exist. In the study, however, bone recovery occurred primarily between the 40- and 60-day period.[222]

The dog/canine animal model also offers valuable insights into understanding of expedited tooth movement. The concept of cortical bone resistance impeding tooth movement was tested by undermining the septal bone in a canine model.[212] This work demonstrated that the cortical bone structure has to be resorbed during tooth movement, and surgical removal of this localized bone will result in more rapid tooth movement. Doubling in the rate of tooth movement over a period of 6 weeks was demonstrated in a split-mouth study in a canine model. Mostafa et al.[223] demonstrated that the injury (corticotomy) has only a transient effect, and a short window of opportunity exists to effect the tooth movement. Major differences were seen in the first 2 weeks between the groups, and the authors suggest that based on histology there was decreased hyalinization in the experimental groups. Their histologic images are quite different from the rodent model, with no rapid declines in mineral content of bone being apparent.[223] This leads to the question whether what is seen in the rodents would really be observed in humans. In a similar split-mouth study, it was observed that a higher rate of tooth movement occurred in the experimental group, with the rate of tooth movement in a canine model being higher in the maxilla than in the mandible.[224] Similar to other studies, there are greater differences between the control and experimental groups, in the first 2 to 3 weeks after which in these animal models the differences diminish. The transient nature of the effect reinforces the need for the tooth movement to begin immediately after the corticotomy. In humans, if the effect is primarily seen for 6 weeks, only a small portion of a phase of treatment, e.g., canine retraction, would be complete, and the question remains if a second invasive surgery would be warranted.

Based on the current literature, there are inconsistencies and vast differences in histology and interpretation of the histology between rodent and canine animal models. This poses a problem, and it is unclear as are the mechanism(s) of expedited tooth movement. The rate-limiting step in tooth movement is resorption of bone by osteoclasts. However, in rodents, the bone mineral "disappears" by a method other than osteoclastic resorption, and transport of the tooth occurs within the compliant bone matrix. In larger animal models, the corticotomies do not seem to "devastate" the bone—that is, the BV/TV does not drop from 60% to 20% in a matter of weeks.

Molar protraction even when using contemporary methods of anchorage can take approximately 20 to 24 months. This type of tooth movement would lend itself to investigation

into procedures that expedite the rate of tooth movement. In contrast to animals, in humans, the two-rooted mandibular molar moves mesially, and the rate of tooth movement[144] is diminished. Other nonultrainvasive methods such as vibration (Acceledent type of device), and microperforations (Propel-type devices), also are believed to alter the rate of bone turnover, but animal studies do not clearly demonstrate an altered, secondary osteonal-bone remodeling.

SUMMARY

Bone physiologic, metabolic, and cell-kinetic concepts have important clinical applications in orthodontics and dentofacial orthopedics. The application of fundamental concepts is limited only by the knowledge and imagination of the clinician. Modern clinical practice is characterized by a continual evolution of methods based on fundamental and applied research.

Acknowledgments

WER was supported by National Institute of Dental Research (National Institutes of Health [NIH]) grants DE09237 and DE09822), NASA-Ames grants NCC 2-594 and NAG 2-756, and private donors through Indiana University Foundation. The author gratefully acknowledges the assistance of faculty and staff members at the University of the Pacific School of Dentistry and Indiana University School of Dentistry. SSH was supported by the American Association of Orthodontist Foundation, NIH R03, Delta Dental, and funds from the Ohio State University and University of Kentucky. SSH is extremely grateful for Dr. Cristina Exposto for her editorial assistance.

REFERENCES

1. Roberts WE, Hartsfield Jr JK. Bone development and function: genetic and environmental mechanisms. *Semin Orthod*. 2004;10:100–122.
2. Burn AK, Herring SW, Hubbard R, Zink K, Rafferty K, Lieberman DE. Dietary consistency and the midline sutures in growing pigs. *Orthod Craniofacial Res*. 2010;13(2):106–113.
3. Abed GS, Buschang PH, Taylor R, Hinton RJ. Maturational and functional related differences in rat craniofacial growth. *Arch Oral Biol*. 2007;52(11):1018–1025.
4. Hylander WL. Mandibular function in *Galago crassicaudatus* and *Macaca fascicularis*: an in vivo approach to stress analysis of the mandible. *J Morphol*. 1979;159(2):253–296.
5. Hylander WL. In: Carlson DS, ed. *Patterns of Stress and Strain in the Macaque Mandible*. Ann Arbor, MI: Center for Human Growth and Development; 1981.
6. Iwasaki LR, Crosby MJ, Marx DB, Gonzalez Y, McCall Jr WD, Ohrbach R, et al. Human temporomandibular joint eminence shape and load minimization. *J Dent Res*. 2010;89(7):722–727.
7. Behrents RG. Adult facial growth. In: Enlow DH, ed. *Facial Growth*. 3rd ed. WB Saunders; 1990.
8. Shirakura M, Tanimoto K, Eguchi H, Miyauchi M, Nakamura H, Hiyama K, et al. Activation of the hypoxia-inducible factor-1 in overloaded temporomandibular joint, and induction of osteoclastogenesis. *Biochem Biophys Res Commun*. 2010;393(4):800–805.
9. Proffit WR. *Contemporary Orthodontics*. 5th ed. St. Louis: Mosby Year Book; 2013.
10. Roberts WE, Smith RK, Zilberman Y, Mozsary PG, Smith RS. Osseous adaptation to continuous loading of rigid endosseous implants. *Am J Orthod*. 1984;86(2):95–111.
11. Roberts WE, Turley PK, Brezniak N, Fielder PJ. Bone physiology and metabolism. *J Calif Dent Assoc*. 1987;15:54–61.
12. Huja SS, Fernandez SA, Hill KJ, Li Y. Remodeling dynamics in the alveolar process in skeletally mature dogs. *Anat Rec Part A Discov Mol Cell Evol Biol*. 2006;288(12):1243–1249.
13. Roberts WE, Chase DC. Kinetics of cell proliferation and migration associated with orthodontically-induced osteogenesis. *J Dent Res*. 1981;60(2):174–181.
14. Roberts W, ed. *Advanced Techniques for Quantitative Bone Cell Kinetics and Cell Population Dynamics*. Ottawa: University of Ottawa Press; 1976.
15. Roberts WE, Chase DC, Jee SS. Counts of labelled mitoses in the orthodontically-stimulated periodontal ligament in the rat. *Arch Oral Biol*. 1974;19(8):665–670.
16. Roberts WE, Jee WS. Cell kinetics of orthodontically-stimulated and non-stimulated periodontal ligament in the rat. *Arch Oral Biol*. 1974;19(1):17–21.
17. Roberts WE, Mozsary PG, Klingler E. Nuclear size as a cell-kinetic marker for osteoblast differentiation. *Am J Anat*. 1982;165(4):373–384.
18. Roberts WE, Ferguson DJ. Cell kinetics of the periodontal ligament. In: Norton LA, Burstone CJ, eds. *The Biology of Tooth Movement. 4*. Boca Raton, FL: CRC Press, Inc; 1989:56–67.
19. Katona TR, Paydar NH, Akay HU, Roberts WE. Stress analysis of bone modeling response to rat molar orthodontics. *J Biomech*. 1995;28(1):27–38.
20. Rapperport DJ, Carter DR, Schurman DJ. Contact finite element stress analysis of porous ingrowth acetabular cup implantation, ingrowth, and loosening. *J Orthop Res Off Publ Orthop Res Soc*. 1987;5(4):548–561.
21. Roberts WE, Garetto LP, Katona TR. Principles of orthodontic biomechanics: metabolic and mechanical control mechanisms. In: Carlson DS, Goldstein SA, eds. *Bone Biodynamics in Orthodontic and Orthopaedic Treatment*. 27th ed. Ann Arbor: University of Michigan Press; 1992:189–256.
22. Siegele D, Soltesz U. Numerical investigations of the influence of implant shape on stress distribution in the jaw bone. *Int J Oral Maxillofac Implants*. 1989;4(4):333–340.
23. Roberts WE, Smith RK, Cohen JA. *Change in Electrical Potential within Periodontal Ligament of a Tooth Subjected to Osteogenic Loading. Factors and Mechanisms Influencing Bone Growth*. New York: Alan R. Liss, Inc; 1982:527–534.
24. Huja SS, Roberts WE. Mechanism of osseointegration: characterization of supporting bone with indentation testing and backscattered imaging. *Semin Orthod*. 2004;10:162–173.
25. Yip G, Schneider P, Roberts WE. Micro-computed tomography: high resolution imaging of bone and implants in three dimensions. *Semin Orthod*. 2004;10(2):174–187.
26. Huja SS, Katona TR, Moore BK, Roberts WE. Microhardness and anisotropy of the vital osseous interface and endosseous implant supporting bone. *J Orthop Res Off Publ Orthop Res Soc*. 1998;16(1):54–60.
27. Huja SS, Hay JL, Rummel AM, Beck FM. Quasi-static and harmonic indentation of osteonal bone. *J Dent Biomech*. 2010:736830.
28. Huja SS, Katona TR, Roberts WE. Microhardness testing of bone. In: An YH, Draughn RA, eds. *Mechanical Testing of Bone and the Bone-Implant Interface*. Boca Raton, FL: CRC Press; 2000:247–256.
29. Huja SS, Phillips CA, Fernandez SA, Li Y. Tissue level mechanical properties of cortical bone in skeletally immature and mature dogs. *Vet Comp Orthop Traumatol*. 2009;22(3):210–215.
30. Keeting PE, Scott RE, Colvard DS, Han IK, Spelsberg TC, Riggs BL. Lack of a direct effect of estrogen on proliferation and differentiation of normal human osteoblast-like cells. *J Bone Miner Res Off J Am Soc Bone Miner Res*. 1991;6(3):297–304.
31. Roberts WE, Garetto LP, Simmons KE. Endosseous implants for rigid orthodontic anchorage. In: Bell WH, ed. *Surgical Correction of Dentofacial Deformities. 2*. Philadelphia: WB Saunders; 1992.
32. Currey J. *The Mechanical Adaptations of Bone*. New Jersey: Princeton University Press; 1984:80–87.

33. Martin RB, Burr DB. *Structure, Function and Adaptation of Compact Bone*. 1st ed. New York: Raven Press; 1989:186–213.
34. Roberts WE. Bone tissue interface. *J Dent Educ*. 1988;52(12):804–809.
35. Enlow DH. *Facial Growth*. 3rd ed. Philadelphia: WB Saunders; 1990.
36. Drinkwater BL, Nilson K, Chesnut 3rd CH, Bremner WJ, Shainholtz S, Southworth MB. Bone mineral content of amenorrheic and eumenorrheic athletes. *N Engl J Med*. 1984;311(5):277–281.
37. Colditz GA, Stampfer MJ, Willett WC, Hennekens CH, Rosner B, Speizer FE. Prospective study of estrogen replacement therapy and risk of breast cancer in postmenopausal women. *JAMA*. 1990;264(20):2648–2653.
38. Roberts WE. Bone physiology, metabolism, and biomechanics in orthodontic practice. In: Graber TM, Vanarsdall Jr RL, Vig KWL, eds. *Orthodontics (Current Principles and Techniques)*. 3rd ed. St. Louis: Mosby-Year Book, Inc; 2012:287–343.
39. Martin RB, Burr DB, Sharkey NA. *Skeletal Tissue Mechanics*. New York: Springer-Verlag; 1998.
40. Parfitt AM. Osteonal and hemi-osteonal remodeling: the spatial and temporal framework for signal traffic in adult human bone. *J Cell Biochem*. 1994;55:273–286.
41. Roberts WE. Bone physiology, metabolism, and biomechanics in orthodontic practice. In: Graber TM, Vanardall Jr RL, Vig KWL, eds. *Orthodontics: Current Principles and Techniques*. St. Louis: Elsevier Mosby; 2005:221–292.
42. Robling AG, Castillo AB, Turner CH. Biomechanical and molecular regulation of bone remodeling. *Annu Rev Biomed Eng*. 2006;8:455–498.
43. Huja SS. Are we any closer to understanding the mechanisms of expedited tooth movement? In: Kapila SDNJ, Hatch N, eds. *Expedited Orthodontics: Improving the Efficiency of Orthodontic Treatment Through Novel Technologies*. Craniofacial Growth Series; 651. Ann Arbor: Department of Orthodontics and Pediatric Dentistry and Center for Human Growth and Development, The University of Michigan; 2015:47–64.
44. Parfitt AM. Targeted and nontargeted bone remodeling: relationship to basic multicellular unit origination and progression. *Bone*. 2002;30:5–7.
45. Midgett RJ, Shaye R, Fruge Jr JF. The effect of altered bone metabolism on orthodontic tooth movement. *Am J Orthod*. 1981;80(3):256–262.
46. Shank SB, Beck FM, D'Atri AM, Huja SS. Bone damage associated with orthodontic placement of miniscrew implants in an animal model. *Am J Orthod Dentofac Orthop Off Publ Am Assoc Orthod Its Const Soc Am Board Orthod*. 2012;141(4):412–418.
47. Burr DB, Martin RB, Schaffler MB, Radin EL. Bone remodeling in response to *in vivo* fatigue microdamage. *J Biomech*. 1985;18:189–200.
48. Huja SS, Hasan MS, Pidaparti R, Turner CH, Garetto LP, Burr DB. Development of a fluorescent light technique for evaluating microdamage in bone subjected to fatigue loading. *J Biomech*. 1999;32(11):1243–1249.
49. Parfitt AM, Drezner MK, Glorieux FH, Kanis JA, Malluche H, Meunier PJ, et al. Bone histomorphometry: standardization of nomenclature, symbols, and units. *J Bone Miner Res*. 1987;2:595–610.
50. Parfitt AM. The physiologic and clinical significance of bone histomorphometric data. In: Recker RR, ed. *Bone Histomorphometry: Techniques and Interpretation*. Boca Raton, FL: CRC Press, Inc.; 1983:143–223.
51. Jones HH, Priest JD, Hayes WC, Tichenor CC, Nagel DA. Humeral hypertrophy in response to exercise. *J Bone Jt Surg*. 1977;59-A:204–208.
52. Frost HM. Wolff's law and bone's structural adaptation to mechanical usage: an overview for clinicians. *Angle Orthod*. 1994;64:175–188.
53. Roberts WE, Marshall KJ, Mozsary PG. Rigid endosseous implant utilized as anchorage to protract molars and close an atrophic extraction site. *Angle Orthod*. 1990;60(2):135–152.
54. Roberts WE, et al. Remodeling of mineralized tissues, part II: control and pathophysiology. *Semin Orthod*. 2006;12:238–253.
55. Mundy GR, Bonewald LF. Transforming grwoth factor beta. In: Gowen M, ed. *Cytokines and Bone Metabolism*. Boca Raton, FL: CRC Press; 1992.
56. Boyle WJ, Simonet WS, Lacey DL. Osteoclast differentiation and activation. *Nature*. 2003;423(6937):337–342.
57. Amizuka N, Shimomura J, Li M, Seki Y, Oda K, Henderson JE, et al. Defective bone remodelling in osteoprotegerin-deficient mice. *J Electron Microsc*. 2003;52(6):503–513.
58. Melsen B. The cranial base. The postnatal development of the cranial base studied histologically on human autopsy material. *Aarhus Acta Odontologica Scand*. 1974;32(suppl 62).
59. Roberts WE, Helm FR, Marshall KJ, Gongloff RK. Rigid endosseous implants for orthodontic and orthopedic anchorage. *Angle Orthod*. 1989;59(4):247–256.
60. Noorda CB. *Modeling and Remodeling in the Cortical Bone of Both Growing and Adult Rabbits*. San Francisco: University of the Pacific; 1986.
61. Brockstedt H, Bollerslev J, Melsen F, Mosekilde L. Cortical bone remodeling in autosomal dominant osteopetrosis: a study of two different phenotypes. *Bone*. 1996;18(1):67–72.
62. Frost HM. The regional acceleratory phenomenon: a review. *Henry Ford Hosp Med J*. 1983;31(1):3–9.
63. Roberts WE, Arbuckle GR, Katona TR, eds. *Bone Physiology of Orthodontics: Metabolic and Mechanical Control Mechanisms Symposion der Deutschen Gesellschaft für Kieferorthopadie*. Munich: Urban & Vogel; 1992.
64. Roberts WE, Garetto LP. Physiology of osseous and fibrous integration. *Alpha Omegan*. 1992;85(4):57–60.
65. Jeffcoat MK, Williams RC, Kaplan ML, Goldhaber P. Nuclear medicine techniques for the detection of active alveolar bone loss. *Adv Dent Res*. 1987;1(1):80–84.
66. Reddy MS, English R, Jeffcoat MK, Tumeh SS, Williams RC. Detection of periodontal disease activity with a scintillation camera. *J Dent Res*. 1991;70(1):50–54.
67. Nikolai RJ. *Bioengineering Analysis of Orthodontic Mechanics*. Philadelphia: Lea & Febiger; 1985.
68. Roberts WE, et al. Bone physiology: evaluation of bone metabolism. *J Am Dent Assoc*. 1991:122–159.
69. Roberts WE, Simmons KE, Garetto LP, DeCastro RA. Bone physiology and metabolism in dental implantology: risk factors for osteoporosis and other metabolic bone diseases. *Implant Dent*. 1992;1(1):11–21.
70. Rhodes WE, Pflanzer R. *Human Physiology*. Philadelphia: WB Saunders; 1989.
71. Frost HM. *Bone Remodeling and Its Relationship to Metabolic Bone Diseases*. Springfield, IL: Charles C Thomas; 1973.
72. Heaney RP. Estrogen-calcium interactions in the postmenopause: a quantitative description. *Bone Miner*. 1990;11(1):67–84.
73. Rigotti NA, Neer RM, Skates SJ, Herzog DB, Nussbaum SR. The clinical course of osteoporosis in anorexia nervosa. A longitudinal study of cortical bone mass. *JAMA*. 1991;265(9):1133–1138.
74. Consensus conference report on osteoporosis. *J Am Dent Assoc*. 1984:254–799.
75. Eriksen EF, Mosekilde L. Estrogens and bone. *J Bone Miner Res*. 1990.
76. Henrich JB. The postmenopausal estrogen/breast cancer controversy. *JAMA*. 1992;268(14):1900–1902.
77. Love RR, Mazess RB, Barden HS, Epstein S, Newcomb PA, Jordan VC, et al. Effects of tamoxifen on bone mineral density in postmenopausal women with breast cancer. *N Engl J Med*. 1992;326(13):852–856.
78. Goldfrank D, Haytoglu T, Frishman WH, Mohammad Z. Raloxifene, a new selective estrogen receptor modulator. *J Clin Pharmacol*. 1999;39(8):767–774.
79. Heaney RP. Calcium, bone health in osteoporosis. In: Peck WA, ed. *Bone and Mineral Research*. Amsterdam: Elsevier Science; 1990.
80. Johnston Jr CC. Osteoporosis–extent and cause of the disease. *Proc Soc Exp Biol Med Soc Exp Biol Med* (New York, NY). 1989;191(3):258–260.
81. Malluche H, Faugere MC. Renal bone disease 1990: an unmet challenge for the nephrologist. *Kidney Int*. 1990;38(2):193–211.
82. Calciolari E, Donos N, Park JC, Petrie A, Mardas N. A systematic review on the correlation between skeletal and jawbone mineral density in osteoporotic subjects. *Clin Oral Implants Res*. 2015.
83. Becker AR, Handick KE, Roberts WE, Garetto LP. Osteoporosis risk factors in female dental patients. A preliminary report. *Ind Dent Assoc J*. 1997;76(2):15–19, quiz 20.
84. Khan AA, Morrison A, Hanley DA, Felsenberg D, McCauley LK, O'Ryan F, et al. Diagnosis and management of osteonecrosis of the jaw: a systematic review and international consensus. *J Bone Miner Res*. 2015;30(1):3–23.
85. Helm NB, Padala S, Beck FM, D'Atri AM, Huja SS. Short-term zoledronic acid reduces trabecular bone remodeling in dogs. *Eur J Oral Sci*. 2010;118(5):460–465.
86. Huja SS. Effect of zoledronic acid on bone healing subsequent to mini-implant insertion. *Angle Orthod*. 2011.
87. Blackwell KA, Raisz LG, Pilbeam CC. Prostaglandins in bone: bad cop, good cop? *Trends Endocrinol Metab*. 2010;21(5):294–301.

88. Viecilli RF, Katona T, Chen J, Roberts E, Hartsfield Jr J. Comparison of dentoalveolar morphology in WT and P2X7R KO mice for the development of biomechanical orthodontic models. *Anat Rec*. 2009;292(2): 292–298 (Hoboken, NJ : 2007).
89. Viecilli RF, Katona TR, Chen J, Hartsfield Jr JK, Roberts WE. Orthodontic mechanotransduction and the role of the P2X7 receptor. *Am J Orthod Dentofac Orthop Off Publ Am Assoc Orthod Its Const Soc Am Board Orthod*. 2009;135(6):694.e1–694.e16, discussion 5.
90. Hanada R, Leibbrandt A, Hanada T, Kitaoka S, Furuyashiki T, Fujihara H, et al. Central control of fever and female body temperature by RANKL/RANK. *Nature*. 2009;462(7272):505–509.
91. Cowin SC. *Bone Mechanics*. Boca Raton, FL: CRC Press; 1989.
92. Riggs BL. Overview of osteoporosis. *West J Med*. 1991;154(1):63–77.
93. Lanyon LE. Control of bone architecture by functional load bearing. *J Bone Miner Res Off J Am Soc Bone Miner Res*. 1992;7(suppl 2):S369–S375.
94. Rubin CT, Lanyon LE. Regulation of bone mass by mechanical strain magnitude. *Calcif Tissue Int*. 1985;37(4):411–417.
95. Rubin CT, Lanyon LE. Osteoregulatory nature of mechanical stimuli: function as a determinant for adaptive remodeling in bone. *J Orthop Res Off Publ Orthop Res Soc*. 1987;5(2):300–310.
96. Rubin CT, McLeod KJ, Bain SD. Functional strains and cortical bone adaptation: epigenetic assurance of skeletal integrity. *J Biomech*. 1990;23(suppl 1):43–54.
97. Frost HM. Skeletal structural adaptations to mechanical usage (SATMU): 2. Redefining Wolff's law: the remodeling problem. *Anat Rec*. 1990;226(4):414–422.
98. Robling AG, Burr DB, Turner CH. Skeletal loading in animals. *J Musculoskelet Neuronal Interact*. 2001;1(3):249–262.
99. Carter DR. Mechanical loading history and skeletal biology. *J Biomech*. 1987;20(11–12):1095–1109.
100. Frost HM. *Intermediary Organization of the Skeleton*. Boca Raton, FL: CRC Press; 1986.
101. Brunski JB. Forces on dental implants and interfacial stressed transfer. In: Laney WR, Tolman DE, eds. *Tissue Integration in Oral, Orthopedic, and Maxillofacial Reconstruction*. Chicago: Quintessence; 1992.
102. Brunski JB, Skalak R. Biomechanical considerations. In: Worthington P, Branemark P-I, ed. *Advanced Osseointegration Surgery*. Chicago: Quintessence; 1992.
103. Herring SW, Teng S. Strain in the braincase and its sutures during function. *Am J Phys Anthropol*. 2000;112(4):575–593.
104. Mao JJ, Wang X, Kopher RA. Biomechanics of craniofacial sutures: orthopedic implications. *Angle Orthod*. 2003;73(2):128–135.
105. Haas AJ. The treatment of maxillary deficiency by opening the midpalatal suture. *Angle Orthod*. 1965;35:200–217.
106. Lee H, Ting K, Nelson M, Sun N, Sung SJ. Maxillary expansion in customized finite element method models. *Am J Orthod Dentofac Orthop Off Publ Am Assoc Orthod Its Const Soc Am Board Orthod*. 2009;136(3):367–374.
107. Chang HN, Garetto LP, Potter RH, Katona TR, Lee CH, Roberts WE. Angiogenesis and osteogenesis in an orthopedically expanded suture. *Am J Orthod Dentofac Orthop Off Publ Am Assoc Orthod Its Const Soc Am Board Orthod*. 1997;111(4):382–390.
108. Chang HN, Garetto LP, Katona TR, Potter RH, Roberts WE. Angiogenic induction and cell migration in an orthopaedically expanded maxillary suture in the rat. *Arch Oral Biol*. 1996;41(10):985–994.
109. Roberts WE, Wood HB, Chambers DW, Burk DT. Vascularly oriented differentiation gradient of osteoblast precursor cells in rat periodontal ligament: implications for osteoblast histogenesis and periodontal bone loss. *J Periodontal Res*. 1987;22(6):461–467.
110. Doherty MJ, Ashton BA, Walsh S, Beresford JN, Grant ME, Canfield AE. Vascular pericytes express osteogenic potential in vitro and in vivo. *J Bone Miner Res Off J Am Soc Bone Miner Res*. 1998;13(5):828–838.
111. Parr JA, Garetto LP, Wohlford ME, Arbuckle GR, Roberts WE. Sutural expansion using rigidly integrated endosseous implants: an experimental study in rabbits. *Angle Orthod*. 1997;67(4):283–290.
112. Sim Y. *Cell Kinetics of Osteoblast Histogenesis in Evolving Rabbit Secondary Haversian Systems Using a Double Labeling Technique with ^{3}H-Thymidine and Bromodeoxyuridine*; 1995. (PhD thesis).
113. Baccetti T, McGill JS, Franchi L, McNamara Jr JA, Tollaro I. Skeletal effects of early treatment of Class III malocclusion with maxillary expansion and face-mask therapy. *Am J Orthod Dentofac Orthop off Publ Am Assoc Orthod Its Const Soc Am Board Orthod*. 1998;113(3):333–343.
114. Gautam P, Valiathan A, Adhikari R. Maxillary protraction with and without maxillary expansion: a finite element analysis of sutural stresses. *Am J Orthod Dentofac Orthop Off Publ Am Assoc Orthod Its Const Soc Am Board Orthod*. 2009;136(3):361–366.
115. Suda N, Ishii-Suzuki M, Hirose K, Hiyama S, Suzuki S, Kuroda T. Effective treatment plan for maxillary protraction: is the bone age useful to determine the treatment plan? *Am J Orthod Dentofac Orthop Off Publ Am Assoc Orthod Its Const Soc Am Board Orthod*. 2000;118(1):55–62.
116. Heymann GC, Cevidanes L, Cornelis M, De Clerck HJ, Tulloch JF. Three-dimensional analysis of maxillary protraction with intermaxillary elastics to miniplates. *Am J Orthod Dentofac Orthop Off Publ Am Assoc Orthod Its Const Soc Am Board Orthod*. 2010;137(2):274–284.
117. Nguyen T, Cevidanes L, Cornelis MA, Heymann G, de Paula LK, De Clerck H. Three-dimensional assessment of maxillary changes associated with bone anchored maxillary protraction. *Am J Orthod Dentofac Orthop*. 2011;140(6):790–798.
118. Liou EJ, Huang CS. Rapid canine retraction through distraction of the periodontal ligament. *Am J Orthod Dentofac Orthop Off Publ Am Assoc Orthod Its Const Soc Am Board Orthod*. 1998;114(4):372–382.
119. Cope JB, Samchukov ML, Cherkashin AM. Mandibular distraction osteogenesis: a historic perspective and future directions. *Am J Orthod Dentofac Orthop Off Publ Am Assoc Orthod Its Const Soc Am Board Orthod*. 1999;115(4):448–460.
120. McCarthy JG, Stelnicki EJ, Grayson BH. Distraction osteogenesis of the mandible: a ten-year experience. *Semin Orthod*. 1999;5(1):3–8.
121. Peltomaki T. Stability, adaptation and growth following distraction osteogenesis in the craniofacial region. *Orthod Craniofacial Res*. 2009;12(3):187–194.
122. Gursoy S, Hukki J, Hurmerinta K. Five-year follow-up of maxillary distraction osteogenesis on the dentofacial structures of children with cleft lip and palate. *J Oral Maxillofac Surg Off J Am Assoc Oral Maxillofac Surg*. 2010;68(4):744–750.
123. Rachmiel A, Aizenbud D, Ardekian L, Peled M, Laufer D. Surgically-assisted orthopedic protraction of the maxilla in cleft lip and palate patients. *Int J Oral Maxillofac Surg*. 1999;28(1):9–14.
124. Ahn JG, Figueroa AA, Braun S, Polley JW. Biomechanical considerations in distraction of the osteotomized dentomaxillary complex. *Am J Orthod Dentofac Orthop Off Publ Am Assoc Orthod Its Const Soc Am Board Orthod*. 1999;116(3):264–270.
125. Krimmel M, Cornelius CP, Roser M, Bacher M, Reinert S. External distraction of the maxilla in patients with craniofacial dysplasia. *J Craniofacial Surg*. 2001;12(5):458–463.
126. Vachiramon A, Urata M, Kyung HM, Yamashita DD, Yen SL. Clinical applications of orthodontic microimplant anchorage in craniofacial patients. *Cleft Palate-Craniofacial J Off Publ Am Cleft Palate-Craniofacial Assoc*. 2009;46(2):136–146.
127. Liou EJ, Polley JW, Figueroa AA. Distraction osteogenesis: the effects of orthodontic tooth movement on distracted mandibular bone. *J Craniofacial Surg*. 1998;9(6):564–571.
128. Raoul G, Wojcik T, Ferri J. Outcome of mandibular symphyseal distraction osteogenesis with bone-borne devices. *J Craniofacial Surg*. 2009;20(2):488–493.
129. Reitan K, Rygh P. Biomechanical principles and reactions. In: Graber TM, Vanarsdall Jr RL, eds. *Orthodontics: Current Principles and Techniques*. St. Louis: Mosby; 1994:96–192.
130. Rygh P, Moxham BJ, Berkowitz BKB. The effects of external forces on the periodontal ligament—the response to horizontal loads. In: Berkowitz BKB, Moxham BJ, Newman HN, eds. *The Periodontal Ligament in Health and Disease*. Oxford: Pergamon Press; 1982:269–290.
131. Burstone CJ, Pryputniewicz RJ, Bowley WW. Holographic measurement of tooth mobility in three dimensions. *J Periodont Res*. 1978;13.
132. Miura F. Effect of orthodontic force on blood circulation in periodontal membrane. In: Cook JT, ed. *Transactions of the Third International Orthodontic Congress*. St. Louis: Mosby; 1975.
133. Roberts WE, Goodwin Jr WC, Heiner SR. Cellular response to orthodontic force. *Dent Clin N Am*. 1981;25(1):3–17.

134. Carter DR, Smith DJ, Spengler DM, Daly CH, Frankel VH. Measurement and analysis of in vivo bone strains on the canine radius and ulna. *J Biomech*. 1980;13(1):27–38.
135. Davidovitch Z. Tooth movement. *Crit Rev oral Biol Med Off Publ Am Assoc Oral Biol*. 1991;2(4):411–450.
136. Kimura R, Anan H, Matsumoto A, Noda D, Maeda K. Dental root resorption and repair: histology and histometry during physiological drift of rat molars. *J Periodontal Res*. 2003;38(5):525–532.
137. Brudvik P, Rygh P. The repair of orthodontic root resorption: an ultrastructural study. *Eur J Orthod*. 1995;17(3):189–198.
138. Huang XF, Zhao YB, Zhang FM, Han PY. Comparative study of gene expression during tooth eruption and orthodontic tooth movement in mice. *Oral Dis*. 2009;15(8):573–579.
139. Yamaguchi M, Hayashi M, Fujita S, Yoshida T, Utsunomiya T, Yamamoto H, et al. Low-energy laser irradiation facilitates the velocity of tooth movement and the expressions of matrix metalloproteinase-9, cathepsin K, and alpha(v) beta(3) integrin in rats. *Eur J Orthod*. 2010;32(2):131–139.
140. Deguchi T, Takano-Yamamoto T, Yabuuchi T, Ando R, Roberts WE, Garetto LP. Histomorphometric evaluation of alveolar bone turnover between the maxilla and the mandible during experimental tooth movement in dogs. *Am J Orthod Dentofac Orthop Off Publ Am Assoc Orthod Its Const Soc Am Board Orthod*. 2008;133(6):889–897.
141. Burstone CJ. Application of bioengineering to clinical orthodontics. In: Graber TM, Vanarsdall RL, eds. *Orthodontics: Current Principles and Techniques*. 3rd ed. St. Louis: Mosby; 2000.
142. Manhartsberger C, Morton JY, Burstone CJ. Space closure in adult patients using the segmented arch technique. *Angle Orthod*. 1989;59(3):205–210.
143. Yoo SK, Warita H, Soma K. Duration of orthodontic force affecting initial response of nitric oxide synthase in rat periodontal ligaments. *J Med Dent Sci*. 2004;51(1):83–88.
144. Roberts WE, Arbuckle GR, Analoui M. Rate of mesial translation of mandibular molars using implant-anchored mechanics. *Angle Orthod*. 1996;66(5):331–338.
145. Rodan GA. Perspectives: mechanical loading, estrogen deficiency, and the coupling of bone formation to bone resorption. *J Bone Miner Res Off J Am Soc Bone Miner Res*. 1991;6(6):527–530.
146. Thompson DD, Rodan GA. Indomethacin inhibition of tenotomy-induced bone resorption in rats. *J Bone Miner Res Off J Am Soc Bone Miner Res*. 1988;3(4):409–414.
147. Tucker A, Sharpe P. The cutting-edge of mammalian development; how the embryo makes teeth. *Nat Rev Genet*. 2004;5(7):499–508.
148. Bosshardt DD, Schroeder HE. How repair cementum becomes attached to the resorbed roots of human permanent teeth. *Acta Anat*. 1994;150(4):253–266.
149. Smith RK, Roberts WE. Cell kinetics of the initial response to orthodontically induced osteogenesis in rat molar periodontal ligament. *Calcif Tissue Int*. 1980;30(1):51–56.
150. Yee JA. Response of periodontal ligament cells to orthodontic force: ultrastructural identification of proliferating fibroblasts. *Anat Rec*. 1979;194(4):603–614.
151. King GJ, Thiems S. Chemical mediation of bone resorption induced by tooth movement in the rat. *Arch Oral Biol*. 1979;24(10–11):811–815.
152. Yee JA, Kimmel DB, Jee WS. Periodontal ligament cell kinetics following orthodontic tooth movement. *Cell Tissue Kinet*. 1976;9(3):293–302.
153. Rodan GA, Martin TJ. Role of osteoblasts in hormonal control of bone resorption–a hypothesis. *Calcif Tissue Int*. 1981;33(4):349–351.
154. Roberts WE, Morey ER. Proliferation and differentiation sequence of osteoblast histogenesis under physiological conditions in rat periodontal ligament. *Am J Anat*. 1985;174(2):105–118.
155. Chambers TJ, McSheehy PM, Thomson BM, Fuller K. The effect of calcium-regulating hormones and prostaglandins on bone resorption by osteoclasts disaggregated from neonatal rabbit bones. *Endocrinology*. 1985;116(1):234–239.
156. Holtrop ME, Raisz LG, Simmons HA. The effects of parathyroid hormone, colchicine, and calcitonin on the ultrastructure and the activity of osteoclasts in organ culture. *J Cell Biol*. 1974;60(2):346–355.
157. Roberts WE. Cell population dynamics of periodontal ligament stimulated with parathyroid extract. *Am J Anat*. 1975;143(3):363–370.
158. Roberts WE. Cell kinetic nature and diurnal periodicity of the rat periodontal ligament. *Arch Oral Biol*. 1975;20(7):465–471.
159. Sims NA, Walsh NC. Intercellular cross-talk among bone cells: new factors and pathways. *Curr Osteoporos Rep*. 2012;10(2):109–117.
160. Zhao N, Lin J, Kanzaki H, Ni J, Chen Z, Liang W, et al. Local osteoprotegerin gene transfer inhibits relapse of orthodontic tooth movement. *Am J Orthod Dentofac Orthop*. 2012;141(1):30–40.
161. Roberts WE, Klingler E, Mozsary PG. Circadian rhythm of mechanically mediated differentiation of osteoblasts. *Calcif Tissue Int*. 1984;36(suppl 1):S62–S66.
162. Roberts WE, Aubert MM, Sparaga JM, Smith RK. Circadian periodicity of the cell kinetics of rat molar periodontal ligament. *Am J Orthod*. 1979;76(3):316–323.
163. Atkinson SR. Balance: the magic word. *Am J Orthod Dentofac Orthop Off Publ Am Assoc Orthod Its Const Soc Am Board Orthod*. 1964.
164. Parfitt AM. Bone remodeling: relationship to the amount and structure of bone, and the pathogenesis and prevention of fractures. In: Riggs BL, Melton III LJ, eds. *Osteoporosis: Etiology, Diagnosis, and Management*. New York: Raven Press; 1988:45–93.
165. Sanjideh PA, Rossouw PE, Campbell PM, Opperman LA, Buschang PH. Tooth movements in foxhounds after one or two alveolar corticotomies. *Eur J Orthod*. 2010;32(1):106–113.
166. Jang I, Tanaka M, Koga Y, Iijima S, Yozgatian JH, Cha BK, et al. A novel method for the assessment of three-dimensional tooth movement during orthodontic treatment. *Angle Orthod*. 2009;79(3):447–453.
167. Goodacre CJ, Brown DT, Roberts WE, Jeiroudi MT. Prosthodontic considerations when using implants for orthodontic anchorage. *J Prosthet Dent*. 1997;77(2):162–170.
168. Helm FR, et al. Bone remodeling response to loading of rigid endosseous implants. *J Dent Res*. 1987.
169. Albrektsson T, Jacobsson M. Bone-metal interface in osseointegration. *J Prosthet Dent*. 1987;57(5):597–607.
170. Branemark PI. Osseointegration and its experimental background. *J Prosthet Dent*. 1983;50(3):399–410.
171. Cope JB, Owens SE. Historical development and evolution of temporary anchorage devices. In: Cope JB, ed. *OrthoTADs: The Clinical Guide and Atlas. 1*. Dallas, Texas: Under Dog Media; 2007:3–20.
172. Papadopoulos MA, Papageorgiou SN, Zogakis IP. Clinical effectiveness of orthodontic miniscrew implants: a meta-analysis. *J Dent Res*. 2011;90(8):969–976.
173. Papageorgiou SN, Zogakis IP, Papadopoulos MA. Failure rates and associated risk factors of orthodontic miniscrew implants: a meta-analysis. *Am J Orthod Dentofac Orthop*. 2012;142(5):577–595 e7.
174. Umemori M, Sugawara J, Mitani H, Nagasaka H, Kawamura H. Skeletal anchorage system for open-bite correction. *Am J Orthod Dentofac Orthop*. 1999;115(2):166–174.
175. Wilmes B, Nienkemper M, Ludwig B, Nanda R, Drescher D. Upper-molar intrusion using anterior palatal anchorage and the Mousetrap appliance. *J Clin Orthod*. 2013;47(5):314–320, quiz 28.
176. Aldikacti M, Acikgoz G, Turk T, Trisi P. Long-term evaluation of sand-blasted and acid-etched implants used as orthodontic anchors in dogs. *Am J Orthod Dentofac Orthop Off Publ Am Assoc Orthod Its Const Soc Am Board Orthod*. 2004;125(2):139–147.
177. Schatzle M, Mannchen R, Zwahlen M, Lang NP. Survival and failure rates of orthodontic temporary anchorage devices: a systematic review. *Clin Oral Implants Res*. 2009;20(12):1351–1359.
178. Alves Jr M, Baratieri C, Nojima LI. Assessment of mini-implant displacement using cone beam computed tomography. *Clin Oral Implants Res*. 2011;22(10):1151–1156.
179. Branemark P-I. Introduction to osseointegration. In: Branemark P-I, Zarb GA, Albrektsson T, eds. *Tissue-Integrated Prostheses: Osseointegration in Clinical Dentistry*. Chicago, IL: Quintessence Publishing Co.; 1986:11–76.
180. Roberts EW, Poon LC, Smith RK. Interface histology of rigid endosseous implants. *J Oral Implantol*. 1986;12(3):406–416.
181. Mardas N, Dereka X, Donos N, Dard M. Experimental model for bone regeneration in oral and cranio-maxillo-facial surgery. *J Invest Surg*. 2014;27(1):32–49.

182. Kieswetter K, Schwartz Z, Dean DD, Boyan BD. The role of implant surface characteristics in the healing of bone. *Crit Rev Oral Biol Med Off Publ Am Assoc Oral Biol*. 1996;7(4):329–345.
183. Garetto LP, Chen J, Parr JA, Roberts WE. Remodeling dynamics of bone supporting rigidly fixed titanium implants: a histomorphometric comparison in four species including humans. *Implant Dent*. 1995;4:235–243.
184. Lemons JE. Retrieval and analysis of explanted and in situ implants including bone grafts. *Oral Maxillofac Surg Clin N Am*. 2010;22(3):419–423, vii.
185. Huja SS. Mechanisms of bone adaptation to orthodontic anchors. In: Cope JB, ed. *Temporary Anchorage Devices in Orthodontics*. Dallas, TX: Under Dog Media; 2005:21–33.
186. Vandeweghe S, Coelho PG, Vanhove C, Wennerberg A, Jimbo R. Utilizing micro-computed tomography to evaluate bone structure surrounding dental implants: a comparison with histomorphometry. *J Biomed Mater Res B Appl Biomater*. 2013;101(7):1259–1266.
187. Liou EJ, Pai BC, Lin JC. Do miniscrews remain stationary under orthodontic forces? *Am J Orthod Dentofac Orthop*. 2004;126(1):42–47.
188. Pittman JW, Navalgund A, Byun SH, Huang H, Kim AH, Kim DG. Primary migration of a mini-implant under a functional orthodontic loading. *Clin Oral Investig*. 2014;18(3):721–728.
189. Kuroda S, Yamada K, Deguchi T, Hashimoto T, Kyung HM, Takano-Yamamoto T. Root proximity is a major factor for screw failure in orthodontic anchorage. *Am J Orthod Dentofac Orthop*. 2007;131 (4 suppl):S68–S73.
190. Liu ZJ, Herring SW. Bone surface strains and internal bony pressures at the jaw joint of the miniature pig during masticatory muscle contraction. *Arch Oral Biol*. 2000;45(2):95–112.
191. El-Beialy AR, Abou-El-Ezz AM, Attia KH, El-Bialy AM, Mostafa YA. Loss of anchorage of miniscrews: a 3-dimensional assessment. *Am J Orthod Dentofac Orthop Off Publ Am Assoc Orthod Its Const Soc Am Board Orthod*. 2009;136(5):700–707.
192. Chaddad K, Ferreira AF, Geurs N, Reddy MS. Influence of surface characteristics on survival rates of mini-implants. *Angle Orthod*. 2008;78(1):107–113.
193. Kim SH, Choi JH, Chung KR, Nelson G. Do sand blasted with large grit and acid etched surface treated mini-implants remain stationary under orthodontic forces? *Angle Orthod*. 2012;82(2):304–312.
194. Kanomi R. Mini-implant for orthodontic anchorage. *J Clin Orthod*. 1997;31:763–767.
195. Shirck JM, Firestone AR, Beck FM, Vig KW, Huja SS. Temporary anchorage device utilization: comparison of usage in orthodontic programs and private practice. *Orthod (Chic)*. 2011;12(3):222–231.
196. Schnelle MA, Beck FM, Jaynes RM, Huja SS. A radiographic evaluation of the availability of bone for placement of miniscrews. *Angle Orthod*. 2004;74(6):832–837.
197. Russell WMS, Burch RL. *The Principles of Humane Experimental Technique*. London: Methuen; 1959.
198. Huja SS. Bone anchors—can you hitch up your wagon? *Orthod Craniofac Res*. April 2015;18(suppl 1):109–116. http://dx.doi.org/10.1111/ocr.12082.
199. Melsen B, Huja SS, Chien HH, Dalstra M. Alveolar bone preservation subsequent to miniscrew implant placement in a canine model. *Orthod Craniofacial Res*. 2015;18:77–85.
200. Graham JW. Temporary replacement of maxillary lateral incisors with miniscrews and bonded pontics. *J Clin Orthod*. 2007;41(6):321–325.
201. Meta IF, Fernandez SA, Gulati P, Huja SS. Alveolar process anabolic activity in C3H/HeJ and C57BL/6J inbred mice. *J Periodontol*. 2008;79(7):1255–1262.
202. Bentolila V, Boyce TM, Fyhrie DP, Drumb R, Skerry TM, Schaffler MB. Intracortical remodeling in adult rat long bones after fatigue loading. *Bone*. 1998;23(3):275–281.
203. Teixeira CC, Khoo E, Tran J, Chartres I, Liu Y, Thant LM, et al. Cytokine expression and accelerated tooth movement. *J Dent Res*. 2010;89(10):1135–1141.
204. Komori T. Mouse models for the evaluation of osteocyte functions. *J Bone Metab*. 2014;21(1):55–60.
205. Huja SS, Fernandez SA, Phillips C, Li Y. Zoledronic acid decreases bone formation without causing osteocyte death in mice. *Arch Oral Biol*. 2009;54(9):851–856.
206. Parr JA, Garetto LP, Wohlford ME, Arbuckle GR, Roberts WE. Implant-borne suture expansion in rabbits: a histomorphometric study of the supporting bone. *J Biomed Mater Res*. 1999;45(1):1–10.
207. Huja SS, Rummel AM, Beck FM. Changes in mechanical properties of bone within the mandibular condyle with age. *J Morphol*. 2008;269(2): 138–143.
208. Allen MR, Burr DB. Mandible matrix necrosis in beagle dogs after 3 years of daily oral bisphosphonate treatment. *J Oral Maxillofac Surg Off J Am Assoc Oral Maxillofac Surg*. 2008;66(5):987–994.
209. Allen MR, Burr D. Mineralization, microdamage, and matrix: how bisphosphonates influence material properties of bone. *Int Bone Miner Soc Knowl Environ*. 2007;4(2):49–60.
210. Fleming PS. Accelerating orthodontic tooth movement using surgical and non-surgical approaches. *Evid Based Dent*. 2014;15(4):114–115.
211. Gkantidis N, Mistakidis I, Kouskoura T, Pandis N. Effectiveness of non-conventional methods for accelerated orthodontic tooth movement: a systematic review and meta-analysis. *J Dent*. 2014; 42(10):1300–1319.
212. Ren A, Lv T, Kang N, Zhao B, Chen Y, Bai D. Rapid orthodontic tooth movement aided by alveolar surgery in beagles. *Am J Orthod Dentofac Orthop Off Publ Am Assoc Orthod Its Const Soc Am Board Orthod*. 2007;131(2):160.e1–160.e10.
213. Sebaoun JD, Kantarci A, Turner JW, Carvalho RS, Van Dyke TE, Ferguson DJ. Modeling of trabecular bone and lamina dura following selective alveolar decortication in rats. *J Periodont*. 2008;79(9):1679–1688.
214. Wilcko MT, Wilcko JM, Bissada NF. An evidence-based analysis of periodontally accelerated orthodontic and osteogenic techniques: a synthesis of scientific perspectives. *Semin Orthod*. 2008;14:305–316.
215. Canalis E. In: Favus MJ, ed. *Regulation of Bone Remodeling*. Kelseyville, CA: American Society for Bone and Mineral Research; 1994.
216. Mueller M, Schilling T, Minne HW, Ziegler R. A systemic acceleratory phenomenon (SAP) accompanies the regional acceleratory phenomenon (RAP) during healing of a bone defect in the rat. *J Bone Miner Res Off J Am Soc Bone Miner Res*. 1991;6(4):401–410.
217. Bogoch E, Gschwend N, Rahn B, Moran E, Perren S. Healing of cancellous bone osteotomy in rabbits–Part I: regulation of bone volume and the regional acceleratory phenomenon in normal bone. *J Orthop Res*. 1993;11(2):285–291.
218. Tricker ND, Dixon RB, Garetto LP. Cortical bone turnover and mineral apposition in dentate bone mandible. In: Garetto LP, Turner CH, Duncan RL, Burr DB, eds. *Bridging the Gap Between Dental and Orthopaedic Implants*. Indianapolis, IN: School of Dentistry Indiana University; 2002:226–227.
219. Garetto LP, Tricker ND. Remodeling of bone surrounding at the implant interface. In: Garetto LP, Turner CH, Duncan RL, Burr DB, eds. *Bridging the Gap Between Dental and Orthopaedic Implants*. Indianapolis, IN: School of Dentistry Indiana University; 2002:89–100.
220. Baloul SS, Gerstenfeld LC, Morgan EF, Carvalho RS, Van Dyke TE, Kantarci A. Mechanism of action and morphologic changes in the alveolar bone in response to selective alveolar decortication-facilitated tooth movement. *Am J Orthod Dentofac Orthop*. 2011;139(4 suppl): S83–S101.
221. Dibart S, Yee C, Surmenian J, Sebaoun JD, Baloul S, Goguet-Surmenian E, et al. Tissue response during piezocision-assisted tooth movement: a histological study in rats. *Eur J Orthod*. 2013.
222. Dibart S, Yee C, Surmenian J, Sebaoun JD, Baloul S, Goguet-Surmenian E, et al. Tissue response during piezocision-assisted tooth movement: a histological study in rats. *Eur J Orthod*. 2014;36(4):457–464.
223. Mostafa YA, Fayed MMS, Mehanni S, ElBokle NN, Heider AM. Comparison of corticotomy-facilitated vs standard tooth-movement techniques in dogs with miniscrews as anchor units. *Am J Orthod Dentofac Orthop Off Publ Am Assoc Orthod Its Const Soc Am Board Orthod*. 2009;136(4):570–577.
224. Kim YS, Kim SJ, Yoon HJ, Lee PJ, Moon W, Park YG. Effect of piezopuncture on tooth movement and bone remodeling in dogs. *Am J Orthod Dentofac Orthop*. 2013;144(1):23–31.

5

Application of Bioengineering to Clinical Orthodontics

Charles J. Burstone

OUTLINE

Sign Conventions, 155
Biomechanics of Tooth Movement, 155
Centers of Rotation, 156
Bracket Path and the Required Force System, 158
Force Magnitude and the Rate of Tooth Movement, 159
Relationship of Force Magnitude to Pain and Tooth Mobility, 162
Optimal Force and Stress, 163
The Orthodontic Appliance, 164
Active and Reactive Members, 164
Moment-to-Force Ratio, 164
Load-Deflection Rate, 164
Maximal Elastic Moment, 164
Manner of Loading, 165
Mechanical Properties of Metals, 166
Basic Behavior of Alloys, 166
Elastic Limit, 167
Modulus of Elasticity, 167
Shape-Memory Alloys, 167
Ideal Orthodontic Alloys, 168
Wire Cross Section, 168
Selection of the Proper Wire (Alloy and Cross Section), 169
Wire Length, 173
Amount of Wire, 173
Stress Raisers, 174
Sections of Maximal Stress, 175
Direction of Loading, 175
Attachment, 176
Forces from a Continuous Arch, 177
Principles of Spring Design, 179
Fiber-Reinforced Composite Applications, 182
The Role of Friction, 182
3D Biomechanics, 183
Summary, 184

An important aspect of the scientific revolution that has taken place in biology over the past decade is the broad application of the physical sciences to living tissues. Physics, engineering, and mathematics can be applied similarly, with great profit to the field of orthodontics. Of all the possible applications, this chapter concerns itself with only one, the biophysics of the orthodontic appliance. The chapter covers whether theoretic mechanics can help in the design and clinical manipulation of an orthodontic appliance.

Theoretic mechanics offers potential benefit in orthodontics in three primary ways:

- Basic principles of engineering and physics can point the way toward improved design of orthodontic appliances. If the orthodontic specialty relies only on trial-and-error procedures for the development of new appliances, the developmental horizons become sharply limited. Empiricism in orthodontic design must give way to a new discipline of orderly appliance development using concepts from the physical sciences. In a less ambitious way, theoretic mechanics may help in the design of a new appliance by enabling clinicians to apply knowledge that has been gained from some of the older appliances. For example, if an existing appliance works well for a given type of tooth movement, clinicians can use the force system developed by that appliance as a basis for designing a new one. Although the trial-and-error method has proven valuable in the past, presumably the application of biophysical principles will give rise to a more orderly development of orthodontic appliances.
- Study of the biophysics of tooth movement can yield important information. If researchers and clinicians can quantify the force systems applied to teeth, they can understand clinical, tissue, and cellular responses better. To make valid judgments about the response of teeth to orthodontic forces, clinicians first must define fully the force system acting on the tooth. Theoretic mechanics also can be helpful in formulating useful concepts of stress distribution in the periodontal ligament (PDL) as it relates to bone remodeling.
- A knowledge of physics can enable better treatment results. Each time clinicians adjust an archwire or any other orthodontic appliance, they make certain assumptions about the relationship between the appliance and the biology of tooth movement. As these assumptions about the relationship between forces and tooth movement come closer to reality, the quality of orthodontic treatment will improve. For instance, many of the undesirable side effects that occur during orthodontic treatment can be attributed directly to a lack of understanding of the physics involved in a given adjustment. Many variables in orthodontic treatment cannot be controlled fully, such as growth and tissue response to appliances; however, the force placed on the tooth is a

controllable variable. In a sense, clinicians have the duty to understand the physics of these forces so they can better control the one variable they are in a position to affect so strongly.[1,2]

SIGN CONVENTIONS

A universal sign convention is available for forces and moments in dentistry and orthodontics. The convention is as follows: Anterior forces are positive (+), and posterior forces are negative (−); lateral forces are positive (+), and medial forces are negative (−) (Fig. 5-1). Forces acting in a mesial direction are positive (+), and forces acting in a distal direction are negative (−). Buccal forces are positive (+), and lingual forces are negative (−) (Fig. 5-2).

Moments (couples) that tend to produce mesial, buccal, or labial crown movement are positive (+), and moments that tend to produce distal or lingual crown movement are negative (−) (see Fig. 5-2 and *B, C*). The same convention is used for groups of teeth (a segment or an entire arch) and for establishing signs for orthopedic effects on the maxilla and mandible. However; any sign convention is acceptable, such as the right hand rule of thumb, if it is made clear to the reader by diagram.

BIOMECHANICS OF TOOTH MOVEMENT

From a clinical viewpoint, two major problems related to tooth movement can be considered: (1) the type of force system required to produce a given center of rotation and (2) the optimal force magnitudes for tooth movement. Solving these problems requires a thorough understanding of the forces and moments that may act on the teeth and also detailed documentation of tooth movement and the response in the PDL.

The forces delivered by an orthodontic appliance can be determined by direct measurement with suitable instruments or, partly, by mathematical calculation.[3-6] The load-deflection rates of orthodontic springs or wires can be measured with electronic strain gauges or mechanical gauges. Most orthodontic appliances deliver a complicated set of forces and moments. In clinical studies, therefore, appliances of simple construction, in which forces are determined more easily and accurately, are useful. For the same reason, a clinical study in which force variables are controlled is likely to yield more information than are data taken from patients in a routine orthodontic practice.

The problems inherent in studying the response of a tooth subjected to a force system are much more complex and difficult to solve than those of simple measurement of the forces. Observations can be made on three levels to describe the response of a tooth to forces: the clinical level, the cellular and biochemical levels, and the stress–strain level. The clinical level allows the study of phenomena such as the rate of tooth movement, pain response, tooth mobility, alveolar bone loss, and root resorption. The cellular and biochemical levels give insight into the dynamics of bone and connective tissue changes in the PDL.[7,8]

Perhaps the most important and least understood level is the stress–strain level of activity in the PDL. The ability to determine accurately the level of stress (the force per unit area) in different areas of the PDL may well offer the best means of correlating the application of force on a tooth with the response of the tooth. Currently, to place strain gauges in

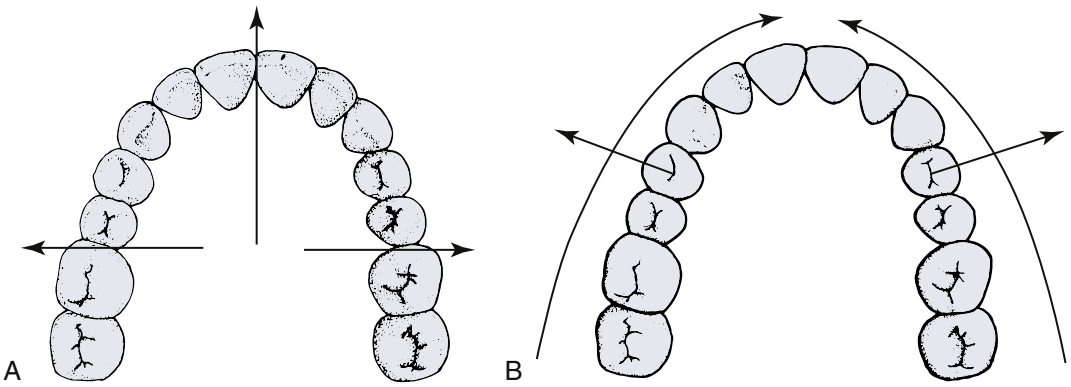


FIGURE 5-1 Sign conventions. **A,** Lateral and anterior forces are positive. **B,** Buccal, labial, and mesial forces are positive. (From Burstone CJ, Koenig HA. *Am J Orthod.* 1974;65:270. With permission from the American Association of Orthodontists.)

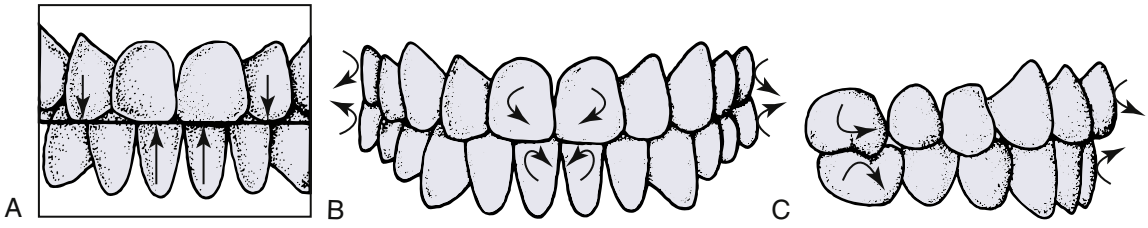


FIGURE 5-2 Sign conventions. **A,** Extrusive forces are positive. **B, C,** Moments (couples) that tend to move crowns in a mesial, buccal, or labial direction are positive. (From Burstone CJ, Koenig HA. *Am J Orthod.* 1974;65:270. With permission from the American Association of Orthodontists.)

the PDL to measure stress distributions is impossible; therefore, knowledge of stress phenomena must depend on another approach. For example, a mathematic model of the tooth and surrounding structures can be constructed based on certain assumptions, and theoretic stress levels can be calculated from these models if the forces applied to the teeth are known. Unfortunately, these mathematic models are no better than the assumptions on which they are based. All such calculations, therefore, should be verified by clinical or animal experimentation whenever possible.

Centers of Rotation

Tooth movement often is described in general terms: tipping, bodily movement, and root movement. More specific descriptions can be devised by locating a center of rotation relative to three mutually perpendicular planes, which are (1) a buccolingual or labiolingual plane oriented through the long axis of the tooth, (2) a mesiodistal plane also oriented through the long axis of the tooth, and (3) a transverse plane that intersects the buccolingual or labiolingual and mesiodistal planes at right angles. To define fully the changes in the position of a tooth, one must use all three planes of reference. For simplicity's sake, the following discussion considers two-dimensional representations of teeth, and therefore only one plane of space is described. If certain simple assumptions are made about stress distributions (e.g., a uniformly varying distribution for pure rotation and a uniform distribution for pure translation), if a linear stress–strain relationship is postulated, and if axial loading is ignored, to mathematically predict the force system required for various centers of rotation is possible. These calculations will err because the realities of tooth and PDL differ from idealized assumptions. A typical central incisor is used to represent the application of this theory to centers of rotation. For purposes of presentation, actual force values are not used.

As a tooth translates (bodily movement), a relatively uniform stress distribution along the root is found. In terms of a center of rotation, the center of rotation for translation is at infinity (Fig. 5-3, *A*). A single force acting through the center of resistance of a root affects pure translation of a tooth. The center of resistance in a single-rooted tooth with a parabolic shape is calculated by multiplying the distance from the alveolar crest to the apex by 0.33. The center of resistance coincides with the centroid, which in this case is the geometric center of the part of the root between the alveolar crest and the apex. A force placed near the center of the root therefore should produce pure translation.[9,10]

If a pure moment (a couple) is placed anywhere on a tooth, a center of rotation is created near the center of resistance of the tooth. In Figure 5-3, *B*, the clockwise moment tends to displace the crown in a lingual direction and the root in a labial direction, with a center of rotation near the middle of the root. Unlike pure translation, pure rotation does not produce a uniform stress distribution in the PDL but rather a uniformly varying distribution, with the highest stress at the apex and the next highest at the alveolar crest. No stress is found at the center of resistance. The center of rotation is located at the level of the root where the stress is zero.

Pure translation (center of rotation at infinity) and pure rotation (center of rotation near the center of resistance) can be considered the two basic types of tooth displacements. Other centers are a combination of pure rotation and pure translation; that is, any center of rotation can be produced by combining a single force through the center of resistance of a root and a pure moment if the proper force-to-couple ratio is used.[11]

In Figure 5-4, *A*, a lingual force is acting through the center of resistance of the tooth. If a couple is added in a clockwise direction, the center of rotation moves from infinity toward the center of resistance. If the magnitude of the couple is small (relative to the force at the center of resistance), the center of rotation lies at the apex of the root. As the magnitude of the couple increases, the center of rotation shifts from the apex toward the center of resistance; finally, as the couple continues to increase, the center approaches the center of resistance of the tooth. The relative magnitude of the couple and force acting through the center of resistance determines the specific type of lingual tipping of the tooth if the direction of the moment is clockwise.

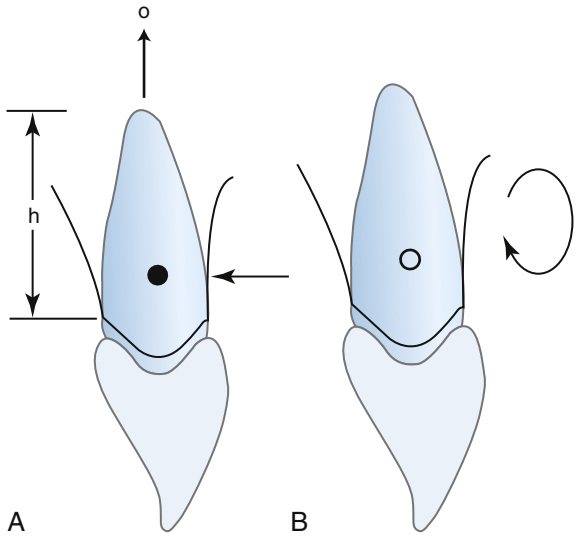


FIGURE 5-3 Basic tooth movements. **A,** A force acting through the center of resistance of a tooth produces a translation center of rotation at infinity. **B,** A couple acting on the tooth produces a center of rotation at the center of resistance.

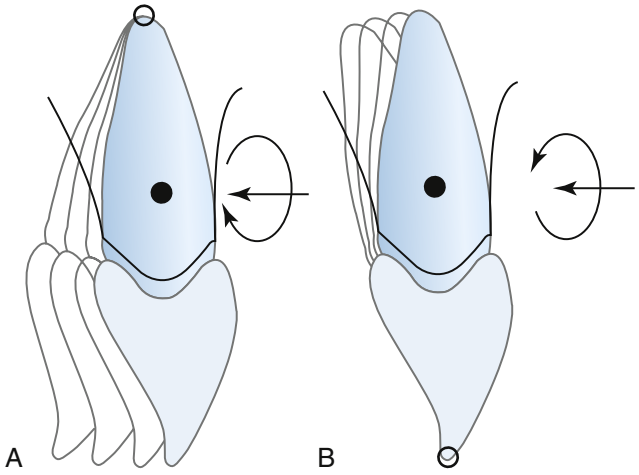


FIGURE 5-4 A couple and a force acting through the center of resistance. **A,** A negative couple produces lingual tipping of the incisor crown. **B,** A positive couple produces incisor lingual root movement.

However, a counterclockwise moment plus a force acting through the center of resistance places the center of rotation somewhere between the center of resistance and infinity (Fig. 5-4, *B*). More specifically, as the couple-to-force ratio increases, the center of rotation moves from the incisal edge to the level of the bracket and finally approaches the center of resistance. Control of the center of rotation during tooth movement then is based on two components: placement of a single force through the center of resistance of the tooth and use of a couple of proper direction and magnitude.[12]

In most instances, however, anatomic limitations in the oral cavity make placing a force through the center of resistance impractical. Therefore, a force system must be placed on the crown of the tooth (at a bracket or tube) equivalent to the required couple and force acting through the center of resistance. The two force systems are equivalent if the sums of their individual forces (F_x, F_y, and F_z) are equal and the sums of their moments around any axis of rotation are equal.

The following equivalent force systems, acting on a bracket of an incisor, are typical effects of changing the couple-to-force ratio:

- A lingually directed single force placed on the crown of a tooth produces a center of rotation somewhere between the center of resistance and the apex (Fig. 5-5, *A*).
- If a counterclockwise moment (lingual root torque) of sufficient magnitude is added, the center of rotation moves to the apex (Fig. 5-5, *B*).
- If an additional moment in the same direction is placed on the tooth, the center of rotation moves toward infinity, and at this instance the tooth is translating, or moving bodily (Fig. 5-5, *C*).
- Further increases in the magnitude of the couple place the center of rotation incisal to the crown of the tooth (Fig. 5-5, *D*), then at the incisal edge, and finally at the bracket.
- Any further increase in the moment tends to move the center of rotation toward the center of resistance.

In Figure 5-6 the moment-to-force (M/F) ratio is plotted against the center of rotation for incisor roots of 7, 10, and 15 mm. The moment and force are applied at the bracket. In all cases, the distance of the bracket is 6 mm from the alveolar crest. The direction of the force applied to the incisor is lingual (−), and the sense of the moment is lingual root torque (+). As the M/F ratio approaches zero in the three examples, the center of rotation approaches the center of resistance. As the ratio increases, the center of rotation is found at the apex of the root, at infinity, at the incisal edge, at the bracket, and at the alveolar crest. As the ratio becomes infinitely large, the center of rotation approaches the center of resistance. A slight variation in the M/F ratio can make a significant difference in the positioning of the center of rotation, except with ratios that produce centers of rotation near the center of resistance. Furthermore, the same M/F ratio produces different centers of rotation, depending on the length of the root.

Nägerl et al.[13-15] developed a general theory of tooth movement based on assumptions about three-dimensional linear elasticity. The general theory states that in any given plane, the distance from the applied force to the center of resistance (a) multiplied by the distance from the center of resistance to the center of rotation, equals a constant (s^2), which represents the distribution of the restraining forces in the PDL. As shown in Figure 5-7, regardless of where the force is applied in a given plane, s^2 remains the same. Experimental determination of s^2 offers the possibility of determining centers of rotation for given teeth with similar morphologic characteristics, which should minimize the number of recordings required experimentally.

The preceding analyses have a number of clinical implications:

1. The location of the center of rotation depends on the ratio between the moment and the force applied to the tooth (M/F ratio) and not on the absolute value of either. For example, if a single force is placed on the crown of a tooth, the center of rotation should be the same, regardless of the magnitude of the force. A light or a heavy force tends equally to move the crown in one direction and the root in the other. This is in sharp contrast to the oft-repeated idea that lighter forces do not displace root apices as much as heavy forces. This statement would be accurate only if a properly directed moment, as well as a force, were applied to the tooth in question. If the moments were identical, the tooth with the lower force would possibly have minimal root displacement.[16] Research suggests that if identical M/F ratios are delivered to a tooth, the center of rotation may not be exactly identical. Heavier single forces on the crown

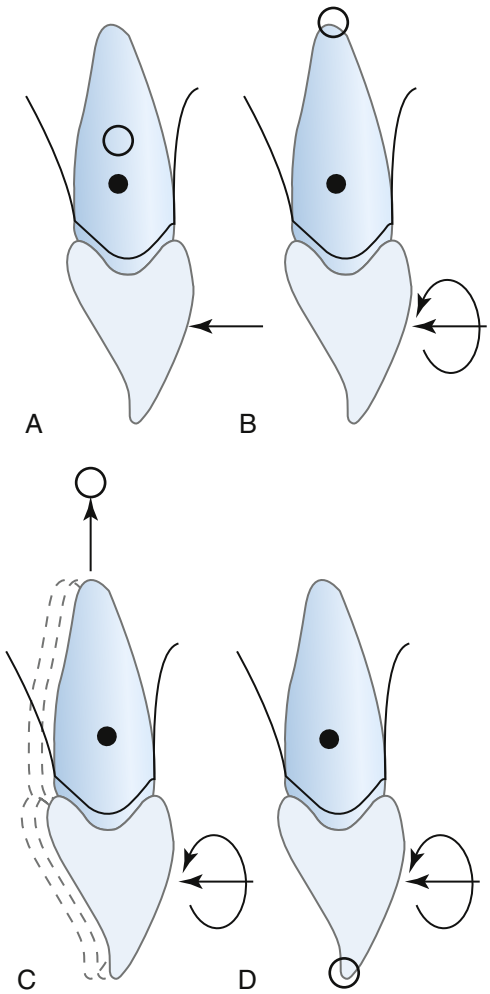


FIGURE 5-5 A single force acting on the crown of a tooth produces a center of rotation (*open circle*) slightly below the center of resistance. **A,** If increasingly larger couples are added to the force in the direction shown in **B** to **D,** the center of rotation will be found at the apex (**B**), the infinity (**C**), or the incisal edge (**D**).

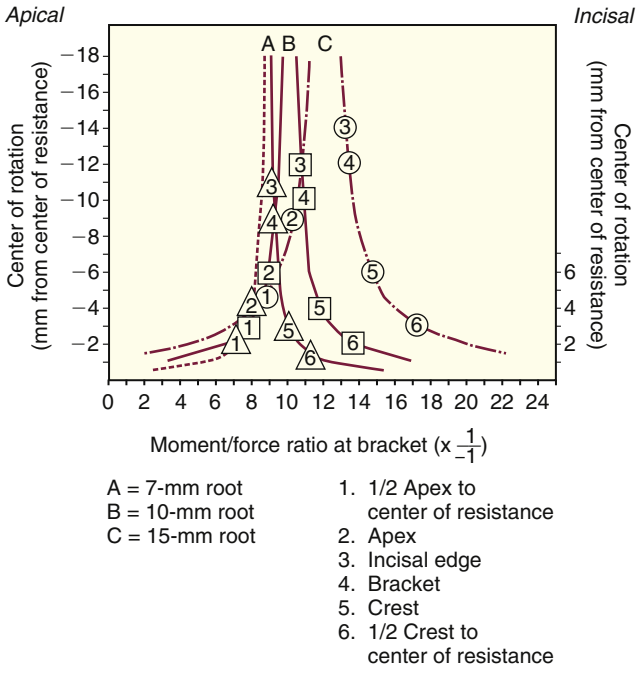


FIGURE 5-6 Moment-to-force ratio plotted against the center of rotation for three incisor root lengths. Note that identical moment/force values produce different centers of rotation if the root length varies.

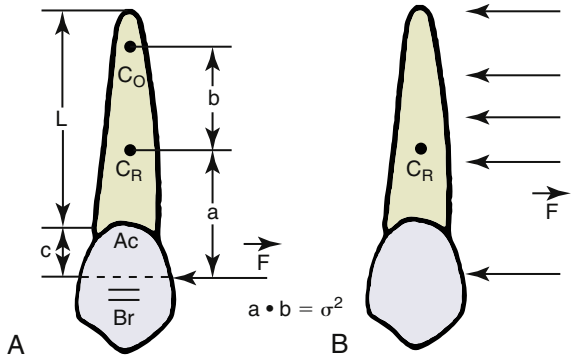


FIGURE 5-7 A, A general theory of tooth movement states that the product of the distance a times b equals s^2. B, s^2 is a constant provided the single forces are parallel and act in the same plane. Any of the individual forces shown in B produces the same s^2. (Redrawn from Nägerl H, Burstone CJ, et al. *Am J Orthod.* 1991;49:337.)

tend to move the center of rotation slightly apically rather than occlusally.

2. The M/F ratio is crucial to the establishment of a center of rotation. Small miscalculations in this ratio can change the type of tooth movement affected. Because stress distributions in the PDL are altered as well, the ease of tooth movement also may be affected.
3. The load deflection rate of the force acting on the crown and the torque deflection (angular) rate of the moment often may be different. For example, if a lingual force dissipates faster than the moment (torque), the center of rotation changes. In this case, the center of rotation shifts, rather than remaining constant, because of the change in the M/F ratio.

Laser holography is a new, noninvasive technique for studying tooth movement in closer detail.[9,10,17,18] In Figure 5-8, the location of the center of rotation is plotted against the M/F ratio for a typical maxillary central incisor. The experimental curves are similar to those derived from previously discussed theory.

In addition to analytic methodology and laser holography, numeric techniques have been used to determine centers of rotation under different loading conditions and estimate stress in the PDL. These techniques use a three-dimensional finite element method. The tooth and the alveolar process are broken down into elements (Fig. 5-9) by accurately determining the shape of the elements and the constitutive mechanical behavior of each element. Centers of rotation are determined using assumed forces on the teeth.[19-22]

As researchers become more knowledgeable, refinements in mathematic models will provide the clinician with good estimates of the M/F ratios needed to produce required centers of rotation for teeth of different geometric configurations and periodontal supports and for groups of teeth or segments.

The lack of knowledge about the variation in tooth morphology and support should not deter clinicians from making their best estimates of the relationship between the forces exerted by an appliance and the centers of rotation produced. An understanding of basic theory can guide the clinician in adjusting appliances when undesirable centers of rotation result.

Nägerl et al.[13,14] developed a concept of tooth motion that is not as limiting in three-dimensional space as the center of rotation concept. Tooth movement is envisioned as a screw oriented in a coordinate system. The screw forms an axis around which the tooth rotates, and translation may occur in or out along that axis.

Bracket Path and the Required Force System

The clinician, by tradition, has been taught to place brackets in a correct and standardized manner and then to reason what a straight wire will do when a discrepancy exists. In Figure 5-10, *A*, the flared upper incisor (brown) must be brought lingually (green). The center of rotation is the solid blue circle and the center of resistance is the open purple circle. The bracket path from the beginning to the final position is shown by the dotted gray arrows and line. The bracket translates lingually and occlusally and also rotates in a counterclockwise direction. Some orthodontists may think that the bracket path (gray arrows) shows the force and moment direction and that should be the correct force system for achieving the depicted goal. This interpretation is not correct.

In Figure 5-10, *B*, only the enlarged brackets are shown; a straight edgewise wire is inserted. What is the force system delivered to the incisor? For simplicity, let us assume the discrepancy is between two central incisors and the incisors are in the same plane. The gray arrows are in the same direction as the bracket path and unfortunately are not correct in both force and moment direction. The actual force system depends on wire-deflection properties that are better described by beam theory (complicated involving large deflections and friction). Thus, the clinician cannot just look at the bracket discrepancy and assume that a straight wire will give the proper force system.

The correct direction of the force system to tip the tooth around a point near the root apex is shown in Figure 5-10, *C*. Note that the direction of the moment should be counterclockwise (lingual root torque) and not clockwise like the bracket path. Also, the direction of force parallels the displacement of

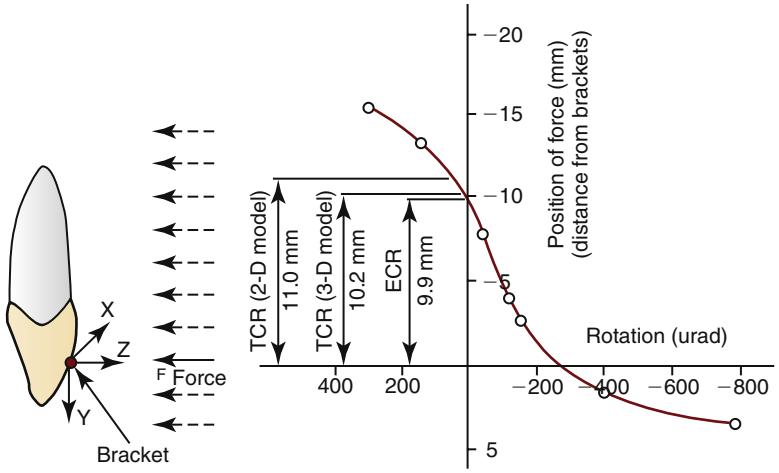


FIGURE 5-8 Center of rotation measured from the centroid of the paraboloid of revolution (h/3) as a function of the moment-to-force ratio at the bracket. The center of rotation approaches infinity as the line of action of the applied force approaches the centroid. *TCR*, Theoretic center of resistance; *ECR*, experimental center of resistance.

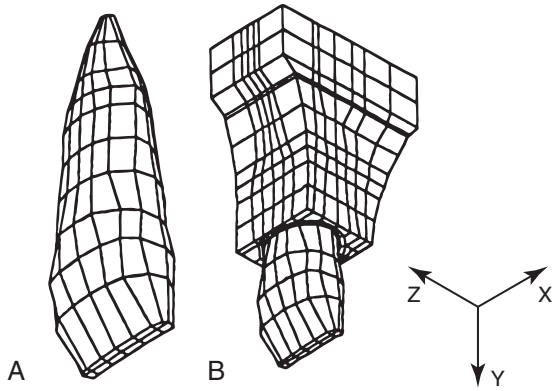


FIGURE 5-9 Three-dimensional model for tooth A and tooth-periodontal ligament-alveolar bone system B. (Redrawn from Tanne K, Koenig HA, Burstone CJ. Moment to force ratios and the center of rotation. *Am J Orthod Dentofac Orthop.* 1988;94(5)426–431.)

the purple center of resistance circles. A line connecting the center of resistance from the start to the final is a better indicator of the force direction than a line connecting the brackets (bracket path).

Since the initial force system when the archwire is placed into the bracket and the subsequent force systems when the archwire works out may be incorrect, there is no assurance that the straight wire as used here gives the best force system. On the other hand, if teeth eventually are in full or almost full alignment, the straight wire shape is correct. The straight wire works best as a final finishing arch.

Force Magnitude and the Rate of Tooth Movement

Considerable debate in orthodontics has centered on the relationship of force magnitude and the rate of tooth movement.[23-27] Attempts at correlations of this type are more likely to succeed if the rate of tooth displacement is used rather than the total displacement (absolute displacement) value. The rate of tooth movement is defined as the displacement of a tooth per unit time, and it usually is measured in millimeters per hour, per day, or per week. As the increments of time become shorter, the dynamics of tooth movement become clearer, and thus daily rather than weekly or monthly recordings are preferred.

The average daily rate can be established by dividing the absolute displacement by the number of days when measurements are made at less frequent intervals than daily. Such methods tend to smooth out rate curves and eliminate much of the rate fluctuation that can be observed each day. For practical purposes, if daily increments of movement cannot be measured (which is the case in many clinical studies), average rates of tooth movement must be used. However, when average rates are used, their limitations should always be kept in mind.

Two possible force–rate relationships can be studied. One approach relates force magnitude and tooth displacement. The other approach, which perhaps is more logical, attempts to relate stress–strain phenomena in the PDL (force per unit area and displacement per unit length) with tooth displacement. Until better experimental methods are devised, the latter method is limited by the fact that the stress–strain values must be calculated from mathematic models rather than by experimentation in the living.

The difficulty of correlating forces and tooth movement is compounded by the large number of variables that can influence the recorded rate of tooth displacement. Connective tissue forces operating through gingival and transseptal fibers or forces from the tongue, perioral musculature, and muscles of mastication may alter the force system acting on the tooth. For these reasons, the clinician should be aware of certain limitations in predicting tooth movement. If a relatively constant force is placed on a tooth, a typical type of graph is obtained when the rate of tooth movement is plotted against time (Fig. 5-11). Tooth movement can be differentiated into three phases: initial phase, lag phase, and postlag phase. The initial phase, which is characterized by a period of rapid tooth movement, normally lasts a few days. The onset of displacement immediately after application of a force and its rapidity suggest that tooth movement in the initial phase largely represents displacement of the tooth in the periodontal space. The initial phase is followed immediately by the lag phase, during which the tooth does not move or shows a relatively low rate of

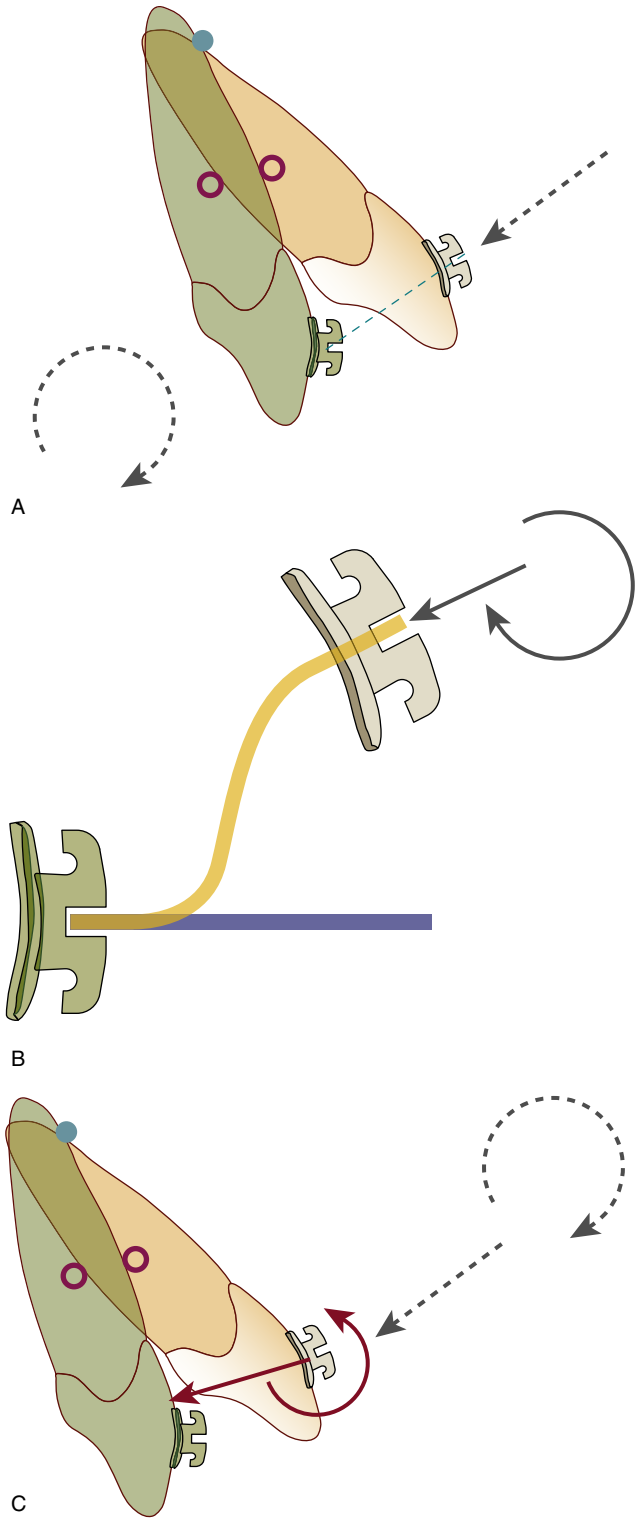


FIGURE 5-10 Flared upper incisor (*brown*) requires retraction. **A,** Center of rotation, solid blue circle. Center of resistance, purple circles. Dotted gray arrows, bracket path direction. **B,** Enlarged view of brackets only. Gray force arrows based on bracket path are not what is produced. The force system is also incorrect to achieve treatment goal. A straight wire if inserted into the bracket will deliver the wrong force system initially. **C,** Red arrows, correct force system to produce desired tooth movement. Compare to arrows based on bracket path (*gray arrows*) where moment is opposite and force direction is in error.

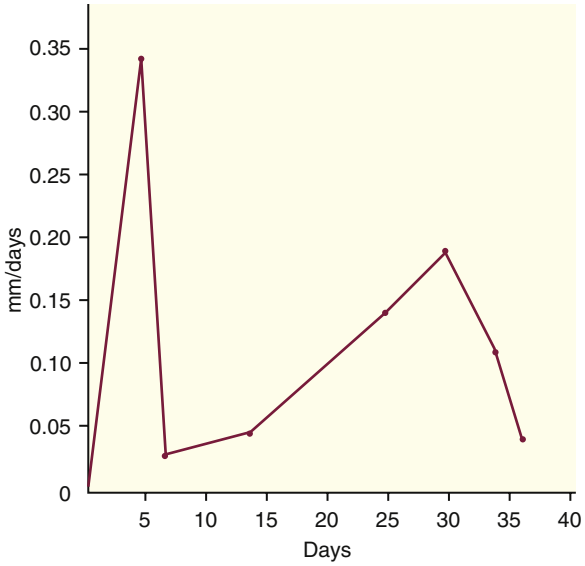


FIGURE 5-11 Typical tooth movement graph in which the rate of movement is plotted against the number of days after application of a continuous force (125 *g*). Rates are given for the reciprocal closure of a diastema between two central incisors.

displacement. A number of explanations have been postulated for the presence of a lag period. Some have suggested that the lag is caused by nonvitalization (hyalinization) of the PDL in areas of maximal stress and that no tooth movement can occur until the area of nonvitalization has been removed by cellular processes.[25,28,29] Others believe that the lag period may represent the interval required for absorption of the thicker compact bone of the lamina dura; hence the rate of tooth movement is reduced. The third phase of tooth movement, the postlag phase, occurs when the rate of tooth movement gradually or suddenly increases (see Chapters 3 and 4).

The great variation of response in tooth movement to relatively identical force systems is impressive. However, this is not surprising because the force applied to a tooth is but one of many variables that determine its displacement. If constant force were applied to a tooth, the tooth might be expected to move at a constant rate through the alveolar process. Nevertheless, clinical measurements show a lack of constancy in rate not only during the initial and lag phases but also during subsequent tooth movement.

The crucial issue in orthodontic therapy is the relationship between force magnitude and the rate of tooth movement. The most obvious and appealing relationship is a linear one, in which more force implies greater tooth movement. Nevertheless, appealing as the simplicity of this assumption might be, it does not correspond to the facts observed histologically and clinically in all phases of tooth movement.

During the first part of the initial phase, when the tooth is deflected through the periodontal space, something approaching a linear relationship may exist. As the stress levels in the PDL increase, a faster rate of tooth movement is expected. Thus, a light force may take days to move a tooth through the width of the PDL, whereas a heavy force, such as a dental separator, can accomplish the same result in seconds. The responses to light and to heavy forces show no gross difference if the absolute displacement is measured after 2 or 3 days or if the average rate is calculated for a similar period.

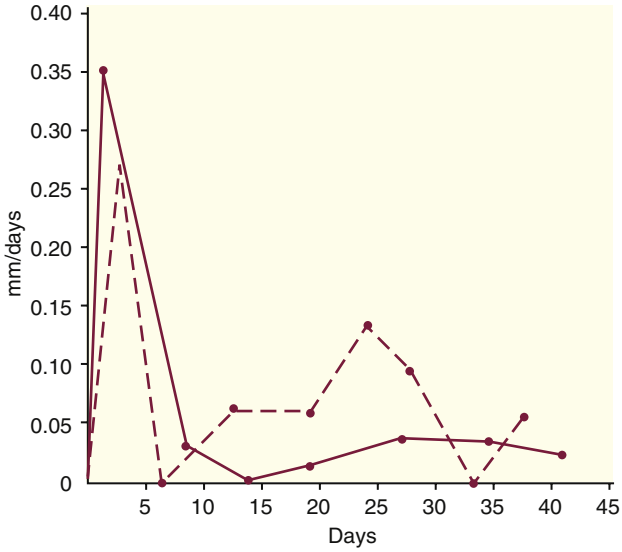


FIGURE 5-12 Rates of tooth movement with different force applications (*dotted line*, 200 *g*; *solid line*, 10 *g*). The rates given are for reciprocal closure of a diastema between two central incisors.

In Figure 5-12, the average rates of tooth movement are plotted against the number of days after insertion of a continuous-force appliance. Application of 10 and 200 *g* of force on a central incisor produces a similar initial response. Fairly light forces apparently can move a tooth through a greater part of the width of the PDL. After this has been accomplished, higher forces may be expected to produce additional compression of the ligament. This additional compression is of lesser magnitude than the initial tooth movement and therefore is not evident in gross measurements of tooth movement. Variables such as the width of the PDL are much more important than force magnitude in determining the initial absolute displacement of a tooth.

During the lag phase of tooth movement, two processes can be observed in areas of pressure: direct resorption may occur on the bone surface facing the root, or indirect (undermining) resorption may start in the medullary spaces and work toward the tooth.[28,29] Currently, the best that can be hoped for is the development of a working hypothesis to explain and relate these bony changes to stress levels in the PDL. Until data are available to correlate measured or calculated stress levels with histologically observed bony resorption, assumptions of this type must be considered tentative. Theoretically, if low-level stress is introduced into the PDL, no bony transformation will occur. If the stress level is increased, a point will be reached at which bone resorption can begin (i.e., a threshold stress level). Nothing is known about the level of stress that can initiate bone resorption and apposition. Clinically, however, relatively small force magnitudes (e.g., 10 *g* or less on the maxillary incisors) can tip teeth.[30] If it even exists, the threshold force (the lowest possible force that will move a tooth) has a low magnitude, at least for simple tipping movements. Stress in the PDL above the threshold level can be expected to increase the amount of frontal resorption on the alveolar process. As the stress value increases, cellular activity can be expected to decline because areas of maximal stress undergo further compression of blood vessels and cellular elements. Histologically, these areas appear hyalinized and may be described as at least partly nonvital. Logically, then, bone resorption also declines because direct bone resorption requires vital connective tissue. Undermining resorption, however, is associated with high stress on the PDL and is a response to a compressed, nonvital area of connective tissue.

At a low stress level, little indirect resorption occurs; in fact, bone apposition can occur in the medullary spaces. As the stress increases, the magnitude of cellular response also increases; however, here, too, a limiting factor comes into play. A force can be only great enough to displace a tooth against the alveolar process; when this occurs, the stress level is limited, and for that particular interval, no greater stimulus for indirect resorption may be expected.

Under most clinical conditions, tooth displacement after the lag phase is caused by a combination of direct and indirect resorption. Concomitant bony and connective tissue changes in the areas of tension most likely occur following resorption of bone in the existing pressure regions.

What, then, is the relationship between force magnitude and the rate of tooth movement? At lower force magnitudes, most increases in force enhance the rate of tooth movement. At higher magnitudes, increases in force are partly responsible for hyalinization of the PDL and result in a longer lag phase. Eventually, undermining resorption removes the area of hyalinization, and the tooth moves rapidly into the newly created space. For these reasons, a wide range of forces, from light to heavy, can move teeth rapidly, provided that the force is continuous.

Lighter forces are more likely to move teeth gradually; heavy forces produce a considerable lag phase followed by a period of rapid movement. Over a long interval, the average rate of movement for heavy continuous force may be greater than that seen with light, continuous force. The complexity of the tissue changes and the large number of variables suggests that postulation of any simple relationship between force magnitude and the rate of tooth movement is plagued by inherent difficulties.

One of the variables to be considered in evaluating force magnitude and rate of movement is the type of tooth movement (center of rotation). For identical forces applied on the crown of a tooth, different centers of rotation can be obtained by altering the applied couple. Although the force remains the same, the change in the moment modifies the stress distribution in the PDL. Imagine a simple model of the tooth in its alveolus (a linear stress–strain relationship in the PDL is assumed). For simplicity's sake, the model has a uniformly thick PDL and smooth alveolar walls. This oversimplifies the nature of the tooth and its surrounding structures; the PDL is not uniformly thick, and the lamina dura is an irregular cribriform plate pierced by many holes. Nonetheless, the tooth model, despite all its difficulties, may prove most useful in comparing stress distribution for different types of tooth movements.

In pure translation (bodily tooth movement), a relatively uniform stress distribution is found in the PDL (actually, variations in the thickness of the PDL alter the stress distribution). Figure 5-13 shows an imaginary stress distribution for a force system causing translation of a central incisor in a lingual direction. If movement of the crown is reduced, the center of rotation shifts from infinity toward the apex of the root. Although the lingual force is identical with that used for bodily movement, the stress distribution is greatly changed. In tipping with a center of rotation at the apex, the maximal stress is found at the alveolar crest, and it is of considerably greater magnitude than that seen in translation. If the moment is increased beyond the amount required for translation, the center of rotation

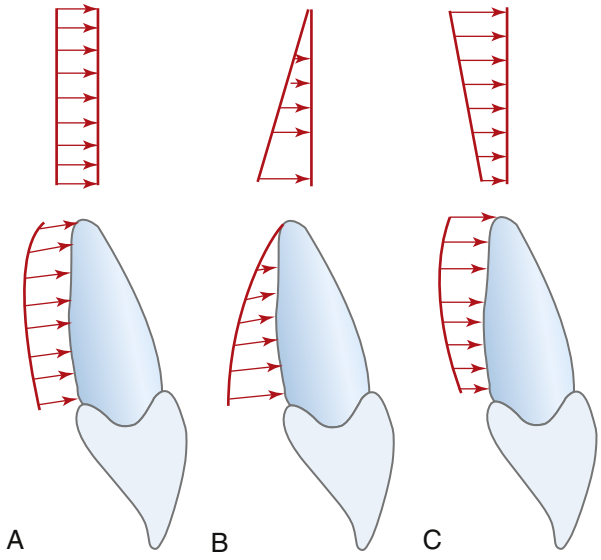


FIGURE 5-13 Theoretic stress distributions in the periodontal ligament. A, Translation. B, Center of rotation at the apex. C, Center of rotation at the incisal edge. The force magnitude is identical in the three situations.

shifts from infinity toward the incisal edge. With the center of rotation at the incisal edge, a nonuniform stress distribution once again is observed. In this case, the maximal stress is at the apex, and it is of greater magnitude than the stress found in translation. One should emphasize that in the three processes (translation, tipping at the apex, and rotation around the incisal edge), the lingual force is identical.

To develop the same maximal stress level of bodily tooth movement as that seen in rotation around the apex and the incisal edge, the lingual force and moment applied to the crown of the tooth must be increased. The center of rotation is an important factor that determines stress distribution in the PDL, and hence the degree and type of cellular reaction. Therefore, in any attempt to relate force magnitude and the rate of tooth movement, the force system must be well defined. This requires knowledge not only of the magnitude but also of the constancy of all forces and moments acting on a tooth crown.

Figure 5-14 shows a tooth in which stress was studied using the finite element method. If a force is placed to produce translation, as shown in Figure 5-15, it is apparent that the stress is not as uniform as that described in the simplistic explanations given previously.[20,31-34] The simplistic explanations may aid in developing an understanding of the ways in which stress distributions vary with the same magnitude of force. One should remember, however, that a tooth is a three-dimensional irregular body that has a most complicated stress distribution, even during translation.

Not all variables that influence the rate of movement involve stress levels in the PDL. Some involve the structure of the supporting bone of the alveolar process. Clinical observation also suggests a great variation in connective tissue response to the stress of orthodontic tooth movement. The response of tissues to mechanical stress is as yet obscure. However, the biochemical mechanisms by which stress and strain cause bone resorption and apposition are beginning to be better understood. Strain, working through biochemical mediators, such as prostaglandins and piezoelectric effects, could be responsible for the biologic response to orthodontic forces.

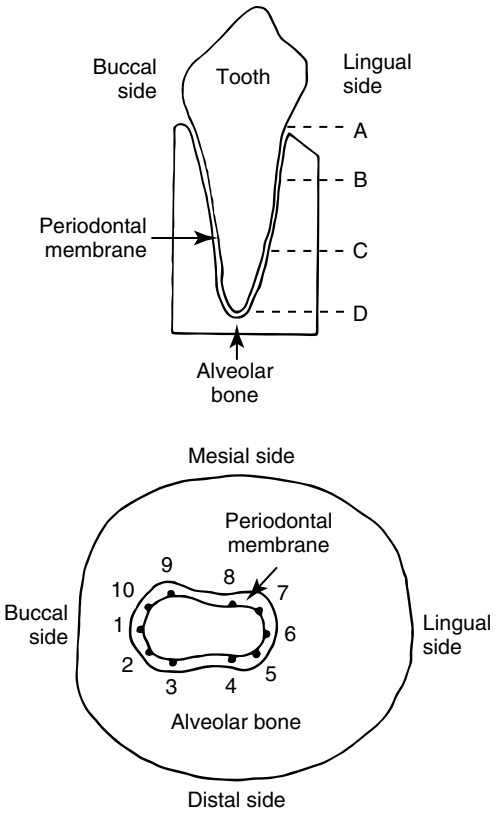


FIGURE 5-14 Occlusogingival levels (A to D) and horizontal points (*1 to 10*) where the principal stresses were determined. (Redrawn from Tanne K, Sakuda M, Burstone CJ. *Am J Orthod Dentofac Orthop.* 1987;92:499.)

Relationship of Force Magnitude to Pain and Tooth Mobility

After an active orthodontic appliance has been inserted, pain or discomfort may develop. Objective evaluation of pain is difficult because the pain response is determined by the central nervous system and local tissue changes. Not surprisingly, however, one finds a wide range of pain reactions among individuals in whom similar forces have been placed on the teeth.

To evaluate the relationship of force to pain, a classification of pain response is helpful. Three degrees of pain responses may be described. First-degree pain is caused only by heavy pressure placed on the tooth with an instrument such as a band pusher or a force gauge. A first-degree response usually is elicited most easily by placing a force in the same direction as the force produced by the appliance. The patient is not aware of first-degree pain unless the teeth being moved with the orthodontic appliance happen to be manipulated. A second-degree pain response is characterized by pain or discomfort during clenching of the teeth or heavy biting. The patient still is able to masticate a normal diet without difficulty. If spontaneous pain is present or the patient is unable to masticate food of normal consistency, a third-degree response exists. Proper interpretation of pain data requires testing of the patient's response to heavy forces placed on the teeth before orthodontic treatment is begun. In this way, the patient influences and even controls future pain and discomfort.

The two types of pain based on time of onset are immediate pain and delayed pain. Immediate pain responses are associated

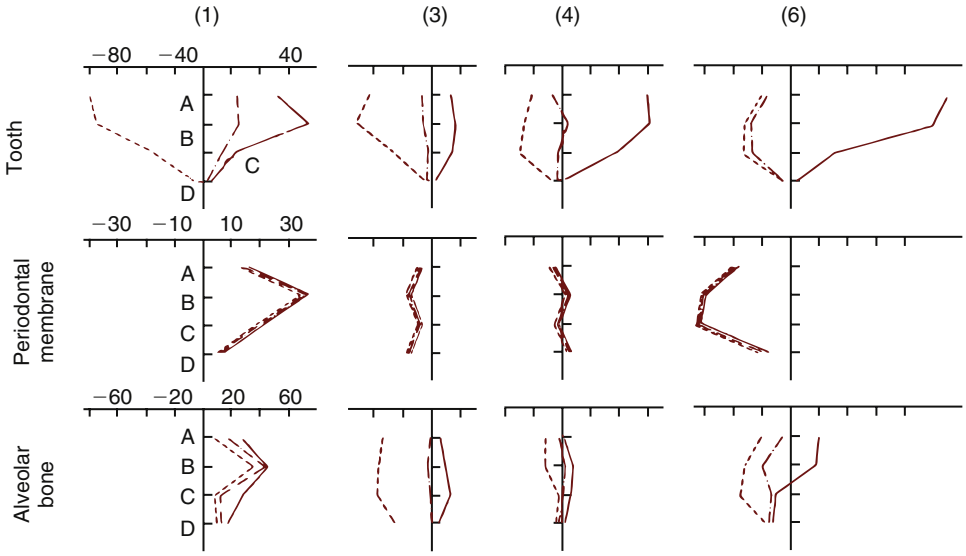


FIGURE 5-15 Stress distribution (g-cm^2) in loading condition for a lingual force of 100 g and a buccal crown couple of 858.5 g-mm (L_2). Three principal stresses—maximal (—), intermediate (---), and minimal (–)—are shown for four significant points (1, 3, 4, and 6) at various occlusogingival levels (*A to D*). (Redrawn from Tanne K, Sakuda M, Burstone CJ. *Am J Orthod Dentofac Orthop.* 1987;92:499.)

with sudden placement of heavy forces on a tooth. For example, a hard figure-eight tie between spaced incisors usually causes an acute pain reaction that gradually subsides. Some hours after an orthodontic adjustment, a delayed response may become evident. Delayed pain responses are caused by a variety of force values, from light to heavy, and represent hyperalgesia of the PDL. The degree of response usually lessens with time. A decreasing gradient is observed when, for instance, the pain reaction passes from a third-degree to a second-degree response and finally to a first-degree or zero-degree response. Although a gradient of this type usually is observed, situations arise in which the pain reaction suddenly increases, for no apparent reason, several days after a continuous force was applied.

Generally, the magnitude of force applied to a tooth has a definite correlation to the patient's experience of pain. Clinical studies have shown that the incidence of second- and third-degree pain is greater with application of heavier forces to the teeth. Not only is a greater degree of pain evident with a heavy force, but also the total number of days in which an abnormal pain response can be elicited is higher. This is not to imply, however, that a linear relationship exists.

The generalization that greater forces produce greater degrees of pain for longer periods is an oversimplification. As with the rate of tooth movement, force magnitude is only one of the factors determining the patient's final response.

An understanding of pain that occurs during orthodontic treatment also is obscured by a lack of knowledge about the reason pain is produced. This difficulty is aggravated by the fact that the PDL has no pain fibers. When the mechanisms of pain production during tooth movement become known, orthodontists will understand better the relationship of force and pain response.

Optimal Force and Stress

The effect of the couple-to-force ratio on the center of rotation of a moving tooth has been considered. Another question of great clinical significance concerns the appropriate magnitude of force and couple to achieve the most desirable response. In other words, what force magnitudes are optimal for tooth movement?[35-37]

From a clinical standpoint, an optimal force is one that produces a rapid rate of tooth movement without discomfort or ensuing tissue damage (particularly alveolar bone loss and root resorption) to the patient. From a histologic viewpoint, an optimal force is one that produces a stress level in the PDL that basically maintains the vitality of the tissue throughout its length and initiates a maximal cellular response (apposition and resorption). Optimal forces, therefore, produce direct resorption of the alveolar process. Because optimal forces require no period for repair, such forces apparently can be made to act continuously.

Histologic studies that correlate forces on the crown or stress in the PDL with tissue responses are most helpful in establishing the levels of optimal force for different situations. Unfortunately, the difficulty involved in obtaining human material is a limiting factor in this type of investigation. At the clinical level, where a greater wealth of material is available, the orthodontist is limited to gross tooth and bone changes or the patient's symptoms. This is not to imply that careful clinical observation is not helpful in determining optimal forces. Lack of pain, minimal mobility, and the absence of a considerable lag period immediately after appliance adjustment are clinical responses that suggest desirable stress levels in the PDL. To use the rate of tooth movement alone as an indicator of optimal force is dangerous, however. Rate is deceptive because heavy and light continuous forces can move teeth rapidly. It does not necessarily follow that because the teeth move rapidly, the forces used are optimal. Long-term histologic and clinical studies are needed to define further the nature of optimal force.

Although study of the biomechanics of tooth movement shows great promise, use of mathematic formulations to describe biologic phenomena is risky because mathematic oversimplification of highly dynamic, variable vital structures and reactions can mislead as well as inform. Biomechanical assumptions, therefore, must be checked against observations made on

the clinical and histologic levels. This type of multidisciplinary approach offers the best hope for solving orthodontic problems involving force systems and tooth movement.

THE ORTHODONTIC APPLIANCE

In designing any orthodontic appliance, the orthodontist starts by subscribing to certain assumptions about the nature of an optimal force system to move teeth. An optimal force system is one that (1) accurately controls the center of rotation of the tooth during tooth movement, (2) produces optimal stress levels in the PDL, and (3) maintains a relatively constant level of stress as the tooth moves from one position to the next. For the sake of argument, assume that these objectives for the orthodontic appliance are correct; one then must decide what is needed to design an appliance to deliver this type of force system.

Active and Reactive Members

An orthodontic appliance can be considered to have active and reactive members. The active member is the part involved in tooth movement; the reactive member serves as anchorage and involves the teeth that will not be displaced. A member sometimes can play an active and a reactive role simultaneously. For example, this is clearly the case when reciprocal anchorage is used.

On the subclinical level, the three primary objectives are to control the center of rotation of the tooth, produce desirable stress levels in the PDL, and maintain a relatively constant level of stress. At the clinical level of observation, the focus becomes the forces and moments produced by an orthodontic appliance. Specifically of interest are three important characteristics involving active and reactive members: (1) the M/F ratio, (2) the load-deflection rate, and (3) the maximal force or moment of any component of the appliance.

Moment-to-Force Ratio

To produce different types of tooth movements, the ratio between the applied moment and the force on the crown must be changed. As the M/F ratio is altered, the center of rotation changes. Crown tipping, translation, and root movement are examples of different types of tooth movements that can be produced with the proper M/F ratio. The important thing for the orthodontist to remember is that in few cases can desirable tooth movement be produced by applying a single force to the crown. If a modern orthodontic appliance is under consideration, an active member must be capable of producing the desired moment and force.

The M/F ratio is equally significant in the reactive member of the appliance. For example, if the practitioner is considering preserving anchorage of the posterior segments in an extraction case, to introduce a moment that tends to move roots forward and crowns back is desirable so that, combined with the mesial forces acting on the posterior segment, a more uniform distribution of stress is achieved in the PDL. A more uniform distribution of stress in the posterior segment minimizes forward displacement. The tip-back bend is one example in which a moment of this type is added to the posterior segment to enhance anchorage. In short, the M/F ratio determines the control that the orthodontic appliance has over the active and reactive units; specifically, it controls the center of rotation of the tooth or a group of teeth.

Load-Deflection Rate

The second characteristic of an orthodontic appliance, the load-deflection (or torque-twist) rate, is a factor in the delivery of a relatively constant force.[38] By definition, the load-deflection rate gives the force produced per unit activation. As the load-deflection rate declines for a tooth that is moving under a continuous force, the change in force value is reduced. For active members, a low load-deflection rate is desirable for two important reasons: (1) a mechanism with a low load-deflection rate maintains a more desirable stress level in the PDL because the force on a tooth does not radically change magnitude every time the tooth has been displaced, and (2) a member with a low load-deflection rate offers greater accuracy in controlling force magnitude. For example, if a high load-deflection spring is used (e.g., an edgewise vertical loop), the load-deflection rate might be 1000 *g*/mm; this means that an error in adjustment of 1 mm could produce an error in force value of about 1000 *g*. However, if a low load-deflection spring is used, such as one with a rate of 10 *g*/mm, an error of 1 mm in activation affects the force value by only 10 *g*. Flexible members with low load-deflection rates require long ranges of activation to build up to optimal force values; hence, they give the orthodontist greater control over the magnitude of force used.

If a low load-deflection rate is desirable for the active member of the appliance, the opposite is true for the reactive member. The reactive member should be relatively rigid; that is, it should have a high load-deflection rate. The anchorage potential of a group of teeth can be enhanced if the teeth displace as a unit. If individual teeth in the reactive unit tend to rotate around separate centers of rotation, higher stress distributions are produced in the PDL and the teeth can be more easily displaced. Another factor to consider is that the equal and opposite forces produced by the active members usually are distributed to localized areas, with just one or a few teeth involved. Localized tooth changes in these areas can be minimized if the reactive members of the appliance are sufficiently rigid. In short, the load-deflection rate is an indicator of the force required per unit deflection. In the reactive part of the appliance, a high load-deflection rate is needed when the orthodontist is dealing with a relatively rigid member.

Maximal Elastic Moment

The last characteristic of an orthodontic appliance that must be evaluated is the maximal elastic load or moment, which is the greatest force or moment that can be applied to a member without causing permanent deformation. Active and reactive members must be designed so they do not deform if activations are made that allow optimal force levels to be reached. In designing an appliance, a good idea is to go beyond required force needs and create a safety factor. Thus, permanent deformation or breakage will not occur from accidental overloading, which can be caused by abnormal activation of an appliance or by abnormal forces during mastication.

All three of the important characteristics of an orthodontic appliance—the M/F ratio, the load-deflection rate, and the maximal elastic load or moment—are found within the elastic range of an orthodontic wire and therefore may be called spring characteristics. Beyond this range are the plastic changes that can occur in a wire up to the point of fracture. Although plastic changes are important in the design of an orthodontic appliance, they are not considered in detail in this discussion.

The designer controls a number of variables that influence spring characteristics; these variables are discussed individually in the following sections. The orthodontist always should keep in mind the relationship between these variables and the three important characteristics previously examined.

Manner of Loading

If an active member is to deliver continuous force for tooth movement, it must be able to absorb and release energy. Energy absorption in a flexible member results from the elastic deformations that occur during application of a force or load. Elastic deformations are changes in form or configuration that are reversible when the load is removed.

To understand the different types of loading and their significance, one must visualize a structural axis centrally positioned along a round wire (Fig. 5-16). A force acting along the structural axis of the wire may produce compression or tension, shortening or elongating the wire. Thus, in tension and compression, the axial load may increase or decrease the length of the structural axis. This change is produced by force acting along the structural axis and therefore is called an axial load. If a moment operates around the structural axis (i.e., at right angles to the lines of the structural axis), torsion is produced (Fig. 5-17, *A*). In torsion, the wire rotates around the structural axis, with the greatest elastic deformation occurring at the periphery of the wire. Bending, or flexure, is produced when the structural axis changes its configuration transversely or at right angles to its original structural axis. Bending can be produced by moments acting at right angles to the cross section of the wire (Fig. 5-17, *B*) or by a transverse force acting on the wire (Fig. 5-17, *C*).

The typical orthodontic appliance usually is not loaded in a simple manner. Tension, compression, torsion, and bending commonly are combined into a more complicated type of loading pattern referred to as *compound loading*. Figure 5-18, *A*, shows two vertical loops in a round wire that can be used clinically to move a tooth buccally or lingually by displacing the central section at right angles to the surface of the paper. In reality, a compound deformation takes place, with bending occurring at point B and torsion or twisting at point A. During activation of this particular member, bending or torsion occurs. Figure 5-18, *B*, shows a vertical loop that can be used as a retraction spring. In a loop of this type, if the horizontal arms are kept parallel during activation, the loading pattern is fairly complicated. Not only are horizontal forces required, but also two equal and opposite couples must be used to keep the horizontal arms parallel. Even though the loading pattern is more complicated in this example, the vertical loop undergoes only bending.

Certain types of loads, whether forces or moments, can produce certain changes in the structural axis of a wire. These changes are referred to as *compression, tension, torsion*, and *flexure*. A more sophisticated system of categorizing changes in a wire is to describe such changes in terms of the stress distribution throughout the length of the wire. This approach is not included in this discussion because a knowledge of stress–strain phenomena is required of the reader. One should remember, however, that resisting forces act throughout the wire during loading, resulting in certain internal factors of stress and strain. Reference is made to stress and strain only when absolutely necessary to develop a point.

Axial loads that produce compression or tension are not useful for spring design because the load-deflection rate is high. An axial pull on a wire often is observed not to produce much elastic deformation, even with a heavy force, because the force is distributed uniformly as stress over each cross section of the wire. However, when nonuniform stress is distributed over various cross sections of wire (as in torsion and bending), the load-deflection rate may be low. For this reason, loading that leads to torsion and flexure is useful in the design of active or flexible members of an appliance.

If the maximal load or maximal torque were maintained as a constant, the load-deflection rate would be lowest in two particular types of loading: torsion and bending produced by moments alone. Such a low load-deflection rate might exist for a given maximal load because each cross section of the wire, from one end to the other, undergoes the same amount of torsion

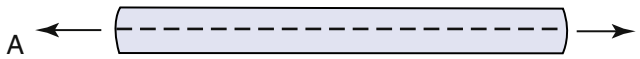


FIGURE 5-16 Axial loading. **A,** Tension. **B,** Compression. The force acts along the structural axis (*dotted line*).

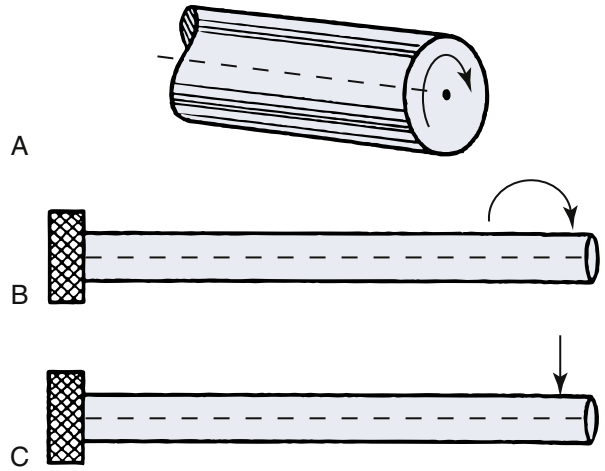


FIGURE 5-17 Torsion and bending. **A,** Torsion is produced by a couple acting around the structural axis. **B,** Pure bending is produced by the application of a couple. **C,** Bending is produced by a transverse force.

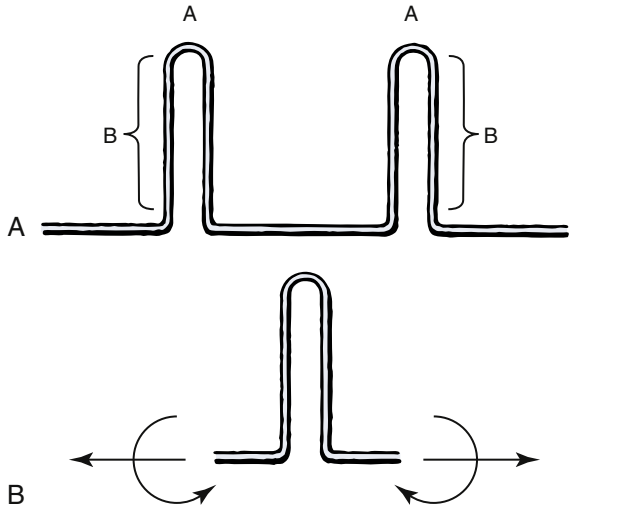


FIGURE 5-18 **A,** Displacement of the central portion of a loop at right angles to the page requires a force and a moment. Bending occurs at **B** and torsion at **A**. **B,** Opening a vertical loop requires a force and a moment to keep the horizontal arms parallel.

or bending. Loading of this type is ideal for spring design, but unfortunately in most instances more than a moment must be delivered to a tooth. Transverse loads therefore must be introduced, and these do not produce uniform changes along a wire unless the diameter of the wire differs along its length (i.e., a tapering wire).

In designing an orthodontic appliance, when should the orthodontist take advantage of tension, compression, torsion, and bending? A number of factors determine the manner of loading that should be used on a given member. For example, one configuration may be superior to another based on simplicity of design, general space available, or comfort in the mouth. Also, the M/F ratio needed to control the teeth partly determines the configuration to be used. For example, if equal and opposite moments are required, pure torsion or bending in a wire may be used. However, if moments and forces are required, primarily bending properties most likely will be used. In considering a reactive member, the forces should be distributed as axial loads whenever possible. For example, a transpalatal lingual arch can preserve widths of the posterior segments better than can a horseshoe lingual arch because the horseshoe-type arch bends more easily in this plane of space. Therefore, one of the early steps in the design of an orthodontic member is to decide on its basic configuration, keeping in mind the objectives listed.

Of the variables that influence the spring characteristics of load-deflection rate (torque-twist rate), maximal elastic load or maximal elastic torque, and M/F ratio, only bending is considered in the following sections. Because most orthodontic configurations take advantage of bending as a major type of elastic deformation, many of the concepts (if not the exact formulations) also apply to torsion and axial loading.

Mechanical Properties of Metals

The mechanical properties of an alloy to be used in an orthodontic wire can be described on at least three levels. The most superficial is the observational level, on which the clinician operates. On this level, forces and deflection can be noted and measured. In other words, a certain amount of force in grams can be applied, and the wire will deflect by a certain number of millimeters. On the observational level, the orthodontist is limited in how much can be understood and predicted about the nature of appliances.

The second level of description is the stress–strain level. On this level the orthodontist is dealing with pounds per square inch and deflection per unit length. These values cannot be measured directly, but they can be calculated from measurements made on the observational level. Most of the engineering formulations that can be used to predict changes in bodies subject to loads are based on stress–strain phenomena.

The third level of description is the atomic and molecular levels. An understanding of events at the atomic and molecular levels enhances the ability to predict responses and design new structures.

Basic Behavior of Alloys

Figure 5-19 is a theoretic diagram that plots load against deflection. It might represent, for instance, the load-deflection characteristics of an open coil spring. Deflection can be seen that from O to P_{max} (maximal elastic load) on the graph, a linear relationship exists between load and deflection. As the force increases, the deflection increases proportionately; this proportionality is referred to as *Hooke's law*. Load divided by deflection is a constant through this range and already has been defined as the load-deflection rate. At P_{max} a point is reached where load and deflection are no longer proportionate. Near P_{max}, permanent deformation is being produced in the spring, which will not return to its original shape. P_{max} represents the highest load that can be placed on the spring without permanent deformation; that is, the maximal elastic load. All the behavior found to the left of P_{max} on the graph lies in the elastic range, and behavior to the right lies in the plastic range. Elastic behavior is the ability of a configuration to return to its original shape after unloading; plastic behavior is the occurrence of permanent deformation in a configuration during loading. Finally, at the extreme right of the graph, the ultimate load (P_{ult}) is reached, at which point the spring will break. The diagram is a schematic one, and loads and deflections may not be so regular in commonly used orthodontic springs.

Load-deflection diagrams of the type shown have a limited application because a separate diagram is required for every orthodontic member. However, if the stress–strain level is studied, one can make generalizations about orthodontic alloys that apply to any given alloy regardless of the configuration. The graph shown in Figure 5-20 plots stress against

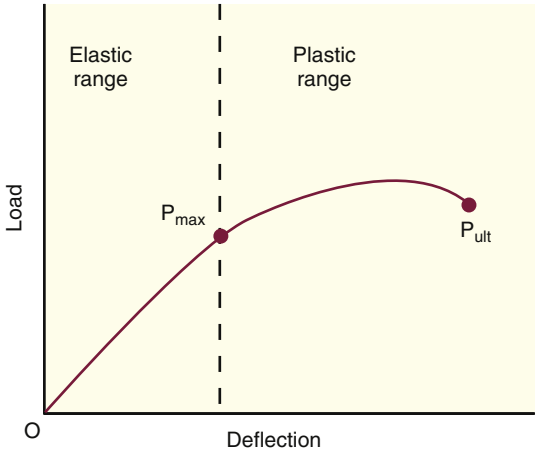


FIGURE 5-19 Load deflection. Note the linear relationship between load and deflection in the elastic range. P_{max}, Maximal elastic load; P_{ult}, maximal load before fracture.

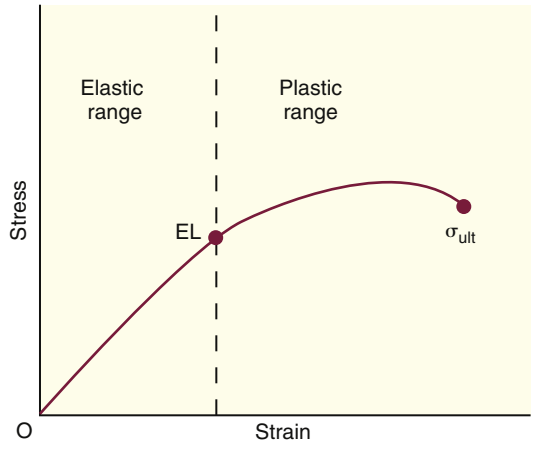


FIGURE 5-20 Stress–strain relationship. Note the linear relationship between load and deflection in the elastic range. *EL*, Elastic limit; σ_{ult}, tensile strength.

strain.* A diagram of this type corrects for the dimensions of the wire. The graph is identical in form to that of the load-deflection rate, except that the units are different. From O to EL (the elastic limit) is a straight line, denoting a linear relationship between stress and strain. This relationship is comparable to the relationship seen on the observational level between load and deflection. The ratio of stress to strain is referred to as the *modulus of elasticity* (E). As might be expected, this mechanical property determines the load-deflection rate of a spring.[39] The EL is the greatest stress that can be applied to the alloy without permanent deformation. The EL is analogous to the maximal elastic load and therefore is the mechanical property that determines the ability of a member to withstand permanent deformation. A number of other terms describe this general part of the curve, such as yield point, yield strength, and proportional limit; these points are close to the EL, although they differ by definition. Finally, at the ultimate stress (tensile strength), the wire will fracture. As with loads and deflections, most actual alloys do not present such a regular and definitive pattern.

An explanation of elastic and plastic behavior would not be complete without a brief mention of atomic and molecular events. Fundamentally, elastic behavior involves interatomic bonding. Because atoms are pulled apart, a fairly definite relationship exists between stress and strain. However, plastic behavior involves displacement along slip planes, which are molecular, not atomic. Plastic behavior, therefore, is not as linear as elastic behavior.

Elastic Limit

The EL determines the maximal elastic load of a configuration. With respect only to the mechanical properties of the wire, the maximal elastic load varies directly and linearly with the EL. Manufacturers' data usually include the yield point or the tensile strength. The yield point is close to the elastic limit, but the tensile strength is higher.*

In a given alloy (e.g., 18-8 stainless steel), a number of factors determine the elastic limit. The amount of work hardening produced during cold drawing of the wire sharply influences the EL.† Wires that have been considerably cold worked have a hard temper and therefore a high EL.[40] Small, round wires may have particularly high ELs because the percentage of reduction by cold working is high. Also, the cold-worked outer core becomes proportionately greater in a wire of smaller cross section. Too much work hardening, however, produces a structurally undesirable wire that becomes highly brittle and may fracture during normal use in the mouth. Far better is to have a slightly lower EL so that an orthodontic member can deform permanently rather than break under accidental loading. Because the work hardening required to reduce the diameter of a wire increases the EL, anodic reduction is a poor method for reducing the size of the wire. Anodic reduction does not cold work a metal; therefore, the wire produced by that method has a lower EL than a work-hardened one, a circumstance that could lead to permanent deformation.

Many orthodontic alloys, such as Elgiloy and gold, can be heat treated to raise the EL, but the most commonly used alloy, 18-8 stainless steel, cannot. However, a stress relief process at 8508°F for 3 minutes or longer raises the apparent elastic limit of 18-8 stainless steel. Stress relief removes undesirable residual stress introduced during manufacturing and during fabrication by the orthodontist. If a single stress release is used, the optimal time to perform it is after all required bends and twists have been placed in the wire.

Modulus of Elasticity

The mechanical property that determines the load-deflection rate of an orthodontic member is the modulus of elasticity (E).† Load-deflection varies directly and linearly with E (in torsion, linearly, and directly as the modulus of rigidity). Steel has an E approximately 1.8 times greater than that of gold. A reactive member made of stainless steel is 1.8 times as resistant to deflection as one made of gold. With edgewise brackets and a 0.022-inch × 0.028-inch archwire, for example, a steel wire gives greater control over the anchorage unit. However, activations made in a steel wire for tooth movement, if identical to the ones made in a gold wire of similar configuration, produce a load-deflection rate almost twice as high. For this reason, steel and gold are not directly interchangeable in the design of an orthodontic appliance.[39,42]

Steel alloys are the alloys most commonly used for orthodontic wires. The E of most steel alloys is almost identical. Unlike the EL, the E is constant for a given alloy and is not influenced by work hardening or heat treatment.* Therefore, hard-temper wires do not have higher load-deflection rates than soft-temper wires. When changing the E of a piece of stainless steel, a new alloy must be chosen because nothing can be done to a steel alloy that will alter its E greatly.

Shape-Memory Alloys

Within recent years, two new alloys have been introduced in orthodontics: nickel titanium and beta-titanium (TMA).

Nickel titanium (nitinol) was developed by William F. Buehler in the early 1960s. The original alloy contained 55% nickel and 45% titanium, which resulted in a 1:1 stoichiometric ratio of these elements. The unique feature of this bimetallic (NiTi) compound is its memory, which is a result of temperature-induced crystallographic transformations. Andreasen and Hilleman[43] and Andreasen and Morrow[44] suggested that the orthodontist use these shape changes to apply forces. The shape-memory principle is not used clinically. Instead, nitinol is used for its low force and high springback. The low E of nitinol, only 0.26 that of stainless steel, means that a 0.018-inch wire has the approximate stiffness of a 0.013-inch stainless-steel wire. The most dramatic characteristic of nitinol, however, is its resistance to permanent deformation. NiTi wires can be activated over twice the distance of stainless steel, with minimal permanent deformation. Because permanent deformation is time dependent, though, additional small deformation occurs between adjustments. After bends or twists are placed, if the wire is activated in a direction opposite that used in forming the configuration, it easily deforms permanently.[45] Nitinol, therefore, is most useful when low forces and large deflections are needed in relatively straight wires. Nitinol is more brittle than stainless steel and cannot be joined by soldering or welding.[43]

A new generation of shape-memory alloys has been introduced into orthodontics and is referred to as *superelastic*.[46-50] Unlike nitinol, these alloys have a much lower transition temperature—either slightly below or slightly higher than mouth

* Stress in a wire is force per unit area applied to a cross section. Strain is deflection per unit length of the wire.

† Residual stress or other mechanisms can give an E that is slightly lower after work hardening.[17,41]

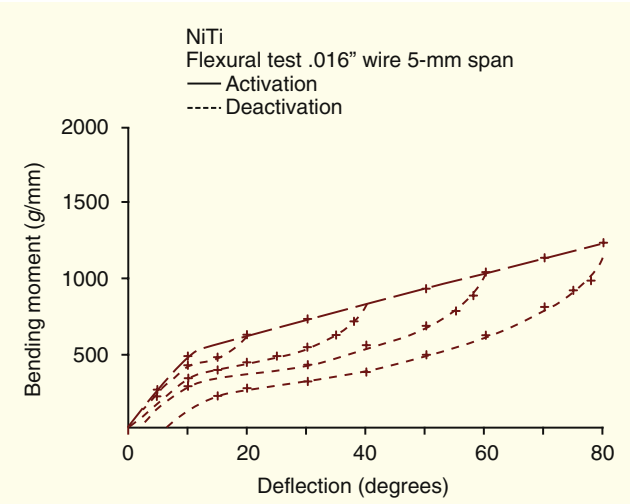


FIGURE 5-21 Activation and deactivation curves for NiTi wire. Unlike with stainless steel and nitinol wires, the unloading curves for NiTi wire change at different activations. (Redrawn from Tanne K, Sakuda M, Burstone CJ. *Am J Orthod Dentofac Orthop.* 1987;92:499.)

temperature. Generally speaking, the austenitic form of these alloys has a slightly higher springback than nitinol and may be less brittle. Figure 5-21 shows a loading and unloading curve for a superelastic NiTi wire at various activations. At the larger activations, part of the unloading curve is relatively flat. The clinical significance of this finding is that more constant forces are delivered to the teeth during the deactivation. Another interesting finding is that the stiffness is greater for small activations than for large activations. The typical orthodontic wire, however, is different because it has a relatively constant load-deflection rate, and it delivers increasing force, depending on the amount of activation.

The superelastic NiTi wires are available in different degrees of stiffness. A true comparison of stiffness should be made at mouth temperature because some wires may appear to have lower forces, due to the fact that they are partly martensitic at room temperature.[51] Forces increase as a phase transformation occurs with mouth temperature. Although the final transition temperature of some superelastic NiTi wires is below mouth temperature, others are not activated fully until they reach 37°C or higher. These wires have both superelastic and shape-memory properties. Generally, the heat treatment process performed during manufacture to raise the transition temperature allows for wires that deliver lower forces at mouth temperature; hence, the orthodontist may be able to achieve full bracket engagement with larger wires earlier in treatment.

The superelastic NiTi wires and nitinol are limited in that they are not formed easily; also, through permanent deformation, they lose bends that are placed unless a heat treatment process is carried out. NiTi wires are brittle, and they usually are used in procedures that call for relatively straight wires and large deflections without permanent deformation.

TMA has a modulus of elasticity between that of steel and nitinol (approximately 0.4 times that of stainless steel).[52,53] TMA can be deflected up to two times as much as steel without permanent deformation. Unlike nitinol, TMA is not significantly altered by the placement of bends and twists and has good ductility, equivalent to or slightly better than that of stainless steel, and can be welded without significant reduction in yield strength. Finger springs and hooks can be welded directly without solder reinforcement[54,55] (Fig. 5-22).

Another new alloy introduced into orthodontics is titanium niobium. The alloy has low springback (equivalent to stainless steel) and is much less stiff than TMA. The alloy is useful when a highly formable wire with low forces in small activations is required.

Ideal Orthodontic Alloys

The ideal orthodontic wire for an active member is one that gives a high maximal elastic load and a low load-deflection rate. The mechanical properties that determine these characteristics are the EL and E. The ratio between the EL and E determines the desirability of the alloy: the higher the ratio, the better the spring properties of the wire. In the commercial development of new wires, the orthodontist should look for alloys that have a high EL and a low E. Small differences in the EL or E do not appreciably alter the ratio. For an alloy to be considerably superior in spring properties, it must possess a significantly higher ratio.

In the reactive member of an appliance, however, not only is a sufficiently high EL required, but also a high E is desirable. Because common practice is to use the same size slot or tube opening throughout a hookup, different alloys can be combined in the same appliance to serve the needs of active and reactive members.

Four other properties of wire should be considered when evaluating an orthodontic wire: (1) the alloy should be reasonably resistant to corrosion caused by the fluids of the mouth, (2) the wire should be sufficiently ductile so that it does not fracture under accidental loading in the mouth or during fabrication of an appliance, (3) the wire should be able to be fabricated in a soft state and later heat-treated to hard temper, and (4) the alloy should allow easy soldering of attachments.

A thorough knowledge of the mechanical and physical properties of an alloy is important in the design of an orthodontic appliance. However, these are but two of the many variables that determine the final form of an orthodontic mechanism.

Wire Cross Section

One of the crucial factors in the design of an orthodontic appliance is the cross section of the wire to be used. Small changes in cross section can influence the maximal elastic load and the load-deflection rate greatly.[38,56]

The maximal elastic load varies directly as the third power of the diameter of round wire, and the load-deflection rate varies directly as the fourth power of the diameter. The most obvious means of reducing the load-deflection rate of an active member may seem to be to reduce the size of the wire. However, the fallacy in reducing the size of the cross section is that the maximal elastic load also is reduced at an alarmingly high rate (as d^3). When designing active members, a good policy is to use as small a cross section as is consistent with a safety factor so that undue permanent deformation does not occur. Beyond this, any attempt to reduce the size of the cross section to improve spring properties may well lead to undesirable permanent deformation.

The fact that the load-deflection rate varies as the fourth power of the diameter in round wires suggests the crucial importance of selecting a proper cross section. For example, 0.018-inch wire is not interchangeable with 0.020-inch

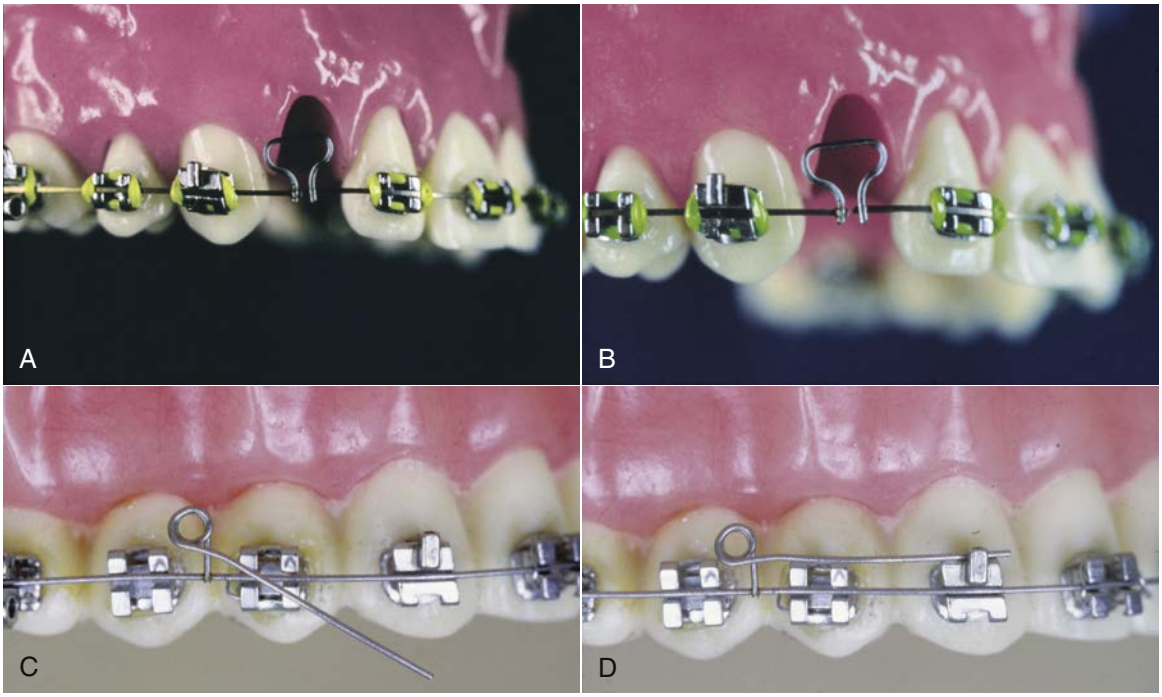


FIGURE 5-22 A, A vertical loop is welded to a continuous arch. B, The arch is cut between the loop forming a vertical loop arch. C, D, Welded helical spring used to erupt a high cuspid. The cuspid is now erupted and engaged in the arch wire. TMA wires can be welded directly together without significantly affecting the mechanical properties.

wire because with a similar activation (forgetting about play in the bracket), the 0.020-inch wire delivers almost twice as much force. The dramatic difference between wire sizes can be demonstrated further by comparing two similar activations in a 0.020-inch and a 0.010-inch round wire. The 0.020-inch wire delivers not twice as much force but rather 16 times as much force, the load-deflection rate varying as the fourth power of the diameter.

In selecting a proper cross section for the rigid reactive members of an appliance, the load-deflection rate, rather than the maximal elastic load, is the prime consideration. Normal circumstances require a large enough wire cross section to give sufficient rigidity so that a sufficiently high load-deflection rate is ensured.

What is the optimal cross section for a flexible member? Generally, for multidirectional activations in which the structural axis is bent in more than one plane, a circular cross section is the structure of choice. Furthermore, because round wire is so widely available for commercial purposes, the mechanical properties of the wire and the cross-sectional tolerances are far superior to those of other cross sections. One of the drawbacks to round wire is that it must be properly oriented or activations may not operate in the intended plane. Moreover, round wire may rotate in the bracket, and if certain loops are incorporated into the configuration, these can roll into the gingiva or the cheek. Many orthodontic wire configurations undergo unidirectional bending. For example, an edgewise vertical loop used for anterior retraction has a structural axis that bends in only one plane. For unidirectional bending, flat wire is the cross section of choice; more energy can be absorbed into a spring made of a flat wire than with any other cross section. This principle has been used for years in watch springs and other commercial designs. Hence, flat (ribbon) wire can deliver lower load-deflection rates without permanent deformation more successfully than any other type of cross section. Another advantage of flat wire is that the problem of orientation is much easier to solve than with a round cross section. Flat wire can be anchored definitely into a tube or bracket so that it will not spin during deactivation of a given spring. Flat wire also can be used in certain situations when considerable tooth movement is required in one plane but limited tooth movement is needed in the other. For instance, if continuous ribbon wires are used (long-axis oriented occlusogingivally), positive leveling can be achieved occlusogingivally over a limited range, and buccolingual and labiolingual tooth alignment can be effected over a long range of action. A configuration of this type is useful when most of the problems are in the horizontal rather than the vertical plane.

For the reactive member, square or rectangular wire is superior to round wire because of the ease of orientation and greater multidirectional rigidity of the former, which leads to more definite control of the anchorage units. In the edgewise mechanism, the assumption may be made that greater rigidity is needed buccolingually or labiolingually than occlusogingivally because an edgewise wire is used. This may or may not be true, depending on the intended use of the edgewise mechanism.

Selection of the Proper Wire (Alloy and Cross Section)

Selection of the proper size wire should be based primarily on the load-deflection rate required and secondarily on the magnitude of the forces and moments needed. Many orthodontists select a cross section of wire based on two other factors, which, although valid, are not as significant:

1. Some clinicians believe that increasingly heavy wires are needed in a replacement technique to eliminate the play between wire and bracket. In an edgewise appliance, however,

TABLE 5-1 Factors Influencing Load-Deflection Rate, Maximal Load, and Maximal Deflection

Design Factor	Load-Deflection Rate	Maximal Load	Maximal Deflection
Addition of wire without changing length	Decreases	No change	Increases
Activation in direction of original bending		Increases	Increases
Material properties of wire	Increases as E	Increases as Sp	Increases as Sp/E
Wire cross section (d) (round)	d^4	d^3	1/d
Wire cross section (b, h) (rectangular)	bh^3	bh^2	1/h
Length (L) (cantilever)	$1/L^3$	1/L	L^2

d, Diameter; *h*, diameter in the direction of bending; *b*, direction at right angle to h; *E*, modulus of elasticity; *Sp*, proportional limit.

the ligature wire minimizes much of the play in a first-order direction because it can fully seat within the brackets. Therefore, the clinician does not select a 0.018-inch over a 0.016-inch wire primarily because of the difference in play.

2. A wire also may be selected because it is believed that the smaller the wire, the greater the maximum elastic deflection possible; in other words, the smaller the wire, the more it can be deflected without permanent deformation. This is true, but maximal elastic deflection varies inversely with the diameter of the wire. A 0.016-inch wire has only 1.15 times as much maximal elastic deflection as a 0.018-inch wire; therefore, the difference is negligible from a clinical standpoint. If the difference is 2:1 (as in 0.010-inch versus 0.020-inch wire), this factor becomes clinically significant.

The primary reason for selecting a particular wire size is the stiffness of the wire (i.e., its load-deflection rate). In a replacement technique, for example, the orthodontist might begin with a 0.014-inch wire that, deflected over 2 mm, gives the desired force. After the tooth had moved 1 mm, the wire can be replaced with a 0.018-inch wire, which gives almost the same force with 1 mm of activation.

Small differences in cross section produce big changes in load-deflection rates because in round wires, the load-deflection rate varies as the fourth power of the diameter (Table 5-1). In bending, the stiffness, or load-deflection rate, is determined by the moment of inertia of the cross section of the wire with respect to the neutral axis. Clinicians are interested in the relative stiffness of the wire they use, but they have neither the time nor the inclination to use engineering formulas to determine these degrees of stiffness. Therefore, a simple numbering system has been developed, based on engineering theory, which gives the relative stiffness of wires of different cross sections if the material composition of the wire is the same.[56] The cross-sectional stiffness number (C_s) uses 0.1-mm (0.004-inch) round wire as a base of 1.

A 0.006-inch wire has a C_s of 5.0, which means that for the same activation, five times as much force is delivered. Tables 5-2 and 5-3 list, under the C_s column, stiffness numbers based on nominal cross sections. Manufacturing variation or mislabeling of wires obviously can change the actual C_s significantly.

TABLE 5-2 Cross-sectional Stiffness Numbers (Cs) of Round Wires

CROSS SECTION		
(in)	(mm)	Cs
0.004	0.102	1.00
0.010	0.254	39.06
0.014	0.356	150.06
0.016	0.406	256.00
0.018	0.457	410.06
0.020	0.508	625.00
0.022	0.559	915.06
0.030	0.762	3164.06
0.036	0.914	6561.00

(From Burstone CJ. *Am J Orthod.* 1981;80:1. With permission from the American Association of Orthodontists.)

TABLE 5-3 Factors Influencing Load-Deflection Rate, Maximal Load, and Maximal Deflection

	CROSS SECTION		C_S	
Shape	(in)	(mm)	First Order	Second Order
Rectangular	0.010×0.020	0.254×0.508	530.52	132.63
Rectangular	0.016×0.022	0.406×0.559	1129.79	597.57
Rectangular	0.018×0.025	0.457×0.635	1865.10	966.87
Rectangular	0.021×0.025	0.533×0.635	2175.95	1535.35
Rectangular	0.0215×0.028	0.546×0.711	3129.83	1845.37

	CROSS SECTION		
Shape	(in)	(mm)	Cs
Square	0.016×0.016	0.406×0.406	434.60
Square	0.018×0.018	0.457×0.457	696.14
Square	0.021×0.021	0.533×0.533	1289.69

(From Burstone CJ. *Am J Orthod.* 1981;80:1. With permission from the American Association of Orthodontists.)

Two C_s numbers are given for rectangular wires—one for the first-order direction and one for the second-order direction.

The C_s of wire with a cross section of 0.016 inch is 256; this means that for an identical activation, it delivers 256 times as much force as a 0.004-inch round wire. The C_s of 0.018-×0.025-inch wire in a first-order direction is 1865. Because the C_s for 0.016-inch wire is 256, a 0.018-×0.025-inch wire in a first-order direction delivers 7.3 times as much force for the same activation. The assumption, for purposes of comparison, is that the wire configuration and the alloy are identical and only the cross section varies. Any two sections of wire can be compared for stiffness simply by dividing the C_s number of one into the other.

Figure 5-23 presents a graph of the C_s numbers for 0.014- to 0.018-×0.025-inch wires. Although the full spectrum of available wire cross sections is not shown, it is apparent that load-deflection rates can vary by a factor of 10 or more if different sized wires of a constant material (e.g., stainless steel) are used.

In the past, the cross section of wires has been varied to produce different degrees of stiffness. The overall stiffness of

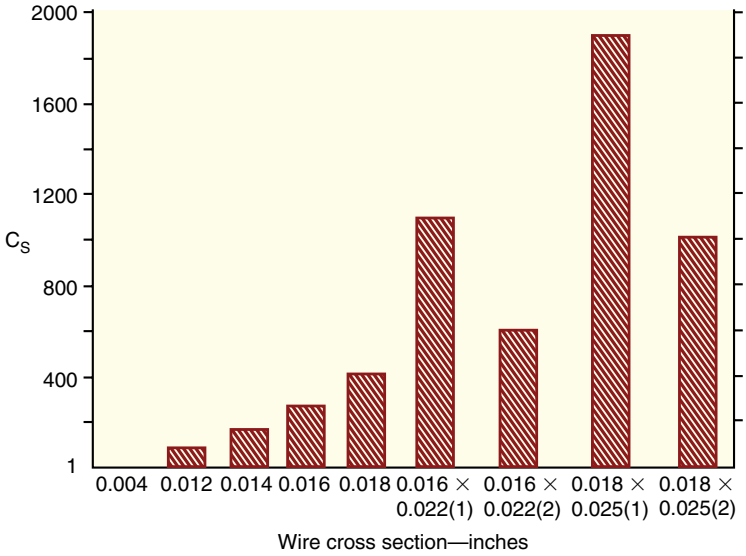


FIGURE 5-23 Cross-section stiffness (C_s) numbers of orthodontic wires. Forces for any given activation are proportionate to the number. With varying cross sections, stiffness can vary by a factor of 10 or more.

an appliance (S) is determined by two factors: one relates to the wire itself (W_s), and the other is the design of the appliance (A_s):

$$S = W_S \times A_S$$

where S is the appliance load-deflection rate; W_s, the wire stiffness; and A_s, the design stiffness factor. In general terms

$$\text{Appliance stiffness} = \text{wire stiffness} \times \text{design stiffness}$$

As the appliance design is changed by increasing wire between the brackets or by adding loops, the stiffness can be reduced as the design stiffness factor changes; however, the orthodontist is not concerned only with ways in which wire stiffness can be altered. Wire stiffness is determined by the cross section and the material of the wires:

$$W_S = M_S \times C_S$$

where W_s is the wire stiffness number; M_s, the material stiffness number; and C_s, the cross-sectional stiffness number. In general terms

$$\text{Wire stiffness} = \text{material stiffness} \times \text{cross-sectional stiffness}$$

Wire stiffness is determined by a cross-sectional property (e.g., moment of inertia) and a materials property (the E). In the past, because most orthodontists used only stainless steel with almost identical Es, only the size of the wire was varied and no concern was expended on the material property, which determines wire stiffness. With the availability of new materials, one can use the same cross section of wire but with different materials with differing degrees of stiffness to produce the wide range of forces and load-deflection rates required for comprehensive orthodontics.

Just as a simple numbering system proved useful for describing the relative stiffness of wires based on cross section, a similar numbering system can be used to compare relative stiffness based on the material. The material stiffness number (M_s) is based on the E of the material. Because steel currently is the most commonly used alloy in orthodontics, its M_s has been arbitrarily set at 1.0. Table 5-4 shows typical stiffness numbers for other alloys. Although the E is considered a constant, the history of the wire (particularly the drawing process) may have some influence on the modulus. Furthermore, differences in chemistry may make small changes in the recorded modulus. For practical clinical purposes, however, the M_s can be used to determine the relative amount of force a wire gives per unit activation. Note that TMA has an M_s of 0.42, which means that for the same appliance and wire cross section, a given activation delivers approximately 0.4 as much force as steel. Elgiloy wires deliver slightly more force than comparable wires of stainless steel, but for all practical purposes this increase is negligible.

In addition to new alloys, braided wires have been introduced into orthodontics. Braids take advantage of smaller cross sections, which have higher maximal elastic deflections and in the process produce wires that have a relatively low stiffness. If the reader were to pretend that a braid was a solid wire and if nominal cross sections were used, it would be possible to establish an apparent E. Based on an apparent modulus, the material stiffness numbers are given for representative braided wires in Table 5-4. For instance, a 0.018-inch Respond wire braid has an M_s of 0.07 and delivers only 0.07 times the force of a 0.018-inch steel wire. The variation in M_s is depicted in a graph in Figure 5-24.

The load-deflection rate can be changed by keeping the wire size constant but varying the load-deflection rate significantly by altering the cross section. Maintaining a cross section of 0.018- × 0.025-inch wire, the wire stiffness (W_s) can be changed by using different materials. To obtain the Ws, the M_s must be multiplied by the C_s. For example, in a second-order direction for TMA,

$$W_S = M_S \times C_S \quad W_S = 0.42 \times 967$$
$$W_S = 406.1$$

TABLE 5-4 Material Stiffness Numbers (Ms) of Orthodontic Alloys and Braided Steel Wires*

Material	M_s
Alloys	
Stainless steel (ss)	1.00
TMA	0.42
Nitinol	0.26
Elgiloy blue	1.19
Elgiloy blue (heat treated)	1.22
Braids	
Twist-flex	0.18-0.20
Force-9	0.14-0.16
D-rect	0.04-0.08
Respond	0.07-0.08

*Based on E = 25 × 106 psi.
(From Burstone CJ. *Am J Orthod.* 1981;80:1. With permission from the American Association of Orthodontists.)

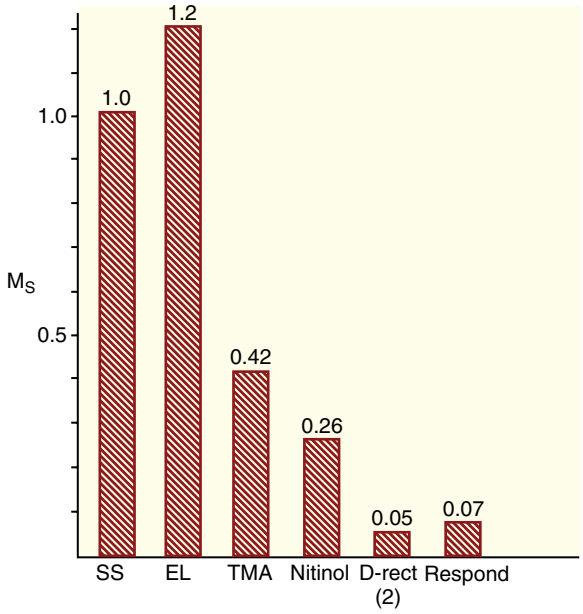


FIGURE 5-24 Material stiffness (M_s) numbers. Stainless steel has a base number of 1.0. The numbers for the other alloys and braids denote their stiffness in comparison with stainless steel. With variations in material, a range of stiffness is available equivalent to that for cross sections.

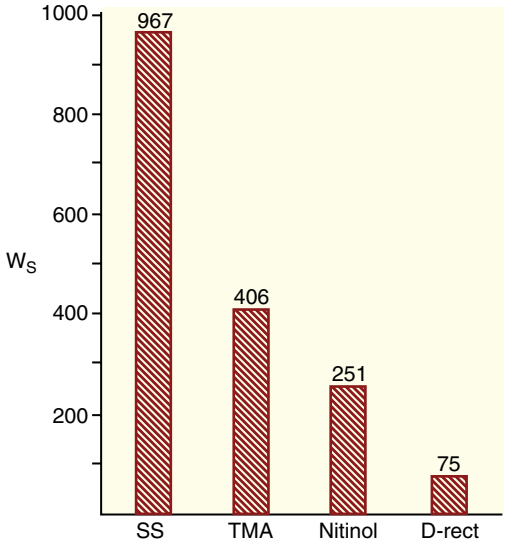


FIGURE 5-25 Wire stiffness (W_s) numbers of 0.018- × 0.025-inch wires in second-order direction. Forces for the same activations are proportionate to the W_s numbers. A full range of forces can be obtained by keeping a constant cross section (e.g., 0.018 × 0.025 inch) but using different materials.

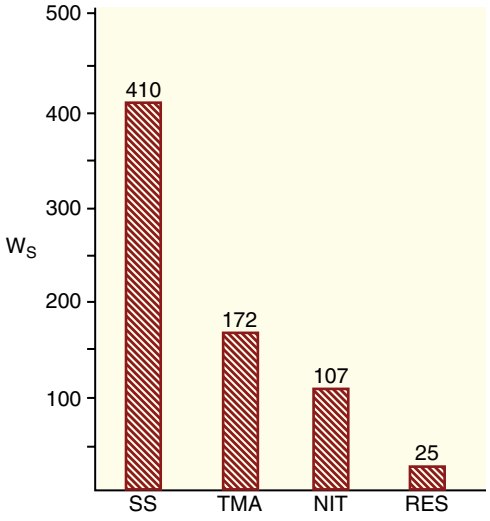


FIGURE 5-26 W_s numbers for 0.018-inch round wires. Figures 5-27 and 5-28 show wide stiffness ranges when the occlusogingival wire dimension is kept constant at 0.018 inch.

A 0.018- × 0.025-inch TMA wire has a W_s of 406.1, which is equivalent to a 0.018-inch round steel wire. Nitinol wire has a W_s of 251.4, which is similar to that of 0.016-inch steel wire. Braided wire (0.018 × 0.025 inch) with a W_s of 75.4 is similar to 0.012-inch steel wire. A full range of forces can be obtained by varying the material of the wire while keeping the cross-section the same (Fig. 5-25). The W_s numbers for 0.018-inch round wire of different materials are shown in Figure 5-26.

Using the principle of variable cross-sectional orthodontics, the amount of play between the attachments and the wire can be varied, depending on the stiffness required. With small, low-stiffness wires, excessive play may lead to lack of control over tooth movement. However, if the principle of variable-modulus orthodontics is used, the clinician determines the amount of play required before selecting the wire. In some instances, more play is needed to allow the brackets freedom of movement along the archwire. In other situations, minimal play is allowed to ensure good orientation and effective third-order movement. After the desired amount of play has been established, the correct wire stiffness can be produced by using a material with a proper M_s. In this way, the play between the wire and the attachment is not dictated by the stiffness required but rather is under the full control of the operator.

The variable-modulus principle allows the orthodontist to use oriented rectangular wires or square wires in light force and heavy force applications and stabilization. A rectangular wire orients in the bracket and thus offers greater control in

delivering the desired force system; it is easier to bend because the orientation of the wire can be checked carefully. More importantly, when placed in the brackets, the wire does not turn or twist, allowing the forces to be dissipated in improper directions.

Wire Length

The length of a member may influence the maximal elastic load and the load deflection in a number of ways, depending on the configuration and loading of the spring. The cantilever has been chosen to demonstrate the effect of length because the cantilever principle is widely used in orthodontic mechanisms. A finger spring may be visualized for the following discussion.

Figure 5-27 shows a cantilever attached at *B* with a vertical force applied at *A*. The distance *L* represents the length of the cantilever measured parallel to its structural axis. In this type of loading, the load-deflection rate varies inversely as the third power of the length; in other words, the longer the cantilever, the lower is the load-deflection rate. The maximal elastic load varies inversely as the length of the cantilever. Again, the longer the cantilever, the lower is the maximal elastic load.

Increasing the length of the cantilever is a better way to reduce the load-deflection rate than is reducing the cross section. Increasing the length of the cantilever greatly reduces the load-deflection rate, yet the maximal elastic load is not changed radically because it varies linearly with the length. Adding length within the practical confines of the oral cavity is an excellent way to improve spring properties.

Another way of loading the cantilever is shown in Figure 5-28, this time by means of a couple or moment applied to the free end. With a couple applied at the free end, the moment-deflection rate varies inversely as the second power of the length. Interestingly, the maximal elastic moment is not affected at all by changes in length. The length may be doubled or tripled, but the maximal elastic moment remains the same. This is a most desirable type of loading because additional length can reduce the moment-deflection rate, but the maximal elastic moment is not reduced. However, the principle can be applied only if moments alone are required for a given tooth movement.

Increasing the length of a wire with vertical loops is one of the more effective means of reducing load-deflection rates for flexible members and at the same time only minimally altering their maximal elastic loads. However, limitations exist on how much the length can be increased. The distance between brackets in a continuous arch is predetermined by tooth and bracket width. Vertical segments in the wire are limited by occlusion and the extension of the mucobuccal fold. An application that shows the way in which added length in a wire achieves more constant force delivery without radically sacrificing maximal elastic load is the use of a 0.018-×0.025-inch TMA intrusion arch (Fig. 5-29). The arch is used to depress maxillary anterior teeth in the correction of deep overbite.[57-59] The long distance from an auxiliary tube on the buccal stabilizing segment to the midline of the incisor is responsible for a more constantly delivered depressive force on the anterior teeth.[60,61]

Amount of Wire

Additional length of wire may be incorporated in the form of loops or helices or some other configuration. This tends to lower the load-deflection rate and increase the range of action of the flexible member. The maximal elastic load may or may not be affected.

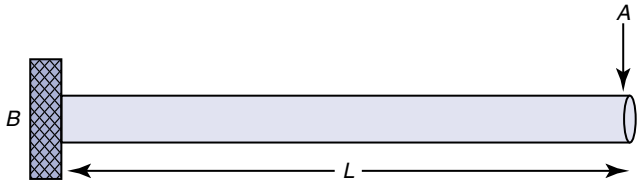


FIGURE 5-27 Cantilever (*B*) with load (*L*) applied at the free end (*A*).

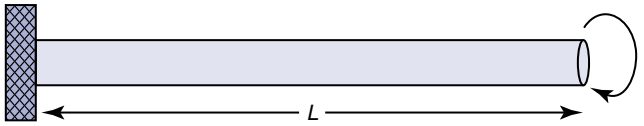


FIGURE 5-28 Cantilever with an applied couple; the effect is uniform bending along the wire.

When a member is to incorporate additional wire, the parts of the configuration where additional wire should be placed must be located properly, and the form the additional wire should take must be determined. If location and formation are done properly, lowering the load-deflection rate without changing the maximal elastic load should be possible merely by adding the least amount of wire that will achieve these ends.

Consider the problem of the cantilever in relation to the placement of the additional wire. In Figure 5-30 a cantilever is shown with the vertical force of 100 *g* at the free end. Imaginary vertical sections can be cut along the length of the wire, and at each of these sections a bending can be calculated. The bending moment is found by multiplying the load at the end of the cantilever by the perpendicular distance to the section in question; therefore, the bending moment at the point of force application at the free end of the wire is zero. Approximately 1 mm closer to the point of support, it is 100 *g*/mm. At 2 mm closer, it is 200 *g*/mm. Finally, at the point of support, the bending moment is 1000 *g*/mm. The bending moment represents an internal moment resisting the 100-*g* force applied to the free end of the cantilever. The significance of the bending moment is that the amount of bending at each cross section of the wire is directly proportionate to the magnitude of the bending moment; in other words, the greater the bending moment at any particular cross section, the more the wire is going to bend at that point.

The optimal place for additional wire is at cross sections where the bending moment is greatest. In the case of the cantilever, the position for additional wire is at the point of support because the bending moment is greatest there: 1000 *g*/mm. Helical coils can be used to reduce the load-deflection rate. Figure 5-31 illustrates the proper positioning of a helical coil for this purpose. The load-deflection rate is maximally lowered for the amount of wire used if the helix is placed at the point of support.

Placement of additional coils at the point of support in a cantilever does not change the maximal elastic load. A straight wire of a given length and a wire with numerous coils at the point of support have identical maximal elastic loads, provided they have the same length measured from the force to the point of support. This should not be surprising because the maximal elastic load is a function of this length of the configuration rather than of the amount of wire incorporated into it. This is true of many other configurations as well: the load-deflection rate can be lowered without changing the maximal elastic load

FIGURE 5-29 Intrusion arches deliver relatively constant force because of the sizable distance between the molar tube and incisor tie. **A,** An intrusion arch showing a canine bypass. **B,** A patient with deep overbite requiring intrusion; 11 mm (60 *g*) of activation. **C,** After intrusion.

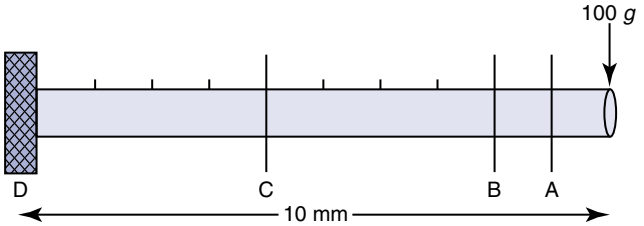


FIGURE 5-30 Cantilever with load applied at the free end. **A** to **C,** Imaginary perpendicular sections. **D,** Point of support.

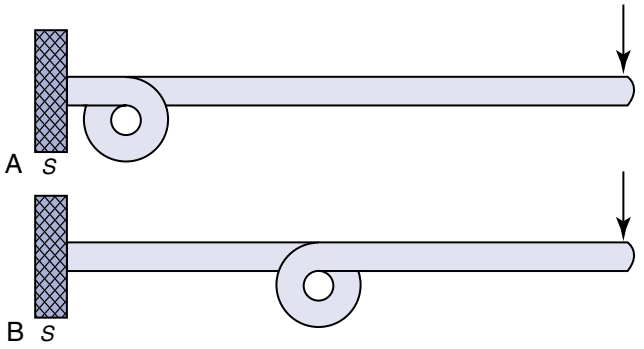


FIGURE 5-31 Placement of a helical coil in a cantilever. **A,** Correct. **B,** Incorrect. *S,* Point of support.

if additional wire is incorporated properly. This is important from a design standpoint because it provides a method of lowering the load-deflection rate without subsequently reducing the maximal elastic load.

As mentioned previously, to achieve this objective using the minimal amount of wire, the best placement of additional wire is at cross sections where the bending moment is greatest. A practical way of deciding where these parts of a wire might be is to activate a configuration and see where most of the bending or torsion occurs. The sections where the bending or torsional moments are greatest are the cross sections with the greatest stress. The configuration of the additional wire should be such that maximal advantage can be taken of the bending and torsional properties of the wire. In short, the amount of wire used is not what is important in achieving a desirably flexible member but rather the placement of the additional wire and its form.

Although additional wire is helpful in the design of flexible members, it should not be used in reactive or rigid members. Loops and other types of configurations diminish the rigidity of the wire and thus may be responsible for some loss of control over the anchor units.

Stress Raisers

From a theoretical point of view, the force or stress required to deform a wire permanently can be calculated; however, in many

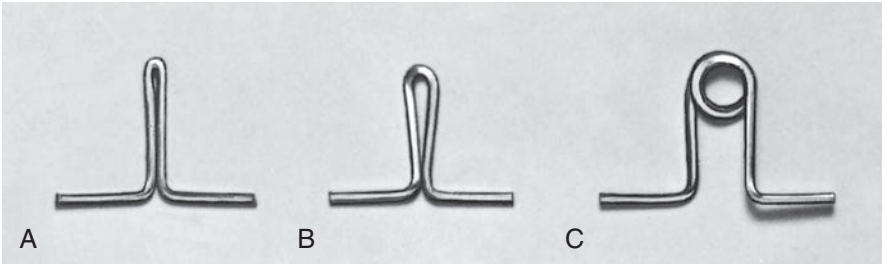


FIGURE 5-32 Vertical loops. **A,** Squashed loop. **B,** Plain loop. **C,** Loop with a helical coil.

cases the wire deforms at values much lower than the predicted one because local stress raisers increase the stress values in a wire far beyond what might be predictable by commonly used engineering formulas.

Two common stress raisers are sudden changes in cross section and sharp bends. Any nick in a wire tends to raise the stress at that cross section and hence may be responsible for permanent deformation or fracture at that point. For this reason, one should not use a file to mark a wire, particularly the small cross-sectional wires used in the flexible member of an appliance.

A sharp bend in a wire also may result in higher stress than that which might be predicted for a cross section of wire. A sudden, sharp bend deforms far more easily than a more rounded or gradual bend. Unfortunately, with a continuous archwire, the orthodontist is limited in space between brackets, and many times must make sharp bends because of this limitation. Flexible members should be designed with gradual bends so that they are less troubled by permanent deformation than comparable members with sharp or sudden bends.

For example, three vertical loops might be compared: a squashed one, a plain one, and one with a helical coil (Fig. 5-32). In terms of permanent deformation, the poorest design is loop A, which because of its squashed state has a sharp bend at its apex. The plain vertical loop (B) is slightly superior because the bending is more gradual; nevertheless, a fairly sharp bend occurs at its apex. The configuration with the most gradual bending is the loop with a helical coil (C). Not only does the helical coil enhance the flexible properties of the spring because of its additional wire, but also the lack of sharp bends further increases its range of action without permanent deformation. (Proper direction of activation is discussed later.)

Sections of Maximal Stress

Certain sections along a wire are points of maximal stress; these sections may be called *critical sections*. As discussed previously, sections in which the bending moments are greatest are areas of high stress. These critical sections are important from a design viewpoint because they are the locations where permanent deformation is most likely to occur.

A number of precautions should be observed at a critical section. First, stress raisers should be avoided in these sections at all costs. A nick in a wire, for instance, might not be so disastrous if the stress is low but might well lead to deformation or fracture where the stress level is high. Second, the EL of the wire should be watched carefully at a critical section. Operations such as soldering may overheat the wire, reducing its EL. Lowering the EL at another place in the wire where stress is low may not be too undesirable but could be responsible for failure at a critical section. Therefore, in high-stress areas an auxiliary should be attached by some means other than soldering; if soldering is used, it should be done with considerable care.

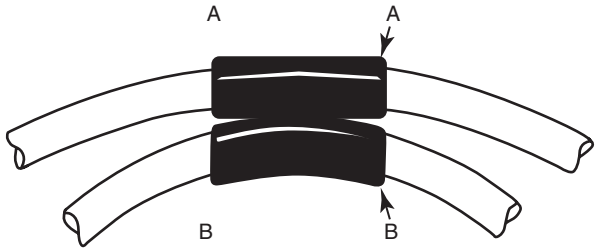


FIGURE 5-33 Face-bow: the anterior portion of the inner and the outer bow. **A,** The juncture of the solder and the outer bow is a stress point. **B,** Another stress point is found at the juncture of the solder and the inner bow.

An example of permanent deformation or fracture produced by a sudden change in cross section and a lowering of the EL can be seen in the face-bow in Figure 5-33.[62,63] A stress point is found at the juncture of the solder and the outer bow (A) and secondarily at the juncture of the solder and the inner bow (B). At A, the wire may be structurally weak for two reasons: the stress point associated with the sudden change of cross section and a lowering of the EL because of the soldering operation. A also happens to be a critical section where stress is high; therefore, it is a predictable area of fracture in a face-bow of this design.

If the orthodontist is in doubt about which parts of an appliance have critical sections, the appliance can be activated in a typical manner and the parts that exhibit the most bending or torsion noted. These generally have high stress along their cross sections.

One must keep in mind three rules in the design of critical sections: (1) all stress raisers should be eliminated as much as possible, (2) a larger cross section can be used to strengthen this part of the appliance, and (3) the appliance may be so designed that it elastically rather than permanently deforms under normal loading. Many times, a highly flexible member is more serviceable than a rigid one because the flexible member can deflect out of the way of the oncoming load. A light, flexible spring can withstand occlusal trauma far better than a more rigidly constructed one because it can displace elastically away from an occlusal force. Because the wings of airplanes are flexible, they are less apt to fracture under normal flying conditions. Similarly, increasing the flexibility of a member may be a way of preserving the integrity of the appliance.

Direction of Loading

Not only is the manner of loading important, but also the direction in which a member is loaded can influence its elastic

properties greatly. If a straight piece of wire is bent so that permanent deformation occurs and an attempt is made to increase the magnitude of the bend, bending in the same direction as had originally been done, the wire is more resistant to permanent deformation than if an attempt had been made to bend in the opposite direction. The wire is more resistant to permanent deformation because a certain residual stress remains in it after placement of the first bend. A flexible member will not deform as easily if it is activated in the same direction as the original bends were made to form the configuration. If a bend is made in an orthodontic appliance, the maximal elastic load is not the same in all directions; it is greatest in the direction identical to the original direction of bending or twisting. The phenomenon responsible for this difference is known as the Bauschinger effect.

Figure 5-34 shows a vertical loop with a coil at the apex and a number of turns in the coil under different directions of loading. The type of loading in A tends to wind the coil, increasing the number of turns in the helix and shortening the length. The type of loading in B tends to unwind the helix, reducing the number of coils and lengthening the spring. The loading in Figure 5-34, *A*, tends to activate the spring in the same direction as it originally was wound and thus is the correct method of activation. In many configurations in which residual stress is high, such as a vertical loop that uses a number of coils at the apex, the range of action can vary 100% or more between correct and incorrect loading. Obviously this is a much more significant factor in design than are small differences in the mechanical properties of the wire.

The same principles can be applied to less complicated configurations, such as a continuous archwire. The orthodontist should be sure that the last bend in an archwire is made in the same direction as the bending produced during its activation. For example, if a reverse curve of Spee is to be placed in an archwire, the curve first should be overbent and then partly removed; only then will activation of the archwire occur in the same direction as the last bend (Fig. 5-35). The same is true of a series of tip-back bends. The tip-back bend should be increased beyond the amount required, and then some of it should be removed. In this way, bending during activation of the tip-back bend when it is placed in the brackets occurs in the same direction as the last bend.

Attachment

If forces and moments are to be delivered to a tooth, some means of attachment are necessary. If forces alone were sufficient without the use of moments, the attachment could be relatively simple; however, this usually is not the case. Most orthodontic movements require moments and forces. Moments and forces can be produced if a noncircular wire is oriented in a noncircular bracket. The edgewise bracket and tube are excellent examples of the use of a noncircular cross section for wire and attachment. However, loops can be used to obtain an orientation of round wire that allows moments and forces to be delivered.

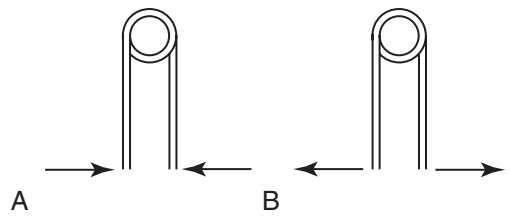


FIGURE 5-34 Activation of a loop with a helical coil. **A,** Correct. **B,** Incorrect.

What are the optimal dimensions for a bracket or tube? This question has no definitive answer unless the objectives and design of an orthodontic appliance are specified fully. However, some of the factors involved in making this decision can be discussed.[64]

The starting point in the design of a bracket is determining the width of the bracket (mesiodistal dimension). From a theoretic viewpoint, a system of forces and moments can be produced regardless of the mesiodistal width of the brackets. However, width becomes important for two reasons. First, wider brackets minimize the amount of play between the arch wire and attachments. A certain amount of leeway must exist between the archwire and the bracket, or easy bracket engagement becomes impossible. However, if a bracket is too narrow, considerable play may exist in all planes between wire and bracket. If the bracket is wider, the archwire has a much more positive purchase. Second, the greater the distance between the brackets, the lower the load-deflection rate. Because at least part of the movement required to treat a patient is produced by adjustments between brackets, it is desirable that the distance between attachments be as great as possible. One of the problems of the continuous arch is the sharp limitation of space between brackets, no matter how narrow the brackets might be.

The decision on proper bracket width lies between two extremes. At one end, a bracket might be as wide as the tooth. In this instance, however, the interbracket distance is not enough to produce sufficient flexibility for adjustment. At the other end, a knife-edge bracket offers the greatest interbracket distance and thus the most desirable load-deflection rates. With a knife-edge bracket, however, delivering the necessary moments and forces to achieve full control over tooth movement is impossible.[65] Generally speaking, the ideal bracket width is one that is as narrow as possible, yet still capable of obtaining positive purchase on an archwire so that moments can be delivered to teeth.

Optimal occlusogingival slot dimensions are determined by the maximal elastic loads required from the active and reactive members. A safe rule is to design based on the reactive members, ensuring that the bracket and tube slots are large enough for rigid control of anchor units. Designing primarily based on the active members is a mistake that may lead to the use of slots that are too small to control the anchor teeth and withstand the forces of mastication.

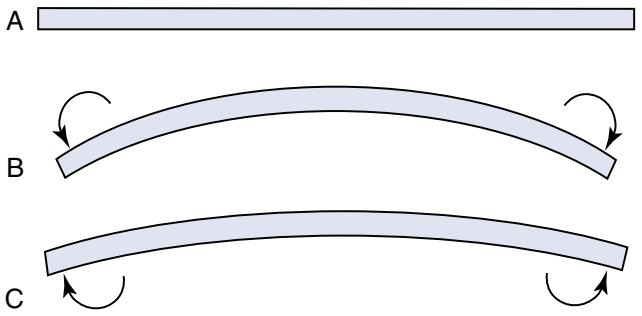


FIGURE 5-35 Placing a reverse curve of Spee in a lower arch.* **A,** Original straight wire. **B,** Wire overbent. **C,** Final configuration. Note that the last bends are in the same direction as the activation in the mouth.

A vexing problem in orthodontic treatment arises when appliance requirements change between stages of treatment. Teeth that at one time are being moved actively may later become reactive units. For convenience, in the typical strap-up, the same dimensions are used in all slots throughout the arch; yet the active and reactive requirements are not the same throughout the arch. One objective in the design of an appliance is to ensure a positive fit between different cross sections of wire, depending on the needs of the case. An edgewise bracket can become adjustable in a buccolingual or labiolingual direction by means of a ligature tie, but it is not adjustable occlusogingivally. Thus, no definite answer can be given to the question of optimal occlusogingival slot dimension.

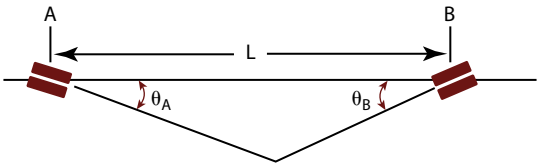


FIGURE 5-36 The geometry of the wire to an edgewise bracket is defined by the interbracket distance (L) from the angles of the brackets with respect to the interbracket axis. (From Burstone CJ, Koenig HA. *Am J Orthod.* 1974;65:270. With permission from the American Association of Orthodontists.)

Class

	I	II	III	IV	V	VI
$\frac{\theta_A}{\theta_B}$	1.0	0.5	0	−0.5	−0.75	−1.0
Lower left quadrant						

FIGURE 5-37 The six basic geometries based on the ratio q_A/q_B. Classes are independent of interbracket distance. Position *A* is the canine; position *B*, the premolar.

The decision depends on many factors, including the general concept of treatment and the basic design of an orthodontic appliance.

Forces from a Continuous Arch

A multibanded appliance, such as that used in edgewise mechanics, produces a complicated set of forces and moments. For example, a straight (ideal) arch placed between irregular brackets on malaligned teeth may deliver desirable and undesirable forces. An analysis of two-tooth segments (two teeth connected by a straight wire) can demonstrate some of the problems encountered when adjacent brackets are connected by a continuous wire or arch.[4,6,66-70]

The force system produced in a two-tooth segment is determined by the angle of the bracket (θ_A and θ_B) with respect to the straight wire and the interbracket distance (Fig. 5-36). Based on the ratio θ_A/θ_B, six classes of force systems can be described (Fig. 5-37). The force system for each class is given in Table 5-5. Note that the ratio of the moments at bracket A with respect to bracket B is constant for each class. Lines 2 and 3 of Table 5-5 give the force systems acting on the wire; line 5 reverses the direction, showing the forces acting on the teeth. What is apparent is that forces, moments, and their ratios may not be correct to produce the desired changes in a malocclusion without side effects.[71,72]

For example, Figure 5-38, *A*, shows a Class I geometry in which the second premolar is supererupted in relation to the first molar. A straight wire placed on the premolar produces a desirable intrusive force; unfortunately, however, the moment produced on the premolar is undesirable, displacing the root mesially. This can be avoided by using a noncontinuous configuration, the rectangular loop. Figure 5-38, *B*, shows the force system from the rectangular loop, which can be designed to produce an intrusive single force and no moment on the premolar. Figure 5-39 shows a rectangular loop used to rotate and extrude a premolar. Rectangular loops and other loop designs offer the potential for delivering desired force systems with

TABLE 5-5 Force Systems by Class

Class	I	II	III	IV	V	VI
$\frac{\theta_A}{\theta_B}$	1.0	0.5	0	−0.5	−0.75	−1.0
$\frac{M_A}{M_B}$	1.0	0.8	0.5	0	−0.4	−1.0
Force system on wire at yield (L = 7 mm)	531.4↑ ↓531.4 1860 1860	477.4↑ ↓477.4 1468 1860	398.0↑ ↓398.0 930 1860	265.7↑ ↓265.7 1860	160.0↑ ↓160.0 740 1860	1860 1860
Force system on wire at yield (L = 21 mm)	177.0↑ ↓177.0 1860 1860	160.0↑ ↓160.0 1468 1860	133.0↑ ↓133.0 930 1860	88.6↑ ↓88.6 1860	53.3↑ ↓53.3 740 1860	1860 1860
Relative force system on teeth (L = 7 mm)	531.4↑ ↓531.4 1860 1860	477.4↑ ↓477.4 1468 1860	398.0↑ ↓398.0 930 1860	265.7↑ ↓265.7 1860	160.0↑ ↓160.0 740 1860	1860 1860

(From Burstone CJ, Koenig HA. Force systems from an ideal arch. *Am J Orthod.* 1974;65:270. With permission from the American Association of Orthodontists.)

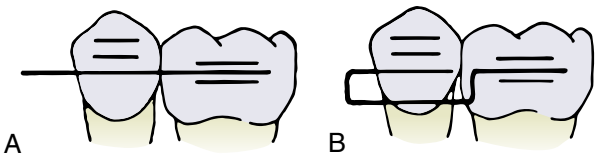


FIGURE 5-38 A, A straight wire (ideal arch) between the first molar and premolar produces an undesirable positive movement that displaces the premolar root mesially. B, A rectangular loop used for the same bracket malalignment produces no side effects on the premolar because only an intrusive force is delivered.

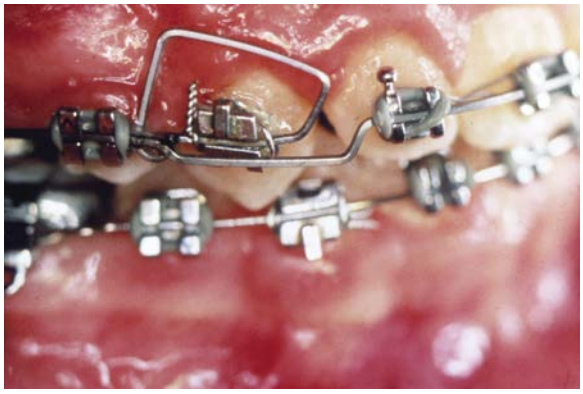


FIGURE 5-39 A rectangular loop is used to rotate and extrude the upper cuspid without a side effect. Note bypass arch for added anchorage.

minimal side effects, which is not usually possible with the continuous arch.[57,58] Loops are not used only to lower forces; they change the entire force system.

Straight wires may reduce desirable or undesirable force systems. The undesirable components of the forces can produce unwanted tooth movement during the leveling process. These side effects can be eliminated by the placement of suitable bends, use of bypass arches, and selection of anchorage teeth not adjacent to malaligned teeth.[60,61,73-75] In addition, forming properly designed loops can change a force system from the straight wire.

Figure 5-40, *A*, shows a canine with its root apex forward. A straight wire, as it moves the root distally, also erupts the incisors, producing deep overbite. To prevent this side effect, a bypass arch was constructed. In Figure 5-40, *B*, the continuous 0.018-inch stainless-steel arch steps occlusally bypass the canine. The axial inclinations have been corrected by a 0.017- × 0.025-inch TMA cantilever root-spring. The reactive forces have been distributed to the whole arch rather than to the incisors. Final leveling can be carried out safely with a straight wire (Fig. 5-40, *C*).

With a high canine, as in Figure 5-41, *A*, a straight wire tends to tip the buccal segment toward the canine. To avoid this effect, a bypass arch was formed so that the entire arch was an anchorage unit. A separate NiTi arch from auxiliary tubes on the first molars brought the canine into alignment (Fig. 5-41, *B*). Instead of the NiTi secondary arch, a cantilever can be used from the molar auxiliary tube (Fig. 5-42).

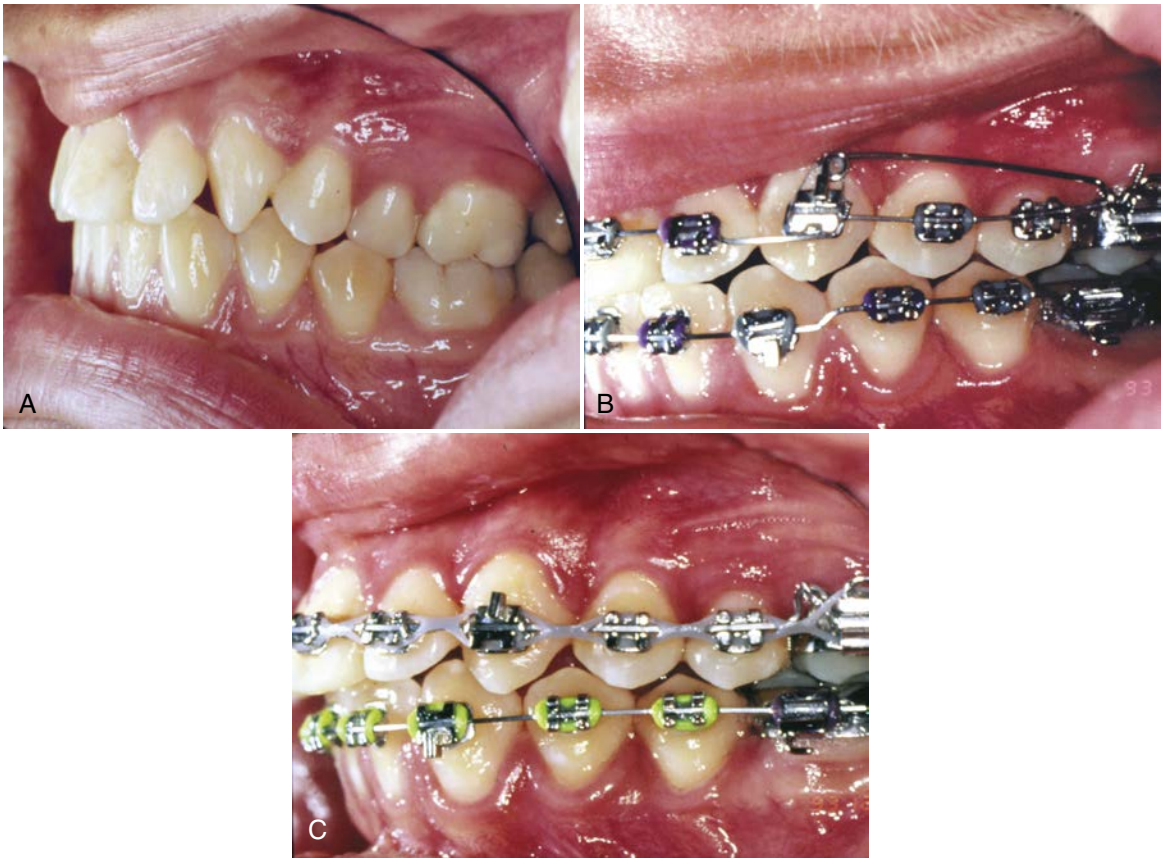


FIGURE 5-40 A 0.017- × 0.025-inch TMA root spring is used to move the canine root distally before final leveling. A, Before treatment. B, After root correction. C, After final leveling.

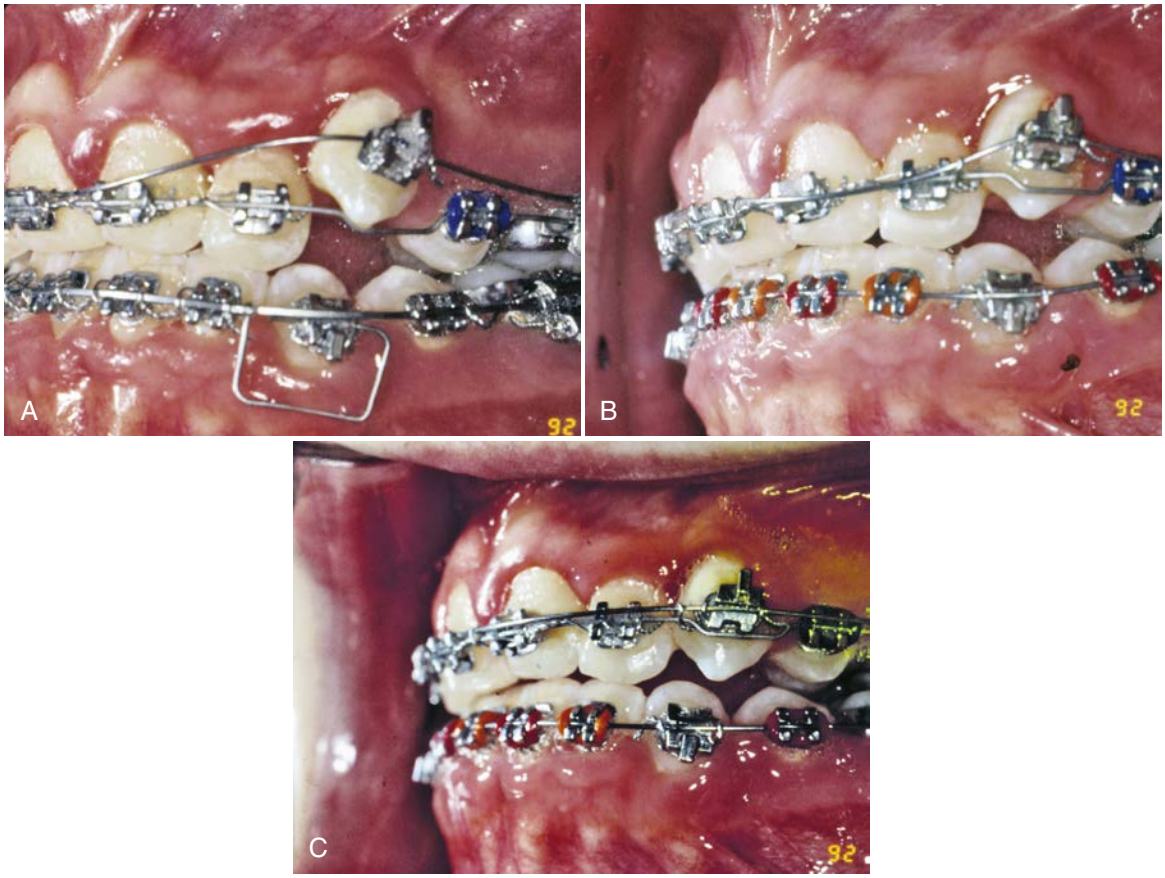


FIGURE 5-41 A cuspid bypass arch prevents side effects. A separate, secondary NiTi wire erupted the cuspid. **A,** Before. **B,** Intermediate. **C,** After.

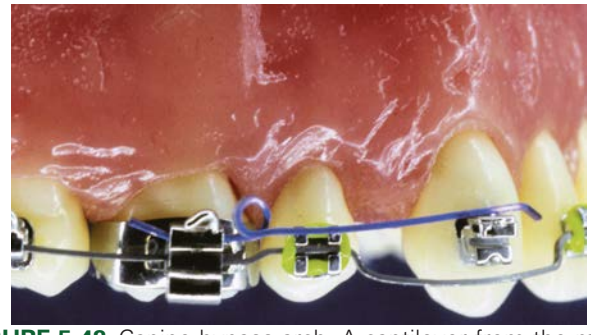


FIGURE 5-42 Canine bypass arch. A cantilever from the molar tube erupts the canine.

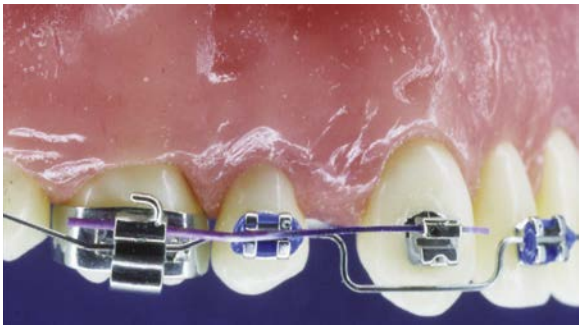


FIGURE 5-43 Canine bypass arch made of 0.016-×0.022-inch TMA wire torques the canine root lingually. The wire is placed ribbonwise in the canine bracket.

A 0.018-×0.025-inch TMA wire connects auxiliary tubes on the canine and first molars to torque the root of the canine lingually (Fig. 5-43). The long span reduces the torque-twist rate, increasing the range of action and delivering more constant torque. The bypass arch gives full anchorage control.

Teeth other than the canine can be bypassed. With the high central incisor, a straight wire tends to tip the adjacent teeth toward the high incisor (Fig. 5-44). A continuous bypass arch with a secondary NiTi wire is used to extrude the incisor without distributing forces to the adjacent teeth.

If a straight wire is used with first molars that are rotated mesially, the side effects can include molar expansion, arch form changes, and prevention of molar distalization. A lingual or transpalatal arch that delivers equal and opposite couples uses reciprocal anchorage and thus more efficiently rotates molars without side effects. This is an example of selecting teeth for anchorage and not relying on a continuous wire that connects adjacent teeth. Connecting only two teeth improves the accuracy of activation. Figure 5-45 shows a 0.032-inch, round, TMA transpalatal arch that attaches to a precision hinge-cap lingual attachment.[76] Equal rotation bends produce equal and opposite couples with no horizontal forces.

Principles of Spring Design

An understanding of the relationships between bioengineering parameters and force systems can offer a rational basis for the design of orthodontic appliances. To achieve an optimal force

system, not only must the force magnitude be correct, but also the force must be delivered constantly, and the required M/F ratio must be produced to control the center of rotation. In this section, these factors are considered together, using the example of an anterior or canine retraction spring.[57,58,77,78]

For a canine that needs translation, or bodily movement, a force must be applied through the center of resistance of the tooth (Fig. 5-46). If the force is 200 *g*, 200 *g* must be delivered at the bracket, as well as a moment of 2000 *g*/mm (provided the distance between the bracket and the center of resistance is 10 mm). In other words, a 10:1 M/F ratio is produced. If a simple vertical loop is used for space closure, a moment that encourages the root to move distally is provided during activation. For a loop 6 mm long, the M/F ratio typically is low, about 2.2:1.24. This ratio is too low to control the root and prevent it from being displaced mesially. A number of strategies can be used to increase the M/F ratio during activation.[75,79] The loop can be made as long as possible in an apical direction. Increasing the length of the loop to 11 mm approximately doubles the M/F ratio. However, a loop can be extended apically only so far before it causes irritation in the mucobuccal fold. Another strategy involves increasing the amount of wire found gingivally at the top of the loop. Figure 5-47 shows that by increasing the gingival amount of wire (dimension G), the M/F ratio is increased and the load-deflection rate is reduced. One advantage of the T-loop design over a simple vertical loop is that the T-loop produces a much higher M/F ratio to control the root and a low load-deflection rate, thereby ensuring greater force constancy.[77,80,81]

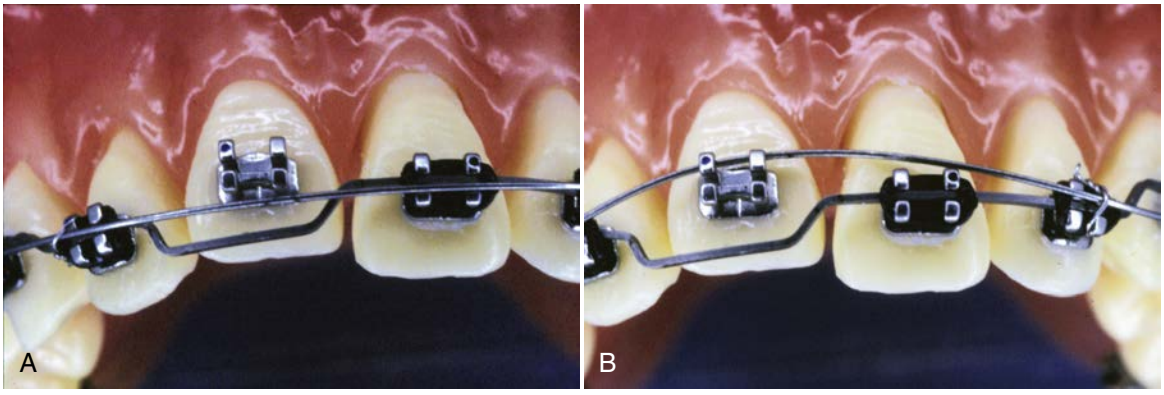


FIGURE 5-44 Incisor bypass arch. A secondary NiTi wire erupts the central incisor without side effects on the adjacent teeth. **A,** Activated. **B,** Passive.

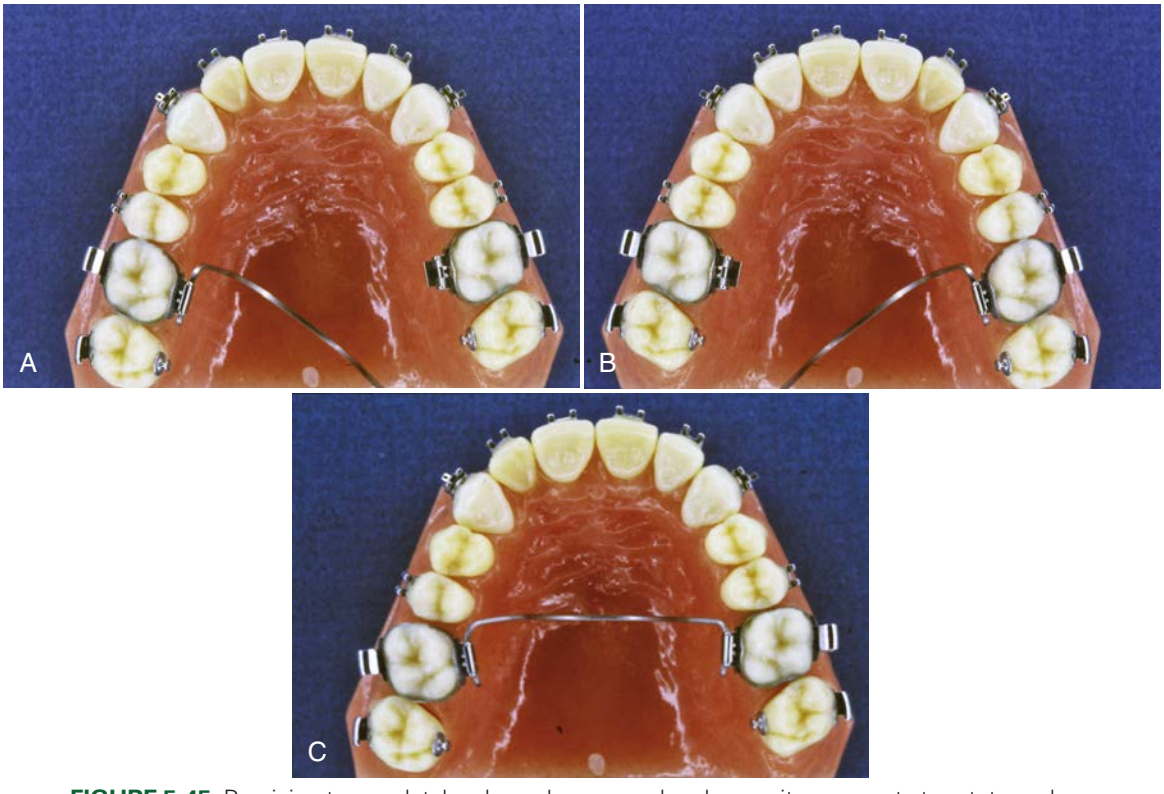


FIGURE 5-45 Precision transpalatal arch produces equal and opposite moments to rotate molars. If both arches are angled equally, no horizontal forces are produced **(A, B). C,** Transpalatal arch inserted showing rotated molars.

The moment produced by a retraction spring during activation is called the *activation moment*. The activation moment depends on the change in angle that the horizontal arms of the spring make with the bracket when a loop is pulled apart. Even if the design is improved by use of a configuration such as a T-loop, the M/F ratio may not be high enough to affect translation. To achieve a higher M/F ratio, an angulation or a gable-type bend must be put in the spring. The moment produced by gabling is known as the *residual moment*. Ideally, a retraction spring delivers a relatively constant M/F ratio. If the ratio changes each time the tooth moves, the tooth will not have a constant center of rotation. Two principles to remember in obtaining a constant M/F ratio are to (1) use the highest activation moment and the lowest residual moment that are possible and to (2) lower the force-deflection and moment-deflection rates.

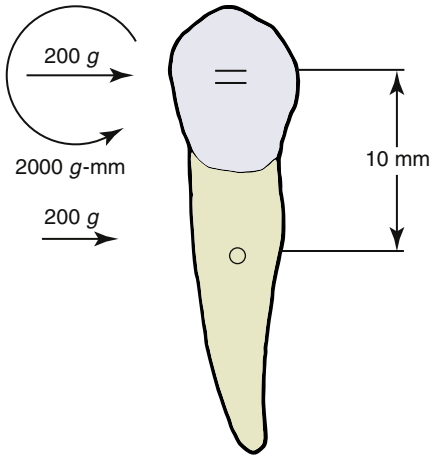


FIGURE 5-46 A force acting at the center of resistance (CR) of a tooth translates the tooth. A couple (moment) and a force (*white arrows*) acting at the bracket can produce the same effect. Note that the magnitude of the moment is equal to the force multiplied by the distance from the bracket to the center of resistance. (Redrawn from Burstone CJ, Koenig HA. *Am J Orthod.* 1976;70:1. With permission from the American Association of Orthodontists.)

An important factor in the use of a loop for space closure is the mesiodistal position of the loop. If the loop is placed midway between the attachments, equal and opposite activation moments are produced.[79] If the loop is positioned off-center to the distal, the posterior teeth receive a larger tip-back moment. In addition, intrusive forces are delivered anteriorly. This concept can be applied in anchorage cases when mesial movement of posterior teeth is not indicated. Conversely, placing a loop mesially off-center increases the moment to the anterior teeth and can be useful in bringing buccal segments forward.

Use of a T-loop for en masse space closure is shown in Figure 5-48. Both the anterior and posterior segments are steel; the active T-loop is TMA, which lowers the force-deflection rate and increases the range of activation. The loop (spring) is attached to auxiliary tubes on the first molar and canine. Centering the spring produces approximately equal translation of posterior and anterior segments during space closure. T-loops were used to affect differential space closure, to hold anchorage, and to correct deep overbite (Fig. 5-49). The upper loop was placed distally off-center. Note that the upper-anterior teeth have tipped somewhat lingually; this is to be expected because the moment to the incisors is sufficient to produce only tipping around their apices, not translation. A separate stage of en masse anterior root movement is required after space closure.

One of the problems with using sliding mechanics for canine retraction is the unpredictability of frictional forces. Much of the friction arises from the tendency of the wire to prevent tipping and rotation. In Figure 5-50, a T-loop is used to deliver force during canine retraction. The loop also produces moments that prevent tipping and rotation; hence, friction is reduced. Another approach to reducing friction with sliding mechanics is shown in Figure 5-51; flexible 0.016- × 0.022-inch TMA extensions with helices attached to canine and molar

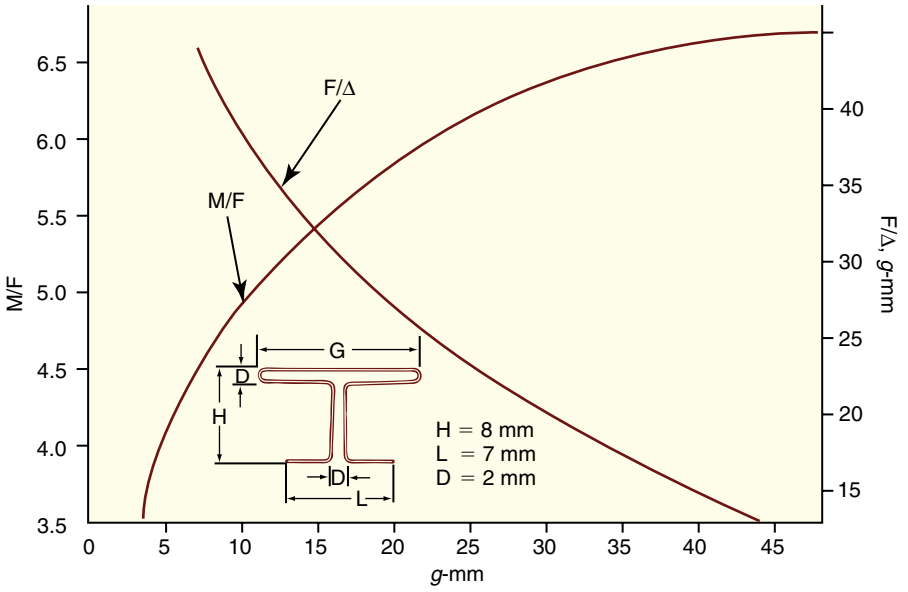


FIGURE 5-47 As the gingival horizontal length increases, the moment-to-force ratio increases and the F/D continues to decrease. (Redrawn from Burstone CJ, Koenig HA. *Am J Orthod.* 1976;70:1. With permission from the American Association of Orthodontists.)

auxiliary tubes place the force closer to the center of resistance of the canine. Unlike rigid "power" arms, the extensions have a relatively low force-deflection rate of 40 *g*/mm and a typical activation of 6 mm.

Fiber-Reinforced Composite Applications

The design of fixed orthodontic appliances historically has required materials with excellent engineering properties such as metals and ceramics. Polymers, although user friendly, are structurally weak and hence have limited use, mainly in removable appliances.

A new generation of materials that incorporates long fibers, such as glass or carbon, has been developed that possesses properties approaching those of metals. To optimize the mechanical properties, fibers are wetted and oriented, and the plastic matrix is added under controlled manufacturing conditions. Wires or strips that are impregnated by the matrix and are unpolymerized are called *pre-pregs*. These wires can be shaped passively to the shape of the arch and, when light-cured, harden to form a stiff orthodontic component.[82,83]

The application of a fiber-reinforced composite (FRC) is shown in Figure 5-52. The upper-left second molar has tipped forward into the first molar extraction space, and the treatment plan calls for some mesial molar movement to close the space and mesial root movement to correct the axial inclination. Traditionally, metal or ceramic brackets are placed on all the anterior teeth to afford sufficient anchorage. In this patient, a pre-preg composed of long fiberglass and Bis-GMA resin was formed to the shape of the upper arch and then light-cured to form a metal-like rigid, strong, and passive anchorage unit. Attachments were placed only on the second molar and on the FRC in the region of the first bicuspid. A simple wire segment connects the two attachments. The long interattachment span and the use of only a few brackets increase biomechanical efficiency and patient comfort. The correction of the upper-second molar axial inclination and space closure is shown in Figure 5-52, *B*. An additional FRC has been placed in the lower arch to facilitate correction of the terminal molar. Following major corrections with an FRC segment, some but not all brackets may be needed for finishing detail.

The development of FRC segments, or arches, offers a new dimension in appliance design. Anchorage is enhanced because play is eliminated between wire and the bracket. Because these wires accurately fit the teeth, the possibility of undisturbed teeth and a completely passive wire is now possible. In addition, the glass fibers are good for anterior aesthetics.

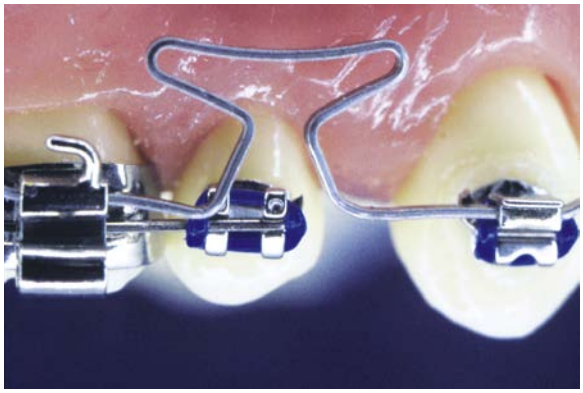


FIGURE 5-48 Centered T-loop is connected to auxiliary tubes on the canine and molar.

The Role of Friction

Tooth movement is determined by the total forces applied to the teeth. This includes not only forces from the appliance but also muscular forces that are produced during function. In addition, frictional forces can operate that can considerably alter the force system.

The mechanical principles and their application that govern friction are complicated and beyond the scope of this chapter. However, a discussion of some basic principles can be very useful for the clinician. The classic formulas below define a relationship between friction force (F_F), coefficient of friction (μ), and forces operating at 90 degrees to the archwire (i.e., normal forces [F_N]). M is a moment in loading by a couple and W is the bracket mesial–distal width (Fig. 5-53).

$$F_F = \mu \times F_N$$

$$F_F = 2\mu \cdot M/W$$

Thus, friction force is produced by many possible appliance activations: buccal, lingual, apical, and occlusal forces. Friction force is also produced by moments acting on the archwire by "tip"

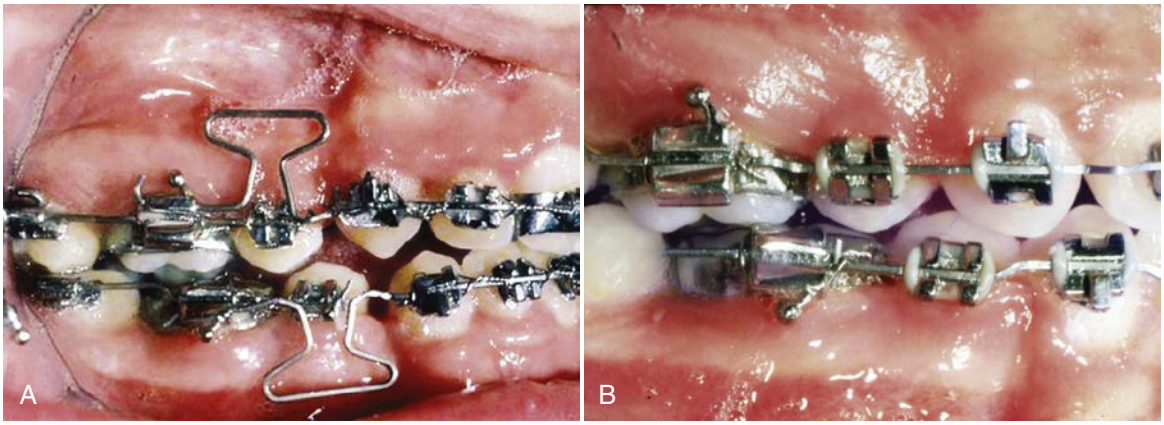


FIGURE 5-49 Differential space closure. Greater tip-back moment was achieved on the posterior teeth by putting the loop distally off center. **A,** After en masse anterior tipping. **B,** After en masse anterior root movement.

or "torque." The ligation mechanism produces normal forces adding to the friction force. The purpose of ligation is to keep the archwire from being displaced from the bracket. Any additional ligation force will add to the friction force and usually is not desired. The coefficient of friction (μ) is determined by such factors as the material, the material interface if more than one material is used, and lubricants. From an orthodontic perspective, the force system used is the main determinant of the friction force.

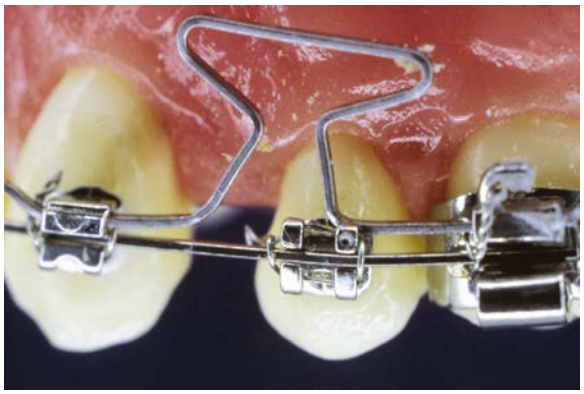


FIGURE 5-50 Canine retraction using sliding mechanics. T-loop moments minimize friction.

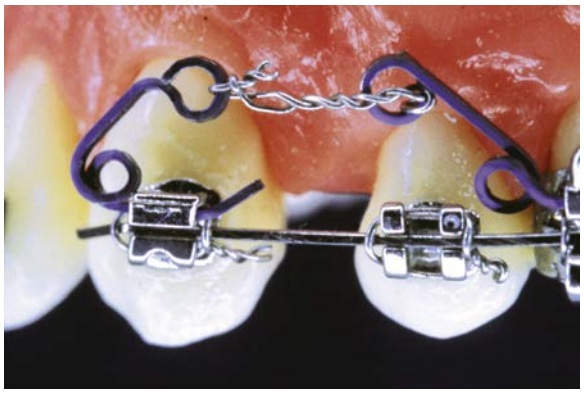


FIGURE 5-51 Canine retraction using sliding mechanics. Flexible extensions deliver more constant force, and the force is closer to the center of resistance of the canine.

Which bracket will produce the most friction force: a narrow or wide bracket? It depends on the stage of tooth movement. If there is initially play between the wire and the bracket, there is little friction—hence, a narrow bracket produces less friction until the wire engages the bracket. Once there is engagement with a single force (e.g., an occlusal force), bracket width does not make any difference if the force magnitude is the same. With torque and tip moments, for the same magnitude moment, the wider bracket produces less friction.

In special situations such as canine retraction, different stages of tooth movement can be identified during sliding mechanics. Initially, the canine will tip; later, the tooth will translate followed by root correction. Translation requires a larger moment with a distal force, and therefore, frictional forces are greater during the translational stage of retraction. Although there has been much discussion about the role of the bracket ligation method as the cause of friction, it should be recognized that most friction is produced by the forces required to correct the malocclusion. Particularly, moments produce large frictional forces since the bracket is far from the center of resistance at the root; hence, for translational or bodily movements, high moments are a necessity.[84]

Friction can be either good or bad depending on the application. With too much friction, force is lost and tooth movement can be reduced. On the other hand, commonly too much force is used and friction reduces the force to more acceptable biological levels. Friction during space closure can also be helpful in reducing tipping.

When heavier normal forces are applied to an archwire, wire abrasion and indenting can occur. Calculation of the friction force becomes more complicated, and the simple, classic formulas are not applicable. It has been demonstrated that vibration or cyclic loading can reduce friction associated with the ligation force.[85]

3D Biomechanics

Orthodontic treatment has always been in three dimensions; limitations in methodology and visualization have been responsible for two-dimensional thinking. Now, with widespread applications of 3D imaging, the necessity of 3D force analysis becomes imperative.

In three dimensions, the center of resistance becomes an axis of resistance. Unlike a center of mass, in three dimensions all axes in a restrained body may not intersect at a point. This can

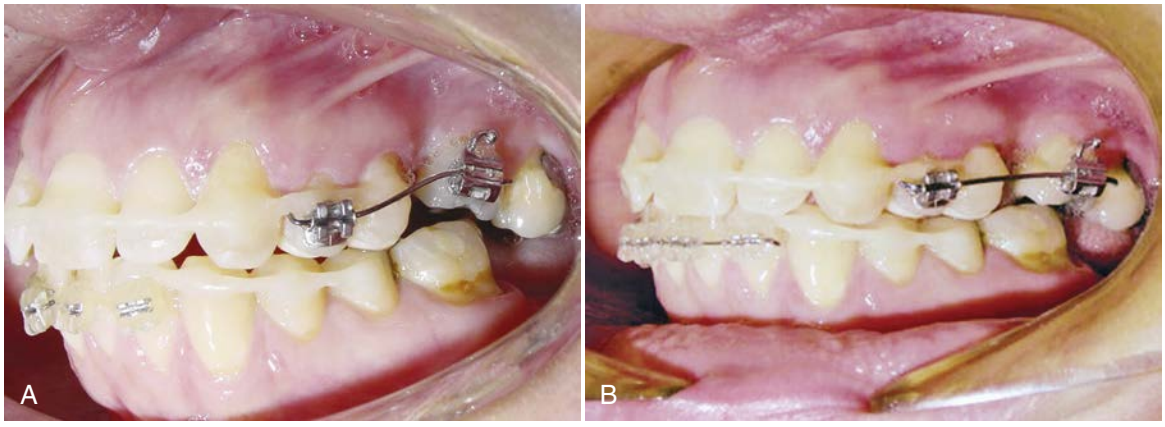


FIGURE 5-52 A fiber-reinforced composite wire (FRC) is used for anchorage instead of brackets to upright the upper-left second molar. The aesthetic FRC connects rigidly and passively the anterior teeth. **A,** Before. **B,** After. (Courtesy Philip Depasquale, Avon, CT.)

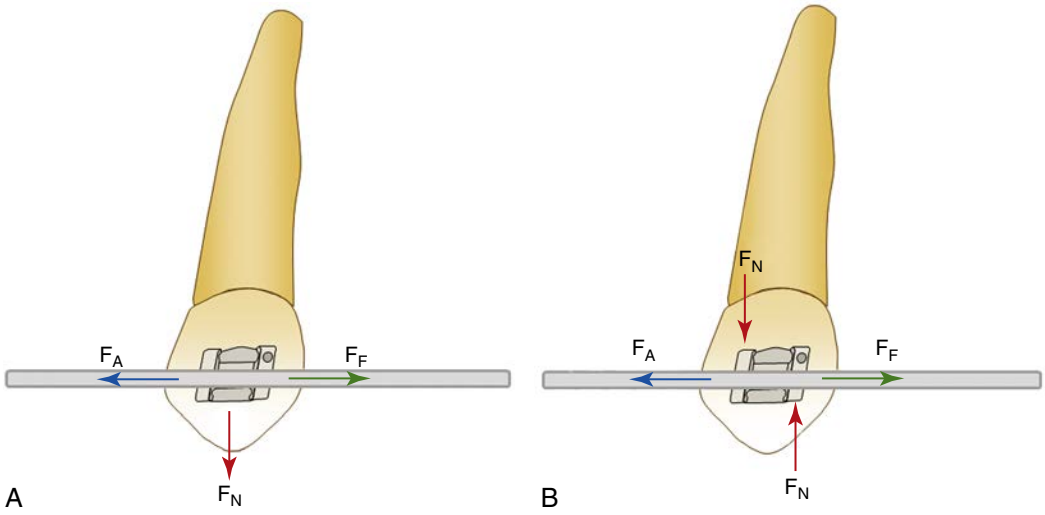


FIGURE 5-53 Normal forces (F_N) in respect to an archwire produce friction forces (F_F). F_A is the applied force. The force that the tooth feels is the applied force minus the friction force. **A,** F_N is a single occlusal force. **B,** A couple or pure moment is applied to the bracket of the canine. These normal forces also lead to friction.

occur if there is asymmetric root morphology or PDL constitutive behavior is asymmetric. Thus, the center of resistance concept becomes a useful fiction.[86]

Also, much variation can exist in generally finding a center of resistance. Therefore, the center of resistance can be considered instead of a point as a sphere. Most likely, in most clinical situations, the radius of a sphere encompassing all intersecting axes is relatively small; however, more research is need. The clinician can still visualize one plane at a time for convenience. It should be remembered that different projections are required in any study to properly localize all axes in three dimensions.

In this chapter, for purposes of simplicity, the discussion and diagrams have shown only one plane. The clinician, however, must think three dimensionally if major side effects are to be avoided.

SUMMARY

Design of an orthodontic appliance requires a thorough understanding of biological and physical variables. Design is an area in which the concepts of biology and physics can be wedded to form a true biomechanical discipline.

Although the author's purpose is not to attempt a cookbook approach to appliance design, there is merit in pointing out the steps that may go into the design of an orthodontic appliance:

1. The orthodontist, as a designer, must make certain assumptions about the nature of forces and tooth movement. Objectives relative to tooth movement must be biologically sound, recognizing not only the tissue changes around the tooth but also the generalized growth changes in the face. The biological objectives of treatment must come first because without them appliance design has no basis.
2. A basic configuration must be selected, depending on the limitations of space in the oral cavity. The configuration should be determined partly by patient hygiene and comfort. Most important, the configuration must be capable of delivering the required force system, should have a desirable maximal elastic load and load-deflection rate, and must deliver the needed M/F ratio. The members should be analyzed so that stress distribution at critical sections does not result in failure.
3. After the general configuration has been established, its dimensions can be determined. Length is established consistent with patient comfort and oral hygiene.
4. When the dimensions of a component are known, the type of material or alloy from which it is to be made can be chosen.[87]
5. Finally, the cross section of the wire is determined.

This is not to suggest that appliances can always be designed by following the same logical sequence. The many variables in appliance design are related and cannot be separated. Furthermore, this list of steps is not meant to suggest that all that is needed in designing an appliance is a group of engineering formulas. Background in the physical sciences can help in the design of appliances, but appliance development still requires a certain amount of intuition, as well as clinical and laboratory experimentation. Basic science, rather than trial and error, offers the greatest possibilities in the development of the orthodontic appliances of the future.

This chapter has discussed the scientific basis of biomechanics and clinical orthodontics. It includes concepts and terminology that are common to physics and engineering. If orthodontists can speak the same language as the physical scientists, they have a better chance to integrate biology and physics as a true science of orthodontic biomechanics is developed.

REFERENCES

1. Burstone CJ. Orthodontics as a science: the role of biomechanics. *Am J Orthod Dentofac Orthop*. 2000;117(5):598–600.
2. Burstone CJ. Biomechanical rationale for orthodontic therapy. In: Melsen B, ed. *Controversies in Orthodontics*. Chicago: Quintessence; 1991.
3. DeFranco JC, Koenig HA, Burstone CJ. Three-dimensional large displacement analysis of orthodontic appliances. *J Biomech*. 1976;9:793.
4. Koenig HA, Burstone CJ. Analysis of generalized curved beams for orthodontic applications. *J Biomech*. 1974;7:429.

5. Solonche DJ, Burstone CJ, Yeamans E. An automated device for determining load-deflection characteristics of orthodontic appliances. In: *Proceedings of the Twenty-Fifth Annual Conference of engineering in Medicine and Biology*. ; 1972.
6. Timoshenko S, Goddier JN. *Theory of Elasticity*. 2nd ed. New York: McGraw-Hill; 1951.
7. Melsen B. Biological reaction of alveolar bone to orthodontic tooth movement. *Angle Orthod*. 1999;69(2):151–158.
8. Melsen B. Tissue reaction to orthodontic tooth movement: a new paradigm. *Eur J Orthod*. 2001;23(6):671–681.
9. Burstone CJ, Pryputniewicz RJ. Holographic determination of centers of rotation produced by orthodontic forces. *Am J Orthod*. 1980;77:396.
10. Burstone CJ, Pryputniewicz RJ, Bowley WW. Holographic measurement of tooth mobility in three dimensions. *J Periodont Res*. 1978;13:283.
11. Kusy RP, Tulloch JFC. Moment/force ratios in mechanics of tooth movement. *Am J Orthod Dentofac Orthop*. 1986;90:27.
12. Smith RJ, Burstone CJ. Mechanics of tooth movement. *Am J Orthod*. 1984;85(4):294.
13. Nägerl H, et al. Centers of rotation with transverse forces: an experimental study. *Am J Orthod*. 1991;99:337.
14. Nägerl H, et al. Basic biomechanical principles of tooth movement. In: Hösl E, Baldauf A, eds. *Mechanical and Biological Basics in Orthodontic Therapy*. Heidelberg: Hüthig Buch Verlag Heidelberg; 1991.
15. Nägerl H, et al. Theoretische and experimentelle aspekte zur initialen Zahnbewegung. *Orthod Kieferorthop Quint*. 1991:43.
16. Christiansen R, Burstone CJ. Centers of rotation within the periodontal space. *Am J Orthod*. 1969;55:353.
17. Burstone CJ, Every TW, Pryputniewicz RJ. Holographic measurement of incisor extrusion. *Am J Orthod*. 1982;82:1.
18. Pryputniewicz RJ, Burstone CJ. The effects of time and force magnitude on orthodontic tooth movement. *J Dent Res*. 1979;58:1154.
19. Tanne K, Burstone CJ, Sakuda M. Biomechanical responses of tooth associated with different root lengths and alveolar bone heights: changes of stress distributions in the PDL. *J Osaka Univ Dent Sch*. 1989;29:17.
20. Tanne K, Sakuda M, Burstone CJ. Three-dimensional finite element analysis for stress in the periodontal tissue by orthodontic forces. *Am J Orthod Dentofac Orthop*. 1987;92:499.
21. Tanne K, et al. Effect of moment-to-force ratios on stress patterns and levels in the PDL. *J Osaka Univ Dent Sch*. 1989;29:9.
22. Tanne K, et al. Patterns of initial tooth displacements associated with various root lengths and alveolar bone heights. *Am J Orthod Dentofac Orthop*. 1991;100(1):66.
23. Dellinger EL. Histologic and cephalometric investigation of premolar intrusion in the *Macaca speciosa* monkey. *Am J Orthod*. 1967;53:325.
24. Fortin JM. Translation of premolars in the dog by controlling the moment-to-force ratio in the crown. *Am J Orthod*. 1971;59:541.
25. Smith R, Storey E. The importance of force in orthodontics. *Aust Dent J*. 1952;56:291.
26. Storey E, Smith R. Force in orthodontics and its relation to tooth movement. *Aust Dent J*. 1952;56:11.
27. Wainwright WM. Faciolingual tooth movement: its influence on the root and cortical plate. *Am J Orthod*. 1973;64:278.
28. Reitan K. The initial tissue reaction incident to orthodontic tooth movement as related to the influence of function. *Acta Odontol Scand Suppl*. 1951;6:1–240.
29. Sandstedt GW. Einige Beiträge zur Theorie der Zahnregulierung. *Nord Tand Tidsskr*. 1904;5:236, 1906;6:1.
30. Groves Jr MH. *Threshold Force Values of the Anterior Retraction*. [Thesis]. Indianapolis: Indiana University School of Dentistry; 1959.
31. Choy K, Pae EK, Park YC, et al. Effect of root and bone morphology on the stress distribution in the periodontal ligament. *Am J Orthod Dentofac Orthop*. 2000;117(1):98–105.
32. Tanne K, Yoshida S, Kawata T, et al. An evaluation of the biomechanical response of the tooth and periodontium to orthodontic forces in adolescent and adult subjects. *Br J Orthod*. 1998;25(2):109–115.
33. Toms SR, Eberhardt AW. A nonlinear finite element analysis of the periodontal ligament under orthodontic tooth loading. *Am J Orthod Dentofac Orthop*. 2003;123(6):657–665.
34. Yoshida N, Jost-Brinkmann PG, Koga Y, et al. Experimental evaluation of initial tooth displacement, center of resistance, and center of rotation under the influence of an orthodontic force. *Am J Orthod Dentofac Orthop*. 2001;120(2):190–197.
35. Iwasaki LR, Beatty MW, Randall CJ, et al. Clinical ligation forces and intraoral friction during sliding on a stainless steel archwire. *Am J Orthod Dentofac Orthop*. 2003;123(4):408–415.
36. Iwasaki LR, Haack JE, Nickel JC, et al. Human tooth movement in response to continuous stress of low magnitude. *Am J Orthod Dentofac Orthop*. 2000;117(2):175–183.
37. Ren Y, Maltha JC, Kuijpers-Jagtman AM. Optimum force magnitude for orthodontic tooth movement: a systematic literature review. *Angle Orthod*. 2003;73(1):86–92.
38. Burstone CJ. The application of continuous forces to orthodontics. *Angle Orthod*. 1961;31:1.
39. Goldberg AJ, Vanderby Jr R, Burstone CJ. Reduction in the modulus of elasticity in orthodontic wires. *J Dent Res*. 1977;56:1227.
40. Goldberg AJ, Burstone CJ, Koenig HA. Plastic deformation of orthodontic wires. *J Dent Res*. 1983;62(9):1016.
41. Burstone CJ, Goldberg AJ. Maximum forces and deflections from orthodontic appliances. *Am J Orthod*. 1983;84(2):95.
42. Goldberg AJ, Morton J, Burstone CJ. The flexure modulus of elasticity of orthodontic wires. *J Dent Res*. 1983;62(7):856.
43. Andreasen GF, Hilleman TB. An evaluation of 55 cobalt substituted nitinol wire for use in orthodontics. *J Am Dent Assoc*. 1971;82:1373.
44. Andreasen GF, Morrow RE. Laboratory and clinical analysis of nitinol wire. *Am J Orthod*. 1978;73:142.
45. Lopez I, Goldberg J, Burstone CJ. Bending characteristics of nitinol wire. *Am J Orthod*. 1979;75:569.
46. Burstone CJ, Farzin-Nia F. Production of low-friction and colored TMA by ion implantation. *J Clin Orthod*. 1995;29(7):453.
47. Burstone CJ, Qin B, Morton JY. Chinese NiTi wire: a new orthodontic alloy. *Am J Orthod*. 1985;87(6):445.
48. Miura F, et al. The super-elastic Japanese NiTi alloy wire of use in orthodontics, I. *Am J Orthod Dentofac Orthop*. 1986;90:1.
49. Miura F, et al. The super-elastic Japanese NiTi alloy wire of use in orthodontics. III. Studies on the Japanese NiTi alloy coil springs. *Am J Orthod Dentofac Orthop*. 1988;94:89.
50. Tripolt H, Burstone CJ, Bantleon P, et al. Force characteristics of nickel-titanium tension coil springs. *Am J Orthod Dentofac Orthop*. 1999;115(5):498–507.
51. Santoro M, Nicolay OF, Cangialosi TJ. Pseudoelasticity and thermoelasticity of nickel-titanium alloys: a clinically oriented review. II. Deactivation forces. *Am J Orthod Dentofac Orthop*. 2001;119(6):594–603.
52. Burstone CJ, Goldberg J. Beta titanium: a new orthodontic alloy. *Am J Orthod*. 1980;7:2.
53. Goldberg AJ, Burstone CJ. An evaluation of beta titanium alloys for use in orthodontic appliances. *J Dent Res*. 1979;58:593.
54. Burstone CJ. Welding of TMA wire clinical applications. *J Clin Orthod*. 1987;21(9):609.
55. Nelson K, Burstone CJ, Goldberg AJ. Optimal welding of beta titanium orthodontic wires. *Am J Orthod*. 1987;92:213.
56. Burstone CJ. Variable-modulus orthodontics. *Am J Orthod*. 1981;80:1.
57. Burstone CJ. The rationale of the segmented arch. *Am J Orthod*. 1962;11:805.
58. Burstone CJ. Mechanics of the segmented arch technique. *Angle Orthod*. 1966;36:99.
59. Burstone CJ. Deep overbite correction by intrusion. *Am J Orthod*. 1977;72:1.
60. Shroff B, et al. Segmented approach to simultaneous intrusion and space closure: biomechanics of the three-piece base arch appliance. *Am J Orthod Dentofac Orthop*. 1995;107:136–143.
61. Shroff B, et al. Simultaneous intrusion and retraction using a three-piece base arch. *Angle Orthod*. 1997;67(6):455–462.
62. Baldini JC, Haack DC, Weinstein S. The manipulation of lateral forces produced by extraoral appliances. *Angle Orthod*. 1981;51:301.
63. Hershey GH, Houghton CW, Burstone CH. Unilateral facebows: a theoretical and laboratory analysis. *Am J Orthod*. 1981;79:229.

64. Gmyrek H, Bourauel C, Richter G, et al. Torque capacity of metal and plastic brackets with references to materials, application, technology and biomechanics. *J Orofac Orthop*. 2002;63(2):113–128.
65. Andreasen GF, Quevedo FR. Evaluation of friction forces in the 0.022 × 0.028 edgewise bracket in vitro. *J Biomech*. 1970;3:151.
66. Burstone CJ. Precision lingual arches: active applications. *J Clin Orthod*. 1989;23(2):101.
67. Burstone CJ, Koenig HA. Force systems from an ideal arch. *Am J Orthod*. 1974;65:270.
68. Burstone CJ, Koenig HA. Creative wire bending: the force system from step and V bends. *Am J Orthod Dentofac Orthop*. 1988;93:59.
69. Koenig HA, Burstone CJ. Force systems from an ideal arch: large deflection considerations. *Angle Orthod*. 1989;59(1):11.
70. Ronay F, et al. Force system developed by V bends in an elastic orthodontic wire. *Am J Orthod Dentofac Orthop*. 1989;94(4):295.
71. Halazonetis DJ. Ideal arch force systems: a center-of-resistance perspective. *Am J Orthod Dentofac Orthop*. 1998;114(3):256–264.
72. Mulligan TF. Understanding wire/bracket relationships. *J Orofac Orthop*. 2002;63(6):493–508.
73. Burstone CJ. Diagnosis and treatment planning of patients with asymmetries. *Semin Orthod*. 1998;4(3):153–164.
74. Burstone CJ. Biomechanics of deep overbite correction. *Semin Orthod*. 2001;7(1):26–33.
75. Choy K, Pae EK, Kim K, et al. Controlled space closure with a statistically determinate retraction system. *Angle Orthod*. 2002;72(3):191–198.
76. Burstone CJ. The precision lingual arch: hinge cap attachment. *J Clin Orthod*. 1994;28(3):151.
77. Burstone CJ. The segmented arch approach to space closure. *Am J Orthod*. 1982;82:361.
78. Burstone CJ, Koenig HA. Optimizing anterior and canine retraction. *Am J Orthod*. 1976;70:1.
79. Kuhlberg AJ, Burstone CJ. T-loop position and anchorage control. *Am J Orthod Dentofac Orthop*. 1997;112:12–18.
80. Chen J, Markham DL, Katona TR. Effects of T-loop geometry on its forces and moments. *Angle Orthod*. 2000;70(1):48–51.
81. Manhartsberger C, Morton J, Burstone CJ. Space closure in adult patients using the segmented arch technique. *Angle Orthod*. 1989;59:205.
82. Burstone CJ, Kuhlberg AJ. Fiber-reinforced composites in orthodontics. *J Clin Orthod*. 2000;34(5):271–279.
83. Freilich MA, Karmaker AC, Burstone CJ, et al. Development and clinical applications of a light-polymerized fiber-reinforced composite. *J Prosthet Dent*. 1998;80(3):311–318.
84. Thorstenson GA, Kusy RP. Resistance to sliding of self-ligating brackets versus conventional stainless steel twin brackets with second-order angulation in the dry and wet (saliva) states. *Am J Orthod Dentofac Orthop*. 2001;120(4):361–370.
85. Liew CF, Brockhurst P, Freer TJ. Frictional resistance to sliding archwires with repeated displacement. *Aust Orthod J*. 2002;18(2):71–75.
86. Viecilli RF, Budiman A, Burstone CJ. Axes of resistance for tooth movement: does the center of resistance exist in 3-dimensional space? *Am J Orthod Dentofac Orthop*. 2013;143(2):163–172.
87. Kusy RP, Greenberg AR. Effects of composition and cross section on the elastic properties of orthodontic archwire. *Angle Orthod*. 1981;51:325.

6

Clinically Relevant Aspects of Dental Materials Science in Orthodontics

Theodore Eliades and Losif Sifakakis

OUTLINE

Impact of Appliance Properties on Mechanics, 187
Stainless Steel Brackets, 187
Stiffness, 187
Roughness, 188
Base-Wing Joint, 188
Hardness, 189
Friction, 189
Material Properties and Torque, 190
Titanium Brackets, 191
Ceramic Brackets, 191
Brittleness and Fracture, 191
Ceramics in Wet Environments, 191
Effect of Grain Size, 192
Archwires, 192
Clinical Impact of NiTi Archwire Properties, 192
Superelastic and Nonsuperelastic NiTi in Crowding Alleviation, 193
Photocuring and Adhesives, 193
Photocuring, 193
Light Intensity, 193
Lamps, 194
Biological Properties of Blue Light and Adhesives, 194
Blue Light Effects, 194
Grinding of Adhesives: Production of Aerosol and Estrogenic Action, 195
Materials for Fixed Retainers, 195
Properties of Wires and Composite Resins Used for Fixed Retainers, 196
Wires, 196
Summary, 197
Acknowledgments, 197

The application of dental materials science in orthodontics coincided with the use of gold and steel wire alloys by Edward. Angle, although the father of the specialty might not have imagined the impact that materials and technological advancements would have in current orthodontic practice. In the United States, the emphasis on mechanics received extraordinary attention, possibly because of the unique educational system and the requirement for a previously earned degree prior to applying to dental school. This prerequisite allowed for the cultivation and growth of materials research because dental graduates entering orthodontic programs were equipped with a bachelor's level of formal training in natural or engineering sciences, thus bringing a new perspective to traditional and empirically taught concepts of mechanics.

The emphasis of this chapter will be on the actual differences that material properties may have on mechanotherapy rather than typically reporting various properties of metallic and polymeric materials. Moreover, a new section will briefly summarize the materials science of fixed retention, assessing the evidence from the mechanics and properties of wires and composites used for the fabrication of fixed retainers.[1]

IMPACT OF APPLIANCE PROPERTIES ON MECHANICS

Stainless Steel Brackets

The following paragraphs will examine these parameters for brackets and analyze their importance in defining the performance of metallic, polymeric, and ceramic appliances (Table 6-1). For a more formal treatise of the subject the reader is referred to Brantley and Eliades' orthodontic materials textbook, where the atomic arrangement, bonding, and mechanical properties are analyzed.[2]

Stiffness

Stiffness reveals the resistance of the appliance to deformation (within the elastic range). High stiffness (or high modulus of elasticity) implies high resistance to deformation and as such is suitable for the areas where no deflection is preferred. These include the slot walls and wings, which should not be compliant to allow for efficient transmission of the loads applied by an activated archwire to the tooth. The reader would appreciate the importance of stiffness by considering the train/railway analogy: railway alloys receive a special surface treatment to gain high stiffness so that they are not deformed during the course of movement,

TABLE 6-1 The Role of Mechanical Properties in Treatment

Property	Effect on Mechanotherapy/Performance
Modulus of elasticity (*E*)	Low E allows for deformation at low forces, facilitating ease in deboning (peel-off effect) because of plastic deformation of base. High E provides resistance to deformation during engagement of archwires (especially torque) of wings and slot
Hardness	Structural integrity against the applied loads during engagement of wire into the brackets slot
Roughness	Full seating of wire into the slot, shielding against high forces arising from the decreased contact points between wire and slot surfaces

Adapted from Eliades T, Pandis N, eds. Self-Ligation in Orthodontics. London: Wiley-Blackwell; 2009.

BOX 6-1 The Role of Stiffness in Different Bracket Components

Various bracket components require different stiffness variants. Wings and slot should be of high stiffness, whereas the base should be compliant for different reasons: wings must withstand the loads applied without being deformed, whereas the base must furnish easy peel-off effect.

and no obstacles are presented to impede rolling of the wheels. To this end, ceramic brackets show higher stiffness as a result of the arrangement of atoms and bonding inside their structure, and for this reason they present better performance when it comes to transmission of loads, whereas plastic brackets, apart from their lower stiffness, which in most cases makes them unsuitable for this task, show several other disadvantages.[2] Loading rate and ambient temperature changes substantially alter the stress–strain characteristics of polymers.[2] In addition, when cyclic loading involves increased strain rates, a thermal softening effect may be induced, leading to a reduction in the fatigue life.[5]

Although high stiffness is preferred for some components of the brackets, for others, such as the base, this is an undesirable feature. For bases, a compliant or low-stiffness alloy is preferred because this bracket component is not an active part of treatment. Box 6-1 summarizes the role of stiffness on desired bracket characteristics, and Figure 6-1 depicts the variable hardness of bracket components.[6]

Roughness

Roughness may differentiate the force because of the effective contact between the wire and bracket. At the microscopic level, this contact is not continuous but possesses a profile of opposing peaks arising from the variation of surfaces between the two materials coming in contact. Figure 6-2 illustrates the roughness of slot surfaces. The implication of roughness in friction during sliding mechanics has been overestimated, and several research and review articles have demonstrated that the rougher wires did not consistently show the highest friction.[7]

Base-Wing Joint

The puzzle of combining both features (a stiff wing and compliant base) was solved with the application of welding or soldering of two different alloys.[8,9] Alloy soldering is used to join the two bracket components. Initially, stainless steel

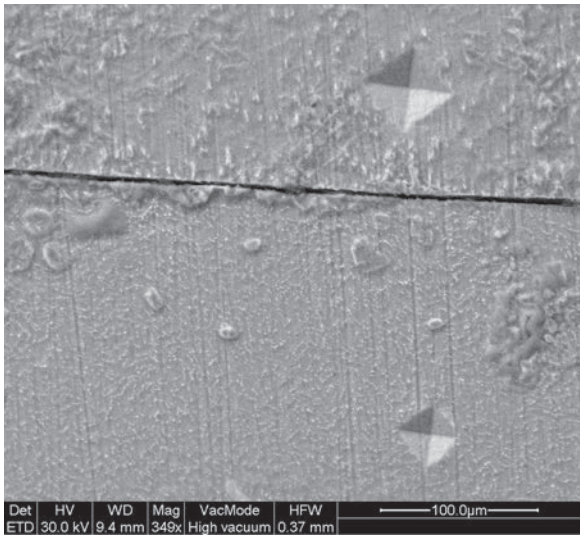


FIGURE 6-1 Secondary electron image of a Ti bracket demonstrating the difference in hardness between the wings *(small pyramid indent)* and base *(large pyramid indent)* (original magnification ×349). The difference in the size of pyramids corresponds to the different alloys used for bracket manufacturing.

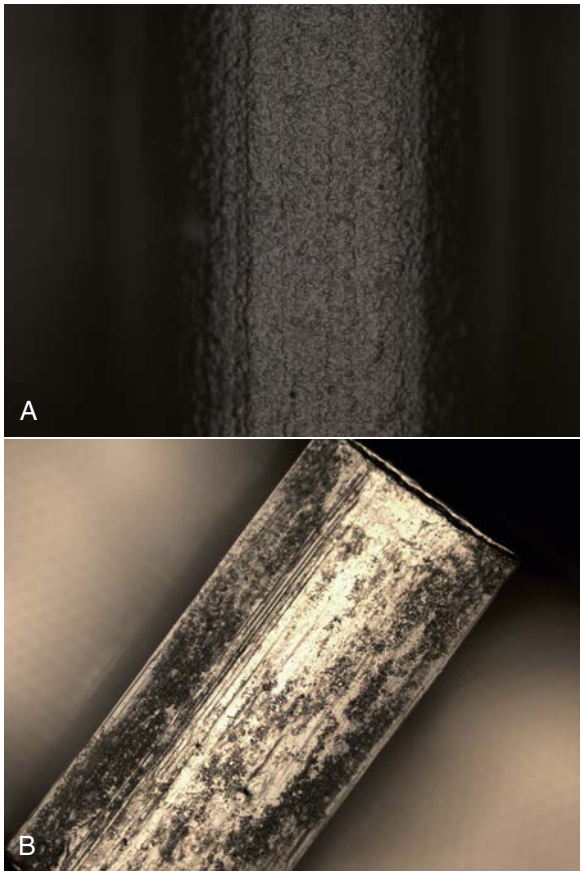


FIGURE 6-2 Optical dark field images of the slot surface of a ceramic (A) and stainless steel bracket (B) demonstrating the increased roughness of the surfaces (original magnification ×10).

BOX 6-2 Bracket-Wing Joints in Metallic Brackets

Base-wing joint is achieved with alloy (Ag, Ni, Au) soldering with potential problems with galvanic corrosion or ionic release, or laser welding with lack of soldering medium and absence of unfavorable effects. MIM brackets are one-piece appliances and show lack of galvanic corrosion, but have higher porosity and are made of one alloy type, which does satisfy the requirements for a stiffer wing component and compliant base.

brackets were brazed with silver (Ag)-based filler alloys, which are also the most frequently used brazing filler metals for stainless steel in industrial applications. However, orthodontic silver brazing alloys contain the cytotoxic element cadmium (Cd), which is added to lower the melting temperature and improve wetting.[10,11] Moreover, Ag-based brazing alloys introduce a galvanic couple with stainless steel alloys, inducing release of metallic ions with copper (Cu) and tin (Zn), the most easily leached out elements from Ag brazing alloys. Because brazing alloys that contain Cu and Zn are cytotoxic, galvanic corrosion, which is the main reason for the progressive dissolution of brazing filler metal,[12] may have both mechanical (detachment of the base-wing component) and biologic implications.[13-17]

To overcome this problem, several manufacturers have introduced gold-based brazing materials. However, this may lead to dissolution of stainless steel, which is less noble than the gold alloys, and this may be the explanation for the in vivo corrosion of bracket bases as well as for Ni leaching from stainless steel alloys.[18] Metal ion release[19] from brackets and orthodontic appliances in general is of great concern because of the adverse effects of allergic reactions or cytotoxicity.[20]

Brackets produced by MIM technology are actually single-piece appliances and thus supposedly free from the corrosion risk associated with the galvanic couple of brazing alloys with stainless steel. However, appliances manufactured with the MIM method have shown extended porosity.[8]

Last, laser welding has not been expanded, and a limited number of products are fabricated with this method. The main advantages of the method relate to the fact that although two alloys with different stiffnesses can be used, the absence of an intermediate phase and potential corrosion risk optimize the prospective performance of the final product. Box 6-2 summarizes the characteristics of base-wing joint.

Hardness

An increased hardness, as in the case with stiffness, is necessary to facilitate surface integrity and preclude binding of the wire onto the bracket slot walls. The latter may affect the force levels experienced by the tooth by increasing the friction variants, thus necessitating an increase in force to achieve tooth movement.[2,19] This is based on the significance of surface roughness on slot–archwire interfacial sliding during displacement of the bracket along the archwire.

The Vickers hardness number (VHN) of MIM-manufactured brackets has been found to vary between 154 and 287, which found much lower than the hardness (400 VHN) of wing components of conventional stainless steel. This difference may have significant effects on the wear phenomena encountered during the archwire activation into the bracket slot.[21] Stainless steel archwires demonstrate a hardness of 600 VHN while the hardness of NiTi archwires ranges from 300 to 430 VHN. The clinical significance of the hardness finding may pertain to the fact that low-hardness wing components may complicate the transfer of torque from an activated archwire to the bracket and may preclude full engagement of the wire to the slot wall and possible plastic deformation of the wing.[21-23]

BOX 6-3 The Overestimation of Actual Clinical Impact of Friction

In vitro assessment of friction includes many assumptions and oversimplifications such as horizontal pathway of wire into the slot; arbitrary movement rate; absence of aging of materials; lack of vibrational movements arising from mastication, which cancel out the frictional resistance; and measurement of an irrelevant component (force required to introduce sliding). Recent clinical trials testing brackets or wires with different fiction do not show difference in treatment duration.

Friction

Bracket–archwire friction has received wide attention during the past 15 years. A number of critical reviews have demonstrated the clinical irrelevance of the typical in vitro assessment of friction protocols presented in a large number of studies during the past decade.[24-29] Various factors related to the oversimplification of experimental configurations and an overwhelming number of assumptions in the experimental design have deprived ex vivo friction assessment from the clinical relevance and scientific soundness required to establish this issue as clinically vital. These briefly include the inappropriateness of the use of the terms *friction* and *sliding resistance* interchangeably, the clinically irrelevant choice of rate of wire sliding onto slot walls, lack of intraoral aging of materials, and study of variables, which possess little or no relevance with the actual clinical analogue.[29] A thorough analysis of the incoherence of the majority of friction research protocols is provided in the Brantley and Eliades orthodontics text.[3] Box 6-3 summarizes the factors that render conventional friction protocols methodologically incoherent and clinically irrelevant.

During the past 5 years, the results of a body of evidence on this issue derived from clinical trials suggest that the bracket–archwire free play may not be the most critical factor in altering tooth movement rate.[30-33] In general, the majority of clinical trials is not supportive of a faster tooth movement rate in "low-friction" brackets.[30-33]

The large clearance and presumed lower binding of a low-friction bracket–wire combination relative to a conventional one may be eliminated as archwires of larger cross sections are gradually inserted in the bracket slot. The clinician may empirically appreciate the free play with low-friction brackets, especially in cases of extreme tooth malalignment, where full engagement of a large-diameter NiTi wire to conventional brackets requires increased pressure. In this first stage, there is a definitive advantage of low-friction brackets relative to conventional appliances; this situation, however, changes dramatically as treatment progresses and wires of higher stiffness are engaged in the bracket. Completion of the correction of rotations and achievement of proper buccolingual crown inclination (torque), which are frequently required in the mandibular and maxillary incisors, respectively, necessitate the presence of a couple of forces. This assumes the formation of contacts of wire inside the bracket slot walls to

generate force, and thus the major advantage of low-friction brackets, namely, the free play, is eliminated as tooth crowns gradually obtain their proper spatial orientation.[34]

Apart from the foregoing factors, in vitro studies dealing with this issue have shown that friction increases with increased roughness of the wire or bracket surfaces, although the opposite has also been suggested. Studies have indicated that beta-titanium and NiTi wires and ceramic brackets have shown increased friction due to their roughened surfaces arising from the manufacturing process. Nonetheless, Kusy and Whitley[28] have shown that although beta-Ti (titanium molybdenum alloy, or TMA) wires exhibited the highest coefficient of friction, the highest roughness was obtained from NiTi wires, with evidence of mass transfer from the beta-titanium wire to the stainless steel and polycrystalline alumina contact flats, attributed to the relatively low compressive yield strength of the beta-titanium wire alloy.

Moreover, frictional testing in most cases involves dry and relatively clean samples (i.e., wires and brackets), and therefore no biofilm or calcified regions are included.[35] The adsorption of these intraoral integuments might increase the surface roughness and resistance to shear forces. A prominent fluctuation of the curve of the frictional loss over the measured displacement of the bracket along the wire has been noted; this could be attributed to the complexes precipitated intraorally.

Material Properties and Torque

Proper buccolingual crown inclination is a key factor to achieving appropriate interincisal inclination and adjusting for minor discrepancy in arch length[36] and may concomitantly be critical in avoiding relapse of deep bite correction, especially in a typical Angle Class II, Division 2, case. A torque transfer of the activated archwire to a bracket assumes ideal materials, no torque loss due to slot design, accuracy of prescription with minimum deviation from actual and reported prescription values, and full expression of the prescribed value. Unfortunately, none of the foregoing assumptions is valid, and the reason is that we deal with real materials that possess various defects.[23]

Let us examine the sequence of events associated with the engagement of a rectangular wire into a bracket slot. First, the edges of the wire will have to be twisted to come in contact with the slot walls, to an extent determined by the size of the slot and the cross section of the wire. The rotation of the wire into the slot prior to contact with the bracket is expressed as *torque loss*, alternatively termed *free play*, *clearance*, or *slack*. The value of this loss has been found to show a difference between theoretic estimation and experimentally derived values, with higher loss occurring in measurements. The source of this discrepancy may be attributed to the rounded edges of the bracket and slot, as well as the tolerance in size; that is, the slot is slightly larger than described and the wire smaller than defined by the manufacturer. The magnitude of this slack is on the order of 10 degrees for a 0.016-×0.022-inch archwire engaged into an 0.018-inch slot,[37] thereby eliminating the torque transferred to the a maxillary central incisor with a Roth prescription appliance, leading to undertorqued anteriors. As the wire is brought in contact with the slot walls, there is a tendency for the former to be pushed out of the slot because the stresses developed tend to displace it labially. Thus, a second factor—namely, the efficiency of ligating medium—comes into play. Elastomeric ligatures are a poor means to secure the wire in place because of the relaxation they present, which reaches high values within the first 24 hours.[38] For this reason, stainless steel ligatures are suggested to increase friction, which in this case is desirable, because otherwise there will be no adequate inclination change.

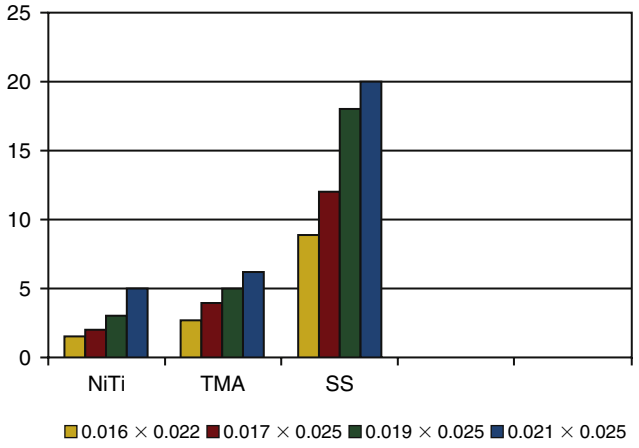


FIGURE 6-3 Graph depicting the relative torsional stiffness of wires of various cross sections and compositions demonstrating that increasing the size of the wire does not secure more effectiveness in torquing crowns. (Compiled from data presented by Kusy RP. On the use of nomograms to determine the elastic property ratios of orthodontic arch wires. *Am J Orthod.* 1983;83:374–381.)

Having eliminated the foregoing variables on slack, the next step is the application of stress of wire onto the slot wall surfaces. As the wire applies a force to the bracket, the wire or the bracket will experience a surface alteration, which will be determined by the difference in hardness. The harder material will leave an imprint in the softer material, and in most cases that is the stainless steel on NiTi and TMA wires and stainless steel wire in Ti or plastic brackets. Also, TMA wires will leave traces of material in ceramic brackets. The resultant wear will presumably take out some of the activation of the wire, further decreasing the potential for torquing the crown of the tooth.

When the residual activation finally outweighs all the abovementioned obstacles, the torsional stiffness of the materials may modulate the torque expression.[39,40] Kusy[41] very effectively described this relationship with the construction of nomograms, where the relative stiffness in torsion of various sizes of archwires is provided in a scale. Figure 6-3 depicts the relative torsional stiffness of various alloys of different cross sections.

Thus, although a 0.017-×0.025-inch NiTi archwire in a 0.018-inch slot will result in decreased play compared with a 0.016-×0.022-inch archwire in the same slot, its torsional stiffness is much lower than the latter. This reveals that a larger cross section is not the critical factor in determining torque efficiency. The emphasis placed on the size of wire as an indication of its stiffness derives from the fallacy that a larger wire will always express the slot prescription more efficiently than one of a smaller cross section. This concept has its roots in the time when orthodontists were able to increase the stiffness of the wire only by incrementally increasing its size. With new alloys with different moduli, it is the combination of size and modulus that determines the stiffness of the wire, not the size alone.[42] Box 6-4 recapitulates the requirements for effective torque transmission with the straight wire technique.

BOX 6-4 Factors Affecting Efficient Torque Expression with the Straightwire Technique

Torquing with the Straightwire technique requires large cross-section stainless steel wires or NiTi wires with a pretorque of almost 40° of activation; stiff wings and wires; hard wing and wire surfaces; and an inelastic ligating medium, with minimum relaxation with time.

BOX 6-5 Clinical Factors with Titanium Brackets

The reduced hardness of titanium brackets imposes several unfavorable implications in sliding and torquing with harder alloys such as stainless steel wires, and thus their use should be limited to cases of proven allergic reaction to nickel.

Titanium Brackets

The introduction of Ti brackets was based on the biocompatibility and lack of allergenic elements, such as Ni, in these appliances. The results of the limited available evidence on Ti brackets indicated that there are substantial structural differences in composition, structure, and manufacturing processes among currently available Ti brackets. These have been found to be single-piece appliances or two separate parts joined together by laser welding, composed of Ti, with a Vickers hardness close to grade II commercially pure (cp) Ti or of a Ti alloy type (Ti-6Al-4V).[43]

These brackets present differences in hardness, which, in general, is much lower than the stainless steel and NiTi archwire alloys. This may induce increased wear rate during orthodontic treatment. It is well known that pure Ti and its alloys have poor wear resistance and require surface modification treatments before being used for biological applications. The use of Ti-6Al-4V alloy with a friction coefficient of 0.28 may have different frictional coefficients from the values available in literature because the latter have been calculated for the cp Ti friction coefficient of 0.34. Box 6-5 notes the clinical implications of reduced hardness of Ti brackets.

The clinical significance of the hardness findings may pertain to the fact that a low-hardness wing component may complicate the transfer of torque from an activated archwire to bracket. The low-hardness–induced wear may preclude a full engagement of the wire to the slot walls and possible plastic deformation of the wing. Thus, whereas Ti brackets may be a viable alternative in the rare cases of proven allergic reaction to Ni-containing products, their reduced hardness may be implicated in wire binding in the slot during mechanotherapy.

Ceramic Brackets

Ceramic brackets were introduced for superior aesthetics, but there are several issues that arose from their large-scale use. Most of the research efforts on this topic were focused on their fracture strength and deboning characteristics, following an unfavorable deboning pattern reported during the early 1990s.

The observation that ceramic brackets fracture frequently, usually at the wings and most often during deboning, is a universal, empirically derived knowledge for most readers. But why do these wires fracture clinically at rates much higher than the ones based on laboratory tests?

Brittleness and Fracture

We shall first provide substantiation for the brittleness of ceramics. These materials are composed of atoms bound together with such strong forces that their flexibility is notably impaired. As a result, the application of a force on ceramics leads only to a minimum elastic deformation with absence of permanent deformation. It follows that these materials maintain their dimensions and shape after fracture because no deformation has set in: this is why broken pieces of china can be glued together. In contrast to this effect, metals and polymers can absorb some of the energy provided during load application by altering their shape or dimensions, thus presenting ductile fracture. The bonding energy and their strong directional characteristics are what make these materials unable to deform. Also, the atomic packing factor in these materials is high, implying that a dense distribution of atoms in a three-dimensional array results in a high crystal density. Therefore, no plastic deformation of the wings is possible, and when the force exceeds a certain value, the wing fractures.

Nonetheless, the foregoing discussion fails to explain the observed higher rate of ceramic fractures clinically compared with that expected from laboratory studies, where ceramic brackets seem to pass the tests. Figure 6-4 illustrates a fractured ceramic bracket.

The mechanism underlying this effect was proposed by Sir Griffith in the early 1920s, when he provided substantiation to the noted deviation of the theoretical strength of ceramics from experimentally estimated strength.[5] Ceramic brackets, like most brittle ceramics, include manufacturing process–induced defects in the form of voids or microcracks, which are most visible in everyday utensils, when glassware subjected to many cycles of dishwashing is examined under bright light. Factors that predispose to crack growth and fracture include manufacturer-controlled and operator-dependent variables; the former relates to the design of the bracket with the presence of many sharp edges and corners enhancing this phenomenon acting as stress raisers, whereas the latter relates to the accidental contact with instruments or burs, which initiate crack growth and propagation.

Ceramics in Wet Environments

Apart from the foregoing discussion, the exposure of ceramics or brittle materials to wet environments may further contribute to the reduction in strength. Assuming a brittle material, the critical stress (σ_{crit}) to induce fracture in a material with crack of size c can be expressed as

$$\sigma_{crit} = (2E\gamma/\pi c)^{1/2}$$

where E is the modulus of elasticity and γ is the critical surface tension of the brittle ceramic.

When this material is exposed to a wet medium (i.e., water or saliva), the previous equation of the critical stress is altered because the critical surface tension (γ) of the material is the only variable that is altered; actually, it is reduced. Because γ is on the numerator, it follows that a reduction in γ will result in a lower value for the whole term, and thus the new critical stress would be decreased relative to one corresponding to the dry state. That means that when the ceramic brackets come into contact with water, their fracture strength is decreased.

Experimental work published in the field has verified this proposal, demonstrating that alumina (aluminum oxide) and zirconia (zirconium oxide) show decreased fracture toughness, faster

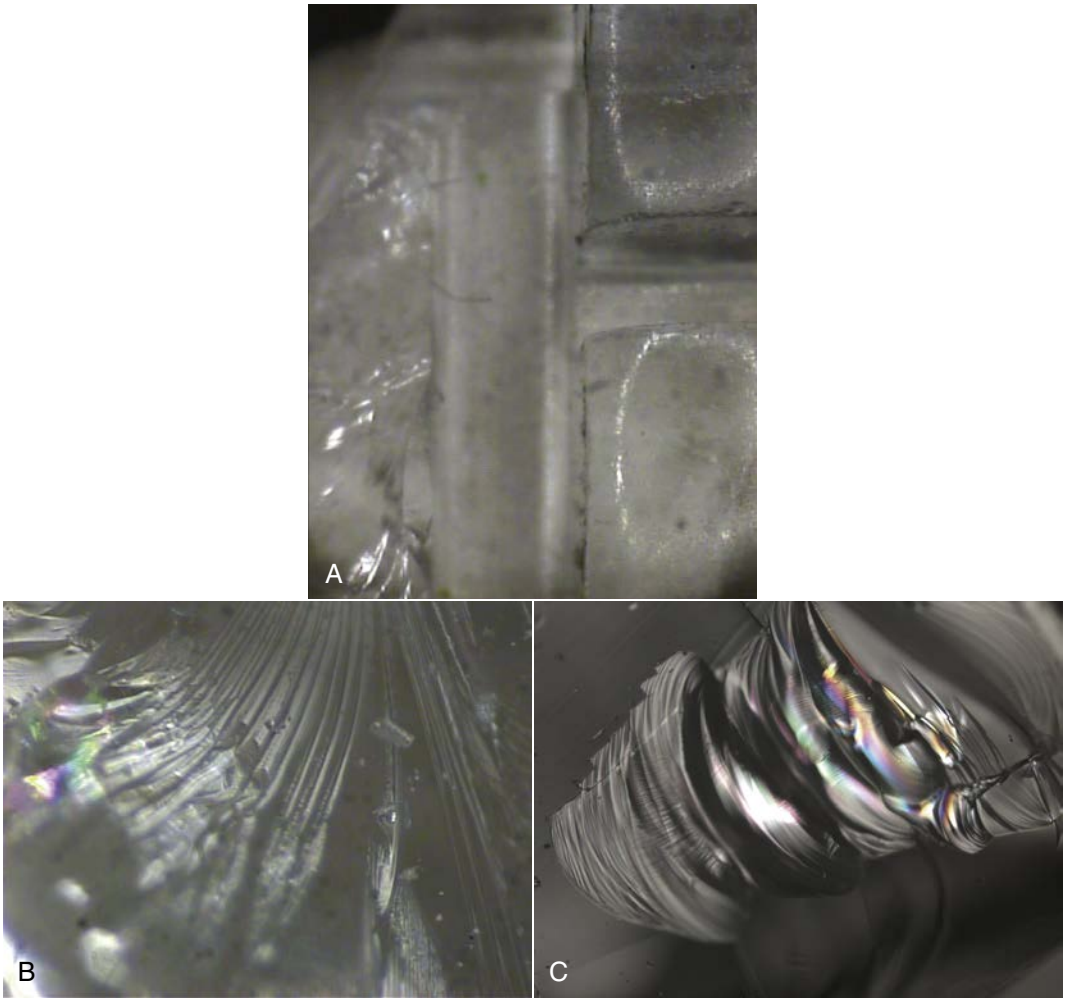


FIGURE 6-4 Optical light transmission image of a ceramic bracket showing a fractured wing (A). The radial pattern corresponds to the fracture plane (B). (C), Under a dark field mode, the dramatic surface effects in B are shown.

crack growth rate, and reduced bending strength when they are exposed to water, normal saline, or Ringer's solution.[4] The presence of electrolytes, enzymes, flora, and other factors may further increase the detrimental impact of aging on strength.

Effect of Grain Size

The different behavior of polycrystalline ceramic brackets compared with the single-crystal appliances to crack growth is attributed to the fact that the crack is impeded at the grains' boundaries of the former, where cracks propagate easier in the latter. It is interesting to note that the same reason for the superb aesthetics of single-crystal brackets arising from their high light transmittance, associated with a reduced light scattering due to the lack of grain boundaries, is also responsible for their reduced fracture toughness. Accordingly, the size of grains may also play a role in determining fracture properties of ceramics: the ones possessing small grain size tend to favor the initiation of cracking because the increased packing capacity of grain boundaries allows for the initiation of the crack in the periphery of force application.[5] On the contrary, large-grained ceramics show a higher resistance to crack initiation because the presence of crystals at the front of crack initiation does not favor crack formation. When the crack is initiated, there are fewer boundaries to inhibit its propagation and thus their fracture toughness is lower than that of their small-grain counterparts.

ARCHWIRES

Clinical Impact of NiTi Archwire Properties

The materials chapter in the 5th edition of this book clarified the concept of phase transformation of superelastic NiTi wires. In the current edition, this section will deal with the actual impact of these crystallographic changes on orthodontics. NiTi archwires have become an integral part of orthodontic treatment because their low load-to-deflection ratio provides a desirable force level and better control of force magnitude.[42] Their initial classification included three categories: superelastic, nonsuperelastic, and true shape memory. This classification was confusing with respect to the meaning of the terms, and as a result, an alternative, structure-based classification was proposed by Kusy as (1) martensitic stabilized, which shows a stable martensitic structure, and thus no shape memory or superelasticity is expressed; (2) martensitic active, also termed thermoactive, in which an increase in the temperature leads to the transformation of the martensitic back to the austenitic structure;

and (3) austenitic active, which demonstrates a pseudo-elastic behavior, where the martensitic structure transformation of these alloys is stress induced, resulting from the activation of the wire. The words *martensite* and *austenite* are named after two prominent metallurgists, Adolf Martens and Sir William Chandler Roberts-Austen, respectively.

The majority of studies investigating the mechanical properties and structural conformation of NiTi wires has used three main routes to elucidate certain aspects of the wire structure and performance.[44-48] The most commonly used method consists of deflection curves, or cantilever testing of segments of archwires under various loading patterns. A problem with this method may be that superelasticity is, by definition, a property referring to the crystallographic structural elements of the material, and depending on the mechanical test, the response of the wires to loading may differ. Also, it is possible that nominally identical curve patterns are derived from different crystallographic structures.

On the other hand, X-ray diffraction studies of archwires are limited by the inherently near-surface nature of this technique, which shows a 50-micron penetration depth, thus providing evidence for the surface layers of the material. Alternatively, differential scanning calorimetry (DSC), which in principle determines the enthalpy for structural transformations, can provide information about the bulk material.[48]

Apart from the limitations on the analytical tools used in relevant research, there is some skepticism over the fact that the actual clinical performance of these wires in the intraoral environment has not been studied to the same extent as their mechanical properties.[49] Studies assessing the rate of tooth movement during treatment using different archwire alloys showed no significant differences among superelastic, nonsuperelastic NiTi wires, and multistranded stainless steel wires.[50]

Superelastic and Nonsuperelastic NiTi in Crowding Alleviation

A recent, randomized, controlled clinical trial has reported no difference in the duration of alignment CuNiTi thermoactive archwires and NiTi wires.[34] This finding, which verified previous clinical research, was attributed to two potential factors: an alteration of transformation temperature ranges occurred in vivo, limiting the transformation of NiTi archwires, or the overall irrelevance of laboratory-derived mechanical behavior of wires to the loading conditions under clinical conditions.

The first hypothesis uses the effect of oral cavity conditions as a key variable affecting the clinical performance of wires. The effect of intraoral conditions on transformation of CuNiTi wires derives from a single study, which assessed the DSC parameters of intraorally exposed and as-received wires.[51] This investigation reported no difference between as-received and clinically retrieved wires in key variables related to phase transformation, except for a significant reduction in heating enthalpy associated with the martensite-to-austenite transition in the 27°C CuNiTi archwires. Therefore, the lack of difference between NiTi and CuNiTi specimens cannot be assigned to intraorally induced changes in the phase transformation pattern of the latter. The temperature sensitivity of superelastic NiTi wires indicates that variations in mouth temperature could cause a stress fluctuation in NiTi wires during orthodontic treatment.[45]

BOX 6-6 Effect of NiTi Structure on Clinical Parameters

The finding that no difference exists between CuNiTi and NiTi with respect to the duration of treatment implies that the loading pattern of wire inside the slot does not allow for expression of superelastic properties either because of aging or free play. It seems that crystallographic structure of materials possesses little importance in clinical outcome.

An alternative hypothesis pertains to the differences of loading conditions between the laboratory conditions and the oral cavity.[37] Such a pattern cannot be simulated in laboratory configurations and may differentiate the performance of the material. This effect, along with the unrealistic force variants at which plateau levels are reached in the stress–strain curve of NiTi wires, may preclude the expression of the full spectrum of properties of NiTi archwires. Box 6-6 summarizes the reason for absence of clinical effects from crystallographically different NiTi wires.

The foregoing considerations, coupled with the complexity of the oral environment, might explain the results of a study[50] on the effect of using archwires of different surfaces on the rate of space closure with sliding mechanics in vivo. Results from this investigation suggested that there was no difference with respect to the time required for space closure in vivo between conventional and ion-implanted beta-Ti wires.

PHOTOCURING AND ADHESIVES

Photocuring

Photocuring in orthodontic bonding has received wide acceptance because of the favorable characteristics of this technique, which include both material and handling advantages. Light-cured adhesives show decreased oxygen inhibition of polymerization, shorter polymerization reaction, and extended working time, which allows for extended handling in the positioning of bracket, thus being ideal for educational purposes.[52]

The development of various types of light sources for use in polymerization has resulted in a multiplicity of factors taking an active role in the polymerization kinetics of the polymeric material.

Light Intensity

When a light beam hits an orthodontic adhesive surface, the extent of light penetration into the relatively thin layer of material depends on the distance of the source from the material surface and the path that the incident beam will have to travel to reach the adhesive.[53]

Likewise, the literature shows a wide array of studies from the field of restorative dentistry dealing with the effect of material composition on the light penetration and degree of cure. In general, the translucency of the composite, which allows for the penetration of light away from the light source, increases with an increasing matching of the refractive indices of the matrix and fillers. Matrix includes the co-monomer system of bis-GMA/TEGDMA mixture, which is used to facilitate the combined favorable features of both monomers, along with several other organic constituents such as polymerization inhibitors, initiators, colorants, etc.[54,55] The choice of these specific monomers is due to the increased molecular weight of bis-GMA, which offers stability and a thicker consistency, and the short-chained

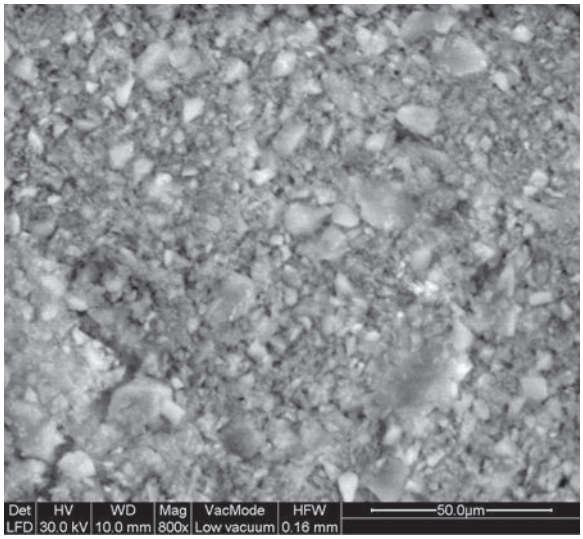


FIGURE 6-5 Secondary electron image of a ground light-cured orthodontic adhesive depicting the variability in filler size (original magnification ×800).

TEGDMA, which contributes to the larger degree of cure because of its decreased molecular weight and higher mobility. Varying the proportion of these constituents, as well as other materials such as amines, accelerators, inhibitors, and initiators, may slightly affect the refractive index of the matrix, which in methacrylate resins is about 1.5.[54]

Fillers, on the other hand, which are contained in the adhesive in a ratio of 60% to 70% per weight in the form of silica particles and barium glasses, possess an index of 1.55 at the wavelength of the photoinitiator.[56] Figure 6-5 depicts the variability in filler size of an adhesive.

The evidence available in the field of composite resins suggests that maximum light scattering occurs at particle size equivalent of the half of the wavelength of a photoinitiator of the polymerization, which for camphorquinone is 468 nm. Almost all the light-cured adhesives use camphorquinone as a photoinitiator of the polymerization. This molecule is contained in the resinous phase at a concentration of 0.2% to 1% of the matrix and shows a peak absorbance wavelength of 468 nm, which implies that increased light intensity in other frequencies may not be effective to excite the molecule.[54] This is an important factor that must be considered in the selection of lamps based on the peak intensity reported by the manufacturer; it is critical that this peak should correspond to the absorbance wavelength of the photoinitiator. Because of its yellowish tint and resultant undesirable matching in uncured versus cured materials, camphorquinone has been recently replaced in some composite resins by 1-phenyl-1,2-propanedione (PPD) with a peak absorbance in the area of 390 to 410 nm.[57]

Lamps

Light-cured orthodontic adhesives require a light-curing source with sufficient intensity and defined wavelength to initiate the polymerization reaction. Increased light intensity and curing time have been advocated for fast polymerization and high degree of cure.[58,59] Recently, various types of commercially available light-curing units have shown comparable bond strength values to those produced by conventional halogen lights at shorter irradiation times.[60] The wide array of new light-curing sources includes plasma arc, laser, and LED lights, which were integrated in the profession to facilitate short irradiation times.

Plasma lamps present very high intensity compared to halogen lights (1600 to 2100 mW/cm^2), an effective spectrum of 450 to 500 nm, and a significantly higher cost, which nevertheless is counterweighed by their increased life span of 5000 hours relative to 40 to 100 hours for halogen. Orthodontic bonding with these light sources can be achieved with only 6 seconds of irradiation for stainless steel brackets or 3 seconds for ceramic brackets.[61]

Laser lights show an intensity of 700 to 1000 mW/cm^2, with a basically monochromatic spectrum of variable wavelength (454, 458, 466, 472, 477, 488, and 497 nm) and are costly but have an almost infinite life span. Application of these light sources to orthodontic bonding has shown that 5 seconds of irradiation provided bond strength values comparable to those found for halogen.[62]

Light-emitting diode (LED) curing units yield a maximum intensity of 1100 mW/cm^2 at a spectrum of 420 to 600 nm, have a cost comparable to that of conventional halogen lights, and possess a nearly infinite life span, while offering handling advantages with a cordless photocuring option. The results of bond strength studies show contradictory evidence on the performance of these lights, with most investigations demonstrating comparable bond strength to halogen lights at the same irradiation duration and reduced strength when shorter time frames are applied.[59,63-65] Conventional bond strength protocols have not been proved to be clinically relevant,[66] and therefore, care should be exercised in extrapolating laboratory results to a clinical situation.

Biological Properties of Blue Light and Adhesives

Apart from standard cytotoxicity assays reported in the literature, the use of high-intensity curing lamps and polymeric molecules has given rise to the investigation of potentially unfavorable effects relevant to orthodontic treatment, which could be proved hazardous to the patients and treatment provider. These include the effect of blue light on mucosa, the action of ground adhesive particulates at deboning as aerosol, and the examination of the role of bis-GMA–based adhesive resins as endocrine disruptors.

Blue Light Effects

Although initially blue light was characterized as relatively harmless, more recent studies have shown that it affects several aspects of cell physiology. Particularly, it has been reported that it disturbs mitochondrial function, thus causing an oxidative stress leading to activation of the stress-responsive pathways.[67] The group of investigations cited earlier suggested that blue light induces effects on the DNA integrity, cellular mitosis, and mitochondrial status in various cell types through the generation of reactive oxygen species (ROS). Investigations in the field have used a variety of mouse and human, normal and transformed cell types, as well as a vast array of assays, which extended from assessment of cell vitality to markers of cell metabolism and oxidative status. This multiplicity of testing protocols has resulted in a variety of effects described.

The result of the sole investigation adopting the time exposures seen in an orthodontic routine bonding[67] has shown that blue light did not affect the viability of these cells, and no immediate effect on the regulation of proliferation was noted 24 hours after irradiation. One week after treatment, however,

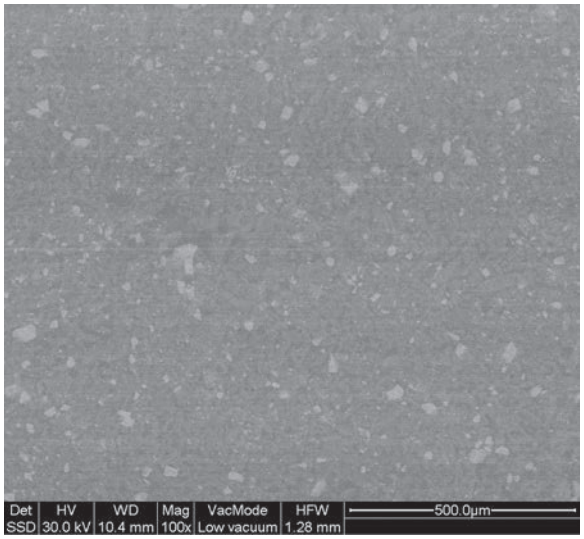


FIGURE 6-6 Backscattered electron image of the particulates of a chemically cured adhesive produced by grinding as occurred during deboning (original magnification ×100).

all types of irradiation induced a significant inhibition of cell proliferation compared with untreated cultures.

The source of this effect has been the subject of several investigations, which have reported that exposure to blue light leads to the generation of ROS, proposing that these are responsible for the adverse biological effects of blue light. However, in a study using a simulation of photocuring in orthodontics,[67] the use of a potent antioxidant agent did not annul the inhibitory effect of irradiation on cell proliferation.

In summary, there is evidence that the biological effects of blue light are confined to long-term effects and are not mediated by oxidation mechanism or DNA damage. The array of effects described suggests that high energy sources such as plasma lamps should be used with caution, especially when bonding mandibular tubes where a close contact between the tissue and the lamp tip occurs.

Grinding of Adhesives: Production of Aerosol and Estrogenic Action

A recent review on the subject has presented the potential action of orthodontic adhesives as endocrinologic disruptors and summarized the available evidence.[52] A substantial body of literature has demonstrated the cohort of phenomena accompanying the exposure of organisms to bisphenol-A (BPA).[68-71] Moreover, the effects of BPA on tissues follow a nonmonotonic curve pattern, which is characterized by intense reactivity at low levels and no response at very high ones, respectively.[71] The concept of "critical concentration," referring to the required amount of substance to induce effects, may not apply in the case of exposure to BPA.[71,72]

In orthodontics, the removal of the brackets and cleanup of the enamel surface that follow the completion of orthodontic treatment involve grinding of the adhesive layer with rotary instruments at low or high speed. The aerosol produced by this process contains polymer matrix and filler degradation by-products as well as particulates arising from the wear of bur. Figure 6-6 illustrates the morphologic condition of particulates produced after the use of rotary instruments on an adhesive. The potential hazardous nature of aerosol is twofold: first, it relates to the production and circulation of a dust with a sufficiently small aerodynamic diameter to reach the alveoli of the lungs.[73-75] Second, there is a potential hormone-disrupting action of these particles derived from the presence of a double benzoic ring in the bis-GMA monomer, which, under specific conditions, has been reported to lead to the formation and release of BPA.[76-78] Although bulk, intact orthodontic adhesive specimens have not demonstrated BPA release or estrogenic action,[79,80] the biological properties of particulates examined with a standard in vitro assay[81,82] have shown opposite effects.[78] Grinding, especially without water spray, increases the temperature locally, with unpredictable effects on the composition and formation of resin by-products, while, concurrently, this process dramatically increases the effective surface area of the material with host tissues, enhancing the reactivity of the material, with potentially altered outcome on the tissue–material interactions. In the broader biomedical literature, the difference between the biological properties of bulk materials and their particulates has been established for inert alloys such as Ti.[82] Box 6-7 notes the potentially estrogenic action of adhesive in a particulate form.

BOX 6-7 Estrogenic Action of Adhesives

Grinding of the adhesive at deboning results in a twofold hazardous sequla: generation of particles, which act as an aerosol with detrimental action on the respiratory system, and potential estrogenicity, owing to the incorporation of Bis-GMA monomer, which gives rise to bisphenol-A formulation with xeno-estrogenic properties. It is interesting to note that no release of bisphenol-A and no estrogenicity have been reported for chemically cured and light-cured adhesives when they are not ground.

Grinding of the adhesive introduces heat into the material, exposing the matrix to severe heat shock and mechanical aging. It has been long known that grinding and polishing increase the C—C bond conversion to C═C on the surfaces of resin composites mainly due to the heat produced. The chemical alterations induced in the abraded powder compounds possessing double benzoyl rings, including bis-GMA, are unknown. It may be hypothesized that this procedure may accelerate or induce formation of BPA, with endocrinologic disruption as an outcome.

This finding is of interest considering that receptors for estrogen have been identified in human gingival tissues, and thus this tissue can be a target organ for sex hormones.[83,84] It has been reported that the oral mucosa of premenopausal women was significantly more sensitive to sodium lauryl sulfate in toothpastes than that of postmenopausal women.[85] This might indicate a sex hormone influence on the oral epithelium reactivity to chemical challenge.

Because a given day in practice may include several appointments involving removal of bonded orthodontic appliances, the treatment provider, patients, and staff are exposed to substantial amounts of adhesive aerosol on an almost daily basis. Care should be taken to apply preventive measures such as mask and protective glasses, access to fresh air, and use of suction.

MATERIALS FOR FIXED RETAINERS

Fixed retention is used widely after orthodontic treatment in order to prevent the relapse of the malocclusion in cases with higher relapse tendency. Since their first use 40 years ago, various indications were suggested.[86,87] Unfortunately, definite

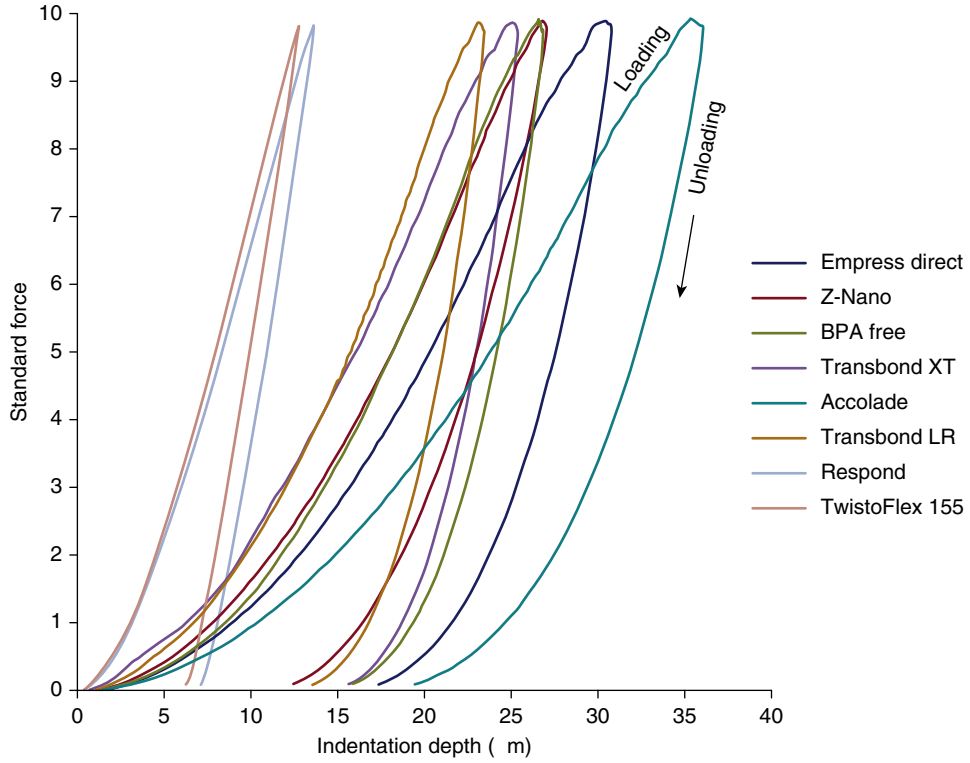


FIGURE 6-7 Force indentation depth loading–unloading curves for various materials used for fixed retainers alongside some dental restorative composites for purposes of comparison.

retention protocols are still unavailable, and the extent of their use is largely based on the experience or the beliefs of the orthodontist. Nowadays, stainless steel archwires are preferred, in the form of either a light round multistrand archwire bonded on all or only four anterior teeth or a heavy round solid wire bonded only on the canines (canine-and-canine). Several less common alternatives have been described in the recent orthodontic literature, regarding the design or the preferable material.[88]

Properties of Wires and Composite Resins Used for Fixed Retainers

It's rather difficult to make a safe assumption about the biomechanical performance of a fixed retainer, since it's influenced by a variety of factors, such as the physical and mechanical properties of the wire or the composite, the tooth dimensions, and the properties of the periodontal ligament. Moreover, the effect of aging on the properties of the retainer should not be underestimated.[7,89-91] Figure 6-7 illustrates the force indentation depth loading–unloading curves for some materials used for fixed retainers along with some dental restorative composites for comparison purposes.

Wires

Stiffness is a measure of resistance to deformation mainly in bending and is dependent on the modulus of elasticity. Stiffness in torsion of the retainer wire is important too since the main movement in the unexpected posttreatment changes observed long term after debonding[92,93] is the labiolingual rotation of the tooth (Fig. 6-8). Stiffness tells how much force will be applied for a certain deflection of the retainer wire and is the same for wires of the same alloy, regardless of their hardness. Size is the most potent variable for controlling stiffness of fixed retainers,[94] and for multistrand archwires the stiffness is influenced also by the composition and the

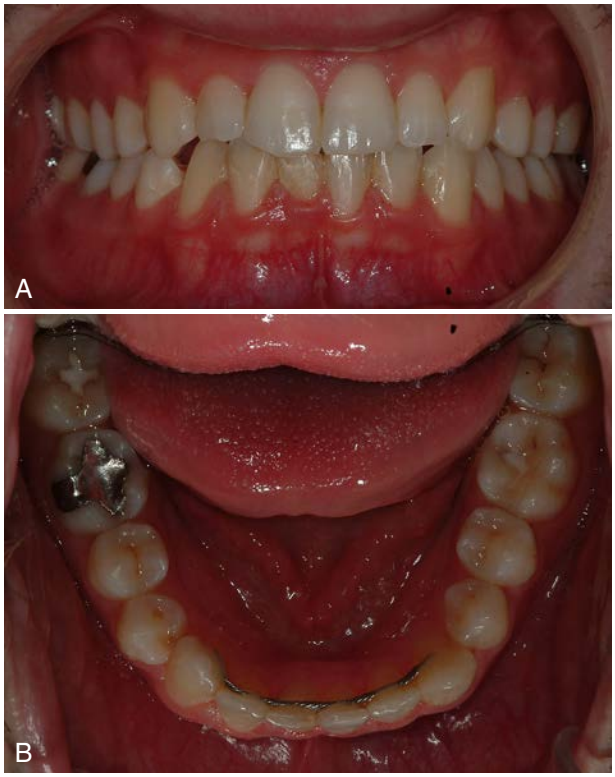


FIGURE 6-8 Frontal occlusion view and mandibular arch of a patient 5 years after deboning. Unexpected labiolingual rotation of both canines was observed in the direction of straightening/untwisting of the multistrand fixed retainer (7-strand 0.027-inch steel), which was still bonded on all anterior teeth. Deformation of the wire is clinically visible, at least between the left lateral incisor and canine.

number of the strands.[95] For a given length of a multistrand archwire, stiffness is reduced by increasing the length of the strands, in comparison with a solid archwire or a multistrand archwire with a lower pitch. However, the tight twisting of the strands increases the stiffness so finally the twisted archwires offer essentially the same stiffness as multiple straight strands but lower stiffness as a solid archwire.[96,97] The forces recorded in vitro on a canine or lateral incisor bonded on various types of lingual canine-to-canine fixed retainer wires might exceed 1 N and are large enough to produce unwanted tooth movement during retention.[98]

In conventional stainless steel wires, range decreases with increasing wire thickness, but in multistrand wires this property is not influenced to the same extent as in solid wires by the cross-section size and appears to be nearly constant for a particular wire configuration.[96] For a given overall wire diameter and helix angle, the ranges of multistranded wires were independent of wire configurations.[99] The increased length of the strands for a given length of an archwire increases its range, in comparison with a solid archwire or a multistrand archwire with a lower pitch.[96,97] The strength of round wires in bending and torsion is proportional to the cube of the wire diameter, but range is inversely related to the diameter. In bending, strength is inversely proportional to the length, but strength in torsion is not dependent on length.[94]

Heat treatment of stainless steel archwires increases the malleability of the wire while improving its properties by relieving the stresses retained from archwire formation into an arch, loops, or coils.[94,96,97] Heating of austenitic stainless steel wires above 650°C should not be done because loss of the wrought microstructure causes degradation of mechanical properties.[100] However, in these archwires, the desirable ease of formation becomes undesirable ease of deformation.[94,101] In the cases of unexpected post-treatment changes observed long term after deboning, a 3-strand 0.0195-inch, heat-treated stainless steel fixed retainer was still in place.[92,93] Clinical observations expand the incidence of these movements in several cases of 7-strand 0.027-inch steel canine-to-canine retainers, still bonded on all lower anterior teeth.

BOX 6-8 Wires Used for Fixed Retention

Most common types of fixed retainers are the light round multistrand steel archwire bonded on all anterior teeth and the heavy round solid steel wire bonded only on the canines. The latter displays lower detachment rate, but the former type induces frequent relapse of incisors not bonded to the retainer. In order to avoid the unexpected movements of teeth bonded on fixed retainers, archwires with higher bending and torsional stiffness may be more suitable for the construction of fixed retainers.

BOX 6-9 Adhesives Used for Fixed Retention

The adhesive resins indicated for lingual retainers should be hard and inelastic with high abrasion resistance and bond strength. An optimal degree of conversion and minimal polymerization shrinkage are extremely important in order to ensure minimal solubility and microleakage and decrease the levels of residual monomers.

This type of loading approximated the vertical incisor bite force during mastication, in a similar way as anterior biting. The results demonstrated residual forces and moments in all retainer types tested, of the highest magnitude in cases of the above-mentioned softer archwires. These findings suggest that the evaluated lower canine-to-canine fixed retainers may not be passive after short- or long-term use, especially these high formable/low yield strength retainers. Archwires with higher bending and torsional stiffness may be more suitable for the construction of fixed retainers.

Regarding the failure rates of the fixed retainers, the heavy canine-and-canine wire displayed a lower detachment rate in comparison with thinner, flexible spiral wire canine-to-canine retainers; however, the former type induced frequent relapse of incisors not bonded to the retainer.[102,103] Box 6-8 summarizes the main points regarding the archwires used for the construction of fixed retainers.

SUMMARY

This chapter analyzed selective aspects of orthodontic materials with direct implications in mechanics, treatment duration and hazardous nature of materials, and applications to patient and care provider. In the last section, the advantages and disadvantages of the materials used for the construction of fixed retainers are discussed. Archwires with higher bending and torsional stiffness may be more suitable for the construction of fixed retainers. It is advisable to use adhesives with high hardness, especially designed for fixed retainers (Box 6-9).

Acknowledgments

The collaboration of William A. Brantley (Ohio State), Spiros Zinelis (University of Athens), and Christoph Bourauel (University of Bonn) in research resulting in some of the evidence presented in this chapter is acknowledged.

REFERENCES

1. Matasa CG. Biomaterials in orthodontics. In: Graber TM, Gioka C, Eliades T, et al., eds. *Orthodontics: Current Principles and Techniques*. St. Louis: Elsevier Mosby; 2005:345–389.
2. Brantley WA. Structures and properties of orthodontic materials. In: Brantley WA, Eliades T, eds. *Orthodontic Materials: Scientific and Clinical Aspects*. Stuttgart: Thieme; 2001:1–27.
3. Eliades T, Brandley WA, Eliades G. Orthodontic brackets. In: Brandley WA, Eliades T, eds. *Orthodontic Materials*. New York: Thieme; 2001: 146–147.
4. Eliades T, Gioka C, Zinelis S, et al. Plastic brackets: hardness and associated clinical implications. *World J Orthod*. 2005;5:62–66.
5. Suresh S. *Fatigue of Materials*. Cambridge, UK: Cambridge Solid State Science Series; 1991.
6. Eliades T, Zinelis S, Brantley WA. The significance of bracket material properties in orthodontic mechanics. In: Eliades T, Pandis N, eds. *Self-Ligation in Orthodontics*. London: Wiley-Blackwell; 2009: 21–31.
7. Eliades T, Bourauel C. Intraoral aging of orthodontic materials: the picture we miss and its clinical relevance. *Am J Orthod Dentofac Orthop*. 2005;127:403–412.

8. Zinelis S, Annousaki O, Makou M, et al. A metallurgical characterization of orthodontic brackets produced by the metal injection molding (MIM). *Angle Orthod*. 2005;75:1024–1031.
9. Zinelis S, Annousaki O, Eliades T, et al. Elemental composition of bracket brazing materials. *Angle Orthod*. 2004;74:394.
10. Berge M, Gjerdet NR, Erichsen ES. Corrosion of silver soldered orthodontic wires. *Acta Odont Scand*. 1982;40:75–79.
11. Pflaum M, Kielbassa C, Garmyn M, et al. Oxidative DNA damage induced by visible light in mammalian cells: extent, inhibition by antioxidants and genotoxic effects. *Mutat Res*. 1998;408:137–146.
12. Maijer R, Smith DC. Corrosion of orthodontic bracket bases. *Am J Orthod*. 1982;81:43–48.
13. Barret RD, Bishara SE, Quinn JK. Biodegradetion of orthodontic appliances. Part I. Biodegradation of nickel and chromium in vitro. *Am J Orthod Dentofacial Othop*. 1993;103:8–14.
14. Eliades T, Athanasiou AE. *In vivo* aging of orthodontic alloys: implications for corrosion potential, nickel release and biocompatibility. *Angle Orthod*. 2002;72:222–237.
15. Eliades T, Pratsinis H, Kletsas D, et al. Characterization and cytotoxicity of ions released from stainless steel and nickel-titanium orthodontic alloys. *Am J Orthod Dentofac Orthop*. 2004;125:24–29.
16. Eliades T, Zinelis S, Eliades G, et al. Characterization of as-received, retrieved, and recycled stainless steel brackets. *J Orofac Orthop*. 2003;64:80–87.
17. Eliades T, Zinelis S, Papadopoulos MA, et al. Nickel content of as-received and retrieved NiTi and stainless steel archwires: assessing the nickel release hypothesis. *Angle Orthod*. 2004;74:151–154.
18. Eliades T. Orthodontic materials research and applications: part 2. Current status and projected future developments in materials and biocompatibility. *Am J Orthod Dentofac Orthop*. 2007;131:253–262.
19. Petoumenou E, Arndt M, Keilig L, et al. Nickel concentration in the saliva of patients with nickel-titanium orthodontic appliances. *Am J Orthod Dentofac Orthop*. 2009;135:59–65.
20. Castro L, Merino S, Levenfeld B, et al. Mechanical properties and pitting corrosion behaviour of 316 L stainless steel parts obtained by a modified metal injection moulding technique. *J Mater Proces Techn*. 2003;143:397–402.
21. Zinelis S, Eliades T, Eliades G, et al. Comparative assessment of the roughness, hardness, and wear resistance of aesthetic bracket materials. *Dent Mater*. 2005;21:890–894.
22. Gioka C, Bourauel C, Zinelis S, et al. Titanium orthodontic brackets: structure, composition, hardness and ionic release. *Dent Mater*. 2004;20:693–700.
23. Gioka C, Eliades T. Materials-induced variation in the torque expression of preadjusted appliances. *Am J Orthod Dentofac Orthop*. 2004;125:323–328.
24. Cacciafesta V, Sfondrini MF, Ricciardi A, et al. Evaluation of friction of stainless steel and esthetic self-ligating brackets in various bracket-archwire combinations. *Am J Orthod Dentofac Orthop*. 2003;124:395–402.
25. Griffiths HS, Sherriff M, Ireland AJ. Resistance to sliding with 3 types of elastomeric modules. *Am J Orthod Dentofac Orthop*. 2005;127:670–675.
26. Khambay B, Millett D, McHugh S. Evaluation of methods of archwire ligation on frictional resistance. *Eur J Orthod*. 2004;26:327–332.
27. Pizzoni L, Ravnholt G, Melsen B. Frictional forces related to self-ligating brackets. *Eur J Orthod*. 1998;20:283–291.
28. Kusy RP, Whitley JQ. Effects of sliding velocity on the coefficients of friction in a model orthodontic system. *Dent Mater*. 1989;5:235–240.
29. Burrow SJ. Friction and resistance to sliding in orthodontics: a critical review. *Am J Orthod Dentofac Orthop*. 2009;135:442–447.
30. Miles PG. Self-ligating vs conventional twin brackets during en-masse space closure with sliding mechanics. *Am J Orthod Dentofac Orthop*. 2007;132:223–225.
31. Miles PG. Smartclip versus conventional twin brackets for initial alignment: is there a difference? *Aust Ortod J*. 2005;21:123–127.
32. Pandis N, Strigou S, Eliades T. Maxillary incisor torque with conventional and self-ligating brackets: a prospective clinical trial. *Orthod Craniofac Res*. 2006;9:193–198.
33. Shivapuja PK, Berger J. A comparative study of conventional ligation and self-ligation bracket systems. *Am J Orthod Dentofac Orthop*. 1994;106:472–480.
34. Pandis N, Polychronopoulou A, Eliades T. CuNiTi vs. NiTi wires in mandibular crowding alleviation: a randomized control trial of efficiency. *Am J Orthod Dentofac Orthop*. 2009;136(152.e1-7):152–153.
35. Eliades T, Eliades G, Athanasiou AE, et al. Surface characterization of retrieved orthodontic NiTi archwires. *Eur J Orthod*. 2000;22:317–326.
36. O'Higgins EA, Kirschen RH, Lee RT. The influence of maxillary incisor inclination on arch length. *Br J Orthod*. 1999;26:97–102.
37. Sebanc J, Brantley WA, Pincsak JJ, et al. Variability of effective root torque as a function of edge bevel on orthodontic arch wires. *Am J Orthod*. 1984;86:43–51.
38. Taloumis LJ, Smith TM, Hondrum SO, et al. Force decay and deformation of orthodontic elastomeric ligatures. *Am J Orthod Dentofac Orthop*. 1997;111:1–11.
39. Meling TR, Odegaard J, Meling EO. On mechanical properties of square and rectangular stainless steel wires tested in torsion. *Am J Orthod Dentofac Orthop*. 1997;111:310–320.
40. Meling TR, Odegaard J. The effect of cross-sectional dimensional variations of square and rectangular chrome-cobalt archwires on torsion. *Angle Orthod*. 1998;68:239–248.
41. Kusy RP. On the use of nomograms to determine the elastic property ratios of orthodontic arch wires. *Am J Orthod*. 1983;83:374–381.
42. Burstone CJ. Variable modulus orthodontics. *Am J Orthod*. 1981;80:1–16.
43. Zinelis S, Annousaki O, Eliades T, et al. Metallographic structure and hardness of titanium orthodontic brackets. *J Orofac Orthop*. 2003;64:426–433.
44. Bourauel C, Scharold W, Jäger A, et al. Fatigue failure of as-received and retrieved NiTi orthodontic archwires. *Dent Mater*. 2008;24:1095–1101.
45. Iijima M, Ohno H, Kawashima I, et al. Mechanical behavior at different temperatures and stresses for superelastic nickel-titanium orthodontic wires having different transformation temperatures. *Dent Mater*. 2002;18:88–93.
46. Meling TR, Odegaard J. The effect of temperature on the elastic responses to longitudinal torsion of rectangular nickel titanium archwires. *Angle Orthod*. 1998;68:357–368.
47. Meling TR, Odegaard J. The effect of short-term temperature changes on superelastic nickel-titanium archwires activated in orthodontic bending. *Am J Orthod Dentofac Orthop*. 2001;119:263–273.
48. Eliades G, Brantley WA. Instrumental techniques for study of orthodontic materials. In: Brantley WA, Eliades T, eds. *Orthodontic Materials: Scientific and Clinical Aspects*. Stuttgart: Thieme; 2001:50–74.
49. Segner D, Ibe D. Properties of superelastic wires and their relevance to orthodontic treatment. *Eur J Orthod*. 1995;17:395–402.
50. Kula K, Phillips C, Gibilaro A, et al. Effect of ion implantation of TMA archwires on the rate of orthodontic sliding space closure. *Am J Orthod Dentofac Orthop*. 1998;114:577–580.
51. Biermann MC, Berzins DW, Bradley TG. Thermal analysis of as-received and clinically retrieved copper-nickel-titanium orthodontic archwires. *Angle Orthod*. 2007;77:499–503.
52. Eliades T. Orthodontic materials research and applications: part 1. Current status and projected future developments in bonding and adhesives. *Am J Orthod Dentofac Orthop*. 2006;130:445–451.
53. Al-Hiyasat AS, Darmani H, Elbetieha AM. Leached components from dental composites and their effects on fertility of female mice. *Eur J Oral Sci*. 2004;112:267–272.
54. Watts DC, Silikas N. In situ photopolymerization and polymerization shrinkage phenomena. In: Eliades G, Watts DC, Eliades T, eds. *Dental Hard Tissues and Bonding: Interfacial Phenomena and Related Properties*. Heidelberg: Springer; 2005:123–154.
55. Watts DC, Amer O, Combe E. Characteristics of visible light-cured composite systems. *Br Dent J*. 1984;156:209–215.
56. Suzuki H, Taira M, Wakasa K, et al. Refractive-index-adjustable fillers for visible-light-cured dental resin composites: preparation of TiO_2-SiO_2 glass powder by the sol-gel process. *J Dent Res*. 1991;70:883–888.

57. Sun G, Chae K. Properties of 2,3-butanedione and 1,2-propanedione as a new photo-sensitizers for visible light cured dental resin composites. *Polymer*. 2000;41:6205–6212.
58. Kauppi MR, Combe EC. Polymerization of orthodontic adhesives using modern high-intensity visible curing lights. *Am J Orthod Dentofac Orthop*. 2003;124:316–322.
59. Mills RW, Jandt KD, Ashworth SH. Dental composite depth of cure with halogen and blue light emitting diode technology. *Br Dent J*. 1999;186:388–391.
60. Yoon TH, Lee YK, Lim BS, et al. Degree of polymerization of resin composites by different light sources. *J Oral Rehab*. 2002;29:1165–1173.
61. Signorelli MD, Kao E, Ngan PW, et al. Comparison of bond strength between orthodontic brackets bonded with halogen and plasma arc curing lights: an *in-vitro* and *in-vivo* study. *Am J Orthod Dentofac Orthop*. 2006;129:277–282.
62. Lalani N, Foley TF, Voth R, et al. Polymerization with the argon laser: curing time and shear bond strength. *Angle Orthod*. 2000;70:28–33.
63. Koupis NS, Eliades T, Athanasiou AE. Clinical evaluation of bracket bonding using two different polymerization sources. *Angle Orthod*. 2008;78:922–925.
64. Niepraschk M, Rahiotis C, Bradley TG, et al. Effect of various curing lights on the degree of cure of orthodontic adhesives. *Am J Orthod Dentofac Orthop*. 2007;132:382–384.
65. Talbot TQ, Blankenau RJ, Zobitz ME, et al. Effect of argon laser irradiation on shear bond strength of orthodontic brackets: an *in vitro* study. *Am J Orthod Dentofac Orthop*. 2000;118:274–279.
66. Eliades T, Brantley WA. The inappropriateness of conventional orthodontic bond strength assessment protocols. *Eur J Orthod*. 2000;22:13–23.
67. Taoufik K, Mavrogonatou E, Eliades T, et al. Effect of blue light on the proliferation of human gingival fibroblasts. *Dent Mater*. 2008;24: 895–900.
68. Palanza P, Howdeshell KL, Parmigiani S, et al. Exposure to a low dose of bisphenol A during fetal life or in adulthood alters maternal behavior in mice. *Environ Health Perspect*. 2002;110:415–422.
69. Quesada I, Fuentes E, Viso-León MC, et al. Low doses of the endocrine disruptor bisphenol-A and the native hormone 17ß-estradiol rapidly activate transcription factor CREB. *FASEB*. 2002;16: 1671–1673.
70. Timms BG, Howdeshell KL, Barton L, et al. Estrogenic chemicals in plastic and oral contraceptives disrupt development of the fetal mouse prostate and urethra. *Proc Nat Acad Sci U S A*. 2005;102:7014–7019.
71. Welshons WV, Thayer KA, Judy BM, et al. Large effects from small exposures. I. Mechanisms for endocrine disrupting chemicals with estrogenic activity. *Environ Health Perspect*. 2003;111:994–1006.
72. Pulgar R, Olea-Serrano MF, Novillo-Fertrell A, et al. Determination of bisphenol A and related aromatic compounds released from bis-GMA-based composites and sealants by high performance liquid chromatography. *Environ Health Perspect*. 2000;108:21–27.
73. Chen Y, Craig L, Krewski D. Air quality risk assessment and management. *J Toxicol Environ Health A*. 2008;71:24–39.
74. Day CJ, Price R, Sandy JR, et al. Inhalation of aerosols produced during the removal of fixed orthodontic appliances: a comparison of 4 enamel cleanup methods. *Am J Orthod Dentofac Orthop*. 2008;33:11–17.
75. Ireland AJ, Moreno T, Price R. Airborne particles produced during enamel cleanup after removal of orthodontic appliances. *Am J Orthod Dentofac Orthop*. 2003;124:683–686.
76. Atkinson JC, Diamond F, Eichmiller F, et al. Stability of bisphenol A, triethylene-glycol dimethacrylate, and bisphenol A dimethacrylate in whole saliva. *Dent Mater*. 2002;18:128–135.
77. Gioka C, Eliades T, Zinelis S, et al. Characterization and *in vitro* estrogenicity of orthodontic adhesive particulates produced by simulated deboning. *Dent Mater*. 2009;25:376–382.
78. Eliades T, Gioni V, Kletsas D, et al. Oestrogenicity of orthodontic adhesive resins. *Eur J Orthod*. 2007;29:404–407.
79. Eliades T, Hiskia A, Eliades G, et al. Assessment of bisphenol-A release from orthodontic adhesives. *Am J Orthod Dentofac Orthop*. 2007;131:72–75.
80. Soto AM, Murai JT, Siiteri PK, et al. Control of cell proliferation: evidence for negative control on estrogen-sensitive T47D human breast cancer cells. *Cancer Res*. 1996;46:2271–2275.
81. Tarumi H, Imazato S, Narimatsu M, et al. Estrogenicity of fissure sealants and adhesive resins determined by reporter gene assay. *J Dent Res*. 2000;79:1838–1843.
82. Okafor CC, Haleem-Smith H, Laqueriere P, et al. Particulate endocytosis mediates biological responses of human mesenchymal stem cells to titanium wear debris. *J Orthop Res*. 2006;24:461–473.
83. Seki K, Koshi R, Sugano N, et al. Microarray analysis of bisphenol A-induced changes in gene expression in human oral epithelial cells. *Acta Biochim Biophys Sin*. 2007;39:879–884.
84. Vittek J, Hernandez MR, Wenk EJ, et al. Specific estrogen receptors in human gingiva. *J Clin Endocrinol Metab*. 1982;54:608–612.
85. Herlofson BB, Barkvoll P. Oral mucosal desquamation of pre- and post-menopausal women. A comparison of response to sodium lauryl sulphate in toothpastes. *J Clin Periodontol*. 1996;23:567–571.
86. Zachrisson BU. The bonded lingual retainer and multiple spacing of anterior teeth. *J Clin Orthod*. 1983;17:838–844.
87. Bearn DR. Bonded orthodontic retainers: a review. *Am J Orthod Dentofacial Orthop*. 1995;108:207–213.
88. Eliades T, Voutsa D, Sifakakis I, et al. Release of bisphenol-A from a light-cured adhesive bonded to lingual fixed retainers. *Am J Orthod Dentofacial Orthop*. 2011;139:192–195.
89. Ramoglu SI, Usumez S, Buyukyilmaz T. Accelerated aging effects on surface hardness and roughness of lingual retainer adhesives. *Angle Orthod*. 2008;78:140–144.
90. Chudoba T. Measurement of hardness and Young's modulus by nanoindentation. In: Cabaleiro A, De Hosson JT, Lockwood DJ, eds. *Nanostructured Coatings*. New York: Springer Science; 2006:216–260.
91. Hirayama S, Iwai H, Tanimoto Y. Mechanical evaluation of five flowable resin composites by the dynamic micro-indentation method. *J Dent Biomech*. 2014;5:1–8.
92. Katsaros C, Livas C, Renkema AM. Unexpected complications of mandibular lingual retainers. *Am J Orthod Dentofacial Orthop*. 2007;132:838–841.
93. Renkema AM, Renkema A, Bronkhorst E, et al. Long-term effectiveness of canine-to-canine bonded flexible spiral wire lingual retainers. *Am J Orthod Dentofacial Orthop*. 2011;139:614–621.
94. Thurow RC. *Edgewise Orthodontics*. 4th ed. St. Louis: Mosby; 1982.
95. Oltjen JM, Duncanson Jr MG, Ghosh J, et al. Stiffness-deflection behavior of selected orthodontic wires. *Angle Orthod*. 1997;67:209–218.
96. Ingram Jr SB, Gipe DP, Smith RJ. Comparative range of orthodontic wires. *Am J Orthod Dentofacial Orthop*. 1986;90:296–307.
97. Kapila S, Sachdeva R. Mechanical properties and clinical applications of orthodontic wires. *Am J Orthod Dentofacial Orthop*. 1989;96:100–109.
98. Sifakakis I, Pandis N, Eliades T, et al. *In vitro* assessment of the forces generated by lingual fixed retainers. *Am J Orthod Dentofacial Orthop*. 2011;139:44–48.
99. Rucker BK, Kusy RP. Theoretical investigation of elastic flexural properties for multistranded orthodontic archwires. *J Biomed Mater Res*. 2002;62:338–349.
100. Brantley WA, Alapati SB. Heat treatment of dental alloys: a review. In: Pardhi Y, ed. *Metallurgy – Advances in Materials and Processes*. Rijeka: InTech; 2012:1–18.
101. Baysal A, Uysal T, Gul N, et al. Comparison of three different orthodontic wires for bonded lingual retainer fabrication. *Korean J Orthod*. 2012;42:39–46.
102. Störmann I, Ehmer U. A prospective randomized study of different retainer types. *J Orofac Orthop*. 2002;63:42–50.
103. Al-Nimri K, Al Habashneh R, Obeidat M. Gingival health and relapse tendency: a prospective study of two types of lower fixed retainers. *Aust Orthod J*. 2009;25:142–146.

7

The Role of Evidence in Orthodontics

David L. Turpin and Greg Huang

OUTLINE

Introduction, 200
The Hierarchy of Evidence, 201
Systematic Reviews and Meta-analyses, 203
How Much Evidence Do We Need?, 203
Where to Find the Evidence, 205
The Future of Evidence-Based Orthodontics, 205
Conclusions, 206

INTRODUCTION

The field of orthodontics has the distinction of being the first recognized specialty in dentistry, and we should all be proud of our profession's history, which is older than 100 years. With our designation as a specialty, we also inherited some responsibilities—one being to lead in the acquisition, evaluation, and dissemination of scientific knowledge. We have accepted this challenge from the beginning, but the mechanisms by which we acquire, assess, and transfer knowledge have changed considerably over the past century. In this chapter, we describe the evolution to evidence-based orthodontics and why we should all utilize this approach in providing care to our patients.

Edward Angle, the father of modern orthodontics, was constantly trying to improve his craft, and certainly, he should be credited with the development of many important orthodontic ideas and principles, some of which we still utilize. His schools on both coasts of the United States have influenced orthodontists not only in North America but also in Europe, Asia, and Australia. His textbooks are still on the reading lists at many orthodontic programs. In the late nineteenth and early twentieth centuries, he was "the man," and as "the man," Dr. Angle exemplified the "Age of the Expert." Few dared to question Dr. Angle's approach to orthodontics, and when this happened, it sometimes led to bitter debates.

Upon Dr. Angle's death in 1930, no one stepped forward to continue his school of "orthodontia."[1] Instead, the responsibility for educating future orthodontists shifted to dental schools, and we entered the "Age of Education." Medicine and dentistry became professions associated with university settings, and standards were developed to insure educational uniformity nationwide. This model certainly has many advantages, and has become the accepted mechanism for training health care providers all over the world.

With the beginning of World War II, scientific innovation flourished. Entire countries underwent tremendous advances and development in response to challenging new conditions. Scientists developed new medications, surgical techniques, sources of energy, etc., and we entered the "Age of Science." The scientific method was embraced to answer our health care questions, and orthodontists were busy conducting studies to improve our understanding of diagnosis and treatment planning. There was much to be learned, and orthodontics was as productive as any field of dentistry. However, much of our research was conducted without rigorous guidelines, and upon reflection, suffered from various types of problems that accompany retrospective or observational studies.

As we entered the latter part of the twentieth century, a shift occurred in medicine. New methods were being proposed to conduct, analyze, and synthesize clinical research. Archie Cochrane and David Sackett, two physicians who both had formal training in epidemiology, were bothered greatly that medicine was often practiced with an unscientific approach. Through their efforts,[2,3] medicine was nudged toward the "Age of Evidence." They wanted physicians to recognize good evidence from bad evidence, and they felt there was value in synthesizing evidence from multiple studies. More important, they wished for this information to then be applied routinely in the clinical setting. This was the birth of evidence-based medicine.

The American Dental Association (ADA) became interested in evidence-based dentistry (EBD) during the late 1990s, and by 2000, the organization had defined EBD as "an approach to oral healthcare that requires the judicious integration of systematic assessments of clinically relevant scientific evidence, relating to the patient's oral and medical condition and history, with the dentist's clinical expertise and the patient's treatment needs and preferences."[4] This definition has served the dental profession well, and the ADA has continued to take a leadership role in promoting EBD. They have created a center for EBD with a vision to "Lead in the promotion of oral health by disseminating the best available scientific information and helping practitioners implement it into clinical practice."[4] The ADA has set up recurring meetings and educational programs to encourage dentists to become familiar with evidence-based principles, and they have also created the ADA Center for EBD, an online resource that is accessible to everyone. One of the features of the ADA Center for EBD is a listing of all of the systematic reviews, meta-analyses, and practice guidelines in the field of dentistry, sorted by various topics. The ADA also

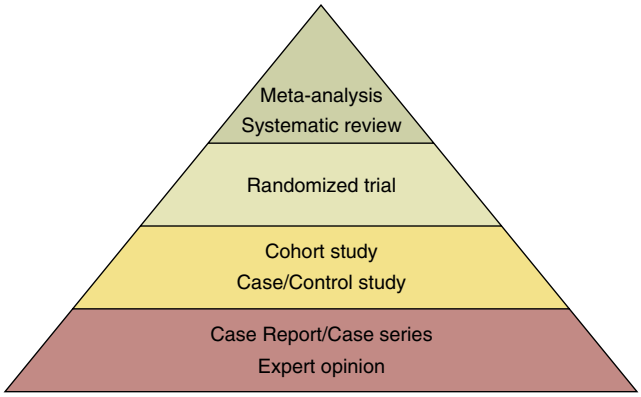


FIGURE 7-1 Hierarchy of evidence.

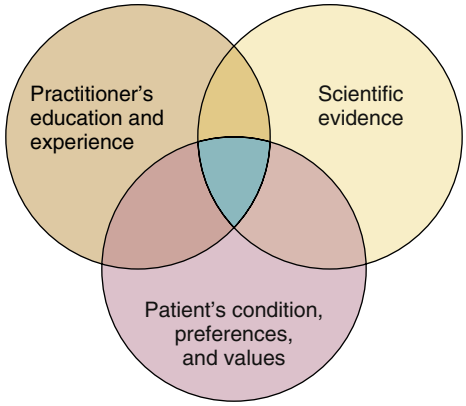


FIGURE 7-2 Evidence-based care is the intersection of three primary factors.

has established panels that summarize systematic reviews and meta-analyses, and translate them into critical summaries for dental professionals, as well as plain language summaries for patients.

In concert with the ADA's shift toward evidence-based practice, the Council on Dental Accreditation (CODA), which oversees the education of dentists and dental specialists in the United States, has also incorporated EBD into dental curricula. Currently, CODA lists "evidence-based care" as one of the "core principles" necessary in the dental educational environment.[5] Likewise, in the CODA standards for Graduate Programs, it is stated that, "Advanced specialty education programs must include instruction or learning experiences in evidence-based practice".[6] With early and intensive exposures to EBD being mandated during education programs, it hopefully will only be a matter of time before all dentists employ an evidence-based approach to practice.

Let's explore the elements of an evidence-based practice. The evidence, naturally, is one fundamental component. The Hierarchy of Evidence allows us to understand the different types of study designs, along with their relative strengths and weaknesses (Fig. 7-1). Thus, we are able to evaluate the merits of a specific publication. The top components of the Hierarchy of Evidence, the systematic review and meta-analysis, allow us to evaluate the strength of the body of literature that may exist for a specific clinical question. Another element of evidence-based care is a focus on patient values and preferences rather than a paternalistic model in which the practitioner dictates treatment. Finally, we must consider the role of the practitioner, who plays a critical role in the delivery of evidence-based care. It is the practitioner who must incorporate his or her own education and experience, as well as the scientific literature and the patient's preferences, in order to arrive at final recommendations for the patient (Fig. 7-2).

How different is our approach to scientific evidence? Just consider the changes that have occurred over the past 20 years. The Internet and digital media have become our routine mechanism for accessing evidence. PubMed, the ADA Evidence-based Dentistry Website, the Cochrane Collaboration, and the AAO Evidence-based Literature Website are only a few of the web-based resources that are available. Most of these sites have at least partial access for everyone, and while some information is targeted toward professionals, these sites often have information that is summarized in a fashion that is friendly to patients seeking guidance.

When reading an article, it is important to understand where it falls on the Hierarchy of Evidence, in order to understand the relative strength of the information. It is also important to judge a few critical parameters under which the study was conducted. Was there a systematic method to select patients? Was a control or comparison group utilized? Was blinding employed to avoid introducing bias? What was the source of funding? All these are simple, but extremely important, elements of evaluating research.

The development of guidelines for conducting systematic reviews has helped to standardize the process and has resulted in more valid and useful reviews. With more than 200 systematic reviews on orthodontic topics, it is often possible to update our knowledge on a particular topic with a 10- or 15-minute Internet search. This is a huge improvement over looking up references in Index Medicus, and then searching through heavy, bound issues of orthodontic journals in all corners of the library!

While much of the information mentioned above is familiar to our recently trained orthodontists, those of us graduating 15 or 20 years ago have witnessed a real revolution in producing, accessing, and utilizing orthodontic literature. Evidence-based methods are an enormous improvement in allowing us to obtain information in a quick and efficient manner.

Entire books have been devoted to evidence-based practice, and it is not our intent to undertake such an effort here. However, in the remainder of this chapter, we would like to briefly address some additional topics regarding an evidence-based practice:

- Comment on the Hierarchy of Evidence
- Review the basic elements of systematic reviews and meta-analyses
- Discuss the quality and quantity of evidence we need to make clinical recommendations
- Provide references to access evidence
- Discuss the future of evidence in orthodontics

THE HIERARCHY OF EVIDENCE

While our patients may not routinely demand that we cite the best evidence as we make our treatment recommendations, keep in mind that orthodontic evidence is abundantly available on the Internet. Therefore, if you are not incorporating the evidence into the fabric of your practice, you may be caught

"...the best balance, the best harmony, the best proportions of the mouth in its relationsto the other features require that there shall be the full complement of teeth..."

Edward Angle
Treatment of Malocclusion of the Teeth, 1907

FIGURE 7-3 Old Glory—illustrating a full complement of teeth. (From Wahl N: Orthodontics in 3 millennia. Chapter 12: Two controversies: Early treatment and occlusion, *American Journal of Orthodontics and Dentofacial Orthopedics* 130(6): 799-804, 2006.)

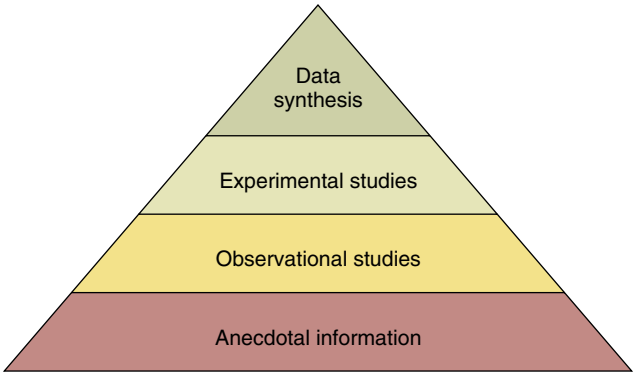


FIGURE 7-4 Anecdotal evidence is low on the evidence pyramid.

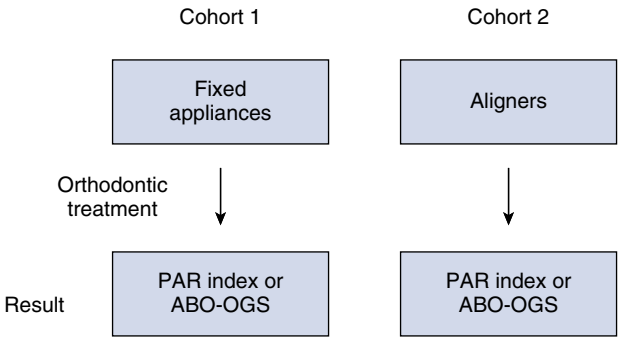


FIGURE 7-5 An example of a cohort study.

off guard, as patients and parents have become increasingly sophisticated about asking for the research that may or may not accompany various treatment options. Every orthodontist should be familiar with the study designs in the Hierarchy of Evidence, which is illustrated in Figure 7-1.

Some have recommended that the term *Hierarchy of Evidence* be replaced with the term *Levels of Evidence,* as the word *hierarchy* might imply a system that was too simplistic and rigid.[7,8] While the relative position of the study designs in the schema were minimally affected, the term *levels* allowed a little more latitude in evaluating the strength of an individual study. Higher level studies will generally provide stronger evidence, and a well-conducted systematic review, if available, would usually provide the best evidence-based information. Therefore, it makes sense to start any search by looking for a systematic review or meta-analysis on a topic. However, it would be unwise to believe that we can blindly apply a hierarchical system to our literature without some degree of judgment and thought. Sometimes, a well-conducted observational study can provide better evidence than a poorly conducted randomized trial. The point is, whether we use the word *Hierarchy* or *Levels* of Evidence, we can never stop thinking critically when we are assessing evidence.

Let's briefly review the Levels of Evidence. At the bottom is expert opinion. While this might have been highly regarded in the past, one person's view of an issue without any evidence is not very convincing today. There is simply too much possibility for bias.

The next type of evidence is the case report or the case series. As the name implies, these are simply one or a few cases illustrating a certain concept or type of treatment. Edward Angle based his non-extraction philosophy from several skulls, including "Old Glory," that all showed excellent occlusion and displayed a full complement of teeth (Fig. 7-3).[9] He extrapolated that the condition represented by the series of skulls could be applied to everyone. Obviously, the selection of cases is subject to bias, and they may or may not be very generalizable. Thus, this level of evidence is considered anecdotal (Fig. 7-4).

The next two study designs, case/control studies and cohort studies, could be labeled "observational studies" because investigators are simply observing the results of treatment rather than manipulating any treatment parameters. The case/control study is not a common design in orthodontics, so we will only say that it is utilized more in the medical field, especially when investigating diseases with rare outcomes. The type of study that is very common in orthodontics is the cohort study, in which two cohorts (or groups) undergo different treatments, and then their outcomes are compared (Fig. 7-5). Cohort studies are well suited to orthodontic research and can be conducted in a retrospective or prospective manner. In the retrospective flavor, one could go back in time and create cohorts that were treated with different techniques and compare their outcomes. The main advantage to this design is that all the treatment has been completed, and only the records need to be identified. However, one potential problem is that patients may have received different treatments for a reason, and perhaps the patients who make up the different cohorts are not very similar in their initial characteristics. Consider a comparison of occlusal outcomes in patients treated with fixed appliances versus aligners, in which the initial severity of the malocclusions may be quite different in the two groups if measures are not taken to match on this parameter.

Prospective cohort studies usually take more time to complete than retrospective cohort studies, as data are being gathered at some predetermined point in the future, usually when treatment is finished. However, there might be opportunities to standardize on some parameters of recruitment and treatment. This may help in creating cohorts that are more evenly matched on initial characteristics, and it also may allow better comparison of different treatments. For example, we might be interested in assessing how well unilateral crossbites are corrected in the mixed dentition using different techniques. If Practitioner A always uses a quad helix and Practitioner B always uses a Hyrax, then we could compare all the 8- to 11-year-old patients whom they treat with their respective techniques over a 1-year period,

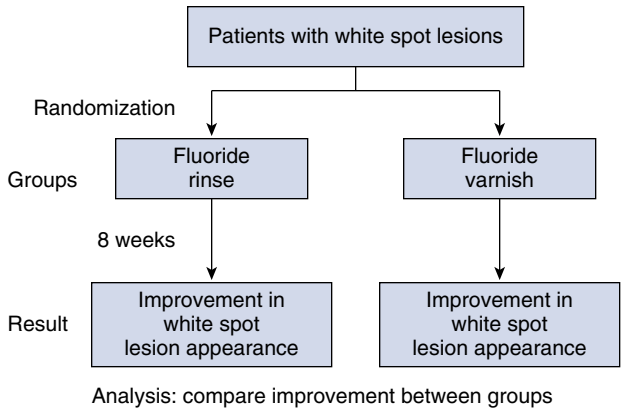


FIGURE 7-6 An example of a randomized trial.

and assess them initially, at the time of expander removal, and at 1 year after expander removal.

Finally, we move to the randomized trial, which is actually very similar to the design of a prospective cohort study (Fig. 7-6). However, there is one very important distinction: all subjects who are eligible for inclusion into the study are randomly assigned to one of the treatment arms. This step helps to ensure that the patients are distributed in an equitable manner into the various treatment groups. Given enough patients, this will help to create groups of patients in each arm of the trial that are as equal as possible in all characteristics, with the exception being the intervention. Randomized trials are considered an experimental design, rather than an observational design, due to the fact that investigators are prospectively manipulating the conditions under which patients will receive care.

Randomized trials should result in the least bias, and when possible, this design should be employed. However, they are expensive, time-consuming, and sometimes not possible to conduct due to ethical issues. We are not likely to find the resources to conduct randomized trials for all our orthodontic questions. However, we should consider conducting randomized trials, whenever possible, for our more important orthodontic questions.

SYSTEMATIC REVIEWS AND META-ANALYSES

At the top of the Levels of Evidence are two types of studies that are considered data synthesis. These are the systematic review and the meta-analysis. Actually, these designs are very similar, and both involve the systematic searching and evaluation of the body of literature that exists for a specific clinical question. The Cochrane Collaboration has published handbooks on conducting these reviews, which has improved their quality tremendously over the past decade.[10] The steps in conducting these studies are similar, but if the data identified during the systematic review are similar enough to allow pooling from several studies, then the data can be summarized using forest plots. This pooling of data is the key feature that distinguishes a systematic review from a meta-analysis. Some consider a systematic review as a qualitative evaluation of the evidence, while a meta-analysis is a quantitative assessment.

When planning a systematic review or meta-analysis, several elements are important to specify *a priori*. First, a very focused and precise question should be formulated. The PICO format is often suggested, with the letters indicating the Population, Intervention, Comparison or Control group, and the Outcome. An example of a PICO question might be, "Among adolescents with posterior crossbites (population), does tooth-borne expansion (intervention), compared with TAD-borne expansion (comparison group), result in comparable amounts of skeletal expansion as measured by CBCT (outcome)?" Second, the databases and references that will be searched should be described. For example, authors might state that they will search PubMed, Embase, and ClinicialTrials.gov. Third, inclusion and exclusion criteria for the studies should be specified, in order to fairly decide which studies will and won't be included. Some reviewers, such as those conducting Cochrane reviews, will limit their searches to randomized trials. If randomized trials are available, then it makes sense to limit reviews to these studies, as they should provide the best evidence. However, in the field of orthodontics, employing that criterion would result in no eligible studies for many topics, and therefore, most orthodontic systematic reviews and meta-analyses do include observational studies. Oftentimes, the conclusions of these reviews frustrate clinicians, as they report that, due to limitations in the quality of the current evidence, conclusions must be viewed with caution, and additional research is warranted.

HOW MUCH EVIDENCE DO WE NEED?

On that note, it is helpful to consider how much evidence we need to make robust clinical recommendations. After all, applying the results of research to the clinical setting is the ultimate goal of an evidence-based approach to practice. Is one cohort study enough, or do we need a couple of randomized trials, or is a systematic review of well-conducted randomized trials necessary? There is a famous article in the *British Medical Journal* that pokes fun at the shortcomings of evidence-based practice.[11] The authors conduct a systematic review of randomized trials on the use of parachutes to prevent death and trauma during free fall. Disappointingly, they find no randomized trial and conclude that "the effectiveness of parachutes has not been subjected to rigorous evaluation by using randomized controlled trials." Obviously, in this instance, it is not necessary or desirable to conduct a randomized trial. We know jumping out of a plane with no parachute is a very bad idea. However, the "interventions" in this experiment are at two extreme ends of the spectrum, as are the outcomes.

In our profession, a parallel question might be, "Does an expander correct a crossbite better than no expander?" The answer would be yes, without performing a randomized trial, because we know most crossbites do not self-correct. Of course, this is a much different question from the one we usually ask in orthodontics, as we often wish to determine which treatment or therapy might be most effective or efficient. Orthodontics has been referred to as the 6-mm specialty (the amount of correction from Class II to Class I molar), and with outcomes of this magnitude, more rigorous study designs must be employed. For example, if I asked, "What is the best and most stable way to perform rapid maxillary expansion," does our profession have a robust answer? Is tooth-borne better than tissue-borne, and is TAD-borne even better? What is the optimal age to perform expansion, and up to what age is acceptable? And what is the optimum regimen for activating a rapid palatal expander? To answer these questions, we would need well-conducted trials to assess the less dramatic differences that might exist among these orthodontic interventions.

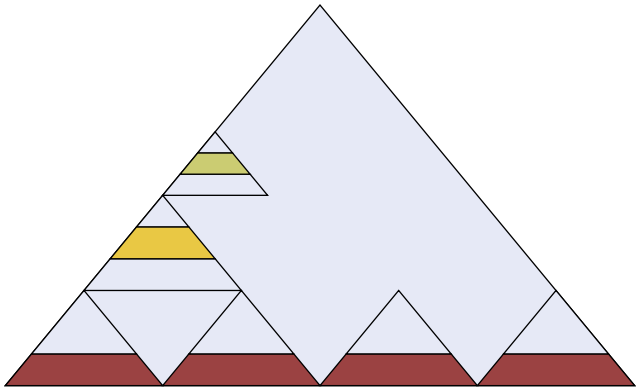


FIGURE 7-7 An illustration of the body of evidence on a particular topic, which consists of a few randomized trials, a few observational studies, and many anecdotal studies.

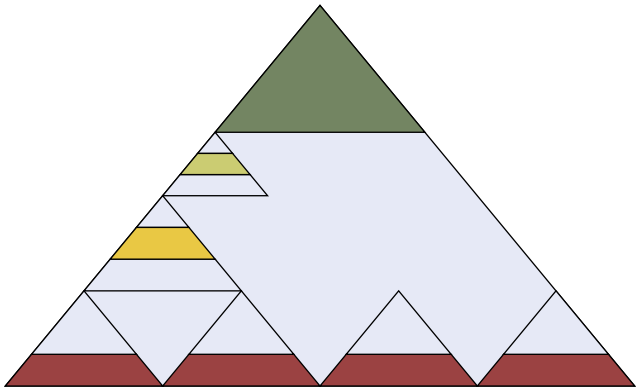


FIGURE 7-8 An illustration of the body of evidence included in a systematic review, which consists of a few randomized trials, a few observational studies, and many anecdotal studies.

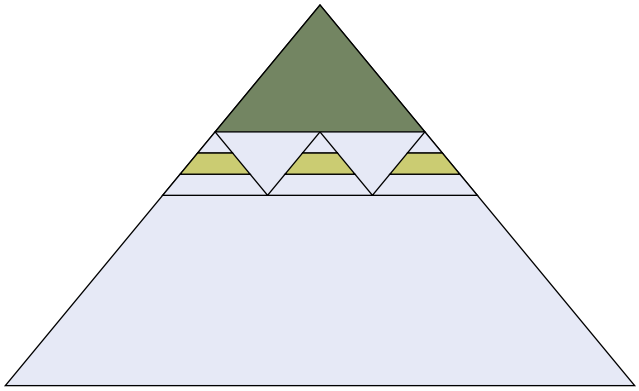


FIGURE 7-9 An illustration of the body of evidence included in a systematic review, which consists of only randomized trials.

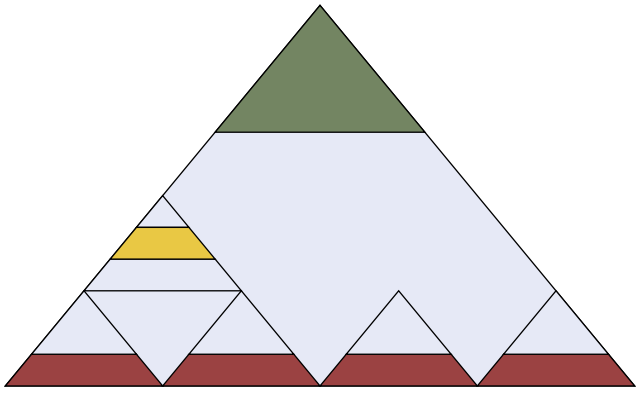


FIGURE 7-10 An illustration of the body of evidence included in a systematic review, which consists of a few observational studies and many anecdotal studies.

To help judge the strength of our evidence, many scales have been developed by various entities. The Centre for Evidence-based Medicine has a chart showing the levels of evidence,[12] and two additional scales are the Strength of Recommendation Taxonomy (SORT)[13] and the Grading of Recommendation Assessment, Development and Evaluation (GRADE).[14] However, these scales can be confusing, as they use phrases like "Level 2" evidence or "Code A" evidence or the "GRADE recommendation is strong." Adding to the confusion is the fact that the scales have been evolving and continue to do so.

At this time, we would like to introduce some simple diagrams that help to depict the level of evidence that exists on a topic. First, take a look at the levels of evidence (see Fig. 7-4), with the anecdotal evidence coded in red, the observational studies coded in yellow, the randomized trials coded in light green, and the systematic reviews and metaanalyses coded in dark green. If we searched on the expansion literature, we would find just a few randomized trials, some observational studies, and many case series. This could be depicted schematically as in Figure 7-7. We would also find several systematic reviews, based on the body of evidence I just mentioned. One of these systematic reviews could be depicted as in Figure 7-8, where the top green triangle represents the existence of a systematic review, and the lower triangles depicts the various types of studies found in this review in their relative proportions. As another example, what if we asked about the evidence comparing 2-phase to comprehensive Class II treatment? There is a Cochrane systematic review that addresses this topic that only includes randomized trials.[15] This would be represented in Figure 7-9—a systematic review including only randomized trials.

Now, back to our question: "What is the quality and quantity of evidence needed to make a robust clinical recommendation in orthodontics?" Well, to be fairly confident of an answer regarding different treatments or techniques, we probably need to have multiple well-conducted randomized trials that report consistent findings. For example, if there are three well-conducted randomized trials on 2-phase versus 1-phase Class II treatment, and they all indicate that patients will arrive at similar outcomes with both strategies, then we can be fairly confidant of this finding, especially if no randomized trials provide opposing evidence. Similarly, if investigators were to perform a systematic review on this topic, and identified well-conducted randomized trials that were all consistent in their findings, we would have pretty good confidence about the conclusions of the systematic review (see Fig. 7-9). On the other hand, if our only systematic review on this topic included many case series and perhaps a few cohort studies, this would not be considered strong evidence, as these types of studies have a high potential for bias (Fig. 7-10).

Consider another question: "Are remineralization agents effective in improving the appearance of white spot lesions that form during orthodontic treatment?" Using the best evidence available, a recent systematic review of seven randomized trials, the answer is: "There is a lack of reliable evidence to support the effectiveness of remineralizing agents for the treatment of post-orthodontic white spot lesions."[16] How convincing is this evidence? It is depicted by Figure 7-9, a systematic review that only includes randomized trials. This is relatively strong evidence that we do not have a reliable agent to address white spot lesions at this time.

As mentioned earlier, there are now more than 200 systematic reviews and meta-analyses on orthodontic topics. Because much of our literature consists of observational data, many systematic reviews and meta-analyses include these types of studies. This helps to explain why we are routinely cautioned when we read the conclusions of these reviews—they look like the diagram depicted in Figure 7-8. On the other hand, when we have multiple randomized trials that have similar findings, or when we have systematic reviews of randomized trials that have similar findings, we should really take note. This may be information that we can be relatively confident about. However, this brings up a very important issue—what we believe is best and true today may not be what we think is best and true tomorrow. Medicine has many examples of this, and in fact, some of our well-accepted guidelines about dietary cholesterol are expected to be reversed on the basis that the recommendations were based upon "insufficient evidence."[17]

WHERE TO FIND THE EVIDENCE

Where you search for information may influence the quality of advice you receive. For instance, one may look for information that is posted on the Web. Many Websites provide little oversight of the material they post, so information may or may not be accurate. On the other hand, there are other sites in which information is highly reviewed and/or refereed, and this information is updated as the knowledge base changes. The Cochrane Collaboration comes to mind as having a well-deserved reputation for maintaining the highest standards in summarizing evidence that is of the highest quality.[18] Likewise, journals come in all flavors, some not refereed at all, some "lightly" reviewed, and some highly refereed by experts in the field. Only a few orthodontic journals have a respected Impact Factor (IF), and this is usually due to a thorough peer-review process that strives to identify and publish only the most meritorious work. The quality of material published in most other venues can drop off quickly if a rigorous review process is not in place.

PubMed provides an opportunity to easily conduct searches of most current journals, even in fields other than orthodontics.[19] Once references are identified, university students or faculty members usually have access to many publications via their university affiliations. This privilege is available through the institutional subscription paid for by the university. Although open source for all published scientific journals is something appealing to scholars and some publishers, it is still not universally available. Someone still has to pay the cost of publishing scientific findings. Options include charging the authors for every page published, while others favor advertising. Banner advertising is the most common type, but in the future it may be common for publishers to electronically push groups of articles that have common interest to specific subscribers, with all paid for by commercial entities. As long as the advertisers do not select the articles, this convenience to readers is considered ethical. Many organizations already select and screen studies of the highest level in specific fields to provide to members of the organizations. They might be notified by e-mail every month or whenever another study is added to the collection. The American Dental Association (ADA)[20] and the American Association of Orthodontists (AAO)[21] provide this member service, as do the librarians of most universities and scholarly organizations.

Evidence-based clinical recommendations are developed through critical evaluation of the collective body of evidence on a particular topic to provide practical applications of scientific information that can assist orthodontists in clinical decision making. In addition to scientific journals, dental schools, and approved courses, sources of this information may be found in the following locations:

1. **PubMed**[19] - The National Library of Medicine's searchable database of more than 12 million indexed citations from more than 4600 medical, dental, health, and scientific journals.
2. **Cochrane Collaboration**[18] - An international nonprofit organization that develops evidence-based systematic reviews on health care interventions.
3. **ADA Center of Evidence-Based Dentistry**[20] - A resource for evidence-based dentistry that is periodically updated and accessible to dentists and the public. It provides a resource on evidence-based terms, houses guidelines, and catalogs systematic reviews for general dentistry, as well as specialty areas. It also provides critical summaries of some systematic reviews.
4. **Websites for various dental specialty organizations** - An example is the evidence-based Website that the AAO Library[21] maintains at its member Website.
5. **Textbooks** - There are several textbooks on evidence-based dentistry and two textbooks on evidence-based orthodontics.[22,23]
6. **Evidence-based Journals** - There are two evidence-based journals, the *Journal of Evidence-based Dental Practice* and *Evidence-based Dentistry*.[24,25]

THE FUTURE OF EVIDENCE-BASED ORTHODONTICS

Orthodontists have been asking "Why, What, and How" for more than a century. For example, "Why do some patients have such bad growth patterns, what is the best way to treat them, and how can we ensure that these corrections will be stable?" Or, "Why do some patients exhibit severe root resorption, what can we do to minimize it, and how can we predict who will be susceptible?" Although we have volumes of publications on orthodontic treatment, is seems that our research has often failed to produce sound, conclusive evidence for many important questions. Obviously, our traditional methods of research have not been adequate, and this is why our profession needs to employ an evidence-based approach toward education, research, and practice.

One exciting opportunity for our profession is the emergence of dental practice–based research networks. Borrowing from a medical model in which physicians conduct clinical research in their own practices, the National Institutes of Health has developed regional and national dental research networks over the past decade. The most recent endeavor in the United States,

FIGURE 7-11 The National Dental Practice-Based Research Network is funded until 2019. (National Dental Practice-Based Research Network (Website): http://www.nationaldentalpbrn.org. Accessed 09.28.15.)

the National Dental Practice-based Research Network, targets questions related to general dentistry (Fig. 7-11).[26] However, specialists are encouraged to join the network and to submit projects that would be of importance to their field. The American Association of Orthodontists (AAO) is aware of this opportunity and has encouraged orthodontists to submit important questions to this national network.

The advantages of this type of research are many. First, the research topics originate from clinicians and reflect the real-life questions that practitioners would like to have answered. Second, experienced researchers can assist with designing and implementing studies. This helps to ensure the validity of the findings. Third, the research is conducted in private offices, rather than in the sometimes artificial setting that can be associated with university research. Fourth, the network is able to allow simultaneous participation from the network practitioners. Therefore, the results will be much more informative regarding practice patterns and will also be more generalizable. Fifth, the pool of potential subjects is greatly expanded by utilizing a network or practitioners, which is particularly helpful if conditions or treatments may be relatively rare, like severe root resorption. Sixth, researchers will be able to work with network statisticians and epidemiologists in analyzing and interpreting the data. And finally, once the results of the investigation are obtained, the network provides the ideal vehicle for dissemination of information.

The current National Network in the United States was awarded more than $66 million by the National Institute of Dental and Craniofacial Research to conduct practice-based research in dentistry. Hopefully, by the time you are reading this chapter, our profession will have launched two or three studies in this setting, and perhaps you will even be a participant in one of the studies.

Another trend will be the accumulation of more and more systematic reviews and meta-analyses, which will continue to be conducted and revised with better methods and also based upon better evidence. These reviews will eventually cover almost every topic in orthodontics and will become the standard method for obtaining information. The American Board of Orthodontics modifies its reading list for the written exam every year, and based on recent trends, perhaps within a decade, most of the references on this list could be systematic reviews and meta-analyses. Having teams of experienced academicians search and summarize the literature makes sense. It is a tedious and methodical process and probably not a responsibility that the average busy practitioner would wish to shoulder. However, it is absolutely the responsibility of every clinician to be very familiar with the components of systematic reviews and meta-analyses. As methods to perform reviews continue to improve, we can feel more confident in the conclusions that are published. However, practitioners must never relinquish the skills to judge the quality of our literature. These skills will increasingly be the ones that apply to judging systematic reviews and meta-analyses.

Technology will also help us. We will be asking our smartphones and smartwatches to retrieve the evidence for a particular clinical question. Our patients will, too, and we may be pressed to stay as current as some of them. As the evidence becomes more available, perhaps it will help our profession to arrive at some consensus on the effectiveness and stability of various treatments, techniques, and procedures. In a nutshell, the future is promising, due to better evidence, better summaries of our evidence, and better access to our evidence.

CONCLUSIONS

In conclusion, we should all be grateful that evidence-based methods have become incorporated into the specialty of orthodontics over the past decade. They have helped us to assess the state of our literature and to recognize its deficiencies. Evidence-based methods have also challenged us to conduct more rigorous studies. After more than a century as a specialty, we still have many important questions to answer. Evidence-based methods will help us to investigate these questions and then allow us to translate this knowledge into providing the safest and most effective, efficient, predictable, and stable treatments to our patients. This is the reason we are all part of the orthodontic profession, and this is why evidence-based orthodontics is so important.

REFERENCES

1. Wahl N. A little house in Pasadena. *Angle Orthod*. 1989;59(1):69–76.
2. Cochrane A. *Effectiveness and Efficiency: Random Reflections on Health Services*. Nuffield Provincial Hospitals Trust; 1972.
3. Sackett DL, Haynes RB, Guyatt GH, Tugwell P. *Clinical Epidemiology: A Basic Science for Clinical Medicine*. 2nd ed. Lippincott Williams & Wilkins; 1991.
4. ADA Evidence-Based Dentistry Website. http://ebd.ada.org/en/about/. Accessed 03.12.15.
5. CODA. Accreditation Standards for Dental Education Programs. Revised July 1, 2013. Educational Environment, 11–13.
6. CODA. Accreditation Standards for Advanced Specialty Education Programs in Orthodontics and Dental Facial Orthopedics. Revised Feb 6, 2015. Standard 4, 25.
7. The 2011 Oxford CEBM Evidence Levels of Evidence (Introductory Document). Oxford Centre for Evidence-Based Medicine. http://www.cebm.net/index.aspx?o=5653. Accessed 03.12.15.
8. Explanation of the 2011 Oxford Centre for Evidence-Based Medicine (OCEBM) Levels of Evidence (Background Document). Oxford Centre for Evidence-Based Medicine. http://www.cebm.net/index.aspx?o=5653. Accessed 03.12.15.
9. Angle EA. *Treatment of Malocclusion of the Teeth*. 7th ed. SS White; 1907.

10. In: Higgins JPT, Green S, eds. *Cochrane Handbook for Systematic Reviews of Interventions*. The Cochrane Collaboration; 2011 Version 5.1.0 [updated March 2011].
11. Smith GC, Pell JP. Parachute use to prevent death and major trauma related to gravitational challenge: systematic review of randomised controlled trials. *BMJ*. December 20, 2003;327(7429):1459–1461.
12. OCEBM Levels of Evidence Working Group. The Oxford levels of evidence 2. Oxford Centre for Evidence-based Medicine. http://www.cebm.net/index.aspx?o=5653. Accessed 03.16.15.
13. Ebell MH, Siwek J, Weiss BD, et al. Strength of recommendation taxonomy (SORT): a patient-centered approach to grading evidence in the medical literature. *Am Fam Physician*. February 1, 2004;69(3):548–556.
14. Guyatt GH, Oxman AD, Vist GE, GRADE Working Group, et al. GRADE: an emerging consensus on rating quality of evidence and strength of recommendations. *BMJ*. April 26, 2008;336(7650):924–926.
15. Thiruvenkatachari B, Harrison JE, Worthington HV, O'Brien KD. Orthodontic treatment for prominent upper front teeth (Class II malocclusion) in children. *Cochrane Database of Systematic Reviews*. 2013;(11). Art. No.: CD003452.
16. Chen H, Liu X, Dai J, Jiang Z, Guo T, Ding Y. Effect of remineralizing agents on white spot lesions after orthodontic treatment: a systematic review. *Am J Orthod Dentofacial Orthop*. March 2013;143(3):376–382.
17. US government is poised to withdraw longstanding warnings about cholesterol. http://www.washingtonpost.com/blogs/wonkblog/wp/2015/02/10/feds-poised-to-withdraw-longstanding-warnings-about-dietary-cholesterol/. Accessed 03.12.15.
18. Cochrane Collaboration. http://www.cochrane.org/. Accessed 03.12.15.
19. PubMed. http://www.pubmed.gov. Accessed 03.12.15.
20. American Dental Association. http://ebd.ada.org/. Accessed 03.12.15.
21. AAO Evidence-based Orthodontic Research. https://www.aaoinfo.org/library/research/evidence-based-orthodontic-research. Accessed 03.12.15.
22. Huang GJ, Richmond, Vig KWL. *Evidence-Based Orthodontics*. Wiley-Blackwell; 2011.
23. Miles P, Rinchuse DJ, Rinchuse DJ. *Evidence-Based Clinical Orthodontics*. Quintessence; 2012.
24. Evidence-Based Dentistry. http://www.nature.com/ebd/index.html. Accessed 03.12.15.
25. Journal of Evidence-Based Dental Practice. http://www.jebdp.com. Accessed 03.12.15.
26. National Dental Practice-Based Research Network. http://www.nationaldentalpbrn.org. Accessed 03.12.15.

8

The Decision-Making Process in Orthodontics

Tung Nguyen and William Proffit

OUTLINE

Background Concepts, 208
- Quality of Life: The Modern Health Care Paradigm, 208
- Understanding Normal versus Ideal Occlusion, 209
- Dental and Skeletal Compensations: Nature's Way of Camouflaging Discrepancies, 210
- Recognizing Orthodontic Problems, 211
- Limitations of Orthodontic Treatment, 213

Patient Evaluation: The Diagnostic Process in Orthodontics, 215
- Overview of the Problem-Oriented Approach, 215
- Collection of Interview Data, 216
  - *First Contact, 216*
  - *Interview at First Appointment, 216*
  - *Meeting the Patient and Eliciting the Chief Concern, 216*
- Clinical Evaluation, 217
  - *Facial Proportions and Appearance, 217*
  - *Intraoral Examination, 217*
- Diagnostic Records, 219
  - *What Records Are Needed?, 219*
  - *Technologic Advances in Diagnosis, 219*
- Analysis of Diagnostic Records, 222
  - *Cast Analysis, 223*
  - *Cephalometric Analysis, 223*
- Classification: Organizing the Database, 226
  - *Angle Classification, 226*
  - *Systematic Description: Ackerman-Proffit Classification, 226*
- Summary of Diagnosis, 229

Treatment Planning: The Problem-Oriented Approach, 230
- Prioritizing the Problem List, 230
- Considerations in Evaluating Treatment Possibilities, 232
  - *To Extract or Not to Extract?, 232*
  - *Therapeutic Modifiability, 234*
- Presenting the Tentative Treatment Plan, Finalizing It, and Obtaining Informed Consent, 235
  - *The Patient–Parent Conference, 235*
  - *Informed Consent, 235*
  - *The Final Step: The Treatment Plan Details (Mechanotherapy Plan), 236*

Many of the ideas presented here originated with Dr. James L. Ackerman, and the diagnosis/treatment planning scheme reflects his development of these ideas during a distinguished career in academics and private practice. We thank him for his contributions to this work and to the orthodontics specialty.

This chapter is written primarily for residents in orthodontics and for current practitioners who want an update on the changes in diagnosis and treatment planning that have affected orthodontics in recent years. Because the purpose is to outline the basics of orthodontic assessment and treatment planning without going into detail about all aspects of the process, it should be used as a companion piece to Chapters 1, 6, and 7 in *Contemporary Orthodontics*.[1] It is also a natural introduction to Chapter 9, which is a detailed analysis of dentofacial appearance from an orthodontic point of view.

A competent orthodontist today must use broad background information in the life sciences and social sciences in assessing and planning treatment for patients, and a comprehensive evaluation is a necessity. The goal of this chapter is to review the many steps in the decision-making process in orthodontics while demonstrating how a comprehensive evaluation can be quickly and seamlessly telescoped into a practical guide for everyday practice.

BACKGROUND CONCEPTS

Before we begin the steps in evaluation of a patient and continue to treatment-planning principles, some important concepts underlying orthodontic diagnosis and treatment planning are important to discuss.

Quality of Life: The Modern Health Care Paradigm

Until fairly recently, the goal of health care was conceived as primarily the control of disease and infirmity. Orthodontists struggled for many years to fit "correction of malocclusion" within the confines of this narrow definition. Today, the

concept of health is a state of complete physical, mental, and social well-being, and the goal of treatment now is to maintain and enhance the patient's quality of life.

For orthodontics, this has tipped the scales from greatest emphasis on dental occlusion, usually defined as the static relationship of the teeth when they are brought into occlusion, to far more weight placed on facial appearance and tooth display. It is simply a fact of life that appearance, particularly facial appearance, has a greater effect on how one is perceived in social interactions than performance. The evolutionary psychologist Geoffrey Miller said it well: "Our vast social-primate brains evolved to pursue one central social goal: to look good in the eyes of others."[2] People seek and value orthodontic treatment primarily as a way to overcome or minimize a social handicap that decreases their quality of life.

This does not mean that dental function is an unimportant component of quality of life. It does mean that malocclusion is an inadequate description of orthodontic problems and that ideal occlusion is not the primary goal of modern treatment. Should orthodontic treatment be restricted to only those with major deviations from normal occlusion? Not in a modern view. Nearly every specialty of medicine and dentistry now offers interventions that are enhancements of normal traits or conditions rather than correction of disease. Medical examples are cosmetic plastic surgery and medications like fluoxetine (Prozac) for patients who desire to feel "better than well."[3] In dentistry, tooth whitening, clinical crown lengthening, and porcelain veneers are examples of enhancement technologies designed to transform normal conditions to states beyond normal. This trend has had remarkable implications for orthodontic diagnosis and treatment planning, in the context of altered goals of treatment.

In an operational sense, the goals of modern orthodontic treatment are to:

- Improve smile and facial appearance with resultant improvement in an individual's social well-being and quality of life.
- Establish normal oral function and performance, allowing for an adequate range of physiologic adaptation.
- Obtain optimal proximal and occlusal contact of teeth (occlusion), defining optimal occlusion in the context of the preceding goals. It is apparent now that there is no one ideal occlusion that is optimal for every patient.
- Achieve stability of the dentition within the bounds of expected physiologic rebound.

Understanding Normal versus Ideal Occlusion

The concept of ideal dental occlusion is a surprisingly recent one and dates back only to Edward Angle, who for all practical purposes invented dental occlusion[4] (the term was not used to describe dental relationships previously) and popularized the terms *occlusion* and *malocclusion* in his development of a classification with four categories:

- Normal occlusion, defined from two characteristics:
  - alignment of the teeth within each arch along a "line of occlusion" through the central fossa of the maxillary teeth and along the cusp tips of the mandibular teeth (Fig. 8-1).
  - occlusion, with the mesiobuccal cusp of the maxillary first molar occluding with the central fossa of the lower first molar. With the teeth aligned along the line of occlusion, this would produce normal occlusion (Fig. 8-2).
- Class I malocclusion, with a normal molar relationship but teeth not aligned along the line of occlusion.

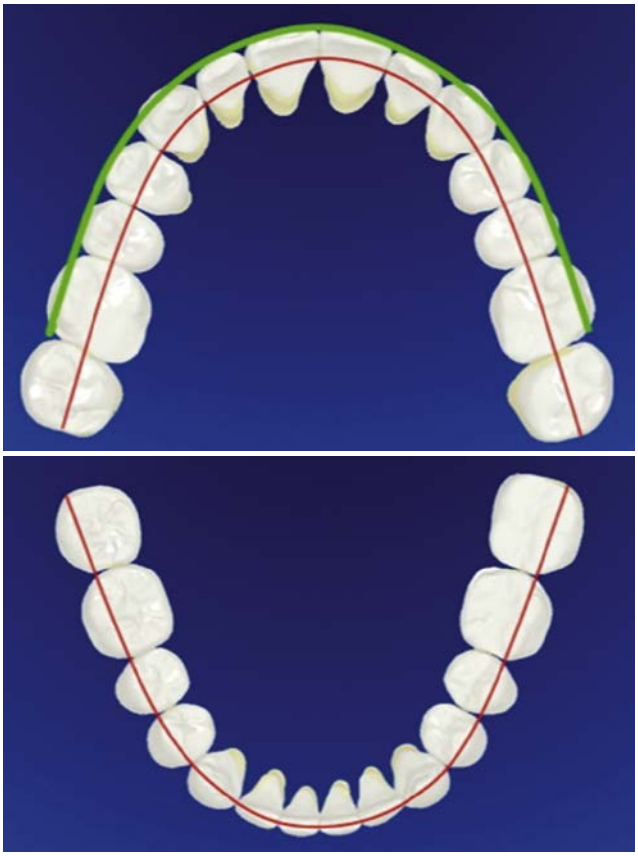


FIGURE 8-1 Bonwill and Angle proposed that if the buccal occlusal line of the mandibular teeth coincided with the central fossae line of the maxillary teeth, ideal occlusion would result. The only recent addition to this concept is that from an appearance point of view, the line corresponding to the line of occlusion that one sees when an individual smiles is the facial occlusal line (the esthetic line of the dentition), which is shown in green. The anterior segment of the esthetic line of the dentition is called the *smile arc*.

- Class II malocclusion, with the lower molar distally positioned relative to the upper molar.
- Class III malocclusion, with the lower molar positioned mesially relative to the upper molar.

The relationship of the teeth to the line of occlusion was not defined for Class II and III.

In the United States, even with a minor deviations from ideal being accepted as normal, two-thirds of the population have one of the forms of malocclusion.[5] Usually a typical trait in a population is considered normal and an atypical trait (in this case an ideal arrangement of the teeth) is considered abnormal. In the older model of health care where practitioners treated abnormal conditions or corrected deformities, how could orthodontists justify treating malocclusion if it was neither atypical nor a major abnormality? Even before the quality of life paradigm was widely accepted, they did it because patients wanted it, for reasons that we now understand more clearly.

The modern goals of orthodontic treatment make it clear that problems needing orthodontic treatment could arise in the absence of malocclusion. For example, ideal occlusion accompanied by dentoalveolar protrusion with excessive facial convexity and lip incompetence, in a patient whose chief concern is social problems

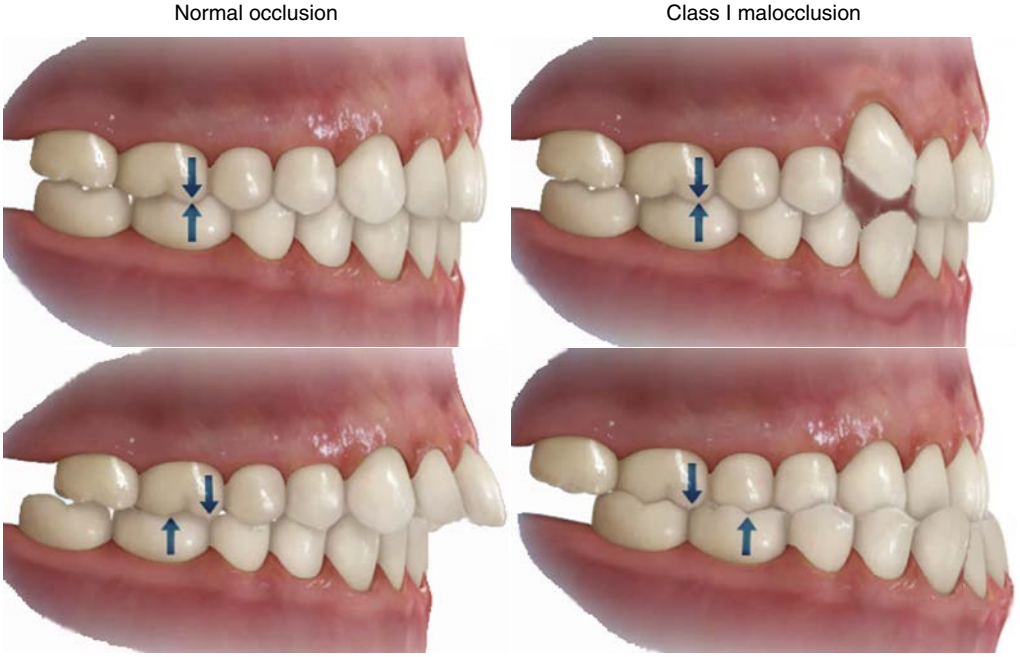


FIGURE 8-2 Angle suggested that in ideal occlusion, the mesiobuccal cusps of the maxillary first molars should rest in the buccal grooves of the mandibular first molars. He called the permanent first molars the keys to occlusion and designated this ideal molar relationship Class I. He observed that two variations of this molar relationship exist and he designated these Class II and Class III. Subsequently, the relationship of the canines also became part of the description of Class I, II, and III, probably as a result of Simon's influential but erroneous belief that the maxillary canines were the keys to occlusion. Nonetheless, the maxillary canines should ideally fit in the embrasures between the mandibular canines and first premolars.

related to appearance, is a definite indication for treatment (one that Angle refused to accept—if you did not like the way you looked with protrusive teeth in ideal occlusion, he questioned your perception). Nor are all malocclusions indications for treatment. Malocclusions are only indications for treatment if they create a problem for the patient (or potential problem for a child), in social interactions or functionally. Nonetheless, we are not suggesting completely abandoning the term *malocclusion* but rather restricting its use to contexts where its meaning cannot be misconstrued, and using *orthodontic problem* in the larger context of indications for treatment.

Are there disease/pathology considerations related to malocclusion, so that orthodontics is needed to treat or control them? Until fairly recently, it was thought that unstable occlusion promotes periodontal disease, because drift of teeth is associated with periodontal bone loss. This is a misinterpretation of cause and effect.[6] In fact, it is loss of periodontal attachment resulting from periodontal disease that causes teeth to drift, not vice versa. It was also believed that traumatic occlusion was a primary factor in causing periodontal problems; it is recognized now that occlusal trauma, if associated at all with a periodontal problem, is a secondary, not a primary, factor in its causation.

Another possible misinterpretation of the role of occlusion in oral health resulted from erroneous conclusions about the "high filling" effect. If a restoration is placed so that it creates a premature occlusal contact, the patient often develops tooth pain lasting days or even weeks. This finding supported the theory that faulty occlusion created microtrauma that caused pulpal hyperemia as well as periodontal lesions. In orthodontics today, composite is often added to the occlusal surfaces of the posterior teeth temporarily to serve as mini–bite planes without patients complaining of dental pain. It is likely that pulpal hyperemia associated with a high filling is primarily the result of caries removal during tooth preparation for restoration, with occlusal trauma being only a secondary or compounding factor in causing pain.

The same type of misinterpretation of causality has been responsible for erroneous conclusions regarding occlusion and temporomandibular pain/dysfunction (TMD).[7] It is now widely accepted that flawed occlusion is not a primary factor in causing TMD. In some individuals, occlusal prematurities can lead to the parafunctional habit of clenching or bruxing, thereby secondarily causing muscle spasm, fasciitis, or tendonitis that results in pain in the region of the TM joint. However, there is no evidence that malocclusion without premature contacts leads the TMD or that any specific occlusal scheme within the normal range of variation provides more efficient mastication.

Dental and Skeletal Compensations: Nature's Way of Camouflaging Discrepancies

Perhaps the most common misperception of beginning residents in orthodontics is that if theoretically ideal occlusion can be defined, there must be an ideal skeletal pattern (jaw relationship) as well. Nothing could be further from reality. When ideal occlusion exists, it is because there have been 3D compensatory linear and rotational adaptations in the relationship of the dentition to the underlying skeleton. These structural alterations can be as far removed from the dentition as the cranial base, the

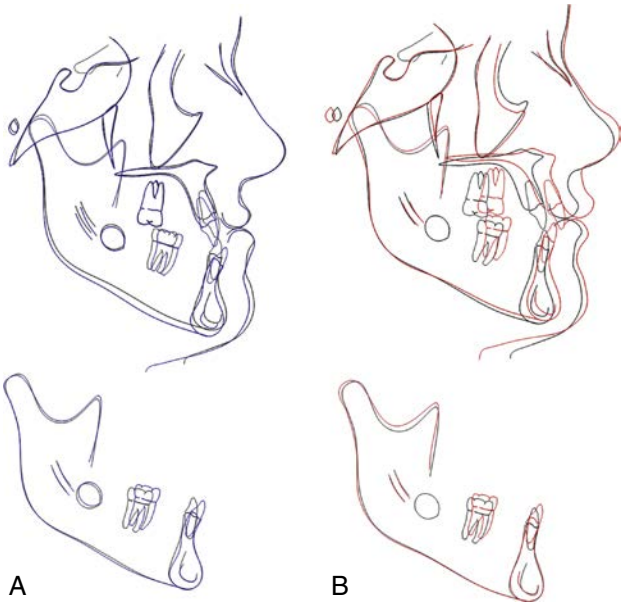


FIGURE 8-3 In preparation for orthognathic surgery, removal of dentoalveolar compensation for the skeletal deformity is often required to optimally position the jaw in a harmonious relationship. This figure of steps in treatment of a Class III problem due to maxillary deficiency shows (A) (black to blue) cranial base and mandibular superimpositions of pretreatment (black) to presurgery (blue), and (B) presurgery to posttreatment (red). Note the retraction of the maxillary incisors and proclination of mandibular incisors in preparation for surgery, so that an adequate amount of maxillary advancement for acceptable soft tissue relationships could be done without dental interferences.

nasomaxillary complex, and the condylar neck and ramus of the mandible. In addition, the corpus of the mandible and the maxillary and mandibular dentoalveolar structures are morphologic features that are remarkably adaptable.

Thus, dental compensations can effectively mask underlying anteroposterior skeletal discrepancies. The simplest demonstration of this is that in Class III skeletal patterns, where either the mandible is too far forward or the maxilla is too far back, the maxillary teeth almost always tip facially (procline), and the mandibular teeth tip lingually (retrocline). The same thing happens in reverse in Class II skeletal patterns, although often not as extensively. The extent to which the teeth are able to compensate determines whether the teeth will be in normal occlusion despite a jaw relationship that predisposes to malocclusion or whether a malocclusion less severe than the jaw discrepancy will develop. Much of orthodontic treatment is simply directed toward completing nature's insufficient compensation, but when the jaw discrepancy is so great that acceptable facial appearance and function can be obtained only by surgery, preparation for the surgical treatment often requires removal of dental compensation so the jaws can be properly positioned (Fig. 8-3 and Case Study 8-1).

Recognizing Orthodontic Problems

Data from the third U.S. National Health and Nutrition Examination Survey (NHANES-III) provide a clear picture of malocclusion in the U.S. population in the 1990s[8] and an important background for evaluating orthodontic treatment need.

Incisor crowding occurs in the majority of all racial and ethnic groups, with only 22% of American adults having well-aligned lower incisors. Incisor irregularities are severe enough in 15% of the population to produce major effects on appearance, function, or both. About 20% of the population have major deviations from the ideal bite relationship (excess or inadequate overbite and overjet), and in 2% the deviations are severe enough to be disfiguring. Less than 10% of the population have posterior crossbites, more than a 6-mm overjet, or more than 6 mm of overbite. Discrepancies in molar relationship of more than 6 mm (i.e., Class II or Class III molars) occurred in 11% to 15% of the people surveyed, with differences between racial/ethnic groups.

If one calculates from the NHANES data the percentage of individuals who fall into Angle's three malocclusion groups, by far the greatest number are Class I (50% to 55%). The next highest group is Class II (15%), and the smallest number is Class III (less than 1% for all other groups but 5% for those of Asian descent). Even with acceptance of minor irregularity and bite deviations, only 30% were scored as normal.

Dentofacial appearance and associated psychosocial issues, not just the way teeth fit together, play a major role in defining orthodontic treatment need. For this reason, determining treatment need just from an examination of dental casts or radiographs is difficult at best. The Index of Treatment Need (IOTN), developed in the United Kingdom,[9] places patients in one of five grades ranging from "no need for treatment" (grade 1) to "treatment required" (grade 5). IOTN has two components: dental health (based on deviations from ideal occlusion) and esthetic concerns (based on the way patients identify themselves relative to a graded set of photographs of malocclusions). As might be expected, a high correlation exists between scores on the two components of the index, which provides some confidence in using the dental health component alone as an indicator of treatment need. A consensus panel of orthodontists established the significance of various occlusal discrepancies, and IOTN grades seem to reflect clinical judgments better than previous methods.

Applying IOTN to the NHANES-III survey data indicates that 57% to 59% of each of the American racial and ethnic groups have at least some degree of orthodontic treatment need. Treatment is much more frequent in higher income groups, but 5% of the lowest income group and 10% to 15% of intermediate income groups report being treated. Although all states are now required to include orthodontic services as part of their Medicaid programs, which in itself is testimony to the importance of overcoming severe orthodontic problems, fewer than 1% of orthodontic patients have their treatment covered through social programs. Despite this, nearly 10% of the adolescents in the lowest income groups and 15% of those in modest income groups now report receiving orthodontic treatment. This shows that even families with modest resources often give orthodontics a high priority because of its role in improving social well-being and a person's social potential in life, as well as quality of life.

Why are orthodontic problems so prevalent? Tooth irregularities and jaw disproportions are developmental conditions resulting from a combination of genetic, epigenetic, and environmental factors. In most instances, malocclusion and dentofacial deformity are caused not by some pathologic process but by moderate distortions of normal development due to intrinsic and extrinsic factors. Occasionally a single specific cause is apparent; for example, an anterior open bite in the early transitional dentition may result from thumb sucking. A more dramatic example of a condition arising from extrinsic causes is mandibular asymmetry secondary to a subcondylar fracture of the jaw in childhood (Fig. 8-4). In some craniofacial syndromes,

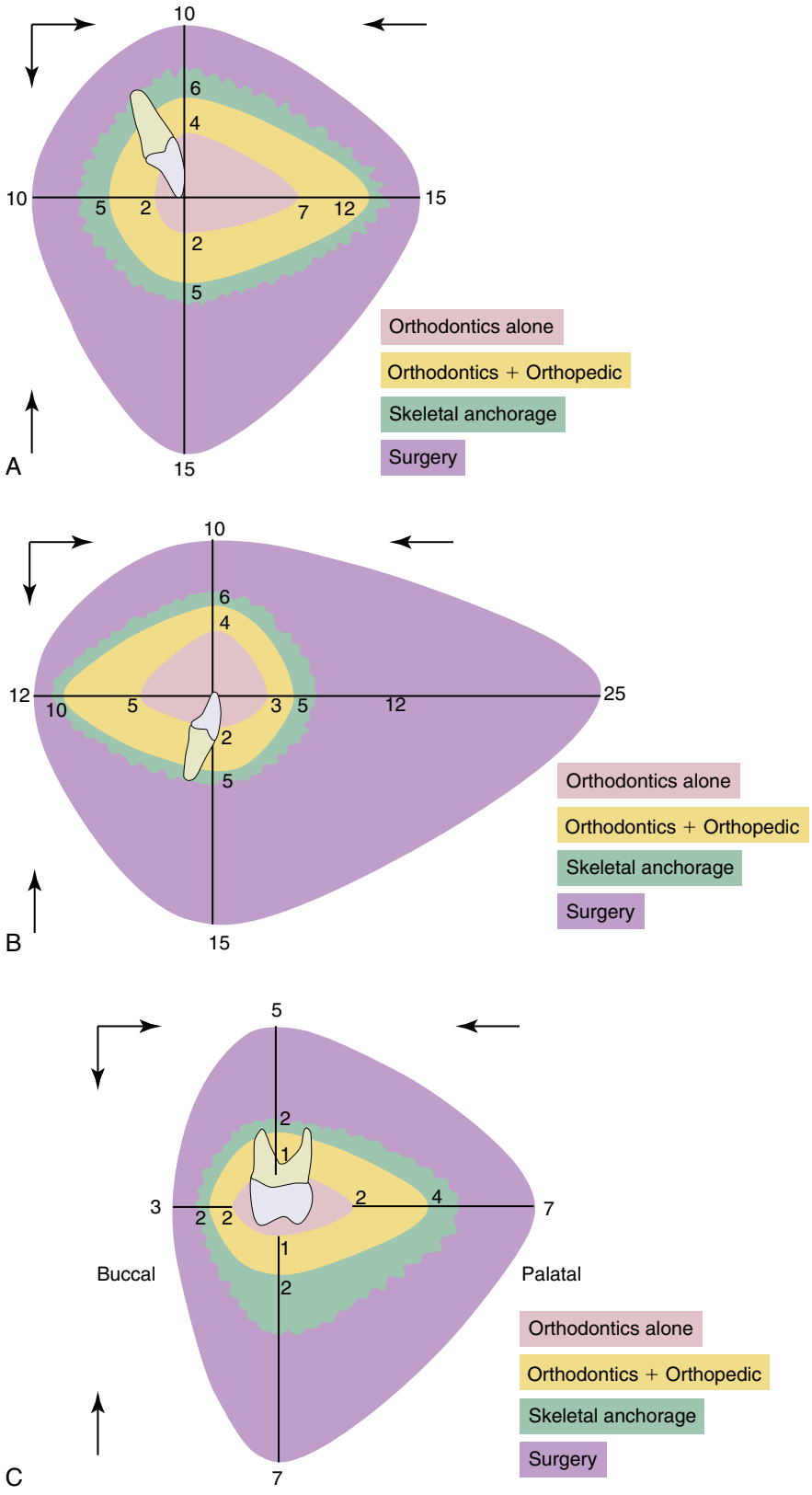


FIGURE 8-4 A-C, The anteroposterior, vertical, and transverse millimetric range of treatment possibilities in orthodontics can be expressed as an envelope of discrepancy. What is meant by treatment possibilities is the amount of tooth movement that can be accomplished by orthodontics alone, orthodontics plus dentofacial orthopedics with or without skeletal anchorage, or orthodontics plus orthognathic surgery. The different colored zones describe the range of potential tooth movement. The arrows designate the direction of the movement in the diagram. The pink zone represents the envelope for orthodontics alone, the yellow zone depicts orthodontics plus orthopedics, the green zone shows skeletal anchorage, and the blue zone any combination of the above with orthognathic surgery. The reason the green zone is shown in "fuzzy" fashion is that there is only sufficiently reliable data to make estimates at this point. The same limitation is the reason there is not a figure depicting the mandibular transverse envelope.

characteristic malocclusions develop from the influence of multiple genes. Most often, however, deviations from normal occlusion result from a complex interaction among many factors that influence growth and development, and it is impossible to describe a specific etiologic factor.

Fundamental to good taxonomy, the science of classification, is the concept that etiology should be included in any classification. The major weakness of all current classifications of malocclusion is that they totally ignore etiology. One of the most exciting aspects of current research to clarify the biological basis of orthodontics is the prospect that advances in genetics will allow differentiating patients within the classic Class I/II/III classification so the specific types of treatments can be related to their pattern of growth.

Limitations of Orthodontic Treatment

One of the most important concepts for a beginning orthodontic resident to grasp is the range of tooth movement that can be accomplished within the biological limits of the system. One way to describe the theoretical boundaries of the potential range of tooth movement is the envelope of discrepancy (Fig. 8-5).

The envelope can be thought of as an elastic 3D, asymmetric closed container. Orthodontics alone rearranges the contents of the container; growth modification treatment and surgery change the shape of the container. For any characteristic of malocclusion, four ranges of correction exist: (1) the amount that can be accomplished by orthodontic tooth movement alone; (2) a larger amount that can be accomplished by orthodontic tooth movement aided by absolute anchorage (bone anchors);

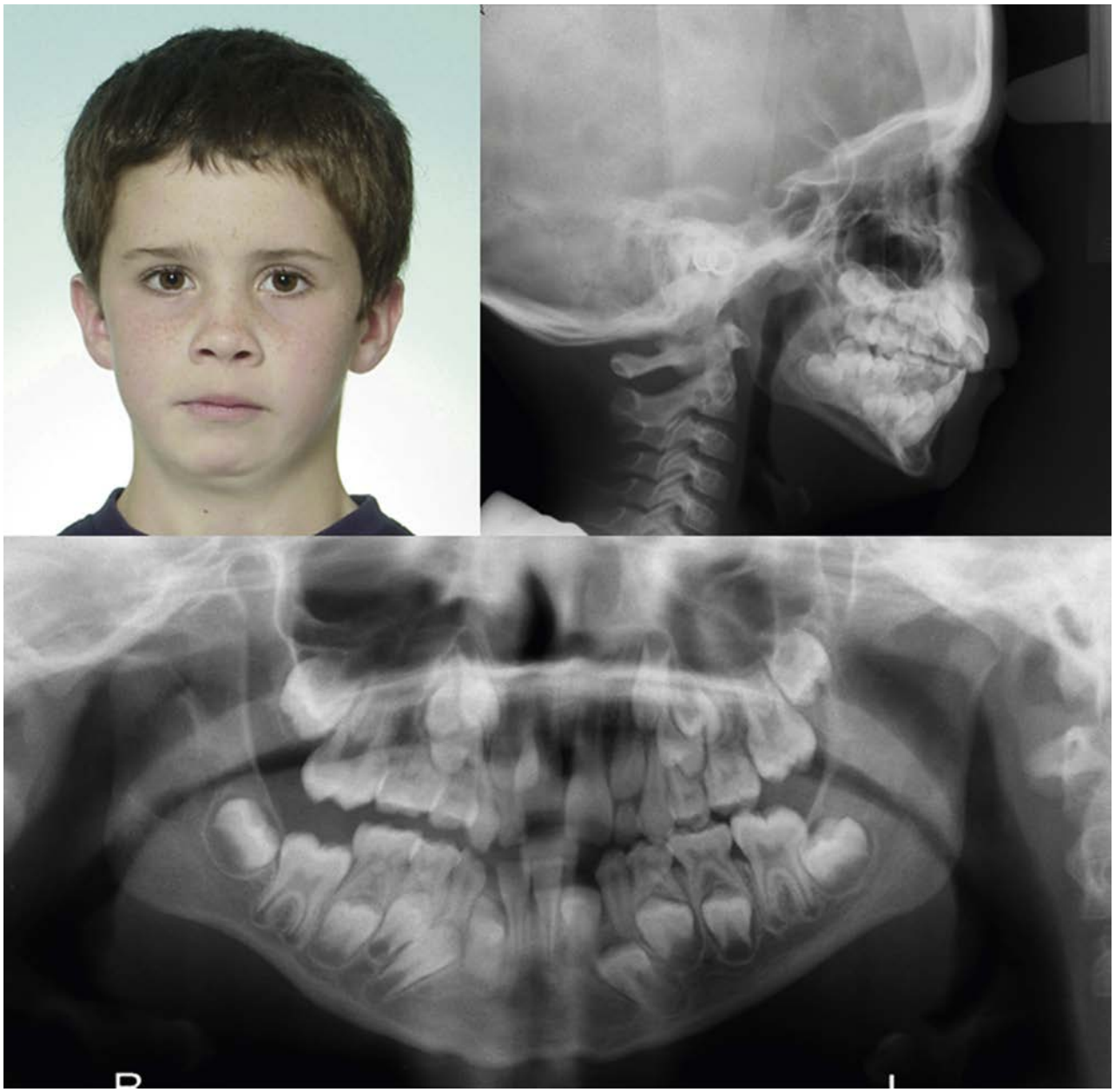


FIGURE 8-5 A patient with an untreated right condylar fracture. The asymmetry can be seen with the chin deviation to the right on the clinical photos, a shorter right condylar neck on the panoramic film, and two distinct lower mandibular border on the cephalometric film. Asymmetries should be diagnosed with a CBCT.

(3) an additional amount that can be achieved by functional or orthopedic treatment to modify growth; and (4) a still larger amount that requires surgery as part of the treatment plan. The magnitude of the potential changes shown in the diagram is a combination of good data for some dimensions and an educated guess for others.

Thus, if a patient presents with an overjet of 7 mm, attributable to forward position of the maxillary incisors, correction of the overjet by retracting the incisors is just within the range of orthodontic tooth movement. Maxillary premolar extractions would provide 7 to 8 mm of space, but 7-mm incisor retraction might require bone anchors to close the extraction space solely by retraction. Correction of greater overjet (which almost surely would have a component of mandibular deficiency as a cause) would require redirection of facial growth with dentofacial orthopedics, and if the mandibular deficiency were severe enough (greater than 10 to 12 mm), a combination of orthodontics and orthognathic surgery would be needed. The same reasoning applies to the transverse and vertical possibilities of orthodontic treatment. In general, orthodontic and growth modification treatment can create larger sagittal (anteroposterior) corrections than in the vertical or transverse planes of space.

The timing of treatment is a factor in the amount of change that can be produced. The amount of tooth movement that is possible is about the same in children as it is in adults. However, the growth modification range diminishes steadily as a child matures and disappears after the adolescent growth spurt, so some Class II and Class III conditions that could have been treated in a growing child with growth modification and tooth movement would require surgery if treated later on. Controlling excessive vertical growth in children is difficult, and TADs can be useful for this in older patients.

The envelope of discrepancy was developed from cephalometric data and thus uses tooth movement relative to the underlying jaw and jaw relationships relative to the cranial base. It is compatible, however, with the newer concept that soft tissue relationships are the key to both dentofacial appearance and treatment limitations.[10] For that reason, the orthodontist must plan treatment within the patient's limits of soft tissue adaptation and soft tissue contours. This requires greater emphasis on soft tissue function and dentofacial appearance during the clinical examination. Limitations in orthodontic treatment related to the soft tissues include (1) pressures exerted on the teeth by the lips, cheeks, and tongue; (2) limitations of the periodontal attachment; (3) neuromuscular influences on mandibular position; (4) the contours of the soft tissue facial mask; and (5) lip–tooth relationships and anterior tooth display during facial animation. The physiologic limits of orthodontic treatment (i.e., the ability of the soft tissue to adapt to changes in tooth and jaw positions) are related to the first three of these limitations and are often narrower that the anatomic limits of treatment shown in the envelope of discrepancy. For instance, in expansion of the lower arch (Fig. 8-6), the envelope is zero for expansion across the canines, broadening to about 4 mm in the molar region. With modern orthodontic appliances and bone anchors, it is quite possible to move the teeth beyond the point of acceptable adaptation, physiologically and esthetically. The esthetic soft tissue limitations relate to facial contours created by jaw and tooth position and to the display of the dentition, both of which must be kept in mind when the acceptability of tooth movement is being determined.

Edward Angle sought perfection in dental alignment and occlusion and thought that this natural condition would have been present if something had not interfered with normal development. The modern view is just the opposite: nature does not intend for the orthodontist to achieve perfection but rather contends with the orthodontist trying to achieve it. Treatment "failures" are generally the result of poor treatment response rather than inadequate treatment. Rather than designating orthodontic outcomes as successes and failures, it is more logical to categorize patients as responders and nonresponders to the treatment procedures. Similarly, since posttreatment relapse is determined by physiologic adaptation and any subsequent growth, postretention patients can be characterized as adapters and nonadapters to the changes that were made. Using this construct, the orthodontic treatment population can be represented by a bell-shaped curve, with the most favorable responders and adapters at one end and the most unfavorable at the other end. For the most part, patients who are presented at meetings as the dramatic successes and failures are merely the outliers on a normal distribution curve. Any individual's position on that curve will be determined, to a great extent, by soft tissue influences on the treatment process and outcome.

Limitations, of course, also relate to the hazards of orthodontic treatment. Known hazards include root resorption, decalcification, pulpal devitalization, fenestrations or dehiscences of alveolar cortical plates, and general alveolar bone loss. It must be kept in mind that some patients are more susceptible to these hazards than others, and advances in genetic analysis may clarify this.

Another major task for the orthodontist in treatment planning is to estimate the effectiveness and efficiency of possible treatment plans, so that the best combination of these important factors can be selected. This determination

FIGURE 8-6 The amount of dental expansion one can safely achieve with orthodontics is related to three major factors: (1) stability, determined largely by the soft tissues; (2) appearance, particularly in regard to profile; and (3) the periodontium, particularly the labial cortical plate of bone and the amount of attached gingivae on the facial aspects of the tooth roots. These constraints make it infeasible to move the mandibular teeth facially more than 2 to 3 mm. To do otherwise would simply be an invitation for instability, unfavorable facial changes, and unfortunate periodontal sequelae. Of course, there are exceptions to every rule.

is the research goal of both careful retrospective studies of treatment outcomes and randomized prospective clinical trials when these are possible. For example, the now classic Class II clinical trials of recent years, which studied the response to early (preadolescent) versus later (adolescent) treatment, showed that despite some individuals responding well to early treatment, on average there was no significant difference in outcome between early two-stage and later one-stage treatment. Does that mean there should be no preadolescent Class II treatment? Of course not. It does mean that patients should be selected for preadolescent treatment for problems specific to that individual child. Relying on these types of data in planning treatment is what is meant by "evidence-based orthodontics."

PATIENT EVALUATION: THE DIAGNOSTIC PROCESS IN ORTHODONTICS

Overview of the Problem-Oriented Approach

Decision making in orthodontics requires the establishment of a prioritized problem list before considering treatment options. In this method, the prioritized problem list becomes the "diagnosis." Essential to the establishment of a complete problem list is the creation of an adequate database. The elements of the database are:

1. Questionnaire and interview data.
2. Clinical examination data including the systematic description of the patient's dentofacial traits (classification).
3. Data from diagnostic records (Fig. 8-7).

The problem list is derived from the database and is prioritized. Tentative solutions are then proposed for the individual problems. Favorable or unfavorable interactions among the tentative solutions are considered, and one or more alternative unified treatment plans are synthesized. The alternatives are presented to the patient, parent, or both, and with their input, an individualized treatment plan and mechanotherapy are established (Fig. 8-8).

The more systematically an orthodontist approaches the collection of adequate diagnostic data, and the more thoroughly he or she interprets these data in terms of expected treatment responses, the better the probabilities will be for successful correction. At the same time, it is necessary to keep in mind the inherent uncertainties in response to treatment so that there is no reluctance to adapt treatment to meet an unexpected turn of events.

Effective use of the problem-oriented approach in evaluation of orthodontic patients requires generation of an appropriate database prior to generating a problem list. In this context, appropriate simply means gathering the information needed for a specific patient. That can vary from a minimal amount when a modest problem in an adult merely requires enhancement, such as the ironing out of a few wrinkles in a smile, to a cone beam CT scan and/or TM joint magnetic resonance imaging (MRI) in a patient with a severe jaw discrepancy and impaired jaw function.

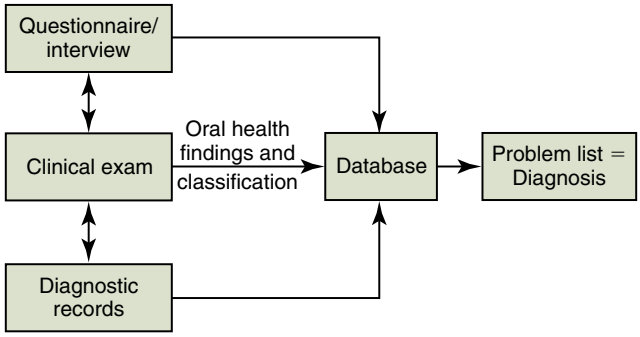


FIGURE 8-7 This flow chart shows the elements of the database and how a problem list is derived from the database. What has changed in recent years is that it is no longer considered necessary to have "complete" orthodontic records before systematically describing the patient's orthodontic condition. Today, the systematic description (i.e., classification) is accomplished during the clinical examination.

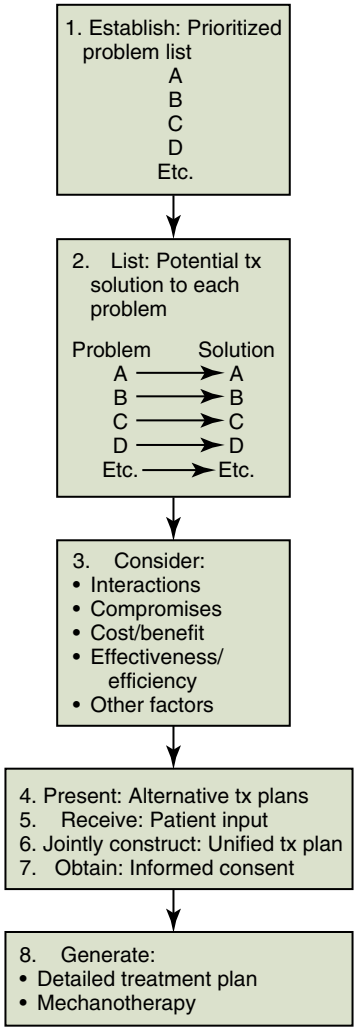


FIGURE 8-8 This flow chart outlines the eight steps that must be taken from the time a prioritized problem list is established to when a detailed treatment plan and mechanotherapy are generated. The essential components of the process are (1) being sufficiently careful to not overlook any aspect of the problem, (2) taking enough time to solicit patient–parent input, and (3) being certain that the patient–parent decision is an informed one. The number of visits required to accomplish these eight steps is determined by the complexity of the orthodontic condition and the orthodontist's practice management style.

Collection of Interview Data

First Contact

Data collection and development of the diagnostic database begin with the very first encounter with the patient or parent. This first contact is almost always by telephone, and important demographic information, including the e-mail address, should be obtained at this time. The patient's age, the source of the referral, the family dentist, and other patients and families whom the prospective patient knows are all clues regarding what the patient and family may already know of an orthodontist's practice. Obviously, if the call is to set up an appointment for the sibling of a patient already in the practice, there is usually instant rapport. If the prospective new patient is another patient's best friend in school, it is likely that she or he already has great awareness of the practice. Today, the majority of patients' parents have had orthodontics themselves, and it is generally easy to quickly integrate them into one's practice routine.

Just as it would be an error in taking for granted that the parent is an old hand at orthodontics, there is an equal danger of having the caller feel the receptionist is talking down to them. The ease or difficulty with which the receptionist can schedule the first appointment may indicate the types of demands this family may make and the cooperation that might be received in the future. All information should be entered directly into the office's computer system, including discrete notes regarding the receptionist's first impressions. When he or she takes the phone call, the receptionist should have a window opened on the computer screen that will serve as a checklist to guide in systematically collecting these data. Usually, the receptionist will be able to glean the motivation for treatment during the first call. This is the first step in building an orthodontic database.

At the end of the initial telephone call regarding scheduling a patient evaluation, the caller should be informed that if they would be kind enough to fill out a patient questionnaire and have it ready at the first visit, as it will greatly facilitate the initial appointment. The caller should have the option of downloading the questionnaire from the office website, having it e-mailed as an attachment, or having it mailed as a hard copy.

Interview at First Appointment

A well-designed patient information questionnaire allows the patient or parent to provide the medical and dental health history so that all positive findings prominently stand out. Then, a simple glance at the questionnaire alerts the clinician to the questions that require follow-up. A few major questions always must be asked. The first, of course, is when the patient last saw his or her physician. If it was within the past year and was for a regular checkup, this usually is a good sign.

Another important question is whether the patient has ever been hospitalized and, if so, for what reason. For prospective orthodontic patients, one usually includes a specific question as to whether the patient has had tonsillectomy, adenoidectomy, or both. This may be a clue that the patient had an earlier airway problem, which might have affected jaw and tongue posture. Sometimes the admission to the hospital was the result of trauma, and it is important to know whether the jaws, face, or teeth were involved. If the injury involved one or more teeth, a closer evaluation of the vitality of the teeth involved is clearly indicated, and the patient or parent should be made aware that orthodontic tooth movement can possibly exacerbate periapical symptoms. Because parents do not realize the relationship between overall health, dental health, and dentofacial development, persistence in pursuing these questions is important.

The next issue that must be considered is whether the patient is taking any medications. Occasionally parents are reluctant to inform the orthodontist in front of the child that seizures (epilepsy) has been a problem, but they will indicate that phenytoin (Dilantin) or some other anticonvulsant drug is being taken. This will not only influence the management of the child in regard to medical emergencies but also influence tooth movement if there is gingival hyperplasia. If a patient is taking medication typically prescribed for attention-deficit disorder, the issue of potential compliance with treatment should be explored further. If a patient has recently been prescribed isotretinoin (Accutane) for severe cystic acne, the orthodontist and patient should be aware that severe lip dryness with cracking is a common side effect of this medication and that becoming pregnant while taking Accutane is associated with a high risk of birth defects.[11] In adults being treated for arthritis or osteoporosis, high doses of prostaglandin inhibitors or resorption-inhibiting agents may impede orthodontic tooth movement. These examples should serve as a reminder that an orthodontist must know the contraindications of orthodontic treatment and be able to rule out that any of these factors are involved with any given patient.

Meeting the Patient and Eliciting the Chief Concern

Before the orthodontist meets the patient at the first appointment, the demographic and historical information should be reviewed, and photographs and a panoramic radiograph should be available. The advent of digital photography and radiography has markedly improved the ease and efficiency of obtaining these records immediately prior to the orthodontist meeting the patient.

Some practitioners prefer to have the parent present when he or she meets the patient; most find it advantageous to examine a child patient independently first and then invite the parent to the treatment area to receive the report. It is sometimes far easier to establish rapport with the patient if there is no parent present. "Helicopter" parents who insist on hovering over their children in every situation should be given the choice of being present if they wish. It is poor form for a staff member or orthodontist to say that the parent's presence or absence is an "office policy," because most people resent rigid adherence to some arbitrary rule.

At the first meeting, the orthodontist should not assume that appearance is the patient's major concern just because the teeth appear unattractive. Nor should the dentist focus on the functional implications of, for instance, a crossbite with a lateral shift, without appreciating the patient's concern about what seems to be a trivial space between the maxillary central incisors. As we have noted, for an individual with what appears to be reasonably normal function and appearance and appropriate psychosocial adaptation, the major reason for treatment may well be a desire to improve appearance "beyond normal." The greater orientation of modern family practice toward cosmetic dentistry increases the chance that a patient may be referred to an orthodontist for comprehensive treatment to improve dental and facial appearance.

From the outset, the orthodontist must determine whether the prospective patient is a suitable candidate for treatment, because there are notable exceptions to the validity of self-determination of the need for orthodontic intervention. An example is an adult patient with body dysmorphic disorder (BDD), which is a condition marked by excessive preoccupation

with an imaginary or minor defect in a facial feature or localized part of the body.[12] These individuals almost always have unreasonable expectations as to how a change in one or more of their dentofacial features will alter their sense of social well-being and quality of life. They can be very persuasive, and they often goad health professionals into performing treatments against the professionals' better judgment. If BDD is suspected, the patient and the orthodontist are both well served by seeking a consultation with a mental health professional. Potential adult patients who might have a mild form of BDD may be more prevalent than currently suspected. The orthodontist also must be wary of parents (the "pageant mom") who push for early treatment with questionable benefit or adolescents who have unrealistic expectations about what orthodontic treatment might accomplish for them.

Perhaps the easiest and most direct way to find out how the patient feels about orthodontic treatment is to ask the simple question, "Do you think you will need braces?" Most children today think that braces are inevitable and thus usually answer affirmatively. Occasionally, the reaction is a shrug of the shoulders. Only rarely will a child say that although his (or her) parents think he should have braces, he does not want them. For the patient who responds with a shrug, the important follow-up question is, "If your parents and I think you will be helped by braces, will you go along with the recommendation?" Children or adolescents who do not appear to be motivated to have treatment rarely exercise good oral hygiene or elastic wear during treatment. From almost anyone's perspective, it is much more acceptable to have treatment that is done for you than to have treatment that is done to you. In this circumstance, there is merit in telling the parent that postponing treatment until the patient is either more mature or simply more motivated may be the best alternative.

For the individual who is convinced he or she will require orthodontics, an important question is whether any one feature is of greater concern than another. It is important to know this for two reasons. First, one of the most embarrassing mistakes an orthodontist can make is failing to address an issue that is of major concern to the patient. The orthodontist may or may not agree with the patient's assessment—that judgment comes later. At this stage, the objective is to find out what is important to the patient. Second, to allow for the most effective treatment planning, it is important to take into consideration what dentofacial trait or traits are most important to the patient or parent when prioritizing the problem list.

Clinical Evaluation

Facial Proportions and Appearance

Although the sequence of steps in completing the database can vary depending on the complexity of the case (see further discussion later), a comprehensive clinical examination usually follows immediately after the interview with the patient. The orthodontist should make some diagnostic determinations "from the doorway" regarding the patient's face, posture, and expression. One can often tell from the first moment whether the orthodontic problem will be largely a dental one or a difficult skeletal or facial problem.

The evaluation of facial appearance should be done with the patient's head in a natural head position (NHP) (i.e., standing or sitting up, looking at the horizon), not with the patient prone in a dental chair. To determine lip incompetence (more than 4-mm separation at rest), the frontal view should be assessed first in repose and then with the lips sealed, which would show evidence of lip strain to confirm incompetence. The patient is then observed during facial animation while speaking (when there should be 2-mm exposure of the teeth) and in a social (posed) smile (when there should be exposure of at least half the crown of the maxillary incisors but not more than 2 to 3 mm of gingiva). Greater exposure is expected in the spontaneous (gratification) smile. After all, it is ultimately anterior tooth display (the social smile zone) over which the orthodontist has the greatest control (see Chapter 9).

In assessing the face in its broadest context, one tries to rule out any genetic defects or partial expression of genetic defects. The distance separating the eyes can often give a clue to this kind of problem. In a number of genetic defects affecting the face and teeth, one frequently finds hypertelorism (eyes that are too far apart). Malformations of the ears may be associated with one of the brachial arch syndromes, which can affect the mandibular condyle.

Although orthodontists in private practice rarely treat patients with malformations other than cleft lip and/or palate, it is important to consider the possibility of other syndromes. A patient with severe mandibular retrognathia at age 15 years, for instance, may have had a Pierre Robin sequence and earlier in development may have had a more pronounced problem. Frequently, knowledge of this type does not markedly affect the treatment plan, but it does often temper the treatment goals based on therapeutic modifiability. Sometimes a surgical approach to treatment will be selected, based on the recognition that one is dealing with the result of a pathologic process rather than normal anatomic variation. For a more complete account of this subject, see Gorlin et al.[13]

After having assessed the overall head and face, the orthodontist then focuses on the lower face, which is most easily affected by tooth position. Lip prominence is evaluated relative to the nose and chin. A large nose and well-developed chin can easily mask what otherwise would be judged a protrusive dentition, and thereby keep it from being a problem. Similarly, the opposite situation of a small nose and weak chin can create a protrusion problem.

Although the facial photographs and lateral cephalogram that are obtained after the clinical examination are helpful in assessing certain aspects of facial appearance, some features must be examined chairside. Assessment of the dental midline as it relates to the midline of the face and the symmetry of the face are examples. Lip competence is another. Can the patient or does the patient keep his or her lips approximated when at rest, and is this done with ease or strain? Does the patient, when in repose or smiling, have a high upper lip line showing a wide band of gingiva? Chapped lips and inflamed gingiva in the maxillary anterior region are often indications of a patient who is a mouth breather or whose oral seal is inadequate because of extremely protrusive teeth.

Intraoral Examination

Health of hard and soft tissues. Once the visual and tactile examination of the face is complete, an evaluation should be made of the intraoral hard and soft tissues. This will immediately reveal the general oral health of the patient. Just as the orthodontist should view the overall health of the child broadly, he or she should also look at oral health from the broadest possible perspective. What has been the caries incidence? How faithful has the child been with home care, and, generally speaking, what is the oral health picture? Some orthodontists begin plaque control programs for children before initiating orthodontic treatment. In

university clinics, patients are usually not accepted for treatment until they can demonstrate adequate home care. The experienced orthodontist knows that this is as fundamental to success in orthodontics as the appliance that is used.

Poor gingival health adversely affects tooth movement and may progress to a more significant periodontal problem. Most periodontal sequelae of orthodontic treatment are self-correcting once the orthodontic appliances are removed, but enamel decalcification resulting from poor hygiene can mar an otherwise beautiful orthodontic result. It has been suspected that systemic disorders such as allergies may be associated with root resorption, but when this hypothesis was tested, no statistically significant correlations were found.[14] It is believed that prolonged treatment can increase the risk of root resorption, devitalization, and obliteration of the pulp chambers, although at present little is known about the undoubted molecular genetic basis underlying these processes. Hazards of orthodontic treatment of this type can be minimized with careful diagnosis and treatment planning.

As part of visual and tactile examination of the dentition, it is important to count the teeth. It is particularly easy to overlook a missing lower incisor. A quick check should be made for mobility of primary or permanent teeth. In the mixed dentition, one should palpate for unerupted canines, since it often is not possible to ascertain from the radiographs whether these teeth are erupting labially or lingually. Ankylosed primary teeth usually appear submerged. Tapping these teeth with the handle of a dental instrument usually produces a somewhat higher "ring" than a normal tooth. Any other abnormalities of the hard tissues should be noted, such as enamel defects and internal or external root resorption.

Examination of the intraoral soft tissues for a prospective orthodontic patient begins with checking the buccal and labial mucosa, the tongue and sublingual areas for possible abnormalities. Significant oral pathology in children is a rare finding. However, the orthodontist should take particular note of unusual frenum attachments. Two points should be noted: (1) Is there a heavy frenum attachment in the area of a maxillary midline diastema? The diastema may or may not be caused by the frenum in such cases, and surgical removal of the frenum by itself is not effective in closing the diastema. Orthodontists often recommend frenum removal after closing the diastema and laser surgery now is often recommended; the extent to which this is helpful is not well documented and remains controversial.[15] (2) Is there gingival clefting or recession in the lower incisor region near a high frenum attachment? Such an attachment often causes periodontal problems, and surgical repositioning of a frenum of this type is indicated, perhaps in conjunction with a free gingival graft to prevent further recession.

Gingivitis is relatively common in children; it is generally due to poor oral hygiene, although it can be exacerbated by faulty tooth alignment such as a high labially positioned maxillary canine. Severe periodontal problems, however, are uncommon in children even in the presence of severe malocclusion, and discovery of bone loss should lead to suspicion of underlying systemic illness such as diabetes, hormonal imbalances, or blood dyscrasias. Occasionally, aggressive juvenile periodontitis (rapid bone loss around central incisors and first molars for no apparent cause) is observed in children referred for orthodontic treatment. Although periodontal treatment methods have improved and a recurrence of this problem is unlikely to recur after treatment,[16] the prognosis for involved teeth in this situation is questionable, and orthodontic treatment may be indicated to prepare the patient for ultimate prosthetic replacements.

Two other periodontal problems often are observed in patients who are candidates for orthodontic treatment. These are clefts of the gingiva around severely protrusive or badly rotated mandibular incisors and gingival hyperplasia and fibrosis in children on seizure medication such as Dilantin. Patients who have gingival clefts and poor oral physiotherapy will frequently require periodontal surgery to provide a wider zone of attached gingiva, while those on Dilantin or equivalent drugs may require gingivectomy or gingivoplasty while under orthodontic treatment. Both types of periodontal surgeries can be performed while orthodontic appliances are in place, but early consultation with the family dentist or periodontist is necessary before proceeding with an orthodontic treatment plan. New surgical techniques with lasers can deal effectively with removing excess gingiva such as an operculum distal to a permanent second molar or performing a soft tissue uncovering of an unerupted tooth.[17]

In adults and children, the orthodontist should use a periodontal probe during the gingival evaluation. In a child, it is important to probe the sites of aggressive juvenile periodontitis: the maxillary molars and mandibular incisors. In an adult, bleeding on gently sweeping the probe along the gingival margin is an indication of a periodontal condition that can simply be marginal gingivitis on the one hand or more serious periodontal disease with loss of attachment and alveolar bone loss at the other extreme. It is not necessary to chart pocket depths. The goal is to see whether chronic inflammation that leads to easy bleeding is present. The basic principle is that orthodontic tooth movement in the absence of inflammation is similar to the physiologic response related to tooth migration or drift. If the same tooth movement is attempted in the presence of inflammation, the process becomes pathologic and more like periodontal breakdown and disease. Therefore, in adult patients, it is necessary for either the general dentist or a periodontist to perform initial periodontal preparation before orthodontics to eliminate any inflammation.

Soft tissue function. The size of the tongue is often hard to assess, but an attempt should be made to evaluate its general dimensions at rest and when protruded. It is important to ask the patient to raise the tongue to the roof of the mouth with the mouth open. Inability to do this suggests ankyloglossia, and the patient may benefit from surgery to allow better tongue movement.

Speech evaluation properly belongs in the hands of trained speech specialists, but sometimes parents seek orthodontic treatment as a way to help their child with speech problems, and an orthodontist should be able to discuss errors in speech that could be related to malocclusion versus those that are not (Table 8-1). Correcting the orthodontic condition is unlikely to remedy even related speech errors without associated speech therapy. Orthodontic treatment will, of course, have no effect on other common speech errors of children, such as substituting one sound for another.

Jaw function. An important part of the clinical examination is to establish the path of closure of the mandible and to determine if the maximum intercuspal position (centric occlusion) corresponds with the retruded contact position (centric relation). If these positions do not correspond, one should note any premature contacts and convenience shifts that

TABLE 8-1 Speech Difficulties Related to Malocclusion

Speech Sound	Problem	Related Malocclusion
/s/, /z/ (sibilants)	Lisp	Anterior open bite, large gap between incisors
/t/, /d/ (lingua-alveolar stops)	Difficulty in production	Irregular incisors, especially lingual position of maxillary incisors
/f/, /v/ (labiodental fricatives)	Distortion	Skeletal Class III
th, sh, ch (linguodental fricatives [voiced or voiceless])	Distortion	Anterior open bite

might exist. It is normal to have a 2-mm forward shift from the most retruded position; it is not normal to have a lateral shift. The patterns of wear on the cusps and incisal edges of teeth often indicate parafunctional movements of the jaws. Grinding or clenching of the teeth can affect orthodontic treatment, particularly in regard to the vertical dimension.

During the clinical examination, the TM joints should be palpated, and any crepitus or pain in the joints should be noted. Even when severe occlusal disharmonies are present, children presenting for orthodontic treatment rarely have TMD problems. The orthodontist is more likely to encounter these problems during or after treatment in older adolescents or in adults whose tolerance of muscular imbalances is reduced. The tolerance of children for occlusal disharmonies does not mean that these are unimportant in orthodontic diagnosis. It is particularly important that occlusal shifts and slides are detected and corrected during the orthodontic treatment. For a complete evaluation of TM joint function, the reader should refer to texts on this subject.[18]

Diagnostic Records

What Records Are Needed?

The final goal of the clinical evaluation is to determine what diagnostic records are needed for this particular patient. As we have noted above, it is neither necessary nor desirable to order the same set of records for every patient. As a rule, facial and intraoral photographs and a panoramic radiograph are needed for every patient (and are ordered before the orthodontist sees the patient so they can be available during the clinical examination). For some patients who will not receive comprehensive orthodontic treatment, everything else is optional except photographs of any soft tissue lesions that were noted in the clinical exam. For those who will be treated comprehensively, dental casts and a lateral cephalometric radiograph are added routinely; for those with impacted teeth, a small field-of-view (FOV) cone beam CT image is recommended; for those with asymmetry, a frontal ceph and often a large FOV cone beam CT is needed; for those with suspected injury or pathology within the TM joint, MRI is indicated.

Virtual versus physical dental casts. Taking impressions and pouring up dental casts is rapidly being superseded by obtaining digital images directly from intraoral scans, and storage of dental casts now has been almost completely eliminated by scanning casts and analyzing the resulting virtual images. It doesn't matter whether virtual or physical casts are obtained—it does matter that 3D rather than 2D images of the occlusal surfaces are obtained (i.e., just photographs are not good enough).

An initial complaint regarding virtual models is the lack of "haptic" feel as the clinician articulates the models, but they have gained popularity because of their convenience. In practices where 75 to 100 or more patients are seen each day, the task of "pulling models" becomes onerous for the orthodontic staff. In a multioffice practice where patients are occasionally seen at more than one location, the innovation of using all digital records and cloud-based storage has been a real boon. Scanning methodology to obtain virtual models is discussed below in the technologic advances section.

Head orientation for cephalograms. In modern cephalometrics, it is important that the cephalograms are obtained with the head oriented in NHP rather than automatically using the ear rods to establish the sagittal orientation and the Frankfort plane to establish vertical orientation. NHP is the orientation of the head that one presents to the world. It is obtained by having the relaxed patient look at the horizon or, more practically in an enclosed X-ray room, into his or her own eyes in a mirror.

The more severe the facial anomaly, the more likely it is that unthinking use of a cephalostat will produce an image in a distorted head position. For most patients, the ears are bilaterally symmetrical, and a line between them represents the true transverse axis, so using ear rods does not put the head in an unnatural orientation, but it is important to evaluate this. If the ears are not symmetric, putting the ear rod only in the right ear (to establish the distance between the X-ray source and patient) and then establishing head position (using the mirror) can prevent distortion.

Similarly, for most patients, Frankfort plane is a good estimator of the true horizontal plane, but for a significant number of patients, the true horizontal deviates from Frankfort, and it can be difficult to orient the head with Frankfort level. It is much better to let the patient orient the head while looking into the mirror and then fix that position with the anterior bar of the cephalostat. This also can be done using only one ear rod if necessary.

Additional records. Technologic advances have significantly added to the possible additional records that could be obtained. They are considered in detail in the following section.

Technologic Advances in Diagnosis

New methods that have affected current orthodontic practice and have even greater potential for changing the way orthodontists will practice in the future include digital photography, videography, 3D photography, computer imaging, virtual dental models, cone beam computed tomography, stereolithographic models, custom milling of attachments, and robotic wire bending. Nonetheless, technologic innovations should not be confused with fundamental changes in orthodontic thinking. It is similar to when recorded music became digital. The tone of the music improved, but the tune remained the same. This can be the litmus test for a clinician considering the adoption of any new technology. Will it change the tune or simply the tone? Only time tell which technology will survive. The new technologies in orthodontics are summarized next.

Digital photography, videography, and 3D photography. The conversion of photography from an analog to a digital process has revolutionized imaging in all fields, with orthodontics very much the beneficiary of this stunning technologic advance. The ability of digital video to capture the dynamics of anterior

tooth display during speech and smiling has not yet been fully embraced by orthodontists despite the potential value of viewing the rapid changes that occur during function. This appears to be a rational judgment because current studies do not indicate that digital video is significantly better than clinical photography in evaluating smile characteristics.[19]

3D facial photography had great promise for orthodontic applications when it was introduced in the early twenty-first century, but this technology also has yet to live up to its potential. Soft tissue asymmetries can be evaluated more precisely with 3D photography and imaging mirroring software, but the benefit to the patient is limited unless major surgical interventions are planned. Perhaps the best use of this technology lies with research. Initially it was touted as a noninvasive, radiation-free method for quantification of longitudinal soft tissue changes during growth; it is more often used now to evaluate posttreatment soft tissue outcomes, especially with surgical correction of cleft lip/palate and facial syndromes.[20] One must be cautious, however, when interpreting longitudinal data obtained from 3D photography studies because registration on stable soft tissue regions still presents a major challenge.

Another growing area of research utilizing this technology is 3D facial norms, initially developed by Coenraad Moorrees. Improvements in spherical harmonic algorithms and their availability through NIH-funded centers (Fasebase.org) will make 3D population norms and morphometrics a reality in the near future. If and how these data will be used for clinical orthodontics remains unknown. Medicine is currently facing the same dilemma with so called personalized/precision medicine versus guideline-based medicine.[21]

Computer imaging. The ability to morph images with special computer software and the creation of algorithms that can simulate the facial outcomes of tooth and jaw movement provide an excellent treatment planning and communication tool in orthodontics[22] (Case Study 8-2).

This can be particularly useful in helping patients understand alternative treatment possibilities. That is the key to true informed consent and is discussed in more detail in that section below.

Cone beam computed tomography. Cone beam computed tomography (CBCT) produces 3D volumetric images that can be reliably measured.[23] Among the advantages of CBCT imaging are true-scale images without magnification artifacts, the ability to correct errors in head positioning after image capture, and being able to see around extraneous structures that would otherwise obscure the desired view. Surface reconstruction of CBCT images has greatly enhanced treatment planning for impacted canines and asymmetric craniofacial growth (Fig. 8-9). The ability to rotate and visualize an impacted canine in any manner around the x-, y-, and z-axes gives the clinician the information needed to bring the tooth into occlusion while avoiding damage to adjacent teeth. The same approach can be applied to virtual surgery to correct jaw asymmetries. By merging digital models of the teeth with CBCT skeletal data, surgeons can better visualize precise surgical movements as well as the impact of these movements to the soft tissue.

This technology has opened new avenues of orthodontic research. New data are emerging from CBCT studies that give us a better understanding of how the TM joint, sutures of the midface, and airway are affected with orthodontic and orthopedic appliances. Yet, this technology is not without cost. The typical CBCT scan has 3 to 20 times the radiation dose of digital panoramic and cephalometric radiographs, and the stochastic effect of radiation on growing children is not fully understood. Practicing clinicians should adhere to the principles of ALARA (As Low As Reasonably Achievable), balancing between radiation risks and the benefits of having more information. While the risk-benefit ratio in diagnosis and treatment planning for impacted canines and jaw asymmetries is high enough to justify CBCT, it is questionable that CBCT scanning of patients without impacted teeth or major jaw discrepancies provides enough additional benefit to the patient to justify the increased radiation. Unfortunately, low-dose CBCT units to this point are not the answer to this problem, because often the decreased radiation comes at the expense of image quality.

Intraoral scanning. Plaster models of the teeth have been used to obtain 3D diagnostic records since the beginning of orthodontics. The advent of laser-scanned dental impressions and more recently intraoral scanning to produce a three-dimensional image of the teeth overcomes the problem of having to pour and trim plaster casts, while eliminating a major component of laboratory work and the need to store and retrieve the models for use when patients are seen. Now it is possible to view a virtual dentition on a computer screen by rotating the virtual models to allow the same type of 3D view as hand-held models. The accuracy of these virtual models for tooth setups in planning treatment is acceptable, but care must be taken to avoid inadvertent reduction in tooth width because of overlap in the digital images.[24]

Intraoral scanning is among the faster growing technologies in dentistry, and some foresee the end of impressions and casts. These scanners incorporate a variety of technologic modalities ranging from light-based to red or blue laser-based emitters combined with 3D stitching software to create a virtual model of the dentition and adjacent gingiva. However, the scan time and discomfort to patients is higher when compared to conventional impressions.[25] Scan times vary depending on the unit but typically range from 7 to 15 minutes, too long for patient comfort, although emerging technology will further decrease this. Much like the arms race between VHS and Betamax in the early 80s or Blu-ray versus HD-DVD in recent years, the better technology might not emerge as the winner, and only time will decide which technology gains mass acceptance in the orthodontic community.

Software for virtual dental models has emerged rapidly in recent years (Fig. 8-10). Semi-automated software can measure for arch length and Bolton discrepancies, and multiple virtual treatment setups can be performed with minimal effort. One of the biggest advantages of virtual models is fabrication efficiency when combined with other 3D commercial CAD-CAM products. Initially, records for Invisalign or Insignia CAD-CAM brackets were PVS impressions that were sent to the company's lab, digitized, and a virtual setup created for the clinician's approval. Intraoral scanning and cloud-based uploading has decreased the time for this process from weeks to days. Furthermore, 3D tooth movement software allows the clinician to change the virtual treatment setup in any manner without the need for communication with the laboratory technician.

In a somewhat humorous vein, it is interesting how deeply ingrained orthodontic traditions and rituals are, such as insisting on having simulated plaster bases on virtual orthodontic models. Edges and angles on conventional plaster models served two purposes: the back sides were trimmed to establish

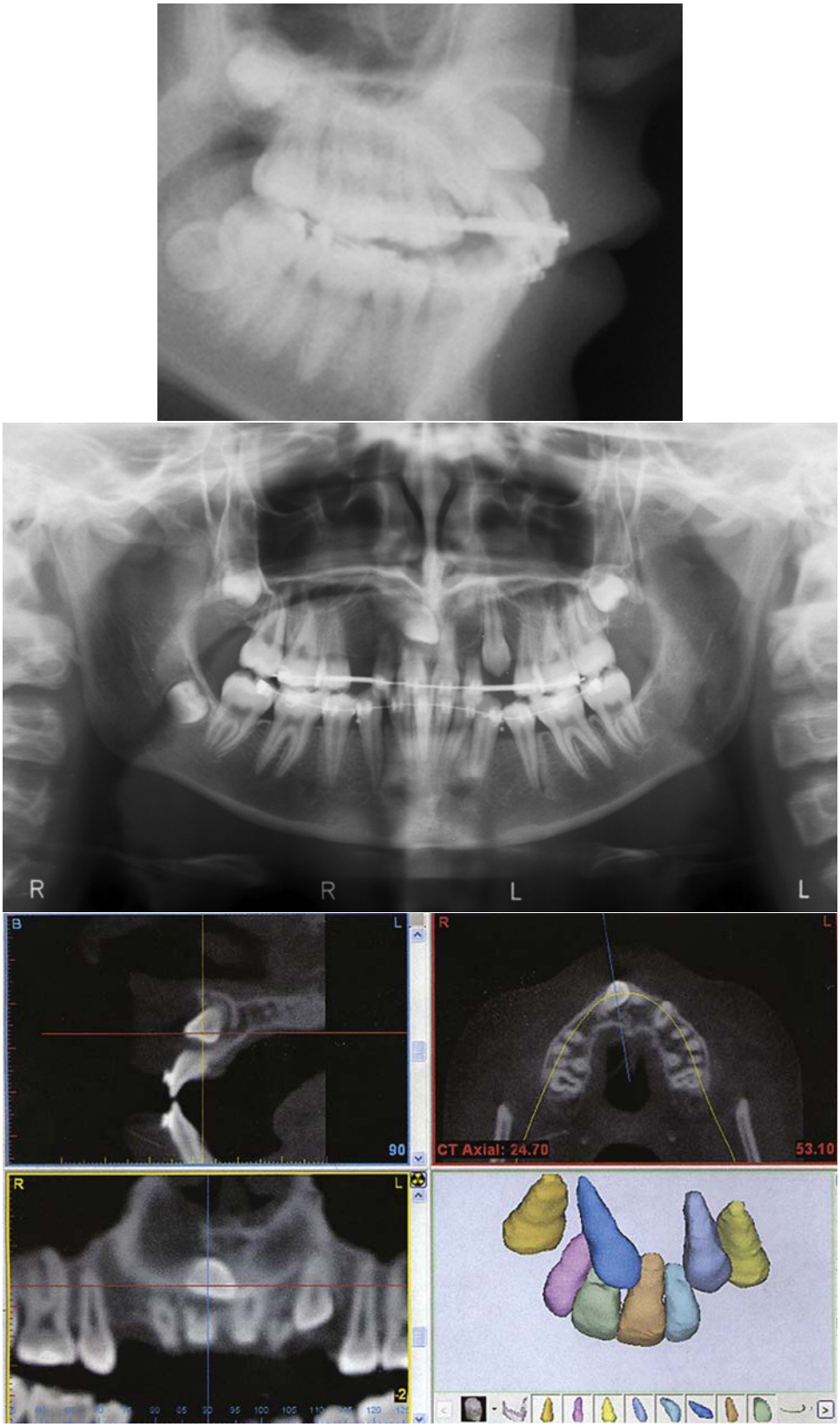


FIGURE 8-9 3D radiographic imaging with CBCT can be a valuable adjunct to panoramic radiography and periapical localizing films in assessing impacted canines. For this patient, although the position of the impacted canine and the significant resorption of the root of the central incisor can be seen clearly from the panoramic image and from the lateral cephalogram, the 3D images add important information as to the path the canine would have to be moved to avoid further damage to the root of the central incisor if it is to be saved, and whether it would be prudent to try to save the incisor or extract it. For difficult situations like this one, 3D imaging now is indicated.

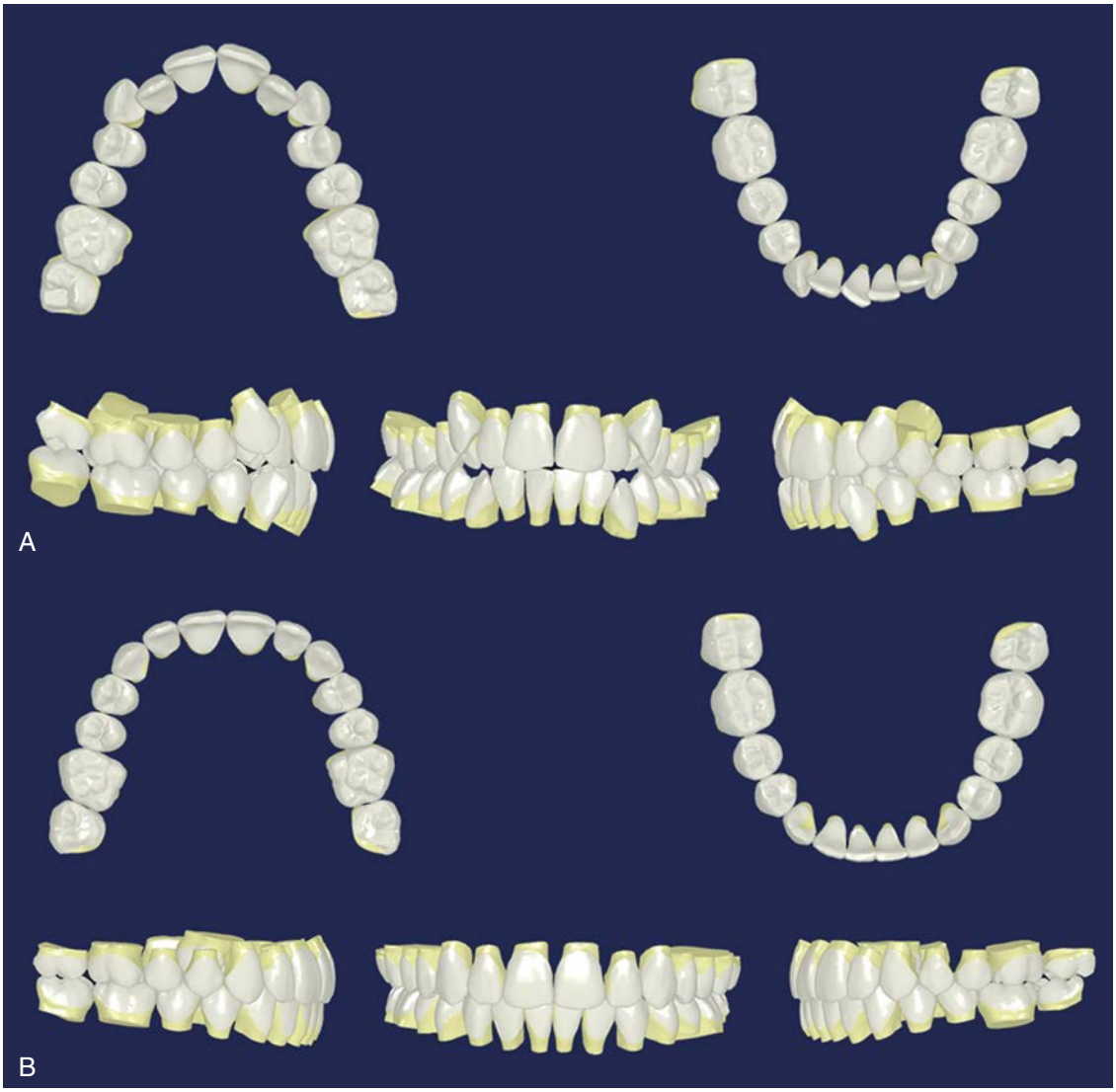


FIGURE 8-10 For complex orthodontic problems or borderline extraction cases, it is sometimes useful in diagnosis and treatment planning to look at 3D (virtual) setups. **A,** A virtual scan of the initial malocclusion and nonextraction digital setup (**B**) can be performed digitally by the clinician with 3D Imaging software or through commercial vendors.

the occlusal relationships, and the symmetric base made it more obvious if the dental arches were asymmetric. Today, the dental occlusion is documented within the software, but the bases are primarily decorative yet they remain.

By far the greatest limitation of plaster casts and virtual models is that although they are excellent facsimiles of the crowns of teeth, they give no clue about three important traits:

1. The inclinations of tooth roots in relationship to their alveolar housing. After all, the critical elements in the biology of tooth movement are the tooth roots in relationship to their surrounding bone.
2. The relationship of tooth crowns to the soft tissues of the tongue and lips. In the long run, it is this relationship that determines functional stability of the dentition.
3. The relative inclinations of tooth crowns in relation to the overall skeletal and soft tissue facial framework. In the end, dentofacial appearance is a critical factor in assessing orthodontic outcome.

Emerging technology from SureSmile and other companies is aimed at overcoming these limitations by merging CBCTs with intraoral scans. A successful system of this type will be a step toward defining the biological bony limits of tooth movement and give us quantifiable outcomes such as tip and torque. Like any new technology, this needs to be validated with scientific rigor—and the question of risk versus benefit remains when adding CBCT to the standard orthodontic records.

Analysis of Diagnostic Records

Analysis of the diagnostic records is the final step in assembling the diagnostic database. This is best discussed in the context of analyzing dental alignment and occlusion from the dental casts; evaluating jaw relationships and tooth–jaw relationships (i.e., how the maxillary teeth relate to the maxilla and the mandibular teeth relate to the mandible); and analyzing 3D images when indicated to obtain a more detailed picture of skeletal and dental relationships.

Cast Analysis

Analysis of the dental casts was the key diagnostic procedure in Edward Angle's era and was used to generate the appropriate Angle classification. From the beginning, that had to be supplemented with information as to the location and severity of dental crowding. In the mid-twentieth century, after cephalometric radiography turned from a research tool for investigation of facial growth into a clinical tool, skeletal and dental components of malocclusion were differentiated, and cast analysis was relegated to a secondary position.

It still is valuable, however, to evaluate the location and severity of dental crowding and to evaluate tooth-size relationships within the dental arches. Both of these now can be done more easily with virtual casts (digital images) than with physical ones. Quantification of crowding is a component, but certainly not the deciding one, of the decision to expand the dental arches or obtain additional space by extraction. Most patients do not have a tooth-size discrepancy, but it is critically important to know whether they do. In a modern orthodontic practice, the amount of crowding in each arch and the amount/location of tooth-size discrepancy should be measured and added to the database as discrete measurements.

Cephalometric Analysis

Development of clinical cephalometrics. It is not possible to establish the true nature of a malocclusion without information about the underlying skeletal relationships, and this cannot be gained from dental casts or photographs. Prior to cephalometrics, these relationships were evaluated (usually quite well) from the patient's soft tissue profile and general appearance. Cephalometric analysis in modern usage provides more detail about dental and skeletal relationships, but it still supplements rather than supplants a careful clinical evaluation of the patient. At best, the radiographic image (often referred to as a cephalogram) is merely a static 2D representation of the hard tissues involved in a complex 3D system.

The problem in using cephalograms to obtain clinically useful information is that the image contains far too much information. The initial solution was to use tracings of clearly defined structures as researchers had done and then use angles and distances between landmarks on the tracing to evaluate relationships between structures. A small set of these measurements then was chosen to compare a specific patient to "normal" individuals (those with ideal occlusion), so that the patient could be described relative to the Angle classifications and subclassifications.

With the proliferation of named analyses (just different sets of measurements) as time passed, analyses began to become ends in themselves instead of guides in making decisions about relationships. Since the typical cephalometric analysis chooses one or two specific measurements from the multitude of measurements that might be used to evaluate a single criterion, there is not and will not be any single analysis that is ideal for every patient. Instead, certain measurements will be useful in providing information about certain patients but not so useful for others.

An alternative way to compare an individual patient with normal controls is to compare the patient's tracing to a composite image based on a group of normal patients, i.e., with landmark positions and relationships created from an average of the group. Despite the difficulty of doing this manually, templates based on a normal reference group (from the Bolton growth study and later from other growth studies) were created. Doing the comparison of the reference and patient tracings is more intuitive than using measurements, because it does not require a digital-to-analog conversion of data—and the human brain is an analog computer, not a digital instrument. Template superimpositions, however, were never used for cephalometric analysis by most orthodontists.

With the replacement of X-ray films with digital X-ray images, manual tracing of films was replaced by landmark identification on a computer screen. Although good data do not exist, it is generally conceded that landmark identification on the digital images is not better than it was previously and probably not as good. It is much easier and faster, however, to have the computer make angular and linear measurements. The advent of digitized landmarks and multiple measurements has made it easy to assess a cephalogram using many different analyses—and to become confused by contradictory interpretations of the measurements among the varied analyses. For detailed information about cephalometric landmarks and the measurements incorporated into various analyses, the reader is referred to Chapter 6 in *Contemporary Orthodontics*[1] and textbooks that focus on cephalometrics.[26,27] One could, of course, superimpose a reference template on the image on the computer screen and just look at it, but that rarely is done.

Goals of modern cephalometric analysis. In this chapter, we wish to emphasize that it is the relationships between skeletal and dental units and not any particular set of measurements that should be evaluated for a given patient. This concept is most easily grasped in terms of block diagrams of the relationships in question (Fig. 8-11). There are five key units in understanding anteroposterior and vertical relationships: the cranial base, maxilla, maxillary dentition, mandibular dentition, and mandible. Although orthodontists are primarily concerned with the position of the teeth and the jaws, it must be kept in mind that there is no certainty that the cranial base will have escaped malformation in individuals who have deviations from ideal proportion elsewhere. It is safe to say that the greater the malocclusion severity, the greater is the chance that there will be deviations in the cranial base as well as in the jaws and the teeth.

Can a series of small deviations in relationships as evaluated cephalometrically add up to a big problem? Can a large deviation in one relationship be compensated by a series of small deviations in other relationships? The answer to both questions is yes. This concept of potential dental and skeletal counterpart compensations that take place during growth of the dentofacial complex is best illustrated by Enlow's classic diagram (Fig. 8-12).

Perhaps the modern approach to cephalometric analysis, developing the ability to evaluate the pattern of relationships that exist for individual patients, is best put into perspective in the context of what now is called "learning to see data." The characteristic of experts in many fields is their ability to quickly view a series of gauges, dials or images and pick out patterns in what initially is a confusing, almost chaotic overload of information. For an experienced orthodontist, a look at a cephalometric radiograph is enough to understand the pattern of dental and skeletal relationships without relying on a series of measurements. Psychologists call this perceptual learning. Recent work has demonstrated that having students (in this case, airplane pilots) repeatedly scan a set of instruments and receive immediate feedback as to what the pattern indicates greatly reduces the time to reach the expert level. Future instruction in

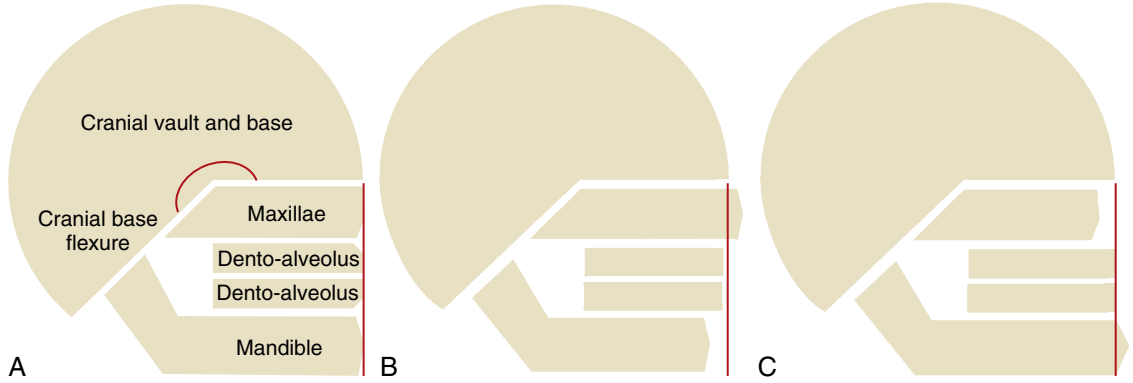


FIGURE 8-11 A, Schematic representation of normal jaw proportions and ideal dental relationships. B, Schematic depiction of a jaw disproportion characterized by maxillary excess and mandibular retrusion, yet ideal dental relationships. C, Schematic showing a jaw disproportion characterized by maxillary deficiency and mandibular excess. Again, in this illustration normal occlusion is due to dentoalveolar compensation.

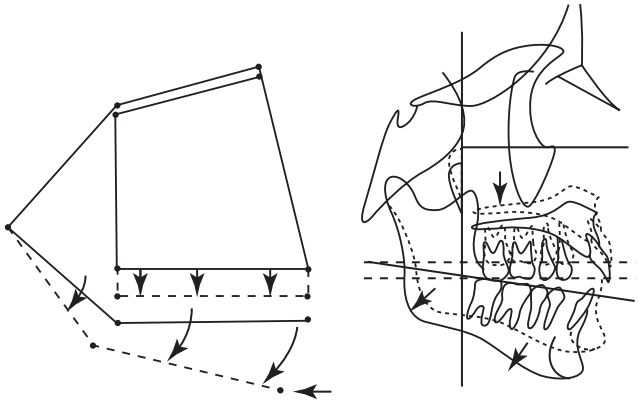


FIGURE 8-12 Enlow proposed that without dental and skeletal counterpart compensations for underlying jaw disproportions, malocclusion results. This example shows a hypothetical situation simulating exuberant vertical growth of the maxillae and dentoalveolus, which in turn caused the mandible to rotate backward causing a concomitant anteroposterior jaw discrepancy with no dental compensation, producing a Class II skeletal and dental open bite malocclusion. (From Enlow DH. *Essentials of Facial Growth*. 4th ed. Philadelphia: WB Saunders; 1996.)

cephalometric analysis undoubtedly will be based more strongly on this type of feedback. After all, perceiving patterns, not making measurements, is the diagnostic goal. Following is a brief description of the type of information regarding the anteroposterior and vertical dental and skeletal relationships that can be gleaned from a lateral cephalogram (mostly by just looking at it with a trained eye) and added to the database for a patient.

Evaluation of Class II malocclusion. In any patient, a Class II dental relationship may be due to any combination of four major factors: (1) maxillary skeletal excess, (2) maxillary dental excess, (3) mandibular skeletal deficiency, and (4) mandibular dental deficiency.

Maxillary skeletal excess can also be called midface protrusion, and the increased facial convexity that accompanies it is one of the easy ways to recognize it. Nasal prominence, heavy orbital and malar ridges, and increased convexity of the facial profile occur together in true midface protrusion. Unless there is a compensating malposition of the maxillary dentition, skeletal maxillary excess or midface protrusion will have a naturally accompanying maxillary dental protrusion as well. Once the relationship of the maxilla to the cranial base has been examined, it is necessary to examine additionally only the relationship of the maxillary dentition to the overlying maxilla.

Since Angle's concepts continue to color our view of the primary anatomic cause of Class II malocclusion, there has been a concentration of attention on mandibular deficiency. The dental occlusion would be the same whether the mandible were small in absolute terms, of reasonably normal size but positioned distally, or rotated so that its effective length was reduced. The latter situation represents an interaction between sagittal and vertical components. McNamara's method of cephalometric evaluation can be credited with making orthodontists aware of the fact that more Class II malocclusions result from mandibular deficiency than maxillary excess.[28] A patient can have Class I occlusion as the result of overclosure of the mandible with short lower face height and a dental deep bite. These individuals are said to have "masked" Class II skeletal patterns. Using Enlow's terminology, these people have had compensatory counterpart alterations in the sagittal and vertical relationships of the teeth and jaws, masking the true nature of the problem.

Mandibular dental deficiency is obvious from the dental casts where there are either small or missing teeth or both. It is possible for the dentition to be relatively deficient, however, in terms of being positioned distally on the mandible. In Class II malocclusion, this can occur simultaneously with protrusion of maxillary incisors as one often finds in inveterate thumb suckers.

Evaluation of Class III malocclusion. Measurements to provide recognition of maxillary skeletal deficiency or midface retrusion rarely are included in traditional cephalometric analyses, and if this is suspected, visual observations of the relationship of the maxilla to a true vertical line dropped from nasion can be helpful. A mandible that is rotated and overclosed can simulate mandibular skeletal excess. The interaction between the sagittal and vertical deviations makes it important to consider the effect of the vertical on sagittal relationships in Class III and Class II patients.

Vertical skeletal problems. The Angle classification focused cephalometric attention on the anteroposterior plane of space

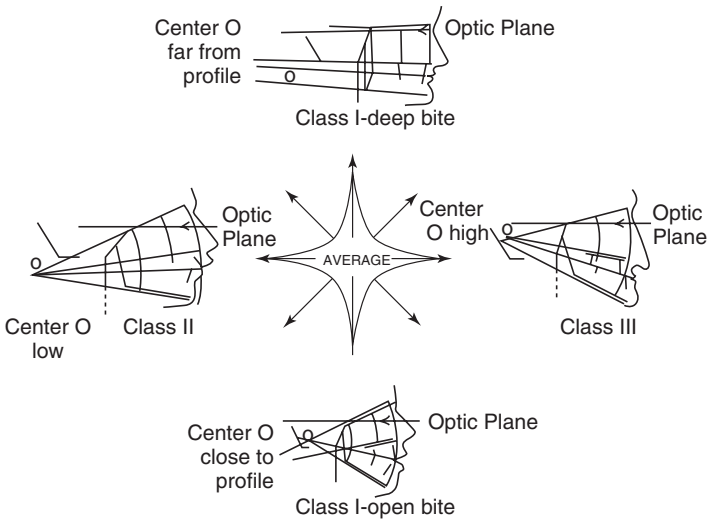


FIGURE 8-13 Sassouni's major contribution to cephalometric analysis was his observation that the relative parallelness or convergence of the horizontal planes of the face—i.e., the anterior cranial base, palatal plane, occlusal plane, and mandibular plane—are related to anterior and posterior face height and frequently reflects a tendency toward deep bite or open bite as seen in Sassouni's original diagram. The more parallel the planes, the greater the deep bite tendency. His "Center O" (outlined in yellow) is the approximation of where the planes converge posteriorly. The theory is that the further Center O is from the profile, the less convergent are the planes. This is seen most dramatically in the Class I deep bite and open bite representations. Note that the profiles in this schematic drawing coincide with those in Figure 8-18. (Modified from Sassouni V. The Class II syndrome: differential diagnosis and treatment. *Angle Orthod* 1970;40:334–341.)

and directed attention away from the vertical plane of space. Much work has been spent on correcting this disproportionate emphasis, but cephalometric standards for vertical relationships remain less well developed than anteroposterior standards. Four major vertical problems exist: (1) anterior open bite, (2) anterior deep bite, (3) posterior open bite, and (4) posterior collapsed bite with overclosure. As with the sagittal and transverse planes of space, skeletal and dental effects must be distinguished to make an accurate evaluation of the situation. Since bite depth is determined by the contact relationships of the teeth, the terms *skeletal open bite* or *skeletal deep bite,* in a sense, are inherent contradictions.

This leads to the first method for detection of vertical dysplasia—namely, measuring anterior face height. Both anterior and posterior skeletal vertical dimensions need to be examined, however, and analysis of the posterior vertical is not done easily. No outstanding method of establishing posterior vertical standards has yet emerged.

Another method for evaluating vertical proportions relies on the convergence or divergence of the mandibular, occlusal, and palatal planes, as suggested by Sassouni (Fig. 8-13).[29] If these three planes converge acutely and meet at a point close behind the face, posterior vertical dimensions are relatively smaller than anterior vertical ones. This produces a skeletal tendency toward anterior open bite, which is now routinely called *skeletal open bite.* It also implies a short ramus and an obtuse gonial angle, although these features do not necessarily have to be present. The open bite tendency is accentuated if the palatal plane is tipped up anteriorly and down posteriorly, a condition that is seen often enough to demonstrate that the skeletal problems leading to open bite are not exclusively in mandibular positioning.

Palatal, occlusal, and mandibular planes that run almost parallel to each other, on the other hand, lead to a skeletal predilection toward anterior deep bite. Individuals with this condition tend to have a longer ramus and a nearly perpendicular gonial angle. The Wits cephalometric analysis[30] recognizes that the relationship of the anteroposterior cant (pitch) of the occlusal plane in relationship to the maxillae and mandible offers a strong clue about the sagittal relationship of the jaws.

The interaction between sagittal and vertical factors is perhaps nowhere seen better than in the person who has a short ramus, steep and convergent mandibular plane angle, and a Class II malocclusion including elements of true as well as relative mandibular deficiency. The label is Class II malocclusion in such a patient, but the problem is frequently more a vertical than a horizontal one. Especially as such patients reach their adult years, even surgical correction can be very difficult and relapse tendencies can be great. The interaction between sagittal and vertical problems extends into an interaction between these factors and the structure of the cranial base.

Vertical dental problems. Dental vertical problems refer to too much or too little eruption of teeth in relation to their own supporting bone. A common example is excessive eruption of mandibular incisors, which is a usual concomitant of most Class II malocclusions. The lower incisors continue to erupt past the anteriorly positioned upper incisors and frequently contact the palate. If there are no skeletal vertical disproportions, this will be observed as a lengthening of the distance from the apex of the mandibular incisor roots to the lower border of the mandible.

Similarly, an open bite may be caused by insufficient incisors in either arch. This can be seen cephalometrically by a decrease in the distance from the incisor to the mandibular plane or palatal plane. Overall proportions must be taken into account in

judging this, since excessive eruption of posterior teeth is also a factor in open bites. The relationship of the upper molar roots to the height of the palatal vault, which is easily observed cephalometrically, can be a great help in evaluation. The root apices of the upper molar in an adult should be at 2 to 3 mm below the height of the palatal vault. Distances in excess of this indicate excessive eruption; roots that are above the height of the palatal vault indicate a deficiency in eruption.

If anterior teeth meet and posterior teeth do not, there is by definition a posterior open bite, which is almost always related to failure of dentoalveolar development in one or both arches. In this situation, the orthodontist must rule out the possibility of primary failure of eruption (PFE).[31,32] PFE is characterized by a nonsyndromic eruption failure of secondary teeth in the absence of mechanical obstruction. The hallmark features of this condition are (1) infraocclusion of affected teeth, (2) increasing significant posterior open bite malocclusion accompanying normal vertical facial growth, and (3) inability to move affected teeth orthodontically. At the present time it has no orthodontic or surgical remedy—the unerupted teeth do not respond to orthodontic force, and repositioning them surgically rarely is practical.

If eruption of both anterior and posterior teeth is deficient, the result will be an overclosure of the mandible. This can be established by evaluating anterior facial proportions and confirmed from an excessive freeway space.

Classification: Organizing the Database

Angle Classification

Even though several treatises on orthodontics had already been written by the beginning of the twentieth century, these authors had no acceptable method for describing irregularities and abnormal relationships of the teeth and jaws. The Angle classification, described at the beginning of this chapter, was readily accepted by the dental profession, because it was simple and brought order out of what previously had been confusion regarding dental relationships.

It was recognized almost immediately, however, that there were deficiencies in the Angle system. In 1912, a report to the British Society for the Study of Orthodontics suggested that malocclusions be classified with regard to deviations in the transverse dimension, the sagittal dimension, and the vertical dimension.[33] Critics also pointed out that Angle's method disregarded, both in classification and in treatment planning, the relationship of the teeth to the face.[34] In the 1920s, Simon, in a technique he called *gnathostatics,* used a facebow transfer and mounting to relate the dental models to the rest of the face and cranium in all three planes of space.[35] Simon's facial reference line (the Frankfort plane) was more rational than Angle's skeletal reference, the key ridge (the lowest extent of the zygomaticomaxillary suture).

If it had not been for the introduction of cephalometrics in the 1930 and 1940s, gnathostatics probably would have made a more lasting impact on orthodontics. With the advent of the lateral cephalogram, many of the relationships that could be determined from gnathostatic casts could more easily be observed on a cephalometric radiograph. Arguably, radiographic cephalometrics, although an important milestone in the evolution of orthodontics, in many ways hindered viewing the teeth and jaws as a 3D issue.

As orthodontic treatment became more widespread and treatment possibilities other than arch expansion were considered, several other problems with the Angle classification emerged, all of which revolved around its narrow focus on the dentition and absence of a diagnosis that points logically to a treatment plan. This difficulty becomes apparent when it is recognized that malocclusions having the same Angle classification may, indeed, be only analogous (having only the same occlusal relationships) rather than homologous (having all characteristics in common). Despite the informal additions to Angle's system that most orthodontists use, there is a tendency to treat malocclusions of the same classification in a similar manner. Homologous malocclusions require similar treatment plans, whereas analogous malocclusions may require different treatment approaches. Some poor responses to treatment are undoubtedly related to this fault in diagnosis.

Figure 8-14 illustrates two nearly identical Angle Class II, Division 1 malocclusions in children of the same age. There are differences in skeletal proportions and in the relationships of the teeth to their respective jaws, both of which affect the profile, and the two cases should not be treated exactly the same. These are analogous malocclusions. For one, an effort has to be made to retract the maxillary teeth without further proclining the mandibular incisors. For the other, proclining the mandibular incisors might be justifiable, and interarch Class II mechanics could probably retract the maxillary teeth while proclining the mandibular teeth.

Since Angle and his followers did not recognize any need for the extraction of teeth, the Angle system does not take into account the possibility of arch-perimeter problems. The reintroduction of extraction into orthodontic therapy has made it necessary for orthodontists to add arch-perimeter analysis as an additional step in classification.

A final, but not inconsequential, difficulty with Angle's classification procedure is that it does not indicate the complexity and severity of the problem. In modern orthodontics, it is necessary to organize the database using a more complete system.

Systematic Description: Ackerman-Proffit Classification

Development of the classification scheme. To overcome the difficulties just discussed, we recommend using a classification scheme in which five or fewer characteristics of orthodontic problems and their interrelationships are assessed.[36,37] The scheme has three major components: (1) dentofacial appearance, (2) dental alignment/symmetry, and (3) spatial relationships of the teeth and jaws.

To fully describe the spatial relationships of the jaws and teeth and their relationship to the facial soft tissues is exactly analogous to what is necessary to describe the position of an airplane in space, which must be combined with rotation about three perpendicular axes (yaw, pitch, and roll) (Fig. 8-15). In engineering terminology, the object has 6 degrees of freedom. In orthodontics, introduction of the rotational axes into the description of orthodontic problems significantly improves the precision of the description and therefore facilitates development of the problem list (Fig. 8-16).

Our representation of the interaction of the five major characteristics of malocclusion, in which both the translational and rotational components are combined in a single Venn diagram, is shown in Figure 8-17. A Venn diagram offers a visual demonstration of interaction or overlap among parts of a complex structure. A collection or group in this system is defined as a set, and all elements contained in a set have some common property. Common to all dentitions is their effect on anterior tooth

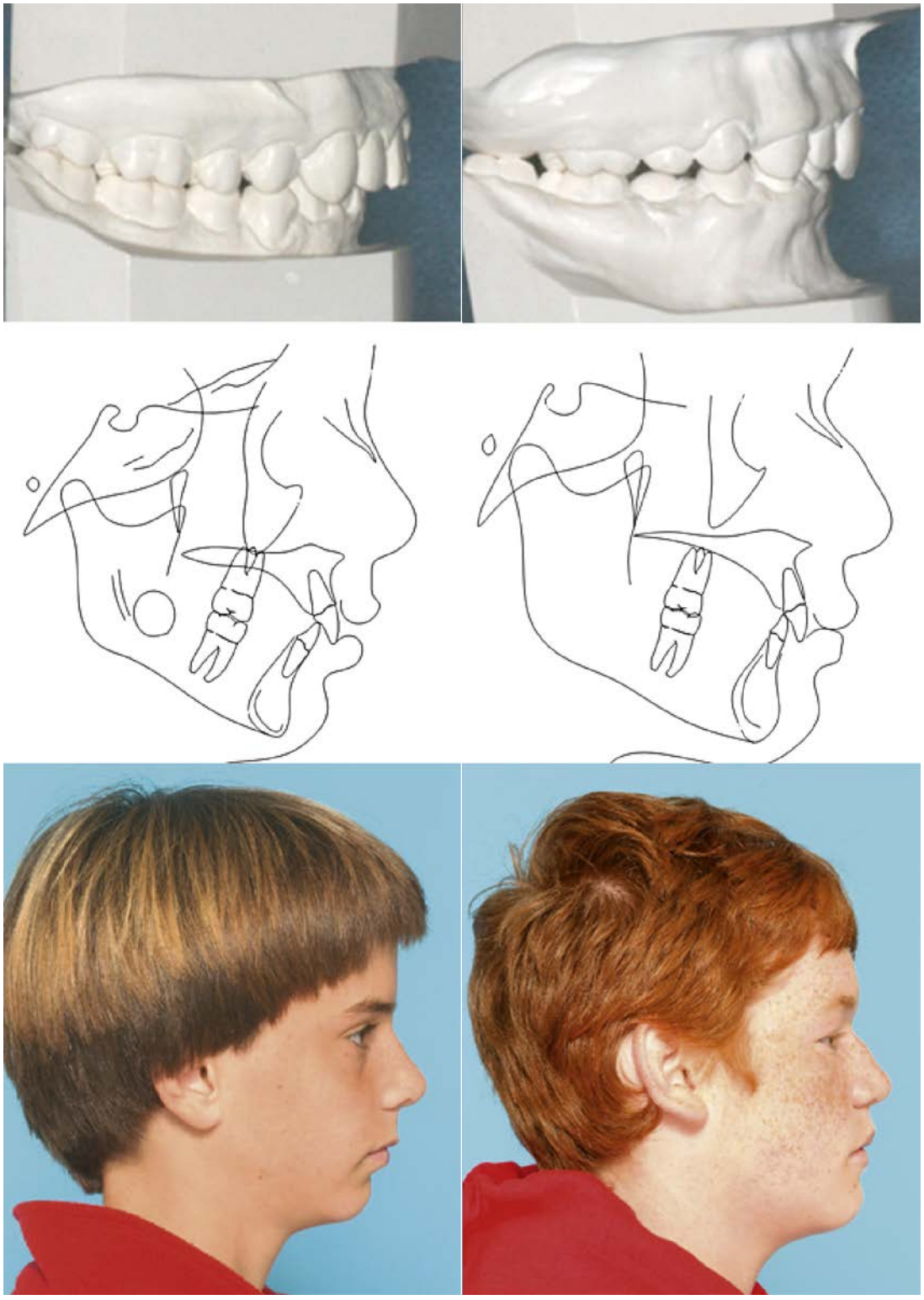


FIGURE 8-14 Shown here are two patients of nearly the same age who have similar orthodontic conditions when only the characteristics of their dental occlusion are taken into consideration. Their underlying skeletal patterns and resultant dental compensations (e.g., mandibular incisor inclinations) are quite different. These two analogous patients require quite different treatment plans.

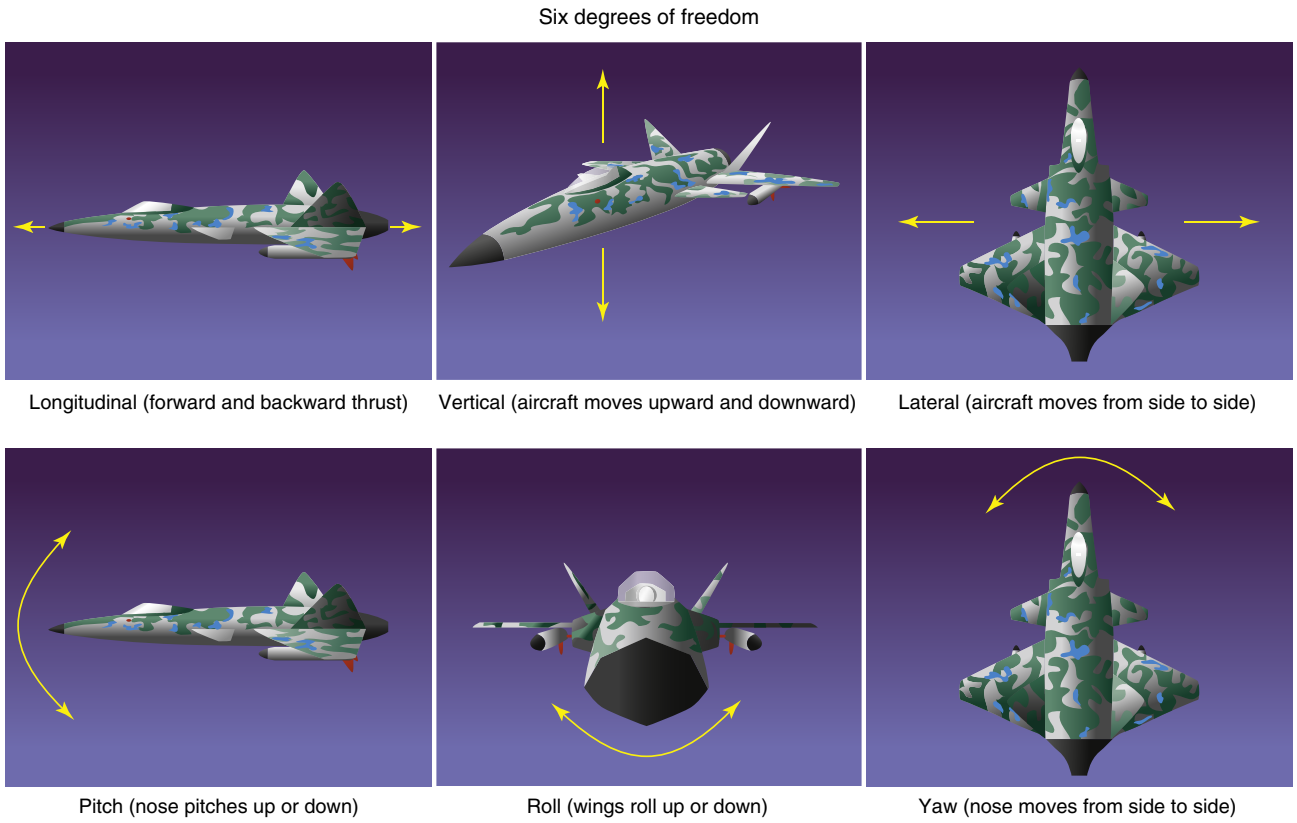


FIGURE 8-15 An airplane's ability to maneuver in space can be described as 6 degrees of freedom. An airplane's attitude, or orientation relative to the direction of travel, is analogous to the orientation of the dentition within the dentofacial complex. Pitch, roll, and yaw are descriptors that can be used for the esthetic line of the dentition. Pitch represents the occlusal plane in sagittal view, roll is analogous to the occlusal plane in frontal view, and yaw is a way of describing rotation of the dentition and jaws around a vertical axis. Yaw problems are manifested as midline deviations or facial asymmetries.

display and the soft tissue drape. We represent this as the framework or "universe" within which all other deviations from the theoretical ideal reside.

Also common to all dentitions is the degree of alignment, arch form, and symmetry of the teeth within the dental arches. For this reason, alignment, arch form, and symmetry are represented within the overall framework represented by the face and smile. Any deviation from the line of occlusion is described and included in this collection of possible discrepancies.

If the teeth are perfectly aligned in both arches, by definition ideal occlusion will occur when the mesiolingual cusps of the maxillary first molars rest in the central fossae of the mandibular first molars, provided the curves of Spee are harmonious and there is no tooth-size discrepancy. This, of course, is the original Angle concept.

Relationships of the teeth and jaws must be considered in all three planes of space, and deviations large enough to be orthodontic problems may occur in any or all of these planes. These are represented by the interacting sets within the alignment/symmetry set.

Application of systematic description. Although it helps to understand the logic of the system, it is understood best by viewing how it is used in organizing the database into the five characteristics of orthodontic problems. The evaluation is carried out in five steps corresponding to each of the five characteristics, or descriptors.

In this classification, a patient with ideal occlusion accompanied by excellent facial balance and a balanced smile requires no descriptors at all to characterize the situation, since no orthodontic problem exists. A patient with crowding/malalignment of the incisor teeth, but excellent balance of the face and smile and no other problems, requires only one descriptor: the location and severity of the resulting malalignment. If the patient has problems related to facial appearance and/or occlusal discrepancies, these descriptors would be added. The steps in organizing the database would be, in sequence:

Step 1: Evaluation of dentofacial appearance. This includes assessing anterior tooth display, as well as the relative convexity and concavity and divergence of the face in profile view and vertical proportions of the face. As discussed previously, faces can be categorized in profile view by their relative convexity and divergence (Fig. 8-18). In anterior view, the vertical characteristics of the face can be expressed by the proportion of facial width and height. In doing so, patients present along a spectrum from short and wide (brachyfacial) to long and narrow (dolichofacial). Average facial proportions are more or less of ovoid shape, and these faces are called *mesofacial.* Dolichofacial individuals often have anterior open bite dental and skeletal relationships, and

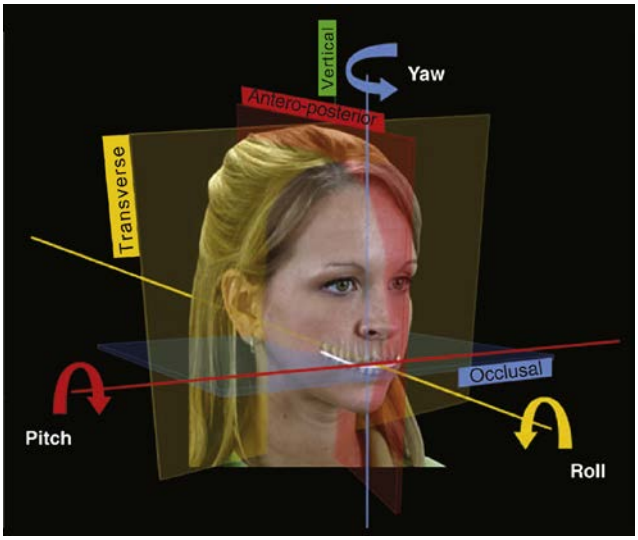


FIGURE 8-16 The face and dentition depicted with 6 degrees of freedom. The classic anatomic planes of the face are coronal, sagittal, and transverse. In the Ackerman-Proffit classification, the sagittal plane is referred to as anteroposterior, the coronal plane is designated vertical, and the transverse plane is the same as the occlusal plane and is used as a reference for the relative widths of the dental arches and any crossbite relationships that might exist. When the three discs representing the three planes of space are rotated they demonstrate pitch, roll, and yaw of the occlusal plane. When the vertical disc rotates it creates roll of the occlusal plane, when the sagittal disc rotates it creates pitch of the occlusal plane, and when the transverse plane rotates it simulates yaw. From Proffit, William R., Henry W. Fields, and David M. Sarver. *Contemporary Orthodontics*. St. Louis, MO: Elsevier/Mosby, 2013. Figure 6-66, p. 209.

brachfacial individuals often have dental and/skeletal anterior deep bite relationships. In most instances, the clinician simply classifies faces from a vertical standpoint as short, average, or long (Fig. 8-19). In terms of anterior tooth display, a smile is characterized by how well the teeth and gingival fit within the smile zone, which is defined by the lips. The lateral and sagittal cants of the occlusal plane and esthetic line of occlusion, as well as the rotation of the maxillae and mandible around a true vertical axis, are described using the terms *pitch*, *roll*, and *yaw*. These features can be assessed when evaluating anterior tooth display. A more detailed analysis of facial form and appearance is presented in Chapter 9 of this volume.

Step 2: Analysis of the dental alignment and intra-arch symmetry. Alignment is the key word in this group; among the possibilities are ideal, crowded (arch length deficiency), spaced, and mutilated. It is obviously important to count the teeth in order to ascertain which teeth are present or absent.

If the maxillary and mandibular dental midlines do not correspond, the fault should be determined by looking at the midline of the face to decide whether the maxillary or mandibular midline deviates or both are at fault, and whether the deviation is the result of an intra-arch alignment issue or whether it is a yaw problem in which either the maxilla or mandible has rotated slightly around an imaginary vertical axis.

Step 3: Lateral dimensions (transverse plane of space). The faciolingual relationships of the posterior teeth are noted and whether posterior crossbite is present. A judgment is also made as to whether the deviation from ideal proportions and occlusion is basically dentoalveolar, skeletal, or a combination of the two. Most patients have components of both, with one or the other predominating. If a bilateral palatal crossbite is the result of a narrow palatal vault, it would be called a skeletal problem; constriction of the maxillary dental arch alone with normal palatal width would be designated a dentoalveolar problem. Dental compensation for a narrow maxilla, with the teeth tipped facially, often is observed.

As a general rule, *maxillary* or *mandibular* is used to indicate where the problem is located. "Maxillary palatal crossbite" implies a narrow maxillary arch, while "mandibular buccal crossbite," describing the same dental relationship, indicates excess mandibular width as the cause.

The lateral cant of the occlusal plane (roll) is evaluated in relationship to both the intercommissure line and the interpupillary line (Fig. 8-20).

Step 4: Anteroposterior dimensions (sagittal plane of space). In this dimension, the Angle classification is useful, but the goal is to evaluate overjet/reverse overjet in terms of whether it is due to deviations in skeletal, dentoalveolar, or both relationships. In Class II or Class III patients, it is important to distinguish which jaw is affected and to also distinguish skeletal from dentoalveolar problems.

It is important to understand the terminology. For example, a Class II patient has a Class II malocclusion; a skeletal Class II could be due largely to mandibular deficiency (and that should be specified) but could have a dentoalveolar component (also to be specified); and a dental Class II would be due largely to displacement of the mandibular teeth forward on the mandible and/or displacement of the maxillary teeth distally on the maxilla. Class III would be described similarly.

It also is important to evaluate the sagittal cant of the occlusal plane (pitch). Doing this from a ceph is problematic unless it was taken in NHP.

Step 5: Vertical dimensions (vertical plane of space). Bite depth is used to describe the vertical relationships. Again, one must determine whether the problem is skeletal, dentoalveolar, or a combination, and the interaction of vertical and a-p relationships must be kept in mind.

A steep mandibular plane, 35 degrees or greater to the Frankfort plane, indicates an open bite tendency, which would be affected or even prevented by dental compensation in the form of excessive eruption of the incisors. A depression in the lower border of the mandible (antegonial notching) just anterior to the gonial angle indicates deficient vertical growth at the condyle and a degree of compensation by addition of bone in the muscle attachment area (Fig. 8-21). A palatal plane that tips down posteriorly also is an indicator of an open bite tendency, because this often leads to downward–backward rotation of the mandible.

Similarly, a relatively flat mandibular plane represents a deep bite tendency, which would be affected by the extent to which deficient incisor eruption provides compensation or excessive eruption makes the deep bite worse.

Summary of Diagnosis

By working from a comprehensive database, orthodontic diagnosis becomes the process of systematically synthesizing the manifold factors involved in a complex situation into a discrete list of problems, described in a way that indicates the anatomic source and severity of the problem and suggests a tentative solution. The information source is a combination of

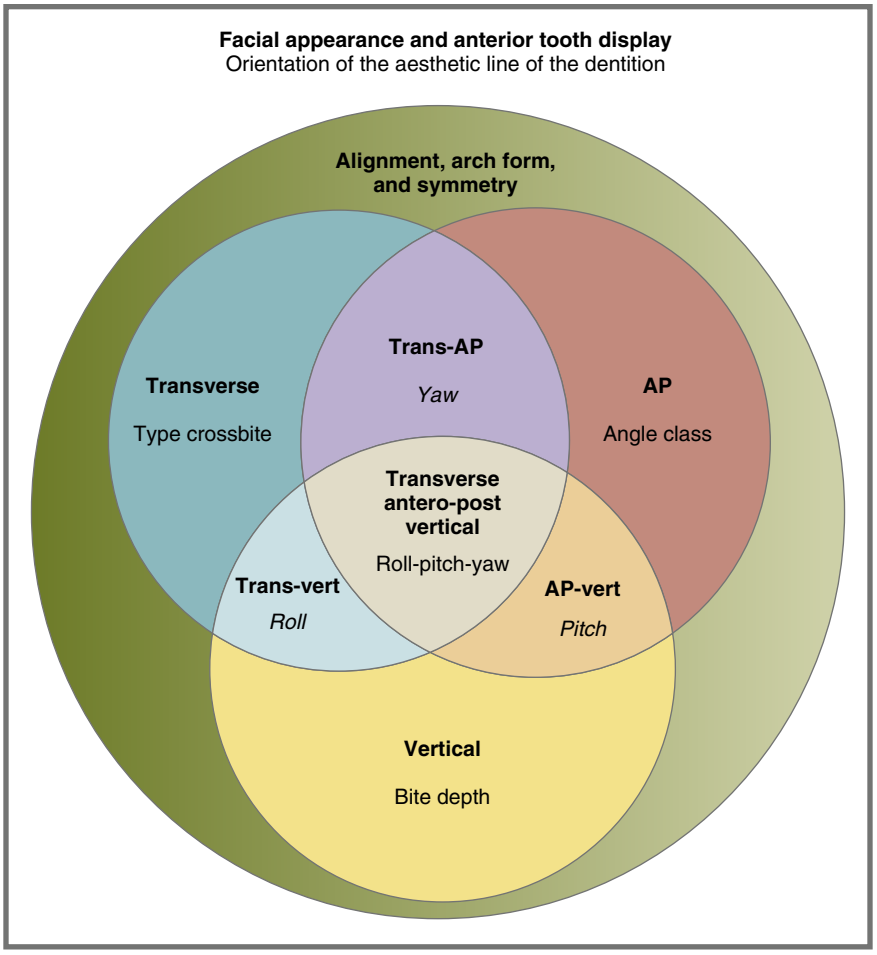


FIGURE 8-17 Ackerman-Proffit Orthogonal Analysis. When the disks representing the three planes of space are stacked as a Venn diagram, we see the interactions between the antero-posterior, transverse, and vertical dimensions. The three overlapping disks are shown atop a disk representing dental arch alignment, symmetry, and arch form, and all four disks are shown on a box representing the framework of facial appearance, anterior tooth display, and orientation of the esthetic line of the dentition. With this classification five or fewer characteristics can fully describe the dentofacial traits of any orthodontic condition.

the components of the database, and classification by the characteristics of dentofacial appearance, alignment of the teeth and occlusal/jaw relationships is used to organize it while being sure nothing important is overlooked. Classification can be accomplished clinically, without the aid of a cephalogram, simply by careful observation of the patient's occlusion and facial appearance. When the orthodontist trains himself or herself to make these judgments at the initial examination, and to substantiate the clinical original estimate from carefully looking at the cephalogram and making selected measurements if there are any doubts about relationships, diagnosis becomes a more natural process.

Proper diagnosis in clinical orthodontics is equivalent to a good hypothesis in basic research. A well-stated hypothesis is a question so well phrased that the path to an answer is inherent in the question. A well-stated orthodontic problem list automatically suggests alternative treatment plans. This analogy can be taken one step further. Orthodontic treatment is to diagnosis what the experiment is to a research hypothesis. The results tend to support or reject one's diagnosis or hypothesis.

TREATMENT PLANNING: THE PROBLEM-ORIENTED APPROACH

Prioritizing the Problem List

The problem list was generated using the interview to identify health problems and then identifying the orthodontic problems and their severity in the five steps in classification. The first step in planning treatment is to put the problems in priority order (see Fig. 8-8). This is a critically important step, because the same problem list prioritized differently will result in a different treatment plan. What are the important considerations in prioritization?

First, the patient's health problems are separated from the list of orthodontic problems, to be considered separately and dealt with before orthodontic treatment begins. This is not because they are more important but because health problems must be under control before active orthodontics begins. As we have noted, uncontrolled diabetes and active periodontal disease contraindicate orthodontic treatment because alveolar bone loss will be accelerated by tooth movement. This is not true when diabetes is under control and active periodontal

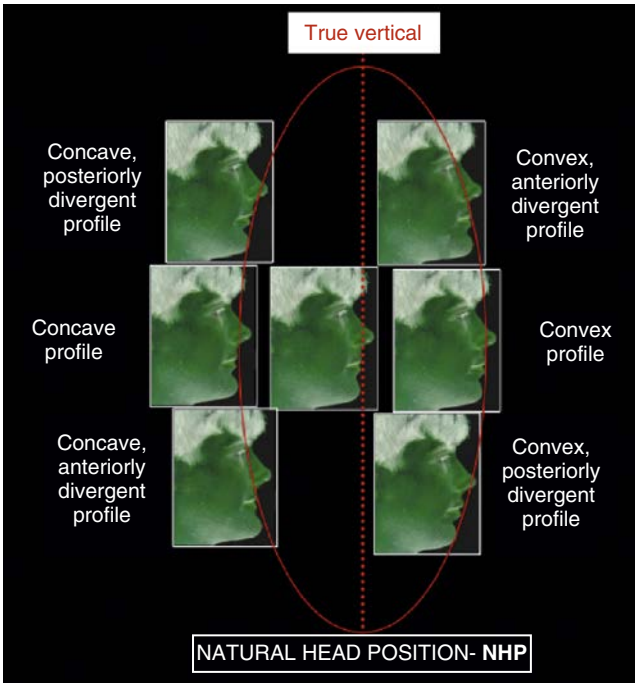


FIGURE 8-18 This illustration demonstrates that any facial pattern can be adequately described using the relative convexity or concavity of the facial profile and the relative anterior divergence or posterior divergence of the chin in relationship to the midface and upper face. Adding a description of face height, as well, nicely completes the picture (see Fig. 8-19).

disease has been eliminated. The same principle applies to other diseases—orthodontics usually is quite feasible when health problems have been brought under control but not when active disease is present.

Second, the orthodontic problems are prioritized, with the patient's chief concern in mind. This is a critically important step, because the same problem list prioritized differently will result in a different treatment plan. It is important to remember that from the patient's perspective, unless the chief concern was corrected during treatment, the treatment was not successful—even if it corrected what the doctor thought was the patient's most important problem.

That does not mean that the patient's chief concern is necessarily the first priority. There certainly is a role for patient education in the process of prioritizing the problem list—but agreement between the doctor and patient about the treatment priorities is the key to true informed consent (see further discussion below).

Then the treatment possibilities for the orthodontic problems are listed and evaluated, starting with the most important problem and continuing with each additional problem in priority order, taking into account the considerations in evaluation that are discussed immediately below.

There are two major advantages to this "individual problem–individual plan" approach before a final synthesis into a unified plan takes place. The first is that there is less chance of rejecting a treatment possibility too soon or never thinking of it at all. The second, which is even more important, is that this approach allows the orthodontist to keep the patient's various problems in perspective as to their priority in treatment. Since

Short face Average face Long face

FIGURE 8-19 Classically, the face width to face height ratio defines three basic types of faces. Today, the important observation rests with recognizing either an increased or decreased lower face height. This illustration shows computer-altered lower face heights ranging from short lower anterior face height to long lower face height. Long, narrow faces with increased lower face height usually have a tendency toward anterior open bite. Lower anterior face height is a reflection of the underlying skeletal pattern. Individuals with short lower face heights usually have relatively parallel horizontal facial planes—i.e., palatal plane, occlusal plane, and mandibular plane. Patients with long lower face heights have horizontal facial planes that tend to converge posteriorly (see Fig. 8-13).

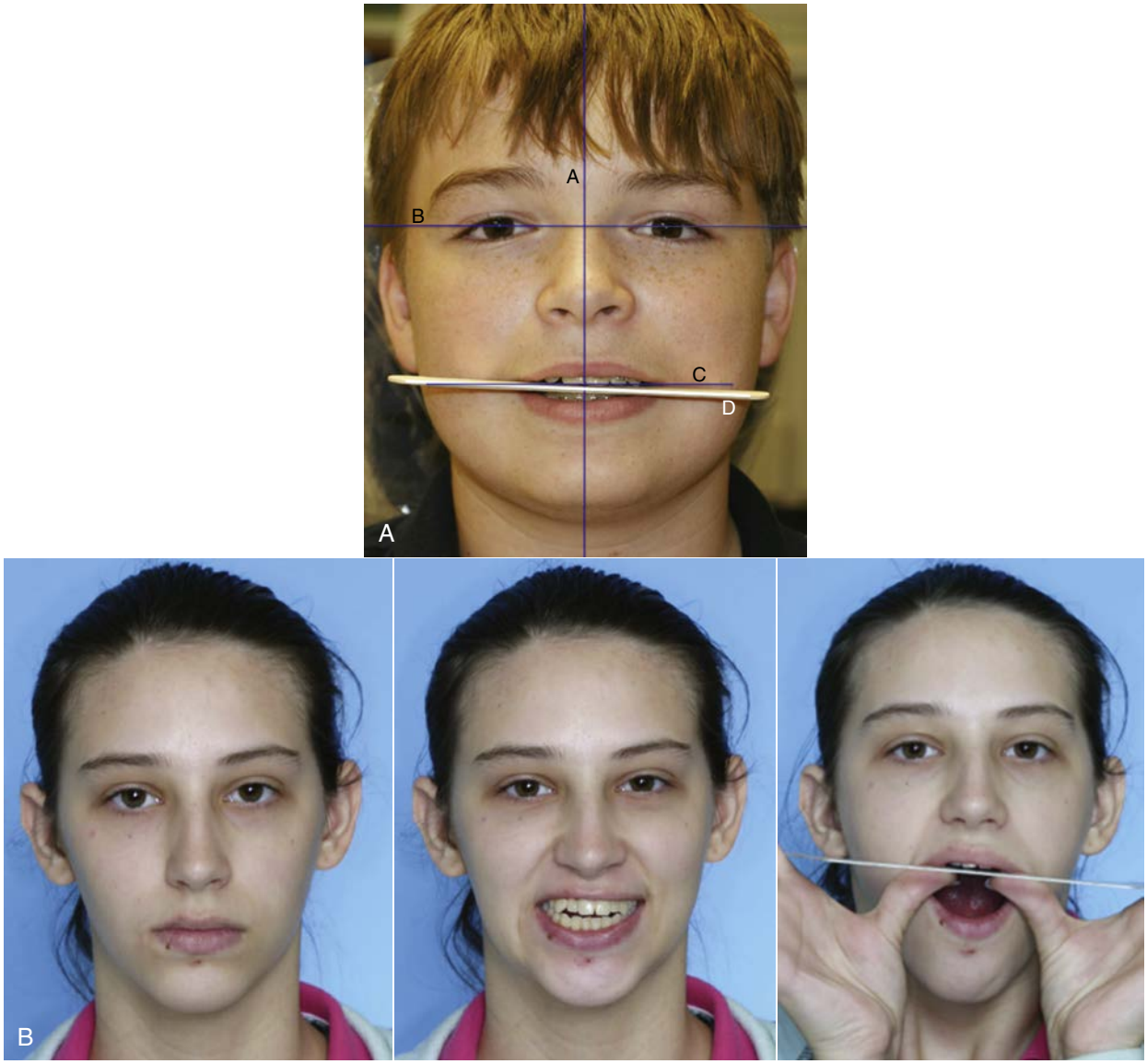


FIGURE 8-20 Determining the anteroposterior and lateral cants of the occlusal plane (pitch and roll) is important in diagnosis and treatment planning, particularly since we have the means today via skeletal anchorage to alter occlusal plane cants without having to resort to orthognathic surgery. There are two useful methods for ascertaining occlusal plane cants. One is a tongue depressor (A) and the other a Fox plane (B). Certainly the tongue depressor is more convenient because it is disposable, but for complex occlusal plane cants particularly in the posterior region there is still not a good substitute for the Fox plane. It is hoped that in the era of 3D imaging there will be new and more precise ways of measuring these types of cants and asymmetries.

compromises are invariably necessary in treatment planning, it is important that the most relevant issues are favored at the sacrifice of less significant factors. With our fetish about achieving theoretically ideal occlusion, we as orthodontists sometimes overlook the patient's "chief concern" because of our compulsion about how the teeth fit. We must satisfy not only ourselves but the patient as well.

Considerations in Evaluating Treatment Possibilities

To Extract or Not to Extract?

There are two major considerations in this important decision: the management of dental crowding or incisor protrusion (which, for incisor crowding, can be considered two aspects of the same thing) and the possibility of camouflage for skeletal problems.

Management of crowding or protrusion. If a patient presents with severe crowding or with procumbent maxillary and mandibular incisors, marked facial convexity, and severe lip protrusion, it should be obvious that removing premolars to alleviate crowding or allow retraction of the anterior teeth would be the best strategy for solving the problem. Unfortunately, most patients do not present such a clear-cut choice, and a debate about extraction versus expansion has raged in orthodontics for more than 100 years.[38] To better understand the grounds for

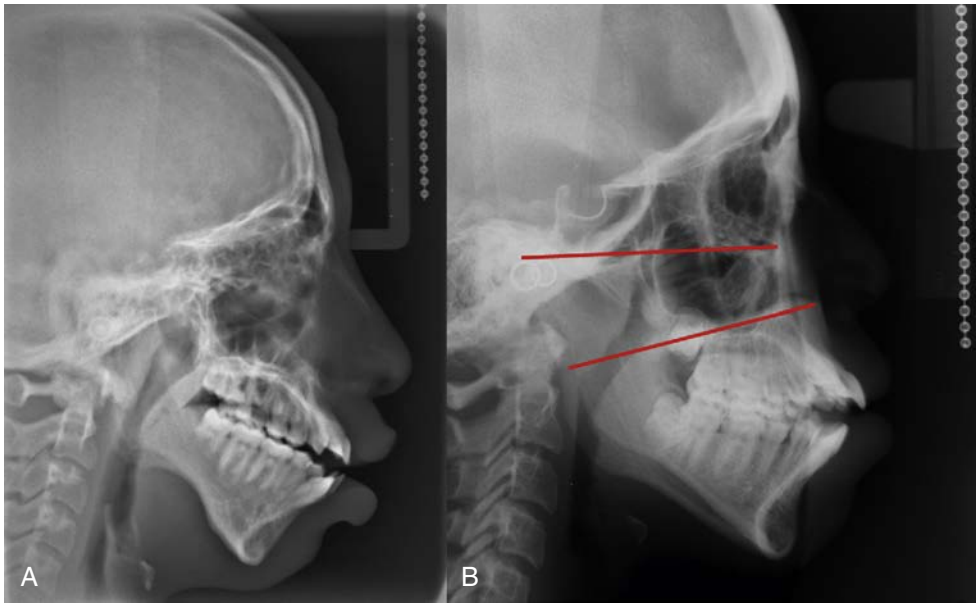


FIGURE 8-21 Cephalometric analysis is a useful tool in the diagnosis of open bite patterns. **A,** Steep mandibular plane (SN-GoGn) and antegonial notching result from deficient vertical growth at the condyle and compensatory bone apposition at the site of muscle attachment. **B,** Open bite can also result from a posteriorly tipped palatal plane in relation to Frankfort horizontal.

the debate, we must consider the pros and cons of dental arch expansion.

There are three reasons why an orthodontist cannot under usual circumstances significantly expand the dental arches. First, the tissues over the labial surfaces of the teeth cannot usually tolerate the teeth being moved into a more facial position. Bone tends to resorb vertically or, if the roots are moved out in advance of the rest of the teeth, fenestration or dehiscence of the labial cortical plate occurs. If a dehiscence is produced, the gingiva in later life may recede in that area.

The second reason is that the teeth will be unstable if they are moved labially or buccally "off their bony base"[39] and into positions where the soft tissue equilibrium can no longer be maintained. The intermolar and especially the intercanine widths for the most part have to be maintained close to their original dimensions during and after treatment, although it is not possible to predict arch stability on the basis of arch dimensions alone.[40] Depending on the initial incisor position, slight incisor advancement may be tolerated by their bony support and soft tissue constraints. Somewhat more lateral expansion in the molar area may be tolerated than in the premolar region (see Fig. 8-6).

Third, major dental arch expansion, particularly when the anterior teeth are moved facially, can have an adverse effect on facial appearance. It is simply not true that arch expansion always creates a more esthetic treatment outcome—an already convex face that has been made more convex as a result of orthodontic treatment does not become more attractive. By the same token, retracting the anterior teeth into an extraction space has the potential to have an adverse esthetic effect in a patient who already has a flat or concave profile. Thus, the general rule in regard to profile is "the principle of opposites." If a profile is convex, consider whether making it less convex would be enhance facial appearance, and if a profile is concave, consider whether making it less concave would be an enhancement. This realization is what caused orthodontists to reject Angle's dogmatic proscription against tooth extractions.

Incisor repositioning for camouflage. Changing the positions of anterior teeth to compensate for an underlying skeletal disproportion is called "camouflaging the skeletal discrepancy." If a jaw discrepancy exists but is not too severe, camouflage allows correction of the dental relationships while maintaining an acceptable facial appearance. In minor skeletal disproportions, it almost always makes more sense to camouflage the discrepancy with tooth movement rather than correcting the jaw discrepancy with orthognathic surgery. On the other hand, if a major skeletal disproportion is accompanied by a significant facial imbalance, there is a limit to the amount that the situation can be effectively camouflaged with orthodontics alone. The limits of camouflage are not defined by the amount of tooth movement that is possible, but by the patient's acceptance of the resulting appearance.[41]

At the tentative treatment plan stage, a key decision is establishing a target for the anteroposterior position of the incisors after treatment. If the incisors do not provide enough lip support and this is a problem for the patient, they can and should be proclined, but it must be recognized that periodontal health becomes an important consideration and that permanent retention will be required. If the incisors are too protrusive, so that lip separation at rest is apparent, the retraction requirements can be defined as the degree to which they have to be retracted: minimum, moderate, and maximum retraction. This defines the amount of extraction space required for retraction of the anterior teeth. Sometimes it is necessary to define the retraction requirements for each arch separately, especially when the goal is camouflage.

Visual treatment objectives in the extraction decision. To a great extent the retraction requirements influence the decision regarding which teeth are to be extracted and whether skeletal anchorage might be required. In the past, a method of

FIGURE 8-22 The decision to attempt treating this borderline extraction patient with nonextraction treatment related to her already pleasing facial balance and her attractive smile. During the course of treatment, the patient and her parents became concerned about what they saw as unfavorable changes in her dentofacial appearance related to the tooth movement. The orthodontist was concerned about the potential that the teeth might be less stable due to having moved the teeth "off basal bone" and perhaps beyond the limits of soft tissue adaptation. The patient, parent, and orthodontist jointly agreed to have four first premolars removed.

representing treatment goals two-dimensionally (sagittally and vertically) was by simulating the proposed skeletal and dental changes on the cephalometric tracing and estimating the facial soft tissue changes likely to result. This approach produced a visualized treatment objective (VTO) that could be achieved through the planned mechanotherapy. This was particularly effective in planning treatment for nongrowing patients (adults) and for planning surgical treatment. It was and is more difficult to produce reasonable VTOs for growing patients because of the limitations in our ability to forecast growth.

If we accept appearance and stability as the valid criteria for extraction in orthodontics, how well can we determine a priori in a child what the face will look like later on in adulthood and what the new functional environment will be after treatment? In fact, one cannot always predict what the face and dentition would look like after extracting teeth. For that reason, in borderline extraction cases, it is wise to judge the patient's response to initial alignment of the teeth before making a decision to extract (Fig. 8-22). Such an initial decision, of course, should be discussed at the patient–parent conference. It is one way to remove all doubt about the need for extraction, and both adult patients and parents usually appreciate such an approach even if the final decision is to extract. In essence, a reasonably longer treatment time is traded for a more predictable outcome.

In orthodontics, we have tended to be extreme in our views regarding extractions. In the early twentieth century, Angle's influence made it a sin to extract in any case; by mid-century, nearly all irregularities became extraction cases; and at the turn of the twenty-first century, extraction percentages were back to about where they were 100 years previously.[42] A recent study of the 2000–2012 period showed that there seems to be an equilibrium now in extraction percentages in a university clinic where these decisions are made individually by a group of attending orthodontists, with less change in extraction rates than previously.[43] It is clear now that many patients can be treated satisfactorily with extraction or expansion by controlling the amount of retraction or proclination of the anterior teeth.

Therapeutic Modifiability

A second important consideration is therapeutic modifiability, which is best considered in the context of the ratio between benefit and cost/risk for both treatment problems and treatment procedures. Some problems are more difficult and expensive to treat than others, whatever the method to treat them. For example, severe anterior open bite is very difficult to correct while a patient is growing; in contrast, severe deep bite is much easier to modify. Some treatment procedures are more difficult

and expensive, whatever the problem to which they would be applied. An excellent example is that single bone screws are easier and less expensive than bone plates because the orthodontist can place bone screws but would not want to reflect flaps as is necessary for bone plates.

The judgment when therapeutic modifiability is evaluated is whether there would be enough additional benefit from the more difficult and expensive choice to justify choosing it. You could be appropriately reluctant to begin treatment of anterior open bite while starting deep bite treatment at an earlier age; you could choose bone plates over orthognathic surgery for less severe anterior open bites while acknowledging the greater benefit from surgery in the more severe cases.

Interaction among potential solutions to problems. An additional consideration is the interaction among the problems and their potential solutions, since all of the factors eventually must be integrated into a unified treatment plan. It is quite possible that the solution to one problem would make another problem worse. For instance, if two of a patient's problems are maxillary constriction with bilateral maxillary palatal crossbite and excessive vertical height of the face accompanied by an anterior open bite, the potential solutions to these two problems would not be compatible. Maxillary expansion to correct the crossbite probably would increase vertical face height, which in turn would increase face height and exacerbate the anterior open bite. This interaction is well demonstrated in Case Study 8-2 (Fig. 8-24, *A–F*). Would you do maxillary expansion to correct the crossbite despite the effect on face height and open bite? Or accept the crossbite and focus on correcting the open bite? It is a far happier circumstance if correcting one problem also addresses another problem on the list, but it may be necessary to leave a less important problem uncorrected or partially corrected in order to correct a more important one.

Patient cooperation. A final consideration would be the patient cooperation needed during treatment. The more cooperation that is needed, the greater the chance that the patient's activity (or lack of it) will compromise the treatment.

Many orthodontists have the distinct impression that an increasing proportion of their practice today consists of children who are less compliant with treatment than patients in the past. This has led many practitioners to use "noncompliance" appliances whenever feasible. The two major types of noncompliance treatments are the Herbst appliance, which is nearly 100 years old, and skeletal bone anchors, a recent advance. Although both methods have considerable merit, their availability tends to drive treatment-planning decisions. The old expression "If one's only tool is a hammer, everything begins to look like a nail" is very apt in describing the impact of noncompliance treatment on the decision-making process in orthodontics. In a more perfect orthodontic world where patient compliance was not such an important factor, a broader choice of appliances would be considered—and for compliant patients, better results might be obtained.

Presenting the Tentative Treatment Plan, Finalizing It, and Obtaining Informed Consent

The Patient–Parent Conference

In the patient–parent conference that precedes the beginning of treatment, it should be emphasized that treatment plan to be discussed is a general strategy for treatment, based on the solution to problems requiring such things as tooth extractions or arch expansion, growth modification, control of tooth eruption or surgical orthodontics, and on explaining the patient's role in obtaining success in treatment.

As an example of the plan to be presented to the patient and parent, refer to the patient records shown in Case Study 8-2. The crowding, and the patient's concern about it, gives the alignment problem a high priority. Tentative treatment plans are immediately suggested by this listing of problems: extraction of a dental unit in all quadrants to solve the crowding or reducing the mesiodistal widths of maxillary teeth or an asymmetric extraction pattern to deal with the tooth-size discrepancy, with the parent or patient able to play a role in deciding which approach would be best.

Obviously, before an overall treatment plan can be written and a precise course of action outlined, all of the other problems and their tentative solutions and interactions must be considered. At the conference, there would be a similar discussion of possibilities for other problems, and a choice would be made there.

Informed Consent

The style of this conference reflects a major change in orthodontics that has resulted from reinterpretation of the legal doctrine of informed consent, which now emphasizes the ethical imperative of greater respect for patient autonomy in the decision-making process.[44] In the modern world, the orthodontist no longer makes decisions alone. He or she now does so jointly with the patient, parent, or both. Arguably this change in approach has had greater impact on orthodontic treatment planning than any technical innovations, even those as dramatic as current computer applications.

Informed consent sequence. Informed consent often is considered just the review of possible complications of treatment and a signature on that form. It is important to do that, but problems related to informed consent almost always involve a difference between what the doctor and patient understood about the goals of treatment and the expected outcome. The sequence for the parent–patient conference should be:

- "Johnny (or Mr. Jones), these are your orthodontic problems …."
- "I think the most important one of those is …. Do you agree?" (further discussion if needed)
- "Based on that, our plan for treatment is to …." (laid out in steps, with discussion about each problem and the possible ways to deal with it)
- "There are some possible potential problems that you should know about:
  - Your cooperation with treatment is important. You will need to ….
  - It is unlikely, but there are some complications you should know about:
    - decalcification
    - root resorption
  - (anything else pertinent to that specific patient)"
- "In summary, my staff and I look forward to working with you. Our goal is to help you with these problems to the best of our ability." (Note what that says about the ownership of the problems: they belong to the patient, not the doctor.)

Computer image predictions. At this conference, should patients and parents be shown computer image predictions of the effect on the profile of alternative treatment plans? There

is no doubt that when the alternative plans are orthodontic camouflage versus orthognathic surgery, the predictions help patients to understand the proposed treatment in a way they rarely gain from just a verbal description. A clinical trial established that patients who were shown their predictions appreciated the doctor's openness in doing this and that those who saw the predictions were more satisfied with their treatment than those who did not see them. It appears, therefore, that there is considerable benefit in showing the predictions and minimal risk in doing so.[45]

Treatment response. In the past, the great variation in treatment response among patients was too often ignored, and as a result rigid treatment plans were often set and almost religiously adhered to during the course of treatment. It is now well understood that each time a patient presents during treatment, the treatment plan has to be reconsidered in light of the treatment response and/or some element of the original problem that might have been overlooked. That applies primarily but not exclusively to the mechanotherapy plan—sometimes the strategic plan also has to be modified during treatment. That should be done only after a discussion with the patient or parent.

The Final Step: The Treatment Plan Details (Mechanotherapy Plan)

As part of informed consent, the patient–parent has understood and accepted the treatment plan concept (strategy). Since the mechanotherapy plan is for the doctor's use in maintaining the sequence of treatment and specifying the planned treatment procedures, it can be written as sparely or elaborately as desired. Typically, it would specify how the steps in treatment presented to the patient were to be accomplished biomechanically. After all, the goal and the strategy should be similar for patients with the same problems, but there are many ways to accomplish the desired tooth movement, growth modification, or other types of treatments.

Applications of skeletal anchorage. One of the fundamental principles in orthodontics is anchorage control. Using temporary titanium screws or plates (TADs) to obtain desired tooth movement or growth modification, while limiting unwanted dental side effects, has become a routine part of clinical orthodontics. To some extent, this has extended the envelope of tooth movement, blurring the line between orthodontic camouflage and orthognathic surgery. Moderate open bites[46] and, to a limited extent, Class III skeletal malocclusion[47] can be treated successfully using skeletal anchorage; transverse problems such as posterior crossbites, however, have met disappointing results.[48] The clinician has to balance the decreased morbidity for the patient with skeletal anchorage against improved facial harmony with orthognathic surgery to assure that patient treatment objectives are met.

Perhaps the most common use for skeletal anchorage is as a substitute for extraoral anchorage devices to retract incisors in noncompliant patients. It is important to remember that these devices make it easier to move the teeth within the biological boundaries of the bone; it does not create new bone; therefore, overzealous retraction of incisors for severe Class II camouflage could result in root dehiscence and resorption. More generally, skeletal anchorage does not change the limitations in tooth movement discussed previously.

Stereolithographic models and printing. One of the remarkable benefits of 3D imaging in orthodontics, the use of stereolithographic models in planning treatment and fabricating appliances, was not appreciated until fairly recently. The concept of producing a stereolithographic model on which an orthodontic appliance could be constructed came as a surprise when it was announced by Align Technology 20 years ago, but broader use of this technology is developing rapidly. Stereolithographic models have been used in oral-maxillofacial surgery during the past decade as guides in modifying bone plates for rigid fixation in orthognathic surgery, placing dental implants, or placing TADs.[49] In the near future, 3D printing will be possible for most materials including titanium, stainless steel, or even bone. Will it replace traditional wire fabrication, and if so, how will this change the practice of orthodontics? That, of course, remains to be seen.

Custom milling of attachments and robotic wire bending. Application of computer-assisted design and manufacturing (CAD-CAM) to clinical practice in orthodontics has made great strides recently, with two general approaches. The first application is the creation of customized brackets for individual patients, which will allow the use of "straight wires" to attain closer and closer approximations to theoretically ideal occlusion (Insignia, Ormco Corp., Monrovia, CA), versus the alternative approach of using laser scans of the positions of the attachments on the teeth and then having the archwires bent by a robot (SureSmile, Orametrix, Houston, TX). Of course, it is possible to use these approaches in combination (Incognito, 3M Unitek, Minneapolis, MN). These techniques have considerable appeal, and it will be interesting to see which methodology prevails in the future.

Problems with increased commercialization. It is an increasing concern that the commercialization of orthodontics has begun to short-circuit certain aspects of diagnosis and treatment planning. It can be argued that the commercialization of orthodontics began with Angle's patenting of his various appliance systems in the early 1900s. Yet, it has only been in recent years that direct marketing to consumers by the pharmaceutical industry has influenced commercial companies in dentistry and orthodontics to do the same. The classic example has been Invisalign's advertising campaign to influence prospective orthodontic patients to seek treatment with this method. Unfortunately, other orthodontic suppliers are beginning to follow this malign example.

The impact, of course, is that patients may arrive with a treatment plan already in their minds. In this circumstance, the orthodontist needs to keep in mind that patient health considerations and the other aspects of a comprehensive evaluation are necessary. From this perspective, patient education is an important consideration in obtaining informed consent, even if a quick evaluation suggests that what the patient says he or she wants would be satisfactory mechanotherapy.

CASE STUDY 8-1

Patient History

Patient A.B. was a healthy and socially well-adjusted 12-year-old African American girl who presented for an orthodontic evaluation with a chief complaint of "crooked teeth" and an "underbite" (which was interpreted to represent her negative overjet). There is no history of caries or periodontal disease.

Initial Records

Photographs and a panoramic radiograph were obtained by the treatment coordinator before the clinical examination by the orthodontist (see Fig. 8-23, *A*). A brief review of the photographs and panoramic radiograph allowed the orthodontist to make the four major treatment-planning

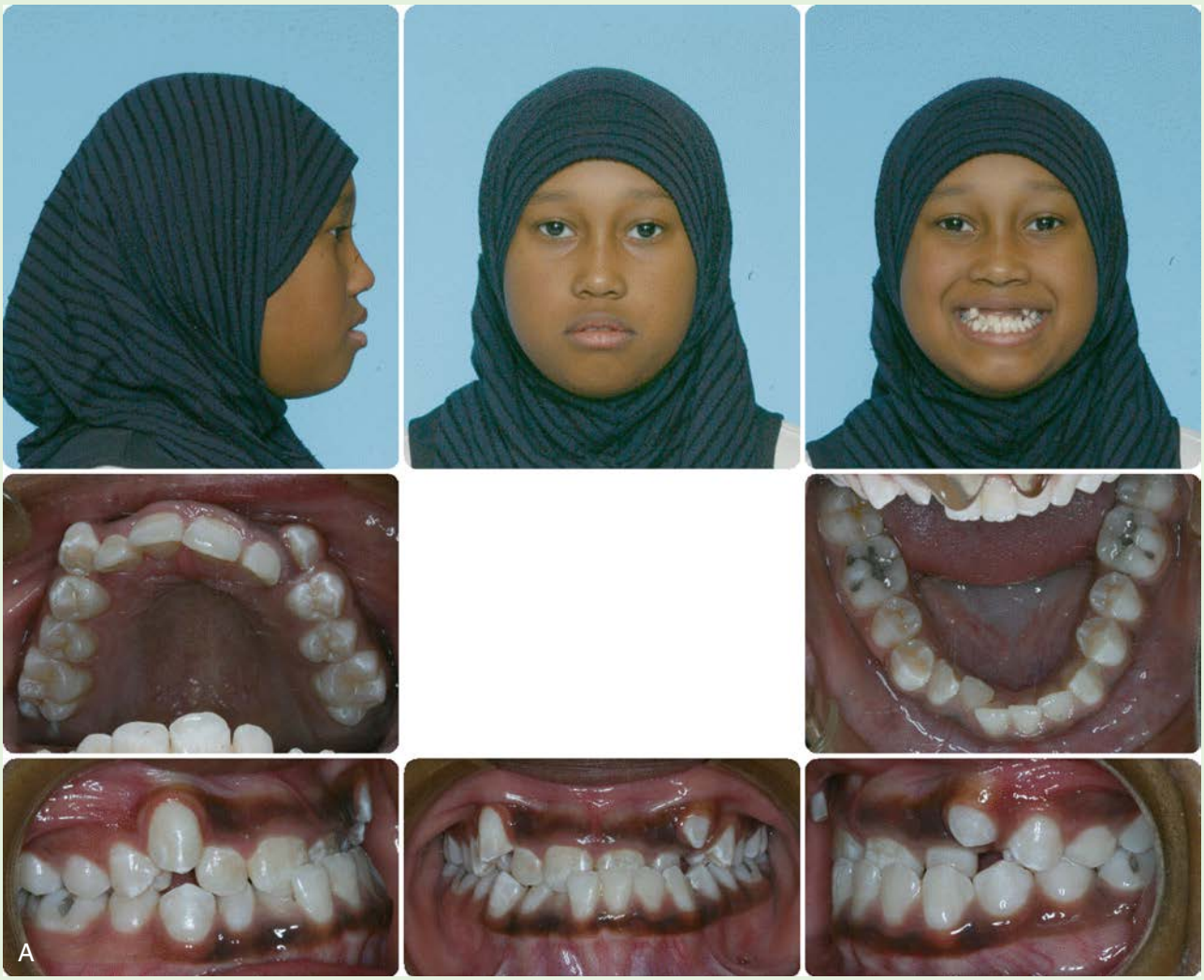


FIGURE 8-23 **A,** Patient A.B. Pretreatment photographs. **B,** Patient A.B. Pretreatment cone beam-computed tomography.

Continued

CASE STUDY 8-1—cont'd

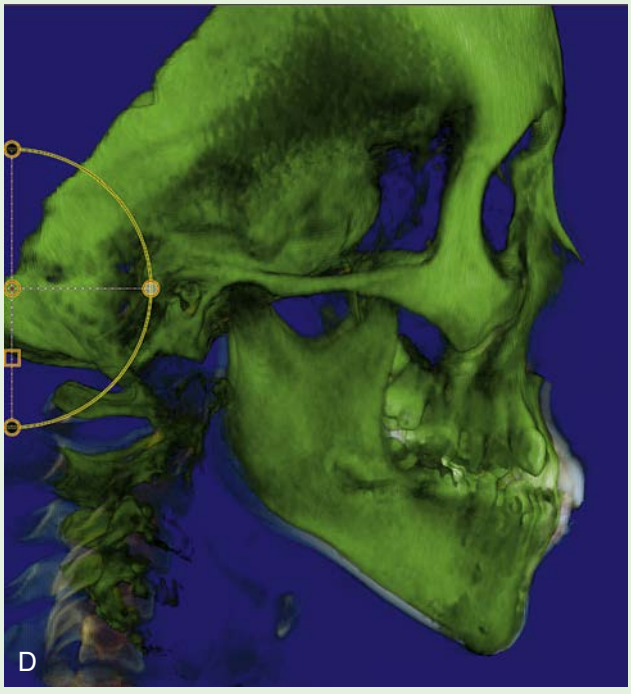


FIGURE 8-23, cont'd **C,** Patient A.B. Posttreatment photographs. **D,** Patient A.B. 3D cranial base registration.

CASE STUDY 8-1—cont'd

decisions in orthodontics, which in turn determine the number of visits required for a complete diagnosis and treatment plan:

1. Extraction versus nonextraction: A.B.'s crowding was sufficient to warrant maxillary premolar extractions, but this treatment option would worsen her overjet.
2. Surgical orthodontics versus nonsurgical treatment: A.B.'s facial appearance was acceptable, but her dental relationship put her at the borderline between orthodontic camouflage and orthognathic surgery.
3. Treat now versus treat later: A.B. is too old to attempt conventional protraction facemask orthopedic therapy. With the unpredictable nature of the Class III growth, possible treatment options include (1) limited Phase I treatment to extract the maxillary 1st premolar and align the incisor, (2) skeletal anchorage growth modification, or (3) wait until cessation of growth and treat with orthognathic surgery.
4. Conventional anchorage versus skeletal anchorage: this will largely depend on the treatment choice decided by the orthodontist and family.

Clinical Examination

The clinical examination revealed the following:

1. Dentofacial appearance
   a. Increase maxillary gingival display
   b. Increased mandibular incisor display
   c. Slightly convex profile resulting primarily from maxillary retrognathism
   d. Lip protrusion and increased interlabial gap
2. Alignment, arch form, and symmetry
   a. An 11-mm maxillary arch-perimeter deficiency, 2-mm mandibular arch-perimeter deficiency
   b. Elliptical maxillary and mandibular arch form with slight asymmetry of the mandibular anterior segment
3. Transverse relationships
   a. Maxillary midline deviation of 1 mm to the right
4. Anteroposterior relationships
   a. Class III skeletal with maxillary retrognathia
   b. Class III dental relationship with 2-mm overjet
   c. Maxillary and mandibular anterior dental compensation with retroclination of the maxillary incisors and retroinclination of mandibular incisors
   d. Anterior crossbite
5. Vertical relationships
   a. Anterior deep bite (50% overbite)

Prioritized Problem List

- Class III skeletal and dental, negative overjet and compensated incisors
- Crowding
- Slightly increased gingival display
- Anterior deep bite
- Slight midline deviation

Potential Solutions to the Individual Problems

- Class III—protract maxilla, restrain mandibular growth, LeFort advancement to position the maxilla anteriorly
- Crowding—extract maxillary premolar, distalize maxillary arch, procline maxillary incisors, procline mandibular incisors
- Increased gingival display—intrude maxillary incisors, impact the anterior maxilla with orthognathic surgery
- Anterior deep bite—intrude maxillary incisors, impact anterior maxilla with orthognathic surgery
- Midline deviation—elastics

Interactions and Risk/Benefit Considerations of Potential Solutions

- The interactions of the potential solutions to the prioritized problems show two clear options: skeletal anchorage orthopedics or orthognathic surgery.
- The risks of skeletal anchorage orthopedics include failure of bone anchors, lack of compliance with elastics, and sutural maturation/interdigitation that prevents skeletal change, while the risk of orthognathic surgery includes increased morbidity.
- The decision to extract in the maxillary makes the biomechanics of alignment easier for the orthodontist but complicates the anteroposterior goals unless adequate maxillary orthopedics can be obtained.

Mechanotherapy

Alternatives

- Fixed appliances with or without extractions if A.B. and her parent chose orthognathic surgery
- A combination of fixed appliances and bone anchor placement for orthopedic treatment
- A maxillary Hawley retainer and a bonded mandibular canine-to-canine fixed retainer

At this point in the process, the orthodontist turns over the following tasks to the treatment coordinator.

Patient–Parent Input

Given the two alternatives, the family expressed a preference for a nonextraction, skeletal anchorage orthopedics. A.B. and her family wanted treatment now due to a self-esteem issue associated with her malocclusion.

Informed Consent

A modification of the AAO informed consent booklet was used to outline the risk/benefit considerations of treatment, including retention considerations. The patient and parent were informed about the nature of Class III skeletal growth and the potential need for additional treatment including orthognathic surgery should she outgrow her correction. The estimate of treatment time and the cost of treatment were discussed.

Supplemental Records

A cone beam computed tomography (CBCT) scan and dental impressions were taken to confirm the findings from the clinical examination (see Fig. 8-23, *B*). The CBCT was taken to evaluate zygomatic bone thickness required for Class III skeletal anchorage orthopedics.

Detailed Treatment Plan

At a later date, the supplemental records were reviewed and the prioritized problem list, detailed treatment plan, and stepwise outline of the mechanotherapy were recorded and placed in a readily accessible part of the patient's file.

Posttreatment Evaluation

3D cranial base registration is shown in Figure 8-23, *D*. The initial CBCT is shown in green, while the posttreatment CBCT is overlain in ivory.

CASE STUDY 8-2

Patient History

Patient A.D., a healthy, 14-year-old Caucasian girl presented for an orthodontic evaluation with a chief complaint of "a space between my upper front teeth" and "the next tooth over is in toward the roof of my mouth" (which was interpreted to mean a palatally displaced lateral incisor). None of her parents or siblings had yet had orthodontic treatment, although they, A.D., and her brother had been evaluated by two other orthodontists.

Initial Records

Photographs and a panoramic radiograph were obtained by the treatment coordinator before the clinical examination by the orthodontist (see Fig. 8-24, *A* and *B*).

Triage

A brief review of the photographs and panoramic radiograph allowed the orthodontist to make the following critical preliminary judgments:

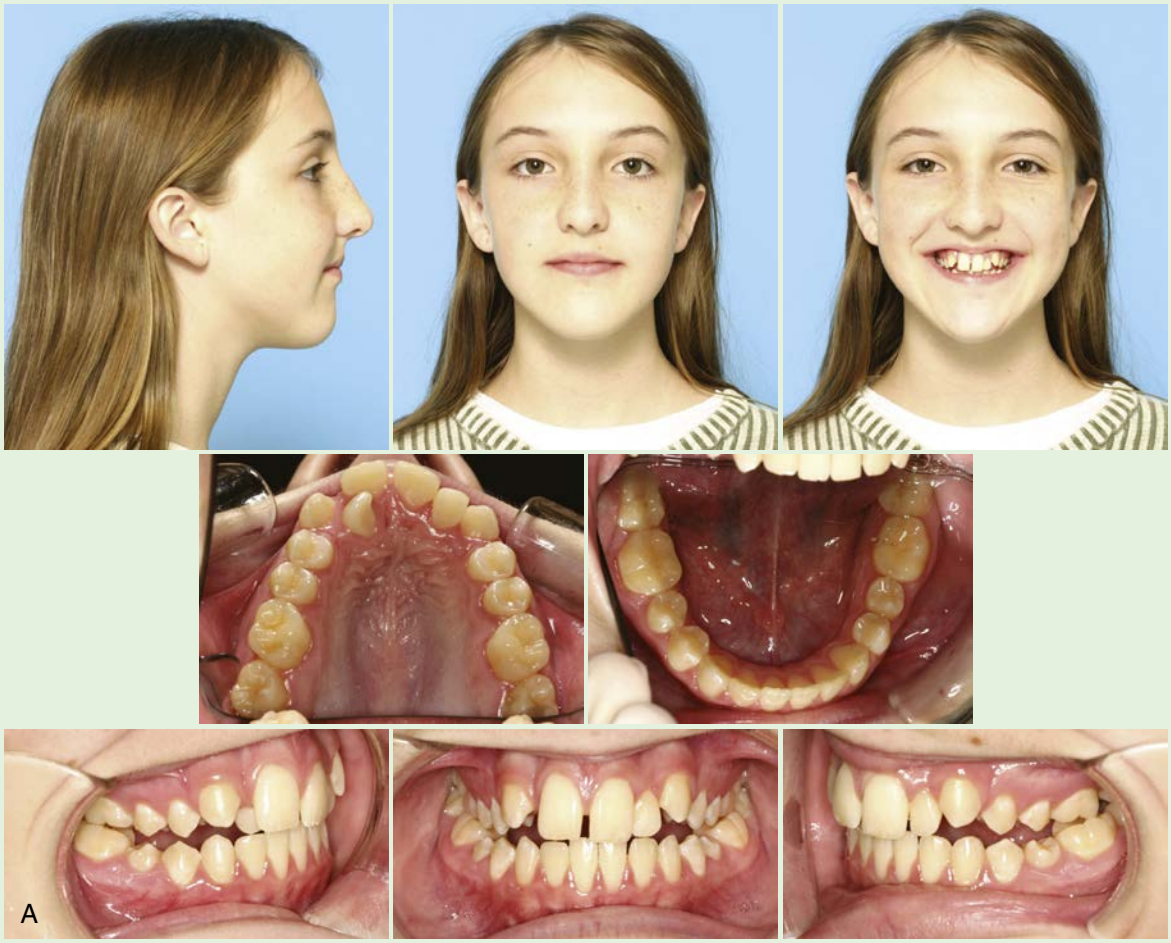


FIGURE 8-24 **A,** Patient A.D. Pretreatment photographs. **B,** Patient A.D. Pretreatment panoramic radiograph.

CASE STUDY 8-2—cont'd

FIGURE 8-24, cont'd **C,** Patient A.D. Pretreatment cephalogram. **D,** Patient A.D. Treatment predictions.

1. A.D.'s crowding, although the maxillary right lateral incisor was largely blocked out of the arch, was insufficient to warrant tooth extractions.
2. Several of the characteristics of A.D.'s orthodontic condition possibly required either orthognathic surgery or skeletal anchorage to completely resolve the problems.

Clinical Examination

The clinical examination revealed the following:

1. Dentofacial appearance
   a. Dolichofacial appearance with long lower face height
   b. Excessive posterior gingival show on smile
   c. Downward posterior cant (pitch) of the esthetic line of the dentition
   d. Mild dental cant (roll) of the anterior teeth slightly up on the patient's right side
   e. Maxillary midline shifted 2 mm to the right side
   f. Concave, straight profile with retrusive lips and thin upper lip vermilion
   g. Obtuse nasolabial angle
   h. Flat mentolabial fold
2. Alignment, symmetry, and arch form
   a. "V-shaped" maxillary arch and "U-shaped" mandibular arch
   b. A 4-mm maxillary arch-perimeter deficiency in the region of the blocked out maxillary right lateral incisor, although there is a 1-mm maxillary midline diastema
   c. Maxillary midline is shifted 2 mm to the patient's right.
   d. Tooth-size discrepancy due to the maxillary right lateral incisor
   e. Maxillary right permanent first molar is rotated mesially, contributing to the arch-perimeter deficiency affecting the maxillary right lateral incisor.
   f. Maxillary canines in slight labial ectopic position
3. Transverse relationships
   a. High constricted palatal vault
   b. A 7-mm maxillary transverse deficiency at the level of the first and second molars
   c. Bilateral maxillary palatal crossbite
4. Anteroposterior relationships
   a. Class II, with 4-mm overjet and palatal crossbite of the maxillary right lateral incisor

Continued

CASE STUDY 8-2—cont'd

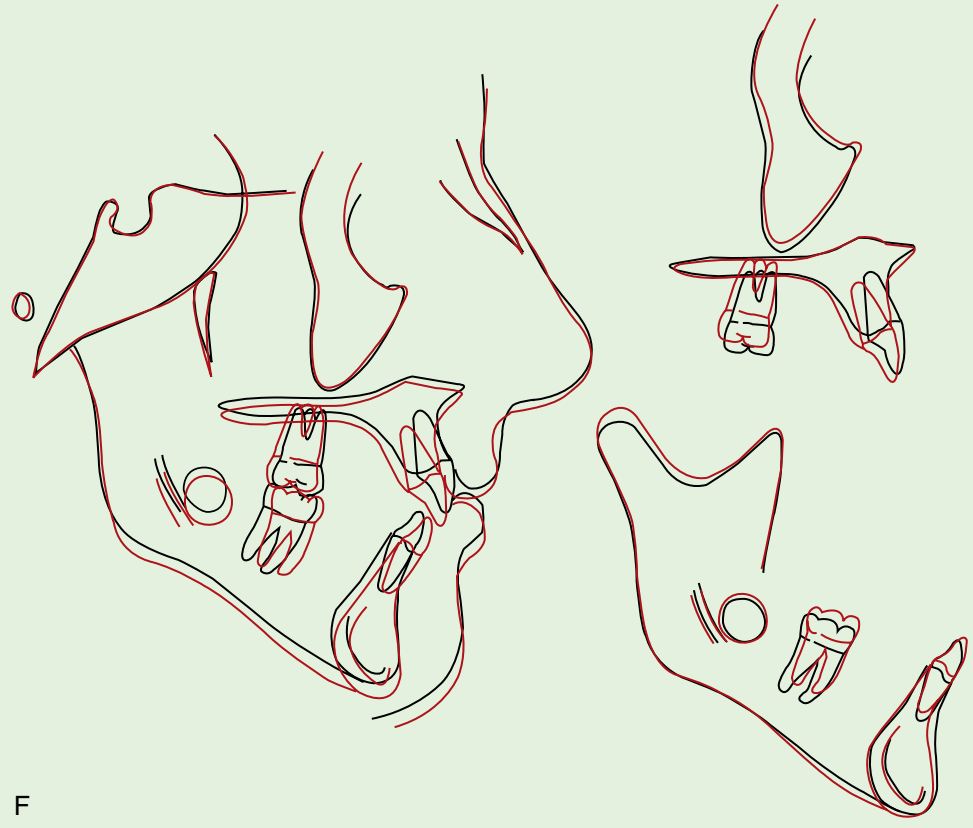


FIGURE 8-24, cont'd **E,** Patient A.D. Posttreatment photographs. **F,** Patient A.D. Cephalometric superimpositions.

CASE STUDY 8-2—cont'd

5. Vertical relationships
   a. Lateral open bites in the canine and premolar regions on the right and left sides
   b. Anterior open bite tendency
   c. Only three points of occlusal contact—maxillary and mandibular second molars and the maxillary right lateral incisor

Prioritized Problem List

- Maxillary midline diastema
- Palatoversion of the maxillary right lateral incisor
- Unesthetic anterior tooth display
- Bilateral maxillary palatal crossbites
- Lateral open bites
- Anterior open bite tendency
- Increased lower face height

Potential Solutions to the Individual Problems

- Maxillary midline diastema—After maxillary expansion, redistribute maxillary anterior space to close the diastema.
- Palatal displacement of the maxillary right lateral incisor—After posterior expansion and redistribution of maxillary anterior spacing, level and align lateral incisor.
- Esthetic anterior tooth display—If possible, intrude maxillary posterior teeth and change orientation of the esthetic line of the dentition and level and align teeth.
- Bilateral maxillary palatal crossbites—Rapid palatal expansion (10 mm)
- Lateral open bites—Either extrusive forces on the maxillary canines and premolars or intrusive forces on the molars
- Anterior open bite tendency—Intrusion of maxillary molars and autorotation of the mandible or extrusion of mandibular incisors
- Increased lower face height—Intrude maxillary posterior teeth or consider a LeFort I osteotomy to superiorly reposition the maxillae and gain autorotation of the mandible.

Interactions and Risk/Benefit Considerations of Potential Solutions

- Expanding the maxillary arch has the potential for increasing face height, which is already long, particularly since the maxillary molars manifest buccal crown inclination.
- Maxillary expansion can increase the open bite tendency by tipping the maxillary molars, effectively bringing the palatal cusps inferiorly.
- The risk of maxillary expansion is that it might ultimately lead to instability of the maxillary dental arch.

Treatment Possibilities

Alternatives

1. Rapid palatal expansion to correct posterior crossbites and skeletal anchorage to control the vertical dimension, particularly posteriorly.
2. Surgical correction of the posterior crossbites and open bites using a LeFort I maxillary osteotomy.

Supplemental Records

A lateral cephalogram and dental impressions were taken for further study. Video imaging simulated the potential facial outcomes that might result from orthodontics plus surgery (see Fig. 8-24, *C*).

Patient–Parent Conference

All records were reviewed with the patient and parent. The family was told about the treatment alternatives and was shown bone anchors, a rapid palatal expansion appliance, and multibonded appliances, as well as simulations of orthognathic surgical technique and the potential changes that might be derived from surgery in this case.

Given the two treatment alternatives, the family expressed a strong preference for the less invasive treatment plan, despite the fact that a more optimal facial outcome might have resulted from orthognathic surgery (see Fig. 8-24, *D*).

Unified Treatment Plan

- Relieve crowding of the maxillary right lateral incisor with 10 mm of rapid palatal expansion and correct posterior crossbites.
- Intrude maxillary posterior teeth with the aid of skeletal anchorage.
- Improve appearance of anterior tooth display with multibonded appliances.

Informed Consent

A modification of the AAO informed consent booklet was used to outline the risk/benefit considerations of treatment, including retention considerations. The 2-year estimate of treatment time and the cost of treatment were discussed.

Detailed Treatment Plan and Mechanotherapy

After the conference, the prioritized problem list, detailed treatment plan, and stepwise outline of the mechanotherapy were recorded and placed in a readily accessible part of the patient's file. The plan was similar to one of the original treatment alternatives (i.e., rapid palatal expansion to correct posterior crossbites, skeletal anchorage to control the vertical dimension, particularly posteriorly and fixed appliances).

Posttreatment Evaluation

The outcome of treatment is shown in Figure 8-24, *E* and *F*.

REFERENCES

1. Proffit WR, Fields Jr HW, Sarver DM, eds. *Contemporary Orthodontics*. 5th ed. St. Louis: Elsevier; 2013.
2. Miller G. *Spent: Sex, Evolution and Consumer Behavior*. New York: Viking Penguin; 2010.
3. Elliott C. *Better Than Well: American Medicine Meets the American Dream*. New York: WW Norton & Co.; 2003.
4. Ackerman JL, Ackerman MB, Kean MR. A Philadelphia fable: how ideal occlusion became the philosopher's stone of orthodontics. *Angle Orthod*. 2007;77:192–194.
5. Brunell JA, Bhat M, Lipton JA. Prevalence and distribution of selected occlusal characteristics in the US population. *J Dent Res*. 1996;75:706–713.
6. Bollen A, Cunha-Cruz J, Bakko DW, et al. The effects of orthodontic therapy on periodontal health: a systematic review of controlled evidence. *J Am Dent Assoc*. 2008;139:413–422.
7. *National Institute of Dental and Craniofacial Research*. Less Is Often Best in Treating TMJ Disorders. http://www.nidcr.nih.gov/OralHealth/Topics/TMJ/LessisBest.htm.
8. Proffit WR, et al. Prevalence of malocclusion and orthodontic treatment need in the United States: estimates from the N-HANES III survey. *Int J Adult Orthod Orthog Surg*. 1998;13:97–106.
9. Brook PH, Shaw WC. The development of an index for orthodontic treatment priority. *Eur J Orthod*. 1989;11:309–320.
10. Ackerman JL, Proffit WR. Soft tissue limitations in orthodontics: treatment planning guidelines. *Angle Orthod*. 1997;67:327–336.

11. Honein MA, Lindstrom JA, Kweder SL. Can we ensure the safe use of known human teratogens? the iPLEDGE test case. *Drug Saf*. 2007;30:5–15.
12. Phillips K. Body Dysmorphic Disorder: Clinical Aspects and Relationship to Obsessive Compulsive-Disorder. *Focus*. 2015;13(2):162–174. http://dx.doi.org/10.1176/appi.focus.130205.
13. Gorlin RJ, Cohen MM, Hennekam RCM, eds. *Syndromes of the Head and Neck*. 4th ed. New York: Oxford University Press; 2001.
14. Hartsfield JK, Everett ET, Al-Qawasmi RA. Genetic factors in external root resorption and orthodontic treatment. *Crit Rev Oral Biol Med*. 2004;15:115–122.
15. Delli K, Livas C, Sculean A, Katsaros C, Bornstein MM. Facts and myths regarding the maxillary midline frenum and its treatment: a systemic review of the literature. *Quintessence Int*. 2013;44:177–187.
16. Merchant SN, Vovk A, Kalash D, et al. Localized aggressive periodontitis treatment response in primary and permanent dentitions. *J Periodontol*. 2014;85:1722–1729.
17. Kravitz ND, Kusnoto B. Soft-tissue lasers in orthodontics: an overview. *Am J Orthod Dentofac Orthop*. 2008;133(suppl 4):S110–S114.
18. Okeson JP. *Management of Temporomandibular Disorders and Occlusion*. 7th ed. St. Louis: Elsevier; 2012.
19. Schabel BJ, Baccetti T, Franchi L, McNamara JA. Clinical photography vs digital video clips for the assessment of smile esthetics. *Angle Orthod*. 2010;80:678–684.
20. Trotman CA. Faces in 4 dimensions: why do we care, and why the fourth dimension? *Am J Orthod Dentofac Orthop*. 2011;140:895–899.
21. Goldberger JJ, Buxton AE. Personalized medicine vs guideline-based medicine. *JAMA*. 2013;309:2559–2560.
22. Lu C, Ko E, Huang C. The accuracy of video imaging prediction in soft tissue outcome after bimaxillary orthognathic surgery. *J Oral Maxillofac Surg*. 2003;61:333–342.
23. Grauer D, Cevidanes LSH, Styner MA, Ackerman JL, Proffit WR. Pharyngeal airway volume and shape from cone-beam computed tomography: relationship to facial morphology. *Am J Orthod Dentofac Orthop*. 2009;136:805–814.
24. Im J, Cha JY, Lee KJ, Yu HS, Hwang CJ. Comparison of virtual and manual tooth setups with digital and plaster models in extraction cases. *Am J Orthod Dentofac Orthop*. 2014;145:434–442.
25. Grunheid T, McCarthy SD, Larson BE. Clinical use of a direct chairside oral scanner: an assessment of accuracy, time and patient acceptance. *Am J Orthod Dentofac Orthop*. 2014;146:673–682.
26. Jacobson A. *Radiographic Cephalometry: From Basics to Videoimaging*. Chicago: Quintessence; 1995.
27. Athanasiou AE. *Orthodontic Cephalometry*. St. Louis: Mosby; 1995.
28. McNamara Jr JA. A method of cephalometric evaluation. *Am J Orthod*. 1984;86:449–469.
29. Sassouni V. The class II syndrome: differential diagnosis and treatment. *Angle Orthod*. 1970;40:334–341.
30. Jacobson A. The Wits appraisal of jaw disharmony. *Am J Orthod*. 1975;67:125–138.
31. Frazier-Bowers SA, Koehler KE, Ackerman JL, et al. Primary failure of eruption: further characterization of a rare eruption disorder. *Am J Orthod Dentofac Orthop*. 2007;131:e1–e11.
32. Proffit WR, Frazier-Bowers SA. Mechanism and control of tooth eruption: overview and clinical implications. *Orthod Craniofac Res*. 2009;12:59–66.
33. Bennett NG. Report of the committee on orthodontic classification. *Oral Health*. 1912;2:321–327.
34. Case CS. *A Practical Treatise on the Techniques and Principles of Dental Orthopedia and Prosthetic Correction of Cleft Palate*. Chicago: CS Case Co.; 1921.
35. Simon P. *Fundamental Principles of a Systematic Diagnosis of Dental Anomalies*. (translated by Lischer BE). Boston: Stratford; 1926.
36. Ackerman JL, Proffit WR. The characteristics of malocclusion: a modern approach to classification and diagnosis. *Am J Orthod*. 1969;56:443–454.
37. Ackerman JL, Proffit WR, Sarver DM, et al. Pitch, roll, and yaw: describing the spatial orientation of dentofacial traits. *Am J Orthod Dentofac Orthop*. 2007;131:305–310.
38. Case CS. The question of extraction in orthodontics. *Am J Orthod*. 1964;50:658–691 (reprinted from 1918).
39. Brodie AG. The apical base: zone of interaction between the intestinal and skeletal system. *Angle Orthod*. 1966;36:136–151.
40. Little RM. Stability and relapse of dental arch alignment: review article. *Br J Orthod*. 1990;17:235–241.
41. Mihalik CA, Proffit WR, Phillips C. Long-term follow-up of class II adults treated with orthodontic camouflage: a comparison with orthognathic surgery outcomes. *Am J Orthod Dentofac Orthop*. 2003;123:266–278.
42. Proffit WR. Forty-year review of extraction frequencies at a university orthodontic clinic. *Angle Orthod*. 1994;64:407–414.
43. Jackson T, Guez C, Proffit WR, [Master's thesis]. The Modern Likelihood of Orthodontic Tooth Extraction. Journal of Dental Research. 2016 (submitted).
44. Carr KM, Fields Jr HW, Beck FM, et al. Impact of verbal explanation and modified consent materials on orthodontic informed consent. *Am J Orthod Dentofac Orthop*. 2012;141:174–186.
45. Phillips C, Hill BJ, Cannac C. The influence of video imaging on patients' perceptions and expectations. *Angle Orthod*. 1995;65:263–270.
46. Scheffler NR, Proffit WR, Phillips C. Outcomes and stability in patients with anterior open bite and long anterior face height treated with temporary anchorage devices and a maxillary intrusion splint. *Am J Orthod Dentofac Orthop*. 2014;146:594–602.
47. Nguyen T, Cevidanes L, Paniagua B, Zhu H, de Paula L, DeClerck H. Use of shape correspondence analysis to quantify skeletal changes associated with bone-anchored class III correction. *Angle Orthod*. 2014;42:329–336.
48. Lagravère MO, Carey JP, Heo G, Toogood RW, Major PW. Transverse, vertical, and anteroposterior changes from bone-anchored maxillary expansion vs traditional rapid maxillary expansion: a randomized clinical trial. *Am J Orthod Dentofac Orthop*. 2010;137:304, e1-e12.
49. Mehra P, Miner J, D'Innocenzo R, Nadershah M. Use of 3-d stereolithographic models in oral and maxillofacial surgery. *J Maxillofac Oral Surg*. 2011;10:6–13.

9

Special Considerations in Diagnosis and Treatment Planning

David M. Sarver

OUTLINE

Purposes and Goals of Orthodontic Treatment, 246
Orthodontics and Quality of Life, 246
Role of the Orthodontist in Total Facial Aesthetic Planning, 246
Records, 248
Facial Photographs, 248
Digital Video Technology in Orthodontic Records, 251
Intraoral Photographs, 252
Cephalometric Radiographs, 253
Contemporary Applications of Cephalometry, 254
Computer Imaging in Contemporary Treatment Planning, 255
Clinical Examination of Facial Symmetry and Proportion: Its Significance in Treatment Planning, 255
Application of Database Programs to Clinical Information, 255
Evaluation of Facial Proportions (Macroaesthetics), 259
Frontal Vertical Facial Relationships, 259
Increased Face Height, 260
Decreased Face Height, 260
Transverse Facial and Dental Proportions, 261
Central Fifth of the Face, 262
Medial Two-Fifths of the Face, 262
Outer Two-Fifths of the Face, 262
Evaluation of Nasal Proportions, 264
Radix, 264
Nasal Dorsum, 264
Nasal Tip, 264
Columella, 264
Nasolabial Angle, 264
Lip Projection, 264
Excessive versus Inadequate Lip Projection, 265
Effects on the Labiomental Sulcus, 265
Chin Projection, 265
Throat Form, 265
Miniaesthetics (Evaluation of the Smile), 265
Importance of the Smile in Orthodontics, 265
Analysis of the Smile, 268
Diagnostic Smile Analysis: Measurement of Characteristics, 270
Vertical Characteristics of the Smile, 271
Transverse Characteristics of the Smile, 272
Oblique Characteristics of the Smile, 276
Dental Microaesthetics and Its Applications to the Smile, 276
Gingival Shape and Contour, 276
Bracket Placement in Preparation for Changes in Gingival Shape, 281

Orthodontic diagnosis and treatment planning are in a period of remarkable change, away from a previous focus on dental occlusion and hard tissue relationships and toward a greater emphasis on soft tissue adaptation and proportions.[1,2] This change has been driven by new technology and interpretation of cosmetic dental principles into the treatment planning process. Dentists themselves have become more aware of the developments in cosmetic dentistry and the benefits now available to their patients. Television and advertising spread consistent images of aesthetic faces and smiles worldwide. Parents and patients now notice and disapprove of the aesthetic liability of unattractive smiles and as a result, orthodontists know that treatment success must be judged by more than the dental occlusion. Chapter 8 of this book has cogently discussed the current philosophical changes in the approach to diagnosis and treatment planning, departing from the traditional model consisting of an initial visit followed by records and subsequent analysis and treatment plan presentation. Until the past decade, the emphasis in planning orthodontic treatment was based on photographs, model analysis, and cephalometric analysis. The contemporary approach involves a much more detailed clinical examination where many aspects of the treatment plan reveal themselves as a function of the systematic evaluation of the functional and aesthetic presentation of the patient. Because Chapter 8 also covered the functional aspects of orthodontic treatment, our intent in this chapter is to present the components of clinical assessment of the hard tissue and soft tissue that logically lead the clinician to the final treatment plan. In planning treatment, to visualize and establish the appearance of the teeth and face that is desired as a treatment outcome and to work backward ("retroengineer") to the hard tissue relationships that are needed to obtain these soft tissue proportions and lip–tooth relationships make much more sense. This approach is not completely contradictory to the records-based approach, where the hard tissues are analyzed (cephalometric head film and models) and the outward soft tissue is a secondary consideration but should be considered an expansion of how we diagnose and form the treatment plan. It is important that in many

instances the clinical observations should override the cephalometric impression.

The purpose of this chapter is to explore in detail the new concepts that underlie clinical application of the soft tissue paradigm with the major emphasis on appearance and aesthetics. The next objective will be to illustrate how treatment approaches change when these concepts are applied. The focus is on the things that have not been emphasized in previous discussions of orthodontic diagnosis and treatment planning. The author recognizes that the reader will have a background in those fundamentals, because Chapter 8 covers that in exquisite detail.

PURPOSES AND GOALS OF ORTHODONTIC TREATMENT

Orthodontics and Quality of Life

In general, the goal of orthodontic treatment is to improve the patient's life by enhancing dental and jaw function and dentofacial aesthetics. From this perspective, the role of orthodontics is analogous to that of several other medical specialties, such as orthopedics and plastic surgery, in which the patient's problems often do not result from disease but rather from distortions of development. As the health care field has evolved from a disease-oriented focus to a wellness model,[3] orthodontics now is viewed more clearly as a health service dedicated to establishing emotional and physical wellness. Dental and facial distortions create a disability that can influence physical and mental health. Appropriate treatment can be important for the patient's well-being.

In diagnosis, whether in orthodontics or other areas of medicine or dentistry, practitioners must not concentrate so closely on their own specialized areas (and a medical or dental professional does not have to be a specialist to take a specialized point of view) when assessing patients' overall conditions that they overlook other significant problems. The problem-oriented approach to diagnosis and treatment planning has been advocated widely in medicine and dentistry as a way to overcome this tendency to concentrate on only one part of a patient's problem. The essence of the problem-oriented approach is the development of a comprehensive database of pertinent information that precludes the possibility of problems being overlooked. We suggest that the problem-oriented treatment-planning approach has grown further to include examination, documentation, and assessment of patient attributes that are normal or positive. The reason for this is the recognition that in orthodontic treatment we may often have an unwittingly negative impact on patient attributes that are good.[4-6] The most commonly cited example of this is the case in which correction of a Class II malocclusion secondary to mandibular deficiency is performed via maxillary first premolar extraction and reduction of overjet. This plan corrects the "problem" of the Class II malocclusion but at the expense of a normal or ideal midface. This leads us to an expanded concept of "problem-oriented treatment planning" to include a "protection strategy" (Fig. 9-1). In other words, identification of both the problems and the normal or ideal attributes of a patient are important as illustrated in the example in Figure 9-2. If the ideal attributes at the initial presentation are not recognized, then the treatment plan, which is focused solely on the problems, may lead to deleterious effects on the ideal attributes.

Chapter 8 gives the reader a basic understanding of the problem-oriented approach to orthodontic diagnosis and treatment planning and of dental cast and cephalometric analysis; therefore, the focus of this chapter is on soft tissue considerations that previously have not received as much consideration in orthodontic evaluation as they should have and on the integration of traditional orthodontic treatment procedures with other treatment (periodontal, restorative, surgical) that can improve the outcome for many patients.

Role of the Orthodontist in Total Facial Aesthetic Planning

One of the first major influences in changing orthodontic thinking and approach began when orthognathic surgery became a more refined and less traumatic procedure; it rapidly became a reasonable treatment option for orthodontists to incorporate into their differential diagnoses and treatment planning strategies. The facial changes created by improvement of skeletal malformations were truly remarkable, and the facial effect of orthognathic treatment moved to the forefront of orthognathic surgical goal setting.

The retreatment of orthodontically camouflaged cases and the recognition of the effect of orthodontics and growth modification on the face have changed the focus of "routine" orthodontic treatment. The goals of orthognathic treatment for the improvement of facial appearance may be attained readily by

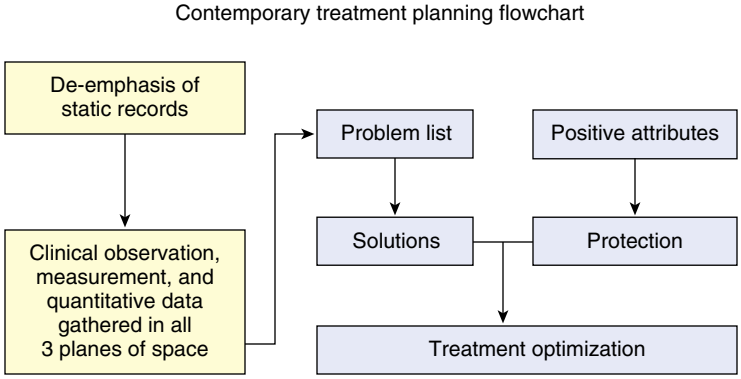


FIGURE 9-1 We suggest an expansion of problem-oriented treatment planning to include a "protection strategy" to avoid unwittingly negative impacts on a patient's positive attributes.

orthodontic methods in children, but the tools are different for different ages: orthognathic surgery in the adult for skeletal modification and growth modification in the adolescent.

The aesthetic and functional goals for growing patients should be the same as they are for adult patients. Growth modification techniques may allow the orthodontist to direct growth to achieve dramatic facial changes similar to those produced by surgery; these changes are an important part of patient motivation and satisfaction. Individualized treatment plans with differential functional and aesthetic options of

FIGURE 9-2 A classic example of problem-oriented treatment planning with the focus on hard tissues only is best illustrated by the Class II mandibular deficiency treated with premolar extraction rather than a contemporary growth modification approach. **A**, This 18-year-old female had undergone premolar extraction for retraction of her anterior teeth to correct her overjet, resulting in an obtuse nasolabial angle, an exaggeration of nasal projection, and inadequate lower facial projection. **B**, Seeking aesthetic improvement 4 years after her original orthodontic treatment, she underwent retreatment through orthodontic decompensation of her previous orthodontic treatment combined with a surgical bimaxillary advancement and rhinoplasty to an obvious aesthetic improvement. **C,** This adult patient in her 40s was prompted to seek treatment after the previous patient, her niece, had completed her treatment. Impressed with the outcome, she presented for correction of her aesthetic issues, and the same plan was recommended and completed. **D**, The final outcome for her was just as outstanding as that for her niece. What is the point here? The young adolescent was treated in the exact same manner her aunt had been fully treated three decades before, to the same unfortunate outcome.

FIGURE 9-3 This "selfie" illustrates the emphasis on frontal aesthetics and its importance in defining our aesthetic problem list. (Photo © istock.com.)

treatment should be planned and discussed with the parents of young patients. Adult patients may choose more aggressive approaches to treatment, whereas the parents of adolescent patients are more cautious. This occurs probably for two reasons: (1) the parents are making decisions on behalf of the child and tend to be more conservative, whereas the adult is making personal treatment decisions; and (2) some surgical decisions are not needed or indicated during adolescence.

Before cephalometric radiography, astute clinicians looked at the face and made general correlations with the way tooth movement might affect the esthetics of the face. Cephalometrics quantified dentoskeletal relations, established norms, and focused the profession on anatomic relations, including the skeletal pattern. Cephalometric diagnostic guidelines, however, pay only marginal attention to the soft tissue profile; although many analyses include soft tissue evaluation, it is usually limited to the profile. The emphasis on cephalometrics that developed in clinical orthodontic diagnosis and treatment planning departed from the original vision of its developers. Brodie[7] cautioned that cephalometrics was never intended as the sole decision maker in orthodontic treatment plans and that its main strength was in quantification of growth and research.

Interestingly, orthodontics is now coming full circle, looking carefully at dentofacial proportions before the cephalometric radiograph is even considered. Of even greater significance is the modern emphasis on the frontal evaluation and the effect of orthodontics and surgery on the frontal vertical relationships. The current popularity of "selfies" is instructive in that the subject's emphasis is the frontal face, generally on smile (Fig. 9-3). Rarely will you see a person taking a profile selfie on his or her phone camera.

The greater recognition of facial planning is only one leg of the contemporary diagnostic tripod. We recommend that aesthetic planning be approached with three major divisions in mind: macroaesthetics (the face), miniaesthetics (the smile), and microaesthetics (tooth and gingival shape and form). (To see case studies demonstrating this approach, please go to www.orthodontics-principles-techniques.com.) Contemporary records should reflect this approach and facilitate the complete three-dimensional evaluation of the patient's aesthetic presentation.

Records

Facial Photographs

For ideal photographic representation of the face, we recommend that the camera be positioned in the portrait position to maximize use of the photographic field. Orienting the camera in the landscape position captures much of the background that is unneeded and detracts from the image by diminishing the size of the face in the picture.

The following facial photographs are recommended as the expected routine for each patient:

1. Frontal: The patient assumes a natural head position and looks straight ahead into the camera. Four types of frontal photographs (Fig. 9-4) are useful:
   a. *Frontal at rest*. If lip incompetence is present, the lips should be in repose and the mandible in rest position.
   b. *Frontal view with the teeth in maximal intercuspation, with the lips closed, even if this strains the patient*. This photograph serves as clear documentation of lip strain and its aesthetic effect, and the lips-together picture is recommended in patients who have lip incompetence. If lips-apart posture is present, then an unstrained image is also recommended as an additional image. The reason for this image is to allow visualization of the philtrum–commissure height relationship, etiologic in the differential diagnosis of excessive gingival display on smile.
   c. *Frontal dynamic* (*smile*). As described in more detail later in this chapter, the smile can vary with emotion. A patient who is smiling for a photograph tends not to elevate the lip as extensively as a laughing patient. The smiling picture demonstrates the amount of incisor show on smile (percentage of maxillary incisor display on smile) and any excessive gingival display. However, the still image of the smile can be variable. Think about it—we squeeze off a picture that is about a 1/125-second duration of a process that has a start and a finish (from the lips together through the smile animation back to the lips being together). Also, having a child relax during the photograph session can also be a challenge. Many times we can only obtain a forced smile at best. Because of this variability, we recommend the addition of digital video clips as part of the patient record.[6,7] This recommendation will be discussed in detail later in this section.
   d. *A close-up image of the posed smile*. This view now is recommended as a standard photograph for careful analysis of the smile relationships. The posed-smile photograph is discussed in greater detail later in the chapter.
2. *Oblique* (*three-quarter*, *45-degree*)
   Patient in natural head position looking 45 degrees to the camera. Three views are useful (Fig. 9-5).
   a. *Oblique at rest*. This view is useful for examination of the midface and is particularly informative of midface deformities, including nasal deformity. Orthodontists should recognize that persons are not seen just on profile or frontally and that the three-quarter view is particularly valuable in assessing the way a patient's face is viewed by others. This view also reveals anatomic characteristics that are difficult to quantify but are important aesthetic factors, such as the chin–neck area, the prominence of the gonial angle, and the length and definition of the border of the mandible. The view also permits focus on lip fullness and vermilion display. For a patient with obvious

FIGURE 9-4 Recommended frontal images. **A,** Frontal at rest. If lip incompetence is present, the lips should be in repose and the mandible in rest position. **B,** Frontal view with the teeth in maximal intercuspation, with the lips closed, even if this strains the patient. This photograph serves as clear documentation of lip strain and its aesthetic effect, and the lips-together picture is recommended in patients who have lip incompetence. **C,** Frontal dynamic (smile). A patient who is smiling for a photograph tends not to elevate the lip as extensively as a laughing patient. The smiling picture demonstrates the amount of incisor show on smile (percentage of maxillary incisor display on smile), as well as any excessive gingival display. **D,** A close-up image of the posed smile. This is now recommended as a standard photograph for careful analysis of the smile relationships.

facial asymmetry, oblique views of both sides are recommended.

b. *Oblique on smile.* As mentioned in Chapter 8, there are diagnostic limitations of plaster casts, virtual models, and, as far as we are concerned, virtually all static records because they do not reflect the relationships of the teeth to the lips and surrounding soft tissue, especially in evaluation of the smile. Standard orthodontic records consist of the frontal smile, frontal rest, and profile. Often, in clinical practice, a parent will ask why the teeth appear flared, and they attempt to describe what they are seeing by holding their hands next to the child's face in angulated fashion to make sure the orthodontist sees it, too. This observation is often not discernible on the models or on the cephalogram but is readily observable on the patient. So the oblique view of the smile reveals characteristics of the smile not obtainable through those means, and it aids the visualization of both incisor flare and occlusal plane orientation. A particular point for observation is the anteroposterior cant of the occlusal plane. In the most desirable orientation, the maxillary occlusal plane is consonant with the curvature of the lower lip on smile (the smile arc, discussed in detail in the section on smile evaluation). Deviations from this orientation that should be noted as potential problems include a downward cant of the posterior maxilla, an upward cant of the anterior maxilla, or variations of both. In the initial examination

and diagnostic phase of treatment, visualization of the occlusal plane in its relationship to the upper and the lower lip is important.

c. *Oblique close-up smile*. This allows a more precise evaluation of the lip relationships to the teeth and jaws than is possible using the full oblique view.

Figure 9-6 illustrates the close-up smiles on the oblique view of a child presenting for correction of an open bite and his mother.

3. *Profile* (Fig. 9-7): The profile photograph also should be taken in a natural head position. The most common method used for positioning the patient properly is to have the patient look in a mirror, orienting the head on the visual axis. The picture boundaries should emphasize the areas of information needed for documentation and diagnosis. The authors recommend that the inferior border of the image be slightly above the scapula, at the base of the neck. This position permits visualization of the contours of the chin and neck area. The superior border should be only slightly above the top of the head and the right border slightly ahead of the nasal tip.

The inclusion of more background simply adds unneeded information to the photograph. Some clinicians prefer that the left border stop just behind the ear, whereas others prefer a full head shot. Under any circumstance, the hair should be pulled behind the ear to permit visualization of the entire face.

Two profile images are useful (see Fig. 9-7):

a. *Profile at rest*. The lips should be relaxed. Lip strain is illustrated better in the frontal view, so a profile photograph with the lips strained in closure is unnecessary.

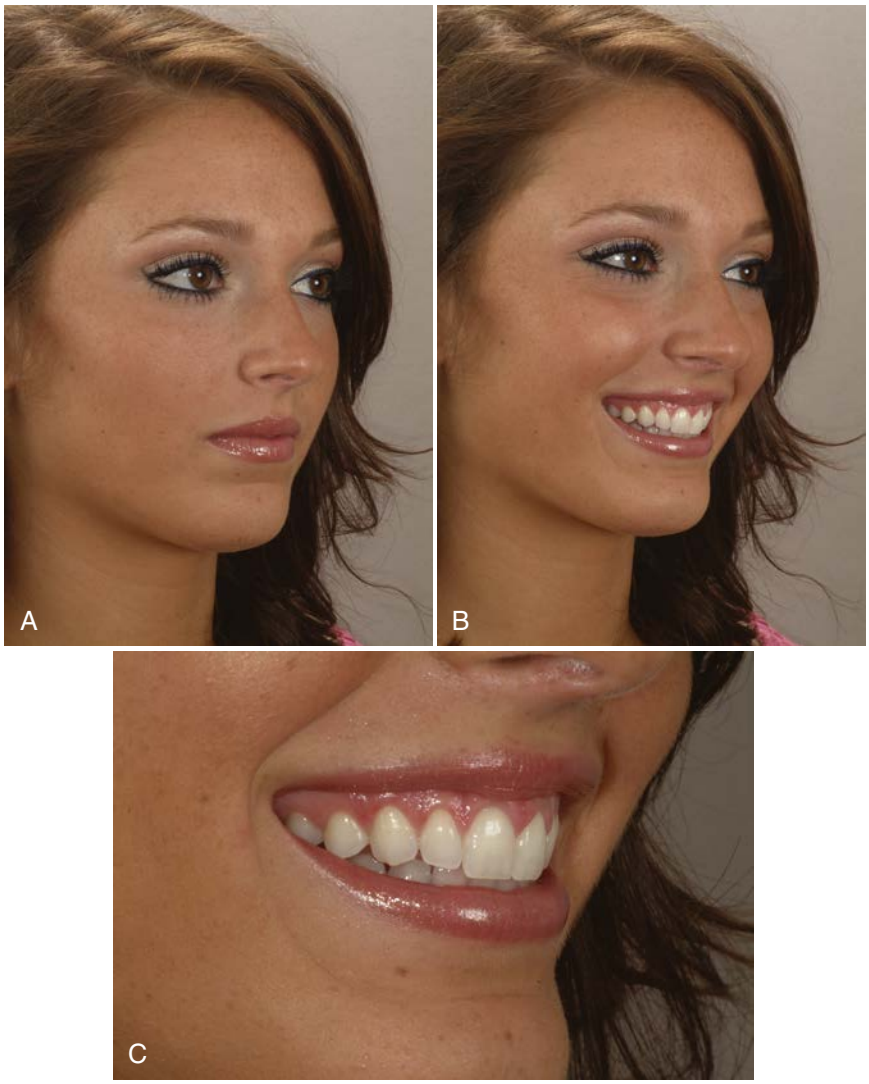


FIGURE 9-5 Oblique (three-quarter, 45-degree) views. Three views are useful. **A,** Oblique at rest. This view can be useful for examination of the midface and is particularly informative of midface deformities, including nasal deformity. This view also reveals anatomic characteristics that are difficult to quantify but are important aesthetic factors, such as the chin-neck area, the prominence of the gonial angle, and the length and definition of the border of the mandible. This view also permits focus on lip fullness and vermilion display. **B,** Oblique on smile. The oblique view of the smile reveals characteristics of the smile not obtainable on the frontal view and certainly not obtainable through any cephalometric analysis. **C,** Oblique close-up smile. This view allows a more precise evaluation of the lip relationships to the teeth and jaws than is possible using the full oblique view.

b. *Profile smile*. The profile-smile image allows one to see the angulation of the maxillary incisors, an important aesthetic factor that patients see clearly and orthodontists tend to miss because the inclination noted on cephalometric radiographs may not represent what one sees on direct examination.

4. *An optional submental view* (Fig. 9-8). Such a view may be taken to document mandibular asymmetry. In patients with asymmetries, submental views can be particularly revealing.

Digital Video Technology in Orthodontic Records

In standard photography, the image represents an approximate 1/125-second exposure of a patient's smile. In taking a picture for a smile, the subject is asked to smile and the picture is snapped. The problem in this process lies in the knowledge that the lips start the smile process at rest and animate over a very short time period. The image is taken somewhere in this dynamic process and may not represent the patient's consistent and repeatable smile. In practical terms, the image may be taken halfway into the smile or halfway to the end of the smile and may not represent the smile the patient uses consistently. Certainly almost all practicing clinicians have had the frustrating experience of looking at the patient's smile image during treatment planning and having the subject show a forced smile or one without the teeth showing (Fig. 9-9) (particularly 12-year-old boys!). The dynamic recording of smile and speech may be accomplished with digital videography. Digital video and computer technology currently enable the clinician to record anterior tooth display during speech and smiling at the equivalent

FIGURE 9-6 A, Close-up smile on oblique view represents a child presenting for correction of an open bite. B, The smile of his mother, who has an anteroposterior cant of the maxilla with the posterior maxilla clearly being inferiorly positioned and incomplete display of the anterior teeth. Note that the child has the same pattern.

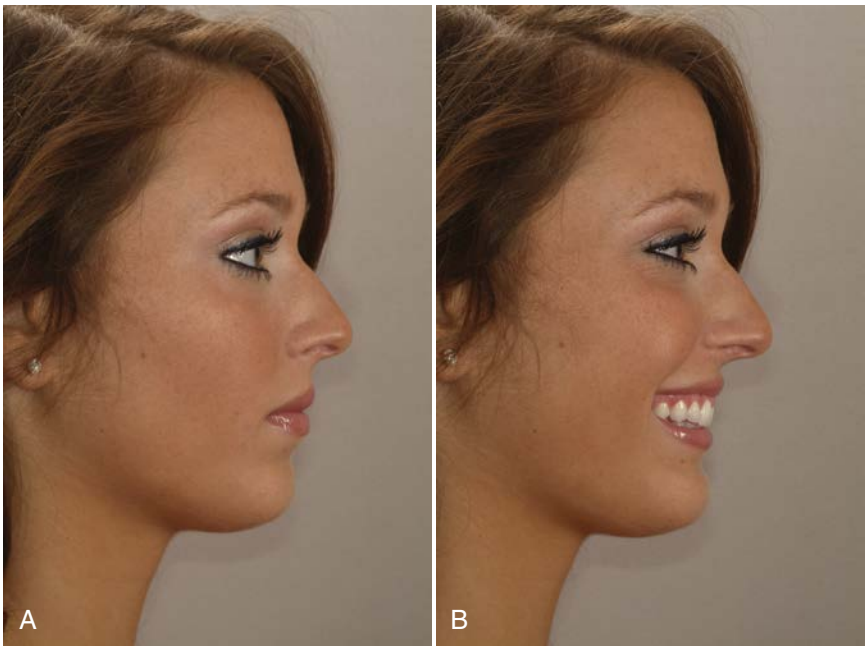


FIGURE 9-7 Two profile images are useful. A, Profile at rest with lips relaxed. B, Profile smile. This image provides a good view of maxillary incisor angulation and overjet in a way that patients see frequently, but clinicians often don't look at their patients' smiles in this orientation.

of 30 frames per second. With video clips of this type, one can review the video clip on a computer screen for repeated playback or set up a printout sequence that reflects the smile curve, which is a series of frames chosen to reflect the animation of the smile from start to finish (Fig. 9-10). This gives the clinician an opportunity to both visualize the smile from start to finish in dynamic viewing and see the individual frames in sequence to visualize the sustained smile—the smile with the most consistent lip incisor position during the smile.

FIGURE 9-8 An optional submental view may be taken to evaluate and document mandibular asymmetries further.

Intraoral Photographs

Standard intraoral photographic series consists of five views: right and left lateral, anterior, and upper and lower occlusal (Fig. 9-11). The occlusal photograph should be taken using a front surface mirror to permit a 90-degree view of the occlusal surface.

The orthodontist also can use specially designed mirrors that improve cheek retraction with the lateral views to obtain more direct views of buccal occlusion. To reflect buccal interdigitation accurately, as much cheek retraction as possible is needed, or one can use a mirror to gain a more perpendicular view. A 45-degree view from the front makes a Class II malocclusion appear to be Class I. Because occlusal relationships are captured more accurately on casts, mirror views of the lateral occlusion are usually not absolutely necessary.

The major purpose of the intraoral photographs is to enable the orthodontist to review the hard and soft tissue findings from the clinical examination during analysis of all the diagnostic data. What is surprising is how often one discovers something on the photographs that was overlooked at the time of oral examination. Another purpose of the intraoral photograph is to record hard and soft tissue conditions as they exist before treatment. Photographs that show white-spot lesions of the enamel, hyperplastic areas, and gingival clefts are essential to document that such preexisting conditions are not caused by any subsequent orthodontic treatment. The authors also recommend, in addition to the standard intraoral images, close-up images taken with a black background for microaesthetics (Fig. 9-12). This is a standard view for cosmetic dentistry and demonstrates tooth shape more clearly, as well as other aesthetic considerations such as color, zeniths, embrasures, connectors, etc.

FIGURE 9-9 The frustrating experience for the clinician is attempting to obtain a natural and repeatable smile. **A,** This patient obviously presents a forced and unnatural appearance. **B,** The same patient at the end of treatment with a more natural smile.

Cephalometric Radiographs

A lateral cephalometric radiograph is needed for diagnosis. Lateral cephalograms have two purposes: (1) they reveal details of skeletal and dental relationships that cannot be observed in other ways, and (2) they allow a precise evaluation of response to treatment. In many instances, an adequate orthodontic diagnosis can be made without a cephalometric radiograph; however, an accurate assessment of a patient's response to treatment is practically impossible without comparing cephalometric films before, during, and after treatment. For this reason, lateral cephalometric films are desirable but not always necessary. Many patients whose dental and skeletal relationships seem perfectly straightforward (e.g., Class I crowding problems) may not actually benefit from a cephalogram sufficiently to warrant the radiographic exposure involved. Treating skeletal malocclusions without cephalometric evaluation is a serious error.

The diagnostic value of lateral cephalometric films can be improved by taking the films with the patient in a natural head position rather than orienting the head to an anatomic plane as was done in the original cephalometric techniques.[2] Cephalometric head-holders fix the patient's head at three points: the external auditory canals bilaterally and the bridge of the nose or forehead. It is possible to obtain a natural head position cephalometric film, controlling both the source-to-subject and subject-to-film distances, without using a head holder at all.[5] Such films are made by having the patient orient the midline of the face (midsagittal plane) to the midsagittal plane of the head holder without actually attaching the head holder. For patients with growth deformities in which the ears are malpositioned, this may be the only feasible approach.

As a general guideline, differences between natural head position and Frankfort plane positioning are most likely to be encountered in patients with jaw discrepancies, so the further out the patient is on the deformity scale, the more important it is to use natural head position. In most patients, the difference between a natural head position film and an anatomically positioned one is in the vertical orientation of the head. The patient's head is positioned with the ear rods placed lightly into the ears, gently manipulated into a relaxed position as the patient looks into a mirror a few feet away or out a window at a distant horizon, and then fixed in the cephalostat. For greater diagnostic usefulness, the patient's lips should be relaxed rather than pulled into a strained closure when the cephalometric film is made. This step is particularly important when one contemplates a vertical repositioning of the teeth by orthodontic intrusion or extrusion or surgical repositioning of the segment. A number of the published soft tissue cephalometric analyses

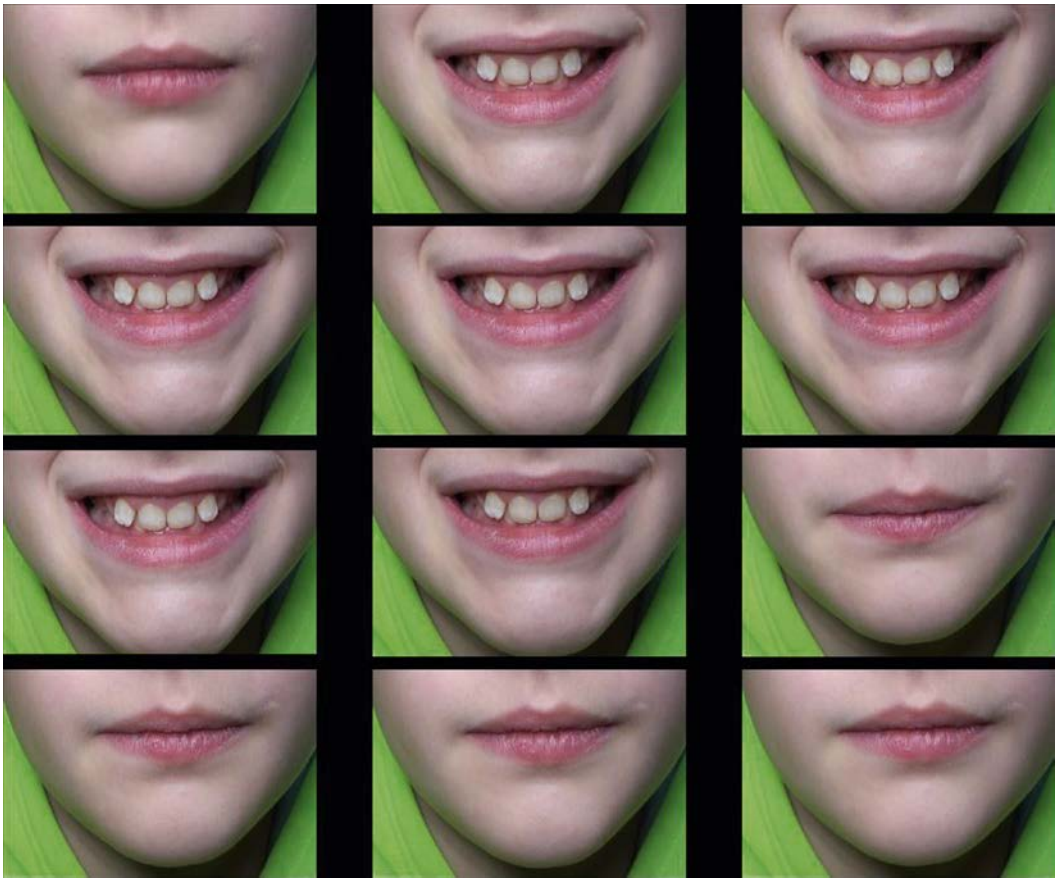


FIGURE 9-10 Digital video clips can be a useful adjunct in smile evaluation. Seeing the series of photographs allows the clinician to see the sustained smile, which we would accept as the posed smile. We currently use additional LED lighting rather than ambient light to give even lighting to eliminate an emphasis of shadowing and buccal corridors, which gives an unrealistic representation of hard and soft tissue relationships.

FIGURE 9-11 A, The right buccal dental photograph, mirror view. B, The left buccal dental photograph, mirror view. C, The frontal centered dental photograph, showing teeth and surrounding soft tissue and excluding retractors and lips. D, The maxillary occlusal dental photograph. E, The mandibular occlusal dental photograph.

FIGURE 9-12 Close-up image with black background for micro aesthetic evaluation. This view demonstrates microaesthetic characteristics such as tooth shape, color, zeniths, embrasures, and connectors.

have been derived from cephalograms with the subject's lips in closed position even if strained. These analyses neglect the vertical component and may present a distorted view of the anteroposterior lip position because of the lip strain factor.

The teeth should be held lightly together in centric (habitual) occlusion when the cephalogram is obtained. If a severe centric relation–centric occlusion discrepancy exists, obtaining a second film with the mandible retruded may be helpful. However, getting the patient into a true centric relation position in the cephalometric head holder can be difficult. For this reason, the centric relation film is of less diagnostic value than might be thought initially, and taking two lateral head films rarely is indicated.

Contemporary Applications of Cephalometry

The original purpose of cephalometrics was for research on growth patterns and the craniofacial complex, but

cephalometric radiographs came to be recognized as valuable tools in evaluating dentofacial proportions and clarifying the anatomic basis for malocclusion. Malocclusion is a result of an interaction between jaw and tooth position and the position the teeth assume as they erupt, which is in turn affected by the jaw relationships. For this reason, two apparently similar malocclusions as evaluated from dental casts may turn out to be different when evaluated more completely, using cephalometric analysis to reveal differences in dentofacial proportions.

Still another use for cephalometrics is to predict changes that should occur in the future for a patient. The result is an architectural plan or blueprint of orthodontic treatment called a *visualized treatment objective* (VTO). The accuracy of the prediction is a combination of the accuracy of predicting the effect of treatment procedures and the accuracy of predicting future growth. Unfortunately, growth predictions and predictions of the effects of treatment on growth remain relatively inaccurate, so a VTO for a growing child often is only a rough estimate of the actual outcome. Nevertheless, preparation of the VTO can be helpful in planning treatment for patients of any age with complex problems. The VTO has matured from its cephalometric origins with pencil tracings to today's incorporation of digital imaging and an expanded vision from the profile only to frontal and dental functional and aesthetic treatment. (Fig. 9-13).

In contemporary patient analysis, the cephalogram is used as an initial diagnostic tool and in the integration of the VTO concept into image projection. The emphasis in this chapter is on the extension of this methodology in modern treatment planning. In modern orthodontics, cephalometric findings no longer are the major determinant of treatment goals.

Computer Imaging in Contemporary Treatment Planning

Patients are keenly interested in knowing what they will look like after treatment both for the face and the smile. Although profile line drawings based on manipulation of cephalometric tracings may provide a reasonable feedback system for the orthodontist, they have little cognitive value to the patient. Computerized cephalometric programs streamline the laborious manual measurement of dimensions and angular relationships on patient cephalograms and have made it much easier to create and use facial VTOs. Before the use of computers in the treatment planning process, the VTO concept required the clinician to use acetate templates of the teeth and jaws to predict what treatment was needed to attain aesthetic and functional goals. Computer imaging technology now allows clinicians to modify facial images to project treatment goals accurately and discuss them with patients. The computer image is much easier for the patient to comprehend than is just the soft tissue profile of a cephalometric tracing. Computer imaging is the next step in the natural progression of the application of technology to orthodontic treatment planning.

For this purpose, integrated profile image–cephalometric planning offers several advantages:

1. Profile visualization for better comprehension of the facial response to the dental and soft tissue manipulation involved in a particular treatment plan.
2. Quantification of the planned dental and osseous movements to reduce the guesswork regarding the facial response to the proposed orthodontic treatment plan.
3. Evaluation of various treatment plans before deciding on the final plan, essentially image "mock-ups" for visualizations.

This is the essence of the concept of image-directed diagnosis because it allows clinicians, at least in adult or surgical cases, to determine beforehand the facial result of proposed treatment.

A VTO is mandatory in the development of a surgical orthodontic treatment plan in which growth effects are not a problem (see Fig. 9-13). Many surgeons are leery of providing imaging projections preoperatively out of the fear that it represents an implied warranty or that a lawsuit may result if the result does not match closely. Studies indicate just the opposite,[8] that patients are more satisfied with their final outcome when their presurgical counseling included imaging than those who did not have imaging. In addition, in one study all patients indicated that they actually liked the final result better than the image projection.[9]

Computer imaging also is useful in counseling and communicating with patients because it allows presentation of treatment options that are often difficult to explain verbally. The counseling phase involves the use of facial- or dental-image modification without any quantitative aspect to the process. This modification is a graphic way of communicating concepts that are difficult to present verbally. For example, facial imaging, smile image modification, or a smile bank can be used to explain to patients how their teeth will look after treatment. A sample of smiles with aligned teeth can be used to demonstrate to patients the changes they may expect in their dental appearance. This process, which is facilitated greatly by the use of computer images, improves the chance of true informed consent.

After the patient decides to proceed with treatment, the clinician must consider ways to maximize the chance of actually producing the outcome the patient desires. The planning phase of computer imaging permits quantification of the treatment plan so that the clinician knows precisely what and how much to do. The principle of quantification and retroengineering as described in the surgical VTO section is also important in smile design. Calibration of a smile image and the ability to measure the amount of change needed to reach smile goals are recent developments (Fig. 9-14).

CLINICAL EXAMINATION OF FACIAL SYMMETRY AND PROPORTION: ITS SIGNIFICANCE IN TREATMENT PLANNING

The clinical examination requires direct measurement of a number of hard and soft tissue relationships, most of which cannot be documented in any form of imaging or models. After the clinical examination data are entered into a database program, the data become as easily retrieved and used as any other digital record.

Application of Database Programs to Clinical Information

The goal of orthodontic diagnosis, as stated previously, is to determine the patient's chief complaint and give it the highest priority in designing treatment. Identification of the patient's problems and specifying potential solutions have been the major focuses of diagnostic efforts.

In contemporary diagnosis, however, orthodontists now include the concept of *treatment optimization*. The term

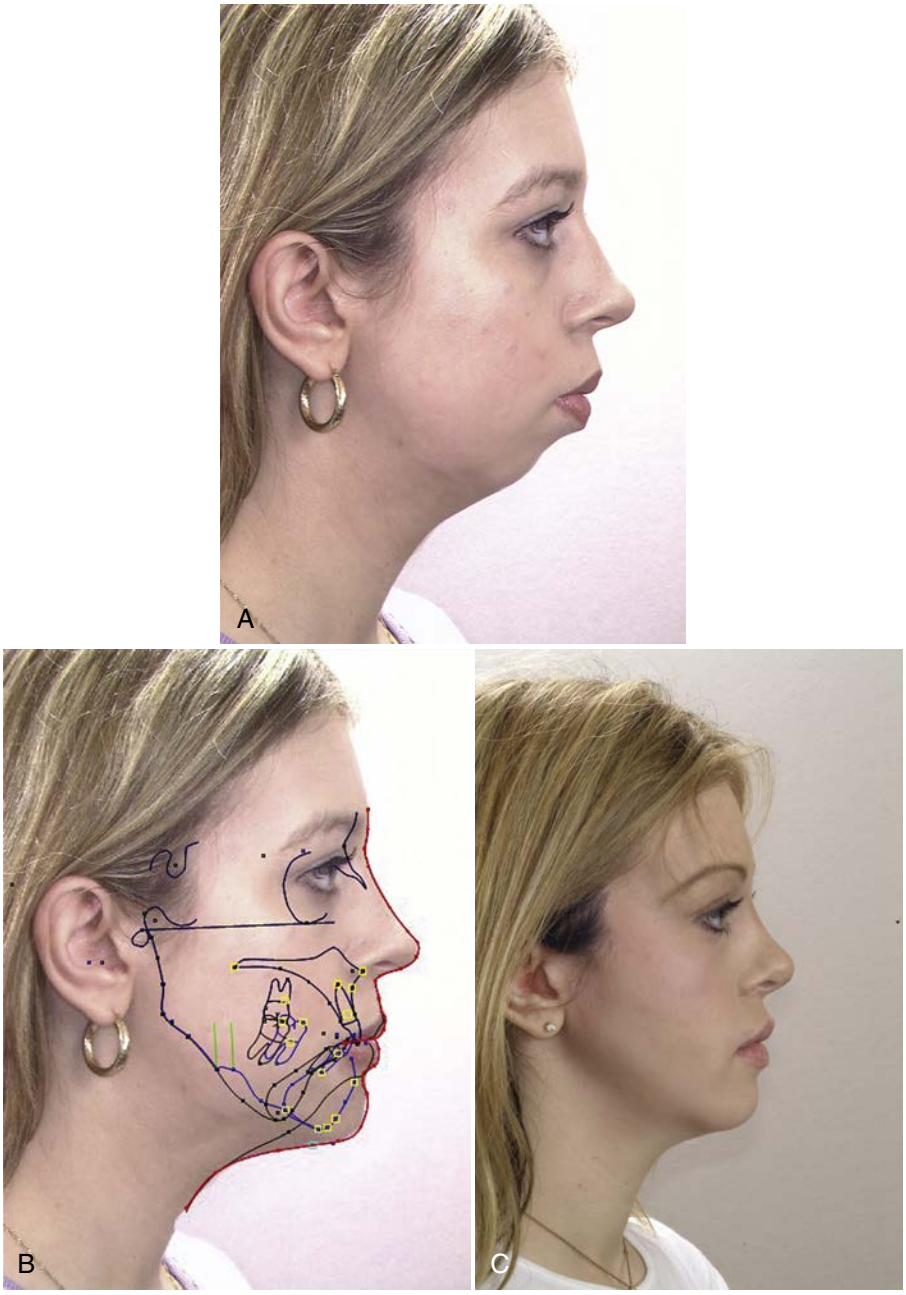


FIGURE 9-13 A, Initial profile image of patient being evaluated for extraction of lower premolars and advancement of the mandible and chin. B, Visualized treatment objective (VTO) of patient demonstrating the soft tissue outline anticipated with these orthodontic and surgical movements, and the amount of movement is retroengineered through a quantitative table reflecting the exact magnitude of movement. C, Final profile image reflects the outcome and its proximity to the VTO.

optimization has become familiar to most orthodontists through the increased use of computer technology. With computer hardware, it is wise periodically to "optimize the hard drive" on your computer to keep it running at its top performance level. What this does is scan the computer disc for damaged or corrupted files and eradicate them. The process also identifies good files and keeps them. Treatment plan optimization is similar. Optimization means that the orthodontist assesses the patient's problems and identifies ways to eliminate them and then identifies the patient's positive attributes and designs treatment to keep them. A familiar example in orthodontics is the correction of Class II malocclusion in a patient with mandibular deficiency and a normal upper face. Extraction of maxillary first premolars and retraction of the upper incisors solves the problem of the Class II malocclusion but ignores the positive attribute of a normal upper face, and the patient then has the normal upper lip retracted and flattened to fit the distorted lower jaw. The more appropriate treatment plan is advancement of the deficient mandible, correcting the Class II malocclusion and maintaining the normal upper face. In miniaesthetics, the smile arc is also an excellent example. If a consonant

FIGURE 9-14 **A,** This patient had an undersized maxillary right lateral incisor, which had been restored with bonding material and a congenitally missing maxillary left lateral incisor (replaced by a removable pontic). In preparation for final restoration, the gingival margins had been placed appropriately, and it was time to coordinate the transition from orthodontic care to restorative care. **B,** Her smile revealed incomplete maxillary incisor display and the placement of the gingival margins relative to the upper lip on smile and incisor heights. **C,** Utilizing the smile image and computer software, we were able to visually outline the treatment targets (the smile arc). Through software calibration of the image to the subject, we then were able to measure the distance of the existing incisal edges on smile to the smile target, thus aiding in the of the veneers to be fabricated in the laboratory. **D,** Utilizing computer software, the intraoral image was calibrated, the known measurements of incisor heights were noted (*in blue*), the projected target distances added to the incisal edges (*yellow*), and the final veneer sizes were calculated and noted in white. **E** and **F,** This coordination of care, and quantification of treatment goals, resulted in an outstanding outcome.

smile arc is not recognized at the beginning of treatment, studies indicate that one-third are flattened by orthodontic treatment.

The problem-oriented treatment-planning process is especially useful in this context because it demands a systematic and thorough approach to evaluation. The generation of the problem list then leads to the diagnosis, and treatment options for each problem lead to a logical treatment plan. In clinical practice, this methodical process may give way to expedience, and the clinician may fall back on techniques and treatment systems rather than individualized treatment plans carefully derived for each patient. The evolution of computer technology has facilitated this process so that the clinician can organize the information to be used in the decision-making process in a streamlined fashion without getting lost in the details.

Database programs work by setting up fields that represent sections of data entry. The data fields allow orthodontists to preload choices through the use of pop-up windows. The database program is designed to facilitate everyday functions in the problem-oriented diagnostic and treatment planning process:

1. *The information-gathering process is thorough but streamlined.* In the traditional examination, measurements are written down on a sheet of paper and then transcribed or dictated into a planning format. The database program facilitates this process by using the computer screen interface as the entry point (Fig. 9-15). Pop-up menus are used for entering data. As the computer cursor is placed on one of the data entry levels, a pop-up box with a number of choices appears, beginning with the most common choice at the top (most easily accessed for entry). As soon as a choice is identified and clicked with

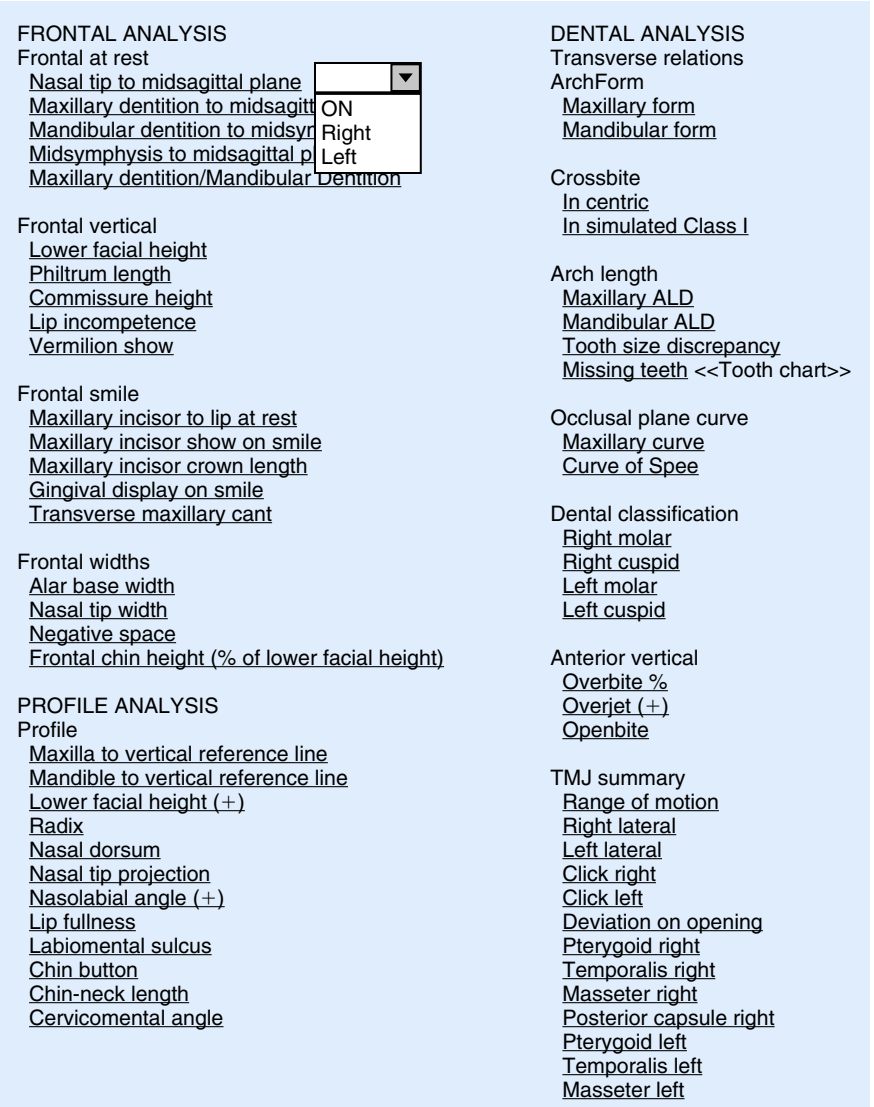


FIGURE 9-15 Database programs facilitate the data-gathering process by using the computer screen interface as the entry point. All components of the clinical examination are displayed on one screen, as in this screen shot, and as the clinician goes through the examination process and calls off each measurement, the data are entered through the use of pop-up menus. The pop-up menus allow data entry, and as each entry is made, the window closes and automatically opens the next for a smooth and rapid transition from one parameter to the next. This process facilitates a thorough examination and evaluation.

the mouse, the menu box closes and the next one is opened, complete with choices. In the clinical setting, this is usually the function of a staff auxiliary member, such as a treatment coordinator. For example, when the field "nasolabial angle," is clicked, a small window with the choices of "obtuse," "normal," and "acute" appears next to the nasolabial angle field, and the clinician can identify the choice by clicking it with a pointing device. After the selection is made, the program moves immediately to the next choice. This greatly streamlines the examination process and forces the clinician to measure each detail of the dentofacial analysis.

The first patient appraisal should be thorough and consistent, maximizing the chance that nothing of importance will be overlooked. Examiners should avoid situations in which they perform a cursory examination, jotting down notes regarding abnormalities they see and not really making note of any other descriptive data, normal or abnormal. Practitioners may assume that most diagnostic decisions can be made from the records they take of patients. Is that bad? Yes, it is, and for several reasons:

a. Static records cannot reflect the dynamic relationships that are important in the overall functional and aesthetic assessment of the patient. For example, the relationship of the upper incisor at rest and on smile to the upper lip is not reflected in radiographs or models and is evaluated poorly in photographs. As described previously in this chapter, dynamic (video) records help but still do not replace careful clinical observation.
b. The comprehensive facial and dental analysis process is streamlined and enhanced greatly by the database program.
c. The notation of normal observations is a powerful medicolegal aid. An unhappy patient may begin to "pick apart" the outcome, and the well-prepared practitioner should be able to document treatment changes with as many observations as possible.

Orthodontists currently enter this information into a computer in the examination room, but other options include the use of laptop and notebook computers. The use of personal digital assistants also is evolving. A handheld personal digital assistant permits even greater mobility, allowing data entry at chairside or any other location in the office or in auxiliary offices. Data may be entered with an electronic pen rather than a mouse and keyboard.

2. *The problem list is generated automatically through the use of predetermined parameters.* Each data entry is processed through the parameter field set by the clinician to test for its range of normality. For example, in the measurement of the maxillary midline to the midsagittal plane, the midline is coincident with the midsagittal plane or it is not. If the midline is not coincident, it is automatically a problem and is identified as such by the software and dropped into the problem list of the appropriate area (in this case, frontal facial examination). In more complex measurements, one may place ranges. For example, a mandibular plane measurement greater than 37 degrees is identified as a high mandibular plane angle; a measurement less than 32 degrees is identified as a low mandibular plane angle. Measurements may be changed by clinicians to fit their ranges because all doctors have their own parameters of what they consider problematic.
3. *Access to information is enhanced greatly in the traditional examination and treatment planning process.* After the clinical examination is finished, the observations then are transcribed into a more formal record system or left on the sheet to be retrieved from the chart when needed. This means that one of three things happens:
   a. If the information is left as is, it is not recalled easily during treatment because the information is not accessible. Also, the information is not transmitted easily to or shared with other doctors or with patients.
   b. The doctor writes the notes in a more organized fashion, which is time-consuming to complete.
   c. If the record is dictated, the doctor's time involvement is reduced significantly because dictation is much faster than handwriting. However, staff time then is needed to transcribe the dictation; therefore, elimination of dictation is desirable if possible.

Database programs require entry of information only one time, and this information can be retrieved and transported into any document, transported into spreadsheets for research purposes, or recalled for the clinician at any time on computer screens in the clinic, consultation area, or even at home.

EVALUATION OF FACIAL PROPORTIONS (MACROAESTHETICS)

Frontal Vertical Facial Relationships

Attractive faces tend to have common proportions and relationships that generally differ from normative values. Treatment planning in the past focused on linear and angular measurements, while the trend today is to recognize the interrelationships of proportions. The ideal face is divided vertically into equal thirds by horizontal lines adjacent to the hairline, the nasal base, and menton (Fig. 9-16). This figure also illustrates two other characteristics of the ideal lower third of the face: the upper lip makes up the upper third and the lower lip and chin comprise the lower two-thirds. What has become increasingly important is to recognize not just the vertical relations but also the relationship to the facial widths—the height-to-width ratio.

FIGURE 9-16 Frontal vertical thirds of the ideal female face with ideal symmetry. The vertical thirds should be roughly equal, with the lower third further subdivided into an upper third and lower two-thirds. In the adult, philtrum height should be equal roughly to commissure height.

Increased Face Height

The patient in Figure 9-17, *A* and *B*, is a good example of vertical maxillary excess (VME). In addition to the long upper third, this patient had a long lower face with excessive incisor at rest, excessive gingival display on smile, and interlabial gap—all of which are characteristics of VME. Surgical correction of the skeletal deformity was performed through superior repositioning of the maxilla, which also dramatically shortened the lower face (Fig. 9-17, *C*). The gummy smile also was improved with the procedure (Fig. 9-17, *D*), and an advancement genioplasty with vertical shortening contributed to the shortening of the lower facial third.

Does a patient with a long lower face always represent a VME problem? Of course not. Figure 9-18, *A*, presents an example of excessive vertical disproportionality of the lower facial height resulting from excessive chin height. Pretreatment facial analysis revealed an excessive lower facial height with normal upper facial thirds. An initial diagnosis of VME was considered, along with the possibility of surgical maxillary impaction. However, the patient had a normal smile line and a normal relationship between the resting upper lip and incisor, which is not associated with VME. The chin height from menton to the lower vermilion was significantly greater than the desired two-thirds of the total height of the lower facial third. This clinical assessment led to the conclusion that the vertical facial disproportionality resulted primarily from excessive chin height rather than VME. The treatment prescribed consisted of a wedge osteotomy and skeletal shortening of the chin to reduce the lower facial height (Fig. 9-18, *B*).

Decreased Face Height

Short lower facial height can result from vertical maxillary deficiency, mandibular deficiency with diminished mandibular body or ramus height, or a short chin height. Characteristics of vertical maxillary deficiency include (1) insufficient incisor

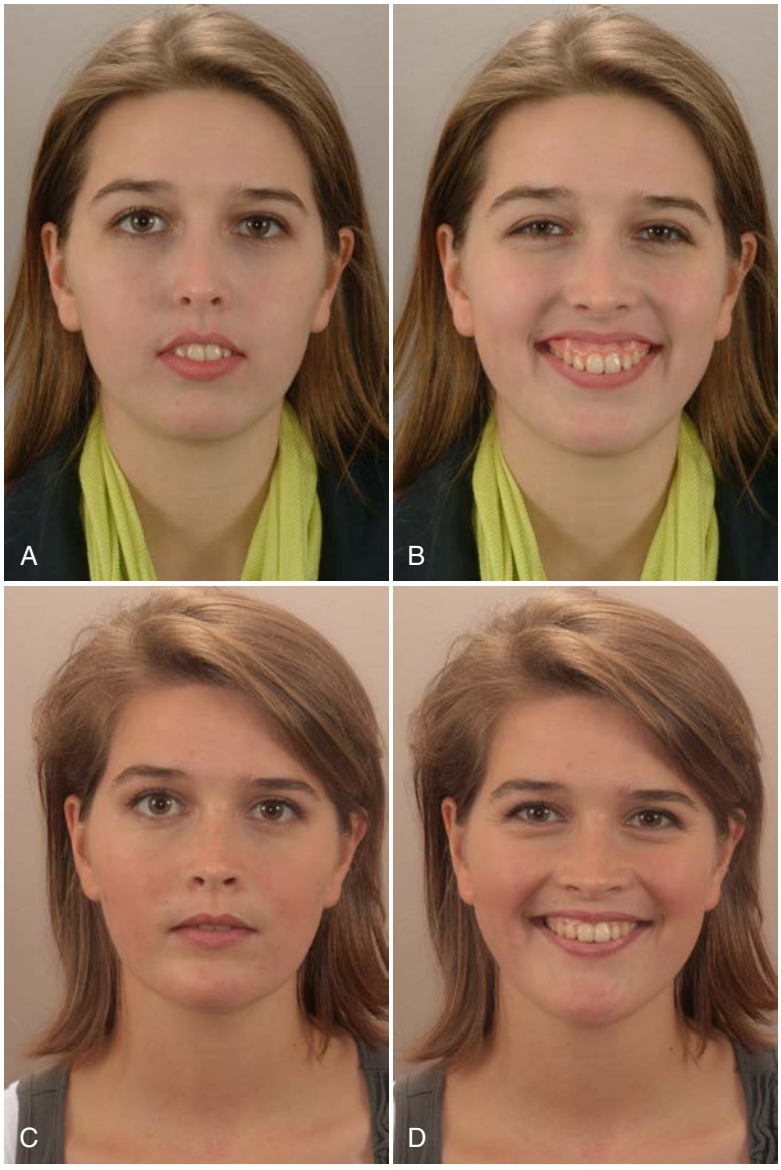


FIGURE 9-17 Pretreatment (**A**, **B**) and posttreatment (**C**, **D**) facial views of a patient with the characteristics of vertical maxillary excess: a long, lower facial height, excessive incisor display at rest, interlabial gap, and excessive gingival display on smile.

display at rest, (2) inadequate upper incisor display on smile, and (3) short lower facial height. Improvement in the adult often includes surgical maxillary downgraft. This surgical procedure lengthens the midface and rotates the chin down as the mandible hinges around the condylar axis.

The patient in Figure 9-19, *A* and *B*, presented with a disproportionately short lower facial third and mandibular deficiency. Clinically, he had diminished incisor display at rest, and on smile, characteristics of vertical maxillary deficiency. Dentally, he had a Class II malocclusion, a deep overbite, and 9 mm of overjet. Surgical correction was required, consisting of maxillary downgraft and mandibular advancement to correct his malocclusion, improve proportionality of his lower facial third, and increase incisor display at rest and on smile (Fig. 9-19, *C* and *D*).

Lower facial height increase in the adolescent may be improved through modification of growth, dental eruption, or both. Some examples of the ways this may be achieved, ranked in order of effectiveness, include the following:

1. *Functional growth modification appliances that increase lower facial height by promoting posterior dentoalveolar eruption*. Eruption occurs more rapidly in some patients than in others and is affected by the amount of freeway space, resting mandibular posture, and amount of wear. This type of treatment is most effective in younger patients in active phases of growth.
2. *Anterior bite plates incorporated into fixed appliances* (*such as a Nance-type appliance*) *or removable appliances*. Anterior bite plates hold the lower incisors against the acrylic while permitting the posterior teeth to erupt freely. The bite plate must be worn continually and also should be worn even after bite opening to maintain the increase in facial height and bite opening.
3. *Cervical headgear to encourage maxillary posterior eruption during growth modification*. Cervical headgear produces an extraoral force in a posterior and inferior direction below the center of resistance of the teeth and the maxilla, resulting in dentoskeletal extrusion and an increase in lower facial height.
4. *Eruptive wire mechanics designed to extrude the mandibular posterior segments*. A reverse curve of Spee commonly is placed in the lower archwire to open the bite through some lower incisor intrusion and a more substantial amount of posterior extrusion, resulting in an increase in the lower facial height (Fig. 9-20).

The analysis of the vertical thirds of the face is an initial barometer of the skeletal structures of the face and serves the clinician well by focusing on the face before the teeth, allowing observation of the gross proportionality of the face before the details are addressed.

Transverse Facial and Dental Proportions

The interrelationships of the widths of the components of the face are important in the overall proportionality of the face (Fig. 9-21). Few linear or angular "normative" measurements or values are available because the interrelationship of these component parts is what is most important. For example, a vertically long, oval face most often is correlated with narrow gonial angles and a narrow nose. A wide nose on a narrow face tends to appear most noticeable and incongruous with the facial type described.

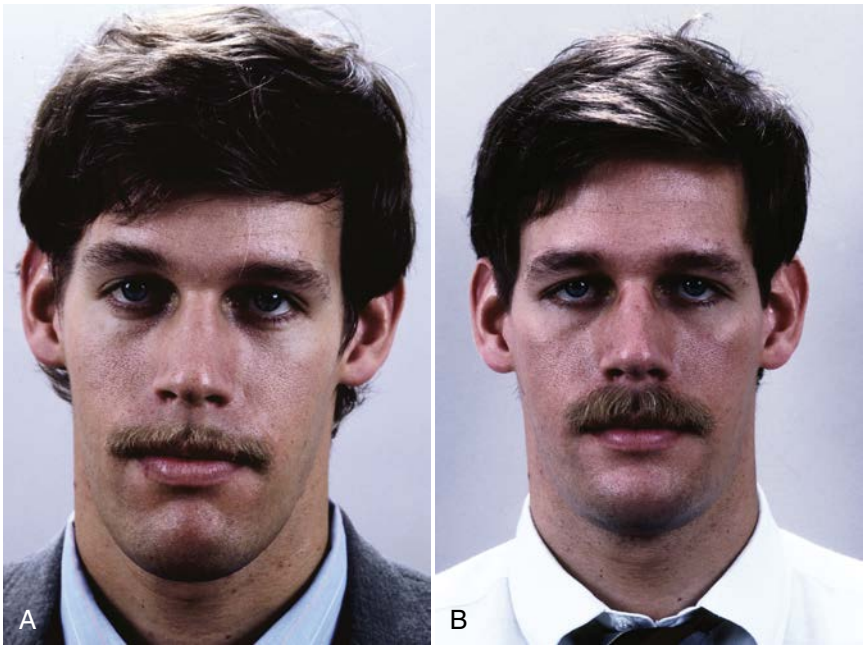


FIGURE 9-18 Before treatment (**A**), this patient had excessive lower facial height. The upper incisor-to-lip relationships were normal, which eliminated vertical maxillary excess as a potential cause of the long lower facial height. The chin height was 75% of the lower facial third, so more ideal facial proportions (**B**) were achieved with an inferior border osteotomy and removal of a wedge of bone above the chin to reduce chin height. (From Sarver DM. *Esthetic Orthodontics and Orthognathic Surgery*. St. Louis: Mosby; 1998.)

FIGURE 9-19 This adult patient presented with a short lower facial height, insufficient incisor display at rest and on smile, a deep bite, and a Class II malocclusion (**A** and **B**). A maxillary downgraft procedure was performed in addition to mandibular advancement surgery to achieve the desired increase in facial height (**C** and **D**).

Central Fifth of the Face

The central fifth of the face is delineated by the inner canthi of the eyes. The inner canthus of the eye is the inner corner of the eye containing the lacrimal duct. A vertical line from the inner canthus should be coincident with the ala of the base of the nose.

Medial Two-Fifths of the Face

A vertical line from the outer canthi of the eyes should be coincident with the gonial angles of the mandible. Disproportionality is a subtle clinical judgment, but procedures to augment or reduce the prominence of the lateral mandibular area are available to improve aesthetic problems here.

Outer Two-Fifths of the Face

The outer two-fifths of the face are measured from the base of the ear to the helix of the ear, which represents the width of the ears. Unless this abnormality is part of the chief complaint, prominent ears are often the most difficult abnormality to discuss with a patient because only in the most severe cases do laypersons recognize their effect on the face. Otoplastic surgical procedures are relatively atraumatic and can improve facial appearance dramatically. They may be recommended to and performed on adolescents and adults, but changing the hairstyle to conceal the ear may be a more practical solution.

FIGURE 9-20 The facial characteristics of this short face, deep bite patient direct the orthodontic mechanics for occlusal correction, facial improvement, and restoration of malformed teeth. **A,** Her frontal relationships were characterized by a short lower facial third and an overclosed facial appearance. **B,** On smile, she did not display all of her maxillary incisor due to mild vertical maxillary deficiency and tooth malformation. **C,** Her profile was characterized by moderate mandibular deficiency and an acute nasolabial angle. Her short lower facial height and overclosed appearance were also apparent on profile. **D,** After 24 months of treatment, her lower facial height was dramatically improved. **E,** The profile also reflected growth modification with improvement in mandibular projection and increase in lower facial height. Lower facial height increase was achieved with cervical headgear and posterior extrusive mandibular arch mechanics. **F,** After restoration of the malformed teeth, incisor display was dramatically improved.

FIGURE 9-21 Sagittal facial proportions: the rule of fifths. From the midsagittal plane, the ideal face is composed of equal fifths, all approximately equal to one-eye width. The commissure width should also be coincident with the medial limbus of the eyes, and the alar width should be coincident with the intercanthal distance.

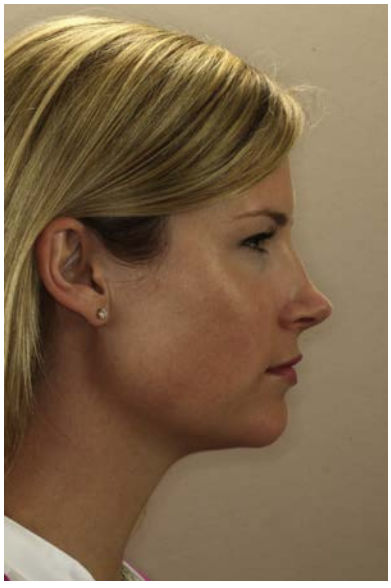


FIGURE 9-22 Profile of a patient with ideal nasal anatomy, illustrating the nasofrontal angle (*radix*), supratip break, double break of the nasal tip, and nasolabial angle.

Evaluation of Nasal Proportions

The nose dominates the middle portion of the face on profile (Fig. 9-22) but is an area that has not been emphasized in orthodontic training. A number of articles in the orthodontic literature have not received the attention from orthodontists they deserve (see the Suggested Readings at the end of the chapter). This is probably because many orthodontists are uncomfortable talking to patients about their noses and often soft tissue traits when the main focus of the patient and the orthodontist is the teeth.

Orthodontic treatment plans and mechanics can affect dramatically the way the nose fits the face. In addition, nasal growth in the adolescent can produce changes that diminish the aesthetic result as the patient matures. The orthodontist needs to understand nasal anatomy and the treatment of nasal deformities well enough to be comfortable with discussions of nasal morphology, recognition of aesthetic problems, and potential treatment.

Radix

On profile, the radix is the area orthodontists generally associate with soft tissue nasion. Radix projection is a discrete but important aspect of nasal aesthetics and is quantified in terms of the nasofrontal angle. Lack of radix projection can make an otherwise normal nose appear to have a dorsal hump.

Nasal Dorsum

One-third to one-half of the nasal dorsum is called the *bony dorsum* because it is formed by the confluence of the two nasal bones. The rest is called the *cartilaginous dorsum* or *septal dorsum* because it is composed of septal cartilage. The septal cartilage combines with the bony nasal septum to divide the nasal cavity into two chambers. On profile, the septal cartilage protrudes in front of the piriform aperture. Most nasal humps are formed by the dorsal border of the septal cartilage in combination with the nasal bones. The removal of an overprojected dorsal hump is one of the most common and familiar of rhinoplastic procedures.

Nasal Tip

The nasal tip is the most anterior point of the nose, and the supratip is just cephalic to the tip. The supratip break is the area just cephalic to the nasal tip where the lobule meets the dorsal portion of the nose. On the aesthetic nose, a slight depression is present on the supratip, which should be more pronounced in the female than in the male. The double break represents the angular formation of the nasal tip created by the distinct definition of the tip cartilages created by the supratip, tip, and infratip.

Columella

The columella is the portion of the nose between the base of the nose (subspinale) and the nasal tip. The columella comprises the cartilaginous nasal septum and membranous septum.

Nasolabial Angle

The nasolabial angle measures the inclination of the columella in relation to the upper lip. The angle should be in the range of 90 to 120 degrees.[1] The morphology of the nasolabial angle is a function of several anatomic features. Procumbency of the maxilla tends to produce an acute nasolabial angle, and maxillary retrusion tends to produce an obtuse nasolabial angle, but the angle is very much affected by nasal form itself.

Lip Projection

Although lip projection appears to be a fairly simple concept, it is a more complex issue in the comprehensive facial analysis in the contemporary hard and soft tissue approach to evaluation. Attempts have been made to quantify lip projection in the orthodontic literature by measurements such as the Rickett's E (aesthetic) line and Holdaway's line. These measurements are dentally oriented and are not facially comprehensive.

For example, it often is stated that in an ideal E-line relationship, the lower lip should be coincident with a line from the nasal tip to the anterior chin, and the upper lip should be about 1 mm behind it. If a patient has a long nose, the E-line describes the problem as dental or maxillomandibular retrusion rather than nasal overprojection.

Lip projection is a function of the following:

- *Lip thickness*: Lip thickness is affected directly by patient age, gender, and ethnicity.
- *Dental protrusion or retrusion*: Hard tissue support of the lips is a recognized determinant of lip position.
- *Maxillomandibular protrusion or retrusion*: Essentially affecting soft tissue support.

Excessive versus Inadequate Lip Projection

Lip projection is difficult to quantify because of its close interrelationship with other structures. Measurement of lip thickness is possible, and enough studies have been published in the literature to provide a sufficient database for this measurement. Lip thickness and its relation to other facial structures heavily influence the perception of lip projection. For example, in a patient with a deficient chin, the lower lip may appear full or procumbent. Advancement of the chin may result in better balance of the lower face and diminish the protrusive appearance of the lips.

The patient in Figure 9-23 is a good example of this interplay of anatomy. This young man was referred for correction of his mild Class II malocclusion. His parents thought that he had protrusive lips (see Fig. 9-23, *A*). His profile (see Fig. 9-23, *B*) confirms that relative to his chin and nose, the lips are more prominent than most orthodontists would consider ideal. A reasonable orthodontic plan would involve four-premolar extraction to create space for retraction of the anterior teeth and reduction of dental protrusion and lip fullness over approximately a 2-year period. A plastic surgeon or oral and maxillofacial surgeon might be inclined to define this patient's profile as chin deficient and recommend chin augmentation. We refer to this as "diagnosis by procedure"—that is, "What I do is what you need."

As the consultation proceeded, the maturational soft tissue changes were discussed (i.e., expected nasal growth and profile flattening). The consultation was visually facilitated through the use of digital image projections, and the parents elected to forego extractions in favor of 1 year of nonextraction treatment to be followed by an advancement genioplasty at the appropriate age (see Fig. 9-23, *C* to *F*).

Much emphasis is now given to nonextraction treatment, but in cases of dental protrusion and an excessively full profile, extraction treatment still has a significant role. The patient in Figure 9-24, *A* to *C*, presented with congenitally missing maxillary lateral incisors and a large maxillary diastema. Facially, she exhibited excessive lip fullness and protrusion. Rather than close the diastema to make space for placement of lateral incisor implants, the treatment plan was to extract the mandibular first premolars, close the space in the maxillary arch to treat her with cuspid substitution, and retract the lower incisors, resulting in protrusion reduction (Fig. 9-24, *D* to *F*).

Effects on the Labiomental Sulcus

The labiomental sulcus is defined simply as the fold of soft tissue between the lower lip and the chin; it may vary greatly in form and depth. The sulcus is affected by facial height, overjet, and chin projection.

Orthodontists commonly see the effect of decreased vertical dentoskeletal relations on the labiomental sulcus because many orthodontic patients have short faces and Class II malocclusions. An exaggerated but excellent analogy of the effect decreased vertical dimension has on chin position is the edentulous patient who has removed his dentures before closing his mouth. The vertical overclosure causes tremendous loss in the vertical dimension, resulting in soft tissue redundancy of the lips expressed as a deep labiomental sulcus.

The adolescent patient in Figure 9-25, *A* and *B*, started treatment with 6 mm of overjet and a 53% lower facial height. Orthodontic treatment included cervical headgear for its orthopedic effect on the lower face and Class II anteroposterior correction. With the downward vector of force application, the posterior maxilla and maxillary dentition are extruded, thus increasing the lower facial height. Eruptive lower archwire mechanics also were used with fixed appliances to extrude the mandibular posterior teeth and increase the lower facial height. The finished result Figure 9-25, *C* and *D*, reflects the longer, lower facial height and decrease in the depth of the labiomental fold achieved by growth and orthodontic and orthopedic treatment.

Chin Projection

Chin projection is determined by two factors: (1) the amount of anteroposterior bony projection of the anterior inferior border of the mandible and (2) the amount of soft tissue that overlays that bony projection. The combination of these two characteristics equals the total amount of chin projection.

NB-Pg is the cephalometric measurement most orthodontists refer to as "chin projection" and is quantified as the amount of bone projecting past the cephalometric NB line (a linear measurement in millimeters). Soft tissue thickness is also a variable in chin projection and has been studied extensively.[1]

Throat Form

Although throat form often is not considered in planning orthodontic treatment, it is an important aspect of facial aesthetics, and one must take into account the effect treatment has on it (Fig. 9-26). An obtuse cervicomental angle often reflects the following:

- *Chin deficiency*: Chin deficiency results in slackening of the submental and platysmal musculature, resulting in an obtuse angle.
- *Lower lip procumbency*: Lower lip procumbency results in lip projection, which simply increases the obtuseness of the lip–chin–throat angle.
- *Excessive submental fat*: Excessive submental fat contributes to the bulk of the neck, increasing the lip–chin–throat angle.
- *Retropositioned mandible*: A retropositioned mandible also results in slackening of the submental musculature and an obtuse angle.
- *Low hyoid bone position*: Low hyoid bone position contributes to the obtuseness of the lip–chin–throat angle through its mechanical location and the attachment of the submental musculature.

Miniaesthetics (Evaluation of the Smile)

Importance of the Smile in Orthodontics

The subject of the smile and facial animation as it relates to communication and expression of emotion should greatly interest orthodontists. Although the English language is replete with words such as *smirk, insipid smile, wry smile, sardonic*

FIGURE 9-23 Because lip prominence is evaluated relative to the chin and nose, orthodontic retraction of anterior teeth or advancement genioplasty may yield a similar result. This patient presented with protrusive lips (**A**), and the profile (**B**) reflected lip fullness with chin deficiency. The patient was offered the option of orthodontic treatment to retract the anterior teeth but chose advancement genioplasty with nonextraction orthodontic treatment. Frontal (**C**) and profile (**D**) relationships are shown after completion of orthodontic treatment. The frontal (**E**) and profile (**F**) changes are shown after advancement of the chin. This case illustrates the nature of differential treatment choices in aesthetic treatment selection.

FIGURE 9-24 A to C, This adolescent patient presented with missing maxillary lateral incisors, dental protrusion, and lip protrusion. D to F, Protrusion reduction with lower first premolar extractions improved the balance of the lips and chin.

FIGURE 9-25 A and B, This growing patient had a Class II malocclusion, deep labiomental sulcus caused by overjet, and diminished lower facial height resulting in lip redundancy. The recommended orthodontic treatment included cervical headgear and extrusive mechanics of the posterior teeth to reduce overjet while increasing lower facial height. C and D, The deep labiomental sulcus was improved through overjet reduction and increase in lower facial height.

smile, ironic smile, inscrutable smile, infectious smile, warm smile, and *enigmatic smile*, all of which conjure up specific images, these descriptions are entirely subjective. An attractive smile helps win elections, and a beautiful smile sells products for companies whose subliminal message in advertisement is "look better, feel younger." A well-treated orthodontic case in which plaster casts meet every criterion of the American Board of Orthodontics for successful treatment may not produce an aesthetic smile.

Interestingly, few objective criteria exist for assessing attributes of the smile, establishing lip–teeth relationship objectives of treatment, and measuring the outcomes of therapy. Without morphometric data for smile characteristics, orthodontists have no choice but to be entirely subjective in assessing smiles. Subjectivity can be reduced, however, by incorporating the measurements described below into the clinical examination.

Analysis of the Smile

The perception of dentofacial aesthetics by orthodontists and patients has differed considerably. A nonposed smile is involuntary (i.e., not obligatory) and is induced by joy or mirth. A smile is dynamic in the sense that it bursts forth but is not

FIGURE 9-26 A, This 19-year-old patient had mandibular and chin deficiency resulting in an obtuse chin–neck angle and full-throat form. B, On oblique view, she had poor definition of her jawline and mandibular angle. C, After surgical advancement of both jaws, combined with soft tissue submental liposuction, platysmal lift, and chin advancement, the throat form was greatly improved on profile. D, Her posttreatment oblique view demonstrates improvement in definition of her jawline and mandibular angle.

sustained. All the muscles of facial expression are recruited in the process, causing a pronounced deepening of the nasolabial folds and squinting of the eyes. A nonposed smile (Fig. 9-27, *A*) is natural in the sense that it expresses authentic human emotion. A posed smile (Fig. 9-27, *B*), by contrast, is voluntary and need not be elicited or accompanied by emotion. Such a smile can be a learned greeting, a signal of appeasement, or an attempt to indicate self-assurance.

A posed smile is static in the sense that it can be sustained. If the smile is typical for a particular individual, a posed smile is natural, but the smile also can be "forced" to mimic a nonposed smile. In the latter circumstance, the smile cannot be sustained and will seem to be strained and unnatural. In the Peck classification, a Stage II smile is a forced or strained posed smile resulting in maximal upper lip elevation. Thus, two types of posed smiles are possible: strained and unstrained. When a person is asked to pose for a photograph, the smile that is desired is a voluntary, unstrained, static, yet natural smile. Hulsey[9] and Rigsbee et al.[10] agree that reproducibility of the posed smile is good.

In a two-and-a-half-year period of observation during adolescence, the changes in smile characteristics in untreated patients and in patients undergoing orthodontic treatment were remarkably small. If the conventional thinking that lip–tooth relationships change over time is correct, these changes must occur gradually or much later in life. These changes are likely a part of aging, rather than part of growth and development.

FIGURE 9-27 A, The nonposed smile is natural in the sense that it expresses authentic human emotion. B, A posed smile is voluntary and is static in the sense that it can be sustained.

Diagnostic Smile Analysis: Measurement of Characteristics

Direct measurement permits the clinician to quantify the resting and dynamic tooth–lip relationships. Observation of the smile is a good start, but quantification of resting and dynamic tooth–lip relationships is critical to smile visualization so that the information gathered in the measurement of smile characteristics then can be translated into terms meaningful to the treatment plan. Systematic measurement of resting tooth–lip relationships and how the dynamics of the smile also interact with the maxillary teeth affect the appearance of the smile virtually lead the clinician to a quantified treatment plan.

The following frontal measurements at rest should be performed systematically:

- *Philtrum height* (Fig. 9-28, *A*). The philtrum height is measured in millimeters from subspinale (the base of the nose at the midline) to the most inferior portion of the upper lip on the vermilion tip beneath the philtral columns. The absolute linear measurement is not particularly important, but what is significant is its relationship to the upper incisor and the commissures of the mouth. The philtrum lengthens with maturation and aging and at a faster rate than the commissures.
- *Commissure height*. The commissure height (Fig. 9-28, *B*) is measured from a line constructed from the alar bases through the subspinale and then from the commissures perpendicular to this line. The difference between philtrum height and commissure height decreases from adolescence to adult life. In adults in whom philtrum height remains considerably shorter than commissure height, the effect is an unwitting frown, so that the individual tends to look angry all the time regardless of whether he or she is angry. This effect can be improved with surgical lengthening of the philtrum (Fig. 9-29).[11]
- *Interlabial gap*. The interlabial gap is the distance in millimeters between the upper and lower lips. An interlabial gap of greater than 4 mm is outside the normal range and is considered lip incompetence.
- *Amount of incisor display at rest* (Fig. 9-28, *C*). The amount of upper incisor display at rest is a critical aesthetic parameter because one of the inevitable characteristics of aging is diminished upper incisor display at rest and on smile. As patients get older, the amount of incisor at rest decreases with age, as does the amount of tooth display on smile. The clinician must be aware of this because the effect of diminishing incisor display results in hastening the aging process in terms of smile appearance. For this reason, in orthodontic treatment the choice to open a deep bite with upper incisor intrusion may correct the occlusion at the cost of making the patient look significantly older.

Measurements on smile are as follows:

- *Amount of incisor display on smile* (Fig. 9-28, *D*). On smile, patients will show either their entire upper incisor or only a percentage of the incisor or gingival display. Therefore, the number of millimeters of crown display on smile is recorded, and this may include the entire crown or, in cases of incomplete incisor display on smile, the amount of incisor shown.
- *Crown height and width*. The vertical height of the maxillary central incisors in the adult is measured in millimeters and is normally between 9 and 12 mm, with an average of 10.6 mm in men and 9.6 mm in women. The age of the patient is a factor in crown height because of the rate of apical migration in the adolescent. The width is a critical part of smile display in that the proportion of the teeth to each other is an important factor in the smile. Most references specify the central incisors to have about an 8:10 width-to-height ratio.
- *Gingival display*. The aesthetically acceptable amount of gingival display on smile varies, but one must always remember the relationship between gingival display and the amount of incisor shown at rest. In broad terms, treating a patient less aggressively in reducing smile gumminess is better when considering that the aging process will result in a natural diminishment of this characteristic. A gummy smile is often more aesthetic than a smile with diminished tooth display.
- *Smile arc*. The smile arc is defined as the relationship of the curvature of the incisal edges of the maxillary incisors and canines to the curvature of the lower lip in the posed social smile. The ideal smile arc has the maxillary incisal edge curvature parallel to the curvature of the lower lip on smile, and the term *consonant* is used to describe this parallel relationship (Fig. 9-28, *E*). Nonconsonant or flat smile arc is characterized by the maxillary incisal curvature being flatter than the curvature of the lower lip on smile. The smile arc relationship is not as quantitatively measurable as the other attributes, so the smile arc is noted merely as consonant, flat, or reversed.

In treating the smile, the social smile in most cases represents a repeatable smile. An important note, however, is that a "maturation" of the social smile may occur in many patients, and the smile may not be consistent from time to time in specific patients.

FIGURE 9-28 A, Philtrum height is measured from the base of the nose to the most inferior portion of the upper lip. B, Commissure height is measured from the alar base to the outer commissure of the lips. C, Incisor display at rest is an important measurement because it reflects the "relative age" of the patient. D, Incisor display and gingival display are recorded within the framework of the smile. In cases of incomplete incisor display on smile, the amount of incisor displayed is measured. In this same patient, crown height is also recorded because the entire crown is visible on smile. E, The smile arc, defined as the relationship of the curvature of the incisal edges of the maxillary teeth to the curvature of the lower lip, is evaluated in the posed social smile.

The author has chosen the social smile as the representation from which to analyze the smile in four dimensions: frontal, oblique, sagittal, and time-specific.

Vertical Characteristics of the Smile

The vertical characteristics of the smile are categorized broadly into two main features: those pertaining to incisor display and those pertaining to gingival display. The patient shows the entire tooth or does not and shows the gingiva or does not. Inadequate incisor display can be a combination of vertical maxillary deficiency, limited lip mobility, and short clinical-crown height. If short clinical-crown height is the primary contributor to the inadequate tooth display, one must differentiate between a lack of tooth eruption (which may take care of itself as a child gets older), gingival encroachment (treated with crown lengthening), and short incisors secondary to attrition, treated by restorative dentistry with laminates or composite buildups (Fig. 9-30).

Another feature of vertical smile characteristics is the relationship between the gingival margins of the maxillary incisors and the upper lip. The gingival margins of the canines should be coincident with the upper lip, and the lateral incisors should be positioned slightly inferior to the adjacent teeth (Fig. 9-31). That the gingival margins should be coincident with the upper lip in the social smile is generally accepted.

FIGURE 9-29 A, While the short philtrum in the adolescent is often a normal maturational characteristic, in the adult it's an unaesthetic feature. B, Soft tissue surgical procedures may be utilized to increased philtrum height.

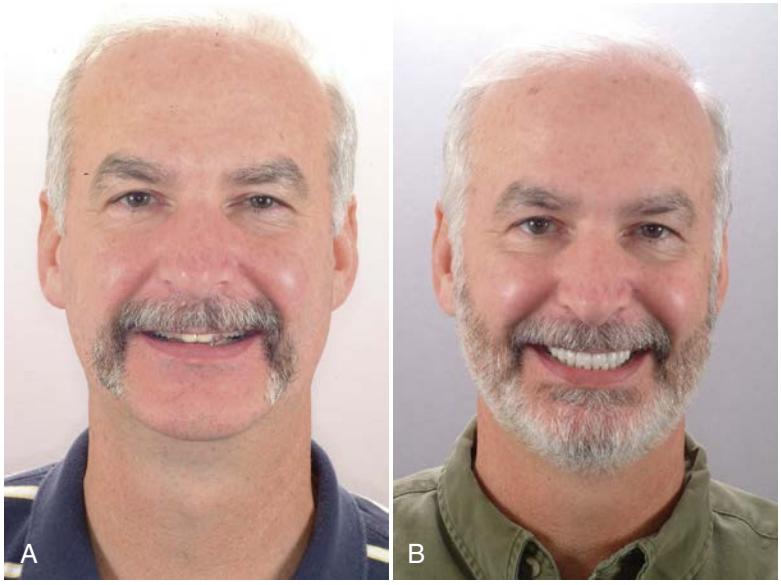


FIGURE 9-30 A flat smile arc can be caused by attrition of the maxillary incisors. A, This patient sought orthodontic consultation for smile improvement and was counseled that his problem was less of an orthodontic problem and more of a restorative problem. B, After laminate veneers were placed, incisal height and smile arc were restored.

However, this is very much a function of the age of the patient because children show more tooth at rest and gingival display on smile than do adults.

Transverse Characteristics of the Smile

Three important influences on the characteristics of the smile in the transverse plane of space are (1) buccal corridor width,[12] (2) arch form, and (3) transverse cant of the maxillary occlusal plane.

Buccal corridor width. This consideration was introduced into dentistry by the removable prosthodontics of the late 1950s. When setting denture teeth, prosthodontists sought to recreate a natural dental presentation transversely. A *molar-to-molar* smile was seen as fake and a tip-off to a poorly constructed denture. More recently, orthodontists have emphasized the diminished aesthetics of an excessively wide buccal corridor, often referred to as *negative space*. In orthodontics as in prosthodontics, the proportional relationship between the

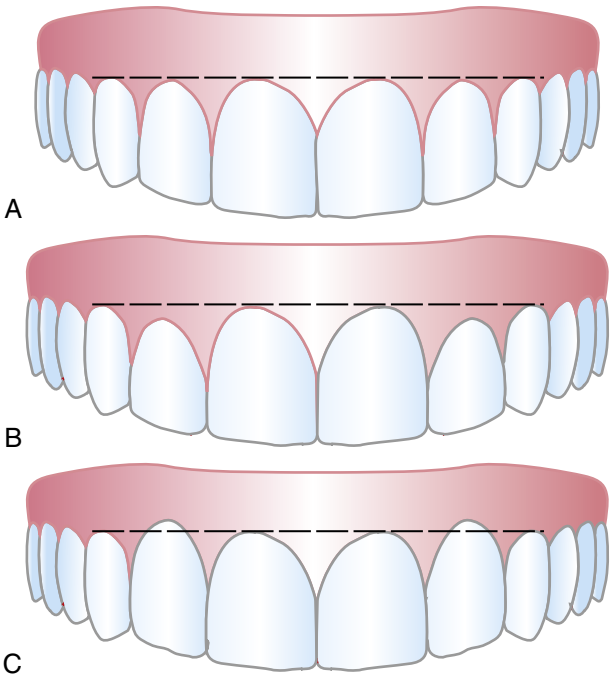


FIGURE 9-31 The gingival margins of the canines should be coincident with the upper lip, and the lateral incisors should be positioned slightly inferior to the adjacent teeth. A, Equal gingival height is acceptable. B, Ideal gingival height relationship. C, Least desirable gingival height relationship.

width of the dental arch and the width of the face must be kept in mind.

The buccal corridor is measured from the mesial line angle of the maxillary first premolars to the interior portion of the commissure of the lips. The corridor often is represented by a ratio of the intercommissure width divided by the distance from first premolar to first premolar.

Arch form. Arch form plays a pivotal role in the transverse dimension of the smile. In patients whose arch forms are narrow or collapsed, the smile also may appear narrow, which is less appealing aesthetically. An important consideration in widening a narrow arch form, particularly in the adult, is the axial inclination of the buccal segments. Patients in whom the posterior teeth are already flared laterally are not good candidates for dental expansion. Patients in whom the premolars and molars are upright have more capacity for transverse expansion in adolescence, but the characteristic is particularly important in the adult in whom sutural expansion is less likely.

Orthodontic expansion and widening of collapsed arch form can improve the appearance of the smile dramatically by decreasing the size of the buccal corridors and improving the transverse smile dimension (Fig. 9-32). The transverse smile dimension (and the buccal corridor width) is related to the lateral projection of the premolars and the molars into the buccal corridors. The wider the arch form in the premolar area, the greater is the amount of the buccal corridor that is filled.

Expansion of the arch form may fill out the transverse dimension of the smile, but two undesirable side effects may result, and one should take care to avoid them. First, excessive expansion obliterates the buccal corridor. Prosthodontists emphasize that this is unaesthetic. Second, when the anterior sweep of the maxillary arch is broadened (Fig. 9-33), the prominence of the incisors relative to the canines is likely to decrease. When these undesirable aspects of expansion are being considered, the clinician must make a judgment in concert with the patient as to what tradeoffs are acceptable in the pursuit of the ideal smile.

When the maxilla is retrusive, the wider portion of the dental arch is positioned more posteriorly relative to the anterior oral commissure. This creates the illusion of greater buccal corridor in the frontal dimension. The patient in Figure 9-34 had a Class III malocclusion caused primarily by maxillary deficiency, vertically (characterized by only 50% of maxillary incisor show on smile) and anteroposteriorly (as evidenced by the flatness of the profile). After orthodontic decompensation, the surgical plan was to advance the maxilla, rotating it clockwise to increase the amount of incisor display at rest and on smile. This occlusal plane rotation not only improves the incisal display but also increases midfacial projection and diminishes mandibular projection. The smile was enhanced greatly by the increased vertical anterior tooth display, but the transverse smile dimension also was improved greatly (see Fig. 9-34, *B*). How was the negative space on smile reduced when there was no maxillary expansion? As the maxilla came forward into the buccal corridor, the negative space was reduced by the wider portion of the maxilla coming forward into the static intercommissure width. Transverse smile dimension, therefore, is a function of arch width and anteroposterior position of the maxillary and mandibular arches.

Transverse cant. The last transverse characteristic of the smile is the transverse cant of the maxillary occlusal plane. A canted or asymmetric smile can be a result of (1) asymmetric vertical growth of the mandible resulting in a compensatory cant to the maxilla, (2) lip curtain asymmetry, or (3) differential gingival heights. A true transverse cant usually is related to asymmetric vertical growth of the mandible, resulting in a compensatory cant to the maxilla and, if present, may be an indication for orthognathic surgery. The appearance of a transverse cant, however, can result from differential eruption and placement of the anterior teeth or differential anterior crown heights requiring soft tissue modification, both of which should be considered in planning orthodontic treatment. Neither intraoral images nor mounted dental casts adequately reflect the relationship of the maxilla to the smile (Fig. 9-35). Only frontal smile visualization permits the orthodontist to visualize any tooth-related asymmetry transversely. The frontal smile photograph (see Fig. 9-35, *B*), not a frontal view of the teeth achieved with a lip retractor, is needed to record what is seen clinically. With good documentation of tooth–lip relationships, the orthodontist subsequently can make any appropriate adaptations in appliance placement or make a decision on the need for differential growth or dental eruption modification of the maxilla in the adolescent or surgical correction in the adult.

Smile asymmetry also may be due to asymmetric lip animation. A differential elevation of the upper lip during smile gives the illusion of a transverse cant to the maxilla. This characteristic emphasizes the importance of direct clinical examination of the smile because this soft tissue animation is documented poorly in static photographic images but is documented best in digital video clips. This can become an important informed consent issue if the patient is concerned about the asymmetry on animation because neither orthodontic tooth movement nor orthognathic surgery will affect it.

FIGURE 9-32 A, This adolescent patient has excessive buccal corridor width or negative space on smile. B and C, The intraoral views demonstrate the transverse deficiency of the maxilla. D, After orthodontic correction of the malocclusion, including orthodontic expansion, the transverse smile dimension is dramatically improved with projection of the teeth into the buccal corridor (E).

FIGURE 9-33 When the anterior sweep of the maxillary arch is broadened, the prominence of the incisors relative to the canines is likely to decrease, and flattening of the smile arc may occur. The clinician must make a judgment in concert with the patient as to what tradeoffs are acceptable in the pursuit of the best possible smile.

FIGURE 9-34 This patient exhibited a Class III malocclusion primarily caused by maxillary deficiency. **A,** Some aspects of vertical maxillary deficiency are present, including 50% of maxillary incisor display on smile. Her transverse smile dimension was characterized by excessive buccal corridors. **B,** Surgical maxillary advancement improved the buccal corridors on smile by bringing a wider portion of the maxilla forward into the buccal corridors, and bringing the maxilla down anteriorly resulted in increased incisor display.

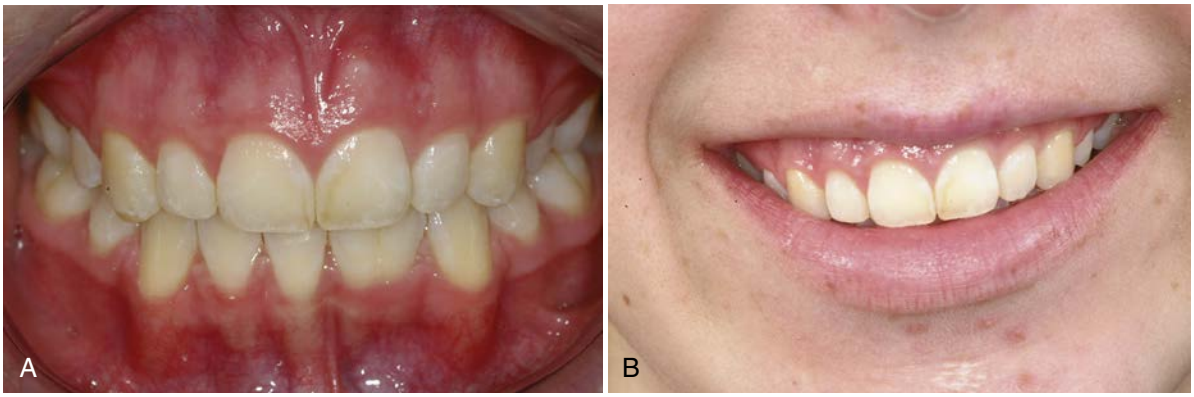


FIGURE 9-35 Neither intraoral images nor mounted casts adequately reflect the relationship of the incisors to the smile. In a close-up intraoral image (**A**), one sees an apparently well-treated occlusion, while a more distant smile view (**B**) reveals the same occlusal relationships with an obvious cant to the maxilla.

FIGURE 9-36 The close-up of the oblique smile, as in this patient in the last stages of preparation for orthognathic surgery to correct severe open bite, facilitates evaluation of the curvature of the molars (when visible), premolars, and anterior teeth in relation to the lower lip on smile. This also enhances closer evaluation of any anteroposterior cant to the palatal and occlusal planes.

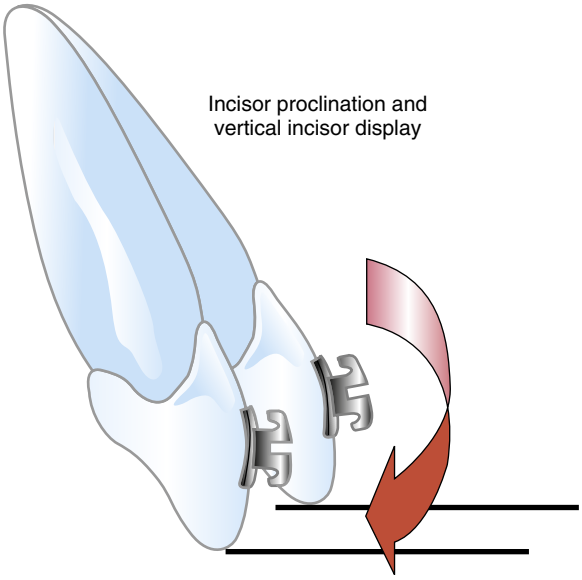


FIGURE 9-37 The amount of proclination of the maxillary incisors can affect how much they are displayed at rest and on smile. In general terms, flared maxillary incisors tend to reduce incisor display, and upright maxillary incisors tend to increase incisor display.

Oblique Characteristics of the Smile

The oblique view of the smile reveals characteristics not obtainable on the frontal view and certainly not obtainable through any cephalometric analysis. The contour of the maxillary occlusal plane from premolar to premolar should be consonant with the curvature of the lower lip on smile (a view of the smile arc, discussed previously). Deviations include a downward cant of the posterior maxilla, upward cant of the anterior maxilla, or variations of both. Figure 9-36 illustrates a patient in preparation for maxillary surgery to close an anterior open bite. Deciding how much the posterior maxilla should be impacted versus the anterior maxilla coming down depends on the amount of incisor display at rest and on smile and on the smile arc relationship, both of which are well visualized in the oblique view.

The amount of incisor proclination also can have dramatic effects on incisor display. In simple terms, flared maxillary incisors tend to reduce incisor display, and upright maxillary incisors tend to increase incisor display (Fig. 9-37). A good example is the patient in Figure 9-38. This patient had an anterior open bite caused primarily by extreme anterior proclination of the maxillary and mandibular incisors. The sagittal view of the smile shows the flare of the maxillary incisors, which resulted in diminished incisor show from the frontal view. The treatment plan involved interproximal reduction of the incisors to provide space for retraction and uprighting of the incisors. The incisors were retracted in such a way to allow the crowns to tip distally so that they also elongated toward the occlusal plane. This movement closed the anterior open bite, increased incisor display as the teeth, and decreased the unaesthetic flare of the incisors.

Dental Microaesthetics and Its Applications to the Smile

Gingival Shape and Contour

Important factors in finishing orthodontics cases for optimal smile aesthetics now include concepts that are important in cosmetic dentistry: gingival shape and contours, tooth proportionality, and crown heights.[13] Most orthodontists have reshaped incisal edges and tooth contours to refine the finish of treatment, but alteration of the gingival contours has been limited primarily to crown lengthening. Of the many concepts from cosmetic dentistry that are important to the microaesthetic smile appearance, two that are important for the final aesthetic outcome of orthodontic cases are gingival shape and gingival contour.[14]

According to the American Academy of Cosmetic Dentistry,[15] "the gingival shape of the mandibular incisors and the maxillary laterals should exhibit a symmetrical half-oval or

FIGURE 9-38 A, The frontal smile relationship demonstrates diminished incisor display on smile. B, On sagittal smile, the flare and proclination of the upper incisors are apparent. C, Before treatment, a mild anterior open bite was present, and the flare of the maxillary incisors contributes to the open bite and the incomplete incisor display on smile. After interproximal reduction and retraction and uprighting of the maxillary incisors, the amount of incisor display increased.

FIGURE 9-38, cont'd D, Interproximal incisor reshaping and retraction of incisors result in uprighting of the anterior teeth, reduced flare, and increase incisor display on smile. E, The saggital smile demonstrates how uprighting of the maxillary incisor improves the smile arc relationship as well. F, This uprighting also results in closure of the anterior open bite.

FIGURE 9-39 **A,** This 12-year-old, prior to treatment, had a nice smile with consonant smile arc but short incisal crown height, **B,** The close-up smile demonstrates the short crown height and slight gingival display on smile. **C,** Her initial intraoral image reflects the diminished crown height. In order to protect her already ideal smile arc, her brackets were place slightly closer to the gingival margin than standard protocol. **D,** Nearing completion of treatment, while the smile arc was ideal, the gingival hypertrophy secondary to oral hygiene issues (possibly exacerbated by the bracket proximity interfering with ideal oral hygiene technique) created periodontal pseudopocketing as well as unaesthetic gingival display and form. **E,** The finishing close-up smile demonstrates the incisal edge placement and the gingival irregularities. **F,** Upon bracket removal, hypertrophy of the papillae and marginal gingival tissues were noted, resulting in poor gingival shape and contour as well as diminished crown height. Upon reflection, in a more contemporary approach, we might have considered crown lengthening prior to bracket placement in order to avoid this unfortunate sequel of orthodontic appliance treatment. **G**, Often brackets are removed, hoping a visit to the hygienist and time would resolve the tissues back to their ideal condition. In our experience, this often does not happen since the tissue is fibrotic and is hard to clean. In today's world, parents are often beyond disappointed in the result. Either an immediate appointment with the periodontist is indicated, or as in this case, a diode laser is employed upon bracket removal to recover an improved condition.

FIGURE 9-39, cont'd H, The aesthetic benefit is immediate, as is the elimination of pseudopockets and inflamed, hypertrophic tissue. Healing is promoted because of the tissue sterilization characteristics of the laser procedure as well. I, One week later, the inflamed gingival tissues were resolved and a more aesthetic and healthy soft tissue resulted. J, The final frontal smile. K, The final close-up smile.

half-circular shape. The maxillary centrals and canines should exhibit a gingival shape that is more elliptical. Thus, the gingival zenith (the most apical point of the gingival tissue) is located distal to the longitudinal axis of the maxillary centrals and canines. The gingival zenith of the maxillary laterals and mandibular incisors should coincide with their longitudinal axis" (see Fig. 9-31).

Recontouring gingival shape and contour now can be accomplished readily, in the orthodontist's office if desired, with a diode laser.[3]

The patient in Figure 9-39, *A*, chose to pursue orthodontic treatment at age 12 for correction of some spacing and slight occlusal discrepancies. Her close-up smile assessment (Fig. 9-39, *B*) showed an ideal smile arc with complete incisor display but had slightly short crowns due to incomplete passive eruption and had slightly more gingival display than ideal. Her deep overbite was shown in her intraoral image (Fig. 9-39, *C*), as was the short height/width ratio of the anterior teeth with some enlargement to the interdental papillae. Assessing the progress of treatment after 9 months of full-fixed appliances (Fig. 9-39, *D* and *E*), we noted that the vertical incisor position had been protected as planned, but she had an excessively gummy smile. This aesthetic shortfall was secondary to the gingival inflammation unfortunately seen in many orthodontic cases. The intraoral image (Fig. 9-39, *F*) permits a more detailed microaesthetic evaluation. Gross gingival inflammation was apparent, but gingival height was disparate, the gingival papilla proportions were excessively large, and the zeniths individually varied. When faced with this situation, the frequent orthodontic action is to remove appliances and immediately refer for cleaning and prophylaxis, followed by observation for soft tissue resolution. This may occur over time, but often the tissues are fibrotic and do not recover as readily as we might wish, and a referral to the periodontist is indicated. While controlling hygiene treatment would have been the ideal approach, this is not always possible in spite of all the promises the patient or parents may give. The periodontal procedure is then met with resistance because of fear of pain, expense, and further delay of the desired aesthetic outcome. In this case, we probed the pocket depths for assessment of removal of the excessive gingival tissues and immediately upon removal of appliances performed simple gingivectomy with a diode laser (Fig. 9-39, *G*). This removed

FIGURE 9-40 A, This patient's smile line was asymmetric because of differential crown heights. B, Three weeks after soft tissue contouring and lengthening of the right central and lateral incisors, the smile was more symmetric and greatly improved.

the excess tissue, cauterizing and sterilizing the ablated wound margin (promoting healing), resulting rapidly in the final outcome (Fig. 9-39, *H* and *J*).

When finishing the anterior aesthetic relationship within the smile framework, the average location of the gingival margins should demonstrate a symmetric level of the margins of the central incisors and a lower location of the lateral incisors and gain a higher and ideally symmetric level on the canines. The patient in Figure 9-40, *A*, was treated to an excellent occlusal and a good aesthetic result. However, the smile line was asymmetric because of differential crown heights of the maxillary right central and lateral incisors, relative to the left side. Using the diode laser, the excess gingiva was excised on the right central and lateral. Three weeks later, the smile was more symmetric and greatly improved (Fig. 9-40, *B*).

Bracket Placement in Preparation for Changes in Gingival Shape

When a dentist is preparing teeth for laminates, it is not uncommon to reshape and idealize the gingival heights and contours with a soft tissue laser before final preparations are made and impressions are taken. In orthodontic treatment, because orthodontists usually are not able to make contour or shape adjustments, their recognition factors are low relative to these problems. Orthodontists must be able to visualize the crown in ideal proportion before bracket placement and in many cases shape and contour the gingiva prior to bracket placement.

The patient in Figure 9-41, *A*, has a disproportionate width-to-height ratio of the maxillary incisors. Most aesthetic dentists strive for a central incisor width-to-height proportion of 8:10, and this patient's incisors have the same width and height. This disproportion could be due to lack of incisor height (gingival encroachment or delayed or incomplete passive eruption requiring gingival reshaping) or incisors that are morphologically wider than ideal in terms of crown shape itself (requiring reshaping of the crown). Bracket placement in orthodontics has traditionally been directed by the relationship of the bracket slot to the incisal edge. Anterior teeth vary in crown height and incisal edge shape. The two most common current methods for placing brackets are to (1) relate the bracket position to the incisal edge or (2) position the bracket in the center of the clinical crown, whatever its dimensions. In this patient, positioning of the bracket a prescribed 4.5 mm from the incisal edge would place the bracket too close to the gingival margin, potentially causing gingival overgrowth and oral hygiene problems. Positioning of the bracket in the center of the clinical crown (Fig. 9-41, *B*) would cause unwanted maxillary incisor intrusion and a reduction in incisor display on smile. In this case, the decision was to improve tooth proportion through laser crown lengthening before bracket placement to maximize the chance of positioning the incisors in their ideal vertical position (Fig. 9-41, *C*).

Bracket positioning for optimal aesthetics. As orthodontists diagnostically move away from the procrustean approach to diagnosis and treatment planning (every patient gets fitted to the same cephalometric analysis, and all patients have their brackets placed the same distance from the incisal edge or cusp tip, according to a chart), concepts of bracket placement also are evolving. Rather than bracket placement being a function of the dental-centric preadjusted appliance demands, placement of the maxillary and mandibular anterior teeth should be directed by concerns such as the following:

1. How much maxillary incisor is displayed at rest (recall the data on incisor display at rest and the aging smile)?
2. How much maxillary incisor is displayed on smile (also a characteristic of the youthfulness of the smile)?
3. The relationship of the anterior teeth to the smile arc.
4. Crown height and width incisor proportionality.
5. Gingival height and contour characteristics.

What is the process to determine the desired bracket placement for each individual patient? As in the macroaesthetic and miniaesthetic treatment goal setting, a systematic approach to these microaesthetic considerations is needed. The authors recommended the following sequence of measurements to be made on the initial examination, recorded in the database program. To determine optimal bracket placement:

1. Determine the incisor display at rest (Fig. 9-42, *A*):
   a. Refer to the chart of incisor display related to age for a guideline as to appropriate incisor display at rest (Fig. 9-42, *B*)

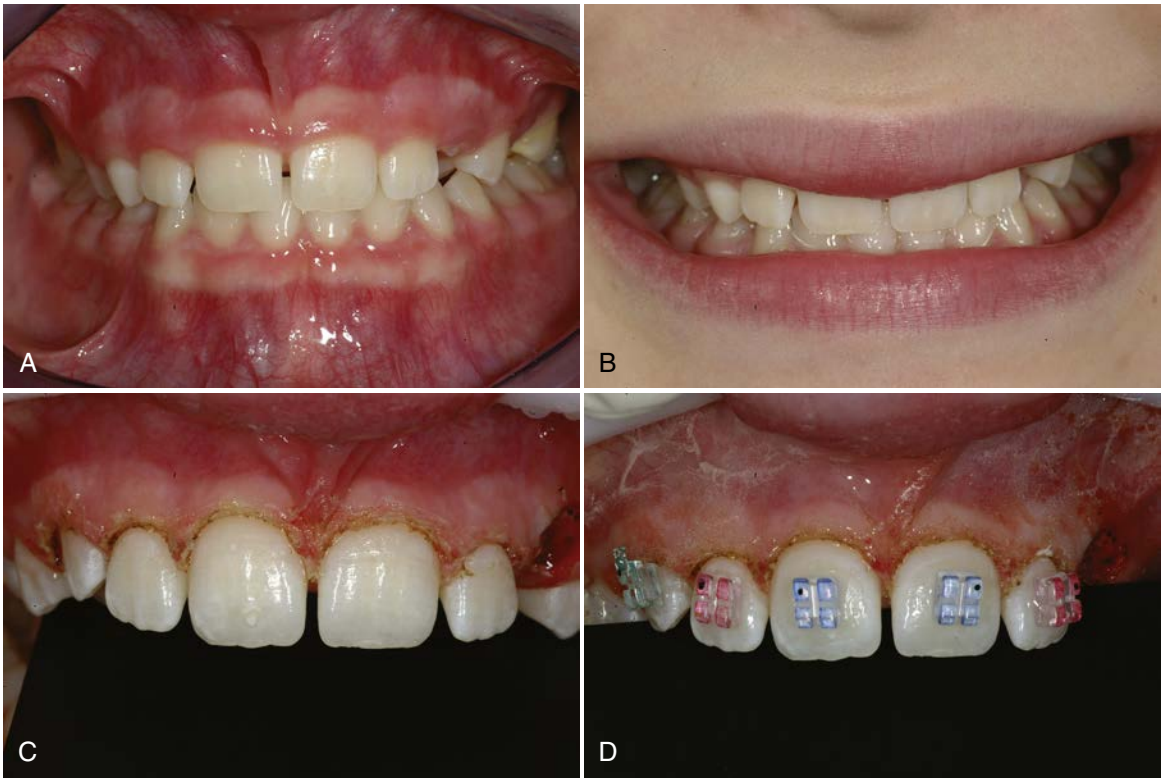


FIGURE 9-41 A, This patient has a disproportionality of the width and height of the maxillary incisors. The short crown heights were due to incomplete passive eruption. Positioning the brackets relative to the incisal edge would position them too close to the gingival margin, which would contribute to poor oral hygiene and result in gingival overgrowth. Positioning the brackets in the center of the clinical crown would cause unwanted incisor intrusion (B) in a patient where this was not desired. C, The decision was made to improve tooth proportion through laser crown lengthening before bracket placement to maximize the chance of positioning the incisors in their ideal vertical position. D, Immediately after bracket placement.

2. Determine how much maxillary incisor is displayed on smile in millimeters (Fig. 9-42, *C*).
3. Determine crown height and width of the incisors. This helps establish whether tooth shape discrepancies exist. If the tooth is too wide, tooth reduction may be desirable before bracket placement. If the incisor is short because of delayed passive eruption or gingival encroachment, crown lengthening may be indicated.
4. Note the number of millimeters of gingival display on smile if present.
5. Refer to chart of gingival display by age (Fig. 9-42, *D*).
6. Determine the smile arc–consonance relationship.
7. Measure gingival height relationships.
   a. Canine–lateral–centrals
   b. Canine–lateral–centrals to upper lip on repeatable social smile
8. Make final decisions and establish applications. For example, if incisor display at rest and on smile is inadequate, then do the following:
   a. Decide how much more tooth display is desired at rest or on smile and how far the incisal edge needs to go to reach the smile arc.
   b. Measure to the center of the clinical crown where most bracket systems require placement.
   c. Place the bracket more apically the distance the incisor is to be moved inferiorly.

The application of this approach to bracket placement is illustrated next.

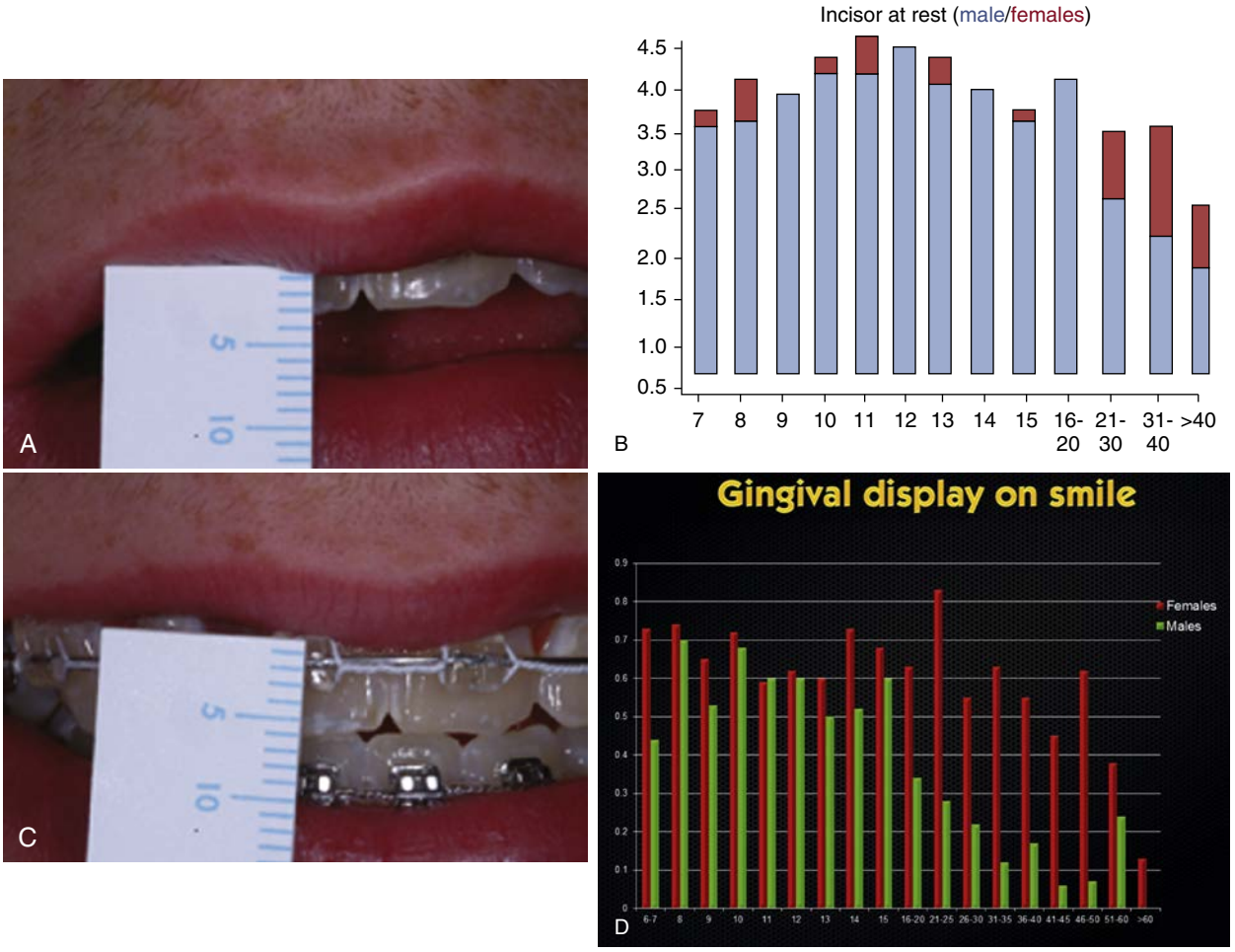


FIGURE 9-42 A, The first step in individualizing bracket placement is to determine how much the maxillary incisor shows at rest. The purpose of this measurement is to place the incisor in the best position for smile display and to anticipate the long-term effects of aging. B, Refer to the chart for appropriate amounts of incisor display for the patient in a particular age group who is to receive brackets. The clinician should consider that these data represent an untreated population, and if smile youthfulness is desired, then greater incisor display may be planned in certain cases. C, Next, the orthodontist asks the patient to smile (social smile) and makes a direct measurement of how much upper incisor shows on smile. D, The amount of gingival display is measured when present and is compared against the chart for gingival display and to the crown height measurement. If gingival display on smile is excessive, the clinician can determine whether it is caused by vertical maxillary excess or short crown height.

CASE STUDY 9-1 SMILE REFINEMENT: MINIAESTHETICS AND MICROAESTHETICS

It is critical in planning smile aesthetics that the orthodontist understand all the principles of mini- and microaesthetics. In presenting a case for smile refinement, we feel it would be beneficial to review some of the important points in this chapter, applied specifically to this case for clarity of teaching.

Miniaesthetics

In smile assessment, there are many characteristics attributable to what is considered the ideal smile. In cosmetic dentistry, there have been numerous publications and quantitative research on what makes the ideal smile. Applications of the principles they have developed along with our orthodontic concepts can be of real significance in treatment planning our cases to be exceptional, and not just normal, as Figure 9-43 offers guidelines on accepted parameters for the ideal smile. At the beginning of this chapter, we described the clinical examination we recommend for smile analysis, taking into account both the mechanism of the smile and the resting characteristics of incisor display. Important aspects of the many miniaesthetic analyses include:

1. The positioning of the upper lip during social smile—the final position of the upper lip during smile and how much gingival display there is can be quite variable, depending on the patient's age. The final position impacts not only how much gingival display there is but also how much incisor display there is on smile. In general terms, the older we get, the less incisor we show on smile, as well as less gingival display. This has clinical impact on us when we are judging whether the adolescent has a gummy smile that will improve with time or whether orthodontic treatment should be directed to increasing incisor display. In both the adolescent and the adult, opening a deep overbite with maxillary incisor intrusion in general is a procedure that has a negative effect on the maturation and aging of smile.
2. Gingival display on smile—as noted above, the amount of gingival display on smile varies with age, and the interaction of gingival display and orthodontic mechanics is linked directly.
3. The percentage of incisor display on smile—in general terms, males show less upper incisor on smile than do females, regardless of age. However, the trend is for both genders to show less incisor as time goes by, but with the male degrading more quickly than the female. In other words, male smiles age more quickly than do female smiles.
4. Buccal corridors—as described previously as a prosthodontics concept from the 1950s, buccal corridors were originally described in terms of denture construction. Translating prosthodontic concepts to today's aesthetic demands and a full dentition is not realistic. Currently, quite a bit of research is being carried out and published on how much buccal corridor show is the ideal amount. Leaving the patients to judge, the current trend is "the wider the better."
5. The smile arc—the smile arc has been described previously in this chapter as the relationship of the maxillary occlusal plane to the lower lip on smile. Ideally, we like for the curvature of both on frontal smile to be parallel, or consonant. However, we would like to emphasize that ideal smile arcs are not always achievable due to other factors such as inherited skeletal patterns, dental attrition, and many other factors. Therefore, we also emphasized that attainment of an ideal smile arc is a guideline, not a rule.

Microaesthetics

In cosmetic dentistry, tooth shape and proportions have been studied extensively. In addition to the height and width proportions of the teeth, the concepts of tooth contacts and connectors can be of real significance in treatment planning the appearance of the smile. Figure 9-44 is a graphic representation of the many attributes we have pieced together to constitute what is considered the ideal microaesthetic analysis.

1. Long axis—the inclination of the anterior teeth should be tipped slightly to the distal aspect.
2. Incisor height/width ratio—incisor height tends to be more variable than incisor width because of the variability of soft tissue biotypes and, in the adolescent, stages of active and passive eruption. The ideal ratio, however, should be 8:10 of width to height.
3. Contact—the contact between the teeth is, of course, where the teeth touch. However, it was described best by Morley and Eubank as, "the point which the teeth appear to touch and makes of the floss pop." The vertical positioning of the contact is important, because it affects the next two attributes.
4. Connector—the connector is "where the teeth appear to touch but does not grab the floss." The accepted ratios to central incisor height are for the connector to be 50% the length of the maxillary incisor height between centrals, 40% of the length of the central incisor between the central incisor and the lateral incisor, and between the lateral incisor and the canine, the percentage should be 30% of central incisor height.
5. Incisal embrasure—what patients sometimes mistake as a space problem is actually a large embrasure. The central incisor embrasure should be smallest, while the width of the embrasure should increase as it progresses away from the midline.
6. Papilla height—40% of the central incisor height is the accepted ideal but is somewhat variable among studies. If the papilla height does not reach the connector, then a gingival embrasure, or an unaesthetic "black triangle," results.
7. Gingival height—the accepted ideal for the most superior aspect of the gingival shape/margin is for the centrals and cuspids to be roughly the same length, with the lateral incisors being slightly shorter with the gingival margin positioned slightly more inferior than the canine and central.
8. Zenith—defined as the apex of the gingival shape and should be slightly distal to the long axis of the tooth on both the maxillary centrals and canines, but on the maxillary lateral it should be coincident.

The 15-year-old female in Figure 9-45 presented with what would be by anyone's standards considered a nice smile. However, her smile was problematic to her, so we undertook a thorough assessment as to what her needs might be in presenting a potential treatment plan. Many options existed, of course, from orthodontics to veneers, but at age 15, the orthodontic option was, without question, the most appropriate. Her occlusal relationships were quite good (Fig. 9-46), but, looking critically, the maxillary lateral incisors would benefit from some facial root torque. In her close-up smile image (Fig. 9-47), adhering to our quantitative clinical assessment, we observed a slightly flat smile arc, incomplete incisor display of the maxillary incisors, and disproportionate height/width ratio of the maxillary lateral incisors. Utilizing computer imaging, the smile targets were identified (Fig. 9-48, *A*) and quantified (Fig. 9-48, *B*) so that we can plan precise bracket positioning to extrude the maxillary incisors to the desired amount to increase incisor display and reach the smile arc target. A visual mockup of the gingivectomy was demonstrated so that the patient and the parents could visualize the periodontal procedure to address the gingival issue, and the extrusion of the incisors is also displayed (Fig. 9-49). The value of informed consent during this entire process is an important aspect of the incorporation of digital imaging into the consultation process when aesthetics is a very important element of the treatment.

The next step in treatment is, of course, to deliver the demonstrated treatment as accurately as possible. Custom bracket placement may be placed according to the orthodontist's design, as well as wire selection. Figure 9-50 represents a calibrated screen image in the planning template in which we have virtually extruded the maxillary incisors to the length we determined to be needed from our clinical exam. The existing dental relationships are in green, while the planned movement is in white. The grid represents 1-mm increments, so on the computer screen we can "grab" the tooth and drag it to the position that we want it to be in. Also, in this case, we can direct the amount of torque desired in the lateral incisors (Fig. 9-51). An indirect bracket setup is then created, and in this case, custom brackets were also produced to allow us to design in the desired torques needed on laterals on the initiation of treatment.

The final results (Fig. 9-52) were quite good, and the patient's parents were quite pleased. This case illustrates the coordination of the direct clinical biometric examination, visualization and quantification of treatment goals, and digitally designed fixed appliances to maximize the outcome.

David M. Sarver, DMD, MS

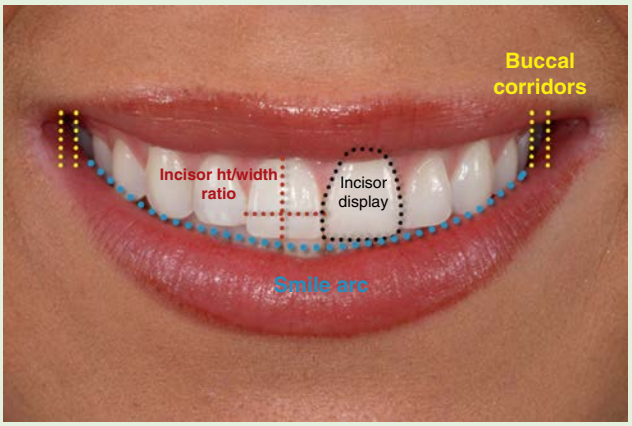


FIGURE 9-43 Here are some guidelines on accepted parameters for the ideal smile.

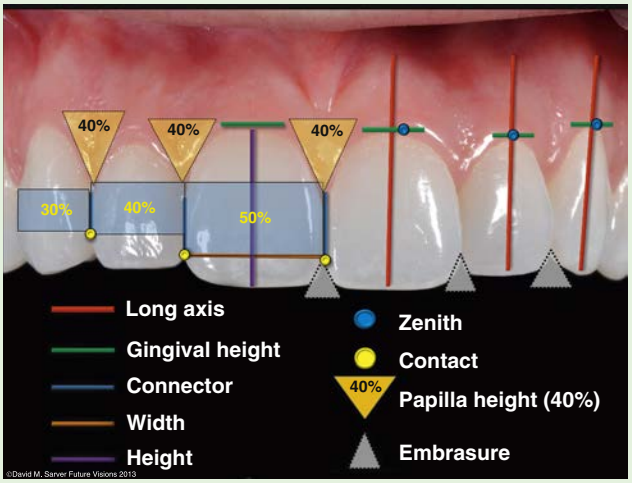


FIGURE 9-44 Ideal microaesthetic characteristics are graphically represented in this figure. This analysis is rather extensive, including orientation of the long axis of the anterior teeth, incisor height/width ratio, contacts, connectors, incisal embrasures, papilla heights, gingival heights, and zeniths.

FIGURE 9-45 This 15-year-old female presented with a very nice occlusion but was not completely happy with her smile. She was unsure as to what she did not like and sought a recommendation as to what might be improved.

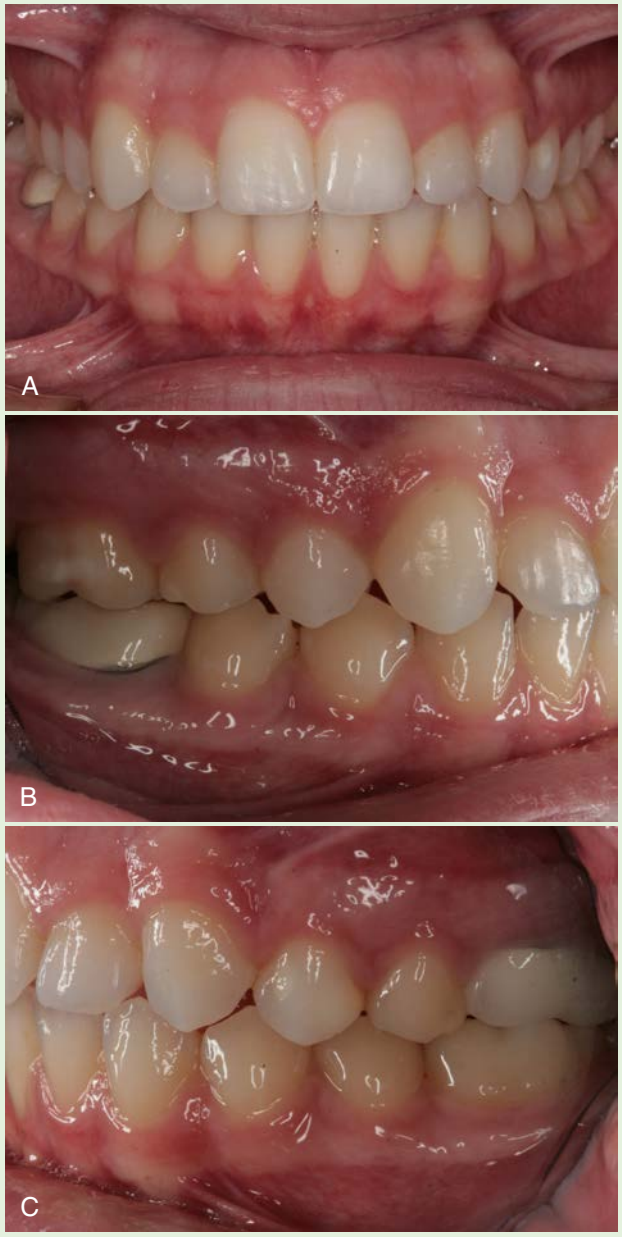
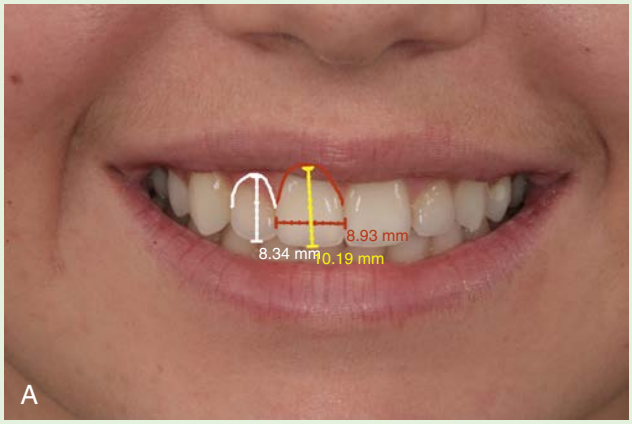


FIGURE 9-46 A, The center intraoral image reflects the short lateral incisors in comparison to maxillary central incisor proportion and palatal root displacement of the lateral incisors. B, The buccal occlusal on the right side was functionally quite good. C, The buccal occlusal on the left was also functionally quite good.

FIGURE 9-47 Her close-up smile reflected a flat smile arc, incomplete incisor display, and disproportionality of the height/width ratio of the maxillary incisors.



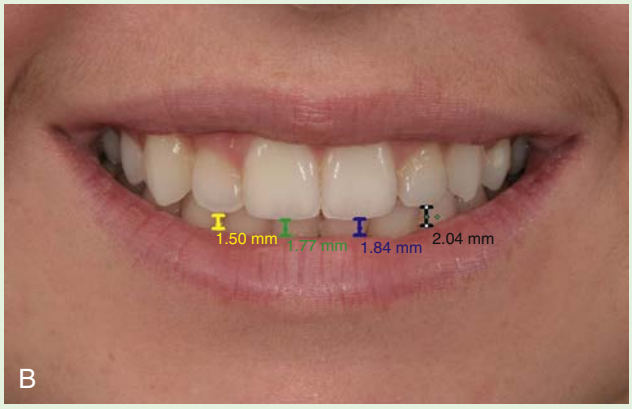


FIGURE 9-48 A, Utilizing computer imaging, we were able to graphically outline proposed improvements—for example, the ideal maxillary right lateral shape and form as outlined in white and its height also measured through a calibrated image. This calibration also allowed quantification of the height/width ratio of the maxillary right central and the distance from the incisal edge to the lower lip curvature representing the smile arc. B, In order to facilitate maxillary bracket placement, precise measurements were made from the incisal edge to the smile arc target, and this update was used in compensating the vertical position of our normal bracket placement. In other words, the center of the bracket was moved superiorly the same amount as the target indicated. This is particularly useful in this case, since the maxillary incisors have a slight transverse cant, and the left side needs to be extruded slightly more than the right.

FIGURE 9-49 In a final graphic illustration of our proposed treatment outcome for the patient and parents, simple cut-and-paste tools were used to simulate extrusion of the incisors to the smile line target and reshaping the gingival margin of the lateral incisors to simulate the potential result of gingivoplasty.

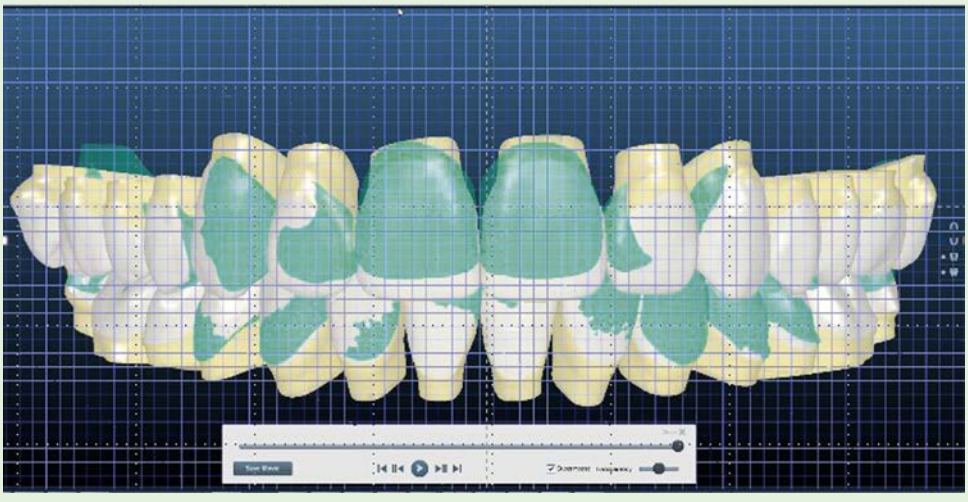


FIGURE 9-50 After intraoral scanning, a three-dimensional rendition of the patient's dentition was loaded for treatment planning. Utilizing tools available in software, we move the incisors downward the prescribed amount determined in the calibrated smile image.

CASE STUDY 9-1 SMILE REFINEMENT: MINIAESTHETICS AND MICROAESTHETICS—cont'd

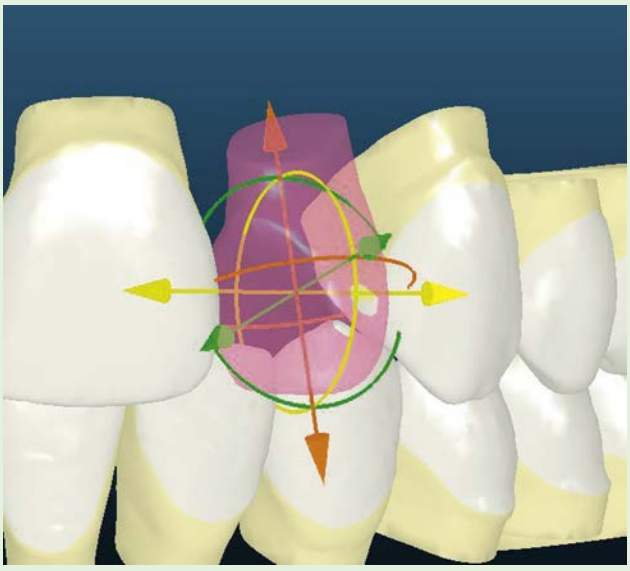


FIGURE 9-51 The CAD (Computer Assisted Design) software allows the clinician to manipulate individual teeth in all planes of space to design both the occlusion and esthetic placement of the teeth to maximum advantage. In this case, the amount of labial root torque in the 3D rendering was transferred to CAM (Computer Assisted Machining) technology to custom fabricate the appliance system to the clinician's precise design.

FIGURE 9-52 **A,** After 9 months of treatment, the patient's final full-facial smile. **B,** The close-up smile reflecting the final outcome and the successful product of the quantified clinical examination, identification and quantification of esthetic targets, and the resulting success of the CAD-CAM customized appliance system. **C** to **E**, The final occlusal relationships.

Continued

REFERENCES

1. Sarver DM, Ackerman JP, Proffit WR. Diagnosis and treatment planning in orthodontics: the modern soft tissue paradigm. In: Graber T, Vanarsdall R, eds. *Orthodontic Practice and Principles*. 3rd ed. St. Louis: Harcourt, Brace, and Mosby; 2000.
2. Sarver DM. Principles of cosmetic dentistry in orthodontics: part 1. Shape and proportionality of anterior teeth. *Am J Dentofac Orthop*. 2004;126:749–753.
3. Mohs E. General theory of paradigms of health. *Scand J Soc Med Suppl*. 1991;46:14.
4. Kiyak HA, Hohl T, West RA, et al. Psychologic changes in orthognathic surgery patients: a 24-month follow-up. *J Oral Maxillofac Surg*. 1984;42:506–512.
5. Morley J, Eubank J. Macroesthetic elements of smile design. *J Am Dent Assoc*. 2001;132(1):39–45.
6. Ackerman MB, Ackerman J. Smile analysis and design in the digital era. *J Clin Orthod*. 2002;36:221–236.
7. Brodie AG. Cephalometric roentgenology: history, technics, and uses. *J Oral Surg*. 1949;7:185–198.
8. Sarver DM, Proffit WR. Diagnosis and treatment planning in orthodontics: the modern soft tissue paradigm. In: Graber T, Vanarsdall R, Vig K, eds. *Orthodontic Practice and Principles*. 4th ed. St. Louis: Elsevier; 2005.
9. Hulsey CM. An esthetic evaluation of lip-tooth relationships present in smile. *Am J Orthod*. 1970;57:132.
10. Riggsbee OH, Sperry TP, BeGole EA. The influence of facial animation on smile characteristics. *Int J Adult Orthodon Orthognath Surg*. 1998;3:233.
11. Frush JO, Fisher RD. The dysesthetic interpretation of the dentogenic concept. *J Prosthet Dent*. 1958;8:558.
12. Sarver DM, Ackerman JL. About face: the re-emerging soft tissue paradigm. *Am J Orthod Dentofac Orthop*. 2000;117:575–576.
13. Park YC, Burstone CJ. Soft-tissue profile-fallacies of hard-tissue standards in treatment planning. *Am J Orthod Dentofacial Orthop*. 1986;90(1):52–62.
14. Sarver DM, Yanosky MR. Principles of cosmetic dentistry in orthodontics: part 2. Soft tissue laser technology and cosmetic gingival contouring. *Am J Dentofac Orthop*. 2005;127:85–90.
15. American Academy of Cosmetic Dentistry. *Diagnosis and Treatment Planning in Cosmetic Dentistry: A Guide to Accreditation Criteria*. Madison, WI: Author; 2004.

SUGGESTED READINGS

Dickens S, Sarver DM, Proffit WR. The dynamics of the maxillary incisor and the upper lip: a cross-sectional study of resting and smile hard tissue characteristics. *World J Orthod*. 2002;3:313–320.

Fattahi T. Aesthetic surgery to augment orthognathic surgery. *Oral Maxillofac Surg Clin North Am*. 2007;19(3):435–447. vii.

Guthrie PB. Rhinoplasty—the other facial osteotomy. *Ann R Australas Coll Dent Surg*. 1991;11:266–272.

Kinnebrew MC, Emison JW. Simultaneous maxillary and nasal reconstruction. *J Craniomaxillofac Surg*. 1987;15(6):312–325.

Morley J, Eubank J. Macroesthetic elements of smile design. http://www.ncbi.nlm.nih.gov/pubmed/11194397 Journal of the American Dental Association (1939) *J Am Dent Assoc*. 2001 Jan;132(1):39–45.

Ronchi P, Chiapasco M. Simultaneous rhinoplasty and maxillomandibular osteotomies. *Int J Adult Orthodon Orthognath Surg*. 1998;13(2):153–161.

Sarver DM, Ackerman MB. Dynamic smile visualization and quantification: Part I. Evolution of the concept and dynamic records for smile capture. *Am J Dentofac Orthop*. 2003;124:4–12.

Sarver DM, Johnston MJ. Orthognathic surgery and esthetics: planning treatment to achieve functional and aesthetic goals. *Br J Orthod*. 1993;20(2):93–100.

Sarver DM, Rousso DR. Facial plastic surgical procedures combined with orthodontic/orthognathic procedures. *Am J Dentofac Orthop*. 2004;126:305–307.

Sarver DM, Weissman SM, Matukas VJ. Incorporation of facial plastic surgery in the planning and treatment of orthognathic surgical cases. *Adult Orthodon Orthognath Surg*. 1991;6(4).

Sarver DM, Yanosky M. Combined orthodontic, orthognathic, and plastic surgical treatment of an adult Class II malocclusion. *J Clin Orthod*. 2005;39(4):209–213.

Sarver DM. Enamelplasty and esthetic Finishing in Orthodontics-Differential Diagnosis of Incisor Proclination-The Importance of Visualization and Records Part 2. *J Esthet Restor Dent*. 2011;23(5):303–313.

Sarver DM. Principles of cosmetic dentistry in orthodontics: Part 1. Shape and proportionality of anterior teeth. *Am J Dentofac Orthop*. 2004;126:749–753. December.

Schendel SA, Carlotti Jr AE. Nasal considerations in orthognathic surgery. *Am J Orthod Dentofacial Orthop*. 1991;100(3):197–208.

Waite PD, Matukas VJ. Indications for simultaneous orthognathic and septorhinoplastic surgery. *J Oral Maxillofac Surg*. 1991;49(2):133–140.

Waite PM, Matukas VJ, Sarver DM. Simultaneous rhinoplasty in orthognathic surgery. *Int J Adult Orthodon Orthognath Surg*. 1988;17:298–302.

10

Psychological Aspects of Diagnosis and Treatment

Leslie A. Will

OUTLINE

Patient Perceptions, 289
Patients with Psychological Disorders, 291
Attention-Deficit/Hyperactivity Disorder, 291
Obsessive-Compulsive Disorder, 291
Body Dysmorphic Disorder, 292
Bipolar Disorder, 292
Panic Disorder, 293
Depression, 293
Eating Disorders, 293
Personality Disorders, 294
"Difficult" Patients, 294
Patients Having Orthognathic Surgery, 294
Psychological Status and Motivation, 294
Expectations, 296
Satisfaction, 296
Patients with Craniofacial Deformities, 298
Psychological Issues, 298
Patients with Clefts of the Lip and Palate, 298
Patients with Acquired Deformities, 299
Summary, 299

Orthodontists learn very early in their careers that moving teeth is only one aspect of taking care of their patients. Every patient is different, and every person comes with his or her unique pattern of responding to others, making decisions, and carrying out plans. Each patient's personal experience, family history, and cultural differences will naturally influence individual responses to orthodontic treatment strategies.

Communication between the orthodontist and the patient is extremely important in achieving treatment goals. In addition to encouraging cooperation and maximizing good treatment results and patient satisfaction, good communication is essential from a medicolegal standpoint. Instead of just giving the patient information, the orthodontist needs to be concerned with what the patient understands and expects from treatment.

Research has shown that patients do not always understand or remember what they have been told about their malocclusion or the orthodontic treatment. Mortensen, Kiyak, and Omnell[1] interviewed 29 pediatric patients aged 6 to 12 years and their parents 30 minutes after an informed-consent discussion. Both the children and their parents were asked about the reasons for treatment, risks, and responsibilities that were mentioned during the informed consent discussion. It was discovered that although an average of 4.7 risks were mentioned by the orthodontist during each discussion, on average the parents remembered 1.5 risks and the children remembered less than 1. Similarly, 2.3 reasons for treatment were mentioned by the orthodontist during the discussion, but the parents on average remembered 1.7 and the children remembered 1.1. Clearly, not all the desired information is being remembered by the patients or their parents.

PATIENT PERCEPTIONS

Psychological factors may influence a patient's perception of his or her malocclusion as well as the treatment plan. It is difficult to know or predict how a patient will view his or her individual situation. Fortunately, there are several research approaches that can give some insight as to how patients see malocclusion, and some generalities have been learned that may help orthodontists assess how their patients are likely to react.

One key method is to show the patient profiles. By altering one aspect of the profile in successive photographs or silhouettes and asking the patient to indicate which profile is most like theirs, it is possible to determine how accurately patients can perceive profiles.[2]

A version of this is the Perceptometrics technique that was developed by Giddon et al.,[3] in which computer alterations are made to photographic images, with the feature of interest "morphed" by the computer so that the feature is moved back and forth in one dimension at gradual, predetermined intervals. By clicking on the image, the range of photographs can be traversed, with each photograph being displayed the same amount of time. Patients can then indicate the beginning and end of the acceptable range of profiles by holding down the computer mouse, and they can also indicate which profile is most attractive. This tool enables clinicians to determine the range of what patients consider acceptable (Fig. 10-1).

Many studies have been done using a variety of methods exploring patient perceptions on many aspects of facial aesthetics. Kitay et al.[4] found that orthodontic patients are less tolerant of variations in their profiles than are nonorthodontic patients. To determine their range of acceptability, 16 patients and 14 nonorthodontic adult patients were asked to respond

FIGURE 10-1 Four frames from animation of horizontal chin distortion including both extremes and two intermediate frames (From *Am J Orthod Dentofacial Orthop.* 114(6):632, Fig. 1.)

to computer-animated distortions of profiles that distorted the lower third of their own faces using the Perceptometrics program. Both groups of subjects were equally accurate in identifying their own profiles. However, the orthodontic patients had a smaller zone of acceptability (ZA) in features in a control face, with a significant disparity between one feature in their own profile and the most pleasing position for that feature. This suggests that the orthodontic patients are motivated to seek treatment by specific features in their own face that they perceived as undesirable.

Using the Perceptometrics technique, Arpino et al.[5] compared the ZA of profiles selected by orthognathic surgery patients, their "significant others," orthodontists, and oral surgeons. Patients with both Class II and Class III jaw discrepancies evaluated their own photographs with four features altered horizontally (upper lip, lower lip, both lips together, and chin) and one feature, lower facial height, altered vertically. Although there was some variation, the magnitude of the ZA was smallest for the patient, followed by the surgeon, the significant other, and finally the orthodontist. Whereas the patient and the significant other groups differed in only two instances, the orthodontists and oral surgeons had significantly different ZAs for all but the Class II bimaxillary relationship. These results show that orthodontists are most tolerant of different profiles, while the patients themselves are least tolerant, perhaps reflecting the reality that orthodontic treatment compared with orthognathic surgery is a slower biological process with a wider range of acceptable outcomes.

Hier et al.[6] used the same technique to compare the preferences for lip position between orthodontic patients and untreated subjects of the same age. They found for both males and females that the untreated subjects preferred fuller lips than did orthodontically treated subjects, which is greater than Ricketts' ideal measurement of lip protrusion to the E-line, between the tip of the nose and soft tissue pogonion.

Miner et al.[7] compared the self-perception of pediatric patients with the perceptions of their mothers and their treating orthodontists. Using the Perceptometrics technique, the upper lip, lower lip, and chin were distorted as the images moved from retrusive to protrusive extremes in counterbalanced order. The patients, mothers, and clinicians were asked to indicate the ZA for each feature and the most accurate representation of the child's profile, as well as indicate the ZA for a neutral female face. Both patients and mothers were found to overestimate the protrusiveness of the child's actual mandible, and both groups preferred a more protrusive profile for both the child and the neutral face. In addition, the mothers had the smallest tolerance for change in the soft tissue profile. These studies are valuable for pointing out the inherent inaccuracies of patient's perceptions as well as the differing preferences of patients.

These techniques are also useful for exploring perceptions of different racial and ethnic groups. In a study by Mejia-Maidl et al.,[8] 30 Mexican Americans and 30 whites of varying age, sex, education, and acculturation indicate their perceptions of four profiles of individuals of Mexican descent. Using the Perceptometrics program, the authors found that in general, Mexican Americans preferred less protrusive lips than did the whites. In addition, there was a wider ZA or tolerance for male lip positions and female lower-lip positions among the whites than among the Mexican Americans of low acculturation. These observations were not true of highly acculturated Mexican Americans, who may have assimilated American cultural aesthetic preferences. Park et al.[9] compared the perceptions of Korean-American orthodontic patients with those of white orthodontists and Asian-American orthodontists. Statistically significant differences were found between the Korean-American patients and the white orthodontists for the acceptable and preferred positions of the female nose and the male chin, finding that the Korean Americans preferred a more protrusive nose for females and a more retrusive chin for males. McKoy-White et al.[10] compared the ZA for black females among black female patients, black orthodontists, and white orthodontists. The patients were also asked to correctly identify their most accurate pretreatment and posttreatment profile. It was found that the white orthodontists preferred flatter profiles than did the black women, who in turn preferred fuller profiles than the black orthodontists. Although the patients could correctly identify their own posttreatment profile, they all recalled a fuller pretreatment profile than they actually had.

These studies underscore the importance of racial and cultural influences on the esthetic perceptions and preferences of orthodontic patients. Such studies are also valuable for pointing out the inherent inaccuracies of patients' perceptions as well as the cephalometric bases of their facial preferences. Orthodontists need to be sensitive to differences between patients' and their own preferences in formulating treatment plans.

PATIENTS WITH PSYCHOLOGICAL DISORDERS

Different and unanticipated behaviors among patients often become challenging for the orthodontist. When does such behavior become a problem? When are some behaviors simply difficult, while others may be evidence of pathology? How do we recognize these difficulties and deal with them?

The preceding research explores the perception of psychologically healthy individuals. However, many orthodontic patients may have preexisting psychological disorders when they present for orthodontic treatment. Many patients who seek orthodontic treatment are functioning within society while being treated for a psychological disorder. Although these patients are usually controlled, it is important to recognize how these disorders may be manifested. In addition, orthodontists should be aware of side effects of medications that may have implications for oral health.

Common psychological conditions are delineated by the American Psychiatric Association in the *Diagnostic and Statistical Manual of Mental Disorders*, or *DSM- V*.[11] The most common conditions that orthodontists may encounter are attention-deficit/hyperactivity disorder (ADHD), obsessive-compulsive disorder (OCD), body dysmorphic disorder, bipolar disorder, panic disorder, and depression. In addition, there are some personality disorders and other psychological conditions, such as eating disorders, that may acutely affect adolescents.

Attention-Deficit/Hyperactivity Disorder

ADHD is a chronic disorder characterized by inattention, impulsivity, and hyperactivity. A survey carried out by the Child Trends of National Health Interview in 2013 reported that 8.8% of children ages 3 to 17 were diagnosed with ADHD, including 12% of boys[12] as well as more than 4% of the adult population.[13] Some diagnostic criteria, however, are nonspecific and the disorder may be overdiagnosed, with some signs of ADHD being observed in almost everyone sometime in their life. Nevertheless, the main criterion is that the behavior must cause impairment in the individual's life for a prolonged period of time.[11]

The precise etiology of ADHD is not known. Although it is considered to have a genetic basis in the majority of cases, it is most likely that a combination of genes, rather than a single gene, is responsible.[14] Approximately 20% of cases may be due to prenatal brain injury, such as hypoxia accompanying prematurity or tobacco smoke, or trauma. Food allergies and food additives are suspected as possible aggravating factors.

Medication is considered to be the most effective method of treating ADHD.[15] However, behavioral therapy for parents of children with ADHD may also be useful to assist parents in managing their children most effectively. A recent systematic review examined 403 primary studies that evaluated treatment outcomes of either pharmacologic, behavioral, or combination treatment.[16] The review concluded that combination treatment resulted in the highest proportion of improved outcomes, at 83%. A majority of outcomes improved regardless of treatment duration or age at initiation of treatment. However, it should be noted that longer follow-ups revealed less benefit from treatment, suggesting that some treatment outcomes may not persist to the same extent after several years.

Hyperactivity and the inability to focus can be problems during orthodontic treatment. Patients with ADHD may have trouble sitting still during procedures and may not be compliant in maintaining good hygiene, wearing elastics, or performing other tasks because of forgetfulness and inattentiveness. These patients can be best managed by giving short, clear instructions and giving written instructions or reminders to them or their parents, with follow-up questions to determine their comprehension and rewards for successful compliance. Dental prophylaxis may be needed more frequently to avoid decalcification and caries. To increase the likelihood of treatment success, it may be wise to avoid treatment plans that require a high degree of patient compliance. During treatment, it is often helpful to give the patient breaks during prolonged procedures.

Obsessive-Compulsive Disorder

OCD is characterized by intrusive thoughts and repetitive, compulsive behaviors.[11] The patient's behavior is intended to reduce the anxiety that accompanies the intrusive thoughts. This disorder affects 1% to 4% of the population and is often associated with eating disorders, autism, or anxiety disorders.[17]

Although OCD is also considered to be genetic in etiology,[18] specific genes causing OCD have not been identified, and the molecular basis of the disorder has not been determined. The clinical variability suggests that the etiology is heterogeneous, with the possibility of gene–gene and gene–environment interactions.[19]

Treatment of OCD can take one of two forms. For milder cases, cognitive-behavioral therapy (CBT) is usually used. During this form of treatment, the patient is exposed to a feared stimulus with increasing intensity and frequency so that the patient will learn to tolerate what had previously caused anxiety. In addition to CBT, more severe cases and adult patients usually require medication, such as selective serotonin reuptake inhibitors (SSRIs), including clomipramine (Anafranil), fluoxetine (Prozac), fluvoxamine (Luvox), paroxetine (Paxil), and sertraline (Zoloft).[18] One side effect of this class of psychopharmacologic agents is xerostomia, so orthodontists should be aware of this possibility and advise patients accordingly.

The greatest predictor of psychiatric problems in adulthood is a similar disorder in childhood. Obsessive-compulsive behaviors can be noticed in a dental setting.[20] Orthodontists are thus well positioned to detect possible problems and refer a patient for evaluation.

Body Dysmorphic Disorder

Body dysmorphic disorder (BDD) is characterized by an intensely negative emotional response to a minimal or nonexistent defect in the patient's appearance. The head and face are common foci for this preoccupation, so orthodontists may see patients who have excessive concerns about their dentofacial appearance.[21] Other characteristics of this disorder involve multiple consultations about their perceived defect, an obsessive concern with appearance, and emotional volatility. This preoccupation may lead to stress and related disorders and behaviors.[22] Patients are likely to become socially isolated since so much time and attention are devoted to this concern.[23]

Diagnosis can be difficult and misleading, without recognition that BDD involves more than obsessive thoughts. Underdiagnosis is also common, because many patients may not seek help. Approximately 1% of the population may suffer from BDD, which may coexist with other disorders, such as depression and OCD.[24,25]

BDD is also treated most successfully with SSRIs, although CBT can help.[26] Using photographic imaging of the patient's own face as a reality check may help with some patients (Giddon DB, personal communication). The cognitive aspect of therapy aims to restructure faulty beliefs that lead patients to focus on an imagined defect. The behavioral therapy works to reduce the social avoidance and repetitive behaviors. It is not known which mode of therapy is better or if a combination is best.[26]

If this disorder is not treated, most patients will seek dental, medical, or surgical treatment to "correct" their flaws, which usually fails.[23] Physical improvement, however, does not signify psychological improvement. Dissatisfied patients may become violent toward themselves or attempt suicide. Thus, orthodontists and other clinicians who are consulted are advised to be particularly wary of such patients, who can disrupt office routines, leading to great frustration for both clinicians and patients.

Even if patients do not have a diagnosis of BDD, they may have excessive concerns about minimal or nonexistent deformities or malocclusion. The orthodontist can screen out patients who may have excessive concerns that are impossible to satisfy by taking a careful history on every patient, particularly probing the extent and nature of aesthetic concerns. Since most patients with aesthetic concerns are not significantly disfigured, these deformities would probably be considered minimal. If patients show an inordinate focus on them, the orthodontist would be well advised to look further before starting treatment, or not begin treatment at all. Polo[27] has developed questions that can be used as starting points to identify patients who may have excessive concern over esthetic problems (Box 10-1).

BOX 10-1 Screening questionnaire for Body Dysmorphic Disorder in orthodontic patients.

- How does the patient rate the severity of the orthodontic concern or defect?
- How would the patient rate the amount of distress or worries produced by the orthodontic concern, defect, or "unattractive" appearance?
- Does this (minor or perceived) defect cause significant distress either socially or related to family/work activities?
- Why is orthodontic treatment sought?
- Have previous evaluations concerning the orthodontic "defect" been performed?
- Why are additional orthodontic evaluations sought?
- Are the expectations for this particular orthodontic procedure reasonable?
- Have requests for other cosmetic procedures ever been obtained?
- Have these other cosmetic procedures been performed? Are these frequent? How many? When?
- Is there a history of dissatisfaction with previous cosmetic procedures? Are these multiple?
- Does the patient report any history of psychiatric or psychological disturbances or any previous referrals for psychological/psychiatric evaluations?

(From Polo M: Body Dysmorphic Disorder: A Screening Guide for Orthodontist, Am J Orthod Dentofac Orthop 139(2):170-173, 2011 Fig. 2.)

With such patients, limits on therapeutic intervention must be set. Patients should be given realistic options with definite endpoints, including the option of no treatment. Concrete comparisons and predictions should be shown to reinforce reality and not lead to unrealistic expectations. Treatment options and the final treatment plan, along with possible obstacles to ideal results, should be put in writing. If ongoing, treatment should be stopped and/or the patient referred to other health professionals.

Bipolar Disorder

Bipolar disorder, formerly known as manic-depressive disorder, consists of two phases: depression and mania. These mood swings are so severe as to interfere with normal life. The lifetime prevalence of this disorder is 1.6%, although the course of the disorder varies. The peak time of onset is between 15 and 24 years, stabilizing in later years.[28]

Accompanying the mood swings can be a variety of other disorders. It is estimated that 50% of patients also abuse illegal substances. Between 25% and 50% attempt suicide, with 10% to 15% being successful.[28]

The pathogenesis of this illness consists of neurochemical abnormalities with an etiology that is at least partially genetic. If one parent is affected, there is a 25% risk that children will be affected, but if both parents are affected, the risk jumps to 50% to 75%. There is a 70% concordance in identical twins.[28]

Treatment for BD with mood stabilizers such as lithium, valproate, or carbamazepine is most important.[28] Drugs that calm agitation, such as chlorpromazine or olanzapine, may also be useful. Antidepressants are not usually prescribed because they may trigger mania.

Of concern in bipolar disorder is that for most patients, 5 to 10 years elapse between the beginning of symptoms and treatment.[28] This is probably due to the fact that for a certain period of time, the mood swings do not seem serious enough to warrant treatment, and people will often try to accommodate to

their symptoms as long as possible before submitting to psychiatric care. During this time, however, these patients may be difficult to manage, with periods of depression and mania. For the orthodontist, bipolar disorder may be manifested with poor hygiene, a lack of compliance, and a general apathy toward treatment. In the patient under treatment, medications can produce xerostomia, with its deleterious effects on the dentition.[28]

Panic Disorder

Panic disorder (PD) is diagnosed when the patient experiences sudden, recurrent panic attacks consisting of heart palpitations, dizziness, difficulty breathing, chest pains, and sweating that are unrelated to any external event and are not due to any medical condition. It is estimated that 2% of males and 5% of females are affected in their young adult years, and the majority have concurrent depression. This condition can be extremely disabling because the patient often avoids certain situations in an effort to prevent recurrences, with the result that patients are socially and vocationally impaired.

A genetic susceptibility to PD combined with environmental stresses is likely, and the heritability is estimated to be 48%.[29] It has been hypothesized that there is a mutation in 13q, with an organic defect in the amygdala and hippocampus, that portion of the midbrain responsible for emotion and memory with input from the visual, auditory, and somatosensory systems.[30] In PD, the amygdala misinterprets sensations from the body, leading to the characteristic extreme reactions.

Treatment for PD consists of medication either by itself or in combination with CBT. Meta analysis has shown that a combination is most effective,[31] although SSRIs have many possible side effects that may affect the oral health of patients, including xerostomia, glossitis, gingivitis, stomatitis, dizziness, headache, and loss of the sense of taste.[32] Suicide has also been reported. In addition, interactions are also possible between SSRIs and erythromycin or codeine.

Depression

Depression is one of the most common psychiatric disorders, affecting an estimated 20% of the population at sometime in their lives.[33] The course of depression may vary widely; it may affect a patient once or recur; it can appear gradually or suddenly and can last a few months or a lifetime. Not only are patients with depression at higher risk for suicide, they also have a higher mortality rate from other causes such as accident, trauma, or homicide. Depression is the leading cause of disability in North America.

Depression can take many forms, but common symptoms, lasting for at least 2 weeks, are a pervasive low mood, a loss of interest in usual activities, significant (5%) weight gain or loss, change in sleep patterns, loss of energy, persistent fatigue, recurrent thoughts of death, and a diminished ability to enjoy life.[34] Adolescents are more apt to be irritable and act out when they are depressed, but patients generally report feeling empty and anxious, with fatigue and decreased energy. It is sometimes difficult to distinguish between "normal" or situational depression, which is a natural response to trauma or illness, and clinical depression, which may be related to underlying endogenous factors. One distinguishing characteristic of normal depression is that these patients still can communicate, make their own decisions, and participate in their own care. Patients with pathologic depression have symptoms that are out of proportion to the circumstances.

The cause of depression is linked to a lack of stimulation of the postsynaptic neurons in the brain. There is an increase in monoamine oxidase (MAO) A, an enzyme that decreases the concentration of serotonin and other monoamines that help maintain a positive mood. As with other psychological disorders, there is a genetic component, although it is poorly defined.[35]

Because orthodontic patients come regularly for appointments and usually interact with the orthodontist and office staff, orthodontists are in a good position to notice whether their adolescent patients exhibit such symptoms or signs of depression. Orthodontists should be particularly attentive to patients who have dropped out of their normal activities, changed their appearance, report insomnia, have abrupt deterioration in academic performance in conjunction with a lack of interest in their usual activities, or show signs of drug or alcohol abuse.

Treatment for depression, as with other disorders, consists of a variety of drugs and psychotherapy.[32,35] In addition to pharmacologic and psychological intervention, alternative therapies such as electroconvulsive therapy, hypnotherapy, meditation, and diet therapy have been suggested. Hospitalization may become necessary if suicide is a possibility. Drugs currently used for depression include SSRIs such as sertraline (Zoloft), fluoxetine (Prozac), citalopram (Celexa), and paroxetine (Paxil); MAO inhibitors; and dopamine reuptake inhibitors such as bupropion (Wellbutrin and Zyban). SSRIs have not been found to work significantly better than placebo for moderate depression but have been found to be effective for severe depression. MAO inhibitors are effective but can have interactions with decongestants or tyramine-rich foods such as cheese. Dopamine reuptake inhibitors are better than SSRIs for fatigue and insomnia.

Nondrug therapies for depression, such as CBT, are directed at helping the patient learn to cope with their symptoms and improve interpersonal communication. In supportive therapy, patients may also discuss their problems with others who can share strategies for coping with their illness. In family therapy, the entire family learns how to undo patterns of destructive behavior.[35]

Eating Disorders

Eating disorders, including anorexia nervosa or bulimia nervosa, affect up to 2% of adolescent and young adult females, although they can affect both sexes at many ages.[34] The fundamental defect lies in the distorted body image that leads patients to control their weight by extreme dieting or vomiting. Patients usually go to great lengths to hide their symptoms and behaviors, so that close family members often are unaware of their existence. Bulimia and anorexia can lead to severe metabolic disturbances and even death, and thus they require treatment.

Both anorexia and bulimia have oral manifestations. Bulimia may lead to dental erosions, which can be sometimes be noted as extruding amalgams, as well as dentinal hypersensitivity and salivary gland hypertrophy. Both conditions can be accompanied by cheilosis.

If orthodontic patients are suspected of having an eating disorder, these concerns should be addressed directly. Therapy for eating disorders consists of CBT, so that patients can develop realistic ideas about how much they should eat, what is good nutrition, and their own body image. SSRIs can also be used.

Personality Disorders

While depression, BDD, and OCD are classified by the *DSM-IV* as Axis I disorders and are predominantly related to mood, personality disorders are classified as Axis II disorders (i.e., disorders that involve maladaptive behaviors and patterns of thinking that lead to problems at home, school, and work). Personality disorders most frequently seen are the narcissistic personality, borderline personality disorder (BPD), and the antisocial personality disorder (APD). It is estimated that the prevalence of these personality disorders ranges from 4.4% to 13% in the United States.[36] Environmental influences such as prior abuse, poor family support, family disruption, and peer influences, as well as biological causes, are important risk factors for the development of such disorders.

Patients with narcissistic personalities believe that they are special and therefore entitled to special treatment. The typical narcissistic patient has a very brittle self-esteem and a strong need for approval, which are manifested as arrogance and demands for special attention. These patients are thus more intolerant of minor complications and are more likely to seek legal recourse when dissatisfied.

BPD has an estimated prevalence of 0.7% to 2.0%.[36] It is characterized by erratic moods, impulsivity, and poorly controlled anger. These behaviors can lead to unstable relationships and chronic interpersonal problems. One interesting feature is that patients with BPD often begin treatment with an extremely positive view of the orthodontist but, with treatment, quickly changes to hatred and anger in response to complications.

APD affects more males than females by a ratio of 4 or 5:1, with an overall prevalence of 2% to 3%.[36] Those affected by APD exhibit unacceptable behavior such as lying, theft, destructive behavior, and aggression to people and animals, accompanied by a lack of remorse.

Patients with any form of personality disorder can be difficult to manage in an orthodontic office, being disruptive and trying for clinicians. Such individuals may be hard to identify or label, even though they may not be compliant and may show some signs of depression or substance abuse and even attempted suicide. Staff members need to handle these patients with evenhandedness, not allowing them to disrupt the office procedures or abuse office personnel. Orthodontists should beware of excessively dependent or manipulative behaviors, which can cause conflict among staff members. If necessary, care can be discontinued and the patient dismissed.

"Difficult" Patients

As noted earlier, patients with no known psychopathology can still be difficult to manage, exhibiting a number of different behaviors that are disruptive, hostile, or otherwise difficult for the orthodontist to handle. According to Groves,[37] they can be categorized into four distinctive types:

- *Dependent clingers* have needs for reassurance from their caregiver that escalate. Patients are initially reasonable in their needs but become progressively more helpless, ultimately becoming totally dependent upon their doctors. These patients must be given appropriate limits with realistic expectations. Clear verbal and written instructions can be helpful in reinforcing the limits of patient access to the professional staff.
- *Entitled demanders* are also needy but manifest it as intimidation and attempts to induce guilt. They have a need to control the situation and often make threats, either overt or implied, in order to get what they want. Their aggressive behavior may be due to feelings of dependency and fear of abandonment. These patients are best dealt with by validating anger but redirecting the feelings of entitlement to realistic expectations of good care. Again, limits must be placed so that office procedures are not disrupted.
- *Manipulative help-rejecters* focus on their symptoms but are resigned toward failure. They seem satisfied with a lack of improvement. Clearly, these patients who are difficult to treat must be involved in all decisions and should have regular appointments. Because they must either agree to all treatment or choose not to proceed, the orthodontist does not have the responsibility for the success of the treatment.
- *Self-destructive deniers* take pleasure in defeating any attempts to help them. They do not seem to want to improve. These patients may be sufficiently depressed to consider not rendering or limiting treatment.

In general, all demanding and needy patients should have limits placed on their behavior at the time when treatment alternatives are discussed. Orthodontists should not promise too much in describing treatment plans and outcomes. They should describe how they would address the orthodontic needs, noting potential problems and obstacles to the treatment, and explain how progress might be evaluated. In fact, some variation of the Perceptometrics method might be helpful to present patients with an acceptable range of suggested treatment outcomes (Giddon DB, personal communication). This will minimize the possibility that patient expectations are too high. As Groves commented, "Difficult patients are typically those patients who raise 'difficult' feelings within the clinician."[37] Orthodontists must learn to address these difficult feelings and deal with them. The orthodontist should remain friendly, unemotional, and professional at all times. Emotional outbursts should be responded to with an acknowledgment of feelings but an expectation of appropriate behavior. Noncompliance must be countered with an appropriate alternative treatment plan. The clinician and staff must avoid being provoked and remain professional and emotionally neutral while maintaining a correct office atmosphere.

PATIENTS HAVING ORTHOGNATHIC SURGERY

Psychosocial aspects of orthognathic surgery patients, who receive orthodontic treatment combined with jaw surgery, deserve special consideration because their treatment often involves changes in appearance which may or may not be the motivation for treatment as well as the fact that surgery entails risks beyond those of orthodontics alone.

Psychological Status and Motivation

When considering the success of combined surgical-orthodontic treatment, there are several aspects to consider. First, what is the psychological and physical situation of the patients that brings them to the point of considering having jaw surgery? What motivates patients to consider this combined treatment? Is their decision a normal reaction to a debilitating dentofacial deformity, or do they have an abnormal perception that surgery will not correct?

Early studies into this area were prompted by reports in the plastic surgery literature which found that many patients seeking plastic surgery had significant psychiatric problems.[38-41] However, two studies examining the psychological profile of

orthognathic patients found that these patients were psychologically normal.[42,43] Although earlier studies had shown that orthognathic patients typically did not suffer from depression or anxiety, Alanko et al., sought to determine whether orthognathic patients were psychologically different from patients who had not been referred for orthognathic treatment.[44] They identified 60 patients with a mean age of 17 and asked them to complete five questionnaires and a structured diary which elicited information regarding their quality of life, experiences with bullying, self-image, and self-esteem. Their results were compared with those from questionnaires completed by a matched group of patients who had not been referred for orthognathic treatment. Each participant was also asked to rate their own dental appearance on a visual analog scale. An objective assessment of each participant's occlusion was provided by an orthodontist using the same scale and study models. The authors determined that those patients who rated their dental aesthetics lower had lower orthognathic quality of life scores and poorer body image than the control subjects, while those who rated their dental esthetics higher only had poorer oral function. The authors concluded that many patients cope very well with their deformities, and that their perception of their dental appearance is a key influence in determining the patients' reaction to their deformity.

The issue of motivation was explored by Ryan et al.,[45] who prospectively interviewed 18 patients, 18 to 40 years old, before having orthognathic surgery. The interviews revealed that the deformity affected the patients in either practical or psychological ways, or in a combination. Practical effects included functional problems including difficulty eating, speaking, or issues such as lip entrapment or biting the cheeks, or structural problems such as asymmetries or overjet. Psychological problems included feeling helpless about their appearance, avoiding social situations, and not applying for jobs due to low self-esteem and fear of rejection. It was noted that those who had functional concerns only did not have low self-esteem. Although they had feelings of embarrassment, these were not related to problems of self-image. Patients coped with these problems in two ways: either avoiding confronting the problem (including social isolation), or continuing normal activities while modifying their behavior to minimize the impact of their condition. This type of behavior modification included altering their rest position to mask their true bite, and not posing for photos. Motivation for treatment has traditionally been classified as external or internal, but this study discussed the practical and psychological motivations that led from the impacts described above. Both factors resulted in the patients deciding that they wanted a change in their lives, whether that meant feeling more confident about themselves or being able to chew more effectively. The authors stressed that both external and internal motivations were present, but that many patients reported a combined external/internal motivation. Although this study did not identify a clear reason for the dissatisfaction of some patients toward their surgery, it very clearly demonstrates that the degree of impact deformities can have on a person's life is not necessarily proportional to the physical deformity, and that the problem as perceived by the patient may be quite different in type and degree than that observed by the clinician.

Quality of life is defined as individuals' perception of their position in life in the context of the culture and value system in which they live and in relation to their goals, expectations, standards, and concerns.[46] This concept that has been increasingly applied to assess how a peoples' physical problems influence their overall well-being; in this context, how dentofacial deformities affect a person's quality of life.

Lee et al.[47] studied patients with dentofacial deformity in order to determine and compare the effect of dentofacial deformity on the patients' quality of life. They asked 76 patients who were referred for orthognathic surgery to complete three different questionnaires that included a generic health-related questionnaire, a generic oral health–related questionnaire, and a condition-specific quality of life measure. Their responses were compared with those from a control group of patients who were referred for removal of asymptomatic third molars. Lee found that in the generic health-related questionnaire, there were no differences in any domain of general health, such as physical functioning, pain, mental health, vitality, and social functioning. However, they found significant differences in the overall oral health quality of life questionnaire in such areas of functional limitation, psychological discomfort, psychological disability, and handicap, with specific problems in areas such as being able to pronounce words properly, being self-conscious or embarrassed because of the mouth or teeth, and feeling that life in general was less satisfying because of problems with the teeth or mouth. Finally, the condition-specific quality of life inventory demonstrated significant differences in five areas, including reporting significantly more problems biting and chewing, and reporting significantly more often disliking seeing a side view of the face, having their photograph taken, or being seen on video. Clearly, these results indicate that patients with dentofacial deformities have a poorer quality of life in a variety of aspects.

De Avila et al.[46] also assessed 50 patients one year before undergoing orthognathic surgery to determine if they exhibited any signs of depression in greater frequency than a nonsurgical population. The patients were given modified QOL (quality of life) questionnaires as well as the Beck Depression Inventory. The adapted questionnaires included 36 questions that evaluated how the physical health influenced the patient's daily life, including emotional state, social activities, energy, and mood. The Beck Depression Inventory asked the patient to rate 21 questions as to how often something bothered the patient (never, occasionally, fairly, and often). The patient was then judged either to have some degree of depression or not depending on the total score. Using this method, 19 patients were judged to have some degree of depression. De Avila noted that the diagnosis of depression was positively correlated with a lack of energy ($p < 0.001$), less social activity ($p = 0.011$), and poorer mental health ($p = 0.008$).This study emphasizes the interrelationships between aspects of mental health and physical health and underlines the necessity of carefully screening patients for potential problems when considering orthognathic surgery.[46]

Yu et al. used QOL instruments to determine motivations for orthognathic surgery in Chinese patients.[48] Two hundred ten healthy patients completed a questionnaire that included self-esteem measures as well as oral health QOL items. Their responses were compared with those from 219 individuals who were not undergoing surgery. Both males and females listed improvement of their facial appearance as the #1 reason for having orthognathic surgery. Men listed occlusion second and self-confidence third, whereas women reversed the positions of these two motivations. Self-esteem was significantly lower in the patient group. The authors concluded that attention needs to be paid to social and cultural motivations of the patient.

Expectations

Regardless of patients' reasons for undergoing orthognathic treatment, patients' ultimate satisfaction with the outcome can often be related to what they expect to gain from the treatment. Ryan interviewed 18 adult patients and found two broad categories of expectations: those related to changes in physical features, and those related to the effect these changes would have on their lives.[49] The authors found that the patients fell into four categories that had been earlier delineated by Ritchie: metamorphosizers, pragmatists, shedders, and evolvers.[50] *Metamorphosizers* had expectations of both physical and psychological problems being fully corrected by the surgery. These patients would be more likely to be dissatisfied and may require more exploration of their expectations. *Pragmatists* expect a physical but not psychological change since they see the impact of the deformity as totally physical. However, these patients may have lower satisfaction because the physical changes may not happen to the extent expected, and there may be unforeseen emotional changes that trouble the patient. A third type of patient, *shedders*, expect little physical change but profound psychological change. These patients also require careful counseling to determine their true motivations and expectations. Finally, *evolvers* have very low expectations for both physical and psychological changes. This, too, poses some risk for dissatisfaction for several reasons: first, there may be more changes, both physical and emotional, than the patient was ready for. Furthermore, without positive expectations, patients may find the postoperative course difficult to manage.

In this study, the investigators made no distinction between realistic and unrealistic expectations, since this would require that judgments be made regarding the likelihood of changes occurring. However, clinicians need to probe in some detail as to what the expectations of the patients are and, in their judgment, whether the expectations are realistic. This will require that the clinician spend as much time as necessary with patients discussing their perceptions and motivations, and a willingness to listen objectively and openly.

Another method of examining expectations was used by Bullen et al.[51] They constructed an average patient profile that was then altered with incremental movements of the lips to create a 13-photo sequence showing gradual differences in lip position. Eighty-five patients who were either in active orthodontic treatment or considering orthodontic and/or orthognathic treatment were then given a photo of their own profile as well as a questionnaire that included the series of constructed profiles. First, the patients were asked how satisfied they were with their current profile. The patients were then asked to choose which of the 13 profiles in the constructed series most closely matched their own. The profile chosen was compared to the individual's profile to measure the difference between the patient's real and perceived profile. The authors found that while the younger patients (15 to 25 years) thought that their lips were more retrusive than they actually were, older patients (over 25 years of age) thought that their lips were significantly more protrusive than they actually were. The authors concluded that older patients, in whom soft tissue changes have continued throughout their adult life, may need additional efforts to make sure they have an accurate view of their facial profile, and thus have an accurate expectation of the outcomes of orthognathic surgery.

Satisfaction

The ultimate goal of orthognathic surgery is patient satisfaction. It might be said that even with a technically successful procedure, success has not been achieved without a satisfied patient. Thus, the factors determining patient satisfaction need to be determined so that, where possible, they can be ensured.

Kiyak et al. were among the first to comprehensively examine patient satisfaction with orthognathic surgery. They gave questionnaires to 74 consecutive orthognathic surgery patients at six time points, from before surgery to 24 months postoperatively. They found that at 1 month and 4 months postoperatively, those who experienced less pain and numbness than expected had a higher self-esteem and were overall more satisfied than those who had more pain and numbness than expected. However, although the levels of satisfaction and self-esteem rose after surgery from 1 to 2 days to 3 weeks to 4 months, the levels were uniformly lower at 9 months. The authors hypothesized that by 9 months, patients might see their results as permanent, so that any problems that remained would decrease their sense of satisfaction.[52] By 24 months, however, the various categories of self-esteem had rebounded, though not quite to preoperative levels, while patient satisfaction was at its highest level. The authors concluded that changes resulting from the orthognathic surgery may persist for at least 2 years and suggested that clinicians work with patients long term to make sure they have adapted well to the surgical outcome.[53]

In 2007, Findlay et al. evaluated the satisfaction of 61 orthognathic patients to determine whether the surgery had any influence on the patients' self-esteem.[54] Preoperatively, patients were given three questionnaires that assessed the patient's extraversion/introversion status, general health, and feelings toward their bodies. They were also asked questions surrounding their motives and expectations with regards to the surgery. At three time points postoperatively, the patients were given some or all of these questionnaires once again to examine changes. They found that 87% of the patients were satisfied with their result. Those who were not satisfied had neuroticism scores on the personality tests that were significantly higher. No associations between satisfaction and general health, sex, extroversion, or self-esteem were found. All dissatisfied patients reported experiencing more pain, swelling, numbness, and scarring than expected, in contrast to the large majority of the group at large. Forty two percent of all patients felt that they had received too little information 3 months after surgery, but gradually this decreased to 26% at 1 year.

Cunningham et al. conducted a similar study, querying 83 preoperative and 100 postoperative patients to determine self-esteem and level of postoperative satisfaction.[55] They found that 95% of patients were satisfied with their treatment at least 9 months after surgery. The reasons given for dissatisfaction were inadequate preparation for postoperative problems and one case of not having as great a change as expected. There was no significant difference in self-esteem between the pre- and postoperative groups. The authors concluded by recommending that preoperative patients be given advice and information regarding the postoperative course and potential problems both verbally and in writing to help prevent dissatisfaction that may persist.

Lee et al. tracked changes in quality of life in 36 patients preoperatively as well as 6 weeks and 6 months postoperatively.[56] A significant increase in quality of life was revealed immediately after surgery as well as long term (Fig. 10-2). Both general mental and physical scores improved after 6 weeks as well as specific improvements in facial aesthetics, psychological discomfort, and social functioning. As would be expected, there was an increase in functional limitation, which later decreased far beyond the preoperative level. Clearly, patients benefit in a variety of ways from orthognathic surgery.

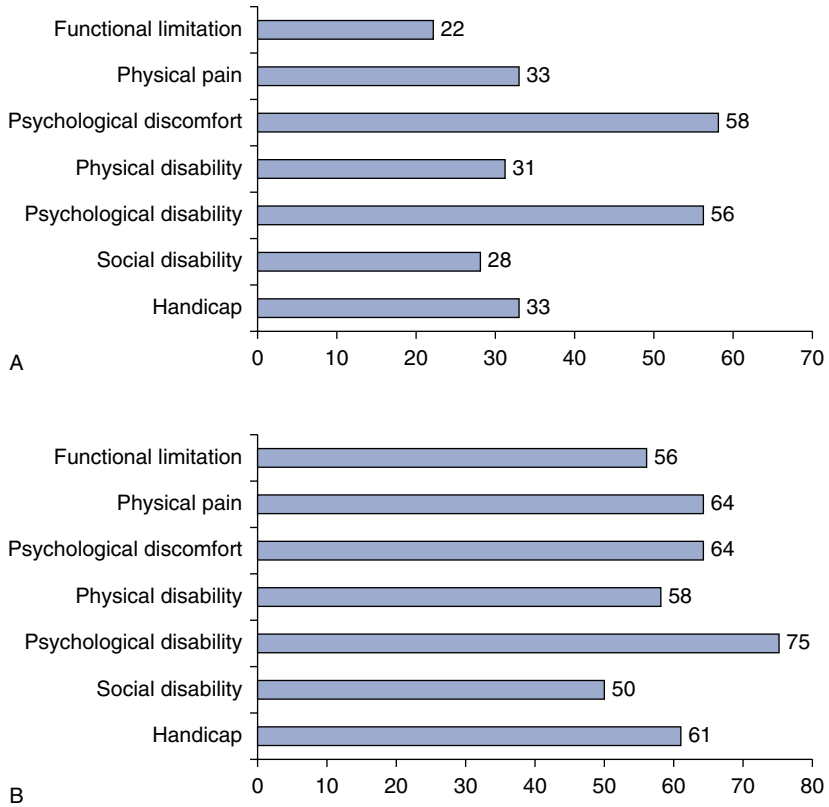


FIGURE 10-2 A, Percentage of subjects with decrease in OHIP-14 score at 6 weeks. B, Percentage of subjects with decrease in OHIP-14 score at 6 months. (From Lee S, McGrath C, Samman N: Impact of Orthognathic Surgery on Quality of Life, J Oral Maxillofac Surg 66(6):1194-1199, 2008.)

Phillips et al. more closely evaluated patients' postoperative course by asking 170 patients to complete a 20-item health-related QOL survey each postoperative day for 90 days.[57] One hundred seventy patients completed at least 30 days' of questionnaires and brought them to clinic visits at 1, 4-6, and 12 weeks. By the end of the first postoperative week, most patients had little postoperative sequelae such as bruising or food collection in the incision. Fewer than 7% reported any significant problems after 2 weeks. Pain and discomfort lasted 2 to 3 weeks after surgery for most patients, although 20% of patients still were taking pain medication after 1 month. The return to daily activities followed a similar pattern with 85% reporting no substantial problems with their daily life by the end of the first month. The problem that lasted the longest was oral function. Problems with chewing persisted significantly longer than problems with opening or eating. By 64 days postoperatively, 75% of patients reported no or slight problems with opening and eating, but the same level of recovery was not reported for chewing until day 70. The authors recommended that similar diaries be routinely completed by all patients as a way for clinicians to track the patient's recovery and quality of life.

Perhaps the ultimate indicator of treatment success is the long-term benefit experienced by patients. Motegi et al. evaluated 93 patients who had received bilateral sagittal split osteotomy for a Class II malocclusion preoperatively as well as 2 and 5 years postoperatively.[58] They evaluated health-related abilities, daily activities, and current symptoms. They found that after both 2 and 5 years, there was significant improvement in all areas. Although there was no control group employed, it was clear that for this group of patients, there was a long-term benefit to orthognathic surgery.

Lazaridou-Terzoudi et al.[59] also looked at long-term benefits as they surveyed 117 patients who had orthognathic surgery 10 to 14 years earlier to assess the patients' perceptions of problems with functioning, socializing, eating, sleeping, and other aspects of self-image and quality of life. The results were compared with two control groups: one group waiting for orthodontic treatment, and a group of comparably aged adults not seeking treatment. They found a linear improvement in all four subscales of function, health, interpersonal relations, and appearance from preoperative state through immediate postoperative state to the present. Compared with both control groups, the orthognathic surgery group reported current functioning in the four areas that was higher than either control group, but their reported level of functioning before surgery was lower than the nontreatment controls in all four areas, and lower than patients awaiting treatment with motivations in appearance and interpersonal relations (Fig. 10-3). The authors also looked at the patients' body image by having them rate their feelings toward different aspects of their physical appearance. In contrast to the improvement of quality of functioning reported in the earlier subscales, the orthognathic patients had lower overall facial body image than either control group, but similar assessments of total body image. In assessing the influence of age on the long-term results, the authors found that the youngest patients, who had undergone surgery in their teens, were significantly less likely to be satisfied with the overall result and the least satisfied with their current appearance. This is consistent with a report by Cunningham et al. that patients in their thirties are more satisfied with their appearance after surgery.[60] In summary, the authors concluded that the improvement in appearance that results

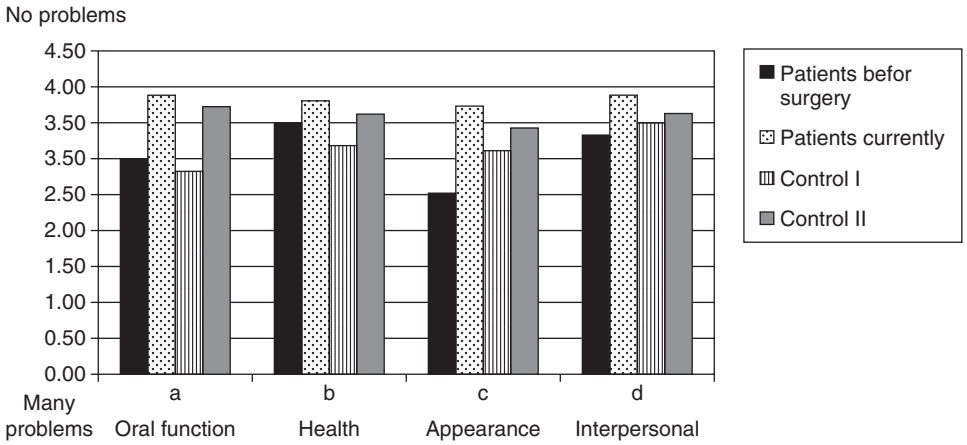


FIGURE 10-3 "Problems" for patients versus controls ([a]*F* =16.78, P = .001; [b]*F* = 7.03, P = .001; [c]*F* =7.08, P =.001; d*F* = 4.25, P =.001, comparison of current status across groups). (From Lazaridou-Terzoudi T, Asuman Kiyak H, Moore R, et al.: Long-term Assessment of Psychologic Outcomes of Orthognathic Surgery, J Oral Maxillofac Surg 61(5):545-552, 2003.)

from orthognathic surgery is associated with improvement in psychosocial adjustment, and that patients should be offered appropriate treatment to address such handicaps.

Soh and Narayanan carried out a systematic review of 21 studies that assessed patient motivations, perceptions, or postoperative changes in QOL in an attempt to objectively evaluate all methods of appraising the quality of life of orthognathic patients.[61] Their conclusions can serve to summarize all the studies discussed here:

1. Orthognathic surgery does result in an improved quality of life. The biggest improvements are seen in the social/emotional and psychological areas.
2. The use of validated QOL questionnaires has significantly improved the ability of clinicians to evaluate details of the affect of surgery on patients.
3. Aesthetic concerns as well as occlusion are the main motivations for patients to undergo orthognathic surgery.
4. When patients are dissatisfied with the outcome of their surgery, most of these complaints are due to short-term surgical sequelae, which can take months to resolve. However, some patients have expectations that are not realistic, and these need to be detected before surgery to attempt to prevent dissatisfaction with outcome.

In conclusion, significant time needs to be taken to understand the patient's motivation for treatment and what they expect. Quality-of-life questionnaires and other instruments may be helpful in gathering information. All patients need to be given detailed information during consultations as well as in writing in order to obtain the best possible outcome and greatest patient satisfaction from surgery.

PATIENTS WITH CRANIOFACIAL DEFORMITIES

Psychological Issues

Orthodontists see patients with facial deformities or other disfigurement. Their orthodontic treatment may be a part of a coordinated surgical-orthodontic plan to address the deformity or may be isolated. Whatever the circumstances, these patients will be fundamentally different psychologically from patients who are not deformed. Pertschuk and Whitaker[62] compared a group of 43 patients with craniofacial anomalies between the ages of 6 and 13 with children matched for age, sex, intelligence, and family income to determine differences in psychological functioning before surgery. They studied levels of anxiety, self-concept, social experiences, intelligence, and personality characteristics and found that the craniofacial patients were more anxious and more introverted and had a poorer self-concept. Their parents reported teasing from peers about their facial appearance. Treatment was being sought out of a desire to improve appearance, but patients could not specify what they wanted to change. When these patients were evaluated 12 to 18 months after surgery, they demonstrated reduced levels of anxiety but more negative social interactions, possibly because their social skills had not developed as the patients grew. Pertschuk and Whitaker thus concluded that modest improvements in psychological adjustment were due to craniofacial surgery.

Pillemer and Cook[63] evaluated 25 patients aged 6 to 16 years at least 1 year after craniofacial surgery and found that these children still exhibited an inhibited personality style, low self-esteem, impaired peer relationships, and greater dependence on significant adults. They concluded that treatment per se does not solve psychological issues, suggesting that long-term follow-up and support from interdisciplinary teams may be of greater benefit.

Sarwer et al.[64] examined 24 adults born with a craniofacial anomaly in terms of their body image dissatisfaction, self-esteem, quality of life, and experiences of discrimination and compared them with an age- and gender-matched control group who were not disfigured. The adults with craniofacial disfigurement reported significantly greater dissatisfaction with their facial appearance, a significantly lower self-esteem, and a significantly lower quality of life than the control group. It is interesting to note that dissatisfaction with facial appearance was correlated with the degree of residual facial deformity. It should also be noted that these problems are by no means universal among adults with facial deformities.

These studies point out the psychological differences in patients whose self-image is intrinsically different. Clinicians should realize that these patients have a much different perspective on orthodontic treatment. Their expectations of treatment outcome may therefore also be quite different, and this should be explored when orthodontic treatment is discussed.

Patients with Clefts of the Lip and Palate

The most common facial deformity that orthodontists will see is cleft lip and palate. Considerable research has been

done examining the psychological aspect of this congenital anomaly. Kapp-Simon[15] explored whether primary school-age children with clefts have negative self-concepts by using the Primary Self-Concept Inventory (PCSI) and compared the results from tests administered to 172 children without noncleft children. Kapp-Simon[15] found that the children with clefts had low self-concepts based on the following areas: social self, emotional state, and helpfulness. It is apparent that this group of children feels stigmatized by their cleft from a very early age and therefore can be expected to have a much different attitude toward orthodontic treatment from those without clefts.

More recently, Broder et al. assessed the quality of life of patients with clefts using the Child Oral Health Impact Profile scale.[65] This instrument is a 34-question inventory that has been validated for use with children and was given to patients aged 7 to 19 years at six craniofacial centers around the United States. It asks the degree to which the patient has difficulty with a variety of items in five general areas: oral symptoms, functional well-being, social-emotional well-being, school/environment, and self-esteem. It was found that those patients who needed surgery had a lower quality of life in all aspects except for self-esteem, which is consistent with earlier reports that the majority of patients with clefts do not "appear to experience major psychosocial problems."[66] The authors also found that with increasing age, girls had a greater reduction in quality of life than boys. This was thought to be due to the greater importance that attractiveness has for girls during adolescence and underscores the importance of listening carefully to patients' concerns during orthodontic visits.

Patients with Acquired Deformities

An important subset of patients with facial deformities includes those who were born without dentofacial deformity but acquired it after birth from trauma, tumor, or disease. Because these patients had normal faces or occlusions prior to the insult, they may have more psychological stress in adapting to their acquired dysmorphic appearance than those patients who have never known any other morphology. Patients who have congenital anomalies have had time to incorporate the defect into their body image,[67] but patients who suddenly lose their normal appearance have a much more difficult time coping with their deformity. Consequently, their expectations from treatment may be quite different from those patients who have never had a "normal" face. Because the responses to acquired or congenital deformities may differ, it is critical that the patient's expectations and wishes be discussed in detail and that the orthodontist discusses realistically what can be achieved.

De Sousa reviewed, in some detail, the psychological issues that may arise with reconstructive oral and maxillofacial surgery.[68] In addition to craniofacial anomalies, mentioned above, facial trauma can create significant problems for patients, not only because of the adaptation required to abrupt changes in facial appearance, but also due to posttraumatic stress disorder. Between 20% and 30% of patients with facial injuries may experience intrusive and disturbing thoughts, reexperiencing the trauma, sleep disturbances, irritability, or depression related to the original injury. In addition, brain injury may have also occurred during the trauma. These patients may need management by a team of physicians including neurologists, neuropsychologists, and psychiatrists to help the patient cope with and recover from the past trauma. Patients with facial cancer also experience anxiety and depression, although levels were found to be lower than in patients with facial trauma. It has been found that a patient's personality, coping ability, and social and family support are significantly related to how well a patient adjusts to their disease.[68] These patients also may need mental-health screening and care to recover psychologically.

SUMMARY

Every patient has individual perceptions, desires, needs, and related behavior, some of which are outside the usual experience of most orthodontists. It is hoped that the information provided in this chapter will help orthodontists to more effectively manage patients with abnormal or difficult perceptions and behaviors. Clear communication is critical when discussing orthodontic problems, proposed treatment, and treatment alternatives and expectations. In addition, the clinician must become completely familiar with the patient's medical and psychosocial history, needs, questions, and perceptions. The patient must also be given clear guidelines for office procedures. In this way, many problems arising from abnormal psychological problems and behaviors can be minimized.

REFERENCES

1. Mortensen MG, Kiyak HA, Omnell L. Patient and parent understanding of informed consent in orthodontics. *Am J Orthod Dentofacial Orthop*. 2003;124(5):541.
2. Tufekci E, Jahangiri A, Lindauer SJ. Perception of profile among laypeople, dental students, and orthodontic patients. Angle Orthod. 2008;78(6):983–987.
3. Giddon DB, Sconzo R, Kinchen JA. Quantitative comparison of computerized discrete and animated profile preferences. *Angle Orthod*. 1996;66(6):441–448.
4. Kitay D, BeGole EA, Evans CA, et al. Computer animated comparison of self perception with actual profiles of orthodontic and nonorthodontic subjects. *Int J Adult Orthodon Orthognath Surg*. 1999;14(2):125.
5. Arpino VJ, Giddon DB, BeGole EA, et al. Presurgical profile preferences of patients and clinicians. *Am J Orthod Dentofacial Orthop*. 1998;114(6):631.
6. Hier LA, Evans CA, BeGole EA, et al. Comparison of preferences in lip position using computer animated imaging. *Angle Orthod*. 1999;69(3):231.
7. Miner RM, Anderson NK, Evans CA, et al. The perception of children's computer-imaged facial profiles by patients, mothers, and clinicians. *Angle Orthod*. 2007;77(6):1034.
8. Mejia-Maidl M, Evans CA, Viana G, et al. Preferences for facial profiles between Mexican Americans and Caucasians. *Angle Orthod*. 2005;75(6):953.
9. Park YS, Evans CA, Viana G, et al. Profile preferences of Korean American orthodontic patients and orthodontists. *World J Orthod*. 2006;7(3):286.
10. McKoy-White J, Evans CA, Viana G, et al. Facial profile preferences of black women before and after orthodontic treatment. *Am J Orthod Dentofacial Orthop*. 2006;129(1):17.
11. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5). 5th ed. Washington: American Psychiatric Association; 2013.

12. Child Trends. *ADHD*; 2014. Available at: http://www.childtrends.org/?indicators=ADHD.
13. Friedlander AH, Yagida JA, Mahler ME, et al. The pathophysiology, medical management, and dental implications of adult attention-deficit/hyperactivity disorder. *J Am Dent Assoc*. 2007;138(4):475–482.
14. Tripp G, Wickens JR. Neurobiology of ADHD. *Neuropharmacology*. July 21, 2009;57(7–8).
15. Kapp-Simon K. Self-concept of primary-school-age children with cleft lip, cleft palate, or both. *Cleft Palate J*. 1986;23(1):24.
16. Arnold LE, Hodgkins P, Caci H, Kahle J, Young S. Effect of treatment modality on long-term outcomes in attention-deficit/hyperactivity disorder: a systematic review. *PLoS One*. February 25, 2015;10:e0116407. http://dx.doi.org/10.1371/journal.pone.0116407.
17. Zohar AH. The epidemiology of obsessive-compulsive disorder in children and adolescents. *Child Adolesc Psychiatr Clin N Am*. 1999;8:445.
18. Fornaro M, Gabrielli F, Albano C, et al. Obsessive-compulsive disorder and related disorders: a comprehensive survey. *Ann Gen Psychiatry*. 2009;8:13.
19. Samuels JF. Recent advances in the genetics of obsessive-compulsive disorder. *Curr Psychiatry Rep*. 2009;11(4):277.
20. Chandna P, Srivastava N, Adlakha VK. Obsessive compulsive disorder in a dental setting. *J Indian Soc Pedod Prev Dent*. 2014;32:330–332.
21. Veale D, Boocock A, Gournay K, et al. Body dysmorphic disorder. A survey of fifty cases. *Br J Psychiatry*. 1996;169:196–201.
22. Anthony MT, Farella M. Body dysmorphic disorder and orthodontics – an overview for clinicians. *Aust Orthod J*. 2014;30:208–213.
23. Phillips KA. Body dysmorphic disorder: the distress of imagined ugliness. *Am J Psychiatry*. 1991;148:1138.
24. Conroy M, Menard W, Fleming-Ives K, et al. Prevalence and clinical characteristics of body dysmorphic disorder in an adult inpatient setting. *Gen Hosp Psychiatry*. 2008;30(1):67.
25. Jakubietz M, Jakubietz RJ, Kloss DF, et al. Body dysmorphic disorder: diagnosis and approach. *Plast Reconstr Surg*. 2007;119(6):1924.
26. Phillips KA. Body dysmorphic disorder: recognizing and treating imagined ugliness. *World Psychiatry*. 2004;3(1):12.
27. Polo M. Body dysmorphic disorder: a screening guide for orthodontists. *Am J Orthod Dentofacial Orthop*. 2011;139:170–173.
28. Friedlander AH, Friedlander IK, Marder SR. Bipolar I disorder. *J Am Dent Assoc*. 2002;133(9):1209.
29. Donner J, Pirkola S, Silander K, et al. An association analysis of murine anxiety genes in humans implicates novel candidate genes for anxiety disorders. *Biol Psychiatry*. 2008;64:672–680.
30. Hamilton SP, Fyer AJ, Durner M, et al. Further genetic evidence for a panic disorder syndrome mapping to chromosome 13q. *Proc Natl Acad Sci U S A*. 2003;100(5):2550–2555.
31. Hofmann SG, Sawyer AT, Korte KJ, et al. Is it beneficial to add pharmacotherapy to cognitive-behavioral therapy when treating anxiety disorders? A meta-analytic review. *Int J Cogn Ther*. 2009;2(2):160–175.
32. Becker DE. Psychotropic drugs: implications for dental practice. *Anesth Prog*. 2008;55:89–99.
33. Kessler R, Walters E. Epidemiology of DSM-III-R major depression and minor depression among adolescents and young adults in the National Comorbidity Survey. *Depress Anxiety*. 1998;7:3–14.
34. Neeley II WW, Kluemper GT, Hays LR. Psychiatry in orthodontics. Part I: typical adolescent psychiatric disorders and their relevance to orthodontic practice. *Am J Orthod Dentofacial Orthop*. 2006;129(2):176.
35. Friedlander A, Mahler M. Major depressive disorder: psychopathology, medical management, and dental implications. *J Am Dent Assoc*. 2001;132:629–638.
36. Coid J. Epidemiology, public health and the problem of personality disorder. *Br J Psychiatry*. 2003;44(suppl):S3–S10.
37. Groves JE. Taking care of the hateful patient. *N Engl J Med*. 1978;298(16):883.
38. Meyer E, Jacobson WE, Edgerton MT, Canter A. Motivational patterns in patients seeking elective plastic surgery. *Psychosom Med*. 1960;22:193–201.
39. Hay GG, Heather BB. Changes in psychometric test results following cosmetic nasal operations. *Br J Psychiatry*. 1973;122:89–90.
40. Hay GG. Psychiatric aspects of cosmetic nasal operations. *Br J Psychiatry*. 1970;116:85–97.
41. Edgerton MT. Motivational patterns of patients seeking cosmetic surgery. *Plast Reconstr Surg*. 1971;48:551–557.
42. Peterson L, Topazian R. Psychological considerations in corrective maxillary and mid-facial surgery. *J Oral Surg*. 1976;34:157–164.
43. Kiyak HA, McNeill RW, West RA, Hohl T, Bucher F, Sherrick P. Predicting psychologic responses to orthognathic surgery. *J Oral Maxillofac Surg*. 1982;40:150–155.
44. Alanko OME, Svedström-Oristo A, Peltomäki T, Kauko T, Tuimisto MT. Psychosocial well-being of prospective orthognathic-surgical patients. *Acta Odontol Scand*. 2014;72:887–897.
45. Ryan FS, Barnard M, Cunningham SJ. Impact of dentofacial deformity and motivation for treatment: a qualitative study. *Am J Orthod Dentofacial Orthop*. 2012;141:734–742.
46. de Avila ED, de Molon RS, Monteiro Loffredo LC, Sgavioli Massucato EM, Hochuli-Vieira E. Health-related quality of life and depression in patients with dentofacial deformity. *Oral Maxillofac Surg*. 2013;17:187–191.
47. Lee S, McGrath C, Samman N. Quality of life in patients with dentofacial deformity: a comparison of measurement approaches. *Int J Oral Maxillofac Surg*. 2007;36:488–492.
48. Yu D, Wang F, Wang X, Fang B, Shen SG. Presurgical motivations, self-esteem, and oral health of orthognathic surgery patients. *J Craniofac Surg*. 2013;24:743–747.
49. Ryan FS, Barnard M, Cunningham SJ. What are orthognathic patients' expectations of treatment outcome – a qualitative study. *J Oral Maxillofac Surg*. 2012;70:2648–2655.
50. Ritchie J, Lewis J. *Qualitative Research Practice*. London: Sage Publications; 2003.
51. Bullen RN, Kook Y-A, Kim K, Park JH. Self-perception of the facial profile: an aid in treatment planning for orthognathic surgery. *J Oral Maxillofac Surg*. 2014;72:773–778.
52. Kiyak HA, West RA, Hohl T, McNeill RW. The psychological impact of orthognathic surgery: a 9-month follow-up. *Am J Orthod*. 1982;81:404–412.
53. Kiyak HA, Hohl T, West RA, McNeill RW. Psychologic changes in orthognathic surgery patients: a 24 month follow up. *J Oral Maxillofac Surg*. 1984;42:506–512.
54. Findlay PM, Atkinson JM, Moos KF. Orthognathic surgery: patient expectations; psychologic profile and satisfaction with outcome. *Br J Oral Maxillofac Surg*. 1995;33:9–14.
55. Cunningham SJ, Hunt NP, Feionmann C. Perceptions of outcome following orthognathic surgery. *Br J Oral Maxillofac Surg*. 1996;34:210–213.
56. Lee S, McGrath C, Samman N. Impact of orthognathic surgery on quality of life. *J Oral Maxillofac Surg*. 2008;66:1194–1199.
57. Phillips C, Blakey III G, Jaskolka M. Recovery after orthognathic surgery: short-term health-related quality of life outcomes. *J Oral Maxillofac Surg*. 2008;66:2110–2115.
58. Motegi E, Hatch JP, Rugh JD, Yamaguchi H. Health-related quality of life and psychosocial function 5 years after orthognathic surgery. *Am J Orthod Dentofacial Orthop*. 2003;124:138–143.
59. Lazaridou-Terzoudi T, Kiyak HA, Moore R, et al. Long-term assessment of psychologic outcomes of orthognathic surgery. *J Oral Maxillofac Surg*. 2003;61:545–552.
60. Cunningham SJ, Crean SJ, Hunt NP, et al. Preparation, perceptions, and problems: a long term follow-up study of orthognathic surgery. *Int J Adult Orthodon Orthognath Surg*. 1996;11:41.
61. Soh CL, Narayanan V. Quality of life assessment in patients with dentofacial deformity undergoing orthognathic surgery – a systematic review. *Int J Oral Maxillofac Surg*. 2013;42:974–980.
62. Pertschuk MJ, Whitaker LA. Psychosocial outcome of craniofacial surgery in children. *Plast Reconstr Surg*. 1988;82(5):741.
63. Pillemer FG, Cook KV. The psychosocial adjustment of pediatric craniofacial patients after surgery. *Cleft Palate J*. 1989;26(3):201.
64. Sarwer DB, Bartlett SP, Whitaker LA, et al. Adult psychological functioning of individuals born with craniofacial anomalies. *Plast Reconstr Surg*. 1999;103(2):412.

65. Broder HL, Wilson-Genderson M, Sischo L, Norman RG. Examining factors associated with oral health-related quality of life for youth with cleft. *Plast Reconstr Surg*. 2014;1331:828–834e.
66. Hunt O, Burden D, Hepper P, Johnston C. The psychosocial effects of cleft lip and palate: a systematic review. *Eur J Orthod*. 2005;27: 274–285.
67. Pruzinsky T, Edgerton MT. Psychologic understanding and management of the plastic surgery patient. In: Georgiade GS, Riefkohl R, Levin LS, eds. *Plastic, Maxillofacial, and Reconstructive Surgery*. Baltimore: Williams and Wilkins; 1997.
68. De Sousa A. Psychological issues in oral and maxillofacial surgery. *Br J Oral Maxillofac Surg*. 2008;46:661–664.

11

Orthodontic Diagnosis and Treatment Planning with Cone-Beam Computed Tomography Imaging

Lucia Cevidanes, Erika Benavides, John B. Ludlow, and Antonio Carlos de Oliveira Ruellas

OUTLINE

Strategies for Assessment of Radiation Dose Risk, 302
Factors That Influence Dose and Risk Estimation, 303
Biological Factors, 303
Technical Factors, 303
Clinical Applications and Potential Limitations of the Use of Cone-Beam Computed Tomography in Orthodontics, 304
Tooth Morphology and Relative Position within the Alveolar Bone, 304
Temporomandibular Joint Health and Disease, 304
Airway Assessment, 305
Dentofacial Deformities and Craniofacial Anomalies, 306
Image Analyses and the Use of Three-Dimensional Surface Models and Superimpositions, 307
Challenges to Advance Our Understanding of Growth and Treatment Responses, 307
Step-by-Step Three-Dimensional Image Analysis Procedures, 309
Treatment Planning with Computer-Assisted Surgery, 313
Surgical Planning and Simulation, 313
Simulation of Soft Tissue Changes, 314
Intraoperative Guidance: Surgical Navigation, 314
Tracking Technology, 315
Final Considerations, 315

The interrelationships of the dentition, craniofacial skeleton, and soft tissues involve the three dimensions (3D). The increasing availability of cone-beam computed tomography (CBCT), over the past 10 years, now allows visualization of these relationships in 3D. Such 3D assessments are likely to promote improved orthodontic diagnosis, treatment planning, evaluation of growth and development, assessment of treatment progress and outcomes, and retention. It is very important that clinicians are prepared and familiar with the technology advances now available.

This chapter describes (1) strategies for assessment of radiation dose risk; (2) clinical applications and potential limitations of the use of CBCT in orthodontics, including suggested optimal imaging selection for each clinical application; and (3) image analyses and the use of 3D surface models and superimpositions.

STRATEGIES FOR ASSESSMENT OF RADIATION DOSE RISK

Because cancer is the principal long-term biological effect of exposure to x-rays, one of the greatest issues facing CBCT in orthodontics is justification of the increased dose of ionizing radiation administered to patients compared with standard two-dimensional (2D) imaging techniques.[1] This is especially important when considering the adolescent and pediatric populations that routinely receive orthodontic treatment in whom cellular growth and organ development is associated with increased radiosensitivity of tissues. In conjunction with a longer life expectancy in which cancer can develop, children may be two times to five times more sensitive to radiation carcinogenesis as mature adults.

The International Commission on Radiological Protection (ICRP) has recommended a calculation called the *effective dose* as the preferred method for comparing risks from different exposures to ionizing radiation.[2] Effective dose is a calculation that considers the most radiosensitive tissues and organs of the body and provides a fractional weighting reflecting the degree of sensitivity for each of those organs. Effective dose is reported in sieverts and for diagnostic imaging is more commonly expressed in millisieverts or microsieverts. Stochastic effects are associated with long-term, low-level (chronic) exposure to radiation, where "stochastic" refers to the likelihood that something will happen: increased levels of radiation exposure make these health effects more likely to occur but do not influence the type or severity of the effect.[3] Because estimation of the risk of the stochastic effects of genetic mutation and cancer formation has evolved with additional data reported from observations of a variety of exposed populations, the ICRP has changed the calculation of effective dose several times. The

most recent change was in 2007[4] and is noteworthy because weights of several tissues located in and around the maxillofacial region were changed, and several other tissues within this region were added to the calculation.[5] Changes in tissue weights have resulted in a 10% increase in weight of tissues located in the maxillofacial area and a 28% increase in weight after adjusting for the distribution of tissues. Newly added tissues for effective dose calculation that are entirely within the maxillofacial area include the oral mucosa, salivary glands, and extrathoracic airways. A recent meta-analysis of effective dose estimation of dental CBCT examinations reported that adult effective doses for any protocol ranged from 46 to 1073 μSv for large fields of view (FOVs), 9 to 560 μSv for medium FOVs, and 5 to 652 μSv for small FOVs. Child effective doses from any protocol ranged from 13 to 769 μSv for large or medium FOVs and 7 to 521 μSv for small FOVs. Mean adult effective doses grouped by FOV size were 212 μSv (large), 177 μSv (medium), and 84 μSv (small). Mean child doses were 175 μSv (combined large and medium) and 103 μSv (small). Large differences were seen among different CBCT units. Additional low-dose and high-definition protocols available for many units extend the range of doses (Fig. 11-1).[3]

The ongoing challenge in the optimization of CBCT is to reduce radiation dose without dramatically decreasing imaging quality and diagnostic information. As an example, the contrast-to-noise ratio was reduced by approximately two thirds when Ludlow and Walker[2] compared QuickScan+ with standard exposure parameters. A potential means of reducing patient risk from CBCT examinations is to limit the area of exposure by using variable fields of view that are sized for the location of the anatomy of interest. However, voxel size is linked to field of view (FOV) in CBCT units, and smaller voxel sizes associated with smaller FOVs may actually increase dose because of increases in exposure that are needed to maintain adequate contrast-to-noise ratio. Another approach is to reduce exposure for diagnostic tasks that theoretically require lower contrast-to-noise ratios or lower signal modulation transfer functions. An example of this type of task might be checking angulation of roots. The combination of careful selection of exposure parameters and FOV may result in an optimal use of dose for specific diagnostic tasks in orthodontic practice.[2]

Factors That Influence Dose and Risk Estimation[1]

Biological Factors

Age has a significant impact on both individual dose and risk. Children are physically smaller, which places peripherally located brain and thyroid tissues closer to the dental area that is being imaged. Even if not directly exposed, these organs receive increased scatter radiation with increased proximity to the location of the scanned volume. Children are also at increased risk from any exposure to ionizing radiation owing to cellular growth and organ development, which increases the radiosensitivity of tissues. In conjunction with a longer life expectancy in which cancer can develop, children may be two times or more sensitive to radiation carcinogenesis than are mature adults.[6,7] Females are at risk for breast cancer and ovarian cancer, and males are at risk for prostate cancer. Because these organs are distant from the maxillofacial area, gender differences do not impact dose and risk estimation for maxillofacial imaging.

Technical Factors

Receptor Technology and Field of View. Two types of receptor technologies are used to acquire image data. Image intensifiers use a round receptor and produce a spherical FOV. Square or rectangular flat panel detectors are incorporated in many CBCT units, and these produce a cylindrical FOV. In general, the cylindrical field is more efficient at capturing the anatomy of the maxillofacial complex when the top of the field includes the temporomandibular joint (TMJ) areas. A cylindrical volume diameter, which captures both TMJ and chin anatomy, requires

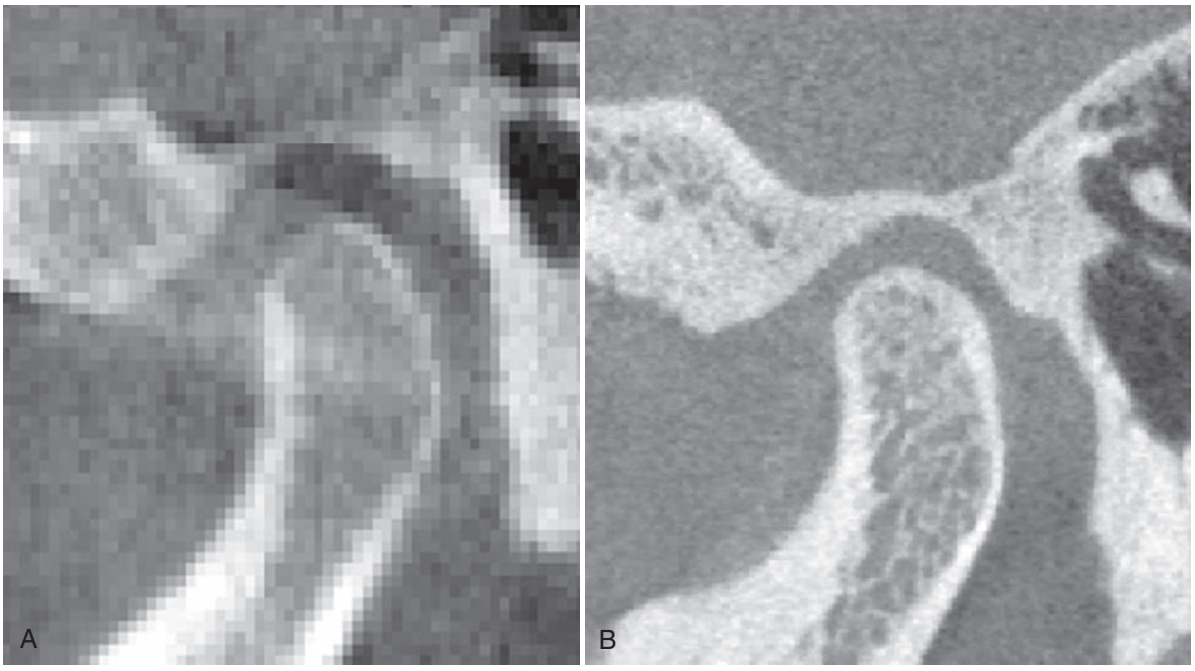


FIGURE 11-1 Images from cross sections of two scans acquired in different cone-beam computed tomography units. **A,** NewTom3D, large field of view. **B,** Accuitomo, small field of view.

a spherical volume diameter that is approximately 25% larger to cover the same anatomy.

mAs. X-ray tube current (mA) and exposure time (s) are directly proportional to dose when other factors remain constant. Some CBCT units produce continuous output of radiation during scanning, with equal scan time and exposure time. Most detectors are unable to record x-ray exposure during the period when the image detector integrates the x-ray energy absorbed in individual receptor pixels and transfers this signal to the computer. To eliminate this unnecessary patient exposure, many CBCT units use a pulsed x-ray source, in which x-ray emission is intermittently turned off during the image acquisition process.

kVp and Beam Filtration. Increasing filtration of the x-ray beam reduces patient exposure to lower energy x-ray photons that are more likely to contribute to patient dose without contributing to image formation.[8] Use of 0.4 mm of additional copper filtration in conjunction with increased kVp was demonstrated to reduce patient dose by an average of 43% with one unit.[9]

Resolution. Exposure must be increased as the voxel size is reduced to create higher resolution images and maintain an adequate signal-to-noise ratio. With some CBCT units, this choice is under operator control, but at other times, the unit dictates which exposure factors may be used with different resolutions.

CLINICAL APPLICATIONS AND POTENTIAL LIMITATIONS OF THE USE OF CONE-BEAM COMPUTED TOMOGRAPHY IN ORTHODONTICS

From a radiation-protection perspective, CBCT should be required when its indication overcomes the fact that conventional images may deliver lower doses to patients. 3D CBCT imaging in orthodontics and orthognathic surgery offers benefits and diagnostic information for the following complex conditions[10]:

Tooth Morphology and Relative Position within the Alveolar Bone

Small or medium FOV high-resolution images that include an arch quadrant or both upper and lower arches are needed to evaluate buccal and lingual plates of the alveolar bone, bone loss or formation, bone depth and height, presence or absence of unerupted teeth, tooth development, tooth morphology and position, amount of bone covering the tooth, and proximity or resorption of adjacent teeth. It is very important that both multiplanar images in the three planes of space and 3D rendered views with or without transparency be used for detailed assessments because isolated views could lead to misdiagnosis (Fig. 11-2). CBCT findings may lead to modifications in the treatment planning (e.g., avoid extraction, help decision of which tooth to extract, evaluate dilacerated roots or placement of bone plates and miniscrews), reduced treatment duration, and additional root resorption control and in the orthosurgical planning.[11–19] Before placing temporary anchorage device (TADs), CBCT is being used as a clinical tool to identify optimal position and to avoid damage to roots.[20] The use of surgical guides based on CBCT data has also been suggested.[21]

Temporomandibular Joint Health and Disease

Small or medium FOV high-resolution images that include one joint at separate right and left acquisitions yield the best quality of images for TMJ assessments (Fig. 11-3). The spectrum of the clinical and pathologic presentation of TMJ osteoarthritis ranges from structural and functional failure of the joint with disc displacement and degeneration (best diagnosed with magnetic resonance imaging [MRI]) to subchondral bone alterations and sclerosis (best diagnosed in multiplanar cross-sectional images from CBCT or CT scans), bone erosions

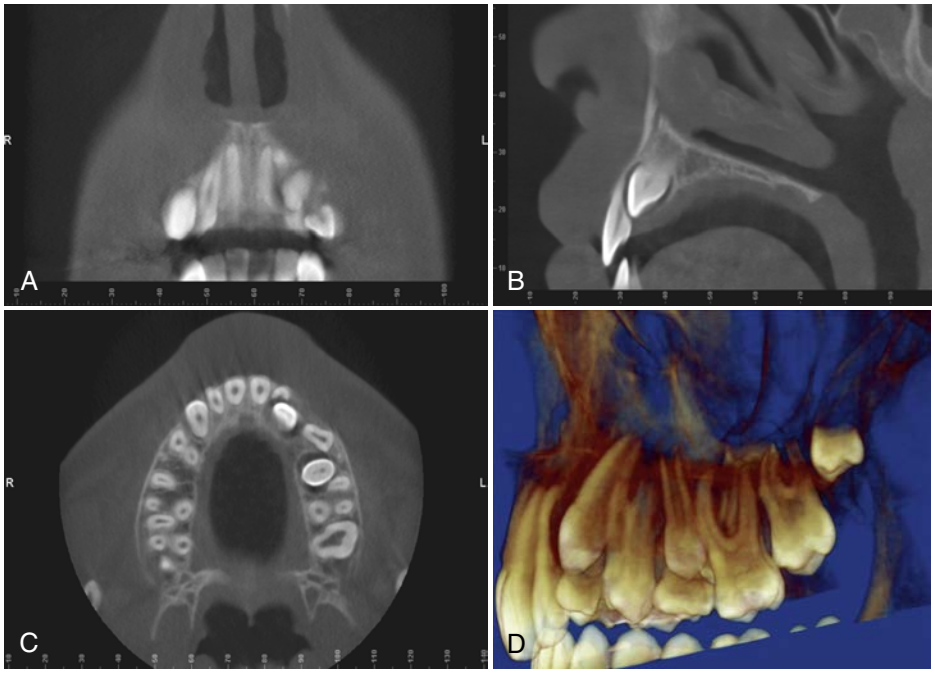


FIGURE 11-2 Multiplanar images in the three planes of space. **B,** Three-dimensional rendered view.

or overgrowth (osteophytes, best diagnosed complementing multiplanar cross-sectional images with assessment of the respective solid 3D surface models constructed from CBCT or CT scans), and loss of articular fibrocartilage and synovitis (best diagnosed with MRI). For detecting TMJ bony changes, panoramic radiography and MRI have only poor to marginal sensitivity.[22] For this reason, CBCT has recently replaced other imaging modalities as the modality of choice to study TMJ bony changes.[23–25] The Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD)[26] was revised recently to include image analysis criteria for various imaging modalities.[22] The RDC/TMD validation project[27–29] concluded that revised clinical criteria alone, without recourse to imaging, are inadequate for valid diagnosis of TMD and had previously underestimated the prevalence of bony changes in the TMJ. TMJ pathologies that result in alterations in the size, form, quality, and spatial relationships of the osseous joint components lead to skeletal and dental discrepancies in the three planes of space.[30,31] In affected condyles, the perturbed growth or bone remodeling, resorption, and apposition can lead to progressive occlusal changes that are accompanied by compensations in the maxilla, "nonaffected" side of the mandible, tooth position, occlusion and articular fossa, and unpredictable orthodontic outcomes (Fig. 11-4).

The intriguing insights into TMJ health and disease provided by recent applications of 3D imaging CBCT[32] have revealed that the TMJ is prone to a myriad of pathologies that could be didactically divided as "degenerative pathologies" and "proliferative pathologies." Such pathologies can dramatically affect other craniofacial structures and be easily recognized, or the TMJ pathology can be challenging to diagnose even to experts when its progression is subtle and limited even though it is still clinically relevant. In any situation, longitudinal quantification of condylar changes has the potential to improve clinical decision making by identifying the most appropriate and beneficial therapy.

The TMJ is unique in relation to the other joints in the body. Adult joint bone surfaces, with the exception of the TMJ, are composed by hyaline cartilage. The TMJ has its articular bone surfaces covered by a thin layer of fibrocartilage that has a tremendous capacity for morphologic adaptation due to function. The threshold between functional physiologic stimulus with its positive biochemical effects on the TMJ and joint overloading that lead to degenerative changes is beyond current knowledge.[33–35] This threshold is influenced by a multitude of factors, including but not limited to the joint loading vectors and their magnitude,[36] and patient inherited or acquired (genetic and mostly epigenetic) factors, including hormonal and autoimmune imbalances. Current methods to detect pathologic conditions in a cross-sectional diagnostic assessment (bone scintigraphy and positron emission tomography [PET]) are highly sensitive; however, they do not have enough specificity because there are no standard normal values for baseline assessments. Longitudinal 3D quantification using CBCT offers a relatively low-cost, low-radiation technology compared with PET-CT and bone scintigraphy and can make a significant difference on treatment planning as an additional biomarker or risk factor tool.

The use of biomarkers to aid diagnosis in TMJ disorders is very promising, but it is not novel. Several biomarkers, including C-reactive protein and markers of inflammation, angiogenesis, and bone turnover (e.g., angiogenin, growth differentiation factor 15 , tissue inhibitors of metalloproteinases 1, chemokine ligand 16, matrix metalloproteinases 3 and 7, epithelial neutrophil activating peptide, plasminogen activator inhibitor-1, vascular endothelial cadherin, vascular endothelial growth factor, granulocyte macrophage colony-stimulating factor, transforming growth factor-β, interferon gamma, tumor necrosis factor-α, interleukin-1α, interleukin-632), have previously been identified in blood and in synovial fluid biopsies of patients with TMJ condylar bone resorption and related to the pathologic progress.[37–39] Such techniques, still currently restricted to academic environments and research centers, are certainly very promising and will complement CBCT 3D techniques that are already clinical tool protocols.

Airway Assessment

Airway morphology and changes over time after surgery, as well as growth and the airway relationship to obstructive sleep apnea, have been recently assessed in either medium or large FOV CBCT scans.[40–43] However, the boundaries of the nasopharynx superiorly with the maxillary and paranasal sinuses and the boundaries of the oropharynx with the oral cavity anteriorly and inferiorly with the larynx boundary are not consistent among subjects, which warrants very careful interpretation of volumetric and 3D assessments and multiplanar cross-sectional

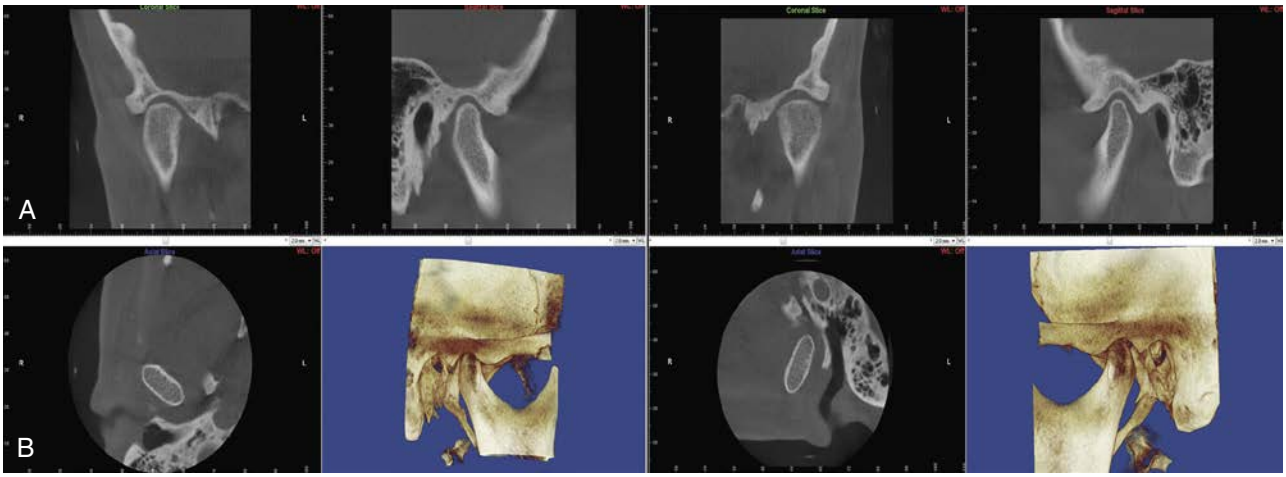


FIGURE 11-3 Right (A) and left (B) temporomandibular joint (TMJ) images acquired with a 6 × 6 field of view. The high-resolution images provide proper assessment of the TMJ bone anatomy.

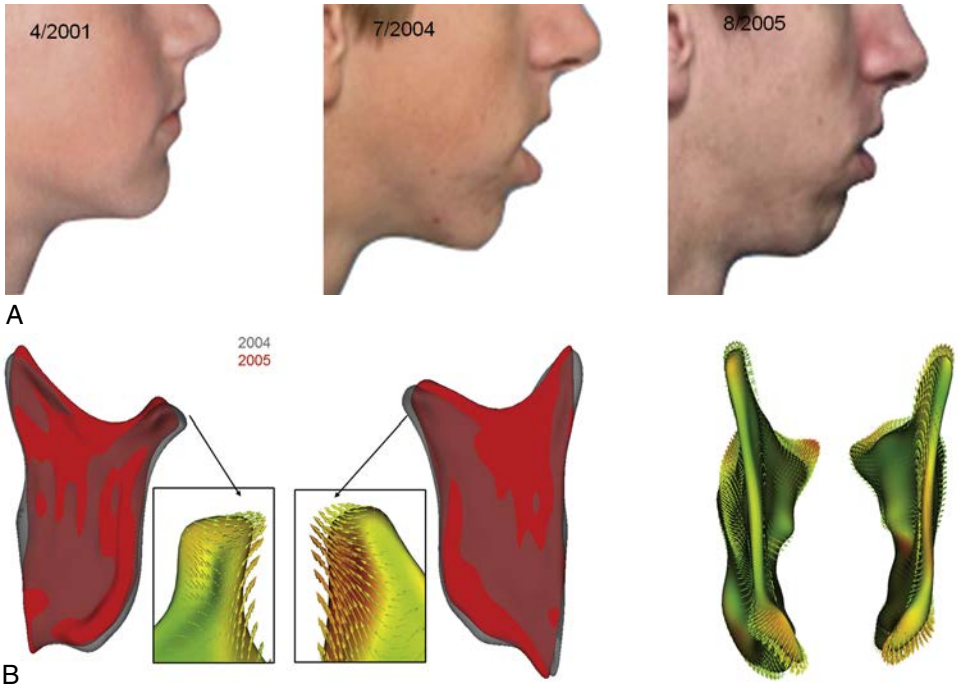


FIGURE 11-4 Degenerative joint disease that led to progressive occlusal and facial changes. **A,** Extraoral photographs: T1, patient at the initial evaluation; T2, 3 years' follow-up; T3, four years' follow-up. **B,** T2/T3 overlay of the right and left sides and the respective vector map based on shape correspondence methodology.

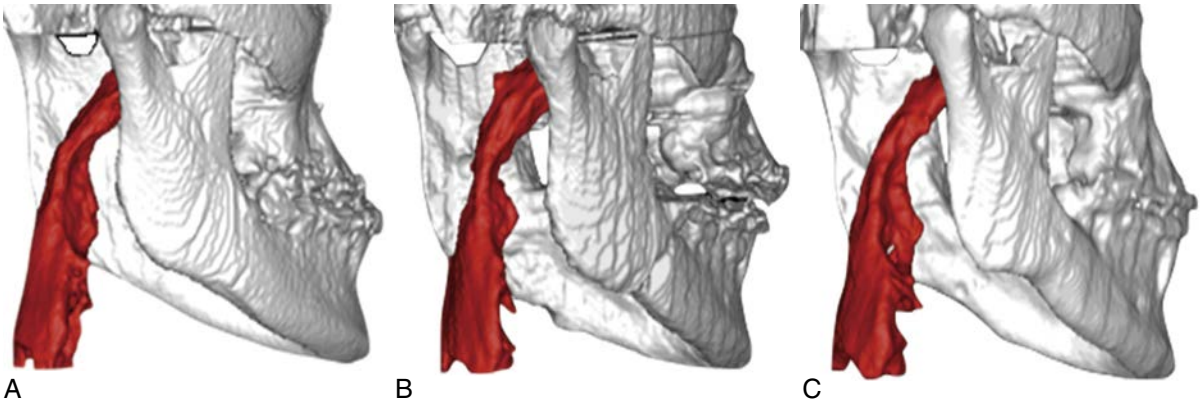


FIGURE 11-5 Airway space assessments. **A,** T1, initial evaluation. **B,** T2, after maxillary advancement surgery, we can notice enlargement of the airway space. **C,** T3, reduction of airway space can be observed 1 year after surgery.

slices of the airway. Additionally, image acquisitions and airway shape and volume vary markedly with functional stage of the dynamic process of breathing and head posture. If head posture is not correctly reproduced in longitudinal studies, differences in head posture will lead to variability in airway dimensions. Longitudinal assessments of mandibular setback have not shown consistent reduction of airway space nor have mandibular propulsion advancement devices shown enlargement of the airway space that might be helpful for obstructive breathing conditions (Fig. 11-5).

Dentofacial Deformities and Craniofacial Anomalies

Large FOV CBCT images offer the ability to analyze facial asymmetry and anteroposterior (AP), vertical, and transverse discrepancies associated with complex craniofacial problems. The virtual treatment simulations using 3D virtual or printed surface models constructed from CBCT or CT images can be used for treatment planning in orthopedic corrections and orthognathic surgery and for printing surgical splints after performing virtually simulated surgery. Computer-aided surgery (CAS) of the jaw is described in more detail in the Image Analysis section of this chapter, and its clinical application has increased because of the possibility to incorporate a high level of precision for accurately transferring virtual plans into the operating room. In complex cases, follow-up CBCT acquisitions for growth observation, treatment progress, and posttreatment observations may be helpful to assess the stability of the correction over time (Fig. 11-6).[44–47]

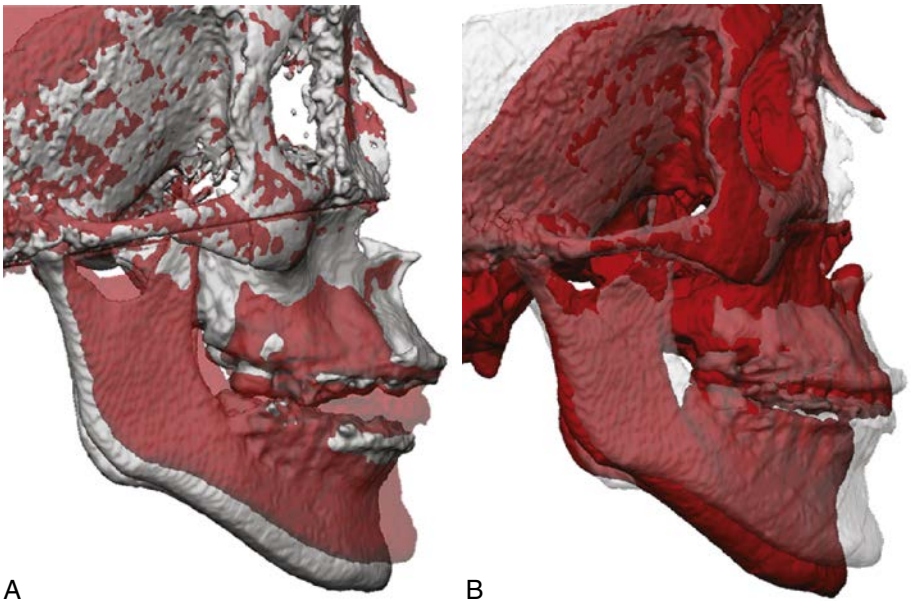


FIGURE 11-6 Overlay of three-dimensional mesh models for follow-up observation. A, Between presurgery and splint removal. B, Between splint removal and 6 years post surgery that shows relapse of the mandibular advancement.

IMAGE ANALYSES AND THE USE OF THREE-DIMENSIONAL SURFACE MODELS AND SUPERIMPOSITIONS

Challenges to Advance Our Understanding of Growth and Treatment Responses

Even with the availability of 3D volumetric images, the clinical questions on the most appropriate quantitative approaches still remain a field for research. Before describing in detail the 3D image analysis procedures in this section of the chapter, we outline below three important challenges.

1. **Construction of 3D volumetric label maps (segmentation)**
   Although assessment of the multiplanar cross-sectional slices is limited to 2D evaluations of each slice and poses challenges toward standardization of which cross-section to analyze,[11] 3D representations of the craniofacial anatomy allow visual and quantitative assessments of the whole 3D anatomic surfaces reconstructed from CBCT scans (Fig. 11-7, *A* and *B*). A distinction must be made between virtual 3D renderings and 3D surface meshes or models that are generated from 3D volumetric label maps. The CBCT volumes can be visualized as projected images (renderings), without the construction of surface models, but these renderings can be used only for perspective visualizations and not for quantitative assessments because they are simply projected images, "ghostlike," and not solid surface meshes (Fig. 11-7, *C*). The 3D surface meshes or models provide additional diagnostic information on size, shape, and exact location of the bone abnormality (Fig. 11-7, *D* and *E*).[48] The process of constructing 3D volumetric label maps, known as *image segmentation*, by examining cross sections of a volumetric data set to outline the shape of structures, remains a challenge.[49–51] Even though image segmentation has been a field of active research for many decades, representation of fine anatomic details remains one of the most time-consuming steps in image processing. A major challenge with segmentation lies with the fact that hard and soft tissues from CBCT have no corresponding Hounsfield units. The same CBCT taken from the same individual may have different intensity levels for the bones depending on their position in the volume and relationship to adjacent anatomy. No standard segmentation method can be expected to work equally well for all tasks. Much commercial software incorporates intensity thresholding algorithms for their segmentation. Although this often works well for thick and dense bones such as the mandibular bone, it often fails for thin bone such as the condyles and labial surfaces of the teeth. The morphology and position of the condyles and maxilla are challenging for adequate segmentation and diagnosis. Therefore, precise segmentation and representation of these anatomic regions are very important.
2. **Image registration**
   For standardized baseline diagnosis or longitudinal assessments, the CBCT scans and their respective 3D surface models require registration in a common coordinate system using a choice of target region or areas of reference. The choice of reference for the registration can also be based on landmarks, surfaces, or voxel gray intensity. Different areas and sources of reference for registration lead to different interpretations of the results. The process of registration involves computing transformations, although the image analysis software can display or allow the user to see it or not. Transformation is a mathematical operation that applies a matrix to move the points of a 3D image or surface model (or both) in multiple planes and degrees of freedom in the 3D space. Longitudinal CBCT scans acquired at different time points can be registered by computing the differences of the head position relative to a stable anatomic structure of reference. The image registration computes the translational (AP, transverse, and vertical) and rotational displacements (pitch, roll, and yaw) in a procedure known as *rigid registration*.[52] The image registration can also compute differences in scale (size changes

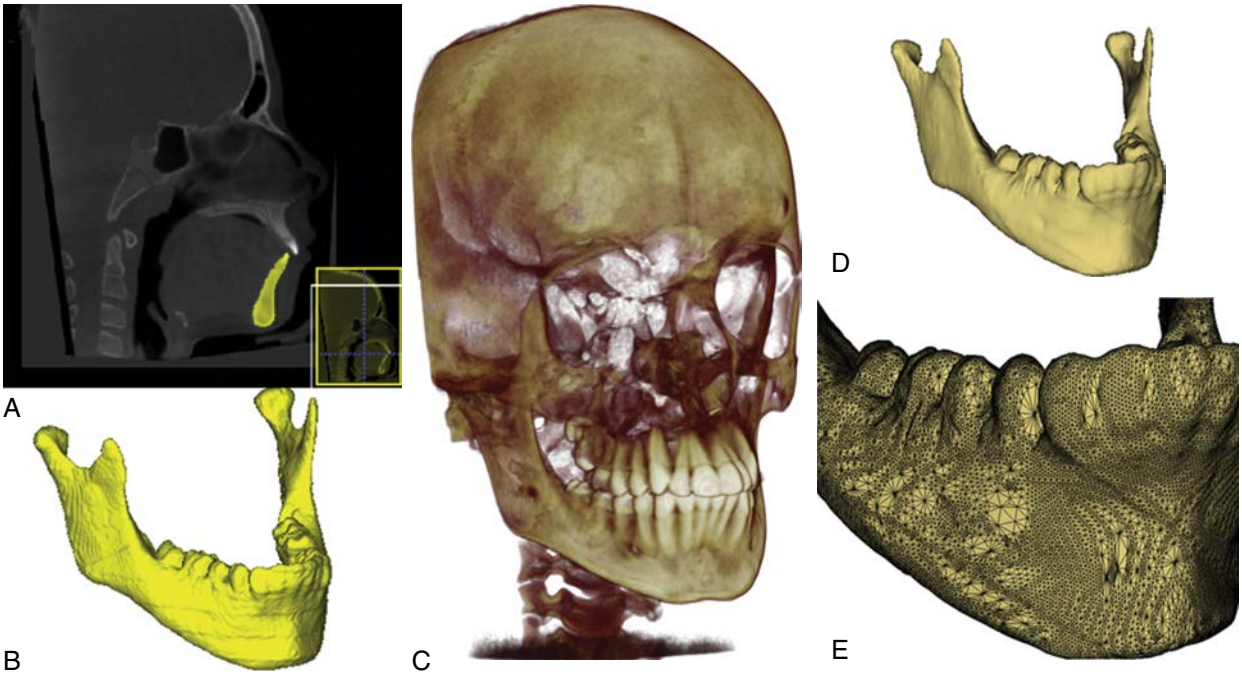


FIGURE 11-7 Image analysis procedures in the construction of three-dimensional (3D) virtual surface models. **A,** Scan acquired and 3D label map being constructed for the mandible. **B,** 3D label maps. **C,** Rendering image. **D,** 3D surface mesh models. **E,** 3D surface mesh models showing the surface mesh in detail.

with growth /or treatment) and shape in image analysis procedures known as *nonrigid registration*. The challenges of using nonrigid registration for clinical studies are that the 3D models are deformed.[53] To avoid distorting or morphing the images, nonrigid registration can be used to compute transformations considering scale and shape differences and then apply only the rigid movements (rotation and translation) to preserve the actual scale and shape features.[54,55]

3. **Quantitative measurements**

Three-dimensional imaging can only advance our understanding of clinical questions if beyond visual assessments, it provides clinicians with improved quantification methods for diagnosis, treatment planning, and assessment of treatment outcomes in short and long terms. The facial changes can be quantified by obtaining measurements in cross-sectional slices of scans or in 3D surface models at two or more time points or between scans or surface models registered relative to a stable structure of reference such as cranial base (a validated voxel-based method to assess posttreatment changes in growing[55] and adult[56] patients). Linear and angular measurements based on landmarks can camouflage bone remodeling because subtraction between time points, for example, is used as a representation of growth and may lead to confusion about what is happening (analysis of rotations, treating shape separately from size, and registering angles on landmarks as vertices).[57] Since the original work of Moyers and Bookstein,[58] advanced morphometric (i.e., that measure morphology or form) methods using semi-landmarks,[59] matrices of interlandmark distances (EDMA),[60] curved distances, or tensor-based morphometry[61] have been proposed. However, these methods involve complex mathematical information that is not easily interpreted by clinicians. Current quantification methods of dental and skeletal displacements and bone remodeling of both the maxilla and the mandible include:

a. **Volume**[52] changes do not capture shape changes as structural changes at specific locations are not sufficiently reflected in volume changes; volume assessment does not reveal the location and direction of proliferative or resorptive changes, which would be relevant for clinical results assessment.
b. **3D linear surface distances in triangular meshes**
   i. Based on **observer-defined landmarks**[62]: Locating 3D landmarks on complex curving structures is not a trivial problem for representation of components of the craniofacial form.[63] As Bookstein[57] noted, there is a lack of literature about suitable operational definitions for the landmarks in the three planes of space (coronal, sagittal, and axial), which leads to errors and variability in landmark location. Gunz et al.[64] and Andresen et al.[65] proposed the use of semi-landmarks (i.e., landmarks plus vectors and tangent planes that define their location), but information from the whole curves and surfaces must also be included. The studies of Subsol et al.[66] and Andresen et al.[65] provided clear advances toward studies of curves or surfaces in 3D, referring to tens of thousands of 3D points to define geometry. For clinical assessments, the challenges remain (1) the awareness of limitations based on measurements derived from 3D landmarks and (2) the choice of landmark anatomic locations that describe and "represent" different components of maxillomandibular growth and response to treatment. Although 3D linear distances are a simplification of complex morphologic changes, they provide relevant clinical evaluation of changes in the space related to differences between time points.

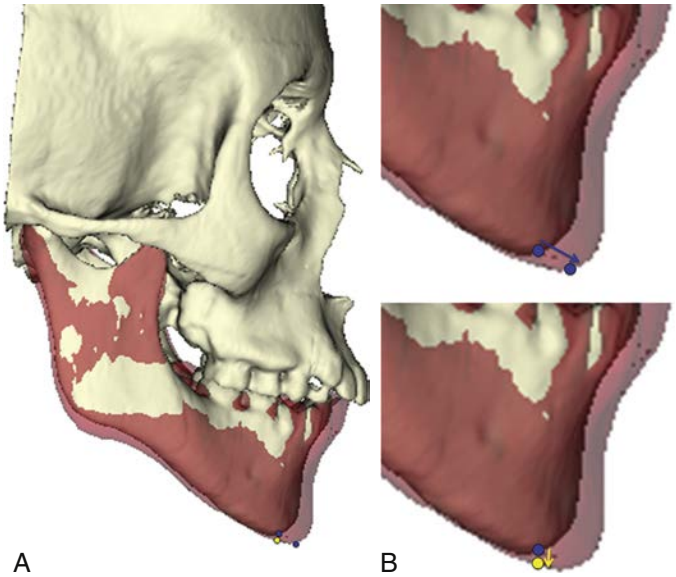


FIGURE 11-8 Mandibular overlay of surface mesh models from two time points (A and B) displaying the differences between measurements based on corresponding points (*blue arrow*) and closest point (*yellow arrow*).

ii. Based on thousands points in triangular meshes automatically defined in the surface models:
   (a) **Closest point measurements** between the surfaces can display changes with color-coded maps as proposed by Gerig et al.[67] However, the closest point method measures the closest 3D linear distances between surfaces, not corresponding distances between anatomic points on two or more longitudinally obtained images (Fig. 11-8). For this reason, the closest point measurements fail to quantify rotational and large translational movements, and this method cannot be used for quantitative longitudinal assessments of growth or treatment changes, nor the physiologic adaptations, such as bone remodeling that follows surgery. This standard analysis is currently used by most commercial and academic software but does not map corresponding surfaces based in anatomic geometry and usually underestimates rotational and large translational movements. Closest point color maps measure surgical jaw displacement as the smallest separation between boundaries of the same structure, which may not be the correct anatomic corresponding boundaries on pre- and postsurgery anatomic structures.
   (b) **Shape correspondence measurements**[68] were developed as part of the National Alliance of Medical Image Computing (NA-MIC, NIH Roadmap for Medical Research) and have been adapted for use with CBCTs of the craniofacial complex.[69,70] The correspondence between thousands of surfaces points is generated by computing point-based models using a parametric boundary description for the computing of shape analysis. The 3D virtual surface models are converted into a corresponding spherical harmonic description (SPHARM), which is then sampled into triangulated surfaces (spherical harmonics-point distributed models [SPHARM-PDM]). This work presents an improvement in outcome measurement as compared with closest point correspondence–based analysis (see Fig. 11-8). The challenges with SPHARM-PDM are that this shape analysis method is currently highly computational intensive, the surface parameterization can only be applied to closed surfaces, and complex anatomy may not be properly represented and require control of the quality of the point-to-point correspondence.
   (c) Quantification of directional changes in each plane of the 3D space (**3D components**). 3D linear distances, based on observer-defined landmarks or automatically defined points, express different amounts of changes in the three axes of the space, and clinical questions require more precise information regarding the location and amounts of changes in each direction (*x*-, *y*-, and *z*-axes). In particular, clinicians plan their expected results of treatment based on anterior, posterior, inferior, superior, medial, or lateral movements and displacements. Quantification of directional changes in each plane of the 3D space (3D components) can be obtained by the distances between projections of the 3D landmarks and require a standardized common *x, y, z* coordinate system across time points and all patients in the Slicer software.

c. **3D angular measurements** can be represented as pitch, roll, and yaw and can be used to evaluate the rotation of the whole skull or only the mandible or maxilla. Evaluations using each of the three views (sagittal, coronal, and axial) allow these angles be measured by the intersection of two lines based on the coordinates of landmarks or by the intersection of two planes being each one from different time points. The challenge of angular assessments is that they also require consistency of a standardized common *x, y, z* coordinate system across time points of all patients.

Step-by-Step Three-Dimensional Image Analysis Procedures

A step-by-step description of image analysis procedures includes (1) image acquisition; (2) construction of 3D volumetric label maps; (3) image registration; (4) conversion to 3D surface models; (5) establishment of a common coordinate system; (6) visual analytics with graphic display of 3D morphologic variability or changes; and (7) quantification of location, directions, and magnitude of 3D morphologic variability or changes:

1. **Acquisition of 3D diagnostic records**
   Diagnosis of maxillomandibular discrepancies is based on data coming from different sources: clinical examination, 3D photographs, CBCT, CT, MRI, and digital dental models. All different records need to be integrated in order to characterize the orthodontic diagnosis and formulate the treatment plan. CBCT, CT, and MRI are acquired as volumetric 3D images (saved in file formats such as DICOM, gipl, or nrrd) that can be used to visualize cross-sectional slices and renderings in various 3D viewer software. These volumetric images can be visualized as 3D projected images (3D renderings) or used to construct polygonal meshes or surfaces of specific anatomic structures of interest. To simplify

the description of image analysis procedures, this chapter specifically describes 3D analysis of images acquired with CBCT because CBCT has been recently widely used in our field. The same image analysis procedures are applicable and can be generalized for images acquired with any 3D imaging modality.

2. **Construction of 3D volumetric files that label with color the anatomic structures of interest**
The CBCT scans (DICOM files) can be opened and visualized in any 3D image analysis software of choice. Examples are 3DMDvultus (3DMD, Atlanta, GA)[71]; Maxilim, Medicim (Mechelen, Belgium)[72]; Dolphin Imaging (Dolphin Imaging & Management Solutions, Chatsworth, CA)[73]; InVivo Dental (Anatomage, San Jose, CA)[74]; SimPlant OMS or Mimics (Materialise, Leuven, Belgium)[75]; and open source tools such as TurtleSeg,[76] ITK-SNAP,[77] and 3D Slicer.[78] In a procedure known as image segmentation, we identify and delineate the anatomic structures of interest in the CBCT scan to obtain a 3D representation of the hard and soft tissues (3D volumetric label map files). To best capture the facial anatomy, our method of choice for the segmentation procedures uses ITK-SNAP[79] software that has received continuous National Institutes of Health (NIH) support for further open source software development. ITK-SNAP was developed, based on the NIH Visualization Tool Kit (VTK) and Insight Tool Kit (ITK), as part of the NIH Roadmap Initiative for National Centers of Biomedical Computing. The automatic segmentation procedures in ITK-SNAP use active contour methods to compute feature images based on the CBCT image gray level intensity and boundaries. It is important to understand that ITK-SNAP is more versatile than other open and commercial software because it allows the adjustment of the parameters for automatic detection of intensities and boundaries as well as allows user interactive editing of contours. On a laptop computer equipped with 1 GB of RAM, the initial segmentation step typically takes about 15 minutes. Manual postprocessing of the segmentation usually takes longer, up to a couple of hours. (Separation of the upper and lower teeth can be particularly tedious.)

For longitudinal CBCT scans or scans of a group of patients, further image analysis procedures are required and described below as performed in Slicer open source software in steps (3) image registration, (4) conversion to 3D surface models, (5) establishment of a common coordinate system, (6) visual analytics, and (7) quantitative measurements as follows:

3. **Image registration**
Both commercial and open source tools now allow registration procedures based on landmarks, surface models, or voxel gray intensity for craniomaxillofacial registration (https://sites.google.com/a/umich.edu/dentistry-image-computing/).[79] The image registration procedures that our research group has found to provide the most reliable results consist of two steps:
   a. **Manual approximation of T1 and T2 scans:** If head position was not controlled and standardized during imaging acquisition, the T1 and T2 scans should be approximated by the observer reorienting one of the scans guided by the best fit of the outlines of a region of reference. The approximation can be visualized in each of the 3D multiplanar cross sections using open source software (Slicer CMF, http://www.slicer.org).[78,80] Videos tutorials are available at http:/www.youtube.com/user/DCBIA.[79]
   b. **Voxel-based registration for longitudinal assessments:** The major strength of the voxel-based registration method is that registration does not depend on how precisely the 3D volumetric label maps represent the anatomic truth or on the location of a limited number of landmarks. In fact, for voxel-based registration methods that use the 3D volumetric label maps as the input of the region of reference, these multiplanar label maps should be extended slightly (1–2 voxels) beyond the actual bone boundaries to provide the complex gray-level information needed for the automatic detection of the bone boundaries during the voxel-based registration. Importantly, the 3D volumetric label maps of the anatomic structures of reference for the registration are not the "clean" 3D surface models and are only used as references to mask anatomic structures that change with growth and treatment. The voxel-based registration methods actually compare voxel by voxel the gray-level values in two CBCT images to calculate the rotation and translation parameters between them. Because not all "voxel-based" registrations are similar in methodology and accuracy, because they can use different structures of reference, different numbers of iterations (e.g., the software can run for 1 minute and compute 100 attempts of best voxel match or run for 10 minutes and compute 1000 comparisons of best fit among thousands of voxels). After registration, the 3D volumetric label maps should be further edited for finer definition of the patient actual bony anatomic contours.

It is also important to understand that the clinical implications that can be derived from 3D registrations or superimpositions depend on the structures selected as reference for registration. Clinicians should become familiar and trained on the new technologies to avoid misunderstandings and incorrect interpretations of the 3D images. Registration on different regions of reference will lead to different interpretations of the results. We have developed a novel sequence of fully automated voxel-wise rigid registration at the cranial base (for overall facial assessments relative to cranial structures that complete their growth early in childhood)[55] and regionally (to assess maxillary and mandibular bone remodeling).[81,82] After these registration steps described earlier, the registered 3D volumetric label maps can then be saved as a 3D triangular or polygonal meshes (3D surface models) in, for example, .obj, .ply, .vtk, or .stl file formats. All image registration procedure conversions from 3D volumetric label maps to 3D surfaces models can be accomplished in Slicer CMF (http://www.slicer.org).[78–80] They were initially developed as part of the(NA-MIC, NIH Roadmap for Medical Research and have been widely used internationally.

4. **Conversion to 3D surface models**
After segmentation, for a single one-time point image, the 3D volumetric label map can be saved or converted as a 3D triangular/polygonal mesh (3D surface model), in .obj, .ply, .vtk, or .3ud file format (see Fig. 11-7). Then it can be visualized in different 3D viewer software programs.
5. **Establishment of a common coordinate system across subjects for group comparisons**
Establishment of a common coordinate system not based on a single line or plane is essential to allow group comparisons and consistent measurements across subjects. To achieve such a common coordinate system, all T1 surface models in

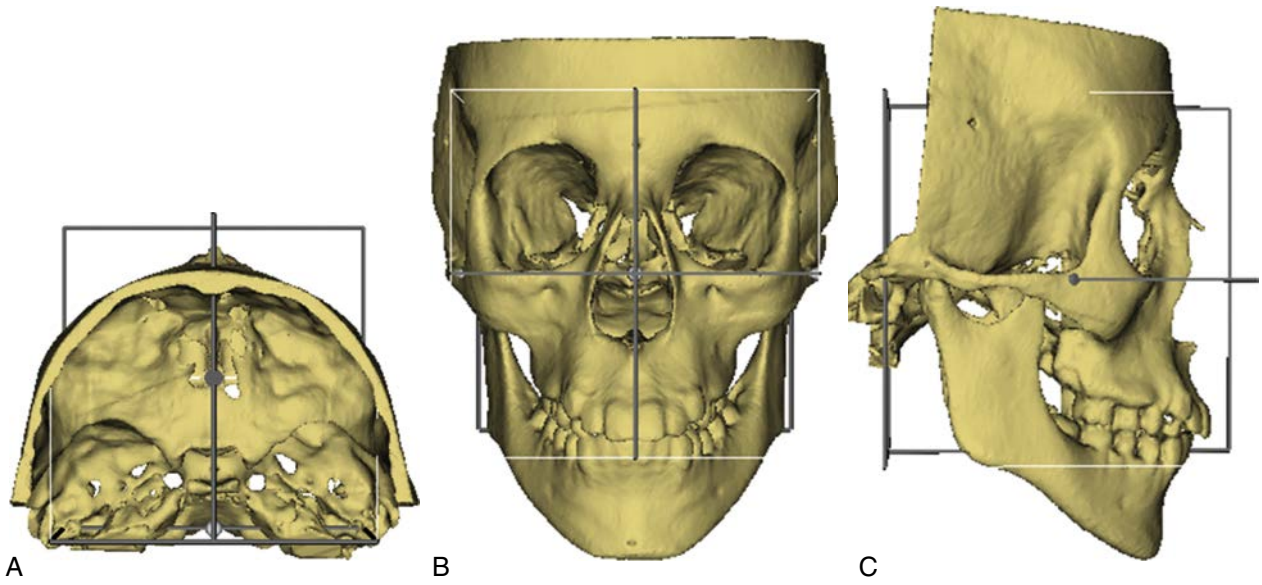


FIGURE 11-9 Head orientation procedure. A, Superior view. B, Frontal view. C, Lateral view.

a study sample can be oriented, for example, in the Slicer software (Fig. 11-9). The 3D orientation can be achieved by using intracranial reference planes defined by at least three landmarks or two landmarks and a plane[83]: Frankfurt horizontal, midsagittal, and transporionic planes. Slicer software displays a fixed 3D coordinate system that can be used as reference to orient the 3D models. Using axial, coronal, and sagittal views of the 3D models, the T1 model can be moved to orient the midsagittal plane vertically and coincident with the vertical (sagittal) plane of the 3D coordinate system. The Frankfurt horizontal plane can be oriented to match with the horizontal (axial) plane. The horizontal transporionic line can be oriented to be coincident with the intersection between the axial and coronal planes in both sides of the head. The matrix generated from this step can then be applied to the T2 registered surface model, obtaining the same head orientation. This same procedure can be repeated for all subjects in the sample with the goal of obtaining a common coordinate system for quantitative steps described in step 5 below. After registration, it is important for visualization and quantification purposes that all models be clipped (cropped) simultaneously with similar boundaries, which can be accomplished in Slicer software.

6. **Visual analytics with graphic display of 3D morphologic variability or changes over time**
   Registered surface models can be visualized using contrasting opaque or semi-transparent colors, and the surface distances between two time points can be graphically displayed with color-coded maps. The overlays provide visual qualitative assessment of the location and direction of changes or morphologic differences. The graphical display of color-coded or "heat" maps may contain information computed using closest or corresponding surface points, statistical significance *P* value maps of group and interaction comparisons, and vector maps and displayed within Slicer CMF (Fig. 11-10).
7. **Quantitative measurements**
   a. **Volume**[52] changes can be measured in the user's software of choice, such as Slicer, ITK-SNAP, Dolphin, or Invivo, to reflect the overall changes in size, such as for the airway, condyles, or bone graft.

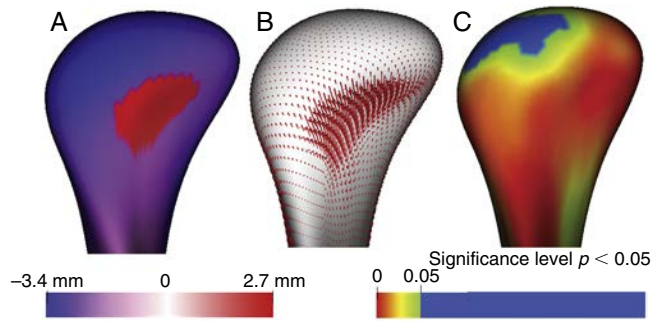


FIGURE 11-10 Assessment of surface distances between two time points displayed with color-coded maps: signed surface distances map (A), vector map (B), and *P* value map (C).

   b. **3D linear surface distances in triangular meshes**
      i. Based on **observer-defined landmarks**[62]: 3D landmarks should be identified using all three views (axial, sagittal, and coronal), for example, in the Slicer software and use a reference surface model for consistency of landmark location. Different 3D surface mesh models can be loaded simultaneously, for example, in the Slicer software, and the landmark coordinates (*x*, *y*, and *z*) are generated and displayed for each landmark on T1 and the registered T2 surface models. Two kinds of quantitative assessments of the differences between landmarks can be performed:
         (a) At one time point (baseline or follow-up): The 3D linear distances correspond to the Euclidean distances between the user-defined landmarks for characterization of dimensions, for example, prior to treatment.
         (b) Differences between two time points (T1 and T2): The 3D linear distances in each plane of the space for displacements of the maxilla or mandible (or

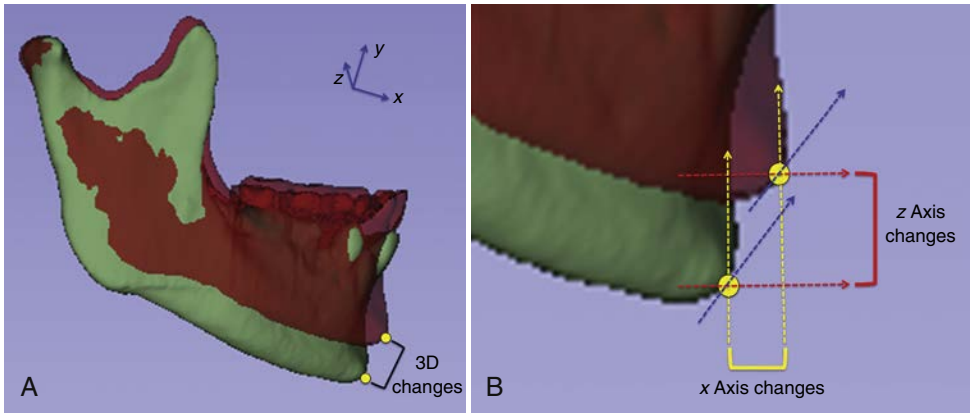


FIGURE 11-11 Representation of the three-dimensional (3D) differences and its components in the 3D space (*x* and *z* components) between two different time points (A and B) that were registered on the cranial base.

both) can be measured. The 3D measurements correspond to the Euclidean distances[84] between the T1 and T2 landmarks 9 (Fig. 11-11).

ii. Based on thousands of points in triangular meshes automatically defined in the surface models:

(a) 3D surface distances computed at the vertices of the triangular meshes can be computed as **closest points** between noncorrespondent surfaces meshes. The computation of the surface distances can be stored as color-coded 3D linear distances within .obj, .ply, or .vtk file formats in the user software of choice, such as Paraview (http://www.paraview.org)[85] or within Slicer (video tutorial, http://www.youtube.com/user/DCBIA).[79] Closest point 3D linear distances measure the closest distances between the vertices of the triangular meshes in two surfaces, not corresponding distances between anatomic points on two or more longitudinally obtained models (Figs. 11-12 to 11-14). Even though this standard analysis currently used by most commercial and academic software programs does not map corresponding surfaces based in anatomic geometry, if used in conjunction with semi-transparent overlays, the graphic display of the color-coded maps can help clinicians and researchers understand the complex overall surface changes. Localized measurements in closest point color-coded maps can be made from point to point or at any radius defined around the landmark.

(b) **Shape correspondence**, as computed using the SPHARM-PDM module[67] in the Slicer software, computes point-based surface models, in which all models have the same number of triangular meshes and vertices in corresponding (homologous) locations. Corresponding surface distances and vectors can then be calculated and graphically displayed in Slicer (see Figs. 11-12 and 11-14; video tutorial available at http://www.youtube.com/user/DCBIA).[79] Localized measurements in corresponding color-coded maps can be made at one point or at any radius defined around the landmark (Fig. 11-15).

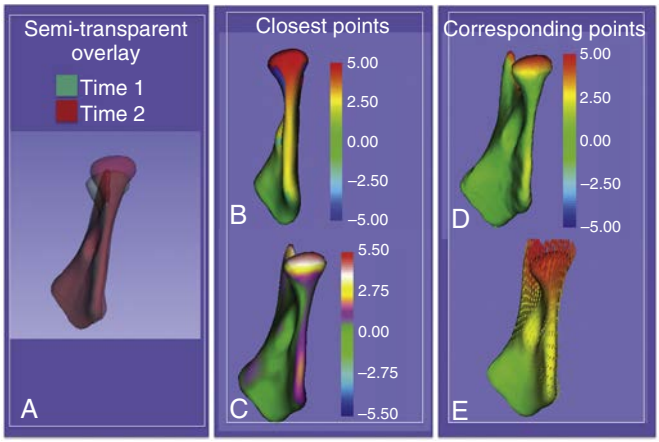


FIGURE 11-12 Posterior views of assessments between two time points. A, Semi-transparent overlay. B and C, Quantitative color-coded maps of the differences between closest distances of two time points represented by color-coded maps with five colors in *B* and seven colors in *C* to better define the regional measurements. D, Quantitative color-coded maps of the differences between corresponding points represented by color-coded maps. E, The vectors of the growth direction based on corresponding points.

(c) Quantification of directional changes in each plane of the 3D space (**3D components**)

The components in each plane of the space of the 3D linear distances can be measured based on observer-defined landmarks or automatically defined points. The distances between corresponding coordinates of corresponding landmarks can be quantified in the transversal (*x*-axis), AP (*y*-axis), and vertical (*z*-axis) direction using Slicer software (see Fig. 11-11).

c. **3D angular measurements:** 3D angular measurements between lines or planes defined in common 3D coordinate systems can be used to quantify pitch, roll, and yaw of the whole skull or only the mandible or maxilla. Evaluations using each of the three views (sagittal, coronal, and axial) allow these angles to be measured by the intersection of two lines based on the coordinates of landmarks or by the intersection of two planes being each from different time points.

Such angular measurements can be accomplished in Slicer for either characterization of facial morphology at any time point or for comparison of rotational changes between time points. Positive and negative values can be used to indicate rotations in different directions, such as clockwise or counterclockwise rotation. The choice of which landmarks or planes should be selected depends on which kind of evaluation researchers would perform to answer their aims. For example, mandibular rotations between two time points can be assessed in the following ways: pitch can be measured as the angle obtained by the intersection of the planes through the right and left condylion and pogonion in each time point or by the intersection of two lines through the pogonion and medium point between right and left condylion in the sagittal view; roll is calculated by the intersection of two lines through the right and left condylion in the coronal view; and yaw is calculated by the intersection of two lines through the right and left condylion in the axial view (Fig. 11-16).

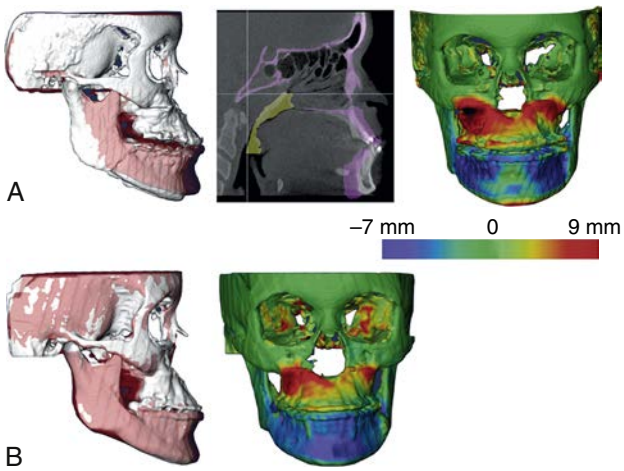


FIGURE 11-13 Assessment of postsurgical changes relative to the cranial base using overlays and closest point color coded maps. The overlays aid verification of whether the closest point color map properly represents the changes. Note that for the large mandibular setback with surgery, at splint removal the closest point color-coded map fails to quantify the changes in the chin. **A,** As shown in the lateral cross-sectional view, the closest point of the time 1 facial surface is close to the lingual surface of the time 2 and the closest point measurements are minimized. **B,** At 1 year after surgery, because the mandibular displacement is smaller, the color-code map better represents the changes.

TREATMENT PLANNING WITH COMPUTER-ASSISTED SURGERY

The methods for CAS systems in jaw surgery follow procedures from the image scanners to the operating room and have included commercially a number of systems, including Medical Modeling (3D Systems Healthcare, Littleton, CO)[86] and Maxilim (Medicim, Mechelen, Belgium).[72] The advantages of these systems are that they do not require time or computer expertise from the surgeon, and for a service fee, the commercial companies construct surface models from CBCTs and impressions or digital dental casts registered to the CBCT, perform the virtual surgery, and print surgical splints. The CAS steps include (1) data acquisition (collection of diagnostic data), (2) image segmentation, (3) quantitative measurements for diagnostic purposes (Fig. 11-17), (4) planning and simulation (preparation of the operative plan by using the virtual anatomy, simulation of the outcome with 3D printed surgical guides, grafts, or prosthetic repair), and (5) intraoperative guidance (assistance for intraoperative realization of the virtual plan). CAS steps 1 to 3 are described in detail in this chapter's section called Step-by-Step Image Analysis Procedures.

Surgical Planning and Simulation

After establishment of the diagnosis, the next step is to use the 3D representations of the anatomy to plan and simulate the surgical intervention. In orthognathic surgery, corrective interventions designate procedures that do not require an extrinsic graft, and reconstructive interventions are designated for situations in which a graft is used. In corrective procedures, it is important to determine the location of the surgical cuts, to plan the movements of the bony segments relative to one another, and to achieve the desired realignment intraoperatively. In reconstructive procedures, problems consist in determining the desired implant or graft shape. In the case of implants and prostheses, the problems are to select the proper device and shape it or to fabricate an individual device from a suitable biocompatible material. With a graft, the difficulties lie in choosing the harvesting site, shaping the graft, and placing the implant or graft in the appropriate location.[87]

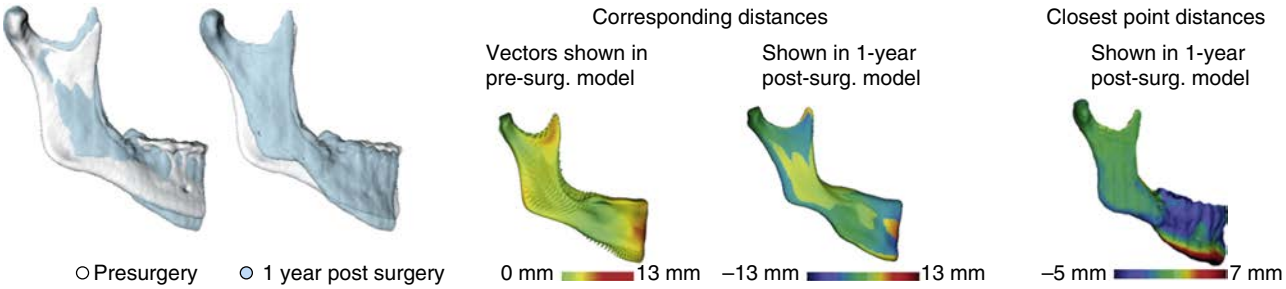


FIGURE 11-14 Comparison of mandibular postsurgical changes relative to the cranial base. Note that the qualitative pattern of changes shown in the semi-transparent overlays is more clearly quantified by the corresponding color-coded maps than the closest point maps.

Virtual osteotomies allow for the planning of cuts and position and size of fixation screws and plates, taking into account the intrinsically complex cranial anatomy; regions of thin (or absent) bone, such as the maxillary sinus anterior wall, create sudden discontinuities in the mesh, and inner structures (e.g., mandibular nerve canal) are often included in the surface model. After the virtual osteotomy, the virtual surgery with relocation of the bony segments can be performed with quantification of the planned surgical movements.[88,89] Relocation of the anatomic segments with six degrees of freedom is tracked for each of the bone fragments. This allows for the correction of the skeletal discrepancy for a given patient and simultaneous tracking of measurements of X, Y, and Z translation and rotation around each of these axes. The segment repositioning produced can be used as an initial suggestion to the surgeon; for discussions of the 3D orthodontic and surgical treatment goals for each patient; and for printing surgical splints if high-resolution scans of the dental structure are registered to the CT or CBCT and if the software tool presents an occlusion detection functionality to detect occlusal contacts, conflicts, and the precise occlusion in the virtual simulations.

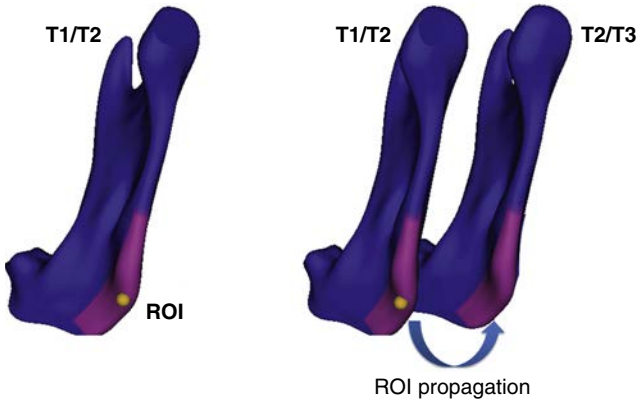


FIGURE 11-15 Corresponding surface distances can be calculated at the same region of interest (ROI) consistently by propagating the same ROI to different color maps. Selecting the ROI in the T1/T2 color map, slicer software allows to propagate the same ROI to the T2/T3 color map.

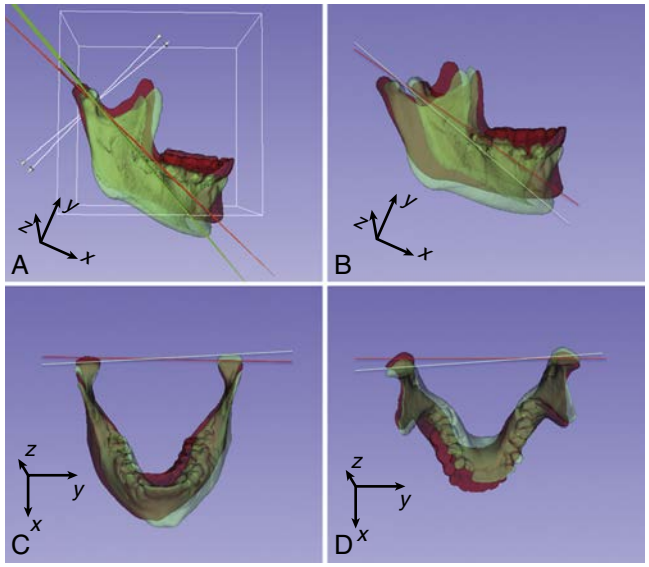


FIGURE 11-16 Representation of three-dimensional angular measurements. **A** and **B,** Pitch. **C,** Roll. **D,** Yaw.

Simulation of Soft Tissue Changes

Methods that attempt to predict facial soft tissue changes resulting from skeletal reshaping use approximation models because direct formulation and analytical resolution of the equations of continuum mechanics are not possible with such geometrical complexity. Different types of models have been proposed, including displacements of soft tissue voxels are estimated with the movements of neighboring hard tissue voxels[90]; bone displacement vectors are simply applied on the vertices of the soft tissue mesh[91]; and multilayer mass-spring models,[92] finite element models,[93,94] and mass tensor models[95] that assume biological properties of soft tissue response. In any case, thorough validation reports for all these methods are still lacking. Comparisons of the simulation with the postoperative facial surface have not yet been performed. Surgical planning functions generally do not fulfill the requirements enumerated earlier for preparation of quantitative facial tissue simulation for surgical planning. Other functionalities that have been incorporated into different software systems include simulation of muscular function,[96] distraction osteogenesis planning,[97] and four-dimensional surgery planning.[98]

Intraoperative Guidance: Surgical Navigation

In surgical procedures, achieving the desired bone segment realignment freehand is difficult. Also, segments must often be moved with very limited visibility, such as under the (swollen) skin. Approaches used currently in surgery rely largely on the

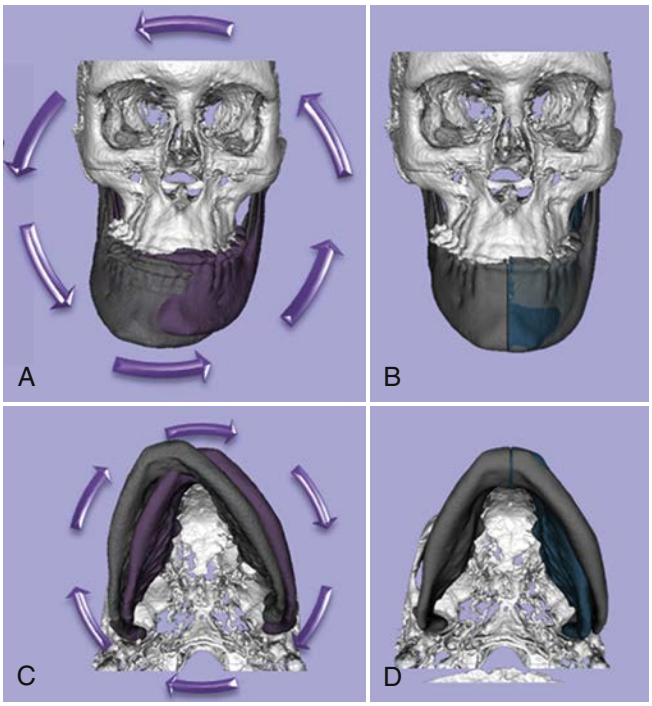


FIGURE 11-17 The use of mirroring techniques requires an initial simulation procedure step to first correct positional asymmetry (roll and yaw) followed by mirroring techniques to assess asymmetry in shape.

clinician's experience and intuition. In maxillary repositioning, for example, a combination of dental splints, compass, ruler, and intuition is used to determine the final position. It has been shown that in the vertical direction (in which the splint exerts no constraint), only limited control is achieved.[99] Although the surgical splint guides the position of the maxilla relative to the mandible, in two-jaw surgeries, the spatial position of the two jaws relative to the face is influenced by the splint precision and the transsurgical vertical assessment. As the splints are made over teeth and guide bone changes away from those teeth, small splint inaccuracies may reflect in significant bone position inaccuracies. The predictability of precise osteotomies in the wide variety of patient morphologies and consequent controlled fractures such as in the pterygoid plates, sagittal split osteotomies, or interdental cuts are still a concern. In reconstructive procedures, the problems of shaping and placing a graft or implant in the planned location also arise. Surgical navigation systems have been developed to help accurately transfer treatment plans to the operating room.

Tracking Technology

Different tracking technologies[100] for the tracking of the displacement of a mobilized fragment in the course of an osteotomy can be used with respective advantages and disadvantages:

1. In ultrasonography, an array of three ultrasound emitters is mounted on the object to be tracked, but the speed of sound value can vary with temperature changes and the calibration procedure is very delicate
2. In electromagnetic tracking, a homogeneous magnetic field is created by a generator coil. Ferromagnetic items such as implants, instruments, or the operation table can interfere strongly with these systems, distorting the measurements in an unpredictable way. Newer systems claim reduction of these effects and feature receivers the size of a needle head, possibly announcing a renewal of interest for electromagnetic tracking in surgical navigation (examples are the 3D guidance trackstar, Ascension, Burlington, VT; StealthStation AXIEM, Medtronic, Louisville, KY; and Aurora, Northern Digital Inc., Ontario, Canada)
3. Infrared (IR) optical tracking devices rely on pairs or triplets of charge-coupled devices that detect positions of IR markers. In these devices, a free line of sight is required between the cameras and markers (Fig. 11-18).

FINAL CONSIDERATIONS

The clinical use of CBCT imaging in orthodontics now has guidelines proposed by European and American associations,[10,101] and the hardware for image acquisition as well as software for image analysis have continued to develop and evolve dramatically in recent years. Careful image analysis requires combined multidisciplinary efforts among orthodontists, oral maxillofacial surgeons, radiologists, and image analysis experts to properly interpret the wealth of information now available for clinicians.

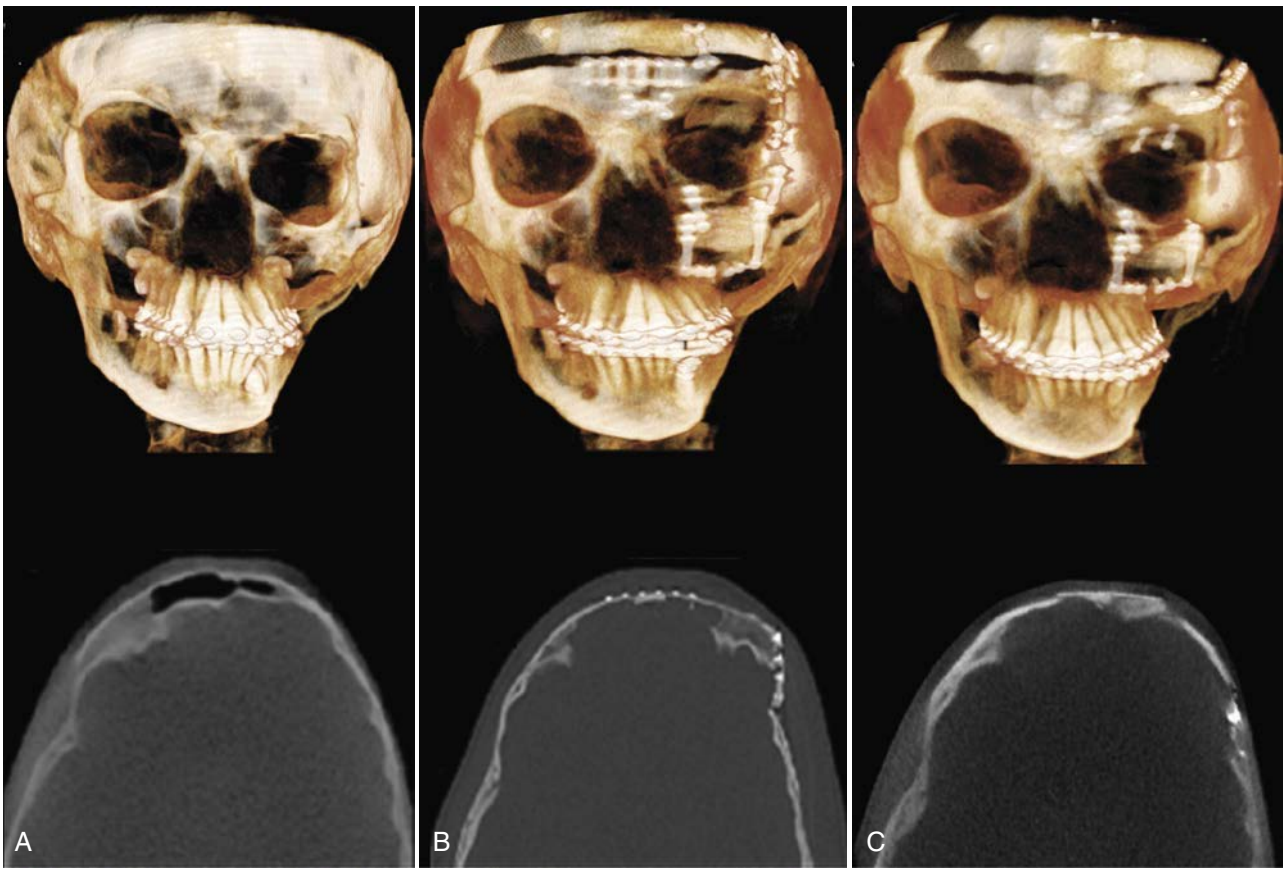


FIGURE 11-18 Improvement of orbital correction with intraoperative surgical navigation. **A,** Contour of the forehead before orbital reconstruction. **B,** Surgical correction with an undesirable outcome. **C,** Improved of symmetry after surgery performed with intraoperative navigation.

REFERENCES

1. Ludlow JB, Timothy R, Walker C, et al. Effective dose of dental CBCT—a meta analysis of published data and additional data for nine CBCT units. *Dentomaxillofac Radiol*. 2015;44:20140197.
2. Ludlow JB, Walker C. Assessment of phantom dosimetry and image quality of i-CAT FLX cone-beam computed tomography. *Am J Orthod Dentofacial Orthop*. 2013;144:802–817.
3. United States Environmental Protection Agency. Radiation Protection. http://www.epa.gov/radiation/understand/health_effects.html.
4. International Commission on Radiological Protection. *The 2007 recommendations of the International Commission on Radiological Protection*. In: Valentin J, ed. St. Louis: Elsevier; 2007:1–332.
5. Ludlow JB, Davies-Ludlow LE, White SC. Patient risk related to common dental radiographic examinations: the impact of 2007 International Commission on Radiological Protection recommendations regarding dose calculation. *J Am Dent Assoc*. 2008;139:1237–1243.
6. Brenner D, Elliston C, Hall E, Berdon W. Estimated risks of radiation-induced fatal cancer from pediatric CT. *AJR Am J Roentgenol*. 2001;176:289–296.
7. Smith-Bindman R, Lipson J, Marcus R, et al. Radiation dose associated with common computed tomography examinations and the associated lifetime attributable risk of cancer. *Arch Intern Med*. 2009;169:2078–2086.
8. Richards AG, Barbor GL, Bader JD, Hale JD. Samarium filters for dental radiography. *Oral Surg Oral Med Oral Pathol*. 1970;29:704–715.
9. Ludlow JB. A manufacturer's role in reducing the dose of cone beam computed tomography examinations: effect of beam filtration. *Dentomaxillofac Radiol*. 2011;40:115–122.
10. Evans C, Scarfe W, Ahmad M, et al. Clinical recommendations regarding use of cone beam computed tomography in orthodontic treatment. Position statement by the American Academy of Oral and Maxillofacial Radiology. *Oral Surg Oral Med Oral Pathol Oral Radiol*. 2013;116:238–257.
11. Molen AD. Considerations in the use of cone-beam computed tomography for buccal bone measurements. *Am J Orthod Dentofacial Orthop*. 2010;137(suppl):S130–S135.
12. Leung CC, Palomo L, Griffith R, Hans MG. Accuracy and reliability of cone-beam computed tomography for measuring alveolar bone height and detecting bony dehiscences and fenestrations. *Am J Orthod Dentofacial Orthop*. 2010;137(suppl):S109–S119.
13. Tai K, Hotokezaka H, Park JH, et al. Preliminary cone-beam computed tomography study evaluating dental and skeletal changes after treatment with a mandibular Schwarz appliance. *Am J Orthod Dentofacial Orthop*. 2010;138. 262.e261–262.e211; discussion 262–263.
14. Becker A, Chaushu S, Casap-Caspi N. Cone-beam computed tomography and the orthosurgical management of impacted teeth. *J Am Dent Assoc*. 2010;141(suppl 3):14S–18S.
15. Botticelli S, Verna C, Cattaneo PM, et al. Two- versus three-dimensional imaging in subjects with unerupted maxillary canines. *Eur J Orthod*. 2011;33:344–349.
16. Katheria BC, Kau CH, Tate R, et al. Effectiveness of impacted and supernumerary tooth diagnosis from traditional radiography versus cone beam computed tomography. *Pediatr Dent*. 2010;32:304–309.
17. Van Elslande D, Heo G, Flores-Mir C, et al. Accuracy of mesiodistal root angulation projected by cone-beam computed tomographic panoramic-like images. *Am J Orthod Dentofacial Orthop*. 2010;137(suppl):S94–S99.
18. Shemesh H, Cristescu RC, Wesselink PR, Wu MK. The use of cone-beam computed tomography and digital periapical radiographs to diagnose root perforations. *J Endod*. 2011;37:513–516.
19. Sherrard JF, Rossouw PE, Benson BW, et al. Accuracy and reliability of tooth and root lengths measured on cone-beam computed tomographs. *Am J Orthod Dentofacial Orthop*. 2010;137:S100–S108.
20. Miyazawa K, Kawaguchi M, Tabuchi M, Goto S. Accurate pre-surgical determination for self-drilling miniscrew implant placement using surgical guides and cone-beam computed tomography. *Eur J Orthod*. 2010;32:735–740.
21. Fayed MM, Pazera P, Katsaros C. Optimal sites for orthodontic mini-implant placement assessed by cone beam computed tomography. *Angle Orthod*. 2010;80:939–951.
22. Ahmad M, Hollender L, Anderson Q, et al. Research diagnostic criteria for temporomandibular disorders (RDC/TMD): development of image analysis criteria and examiner reliability for image analysis. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2009;107:844–860.
23. Alexiou K, Stamatakis H, Tsiklakis K. Evaluation of the severity of temporomandibular joint osteoarthritic changes related to age using cone beam computed tomography. *Dentomaxillofac Radiol*. 2009;38:141–147.
24. Helenius LM, Hallikainen D, Helenius I, et al. Clinical and radiographic findings of the temporomandibular joint in patients with various rheumatic diseases. A case-control study. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2005;99:455–463.
25. Koyama J, Nishiyama H, Hayashi T. Follow-up study of condylar bony changes using helical computed tomography in patients with temporomandibular disorder. *Dentomaxillofac Radiol*. 2007;36:472–477.
26. Dworkin SF, LeResche L. Research diagnostic criteria for temporomandibular disorders: review, criteria, examinations and specifications, critique. *J Craniomandib Disord*. 1992;6:301–355.
27. Schiffman EL, Ohrbach R, Truelove EL, et al. The Research Diagnostic Criteria for Temporomandibular Disorders. V: methods used to establish and validate revised Axis I diagnostic algorithms. *J Orofac Pain*. 2010;24:63–78.
28. Truelove E, Pan W, Look JO, et al. The Research Diagnostic Criteria for Temporomandibular Disorders. III: validity of Axis I diagnoses. *J Orofac Pain*. 2010;24:35–47.
29. Schiffman EL, Truelove EL, Ohrbach R, et al. The Research Diagnostic Criteria for Temporomandibular Disorders. I: overview and methodology for assessment of validity. *J Orofac Pain*. 2010;24:7–24.
30. Kapila S, Conley RS, Harrell WE. The current status of cone beam computed tomography imaging in orthodontics. *Dentomaxillofac Radiol*. 2011;40:24–34.
31. Bryndahl F, Eriksson L, Legrell PE, Isberg A. Bilateral TMJ disk displacement induces mandibular retrognathia. *J Dent Res*. 2006;85:1118–1123.
32. Cevidanes LH, Walker D, Schilling J, et al. 3D osteoarthritic changes in TMJ condylar morphology correlates with specific systemic and local biomarkers of disease. *Osteoarthritis Cartilage*. 2014;22:1657–1667.
33. Roemhildt ML, Coughlin KM, Peura GD, et al. Effects of increased chronic loading on articular cartilage material properties in the lapine tibio-femoral joint. *J Biomech*. 2010;43:2301–2308.
34. Scott CC, Athanasiou KA. Mechanical impact and articular cartilage. *Crit Rev Biomed Eng*. 2006;34:347–378.
35. Verteramo A, Seedhom BB. Effect of a single impact loading on the structure and mechanical properties of articular cartilage. *J Biomech*. 2007;40:3580–3589.
36. Gallo LM, Gössi DB, Colombo V, Palla S. Relationship between kinematic center and TMJ anatomy and function. *J Dent Res*. 2008;87:726–730.
37. Fredriksson L, Alstergren P, Kopp S. Tumor necrosis factor-alpha in temporomandibular joint synovial fluid predicts treatment effects on pain by intra-articular glucocorticoid treatment. *Mediators Inflamm*. 2006;2006:59425.
38. Nordahl S, Alstergren P, Eliasson S, Kopp S. Radiographic signs of bone destruction in the arthritic temporomandibular joint with special reference to markers of disease activity. A longitudinal study. *Rheumatology (Oxford)*. 2001;40:691–694.
39. Alstergren P, Kopp S. Prostaglandin E2 in temporomandibular joint synovial fluid and its relation to pain and inflammatory disorders. *J Oral Maxillofac Surg*. 2000;58:180–186; discussion 186–188.
40. Abramson Z, Susarla SM, Lawler M, et al. Three-dimensional computed tomographic airway analysis of patients with obstructive sleep apnea treated by maxillomandibular advancement. *J Oral Maxillofac Surg*. 2011;69:677–686.
41. Schendel S, Powell N, Jacobson R. Maxillary, mandibular, and chin advancement: treatment planning based on airway anatomy in obstructive sleep apnea. *J Oral Maxillofac Surg*. 2011;69:663–676.

42. Iwasaki T, Saitoh I, Takemoto Y, et al. Evaluation of upper airway obstruction in Class II children with fluid-mechanical simulation. *Am J Orthod Dentofacial Orthop*. 2011;139:e135–e145.
43. Conley RS. Evidence for dental and dental specialty treatment of obstructive sleep apnoea. Part 1: the adult OSA patient and Part 2: the paediatric and adolescent patient. *J Oral Rehabil*. 2011;38:136–156.
44. Edwards SP. Computer-assisted craniomaxillofacial surgery. *Oral Maxillofac Surg Clin North Am*. 2010;22:117–134.
45. Jayaratne YS, Zwahlen RA, Lo J, Cheung LK. Three-dimensional color maps: a novel tool for assessing craniofacial changes. *Surg Innov*. 2010;17:198–205.
46. Kim YI, Park SB, Son WS, Hwang DS. Midfacial soft-tissue changes after advancement of maxilla with Le Fort I osteotomy and mandibular setback surgery: comparison of conventional and high Le Fort I osteotomies by superimposition of cone-beam computed tomography volumes. *J Oral Maxillofac Surg*. 2011;69:e225–e233.
47. Almeida RC, Cevidanes LH, Carvalho FA, et al. Soft tissue response to mandibular advancement using 3D CBCT scanning. *Int J Oral Maxillofac Surg*. 2011;40:353–359.
48. Kapila SD, Nervina JM. CBCT in orthodontics: assessment of treatment outcomes and indications for its use. *Dentomaxillofac Radiol*. 2015;44:20140282.
49. Ma WY, Manjunath BS. EdgeFlow: a technique for boundary detection and image segmentation. *IEEE Trans Image Process*. 2000;9:1375–1388.
50. Lie WN. Automatic target segmentation by locally adaptive image thresholding. *IEEE Trans Image Process*. 1995;4:1036–1041.
51. Moon N, Bullitt E, Leemput K, Gerig G. Model-based brain and tumor segmentation. In: Casturi R, Laurendeau D, Suen C, eds. *Proceedings of the 16th International Conference on Pattern Recognition*. IEE Computer Society; 2002:528–531.
52. Thompson PM, MacDonald D, Mega MS, et al. Detection and mapping of abnormal brain structure with a probabilistic atlas of cortical surfaces. *J Comput Assist Tomogr*. 1997;21:567–581.
53. Maes F, Collignon A, Vandermeulen D, et al. Multimodality image registration by maximization of mutual information. *IEEE Trans Med Imaging*. 1997;16:187–198.
54. Cevidanes LH, Styner MA, Proffit WR. Image analysis and superimposition of 3-dimensional cone-beam computed tomography models. *Am J Orthod Dentofacial Orthop*. 2006;129:611–618.
55. Cevidanes LH, Heymann G, Cornelis MA, et al. Superimposition of 3-dimensional cone-beam computed tomography models of growing patients. *Am J Orthod Dentofacial Orthop*. 2009;136:94–99.
56. da Motta AT, de Assis Ribeiro Carvalho F, Oliveira AE, et al. Superimposition of 3D cone-beam CT models in orthognathic surgery. *Dental Press J Orthod*. 2010;15:39–41.
57. Bookstein F. *Morphometric Tools for Landmark Data*. Cambridge: Cambridge University Press; 1991.
58. Moyers RE, Bookstein FL. The inappropriateness of conventional cephalometrics. *Am J Orthod*. 1979;75:599–617.
59. De Groote I, Lockwood CA, Aiello LC. Technical note: a new method for measuring long bone curvature using 3D landmarks and semi-landmarks. *Am J Phys Anthropol*. 2010;141:658–664.
60. Kenyhercz MW, Klales AR, Kenyhercz WE. Molar size and shape in the estimation of biological ancestry: a comparison of relative cusp location using geometric morphometrics and interlandmark distances. *Am J Phys Anthropol*. 2014;153:269–279.
61. Paniagua B, Alhadidi A, Cevidanes L, et al. Mandibular asymmetry characterization using generalized tensor-based morphometry. *Proc IEEE Int Symp Biomed Imaging*. 2011;2011:1175–1178.
62. Rohr R. *Landmark-Based Image Analysis: Using Geometric and Intensity Models*. London: Kluwer Academic Publishers; 2001.
63. Dean D, Hans MG, Bookstein FL, Subramanyan K. Three-dimensional Bolton-Brush Growth Study landmark data: ontogeny and sexual dimorphism of the Bolton standards cohort. *Cleft Palate Craniofac J*. 2000;37:145–156.
64. Gunz P, Mitteroecker P, Bookstein F. Semilandmarks in three dimensions. In: Slice D, ed. *Modern Morphometrics in Physical Anthropology*. New York: Kluwer Academic Publishers; 2004.
65. Andresen PR, Bookstein FL, Conradsen K, et al. Surface-bounded growth modeling applied to human mandibles. *IEEE Trans Med Imaging*. 2000;19:1053–1063.
66. Subsol G, Thirion JP, Ayache N. A scheme for automatically building three-dimensional morphometric anatomical atlases: application to a skull atlas. *Med Image Anal*. 1998;2:37–60.
67. Gerig G, Jomier M, Chakos M. Valmet: a new validation tool for assessing and improving 3D object segmentation. In: Niessen W, Viergever M, eds. *MICCAI 2001: Proceedings of the International Society and Conference Serien on Medical Image Computing and Computer-Assisted Intervention*. Berlin; 2001:516–528.
68. Styner M, Oguz I, Xu S, Brechbühler C, et al. Framework for the statistical shape analysis of brain structures using SPHARM-PDM. *Insight J*. 2006:242–250.
69. Paniagua B, Cevidanes L, Walker D, et al. Clinical application of SPHARM-PDM to quantify temporomandibular joint osteoarthritis. *Comput Med Imaging Graph*. 2011;35:345–352.
70. Paniagua B, Cevidanes L, Zhu H, Styner M. Outcome quantification using SPHARM-PDM toolbox in orthognathic surgery. *Int J Comput Assist Radiol Surg*. 2011;6:617–626.
71. 3DMD software. 3DMD Vultus. http://www.3dmd.com/3dmd-software.
72. Maxilim. http://www.medicim.com/en/products.
73. Dolphin Imaging. http://www.dolphinimaging.com.
74. Invivo. http://dental.anatomage.com/specialty/orthodontist.
75. Simplant. http://www.dentsply.com/en-uk-implants/digital-solutions/simplant.html/Implants/Digital-solutions/SIMPLANT/Software/c/3000106.html: Simplant.
76. Turtleseg. http://www.turtleseg.org.
77. ITKSNAP. http://www.itksnap.org/pmwiki/pmwiki.php.
78. 3DSlicer. http://www.slicer.org.
79. Videos Tutoriais DCBIA. https://http://www.youtube.com/user/DCBIA. Videos Tutoriais DCBIA; Accessed April 30, 2015.
80. Slicer CMF. Available at https://drive.google.com/a/umich.edu/folderview?id=0B45O4BAuzBh6QzcxSndRcVRMeFk&usp=sharing 3DSlicer CMF.
81. Nguyen T, Cevidanes L, George W, . Validation of 3D mandibular regional superimposition methods for growing patients [abstract]. *J Dent Res*. 2014;93(Spec Iss B):784.
82. Ruellas A, Huanca L, Gomes MT, et al. Comparison and Reproducibility of Two Maxillary Regional Registration Methods. *J Dent Res*. 2015;94(Spec Iss A):0915.
83. Ludlow JB, Gubler M, Cevidanes L, et al. Precision of cephalometric landmark identification: cone-beam computed tomography vs conventional cephalometric views. *Am J Orthod Dentofacial Orthop*. 2009;136.312.e311–310; discussion 312–313.
84. Khambay B, Ullah R. Current methods of assessing the accuracy of three-dimensional soft tissue facial predictions: technical and clinical considerations. *Int J Oral Maxillofac Surg*. 2015;44:132–138.
85. Paraview. http://www.paraview.org.
86. Medical Modeling. http://www.medicalmodeling.com.
87. Chapuis J. *Computer-Aided Cranio-Maxillofacial Surgery*. Bern: University of Bern; 2006.
88. Chapuis J, Schramm A, Pappas I, et al. A new system for computer-aided preoperative planning and intraoperative navigation during corrective jaw surgery. *IEEE Trans Inf Technol Biomed*. 2007;11:274–287.
89. De Momi E, Chapuis J, Pappas I, et al. Automatic extraction of the mid-facial plane for cranio-maxillofacial surgery planning. *Int J Oral Maxillofac Surg*. 2006;35:636–642.
90. Schutyser F, Poorten VV, Van Cleynenbreugel J, et al. An image-based 3D planning environment for hemicricolaryngectomy and reconstruction by tracheal autotransplantation. *Comput Aided Surg*. 2000;5:166–174.
91. Xia J, Samman N, Yeung RW, et al. Three-dimensional virtual reality surgical planning and simulation workbench for orthognathic surgery. *Int J Adult Orthodon Orthognath Surg*. 2000;15:265–282.
92. Teschner M, Girod S, Girod B. 3-D simulation of craniofacial surgical procedures. *Stud Health Technol Inform*. 2001;81:502–508.

93. Westermark A, Zachow S, Eppley BL. Three-dimensional osteotomy planning in maxillofacial surgery including soft tissue prediction. *J Craniofac Surg*. 2005;16:100–104.
94. Chabanas M, Luboz V, Payan Y. Patient specific finite element model of the face soft tissues for computer-assisted maxillofacial surgery. *Med Image Anal*. 2003;7:131–151.
95. Keeve E, Girod S, Kikinis R, Girod B. Deformable modeling of facial tissue for craniofacial surgery simulation. *Comput Aided Surg*. 1998;3: 228–238.
96. Zachow S, Gladilin E, Zeilhofer H, Sader R. *Improved 3D osteotomy planning in craniomaxillofacial surgery. Proceedings of the Medical Image Computing and Computer-Assisted Intervention 4th International Conference*. The Netherlands: Ultrecht; 2001.
97. Gladilin E, Zachow S, Deuflhard P, Hege HC. Anatomy- and physics-based facial animation for craniofacial surgery simulations. *Med Biol Eng Comput*. 2004;42:167–170.
98. Gateno J, Teichgraeber JF, Xia JJ. Three-dimensional surgical planning for maxillary and midface distraction osteogenesis. *J Craniofac Surg*. 2003;14:833–839.
99. Vandewalle P, Schutyser F, Van Cleynenbreugel J, Suetens P. Modeling of facial soft tissue growth for maxillofacial surgery planning environments. *IS4TH*. 2003:27–37.
100. Langlotz F. Localizers and trackers for computer assisted freehand navigation. In: Picard F, Noite L, Digiola A, Jaramaz B, eds. *Hip and Knee Surgery—Navigation, Robotics, and Computer Assisted Surgical Tools*. Oxford University Press; 2004:51–53.
101. European Commission ISSN 1681-6803, Euratom, Luxembourg, 2012. https://ec.europa.eu/energy/sites/ener/files/documents/172.pdf.

12

Upper Airway, Cranial Morphology, and Sleep Apnea

Juan Martin Palomo, Hakan El, Leena Palomo, and Kingman P. Strohl

OUTLINE

Introduction, 319
Airway Complications Have Far-Reaching Effects, 319
Anatomy and Growth, 320
Overview, 320
Hyoid Bone Position and Morphologic Features, 322
Relationship of Different Skeletal Patterns to Airway Morphologic Structure, 323
Airway Measurements and Imaging, 323
Overview, 323
Cone-Beam Computed Tomography, 326
Acoustic Rhinometry, 327
Pharyngometry, 329
Influence of Orthodontic Treatment on the Airway, 329
Treatment Including Extractions, 329
Rapid Maxillary Expansion, 330
Functional Orthopedic Appliances, 330
Orthognathic Surgery, 331
Summary of Orthodontic Treatment Effects on the Airway, 332
Sleep-Disordered Breathing: Airway Disorders and Management, 333
Definitions and Testing Reports, 333
Classifications of Sleep-Disordered Breathing, 334
Obstructive Sleep Apnea, 334
Upper Airway Resistance Syndrome, 335
Central Sleep Apnea Syndrome, 335
Sleep Hypoventilation Syndromes, 335
Epidemiologic Factors, 335
Pathophysiologic Factors, 335
Clinical Presentation, 335
Physical Characteristics, 336
Clinical Prediction of Significant Sleep-Disordered Breathing, 336
Tests and Questionnaires, 336
Friedman Classification, 336
Mallampati Score Method, 337
Kushida Index, 338
Berlin Questionnaire, 338
STOPBang Questionnaire, 338
Epworth Sleepiness Scale, 338
Cephalometric Analysis, 338
Sleep Disorders Questionnaire, 338
Diagnostic Testing of Obstructive Sleep Apnea, 340
Treatment Modalities, 340
Treatment Options, 340
Lifestyle and Behavioral Modification, 341
Positive Airway Pressure, 341
Oral Appliances, 341
Surgical Treatment, 343
Oropharyngeal Exercises, 343
Upper Airway Electrical Neurostimulation, 344
Preventive Management, 344
Importance of a Multidisciplinary Approach, 345
Summary and Conclusions, 347

INTRODUCTION

Air is essential for human life. The path that air takes into the body and through to the lungs is called the *airway*. Since much of the upper airway (UA) is part of the craniofacial complex, the orthodontist can observe the airway and modulate it in case of potential obstructions. This places the orthodontist in a strategic position to intervene when airway complications exist or may develop. Since airway obstructions can have far-reaching effects, the importance of airway assessment has been part of orthodontic literature for over a century. Now, more than ever, with the emergence of state-of-the-art technologies and treatment options, the orthodontist has a responsibility and obligation to recognize respiratory problems.

AIRWAY COMPLICATIONS HAVE FAR-REACHING EFFECTS

When nasal obstruction occurs, the body is forced to breathe through the mouth; the ensuing effects have yet to be fully elucidated. Reportedly, when an individual switches to mouth breathing, the body adapts through extended head posture, anterior tongue position, and low mandibular position.[1] When the nasal obstruction is chronic in young children, these long-term adaptations can affect craniofacial growth. However, disagreement exists among investigators as to whether nasal obstruction is the cause of malocclusion or whether it is the result of the craniofacial pattern. Historically, the first mention of UA obstruction forcing individuals to be mouth breathers

was made by Meyer in 1869.[2] He describes subjects who had to adapt to mouth breathing as clinically having a more nasal voice, an open mouth, and pouting lips.

In 1872, Tomes[3] described the *adenoid facies*, and the concept that facial form can be influenced by enlarged adenoids became accepted as a hypothesis. In 1907, Angle[4] included airway obstruction as an important etiologic factor in malocclusion. Tomes' view was supported by many leading orthodontists, including Todd and Broadbent in 1939,[5] and was later referred to as the "long face syndrome" in the orthodontic literature.[6] According to Moss' functional matrix theory,[7] nasal breathing allows the proper growth and development of the craniofacial complex interacting with other functions such as mastication and swallowing.[8] A narrow maxilla, hypotonia, and a small nose are also descriptions consistent with Moss' functional matrix theory, in which the lack of function (nasal breathing) creates a lack of area development.

A more complete analysis was later suggested by Linder-Aronson, who described in greater detail the craniofacial characteristics of the adenoid facies.[9] The adenoid facies craniofacial characteristics include open-mouth posture, hypotonia, narrow base in the area of the ala of the nose, proclined maxillary incisors, proclined lower lip, increased lower facial height, high mandibular angle, narrow maxilla, high palate, anterior tongue position, frequent mandibular retrognathia, and a vacant facial expression.[1,10,11] Despite still being a topic of controversy, recent articles reinforce the theories that nasal obstruction is the cause of craniofacial changes, as the body attempts to adapt to the abnormality.[12,13]

Meyer was also the first to report that improvements may almost immediately occur after the removal of the obstruction[2]; since then, through the years, several reports show craniofacial improvements after the nasal breathing is normalized at any age but with more dramatic improvements in younger patients.[14,15]

Another adaptation of mouth breathing is that the upper lip's muscular tonicity is lost. A short and flaccid upper lip may be unable to cover the anterior teeth, thereby displaying more maxillary anterior teeth when at rest. Additionally, gingival display is increased and the potential of a gummy smile appearance increases. As a result of lip incompetence, salivary flow to the area is decreased, resulting in reduced effects of salivary cleaning mechanisms. The incidence of caries is increased, and maxillary anterior teeth are most affected. The gingiva also repeatedly alternates from wet (with saliva from the tongue and lip) to dry (as the short incompetent lip returns to its open rest position). This repeated wet-dry cycle results in a histologically incomplete keratinization of the gingiva. Clinically, the gingiva has a red color, rolled gingival margins, and bulbous papilla.[16] Inflammation may occur alone or with hyperplasia. Mouth breathing, increased lip separation, and decreased upper lip coverage at rest have all been associated with higher levels of plaque and gingival inflammation.[17] As a result of long-term plaque accumulation and poor oral hygiene, a mouth breather's gingivitis can progress to pocket formation and bone loss.

Another consequence of mouth breathing is that air brought into the body through the nose is different from air brought in through the mouth. Only the nose is able to filter, warm, moisturize, and dehumidify air; furthermore, each nostril independently and synergistically functions.[18] Small amounts of nitric oxide are made by the nose and sinus mucous membranes. Nitric oxide is lethal to bacteria and viruses and is also known to increase oxygen absorption in the lungs from 10% to 25%. Breathing through the nose increases blood circulation and blood oxygen and carbon dioxide levels, slows the breathing rate, and improves overall lung volumes as a consequence to providing almost double the resistance than when breathing through the mouth.[19] The nasal resistance is crucial to maintain adequate elasticity of the lungs.[20] There are reports of hypoxemia (low levels of oxygen in the blood), hypercarbia (high levels of carbon dioxide in the blood), and hypoventilation after only 24 hours of nasal obstruction, forcing the individual to breathe through the mouth. Oxygenation changes are the foundation of systemic consequences of airway obstruction. Adults who habitually breathe through the mouth, attributable to nasal obstruction, are more likely to have sleep disorders and attention-deficit/hyperactivity disorder (ADHD).[21] Patients with obstructive sleep apnea (OSA) are noted to have high blood pressure as a result of overactivation of the sympathetic nervous system. Exaggerated negative intrathoracic pressure during obstructive apneas further increases left ventricular afterload, reduces cardiac output, and may promote the progression of heart failure. Intermittent hypoxia and postapneic reoxygenation cause vascular endothelial damage, which can progress to atherosclerosis and, consequently, to coronary artery disease and ischemic cardiomyopathy, although the mechanisms of progression have not yet been fully explained. Chronic OSA is also characterized by apnea, hypoxia, and increased sympathetic nervous activity and, when present in heart failure, is associated with increased risk of death. Since heart failure, atherosclerosis, and coronary artery disease remain major causes of mortality in the industrialized countries, the significance of investigating, diagnosis, and managing OSA has been emphasized to prevent its development and progression.[22]

ANATOMY AND GROWTH

Overview

The respiratory tract is the complete path that air takes through the nose or mouth, ending at the lungs. The respiratory tract can be divided into the upper respiratory tract and lower respiratory tract, also known as UA and lower airways (LA). The LA comprises the trachea, bronchi, and lungs. The UA, which is more relevant to orthodontics, includes the nasal cavity, the pharynx, and the larynx. However, no consensus exists among investigators as to which terminology to use when describing the anatomic limits of the UA.[23] According to *Gray's Anatomy* classification, the pharyngeal airway can be divided into the following three regions of interest, with one subdivision very relevant to orthodontics[24,25] (Fig. 12-1):

1. Nasopharynx: Is located between the nares and hard palate.
2. Oropharynx: Is located from the soft palate to the upper border of the epiglottis. The velopharynx or retropalatal oropharynx is located between the soft palate and the posterior pharyngeal wall and is found within the oropharynx.
3. Laryngopharynx or hypopharynx: Is located from the base of the tongue to the inferior border of the cricoid cartilage.

The UA forms the passage for movement of air from the nose to the lungs and also participates in other physiologic functions such as phonation and deglutition.[26] The properties of the UA are a compromise among these different functions, which variably require maintenance of patency (during breathing) or closure of the airway (as in swallowing).

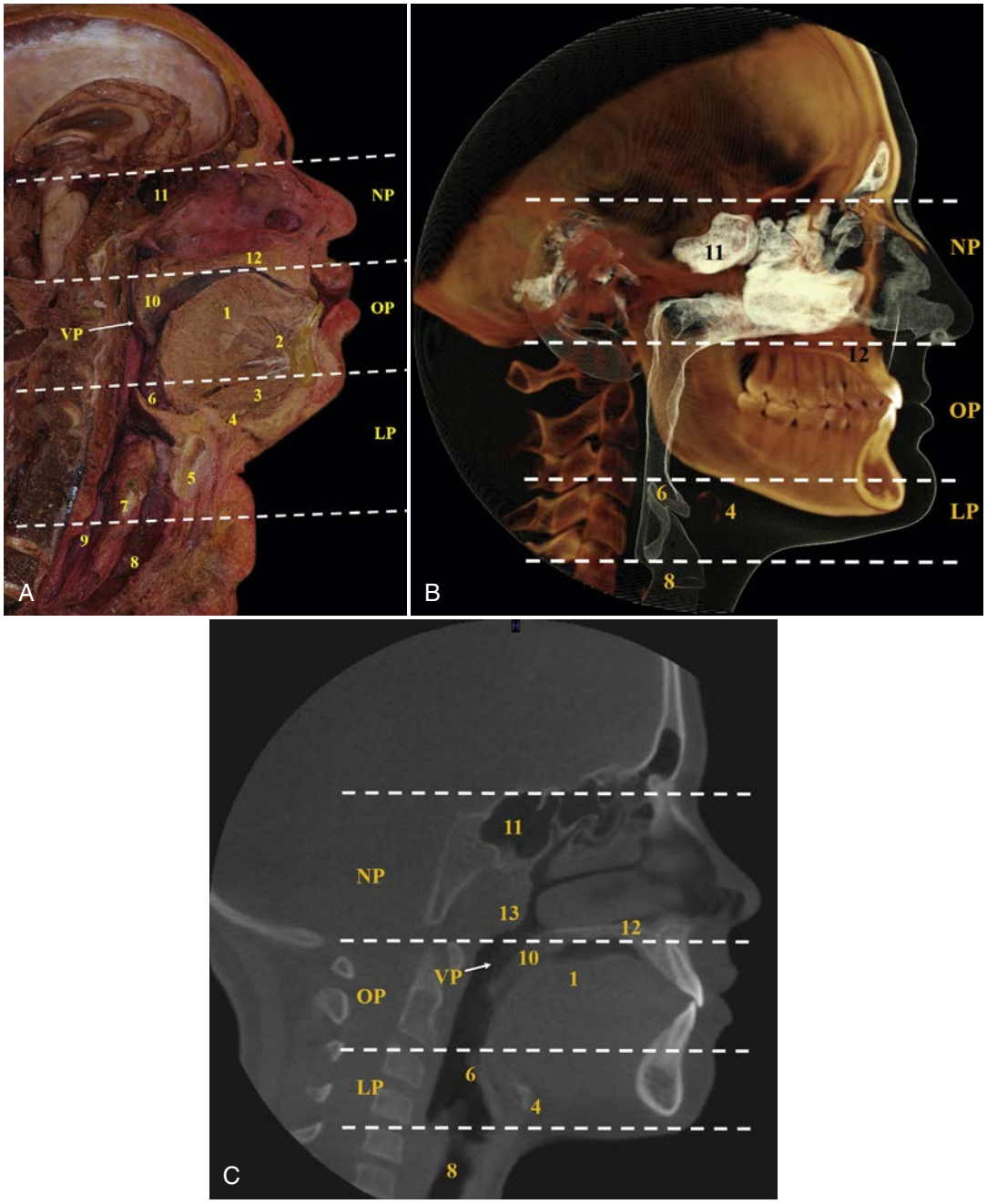


FIGURE 12-1 Anatomic midsagittal view shows human (adult) upper airway and surrounding structures. **A,** Cadaver. **B,** Cone-beam computed tomography (CBCT) volumetric view. **C,** CBCT sagittal slice. *NP,* Nasopharynx; *OP,* oropharynx; *VP,* velopharynx; *LP,* laryngopharynx; *1,* tongue; *2,* genioglossus muscle; *3,* geniohyoid muscle; *4,* hyoid bone; *5,* thyroid cartilage; *6,* epiglottis; *7,* posterior part of cricoid cartilage; *8,* trachea; *9,* esophagus; *10,* soft palate; *11,* sphenoid air-sinus; *12,* hard palate; *13,* pharyngeal tonsils (adenoids). (**A,** Courtesy of Dr. Michael Landers.)

The muscles surrounding the airway that actively constrict and dilate the UA lumen[27,28] can be classified into four groups:

1. Muscles regulating the position of the soft palate: ala nasi, tensor palatini, and levator palatine
2. Tongue: genioglossus, geniohyoid, hyoglossus, and styloglossus
3. Hyoid apparatus: hyoglossus, genioglossus, digastric, geniohyoid, and sternohyoid
4. Posterolateral pharyngeal walls: palatoglossus and pharyngeal constrictors

These groups of muscles interact in a complex fashion to determine the patency of the airway. Soft tissue structures form the walls of the UA and include the tonsils, soft palate, uvula, tongue, and lateral pharyngeal walls (Fig. 12-2).[29]

Taylor and colleagues[30] examined healthy children to elucidate the pattern of bony and soft tissue growth of the oropharynx. Lateral cephalograms of 32 untreated subjects belonging to the Bolton-Brush Growth Study were examined at 6, 9, 12, 15, and 18 years of age. Hard and soft tissue

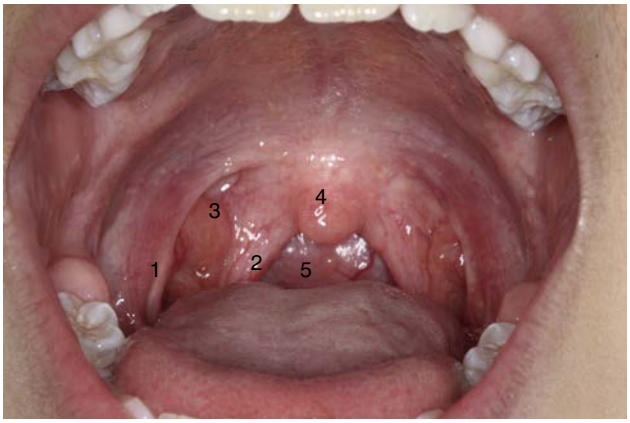


FIGURE 12-2 Intraoral picture of an 8-year-old child shows the palatoglossal fold **(1)**, palatopharyngeal fold **(2)**, palatine tonsils **(3)**, uvula **(4)**, and posterior pharyngeal wall **(5)**.

tracings were compiled at different ages to generate tracing templates. Pharyngeal soft tissues exhibited two periods of accelerated change (ages 6 to 9 years and 12 to 15 years) and two periods of quiescence (ages 9 to 12 years and 15 to 18 years). Beginning at age 9 years, the soft palate increased in both length and thickness. The increase in airway size that occurs from ages 6 to 9 years is thought to occur because of the continued growth of the pharyngeal region, the surgical removal of adenoid tissue, and the natural involution of adenoid tissue, either alone or in combination with one another. These three mechanisms are suspected to play a role in the increase of the airway during the 12- to 15-year-old period. Moreover, inferior regions of the airway located below the tonsils and adenoids also demonstrated these periods of growth and quiescence.

The primary craniofacial bony structures that determine the airway size are the mandible[31] and the hyoid bone[32]; these presumably act by providing the anchoring structures to which muscles and soft tissue attach. In normal, nonobese individuals, the mean minimum cross-sectional area across multiple segments of the UA has been measured using several techniques: estimates vary from 320 mm^2 (acoustic reflection),[24] 59 mm^2 (fast computed tomography [CT] at functional residual capacity [FRC]),[33] 64 mm^2 (magnetic resonance imaging [MRI]),[29] 144 mm,[2,34] 188 mm,[2,35] and 138 mm^2 (conventional CT).[36] This wide range of sizes reflects the differences attributable to individual variability but also to differing locations of measurement, positional change (sitting or supine), and differences imposed by the choice of imaging modality (e.g., mouth open is required for acoustic reflection). The minimum caliber of the UA in the wake state is primarily in the velopharynx,[37] which makes it a site of interest as the potential location of collapse during sleep. The anterior wall of the oropharynx primarily comprises the soft palate, tongue, and lingual tonsils, and the posterior wall is bounded by a muscular wall made up of the superior, middle, and inferior constrictor muscles that lie in front of the cervical spine. The lateral pharyngeal walls are a complex structure made up of muscles (hypoglossus, styloglossus, stylohyoid, stylopharyngeus, palatoglossus, palatopharyngeus, and pharyngeal constrictors), lymphoid tissue, and pharyngeal mucosa. Such complexity of interactions among these different muscles makes the oropharynx an extremely difficult structure to evaluate.

Hyoid Bone Position and Morphologic Features

The hyoid bone is strategic because, as a bone, it is more readily seen in radiographs, as compared with muscles. The hyoid bone is the only bone in the head and neck region without a bony articulation. It freely lies suspended by the same muscles that are integrally related to the pharyngeal region of the airway. As such, the hyoid bone is a useful indicator of how the soft tissue is functioning. Its muscle attachments can be described as belonging either to the suprahyoid group or the infrahyoid group. Arising from the cartilages of the second and third branchial arches, the hyoid bone consists of a central body that posteriorly projects as a left and right greater horn. Attaching to the superior surface of the hyoid bone are the left and right lesser horns.[38] Papadopoulos and colleagues[39] used the hyoid bones from 76 cadavers to demonstrate their lack of symmetry. Among their sample, almost 50% of hyoid bones were found to be asymmetric in the transverse dimension. Nearly 60% of the hyoid bones examined displayed some differences in the extension of the greater horns.

A combination of linear and angular measurements shows that the hyoid bone descends and moves slightly anteriorly up to age 18 years. Anterior movement of the hyoid bone is believed to be related to the forward translation of the mandible that occurs during cephalocaudal skeletal growth. The descent of the hyoid bone may be explained by the growth of the cervical vertebrae. The combined anterior and downward displacement results from the hyoid bone being suspended between the cervical vertebrae and the mandible.[30]

The position of the hyoid bone is determined by the combined activity of the suprahyoid and infrahyoid muscle groups, with the UA dilator muscles being of great importance.

Behlfelt and associates in 1990[40] concluded that age was positively correlated with linear measurements of the sagittal and vertical posture of the hyoid bone. Enlarged tonsils can impinge on the UA, leading to a restricted passage and smaller airway volumes. They observed that children with enlarged tonsils had a more caudally positioned hyoid bone with respect to the mandibular plane (hyoid to mandibular plane [H-MP]). When comparing the sagittal position of the hyoid bone, either in relation to the cervical spine or to the gnathion, no significant differences were found between subjects in the control group and those with enlarged tonsils. Cohen and colleagues[41] found that during growth and development, the tongue increases in size and becomes larger with respect to the intermaxillary space, especially in males. This disproportionate increase in tongue mass relative to the oral cavity may lead to the tongue moving downward as the individual grows. This descent of the tongue may play a role in the inferior and anterior movements of the hyoid bone that occurs as one ages.

Similar to Taylor's group,[30] Nelson and colleagues[42] found that the H-MP distance increased over time, irrespective of whether the person was a snorer or not. Their study demonstrated that snorers have a greater H-MP distance during the prepubertal and pubertal period ($p < 0.10$). Similarly, adult snorers displayed a significantly larger H-MP distance ($p < 0.05$), compared with nonsnoring control subjects. Hyoid bone position can potentially be influenced by anatomic variables, neuromuscular control, and even deposition of fat in the neck region.

Sforza and colleagues[43] hypothesized that excessive submental fat deposition in obese patients could be responsible for inferiorly moving the hyoid bone further. Upon examining lateral cephalograms, Sforza and others[43] showed that differences in the position of the hyoid bone and measurements of the posterior airway space exist between control subjects and snorers. They proposed that the pharyngeal critical (Pcrit) pressure, which represents the airway pressure above which airflow can pass through the UA, is a measure of pharyngeal collapsibility. Upon examining male patients with OSA, significant correlations were found between Pcrit and the distance from the hyoid bone to the posterior pharyngeal wall ($r = 0.29$, $p = 0.03$) and the vertical position of the hyoid bone ($r = 0.32$, $p = 0.02$). A lower hyoid position was theorized to predispose a subject to pharyngeal collapse by influencing the Pcrit pressure, thus contributing to an anatomic deficit in patients with OSA.

Verin and colleagues (2002)[44] demonstrated that segmental UA resistance was correlated with the posterior airway space and the distance from the H-MP and the distance from the hyoid bone to the posterior pharyngeal wall (H-Ph) ($p < 0.05$). Patients with OSA displayed a greater UA resistance, which was linked to various anatomic variables, especially the position of the hyoid bone. A more downward and anterior position of the hyoid bone was demonstrated in subjects with OSA, compared with both snorers and normal subjects. The H-MP distances averaged 12 ± 5 mm in the control group, 18 ± 3 mm in the snorers group, and 24 ± 7 mm in OSA group, showing a statistical significance at all levels. The anterior position of the hyoid bone was not significantly different between the control group and the snorers group (35 ± 5 mm and 39 ± 6 mm, respectively), but both were significantly smaller than in the OSA group (46 ± 1 mm).[44]

Relationship of Different Skeletal Patterns to Airway Morphologic Structure

Since mandibular retrognathia and vertical excess are often associated with airway problems, the question remains whether different sagittal skeletal patterns have different airway sizes. It has been suggested through various three-dimensional (3D) studies that airway morphologic structure varies among patients with different craniofacial characteristics,[45-50] but conflicting findings are also available.[51,52] The primary consideration that arises from this discrepancy is that there is still no methodologic consensus on these studies. The differences in such studies when evaluating airway are patient position (supine or upright), consistency among subjects' ages, and the variability of two-dimensional (2D) and 3D landmarks used to define the UA. Although it has been shown that the airway volume and characteristics change with age, some studies include both adolescents and adults within the same group. Some studies use the Angle classification for stratification of groups, but this stratification could be too general. In a study by El and Palomo, groups were further stratified to Class II maxillary protrusion and mandibular retrusion and Class III maxillary retrusion and mandibular protrusion subgroups.[53] It was observed that subjects with Class II mandibular retrusion had the lowest airway values. When the mandible was in the opposite sagittal relationship—that is, the Class III mandibular protrusion group—the highest oropharyngeal volume, nasal airway volume, and minimum axial cross-sectional areas were seen. The nasal volume was lower in the Class II mandibular retrusion group, compared with Class I subjects, which was also previously found by Kim and colleagues.[47] Although still somewhat controversial, it makes sense that different sagittal positions of the jaws would influence the airway space, especially considering that approved treatment approaches include the movement of the mandible forward (with surgery or through oral appliances [OAs]), and bimaxillary advancement surgery for treatment of OSA (Fig. 12-3).

As for the orientation, subjects with a Class III skeletal pattern present a more vertical orientation of the airway in the sagittal plane, compared with Class I and Class II subjects, whereas a Class II skeletal pattern is associated with a more forward orientation of the airway.[48] Changes in the overall volume, area, or linear measurements of the sagittal airway are not of greatest significance; rather, detecting the location presenting with the minimum axial cross section (i.e., area of maximum constriction) is most critical. This area of maximum constriction is responsible for disturbance in breathing periods and is most susceptible to negative pressure. Furthermore, a correlation between the minimum axial area of the oropharynx and oxygen saturation and quantity has been presented, as well as apnea episodes.[54] Although some results concerning airway morphologic structure and different skeletal patterns are contradictory, most of the studies agree that minimum axial cross-sectional areas present a high positive correlation with the volume,[45,51,55] which means that as the minimal area increases, volume is expected to increase as well. This concept has also been realized by the software developers of the widely used segmentation programs, and most of the commercially available programs on the market today are capable of automatically finding the minimum axial cross-sectional area within the region of interest.

Although most studies have tendencies to relate the airway to skeletal tissue, Solow and Kreiborg presented a "soft tissue stretching hypothesis," proposing that a change in jaw posture caused by mouth breathing could lead to stretching of the lips, cheeks, and musculature, resulting in upright incisors and narrower dental arches, as observed in patients with a long-face and open bite growth pattern.[56] Patients with a vertical growth pattern have shown a narrower airway, both anteroposteriorly and coronally, when compared with patients showing more horizontal growth (Fig. 12-4).[46,48] Most vertical growers may also have a skeletal anteroposterior (AP) malocclusion (Class II or Class III), and often a strong tongue indentation can be noted at the anterior wall of the airway.[48] Joseph and colleagues evaluated the airway of hyperdivergent and normodivergent facial types and found smaller dimensions in the hyperdivergent group, attributing this finding to the retrusion of the maxilla and mandible and a low-set hyoid position, suggesting a compensatory mechanism.[57]

The relationship between the AP position of the jaws and the airway is not completely understood. Not everybody with a retruded mandible will have airway issues, but the airways of those with retruded jaws and/or hyperdivergent growth may need special attention.

AIRWAY MEASUREMENTS AND IMAGING

Overview

The objective measurement of airway patency is an important part of airway assessment. The measurement of nasal cavity geometry has proven to be a great challenge for researchers in modern rhinology. A number of techniques have been used to study the airway, including nasal pharyngoscopy, cephalometric

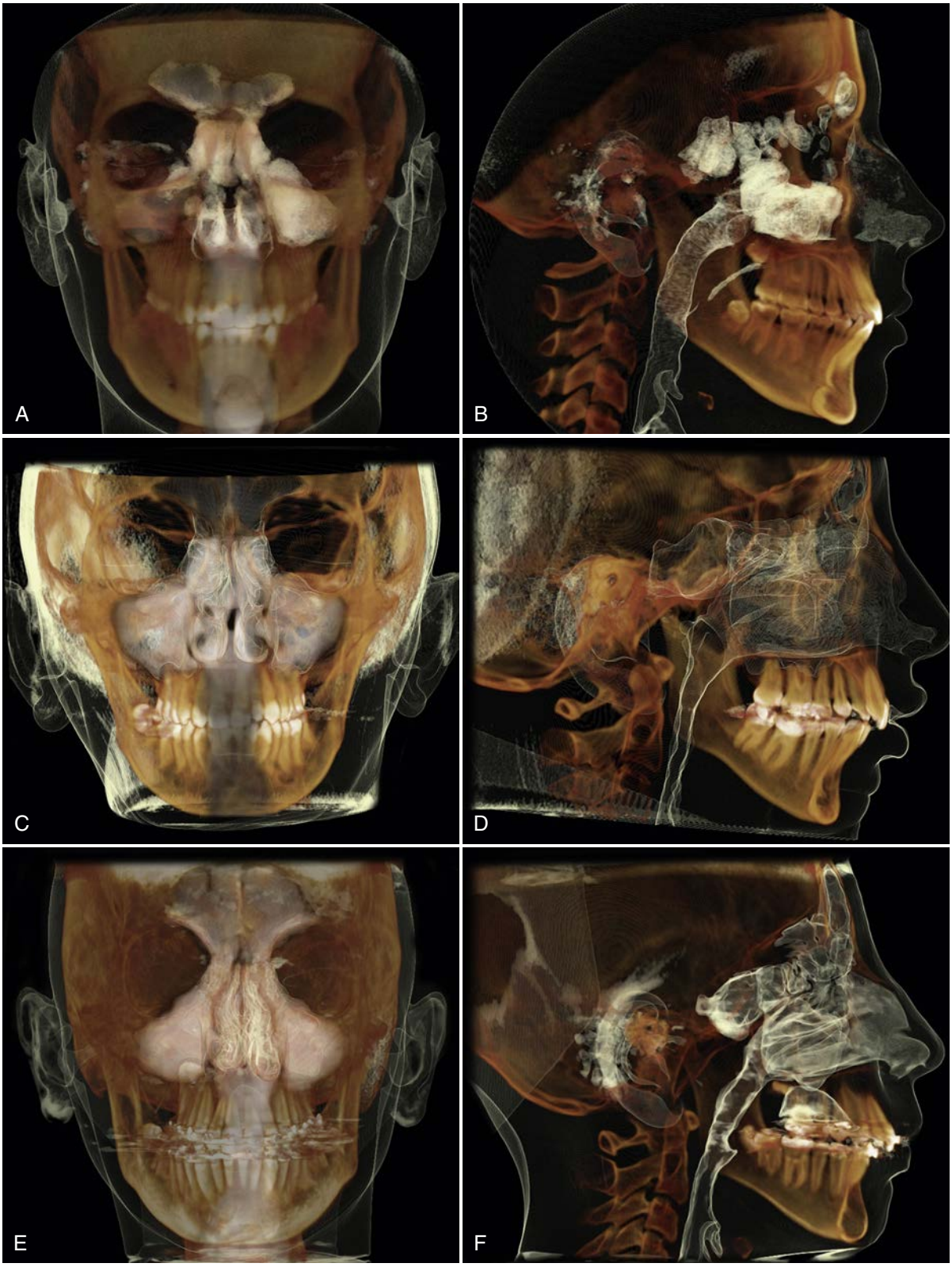


FIGURE 12-3 Airways from the sagittal and coronal views for different anteroposterior skeletal patterns are demonstrated. Class I **(A, B)**, Class II with mandibular retrognathia **(C, D)**, Class II with maxillary prognathia **(E, F)**, Class III with maxillary retrognathia **(G, H)**, and Class III with mandibular prognathia **(I, J)**.

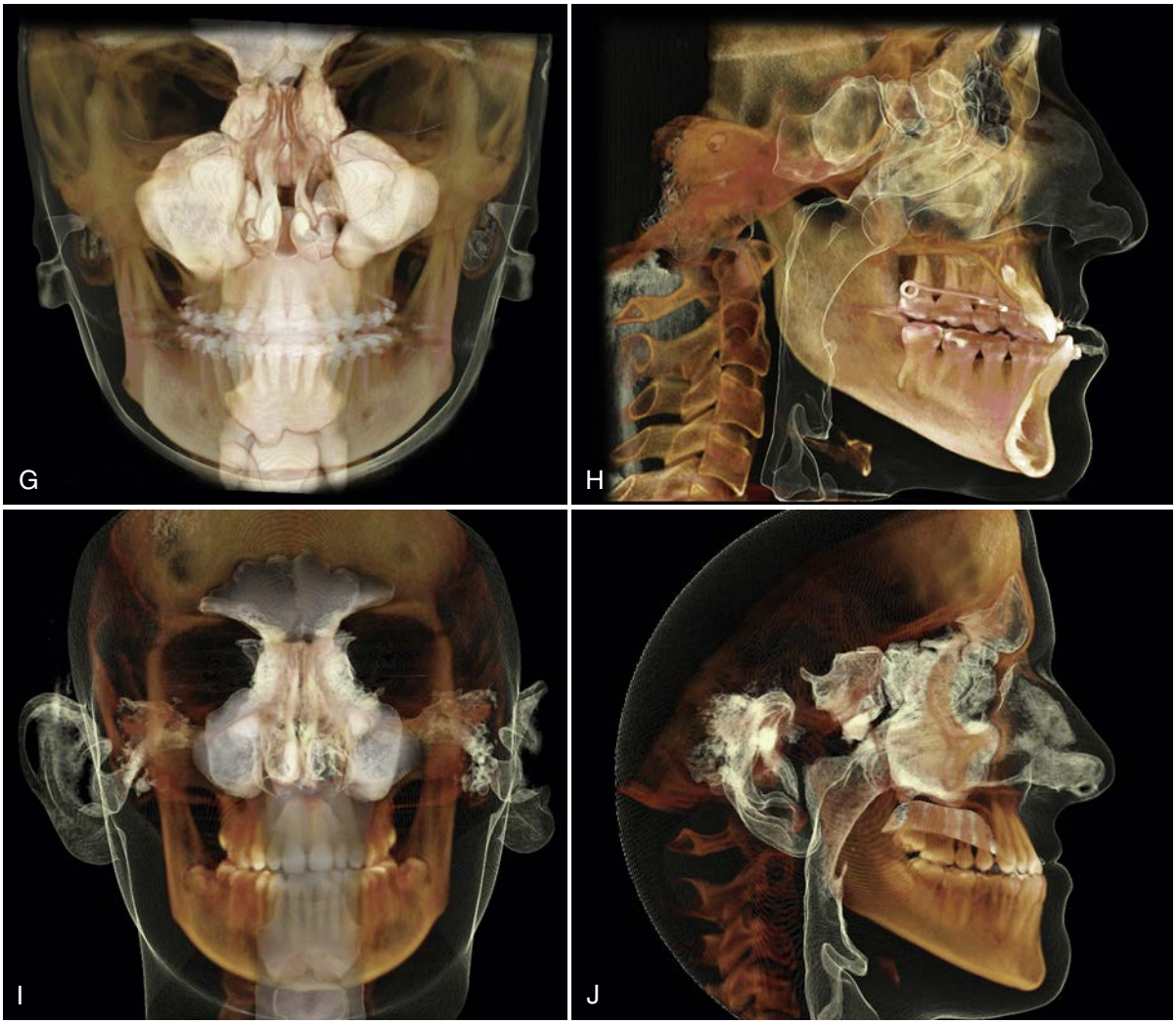


FIGURE 12-3, cont'd

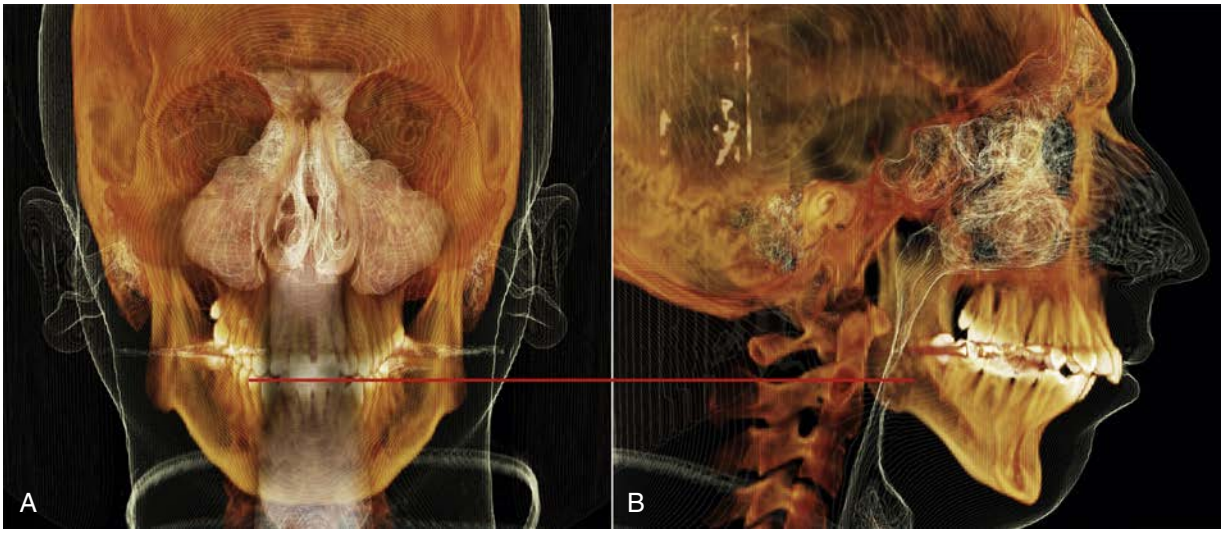


FIGURE 12-4 Three-dimensional image showing the cranial structure and airway of a patient with vertical growth pattern, in a) frontal, and b) lateral views. The red line compares the minimum axial cross sectional area. which appears wide on frontal view, but narrow on lateral view.

radiographs, fluoroscopy, conventional and electron-beam CT, acoustic reflection, and MRI.[58] Although MRI is considered to be very accurate in measuring the soft tissue lining, fat pad, and surrounding structures of the airway in three dimensions,[58] it is not as useful in orthodontics because of the use of metals, which interfere with this imaging modality. Patel and colleagues in 2006 reported that fixed metal orthodontic appliances can produce artifacts and obscure the area of the MRI.[59]

Probably the most common imaging modality found in an orthodontic office is the cephalogram, which provides a 2D radiographic view of the patient profile. Cephalograms have been found useful in identifying airway obstruction, adenoid hypertrophy, and very constricted airways.[60] However, the cephalogram is an image with incomplete information as it attempts to represent a 3D structure in 2D. Since many of the airway deficiencies and changes have been shown to occur in a mediolateral direction, the use of cephalograms for airway assessment is limited. (A classic cephalometric assessment of the airway is described later in this chapter.)

Some studies suggest that nasoendoscopy presently holds the position of gold standard diagnosis for UA obstruction.[60] However, nasoendoscopy has drawbacks as well; primarily, it allows little opportunity for objective measurement but relies, instead, on professional opinion, often causing low interobserver agreement.[61] Acoustic rhinometry (AR) or acoustic pharyngometry and cone-beam computed tomography (CBCT) are more widely used in otolaryngology and orthodontics, respectively. Current trends are transitioning to CBCT as it provides a 3D perspective and can be used to look precisely at hard tissue structures.

UA imaging techniques have also been used to visualize the airway lumen and to define the surrounding structures. In awake subjects, Schwab and colleagues[37] have shown that the normal UA has a longer lateral (coronal) than AP (sagittal) dimension using MRI techniques.

In addition, by using fast cardiovascular CT (cine CT), they also showed that airway size stays fairly constant during inspiration and reaches a minimum during end expiration, suggesting that muscular stabilization of the airway lumen during inspiration against the negative intraluminal pressure is more important than actual dilation, as had previously been believed. According to these authors, most respiratory-related changes (e.g., end-expiratory loss of diameter) are predominantly in the lateral dimension.[37]

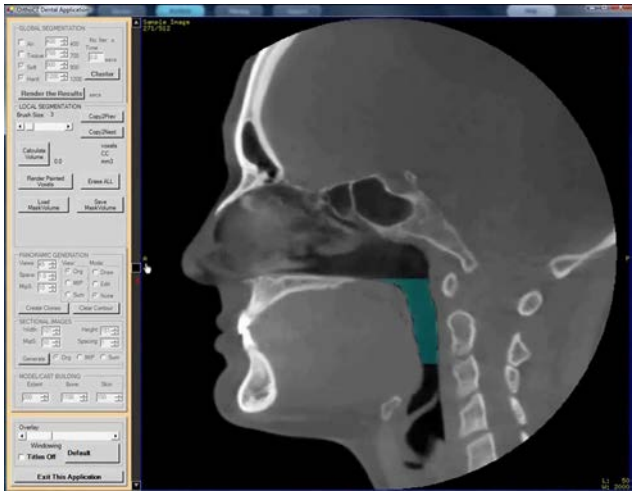
Cone-Beam Computed Tomography

CBCT is a medical imaging modality that has been applied in different fields of medicine (e.g., cardiac imaging, radiotherapy). CBCT is very popular in orthodontics and has brought 3D radiography to clinical orthodontics. The principle behind this technique is a cone-shaped x-ray bundle, with the x-ray source and detector (image intensifier or flat-panel detector) rotating around a point (or field) of interest of the patient. The conical shape of the beam distinguishes this technique from helical CT, which uses a fan-shaped beam. During a CBCT scan, the scanner (x-ray source and a rigidly coupled sensor) rotates, usually 360 degrees, around the head to obtain multiple images (ranging from approximately 150 to 599 unique radiographic views). These 2D images received by the detector are then compiled by the acquiring software into volumetric data, creating a 3D image (primary reconstruction). The scan time can range between 5 and 40 seconds, depending on the unit and settings used.

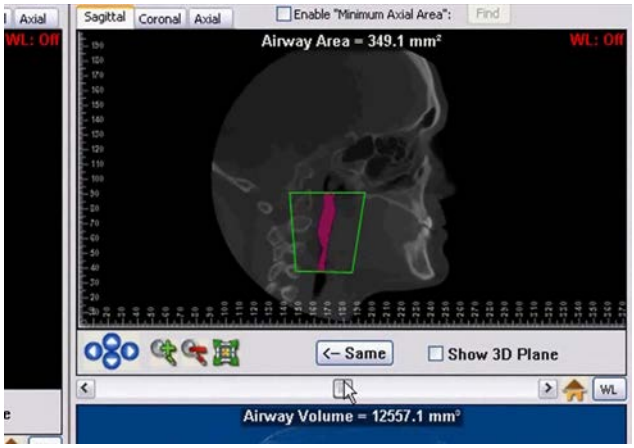
From its introduction, the use of CBCT has been criticized for its additional radiation exposure when compared with more traditional methods. The latest generation CBCT scanners are able to scan a patient with 180-degree rotation and with pulse technology, which uses radiation only when capturing the 2D images, resulting in approximately 2 seconds of total radiation time.[62] In addition, CBCT may now result in less radiation exposure to the patient than the usual combination of a panoramic radiograph and a lateral cephalogram. The combination of less radiation with equal or more diagnostic information may make CBCT more common in the near future.

Osorio and colleagues[63] suggested that CBCT has the potential to eventually emerge as a comprehensive and practical system to evaluate the airway and could become an excellent research and teaching tool for understanding the normal and abnormal airway. The axial plane, which is not visualized on a lateral cephalogram, is the most physiologically relevant plane because it is perpendicular to the airflow. The airway can be accurately assessed through segmentation. In medical imaging, segmentation is defined as the construction of 3D virtual surface models to match the volumetric data.[64] UA segmentation can be performed either manually or semiautomatically. In the manual approach, the user identifies the airway in each slice through the length of the airway (Video 12-1). The software then combines all slices to form a 3D volume. This method is time-consuming and almost impractical for clinical application. In contrast, semiautomatic segmentation of the airway is significantly faster.[65] In the semiautomatic approach, the computer automatically differentiates the air and the surrounding soft tissues by using the differences in density values (gray levels) of these structures. In some programs, the semiautomatic segmentation includes two user-guided interactive steps: (1) placement of initial seed regions in the axial, coronal, and sagittal slices, and (2) selection of an initial threshold (Videos 12-2 to 12-5). El and Palomo[65] showed that manual segmentation was more accurate than semiautomatic segmentation using different commercially available software; however, all of them showed high correlations, suggesting the existence of a systematic error in the derivation of the airway volume. Similar results were reported by Weissheimer and colleagues[66] using a more comprehensive list of software.

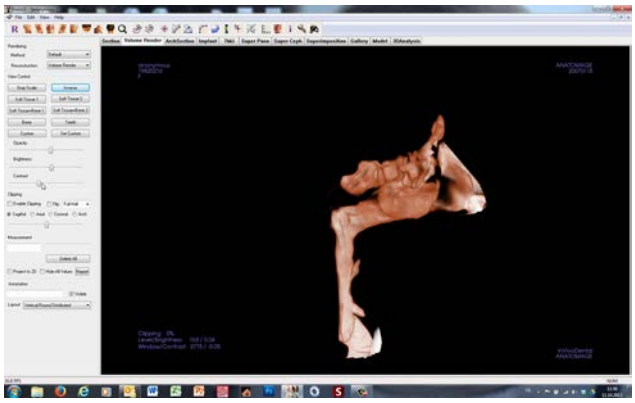
Proper analysis of the airway starts with the orientation of the 3D image, adjusting all three planes of space.[67] For a complete assessment of the airway, using a combination of 2D and 3D measurements is better. Calculating the volume is relatively easy and is probably the first measurement that comes to mind when studying the airway; however, the volume alone is not descriptive enough to describe all of the changes that may have occurred. An example to describe the shortcomings of using only the volume is to think of a birthday party air balloon. The same balloon, tied up with a fixed volume of air inside, can be deformed and have its shape changed without altering the air volume inside. When comparing air volumes, the result would suggest that no deformation took place (Fig. 12-5). Using anatomic limits when measuring the airway is very important to ensure that the same segmented volume is being compared among patients and groups. Unfortunately, no consensus has been reached at this point on which planes or landmarks to use as anatomic limits. The minimum axial area, also known as the *area of maximum constriction* (mm^2), is probably more useful



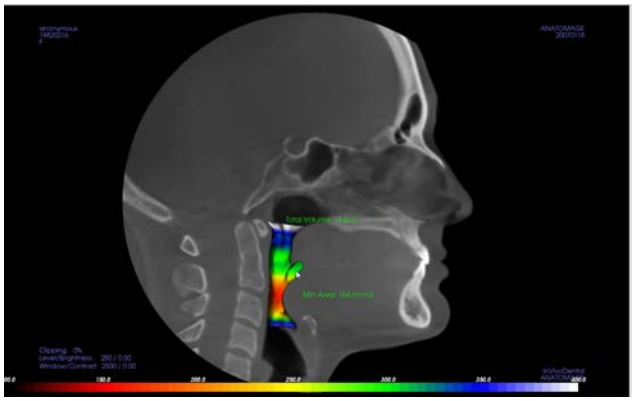
VIDEO 12-1 Manual segmentation of the airway. When performing a manual segmentation of the airway, the user identifies the airway in each slice through the length of the airway. This is a labor-intensive procedure that gives the operator total segmentation control.



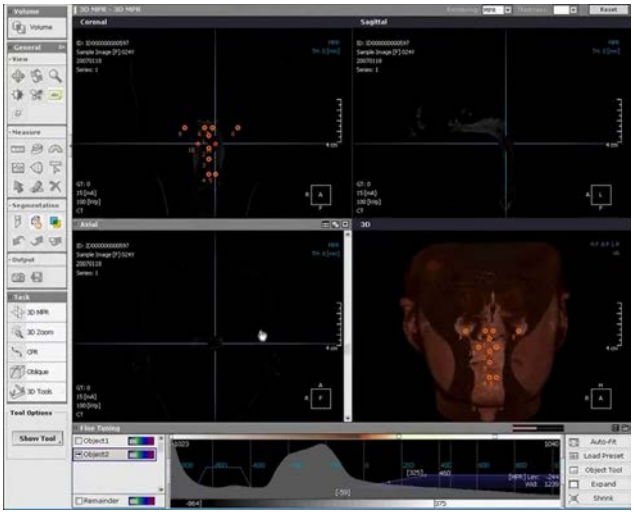
VIDEO 12-2 Airway segmentation using Dolphin 3D v11. (Used with permission from Dolphin Imaging & Management Solutions, Chatsworth, CA). Semiautomatic segmentation of the airway using Dolphin 3D. The software was a pioneer in user-friendly and fast airway segmentation.



VIDEO 12-3 Airway segmentation using InVivo Dental v4.Semiautomatic segmentation of the airway using InVivo Dental 4. In this older version, the segmentation was more manual than automatic. (Used with permission from Anatomage Inc. San Jose, CA.)



VIDEO 12-4 Airway segmentation using InVivo Dental v5.1. (Used with permission from Anatomage Inc., San Jose, CA). Semiautomatic segmentation of the airway using InVivo Dental 5.1. Software updates made segmentation faster and more user-friendly.



VIDEO 12-5 Airway segmentation using OnDemand 3D v1.0. (Used with permission from Cybermed Inc., Seoul, South Korea). Semiautomatic segmentation of the airway using OnDemand 3D. This older version uses a combination of seed points and extensive manual sculpting.

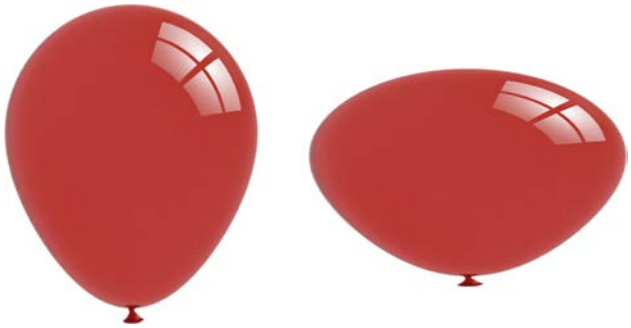


FIGURE 12-5 The same party balloon, before and after distortion, is illustrated. The air volume remains constant; consequently, volumetric analysis, on its own, is not able to describe all the events that may have happened.

than the airway volume. Figure 12-6 shows the most commonly used 2D and 3D measurements of the UA.

Acoustic Rhinometry

AR was introduced by Hilberg and associates in 1985[68] as an objective method for examining the nasal cavity (Fig. 12-7). This technique is based on the principle that a sound pulse propagating in the nasal cavity is reflected by local changes in acoustic impedance. AR is a simple, fast (approximately 30 seconds), and noninvasive technique that became widely accepted in a short period. Most previous investigations of living human subjects have demonstrated reasonably good agreement between the cross-sectional areas in the anterior part of the nasal cavity determined by AR and those determined by imaging techniques such as MRI and CT. However, this does not hold true for the posterior part of the nasal cavity and the epipharynx, in which AR significantly

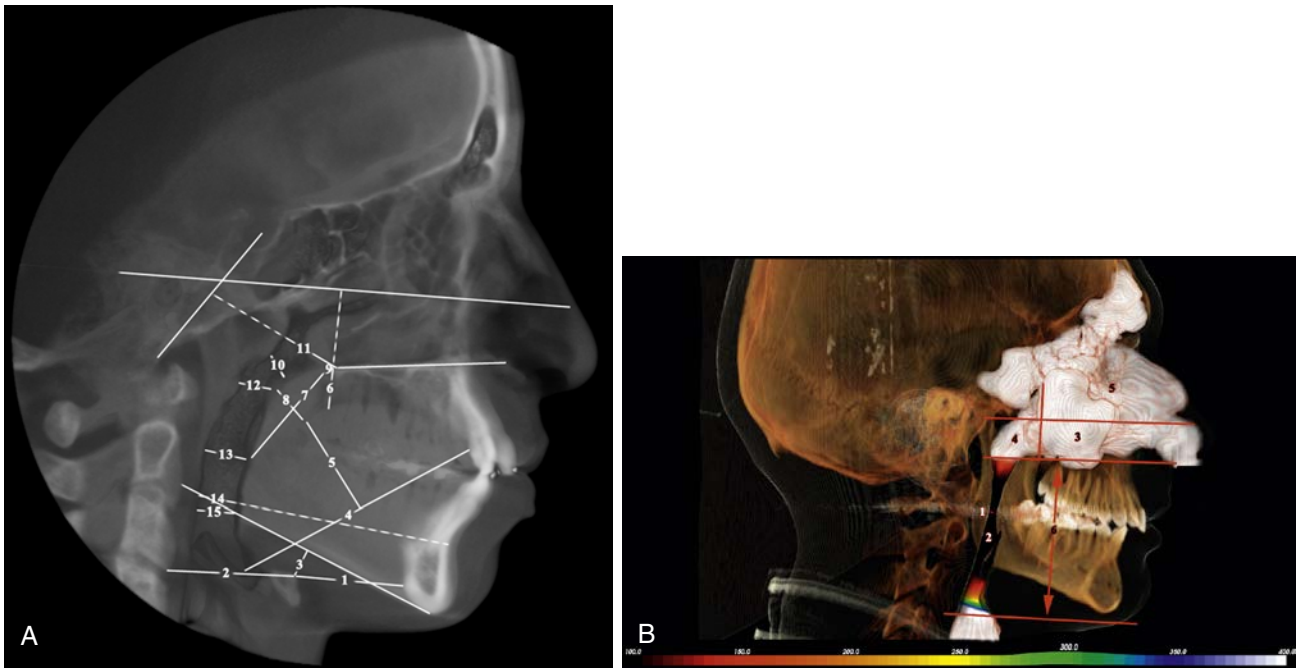


FIGURE 12-6 Common measurement made for airway analysis. **A,** Two-dimensional image: *(1)* line between the most posterior point of symphysis and the most superior and anterior points on the body of the hyoid bone; *(2)* line between the most anteroinferior part of the third cervical vertebra and the most superior and anterior points on the body of the hyoid bone; *(3)* perpendicular distance from the most superior and anterior points on the body of the hyoid bone to the mandibular plane; *(4)* tongue length: distance between the base of the epiglottis to the tip of the tongue; *(5)* tongue height: maximum height of the tongue along a perpendicular line of tongue length line to the tongue dorsum; *(6)* vertical distance between the dorsum of the tongue and posterior nasal spine (PNS) on a perpendicular line to the Frankfort horizontal (FH) plane; *(7)* soft palate length: distance between the PNS point and the tip of soft palate; *(8)* soft palate thickness: maximum thickness of the soft palate measured on a line perpendicular to the PNS and tip of soft palate line; *(9)* soft palate angle: angle that forms between the distance between PNS point and the tip of soft palate and the posterior nasal spine–anterior nasal spine (ANS-PNS) line; *(10)* shortest distance between soft palate and the adenoid tissue; *(11)* distance between AD2 point (intersection of the posterior nasopharyngeal wall and the PNS and the midpoint between the sella-basion [S-Ba] line) and PNS; *(12)* width of the most constricted airway space behind the soft palate along a parallel line to the Gonion-Point B [Go-B] line; *(13)* width of the airway along a parallel line to the Go-B line through the soft palate tip; *(14)* width of the airway space along the Go-B line; *(15)* posterior airway space: shortest distance between the radix of the tongue and the posterior wall of the pharynx. **B,** Three-dimensional image: *(1)* minimum axial cross-sectional area; *(2)* oropharynx volume: superior limit is defined by the ANS-PNS line, inferior limit is the line passing from the most anteroinferior part of the third cervical vertebrae and parallel to the ANS-PNS line; *(3)* nasal passage volume: inferior limit is defined by the ANS-PNS line, superior limit is the last axial slice before the nasal septum fuses with the posterior wall of the pharynx; *(4)* nasopharynx volume: located in the superior part of the ANS-PNS line and posterior of the line that passes from the PNS and is perpendicular to the ANS-PNS line until the most superior part of the nasopharynx; *(5)* nasal cavity: inferior limit is the ANS-PNS line, posterior limit is the line that is perpendicular to ANS-PNS line and passes through the PNS point; and *(6)* vertical oropharyngeal length: distance between the ANS-PNS line and the line passing from the most anteroinferior part of the third cervical vertebrae and parallel to the ANS-PNS line.

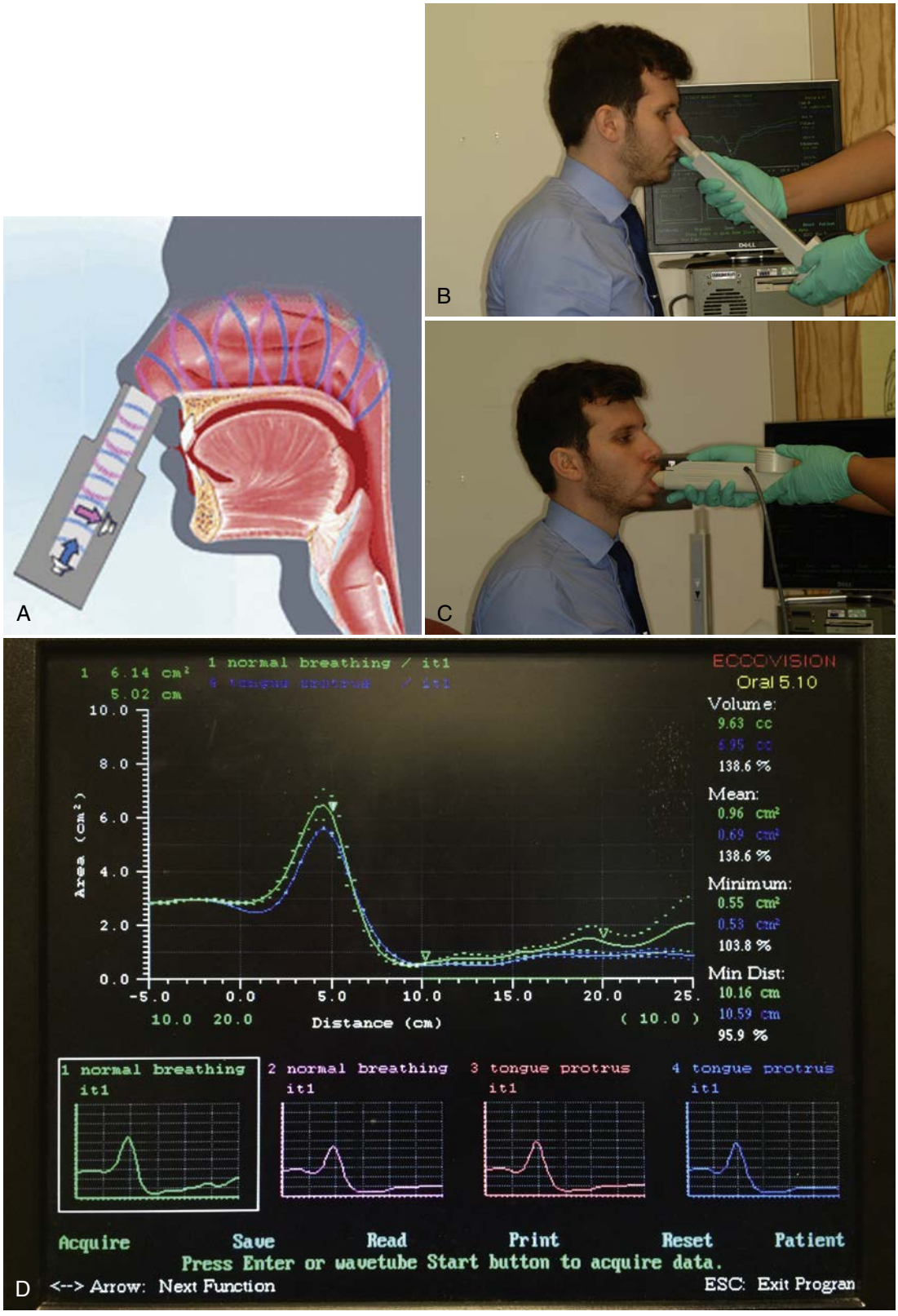


FIGURE 12-7 A, Drawing illustrates how rhinometry measures the airway through sound waves. Capturing rhinometry (B), pharyngometry (C), and data collected through pharyngometry (D). The *x* axis indicates the distance travelled by the sound, and the *y* axis indicates the airway area at that corresponding length. (Courtesy of Dr. Sorapan Smuthkochorn and Divya Venkat.)

overestimates cross-sectional areas compared with MRI and CT scans. So far, few attempts have been made to validate AR by comparison with other methods, including nasal casting, fluid-displacement, and MRI. Kaise and colleagues[69] tested the AR method using small experimental animals, comparing the readings with impression material instilled postmortem into the nasal cavity of the animals and then measured through fluid displacement. The results deemed RA useful and reliable, estimating 73.7 ± 20.0% of the actual volume. Cakmak and colleagues[70] conducted a clinical study of 25 healthy adults that focused on the nasal valve region in particular, also reporting AR as a valuable method for measuring the passage area of the nasal valve. Numminen and associates[71] compared RA to high-resolution CT volumetry, considered one of the best imaging modalities available for evaluating the nasal cavity and paranasal sinus geometry, showing 1% error in segmented volumes when compared with actual volumes. Numminen concluded that AR is clinically useful and shows very good reliability in the anterior and middle parts of the nasal cavities but decreasing accuracy in the posterior part.

Pharyngometry

The acoustic reflection technique may also assess the pharyngeal cross-sectional area. This technique has been previously applied to study the pharynx, glottis, and trachea in humans in vivo. The technique has been validated against CT scans and experimental models.[72] Unlike the nose, the oropharyngeal airway is geometrically more complex and variable and includes mobile structures (soft palate and tongue); therefore establishing a standard operating protocol and an understanding of the possible sources of artifacts is of great importance in obtaining reliable results. Pharyngometry provides a noninvasive assessment of the dimensions, structure, and physiologic behavior of the UA from the oral cavity to the hypopharnyx while the patient breathes. Computer processing of the incident and reflected sound waves from the airways provide an area distance curve that represents the lumen from which minimal cross-sectional area and volume can be derived. This dynamic test measures the dimensions of the airway through the oral cavity and 25 cm down the pharynx. Marshall and associates[73] compared acoustic measurements of the pharyngeal and glottal areas in human volunteers during free breathing with MRI measurements of the same areas and showed no statistically significant differences between the measurements taken using either technique. Of equal importance is testing the repeatability of measurements obtained to ensure the validity of both the technique and the results. In 2004, Kamal[74] showed that measurements of the pharyngeal cross-sectional area in a different session on the same day did not significantly differ from those obtained in different days. Provided that a standard operating protocol is adopted and maintained, repeatability of acoustic reflection results can be achieved. Hatzakis and colleagues,[75] on the other hand, when examining 40 patients between the ages of 3 and 9 years before and after an adenoidectomy, suggested that pharyngometry was not reliable in assessing pharyngeal volumes in a pediatric population. They concluded that the results given by this method were not consistent with their clinical examination and hypothesis of what happened after the surgical procedure.

In 2007, Gelardi and colleagues[76] assessed variations of pharyngometric parameters in patients with sleep disorders and established a correlation between volumetric variations of oro-pharyngo-laryngeal spaces and the presence and severity of disease. A total of 110 patients, 70 with sleep disorders and 40 healthy subjects, had acoustic pharyngometry to evaluate the mouth and hypopharynx. A significant difference in parameters was observed between patients with sleep disorders and those in the control group, especially in patients with macroglossia. The authors concluded that although not a standardized test, acoustic pharyngometry was shown to be a useful method to assess OSA and in postoperative monitoring of UA surgery in patients with sleep disorders. When attempting to maintain good reliability and obtain accurate results, posture may play an important role in determining the pharyngeal area. Flexion of the neck and back, as well as raising the shoulders (which occurs near residual volume), may compress the pharynx and decrease its cross-sectional area.[77]

Pharyngometry is often marketed as a screening method to assess quickly a patient for potential sites of sleep-related UA obstruction and to better determine whether an OA or continuous positive airway pressure (CPAP) device may be appropriate for the patient. Proper sleep-related diagnosis is accomplished through polysomnography, which is discussed later in this chapter.

INFLUENCE OF ORTHODONTIC TREATMENT ON THE AIRWAY

Since orthodontists work around the UA and the changes made by orthodontic treatment are spatially related to the airway, several studies elucidate the relationship.

Treatment Including Extractions

Since the 1911 paper written by Calvin S. Case started the debate that came to be known as the "Great Extraction Debate," extraction treatment is one of the most discussed subjects of orthodontics.[78] For today's orthodontic practice, extraction treatment is a necessity for a not-to-be-underestimated number of patients. It is well documented in the literature that after four premolar extractions using maximum anchorage mechanics, a change in the soft tissue profile, retraction and uprighting of upper and lower incisors, and a slight change in the mandibular plane may be observed.[79-82] On the other hand, minimum anchorage mechanics may sometimes be desired for patients having good facial balance and moderate crowding, as well as when a counterclockwise rotation of the mandible is anticipated. In such cases, mesial molar movement is desired after the resolution of anterior crowding. Closure of extraction sites with mesial movement carries the molar to a narrower part of the arch, which could potentially have an effect on the tongue position.[83] Therefore investigating the effects of extraction treatment on the incisor, molar, and soft tissue position would be worthwhile, as this could potentially affect tongue position and may cause an alteration in the UA anatomy, especially the oropharynx.

Tongue position is considered to be an important factor for the UA since the root and posterior part of the tongue form the anterior wall of the oropharynx. Existing evidence suggests that extraction treatment with maximum anchorage mechanics may cause the tongue's length and height to decrease slightly and move to a more retracted position against the soft palate.[83,84] This movement results in an adaptation and may lead to the narrowing of the UA. However, because one study was a 2D study[83] and the other lacked a control group,[84] reaching definitive judgments is difficult. Valiathan and colleagues assessed 40 subjects (20 with extractions and 20 age- and gender-matched controls without extractions) and reached a conclusion that

extractions do not affect the oropharyngeal dimensions.[85] Stefanovic and associates who analyzed the pharyngeal airway of 62 subjects (31 subjects with extractions and 31 age-, gender-, and treatment duration–matched control subjects) also concluded that extraction of four premolars does not affect the pharyngeal airway volume or the minimum axial cross-sectional area.[86] The latter two studies were performed in university settings using different samples and subjects treated to the supervising faculty's personal treatment philosophy. Therefore interpreting the findings is also difficult because of the lack of data about the anchorage mechanics used. As previously discussed, a minimum axial cross-sectional area is important for the patency of the airway. It has been shown by Valiathan and colleagues that when the anterior teeth are retracted to a new position, predicting how the minimum axial cross-sectional area will respond to this movement and how respiratory function will be affected is impossible.[85]

Another possible explanation for UA reduction after incisor retraction is the movement of the hyoid bone in a posterior and inferior direction.[87] Wang and colleagues reported that this change in hyoid bone position was an adaptation that prevents an encroachment of the tongue into the pharyngeal airway. Shannon, in contrast, evaluated the 3D changes in the hyoid position in extraction and nonextraction subjects and concluded that the hyoid position had no significant change attributable to extractions.[88] Therefore the impact of backward and downward movement of the hyoid on UA dimensions remains controversial.[89]

On the other hand, mesial movement of the molars in to extraction spaces seems to enlarge the space behind the tongue, which is considered to play a vigorous role in improving UA dimensions.[83] Minimum anchorage and vertical control mechanics are also preferred in vertically growing patients to obtain a counterclockwise rotation of the mandible.[90] It has been reported that hyperdivergent patients with an obtuse mandibular plane angle have a narrower AP pharyngeal dimension, compared with normodivergent patients.[47,91,92] Therefore obtaining a counterclockwise rotation and a forward positioning of the mandible in such situations may contribute to enhanced dimensional changes in the UA.

To date, conclusive data on the effect of orthodontic extraction on the dimensional changes of the UA do not exist. Even if the oropharynx narrows, due to extractions, the assumption can be made that extractions may still be beneficial from the standpoint of addressing the malocclusion. To be more specific, whether extractions have an effect over the UA, actual functional assessment of breathing patterns must be evaluated in further studies, and higher quality trials are necessary to verify reliability.[89] Furthermore, no adverse effects of extraction treatment over the nasopharynx area have been reported to date.

Rapid Maxillary Expansion

Nasal resistance to airflow is an important factor in determining the nasal breathing pattern. Several methods such as intranasal and nasopharyngeal surgery have been recommended to increase airflow through the nose.[93] Rapid maxillary expansion (RME) is commonly used to correct maxillary constriction.[94,95] Because of the nature of the procedure, an increase in the nasal cavity width and posterior nasal airway is anticipated, not only attributable to the opening of the median palatal suture[96] but also to an increase in the sagittal and vertical dimensions.[97,98] As a result, an improvement in nasal respiration is expected, along with expansion in patients with a transverse arch discrepancy. Surgically assisted rapid palatal expansion (SARPE), on the other hand, is a frequently used surgical modality of RME preferred in skeletally mature individuals to overcome the resistance of the closed sutures.[99] Studies have documented nasal resistance reduction and intranasal capacity increase with both RME and SARPE treatments.[100,101] When speaking of the success for all treatment modalities, stability is a primary concern. The same holds true for the gain in nasal airway dimensions after RME treatment. In a systematic review by Baratieri and colleagues, changes after RME in growing children have been shown to improve the conditions for nasal breathing, and the results can be expected to be stable for at least 11 months after therapy.[96] El and Palomo reported a twofold increase in the nasal passage volume of patients who underwent RME treatment, compared with controls even at the end of approximately a 2-year treatment.[102]

Lateral and posteroanterior cephalometric records were used earlier for the evaluation of nasal airway. Although these methods proved to be useful in determining the contractions along the airway, they did not provide adequate information on measuring nasal resistance, airflow, minimum axial cross-sectional areas, or volumetric data. Rhinomanometry and AR have proven to be useful methods in providing an objective assessment of the air flow through the nose and to measure nasal cavity dimensions, respectively.[68,103] Furthermore, AR, CT, and MRI measurements show good correlation, especially for the anterior 6 cm of the nasal cavity area, which is most favorably affected by REM.[100] However, due to sound loss in the paranasal sinuses, the same probably does not hold true for the posterior nasal cavity and nasopharynx.[104]

As for the oropharynx area, using CBCT data in growing patients with a unilateral or bilateral posterior crossbite have demonstrated that oropharyngeal airway volume is significantly smaller, compared with patients without constriction.[105] After RME, the increase in intermolar width is a fact, especially in the maxillary arch, which may cause the tongue to reposition more anteriorly in the oral cavity.[106] Additionally, secondary to RME treatment, mandibular position also changes in various directions in patients with different malocclusions.[107] Therefore thinking that these outcomes may most likely cause dimensional changes for the oropharynx is not erroneous. However, the latest studies failed to confirm an enlargement or narrowing of the oropharyngeal volume, either in adults or in adolescents, despite the increase in intermolar width and mandibular positional changes.[102,105,108]

Functional Orthopedic Appliances

The position of the mandible, relative to the anterior cranial base and mandibular length, seems to have an impact over the oropharyngeal airway. Several studies have shown a significant but weak negative correlation between oropharyngeal dimensions and the skeletal configuration according to the A point–nasion–B point (ANB) angle.[45,49,50,109] In addition, mandibular corpus length and oropharyngeal airway volume, along with minimum axial area, have shown a positive correlation.[45,109] Thus thinking that functional appliances that advance the mandible could have a positive impact over the UA is logical, and an increase in the oropharyngeal airway dimensions has been previously reported.[110-112] When the mandible is protruded, a different posture of the tongue caused by increased genioglossus

muscle activity and/or other soft tissue activity may play an important role over airway dimensions.[113] A study by Hänggi and associates, using activator-headgear therapy in patients with a mean age of 10.2 years, showed improved distance behind the tongue (velopharynx) by 2.5 mm on average, resulting in an increase of the oropharyngeal dimensions.[114] Children with large adenoids and tonsils can show growth impairment attributable to abnormal nocturnal growth hormone secretion, pointing out the importance of proper clinical examination by the orthodontist before any treatment. In such cases, adenotonsillectomy may be needed to obtain a significant increase in serum levels of growth hormone mediators and potentially to help bring the somatic growth to normal levels.[115,116]

Iwasaki and colleagues found that a frequently used fixed functional appliance, the Herbst appliance (American Orthodontics, Sheboygan, WI), enlarges the oropharyngeal and laryngopharyngeal airways of Class II subjects at the prepubertal growth spurt stage, compared with an age-, sex-, and Frankfort mandibular angle (FMA)-matched skeletal Class I subjects.[117] One of the most interesting findings in such studies is that the majority of the airway enlargement occurs in the mediolateral dimension—in other words, in the width of the airway.[118-120] This effect cannot be seen in a lateral cephalogram, and identifying it in a posteroanterior film is difficult because of the superimposing anatomic structures. On the other hand, when fixed functional appliances are used in the later stages of growth, when most dental changes take place, no significant posterior airway changes are usually seen after treatment is completed.[121]

Other extensively used orthopedic appliances are headgears, to inhibit the forward maxillary growth, and the face mask for maxillary protraction. Kirjavainen and Kirjavainen found that cervical headgear treatment increased the velopharyngeal airway space but did not significantly affect the rest of the oropharynx or hypopharynx.[122] Although headgear treatment is intended to restrict the forward growth of the maxilla, which may suggest a negative influence over the airway, they speculated that this restriction was only limited to the maxillary alveolar process. Headgear is extensively used by patients during sleep.

The literature shows that the protraction face mask with or without RME can produce favorable skeletal and dental changes for patients with maxillary retrusion. The use of a face mask at an early age, along with an RME appliance, is reported to help obtain greater stability and skeletal effects.[123] However, neither type of treatment protocol seems to create a significant change for the oropharyngeal or nasopharyngeal sagittal airway dimensions when compared with subjects with untreated Class III malocclusions.[124] The literature also emphasizes that sagittal airway dimensions induced by therapy or physiologic growth show great interindividual variability in Class III subjects.

Orthognathic Surgery

Recent advances in surgical techniques, 3D imaging techniques, and surgical simulation programs offer a new perspective on treatment planning where face, airway, and bite are linked.[125] Today, planning and obtaining sagittal maxillary and mandibular movements and/or rotations in three planes of space are possible. The most common surgical procedures can be roughly categorized as concerning the mandibular or maxillary region only and bimaxillary surgical procedures. When the UA is in question, an important aspect of orthognathic surgery is how the skeletal movements and changes will affect the position of the hyoid bone and the tongue. A consensus in the literature suggests that when mandibular setback osteotomy is performed, the hyoid bone tends to move to a more posterior and inferior position, and the tongue is carried to a more posterior position, regardless of whether using bilateral intraoral vertical ramus osteotomy or sagittal split ramus osteotomy.[126-128] As a result, narrowing in the width and depth of the hypopharyngeal and oropharyngeal areas has been reported. However, there seems to be an adaptation of the airway in the oropharyngeal and hypopharyngeal levels after surgery. However, 1-year follow-up shows that the airway is still narrower, compared with its preoperative dimensions.[127] On the contrary, mandibular advancement surgery results in an increase in the dimensions of the oropharyngeal airway.[129-131] Maxillary advancement, on the other hand, creates a significant increase in the nasopharyngeal and oropharyngeal airway dimensions. It has also been reported that hypopharyngeal airway may as well present an enlargement after maxillary advancement.[132] Therefore performing bimaxillary orthognathic surgery rather than only mandibular setback surgery would be advisable, even if the patient exhibits mandibular prognathia. Recent studies also confirm that maxillary advancement, combined with mandibular setback surgery, compensates for the narrowing of the UA, and, as a consequence, sleep quality and efficiency improve.[133-135] Probably the highest gain in the UA is obtained with maxillomandibular advancement surgery, which is a frequently used surgical modality in the treatment of patients with OSA.[136] Additionally, when maxillomandibular advancement surgery is performed in conjunction with genial tubercle advancement, which pulls the geniohyoid and genioglossus muscles forward, the gain in the UA is even better.[137,138]

Craniofacial anomalies involving the midface (Crouzon, Apert, and Pfeiffer syndromes), the ones primarily involving the mandible (Nager and Stickler syndromes and Pierre Robin sequence), and those affecting the midface along with the mandible (Treacher Collins syndrome and hemifacial microsomia) can lead to a decrease in the size of the oropharyngeal and nasopharyngeal airways. In these disorders, the reduced size of the mandible and its retruded position cause retrodisplacement of the tongue and concomitant reduction of the oropharyngeal airway, which may lead to UA obstruction. Distraction osteogenesis (DO) has become an accepted method of treatment for patients requiring reconstruction of a hypoplastic mandible and a severely retruded maxilla to increase airway dimensions.[139,140] DO is debated as a promising surgical technique and an alternative to tracheotomy for long-term management of the airway.[141-143]

Different types of distraction devices are used for the treatment of craniofacial anomalies. These are primarily classified as external and internal distraction devices, and they have been extensively used for the maxillary and mandibular regions according to the patients' needs. It has been shown on CT studies that especially the airway region above the uvula and posterior nasal spine level of young patients with severe midface retrusion significantly improve with LeFort III osteotomy and DO.[140] In addition, a slight difference seems to exist between internal and external distractors used for maxillary distraction in terms of airway enlargement. External distractors, although bulky and having a negative impact on a patient's psychosocial life, appear to provide more extended bone osteogenesis advancement when compared with internal devices.[144-146] Therefore a greater gain is obtained in the UA (Fig. 12-8).

FIGURE 12-8 Figure shows the airway changes in a patient with Crouzon syndrome and treated with rigid external distractor (RED). A and B show frontal and lateral pre-treatment views, while C and D show the equivalent post treatment views. (Courtesy of Dr. Muge Aksu.)

Similarly, mandibular DO has been proposed as a useful method to resolve oropharynx airway obstruction.[147] This effect is primarily due to the displacement of the hyoid bone away from the posterior pharyngeal wall.[148] Furthermore, the small size of the mandible and its retruded position causes a corresponding retrodisplacement of the tongue, which also contributes to a reduction in the airway. Mandibular DO also creates a change in the position of the tongue and is believed to aid in increasing the airway (Fig. 12-9).[149]

Summary of Orthodontic Treatment Effects on the Airway

Studies show that certain orthodontic treatments may impact the UA, but there are limitations on this impact. Therefore some final recommendations in light of current literature can be as follows:

- Extraction treatment does not seem to affect the airway's size, but caution may be taken in patients who have respiratory problems or already constricted airways, possibly avoiding maximum anchorage approaches, if possible.
- RME may be able to help solve the nasal resistance to airflow if the problem originates from the anterior nasal cavity. Therefore in a possible relationship with an ear, nose, and throat (ENT) specialist, the clinician must be aware of the limitations of the procedure.
- Functional appliances are most useful in patients with a horizontal growth pattern of the mandible. If so, using fixed or removable appliances in a timely fashion may increase the dimensions of the airway. On the contrary, vertical-growing patients may not benefit from such a treatment since it is not the sagittal correction but rather a counterclockwise rotation that may increase the airway space.
- When planning surgical treatments, consideration should be given to avoiding large amounts of mandibular setback even if the patient's diagnostic records indicate mandibular prognathia. Bimaxillary surgeries are probably better choices for such patients.

Incorporating the morphologic airway into the orthodontic treatment plan is important. Orthodontics is not only about crooked teeth or jaw discrepancies. Airway patency is more important for patients than aligning teeth.

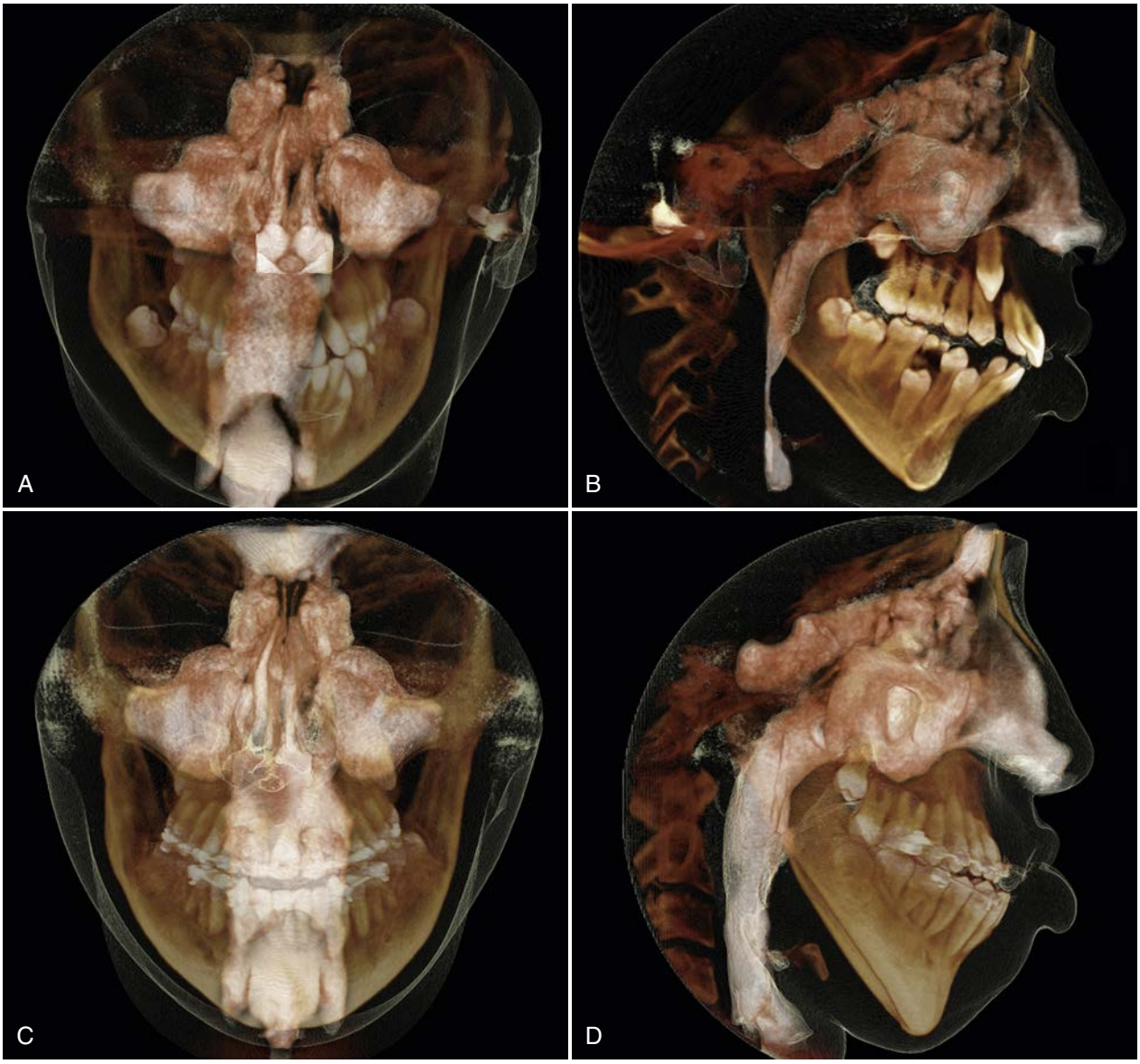


FIGURE 12-9 Figure showing the airway changes in a patient treated with mandibular distractor. A and B show frontal and lateral pre-treatment views, while C and D show the equivalent post mandibular distractor views.

SLEEP-DISORDERED BREATHING: AIRWAY DISORDERS AND MANAGEMENT

Sleep plays a vital role in good health and well-being throughout life. Getting enough quality sleep can help protect mental health, physical health, quality of life, and safety. Inadequate sleep contributes to heart disease, diabetes, depression, falls, accidents, impaired cognition, and a poor quality of life. In children and teenagers, sleep also supports growth and development.

OSA, which the orthodontist will most frequently encounter, is considered part of a group of disorders called *sleep-disordered breathing* (SDB). This class of disorders refers to abnormal respiratory patterning during sleep; but, ironically its presence or a suspicion of disease is made when the patient is awake. A finding of a narrow airway or a report of heavy snoring results only in a pre-test probability for any one of a number of respiratory pattern abnormalities, all which produce decreases in oxygen and increases in carbon dioxide levels, and arousals during sleep. Sleepiness by itself is not specific for SDB.

OSA is estimated to affect approximately 8% of men and 2% of women, averaging 5% of the general population, with many affected individuals going undiagnosed, considering themselves as healthy individuals.[150,151] The orthodontist who treats many patients a day probably encounters several people daily with OSA. Although the role of the orthodontist is not to diagnose SDB, an opportunity to screen for SDB exists. Proper diagnosis can only be done through polysomnography (PSG) or home testing with portable monitors, with PSG being the gold standard. It is important for the orthodontist to recognize the signs and symptoms of SDB and refer the patient to a sleep medicine physician for proper diagnosis. An otorhinolaryngologist (also known as ENT physician), may also be consulted in suspected cases of chronic nasal obstruction or adenotonsillar hypertrophy.[152]

Definitions and Testing Reports

Breathing abnormalities detected during sleep are classified as apnea, hypopnea, respiratory effort–related arousals, and hypoventilation. Apnea is the cessation, or near cessation, of

airflow. It exists when airflow is less than 20% of baseline for at least 10 seconds in adults.[153] In children, the duration criteria are shorter. Apnea is most commonly detected using airflow sensors placed at the nose and mouth of the sleeping patient. Inspiratory airflow is typically used to identify an apnea, although both inspiratory and expiratory airflows are usually abnormal. Some laboratories use surrogate measures instead, such as inspiratory chest wall expansion. Three types of apneas may be observed during sleep:

- OSA occurs when airflow is absent or nearly absent but ventilatory effort persists. It is caused by complete, or nearly complete, UA obstruction.
- **Central apnea** occurs when both airflow and ventilatory effort are absent.
- Mixed **apnea** is mix of intervals during which no respiratory efforts occur (i.e., central apnea pattern) and intervals during which obstructed respiratory efforts occur.

The most common breathing abnormality scored in a sleep study is called *hypopnea*, which is an abnormal reduction of airflow to a degree that is insufficient to meet the criteria for an apnea. As further classified, obstructive hypopneas are due to partial UA obstruction, which can be heard as snoring. Central hypopneas are due to reduced inspiratory effort. Although the criteria for hypopnea vary among sleep laboratories, a common definition is ≥30% reduction of breathing movements or airflow for at least 10 seconds, with ≥3% or 4% oxyhemoglobin desaturation. Similarly to apnea, hypopnea is detected using airflow sensors or surrogate measures, such as chest wall expansion. Airflow is typically used to identify hypopnea, and both inspiratory and expiratory airflows are usually abnormal.

Another class of breathing abnormalities, respiratory effort–related arousals (RERAs), which are episodes during which breathing and oxygenation are maintained at the expense of a great increase in respiratory efforts, results from increased UA resistance. RERAs are terminated by an arousal, which is often characterized by a resuscitative snore or an abrupt change in respiratory measures with arousal and a change in breathing sounds. Patients with RERAs tend to have frequent microarou sals of 3 seconds or less during sleep.

Repetitive RERAs associated with daytime sleepiness was previously called UA resistance syndrome, a subtype of OSA. These patients may exhibit abnormal sleep and cardiorespiratory changes that are typical of OSA.

Sleep hypoventilation is expressed by a reduction in only the oxygen level or an increase in the carbon dioxide level without measurable changes in breathing patterns evident in the airflow monitor. Sleep hypoventilation is usually presumed when persistent oxyhemoglobin desaturation is detected without an alternative explanation.

Some reported measures from a sleep study are common to assess the severity of suspected sleep apnea, either an attended in-house study with sleep measures and cardiopulmonary monitoring or an unattended patient-based home monitor with no sleep measures.

- **Apnea-Hypopnea Index (AHI)**—The AHI is the total number of apneas and hypopneas per hour of sleep. The AHI is most commonly calculated per hour of total sleep and is the current defining measure of disease and disease risk.[154-156] However, an AHI is occasionally calculated per hour of non-REM sleep, per hour of REM sleep, or per hour of sleep in a certain position to provide insight into the sleep-stage dependency or sleep-position dependency. If AHI values are 4 or less, then the patient is within normal limits. OSA is mild when the AHI reflects 5 to 15 episodes per hour of sleep, moderate when the AHI reflects 15 to 29 episodes per hour of sleep, and severe when the AHI reflects 30 and higher episodes per hour of sleep.[157]
- **Respiratory Disturbance Index (RDI)**—The RDI is the total number of events (apneas, hypopneas, and RERAs) per hour of sleep. The RDI is generally larger than the AHI, because the RDI considers the frequency of RERAs, whereas the AHI does not.[153] OSA severity is defined as mild when the RDI reflects 5 to 15 episodes per hour of sleep, moderate when the RDI reflects 15 to 30 episodes per hour of sleep, and severe when the RDI reflects 30 or more episodes per hour of sleep.
- **Reporting oxygen saturation.** Oxygen desaturation is a consequence of SDB. The oxygen desaturation index (ODI) is the number of times that the oxygen saturation falls by more than 3% to 4% per hour of sleep. The percent of sleep time during which oxygen saturation is <90% quantifies the exposure to hypoxemia. This measure and mean oxygen saturation are associated with a risk for cardiovascular disorders and glucose intolerance.[158] Minimum levels (i.e., troughs) of oxygen saturation are important because severe hypoxemia is considered a risk for cardiac arrhythmias.[159]

If sleep is measured by a monitor, then the sleep stages and comments on whether the sleep stages are all present and in order will be reported. One measure of interrupted sleep is the arousal index, calculated as arousals per hour of sleep. The arousal index score is generally lower than the AHI or RDI score because approximately 20% of apneas or hypopneas are not accompanied by arousals. However, the arousal index score can be greater than the AHI or RDI score if arousals that occur are due to causes other than apneas or hypopneas. As examples, arousals can be caused by periodic limb movements, noise, and sleep state transitions.

There are limitations common to the definitions and indexes previously described, and each will differ according to the diagnostic study performed. Specifically, indexes determined by PSG define the number of events per hour of electroencephalographically documented sleep, whereas indexes determined by portable monitoring define the number of events per hour of recording time, subjectively estimated sleep time, or time in bed. In portable monitoring, the tendency is to overestimate the sleep time and thus underestimate the index.

Classifications of Sleep-Disordered Breathing

The syndromic classification of SDB results from a combination of testing results and symptom presentations.

Obstructive Sleep Apnea

OSA is defined as either[160]:

- More than 15 apneas, hypopneas, or RERAs per hour of sleep (i.e., an AHI or RDI >15 events per hour) in an asymptomatic patient, OR
- More than 5 apneas, hypopneas, or RERAs per hour of sleep (i.e., an AHI or RDI >5 events per hour) in a patient with symptoms (e.g., sleepiness, fatigue, inattention) or signs of disturbed sleep (e.g., snoring, restless sleep, respiratory pauses).

OSA syndrome applies only to the latter definition. In both situations, more than 75% of the apneas or hypopneas must have an obstructive pattern.

Upper Airway Resistance Syndrome

Individuals previously diagnosed with upper airway resistance syndrome (UARS) are now classified as having OSA by the most recent *International Classification of Sleep Disorders—Third Edition* (ICSD-3).[161] UARS refers to RERAs accompanied by symptoms or signs of disturbed sleep.

Central Sleep Apnea Syndrome

Central sleep apnea syndrome (CSAS) exists when symptoms or signs of disturbed sleep are accompanied by more than five central apneas plus hypopneas per hour of sleep and normocarbia during wakefulness.[162] The threshold frequency of events that warrants treatment beyond that required for the underlying disease is unknown. The UA has little or no involvement in CSAS. A special case of recurrent central apneas is called Cheyne-Stokes respiration and refers to a cyclic pattern of central apneas and crescendo-decrescendo tidal volumes. Cheyne-Stokes respiration is considered a type of CSA and is commonly associated with heart failure or stroke. UA obstruction does not play a major role in this syndrome.

Sleep Hypoventilation Syndromes

Patients with one of the hypoventilation syndromes generally have mild hypercarbia when awake, which worsens during sleep. The two hypoventilation syndromes are congenital central hypoventilation syndrome (CCHS) and obesity hypoventilation syndrome (OHS).

Epidemiologic Factors

The term *sleep apnea* encompasses a number of different clinical problems. In OSA, the most common form of sleep apnea, episodes of apnea occur during sleep as a result of airway obstruction at the level of the oropharynx and velopharynx (also called the *nasopharynx*). Several studies have shown that OSA is a common disorder that represents a significant public health problem.[163] A large prevalence study in state employees found that undiagnosed SDB "is prevalent and has a wide range of severity in middle aged women and men."[164] In this study, 9.1% of men and 4.0% of women had an apnea and/or a hypopnea index of 15 or more events per hour of sleep. Therefore in the United States alone, more than 3 million men and 1.5 million women meet at least one definition of OSA (apnea and/or hypopnea index of 5 or more plus a complaint of daytime sleepiness). In addition, clinicians are recognizing OSA in their patients with increasing frequency. In the United States, the annual number of patients diagnosed with sleep apnea between 1990 and 1998 increased from 108,000 to over 1.3 million—a 12-fold increase.[165]

Although the pathophysiologic factors do not really differ across ages or genders, subtle differences in the presentations and management of disease are emerging.

OSA in children is a special case for several reasons. The presenting symptoms are more likely to be behavioral problems during the day and below expected performance in school.[166] Sleepiness during the day is less common than hyperactivity, and a consideration of OSA is warranted in those with ADHD. Adenotonsillar hypertrophy by itself or in the presence of obesity, the prevalence of which is increasing, is a major cause for OSA in children. The scoring rules for diagnosis are different in children, with less emphasis on the number of apnea episodes or hypoxemia and more emphasis on the number of hypopneas, RERAs, and arousals from sleep.[167] Adenotonsillary surgery plays a greater role in children than in adults, although one suspects that as the individual ages, he or she might be at greater risk for SDB.

In young women and in women before menopause, OSA is accompanied with more complaints of fatigue and depression than with snoring or sleepiness, and a workup for hypothyroidism is more often negative.[168] The results of a PSG may be dominated by arousals, RERAs, and hypopneas.[153] The special case of pregnancy is also a time when a woman is more vulnerable to OSA because of edema, nasal congestion, progesterone and small lung volumes, resulting in increased oscillation from hyperventilation to apnea.[169]

Pathophysiologic Factors

The preponderance of evidence indicates that the pharynx is abnormal in size and/or is capable of collapsing or being collapsed in patients with OSA. This single tube, the pharynx, is obligated by nature to serve a number of functions that, at face value, seem to conflict. The pharynx must be collapsible because, as an organ for speech and deglutition, it must be able to change shape and close. However, as a conduit for airflow, it must also resist collapsing. The parsimonious solution to this design problem involves a group of muscles that can alter the shape of the pharynx when an individual swallows or speaks but will hold it open when he or she inhales.

With sleep, there is a reduced tonic input to the UA muscles, diminished reflexes that protect the pharynx from collapsing, reduced compensation for resistive loading, and an increased chemoreceptor set point during non–rapid eye movement (non-REM) sleep, which reveals a sensitive hypocapnia-induced apneic threshold. An abnormal pharynx can be kept open in wakefulness by an appropriate compensatory increase in dilator muscle activity,[170] but this compensation fails during sleep and the airway collapses. Partial collapse results in snoring, hypopneas, and, in some cases, prolonged obstructive hypoventilation. Complete closure results in an apnea.

The anatomic location of a UA obstruction may be anywhere from the nose to the glottis, with the most frequent site of primary obstruction being the velopharynx at the level of the soft palate and the oropharynx.[170] Isolated or a few apneas and hypopneas normally occur at sleep onset and in REM sleep; therefore when the events become recurrent, a disease process is recognized. Besides anatomic vulnerability, the physiologic causes for recurrent apneas during sleep are three-fold: ventilation transitions from waking to sleeping and back (for it is *sleep* apnea rather than *wake* apnea), a reduced UA activation in response to an apnea or hypopneas, and a high arousal threshold that awakens the patient with a ventilatory overshoot before a compensatory response occurs during sleep.[171] There is a history of relevant computational approaches for understanding recurrent obstructive and central apneas with models using mechanical properties (lungs, chest wall, and UA), fluid dynamics, circulation, and controller (brainstem) characteristics.[172,173]

Clinical Presentation

The most recognized manifestations of the OSA are stentorian snoring and severe sleepiness; however, both snoring and sleepiness may be denied or minimized by the patient or are less obvious than other symptoms, such as insomnia or fatigue or inattention. Sleepiness may develop fatigue very slowly over years or sleepiness may be considered normal, considering the prevalence of sleep deprivation.[160] The observations of someone

who has seen the patient's sleep behavior and can report on daytime alertness can be helpful; however, if the patient does not consider the report as a problem, then the next step of diagnosis and the adherence to treatment are often viewed with skepticism. When obstructive events during sleep in a habitual snorer are witnessed, these events are a strong predictor of the presence of sleep apnea, but it does not predict its severity.[160] OSA increases with age and is twofold higher in men than in women up until the age of approximately 60 years, after which the opportunity for finding snoring, increased AHI, and OSA is equal.[168]

In adults, some other common reports include:

- Physically restless sleep and reports of insomnia
- Morning dry mouth or sore throat from mouth breathing upon recovery from apnea and/or hypopnea
- Morning confusion and headache from increases in carbon dioxide levels
- Personality changes (irritability and distracted demeanor) and judgment changes resulting from sleepiness
- Night sweats, secondary to increased work of breathing
- Erectile dysfunction, especially in the setting of hyperlipidemia

Physical Characteristics

The patient with OSA exhibits a greater prevalence of hypertension, obesity, a large neck, and a structurally abnormal or crowded UA.[174] The abnormal airway is the critical factor; consequently, OSA is not really an age- or gender-specific disease. The loci of obstruction in the UA are not easily assessed by the clinician while the patient is awake. The orthodontist can detect nasal obstruction, a low hanging soft palate and large uvula, enlarged tonsils and adenoids, and retrognathia or micrognathia. Nasopharyngeal tumors are rare but must be ruled out. Other disorders that can crowd or affect the pharynx include hypothyroidism, acromegaly, amyloidosis, neuromuscular disease, and vocal cord paralysis. Neck size is an important predictor of sleep apnea and, in some cases, is a better predictor than body mass index (BMI; the weight in kilograms divided by the square of the height in meters), presumably because additional tissue can influence the size or compliance of the UA.

Clinical Prediction of Significant Sleep-Disordered Breathing

Clinical features may not reliably predict sleep apnea in all patients suspected of having the disorder or being screened for sleep apnea. Some of the reasons are the nature of the definitions for significant OSA, meaning that the range of severity is from 5 events (1 event every 12 minutes) to 60 events or more. However, several models have been developed that can help the clinician decide which patients could be referred for more definitive testing.[160] The most useful models use the following clinical domains:

- Presence of hypertension (or hypertension treatment)
- BMI 30 or higher
- Neck circumference (or collar size) of >17 inches for men and >16 inches for women
- History of habitual snoring, snorts, observed apneas, and restless sleep
- Observed reports of nocturnal choking or gasping
- Reported or observed sleepiness, inattention, and nocturnal changes in energy

Although questionnaire scorings vary, these reports should be tallied, either as binary traits or in a graduated fashion. A patient with a low clinical score will have a low or very low posttest probability of having important sleep apnea and does not need further evaluation. A patient with a high probability of having clinically important sleep apnea requires further testing. Patients with a clinical score that is intermediate can be potential candidates, but further evaluation is needed. If the context is a screening questionnaire, then some discussion is needed to alert the patient to the cost and treatment implications of only a questionnaire result. If the quiz is conducted in the context of an already suspected sleep apnea (e.g., a referral for an OA), then the questionnaire is used to assess clinical severity.

Clinical severity profiles are being developed and range from severe (very high sleepiness and profiles for abnormal breathing during sleep) to mild (loud snoring without cardiovascular or behavioral indications for immediate treatment). Moderate severity may be initially managed by weight loss or the treatment of an anatomic problem, such as a nasal polyp or rhinitis, leaving the use of a CPAP device or an OA and surgery for consideration after appropriate diagnostic testing.

However, no clinical algorithm or test, including these, is a substitute for clinical judgment. For instance, as previously noted, daytime sleepiness is both ubiquitous and underrecognized in this relatively sleep-deprived society and is therefore not a statistically significant predictor of sleep apnea. Nevertheless, all patients with an otherwise unexplained complaint of excessive daytime sleepiness deserve further evaluation. A sleepy, stentorian snorer who has had witnessed apneas probably should be examined during sleep, regardless of the size of his or her neck.

Tests and Questionnaires

The presence or absence and predicting the severity of OSA must be determined before initiating any kind of treatment.[175] The gold standard of OSA diagnosis is clinical examination, daytime sleepiness, and overnight PSG.[176] Other clinical tools that are more time efficient and clinically feasible are available and include screening questionnaires, indexes, and cephalometric analyses. Nevertheless, no consensus has been reached on which of these scoring questionnaires or indexes are the most valid and reliable.[177] Dentists should play a role in the diagnosis of OSA, which begins with patient history, physical examination, and objective testing.[175,177] Different screening methods have been developed to diagnose OSA to avoid mistakenly diagnosing it as simply snoring.[176] Examples of some screening methods are the Berlin Questionnaire, the Epworth Sleepiness Scale (ESS), the Sleep Disorders Questionnaire (SDQ), the STOPBang Questionnaire, the Kushida Index, apnea prediction score,[178] and the Friedman classification that includes the modified Mallampati (MMP) score.

Friedman Classification

In 1999, Dr. Michael Friedman showed that the combination of many factors such as tonsil size, MMP score, and BMI plays an important role in clinically predicting OSA.[179]

The tonsil size can be graded from 0 to 4 (Fig. 12-10).[180] The MMP scoring system was developed in 1983; it was initially developed to help clinically predict the ease versus difficulty of laryngeal intubation[181] and was performed to minimize the risk of failed intubation that could have fatal consequences since intubation is the only mean of breathing for patients undergoing general anesthesia.[182] This scoring system is based on the direct visualization of the soft palate, uvula, faucial pillar, and hard palate and on the concept of examining the tongue size relative to the oral cavity. Because measuring the size of

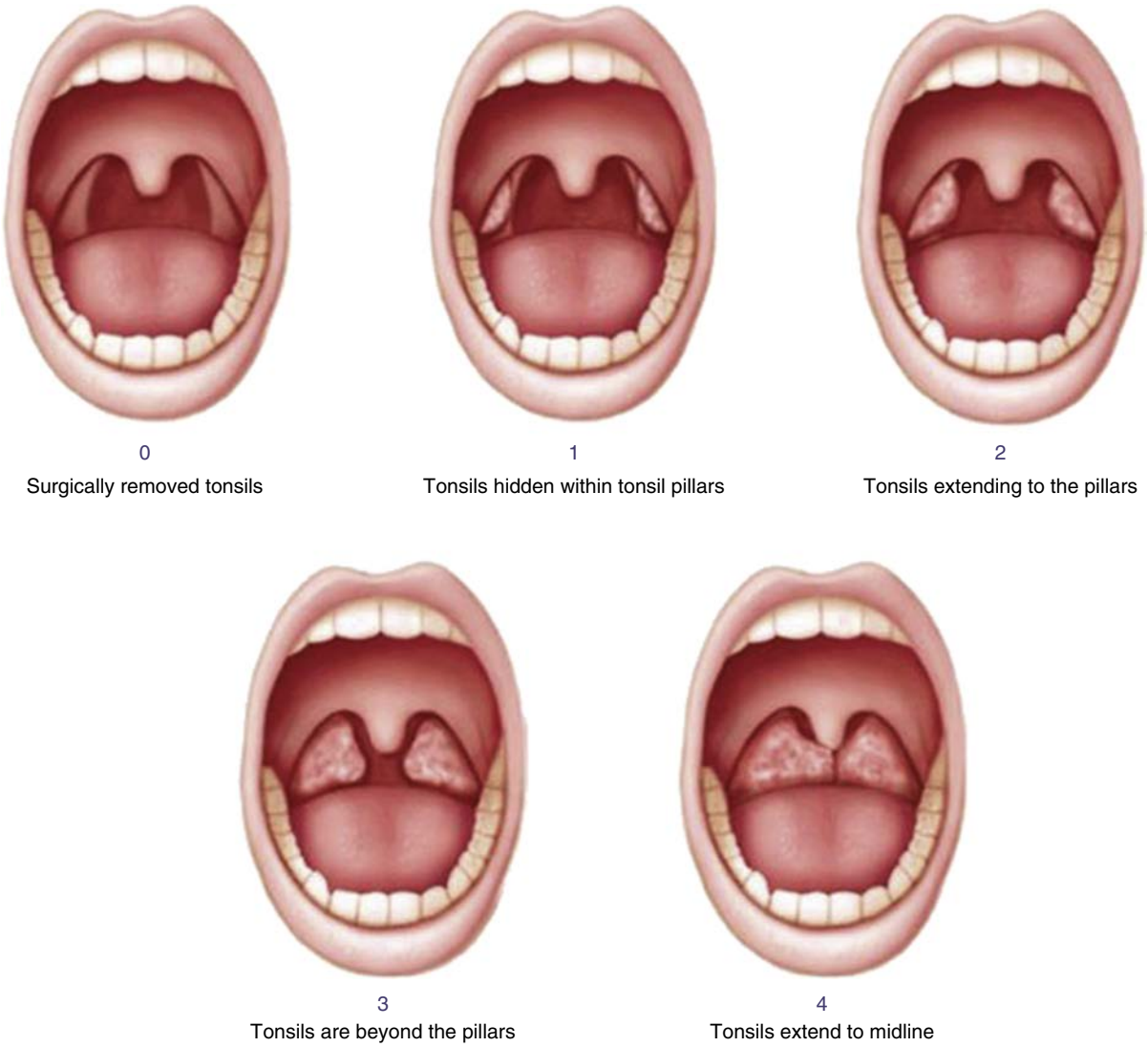


FIGURE 12-10 Tonsil classification. 0: surgically removed tonsils; 1: tonsils hidden within pillars; 2: tonsils extended to the pillars; 3: tonsils beyond the pillars; 4: tonsils extended to the midline.

the tongue relative to the oropharyngeal cavity is not possible, the MMP score is considered an indirect way of assessing the size of the base of the tongue.[182] The original classification was divided into three classes, Classes I, II, and III, that, respectively, coincide with the severity and difficulty of intubation. In 1987, Samsoon and Young suggested a modified Mallampati test (MMT), adding a fourth class (Fig. 12-11).[183] Although it was initially designed for predicting the difficulty of tracheal intubation, today, this clinical tool is used to assess patients with OSA and to detect those who have breathing problems attributable to UA obstruction.[184] A 2006 study reported the MMP score as an independent predictor of OSA, showing that, on average, for every point increase in the MMP score, the OSA increased by more than twofold and the AHI increased by more than five events per hour.[184]

As all different assessment scores and tools, the MMP score shows reliability in some tests for OSA and intubation prediction but was found inaccurate for postsurgical outcome assessments.[185]

It is important to note that the MMP score increases in certain conditions such as pregnancy; therefore it is important that clinicians not confuse high scores when they are caused by pathologic versus physiologic reasons.[186]

Mallampati Score Method

Three steps are followed to determine the MMP score:

Step 1. Patients are asked to take a seated or supine position. A study showed that the accuracy of predicting the intubation using the MMT was observed more in the sitting position; however, both positions are reliable.[187] If seated in an upright position, the head is in neutral position.

Step 2. Patients are asked to protrude their tongue as far forward as they can without emitting a sound. In the Friedman classification, the patient opens his or her mouth wide without protruding the tongue.[179]

Step 3. Through visual observation, a Class I to Class IV MMP score is determined.

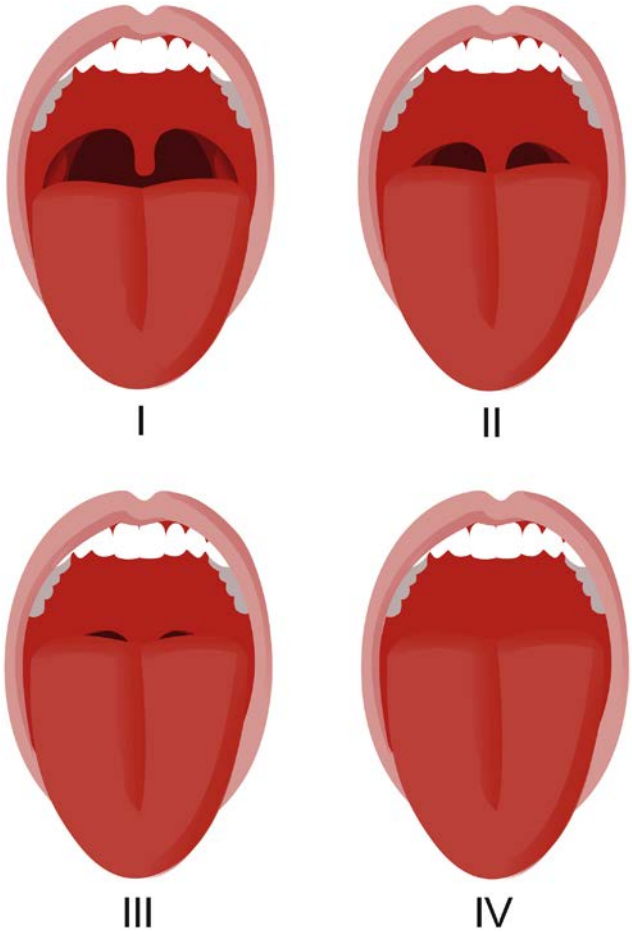


FIGURE 12-11 The modified Mallampati test (MMT) classifications. I: Soft palate, fauces, uvula, and pillars are visible. II: Soft palate, fauces, and uvula are visible. III: Soft palate and base of uvula are visible. IV: Soft palate is not visible.

Kushida Index

The Kushida index was developed in 1997 through a mathematical formula and is considered to have high levels of sensitivity and specificity.[188,189] It combines different measurements with BMI and neck circumference, creating a morphometric mathematical model to predict OSA. After the calculations are made, if the result is 70 or above, then it indicates a high risk of OSA. The Kushida index is calculated as follows:

$$\{P + (Mx - Mn) + 3 \times OJ\} + 3 \times [\text{Max}(BMI - 25, 0)] \times (NC/BMI).$$

where *P,* palatal height; *Mx,* maxillary intermolar distance; *Mn,* mandibular intermolar distance; *OJ,* overjet; *BMI,* body mass index; and *NC,* neck circumference.

The first part of the formula reflects the contribution of the craniofacial dysmorphism to predict OSA through measurements from the oral cavity, whereas the second part reflects the contribution of obesity.

Berlin Questionnaire

The Berlin questionnaire was developed in 1996 at the Conference on Sleep in Primary Care that took place in Berlin, Germany. This 10-question questionnaire is considered to be a very accurate method of predicting OSA.[190] The complete method is explained in Table 12-1.

STOPBang Questionnaire

The STOPBang questionnaire was developed in 2008 based on the Berlin Questionnaire by anesthetist Chung Frances and sleep specialists in Canada to screen patients at high risk for OSA preoperatively. It was first identified as the STOP Questionnaire, which stands for yes or no questions on snoring (S), tiredness (T), observed events (O), and blood pressure (P). It was later modified to STOPBang, adding BMI (B), age (A), neck circumference (N), and gender (G) (Table 12-2). Such modification improved the questionnaire's sensitivity.[191] A 2014 study reports that the STOPBang Questionnaire has the highest sensitivity to diagnose the patient with moderate to severe OSA when compared with other screening tests such as the Berlin Questionnaire, the original STOP questionnaire, and the ESS. Their result showed that with AHI of ≥5 events per hour, AHI of ≥15 events per hour, and AHI ≥30 events per hour as cut-offs, the sensitivities of the STOPBang Questionnaire were 83.6%, 92.9%, and 100%, and the specificities were 56.4%, 43%, and 37%, respectively.[192]

Epworth Sleepiness Scale

The ESS was introduced by Dr. Murray Johns in 1991 and is a tool for assessing daytime sleepiness.[193] It assesses eight situations with a likelihood of falling asleep and assigns scores from 0 to 3, giving a total scale of 0 to 24. ESS was found to play a relatively small role in screening patients at high risk of OSA and cannot be used to predict or screen patients for OSA.[194]

Cephalometric Analysis

Although cephalometrics has an inherent limitation of being a 2D representation of a 3D structure, cephalometric analysis is still common in orthodontic offices. The sagittal and vertical position of different structures such as soft palate, tongue, airway, and hyoid position measurements could help detect an airway problem. In 1984, McNamara described his cephalometric technique,[195] which included assessment of the UA. He suggested taking two measurements, one for the upper pharynx and the other for the lower pharynx dimension. The upper pharynx dimension was described as the minimum distance between the upper soft palate and the nearest point of the posterior pharynx wall. This distance was determined to increase with age, with its norm established at 17.4 ± 3.4 mm. The lower pharynx dimension is the minimum distance between the posterior wall of the pharynx and a point seen in the cephalograms as the intersection between the posterior contour of the tongue with the lower border of the mandible. The lower pharynx dimension was not seen to change with age, and its norm was determined to be 11.3 ± 3.3 mm for females and 13.5 ± 4.3 mm for males (Fig. 12-12).[195] The anteroposterior and vertical positions of the hyoid bone can also be seen in the cephalograms, but a 3D modality such as the CBCT allows a far more comprehensive analysis, including axial and mediolateral assessments.

Sleep Disorders Questionnaire

The SDQ was developed from the sleep questionnaire and assessment of wakefulness in 1993 by retrieving the best questions, creating a multivariate scoring scale (Table 12-3). The four clinical diagnostic scales are sleep apnea (SA), narcolepsy (NAR), psychiatric sleep disorder (PSY), and periodic limb movement (PLM) disorder.[196] In a 2003 study, the 12 SA-SDQ questions proved to be valid and useful in screening OSA with a good degree of sensitivity and specificity.[197] It is important to

TABLE 12-1 Berlin Questionnaire

Ten multiple-choice questions, divided into three categories. Asterisk (*) marks answers that would give a positive score. A category becomes positive when two or more questions score positive. If no or one category is positive, then the patient is at low risk of obstructive sleep apnea (OSA). If two or more categories are positive, then the patient is at high risk of OSA.

Height (m) _____ Weight (kg) _____ Age _____ Male/Female _____

Please choose one response per question.

Category 1

1. Do you snore?
   a. Yes*
   b. No
   c. I do not know

If you snore, please answer 2-4.

2. Your snoring is:
   a. Slightly louder than breathing
   b. As loud as talking
   c. Louder than talking*
   d. Very loud and can be heard in adjacent rooms*

3. How often do you snore?
   a. Nearly every day*
   b. Three to four times a week*
   c. One to two times a week
   d. One to two times a month
   e. Never or nearly never

4. Has your snoring ever bothered other people?
   a. Yes*
   b. No
   c. I do not know

5. Has anyone noticed that you quit breathing during your sleep?
   a. Nearly every day*
   b. Three to four times a week*
   c. One to two times a week
   d. One to two times a month
   e. Never or nearly never

Category 2

6. How often do you feel tired or fatigued after you sleep?
   a. Nearly every day*
   b. Three to four times a week*
   c. One to two times a week
   d. One to two times a month
   e. Never or nearly never

7. During your waking time, do you feel tired, fatigued, or not up to par?
   a. Nearly every day*
   b. Three to four times a week*
   c. One to two times a week
   d. One to two times a month
   e. Never or nearly never

8. Have you ever nodded off or fallen asleep while driving a vehicle?
   a. Yes*
   b. No

If you answer No, please skip to 10.

9. How often does this occur?
   a. Nearly every day*
   b. Three to four times a week
   c. One to two times a week
   d. One to two times a month
   e. Never or nearly never

Category 3

10. Do you have high blood pressure?
    a. Yes*
    b. No
    c. I do not know

From Netzer NC, Stoohs RA, Netzer CM, Clark K, Strohl KP. Using the Berlin Questionnaire to identify patients at risk for the sleep apnea syndrome. Annals of internal medicine. 1999;131(7):485-91.

TABLE 12-2 STOPBang Questionnaire

The patient is at low risk of obstructive sleep apnea (OSA) if "Yes" is chosen fewer than three times and is at high risk of OSA if "Yes" is chosen three or more times.

Height (m) _____ Weight (kg) _____ Age _____ Male/Female BMI _____

Collar size of shirts: S, M, L, XL; or Neck Circumference (measured by staff): _____

Please answer the following questions:

1. Snoring
   Do you snore loudly (louder than talking or loud enough to be heard through closed doors)?
   a. Yes
   b. No

2. Tired
   Do you often feel tired, fatigued, or sleepy during the day?
   a. Yes
   b. No

3. Observed
   Has anyone observed you stop breathing during your sleep?
   a. Yes
   b. No

4. Pressure
   Do you have or are you being treated for high blood pressure?
   a. Yes
   b. No

5. Body Mass Index (BMI)
   Is your BMI more than 35 kg/m^2?
   a. Yes
   b. No

6. Age
   Are you 50 years of age or older?
   a. Yes
   b. No

7. Neck circumference
   (*male*) Is your shirt collar 17 inches (43 cm) or larger?
   (*female*) Is your shirt collar 16 inches (41 cm) or larger?
   a. Yes
   b. No

8. Gender
   Is your gender male?
   a. Yes
   b. No

Published with permission from University Health Network. The STOP Bang Tool is proprietary to the University Health Network. Any reproduction and/or use of the STOP Bang tool without written permission from the University Health Network is prohibited.

note that the SDQ is not a diagnostic tool that can establish the presence or absence of OSA; rather, it is a screening tool that detects potential risk factors.

Diagnostic Testing of Obstructive Sleep Apnea

Diagnosis of OSA requires that the patient be examined during sleep,[174] and the gold standard is to diagnose through PSG.[175] PSG can be performed as an in-laboratory full-night or split-night test that includes the analysis of the following tests: electroencephalogram, electrooculogram, chin electromyogram, airflow analysis, oxygen saturation, respiratory effort, and electrocardiogram, sometimes replaced by heart rate. Body position and excessive movements are also observed during this test.

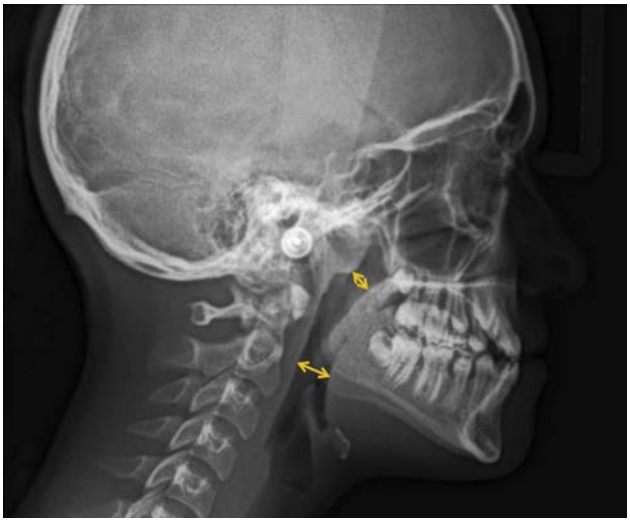


FIGURE 12-12 McNamara's airway analysis, showing upper and lower pharyngeal measurements (*arrows*).

The extent and location of the examination are the subjects of both clinical and cost-benefit concerns, but a full laboratory PSG is likely not necessary in most patients within whom the issue is merely the counting and classification of events for consideration of treatment of moderate to severe presentations of SDB and when no comorbidity, such as heart failure, chronic obstructive pulmonary disease (COPD), or other sleep behaviors exists that might require detection of sleep, arousals from sleep, and other measures for leg movements, seizures, or unusual behaviors (parasomnias). However, the proper diagnosis of OSA through PSG is recommended before any treatment is rendered to avoid potential life-threatening results.

Treatment Modalities

Conservative estimates suggest that 13% of men and 6% of women in the United States have clinically important OSA, and approximately 33% have moderate to severe disease.[198] Despite the transformative benefits of therapy with the CPAP device, many patients remain inadequately treated because they cannot or will not tolerate CPAP therapy. For these patients, other therapies can be considered.[199] Those most commonly considered include OAs[200] and surgery to the UA.[201,202] New forms of therapy include expiratory nasal valves, unilateral hypoglossal stimulation, and muscle exercises.[199,203] Bariatric surgery is indicated for eligible men and women and can reduce OSA to low levels in 85% of patients; the durability of this approach is under active consideration.[198]

Treatment Options

OSA should be approached as a chronic disease, requiring long-term, multidisciplinary management. The desired outcome of treatment includes the resolution of the clinical signs and symptoms, and the normalization of the AHI and oxyhemoglobin saturation.

TABLE 12-3 Sleep-Disorder Questionnaire Showing 34 Check Questions

Question are divided into four groups for scoring. Questions 1-12: Three or more checked boxes means symptoms of sleep apnea. Questions 13-19: Three or more checked boxes means symptoms of insomnia. Questions 20-27: Three or more checked boxes means symptoms of narcolepsy. Questions 28-34: Three or more checked boxes means symptoms of periodic limb movement disorder (uncontrollable leg or arm jerks during sleep) or restless leg syndrome.

Patient Name: _____ **Date:** _____

☐ 1.	I have been told that I snore.	☐ 18.	I wake up earlier in the morning than I would like to.
☐ 2.	I have been told that I stop breathing when I sleep.	☐ 19.	I lie awake for one-half hour or longer before I sleep.
☐ 3.	I have high blood pressure.	☐ 20.	When I am angry or surprised, I feel like my muscles go limp.
☐ 4.	My friends and family say that I am grumpy and irritable.	☐ 21.	I often feel like I am in a daze.
		☐ 22.	I have experienced vivid, dreamlike scenes.
☐ 5.	I have fallen asleep while driving.	☐ 23.	I have fallen asleep in social settings such as the movies or at a party.
☐ 6.	I have noticed my heart pounding or irregularly beating during the night.	☐ 24.	I have trouble at work because of sleepiness.
		☐ 25.	I have dreams soon after falling asleep or during naps.
☐ 7.	I get morning headaches.	☐ 26.	I have "sleep attacks" during the day, no matter how hard I try to stay awake.
☐ 8.	I suddenly wake, gasping for a breath.	☐ 27.	I have had episodes of feeling paralyzed during my sleep or on awakening.
☐ 9.	I am overweight.	☐ 28.	Other than when exercising, I still experience muscle tension in my legs.
☐ 10.	I seem to be losing my sex drive.	☐ 29.	I have noticed (or others have commented) that part of my body jerks during sleep.
☐ 11.	I often feel sleepy and struggle to remain alert.		
☐ 12.	I frequently wake with a dry mouth.	☐ 30.	I have been told I kick at night.
☐ 13.	I have difficulty falling asleep.	☐ 31.	When trying to go to sleep, I experience an aching or a crawling sensation in my legs.
☐ 14.	Thoughts race through my mind and prevent me from sleeping.	☐ 32.	I experience leg pain and cramps at night.
☐ 15.	I anticipate a problem with sleep several times a week.	☐ 33.	Sometimes I cannot keep my legs still at night. I just have to move them to feel comfortable.
☐ 16.	I wake up and cannot go back to sleep.		
☐ 17.	I worry about things and have trouble relaxing.	☐ 34.	Although I slept during the night, I feel sleepy during the day.

No treatment should be rendered without proper diagnosis through PSG.

According to the American Academy of Sleep Medicine, positive airway pressure (PAP) is the treatment of choice for mild, moderate, and severe OSA and should be offered as an option to all patients; however, depending on the severity of the OSA, the patient's anatomy, risks factors, and patient preferences, other options such as OAs and surgery may be adequate.[175]

Lifestyle and Behavioral Modification

According to the American Academy of Sleep Medicine, positive lifestyle modifications or behavioral treatments that can help in the treatment of OSA include the loss of weight to a BMI of 25 kg/m^2 or less, exercise, positional therapy during sleep, and avoidance of alcohol or sedatives before going to sleep.[175] Weight loss alone has not shown success in solving OSA; therefore it should always be accomplished in conjunction with other therapy.[204]

Sleeping in a supine position can affect the airway size and patency, helping the collapse of all structures. Positional therapy consists of a method that keeps the patient sleeping in a nonsupine position. Positional therapy will not always have a positive effect; consequently, PSG should be performed in both the supine and nonsupine positions before deciding if it will be a primary or secondary therapy for a patient. To prevent the patient from sleeping in a supine position, using objects such as tennis balls, pillows, or a backpack is recommended.[205]

Such behavioral changes can help achieve more positive outcomes and could be used as adjunctive therapy. For a patient diagnosed with OSA, however, using behavioral changes as the only treatment could be dangerous.

Positive Airway Pressure

PAP is the treatment of choice for all levels of OSA; it provides pneumatic splinting of the UA and shows positive outcomes in reducing the AHI.[206] PAP may be delivered as continuous (CPAP), bilevel (BPAP), or autotitrating (APAP) modes. The airway pressure can be applied through nasal, oral, or oronasal mask. PAP therapy is also indicated for improving sleepiness and quality of life and as an adjunctive therapy to lowering blood pressure in patients with OSA who also have hypertension. CPAP therapy is highly efficacious and currently the reference standard of treatment in preventing airway collapse. Early CPAP systems and masks were cumbersome and intrusive, but newer systems are light, less noisy, and easier to use. Nonetheless, many patients find the system difficult to tolerate.[207,208]

Oral Appliances

When the OSA is diagnosed as mild to moderate, OAs are considered a viable option.[175] Over the past decade, OAs have gained increasing acceptance as a viable treatment alternative to CPAP therapy for the treatment of OSA (Fig. 12-13). Patients often prefer OAs instead of a CPAP device because of their portability, ease of use, and comfort. OAs are also helpful with patients who snore or have UA resistance syndrome. Although they are currently indicated for mild to moderate OSA, there is increasing evidence of the potential role of OAs in patients with severe OSA who are intolerant or fail a trial of CPAP therapy

FIGURE 12-13 Patient with and without an oral appliance. The oral appliance positions the mandible forward and opens the mouth, increasing the vertical dimension. Intraoral frontal view without the appliance **(A)** and with the appliance **(B)**. Intraoral right side view without the appliance **(C)** and with the appliance **(D)**. (Courtesy of Dr. Aurelio A. Alonso.)

(Fig. 12-14 and Videos 12-6 and 12-7).[209-213] As a general rule, patients with severe OSA are not treated with OAs because of the concern that failed treatment or partial treatment may lead to respiratory failure. CPAP therapy has shown better results than OA therapy in bringing the AHI to <10 events per hour, but when Smith and Stradling substituted an OA for a CPAP device for a month, they reported that OA therapy produced a similar reduction in hypopneas from 29 to 4 events per hour.[214]

Better treatment responses have been found in younger patients,[215-217] patients with smaller neck circumferences,[210] women,[216] and supine-dependent patients with OSA.[216] A 10-year study found that the BMI does not play a role in long-term success of OAs.[218]

Cephalometric variables associated with better treatment responses have included a longer maxilla, shorter facial heights and soft palate, reduced overjet, and shorter distances between mandibular plane and hyoid bone. These variables are consistent with less severe OSA.[210,219,220]

Using an AHI of <5 events per hour as a measure of treatment success, approximately 35% to 40% of patients are successfully treated when using OA therapy. Another 25% showed at least a partial response.[209,210] Some individuals showed a worsening of OSA symptoms while on OA therapy; consequently, proper follow-up is important.[221,222] Overall, two-thirds of patients will experience improvement in OSA symptoms with OA therapy.[215,222]

A randomized cross-over study concluded that OA is effective in the treatment of patients with mild to moderate OSA with fewer side effects and greater patient satisfaction than nasal CPAP therapy.[223] Kyung and colleagues[118] indicated that OAs appear to enlarge the pharynx to a greater degree in the lateral plane than in the sagittal plane at the velopharynx. Such change would be visualized with 3D imaging and allow for proper follow-up.

The three general groups of OAs that are available include soft palate lift devices, tongue retention devices, and mandibular advancement splints (MAS).[224] A cross-over clinical trial comparing different OA designs[225] found MAS to be an effective alternative in treating patients with severe OSA, whereas tongue retention and soft palate lift devices did not achieve satisfactory results. MAS are the predominant type of OA used in clinical practice and have shown the best results. MAS effects include:

1. Enlargement of velopharyngeal airway caliber in the lateral dimension[226,227]
2. Increasing UA neuromuscular tone
3. Stimulation of UA dilator muscles[228,229]

MAS can be one piece (monobloc) or two pieces (bibloc) in design, custom-made or prefabricated, and titratable or nontitratable. Titratable appliances have shown greater reductions in obstructive events than nontitratable OAs, especially in patients with moderate to severe OSA.[217] The use of a dental implant–retained MAS[230] and mini-implants have been reported in edentulous and partially dentate patients.[231,232] Tongue-retaining and tongue-stabilizing devices, which protrude and hold the tongue forward by using suction, have also been suggested as a treatment alternative for edentulous patients.

Contraindications to the use of OA therapy include:

- Multiple comorbid conditions such as heart failure and respiratory failure, as well as when the possibility of central apnea and/or central hypoventilation exists
- Severe periodontal disease, when the risk for teeth mobility and loss is significant
- Severe temporomandibular disorder (TMD), in which the pain and dysfunction are aggravated with mandibular protrusion

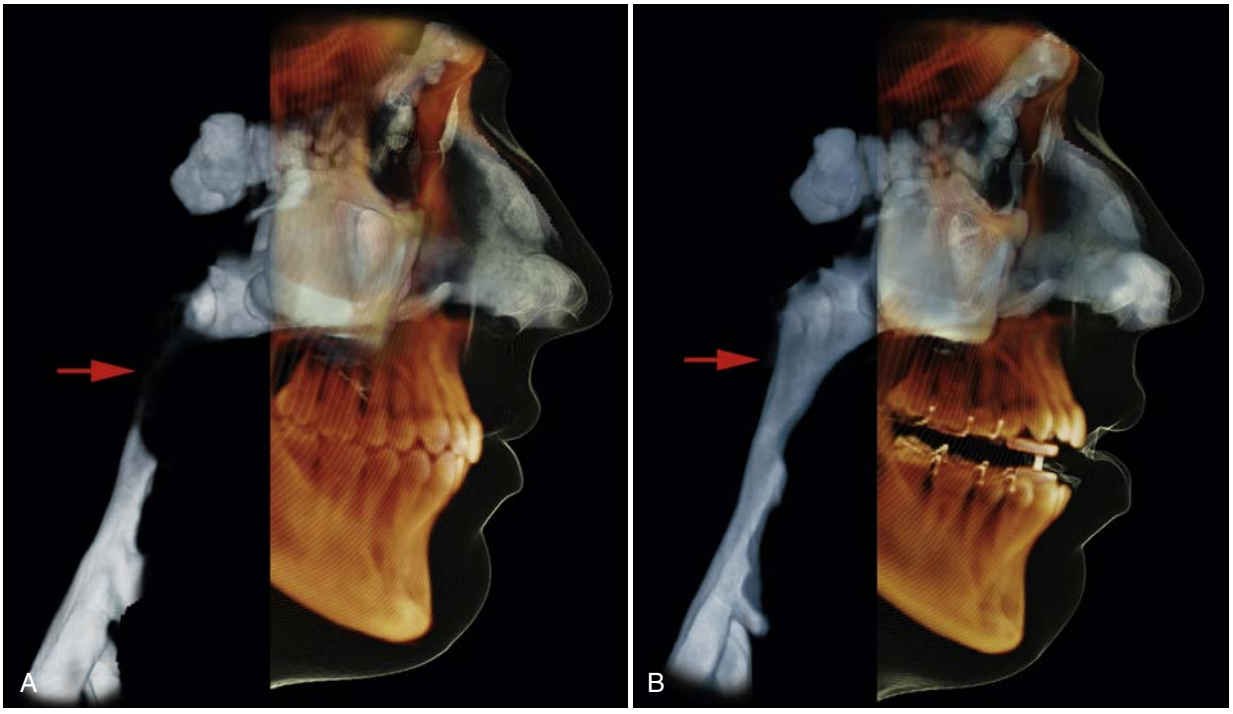
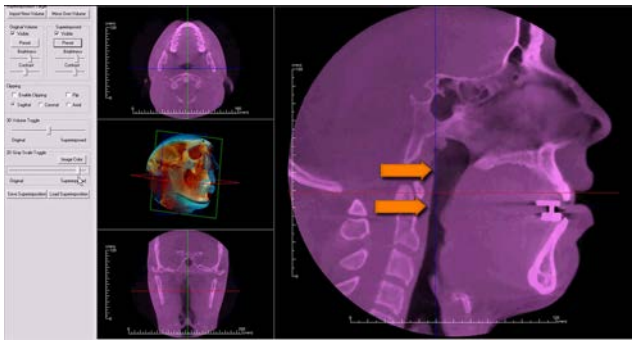
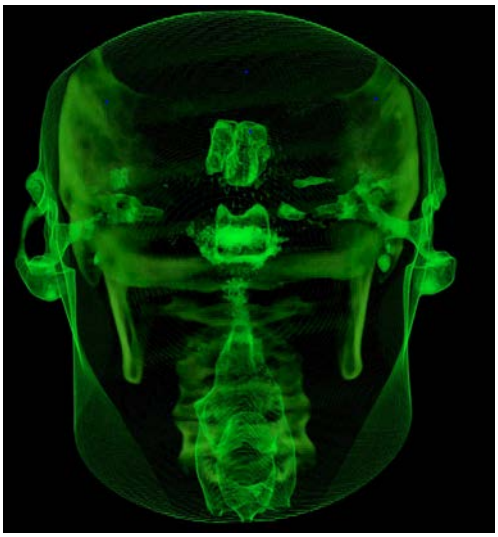


FIGURE 12-14 Three-dimensional images show the patient's airway without (A) and with (B) an oral appliance. With the appliance, the oropharyngeal airway volume went from 2128 mm^3 to 7797 mm^3. A most significant change occurred in the minimum axial area, which went from 1.1 mm^2 to 38.7 mm^2.



VIDEO 12-6 Superimposition of an OSA patient. Superimposition of an OSA patient with *(purple)* and without *(grey)* a mandibular advancement oral appliance, in all three planes of space. (From Anatomage Inc. San Jose, CA.)



VIDEO 12-7 Three dimensional superimposition of an OSA patient. 3D superimposition of an OSA patient with *(orange)* and without *(green)* a mandibular advancement oral appliance. Notice the medio-lateral change in the airway width, which would not be apparent in a lateral cephalogram.

- Severe gag reflex
- Poor coordination or dexterity as required for the placement and removal of OAs

Side effects of wearing an OA include excessive salivation, dry mouth, tooth discomfort, gingival irritation, masticatory muscle tenderness, and temporomandibular joint (TMJ) discomfort. With as few as 6 months of OA use, changes in facial height, as well as tooth and jaw positions, have been noted to occur.[233] Longer term 5-year studies reveal increases in facial height, occlusal changes, incisor inclination, and molar positional changes.[233-235]

Both the American Academy of Sleep Medicine and the American Academy of Dental Sleep Medicine released a joint resolution stating that the evaluation and management of patients with OSA should be overseen by a qualified physician trained in sleep medicine, which is defined as being licensed by a state to practice medicine and maintaining certification from the American Board of Sleep Medicine or a sponsoring sleep medicine board of the American Board of Medical Specialties. Similarly, OA therapy should be provided by a qualified dentist who completes additional education in dental sleep medicine and pursues objective verification of competency in OA therapy.[236]

Surgical Treatment

Although PAP treatment is the first line of treatment for moderate to severe OSA, successful long-term treatment is difficult to achieve.[208,237,238] Surgical procedures may be considered as a secondary option when the patient is intolerant of CPAP or OAs or when CPAP therapy is unable to eliminate OSA.[238] Surgery may also be considered as a secondary therapy in patients with mild to moderate OSA, when the patient is also intolerant of OAs, or when OA therapy provides unacceptable improvement of the clinical outcomes of OSA.[238,239] Surgery for OSA has been shown to improve important clinical outcomes including survival and quality of life.[208,238,239]

Surgical treatment of pediatric SDB with tonsillectomy and adenoidectomy is the recommended first-line treatment. In the pediatric population, resolution of OSA occurs in 82% of patients who are treated with tonsillectomy and adenoidectomy.[238,240]

Surgical treatment alternatives for OSA treatment include:

- **UA bypass procedure or tracheostomy:** This procedure creates an opening in the trachea to bypass the UA where obstruction is causing OSA. A tube or stoma is placed for ventilation.
- **Nasal procedures:** The objective of procedures such as septoplasty, functional rhinoplasty, inferior turbinate reduction, and nasal polypectomy[241-244] is to eliminate the obstruction that is preventing nasal breathing.
- **Tonsillectomy and/or adenoidectomy:** When OSA is properly diagnosed, as previously described in this chapter, such procedures can provide significant improvements in the treatment of OSA in children and young adults.
- **Uvulopalatopharyngoplasty:** The purpose of this procedure is to enlarge the velopharyngeal area, including trimming and reorienting the tonsillar pillars and excising both the uvula and the posterior part of the palate. Uvulopalatopharyngoplasty shows mixed results in the literature. This procedure can also be performed using a laser-assisted method, during which incisions are placed along both sides of the uvula, followed by laser ablation of the uvula rather than excision.
- **Radiofrequency ablation:** This technique consists of placing a temperature-controlled probe in the base of the tongue and/or soft palate with the objective of stiffening the area.[245] Soft palatal implanting of malleable plastic rods is also used with the same objective.
- **Orthognathic surgery:** Simultaneous advancement of both the maxilla and mandible has shown to provide significant enlargement of the velopharyngeal and overall oropharyngeal airway. Whenever possible, this procedure should be performed in conjunction with the orthodontist to achieve proper functional and aesthetic results.

Surgical procedures should be preceded by a thorough evaluation that includes an anatomic examination to identify possible surgical sites; an assessment of any medical, psychologic, or social comorbidities that might affect the surgical outcome; and a determination of the patient's desire for surgery. Tracheostomy as a bypass procedure can eliminate OSA but does not appropriately treat central hypoventilation syndromes.[175]

Maxillary and mandibular advancement can improve PSG parameters that are comparable with CPAP therapy in the majority of patients.

Bariatric surgery as an adjunctive surgery is an effective means to achieve major weight loss and is indicated in individuals with a BMI of ≥40 kg/m^2 or those with a BMI of ≥35 kg/m^2 with important comorbidities and in whom dietary attempts at weight control have been ineffective.[246]

Oropharyngeal Exercises

A less invasive option for the treatment of snoring and/or OSA includes exercises administered by an expert in myologic structure, arrangement, and action of orofacial muscles. As previously described, one of the reasons why OSA could occur in some patients is the larger size and hypotonicity of the oropharyngeal muscles. Oropharyngeal hypotonia may be linked to the pathogenesis of an individual and predispose him or her to OSA. To treat patients with OSA, the muscles responsible for blocking the UA need to be exercised to prevent airway collapse. The goal of the exercise is to strengthen the muscles located around the airway and to increase their tonicity, especially during sleep when muscles tend to relax. Oropharyngeal exercises can also improve stomatognathic function and reduce neuromuscular impairment.[247] The oropharyngeal muscles are the tongue, soft palate, neck muscles, and pharyngeal muscles. UA dilator muscles are very important to the maintenance of the pharyngeal opening and may contribute to the beginning of OSA.[248] In addition, if the neck muscles are flabby and weak, then they can exert pressure on the airway, which may lead to its collapse and obstruction of airway flow.

The oropharyngeal exercises target the soft palate, tongue, and facial muscle, as well as stomatognathic function. They are frequently performed during the day for few minutes in an isotonic (intermittent) and an isometric (continuous) way. Some examples of possible exercises include[248,249]:

1. **Soft palate:** An oral vowel is intermittently and continuously pronounced. The palatopharyngeus, palatoglossus, uvula, and tensor veli palatini and levator veli palatini muscles are recruited in this exercise. The intermittent exercise recruits the pharyngeal lateral wall as well. A blowing exercise is also performed, whether blowing a balloon or inhaling through the nose and exhaling through the mouth while keeping the lips together.
2. **Tongue:** Exercises that target the tongue include brushing the superior and lateral surfaces of the tongue while the

tongue is positioned in the floor of the mouth; placing the tip of the tongue against the front of the palate and sliding the tongue backward, which forces tongue sucking upward against the palate; pressing the entire tongue against the palate; and forcing the back of the tongue against the floor of the mouth while keeping the tip of the tongue in contact with the inferior incisive teeth.

3. **Facial:** The exercises of the facial musculature use facial imitations to recruit the orbicularis oris, buccinator, major zygomaticus, minor zygomaticus, levator labii superioris, levator anguli oris, lateral pterygoid, and medial pterygoid muscles.
   a. Muscle pressure of the orbicularis oris with the mouth closed
   b. Suction movements contracting only the buccinators (These exercises are performed with repetitions and holding position.)
   c. Recruitment of the buccinator muscle against the finger that is introduced in the oral cavity, pressing the buccinator muscle outward
   d. Alternating elevation of the mouth angle muscle
   e. Lateral jaw movements with alternating elevation of the mouth angle muscle
4. **Stomatognathic functions:**
   a. Breathing and speech: Forced nasal inspiration and oral expiration in conjunction with phonation of open vowels, while sitting; balloon inflation with prolonged nasal inspiration and then forced blowing
   b. Swallowing and chewing: Alternate bilateral chewing and deglutition, using the tongue in the palate, closed teeth, without perioral contraction, whenever feeding

A study performed in 2009[248] compared 15 control subjects with 15 matched experimental subjects, diagnosed with moderate OSA, and found statistically significant improvements after only 3 months of oropharyngeal exercises in snoring frequency, daytime sleepiness, sleep quality, and AHI levels, which reduced from 29.8 ± 12.7 to 17.4 ± 15.9 events per hour. The sample size was relatively small but shows promising results to this less invasive approach.

Other types of exercises, such as wind instrument playing and singing, have been studied in the literature and show mixed results.[250] A randomized trial with 25 subjects showed that 4 months of playing a particular wind instrument, the indigenous Australian didgeridoo, for approximately 25 minutes a day, 6 days a week, was associated with a significant reduction in snoring and daytime sleepiness. AHI reduced from a mean of 22 to 12 events per hour in the didgeridoo group, compared with a mean of 20 to 15 events per hour in the control group ($p < 0.05$).[251]

Playing the didgeridoo requires the use of circular breathing, a technique used to produce a continuous tone without break, accomplished by the use of the cheeks as a reservoir of air while breathing through the nose rather than the mouth. It has been suggested that practicing this wind instrument may train airway muscles, leading to less collapse of oropharyngeal muscles at night and resulting in its beneficial effect on OSA. Limitations of the study are a small sample size, and BMI <30. In contrast, a larger study that compared 369 orchestra wind instrument players with 736 orchestra non-wind instrument players found no significant differences between the groups. The study has some limitations, such as being an e-mail survey, lacking proper OSA diagnoses, and the presence of significantly higher BMI levels, as well as male players in the wind instrument group.[252] A more important limitation, which may welcome future studies, was the lack of sample power to stratify properly the different types of wind instruments. Special attention should be given to instruments requiring circular breathing, and this was not the objective of this particular study.

The oropharyngeal exercise option could use further studies and possibly develop into an adjunctive treatment option to the patient with OSA.

Upper Airway Electrical Neurostimulation

UA stimulation using a unilateral implantable neurostimulator for the hypoglossal nerve is a relatively novel and cutting-edge therapy for the treatment of patients with moderate to severe OSA who are intolerant to CPAP therapy. Patients with AHI >65 and/or BMI >32 are not good candidates for this therapy, attributable to a decreased likelihood of response to treatment. This therapy is also contraindicated when central and mixed apneas represent 25% or more of the AHI and when neurologic problems in the UA are due to a condition or previous procedures. The device is implanted in the chest and has a small generator, a breathing sensor lead, and a stimulation lead. The patient can turn on the therapy before bedtime and turn it off in the morning through a remote control (Fig. 12-15). When the device is activated, it senses the person's breathing patterns and delivers a mild stimulation to keep the airway open, acting in a similar fashion as a pacemaker. The hypoglossal nerve is accessed through a horizontal incision in the upper neck at the inferior border of the submandibular gland. The median time for surgical implantation has been reported as 140 minutes (65 to 360 minutes), with most patients spending the night at the hospital.[253] A clinical trial that included 126 patients showed a 68% decrease in AHI, from 29.3 events per hour to 9 events per hour, and a 70% ODI decrease, with an average reduction from 25.4 events per hour to 7.4 events per hour.[253]

Preventive Management

Morbidity is related to loss of alertness and cardiovascular complications. Patients with severe OSA, especially those with excessive sleepiness and neurocognitive impairments, have a two to seven times increased risk of having a motor vehicle accident.[254] Those at highest risk have had a fall-asleep car crash or near miss attributable to sleepiness or inattention. Therefore a health professional with expertise in the diagnosis and management of sleep apnea is expected to inform the patient of this potential risk and to suggest steps such as more sleep, timely diagnosis, and/or treatment, or avoidance of driving until the sleepiness is under better control.

Prospective studies suggest an increased risk of stroke, myocardial infarction, cardiovascular disease, and all-cause mortality.[155,156,255] Below 70 years of age, there is an increased risk for early death, although not necessarily during sleep. Other than for hypertension,[154] however, too few prospective studies have determined whether the treatment of OSA alone is sufficient to reduce risk.

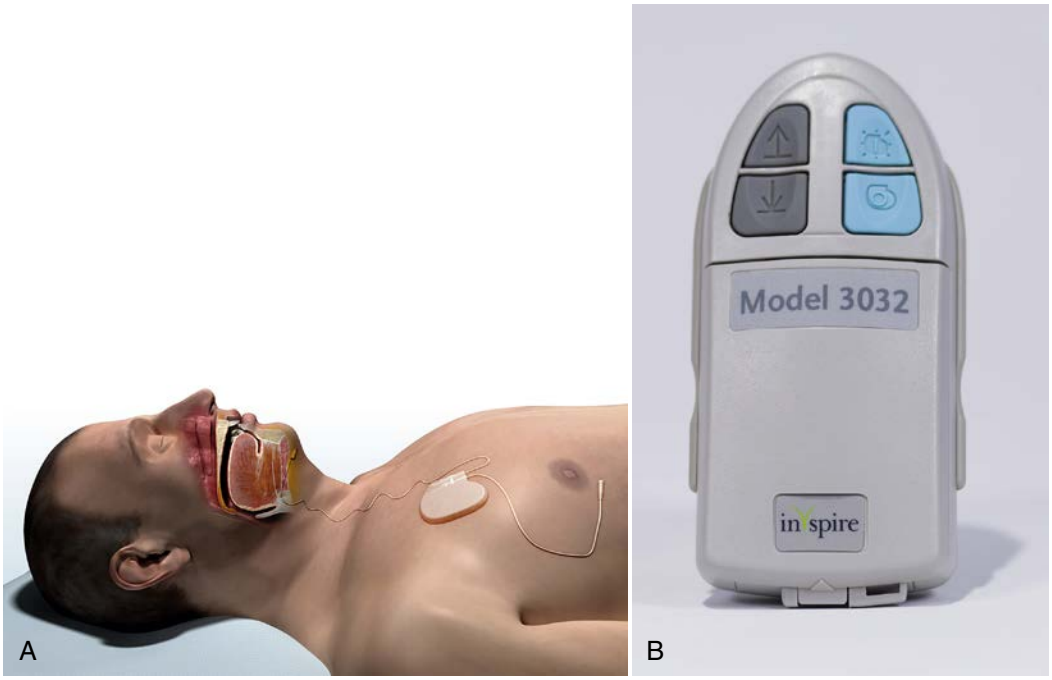


FIGURE 12-15 Upper airway electrical stimulation. **A,** *(From left to right)*Stimulation lead, generator, breathing sensor lead. **B,** Remote control. (Courtesy of Inspire Medical Systems, Inc., Maple Grove, MN.)

IMPORTANCE OF A MULTIDISCIPLINARY APPROACH

The relationship of the UA and orthodontics is such that a teamwork approach is essential. From an orthodontist's screening to physician referral for diagnosis, monitoring throughout orthodontic treatment, airway outcomes and beyond, involvement and coordination among multiple clinicians bring forth specific responsibilities of various specialists to patient care. The concept of a *patient care team* was first introduced in 1968 by the National Institutes of Health (NIH) and has since been recognized by the World Health Organization, The Joint Commission, and the American College of Physician Executives, among others, as a means to improve patient and process outcomes, as well as patient satisfaction.[256]

The importance of this approach is demonstrated in the case of a 62-year-old male patient. A three-unit fixed bridge from #8 to #10 had been placed by a general dentist without incident, other than waxing and waning plaque accumulation around the abutment margins over 3 years before being diagnosed with OSA by a sleep medicine physician. Two years after being fitted with a CPAP device, #8 abutment began to show signs of gingival trauma at the site where the CPAP device contacted #8's facial surface. Since plaque accumulation remained in check, few changes were noticeable. As plaque accumulation waxed and waned around the #8 abutment, gingival trauma was visible as fenestration (Fig. 12-16). However, with habitual plaque accumulation and ensuing inflammation around the gingival margins, secondary trauma from the CPAP device progressed to attachment loss. Unchecked, the combination of trauma and inflammation progressed to pathologic migration of the abutment. The patient's aesthetic complaints to the orthodontist centered on "a change in my smile"; however, secondary trauma from the CPAP device resulted in attachment loss and eventual abscess (Fig. 12-17).

This case underscores the need for primary teamwork skills. Leadership is needed to coordinate between the sleep medicine physician, the dentist, and the orthodontist to ensure that the CPAP fit does not create trauma (Fig. 12-18). Since inflammation is transient, situational monitoring among the physician, dentist, and orthodontist is needed to identify plaque accumulation, gingival inflammation and trauma from the CPAP fit, and ensuing gingival changes. Communication among all specialists is needed for clear and accurate informational exchange. Four key skills—leadership, situational monitoring, mutual support, and communication—are needed among the physician, dentist, periodontist, and orthodontist.

FIGURE 12-16 **A,** Gingival trauma at the point where the CPAP device contacts lip-over-labial surface of #8 abutment. **B,** The CPAP device fits over the lip and the labial surface of teeth #8 and #9. The gingiva has developed a fenestration at the traumatized site. **C,** No clearance is found between the CPAP device and the underlying abutment #8, as evidenced by the inability for a mouth mirror to fit passively between the lip and the facial gingiva of #8. **D,** After switching to a CPAP device of different design and fit, there is ample clearance for the mouth mirror to fit between the lip and facial gingiva of #8. (Courtesy of Dr. Alan Robbins.)

FIGURE 12-17 Gingival trauma from CPAP contact points and pressure has remained for over 2 years. Attachment loss progression and evidence of purulent abscess are demonstrated. (Courtesy of Dr. Alan Robbins.)

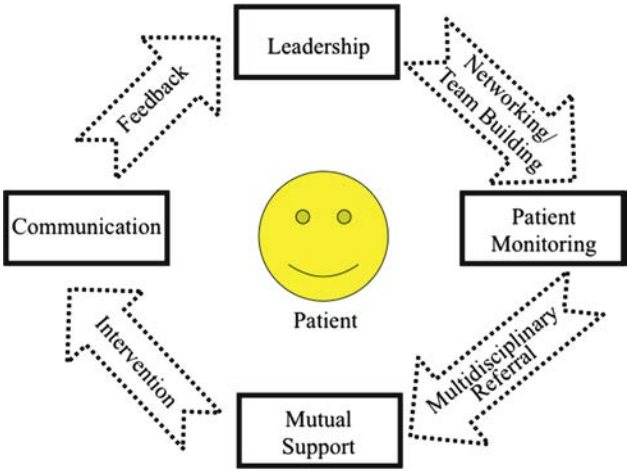


FIGURE 12-18 Teamwork flowchart.

SUMMARY AND CONCLUSIONS

The airway is an integral part of the craniofacial complex, often ignored during clinical examination and when determining the diagnosis and treatment options. Sleep disorders, most commonly OSA, affect a great portion of the adult population. Many patients go undiagnosed. In children, airway problems reportedly have detrimental effects on craniofacial growth and development. The orthodontist is strategically positioned to screen children and adults. The simple incorporation of a few questions and radiographic examination are probably sufficient to identify risks and to refer for proper diagnosis. Official diagnosis of a sleep problem through PSG is required before any treatment is rendered. Even if the orthodontist decides not to treat an airway problem, understanding the condition for the purpose of screening and referral is still an important responsibility of the orthodontist.

Acknowledgments

This chapter would not have been possible without the invaluable contributions provided by Dr. Noha Orabi, Dr. Ehab Ben Nasir, Dr. Ioannis Tsolakis, Dr. Adriano Farina, Dr. Athanasios Xofyllis, and Mrs. Cynthia McConnaughy.

REFERENCES

1. Stellzig-Eisenhauer A, Meyer-Marcotty P. Interaction between otorhinolaryngology and orthodontics: correlation between the nasopharyngeal airway and the craniofacial complex. *GMS Curr Top Otorhinolaryngol Head Neck Surg*. 2010;9. Doc04.
2. Meyer W. On adenoid vegetations in the naso-pharyngeal cavity: their pathology, diagnosis, and treatment. *Med Chir Trans*. 1870;53:191–216.1.
3. Tomes CS. On the developmental origin of the v-shaped contracted maxilla. *Monthly Rev Dent Surg*. 1872;1:2–5.
4. Angle EH. *Treatment of Malocclusion of the Teeth*. 7th ed. Philadelphia: S.S. White Dental Manufacturing; 1907.
5. Todd TW, Cohen MB, Broadbent BH. The role of allergy in the etiology of orthodontic deformity. *American Journal of Orthodontics and Oral Surgery*. 1939;25(8):807–808.
6. Schendel SA, Eisenfeld J, Bell WH, et al. The long face syndrome: vertical maxillary excess. *Am J Orthod*. 1976;70(4):398–408.
7. Moss ML. The functional matrix. In: Kraus BS, Reidel R, eds. *Vistas in Orthodontics*. Philadelphia: Lea & Febiger; 1962:85–98.
8. Prates NS, Magnani M, Vladrighi HC. Respiração bucal e problemas ortodônticos: relação causa-efeito. *Rev Paul Odontol*. 1997;19(4):14–18.
9. Linder-Aronson S. Respiratory function in relation to facial morphology and the dentition. *Br J Orthod*. 1979;6(2):59–71.
10. Linder-Aronson S, Adenoids. Their effect on mode of breathing and nasal airflow and their relationship to characteristics of the facial skeleton and the denition. A biometric, rhino-manometric and cephalometro-radiographic study on children with and without adenoids. *Acta Otolaryngol Suppl*. 1970;265:1–132.
11. McNamara JA. Influence of respiratory pattern on craniofacial growth. *Angle Orthod*. 1981;51(4):269–300.
12. Harari D, Redlich M, Miri S, et al. The effect of mouth breathing versus nasal breathing on dentofacial and craniofacial development in orthodontic patients. *Laryngoscope*. 2010;120(10):2089–2093.
13. Diouf JS, Ngom PI, Sonko O, et al. Influence of tonsillar grade on the dental arch measurements. *Am J Orthod Dentofacial Orthop*. 2015;147(2):214–220.
14. Linder-Aronson S, Woodside DG, Daigle DJ. A longitudinal study of the growth in length of the maxilla in boys between ages 6-20 years. *Trans Eur Orthod Soc*. 1975:169–179.
15. Zettergren-Wijk L, Forsberg CM, Linder-Aronson S. Changes in dentofacial morphology after adeno-/tonsillectomy in young children with obstructive sleep apnoea—a 5-year follow-up study. *Eur J Orthod*. 2006;28(4):319–326.
16. Lite T, Dimaio DJ, Burman LR. Gingival pathosis in mouth breathers; a clinical and histopathologic study and a method of treatment. *Oral Surg Oral Med Oral Pathol*. 1955;8(4):382–391.
17. Wagaiyu EG, Ashley FP. Mouthbreathing, lip seal and upper lip coverage and their relationship with gingival inflammation in 11-14 year-old schoolchildren. *J Clin Periodontol*. 1991;18(9):698–702.
18. Souki BQ, Pimenta GB, Souki MQ, et al. Prevalence of malocclusion among mouth breathing children: do expectations meet reality? *Int J Pediatr Otorhinolaryngol*. 2009;73(5):767–773.
19. Swift AC, Campbell IT, McKown TM. Oronasal obstruction, lung volumes, and arterial oxygenation. *Lancet*. 1988;1(8577):73–75.
20. Cottle MH. A consideration of nasal, pulmonary and cardio-vascular interdependance and nasal-pulmonary function studies. *Rhinology*. 1980;18(2):67–81.
21. Sano M, Sano S, Oka N, et al. Increased oxygen load in the prefrontal cortex from mouth breathing: a vector-based near-infrared spectroscopy study. *Neuroreport*. 2013;24(17):935–940.
22. Kasai T. Sleep apnea and heart failure. *J Cardiol*. 2012;60(2):78–85.
23. Rama AN, Tekwani SH, Kushida CA. Sites of obstruction in obstructive sleep apnea. *Chest*. 2002;122(4):1139–1147.
24. Ayappa I, Rapoport DM. The upper airway in sleep: physiology of the pharynx. *Sleep Med Rev*. 2003;7(1):9–33.
25. Williams PL, Warwick R, Dyson M, Bannister LH, eds. *Gray's Anatomy*. 37th ed. London: Churchill Livingstone; 1989.
26. Nishino T, Hiraga K. Coordination of swallowing and respiration in unconscious subjects. *J Appl Physiol (1985)*. 1991;70(3):988–993.
27. Strohl KP, Fouke JM. Dilating forces on the upper airway of anesthetized dogs. *J Appl Physiol (1985)*. 1985;58(2):452–458.
28. Fouke JM, Teeter JP, Strohl KP. Pressure-volume behavior of the upper airway. *J Appl Physiol (1985)*. 1986;61(3):912–918.
29. Schwab RJ, Gupta KB, Gefter WB, et al. Upper airway and soft tissue anatomy in normal subjects and patients with sleep-disordered breathing. Significance of the lateral pharyngeal walls. *Am J Respir Crit Care Med*. 1995;152(5 Pt 1):1673–1689.
30. Taylor M, Hans MG, Strohl KP, et al. Soft tissue growth of the oropharynx. *Angle Orthod*. 1996;66(5):393–400.
31. Rivlin J, Hoffstein V, Kalbfleisch J, et al. Upper airway morphology in patients with idiopathic obstructive sleep apnea. *Am Rev Respir Dis*. 1984;129(3):355–360.
32. Lyberg T, Krogstad O, Djupesland G. Cephalometric analysis in patients with obstructive sleep apnoea syndrome. I. Skeletal morphology. *J Laryngol Otol*. 1989;103(3):287–292.
33. Burger CD, Stanson AW, Daniels BK, et al. Fast-CT evaluation of the effect of lung volume on upper airway size and function in normal men. *Am Rev Respir Dis*. 1992;146(2):335–339.
34. Suratt PM, Dee P, Atkinson RL, et al. Fluoroscopic and computed tomographic features of the pharyngeal airway in obstructive sleep apnea. *Am Rev Respir Dis*. 1983;127(4):487–492.
35. Kuna ST, Bedi DG, Ryckman C. Effect of nasal airway positive pressure on upper airway size and configuration. *Am Rev Respir Dis*. 1988;138(4):969–975.
36. Shepard Jr JW, Garrison M, Vas W. Upper airway distensibility and collapsibility in patients with obstructive sleep apnea. *Chest*. 1990;98(1):84–91.
37. Schwab RJ, Gefter WB, Pack AI, Hoffman EA. Dynamic imaging of the upper airway during respiration in normal subjects. *J Appl Physiol (1985)*. 1993;74(4):1504–1514.
38. Moore KL, Persaud TVN. *The Developing Human: Clinically Oriented Embryology*. 8th ed. Philadelphia: Saunders–Elsevier; 2007.

39. Papadopoulos N, Lykaki-Anastopoulou G, Alvanidou E. The shape and size of the human hyoid bone and a proposal for an alternative classification. *J Anat*. 1989;163:249–260.
40. Behlfelt K, Linder-Aronson S, Neander P. Posture of the head, the hyoid bone, and the tongue in children with and without enlarged tonsils. *Eur J Orthod*. 1990;12(4):458–467.
41. Cohen AM, Vig PS. A serial growth study of the tongue and intermaxillary space. *Angle Orthod*. 1976;46(4):332–337.
42. Nelson S, Cakirer B, Lai YY. Longitudinal changes in craniofacial factors among snoring and nonsnoring Bolton-Brush study participants. *Am J Orthod Dentofacial Orthop*. 2003;123(3):338–344.
43. Sforza E, Bacon W, Weiss T, et al. Upper airway collapsibility and cephalometric variables in patients with obstructive sleep apnea. *Am J Respir Crit Care Med*. 2000;161(2 Pt 1):347–352.
44. Verin E, Tardif C, Buffet X, et al. Comparison between anatomy and resistance of upper airway in normal subjects, snorers and OSAS patients. *Respir Physiol*. 2002;129(3):335–343.
45. El H, Palomo JM. Airway volume for different dentofacial skeletal patterns. *Am J Orthod Dentofacial Orthop*. 2011;139(6):e511–e521.
46. Alves Jr M, Franzotti ES, Baratieri C, et al. Evaluation of pharyngeal airway space amongst different skeletal patterns. *Int J Oral Maxillofac Surg*. 2012;41(7):814–819.
47. Kim YJ, Hong JS, Hwang YI, Park YH. Three-dimensional analysis of pharyngeal airway in preadolescent children with different anteroposterior skeletal patterns. *Am J Orthod Dentofacial Orthop*. 2010;137(3):306 e1–e11; discussion 306–307.
48. Grauer D, Cevidanes LS, Styner MA, et al. Pharyngeal airway volume and shape from cone-beam computed tomography: relationship to facial morphology. *Am J Orthod Dentofacial Orthop*. 2009;136(6):805–814.
49. Ceylan I, Oktay H. A study on the pharyngeal size in different skeletal patterns. *Am J Orthod Dentofacial Orthop*. 1995;108(1):69–75.
50. Abu Allhaija ES, Al-Khateeb SN. Uvulo-glosso-pharyngeal dimensions in different anteroposterior skeletal patterns. *Angle Orthod*. 2005;75(6):1012–1018.
51. Di Carlo G, Polimeni A, Melsen B, Cattaneo PM. The relationship between upper airways and craniofacial morphology studied in 3D. A CBCT study. *Orthod Craniofac Res*. 2015;18(1):1–11.
52. Alves PV, Zhao L, O'Gara M, et al. Three-dimensional cephalometric study of upper airway space in skeletal class II and III healthy patients. *J Craniofac Surg*. 2008;19(6):1497–1507.
53. El H, Palomo JM. An airway study of different maxillary and mandibular sagittal positions. *Eur J Orthod*. 2013;35(2):262–270.
54. Avrahami E, Englender M. Relation between CT axial cross-sectional area of the oropharynx and obstructive sleep apnea syndrome in adults. *AJNR Am J Neuroradiol*. 1995;16(1):135–140.
55. Tso HH, Lee JS, Huang JC, et al. Evaluation of the human airway using cone-beam computerized tomography. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2009;108(5):768–776.
56. Solow B, Kreiborg S. Soft-tissue stretching: a possible control factor in craniofacial morphogenesis. *Scand J Dent Res*. 1977;85(6):505–507.
57. Joseph AA, Elbaum J, Cisneros GJ, Eisig SB. A cephalometric comparative study of the soft tissue airway dimensions in persons with hyperdivergent and normodivergent facial patterns. *J Oral Maxillofac Surg*. 1998;56(2):135–139; discussion 139–140.
58. Welch KC, Foster GD, Ritter CT, et al. A novel volumetric magnetic resonance imaging paradigm to study upper airway anatomy. *Sleep*. 2002;25(5):532–542.
59. Patel A, Bhavra GS, O'Neill JR. MRI scanning and orthodontics. *J Orthod*. 2006;33(4):246–249.
60. Filho DI, Raveli DB, Raveli RB, et al. A comparison of nasopharyngeal endoscopy and lateral cephalometric radiography in the diagnosis of nasopharyngeal airway obstruction. *Am J Orthod Dentofacial Orthop*. 2001;120(4):348–352.
61. Major MP, Flores-Mir C, Major PW. Assessment of lateral cephalometric diagnosis of adenoid hypertrophy and posterior upper airway obstruction: a systematic review. *Am J Orthod Dentofacial Orthop*. 2006;130(6):700–708.
62. Ludlow JB, Walker C. Assessment of phantom dosimetry and image quality of i-CAT FLX cone-beam computed tomography. *Am J Orthod Dentofacial Orthop*. 2013;144(6):802–817.
63. Osorio F, Perilla M, Doyle DJ, Palomo JM. Cone beam computed tomography: an innovative tool for airway assessment. *Anesth Analg*. 2008;106(6):1803–1807.
64. Grauer D, Cevidanes LS, Proffit WR. Working with DICOM craniofacial images. *Am J Orthod Dentofacial Orthop*. 2009;136(3):460–470.
65. El H, Palomo JM. Measuring the airway in 3 dimensions: a reliability and accuracy study. *Am J Orthod Dentofacial Orthop*. 2010;137(4 Suppl):S50 e1–9; discussion S50–52.
66. Weissheimer A, Menezes LM, Sameshima GT, et al. Imaging software accuracy for 3-dimensional analysis of the upper airway. *Am J Orthod Dentofacial Orthop*. 2012;142(6):801–813.
67. Palomo JM, Valiathan M, Hans MG. 3D orthodontic diagnosis and treatment planning. In: Kapila S, ed. *Cone Beam Computed Tomography in Orthodontics: Indications, Insights, and Innovations*. 1st ed. Hoboken, NJ: Wiley-Blackwell; 2014:221–246.
68. Hilberg O, Jackson AC, Swift DL, Pedersen OF. Acoustic rhinometry: evaluation of nasal cavity geometry by acoustic reflection. *J Appl Physiol (1985)*. 1989;66(1):295–303.
69. Kaise T, Ukai K, Pedersen OF, Sakakura Y. Accuracy of measurement of acoustic rhinometry applied to small experimental animals. *Am J Rhinol*. 1999;13(2):125–129.
70. Cakmak O, Coskun M, Celik H, et al. Value of acoustic rhinometry for measuring nasal valve area. *Laryngoscope*. 2003;113(2):295–302.
71. Numminen J, Dastidar P, Heinonen T, et al. Reliability of acoustic rhinometry. *Respir Med*. 2003;97(4):421–427.
72. Wright S, Haight J, Zamel N, Hoffstein V. Changes in pharyngeal properties after uvulopalatopharyngoplasty. *Laryngoscope*. 1989;99(1):62–65.
73. Marshall I, Maran NJ, Martin S, et al. Acoustic reflectometry for airway measurements in man: implementation and validation. *Physiol Meas*. 1993;14(2):157–169.
74. Kamal I. Test-retest validity of acoustic pharyngometry measurements. *Otolaryngol Head Neck Surg*. 2004;130(2):223–228.
75. Hatzakis GE, Karsan N, Cook J, et al. Acoustic reflectance of pharyngeal structures in children. *Int J Pediatr Otorhinolaryngol*. 2003;67(4):373–381.
76. Gelardi M, Del Giudice AM, Cariti F, et al. Acoustic pharyngometry: clinical and instrumental correlations in sleep disorders. *Braz J Otorhinolaryngol*. 2007;73(2):257–265.
77. Brown IG, Zamel N, Hoffstein V. Pharyngeal cross-sectional area in normal men and women. *J Appl Physiol (1985)*. 1986;61(3):890–895.
78. Wahl N. Orthodontics in 3 millennia. Chapter 2: entering the modern era. *Am J Orthod Dentofacial Orthop*. 2005;127(4):510–515.
79. Bravo LA. Soft tissue facial profile changes after orthodontic treatment with four premolars extracted. *Angle Orthod*. 1994;64(1):31–42.
80. Ismail SF, Moss JP, Hennessy R. Three-dimensional assessment of the effects of extraction and nonextraction orthodontic treatment on the face. *Am J Orthod Dentofacial Orthop*. 2002;121(3):244–256.
81. Saldarriaga JR. Protocol for the treatment of anterior open bite patients: an evaluation. *World J Orthod*. 2007;8(3):285–299.
82. Bishara SE, Cummins DM, Zaher AR. Treatment and posttreatment changes in patients with Class II, Division 1 malocclusion after extraction and nonextraction treatment. *Am J Orthod Dentofacial Orthop*. 1997;111(1):18–27.
83. Germec-Cakan D, Taner T, Akan S. Uvulo-glossopharyngeal dimensions in non-extraction, extraction with minimum anchorage, and extraction with maximum anchorage. *Eur J Orthod*. 2011;33(5):515–520.
84. Chen Y, Hong L, Wang CL, et al. Effect of large incisor retraction on upper airway morphology in adult bimaxillary protrusion patients. *Angle Orthod*. 2012;82(6):964–970.
85. Valiathan M, El H, Hans MG, Palomo MJ. Effects of extraction versus non-extraction treatment on oropharyngeal airway volume. *Angle Orthod*. 2010;80(6):1068–1074.
86. Stefanovic N, El H, Chenin DL, et al. Three-dimensional pharyngeal airway changes in orthodontic patients treated with and without extractions. *Orthod Craniofac Res*. 2013;16(2):87–96.

87. Wang Q, Jia P, Anderson NK, et al. Changes of pharyngeal airway size and hyoid bone position following orthodontic treatment of Class I bimaxillary protrusion. *Angle Orthod*. 2012;82(1):115–121.
88. Shannon TP. Oropharyngeal airway volume following orthodontic treatment: premolar extraction versus non-extraction. Master's thesis. Knoxville, TN: University of Tennessee; 2012.
89. Hu Z, Yin X, Liao J, et al. The effect of teeth extraction for orthodontic treatment on the upper airway: a systematic review. *Sleep Breath*. 2015;19(2):441–451.
90. Martin D. Extractions for function and aesthetics. In: Girardot Jr RA, ed. *Goal Directed Orthodontics*. Los Gatos, CA: Roth Williams International Society of Orthodontics; 2013:427–474.
91. Zhong Z, Tang Z, Gao X, Zeng XL. A comparison study of upper airway among different skeletal craniofacial patterns in nonsnoring Chinese children. *Angle Orthod*. 2010;80(2):267–274.
92. Hong JS, Oh KM, Kim BR, et al. Three-dimensional analysis of pharyngeal airway volume in adults with anterior position of the mandible. *Am J Orthod Dentofacial Orthop*. 2011;140(4):e161–e169.
93. Gluckman J. The clinical approach to nasal obstruction. *J Respir Dis*. 1983;4:13–29.
94. Haas AJ. The treatment of maxillary deficiency by opening the midpalatal suture. *Angle Orthod*. 1965;35:200–217.
95. Haas A. Rapid expansion of the maxillary dental arch and nasal cavity by opening the midpalatal suture. *Angle Orthod*. 1961;31:73–90.
96. Baratieri C, Alves Jr M, de Souza MM, et al. Does rapid maxillary expansion have long-term effects on airway dimensions and breathing? *Am J Orthod Dentofacial Orthop*. 2011;140(2):146–156.
97. Habeeb M, Boucher N, Chung CH. Effects of rapid palatal expansion on the sagittal and vertical dimensions of the maxilla: a study on cephalograms derived from cone-beam computed tomography. *Am J Orthod Dentofacial Orthop*. 2013;144(3):398–403.
98. Woller JL, Kim KB, Behrents RG, Buschang PH. An assessment of the maxilla after rapid maxillary expansion using cone beam computed tomography in growing children. *Dental Press J Orthod*. 2014;19(1):26–35.
99. Bell WH, Epker BN. Surgical-orthodontic expansion of the maxilla. *Am J Orthod*. 1976;70(5):517–528.
100. Doruk C, Sökücü O, Biçakçi AA, et al. Comparison of nasal volume changes during rapid maxillary expansion using acoustic rhinometry and computed tomography. *Eur J Orthod*. 2007;29(3):251–255.
101. Babacan H, Sokucu O, Doruk C, Ay S. Rapid maxillary expansion and surgically assisted rapid maxillary expansion effects on nasal volume. *Angle Orthod*. 2006;76(1):66–71.
102. El H, Palomo JM. Three-dimensional evaluation of upper airway following rapid maxillary expansion: a CBCT study. *Angle Orthod*. 2014;84(2):265–273.
103. Pallanch JF, McCaffrey TV, Kern EB. Evaluating nasal breathing function with objective airway testing. In: Cummings C, Fredrickson JM, Harker LA, et al., eds. *Otolaryngology—Head and Neck Surgery*. 2nd ed. St. Louis: Mosby Year Book; 1993:665–686.
104. Hilberg O. Objective measurement of nasal airway dimensions using acoustic rhinometry: methodological and clinical aspects. *Allergy*. 2002;57(Suppl 70):5–39.
105. Zhao Y, Nguyen M, Gohl E, et al. Oropharyngeal airway changes after rapid palatal expansion evaluated with cone-beam computed tomography. *Am J Orthod Dentofacial Orthop*. 2010;137(4 Suppl):S71–S78.
106. Schmidt-Nowara W, Lowe A, Wiegand L, et al. Oral appliances for the treatment of snoring and obstructive sleep apnea: a review. *Sleep*. 1995;18(6):501–510.
107. Farronato G, Giannini L, Galbiati G, Maspero C. Sagittal and vertical effects of rapid maxillary expansion in Class I, II, and III occlusions. *Angle Orthod*. 2011;81(2):298–303.
108. Malkoç S, Usümez S, Iseri H. Long-term effects of symphyseal distraction and rapid maxillary expansion on pharyngeal airway dimensions, tongue, and hyoid position. *Am J Orthod Dentofacial Orthop*. 2007;132(6):769–775.
109. Trenouth MJ, Timms DJ. Relationship of the functional oropharynx to craniofacial morphology. *Angle Orthod*. 1999;69(5):419–423.
110. Ozbek MM, Memikoglu TU, Gogen H, et al. Oropharyngeal airway dimensions and functional-orthopedic treatment in skeletal Class II cases. *Angle Orthod*. 1998;68(4):327–336.
111. Han S, Choi YJ, Chung CJ, et al. Long-term pharyngeal airway changes after bionator treatment in adolescents with skeletal Class II malocclusions. *Korean J Orthod*. 2014;44(1):13–19.
112. Erbas B, Kocadereli I. Upper airway changes after Xbow appliance therapy evaluated with cone beam computed tomography. *Angle Orthod*. 2014;84(4):693–700.
113. Battagel JM, Johal A, L'Estrange PR, et al. Changes in airway and hyoid position in response to mandibular protrusion in subjects with obstructive sleep apnoea (OSA). *Eur J Orthod*. 1999;21(4):363–376.
114. Hänggi MP, Teuscher UM, Roos M, Peltomäki TA. Long-term changes in pharyngeal airway dimensions following activator-headgear and fixed appliance treatment. *Eur J Orthod*. 2008;30(6):598–605.
115. Bar A, Tarasiuk A, Segev Y, et al. The effect of adenotonsillectomy on serum insulin-like growth factor-I and growth in children with obstructive sleep apnea syndrome. *J Pediatr*. 1999;135(1):76–80.
116. Nieminen P, Löppönen T, Tolonen U, et al. Growth and biochemical markers of growth in children with snoring and obstructive sleep apnea. *Pediatrics*. 2002;109(4):e55.
117. Iwasaki T, Takemoto Y, Inada E, et al. Three-dimensional cone-beam computed tomography analysis of enlargement of the pharyngeal airway by the Herbst appliance. *Am J Orthod Dentofacial Orthop*. 2014;146(6):776–785.
118. Kyung SH, Park YC, Pae EK. Obstructive sleep apnea patients with the oral appliance experience pharyngeal size and shape changes in three dimensions. *Angle Orthod*. 2005;75(1):15–22.
119. Sutherland K, Deane SA, Chan AS, et al. Comparative effects of two oral appliances on upper airway structure in obstructive sleep apnea. *Sleep*. 2011;34(4):469–477.
120. Ryan CF, Love LL, Peat D, et al. Mandibular advancement oral appliance therapy for obstructive sleep apnoea: effect on awake calibre of the velopharynx. *Thorax*. 1999;54(11):972–977.
121. Ozdemir F, Ulkur F, Nalbantgil D. Effects of fixed functional therapy on tongue and hyoid positions and posterior airway. *Angle Orthod*. 2014;84(2):260–264.
122. Kirjavainen M, Kirjavainen T. Upper airway dimensions in Class II malocclusion. Effects of headgear treatment. *Angle Orthod*. 2007;77(6):1046–1053.
123. Kim JH, Viana MA, Graber TM, et al. The effectiveness of protraction face mask therapy: a meta-analysis. *Am J Orthod Dentofacial Orthop*. 1999;115(6):675–685.
124. Mucedero M, Baccetti T, Franchi L, Cozza P. Effects of maxillary protraction with or without expansion on the sagittal pharyngeal dimensions in Class III subjects. *Am J Orthod Dentofacial Orthop*. 2009;135(6):777–781.
125. Arnett GW, Gunson MJ. Esthetic treatment planning for orthognathic surgery. *J Clin Orthod*. 2010;44(3):196–200.
126. Athanasiou AE, Toutountzakis N, Mavreas D, et al. Alterations of hyoid bone position and pharyngeal depth and their relationship after surgical correction of mandibular prognathism. *Am J Orthod Dentofacial Orthop*. 1991;100(3):259–265.
127. Kawamata A, Fujishita M, Ariji Y, Ariji E. Three-dimensional computed tomographic evaluation of morphologic airway changes after mandibular setback osteotomy for prognathism. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2000;89(3):278–287.
128. Enacar A, Aksoy AU, Sencift Y, et al. Changes in hypopharyngeal airway space and in tongue and hyoid bone positions following the surgical correction of mandibular prognathism. *Int J Adult Orthodon Orthognath Surg*. 1994;9(4):285–290.
129. Achilleos S, Krogstad O, Lyberg T. Surgical mandibular advancement and changes in uvuloglossopharyngeal morphology and head posture: a short- and long-term cephalometric study in males. *Eur J Orthod*. 2000;22(4):367–381.
130. Eggensperger N, Smolka K, Johner A, et al. Long-term changes of hyoid bone and pharyngeal airway size following advancement of the mandible. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2005;99(4):404–410.

131. Turnbull NR, Battagel JM. The effects of orthognathic surgery on pharyngeal airway dimensions and quality of sleep. *J Orthod*. 2000;27(3):235–247.
132. Greco JM, Frohberg U, Van Sickels JE. Cephalometric analysis of long-term airway space changes with maxillary osteotomies. *Oral Surg Oral Med Oral Pathol*. 1990;70(5):552–554.
133. Gokce SM, Gorgulu S, Gokce HS, et al. Changes in posterior airway space, pulmonary function and sleep quality, following bimaxillary orthognathic surgery. *Int J Oral Maxillofac Surg*. 2012;41(7):820–829.
134. Gokce SM, Gorgulu S, Gokce HS, et al. Evaluation of pharyngeal airway space changes after bimaxillary orthognathic surgery with a 3-dimensional simulation and modeling program. *Am J Orthod Dentofacial Orthop*. 2014;146(4):477–492.
135. Jakobsone G, Stenvik A, Espeland L. The effect of maxillary advancement and impaction on the upper airway after bimaxillary surgery to correct Class III malocclusion. *Am J Orthod Dentofacial Orthop*. 2011;139(4 Suppl):e369–e376.
136. Mattos CT, Vilani GN, Sant'Anna EF, et al. Effects of orthognathic surgery on oropharyngeal airway: a meta-analysis. *Int J Oral Maxillofac Surg*. 2011;40(12):1347–1356.
137. Prinsell JR. Maxillomandibular advancement surgery in a site-specific treatment approach for obstructive sleep apnea in 50 consecutive patients. *Chest*. 1999;116(6):1519–1529.
138. El AS, El H, Palomo JM, Baur DA. A 3-dimensional airway analysis of an obstructive sleep apnea surgical correction with cone beam computed tomography. *J Oral Maxillofac Surg*. 2011;69(9):2424–2436.
139. Rachmiel A, Aizenbud D, Pillar G, et al. Bilateral mandibular distraction for patients with compromised airway analyzed by three-dimensional CT. *Int J Oral Maxillofac Surg*. 2005;34(1):9–18.
140. Xu H, Yu Z, Mu X. The assessment of midface distraction osteogenesis in treatment of upper airway obstruction. *J Craniofac Surg*. 2009;20(Suppl 2):1876–1881.
141. Perkins JA, Sie KC, Milczuk H, Richardson MA. Airway management in children with craniofacial anomalies. *Cleft Palate Craniofac J*. 1997;34(2):135–140.
142. Sculerati N, Gottlieb MD, Zimbler MS, et al. Airway management in children with major craniofacial anomalies. *Laryngoscope*. 1998;108(12):1806–1812.
143. Boston M, Rutter MJ. Current airway management in craniofacial anomalies. *Curr Opin Otolaryngol Head Neck Surg*. 2003;11(6):428–432.
144. Gosain AK, Santoro TD, Havlik RJ, et al. Midface distraction following Le Fort III and monobloc osteotomies: problems and solutions. *Plast Reconstr Surg*. 2002;109(6):1797–1808.
145. Kubler AC, Speder B, Zoller JE. Fronto-orbital advancement with simultaneous LeFort III-distraction. *J Craniomaxillofac Surg*. 2004;32(5):291–295.
146. Pelo S, Gasparini G, Di Petrillo A, et al. Distraction osteogenesis in the surgical treatment of craniostenosis: a comparison of internal and external craniofacial distractor devices. *Childs Nerv Syst*. 2007;23(12):1447–1453.
147. Morovic CG, Monasterio L. Distraction osteogenesis for obstructive apneas in patients with congenital craniofacial malformations. *Plast Reconstr Surg*. 2000;105(7):2324–2330.
148. Perlyn CA, Schmelzer RE, Sutera SP, et al. Effect of distraction osteogenesis of the mandible on upper airway volume and resistance in children with micrognathia. *Plast Reconstr Surg*. 2002;109(6):1809–1818.
149. Cohen SR, Simms C, Burstein FD. Mandibular distraction osteogenesis in the treatment of upper airway obstruction in children with craniofacial deformities. *Plast Reconstr Surg*. 1998;101(2):312–318.
150. Young T, Peppard PE, Gottlieb DJ. Epidemiology of obstructive sleep apnea: a population health perspective. *Am J Respir Crit Care Med*. 2002;165(9):1217–1239.
151. Carlson JT, Hedner JA, Ejnell H, Peterson LE. High prevalence of hypertension in sleep apnea patients independent of obesity. *Am J Respir Crit Care Med*. 1994;150(1):72–77.
152. Ngiam J, Balasubramaniam R, Darendeliler MA, et al. Clinical guidelines for oral appliance therapy in the treatment of snoring and obstructive sleep apnoea. *Aust Dent J*. 2013;58(4):408–419.
153. Berry RB, Budhiraja R, Gottlieb DJ, et al. Rules for scoring respiratory events in sleep: update of the 2007 AASM Manual for the Scoring of Sleep and Associated Events. Deliberations of the Sleep Apnea Definitions Task Force of the American Academy of Sleep Medicine. *J Clin Sleep Med*. 2012;8(5):597–619.
154. O'Connor GT, Caffo B, Newman AB, et al. Prospective study of sleep-disordered breathing and hypertension: the Sleep Heart Health Study. *Am J Respir Crit Care Med*. 2009;179(12):1159–1164.
155. Punjabi NM, Caffo BS, Goodwin JL, et al. Sleep-disordered breathing and mortality: a prospective cohort study. *PLoS Med*. 2009;6(8):e1000132.
156. Redline S, Yenokyan G, Gottlieb DJ, et al. Obstructive sleep apnea-hypopnea and incident stroke: the sleep heart health study. *Am J Respir Crit Care Med*. 2010;182(2):269–277.
157. Ruehland WR, Rochford PD, O'Donoghue FJ, et al. The new AASM criteria for scoring hypopneas: impact on the apnea hypopnea index. *Sleep*. 2009;32(2):150–157.
158. Punjabi NM, Polotsky VY. Disorders of glucose metabolism in sleep apnea. *J Appl Physiol (1985)*. 2005;99(5):1998–2007.
159. Monahan K, Storfer-Isser A, Mehra R, et al. Triggering of nocturnal arrhythmias by sleep-disordered breathing events. *J Am Coll Cardiol*. 2009;54(19):1797–1804.
160. Strohl KP, Redline S. Recognition of obstructive sleep apnea. *Am J Respir Crit Care Med*. 1996;154(2 Pt 1):279–289.
161. American Academy of Sleep Medicine. *International Classification of Sleep Disorders: Diagnostic & Coding Manual*. 2nd ed. Westchester, MN: American Academy of Sleep Medicine; 2005.
162. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. The Report of an American Academy of Sleep Medicine Task Force. *Sleep*. 1999;22(5):667–689.
163. Pack AI. Obstructive sleep apnea. *Adv Intern Med*. 1994;39:517–567.
164. Young T, Palta M, Dempsey J, et al. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med*. 1993;328(17):1230–1235.
165. Namen AM, Dunagan DP, Fleischer A, et al. Increased physician-reported sleep apnea: the National Ambulatory Medical Care Survey. *Chest*. 2002;121(6):1741–1747.
166. Patino M, Sadhasivam S, Mahmoud M. Obstructive sleep apnoea in children: perioperative considerations. *Br J Anaesth*. 2013;111(Suppl 1):i83–i95.
167. Wise MS, Nichols CD, Grigg-Damberger MM, et al. Executive summary of respiratory indications for polysomnography in children: an evidence-based review. *Sleep*. 2011;34(3):389–398. AW.
168. Lamm J, Poeschel J, Smith S. Obtaining a thorough sleep history and routinely screening for obstructive sleep apnea. *J Am Acad Nurse Pract*. 2008;20(4):225–229.
169. Balserak BI. Sleep-disordered breathing in pregnancy. *Am J Respir Crit Care Med*. 2014;190(4):P1–P2.
170. Dempsey JA, Veasey SC, Morgan BJ, O'Donnell CP. Pathophysiology of sleep apnea. *Physiol Rev*. 2010;90(1):47–112.
171. Decker M, Yamauchi M, Strohl KP. Keep the airway open and let the brain sleep. *Am J Respir Crit Care Med*. 2014;190(11):1207–1209.
172. Longobardo GS, Evangelisti CJ, Cherniack NS. Analysis of the interplay between neurochemical control of respiration and upper airway mechanics producing upper airway obstruction during sleep in humans. *Exp Physiol*. 2008;93(2):271–287.
173. Longobardo GS, Evangelisti CJ, Cherniack NS. Influence of arousal threshold and depth of sleep on respiratory stability in man: analysis using a mathematical model. *Exp Physiol*. 2009;94(12):1185–1199.
174. Patil SP, Schneider H, Schwartz AR, Smith PL. Adult obstructive sleep apnea: pathophysiology and diagnosis. *Chest*. 2007;132(1):325–337.
175. Epstein LJ, Kristo D, Strollo Jr PJ, et al. Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. *J Clin Sleep Med*. 2009;5(3):263–276.
176. Jauhar S, Orchardson R, Banham SW, et al. The Kushida Index as a screening tool for obstructive sleep apnoea-hypopnoea syndrome. *Br Dent J*. 2012;212(1):E2.
177. Kushida CA, Littner MR, Morgenthaler T, et al. Practice parameters for the indications for polysomnography and related procedures: an update for 2005. *Sleep*. 2005;28(4):499–521.

178. Ramachandran SK, Kheterpal S, Consens F, et al. Derivation and validation of a simple perioperative sleep apnea prediction score. *Anesth Analg*. 2010;110(4):1007–1015.
179. Friedman M, Tanyeri H, La Rosa M, et al. Clinical predictors of obstructive sleep apnea. *Laryngoscope*. 1999;109(12):1901–1907.
180. Friedman M, Ibrahim H, Joseph NJ. Staging of obstructive sleep apnea/hypopnea syndrome: a guide to appropriate treatment. *Laryngoscope*. 2004;114(3):454–459.
181. Mallampati SR. Clinical sign to predict difficult tracheal intubation (hypothesis). *Can Anaesth Soc J*. 1983;30(3 Pt 1):316–317.
182. Mallampati SR, Gatt SP, Gugino LD, et al. A clinical sign to predict difficult tracheal intubation: a prospective study. *Can Anaesth Soc J*. 1985;32(4):429–434.
183. Samsoon GL, Young JR. Difficult tracheal intubation: a retrospective study. *Anaesthesia*. 1987;42(5):487–490.
184. Nuckton TJ, Glidden DV, Browner WS, Claman DM. Physical examination: Mallampati score as an independent predictor of obstructive sleep apnea. *Sleep*. 2006;29(7):903–908.
185. Islam S, Selbong U, Taylor CJ, Ormiston IW. Does a patient's Mallampati score predict outcome after maxillomandibular advancement for obstructive sleep apnoea? *Br J Oral Maxillofac Surg*. 2015;53(1):23–27.
186. Kinsella SM, Robinson SM. Increase in Mallampati score during pregnancy. *Br J Anaesth*. 1995;75(6):822.
187. Khatiwada S, Bhattarai B, Pokharel K, et al. Comparison of Modified Mallampati Test between sitting and supine positions for prediction of difficult intubation. *Health Renaissance*. 2012;10(1):12–15.
188. Kushida CA, Efron B, Guilleminault C. A predictive morphometric model for the obstructive sleep apnea syndrome. *Ann Intern Med*. 1997;127(8 Pt 1):581–587.
189. Jung DG, Cho HY, Grunstein RR, Yee B. Predictive value of Kushida index and acoustic pharyngometry for the evaluation of upper airway in subjects with or without obstructive sleep apnea. *J Korean Med Sci*. 2004;19(5):662–667.
190. Netzer NC, Stoohs RA, Netzer CM, et al. Using the Berlin Questionnaire to identify patients at risk for the sleep apnea syndrome. *Ann Intern Med*. 1999;131(7):485–491.
191. Chung F, Yegneswaran B, Liao P, et al. STOP questionnaire: a tool to screen patients for obstructive sleep apnea. *Anesthesiology*. 2008;108(5):812–821.
192. Luo J, Huang R, Zhong X, et al. STOP-Bang questionnaire is superior to Epworth sleepiness scales, Berlin questionnaire, and STOP questionnaire in screening obstructive sleep apnea hypopnea syndrome patients. *Chin Med J (Engl)*. 2014;127(17):3065–3070.
193. Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. *Sleep*. 1991;14(6):540–545.
194. Sil A, Barr G. Assessment of predictive ability of Epworth scoring in screening of patients with sleep apnoea. *J Laryngol Otol*. 2012;126(4):372–379.
195. McNamara Jr JA. A method of cephalometric evaluation. *Am J Orthod*. 1984;86(6):449–469.
196. Douglass AB, Bornstein R, Nino-Murcia G, et al. The Sleep Disorders Questionnaire. I: Creation and multivariate structure of SDQ. *Sleep*. 1994;17(2):160–167.
197. Weatherwax KJ, Lin X, Marzec ML, Malow BA. Obstructive sleep apnea in epilepsy patients: the Sleep Apnea scale of the Sleep Disorders Questionnaire (SA-SDQ) is a useful screening instrument for obstructive sleep apnea in a disease-specific population. *Sleep Med*. 2003;4(6):517–521.
198. Balachandran JS, Patel SR. In the clinic. Obstructive sleep apnea. *Ann Intern Med*. 2014;161(9). ITC1–ITC15; quiz ITC16.
199. Chwiesko-Minarowska S, Minarowski L, Kuryliszyn-Moskal A, et al. Rehabilitation of patients with obstructive sleep apnea syndrome. *Int J Rehabil Res*. 2013;36(4):291–297.
200. Pliska BT, Almeida F. Effectiveness and outcome of oral appliance therapy. *Dent Clin North Am*. 2012;56(2):433–444.
201. Katyal V, Kennedy D, Martin J, et al. Paediatric sleep-disordered breathing due to upper airway obstruction in the orthodontic setting: a review. *Aust Orthod J*. 2013;29(2):184–192.
202. Stuck BA, Maurer JT. Airway evaluation in obstructive sleep apnea. *Sleep Med Rev*. 2008;12(6):411–436.
203. Schwartz AR, Smith PL, Oliven A. Electrical stimulation of the hypoglossal nerve: a potential therapy. *J Appl Physiol (1985)*. 2014;116(3):337–344.
204. Morgenthaler TI, Kapen S, Lee-Chiong T, et al. Practice parameters for the medical therapy of obstructive sleep apnea. *Sleep*. 2006;29(8):1031–1035.
205. Pevernagie DA, Stanson AW, Sheedy 2nd PF, et al. Effects of body position on the upper airway of patients with obstructive sleep apnea. *Am J Respir Crit Care Med*. 1995;152(1):179–185.
206. Gay P, Weaver T, Loube D, et al. Evaluation of positive airway pressure treatment for sleep related breathing disorders in adults. *Sleep*. 2006;29(3):381–401.
207. Yetkin O, Kunter E, Gunen H. CPAP compliance in patients with obstructive sleep apnea syndrome. *Sleep Breath*. 2008;12(4):365–367.
208. Weaver TE, Grunstein RR. Adherence to continuous positive airway pressure therapy: the challenge to effective treatment. *Proc Am Thorac Soc*. 2008;5(2):173–178.
209. Gotsopoulos H, Chen C, Qian J, Cistulli PA. Oral appliance therapy improves symptoms in obstructive sleep apnea: a randomized, controlled trial. *Am J Respir Crit Care Med*. 2002;166(5):743–748.
210. Mehta A, Qian J, Petocz P, et al. A randomized, controlled study of a mandibular advancement splint for obstructive sleep apnea. *Am J Respir Crit Care Med*. 2001;163(6):1457–1461.
211. Petri N, Svanholt P, Solow B, et al. Mandibular advancement appliance for obstructive sleep apnoea: results of a randomised placebo controlled trial using parallel group design. *J Sleep Res*. 2008;17(2):221–229.
212. Walker-Engström ML, Ringqvist I, Vestling O, et al. A prospective randomized study comparing two different degrees of mandibular advancement with a dental appliance in treatment of severe obstructive sleep apnea. *Sleep Breath*. 2003;7(3):119–130.
213. Lam B, Sam K, Lam JC, et al. The efficacy of oral appliances in the treatment of severe obstructive sleep apnea. *Sleep Breath*. 2011;15(2):195–201.
214. Petit FX, Pépin JL, Bettega G, et al. Mandibular advancement devices: rate of contraindications in 100 consecutive obstructive sleep apnea patients. *Am J Respir Crit Care Med*. 2002;166(3):274–278.
215. Hoffstein V. Review of oral appliances for treatment of sleep-disordered breathing. *Sleep Breath*. 2007;11(1):1–22.
216. Marklund M, Stenlund H, Franklin KA. Mandibular advancement devices in 630 men and women with obstructive sleep apnea and snoring: tolerability and predictors of treatment success. *Chest*. 2004;125(4):1270–1278.
217. Lettieri CJ, Paolino N, Eliasson AH, et al. Comparison of adjustable and fixed oral appliances for the treatment of obstructive sleep apnea. *J Clin Sleep Med*. 2011;7(5):439–445.
218. Jauhar S, Lyons MF, Banham SW, et al. Ten-year follow-up of mandibular advancement devices for the management of snoring and sleep apnea. *J Prosthet Dent*. 2008;99(4):314–321.
219. Liu Y, Lowe AA, Fleetham JA, Park YC. Cephalometric and physiologic predictors of the efficacy of an adjustable oral appliance for treating obstructive sleep apnea. *Am J Orthod Dentofacial Orthop*. 2001;120(6):639–647.
220. Ng AT, Darendeliler MA, Petocz P, Cistulli PA. Cephalometry and prediction of oral appliance treatment outcome. *Sleep Breath*. 2012;16(1):47–58.
221. Gotsopoulos H, Kelly JJ, Cistulli PA. Oral appliance therapy reduces blood pressure in obstructive sleep apnea: a randomized, controlled trial. *Sleep*. 2004;27(5):934–941.
222. Sutherland K, Cistulli P. Mandibular advancement splints for the treatment of sleep apnea syndrome. *Swiss Med Wkly*. 2011;141. w13276.
223. Ferguson KA, Ono T, Lowe AA, et al. A short-term controlled trial of an adjustable oral appliance for the treatment of mild to moderate obstructive sleep apnoea. *Thorax*. 1997;52(4):362–368.
224. Annapurna K, Suganya S, Vasanth R, Kumar PR. Prosthodontic approach to treat obstructive sleep apnea. *Ann Med Health Sci Res*. 2014;4(4):481–486.
225. Barthlen GM, Brown LK, Wiland MR, et al. Comparison of three oral appliances for treatment of severe obstructive sleep apnea syndrome. *Sleep Med*. 2000;1(4):299–305.
226. Chan AS, Lee RW, Srinivasan VK, et al. Nasopharyngoscopic evaluation of oral appliance therapy for obstructive sleep apnoea. *Eur Respir J*. 2010;35(4):836–842.

227. Chan AS, Sutherland K, Schwab RJ, et al. The effect of mandibular advancement on upper airway structure in obstructive sleep apnoea. *Thorax*. 2010;65(8):726–732.
228. Tsuiki S, Ono T, Kuroda T. mandibular advancement modulates respiratory-related genioglossus electromyographic activity. *Sleep Breath*. 2000;4(2):53–58.
229. Johal A, Gill G, Ferman A, McLaughlin K. The effect of mandibular advancement appliances on awake upper airway and masticatory muscle activity in patients with obstructive sleep apnoea. *Clin Physiol Funct Imaging*. 2007;27(1):47–53.
230. Hoekema A, de Vries F, Heydenrijk K, Stegenga B. Implant-retained oral appliances: a novel treatment for edentulous patients with obstructive sleep apnea-hypopnea syndrome. *Clin Oral Implants Res*. 2007;18(3):383–387.
231. de Carlos F, Cobo J, Fernandez Mondragon MP, et al. Orthoimplants: an alternative treatment for SAHS? *Sleep Breath*. 2010;14(2):171–174.
232. Ngiam J, Kyung HM. Microimplant-based mandibular advancement therapy for the treatment of snoring and obstructive sleep apnea: a prospective study. *Angle Orthod*. 2012;82(6):978–984.
233. de Almeida FR, Lowe AA, Tsuiki S, et al. Long-term compliance and side effects of oral appliances used for the treatment of snoring and obstructive sleep apnea syndrome. *J Clin Sleep Med*. 2005;1(2):143–152.
234. Martinez-Gomis J, Willaert E, Nogues L, et al. Five years of sleep apnea treatment with a mandibular advancement device. Side effects and technical complications. *Angle Orthod*. 2010;80(1):30–36.
235. Chen H, Lowe AA, de Almeida FR, et al. Three-dimensional com puter-assisted study model analysis of long-term oral-appliance wear. Part 2. Side effects of oral appliances in obstructive sleep apnea patients. *Am J Orthod Dentofacial Orthop*. 2008;134(3):408–417.
236. American Academy of Dental Sleep Medicine. *Policy Statement on the Diagnosis and Treatment of OSA*. 2012. [Cited March 15, 2015]. Available from: http://www.aadsm.org/osapolicystatement.aspx.
237. Kribbs NB, Pack AI, Kline LR, et al. Objective measurement of patterns of nasal CPAP use by patients with obstructive sleep apnea. *Am Rev Respir Dis*. 1993;147(4):887–895.
238. American Academy of Otolaryngology-Head and Neck Surgery. *Position Statement: Treatment of Obstructive Sleep Apnea*. 2012. [Cited March 11, 2015]. Available from http://www.entnet.org/node/549.
239. American Association of Oral and Maxillofacial Surgeons. *Evaluation and Management of Obstructive Sleep Apnea—Overview*. 2013. [Cited March 11, 2105]. Available from http://www.aaoms.org/practice-resources/aaoms-advocacy-and-position-statements/white-papers.
240. Brietzke SE, Gallagher D. The effectiveness of tonsillectomy and adenoidectomy in the treatment of pediatric obstructive sleep apnea/hypopnea syndrome: a meta-analysis. *Otolaryngol Head Neck Surg*. 2006;134(6):979–984.
241. Sugiura T, Noda A, Nakata S, et al. Influence of nasal resistance on initial acceptance of continuous positive airway pressure in treatment for obstructive sleep apnea syndrome. *Respiration*. 2007;74(1):56–60.
242. Nakata S, Noda A, Yasuma F, et al. Effects of nasal surgery on sleep quality in obstructive sleep apnea syndrome with nasal obstruction. *Am J Rhinol*. 2008;22(1):59–63.
243. Friedman M, Tanyeri H, Lim JW, et al. Effect of improved nasal breathing on obstructive sleep apnea. *Otolaryngol Head Neck Surg*. 2000;122(1):71–74.
244. Friedman M, Maley A, Kelley K, et al. Impact of nasal obstruction on obstructive sleep apnea. *Otolaryngol Head Neck Surg*. 2011;144(6):1000–1004.
245. Aurora RN, Casey KR, Kristo D, et al. Practice parameters for the surgical modifications of the upper airway for obstructive sleep apnea in adults. *Sleep*. 2010;33(10):1408–1413.
246. SAGES Society of American Gastrointestinal and Endoscopic Surgeons. *Guidelines for Clinical Application of Laparoscopic Bariatric Surgery*. 2008. [Cited March 2015]. Available from: http://www.sages.org/publications/guidelines/guidelines-for-clinical-application-of-laparoscopic-bariatric-surgery/.
247. De Dios JA, Brass SD. New and unconventional treatments for obstructive sleep apnea. *Neurotherapeutics*. 2012;9(4):702–709.
248. Guimarães KC, Drager LF, Genta PR, et al. Effects of oropharyngeal exercises on patients with moderate obstructive sleep apnea syndrome. *Am J Respir Crit Care Med*. 2009;179(10):962–966.
249. Villa MP, Brasili L, Ferretti A, et al. Oropharyngeal exercises to reduce symptoms of OSA after AT. *Sleep Breath*. 2015;19(1):281–289.
250. Ojay A, Ernst E. Can singing exercises reduce snoring? A pilot study. *Complement Ther Med*. 2000;8(3):151–156.
251. Puhan MA, Suarez A, Lo Cascio C, et al. Didgeridoo playing as alternative treatment for obstructive sleep apnoea syndrome: randomised controlled trial. *BMJ*. 2006;332(7536):266–270.
252. Brown DL, Zahuranec DB, Majersik JJ, et al. Risk of sleep apnea in orchestra members. *Sleep Med*. 2009;10(6):657–660.
253. Strollo Jr PJ, Soose RJ, Maurer JT, et al. Upper-airway stimulation for obstructive sleep apnea. *N Engl J Med*. 2014;370(2):139–149.
254. Strohl KP, Brown DB, Collop N, et al. An official American Thoracic Society Clinical Practice Guideline: sleep apnea, sleepiness, and driving risk in noncommercial drivers. An update of a 1994 statement. *Am J Respir Crit Care Med*. 2013;187(11):1259–1266.
255. Gottlieb DJ, Yenokyan G, Newman AB, et al. Prospective study of obstructive sleep apnea and incident coronary heart disease and heart failure: the Sleep Heart Health Study. *Circulation*. 2010;122(4):352–360.
256. Bosch M, Faber MJ, Cruijsberg J, et al. Review article: Effectiveness of patient care teams and the role of clinical expertise and coordination: a literature review. *Med Care Res Rev*. 2009;66(6 Suppl):5S–35S.

13

Orthodontic Therapy and the Patient with Temporomandibular Disorder

Jeffrey P. Okeson

OUTLINE

Introduction, 353
The Concept of Orthopedic Stability, 353
Finding the Musculoskeletal Stable Position, 355
Evaluating the Patient for Temporomandibular Disorders, 357
A TMD Screen History, 358
A TMD Screen Examination, 358
Muscle Palpation, 358
Temporomandibular Joint Palpation, 358
Range of Mandibular Movement, 360
Occlusal Evaluation, 360
Developing the Orthodontic/TMD Treatment Plan, 362
Managing TMD Symptoms That Arise During Orthodontic Therapy, 363
Summary, 365

INTRODUCTION

There are two main goals of orthodontic therapy. The first, and often the most important to the patient, is improving aesthetics. Although aesthetics may be the primary goal of the patient (and parent), it is certainly not the most important goal. The second goal, which is achieving sound masticatory function, is actually the most important treatment goal of orthodontic therapy. Developing a healthy, orthopedically stable masticatory system enables sound function, which is essential for the lifetime of the patient. No other dental specialist routinely alters the patient's occlusal condition as a part of the therapy. The orthodontist is in a unique position to either improve or worsen the occlusal condition while carrying out the aesthetic goals of the therapy. It therefore behooves the orthodontists to be knowledgeable of normal masticatory function and the goals that need to be achieved to maintain normal function. These goals should be met in all patients, both those with and without masticatory dysfunction.

This chapter will first discuss the principles of normal orthopedic stability in the masticatory system and define treatment goals that will help assure normal masticatory function. Next, concepts of how orthopedic instability may relate to temporomandibular disorders (TMDs) will be reviewed, and also when orthodontic therapy may influence TMD symptoms. A brief TMD history and examination will be presented so that important symptoms will be identified before orthodontic therapy is begun. Lastly, this chapter will provide some clinical considerations regarding the management of TMD symptoms that may arise during the orthodontic therapy.

THE CONCEPT OF ORTHOPEDIC STABILITY

For many years, the dental profession has debated the relationship between occlusion and TMDs. We have often concentrated on the precise contact pattern of the teeth and perhaps overlooked the more significant aspect of orthopedic stability. If occlusion is important to masticatory function, it must relate to how the occlusal contact pattern of the teeth relates to orthopedic stability of the entire masticatory system. In other words, the clinician needs to better understand sound orthopedic principles to more completely appreciate the importance of occlusion and its role on function or dysfunction of the masticatory system.

In establishing the criteria for the optimum, orthopedically stable joint position, the anatomic structures of the temporomandibular joint (TMJ) must be closely examined. The TMJ is made up of the condyle resting within the articular fossa with the articular disc interposed. The articular disc is composed of dense, fibrous connective tissue devoid of nerves and blood vessels.[1] This allows it to withstand heavy forces without damage or creating a painful stimulus. The purpose of the disc is to separate, protect, and stabilize the condyle in the mandibular fossa during functional movements. The articular disc, however, does not determine positional stability of the joint. As in any other joint, positional stability is determined by the muscles that pull across the joint and prevent separation of the articular surfaces. The directional forces of these muscles determine the optimum, orthopedically stable joint position. This is an orthopedic principle that is common to all mobile joints. It might be stated that every mobile joint has a *musculoskeletally stable (MS) position*, that being, the position stabilized by the activity of muscles that pull across it. The MS position is the most orthopedically stable position for the joint and can be identified by observing the directional forces applied by the stabilizing muscles.

The major muscles that stabilize the TMJs are the elevators. The direction of the force placed on the condyles by the temporalis muscles is predominantly in a superior. The temporalis muscles have some fibers that are oriented horizontally; however, because these fibers must transverse around the root of the zygomatic arch, the majority of fibers elevate the condyles in a straight superior direction (Fig. 13-1, *A*).[2] The masseter and medial pterygoid muscles provide forces in a superoanterior direction, which seats the condyles superiorly and anteriorly against the posterior slopes of the articular eminences (Fig. 13-1, *B*). These three muscle groups are primarily responsible

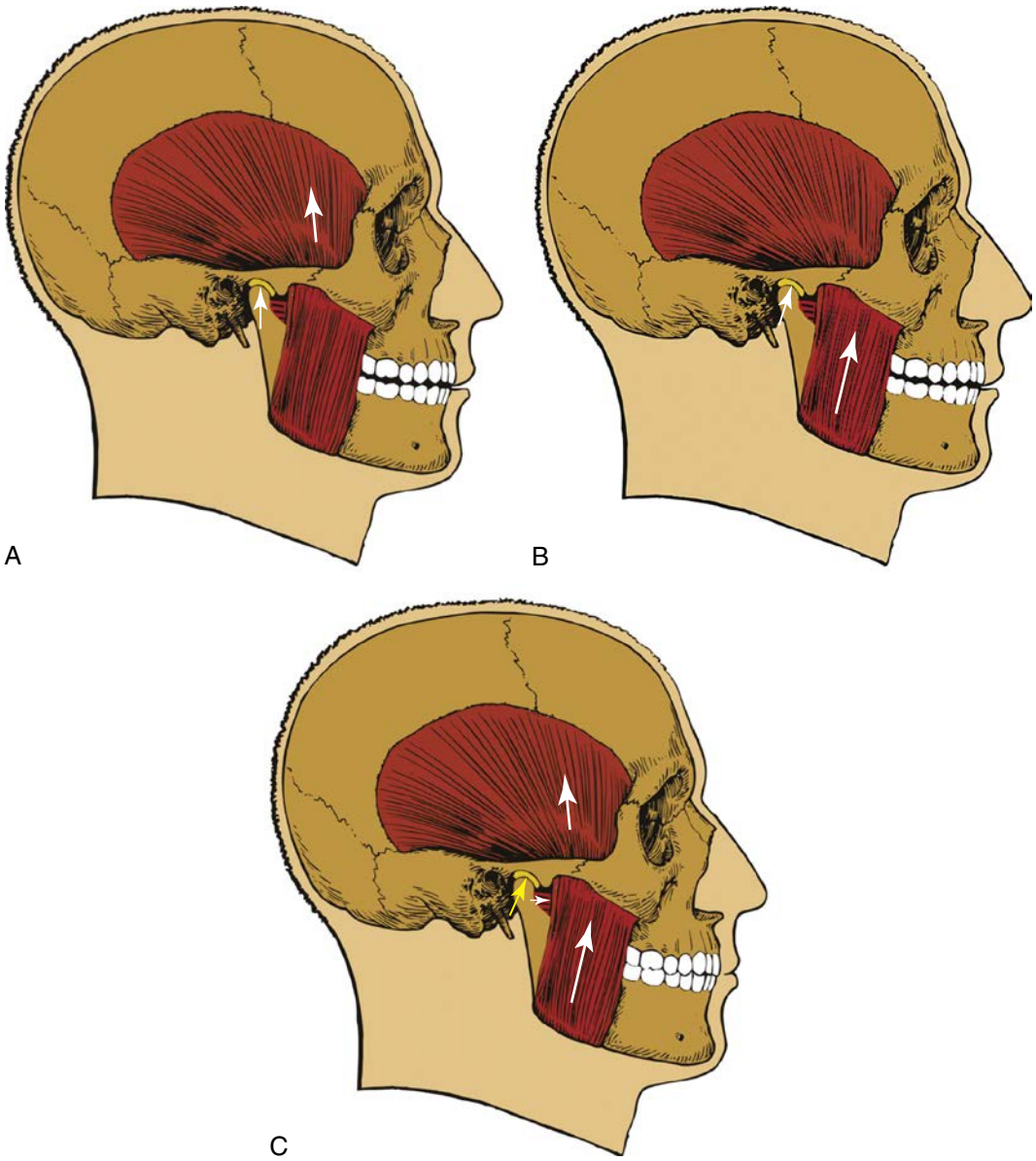


FIGURE 13-1 A, The directional forces applied to the condyles by the temporal muscles are to seat the condyles in a superior position in the fossae (*white arrow*). B, The directional forces applied to the condyles by the masseter and medial pterygoid muscles are to seat the condyles in a superior–anterior position in the fossae (*white arrow*). C, When these forces are combined with the lateral pterygoid muscle (note shown), the condyles are seated into their superoanterior position in the fossae against the posterior slopes of the articular eminences.

for joint position and stability; however, the lateral pterygoid muscles also contribute to joint stability by stabilizing the condyles against the posterior slopes of the articular eminences (Fig. 13-1, *C*).

In the postural position, without any influence from the occlusal condition, the condyles are stabilized by muscle tonus of the elevator and the inferior lateral-pterygoid muscles. The temporalis muscles position the condyles superiorly in the fossae. The masseter and medial pterygoid muscles position the condyles superoanteriorly. Tonus in the inferior lateral pterygoid muscles positions the condyles anteriorly against the posterior slopes of the articular eminences.

Therefore, the most orthopedically stable joint position as dictated by the muscles is described to be when the condyles are located in their most superoanterior position in the articular fossae, resting against the posterior slopes of the articular eminences. This description is not complete, however, until the positions of the articular discs are considered. Optimum joint relationship is achieved only when the articular discs are properly interposed between the condyles and the articular fossae. Therefore, the complete definition of the most orthopedically stable joint position is that position when the condyles are in their most superoanterior position in the articular fossae, resting against the posterior slopes of the articular eminences, with the articular discs properly interposed. This position is the *most MS position* of the mandible.

The most orthopedically stable position just described does not consider the stabilizing effects of the structures at the other

end of the mandible, namely the teeth. The occlusal contact pattern of the teeth also influences stability of the masticatory system. It is important that when the condyles are in their most stable position in the fossae and the mouth is closed, the teeth occlude in their most stable relationship. The most stable occlusal position is the maximal intercuspation of the teeth. This type of occlusal relationship furnishes maximum stability for the mandible while minimizing the amount of force placed on each tooth during function.

In summary, the criteria for optimum orthopedic stability in the masticatory system would be to have even and simultaneous contact of all possible teeth when the mandibular condyles are in their most superoanterior position, resting against the posterior slopes of the articular eminences, with the discs properly interposed. In other words, the MS position of the condyles coincides with the maximum intercuspal position of the teeth.

One additional consideration in describing the occlusal condition is the fact that the mandible has the ability to move eccentrically, resulting in tooth contacts. These lateral excursions allow horizontal forces to be applied to the teeth, and horizontal forces are not generally well accepted by the dental supportive structures; yet the complexity of the joints requires that some teeth bear the burden of these less-tolerated forces. When all the teeth are examined, it becomes apparent that the anterior teeth are better candidates to accept these horizontal forces than posterior teeth because they are further from the force vectors, which results in less force to these teeth. Of all the anterior teeth, the canines are the best suited to accept the horizontal forces that occur during eccentric movements.[3-5] They have the longest and largest roots and therefore the best crown/root ratio.[6] They are also surrounded by dense, compact bone, which tolerates the forces better than does the medullary bone found around posterior teeth.[7]

The laterotrusive contacts need to provide adequate guidance to immediately disocclude the teeth on the opposite side of the arch (mediotrusive or nonworking side). When the mandible moves forward into protrusive contact, the anterior teeth should also provide adequate contact or guidance to disarticulate the posterior teeth.

The following is a summary of the conditions that provide optimum orthopedic stability in the masticatory system. This represents the orthodontic treatment goals for all patients.

1. When the mouth closes, the condyles should be in their most superoanterior position (MS), resting on the posterior slopes of the articular eminences with the discs properly interposed. In this position, there should be even and simultaneous contact of all posterior teeth. The anterior teeth may also contact, but more lightly than the posterior teeth.
2. When the mandible moves into laterotrusive positions, there should be adequate tooth-guided contacts on the laterotrusive (working) side to immediately disocclude the mediotrusive (nonworking) side. The canines (canine guidance) provide the most desirable guidance.
3. When the mandible moves into a protrusive position, there should be adequate tooth-guided contacts on the anterior teeth to immediately disocclude all posterior teeth.
4. When the patient sits upright (in the alert feeding position)[8] and is asked to bring the posterior teeth into contact, the posterior tooth contacts should be heavier than anterior tooth contacts.

FINDING THE MUSCULOSKELETAL STABLE POSITION

Now that the orthopedic treatment goals for all orthodontic therapy have been described, the next question that needs to be asked is, "How can I locate the MS position of the condyles in the fossae?" In order for these treatment goals to be useful, the clinician must be able to repeatedly and reliably locate this treatment position. An easy and effective method of locating the MS position is the bilateral manual-manipulation technique.[9,10] This technique begins with the patient lying back and the chin pointed upward (Fig. 13-2, *A*). Lifting the chin upward places the head in an easier position to locate the condyles near the MS position. The dentist sits behind the patient and places the four fingers of each hand on the lower border of the mandible at the angle. The small finger should be behind the angle with the remaining fingers on the inferior border of the mandible. It is important that the fingers be located on the bone and not in the soft tissues of the neck (Fig. 13-2, *B*). Next, both thumbs are placed over the symphysis of the chin so they touch each other between the patient's lower lip and chin (Fig. 13-2, *C*). When the hands are in this position, the mandible is guided by upward force placed on its lower border and angle with the fingers while at the same time the thumbs press downward and backward on the chin. The overall force on the mandible is directed so the condyles will be seated in their most superoanterior position braced against the posterior slopes of the eminences (Fig. 13-2, *D*). Firm but gentle force is needed to guide the mandible so as not to elicit any protective reflexes.

Locating the MS position begins with the anterior teeth no more than 10 mm apart to ensure that the temporomandibular ligaments have not forced translation of the condyles. The mandible is positioned with a gentle arcing until it freely rotates around the MS position. This arcing consists of short movements of 2 to 4 mm. Once the mandible is rotating around the musculoskeletally stable position, force is firmly applied by the fingers to seat the condyles in their most superoanterior position.

In this superoanterior position, the condyle–disc complexes are in proper relation to accept forces. When such a relationship exists, guiding the mandible to this position should not produce pain. If pain is elicited, it is possible that some type of intracapsular disorder exists. When a pain condition exists, an accurate mandibular position will not likely be found. Therefore, the reason for this pain needs to be investigated and managed before any orthodontic therapy is begun.

Another method of finding the MS position is by using the muscles themselves to seat the condyles. This can be accomplished with a leaf gauge (Fig. 13-3, *A* and *B*).[10,11] The concept behind a leaf gauge is that when only the anterior teeth occlude (disengaging the posterior teeth), the directional force provided by the elevator muscles (temporalis, masseter, medial pterygoid) seats the condyles in a superoanterior position within the fossae. The anterior stop provided by the leaf gauge acts as a fulcrum, allowing the condyles to be pivoted to the MS position in the fossae. A leaf gauge must be used carefully, however, so the condyle will not be deflected away from the stable joint position. If the leaf gauge is too rigid, it may provide a posterior slope, deflecting the mandible posteriorly as the elevator muscles contract. Another error may result if the patient attempts to bite on the leaf

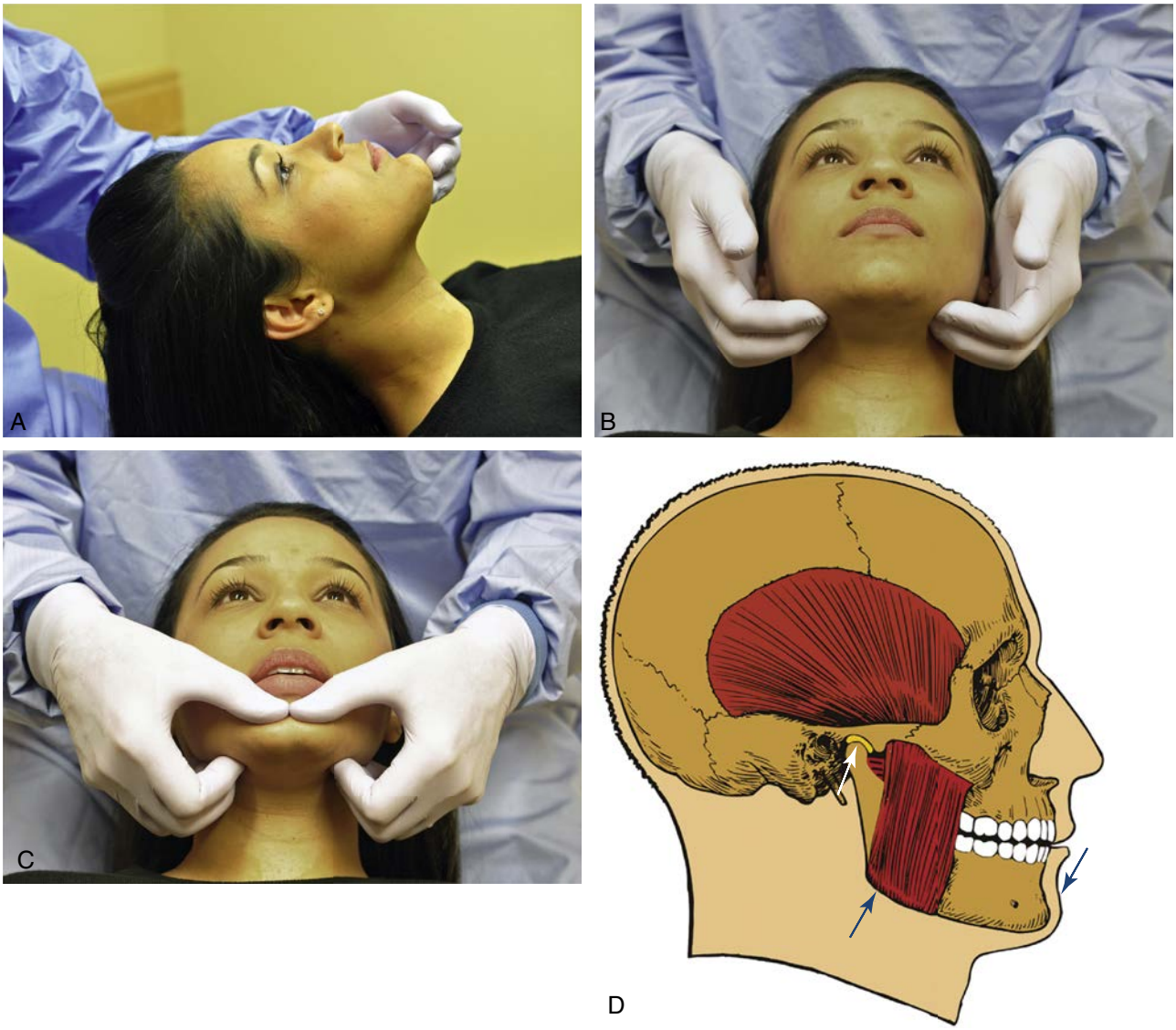


FIGURE 13-2 A, Successfully guiding the mandible into the musculoskeletally-stable position begins with having the patient recline and directing the chin upward. B, The four fingers of each hand are placed along the lower border of the mandible. The small finger should be behind the angle, with the remaining fingers on the inferior border of the mandible. An important point is to place the fingers on the bone and not in the soft tissues of the neck. C, The thumbs meet over the symphysis of the chin. D, Downward force is applied to the chin (*blue arrow*) while superior force is applied to the angle of the mandible (*blue arrow*). The overall affect is to set the condyle superoanterior in the fossae (*white arrow*).

gauge in a slightly forward position, as if to bite off a sandwich. This will lead to protruding the mandible from the MS position.

For effective use of the leaf gauge, the patient must attempt to close down on the posterior teeth with mild force. Enough leaves are placed between the anterior teeth to separate the posterior teeth slightly. The patient is instructed to close by trying to use only the temporalis muscles, avoiding any heavy masseter contraction. At first this is a difficult request; however, by having the patient place two fingers over these muscles, the examiner can demonstrate how they feel when contracting. The patient will quickly learn to contract the temporalis muscles predominantly, which will minimize protrusive forces. Once this has been mastered, the leaves are removed one by one until the teeth become closer, and therefore so that the occlusal relationship can be evaluated in the MS position.

Before beginning any orthodontic therapy, the orthodontist should feel confident that the MS position has been located. Nothing is worse than completing the orthodontic therapy only to learn that the patient was habitually posturing the jaw in a forward position, and that a duel bite has been developed. This condition does not occur often, but being suspicious that it can will help assure that it does not. If the operator feels unsure in locating the stable mandibular position, orthodontic therapy should be delayed until certainty is established.

Still another condition that should raise suspicion is the presence of a unilateral cross-bite. Patients with a unilateral cross-bite often shift the mandible to one side during final tooth contact.

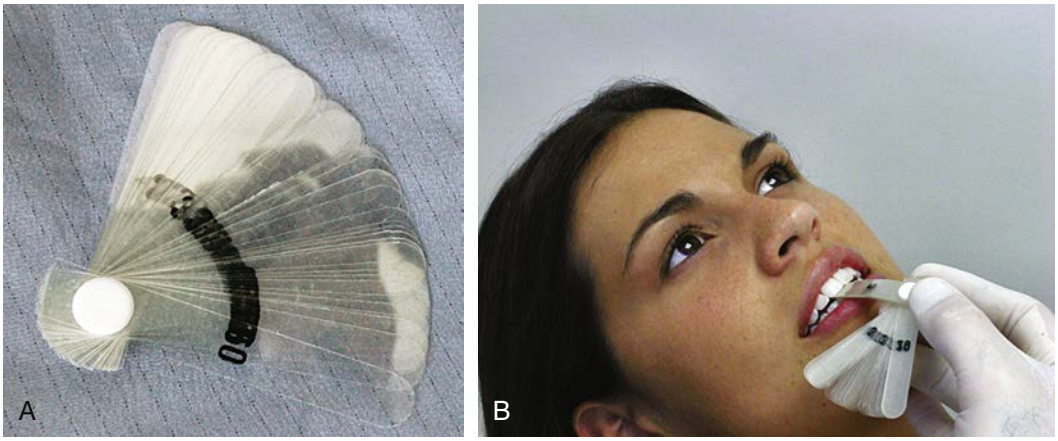


FIGURE 13-3 A, A leaf gauge. B, A leaf gauge may be used to assist in locating the musculoskeletally stable position. The patient is asked to close, and enough leaves are placed between the anterior teeth to separate the posterior teeth slightly. As the patient is asked to close on the posterior teeth, the condyles will be seated to their musculoskeletally stable position. Care should be taken to ensure that the patient does not protrude the mandible while closing and that the leaf gauge doses not exert a retruding force on the condyles. Once the position has been located, the leaves are removed one at a time so that the initial contact in the musculoskeletally stable position can be identified.

This shifting may prevent a condyle from being maintained in a stable position in the fossa. The orthodontist should observe the patient, while the mouth is being closed, for any deviations or deflections as the teeth reach intercuspation. In cases when the shift is great, tomography may be useful in identifying the position of the condyle in the fossa. However, it should be noted that tomograms have limited use in identifying condylar position unless gross positioning abnormalities exist. The reason for this is because tomography, as with any radiograph, only images subarticular bone and not the soft tissues, which are the true articular surfaces of the joint. Since these tissues can vary in thickness, the condylar position may not appear to be seated, when in reality it is in a MS position. Transcranial and panoramic radiographs are even less reliable for identifying the condylar position in the fossa.

As previously stated, being suspicious that a condyle may not be fully seated is the beginning of developing a successful treatment plan. If the operator has any doubt regarding the location of the MS position, a stabilization appliance should be fabricated until a stable and reproducible condylar position is found.

Once the MS position has been reliably located, the relationship of the maxillary and mandibular teeth is observed in this mandibular position. Since the orthodontic treatment goal is to develop the maximum intercuspal position of the teeth in this mandibular position, the orthodontist needs to develop the correct orthodontic strategies that will accomplish this goal. In some instances, the orthodontist may find it useful to mount the patient's casts on an articulator to better visualize the occlusal relationship. This may be especially helpful when a significant intra-arch discrepancy exists. This author does not believe it is necessary to mount every orthodontic case on an articulator. In most growing patients, the orthodontic therapy will likely be completed before final maturation of the condyle/fossae relationship. It is important that the orthodontist always be aware of the MS position of the condyles, and finalize the occlusion in relationship to this position. However, final precision of the position is likely accomplished by the physiology of form and function as the young adult matures. In other words, the orthodontist needs to provide an occlusal condition that is within the physiologic tolerance or adaptability of the patient. In a growing patient, it would be reasonable to assume that this is within a millimeter or two of the MS position of the joint. Once the orthodontic therapy is finalized, the patient's individual loading during function will normally assist in stabilizing the masticatory system. The only point in question is, "How adaptable are the patient's masticatory structures?" Of course this is unknown, and therefore the orthodontist needs to always strive toward developing the occlusal position as close to the MS position as possible. In difficult cases, the articulator may be useful in achieving this goal. However, it should always be remembered that an articulator is merely a tool that may assist in achieving your goal, not a magical instrument that will assure success.

In adult patients, it more important to precisely develop the orthopedically stable position since growth is less likely and adaptability may be less. The articulator may be of greater assistance in these cases, but once again, articulators are not always needed. The clinician needs to assess the dental relationship and then determine if an articulator will assist in accomplishing the treatment goals. Remember that the articulator is only as accurate as the operator who takes the records and mounts the casts.

EVALUATING THE PATIENT FOR TEMPOROMANDIBULAR DISORDERS

Since TMD symptoms are common, it is recommended that every orthodontic patient be screened for these problems, regardless of the apparent need or lack of need for treatment. Since orthodontic therapy will likely influence the patient's occlusal condition, it is important to identify any dysfunction in the masticatory system before therapy is ever begun. Knowing the functional condition of the masticatory system in advance helps prepare the patient and the orthodontist to what can be expected after the therapy has been completed. This

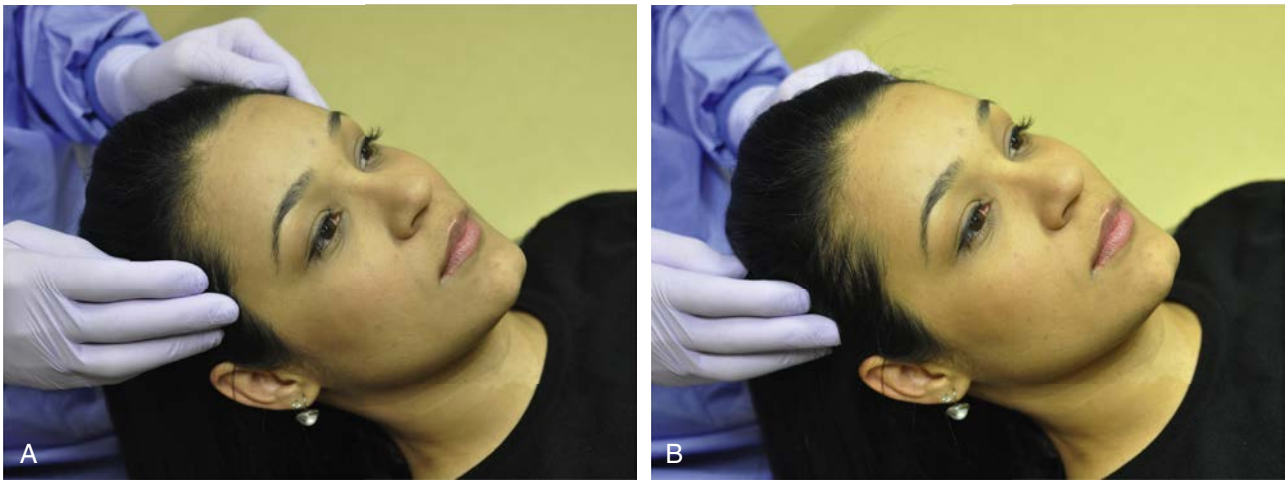


FIGURE 13-4 A, Palpation of the anterior portion of the temporalis muscle. B, Palpation of the posterior portion of the temporalis muscle.

information also helps develop the most appropriate treatment plan that will minimize dysfunction in future years. Nothing is more disheartening to the orthodontist than to be in the middle of orthodontic therapy and have the patient report that a preexisting TMD symptom was a result of the orthodontic therapy. It greatly behooves the orthodontist to be aware of all conditions in the masticatory system *before* any therapy is begun.

A TMD Screen History

The purpose of the screening history and examination is to identify any TMD signs and symptoms of which the patient may or may not be aware (i.e., headaches, ear pain). The screening history consists of several questions that will help alert the orthodontist to any TM disorder symptoms. These can be asked personally by the clinician or may be included in the general health and dental questionnaire that the patient completes prior to developing the treatment plan. The following questions can be used to identify functional disturbances.[12]

1. Do you have difficulty and/or pain opening your mouth, for instance, when yawning?
2. Does your jaw get "stuck, locked, or go out"?
3. Do you have difficulty and/or pain when chewing, talking, or using your jaws?
4. Are you aware of noises in the jaw joints?
5. Do your jaws regularly feel stiff, tight, or tired?
6. Do you have pain in or about the ears, temples, or cheeks?
7. Do you have frequent headaches, neck aches, or toothaches?
8. Have you had a recent injury to your head, neck, or jaw?
9. Have you been aware of any recent changes in your bite?
10. Have you previously been treated for any unexplained facial pain or a jaw joint problem?

If a patient reports positively to any of these questions, the clinician should request additional information to clarify the condition.

A TMD Screen Examination

A screening examination should accompany the screening history.[12] This should be relatively brief and is an attempt to identify any variation from normal anatomy and function. It begins with an inspection of the facial symmetry. Any variation from the general bilateral symmetry should raise suspicion and indicate the need for further examination. The screening examination should include the palpation of facial muscle, and the TMJs as well as observations of jaw movement. The occlusal condition should also be evaluated with respect to the orthopedically stable position of the joint.

Muscle Palpation

Several important muscles of the masticatory system are palpated for pain or tenderness during the screening examination. The temporalis (Fig. 13-4, *A* and *B*) and masseter muscles (Fig. 13-5, *A* and *B*) are palpated bilaterally. Palpation of the muscle is accomplished mainly by the palmar surface of the middle finger, with the index finger and forefinger testing the adjacent areas. Soft but firm pressure is applied to the designated muscles, the fingers compressing the adjacent tissues in a small circular motion. A single firm thrust of 1 or 2 seconds duration is usually better than several light thrusts. During palpation, the patient is asked whether it hurts or is just uncomfortable.

For the muscle examination to be most helpful, the degree of discomfort is ascertained and recorded. This is often a difficult task. Pain is subjective and is perceived and expressed quite differently from patient to patient. Yet the degree of discomfort in the structure can be important to recognizing the patient's pain problem as well as an excellent method of evaluating treatment effects. An attempt is made, therefore, not only to identify the affected muscles, but also to classify the degree of pain in each. When a muscle is palpated, the patient's response is placed in one of four categories.[13,14] A zero (0) is recorded when the muscle is palpated and there is no pain or tenderness reported by the patient. A number 1 is recorded if the patient responds that the palpation is uncomfortable (tenderness or soreness). A number 2 is recorded if the patient experiences definite discomfort or pain. A number 3 is recorded if the patient shows evasive action, eye tearing, or verbalizes a desire not to have the area palpated again. The pain or tenderness of each muscle is recorded on an examination form, which will assist diagnosis and later be used in the evaluation and assessment of progress.

Temporomandibular Joint Palpation

The TMJs are examined for any signs or symptoms associated with pain and dysfunction. Pain or tenderness of the TMJs is determined by digital palpation of the joints when the mandible is both stationary and during dynamic movement. The

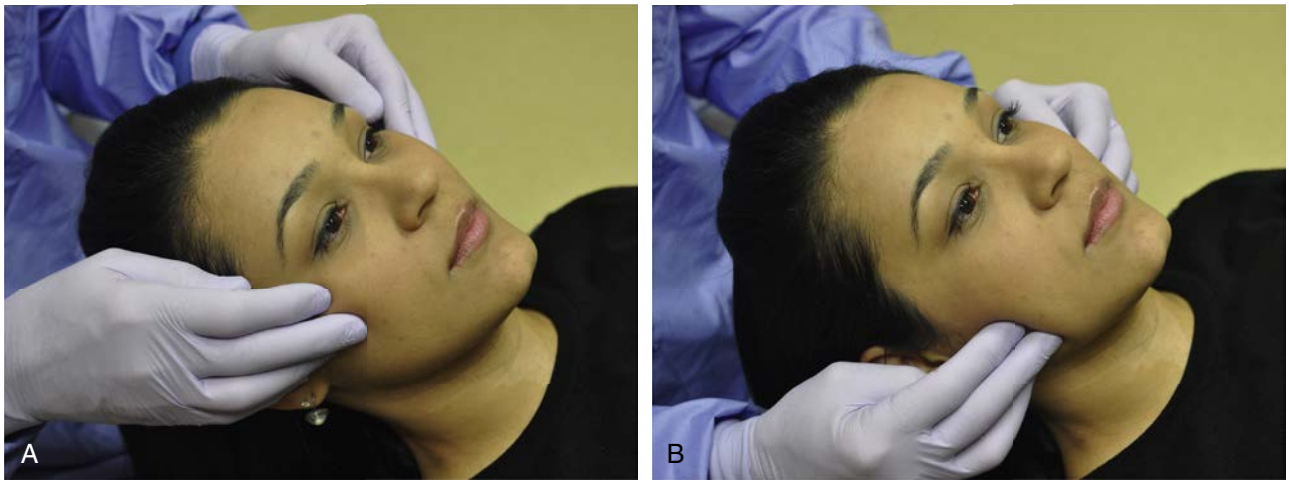


FIGURE 13-5 A, Palpation of the masseter muscle at the superior attachment to the zygomatic arch. B, Palpation of the masseter muscle at its attachment of the lower border of the mandible.

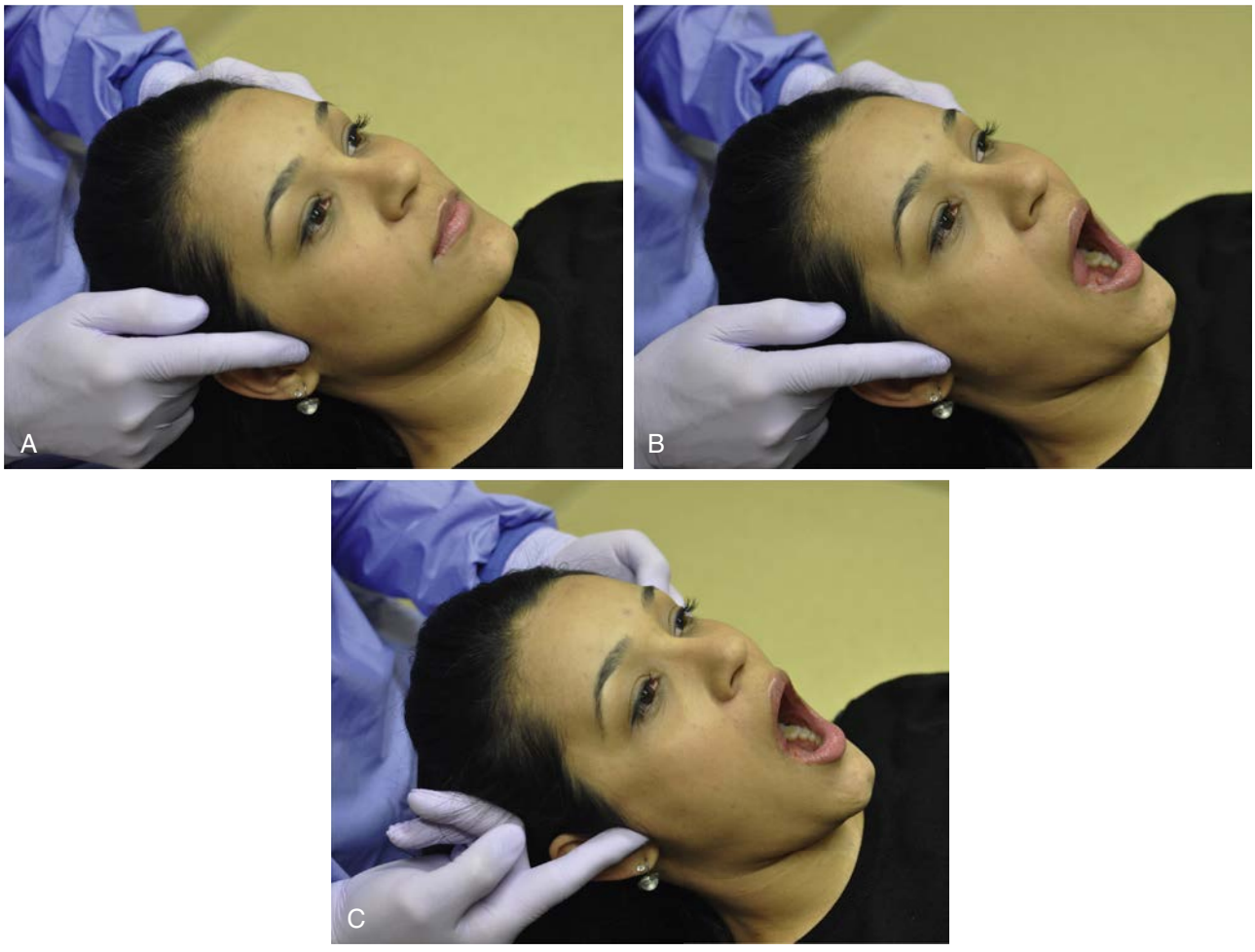


FIGURE 13-6 A, Palpation of the temporomandibular joint in the closed-mouth position. B, Palpation of the temporomandibular joint in the opened-mouth position. C, Palpation of temporomandibular joint with the mouth fully open. The finger is moved behind the condyle to palpate the posterior aspect of the joint.

fingertips are placed over the lateral aspects of both joint areas simultaneously (Fig. 13-6, *A*). If uncertainty exists regarding the proper position of the fingers, the patient is asked to open and close a few times. The fingertips should feel the lateral poles of the condyles passing downward and forward across the articular eminences. Once the position of the fingers over the joints has been verified, the patient relaxes and medial force is applied to the joint areas. The patient is asked to report any symptoms and they are recorded with the same numerical code that is used for the muscles. Once the symptoms are

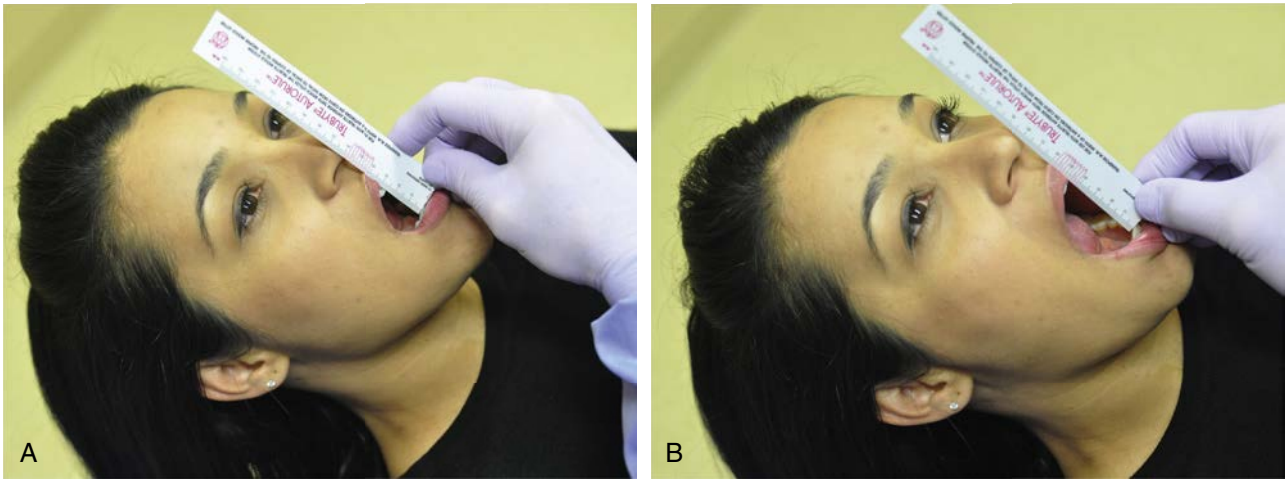


FIGURE 13-7 Measuring mouth opening. **A,** The patient is asked to open the mouth until pain is first felt. The interincisal distance is measured, which is called the maximum comfortable opening. **B,** The patient is then ask to open as wide as possible, even if this is painful. This measurement is called the maximum mouth opening.

recorded in a static position, the patient opens and closes, and any symptoms associated with this movement are recorded (Fig. 13-6, *B*). As the patient opens maximally, the fingers should be rotated slightly posteriorly to apply force to the posterior aspect of the condyle (Fig. 13-6, *C*). Posterior capsulitis and retrodiscitis are clinically evaluated in this manner.

Joint sounds are recorded as either clicks or crepitation. A click is a single sound of short duration. If it is relatively loud, it is sometimes referred to as a pop. Crepitation is a multiple, gravel-like sound described as "grating" and "complicated." Crepitation is most commonly associated with osteoarthritic changes of the articular surfaces of the joint.[15-18]

Joint sounds can be perceived by placing the fingertips over the lateral surfaces of the joint and having the patient open and close. Often they may be felt by the fingertips. A more careful examination can be performed by placing a stethoscope over the joint area. Not only should the character of any joint sounds be recorded (clicking or crepitation), but also the degree of mouth opening associated with the sound. Of equal importance is whether the sound occurs during opening or closing or can be heard during both these movements.

It is not wise to examine the joint for sounds by placing the fingers in the patient's ears. It has been demonstrated that this technique can actually produce joint sounds that are not present during normal function of the joint.[19] It is thought that this technique forces the ear canal cartilage against the posterior aspect of the joint; either this tissue produces sounds or this force displaces the disc, which produces the additional sounds.

Range of Mandibular Movement

A screening examination should also include evaluation of the patient's range of mandibular movement. The normal range[20-22] of mouth opening when measured interincisally is between 53 and 58 mm. Even a 6-year-old child can normally open a maximum 40 mm or more.[23,24] The patient is asked to open slowly until pain is first felt (Fig. 13-7, *A*). At that point, the distance between the incisal edges of the maxillary and mandibular anterior teeth is measured. This is the maximum comfortable opening. The patient is next asked to open the mouth maximally. This is recorded as the maximum opening (Fig. 13-7, *B*). In the absence of pain, the maximum comfortable opening and maximum opening are the same.

FIGURE 13-8 Measuring the distance of lateral eccentric movement using a millimeter ruler.

A restricted mouth-opening is considered to be any distance less than 40 mm. Only 1.2% of young adults[23] open less than 40 mm. Less than 40 mm of mouth opening, therefore, seems to represent a reasonable point to designate restriction; however, one should always consider the patient's age and body size.

The patient is next instructed to move his mandible laterally. A lateral movement less than 8 mm is recorded as a restricted movement (Fig. 13-8). Protrusive movement is also evaluated in a similar manner.

Occlusal Evaluation

The occlusal examination[12] begins with an observation of the occlusal contacts when the condyles are in their optimum orthopedic position (MS position). As already described, this position is located by using a bilateral manual manipulation technique. In this position the mandible can be purely rotated, opened, and closed approximately 20 mm interincisally while the condyles remain in their MS position. Once the MS position is located, the mandible is brought into tooth contact and the occlusal relationship of the teeth in this joint

position is evaluated. Once tooth contact is achieved, the patient is asked to hold the mandible on the first occlusal contact and the relationship of the maxillary and mandibular teeth is noted. Then the patient is requested to apply force to the teeth, and any shifting of the mandible is observed. If the occlusion is not stable in the MS position, a shifting will occur that carries the condyles away from their orthopedically stable positions to the more stable maximum-intercuspal position. This shifting represents a lack of orthopedic stability.

It is important to observe the horizontal and vertical components of the slide. Some slides occur in a straight, anterosuperior direction into intercuspal position. Others have a lateral component. It has been reported[25,26] that slides that deflect the mandible to the left or right are more commonly associated with dysfunction than are slides that create a straight, anteroverticall movement. The distance of the slide is also important in that slides greater than 3 to 4 mm have been associated with increased incidence of TMD symptoms. Conversely, slides of 1 to 2 mm or less, which are very common, do not seem to be related to TMD symptoms. If the patient is asked to apply force to the teeth and no shift occurs, the intercuspal position is said to be coincident with MS position.

If the screening history and examination reveal positive findings, a more thorough history and examination for TM disorders is completed. A thorough TMD examination can be found in other sources.[12]

The clinical significance of joint sounds. Joint sounds most commonly occur as a result of disc displacements.[10] When the disc is displaced during mouth opening, an abnormal translatory movement can occur between the condyle and the disc, causing a clicking sound. Although disc displacements are considered abnormal, they are certainly not uncommon. The published prevalence of disc displacement in asymptomatic children and young adults is approximately 30%.[27] Recent studies suggest that a majority of adolescent preorthodontic patients, regardless of gender, present with some disc displacement.[28,29] The question that needs to be asked is, "Does the click represent pathology?" Often clicks occur in the absence of any pain or signs of progression. In a very interesting study by Magnusson et al.,[30] joint sounds were recorded in a 15-year-old population and then again in the same population at age 20. Of the 35 subjects who had sounds at age 15, 16 (or 46%) no longer had them at age 20. None of these subjects was provided any treatment. It was also interesting to note in this study that of the 38 15-year-olds who did not have joint sounds, 19 (or 50%) did have joint sounds at age 20. These data suggest that a 15-year-old with TMJ sounds has a 46% chance the sound will resolve without treatment by the age of 20. The study also suggests, however, that if a 15-year-old does not have TMJ sounds, there is a 50% chance he or she will acquire a sound by age 20. The authors concluded that joint sounds come and go and are often unrelated to major masticatory symptoms. Ten- and twenty-year follow-up examinations of this same population continue to reveal the lack of a significant relationship between joint sounds and pain or dysfunction.[31,32]

In a similar study, Kononen et al.[33] observed 128 young adults longitudinally over 9 years at ages 14, 15, 18, and 23 years. They reported that although clicking did increase significantly with age from 11% to 34%, there was no predictable pattern, and only 2% of subjects showed consistent findings during the periods of evaluation. They found no relationship between clicking and the progression to locking.

A significant long-term study by de Leeuw et al.[34] found that 30 years after nonsurgical management of intracapsular disorders, joint sounds persisted in 54% of the patients. Although these findings reveal that joint sounds remain in many patients, it is important to note that none of these patients was experiencing any discomfort or even dysfunction from their joint condition. This study, like the others referenced here, suggest that joint sounds are often not associated with pain or even major TM joint dysfunction. This research group[35,36] also found that long-term osseous changes in the condyle were commonly associated with disc dislocation without reduction and not so commonly associated with disc dislocation with reduction. Yet even in the patients with significant alterations in condylar morphology (osteoarthrosis), little pain and dysfunction were noted.

Studies such as these suggest that joint sounds are not always progressive and therefore may not need to be treated. Several studies[33,37-41] report that progression of intracapsular disorders as determined by joint sounds only occurs in 7% to 9% of the patients with sounds. One study suggests, however, that if the disc derangement disorder results in significant catching or locking, the chances that the disorder will progress is much greater.[42]

The long-term studies certainly suggest that joint sounds are not necessarily a significant problem for most patients. However the majority of these studies have evaluated adult populations. What about a young adult who will be undergoing significant occlusal changes with orthodontic treatment? This may pose a different question for the orthodontist. Certainly the orthodontist who is trying to achieve orthopedic stability in the masticatory structures needs to be able to assess the status of the TMJs prior to treatment.

Animal studies demonstrate if a disc displacement is surgically induced, histologic changes are observed in the condylar cartilage.[43] In this study, the investigators also noted that the condylar cartilage on the contralateral control side was also affected, though to a lesser degree. In studies conducted by Legrell et al., surgically induced unilateral disc displacement without reduction in animal models resulted in developmental facial asymmetry. Animals subjected to bilateral disc displacement developed Angle Class II relationship.[44] These results demonstrate that alterations in the disc position can influence three-dimensional growth patterns of the condyle. Similarly, experimentally induced disc displacements without reduction in growing animals have been shown to actually hinder mandibular growth relative to controls. The negative effects were greater on anterior and superior growth of the mandible while posterior growth was relatively unaffected.[45] According to A. Björk, bone apposition on the posterior aspect of the condyle causes clockwise rotation of the mandible, resulting in a receded chin and an open bite, which would be an unfavorable situation for orthodontic treatment.[46]

Although these experimental studies suggest that disc displacements can lead to condylar changes, the question remains, on a clinical level, how often this occurs in the young adult. This question is very important but very difficult to determine. The musculoskeletal system is very adaptive, especially in this young population. It is very likely that the individual with a disc displacement will adapt to this change and show very little clinical consequence. However, this may not be true for every patient. Therefore it is very important for the orthodontist to evaluate the health of the TMJs prior to any treatment. As a general

rule, individuals who are having a problem with adaption have greater degree of pain and dysfunction. Therefore, if the history and examination reveal any pain or significant dysfunction, more investigation is indicated.

If the examination determines the presence of pain or significant dysfunction, additional information may be needed. Images such as cone beam CT or MRIs may be able to provide more information. Cone beam CT three-dimensionally visualizes morphologic alterations in bony structures, degenerative changes, and to some degree the position of the condylar within the fossa.[47] MRIs can identify disc displacement and its degree, location, and direction. MRIs can also detect certain disc conditions such as hypertrophy, and T2-weighted images visualize joint effusion. These data may be useful in understanding the health of these tissues. The interpretation of diagnostic images will be described later in detail through a clinical case.

For patients who exhibit TMD symptoms, abnormality in images, unstable jaw position, and/or facial asymmetry, the orthodontist should consider resolving the TMD symptoms and stabilizing the joint position before any orthodontic therapy is begun. A stabilization appliance may be useful in reducing the TMD symptoms. With this appliance, the causal relationship between the patient's occlusion and TMD symptoms can be better appreciated (as will be discussed later in this chapter). Improvement in soft and hard tissues of the TMJs can also be expected during the occlusal appliance therapy, providing a repeatable and stable jaw position. With a stabilized joint, the orthodontist can develop a treatment plan that will provide optimum occlusal contacts in this position (orthopedic stability).

DEVELOPING THE ORTHODONTIC/TMD TREATMENT PLAN

All potential orthodontic patients should be evaluated for both their aesthetic needs as well as for their functional needs. Once the history and examination data are collected, this information is used to develop a treatment plan that will appropriately meet the overall needs of the patient. When considering the possibilities, the patient can have only aesthetic needs, only TMD needs, or both aesthetic and TMD needs. The type of needs determines the treatment sequence. When the patient has only aesthetic needs, the clinician need only consider the best therapy to fulfill the orthodontic goals. Included with these goals, of course, is not only the best mechanics to move teeth, but also the goals of achieving orthopedic stability, which have already been presented in this chapter.

When a patient presents with only TMD symptoms the clinician should not assume that orthodontic therapy would be a part of the treatment plan. Orthodontic therapy is only indicated for TMD patients when it has been determined that orthopedic instability is present and this instability is contributing to the TM disorder. The mere presence of orthopedic instability is not enough evidence to be certain that it is contributing to the TM disorder. Many individuals have orthopedic instability without any functional complaints or complications. Therefore, when TMD symptoms are present, the clinician should first attempt to determine if the orthopedic instability is contributing to the TM disorder. The best way to identify this relationship is by first providing orthopedic stability reversibly with an occlusal appliance. If the occlusal appliance adequately provides the desirable stability but does not reduce the TMD symptoms it can be assumed that orthopedic stability is not related to the symptoms and orthodontic therapy should not be considered for this patient. It is important to remember that orthodontic therapy can only affect TMD symptoms by changing the occlusal contact pattern of the teeth and the resulting function of the masticatory system (improved orthopedic stability).

If an occlusal appliance successfully reduces the TMD symptoms, the clinician often assumes that the occlusion and its relationship to orthopedic instability are etiologic factors in the TM disorder. Although this may be true, it makes an assumption that the only manner in which an occlusal appliance affects TMD symptoms is by altering the patient's occlusion. This is a very naive assumption. In fact there are several factors that may explain how occlusal appliances reduce symptoms associated with TM disorders. Consider the following factors:

1. *Alteration of the occlusal condition*: All occlusal appliances temporarily alter the existing occlusal condition. A change, especially toward a more stable and optimum condition, generally decreases protective muscle co-contraction, leading to a reduction of symptoms.
2. *Alteration of the condylar position*: Most appliances alter condylar position to either a more MS or a more structurally compatible and functional position. This effect on the joint stability can be responsible for a decrease in symptoms.
3. *Increase in the vertical dimension*: All interocclusal appliances increase the patient's vertical dimension while they are being worn. This effect is universal regardless of treatment goals. It has been demonstrated that increases in vertical dimension can temporarily decrease muscle activity[48-50] and symptoms.[51-53]
4. *Cognitive awareness*: Patients who wear occlusal appliances become more aware of their functional and parafunctional behavior. The appliance acts as a constant reminder to alter activities that may affect the disorder. As cognitive awareness is increased, factors that contribute to the disorder are decreased. The result is a decrease in symptoms.[54-56]
5. *Placebo effect*: As with any treatment, a placebo effect can result.[57,58] Studies[59-62] suggest that up to 40% of the patients suffering from certain TM disorders respond favorably to such treatment. A positive placebo effect may result from the competent and reassuring manner in which the doctor approaches the patient and provides the therapy. This favorable doctor–patient relationship, accompanied by an explanation of the problem and reassurance that the appliance will be effective, often leads to a decrease in the negative emotional state of the patient, which may be the significant factor responsible for the placebo effect.
6. *Increased peripheral input to the CNS*: Evidence suggests that nocturnal muscle hyperactivity appears to have its source at the central nervous system level.[63-65] Any change at the peripheral input level seems to have an inhibitory effect on this CNS activity.[63,66] When an occlusal appliance is placed between the teeth, it provides a change in peripheral input and thus decreases CNS-induced bruxism. The appliance does not cure bruxism; it only inhibits the bruxing tendency while it is being worn. Studies[67-69] show that even with long-term use of an appliance, bruxism seems to return.
7. *Regression to the mean*: Regression to the mean is a statistical term that addresses the common fluctuation of symptoms associated with chronic pain conditions.[70] If one follows the musculoskeletal symptoms of a particular patient, it will be observed that the intensity of pain will usually vary on a daily

bases. Some days will be quite painful, while other days are more tolerable. If the patient is asked to rate the intensity of his pain each day on a visual analog scale, with 0 being no pain and 10 the worst possible pain, the patient may report an average day to be 3. This would represent his mean pain score. However, some days the pain may reach a 7 or 8 but then often with time the pain returns back to its mean level of 3. Patients most commonly report to the dental office when the pain intensity is great since that is often the factor that motivates them to seek treatment. When the clinician provides therapy (such as an occlusal appliance) and the symptoms reduce back to the average level of 3, one must question if the reduction of symptoms was actually the therapeutic effect of the treatment or did the patient's symptoms merely "regressed to the mean." This factor can be very confusing to the clinician and may lead to misdirection of future treatment. Uncontrolled short-term studies that report success of various therapies need to be questioned regarding their actual effect. Is it actually the therapeutic effect of the modality or was it regression to the mean? The importance of well-controlled, blinded studies becomes obvious when attempting to answer this question.[58]

When a patient's symptoms are reduced by occlusal appliance therapy, each of these seven factors must be considered as responsible for the success. All permanent (irreversible) treatment should be delayed until significant evidence exists to determine which factor(s) was important in reducing the symptoms. This can be accomplished by first allowing the patient to wear the appliance for 1 to 2 months to assure that the symptoms have been adequately controlled. It should be noted that the appliance may not need to be worn 24 hours a day.[71] Many patients do very well with only part-time use, most commonly at night. The amount of time needed is dependent upon the type of TMD that is being treated.[72] Once the patient is comfortable for several weeks, perhaps months, the patient should be asked to reduce the use of the appliance. In many instances, the patient can discontinue use of the appliance and not experience a return of symptoms. When this occurs, factors that relate to dental etiology, such as the occlusal condition, condylar position, or vertical dimension, are not likely the causes of the TMD. When this occurs, the patient should be encouraged to wear the appliance occasionally as needed to manage any return in symptoms secondary to bruxism or emotional stress. These patients do not need orthodontic therapy.

If reducing the use of the occlusal appliance reestablishes the original symptoms, then factors such as the occlusal condition, condylar position, or vertical dimension may need to be considered as potential etiologic factors. But which factor is the likely cause? This question needs to be answered before any therapy begins. If the clinician is suspicious that the vertical dimension is the etiology of the TMD symptoms, then an attempt should be made to verify this assumption. The appliance should be gradually thinned while maintaining the same occlusal contacts and condylar position. The significance of the vertical dimension is confirmed if the symptoms return as the appliance is thinned. However, if the symptoms do not return as the appliance reaches the original vertical dimension, the clinician should be more suspicious that the factors responsible for the reduction of symptoms are either the occlusal condition or the condylar position. It is now important to realize that these two remaining factors are accessed together by evaluating the patient's orthopedic stability. The patient's mandible should be bilaterally manipulated to the MS position and the occlusal contact pattern assessed. At this time, the clinician will now be able to observe the orthopedic instability and have some reasonable clinical certainty that this condition is contributing to the TMD symptoms. Since the clinical evidence now suggests that a change in the occlusal condition would likely reduce the TMD symptoms, the clinician should accurately mount the patient's study casts on an articulator and determine the most appropriate method of achieving the goals of orthopedic stability (i.e., orthodontic therapy).

By way of summary, although occlusal appliances may have some diagnostic value, conclusions regarding the rationale for their success must not be hastily made. Before any orthodontic treatment plan for TMD is begun, ample evidence must exist that the treatment will be of benefit to the patient. In reality, only a select group of TMD patients benefit from orthodontic therapy.

The last situation to be discussed is the patient with TMD who also has aesthetic needs. When patients have both needs, the clinician's first efforts should be directed toward resolving the TMD symptoms. The clinician may decide to use an occlusal appliance to help reduce the symptoms. When an appliance is used, it will not only help reduce symptoms but also assist in locating the MS position of the joint. Once the symptoms are reduced, the orthodontic treatment plan is developed with respect to the MS position of the joints, and therapy can begin.

MANAGING TMD SYMPTOMS THAT ARISE DURING ORTHODONTIC THERAPY

Occasionally a patient will present with TMD symptoms while actively undergoing orthodontic therapy. This may pose a challenge to the orthodontist. These symptoms may demand immediate attention, yet traditional TMD therapy would be difficult and likely delay the orthodontic treatment plan. Therefore, the orthodontist needs to have some treatment strategies that will help reduce the patient's symptoms while orthodontic therapy continues.

The first important consideration with a patient who develops new TMD symptoms is to review the orthodontic progress toward achieving orthopedic stability in the masticatory system. This is especially important if the TMD symptoms are related to intracapsular concerns such as joint clicking. The orthodontist should locate the MS position of the condyles using a bilateral manual manipulation technique and determine if the orthodontic therapy is moving toward developing a favorable intercuspal position in this stable joint position. If it is determined that this goal is not being achieved, the orthodontic treatment plan should be redirected in a manner to better accomplish this goal.

Once it is determined that the direction of treatment is correct, but not yet achieved because orthodontic therapy has not been finalized, the patient's specific symptoms can be addressed. The response to symptoms should be appropriate for the type and intensity of the patient's complaints. Although the management of muscle pain disorders is often different than intracapsular disorders, some general treatment strategies can be used to manage both. Much of this management is conservative and easily applied to the patient.

One of the most important things the orthodontist can do for the patient is to provide education. The patient needs to know that TMD symptoms are common and benign. They do not shorten one's life. The nature course of most TMD is to

experience fluctuations of symptoms, often resolving with little to no significant treatment. Therefore, informing the patient of these can be very therapeutic. Since emotional stress can be an etiologic factor of TMD, often worrying about the problem makes the situation even worse.

Education is also important for it can be used to actively bring the patient into the treatment that can help them the most. Some simple behavioral interventions can be most helpful. For example, advising the patient to reduce jaw use to within painless limits goes a long way in symptom reduction. The patient should be instructed to eat softer foods, smaller bites and slower chewing. Don't let it hurt. Increased pain can maintain the pain cycle, which may prolong the pain experience.[73] Sometimes patients chew gum, bite on pencils, or bite their fingernails. These activities can further enhance muscle pain. The patient needs to be informed that it is common to put the teeth together even at times when we are unaware. Bruxing and/or clenching the teeth are good examples of such activity. Making the patient aware of these activities (cognitive awareness) is the beginning of therapy. Once the patient is aware, the patient should be instructed that at any time he catches himself with the teeth in contact and he is not chewing or swallowing, he should immediately puff a little air between the lips and teeth, let the jaw relax, and then allow the lips to seal. This will place the mandible in the postural position, disengaging the tooth. This position minimizes muscle activity and joint loading. Although this technique seems too simple to work, it has been demonstrated to be very effective in reducing TMD symptoms.[74] The concept of "lips together and teeth apart" is powerful in reducing most acute TMD pains.

If the acute TMD symptoms are associated with pain, the clinician may wish to suggest that a mild analgesic be used for 5 to 7 days to reduce the pain. As previously mentioned, pain can reinforce the condition beginning cyclic muscle pain.[73] Mild analgesics can be used to break this cycle. An NSAID such as ibuprofen can be very useful. It should not be take only as needed because it will not effectively break the pain cycle. Therefore, the patient should be instructed to take 400 to 600 mg of ibuprofen three times a day with meals for 5 to 6 days. Most individuals can tolerate this medication without problems, but if there is a history of gastrointestinal upset, other options should be used.

Another very conservative therapy for muscle pain is moist heat. A moist, hot towel can be placed over the painful muscle(s) for 15 to 20 minutes and repeated several times a day as needed. This therapy can be very helpful in reducing acute muscle pain.

Still another option that may be considered relates to sleep quality. If the patient reports poor sleep quality and is waking up with increased pain, one may be suspicious of sleep-related bruxism. When this occurs, a mild muscle relaxant such as cyclobenzaprine, 5 to 10 mg before sleep, may be helpful. This may only be needed for 5 to 7 days.[73]

For many acute TMD symptoms, these simple therapeutic interventions will be adequate to resolve the condition. There is no need to alter the orthodontic treatment. The patient should be reassessed in 7 to 10 days to make sure that the symptoms have been resolved. Once the symptoms have resolved, continue with the orthodontic therapy with the goals of establishing orthopedic stability and an acceptable esthetic.

If in 10 days the symptoms have not adequately resolved, additional steps may be needed. Continue to reinforce the important behavioral aspects of resting the masticatory systems but also consider other factors that may help reduce muscle activity. If the patient is wearing interarch elastics, they should be temporally discontinued. Some patients have a tendency to play with these elastics, which further activates the muscles. Also it may be time to consider temporary methods of disengaging the teeth during sleep. Making a stabilization appliance may be difficult with all the orthodontic brackets and wires so on a short term base one can consider a more generic soft appliance.[75,76] These appliances do not require individual fabrication, making them easy to use with orthodontic brackets and wires. Although these types of appliances have not been shown to be as effective as more traditional, hard-stabilization appliances, they can certainly be used for a brief period of time to hopefully reduce acute TMD symptoms.

The patient should again be reevaluated in 1 to 2 weeks for symptom reduction. If the TMD symptoms have resolved, proceed with the orthodontic treatment plan. If the symptoms have still not resolved, a more significant TMD is present and therefore a more traditional approach to management may be needed. At that time, the orthodontist may need to discontinue the active orthodontic therapy by removing the arch wire and fabricate a more traditional stabilization appliance. The stabilization appliance should provide even contact of all teeth on flat surfaces when the condyles are in the MS position (orthopedic stability). Eccentric guidance is provided by the canines. This appliance may be a little more difficult to fabricate with the orthodontic brackets in place, but certainly not impossible. The brackets can be blocked out with wax on the model, allowing a good fit. In many patients, this appliance may not need to be worn for very long.

Another appliance that may be considered is the anterior bite plane.[77] This appliance provides only anterior tooth contact and can be useful in reducing symptoms. Since the posterior teeth do not occlude, it may be easier to fabricate and adjust. As soon as the symptoms have resolved, the appliance can be removed and active orthodontic therapy can be resumed.

At that time, the orthodontist needs to consider all the etiologic factors that may be contributing to the TMD, such as emotional stress, trauma, deep sources of pain, and uncontrolled parafunctional activity. The patient should receive a complete history and examination for TMD and be managed appropriately.[12] Orthodontic therapy should not be reinitiated until the TMD symptoms have been properly managed.

In those instances when the TMD symptom is clicking and the orthodontic therapy is effectively moving toward establishing orthopedic stability, the clinician needs to be aware that this symptom is relatively common in the young adult and does not always lead to significant consequence. In fact, one study[30] that observed untreated subjects at the age of 15 years and then again at age 20 found that clicking is very common in this age group, and that clicking can come and go unrelated to any major clinical symptoms. Therefore, if the patient reports the onset of a joint sound unrelated to pain and the occlusal condition is being developed in harmony with the stable joint position, patient education regarding the problem may be all that is needed.

On occasion, the clicking joint is associated with significant pain or joint dysfunction, such as catching or locking. When this occurs, active orthodontic therapy should be discontinued and therapy should begin for the specific disorder that has been diagnosed. This might include appliance therapy and/or active physical therapy to the involved joint. The precise treatment for the intracapsular disorder is outside the goals of this chapter, and therefore other texts[78,79] should be pursued for a complete description of therapy.

SUMMARY

The goal of the orthodontist is to develop an esthetic smile and a functional masticatory system. Although initially esthetics is often considered the most important goal, function eventually becomes far more important in the overall success of treatment. Therefore, the orthodontist must always consider how the orthodontic therapy will affect function. To maximize sound orthopedic function, the occlusal condition must be finalized in harmony with the MS position of the TMJs. Accomplishing this goal will maximize the success of masticatory function in future years.

REFERENCES

1. Wink CS, St Onge M, Zimny ML. Neural elements in the human temporomandibular articular disc. *J Oral Maxillofac Surg*. 1992;50(4):334–337.
2. DuBrul EL. *Sicher's Oral Anatomy*. 7th ed. St. Louis, MO: Mosby Yearbook; 1980.
3. Williamson EH, Lundquist DO. Anterior guidance: its effect on electromyographic activity of the temporal and masseter muscles. *J Prosthet Dent*. 1983;49(6):816–823.
4. Williamson EH. Occlusion and TMJ dysfunction. *J Clin Orthod*. 1981;15:333–342.
5. Standlee JP. Stress transfer to the mandible during anterior guidance and group function at centric movements. *J Prosthet Dent*. 1979;34:35–45.
6. Kraus BS, Jordon RE, Abrhams L. *Dental Anatomy and Occlusion*. Baltimore, MD: Waverly Press Inc.; 1973.
7. Goldman HM, Cohen WD. *Periodontal Therapy*. 4th ed. St. Louis, MO: The CV Mosby Co.; 1968.
8. Mohl ND. Head posture and its role in occlusion. *N Y State Dent J*. 1976;42:17–23.
9. Dawson PE. *Evaluation, Diagnosis and Treatment of Occlusal Problems*. 2nd ed. St. Louis, MO: The CV Mosby Co.; 1989.
10. Okeson JP. *Management of Temporomandibular Disorders and Occlusion*. 7th ed. St. Louis, MO: Elsevier/Mosby Publishers; 2013:129–169.
11. Carroll WJ, Woelfel JB, Huffman RW. Simple application of anterior jig or leaf gauge in routine clinical practice. *J Prosthet Dent*. 1988;59(5):611–617.
12. Okeson JP. *Management of Temporomandibular Disorders and Occlusion*. 7th ed. St. Louis, MO: Elsevier/Mosby Publishers; 2013:170–221.
13. Moody PM, Calhoun TC, Okeson JP, Kemper JT. Stress-pain relationships in MPD syndrome patients and non-MPD syndrome patients. *J Prosthet Dent*. 1981;45:84–88.
14. Okeson JP, Kemper JT, Moody PM. A study of the use of occlusion splints in the treatment of acute and chronic patients with craniomandibular disorders. *J Prosthet Dent*. 1982;48(6):708–712.
15. Bezuur JN, Habets LLH, Jimenez Lopez V, Naeije M, Hansson TL. The recognition of craniomandibular disorders, a comparison between clinical and radiograghic findings in eighty-nine subjects. *J Oral Rehabil*. 1988;15:215–222.
16. Cholitgul W, Petersson A, Rohlin M, Akerman S. Clinical and radiological findings in temporomandibular joints with disc perforation. *Int J Oral Maxillofac Surg*. 1990;19(4):220–225.
17. Hansson LG, Petersson A, Vallon-Christersson D. Clinical and radiologic six-year follow-up study of patients with crepitation of the temporomandibular joint. *Swed Dent J*. 1984;8(6):277–287.
18. Widmalm SE, Westesson PL, Brooks SL, Hatala MP, Paesani D. Temporomandibular joint sounds: correlation to joint structure in fresh autopsy specimens. *Am J Orthod Dentofacial Orthop*. 1992;101(1):60–69.
19. Hardison JD, Okeson JP. Comparison of three clinical techniques for evaluating joint sounds. *Cranio*. 1990;8(4):307–311.
20. Agerberg G. Maximal mandibular movement in young men and women. *Swed Dent J*. 1974;67:81–100.
21. Solberg W. *Occlusion-Related Pathosis and Its Clinical Evaluation* Clinical Dentistry. New York, NY: Harper & Row Publishers; 1976:1–29.
22. Gea B. Range of jaw opening in an elderly non-patient population. *J Dent Res*. 1991;70(special issue):419. abstr 1225.
23. Agerberg G. Maximal mandibular movements in children. *Acta Odontol Scand*. 1974;32(3):147–159.
24. Vanderas AP. Mandibular movements and their relationship to age and body weight in children with or without clinical signs of craniomandibular dysfunction. Part IV. *J Dent Child*. 1992;59:338–341.
25. Solberg WK, Woo MW, Houston JB. Prevalence of mandibular dysfunction in young adults. *J Am Dent Assoc*. 1979;98(1):25–34.
26. Rieder C. The prevalence and magnitude of mandibular displacement in a survey population. *J Prosthet Dent*. 1978;39:324–329.
27. Ribeiro RF, Tallents RH, Katzberg RW, et al. The prevalence of disc displacement in symptomatic and asymptomatic volunteers aged 6 to 25 years. *J Orofac Pain*. 1997;11(1):37–47.
28. Nebbe B, Major PW. Prevalence of TMJ disc displacement in a pre-orthodontic adolescent sample. *Angle Orthod*. 2000;70(6):454–463.
29. Drace JE, Enzmann DR. Defining the normal temporomandibular joint: closed-, partially open-, and open-mouth MR imaging of asymptomatic subjects. *Radiology*. 1990;177(1):67–71.
30. Magnusson T. Five-year longitudinal study of signs and symptoms of mandibular dysfunction in adolescents. *Cranio*. 1986;4(4):338–344.
31. Magnusson T, Carlsson GE, Egermark I. Changes in clinical signs of craniomandibular disorders from the age of 15 to 25 years. *J Orofac Pain*. 1994;8(2):207–215.
32. Magnusson T, Egermark I, Carlsson GE. A longitudinal epidemiologic study of signs and symptoms of temporomandibular disorders from 15 to 35 years of age. *J Orofac Pain*. 2000;14(4):310–319.
33. Kononen M, Waltimo A, Nystrom M. Does clicking in adolescence lead to painful temporomandibular joint locking? *Lancet*. 1996;347(9008):1080–1081.
34. de Leeuw R, Boering G, Stegenga B, de Bont LG. Clinical signs of TMJ osteoarthrosis and internal derangement 30 years after nonsurgical treatment. *J Orofac Pain*. 1994;8(1):18–24.
35. de Leeuw R, Boering G, Stegenga B, de Bont LG. Symptoms of temporomandibular joint osteoarthrosis and internal derangement 30 years after non-surgical treatment. *Cranio*. 1995;13(2):81–88.
36. de Leeuw R, Boering G, Stegenga B, de Bont LG. Radiographic signs of temporomandibular joint osteoarthrosis and internal derangement 30 years after nonsurgical treatment. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 1995;79(3):382–392.
37. Randolph CS, Greene CS, Moretti R, Forbes D, Perry HT. Conservative management of temporomandibular disorders: a posttreatment comparison between patients from a university clinic and from private practice. *Am J Orthod Dentofacial Orthop*. 1990;98(1):77–82.
38. Lundh H, Westesson PL, Kopp S. A three-year follow-up of patients with reciprocal temporomandibular joint clicking. *Oral Surg Oral Med Oral Pathol*. 1987;63(5):530–533.
39. Salonen L, Hellden L, Carlsson GE. Prevalence of signs and symptoms of dysfunction in the masticatory system: an epidemiologic study in an adult Swedish population. *J Craniomandib Disord*. 1990;4(4):241–250.
40. Greene CS, Laskin DM. Long-term evaluation of treatment for myofascial pain-dysfunction syndrome: a comparative analysis. *J Am Dent Assoc*. 1983;107(2):235–238.
41. Greene CS, Laskin DM. Long-term status of TMJ clicking in patients with myofascial pain and dysfunction. Published erratum appears in *J Am Dent Assoc*. October 1988;117(5):558. *J Am Dent Assoc*. 1988;117(3):461–465.
42. Brooke RI, Grainger RM. Long-term prognosis for the clicking jaw. Published erratum appears in *Oral Surg Oral Med Oral Pathol*, February 1989;67(2):131. *Oral Surg Oral Med Oral Pathol*. 1988;65(6):668–670.
43. Berteretche MV, Foucart JM, Meunier A, Carpentier P. Histologic changes associated with experimental partial anterior disc displacement in the rabbit temporomandibular joint. *J Orofac Pain*. 2001;15(4):306–319.

44. Legrell PE, Isberg A. Mandibular height asymmetry following experimentally induced temporomandibular joint disk displacement in rabbits. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 1998;86(3):280–285.
45. Bryndahl F, Eriksson L, Legrell PE, Isberg A. Bilateral TMJ disk displacement induces mandibular retrognathia. *J Dent Res*. 2006;85(12):1118–1123.
46. Bjork A, Skieller V. Normal and abnormal growth of the mandible. A synthesis of longitudinal cephalometric implant studies over a period of 25 years. *Eur J Orthod*. 1983;5(1):1–46.
47. Ikeda K, Kawamura A. Assessment of optimal condylar position with limited cone-beam computed tomography. *Am J Orthod Dentofacial Orthop*. 2009;135(4):495–501.
48. Graf H. Bruxism. *Dent Clin North Am*. 1969;13(3):659–665.
49. Christensen J. Effect of occlusion-raising procedures on the chewing system. *Dent Pract Dent Rec*. 1970;10:233–238.
50. Rugh JD, Drago CJ. Vertical dimension: a study of clinical rest position and jaw muscle activity. *J Prosthet Dent*. 1981;45:670–675.
51. Christensen LV, Mohamed SE, Harrison JD. Delayed onset of masseter muscle pain in experimental tooth clenching. *J Prosthet Dent*. 1982;48:579–584.
52. Manns A, Miralles R, Cumsille F. Influence of vertical dimension on masseter muscle electromyographic activity in patients with mandibular dysfunction. *J Prosthet Dent*. 1985;53(2):243–247.
53. Manns A, Miralles R, Santander H, Valdivia J. Influence of the vertical dimension in the treatment of myofascial pain-dysfunction syndrome. *J Prosthet Dent*. 1983;50(5):700–709.
54. Oakley ME, McCreary CP, Clark GT, et al. A cognitive-behavioral approach to temporomandibular dysfunction treatment failures: a controlled comparison. *J Orofac Pain*. 1994;8(4):397–401.
55. Turner JA, Mancl L, Aaron LA. Short- and long-term efficacy of brief cognitive-behavioral therapy for patients with chronic temporomandibular disorder pain: a randomized, controlled trial. *Pain*. 2006;121(3):181–194.
56. Dworkin SF, Turner JA, Mancl L, et al. A randomized clinical trial of a tailored comprehensive care treatment program for temporomandibular disorders. *J Orofac Pain*. 2002;16(4):259–276.
57. Stockstill JW. The placebo effect. The placebo effect in the management of chronic myofascial pain: a review. *J Am Coll Dent*. 1989;56(2):14–18.
58. Dao TT, Lavigne GJ, Charbonneau A, Feine JS, Lund JP. The efficacy of oral splints in the treatment of myofascial pain of the jaw muscles: a controlled clinical trial. *Pain*. 1994;56(1):85–94.
59. Greene CS, Laskin DM. Meprobamate therapy for the myofascial pain-dysfunction (MPD) syndrome: a double-blind evaluation. *J Am Dent Assoc*. 1971;82(3):587–590.
60. Greene CS, Laskin DM. Splint therapy for the myofascial pain–dysfunction (MPD) syndrome: a comparative study. *J Am Dent Assoc*. 1972;84(3):624–628.
61. Le Bell Y, Niemi PM, Jamsa T, Kylmala M, Alanen P. Subjective reactions to intervention with artificial interferences in subjects with and without a history of temporomandibular disorders. *Acta Odontol Scand*. 2006;64(1):59–63.
62. Koh H, Robinson PG. Occlusal adjustment for treating and preventing temporomandibular joint disorders. *J Oral Rehabil*. 2004;31(4):287–292.
63. Okeson JP. *Management of Temporomandibular Disorders and Occlusion*. 7th ed. St. Louis, MO: Elsevier/Mosby Publishers; 2013:102–128.
64. Kato T, Montplaisir JY, Guitard F, et al. Evidence that experimentally induced sleep bruxism is a consequence of transient arousal. *J Dent Res*. 2003;82(4):284–288.
65. Lavigne GJ, Kato T, Kolta A, Sessle BJ. Neurobiological mechanisms involved in sleep bruxism. *Crit Rev Oral Biol Med*. 2003;14(1):30–46.
66. Cassisi JE, McGlynn FD, Mahan PE. Occlusal splint effects on nocturnal bruxing: an emerging paradigm and some early results. *Cranio*. 1987;5(1):64–68.
67. Holmgren K, Sheikholeslam A, Riise C. Effect of a full-arch maxillary occlusal splint on parafunctional activity during sleep in patients with nocturnal bruxism and signs and symptoms of craniomandibular disorders. *J Prosthet Dent*. 1993;69(3):293–297.
68. Sheikholeslam A, Holmgren K, Riise C. A clinical and electromyographic study of the long-term effects of an occlusal splint on the temporal and masseter muscles in patients with functional disorders and nocturnal bruxism. *J Oral Rehabil*. 1986;13(2):137–145.
69. Pierce CJ, Gale EN. A comparison of different treatments for nocturnal bruxism. *J Dent Res*. 1988;67(3):597–601.
70. Whitney CW, Von Korff M. Regression to the mean in treated versus untreated chronic pain. *Pain*. 1992;50:281–285.
71. Davies SJ, Gray RJ. The pattern of splint usage in the management of two common temporomandibular disorders. Part II: the stabilisation splint in the treatment of pain dysfunction syndrome. *Br Dent J*. 1997;183(7):247–251.
72. Okeson JP. *Management of Temporomandibular Disorders and Occlusion*. 7th ed. St. Louis, MO: Elsevier/Mosby Publishers; 2013:395–396.
73. Okeson JP. *Management of Temporomandibular Disorders and Occlusion*. 7th ed. St. Louis, MO: Elsevier/Mosby Publishers; 2013:258–290.
74. Carlson C, Bertrand P, Ehrlich A, Maxwell A, Burton RG. Physical self-regulation training for the management of temporomandibular disorders. *J Orofac Pain*. 2001;15:47–55.
75. Pettengill CA, Growney MR Jr, Schoff R, Kenworthy CR. A pilot study comparing the efficacy of hard and soft stabilizing appliances in treating patients with temporomandibular disorders. *J Prosthet Dent*. 1998;79(2):165–168.
76. Wright E, Anderson G, Schulte J. A randomized clinical trial of intraoral soft splints and palliative treatment for masticatory muscle pain. *J Orofac Pain*. 1995;9(2):192–199.
77. Okeson JP. *Management of Temporomandibular Disorders and Occlusion*. 7th ed. St. Louis, MO: Elsevier/Mosby Publishers; 2013:415–416.
78. Okeson JP. *Management of Temporomandibular Disorders and Occlusion*. 7th ed. St. Louis, MO: Elsevier/Mosby Publishers; 2013:317–361.
79. Okeson JP. Joint intracapsular disorders: diagnostic and nonsurgical management considerations. *Dent Clin North Am*. 2007;51(1):85–103.

14

The Orthodontist's Role in a Cleft Palate–Craniofacial Team

Katherine W.L. Vig and Ana M. Mercado

OUTLINE

Diagnostic Considerations, 368
Prenatal Diagnosis of Cleft Lip/Palate, 368
Ultrasound Technique and Limitations, 368
Advantages of Prenatal Cleft Diagnosis, 368
Disadvantages of Prenatal Cleft Diagnosis, 368
The Team Approach, 368
Role of the Orthodontist, 369
Neonate and Infant (Birth to 2 Years of Age), 369
Primary Dentition Stage (2 to 6 Years of Age), 372
Mixed Dentition Stage (7 to 12 Years of Age), 375
Primary Alveolar Bone Grafting, 377
Secondary Alveolar Bone Grafting, 377
Permanent Dentition Stage, 382
Growth Considerations, 382
Skeletal-Facial Considerations, 383
Orthognathic Surgery, 387
Role of the Orthodontist, 388
Distraction Osteogenesis, 388
Management of the Missing Lateral Incisor Space, 390
Summary, 392
Acknowledgements, 392

Historically, children who were born with facial clefting in addition to other manifested craniofacial anomalies underwent a succession of evaluations and hospitalizations by their independent caregivers. This individualized delivery of care, although considered in the best interests of the patients, resulted in additional hospital admissions and multiple instances of general anesthetic administration. The American Cleft Palate–Craniofacial Association (ACPA) was established in 1943 to foster a team approach and to serve as an advocate for patients with clefts of the lip and/or palate and their families. The association defined the role of the orthodontist on a cleft palate team and recognized a team approach as the most appropriate method to manage the care of patients with orofacial clefts. In 1972, craniofacial teams became established as an extension of the cleft palate team. This development was in response to clinical geneticists and dysmorphologists becoming increasingly aware that orofacial clefts were part of a phenotypic spectrum of craniofacial anomalies.[1]

Currently in the United States, care for individuals with craniofacial anomalies, including orofacial clefs, is delivered in a variety of collaborative models: hospital-based teams, university-based teams, or in independent clinics where multidisciplinary evaluations and treatment are coordinated. The ACPA has published the document "Standards for Cleft Palate and Craniofacial Teams," which describes the essential quality characteristics for team composition and functioning (http://www.acpa-cpf.org/team_care/standards/). The ACPA Commission on Approval of Teams reviews and identifies those cleft palate–craniofacial teams that meet the specified standards. The Cleft Palate Foundation maintains a list of approved cleft palate–craniofacial teams in the United States and around the world (http://www.cleftline.org/parents-individuals/team-care/) and provides information about their services to patients and families.

The team approach to comprehensive care requires the orthodontist to work collaboratively to determine the appropriate timing and sequencing of treatment in the context of the patient's other health care needs. This interactive, evidence-based, and patient-centered care provides the basis for a rational approach to diagnosis and treatment planning. Just as multiple methods and alternative treatment interventions are available, so also the team approach to management advocates that patients and their parents be aware of the choices through a risk-cost-benefit appraisal. This appraisal allows patients to make informed decisions and to understand the consequences of the different options available, especially in light of emerging technologies and treatment modalities for which long-term outcomes are not available. The document "Parameters for Evaluation and Treatment of Patients with Cleft Lip/Palate or other Craniofacial Anomalies"[2] was the product of a consensus conference in 1992 to develop guidelines and practices in the care of patients with craniofacial anomalies. The parameters, revised in 2009,[3] also serve as guidelines for the patient-oriented clinical management of those patients with craniofacial anomalies.

The purpose of this chapter is to consider a rational team approach to the orthodontic management of the patient with cleft lip and/or palate.[4,5] Orthodontic interventions should be confined to discrete stages in skeletodental development of the craniofacial complex and should not be considered as a continuum of treatment from birth to adulthood.

DIAGNOSTIC CONSIDERATIONS

Clefts of the lip and palate occur in approximately 1:700 live births in the United States and are the fourth most common craniofacial birth defect. The incidence varies in different races, and prevalence of cleft type varies by gender. The estimated cost of treatment is about $108,000 for this craniofacial birth defect compared with other birth defects. The high total cost of cleft lip/palate is a reflection of its incidence and also relates to the associated morbidity and mortality.[6]

Cleft lip and/or palate may be diagnosed by prenatal ultrasound, which has become more common because refinements of this diagnostic tool provide images by which congenital and developmental anomalies may be detected more clearly. The ability to make the diagnosis in utero gives the parents the opportunity to be prepared for the birth of their infant with a facial anomaly and to understand the surgical procedures available for repair of the facial cleft. Prenatal counseling provides parents with both a level of awareness and realistic expectations at the time of the delivery. If the diagnosis of a craniofacial anomaly has not been identified prenatally, parents may experience an overwhelming disappointment and sense of guilt when the obstetrician or neonatologist informs them of their baby's birth defect. However, the distinction between a syndromic/nonsyndromic facial cleft and its recurrence risk is the domain of the geneticist, dysmorphologist, or syndromologist, who provides this information to the parents and later to the patient at the appropriate time in his or her maturation.[7]

Prenatal Diagnosis of Cleft Lip/Palate

Ultrasonography is a noninvasive diagnostic tool widely used as a routine component of prenatal care. Ultrasonography serves to confirm fetal viability, determine gestational age, establish the number of fetuses and their growth, check placental location, and examine fetal anatomy to detect any malformations.[8]

Ultrasound Technique and Limitations

Christ and Meininger[9] reported that optimal imaging of the fetal face is not reliable with transabdominal ultrasound until gestational week 15. The position of the face and the disproportionate size of the transducer and fetus make it more difficult to show the anatomy of the fetal nose and lips before that point of gestation. Robinson and coworkers[10] reported that the detection rate for fetal cleft lip is improved when transabdominal sonography is performed at or after 20 weeks of gestation. At that time the lips become more developed and prominent as the fetus grows, allowing for better imaging as long as there is an amniotic fluid interface and no obstructions. Transvaginal sonography, although not routinely used, has been reported to allow earlier visualization of the face and better image resolution, yielding high specificity and sensitivity of prenatal cleft lip detection.[11]

Babcock and McGahan[12] described a systematic approach that allows for fast and thorough sonographic evaluation of the midface structures. Starting with the coronal plane, the soft tissues of the fetal nares and upper lip are examined for continuity (Fig. 14-1, *A*). Once a cleft is detected in the coronal view, anatomic assessment continues in the axial plane, evaluating the superficial skin margin of the upper lip, the underlying orbicularis oris muscle, and tooth-bearing alveolar ridge (Fig. 14-1, *B*). An isolated cleft lip is diagnosed if discontinuity exists in the upper lip with no evidence of disruption in the underlying alveolar ridge. Cleft lip and palate is diagnosed if a cleft is identified in the lip and disruption of the alveolar ridge, whether unilateral or bilateral, is present. Bilateral clefts of lip and palate are well visualized in the sagittal plane because protrusion of the premaxillary segment is evident.[12,13] Isolated clefts of the hard palate are viewed best in the axial plane (Fig. 14-1, *C*).[14] Acoustic shadowing from facial bones, which obscure the palatal structures, makes isolated clefts of the hard palate particularly difficult to visualize and diagnose prenatally.[12] Movement of the tongue above the level of the hard palate may suggest the presence of a secondary palate cleft.[15,16] Color Doppler ultrasonography has been used to visualize abnormal flow of amniotic fluid from the mouth to the nasal cavity, a finding that may suggest the presence of a palatal cleft.[17] Factors that may limit the sensitivity of diagnosing an orofacial cleft during ultrasound screening include an unfavorable position of the fetus, hand or umbilical cord overlying the face, maternal obesity, presence of a multiple gestation, oligohydramnios (reduced amniotic fluid), prior abdominal surgery, and the presence of additional fetal abnormalities.[10,18]

Advantages of Prenatal Cleft Diagnosis

There are several potential advantages for informing parents of a prenatal diagnosis of facial clefting[18]:

1. Psychological preparation of parents and caregivers to allow for realistic expectations at the time of delivery.
2. Education of parents on the management of the cleft: presurgical neonatal orthopedics, plastic surgery for lip and palate closure, and alveolar bone grafting.
3. Preparation for neonatal care and feeding.
4. Opportunity to investigate for other structural or chromosomal abnormalities.
5. Possibility for fetal surgery.

An additional advantage of prenatal diagnosis of cleft lip and palate is the ability of the plastic surgeon to prepare a customized plan of management for surgical repair of the cleft once the sonologist characterizes the specific type of cleft and describes the extent of the anomaly. With this information, the plastic surgeon may wish to educate the parents about the severity of the deformity, the need for any adjunctive intervention before surgery, and the predicted outcome of repair.[16]

Disadvantages of Prenatal Cleft Diagnosis

Parents and professionals report an emotional disturbance and high maternal anxiety after prenatal diagnosis of cleft lip/palate is disclosed.[19,20] However, parents of affected children strongly favor being informed and involved in prenatal testing and counseling decisions and view this preparation as valuable despite acknowledging the increased anxiety and dysfunction during pregnancy.[21] As the sensitivity of ultrasound screening in the detection of facial clefts increases, the potential exists for an increased number of families choosing to terminate the pregnancy even in the absence of other malformations.[18] Factors such as perceived burden, expectation of recurrence, religious and cultural beliefs, professional advice, and gestational age at diagnosis are considered influential in the family's decision to terminate pregnancy.[20,21]

THE TEAM APPROACH

The timing and sequencing of orthodontic treatment are not carried out in isolation from other members of the team but

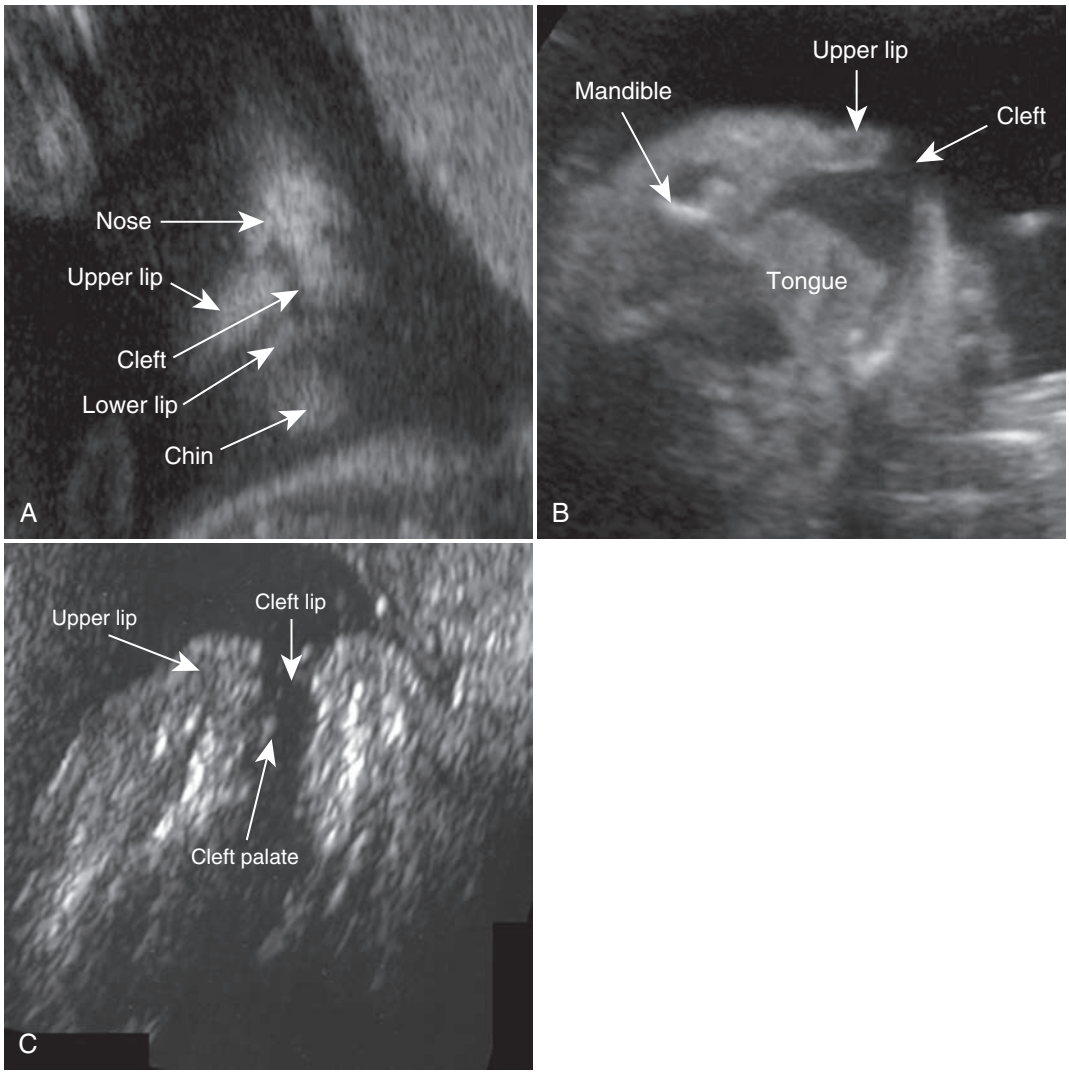


FIGURE 14-1 Transabdominal ultrasonography images of the midface of a fetus with unilateral cleft lip at 21 weeks of gestational age. **A,** Two-dimensional, coronal view: the defect in the upper lip extends into the nostril. **B,** Two-dimensional, axial view at the tongue level: discontinuity in the upper-lip skin and muscle is observed. **C,** Axial view. Discontinuity of the upper lip, alveolar ridge, and hard palate can be visualized.

as a result of collaborative decisions made in a coordinated, patient-centered manner sensitive to the patient's and family's needs (Fig. 14-2). Several texts provide specific details of treatment intervention, but the overall care of affected infants should rely on interdisciplinary team decisions rather than a series of independent, critical events by individual specialists on a team.[22]

The orthodontist serving on a cleft palate team should consider additional priorities other than malocclusion. The timing and sequencing of treatment should be sensitive to other interventions by specialists on the team to provide the affected individual with a patient-centered interdisciplinary approach that follows critical pathways.[23,24] These critical pathways have been well defined in a document on critical elements of care for children with special health problems developed by the Washington State Department of Health in 1997.[25] These guidelines were developed through a consensus process including primary and tertiary care providers, family members, and representatives from a health insurance plan.

ROLE OF THE ORTHODONTIST

Timing and sequencing of orthodontic care may be divided into four distinct developmental periods. These periods are defined by age and dental development and should be considered as time frames in which to accomplish specific objectives. Such sequencing avoids the common tendency to allow an early phase of treatment intervention to extend through infancy, childhood, adolescence, and into adulthood. With the understanding that children born with cleft lip and/or palate should be treated by an interdisciplinary team approach, the following four time periods in the child's development provide a framework for discussing and recommending defined objectives.

Neonate and Infant (Birth to 2 Years of Age)

Presurgical orthodontics or neonatal maxillary orthopedics is initiated during the first or second week following birth unless complications arise from other congenital anomalies or medical problems.[26-28] This treatment may be carried out by the

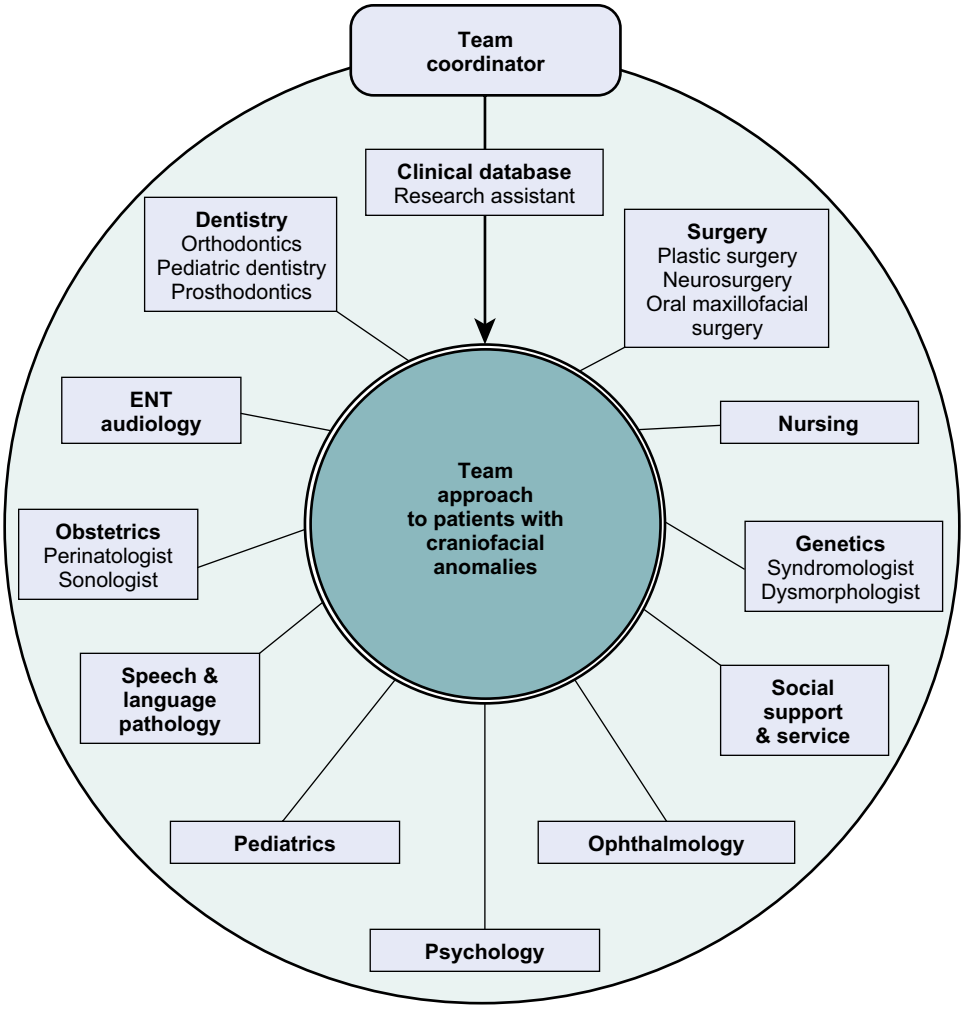


FIGURE 14-2 Members in the team approach to patients with craniofacial anomalies. (Reprinted from Vig KWL, Mercado AM. Contemporary management of craniofacial anomalies: Will past experiences influence and predict the future? In: McNamara JA Jr, ed. *The 40th Moyers Symposium: Looking Forward … Looking Back*. Ann Arbor: Department of Orthodontics and Pediatric Dentistry and Center for Human Growth and Development, The University of Michigan; 2014. Monograph 50, Craniofacial Growth Series.)

orthodontist, the pediatric dentist, or the prosthodontist. The popularity of this early intervention was well accepted in the 1960 and 1970s to eliminate the need for subsequent orthodontic treatment after the segments were aligned. Later reports suggested that although the initial results of lip repair were easier to attain with cosmetic improvement, the procedure seemed to accrue no long-term benefit on the growth of the midface and dentoalveolus. Growth of the nasomaxillary complex and the occlusal results in the primary and mixed dentition appeared clinically similar to those cases without this early treatment intervention. Additionally, the literature also reported that primary bone grafting and associated presurgical maxillary orthopedics further confounded the cause-and-effect relationship.[29] Early or primary bone grafting associated with maxillary orthopedics at the time of primary lip repair may have compromised the long-term follow-up of treated patients. Results suggested that neonatal maxillary orthopedics produced little effect on the developing malocclusions if assessed when the child was 10 years old, especially if treatment had included primary bone grafting to stabilize and prevent maxillary collapse in the infant.[30-32]

Because no long-term benefit was to be gained from these interventions when used indiscriminately on every affected infant, the use of neonatal orthopedics before definitive primary surgical lip repair became an ongoing controversy between clinicians.[30,33] Several institutions continue to advocate early bone grafting and report substantial benefits.[28,34] The perspective of contemporary wisdom is that when provided as an adjunctive procedure to primary definitive lip repair, neonatal maxillary orthopedics does have presurgical benefits. Therefore, the popularity of neonatal maxillary orthopedics has returned, albeit with different objectives than to eliminate future need of orthodontic treatment for the child. A considerable literature has developed on this procedure, and the enthusiasm of many clinicians in this field attests to the variety and complexity of the appliances.[24,35-38] The feature that all these appliances, whether fixed or removable, have in common is the ability to adjust the position of the cleft segments into a more ideal relationship before definitive surgical repair of the lip.

In the 1990s, there was renewed enthusiasm for neonatal maxillary orthopedics with the introduction of Naso-Alveolar

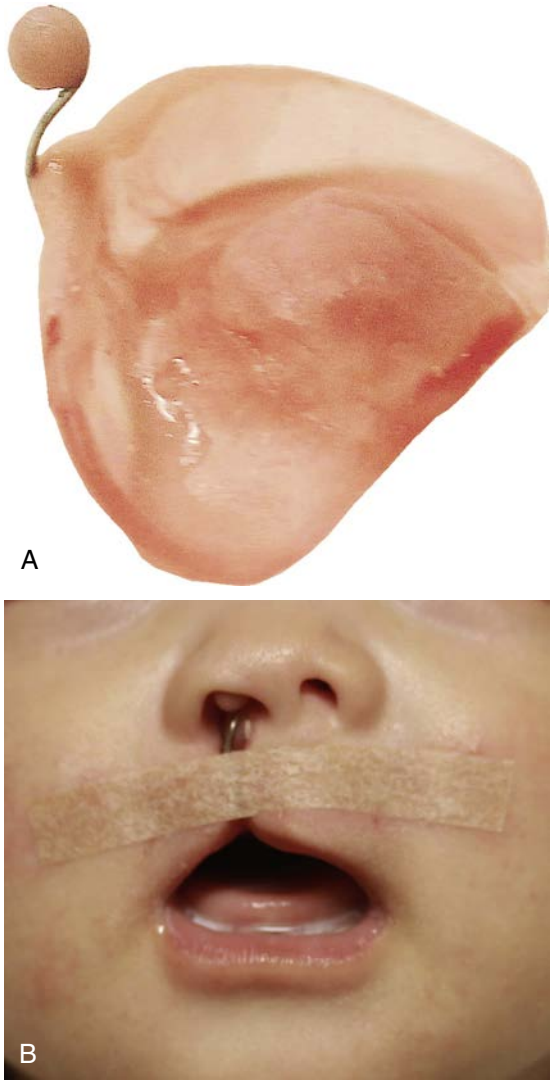


FIGURE 14-3 Presurgical nasoalveolar molding. A, Custom intraoral plate to mold the alveolar segments. From the labial vestibular flange, there is a wire extension with acrylic at the tip that serves as a nasal stent to mold the nasal alar cartilage. B, Molding plate in place in a neonate with unilateral cleft lip and palate. Lip taping is applied to approximate the lip segments.

Molding (NAM). This technique consists of an intraoral molding plate, nasal stents, and extraoral taping.[35,36] The alveolar ridges are molded to reduce the size of the cleft to approximately 1 to 2 mm of each other. Concomitantly, the use of nasal stents and taping lengthens the columella and realigns of the lower lateral alar cartilages (Figs. 14-3, *A* and *B*, and 14-4, *A* to *D*). The goal is to prepare the infant for a one-stage primary lip–nose repair in combination with a gingivoperiosteoplasty surgery to close the alveolar defect. It is advocated that the reorientation and molding of cartilages that is achieved with NAM may eliminate or reduce future surgical procedures for the nose. Longitudinal follow-up from infancy into adolescence is necessary to evaluate the long-term effect of presurgical maxillary orthopedics within the context of overall nasomaxillary growth.

European investigators have created the Dutchcleft, which is the Dutch Inter-center prospective two-arm randomized clinical trial (RCT).[39] Dutchcleft was designed to study the outcome of neonatal maxillary orthopedics in subjects with unilateral cleft lip and palate (UCLP). In this trial the infant presurgical orthopedics technique consisted of passive intraoral plates adjusted every 3 weeks to mold the alveolar segments. The control group received no intraoral plates. Prahl and coworkers[40] found no significant difference between the randomized plate versus control group of infants up to 2 years of age on feeding, nutritional status, or somatic growth. Konst and coworkers[41] assessed speech development at age 2.5 years and found a significant improvement in children treated with intraoral plates compared to children who did not receive plates, with acceptable cost-effectiveness of the intervention in regards to speech development. Bongaarts and coworkers[42] reported that infant orthopedics had no significant effect in maxillary arch dimensions at ages 4 and 6 when compared to similar-aged children who had not been treated with infant orthopedics. Similarly, there was no significant effect of infant orthopedics on dental arch relationships at ages four and six including overjet, overbite, and sagittal occlusion.[43] Only minor group differences were revealed at age 6 by cephalometric analyses of soft tissue, skeletal, and dental structures.[44] In regards to facial appearance, group differences in facial appearance observed at age 4 were no longer observed at age 6.[45] The Dutchcleft investigators alerted that clinicians who promote different methods of infant orthopedics including NAM should evaluate the long-term effects of these interventions under the rigorous methodology of an RCT.[44] In 2011, Uzel and Alparslan[46] published a systematic review of the long-term effects of presurgical infant orthopedics. They analyzed eight RCTs and four controlled clinical trials (CCTs), with follow-up periods of up to 6 years. The systematic review did not show a positive effect of infant orthopedics on the treatment outcomes evaluated until the age of 6 years, including motherhood satisfaction, feeding, speech, facial growth, maxillary arch dimension, occlusion, and nasolabial appearance.[46]

The benefits from early presurgical orthopedic appliances need to be weighed against the increased burden of care because of the number of clinic visits necessary to adjust the appliance during the first year of life.[31,36] Neonatal maxillary orthopedics continues to be practiced in a number of centers in the United States and Europe, although contemporary opinion generally does not consider that the evidence supports the intervention as an essential or desirable routine procedure for all infants born with a facial cleft. Nonetheless, the molding of the segments achieved by these appliances does make definitive lip repair easier for the surgeon, especially for patients with a severely protruding premaxilla caused by a bilateral cleft lip.[24]

The results in aligning the cleft segments with appliances have been considered similar to those produced by surgical lip adhesion. Surgical lip adhesion is a partial lip repair procedure often reserved for wide, complete clefts to convert these into incomplete clefts.[47,48] Narrowing a wide cleft and aligning the alveolar segments under the compression forces of the partially repaired lip is advocated. When the alignment of the segments has been achieved following lip adhesion, then definitive lip repair with muscle continuity is performed. This early surgical repair of the lip by an adhesion technique has much to commend it because the parents are not required to remove or adjust appliances, the cosmetic appearance is

FIGURE 14-4 Neonate with complete unilateral left cleft lip and palate. **A,** Defect before any orthopedic or surgical intervention. The left lower alar cartilage is depressed. The nasal tip is displaced and the columella is deviated over the cleft. **B,** Intraoral view of palate before orthopedics or surgery. The alveolar segments are widely separated and not in alignment. **C,** Defect after presurgical nasoalveolar molding. The left alar cartilage is more elevated as well as the nasal tip. The columella is located more medially. **D,** Intraoral view of palate after nasoalveolar molding. Note close approximation of the alveolar segments.

improved with a minor initial surgical procedure, and postoperative care by the parents is minimal. The most serious problem with this approach is the potential of wound dehiscence and the need for an additional surgery.[29] Currently, lip adhesion is not typically performed, and contemporary primary surgical lip repair restores continuity of the mucosa, skin, and circumoral musculature in the infant's cleft lip with a single procedure.

Definitive lip repair usually is achieved by the time the infant is 3 to 6 months old, and repair of the palate typically is delayed until 12 months to 2 years of age. Palatal repair is another controversial issue, and many methods are available for repairing just the soft palate or the hard and soft palate simultaneously. The rationale for the timing of the palatal repair is related to the developing speech and language skills of the child, which typically evolve around the first year of age. This rationale is usually in conflict with the effect of early surgical repair and the constraints of scar tissue on the growth and development of the nasomaxillary complex. Early repair of the palate and the resulting scar tissue may have an effect on the growth and development of the maxilla, which is reflected in the occlusion as a crossbite of anterior and posterior teeth. The severity of the malocclusion has been associated with certain surgical methods of palate repair, and evidence from unrepaired clefts in children and adolescents indicates that crossbites in the dentition rarely develop in the absence of surgical repair and resulting scar tissue.

Primary Dentition Stage (2 to 6 Years of Age)

At 2 to 3 years of age, the establishment of the primary dentition permits classification of the type of developing malocclusion. This determination may be part of the diagnostic regimen in which the contribution of the skeletal and dental components may be identified.

The facial soft tissues may mask the underlying skeletal deficiency of the midface in young children (Fig. 14-5, *A* to *C*). Growth of the intermaxillary space in three dimensions results in a redistribution of the facial soft tissues as the chubby face of infancy takes on the more mature and defined facial proportions of the child. These facial characteristics start to unmask the underlying skeletal discrepancy more accurately than in the younger child. The dentition often reflects the skeletal relationship, especially if the dentoalveolar component (axial inclination of the teeth) has not compensated for any skeletal discrepancies. Dental compensation for maxillary skeletal deficiency may result in retroclination

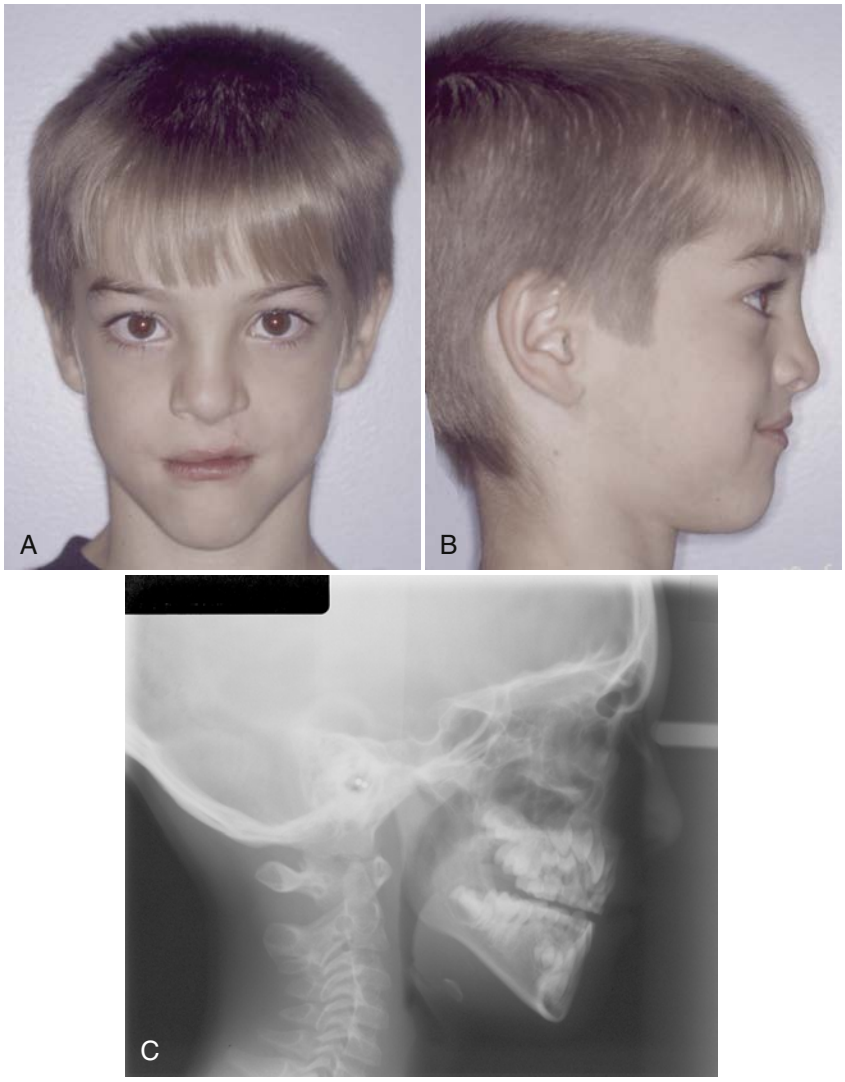


FIGURE 14-5 **A,** Frontal view of a 7-year-old boy with repaired unilateral left cleft lip and palate. **B,** Profile view showing mild bimaxillary retrusion. **C,** Lateral skull radiograph in early mixed dentition.

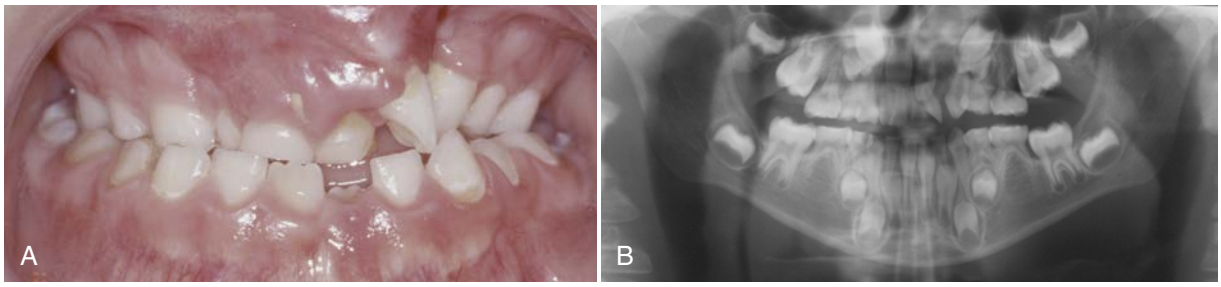


FIGURE 14-6 **A,** Intraoral view in occlusion of the same child as in Figure 14-5. Note the anterior crossbite and the rotated incisor mesial to the cleft. **B,** Panoramic radiograph indicating congenitally absent maxillary lateral incisors, maxillary second premolars, and mandibular second premolars.

of mandibular incisors with proclination of the maxillary incisors to mask the anteroposterior discrepancy, although in children with repaired orofacial clefts, the scar tissue following lip repair may constrain the maxillary incisor axial inclination (Fig. 14-6, *A* and *B*).

Because the primary incisors tend to be more upright than their successors, an anterior crossbite may be unilateral or bilateral with or without a functional shift of the mandible. This shift occurs when the child closes the teeth together. To eliminate mandibular shifts, orthodontic treatment may be

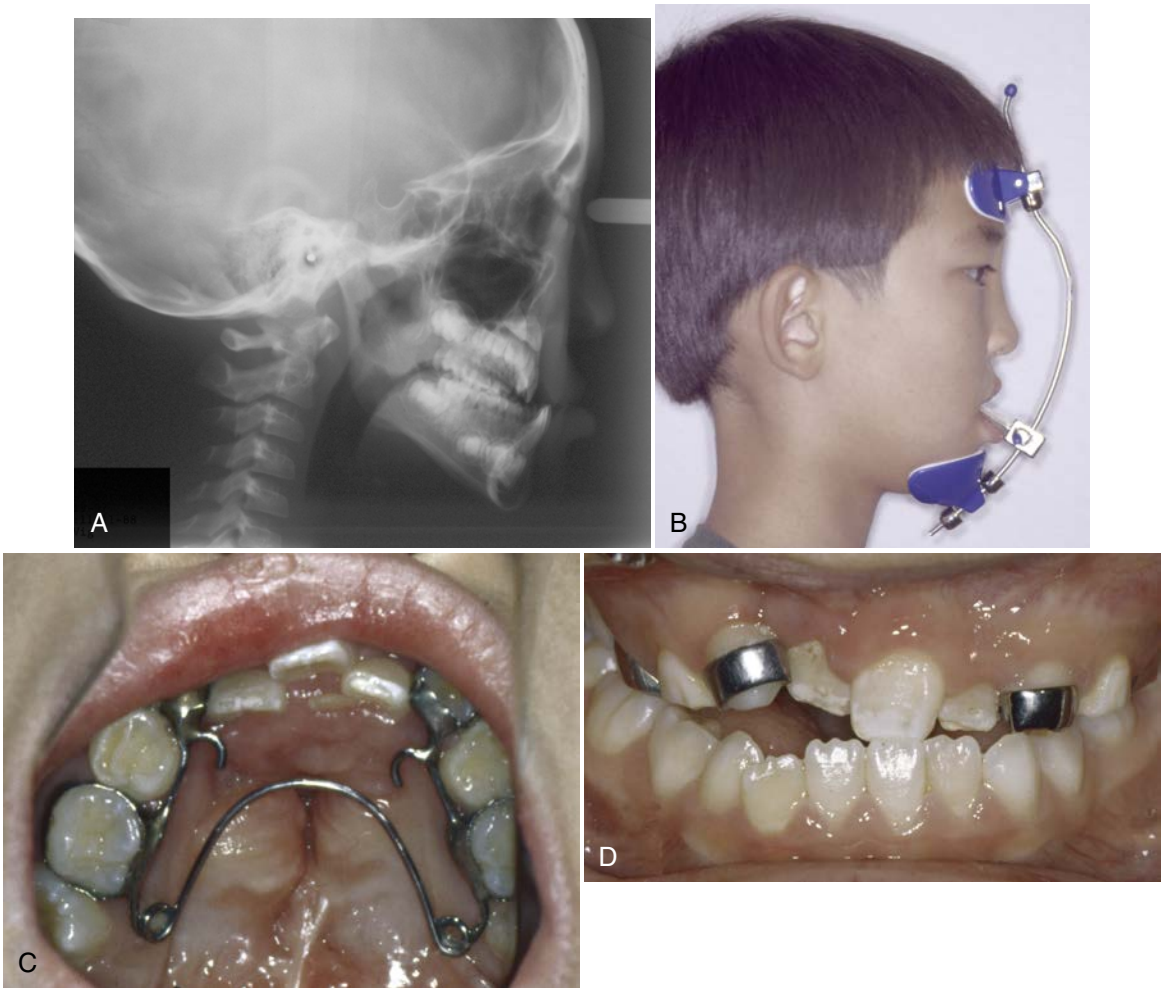


FIGURE 14-7 Eight-year-old boy with repaired unilateral right complete cleft lip and palate with sagittal and transverse maxillary deficiency. **A,** Lateral skull radiograph. Note the 7-mm reverse overjet. **B,** Protraction face mask with elastics attached to palatal hooks on expander. **C,** Palatal expander with bands cemented on the maxillary second primary molars and canines with palatal hooks to attach the protraction face mask. **D,** Lateral and anterior crossbites improving with palatal expansion and maxillary and dental protraction. (D, Adapted from Lidral AC, Vig KWL. The role of the orthodontist in the management of patients with cleft lip and/or palate. In: Wyszynski D, ed. *Cleft Lip and Palate: From Origin to Treatment.* New York: Oxford University Press; 2002.)

indicated to remove the interfering contact by tooth movement. This process may involve the maxillary incisors if an anterior crossbite exists or expansion of the posterior segments to eliminate a posterior crossbite (Fig. 14-7, *A* to *D*). If the dental crossbite relationship is a continuing problem once the dentition is established, it may be a reflection of the underlying skeletal discrepancy for which growth modification and redirection may be indicated with a protraction face mask (see Fig. 14-7, *B*). Some skeletal change occurred in Figure 14-8, *C*, with proclination of the maxillary incisors and forward movement of A point. However, with further growth the compensated incisal relationship may deteriorate. In cases of bilateral cleft lip and palate, severe constriction of maxillary posterior segments often is associated with bilateral crossbite and protrusion/extrusion of the premaxillary segment (Fig. 14-9).

The orthodontist should consider many factors in determining when to initiate orthodontic treatment during the primary dentition stage. These factors include the ability of the child to cooperate, the severity of the malocclusion, timing of secondary bone grafts, and the need for future orthodontic treatment in the early mixed or permanent dentitions. Contemporary opinion recognizes a need for orthodontic treatment in the early mixed and permanent dentitions. However, no strong evidence supports a benefit from routinely treating dental malocclusions in the primary dentition, suggesting that orthodontic treatment may be best delayed until it can be combined with other treatment goals and thus shorten the overall duration of treatment.

Severe skeletal discrepancies in the primary dentition are a more complex problem. Modification or redirection of growth has been advocated, and the use of functional or orthopedic appliances, including the forward protraction face mask, has been reported to have some success.[49,50] More commonly, the "apparent correction" is achieved by a transient change in the position of the teeth only, so that with subsequent growth,

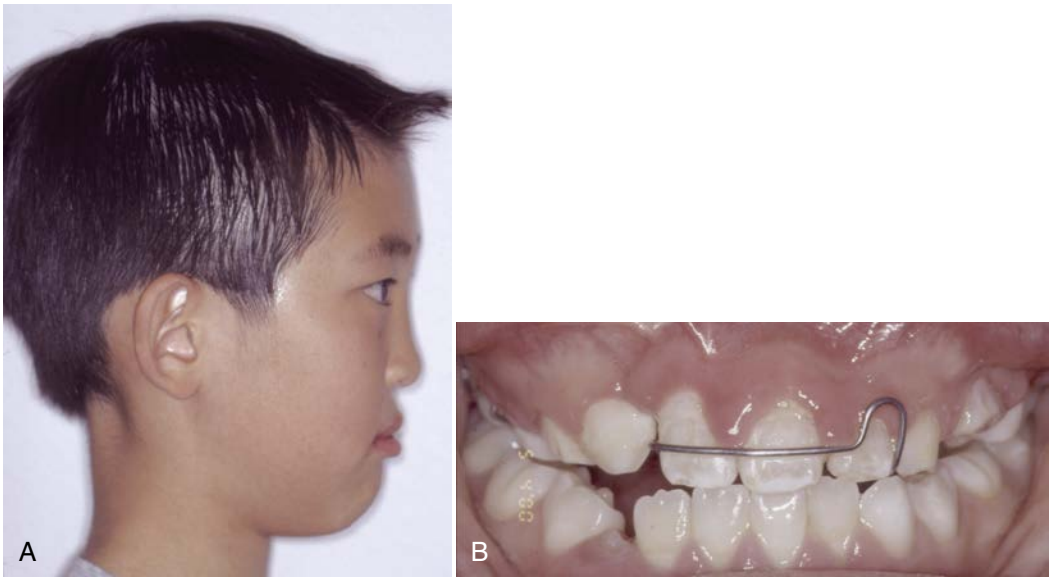


FIGURE 14-8 A, Facial profile after 9 months of protraction face-mask therapy and palatal expansion (same patient as in Fig. 14-7). B, Intraoral view with correction of anterior and posterior crossbites; maxillary retainer is in place. C, Superimposition of initial and postprotraction lateral cephalogram tracings showing correction of reverse overjet and mild maxillary advancement.

the skeletal discrepancy once again is reflected in the reestablishment of the malocclusion. Early treatment procedures, in common with neonatal maxillary orthopedics, require a long-term follow-up period to evaluate the outcome of treatment when the child reaches adolescence (see Fig. 14-8). Excessive changes attributable to therapeutic growth modification now are considered to be the exception rather than a predictable outcome of this early intervention.[51,52] One must carefully consider the severity of the skeletal discrepancy to determine the likelihood of successful growth modification and subsequent long-term results compared with conventional orthognathic surgery at a later stage. A more conservative option may be to provide a combined orthodontic/orthognathic surgery treatment plan than to promote long-term growth modification strategies that ultimately may not be successful.

Mixed Dentition Stage (7 to 12 Years of Age)

The transition to the mixed dentition starts at 6 to 7 years of age with the eruption of the first permanent molars and incisors. Further growth of the craniofacial complex often accentuates a previously mild skeletal discrepancy (Fig. 14-10). As the permanent teeth erupt, children are undergoing a period of psychosocial transition when friendships become more intimate and there is increased independence from parents.[53] In a

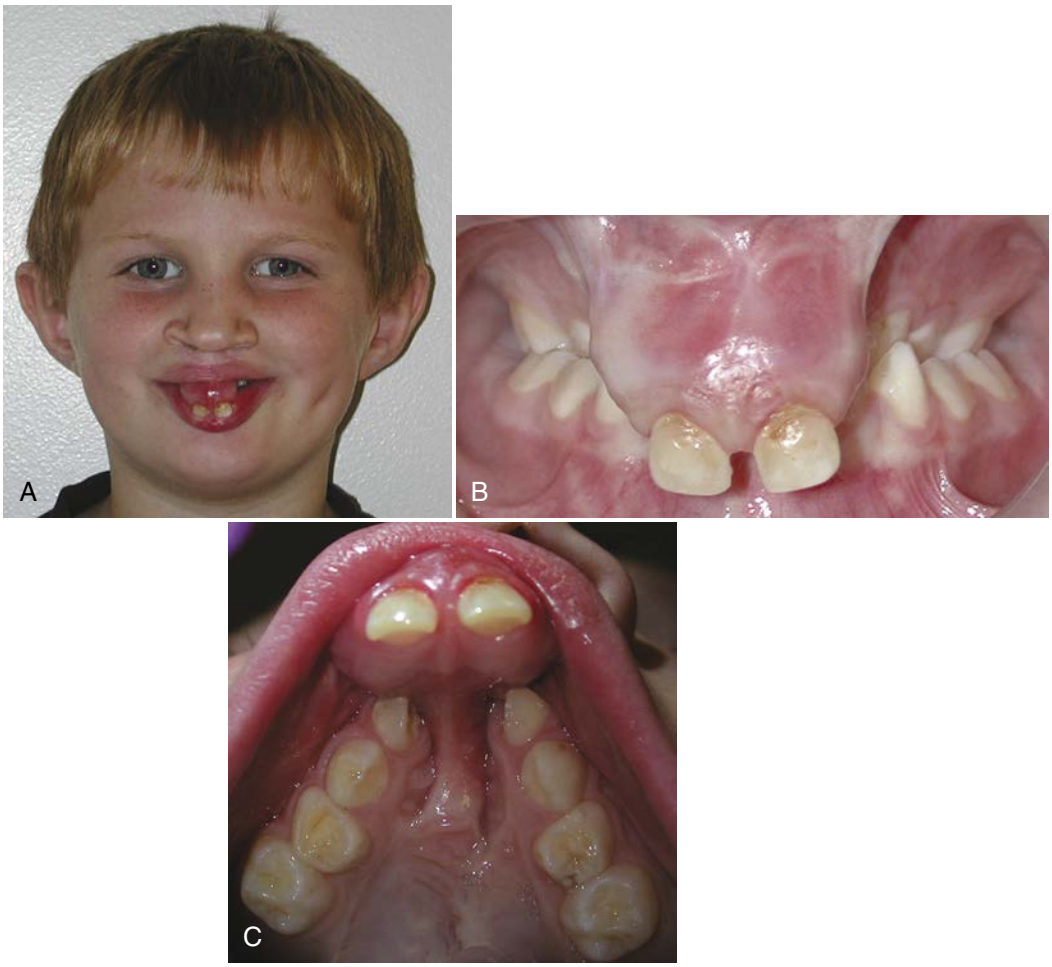


FIGURE 14-9 A, Five-year-old boy with repaired bilateral complete cleft lip and palate. Note the severe extrusion of premaxillary segment. B, Frontal intraoral view showing premaxillary segment out of the plane of occlusion. C, Maxillary occlusal view showing severe constriction of posterior segments and extrusion of premaxillary segment.

study by Ward and coworkers,[54] it was found that the presence of an orofacial cleft decreases the Oral Health-Related Quality of Life (OHRQoL) in children and adolescents. The dissatisfaction with appearance experienced by preadolescents with craniofacial anomalies is related to social withdrawal, social anxiety, and self-consciousness.[55] It is important for the health professionals on the team to be cognizant and empathetic of the psychosocial challenges that children and adolescents with craniofacial anomalies experience. Such challenges may influence the youngsters' participation in treatment decisions and their adherence to treatment protocols.

As the permanent incisors erupt adjacent to the cleft site, they typically are rotated, misplaced, malformed, or hypoplastic. In addition, incisors may be supernumerary, absent, or peg shaped. These characteristics are considered the result of early disruption of the dental lamina at the cleft site reflected in the developing tooth germs (Figs. 14-11 and 14-12). Constriction of the maxilla with a characteristic V-shaped arch form contributes to the posterior crossbite relationships usually seen in the mixed dentition. Tooth-borne maxillary expansion appliances such as a rapid palatal expander, a W-arch, or a Quad-helix can be anchored on the permanent first molars and extended anteriorly to improve arch form while correcting the crossbite (Fig. 14-13). By adding hooks bilaterally to these appliances, a protraction face mask may be used during expansion to treat a mild to moderate skeletal midface deficiency. The correction of an anterior crossbite results from dentoalveolar proclination of incisors with usually a slight and transient skeletal advancement. By incorporating miniplates and miniscrews, the intraoral appliances become bone-borne allowing the orthopedic force from the protraction face mask to be transmitted to the maxilla rather than to the teeth. Such an approach was developed by DeClerck and coworkers[56] and Baek and coworkers,[57] placing miniplates on the zygomatric buttress as anchorage for skeletal maxillary protraction and placing miniscrews in the anterior mandible and in the posterior maxilla to allow the use of intraoral Class III elastics. Both approaches have resulted in favorable short-term orthopedic changes in maxilla while minimizing common side effects such as proclination of the incisors, molar extrusion, bite opening, or clockwise rotation of the mandibular plane.

Because deficiency of tissue is an inevitable consequence of facial clefting, not only are the teeth missing, but the supporting alveolar bone at the cleft site is also compromised. In the past, rehabilitation of the maxillary dentition depended on the expertise of the prosthodontist to replace the missing teeth and

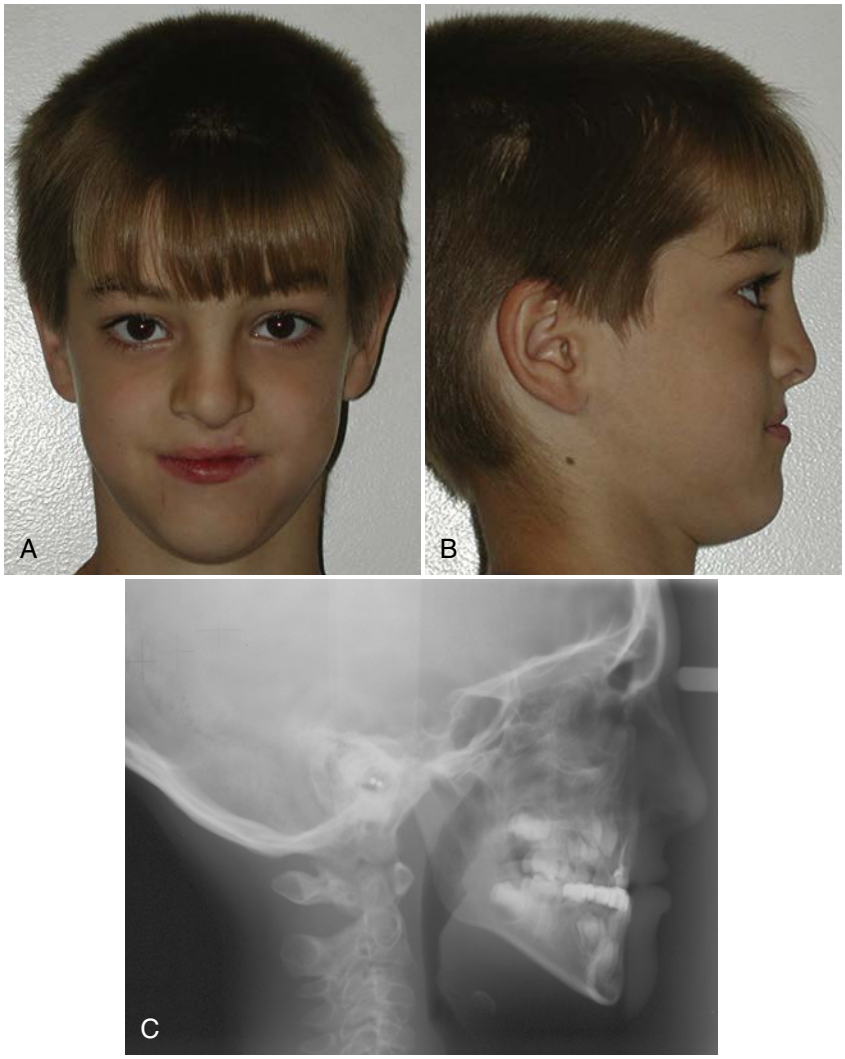


FIGURE 14-10 A, Same patient as in Figures 14-5 and 14-6 shown at 9 years of age. B, Profile view showing mild midfacial deficiency. C, Lateral skull radiograph in mixed dentition with anterior crossbite.

alveolus in the cleft defect with an overdenture. The challenge to restore the missing tissue at the cleft site was resolved with the advent of secondary alveolar bone grafting in the 1970s.[58-60] This bone grafting procedure provided the orthodontist with one of the most important milestones in managing the cleft site: restoration of an uninterrupted, continuous alveolar ridge that allows for eruption of teeth into the graft and orthodontic movement of teeth into the cleft site (Fig. 14-14). Additionally, placement of osseointegrated implants is now possible with prosthetic replacement of missing teeth. The elimination of the residual cleft provided a major advance in the contemporary management of the cleft maxilla and is an example of the outcome of a coordinated and problem-oriented approach to developing new strategies in treatment protocols.[58,61,62]

Primary Alveolar Bone Grafting

Primary bone grafting is performed in the infant cleft site before eruption of the primary incisors, usually at the time of primary surgical lip repair. In the United States, primary alveolar bone grafting was discontinued following a 5-year posttreatment outcome study in 1972 by Jolleys and Robertson,[30] who reported that patients with complete clefts of the lip and palate who had received primary bone grafting had limitation of maxillary growth compared to a control group of patients with clefts but no primary bone grafting. In Eurocleft, the study of treatment outcomes among five European cleft palate centers, it was found that the only center performing primary alveolar bone grafting obtained the worst dentoalveolar relationships suggesting growth impairment of the maxilla.[63] Similarly, poor dentoalveolar outcomes in a center using primary bone grafting were reported by the Americleft intercenter study in North America.[64] Postponing bone grafting surgery until more maxillary growth has occurred is preferred among most cleft palate centers.[65]

Secondary Alveolar Bone Grafting

By definition secondary or delayed alveolar bone grafting is performed after primary lip repair.[59,60] The age at which the bone graft is placed defines whether it is early secondary bone grafting (2 to 5 years), intermediate or secondary bone grafting (6 to 15 years), or late secondary bone grafting (adolescence to adulthood).

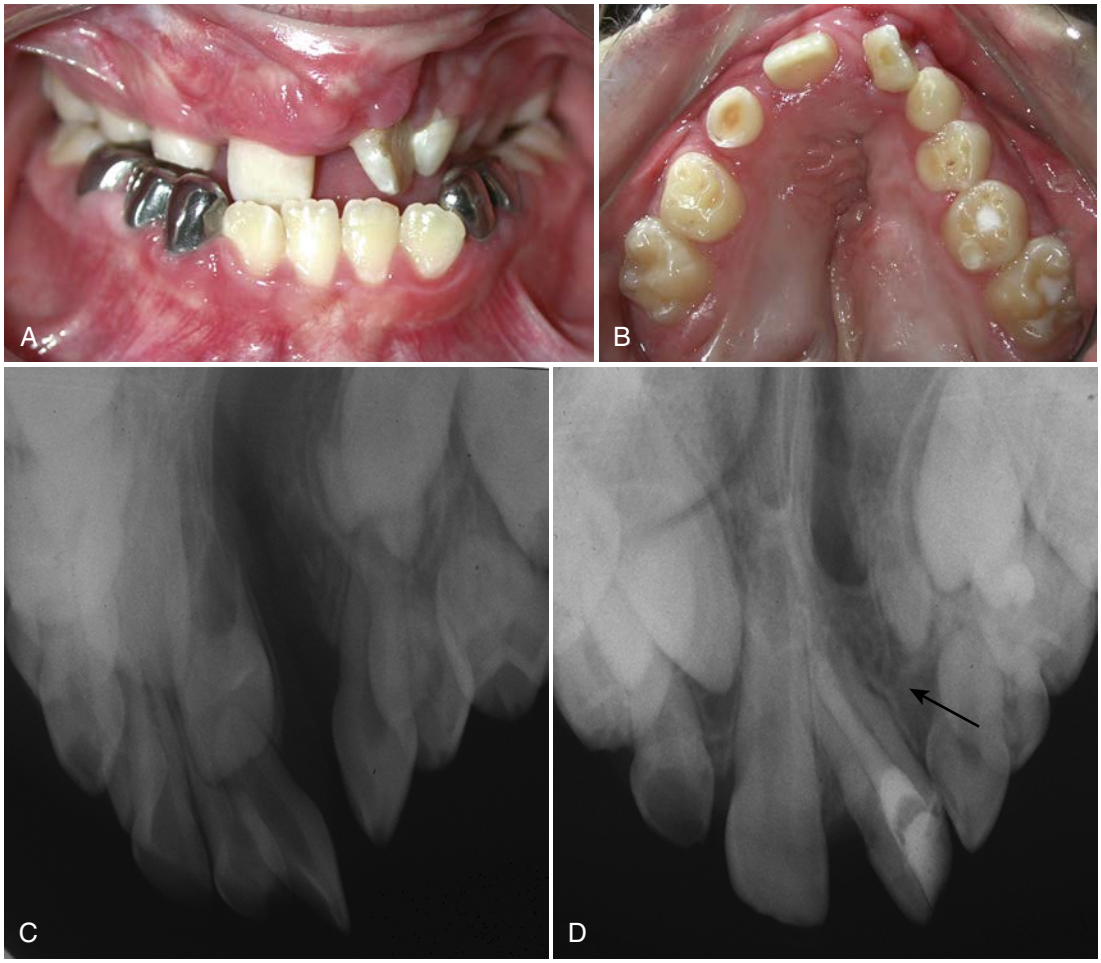


FIGURE 14-11 **A,** Same patient as in Figure 14-10. Intraoral view showing bilateral posterior and anterior crossbites. Note severely rotated maxillary central next to the cleft. **B,** Maxillary occlusal view showing V-shaped arch form with palatal scarring. **C,** Occlusal radiograph showing the bony defect at the cleft site before alveolar bone grafting. **D,** Occlusal radiograph showing successful alveolar bone grafting (the *arrow* points to cancellous bone at the cleft site). Note decayed and endodontally treated maxillary left-central incisor. A developing maxillary left-lateral incisor is now evident at the grafted cleft site.

Intermediate or secondary alveolar bone grafting (6 to 15 years of age). The success of this intervention requires collaborative treatment planning between the orthodontist, surgeon, and other team members.[66-69] Secondary alveolar bone grafting offers five main benefits:

1. Provision of bone support for unerupted teeth and those teeth adjacent to the cleft. If a bone graft is placed before eruption of teeth adjacent to the cleft, it will improve the periodontal support of those teeth. If a bone graft is placed after eruption of the canine, the bone will not improve the crestal height of support and will resorb quickly to its original level.
2. Closure of oronasal fistulae. By using a three-layered closure technique, with the graft sandwiched between the two soft tissue planes, an increased success rate of fistula closure has been reported.
3. Support and elevation of the alar base on the cleft side. This benefit helps to achieve nasal and lip symmetry and provides a stable platform on which the nasal structures are supported. If this procedure is performed alone or is combined with alar cartilage revisions, improved esthetic changes occur.
4. Construction of a continuous arch form and alveolar ridge. This benefits the orthodontist for moving teeth bodily and for uprighting roots into the cleft site. A continuous arch form also benefits the surgeon and prosthodontist by enabling a more esthetic and hygienic prosthesis in preparation for implants to be placed when teeth are missing.
5. Achieve stabilization and some repositioning of the premaxilla in those patients with a bilateral cleft. Controversies concerning alveolar bone grafting require a rational and evidence-based approach for resolution. These controversies relate to the timing of the alveolar bone graft, the sequencing of orthodontic treatment to correct a transverse discrepancy with palatal expansion, and the sites and types of bone for the graft.[70-72]

Timing. The timing of surgery depends more on dental development than on chronologic age. Ideally, the permanent canine root should be half to two thirds formed at the time the

FIGURE 14-12 **A,** Same patient as in Figures 14-5, 14-6, 14-10, and 14-11 shown at 11 years of age undergoing phase I orthodontic treatment (maxillary arch only). **B,** Profile view showing more marked midfacial deficiency. **C,** Intraoral view showing limited bonding of orthodontic appliances in maxillary arch, correction of anterior and posterior crossbites, and alignment of incisors. **D,** Maxillary occlusal view shows palatal expander in place and improved arch form. **E,** Right buccal view showing proclined maxillary incisors, retroclined mandibular incisors, and Class III molar relationship. **F,** Panoramic radiograph showing alignment and uprighting of the endodontically treated maxillary left-central incisor adjacent to the cleft site.

graft is placed (see Fig. 14-14). Permanent canine root formation generally occurs between the ages of 8 and 11 years. Rarely is the graft placed before this time, although occasionally the graft may be placed at an earlier age to improve the prognosis of a lateral incisor. Once teeth have erupted into the cleft site, their periodontal support will not improve with a bone graft. Instead, the height of the crest of alveolar bone resorbs to its original level. For this reason, performing the graft before the eruption of the permanent canine is recommended. If the lateral incisor is on the distal side of the cleft, the graft should be placed earlier. Results from primary bone grafting indicate an adverse effect on maxillary development but, because maxillary growth is almost completed by 10 years of age, performing secondary alveolar bone grafting at this age should have minimal, if any, effect on subsequent facial growth and development.

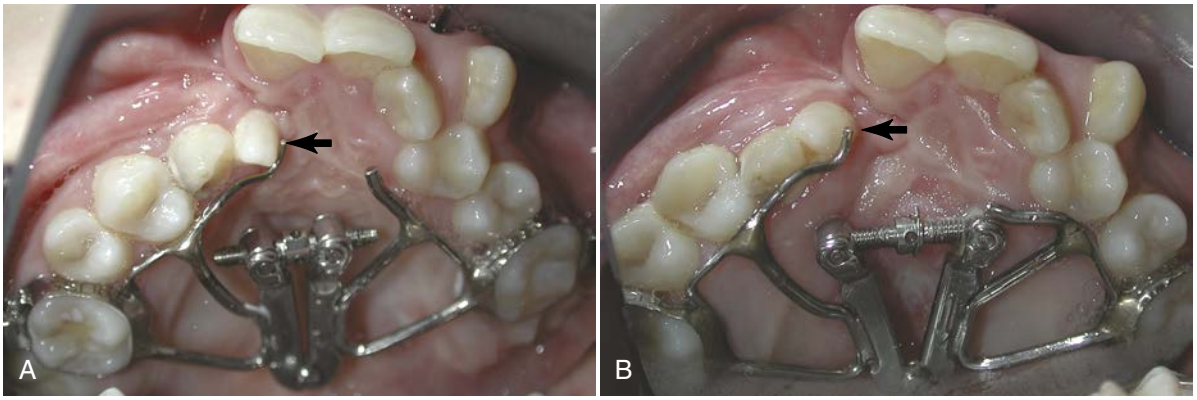


FIGURE 14-13 A, Maxillary occlusal view of 12-year-old patient with repaired, unilateral, right-cleft alveolus. Palatal Spider appliance (*Leone S.P.A. Orthodontic Products, Oxnard, California*) cemented in place. Adjustments of the screw system produce differential expansion in the premolar and canine regions while pivoting around a posterior hinge next to the molars. *Arrow* points to right lateral incisor. B, Maxillary arch after 8 months of expansion. Note improved position of right segment (*arrow* points to lateral incisor) in relation to maxillary central incisor, with minimal expansion at the molar region.

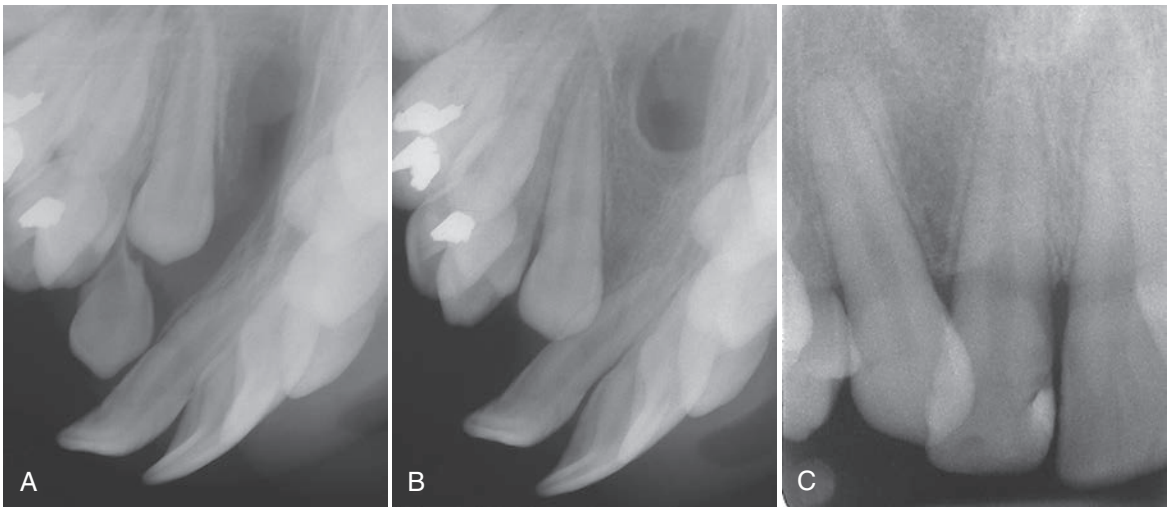


FIGURE 14-14 A, Occlusal radiograph of bone defect at the cleft site before alveolar bone grafting. Lateral incisor is missing and canine has more than two-thirds of its root developed (close to eruption). Note the thin layer of alveolar bone on the central incisor directly adjacent to the cleft. B, Occlusal radiograph taken 7 months after alveolar bone grafting. Note excellent fill of cleft defect with cancellous bone. Canine is erupting through the grafted bone. C, Periapical radiograph taken 4 years after alveolar bone grafting. Note excellent alveolar crest levels adjacent to central incisors and canine.

Sequencing. Secondary bone grafting has been divided into early (2 to 5 years of age), intermediate (6 to 15 years of age), and late (16 years to adult). Since Bergland and coworkers[66] published the results from the Oslo study in which 378 consecutive patients had undergone alveolar bone grafting, contemporary opinion supports the intermediate period as the most appropriate time for grafting. Bone grafting in the intermediate period has the greatest benefits and least risk for interfering with midfacial and skeletodental growth and development. The sequencing of procedures surrounding alveolar bone grafting requires interdisciplinary communication and cooperation resulting in better and more predictable patient care. The general or pediatric dentist ensures that any decayed teeth, especially those adjacent to the cleft, are restored before the grafting procedure. Patient and parents are instructed on good oral hygiene practices to maintain at home. In addition, orthodontic treatment may be required presurgically to reposition maxillary teeth that are in traumatic occlusion or to expand a severely constricted maxilla, thus providing the surgeon better accessibility to the cleft defect. In bilateral cleft lip and palate cases, a vertically extruded premaxilla can be repositioned upward with the use of a labial intrusion archwire, moving the incisors en masse with the bone in vertical alignment with the posterior segments prior to bone grafting (Fig. 14-15). Alternatively, a miniscrew can be placed directly in the premaxilla to stabilize, intrude, or align an edentulous and mobile premaxilla prior to alveolar

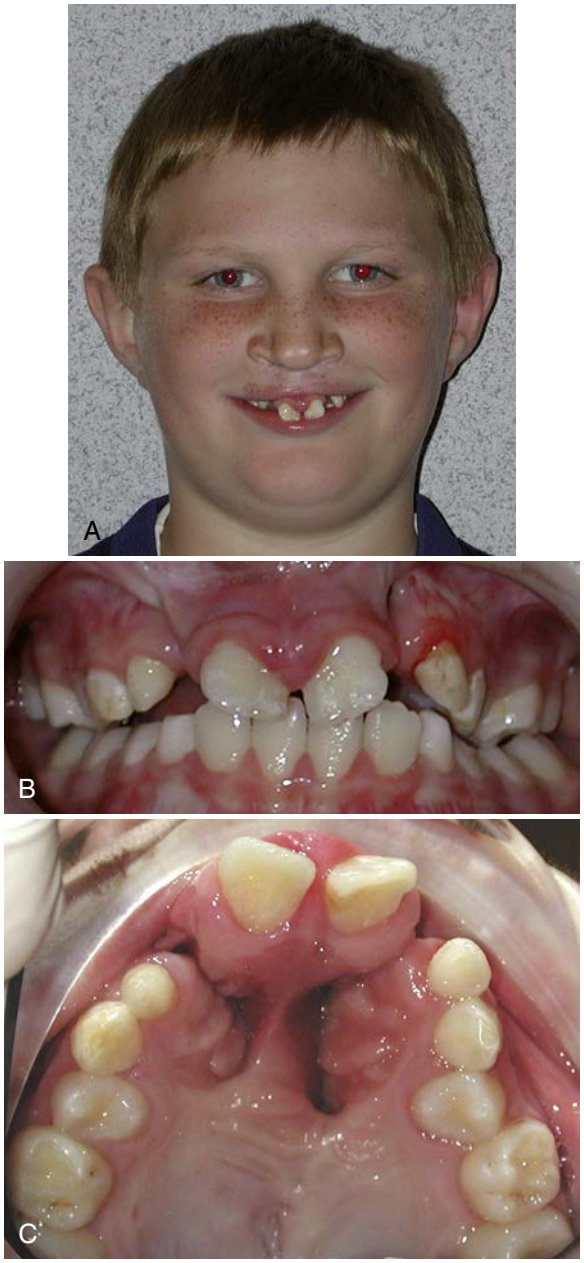


FIGURE 14-15 A, Same patient as in Figure 14-9 shown at 11 years of age after maxillary expansion and orthodontic repositioning of premaxilla. B, Frontal intraoral view showing premaxillary segment in better vertical alignment with the rest of the arch. C, Maxillary occlusal view showing improved arch form. There are persistent palatal fistulas, which are adequately obturated by a Hawley retainer (not shown).

bone grafting.[73] Any erupted teeth adjacent to the cleft that have poor periodontal or endodontic prognosis should be extracted at least 2 months in advance to allow healing of mucosal tissues before surgery.

Surgical technique. The grafting procedure uses tissue lining the cleft defect to construct a nasal floor and close the nasal side of the oral-nasal fistula. The cleft lining is elevated in a subperiosteal plane that leaves bare the osseous margins of the cleft. Cancellous bone taken from the ilium, cranium, or mandibular symphysis is then packed into the cleft defect. Cancellous bone is preferred over cortical bone because it revascularizes more rapidly and is less likely to become infected.[72] Once the cleft defect is packed with bone and the margins are overpacked, soft tissue coverage of the graft is required. The surgeon determines the choice of the donor site from which the bone is harvested. Traditionally, the iliac crest, ribs, and tibia have been used because of their abundant supply of cancellous bone. The morbidity of harvesting bone from these sites results in most patients being hospitalized postsurgically because of complications associated with the donor site more so than with the oronasal recipient site. The cranium has become an alternative site from which to harvest cancellous bone because of the lack of associated discomfort and the amount of hospitalization time involved. However, the operating risks are higher and the abundance of cancellous bone is less than from the iliac crest. The mandibular symphysis is another donor site but should be recommended only when the permanent mandibular canines have been located so as to minimize the chances of injuring these developing teeth.

Orthodontic considerations associated with secondary bone grafting. Orthodontic concerns regarding secondary bone grafting relate to the transverse dimension, incisor alignment, and eruption of the maxillary canines.

The transverse dimension. Orthodontic expansion of the posterior segments (see Fig. 14-7, *C*) preoperatively may improve the occlusion but also widen an existing fistula. The expansion provides better access at surgery for incision and elevation of flaps with closure of the palatal and vestibular oronasal fistulae following the cancellous alveolar bone graft. Expansion also improves the buccolingual orientation of the collapsed posterior segment with the anterior segment, restoring arch symmetry (see Fig. 14-15, *B*). Retention of the corrected crossbite with orthodontic appliances postsurgically may be indicated because the bone graft is unlikely to stabilize the expansion.

Incisor alignment. Alignment of incisors adjacent to the cleft, which typically are rotated, displaced, or tipped, is limited by the available bone into which the roots of the teeth may be moved. If appliances have been placed presurgically, individual orthodontic tooth movements should be delayed until 2 to 6 months following placement of the bone graft. The early movement of the roots into the grafted bone appears clinically to consolidate the alveolar bone and improve the crestal alveolar height. The orthodontist should confirm with the surgeon on the timing of tooth movement into the grafted cleft site. When there are severely rotated incisors near an edentulous space, such as a maxillary central next to a grafted alveolar cleft, the long interbracket span limits the control on force application to effect the derotation. Proff and coworkers[74] reported that placing a miniscrew directly on a well-consolidated graft is an effective method of anchorage to help straighten adjacent teeth.

Eruption of the maxillary canine. Following surgery, the maxillary canine erupts through the grafted bone (see Fig. 14-14, *C*). With orthodontic movement of teeth, sufficient space is created in the arch to allow the canines to erupt successfully. Removal of unerupted supernumerary teeth usually is performed at the time that the bone graft is placed to create an unobstructed path of eruption for the canine. Often the canine will erupt rapidly following the bone graft. If the lateral incisors are malformed or absent, especially in patients with bilateral clefts, the canine is encouraged to erupt adjacent

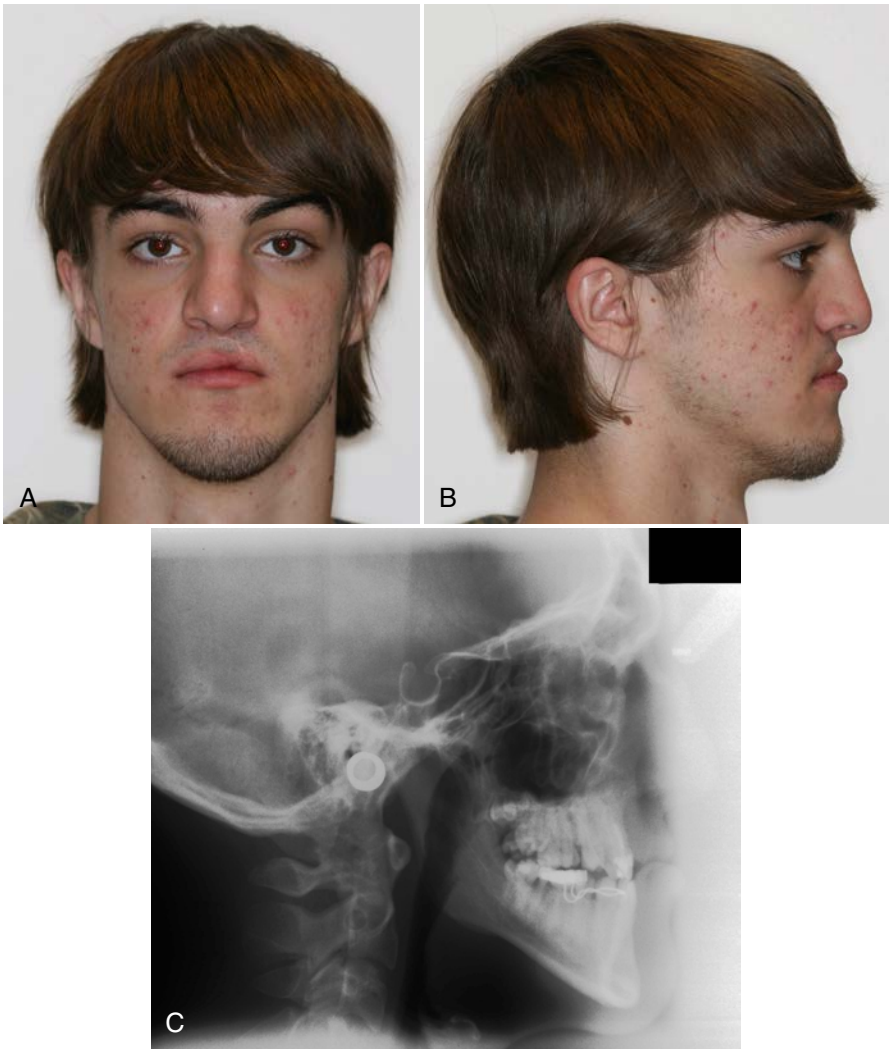


FIGURE 14-16 **A,** Frontal view of same patient as in Figures 14-5, 14-6, and 14-10 to 14-12, hereby shown at age 16. He is interested in combined orthodontic-surgical treatment and future dental implants. **B,** Profile view showing bimaxillary retrusion, especially in the maxilla, concave profile, malar deficiency, and increased lower facial height. **C,** Lateral skull radiograph. Note bimaxillary retrusion, high mandibular-plane angle, and upright incisors in anterior crossbite. (Reprinted from Mercado AM, Vig KWL. Orthodontic principles in the management of orofacial clefts. In: Losee J, Kirschner RE, eds. *Comprehensive Cleft Care*. 6th ed. London: CRC Press Taylor & Francis Group, Inc.; 2015.)

to the central incisors. Closing the edentulous space is an advantage, thus avoiding the need for a prosthetic replacement of the absent lateral incisors. However, "canine substitution" needs to be considered in the context of the occlusion, crown morphology, and the need for orthognathic surgery.

Permanent Dentition Stage

With the eruption of the canines and premolars, the permanent dentition is established. During this time, the adolescent growth spurt and onset of puberty occur. The skeletal discrepancy becomes accentuated and facial appearance and occlusal relationships deteriorate (Figs. 14-16 and 14-17). These changes occur at a time when individuals are most self-conscious about their body image and facial appearance. Facial scars already detract from the cosmetic appearance, and derogatory comments by peers may have a profound psychological effect. Ward and coworkers[54] found that children with clefts reported lower social-emotional well-being compared with noncleft children. This effect was greater in adolescents 15 to 18 years of age than in younger children, pointing to the challenges that adolescents with clefts encounter with peer interactions. During adolescence, involution of the adenoidal lymphoid tissue occurs, often with impairment of speech from the resulting hypernasality. With a decline in cosmetic appearance and speech communication, many patients have a special need for early intervention by the surgeons, orthodontists, speech therapists, and psychologists.

Growth Considerations

Patients with unilateral complete clefts of the lip and palate typically become more maxillary deficient and mandibular prognathic in their appearance, because of sagittal maxillary

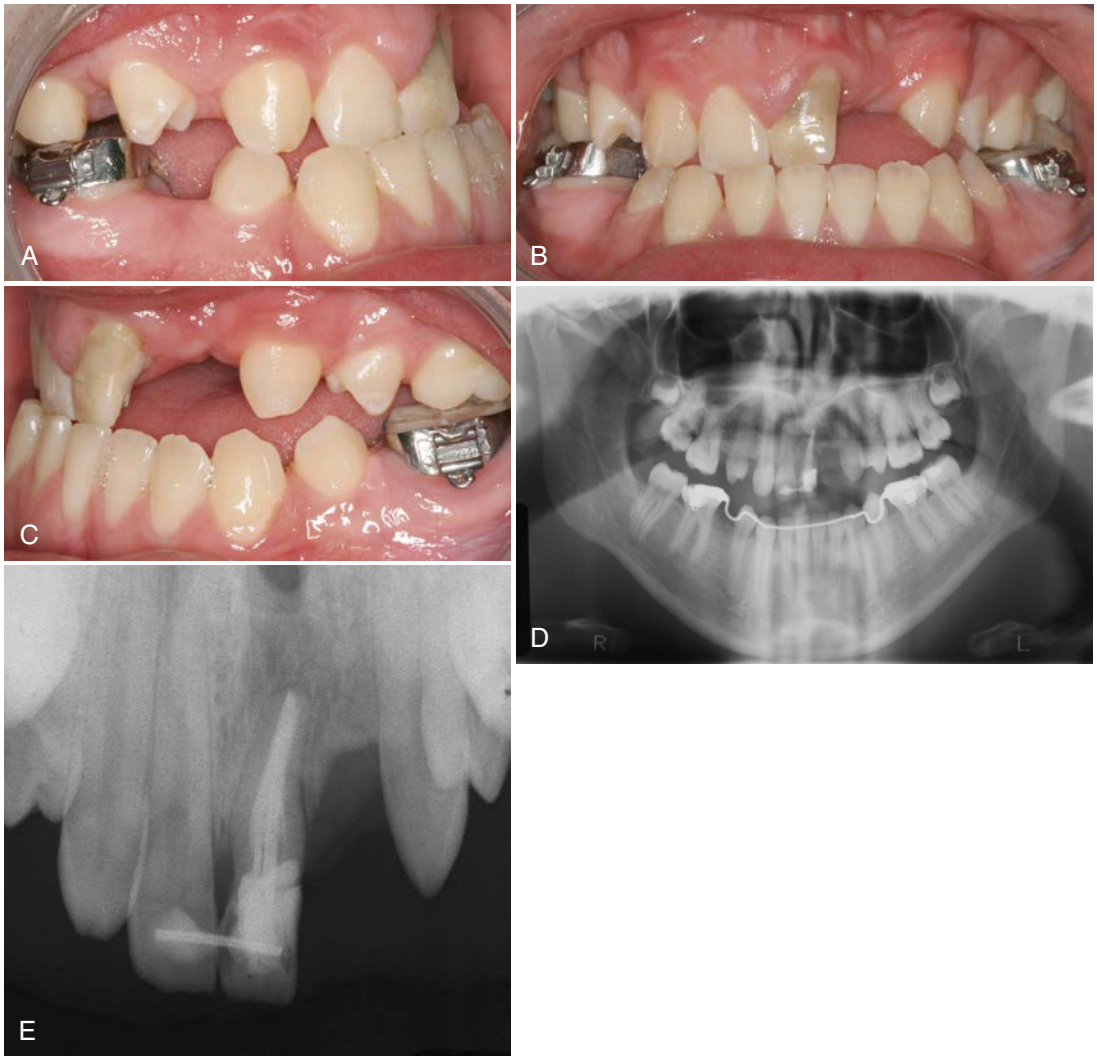


FIGURE 14-17 A to C, Intraoral views of same patient illustrated in Figures 14-5, 14-6, 14-10 to 14-12, and 14-16, shown at age 16. Note right Class III molar and left Class I molar, anterior crossbite with retroclined incisors, signs of gingival recession of #9, and labial aspect of lower incisors. Tooth #9 is discolored due to previous endodontic involvement. Maxillary midline is deviated to the right. Lower lingual holding arch in place. D, Panoramic radiograph showing congenitally missing teeth #4, 7, 13, 20, and 29. Tooth #10 was removed previously due to malformation and lack of root development. Evidence of endodontic treatment and large composite restoration on tooth #9. E, Occlusal radiograph showing adequate alveolar bon-fill at cleft site but lacking in vertical height, especially on distal aspect of #9. Prognosis of #9 is poor. (Reprinted from Mercado AM, Vig KWL. Orthodontic principles in the management of orofacial clefts. In: Losee J, Kirschner RE, eds. *Comprehensive Cleft Care*. 6th ed. London: CRC Press Taylor & Francis Group, Inc.; 2015.)

deficiency. Vertical maxillary deficiency may result in overclosure of the mandible to achieve occlusion of the teeth, thus accentuating the Class III tendency. Clinical evaluation of the extent of overclosure contributing to the Class III relationship by measuring the interocclusal clearance at the premolar region with the patient in resting posture is important. The Class III dental relationship in the sagittal plane may accentuate discrepancies in the transverse plane and are often manifested as posterior crossbites. To evaluate the occlusion, study models are necessary to assess the relationship of the maxillary to mandibular dentition in all three dimensions.

The pattern of facial growth results from the interaction of genetic and environmental factors. Continued growth in early adulthood may enhance or detract from treatment results that have been obtained during childhood and adolescence (Fig. 14-18). These dynamic properties of the face make the management of facial growth challenging and rewarding.[49,50] A patient whose treatment in the permanent dentition allowed camouflage of the skeletal discrepancy is illustrated in Figures 14-19 and 14-20.

Skeletal-Facial Considerations

Examination of facial balance and proportions is critical in determining a treatment plan that combines surgery and orthodontics. This clinical evaluation should be carried out with the patient standing so that one can consider the overall

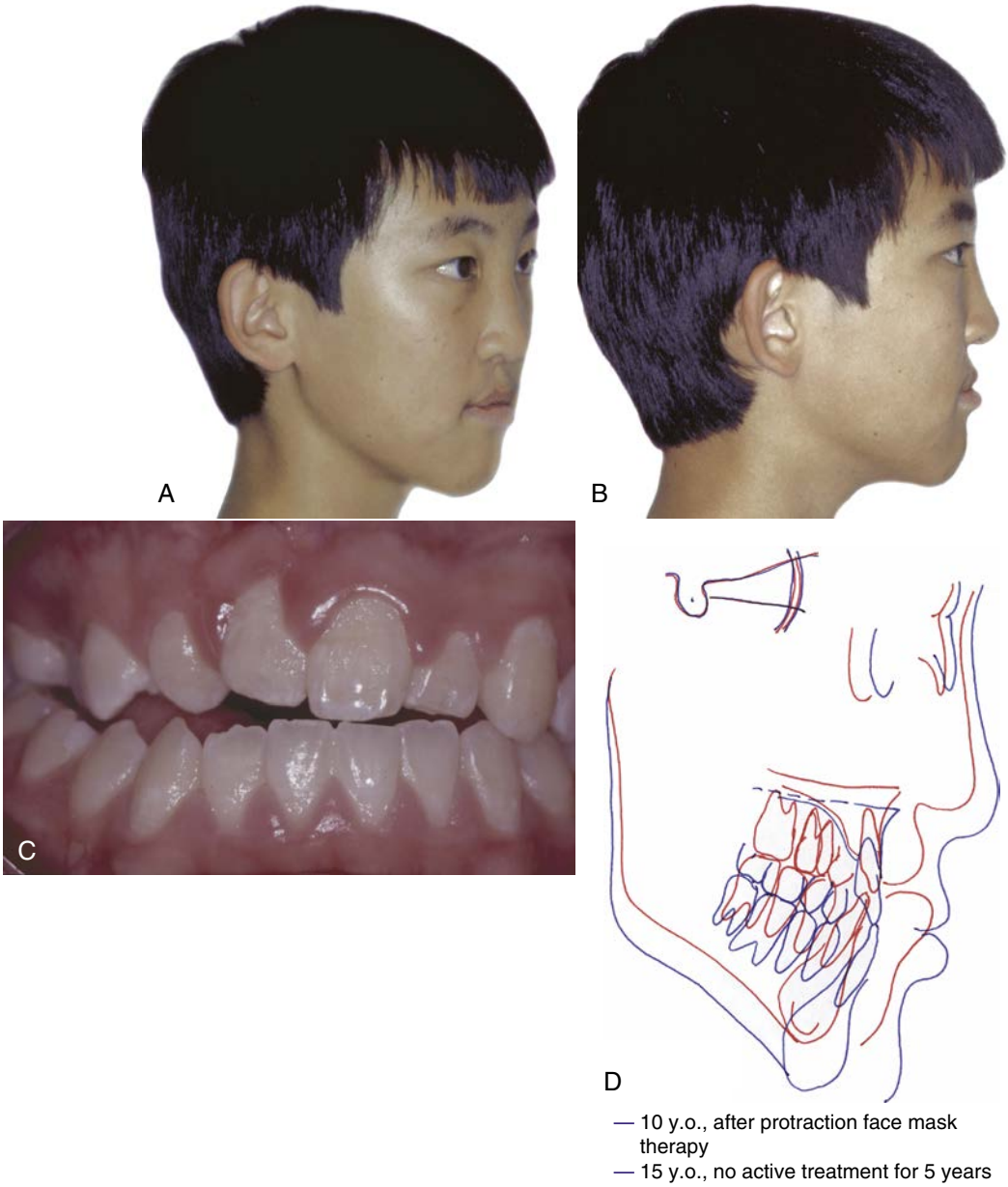


FIGURE 14-18 A, Same patient as in Figures 14-7 and 14-8 shown at 15 years of age. No active orthodontic treatment had been delivered for 5 years after completion of protraction face-mask therapy. B, Profile view shows a straight to slightly concave profile with long, lower facial height. C, Intraoral view shows anterior edge-to-edge relationship, mild incisor rotations, and constricted maxillary-posterior segments. D, Superimposition of lateral cephalogram tracings from immediately after protraction and 5 years after protraction (no active treatment), showing skeletal growth in a predominantly vertical downward pattern.

stature. Full-face and profile assessment provides a database incorporating all three dimensions, and this information should be documented with the patient in resting position and also in occlusion. Cephalometric analysis and prediction tracings provide further information for deciding whether a patient can be treated by orthodontics alone or by orthodontics and an orthognathic surgical procedure. If the skeletal discrepancy is mild and aesthetic concerns are minimal, dental compensation by orthodontic treatment alone may be recommended. A change in axial inclination of the teeth may camouflage the skeletal relationship adequately (see Figs. 14-19 and 14-20). However, one should be cautious because the individual may outgrow the dental correction so that ultimately orthognathic surgery is indicated. The presurgical phase of orthodontic treatment requires decompensation of the dentition so that the maxillary and mandibular teeth are placed in their correct relationship to the underlying skeletal bases. If orthodontic therapy has achieved the ideal relationship of the teeth to the maxillary and mandibular skeletal bases, surgical movements will result in the dentition and the maxilla and mandible being optimally related. With conventional orthognathic surgery techniques for patients with severe

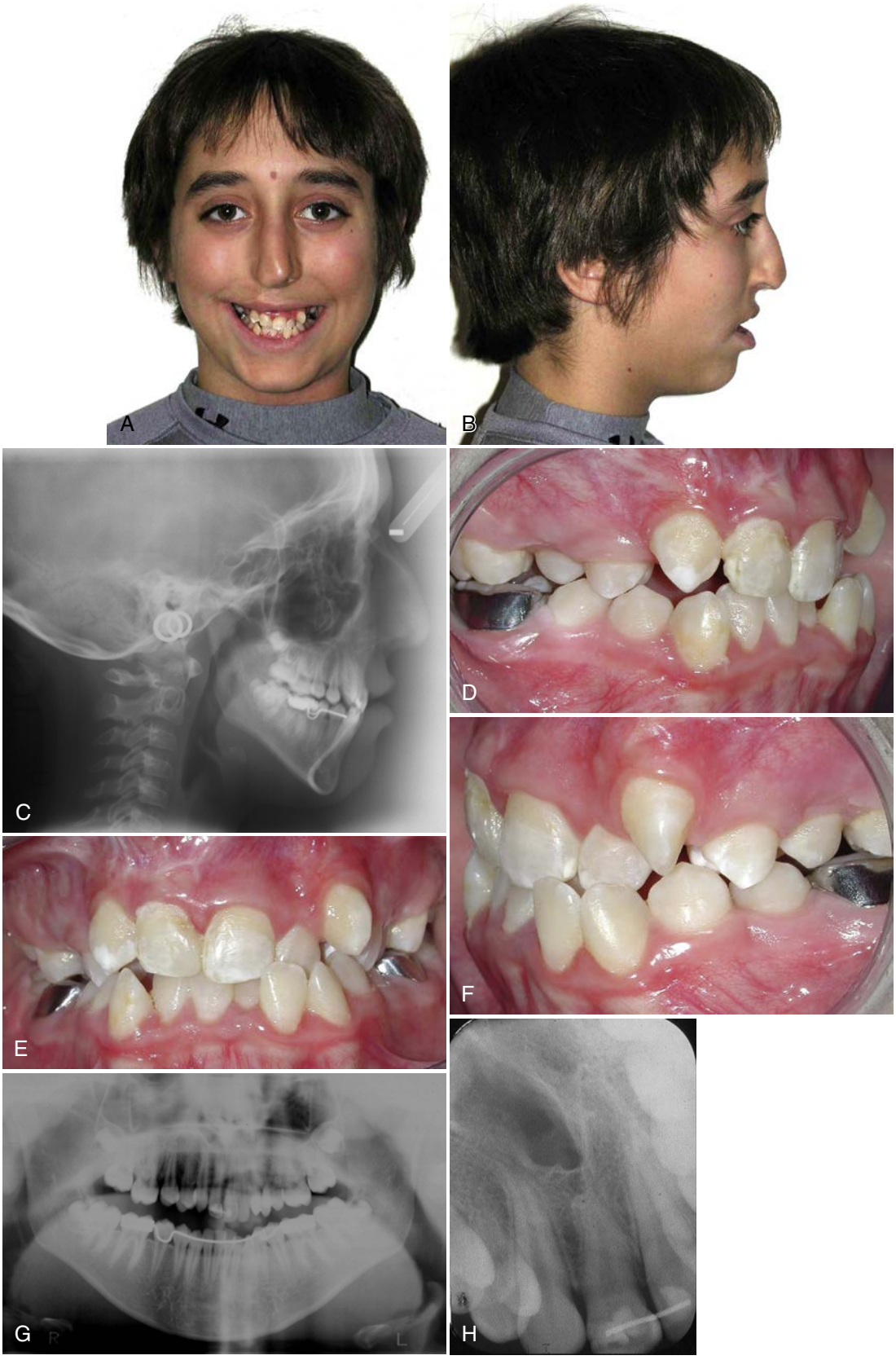


FIGURE 14-19 A, Frontal view of a 13-year-old boy with repaired, complete, and unilateral right cleft lip and palate. He is interested in orthodontic treatment without orthognathic surgery. B, Profile view showing bimaxillary retrusion, mildly convex profile and lip incompetence. C, Lateral skull radiograph. Note bimaxillary retrusion, high mandibular plane angle, and upright incisors. D to F, Intraoral views showing right and left Class I molar, severe crowding in both arches, and significant midline deviation. Lower lingual holding arch in place. G, Panoramic radiograph. Tooth #7 was removed previously due to malformation and lack of root development. H, Periapical radiograph showing adequate alveolar bone fill at cleft site. (Reprinted from Mercado AM, Vig KWL. Orthodontic principles in the management of orofacial clefts. In: Losee J, Kirschner RE, eds. *Comprehensive Cleft Care*. 6th ed. London: CRC Press Taylor & Francis Group, Inc.; 2015.)

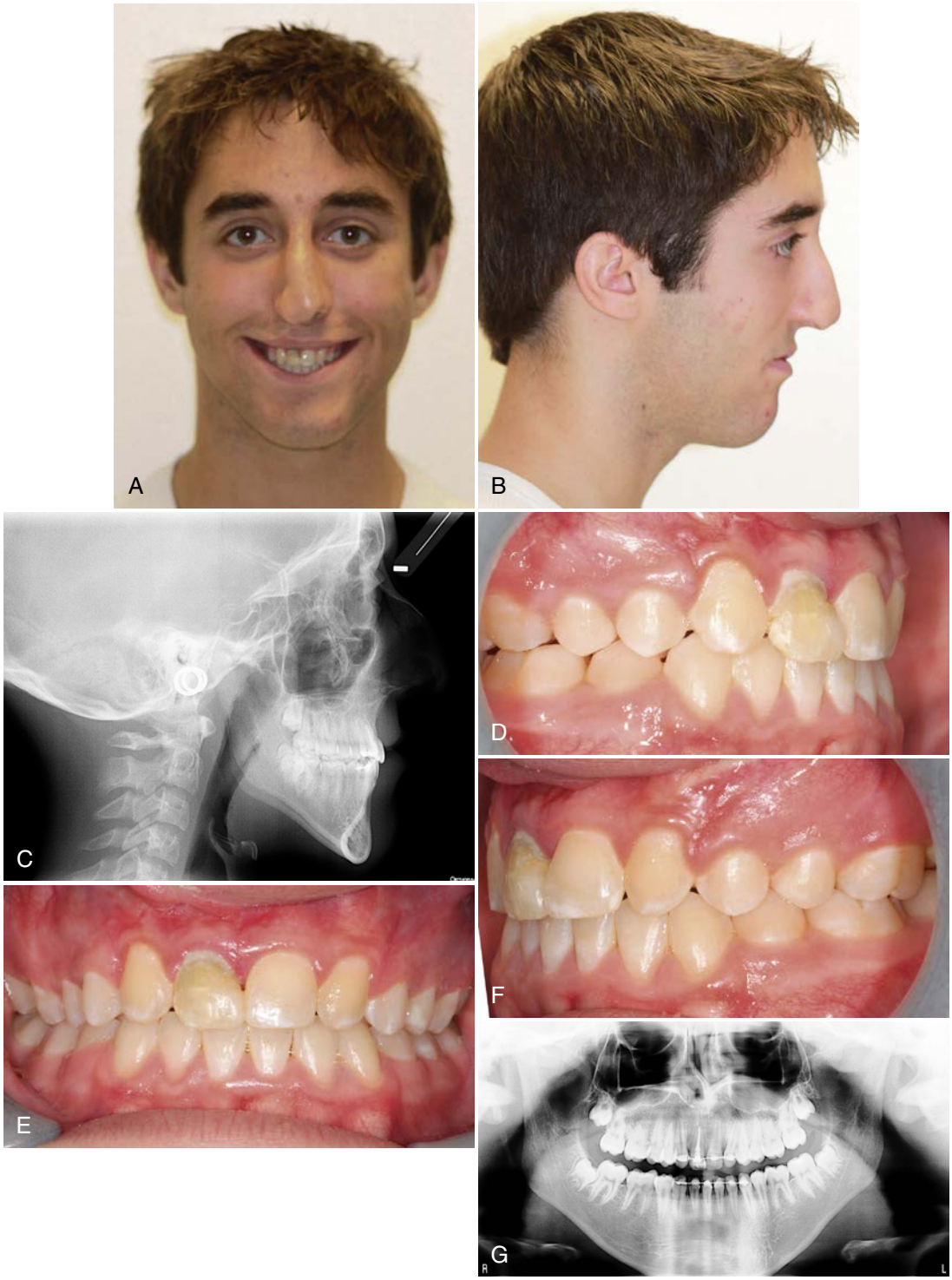


FIGURE 14-20 **A,** Frontal view of same patient as in Figure 14-19, hereby shown at 17 years of age, after completion of orthodontic treatment (nonsurgical). **B,** Profile view showing bimaxillary retrusion and mildly convex profile. **C,** Lateral skull radiograph. Note bimaxillary retrusion, high mandibular plane angle, proclination of upper incisors, and retroclination of lower incisors. **D** to **F,** Intraoral views showing right and left Class I molar. Camouflage orthodontic plan included extraction of teeth #10, 21, and 28, a fan-type maxillary expander, and bilateral canine substitution. Canines should be recontoured to resemble lateral incisors. Tooth #8 is discolored due to trauma and subsequent pulpal necrosis. **G,** Panoramic radiograph showing evidence of endodontic treatment on tooth #8 and lingual bonded retainers. (Reprinted from Mercado AM, Vig KWL. Orthodontic principles in the management of orofacial clefts. In Losee J, Kirschner RE, eds. *Comprehensive Cleft Care*. 6th ed. London: CRC Press Taylor & Francis Group, Inc.; 2015.)

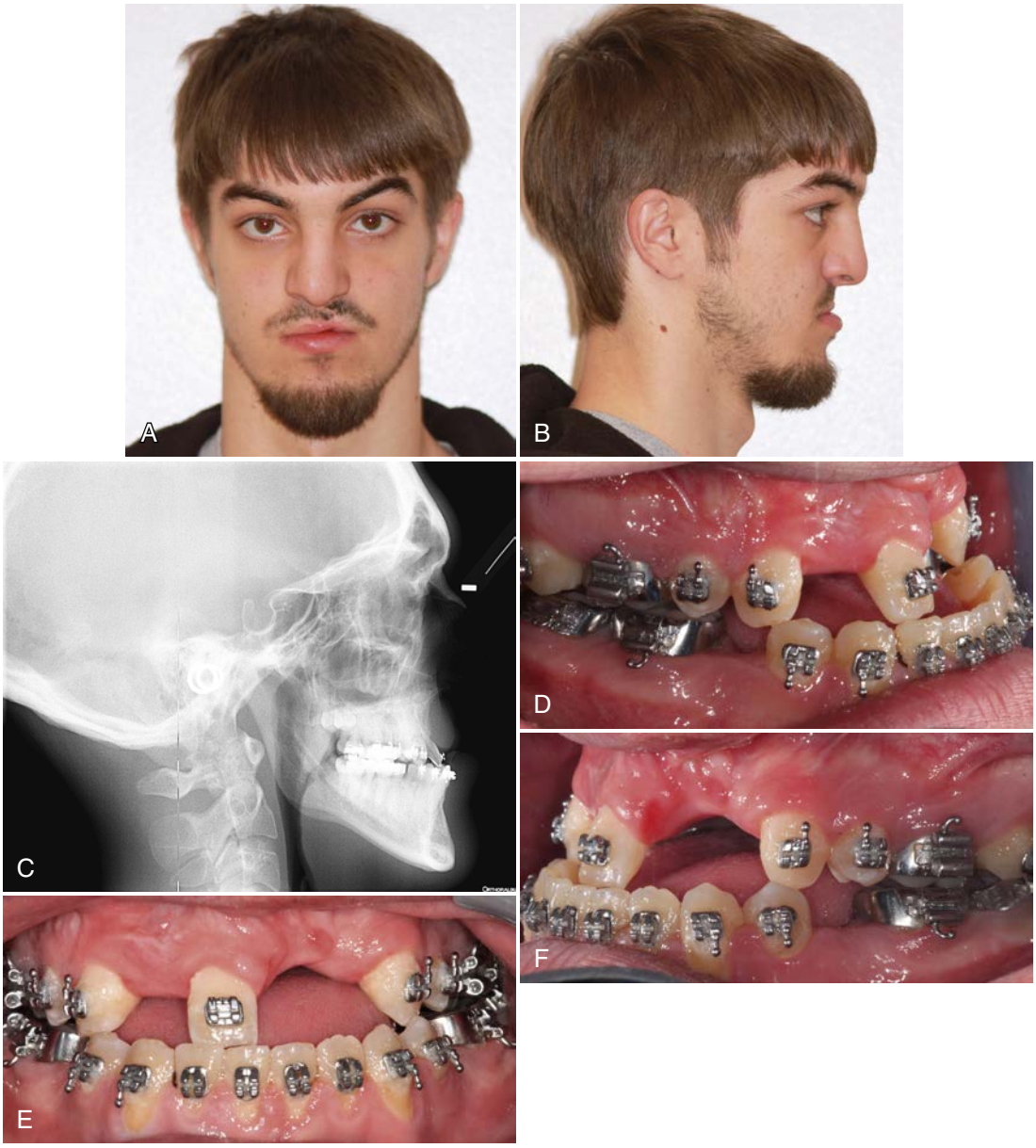


FIGURE 14-21 A, Frontal view of same patient illustrated in Figures 14-5, 14-6, 14-10 to 14-12, 14-16, and 14-17; shown at 19 years following presurgical orthodontic treatment. He has elected for a single-jaw surgery without genioplasty. B, Profile view shows a more accentuated concave profile. C, Lateral skull radiograph showing more proclined maxillary and mandibular incisors. D to F, Intraoral views showing bilateral Class III canine relationship and anterior crossbite. Tooth #9 was extracted due to increased bone loss and mobility. Gingival grafting was done on labial aspect of lower incisor region prior to incisor proclination. (Reprinted from Mercado AM, Vig KWL. Orthodontic principles in the management of orofacial clefts. In: Losee J, Kirschner RE, eds. *Comprehensive Cleft Care*. 6th ed. London: CRC Press Taylor & Francis Group, Inc.; 2015.)

maxillary hypoplasia, surgeons would advance the maxilla as much as possible in the presence of the scar tissue from the lip and palate repairs (Figs. 14-21 to 14-23). The remaining skeletal discrepancy would be corrected with a mandibular setback. Essentially, this was a surgical camouflage of the inability to correct the underlying maxillary hypoplasia predictably. Additionally, the velopharyngeal mechanism may be compromised by maxillary advancement, especially if a pharyngeal flap has been already performed to improve speech.

Orthognathic Surgery

Treatment planning. The timing and sequencing of treatment require close collaboration of the team. The decision to delay surgical orthodontic treatment until growth is stabilized may be sound but not always in the patient's best interest, especially when psychosocial development is affected. In some instances, skeletal surgery may be indicated before growth is completed, knowing that another procedure may be necessary if the patient outgrows the correction. As a general

FIGURE 14-22 **A,** Frontal view of same patient illustrated in Figures 14-5, 14-6, 14-10 to 14-12, 14-16, 14-17, and 14-21; shown at 20 years following orthognathic surgery. He underwent an 8-mm LeFort I advancement. **B,** Profile view shows a more balanced facial profile. **C,** Lateral skull radiograph showing improved maxillary position and rigid fixation. Endosseous implants have been placed for #7, 20, 29. Miniscrews have been placed to hold a bone graft augmenting the bone level in edentulous region of #9 and 10. (Reprinted from Mercado AM, Vig KWL. Orthodontic principles in the management of orofacial clefts. In: Losee J, Kirschner RE, eds. *Comprehensive Cleft Care*. 6th ed. London: CRC Press Taylor & Francis Group, Inc.; 2015.)

rule, skeletal surgery, orthodontic intervention, and final prosthetic rehabilitation should be completed before final soft tissue nose and lip revisions or rhinoplasty are instituted. The outcome of soft tissue surgical revisions, combined with osteotomies for the mobilization of the maxilla and mandible, is often unpredictable until the skeletal discrepancy has been corrected.

Role of the Orthodontist

A coordinated approach to the presurgical phase of orthodontic treatment is indicated. Twelve to 18 months of presurgical orthodontics are usually necessary to align the teeth, correct any compensations in axial inclination of teeth and any dental midline discrepancy, coordinate arches, and localize space for prosthetic replacement of the teeth (see Fig. 14-21). The provision of space for surgical cuts between the crown and the roots of adjacent teeth is also an important part of the presurgical preparations. Ideally, the patient is referred to the surgeon for a presurgical consultation, and the surgical movements are performed on mounted dental casts. Close communication between the surgeon and the orthodontist should identify any occlusal discrepancies that may prevent coordination of arches. The placement of full-size edgewise arch wires, with lugs, provides a means of intermaxillary fixation at the time rigid internal fixation is performed. After surgery is completed, the postsurgical phase of orthodontics details the occlusion, which should be completed within 4 to 6 months (see Figs. 14-22 and 14-23).

Distraction Osteogenesis

With the advent of distraction osteogenesis, correction of severe maxillary hypoplasia solely by advancing the maxilla

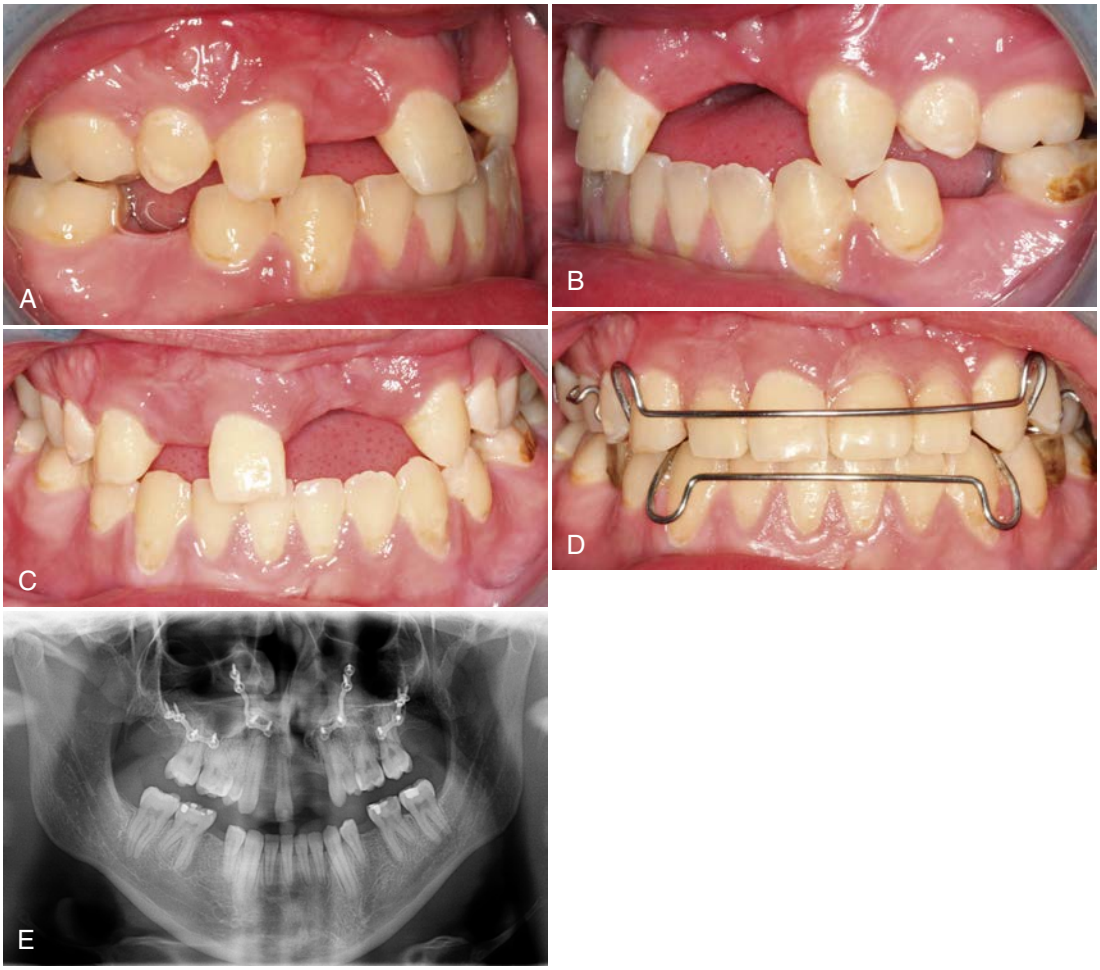


FIGURE 14-23 A to C, Intraoral views of same patient illustrated in Figures 14-5, 14-6, 14-10 to 14-12, 14-16, 14-17, 14-21, and 14-22, shown at age 20 following orthognathic surgery. Note bilateral Class II molar and Class I canine relationships, adequate overbite, and overjet. **D,** Intraoral view with Hawley retainers in place. The maxillary retainer has pontics for temporary replacement of #7, 9, and 10. **E,** Panoramic radiograph taken upon debonding, before implant placement. (Reprinted from Mercado AM, Vig KWL. Orthodontic principles in the management of orofacial clefts. In: Losee J, Kirschner RE, eds. *Comprehensive Cleft Care*. 6th ed. London: CRC Press Taylor & Francis Group, Inc.; 2015)

may be possible.[75-77] Such correction would be accomplished by expansion of the scar tissue at the same time distraction osteogenesis is performed. Briefly, rigid external distraction (RED) involves cementation of an intraoral tooth-borne splint in the maxilla with hooks extending extraorally for traction. The appliance is usually fabricated and delivered by the orthodontist. This tooth-borne method of attachment to the maxilla may place the teeth at risk of periodontal damage. Bone-borne methods of attachment reduce such risk and include miniscrews placed bilaterally in the alveolar ridges to guide the skeletal movements[73] or preadapted miniplates fixated to the anterior surface of the maxilla beside the piriform aperture for traction[78] (Figs. 14-24 to 14-26). A complete LeFort I osteotomy is performed, and a RED device is placed intraoperatively. The vector of the anterior force can be adjusted for each patient during the course of distraction, which is done at home by patients turning the activation screw at a rate of 1 mm per day. Following advancement, the RED device is maintained for 2 to 3 weeks for bone consolidation. This is followed by a retention period during which the patient wears a protraction face mask. Internal distractors, which typically are bone-borne, have also been used in patients with cleft maxillas.[79] The distractor rod penetrates into the oral cavity, thus activation of the system is possible via an intraoral approach. After the desired advancement is achieved, the turning arms are removed and the submucosal components stay in place. This method has the added benefit of allowing for long consolidation periods (3 months).

The benefit of distraction osteogenesis in a hypoplastic maxilla with scar tissue and a compromised blood supply lies in a more gentle skeletal advancement with corticotomy cuts without downfracture of the maxilla. The orthodontist may monitor for hypernasal speech that could occur with advancement of the maxilla. Because the nasomaxillary complex is advanced slowly, at a millimeter per day, speech may be evaluated at intervals as the advancement proceeds and adaptation of the velopharyngeal mechanism occurs. Again, the treatment benefits need to be weighed against the burden of care

FIGURE 14-24 21-year-old male with repaired cleft lip and palate on the right side undergoing comprehensive orthodontic treatment. **A** to **C,** Facial images showing lack of support for upper lip, excessive lower-incisor display, and concave profile due to severe maxillary retrognathia. Patient is interested in orthognathic surgery for maxillary advancement. **D** to **F,** Facial images after 13-mm maxillary advancement using a RED device and bone-anchored plates. Note improved upper lip support, improved smile aesthetics, and more balanced profile.

and considered in the context of the scientific evidence of the probability of achieving quantifiable outcomes of success and failure.[80,81]

In craniofacial anomalies presenting with severe mandibular retrognathia, such as Pierre Robin sequence or Goldenhar syndrome, mandibular advancement is needed to improve a compromised airway. When distraction osteogenesis is done to advance the mandible, there is often an unfavorable side effect in the vertical dimension manifested as an anterior open bite. Miniscrews can be placed bilaterally on the alveolar ridges of maxilla and/or mandible to serve as attachments for intermaxillary elastics during protraction, therefore preventing the development of an open bite (Fig. 14-27).[73]

MANAGEMENT OF THE MISSING LATERAL INCISOR SPACE

Patients with clefts often have an edentulous ridge in the cleft region, either due to congenital agenesis or previous extraction of teeth. In such cases, the most common options for management of the edentulous area include space closure, fixed versus removable prosthesis, and osseointegrated implants.

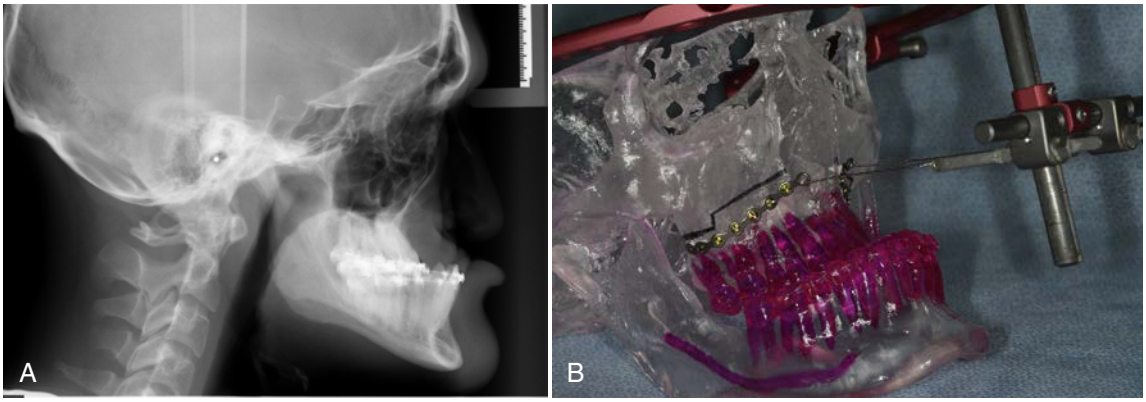


FIGURE 14-25 Same patient illustrated in Figure 14-24. **A,** Cephalometric radiogram before distraction. SNA angle = 62 degrees (severe maxillary retrusion); SNB angle = 81 degrees (normal mandibular position); ANB angle = −19 degrees (severe maxilla-mandibular discrepancy). **B,** To avoid excessive dental movement often associated with tooth borne anchorage, 2-mm plates are adapted along the osteotomy and anchored to the maxilla with a minimum of six screws on each side. Wires extending from the anterior hole of the plate exit via percutaneous punctures adjacent to the ala bilaterally. This allows for skeletal anchorage and a more controlled vector of distraction. In this figure, the plates are shown adapted to a stereolithic model for illustrative purposes. Adaptation is easily accomplished during surgery and a model is not generally required. The wire extensions connect to the adjustable screw system in the vertical bar of the RED.

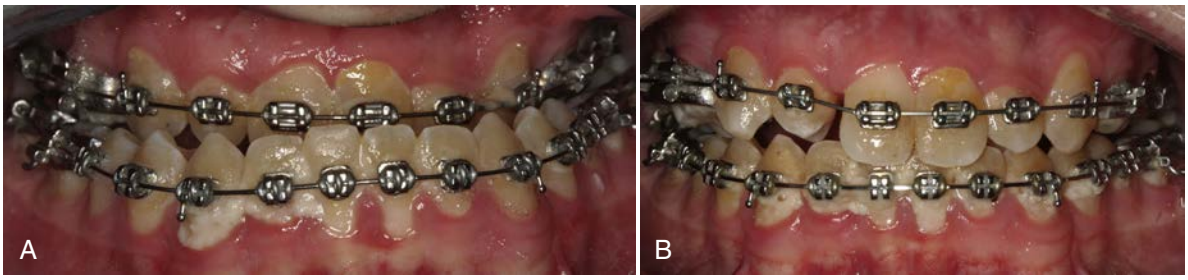


FIGURE 14-26 Same patient illustrated in Figures 14-24 and 14-25. **A,** Frontal intraoral view prior to distraction. Overjet is −10 mm. Overbite is nearly 50%. **B,** Frontal intraoral view after distraction. Overjet is 2 mm. Positive overbite has been maintained.

FIGURE 14-27 Intraoral view of an 8-year-old child with Goldenhar syndrome who underwent mandibular advancement with distraction osteogenesis to improve his airway. Anterior open bite is evident. Miniscrews were inserted in anterior maxilla and composite resin was applied on the miniscrew heads for increased retention of elastics and for patient comfort. Miniscrews could not be placed in anterior mandible due to close proximity of roots. A lower labial arch with hooks was cemented. Patient was instructed to wear intermaxillary elastics between the miniscrews and the hooks, to help close the bite.

The need for prosthetic replacement of the missing lateral incisor can be avoided by allowing mesial eruption of the canine, moving the posterior teeth anteriorly with orthodontics, and cosmetic "reshaping" of the canine (see Fig. 14-20, *E*). If the space is preserved, the options to consider for the replacement of missing teeth at the cleft are fixed versus removable prosthesis. Fixed prostheses are a feasible option for patients with a single tooth missing in the cleft area, patients with failed or nonexistent alveolar bone grafts who need stabilization of the maxillary segments, and patients whose teeth adjacent to the cleft are decayed or malformed. Removable prostheses are a better option for replacing multiple teeth, restoring long edentulous spans, simultaneous obturation of residual palatal fistulas, improvement of lip support by using acrylic flanges, and for transitional replacement of teeth in young patients prior to definitive prosthetic rehabilitation. The orthodontist should work in consultation with the prosthodontist to determine the optimal space distribution between teeth that will allow an unrestricted path of insertion for the prosthesis, and will ultimately result in a functional occlusion with harmonious aesthetics.

Endosseous implants can be used effectively to restore the edentulous cleft area, most commonly when there is a single

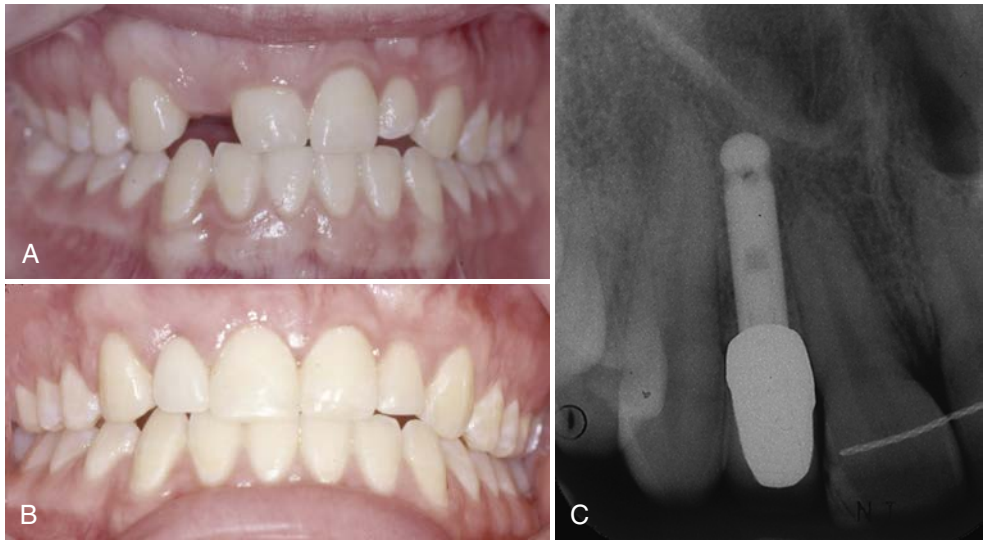


FIGURE 14-28 Sixteen-year-old patient with bilateral cleft lip and unilateral cleft palate on the right alveolus. **A,** Intraoral view after alveolar grafting and orthodontic treatment, showing edentulous ridge in area of missing #7. Tooth #10 is malformed. Note uneven gingival margins. **B,** Intraoral view after implant and crown for replacement of #7 and cosmetic bonding of #8 and 10. Gingivoplasty was done to achieve gingival symmetry and adequate soft tissue contours. **C,** Periapical radiograph showing osseointegration of implant fixture in the former site of the cleft alveolus. Endosteal fixtures of smaller diameters are now available with ideal indication for narrow interdental spaces typical such as cleft sites.

missing tooth, healthy adjacent teeth, and an adequate volume of alveolar bone (Fig. 14-28).[82-84] This requires careful planning with the oral surgeon and the prosthodontist prior to orthodontic distribution of the adequate spaces between the teeth. The orthodontist must ensure that roots of teeth adjacent to the edentulous area are upright and parallel, which will allow the surgeon to insert the endosteal fixture without risk of damaging adjacent roots. For the young adolescent patient who completes orthodontic treatment, it is recommended to postpone implant placement until the patient has completed both sagittal and vertical growth.[85,86] In such cases, a Hawley retainer with pontic teeth can be used to maintain the alignment and arch form (see Fig. 14-23, *C* and *D*). In the postadolescent period, the orthodontist should assess facial growth by superimposing cephalometric radiographs taken 6 months apart and looking for longitudinal changes in the sagittal and vertical dimensions. When serial superimpositions show no significant dimensional changes, then completion of facial growth has been attained. During the growth monitoring period, the edentulous grafted area may show radiographic evidence of resorption over several years in adolescents whose lateral incisor space is preserved for future implant placement. The greater the interval between secondary bone graft and implant placement, the more likely that the residual graft will need to be augmented with a new bone graft.[83] Kearns and coworkers[83] recommend that patients who receive secondary bone grafts in the mixed dentition should be regrafted between 15 and 17 years of age, followed by implant placement within 4 months thereafter.

SUMMARY

The orthodontist's role in the cleft palate team requires close collaboration with the other team members. The rationale of timing and sequencing of orthodontic treatment have been discussed in four periods of development: (1) neonatal or infant maxillary orthopedics, (2) orthodontic considerations in the primary dentition, (3) mixed dentition to include presurgical considerations before an alveolar bone graft is placed, and (4) final treatment in the permanent dentition with orthodontics only or combined with orthognathic surgery. The latter period combines an orthodontic and surgical approach to the correction of dental and skeletal components of malocclusion and facilitation of any necessary prosthodontic treatment. Speech considerations and the communicative skills of the patient with a cleft are important aspects in planning orthognathic surgery for these patients. Subsequent nose and lip revisions for cosmetic improvement also must not be underestimated in the enhancement of the final, soft-tissue, facial aesthetic result following correction of the skeletal and dental discrepancies. Provided that the team members plan the timing and sequencing of appropriate treatment modalities in a closely coordinated, problem-oriented approach, patients with clefts should have optimal functional and aesthetic results. Outcome measures for reporting the results of surgical interventions require the choice of valid and reliable measures to be identified and implemented.[80,81] The ultimate outcome for team-based care is to have a fully rehabilitated patient who is satisfied with the treatment outcomes in terms of speech, occlusion, facial and dental aesthetics, and function. The patient should continue to receive conventional dental and medical routine evaluations similar to any adult to maintain optimal oral health.

Acknowledgements

We thank Dr. Philip Samuels (perinatologist) and Ms. Pamela Foy (sonographer) at The Ohio State University Department of

Obstetrics and Gynecology for their permission to publish Figure 14-1. We are indebted to Dr. Supakit Peanchitlertkajorn for permission to publish Figures 14-3 and 14-4 (neonatal orthopedics). Our gratitude goes to Dr. Peter Larsen for permission to publish Figures 14-24, 14-25, and 14-26 and to Dr. Lawrence Wright, orthodontist who treated the patient in those figures. We thank the past and present members of the Cleft Palate–Craniofacial Team at Nationwide Children's Hospital in Columbus, Ohio, and of The Ohio State University College of Dentistry for their collaboration in the treatment of the cases illustrated in this chapter.

Figure 14-2 was reprinted from Vig KWL & Mercado AM: Contemporary management of craniofacial anomalies: Will past experiences influence and predict the future? In McNamara JA Jr (ed.): *The 40th Moyers Symposium: Looking Forward … Looking Back.* Ann Arbor: Monograph 50, Craniofacial Growth Series, Department of Orthodontics and Pediatric Dentistry and Center for Human Growth and Development, The University of Michigan, Ann Arbor, 2014.

Figures 14-7 and 14-8 were modified from Lidral AC & Vig KWL: Role of the orthodontist in the management of patients with cleft lip and/or palate. In Wyszynski DF (ed.): *Cleft Lip and Palate: From Origin to Treatment*. New York, 2002, with kind permission from Oxford University Press, Inc.

Figures 14-16, 14-17, 14-19, to 14-23 were reprinted from Mercado AM & Vig KWL: Orthodontic principles in the management of orofacial clefts. In Losee J & Kirschner RE (eds.): *Comprehensive Cleft Care*. 2nd ed. London, 2015, with kind permission from CRC Press Taylor & Francis Group, Inc.

REFERENCES

1. Sprintzen RJ, Siegel-Sadewitz VL, Amapt A, et al. Anomalies associated with cleft lip, cleft palate or both. *Am J Med Genet*. 1985;20:585.
2. Parameters for the evaluation and treatment of patients with cleft lip/palate or other craniofacial anomalies, American Cleft Palate-Craniofacial Association. *Cleft Palate-Craniofac J*. 1993;30(suppl 1): S1–S16.
3. Parameters for the evaluation and treatment of patients with cleft lip/palate or other craniofacial anomalies, American Cleft Palate-Craniofacial Association, Revised ed. November 2009. http://www.acpa-cpf.org/teamcare/Parameters%20Rev.2009.pdf.
4. Lidral AC, Vig KWL. The role of the orthodontist in the management of patients with cleft lip and/or palate (Chapter 29). In: Wyszynski D, ed. *Cleft Lip and Palate: From Origin to Treatment*. New York: Oxford University Press; 2002.
5. Mercado AM, Vig KWL. Orthodontic principles in cleft lip and palate care (Chapter 47). In: Losee JE, Kirschner RE, eds. *Comprehensive Cleft Care*. New York: McGraw-Hill, Medical Publishing Division; 2009.
6. Waitzman NJ, Romano PS, Scheffler RM. Estimates of economic costs of birth defects. *Inquiry*. 1994;31:189.
7. Jones MC. Facial clefting: etiology and developmental pathogenesis. *Clin Plast Surg*. 1993;20:599.
8. Grandjean H, Larroque D, Levi S, et al. The performance of routine ultrasonographic screening in the Eurofetus Study. *Am J Obstet Gynecol*. 1999;181:446.
9. Christ J, Meininger M. Ultrasound study of the nose and upper lip before birth. *Ann Plast Surg*. 1983;11:308.
10. Robinson JN, McElrath TF, Benson CB, et al. Prenatal ultrasonography and the diagnosis of fetal cleft lip. *J Ultrasound Med*. 2001;20:1165.
11. Bronshtein M, Blumenfeld I, Kohn J, et al. Detection of cleft lip by early second-trimester transvaginal sonography. *Obstet Gynecol*. 1994;84:73.
12. Babcook CJ, McGahan JP. Axial ultrasonographic imaging of the fetal maxilla for accurate characterization of facial clefts. *J Ultrasound Med*. 1997;16:619.
13. Nyberg DA, Hegge FN, Kramer D, et al. Premaxillary protrusion: a sonographic clue to bilateral cleft lip and palate. *J Ultrasound Med*. 1993;12:331.
14. Cockell A, Lees M. Prenatal diagnosis and management of orofacial clefts. *Prenat Diagn*. 2000;20:149.
15. Christ JE, Meininger MG. Ultrasound diagnosis of cleft lip and cleft palate before birth. *Plast Reconstr Surg*. 1981;68:854.
16. Mulliken JB, Benacerraf BR. Prenatal diagnosis of cleft lip: what the sonologist needs to tell the surgeon. *J Ultrasound Med*. 2001;20:1159.
17. Monni G, Ibba RM, Olla G, et al. Color Doppler ultrasound and prenatal diagnosis of cleft palate. *J Clin Ultrasound*. 1995;23:189.
18. Johnson N, Sandy JR. Prenatal diagnosis of cleft lip and palate. *Cleft Palate-Craniofac J*. 2003;40:186.
19. Matthews MS, Cohen M, Viglione M, et al. Prenatal counseling for cleft lip and palate. *Plast Reconstr Surg*. 1998;101:1.
20. Sagi M, Shiloh S, Cohen T. Application of the Health Belief Model in a study on parents' intentions to utilize prenatal diagnosis of cleft lip and/or palate. *Am J Med Genet*. 1992;44:326.
21. Berk NW, Marazita ML, Cooper ME. Medical genetics on the cleft palate-craniofacial team: understanding parental preference. *Cleft Palate-Craniofac J*. 1999;36:30.
22. *Surgeon General's report: children with special health care needs*. Washington, DC: US Department of Health and Human Services, Office of Maternal and Child Health; 1987.
23. Turvey TA, Vig KWL, Fonseca RJ, eds. *Facial Clefts and Craniosynostosis: Principles and Management*. Philadelphia: WB Saunders; 1996.
24. Vig KWL, Turvey TA. Orthodontic, surgical interaction in the management of cleft lip and palate. *Clin Plast Surg*. 1985;12:735.
25. Washington Department of Health, Division of Family and Community Services, Office for Children with Special Health Care Needs. *Cleft Lip and Palate: Critical Elements of Care*. Seattle: Kaye Smith Business Graphics; 1997.
26. Burston WR. The early treatment of cleft palate conditions. *Dent Pract*. 1958;9:41.
27. McNeil CK. Orthopaedic principles in the treatment of lip and palate clefts. In: Hotz R, ed. *International Symposium on Early Treatment of Cleft Lip and Palate*. Bern, Switzerland: Hans Huber; 1964.
28. Rosenstein SW, Monroe CW, Kernahan DA, et al. The case for early bone grafting in cleft lip and cleft palate. *Plast Reconstr Surg*. 1982;70:297.
29. Hotz MM, Gnoinski WM, Nussbaumer H, et al. Early maxillary orthopedics in cleft lip and palate cases: guidelines for surgery. *Cleft Palate-Craniofac J*. 1978;15:405.
30. Jolleys A, Robertson NRE. A study of the effects of early bone grafting in complete clefts of the lip and palate: a five year study. *Br J Plast Surg*. 1972;25:229.
31. Mølsted K, Dahl E, Skovgaard LT, et al. A multicentre comparison of treatment regimens for unilateral cleft lip and palate using a multiple regression model. *Scand J Plast Reconstr Surg Hand Surg*. 1993;27:277.
32. Ross RB, MacNamera MC. Effect of presurgical infant orthopedics on facial esthetics in complete bilateral cleft lip and palate. *Cleft Palate-Craniofac J*. 1994;31:68.
33. Pruzansky S. Pre-surgical orthopedics and bone grafting for infants with cleft lip and palate. *Cleft Palate-Craniofac J*. 1964;1:164.
34. Santiago PE, Grayson BH, Cutting CB, et al. Reduced need for alveolar bone grafting by presurgical orthopedics and primary gingivoperiosteoplasty. *Cleft Palate-Craniofac J*. 1998;35:77.
35. Grayson BH, Santiago PE, Brecht LE, et al. Presurgical nasoalveolar molding in infants with cleft lip and palate. *Cleft Palate-Craniofac J*. 1999;36:486.
36. Grayson BH, Maull D. Nasoalveolar molding for infants born with clefts of the lip, alveolus, and palate. *Clin Plast Surg*. 2004;31:149.
37. Millard DR, Latham R, Huifen X, et al. Cleft lip and palate treated by presurgical orthopedics, gingivoperiosteoplasty, and lip adhesion (POPLA) compared with previous lip adhesion method: a preliminary study of serial dental casts. *Plast Reconstr Surg*. 1999;103:1630.

38. Winters JC, Hurwitz DJ. Presurgical orthopedics in the surgical management of unilateral cleft lip and palate. *Plast Reconstr Surg*. 1995;95:755.
39. Prahl C, Kuijpers-Jagtman AM, van't Hof MA, et al. A randomised prospective clinical trial into the effect of infant orthopedics on maxillary arch dimensions in unilateral cleft lip and palate (Dutchcleft). *Eur J Oral Sci*. 2001;109:297.
40. Prahl C, Kuijpers-Jagtman AM, van't Hof MA, et al. Infant orthopedics in UCLP: effect on feeding, weight and length. A randomised clinical trial (Dutchcleft). *Cleft Palate-Craniofac J*. 2005;42:171.
41. Konst EM, Prahl C, Weersink-Braks H, et al. Cost-effectiveness of infant orthopedic treatment regarding speech in patients with complete unilateral cleft lip and palate: a randomized three-center trial in the Netherlands (Dutchcleft). *Cleft Palate-Craniofac J*. 2004;41:71.
42. Bongaarts CAM, van't Hof MA, Prahl-Andersen B, et al. Infant orthopedics has no effect on maxillary arch dimensions in the deciduous dentition of children with complete unilateral cleft lip and palate (Dutchcleft). *Cleft Palate-Craniofac J*. 2006;43:665.
43. Bongaarts CAM, Kuijpers-Jagtman AM, van't Hof MA, et al. The effect of infant orthopedics on the occlusion of the deciduous dentition in children with complete unilateral cleft lip and palate (Dutchcleft). *Cleft Palate-Craniofac J*. 2004;41:633.
44. Bongaarts CAM, Prahl-Andersen B, Bronkhorst EM, et al. Infant orthopedics and facial growth in complete unilateral cleft lip and palate until six years of age (Dutchcleft). *Cleft Palate-Craniofac J*. 2009;46:654.
45. Bongaarts CAM, Prahl-Andersen B, Bronkhorst EM, et al. Effect of infant orthopedics on facial appearance of toddlers with complete unilateral cleft lip and palate (Dutchcleft). *Cleft Palate-Craniofac J*. 2008;45:407.
46. Uzel A, Alparslan N. Long-term effects of presurgical infant orthopedics in patients with cleft lip and palate: a systematic review. *Cleft Palate-Craniofac J*. 2011;48:587.
47. Millard DR. A preliminary adhesion. In: Millard DR, ed. *Cleft Craft: The Unilateral Deformity*. vol. 1. Boston: Little, Brown; 1976.
48. Randall P. A lip adhesion operation in cleft lip surgery. *Plast Reconstr Surg*. 1965;35:371.
49. Rygh P, Tindlund RS. Early considerations in the orthodontic management of skeletodental discrepancies. In: Turvey TA, Vig KWL, Fonseca RJ, eds. *Facial Clefts and Craniosynostosis: Principles and Management*. Philadelphia: WB Saunders; 1996.
50. Tindlund RS. Orthopaedic protraction of the midface in the deciduous dentition: results covering 3 years out of treatment. *J Craniomaxillofac Surg*. 1989;17(suppl 1):17.
51. Ishikawa H, Kitazawa S, Iwasaki H, et al. Effects of maxillary protraction combined with chin-cap therapy in unilateral cleft lip and palate patients. *Cleft Palate Craniofac J*. 2000;37:92.
52. Tindlund RS. Skeletal response to maxillary protraction in patients with cleft lip and palate before age 10 years. *Cleft Palate Craniofac J*. 1994;31:295.
53. Berndt TJ. The features and effects of friendship in early adolescence. *Child Dev*. 1982;53:1447.
54. Ward JA, Vig KWL, Firestone AR, et al. Oral health-related quality of life in children with orofacial clefts. *Cleft Palate Craniofac J*. 2013;50:174.
55. Pope AW, Ward J. Factors associated with peer social competence in preadolescents with craniofacial anomalies. *J Pediatr Psychol*. 1997;22:455.
56. De Clerck HJ, Cornelis MA, Cevidanes LH, et al. Orthopedic traction of the maxilla with miniplates: a new perspective for treatment of midface deficiency. *J Oral Maxillofac Surg*. 2009;67:2123.
57. Baek SH, Kim KW, Choi JY. New treatment modality for maxillary hypoplasia in cleft patients. Protraction facemask with miniplate anchorage. *Angle Orthod*. 2010;80:783.
58. Abyholm FE, Bergland O, Semb G. Secondary bone grafting of alveolar clefts. *Scand J Plast Reconstr Surg Hand Surg*. 1981;15:127.
59. Boyne PJ. Use of marrow-cancellous bone grafts in maxillary alveolar and palatal clefts. *J Dent Res*. 1974;53:821.
60. Boyne PJ, Sands NR. Combined orthodontic-surgical management of residual palato-alveolar cleft defect. *Am J Orthod Dentofac Orthop*. 1976;70:20.
61. Turvey TA, Vig KWL, Moriarty J, et al. Delayed bone grafting in the cleft maxilla and palate: a retrospective multidisciplinary analysis. *Am J Orthod Dentofac Orthop*. 1984;86:3.
62. Vig KWL, Turvey TA, Fonseca RJ. Orthodontic and surgical considerations in bone grafting the cleft maxilla and palate. In: Turvey TA, Vig KWL, Fonseca RJ, eds. *Facial Clefts and Craniosynostosis: Principles and Management*. Philadelphia: WB Saunders; 1996.
63. Mølsted K, Brattström V, Prahl-Andersen B, et al. The Eurocleft Study: intercenter study of treatment outcome in patients with complete cleft lip and palate. Part 3: dental arch relationships. *Cleft Palate-Craniofac J*. 2005;42:78.
64. Hathaway RR, Daskalogiannakis J, Mercado AM, et al. The Americleft Study: an intercenter study of treatment outcomes for patients with unilateral cleft lip and palate. Part 2 – dental arch relationships. *Cleft Palate-Craniofac J*. 2011;48:244.
65. Murthy AS, Lehman JA. Evaluation of alveolar bone grafting: a survey of ACPA teams. *Cleft Palate-Craniofac J*. 2005;42:99.
66. Bergland O, Semb G, Abyholm FE. Elimination of the residual alveolar cleft by secondary bone grafting and subsequent orthodontic treatment. *Cleft Palate-Craniofac J*. 1986;23:175.
67. Semb G. *Analysis of the Oslo Cleft Lip and Palate Archive: Long Term Dentofacial Development* [Doctoral thesis]. Oslo, Norway: University of Oslo; 1991.
68. Troxell J, Fonseca RJ, Osbon D. A retrospective study of alveolar cleft grafting. *J Oral Maxillofac Surg*. 1982;40:721.
69. Vig KWL. Timing of alveolar bone grafting: an orthodontist's viewpoint. In: Lehman JA, Serafin D, eds. *Problems in Plastic and Reconstructive Surgery*. vol. 2. Philadelphia: JB Lippincott; 1992.
70. Vig KWL. Orthodontic considerations applied to craniofacial dysmorphology. *Cleft Palate Craniofac J*. 1990;27:141.
71. Vig KWL. Alveolar bone grafts: the surgical/orthodontic management of the cleft maxilla. *Ann Acad Med Singap*. 1999;28:721.
72. Zins JE, Whittaker LA. Membraneous vs endochondral bone autografts: implications for craniofacial reconstruction. *Surg Forum*. 1979;30:521.
73. Vachiramon A, Urata M, Kyung HM, et al. Clinical applications of orthodontic microimplant anchorage in craniofacial patients. *Cleft Palate-Craniofac J*. 2009;46:136.
74. Proff P, Bayerlein T, Gedrange T. Special features of planning and application of orthodontic miniscrews in cleft patients. *J Cranio-Maxillofac Surg*. 2006;34:73.
75. Cohen SR, Burstein FD, Stewart MB, et al. Maxillary-midface distraction in children with cleft lip and palate: a preliminary report. *Plast Reconstr Surg*. 1997;9:1421.
76. Losken HW, Vig KWL, Molina F. Distraction osteogenesis: indications, clinical application, and preliminary case reports. In: Turvey TA, Vig KWL, Fonseca RJ, eds. *Facial Clefts and Craniosynostosis: Principles and Management*. Philadelphia: WB Saunders; 1996.
77. Polley JW, Figueroa AA. Rigid external distraction: its application in cleft maxillary deformities. *Plast Reconstr Surg*. 1998;102:1360.
78. Minami K, Mori Y, Tae-Geon K, et al. Maxillary distraction osteogenesis in cleft lip and palate patients with skeletal anchorage. *Cleft Palate-Craniofac J*. 2007;44:137.
79. Kumar A, Gabbay JS, Nikjoo R, et al. Improved outcomes in cleft patients with severe maxillary deficiency after Le Fort I internal distraction. *Plast Reconstr Surg*. 2006;117:1499.
80. Shaw WC, Roberts CT, Semb G. Evaluating treatment alternatives: measurement and design. In: Turvey TA, Vig KWL, Fonseca RJ, eds. *Facial Clefts and Craniosynostosis: Principles and Management*. Philadelphia: WB Saunders; 1996.
81. Dalston RM, Marsh JL, Vig KWL, et al. Minimal standards for reporting the results of surgery on patients with cleft lip, palate or both: a proposal. *Cleft Palate-Craniofac J*. 1988;25:3.
82. Takahashi T, Fukuda M, Yamaguchi T, et al. Use of endosseous implants for dental reconstruction of patients with grafted alveolar clefts. *J Oral Maxillofac Surg*. 1997;55:576.
83. Kearns G, Perrott DH, Sharma AB, et al. Placement of endosseous implants in grafted alveolar clefts. *Cleft Palate-Craniofac J*. 1997;34:520.
84. Kramer FJ, Baethge C, Bremer B, et al. Dental implants in patients with orofacial clefts: a long-term follow-up study. *Int J Oral Maxillofac Surg*. 2005;34:715.
85. Brugnolo E, Mazzocco C, Cordioll G, et al. Clinical and radiographic findings following placement of single-tooth implants in young patients – case reports. *Int J Periodon Rest Dent*. 1996;16:421.
86. Knobloch LA. Prosthodontic treatment planning considerations for the replacement of missing teeth in the growing patient. *J Ped Dent Care*. 2005;11:8.

15

Patient Management and Motivation for the Child and Adolescent Patient

Patrick Turley and Patricia Turley

OUTLINE

Behavior Guidance, 395
Pain Management, 395
First Impressions, 395
Differences in Behavior Management Between Pediatric Dentistry and Orthodontics, 396
Communication Guidance, 396
Patient At-Home Responsibilities, 397
Oral Hygiene and Diet, 397
Removable Orthodontic Appliances, 398
Extraoral Appliances, 398
Headgear for Class II Correction, 398
Removable Intraoral Appliances, 398
Orthodontic Appointments, 399
Intraoral Elastics, 400
Removable Retainers, 400
Summary, 401

BEHAVIOR GUIDANCE

Behavior guidance skills are an important aspect of providing quality and efficient treatment for the child or adolescent patient. Patients seek our services for the treatment of dental diseases or malocclusions, but performing these services often requires modifying the child's behavior.

The American Academy of Pediatric Dentistry (AAPD) first published guidelines on behavior guidance in 1989,[1] with the most recent revision occurring in 2011.[2] Some of what is discussed in this chapter is based on that document. Behavior guidance is described as "a continuum of interaction involving the dentist, dental team, patient, and the parent directed toward communication and education. Its goal is to ease fear and anxiety while promoting an understanding of the need for good oral health and the process by which that is achieved."

In addition to allowing the orthodontist to perform quality treatment safely and efficiently, behavior guidance allows the doctor and staff to teach appropriate coping mechanisms; guide the child to be cooperative, relaxed, and self-confident in the dental setting; extinguish inappropriate behavior; and establish communication. Finally, behavior guidance allows the clinician to develop and nurture the relationships between the patient and doctor, which ultimately builds trust and allays fear and anxiety.

Pain Management

Pain management is essential for proper behavior guidance. Children respond differently to painful stimuli; hence, the orthodontist must be vigilant in listening to the patient and observing signs of pain such as facial expression, crying, complaining, and body movement.[3–7] Pain perception is strongly related to psychological characteristics. Dental anxiety has been shown to be higher in patients who were high pain responders, and they experienced more pain than low pain responders.[8] Inadequate pain management not only makes the delivery of treatment more difficult; it also makes future treatment more complicated. A painful past medical or dental experience is a common cause of fear and behavior problems in the dental environment.[9,10]

First Impressions

Setting the stage for optimum behavior can begin even before the patient presents to the office. Having a customized website introduces the parent and child to the practice and can begin to allay fears of the unexpected. The initial telephone call is extremely important in establishing rapport with the parent. These calls should be scripted, and every staff member who answers the phone should be trained to obtain all essential information while beginning to establish a trusting relationship. Many orthodontists have treatment coordinators (TCs) who are responsible for conducting most of these new patient calls.

When the child and parent present to the office for a first appointment, the receptionist should be trained to greet them in such a manner that they feel welcomed and special. Some practices have a bulletin board where the day's new patients' names are displayed. A tour of the office gives the patient the chance to view the areas where they will be treated, as well as areas of the practice where they can play and have fun while waiting for their appointments. The child needs to be assured that this first appointment will be an easy one.

Ultimately, it is the orthodontist who is responsible for delivering dental care and managing the associated patient behavior. The doctor must have good communication skills and be caring, supportive, and empathetic. In addition to being a clinician, the orthodontist is a teacher who must explain the dental health problems that the patient has and the procedures needed to resolve those problems. The doctor must guide the parent and child's behavior to achieve optimal care. The communication skills of the orthodontist are a large factor in patient satisfaction.[11,12] Some doctor behaviors are associated with low patient satisfaction; such behaviors include rushing through appointments, not taking the time to explain procedures, not allowing the parent in the treatment room, and being impatient.[13] On the other hand, studies have shown behaviors that are effective in managing uncooperative patients include directing, empathizing, persuading, giving the patient the feeling of control, and operant conditioning.[14–17]

Many factors affect the child's behavior in the dental environment. These factors include the child's age and cognitive level,[18–22] temperament and personality,[23–27] fear and anxiety levels,[18,24,28] the parent's dental anxiety,[29–31] reaction to strangers,[32] and previous dental experience.[18,20,29] Chapter 3 reviews psychological aspects also to be considered.

A parent who had negative dental experiences either as a child or adult may affect his or her child's behavior. In these situations, procedures focused on reducing parental anxiety are important. Although most children behave well when the parent is chairside, the presence of a parent can sometimes impede the communication between the child and dentist. Parenting style may also correlate with child behavior.[33] Children with authoritative parents exhibit more positive behavior and less caries than children with authoritarian or permissive parents. Children attending day care or school also exhibit better behavior than children who do not.[34]

DIFFERENCES IN BEHAVIOR MANAGEMENT BETWEEN PEDIATRIC DENTISTRY AND ORTHODONTICS

In pediatric dentistry, the focus on behavior management (BM) is the successful completion of in-office treatment procedures usually related to restorative dentistry. In very young children, BM techniques may be needed to perform simple intraoral examinations or the prophylaxis of teeth. In older children, BM techniques are necessary to allow restorative dentistry to be accomplished with such procedures as local anesthetic injection, rubber dam and clamp placement, use of a handpiece to remove decay, or the extraction of teeth. In children with a high caries rate, modifying the behavior of the child and the parent regarding oral hygiene practices and diet is also very important.

Behavior management in orthodontics has a different focus. Although some procedures may cause discomfort or pain, such as placing separators, fitting bands on partially erupted molars, or taking impressions on a patient prone to gagging, the focus in orthodontics is on modifying behavior outside the office. All patients must perform certain tasks in the course of their orthodontic treatment. A patient who best complies with assigned tasks is more apt to complete treatment on time with the desired result with and the absence of complications such as white spot lesions and decalcification.

The initial orthodontic examination should be a positive experience, with the orthodontist obtaining as much information as possible about the child's dental condition. Examination and treatment discussions should be done in a private setting so the parent feels free to openly discuss his or her children's needs. Many orthodontic practices have a new patient examination room and a TC who assists the doctor with examinations and case presentations. Based on examination findings, a treatment plan is discussed with the parent to address the problems identified. After the doctor leaves the room, the TC reviews with the parent and child the doctor's findings and treatment recommendations, finally asking if they have any unanswered questions. The TC should listen to the parent's response to make sure there is no misunderstanding. After the treatment discussion is complete, the TC then discusses financial arrangements. Patient education applications are available for iPads and personal computers to help parents visualize such things as malocclusion and treatment.

Communication Guidance

Implementing the procedures on the treatment plan will require optimal patient compliance. The AAPD recommends the following behavior guidance techniques: communication guidance, tell–show–do, voice control, nonverbal communications, positive reinforcement, and distraction.[2] All of these techniques become integrated into the daily routine of a well-functioning practice.

Especially for patients without much experience in the dental setting or for those who might have had a prior negative experience, "tell–show–do" is extremely important. The doctor should explain the exact steps of the appointment using age-appropriate wording. The doctor should show the patient all instruments, materials, and lights to be used during the procedure and match the size of the instruments to the size of the mouth. When treatment starts, the doctor should work quickly with constant positive reinforcement for younger patients such as "I am so proud of you" or "You are such a big boy/girl." Be patient; if the child becomes antsy, stop for a few seconds until the patient composes themselves and then resume treatment. You must be assured the patient is not in pain. If the patient states that something hurts, believe them and use additional pain control measures.

Although certain orthodontic problems can be identified early, such as a crossbite or Class II or III malocclusion, orthodontics is elective and should be postponed until the child is old enough to cooperate. The best guide for acceptable level of cooperation is to schedule beginning orthodontic records. A child who can accomplish impressions, radiographs, and photographs is mature enough to handle band fitting and impressions for an appliance. A child who is deemed too young to initiate orthodontic treatment should be recalled at 6-month intervals until the child is ready to start.

Tell–show–do is commonly used in orthodontics and involves verbal explanation of the procedure appropriate to the level of the patient and demonstration of the procedure in a nonthreatening environment. For example, the procedure of fitting bands on molars to fabricate an expansion appliance could be demonstrated as being like fitting a ring on a finger. We may have to try on a few before we find the correct size. Sometimes when attempting to fit bands on partially erupted permanent molars, the procedure becomes too painful. It is often best to switch to the second primary molar rather than have this

first appointment be a negative one. After bands are fitted, the patient is praised for their cooperation, and a brief tell–show–do is done for the impression. After the impression, praise is offered again for the successful completion of the appointment.

Impressions can be difficult for some patients. They feel the alginate is going to go down their throat, or they feel like they can't breathe, causing a gag reflex and sometimes vomiting. For these patients, the successful completion of an impression can be a game changer for their future happiness in the dental environment. Skinner's[35,36] method of successive approximations can be effective in this situation. It involves first reinforcing a behavior only vaguely similar to the one desired. Then you move on to behaviors that come a little closer to what you want. For patients who are extremely anxious, we may start by taking an impression of their fingers so they can sense what the alginate feels like and what the final product will be. Next try in a lower impression tray, smaller than the ideal but easy to insert; you may even have the patient self try in the tray themselves. If things go well, try in a tray correct for the child's arch, and if that is successful, attempt a lower impression. Not all impressions require extensive vestibular rolls. The orthodontist and staff need to know when to modify their procedures to make it more comfortable for the child. One scoop of alginate (not three) is sufficient to obtain all the teeth in the lower arch while minimizing the gag reflex. Flavors can be mixed into the alginate for anxious patients. Allow the child to select the flavor they want and have them smell the flavor they have chosen. Knowing ahead of time that the patient is a "gagger" obviously helps, but the child may not have had an impression before. Patients who are gaggers should not be seen right after a heavy breakfast or lunch. The lower impression should be taken first; it is easier to tolerate than the upper, and it lets the patient know that they can accomplish this procedure. The technique of distraction works well in these situations. While the tray is being inserted, talk to the patient about a subject other than the impression. If televisions are in view, have the patient focus on the movie and even talk about the scene they are watching. Counting from 1 to 10 gives the patient notice that the procedure will soon be over. Have the patient touch the alginate with their fingers to feel it turn into rubber. Seconds before removing the tray, tell the patient that it is going to come out now, and They will hear a "woosh" sound as the suction is broken. The tray is removed, and the staff, doctor, and parent cheer for the patient as if they just scored the winning goal in a soccer game.

PATIENT AT-HOME RESPONSIBILITIES

Successful orthodontic treatment requires the patient to perform certain procedures away from the office. Various factors influence the child's ability to comply with these tasks. Although the age and gender of the patient do not seem to influence compliance, academic performance is positively correlated.[37] Patients receiving above-average grades at school are also above-average compliers in the orthodontic setting. To obtain compliance with these procedures, the parent and patient must be educated on the importance of the task to the child's treatment, as well as how to perform the task. The method of presenting educational and motivational material is important. Studies have shown that the key to obtaining excellent patient compliance is spending time with patients and having good communication skills.[38,39] For communication to be effective, it must be remembered and understood.[40] Information written at the level of a 12-year-old child has been shown to be most effective.[41] The format of the written information also is important. Headings that stand out, short sentences, and use of the active rather than passive tense are recommended.[42–44] Technical jargon should be avoided.[45]

To help the patient and parent better understand the information being presented to them, we present it in multiple ways. Whenever possible, instruction is provided in oral, written, and visual forms. Supplementing verbal information with written and visual material has a positive effect on information recall, motivation for treatment, compliance, and treatment satisfaction.[39,46–48]

Oral Hygiene and Diet

Optimal oral hygiene requires professional instructions, adequate tools, and patient motivation, which are crucial factors to obtain compliance.[49] The day orthodontic appliances are placed, the patient and parent are given extensive instruction on home care and diet. We show a video that discusses the importance of plaque removal with brushing and flossing. Adjunctive aids such as electric toothbrushes, water picks, topical fluoride, floss threaders, and so on also are discussed. Using the home care kit we provide for each patient, the assistant then demonstrates proper brushing and flossing. Last, written instructions with photographs can also be given as reference. Diet is discussed with the focus on foods that can cause damage to the appliances or foods that can cause damage to the teeth. The avoidance of juices and soda with high acidity is stressed.

At subsequent appointments, oral hygiene instruction (OHI) needs to be a priority in treatment. Research has shown that repeated OHI and motivation significantly improved oral hygiene over patients who receive OHI only at the beginning of treatment.[50,51] Patients who received multiple motivational techniques, including plaque disclosure, demonstration of a horizontal brushing method, video of plaque bacteria, and the viewing of their own plaque under a phase contrast microscope, showed the greatest improvement in gingival health over a 6-month period.[52]

Oral hygiene should be graded and recorded at the beginning of each appointment. The dental staff needs to be trained on what exactly to say to the patient who presents with poor oral hygiene. Instruction should be given in a manner that is not embarrassing. Our responsibility is "to inform, not scorn."[53] Consider first pointing out areas where the patient is brushing well (if there any) and then ask the patient if he can see areas that need improvement. After giving additional OHI, tell the patient that you are confident he can improve his hygiene by the next appointment. Some practices have a reward system for patients who have excellent oral hygiene or no broken appliances at each appointment. Although one study found some improvement in oral hygiene scores, rewards did not improve appointment attendance or appliance wear and maintenance.[37]

New technology brings new ways to motivate patients. Apps and online programs are available that teach oral hygiene and braces care to children and teenage patients. Rather than lecturing patients when they are in the office, these apps engage patients the way they want to be engaged. Patients can also use these apps to send the office pictures if they have a problem such as a broken wire or bracket.[54] Sending motivational text messages to patients regarding their at-home responsibilities may improve compliance. Research showed that at-risk patients who were sent weekly text messages significantly increased their toothbrushing frequency.[55] Instituting a system for sending

motivational text messages could lead to better dietary choices, improved oral hygiene behaviors, and better compliance in wearing appliances and elastics.

Nothing slows the progress of treatment and wreaks havoc on the schedule like loose brackets or bands. Although a small percentage may be attributable to contamination that occurs during the bonding appointment, the majority are caused by patients eating foods they have been asked to avoid or habits such as pencil chewing or even bruxism. For repeat offenders, we stress the estimated completion date (ECD) may not be met and that treatment may be extended and compromised. These discussions should be done in a nonthreatening manner with the parent present.

REMOVABLE ORTHODONTIC APPLIANCES

Many types of removable orthodontic appliances are commonly used in everyday orthodontic practice. Treatment success, however, is based on the patient wearing the appliance as instructed. Optimal compliance includes both wear time (number of hours per day) and wear behavior. Wear behavior is related to whether the appliance was worn every day or whether there were days when the appliance was not worn. Studies have shown that a majority of patients may not wear their appliance as instructed[56]; hence, patient motivation is the highest of priorities when using removable appliances.

For every appliance that we use, we have a written handout that describes the appliance, its purpose, how to wear and care for it, what to expect in regard to discomfort, and what problems may occur along the way. This handout is given to the patient and parent to read before they leave the office, after which any questions they have are addressed. Basic information also can be given at the initial examination or consultation before the start of treatment. Numerous companies have videos that are designed to illustrate and educate the patient and parent in this regard.

EXTRAORAL APPLIANCES

Compliance with extraoral appliances is extremely important for the successful correction of various skeletal-type malocclusions. These appliances must be comfortable and easy to wear. Extraoral appliances can be especially problematic because they are visible and may embarrass the patient.

To obtain acceptable compliance, the orthodontist needs to go to great lengths in devising ways to motivate the patient. An example of the way we motivate patients to wear extraoral appliances involves the use of a face mask or reverse headgear for the correction of the Class III malocclusion. We have previously described using a face mask that is custom made to a plaster mold of the patient's face and attached to an intraoral cemented expansion appliance with elastics.[57] Because we request the child to wear the headgear to school the first 6 weeks, we spend considerable time in educating the parent and child on the importance of compliance. From age 5 to 7 years, children will do whatever the teacher, doctor, or parent requests, of them. It is winning over the parent that is the most difficult. The following methods are important in obtaining the compliance requested. Thorough explanation of the structural etiology of the malocclusion is important, stressing that patients who become adults with this type of malocclusion may require surgery of the jaws in order to correct it. We describe the mode of action of the appliances to be used, keeping an open mind on full-time wear until the appliances are finally delivered. We advise the parent to meet with the teacher to discuss the child's wearing the headgear to school. Plan for the teacher, parent, or TC to perform a tell–show–do to the class. Using a cartoon-type drawing of the face mask, have each member of the class design a face mask with his or her favorite colors, stickers, and so on. Be excited at the delivery appointment when the custom appliance is first shown and delivered. Make sure the appliance fits well and is comfortable. Provide thorough instructions on how and when to wear the appliance and how to store it safely when it is not being worn. Have the child fill out a time card, recording the hours of wear each day. Measure overjet at each appointment, and inform the patient and parent as to the improvement in that measurement, providing praise and encouragement. As the overjet improves, reduce the hours per day as promised. This rewards the patient for his cooperation and sends a message to the parent and child that treatment is progressing as planned.

HEADGEAR FOR CLASS II CORRECTION

Cervical traction headgear is one of the appliances used for Class II correction. The use of this appliance, however, has decreased over the past several decades, in favor of removable functional appliances purposed to stimulate mandibular growth and more recently by fixed-functional or distalizing appliances that require less patient cooperation.[58] Rather than abandoning headgear completely, we accept the fact that a certain percentage of patients will not comply and a certain percentage will.[59] Why totally discard the appliance because of the patients who won't wear it? We have found that patients generally fall into three groups. Group 1 wears the appliance as instructed and reports it as being easy to wear and tolerate. Group 2 wears the appliance but doesn't like it and struggles to get the required hours. Group 3 patients don't like it, can't sleep with it, won't wear it, and parents choose not to force the issue and request other options. Having an open discussion with both the patient and parent at the treatment planning stage can help identify whether or not the patient is a good candidate for cervical headgear. After establishing cervical headgear as the treatment of choice and the child agreeing to wear it, there are certain things that can increase compliance. The appliance should be comfortable and easy to insert and remove. The inner bow should insert easily into the buccal tubes such that it can be inserted and removed with the slightest of finger pressure. Adjustments to rotate molars should wait until the appliance is comfortable to wear and the patient has shown compliance. The innerbow–outerbow connection should be positioned comfortably 3 to 5 mm in front of the upper incisors at the level of the lip embrasure. Adjust the outer bow close to the face to make it easier to sleep in. Provide neck straps that are comfortable and available in assorted colors. Keep forces low at the beginning. Have the patient record her hours and days of wear, and provide praise or rewards for good compliance. Measure overjet and molar relation at each appointment and provide praise as the measures improve. Some patients may not show improvement in overjet and molar relation even though they are wearing the headgear. Don't let them become discouraged. Measurable improvement may take more time for some patients.

REMOVABLE INTRAORAL APPLIANCES

The removable Hawley appliance is a commonly used appliance that can incorporate springs for individual tooth movement, an

anterior bite plate to aid Class I or Class II deep bite correction, activation of the labial bow to retract anterior teeth and close spaces, and even expansion incorporating a jackscrew. Because these appliances are most commonly lost away from home, especially when taken out to eat, we have some patients wear them only at home and most learn to eat with them in their mouths. The Hawley appliance can be made in special colors or designs, and each is delivered along with a case for storage when the appliance is not being worn. The appliance should be brushed along with toothbrushing and especially before placing it in its case, where plaque can dry on the appliance, making it "grungy." Dogs (and cats!) love to chew on these appliances even when they're inside the case; hence, patients are instructed to "beware of dogs." The appliance is easy to wear, and hence compliance is generally good.

A study examining wear time using a temperature-sensitive microsensor found that patients wore a removable appliance only 7.6 hours per day even though 15 to 16 hours per day was prescribed.[60] Research has shown that to improve compliance, wear time must be measured.[61] Newly developed temperature-sensitive microsensors, incorporated into removable appliances by polymerization, can provide data that can be discussed with patients at their appointments.[62–64] Wear time documentation is reported to be well received by patients[65] and has a positive effect on adherence.[66] Wear time measurement is especially important when treatment does not progress satisfactorily. Rather than engaging in a contentious discussion, which can strain the doctor–patient relationship, objective data can be used to discuss patient behavior and possible alternate therapies.

Removable appliances should be delivered in a passive state to allow the appliance to settle or seat into the dental arch and allow the patient to adjust to having something in the mouth. After the appliance has seated and the patient has adjusted to it, the appliance can then be adjusted by tightening the retention clasps and then the active components. The patient should be instructed that some tooth soreness is to be expected and should subside after a few days. Tissue impingement should be reported to the orthodontist so that comfort adjustments can be made.

In the 1980s, the "British invasion" occurred in US orthodontics. No, it wasn't the Beatles, and it was more a "European invasion" that found removable functional appliances now being advocated for Class II malocclusions (See Chapters 16 and 35). As American orthodontists became experienced with these appliances and long-term data began to be published, the effects on mandibular length were less than expected, although they were excellent appliances in correcting the Class II malocclusion. The major problem with them (i.e., the bionator, activator, Frankel appliance), however, was patient compliance. They are bulky appliances; difficult to wear; need to be removed for eating and cleaning; and similar to the cervical headgear, were not worn well by a significant portion of the patients for which they were prescribed. Because of concerns for compliance, these appliances have fallen out of favor in the United States in favor of fixed functional appliances such as the Herbst, MARA, or various spring-loaded modules.[58] See Chapter 16 and 35 for a thorough discussion of these appliances.

An increasing number of adolescents are requesting treatment with clear removable aligners. The appropriateness of such treatment depends on the type and severity of the malocclusion; the experience of the orthodontist; and, of course, the anticipated cooperation of the patient. Although these patients appear committed and are enthusiastic about this treatment choice over traditional braces, adolescents are still adolescents, and enthusiasm can wane. A patient who doesn't wear the trays as instructed, misses an appointment for additional trays, or loses a set of trays can derail the progress of treatment, frustrating the doctor and parents while increasing costs and treatment time. Proper oral hygiene procedures are important, and the avoidance of soda and fruit juices while wearing the trays is stressed. Cases of decalcification with clear aligner therapy have been reported.[67]

ORTHODONTIC APPOINTMENTS

Keeping regular appointments is integral to the success of orthodontic treatment. Missed appointments can lengthen treatment and increase the chances of root resorption, white spot lesions, and periodontal problems.[68–70] Researchers have investigated factors affecting children's adherence to regular dental appointments.[71] Studies have shown a significant correlation with the parents' level of education,[72–80] economic status,[78,79,81–83] and marital status.[73] A missed appointment is usually followed by a telephone call from the office and a message to reschedule. If the family does not respond to the telephone contact, then letters can be sent with increasing seriousness. The last letter is sent registered and informs the family that the orthodontist is discontinuing treatment because of lack of cooperation. What could have been done earlier in treatment to avoid ending up in this situation? There are many situations that can interfere with maintaining orthodontic appointments. A family with multiple children attending multiple schools and participating in different school activities makes scheduling orthodontic appointments difficult. Crises also can upset the routine of life and the routine of keeping orthodontic appointments. Death, divorce, or the diagnosis of a family member with cancer or other serious illness may take precedence over keeping orthodontic appointments, but they shouldn't. Just as children will continue to attend school, and after-school activities, they need to keep their orthodontic appointments. Keeping regular appointments needs to be stressed both in the written material presented to the family as well as in verbal discussions at the beginning of treatment.

Modifying behavior is best done by rewarding behavior that we want repeated. This should be kept in mind when calling patients who have missed their appointment. "Mrs. Jones, this is Cari calling from Dr. Turley's office. Jeremy missed his appointment yesterday, and we were a little concerned because he is one of our patients who is always on time."[53]

Getting child patients to accept some responsibility for keeping their appointments also can improve compliance. Patients assume a lot of responsibility for getting to their after-school activities, such as sports teams and music lessons. Children know when their next practices are scheduled and are commonly involved in arranging transportation. Orthodontic appointments, however, are generally left up to the parent, with some being less than responsible in making it a top priority. When a pattern of missed appointments appears, our staff communicates with the parent, but we also discuss it with the child, especially if they are in high school and responsible for other outside school activities. We first discuss how the child's pattern of missed appointments is going to extend the treatment time, meaning he probably won't be getting his braces off when they

expected. We'll ask him, "Does your mother need to remind you when volleyball practice is? No, of course not. In fact, you're the one who's reminding your parent that you need to be at practice at a certain day and time. Well, you need to start thinking of your orthodontic appointments the same way. If you want your braces off as soon as possible with the best results, when you leave today and make your next appointment, write that date and time in your own phone calendar and remind your parent as the date is approaching that you need to be at that appointment to keep your treatment progressing as scheduled." This adult-type conversation is appreciated by the parent and often changes a noncompliant family into a cooperative one. The doctor's communication skills have been shown to have a positive influence in families adhering to dental appointments.[71] On the other hand, an unpleasant dental visit, dissatisfaction with previous appointments, uncertainty about dental treatment and its import, children's aversion to dental visits, and dental care–related anxiety can have negative effects regarding parents' adherence to keeping dental appointments.[72,77,79–80,83–85]

INTRAORAL ELASTICS

Intraoral elastics are an integral part of treating many orthodontic patients. If possible, the patient should know early in treatment that wearing elastics will be an important part of achieving an ideal result. Patients report pain, laziness, forgetfulness, and embarrassment as reasons for not wearing elastics (or headgear).[86] When elastic wear is scheduled, sufficient time needs to be given to instruct the patient how to place and remove the elastics and how often to wear them. An information sheet showing the position of the elastics is given to the patient. This information sheet should describe when and where to wear the elastics, where to keep elastics at home, how to carry spare elastics when not at home, and how to manage problems with elastics.[87] Researchers found that elastic wear was related to three subcategories of factors: remembering to wear or change elastics; physically having elastics available; and a series of barriers that impeded wearing or changing elastics, such as eating, pain or discomfort, difficulty in placement, and social concerns. Cues from parents or caregivers can help remind forgetful patients. Elastic force can be light at the beginning or delayed until the soreness of the recent archwire adjustment diminishes; then heavier elastics can be initiated. When placing elastics, start with the most posterior tooth and pull forward and engage the most anterior tooth. Multiple packs of elastics should be given so there is never an excuse to not wear them. A pack can be carried in the school backpack, another in the bathroom at home, at one's desk, in the car, or in any other familiar place. Elastic placement should be diagrammed on each pack. In the case of triangular elastics, different colored bracket ties can be used on the teeth to receive the elastics. Colored elastics may motivate some patients. Most patients do well wearing the elastics to bed and after breakfast, but many forget to replace them after lunch.

If elastic wear is found to be inadequate, we recommend they be worn while eating, replacing them when they break. Make patients aware of the goal you're trying to achieve (i.e., how much overjet or midline correction). Measure overjet and occlusal relationships at each appointment, and give praise for positive changes. If progress is ignored at the next appointment, the patient will lose motivation. Patients may telephone the office stating that they have forgotten where to attach the elastics or that they have lost them. Elastics should be diagrammed and the size and force recorded on the treatment card so the front desk staff can correctly assist the patient. "I lost them 4 weeks ago" is not an acceptable excuse for not wearing elastics. Packs of elastics can be mailed to the patient, saving them a trip to the office. Some patients get confused and reverse elastics (e.g., wear a Class III direction rather than Class II). At the end of every appointment, have the patient place her elastics so the doctor can make sure they are being worn correctly. Apprise each patient of the ECD of the treatment. Patients who are not compliant may need to have the ECD extended. Seeing the doctor cross out the ECD and write in a later date or a question mark can be effective in getting the patient's attention. Motivated patients can often accomplish more in 8 weeks than they did the previous 8 months.

REMOVABLE RETAINERS

It is important for patients to have realistic expectations about retention. No retainers hold the teeth 100%, even bonded retainers. Patients can expect to see or feel changes equal in magnitude to their last adjustment appointment. All teeth move when the braces come off. Our hope is that they will move but not become "crooked." See Chapter 33 for further discussion.

We offer three types of retainers at the end of treatment: bonded lingual retainers, clear aligners, and Hawley retainers. The pros and cons of each are discussed at the last adjustment appointment, and impressions are taken that day if a bonded retainer is chosen. Similar to any other removable appliance, a significant percentage of patients will not follow the prescribed regimen for wear and will experience posttreatment tooth movement. Because these patients are now older adolescents or adults, they are often beyond the stage of motivation.[88] Before scheduling the removal of braces, have the patient tell you truthfully if he can adhere to wearing removable retainers. If there is any doubt, a bonded retainer, especially in the lower arch, should be used, with a clear upper aligner for home wear. Hawleys are used for the most compliant patients. I tell patients that one patient per year fails to wear his retainers properly and needs to have braces placed back on to restraighten the teeth (usually the lowers), stating, "I don't want you to be that patient!" We offer a second set of removable retainers at 50% discount if made at the time of debanding. Most patients take advantage of this offer. Some adult patients prone to relapse may benefit from "belt and suspenders," a bonded retainer with a removable retainer to fit over it for insurance. As with other adjunctive appliances, verbal instruction is given in addition to a written handout.

We use a form that both the patient and parent sign indicating the doctor's preference and choice of retainers. The form reinforces the pros and cons of each type, as well as our policy of a 50% discount for a second set. In the past, we would occasionally have a patient not wear or lose her retainers, with relapse occurring and the need for additional treatment. An upset parent would state that she was not adequately informed or given the choice of a bonded retainer. We even had a parent who demanded a new free retainer because she was unhappy with the color chosen by the child without consulting the parent. These issues have not occurred since we initiated the use of this signed form.

Certain catchphrases can help remind and motivate patients to wear their retainers as prescribed. Nighttime wear can be

described as "pajamas for your teeth." When you get ready for bed, you put on your pajamas, and you put on your retainers. We describe retainers as an "insurance policy" for your teeth—an insurance policy you've already paid for! As long as you continue to wear retainers at some interval, you can be reasonably assured of continuing to have straight teeth. Many patients are concerned that they will have to wear retainers every night for the rest of their lives. Although this may be true for a small number of patients, most will be able to reduce their wear over the years. Every night will decrease to every other night, to once a week, and to twice a month as time passes.

SUMMARY

1. The key to obtaining excellent patient compliance is spending time with patients and having good communication skills.
2. For communication to be effective, it must be both understood and remembered.
3. Proper parent and patient education leads to increased compliance.
4. Good verbal, visual, and written explanations are necessary.
5. All staff members should be trained so the messages are clear and consistent.
6. Systems to identify less than optimal behavior and methods to modify behavior and obtain compliance should be developed and continually updated.

REFERENCES

1. American Academy of Pediatric Dentistry. *Proceedings of the Consensus Conference: Behavior Management for the Pediatric Dental Patient*. Chicago: American Academy of Pediatric Dentistry; 1989.
2. American Academy of Pediatric Dentistry. Guideline on behavior guidance for the pediatric dental patient. *Pediatr Dent*. 2011;36(6):179–191.
3. Nutter DP. Good clinical pain practice for pediatric procedure pain: iatrogenic considerations. *J Calif Dent Assoc*. 2009;37(10):713–718.
4. Nutter DP. Good clinical pain practice for pediatric procedure pain: neurobiologic considerations. *J Calif Dent Assoc*. 2009;37(10):705–710.
5. Nutter DP. Good clinical pain practice for pediatric procedure pain: target considerations. *J Calif Dent Assoc*. 2009;37(10):719–722.
6. Nakai Y, Milgrom P, Mancl L, et al. Effectiveness of local anesthesia in pediatric dental practice. *J Am Dent Assoc*. 2000;131(12):1699–1705.
7. American Academy of Pediatric Dentistry. Use of local anesthesia for pediatric dental patients. *Pediatr Dent*. 2010;32(special issue):141–147.
8. Beck VJ, Farella M, Chandler NP, et al. Factors associated with pain induced by orthodontic separators. *J Oral Rehabil*. 2014;41:282–289.
9. Versloot J, Veerkamp JSJ, Hoogstraten J. Children's self-reported pain at the dentist. *Pain*. 2008;137(2):389–394.
10. Klingberg G. Dental anxiety and behavior management problems in pediatric dentistry: a review of background factors and diagnostics. *Eur Arch Dent*. 2007;8(4):11–15.
11. Gale EN, Carlsson SG, Eriksson A, Jontell M. Effects of dentists' behavior on patients' attitudes. *J Am Dent Assoc*. 1984;109(3):444–446.
12. Schouten BC, Eijkman MA, Hoogstraten J. Dentists' and patients' communicative behavior and their satisfaction with the dental encounter. *Community Dent Health*. 2003;20(1):11–15.
13. Reichard A, Turnbull HR, Turnbull AP. Perspectives of dentists, families, and case managers on dental care for individuals with developmental disabilities in Kansas. *Ment Retard*. 2001;39(4):268–285.
14. Wright GZ, Stigers JI. Nonpharmacologic management of children's behaviors. In: Dean JA, Avery DR, McDonald RE, eds. *McDonald and Avery's Dentistry for the Child and Adolescent*. 9th ed. Maryland Heights, MO: Mosby-Elsevier; 2011:27–40.
15. Feigal RJ. Guiding and managing the child dental patient: a fresh look at old pedagogy. *J Dent Educ*. 2001;65(12):1369–1377.
16. Weinstein P, Getz T, Raetener P, Domoto P. The effect of dentists' behavior on fear-related behaviors in children. *J Am Dent Assoc*. 1982;104(1):32–38.
17. Sarnat H, Arad P, Hanauer D, Shohami E. Communication strategies used during pediatric dental treatment: a pilot study. *Pediatr Dent*. 2001;23(4):337–342.
18. Baier K, Milgrom P, Russell S, et al. Children's fear and behavior in private pediatric dentistry practices. *Pediatr Dent*. 2004;26(4):316–321.
19. Rud B, Kisling E. The influence of mental development on children's acceptance of dental treatment. *Scand J Dent Res*. 1973;81(5):343–352.
20. Brill WA. The effect of restorative treatment on children's behavior at the first recall visit in a private pediatric dental practice. *J Clin Pediatr Dent*. 2002;26(4):389–394.
21. Allen KD, Hutfless S, Larzelere R. Evaluation of two predictors of child disruptive behavior during restorative dental treatment. *J Dent Child*. 2003;70(3):221–225.
22. Cunha RF, Delbem ACB, Percinoto C, Melhado FL. Behavioral evaluation during dental care in children ages 0 to 3 years. *J Dent Child*. 2003;70(2):100–103.
23. Klingberg G, Broberg AG. Temperament and child dental fear. *Pediatr Dent*. 1998;20(4):237–243.
24. Arnup K, Broberg AG, Berggren U, Bodin L. Lack of cooperation in pediatric dentistry: the role of child personality characteristics. *Pediatr Dent*. 2002;24(2):119–128.
25. Radis FG, Wilson S, Griffen AL, Coury DL. Temperament as a predictor of behavior during initial dental examination in children. *Pediatr Dent*. 1994;16(2):121–127.
26. Lochary ME, Wilson S, Griffen AL, Coury DL. Temperament as a predictor of behavior for conscious sedation in dentistry. *Pediatr Dent*. 1993;15(5):348–352.
27. Jensen B, Stjernqvist K. Temperament and acceptance of dental treatment under sedation in preschool children. *Acta Odontol Scand*. 2002;60(4):231–236.
28. Arnup K, Broberg AG, Berggren U, Bodin L. Treatment outcome in subgroups of uncooperative child dental patients: an exploratory study. *Int J Paediatr Dent*. 2003;13(5):304–319.
29. Klingberg G, Berggen U, Carlsson SG, Noren JG. Child dental fear: cause related factors and clinical effects. *Eur J Oral Sci*. 1995;103(6):405–412.
30. Johnson R, Baldwin DC. Maternal anxiety and child behavior. *J Dent Child*. 1969;36(2):87–92.
31. Peretz B, Nazarian Y, Bimstein E. Dental anxiety in a students' pediatric dental clinic: children, parents and students. *Int J Paediatr Dent*. 2004;14(3):192–198.
32. Holst A, Hallonsten AL, Schroder U, et al. Prediction of behavior-management problems in 3-year-old children. *Scand J Dent Res*. 1993;101(2):110–114.
33. Baumrind D. Parental disciplinary patterns and social competence in children. *Youth Soc*. 1978;9:239–251.
34. Howenstein J, Kumar A, Casamassimo PS, et al. Correlating parenting styles with child behavior and caries. *Pediatr Dent*. 2015;37:59–64.
35. Skinner BF. *Science and Human Behavior*. New York: Macmillan; 1953.
36. McLeod SA, Skinner BF. *Operant Conditioning*; 2015. Retrieved from http://www.simplypsychology.org/operant-conditioning.html.
37. Richter DD, Nanda RS, Sinha PK, Smith DW. Effect of behavior modification on patient compliance in orthodontics. *Angle Orthod*. 1998;68:123–132.
38. Aljabaa A, McDonald F, Newton JT. A systematic review of randomized controlled trials of interventions to improve adherence among orthodontic patients aged 12 to 18. *Angle Orthod*. 2015;85:305–313.
39. Thickett E, Newton JT. Using written material to support recall of orthodontic information: a comparison of three methods. *Angle Orthod*. 2006;76:243–250.
40. Ley P, Spelman MS. *Communicating with the Patient*. London: Staples Press; 1967:114–116.
41. Ley P, Florio T. The use of reading formulas in health care. *Psychol Health Med*. 1996;1:7–28.
42. Kitching N, Low L, Evers C. Use of language. Entitled to respect. *Nurs Elder*. 1990;2:9.

43. Ong G, Austoker J, Brouwer A. Evaluation of written information sent to women who are called back for further investigation of breast screening in the UK. *Health Educ J*. 1996;55:413–429.
44. Petterson T. How readable are the hospital information leaflets available to elderly patients? *Age Ageing*. 1994;23:14–16.
45. Alexander RE. Patient understanding of postsurgical instruction forms. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 1999;87:153–158.
46. Thomson AM, Cunningham SJ, Hunt NP. A comparison of information retention at an initial orthodontic consultation. *Eur J Orthod*. 2001;23:169–178.
47. Mortensen MG, Kiyak HA, Omnell L. Patient and parent understanding of informed consent in orthodontics. *Am J Orthod Dentofacial Orthop*. 2003;124:541–550.
48. Wright NS, Fleming PS, Sharma PK, Battagel J. Influence of supplemental written information on adolescent anxiety, motivation and compliance in early orthodontic treatment. *Angle Orthod*. 2010;80:329–335.
49. Van der Weijden F, Slot DE. Oral hygiene in the prevention of periodontal diseases: the evidence. *Periodontol 2000*. 2011;55:104–123.
50. Marini I, Bortolotti F, Parenti SI, et al. Combined effects of repeated oral hygiene motivation and type of toothbrush on orthodontic patients: a blind randomized clinical trial. *Angle Orthod*. 2014;84(5):896–901.
51. Boyd RL. Longitudinal evaluation of a system for self-monitoring plaque control effectiveness in orthodontic patients. *J Clin Periodontal*. 1983;10:380–388.
52. Acharya S, Goyal A, Utreja AK, Mohanty U. Effect of three different motivational techniques on oral hygiene and gingival health of patients undergoing multibracketed orthodontics. *Angle Orthod*. 2011;81:884–888.
53. White C. Empowering Patient Communication-Ortho2. 2009;27(2):1–3.
54. Zak AJ. Paving an interactive path toward patient compliance: how Mighty Brace aims to keep young patients engaged with their orthodontic health. *Orthodontic Products*. 2014:16.
55. Schluter P, Lee M, Hamilton G, et al. Keep on brushing: a longitudinal study of motivational text messaging in young adults aged 18-24 years receiving work and income support. *J Public Health Dent*. 2015;75(2):118–125.
56. Schott TC, Ludwig B. Microelectronic wear-time documentation of removable orthodontic devices detects heterogeneous wear behavior and individualizes treatment planning. *Am J Orthod Dentofacial Orthop*. 2014;146:155–160.
57. Turley PK. Orthopedic correction of class III malocclusion with palatal expansion and custom protraction headgear. *J Clin Orthod*. 1988;22(5):314–325.
58. Keim RG, Gottlieb EL, Vogels DS, Vogels PB. 2014 JCO study of orthodontic diagnosis and treatment procedures: part 1 results and trends. *J Clin Orthod*. 2014;10:607–630.
59. Sandler J, Murray A, Thiruvenkatachari B, et al. Effectiveness of 3 methods of anchorage reinforcement for maximum anchorage in adolescents: a 3-arm multicenter randomized clinical trial. *Am J Orthod Dentofacial Orthop*. 2014;146(1):10–20.
60. Witt E, Bartsch A, Sahm G. The wear-time measuring device in orthodontics-cui bono? Reflections on the state-of-the-art wear-timing measurement and compliance research in orthodontics. *Fortschr Kieferorthop*. 1991;52:117–125.
61. Schäfer C. *Patientencompliance-Messung, typologie, erfolgsfaktoren*. Wiesbaden, Germany: Gabler Verlag; 2011.
62. Schott TC, Göz G. Wearing times of orthodontic devices as measured by the TheraMon® Microsensor. *J Orofac Orthop*. 2011;72:103–110.
63. Schott TC, Ludwig B, Glasl BA, Lisson JA. A microsensor for monitoring removable appliance wear. *J Clin Orthod*. 2011;45:518–520.
64. Schott TC, Schlipf C, Glasl B, et al. Quantification of patient compliance with Hawley retainers and removable functional appliances during the retention phase. *Am J Orthod Dentofacial Orthop*. 2013;144:533–540.
65. Schott TC, Schrey S, Walter J, et al. Questionnaire study of electronic wear-time tracking as experimented by patients and parents during treatment with removable orthodontic appliances. *J Orofac Orthop*. 2013;74:217–225.
66. Ackerman MB, Thornton B. Post treatment compliance with removable maxillary retention in a teenage population: a short-term randomized clinical trial. *Orthodontics (Chic)*. 2011;12:22–27.
67. Moshiri M, Eckhart JE. Consequences of poor oral hygiene during clear aligner therapy. *J Clin Orthod*. 2013;47(8):494–498.
68. Zachrisson BU. Cause and prevention of injuries to teeth and supporting structures during orthodontic treatment. *Am J Orthod*. 1976;69:285–300.
69. Skidmore KJ, Brook KJ, Thomson WM, Harding WJ. Factors influencing treatment time in orthodontic patients. *Am J Orthod Dentofacial Orthop*. 2006;129:230–238.
70. Apajalahti S, Peltola JS. Apical root resorption after orthodontic treatment—a retrospective study. *Eur J Orthod*. 2007;29:408–411.
71. Badri P, Saltaji H, Flores-Mir C, Amin M. Factors affecting children's adherence to regular dental attendance: a systematic review. *J Am Dent Assoc*. 2014;145(8):817–828.
72. Brickhouse TH, Farrington FH, Best AM, Ellsworth CW. Barriers to dental care for children in Virginia with autism spectrum disorders. *J Dent Child*. 2009;76(3):188–193.
73. Quiñonez RB, Pahel BT, Rozier RG, Stearns SC. Follow-up preventive dental visits for Medicaid-enrolled children in the medical office. *J Public Health Dent*. 2008;68(3):131–138.
74. Reiss ML, Piotrowski WD, Bailey JS. Behavioral community psychology: encouraging low-income parents to seek dental care for their children. *J Appl Behav Anal*. 1976;9(4):387–397.
75. Talekar BS, Rozier RG, Slade GD, Ennett ST. Parental perceptions of their preschool-aged children's oral health. *J Am Dent Assoc*. 2005;136(3):364–372.
76. Liena Puy MC, Ausina Mārquez V. Compliance with scheduled visits at an area preventive odontology consulting clinic [in Spanish]. *Aten Primaria*. 1997;20(5):255–258.
77. Leroy R, Bogaerts K, Hoppenbrouwers K, et al. Dental attendance in preschool children: a prospective study. *Int J Paediatr Dent*. 2013;23(2):84–93.
78. Denloye OO, Bankole OO, Onyeaso CO. Dental health service utilization by children seen at the University College Hospital: an update. *Odontostomatol Trop*. 2004;27(108):29–32.
79. Goettems ML, Ardenghi TM, Demarco FF, et al. Children's use of dental services: influence of maternal dental anxiety, attendance pattern, and perception of children's quality of life. *Community Dent Oral Epidemiol*. 2012;40(5):451–458.
80. Kelly SE, Binkley CJ, Neace WP, Gale BS. Barriers to care-seeking for children's oral health among low-income caregivers. *Am J Public Health*. 2005;95(8):1345–1351.
81. Hoeft KS, Barker JC, Masterson EE. Maternal beliefs and motivations for first dental visit by low-income Mexican American children in California. *Pediatr Dent*. 2011;33(5):392–398.
82. Amin MS. Utilization of dental services by children in low-income families in Alberta. *J Can Dent Assoc*. 2011;77:b57.
83. Naidu R, Nunn J, Forde M. Oral healthcare of preschool children in Trinidad: a qualitative study of parents and caregivers. *BMC Oral Health*. 2012;12:27.
84. Vanobbergen JN, Van Der Beken R, Gyssels L, et al. Barriers to oral health care access among socially vulnerable groups: a qualitative study. *J Disab Oral Health*. 2007;8(2):63–69.
85. Vignarajah S. Oral health knowledge and behaviours and barriers to dental attendance of school children and adolescents in the Caribbean Island of Antigua. *Int Dent J*. 1997;47(3):167–172.
86. Egolf RJ, Begole EA, Upshaw HS. Factors associated with orthodontic patient compliance with intraoral elastics and headgear. *Am J Orthod Dentofacial Orthop*. 1990;97:336–348.
87. Veeroo H, Cunningham SJ, Newton JT, Travess HC. Motivation and compliance with intraoral elastics. *Am J Orthod Dentofacial Orthop*. 2014;146:33–39.
88. Hyun P, Preston CB, Al-Jewair TS, Tabbaa S. Patient compliance with Hawley retainers fitted with the SMART® sensor: a prospective clinical pilot study. *Angle Orthod*. 2015;85(2):263–269.

16

Optimizing Orthodontics and Dentofacial Orthopedics: Treatment Timing and Mixed Dentition Therapy

James A. McNamara, Laurie McNamara McClatchey, and Lee W. Graber

OUTLINE

The Timing of Treatment Intervention, 404
Modification of Craniofacial Growth, 404
Patient Cooperation, 404
Practice Management, 404
The Cervical Vertebral Maturation Method, 405
When to Intervene, 408
Treatment of Tooth-Size/Arch-Size Discrepancy Problems, 409
Space Maintenance during the Transition of the Dentition, 409
Transpalatal Arch, 409
Lingual Arch, 410
Serial Extraction, 410
Arch Expansion, 412
Types of Expansion, 412
Orthodontic Expansion, 412
Passive Expansion, 412
Orthopedic Expansion, 412
Rationale for Early Orthopedic Expansion, 412
Permanent Dentition, 413
Mixed Dentition, 413
Orthopedic Expansion Protocols, 413
Maxillary Adaptations, 413
Mandibular Adaptations, 414
Mandibular Dental Uprighting and Expansion Appliances, 414
The Schwarz Appliance, 414
Lip Bumper, 416
Spontaneous Improvement of Sagittal Malocclusions, 416
Class II Patients, 416
Class III Patients, 418
The Treatment of Class II Malocclusion, 419
Components of Class II Malocclusion, 419
Available Class II Treatment Strategies, 419
Maxillary Distalization, 419
Extraoral Traction, 419
Maxillary Molar Distalization, 420
Pendulum and Pendex Appliances, 421
Mandibular Enhancement: Functional Jaw Orthopedics, 422
Appliance Selection, 423
Herbst Appliance, 423
Mandibular Anterior Repositioning Appliance, 425
The Twin Block Appliance, 425
Treatment Timing for Class II Malocclusion, 426
Additional Comments Regarding Class II Treatment, 426
Treatment of Class III Malocclusion, 427
Components of Class III Malocclusion, 427
Available Class III Treatment Strategies, 427
Appliance Selection, 427
The Orthopedic Facial Mask, 427
The FR-3 Appliance of Fränkel, 429
The Orthopedic Chin Cup, 429
Additional Comments Regarding Class III Treatment, 432
Summary, 432

This chapter describes the integration of various orthodontic and orthopedic protocols that can be used to treat the myriad of dentoskeletal problems seen routinely in orthodontic practice. Some malocclusions respond well when orthodontic treatment is begun in the mixed dentition; other conditions are treated optimally at the time of the circumpubertal growth spurt or even later. The nature of the problem, as revealed by the process of proper differential diagnosis and treatment planning, determines whether intervention is best begun early (as in the early mixed dentition) or late (late mixed or permanent dentition).

If a late treatment is initiated, a single phase of comprehensive orthodontic therapy usually is involved, the duration of which typically spans anywhere from 12 to 30 months. If treatment begins in the early mixed dentition, however, in most instances, a two-phase treatment protocol is anticipated, with a second phase of fixed orthodontic appliances required in the vast majority of such patients.

The goal of such early treatment is to correct existing or developing skeletal, dentoalveolar, and muscular imbalances to improve the orofacial environment before the eruption of the permanent dentition is complete. By initiating orthodontic and orthopedic treatment at a younger age, the overall need for complex orthodontic treatment involving permanent tooth extraction and orthognathic surgery presumably is reduced.

THE TIMING OF TREATMENT INTERVENTION

A topic of much conversation and debate among orthodontists and even the lay public has been orthodontic treatment timing, with articles concerning this subject appearing not only in refereed orthodontic journals but also occasionally in such publications as *The New York Times*, *The Wall Street Journal*, and *US News and World Report*. These articles reflect one of the dilemmas facing the practicing orthodontist—whether to intervene before the eruption of the permanent dentition is complete.

One can argue that in many patients, it is best to allow for the eruption of all permanent teeth (except for third molars) before initiating orthodontic treatment. By having all teeth erupted fully, treatment often is provided in a relatively straightforward manner within a predictable period of time (12 to 30 months). When dealing with a postpubescent patient in whom most growth has terminated, the clinician usually does not have to contend with unwanted changes associated with aberrant growth patterns. In fact, in some types of malocclusions, as, for example, in a Class III malocclusion characterized by significant mandibular prognathism, definitive orthodontic and surgical treatment is best deferred until the end of the active growth period.

Although deferring treatment of orthodontic problems to the adolescent age period is viewed as an advantage by some clinicians, others view it as a significant disadvantage. Many clinicians seek to intervene in the mixed dentition so as to eliminate or modify skeletal, muscular, and dentoalveolar abnormalities before the eruption of the permanent dentition occurs. On the surface, this concept seems reasonable because it appears more logical to prevent an abnormality from occurring than to wait until it has become developed fully. Not all clinicians, however, use early treatment protocols. The decision concerning whether to intervene before the eruption of the permanent dentition can be viewed on the basis of a number of interactive factors.

Modification of Craniofacial Growth

The name of the *American Journal of Orthodontics* was changed by then editor-in-chief T.M. Graber to the *American Journal of Orthodontics and Dentofacial Orthopedics* in 1985. Ten years later, the orthodontic specialty changed its name from *Orthodontics* to *Orthodontics and Dentofacial Orthopedics*. Both of these changes in designation are a reflection of the importance now given to the orthopedic aspect of orthodontics. Despite this change in emphasis, however, the role of dentofacial orthopedics remains controversial.

During the past 45+ years, there has been much discussion among orthodontists and craniofacial biologists regarding the extent and location of therapeutically induced neuromuscular and skeletal adaptations throughout the craniofacial complex. Most would agree that the downward and forward growth of the maxillary complex of the growing individual can be influenced by such therapeutic techniques as extraoral traction and activator therapy. The ability to widen the transverse dimension of the maxilla through rapid maxillary expansion (RME) no longer is considered particularly controversial, although the long-term stability of this form of treatment only recently has been evaluated thoroughly.

The question of whether the mandible can be increased in length in comparison with untreated controls also has been addressed in numerous experimental and clinical studies (for a review of the literature in this area, see Chapter 15 in McNamara and Brudon[1]). Although controversial, the bulk of scientific evidence indicates that, in growing individuals, mandibular growth can be enhanced over the short term. A recent study of functional jaw orthopedics (FJO) by Freeman and coworkers[2] that considered the long-term effect of the function regulator (FR-2) of Fränkel showed that in late adolescence, the average increase in mandibular growth in the treatment group was 3 mm in comparison with matched untreated Class II subjects. Investigations of other functional appliance systems by our group have led to similar findings of 3 to 5 mm over the long term in comparison with untreated matched control participants.[3-6] Other long-term studies, however, have shown residual mandibular length increases of only 1 to 2 mm.[7,8]

In contrast, there is limited evidence that the growth of the mandible can be diminished substantially[9] either through the use of a chin cup or through orthopedic facial mask (FM) therapy, although a redirection of mandibular growth in a more vertical direction has been observed using a number of orthopedic techniques.[10]

Patient Cooperation

The ability to motivate a patient to comply is an essential ingredient of successful orthodontic therapy, whether initiated in the mixed or permanent dentition. One of the great fears of many orthodontists is that by beginning treatment in the mixed dentition, patient and parental cooperation will wane before fixed appliance therapy has been completed to the clinician's satisfaction. The goals and objectives of treatment must be established clearly in order to prevent unnecessary, prolonged treatment that may "burn out" the patient in the future.

In our opinion, the most significant problem regarding cooperation, particularly in a mixed dentition patient, is in the mind of the orthodontist or the parent rather than in that of the young patient. Every effort must be made to incorporate the patient and parents in treatment decisions and to stress the importance of appliance wear according to the specific needs of the patient. Indeed, motivating parents to provide the home support necessary for treatments that require strict compliance often is the greatest challenge to the clinician, as parents look for quick solutions to complex treatment challenges. Regimens requiring maximal patient cooperation should be used only after it has been determined that this type of appliance is the optimal approach for a given skeletal and neuromuscular imbalance. The treatment time should be estimated reasonably and should be known to the patient and parents at the beginning of the treatment period. Chapter 15 discusses this further.

A critical point in patient cooperation is related to the transition to high school, with the start of ninth grade often coinciding with a decrease in patient motivation. Thus, it is desirable to complete either phase II treatment or a comprehensive single-phase treatment before this time. It has been our experience that most patients who begin phase I orthodontic or orthopedic treatment in the second or third grade finish phase II before high school, although there is substantial individual variation. Scheduling of orthodontic appointments also is much easier during middle school or junior high school years than later.

Practice Management

It is obvious that when patients begin treatment in the mixed dentition, the time from the onset of treatment to the completion of the final fixed appliance phase will extend well beyond the duration of a typical orthodontic protocol initiated in the permanent dentition. When many of the currently used early

treatment protocols were being developed in the late 1970s and early 1980s, there were many instances of prolonged treatments that not only had a negative effect on patient and parental enthusiasm but also became a nightmare from a practice management perspective. Thus, more efficient and effective early treatment protocols have evolved. These protocols have a defined duration as well as a reasonably predictable outcome.

In general terms, an initial phase of treatment is provided that is approximately 1 year in duration followed by intermittent observation during the transition from the mixed to the permanent dentition. The naturally occurring increases in arch space are incorporated into the overall treatment plan by anchoring the permanent first molars in position as the second deciduous molars are lost, usually by placing a transpalatal arch (TPA) in the maxilla and, in about one-third of patients, a lingual arch in the mandible. After all of the permanent teeth have erupted fully into occlusion (except for, perhaps, the erupting second and developing third molars), fixed appliances then are used to align and fine detail the occlusion. Treatment typically is not begun until the eruption of the second molars has taken place or is anticipated within a 6- to 9-month period.

From a practice management perspective, separate charges are levied for the initial phase of early treatment and for the final comprehensive phase of treatment. In our practices, no charges are levied for appointments during the "interim" period when the eruption of the permanent dentition is monitored two or three times a year. These visits are important to monitor any space-holding appliances and are extremely valuable to ensure proper timing for full fixed appliance treatment, a point that must be repeated for parents by staff making recall appointments. We stress to patients and parents that we have two separate stages of "active" treatment so as to mitigate the perception of "being in braces forever."

THE CERVICAL VERTEBRAL MATURATION METHOD

Before we begin a detailed description of the protocols that can be used to treat various malocclusion types, a discussion of the skeletal maturational level of the patient is in order. In some instances, it is desirable to treat the patient at the time when the patient is growing rapidly, as during the circumpubertal growth period, when FJO has been shown to be particularly effective. In other circumstances, we would like to know if a patient has reached his or her growth potential, as in planning corrective jaw surgery or the placement of endosseous implants, so that substantial further growth is not anticipated or desired. We also would like to know if a patient is early in the growth process and would respond skeletally rather than dentally to forces placed against the circumaxillary sutural system (e.g., RME, Facial mask (FM) therapy).

One of the most inaccurate ways of determining a patient's maturational level is to use chronologic age as an indicator. It is well known that in a classroom of 12-year-old children, there may be as much as a 7-year spread developmentally among the children, with girls maturing faster than boys on average by at least 1 year. On the other hand, orthodontists in general have relied on the stage of dental eruption and development[11,12] as a starting point. This scheme of using tooth eruption as an indicator certainly is more accurate than using chronologic age; however, other biological indicators can be used as well.

Biological indicators of skeletal maturity refer mainly to somatic changes at puberty, thus emphasizing the known interactions between the development of craniofacial structures and the modifications in other body regions. Individual skeletal maturity can be assessed by means of several biological indicators, including increase in body height,[13,14] skeletal maturation of the hand and wrist,[15] menarche or voice changes,[16] and changes in the morphology of the cervical vertebrae.[17-19]

Of the skeletal indicators available routinely to the orthodontic practitioner that do not require the taking of additional records (e.g., hand–wrist film), the cervical vertebral maturation (CVM) method has proved clinically useful for us for more than 15 years. This method has been available since the early 1970s when it was developed by Don Lamparski as part of his master of science thesis at the University of Pittsburgh.[17] The CVM method remained relatively unused for the next 25 years, with few references made to it in the literature. The CVM method was reintroduced by our group first in 2000,[20] with an updated and simplified version presented in 2005.[21] The following is a brief summary of the most current CVM method.

There are six stages of cervical maturation in all, as shown diagrammatically in Figure 16-1. For the purpose of this evaluation using the lateral head film, only the bodies of the second, third, and fourth cervical vertebrae (C2, C3, and C4) are considered. Two morphologic characteristics are monitored, the first of which is the presence or absence of a notch or indentation on the inferior border of each of the three vertebral bodies. The second feature is the shape of the third and fourth cervical bodies, which change from trapezoid to rectangular horizontal to square to rectangular vertical (see Fig. 16-1).

The first three stages are differentiated from one another by the presence or absence of the notch. In the first cervical stage (CS-1), the inferior borders of vertebral bodies C2 to C4 are flat (or sometimes slightly convex; Fig. 16-2). From a practical standpoint, the notch must be at least 1 mm in depth at the center of the notching. The third and fourth cervical bodies are trapezoidal in morphology, assuming the shape of a typical wedge of cheese (Fig. 16-3), with the posterior border of the vertebral body taller than the anterior border and the superior surface sloping forward and downward. This stage occurs from approximately the time of the eruption of the deciduous dentition until about 2 years before the peak in skeletal growth. Our research[22] has shown that the ideal age to intervene with FM therapy combined with RME is at CS-1. Maximum skeletal adaptations occur in the midfacial region during this stage as the sutures are more open, in the

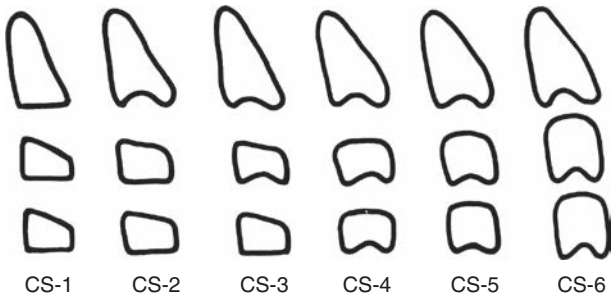


FIGURE 16-1 Schematic representation of the six stages of cervical vertebral maturation, according to the protocol described by Baccetti and coworkers. The second, third, and fourth cervical bodies are shown. Note the increase in notching and the changes in the shape of the third and fourth cervical bodies with maturation. (From Baccetti T, Franchi L, McNamara JA Jr. The cervical vertebral maturation (CVM) method for the assessment of optimal treatment timing in dentofacial orthopedics. *Semin Orthod* 2005;11:119–129.)

younger patient. Less skeletal and greater dentoalveolar adaptations are noted when RME combined with FM therapy is used during later stages (e.g., CS-3, CS-4).

The second cervical stage (CS-2) is characterized by a notch present along the inferior border of the second cervical vertebra (odontoid process). The lower borders of the third and fourth vertebral bodies remain flat (Fig. 16-4). Usually both C3 and C4 retain a trapezoidal shape, again the wedge of cheese appearance. CS-2 can be considered the "get-ready" stage because the peak interval of mandibular growth should begin within 1 year after this stage is evident.

FIGURE 16-2 The first CVM stage (CS-1). Note that the inferior borders of the three cervical bodies are not indented but are flat or slightly convex.

The third cervical stage (CS-3) is characterized by notching of the inferior borders of C2 and C3. C4 remains flat (Fig. 16-5). At least one of C3 and C4 bodies still retains a trapezoidal shape while the other can assume a more rectangular horizontal shape. At this stage, the maximum craniofacial growth velocity is anticipated. It must be remembered that the difference between stages is gradual, not abrupt, so that saying that someone is a "late CS-3" or an "early CS-4" is appropriate, depending on the transitional morphology of the third and fourth vertebrae.

In the fourth cervical stage (CS-4), all three bodies have notches along their inferior bodies, the more important factor being the shape of C3 and C4 (Fig. 16-6). At this stage, both vertebral bodies have a rectangular horizontal rather than a trapezoidal shape. It is easiest to remember this stage as the "bar of soap" stage because the bodies of both C3 and C4 assume this well-known shape (Fig. 16-7). During this stage, continued craniofacial growth can be anticipated but at a lesser rate than is seen at CS-3. Placing an endosseous implant to replace a missing maxillary lateral incisor would be inappropriate at this stage of maturation because of anticipated growth in the future.

The fifth cervical stage (CS-5) can be differentiated from CS-4 on the basis of the shapes of C3 and C4, with these bodies becoming square in shape (Fig. 16-8). All three cervical bodies have notches, so the presence of notching no longer is important in the differential diagnosis. We have found it easy to remember this stage as the "marshmallow" stage, in that the bodies now resemble the soft white puffy confection seen so commonly at summer campfires (Fig. 16-9). When this stage is reached, most substantial craniofacial growth has been achieved. The patient can be evaluated for corrective jaw surgery or the placement of endosseous implants in the aesthetic region. It should be noted that even though CVM staging is useful here, the gold standard for determining the continuation or cessation of significant

FIGURE 16-3 C-3 and C-4 usually have a trapezoidal shape and appear similar to a "wedge of cheese," as shown here.

FIGURE 16-4 The second CVM stage (CS-2). A notch is present in the inferior border of the odontoid process (C2). The vertebral bodies of C3 and C4 are in the shape of a wedge or trapezoid.

FIGURE 16-5 The third CVM stage (CS-3). Distinct notches are present on the inferior border of C2 and C3. At least one or both of the third and fourth cervical bodies still have a trapezoidal shape.

FIGURE 16-6 The fourth CVM stage (CS-4). Notches are present in all vertebrae. The bodies of C3 and C4 are rectangular and horizontal in shape.

FIGURE 16-7 The vertebral bodies of C3 and C4 at CS-4 resemble the shape of an ordinary bar of soap, as shown here.

FIGURE 16-8 The fifth CVM stage (CS-5). The bodies of C3 and C4 now are square in shape, with the posterior height the same as the width.

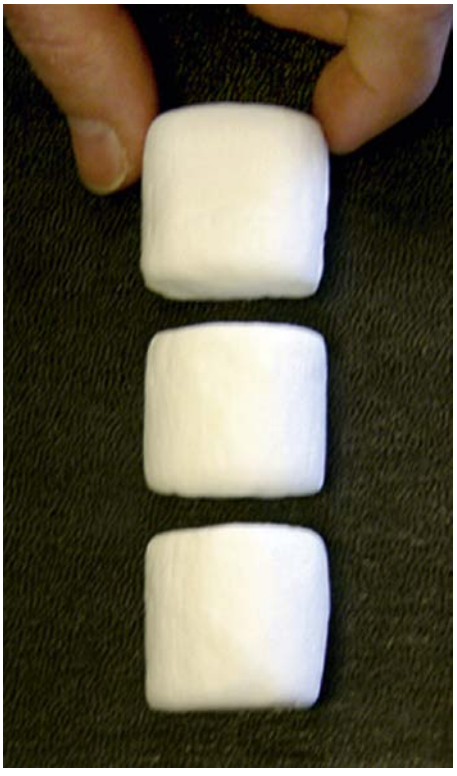


FIGURE 16-9 C3 and C4 at CS-5 resemble the shape of a stack of marshmallows, as imaged here.

FIGURE 16-10 The sixth CVM stage (CS-6). The bodies of C3 and C4 now are rectangular vertical in shape (i.e., greater posterior height than width).

craniofacial growth is the evaluation of two lateral head films taken at least 6 months apart.

It has been our experience that the most difficult stage to determine is the sixth cervical stage (CS-6). At CS-6, at least one of the third and fourth cervical bodies has assumed a rectangular vertical morphology (Fig. 16-10). In addition, the cortical bone appears better delineated in CS-6 than at CS-5.

Estimating patient maturational level by staging the second through fourth cervical vertebral bodies as seen in the lateral head film gives the clinician one additional piece of information that can be used to reach an appropriate diagnosis and treatment. CVM staging should be used in concert with a thorough evaluation of the hard and soft tissue during the treatment planning process as well as other maturational indicators and the family history. As with any subjective clinical evaluation, the precision of the CVM method improves with experience.

WHEN TO INTERVENE

The timing of orthodontic intervention is of critical importance, and the initiation of our treatment protocols varies according to the type of malocclusion being treated. For example, tooth-size/arch-size discrepancy problems typically are treated when the patient is 8 or 9 years of age. Normally, this treatment is initiated after the lower four incisors and the upper central incisors have erupted. In many instances, there is insufficient space to allow for the unimpeded eruption of the upper lateral incisors. Depending on the size of the permanent teeth,

space maintenance, serial extraction, orthopedic expansion, or a combination of these protocols is used.

In the instance of a Class III malocclusion, the onset of treatment usually is earlier than for a Class I patient. An optimal time for the beginning of an early Class III treatment regimen (e.g., orthopedic FM combined with a bonded acrylic splint expander to which have been attached FM hooks) is coincident with the loss of the upper deciduous incisors and the eruption of the upper permanent central incisors (the maxillary permanent first molars should be erupted as well). Reduction of a Class III pattern in which the patient is shifting into anterior crossbite and there is a family history of Class III may start in the primary dentition. This earlier intervention in Class III patients obviously will result in a longer period of time between the start of the initial phase of treatment and the end of the fixed appliance phase after the permanent dentition has erupted.

The timing of treatment of Class II malocclusions differs substantially from that described previously for Class I and Class III malocclusions. In contrast to our positive recommendations concerning early intervention in Class III malocclusions and in many tooth-size/arch-size discrepancy problems, we typically recommend a delay until the circumpubertal growth period (i.e., cervical stage CS-3) before using FJO in patients with Class II malocclusions characterized in part by mandibular skeletal retrusion. Both clinical and experimental studies have shown that there is a greater mandibular growth response with functional appliances when treatment is initiated during the circumpubertal growth period.[23-26] Ideally, functional appliance therapy (e.g., Herbst, twin block, mandibular anterior repositioning appliance [MARA], bionator, FR-2 of Fränkel) will be followed directly by a phase of fixed appliance therapy to align the permanent dentition. In reality, besides the routine use of Class II intermaxillary elastics, we use two Class II correction appliances primarily, both in the early permanent dentition. These two appliances are the stainless steel crown Herbst appliance and the Pendex appliance. Still used are the MARA and modifications of the Pendex, including temporary anchorage device (TAD) secured appliances for patients with maxillary dental protrusion.

In patients who present with severe neuromuscular and skeletal problems that lead to what we have termed "socially debilitating" malocclusions, the initiation of treatment earlier in the mixed dentition sometimes is indicated. From a physiologic standpoint, it may be better to delay treatment until the circumpubertal growth period so that a maximum response to FJO can be achieved. However, earlier intervention may be necessitated because of psychological issues related to the underlying severity of the malocclusion. Fortunately, such "socially debilitating" malocclusions are not observed frequently.

In Class II patients who present with maxillary prognathism, the timing of treatment does not appear to be crucial. Extraoral traction can be used in either the mixed or permanent dentition to treat this type of skeletal imbalance satisfactorily.[27,28]

It also should be noted that in many patients with Class II malocclusions identified in the 7 to 9-year-old age range, treatment is initiated at that time to handle intraarch problems (e.g., crowding, spacing, flaring); interarch discrepancies are addressed later. In other words, the same protocols (e.g., orthopedic expansion, extractions) that can be used for Class I patients may be initiated in Class II patients with arch length discrepancies. The attempt to correct mandibular deficiency, however, is best delayed until near the circumpubertal growth period in patients with mild to moderate Class II sagittal problems.

TREATMENT OF TOOTH-SIZE/ARCH-SIZE DISCREPANCY PROBLEMS

The most common type of malocclusion noted in the mixed dentition usually is described as *crowding*. These patients are referred by the family dentist or by the patient's parents because of obvious dentoalveolar protrusion or lack of sufficient space for permanent tooth eruption. Most commonly, this type of patient presents with a Class I molar relationship or a tendency toward either a Class II or Class III malocclusion.

In the permanent dentition, discrepancies between tooth size and arch size usually are handled by one of three treatment modalities: extraction, interproximal reduction, or arch expansion. Comparable treatment protocols in the mixed dentition are serial extraction and orthopedic expansion, with interproximal reduction usually being reserved for permanent dentition patients only. Additional methods of treating discrepancy problems in the mixed dentition that are not available for use in permanent dentition patients include techniques of space management (e.g., maintenance of leeway space).

Space Maintenance during the Transition of the Dentition

An integral part of any mixed dentition protocol is monitoring the transition from the mixed to the permanent dentition. Significant differences exist between the sizes of the second deciduous molars and the succeeding second premolars.[29] On average, 2.5 mm per side of arch space can be gained in the mandibular arch, and about 2 mm per side can be gained in the maxillary arch. There is wide variation in tooth size among patients, however, and thus each patient must be evaluated with radiographs to determine the relative size of the second deciduous molars and their successors. Simply maintaining available arch space during the transition of the dentition may be sufficient to resolve minor to moderate tooth-size/arch-size discrepancies,[30] particularly if judicious interproximal reduction is used after the permanent dentition has erupted.

Two types of arches are used as holding appliances in the late transition of the dentition: the transpalatal arch (TPA) and the mandibular lingual arch. These arches routinely are cemented in place before the loss of the second deciduous molars.

Transpalatal Arch

The TPA,[1] as the name implies, extends from one maxillary first molar along the contour of the palate to the molar on the opposite side (Fig. 16-11). Although both fixed and removable types of TPAs are available, we routinely use the soldered TPA that is made from 0.036-inch stainless steel wire and that is soldered to the molar bands at their mesiolingual line angles and has an omega-shaped adjustment loop at the midpalatal height of contour.

The major function of the TPA in the mixed dentition is to prevent the mesial migration and mesial rotation of the maxillary first molars during the transition from the second deciduous molars to the second premolars. If desired, this appliance also is capable of producing desired molar rotations and changes in root torque[1] by sequential unilateral activation of the appliance. The TPA also can be used for stabilization of molar position. The TPA is often left in place until the final comprehensive phase of orthodontic therapy is completed. Interestingly, the TPA does not function well as an anchorage appliance in extraction cases.[31]

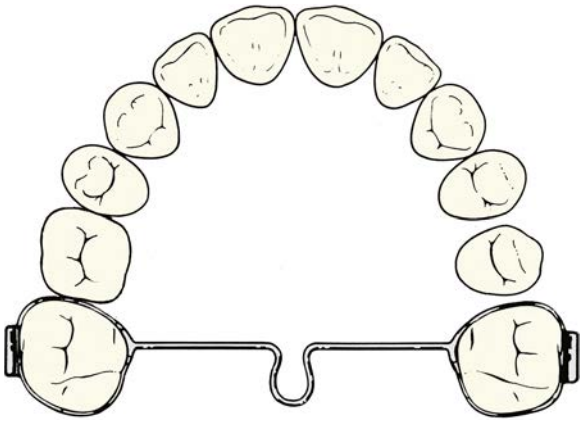


FIGURE 16-11 Transpalatal arch. This arch can be used as both an active appliance and a stabilization appliance during the transition of the dentition. Note the potential net increase in available space after the transition from the second deciduous molar to the second premolar. (Adapted from McNamara JA Jr, Brudon WL. *Orthodontics and Dentofacial Orthopedics*. Ann Arbor, MI: Needham Press; 2001.)

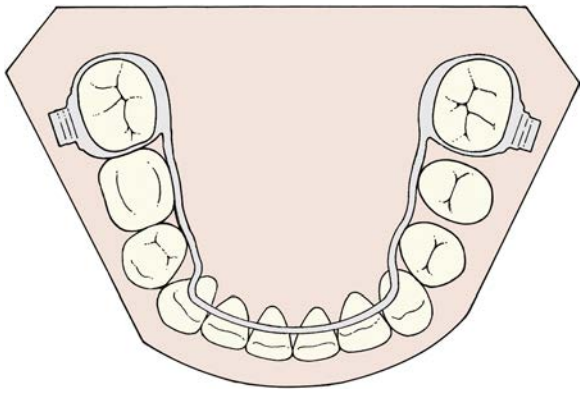


FIGURE 16-12 Lower lingual arch. Note the maintenance of arch space after the loss of the second deciduous molar and the eruption of the second premolar on the left side. Adjustment loops can be placed in the arch in the second premolar region, if desired. (Adapted from McNamara JA Jr, Brudon WL. *Orthodontics and dentofacial orthopedics*. Ann Arbor, MI: Needham Press; 2001.)

Lingual Arch

The lingual arch, usually used in the mandible as part of our early treatment protocol, has a similar function to the TPA in the maxilla, which is as a molar anchorage appliance. The lingual arch, also made of 0.036-inch stainless steel, extends along the lingual contour of the mandibular dentition from the first molar on one side to the first molar on the other (Fig. 16-12). Optional adjustment loops (not shown) can be placed in the lingual arch in the region of the second deciduous molars, providing added ability to adjust arch vertical and horizontal position.

The lower lingual arch is used less frequently (30% of our early treatment patients) than is the TPA (90+% of patients) because many patients undergo early orthodontic treatment who do not require the maintenance of arch space in the lower second premolar region. Thus, the lower lingual arch is indicated only in patients in whom maximum molar anchorage is to be maintained. In contrast to the TPA, the lingual arch usually is removed after the eruption of the second premolars is completed and the proper positioning of these teeth has been achieved.

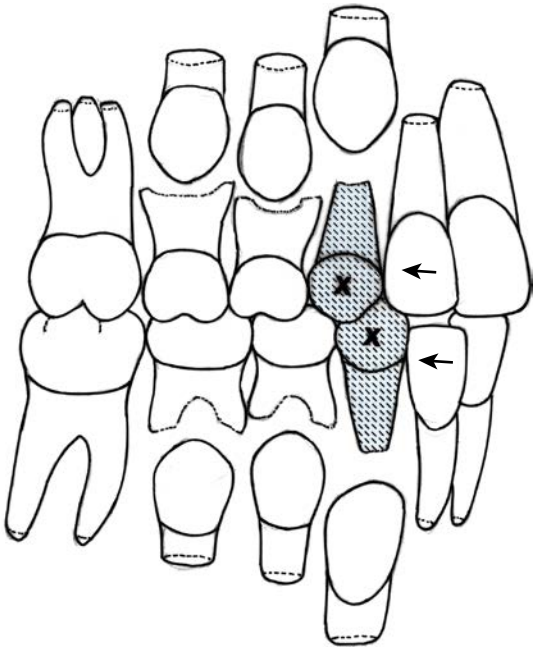


FIGURE 16-13 Serial extraction protocol. The removal of the upper and lower deciduous canines (x) allows for an improvement in the alignment of the upper and lower incisors.

Not only is the lingual arch used in Class I individuals in whom arch space is to be stabilized but also in the treatment of patients with Class III malocclusions. In this instance, molar position is maintained to prevent the forward movement of the molars (thus aggravating the Class III molar relationship) and facilitating the more posterior eruption and potential distal movement of the mandibular premolars.

SERIAL EXTRACTION

Another protocol that is used less frequently in the management of tooth-size/arch-size discrepancies is *serial extraction*. This treatment technique involves the sequential removal of deciduous teeth to facilitate the unimpeded eruption of the permanent teeth. Such a procedure often, but not always, results in the extraction of four premolar teeth. The sequence of serial extraction has been clarified in a series of articles by Dewel[32,33] as well as in several book chapters (See Chapter 34).[34,35]

The typical serial extraction protocol is initiated about the time of the appearance of the permanent lateral incisors, which erupt in rotated positions or initially are prevented from eruption by the deciduous canines. In the most commonly used protocol, the first teeth to be removed are the deciduous canines (Fig. 16-13). The removal of these teeth allows for the eruption, posterior movement, and spontaneous improvement in the alignment of the permanent lateral incisors.

In about 6 to 12 months, the removal of the four deciduous first molars is undertaken (Fig. 16-14). Ideally, the root development of the four first premolars is ahead of that of the permanent canines, so that the first premolars will erupt before the canines. At this stage, if the canines are erupting close to the same time as the first premolars, some clinicians prefer to extract the first premolars at the same time that the

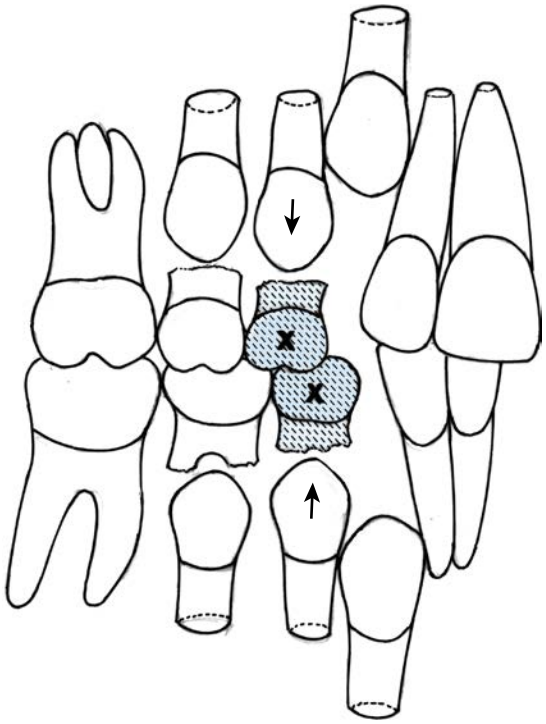


FIGURE 16-14 Serial extraction protocol (continued). The removal of the deciduous first molars encourages the eruption of the first premolars. Some clinicians choose to remove the first premolars at the same time to allow the lower canines to migrate posteriorly before emergence. (From McNamara JA Jr, Brudon WL. *Orthodontics and dentofacial orthopedics*. Ann Arbor, MI: Needham Press; 2001.)

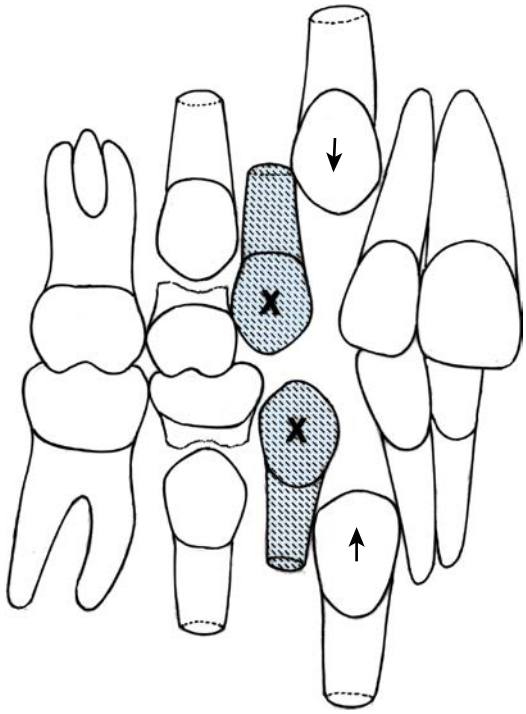


FIGURE 16-15 Serial extraction protocol (continued). The removal of the first premolars encourages the eruption and posterior movement of the permanent canines. (From McNamara JA Jr, Brudon WL. *Orthodontics and dentofacial orthopedics*. Ann Arbor, MI: Needham Press; 2001.)

first deciduous molars are removed, under the assumption that changes that are even more favorable are obtained when the first premolars are removed before they emerge. Before emergence, the permanent canines can move within the jaws toward the space where the first premolar crowns were located.

The next step in the usual protocol is the extraction of the first premolars (Fig. 16-15) after these teeth have been allowed to erupt. It is common to observe that the adjacent canine and potentially second premolars erupt toward the extraction sites, as is shown in Figure 16-16. The lower incisors often upright as well, sometimes too much so. As soon as the second molars near emergence, fixed appliances are used to align and detail the dentition and occlusion (Fig. 16-17).

Graber[34] states that serial extraction may be indicated when it is determined "with a fair degree of certainty that there will not be enough space in the jaws to accommodate all the permanent teeth in their proper alignment." Whereas Proffit and coworkers[36] cite a predicted tooth-size/arch-size discrepancy of 10 mm or greater as an indication for serial extraction, Ringenberg[37] mentions a discrepancy of 7 mm or more. In many serial extraction patients, a TPA is used to maintain maxillary molar position as the transition to the permanent dentition occurs.

From our perspective, the primary factor to be evaluated when making a treatment decision concerning serial extraction is large tooth size. In instances in which tooth sizes are abnormally large (e.g., maxillary central incisor width >10.0 mm),[29] the initiation of serial extraction protocols may be appropriate. Another factor that must be considered is the anteroposterior positioning of the lower incisors relative to the adjacent

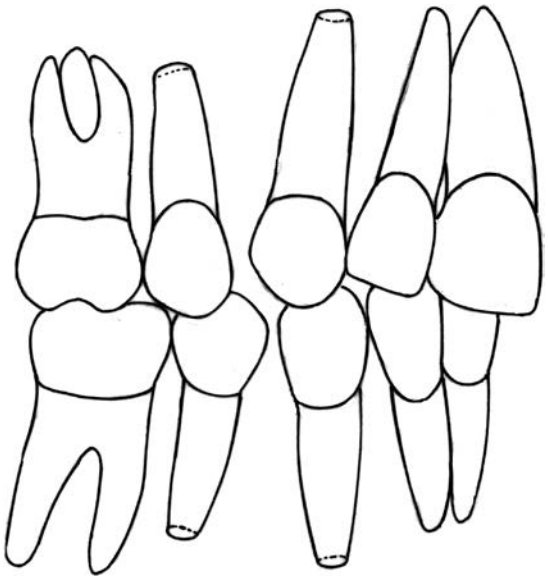


FIGURE 16-16 Serial extraction protocol (continued). The remaining teeth tend to tip toward the extraction sites. The lower incisors often tip lingually as well. (From McNamara JA Jr, Brudon WL. *Orthodontics and dentofacial orthopedics*. Ann Arbor, MI: Needham Press; 2001.)

skeletal elements as well as to the soft tissue, especially the lip musculature.

Serial extraction patients with tooth crowding with extreme bialveolar retrusion and flat facial profiles obviously are not

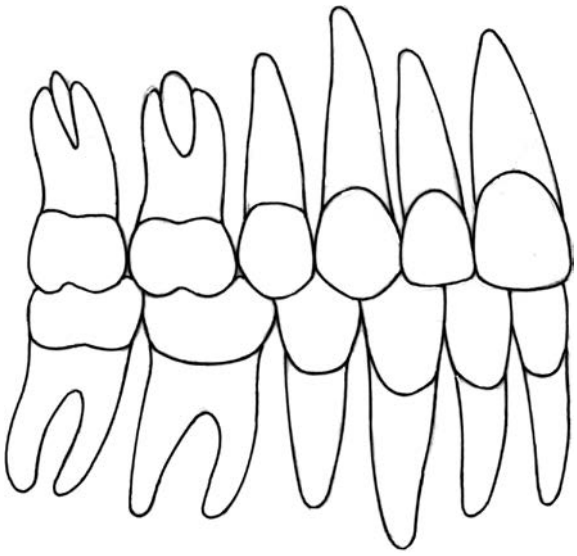


FIGURE 16-17 Serial extraction protocol (continued). After the lower second premolars near emergence, fixed appliances are used to align the teeth and level the occlusal plane. (From McNamara JA Jr, Brudon WL. *Orthodontics and dentofacial orthopedics*. Ann Arbor, MI: Needham Press; 2001.)

recommended because of potential unfavorable facial contour changes. In fact, mild residual crowding of the lower incisors is more preferable to creating a "dished-in" facial appearance. Similarly, a serial extraction protocol in patients with bialveolar protrusion also is not indicated because maximum retraction of the incisors is desirable and is not usually attainable with serial extraction. Maximum anchorage mechanics using fixed appliance therapy potentially combined with extraoral traction[38] or temporary anchorage devices[39] are treatments of choice.

Serial extraction may be combined with RME in certain patients with significant arch-length discrepancy problems who also present with a narrow tapered maxilla and negative space in the corners of the mouth during smiling. The use of RME is particularly appropriate in patients with broad facial contours. The arches can be expanded first to broaden the smile, and then after reevaluation, serial extraction procedures may be initiated subsequently to reduce or eliminate emerging tooth–arch imbalances.

It is well known that serial extraction is not a panacea in all patients who present with dental crowding in the mixed dentition.[37] Great care must be taken to avoid lingual tipping of the lower incisors as well as unfavorable changes in the sagittal position of the upper and lower dentitions. In addition, the initiation of serial extraction procedures may result in unwanted spacing in the dental arches. When used appropriately as based on a solid diagnosis, however, a protocol of sequentially extracting the deciduous dentition has proven to be an efficient, cost-effective, and satisfactory treatment for tooth-size/arch-size discrepancy problems (see Chapter 34 for in-depth discussion).

ARCH EXPANSION

Types of Expansion

It is well known that expansion of the dental arches can be produced by a variety of orthodontic treatments, including those that incorporate fixed appliances. The types of expansions produced can be divided arbitrarily into three categories.

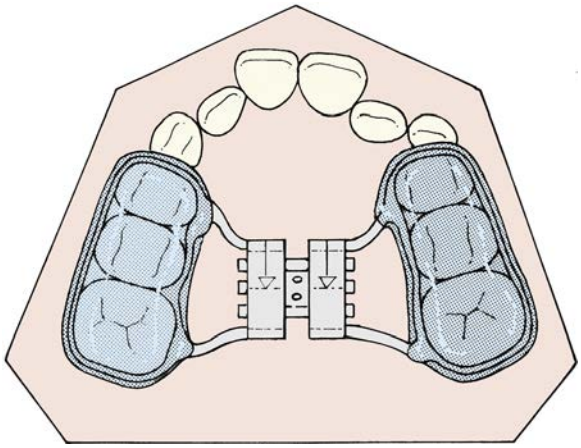


FIGURE 16-18 An acrylic splint RME appliance that is bonded to the maxillary primary molars and the permanent first molars. The occlusal coverage of acrylic produces a posterior bite block effect on the vertical dimension. (Adapted from McNamara JA Jr, Brudon WL. *Orthodontics and dentofacial orthopedics*. Ann Arbor, MI: Needham Press; 2001.)

Orthodontic Expansion

Orthodontic expansion, produced by conventional fixed appliances as well as by various removable expansion plate and finger spring appliances, usually results in lateral movements of the buccal segments that primarily are dentoalveolar in nature. There is a tendency toward a lateral tipping of the crowns of the involved teeth and a resultant relative lingual tipping of the roots. The resistance of the cheek musculature and other soft tissue remains, providing forces that may lead to a relapse or rebound of the achieved orthodontic expansion.[40,41]

Passive Expansion

When the forces of the buccal and labial musculature are shielded from the occlusion, as with the FR-2 appliance of Fränkel,[42] a widening of the dental arches often occurs. This *passive* expansion is not a result of the application of extrinsic biomechanical forces but rather by intrinsic forces such as those produced by the tongue. Brieden and coworkers,[43] in an implant study conducted in patients treated with the FR-2 appliance of Fränkel, have demonstrated that bone deposition occurs primarily along the lateral aspect of the alveolus rather than at the midpalatal suture. A related type of spontaneous arch expansion also has been observed after lip-bumper therapy.[44]

Orthopedic Expansion

Rapid maxillary expansion appliances (Fig. 16-18) are the best examples of true *orthopedic* expansion, in that changes are produced primarily in the underlying skeletal structures rather than by the movement of teeth through alveolar bone.[45-47] RME not only separates the midpalatal suture but also affects the circumzygomatic and circumaxillary sutural systems.[48] After the palate has been widened, new bone is deposited in the area of expansion so that the integrity of the midpalatal suture usually is reestablished within 3 to 6 months.

Rationale for Early Orthopedic Expansion

The cornerstone of the early orthopedic expansion protocol used in the treatment of patients with arch-length discrepancy problems is the actual RME itself. The use of RME is based in

part on our previous studies of the development of the dental arches in untreated individuals, both in the permanent dentition and the mixed dentition.[1,29,49]

Permanent Dentition

When arches are crowded, what is the cause? Howe and coworkers[49] carried out an investigation in which the dental casts of patients with severe crowding were compared with the dental casts of untreated individuals who were classified as having ideal or near-ideal occlusions. No statistically significant differences in tooth size were noted between the uncrowded and crowded populations, regardless of whether aggregate tooth size or the sizes of individual teeth were considered. In contrast, there were statistically significant differences in arch width and arch perimeter.

Maxillary intermolar width was of particular importance as an easily measured clinical indicator. In noncrowded male patients, the average distance between the upper first permanent molars, measured at the point of the intersection of the lingual groove at the gingival margin, was about 37 mm, a value that can be compared with a similar measure in the crowded males of 31 mm. Similar but slightly smaller measures and differences were noted in the female sample.[49] Howe and coworkers concluded that a transpalatal width of 35 to 39 mm suggests a bony base of adequate size to accommodate a permanent dentition of average size (of course, a larger aggregate tooth size requires a larger bony base and vice versa).

Mixed Dentition

Because Howe and coworkers'[49] study was conducted using data from individuals in the permanent dentition, it did not address the issue of the normal development of the dental arches. This question was considered in a second study[50] that examined the nature of normal changes in maxillary and mandibular transpalatal width from the early mixed dentition to the permanent dentition. Longitudinal changes in an untreated population from 7 to 15 years of age were evaluated. The average increase in transpalatal width between the upper first molars was about 2.5 mm.[49,50]

One of the conclusions that can be drawn from the studies cited earlier concerning dental arch development is that by providing some mechanism of widening the bony bases and increasing arch width and perimeter, more space can be obtained for the alignment of the permanent dentition. Of course, the dental arches cannot be expanded *ad libitum* because of the physiologic limits of the associated hard and soft tissues. It seems reasonable, however, to consider increasing arch size at a young age so that skeletal, dentoalveolar, and muscular adaptations can occur before the eruption of the permanent dentition.

Orthopedic Expansion Protocols

Our appliance of choice for use in patients with mixed dentition is the bonded acrylic splint expander (see Fig. 16-18). This appliance, which incorporates a Hyrax-type screw into a framework made of wire and acrylic, is used to separate the halves of the maxilla. It is widely recognized that maxillary expansion is achieved easily in a growing individual, particularly in individuals with mixed dentition.[51,52] The acrylic-splint type of appliance that is made from 3-mm-thick, heat-formed Biocryl has the additional advantage of acting as a bite block because of the thickness of the acrylic that covers the occlusal surfaces of the posterior dentition. The posterior bite block effect of the bonded acrylic splint expander prevents the extrusion of the posterior teeth,[53] a movement often associated with banded RME appliances,[54] thus permitting the use of this type of expander in some patients with steep mandibular planes. It also unlocks the occlusion, immediately aiding resolution of a functional jaw shift into crossbite.

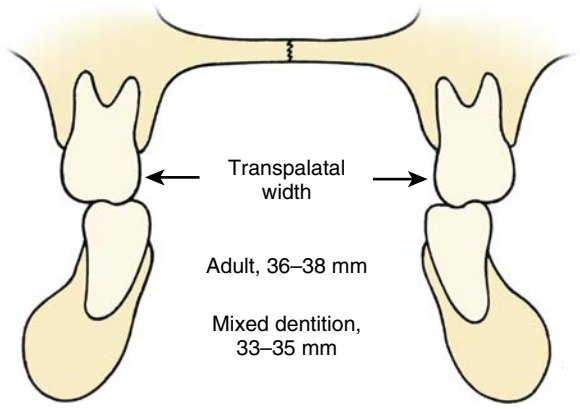


FIGURE 16-19 Frontal cross-sectional view of transpalatal dimensions through the molar region. Ideal transpalatal widths of the adult patient and the mixed dentition patient are shown. (Adapted from McNamara JA Jr, Brudon WL. *Orthodontics and dentofacial orthopedics*. Ann Arbor, MI: Needham Press; 2001.)

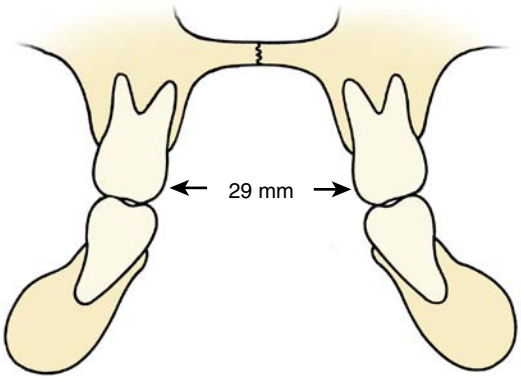


FIGURE 16-20 Frontal cross-sectional view of a patient with a constricted maxilla, as indicated by the intermolar width of 29 mm. (Adapted from McNamara JA Jr, Brudon WL. *Orthodontics and dentofacial orthopedics*. Ann Arbor, MI: Needham Press; 2001.)

Maxillary Adaptations

The treatment protocol that involves the use of a bonded expander is illustrated by the following example. The morphology of a patient in the mixed dentition with an idealized (e.g., 34 to 35 mm) transpalatal width (Fig. 16-19) can be compared with a patient with a narrow (e.g., 29 mm) transpalatal width (Fig. 16-20). A goal of the orthopedic treatment initiated in the mixed dentition is to reduce the need for extractions in the permanent dentition through the elimination of arch-length discrepancies as well as the elimination of bony base imbalances. In instances of restricted transverse dimensions, a bonded RME appliance is placed. The screw of the expander is activated one-quarter turn (90 degrees, 0.20 to 0.25 mm) per day until the lingual cusps of the upper posterior teeth approximate the

buccal cusps of the lower posterior teeth (Fig. 16-21). In contrast to Haas,[47] who recommends full opening of the expansion screw to 10.5 to 11.0 mm (an action that can produce a buccal crossbite), we advocate only as much expansion as is feasible while still maintaining contact between the upper and lower posterior teeth.

After the active phase of expansion is completed, the appliance is left in place for an additional 5 months to allow for a reorganization of the midpalatal suture as well as other sutural systems affected by the expansion and to maximize the effect of the posterior bite block. At the end of the treatment time, the RME appliance is removed, and the patient is given a removable palatal plate to sustain the achieved result (Fig. 16-22).

The active expansion of the two halves of the maxilla produces a midline diastema between the two upper central incisors. During the period after the active expansion of the appliance, a mesial tipping of the maxillary central and lateral incisors usually is observed. Such spontaneous tooth movement is typical after RME, and this movement often is interpreted as being evidence of "relapse" by the patient or the parents. The clinician should advise the family about the probability of such spontaneous tooth migration. At 3 or 4 months after the initiation of RME treatment, brackets often are placed on the upper incisors to close the midline diastema and align the anterior teeth (Fig. 16-23). In limited instances, a utility arch is used to retract, intrude, or protract the upper incisors, depending on the needs of the individual patient.

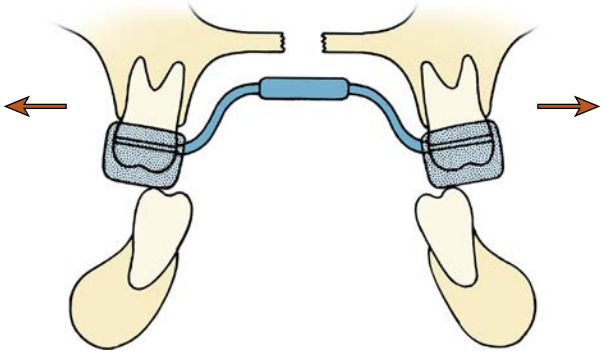


FIGURE 16-21 The effect of the bonded acrylic splint rapid maxillary expansion appliance. Note that the lingual cusps of the upper posterior teeth approximate the buccal cusps of the lower posterior teeth. (Adapted from McNamara JA Jr, Brudon WL. *Orthodontics and dentofacial orthopedics*. Ann Arbor, MI: Needham Press; 2001.)

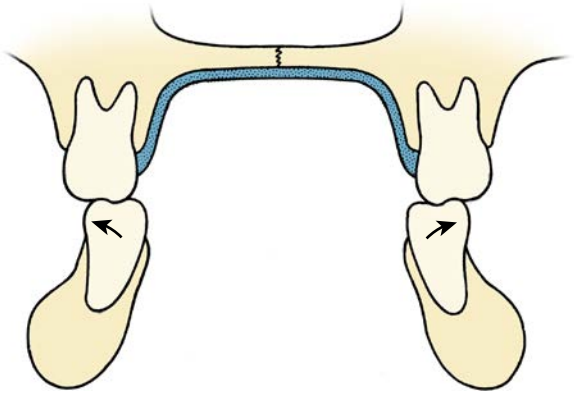


FIGURE 16-22 The same patient during the post–rapid maxillary expansion period. A removable palatal plate has been added to stabilize the intraarch relationship. Note the slight spontaneous uprighting of the posterior mandibular dentition. (Adapted from McNamara JA Jr, Brudon WL. *Orthodontics and dentofacial orthopedics*. Ann Arbor, MI: Needham Press; 2001.)

Mandibular Adaptations

In patients whose lower arch exhibits moderate crowding of the anterior teeth or in whom the posterior teeth are tipped lingually, two types of appliances can be used before RME: the removable Schwarz appliance and the lip bumper. The use of these "decompensating" (i.e., expanding, uprighting) appliances began as a result of our initial experiences using the bonded RME appliances alone. We were able to produce the expected changes in maxillary transverse dimensions with the bonded expander readily, but we made no attempt to widen the lower dental arch actively. After evaluating RME in mixed dentition patients over a 5-year period, we discovered that in some patients, a spontaneous uprighting and "decrowding" of the lower teeth occurred, yet in others, there was no change in the position and alignment of the lower teeth.

Because one of the cardinal rules of orthodontics was that one never should expand the lower arch, we were reluctant to do so. However, because expansion or uprighting was observed in the lower arch on a sporadic basis using RME and because arch expansion was produced routinely by the FR-2 appliance of Fränkel,[42,55-57] we decided to attempt orthodontic expansion or uprighting of the lower dental arch using either the removable Schwarz appliance or the lip bumper before orthopedic expansion of the maxilla. We assumed that expansion of the lower arch would not be stable unless the expansion was followed by maxillary orthopedic expansion.

Mandibular Dental Uprighting and Expansion Appliances

The Schwarz Appliance

The Schwarz appliance is a horseshoe-shaped removable appliance that fits along the lingual border of the mandibular dentition (Fig. 16-24). The inferior border of the appliance extends below the gingival margin and contacts the lingual gingival tissue. A midline expansion screw is incorporated into the acrylic, and ball clasps lie in the interproximal spaces between the deciduous and permanent molars.

The lower Schwarz appliance is indicated in patients with mild to moderate crowding in the lower anterior region and

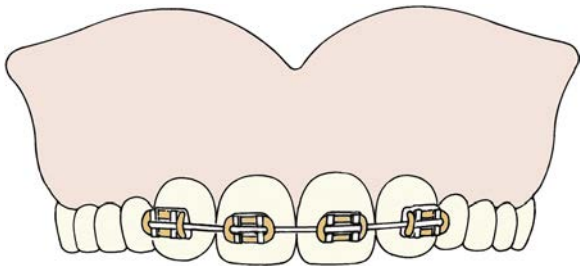


FIGURE 16-23 The placement of brackets on the upper anterior teeth to achieve incisal alignment with mesial movement of incisors providing added space for the permanent canines. (Adapted from McNamara JA Jr, Brudon WL. *Orthodontics and dentofacial orthopedics*. Ann Arbor, MI: Needham Press; 2001.)

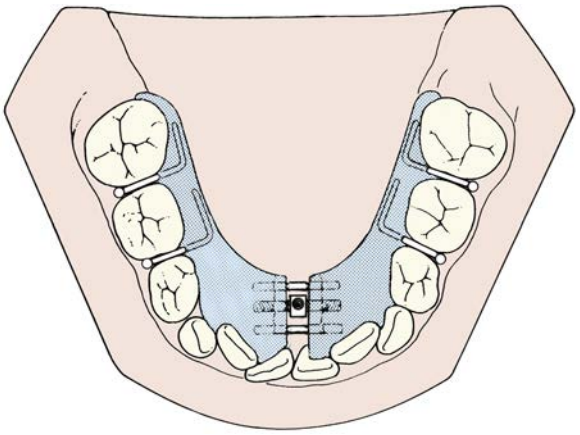


FIGURE 16-24 The removable lower Schwarz appliance that is used for mandibular dental decompensation. This appliance produces an orthodontic tipping (uprighting) of the lower posterior teeth and may create additional arch space anteriorly. (Adapted from McNamara JA Jr, Brudon WL. *Orthodontics and dentofacial orthopedics*. Ann Arbor, MI: Needham Press; 2001.)

especially in instances in which there is significant lingual tipping of the posterior dentition. The appliance is activated once per week, producing 0.20 to 0.25 mm of expansion in the midline of the appliance. Usually the appliance is expanded for 4 to 5 months, depending on the degree of incisal crowding, producing about 4 to 5 mm of arch length anteriorly.

Clinicians frequently have experienced difficulty understanding the reasoning underlying the use of the Schwarz appliance before RME. The following example illustrates the logic for this treatment decision. Figure 16-25, *A*, is a schematic of a bilateral posterior crossbite, a condition that clinically is recognized easily and for which RME is a generally accepted treatment regimen. In this example, the mandibular bony base and dental arch are of normal width, and there is normal posterior dental angulation, whereas the maxilla is constricted.

The example shown in Figure 16-25, *B*, is from a patient who has maxillary constriction but in whom also there has been mandibular dentoalveolar "compensation" (i.e., the positions of the lower teeth have been influenced by the size and shape of the narrow maxilla). No obvious crossbite is present. Even

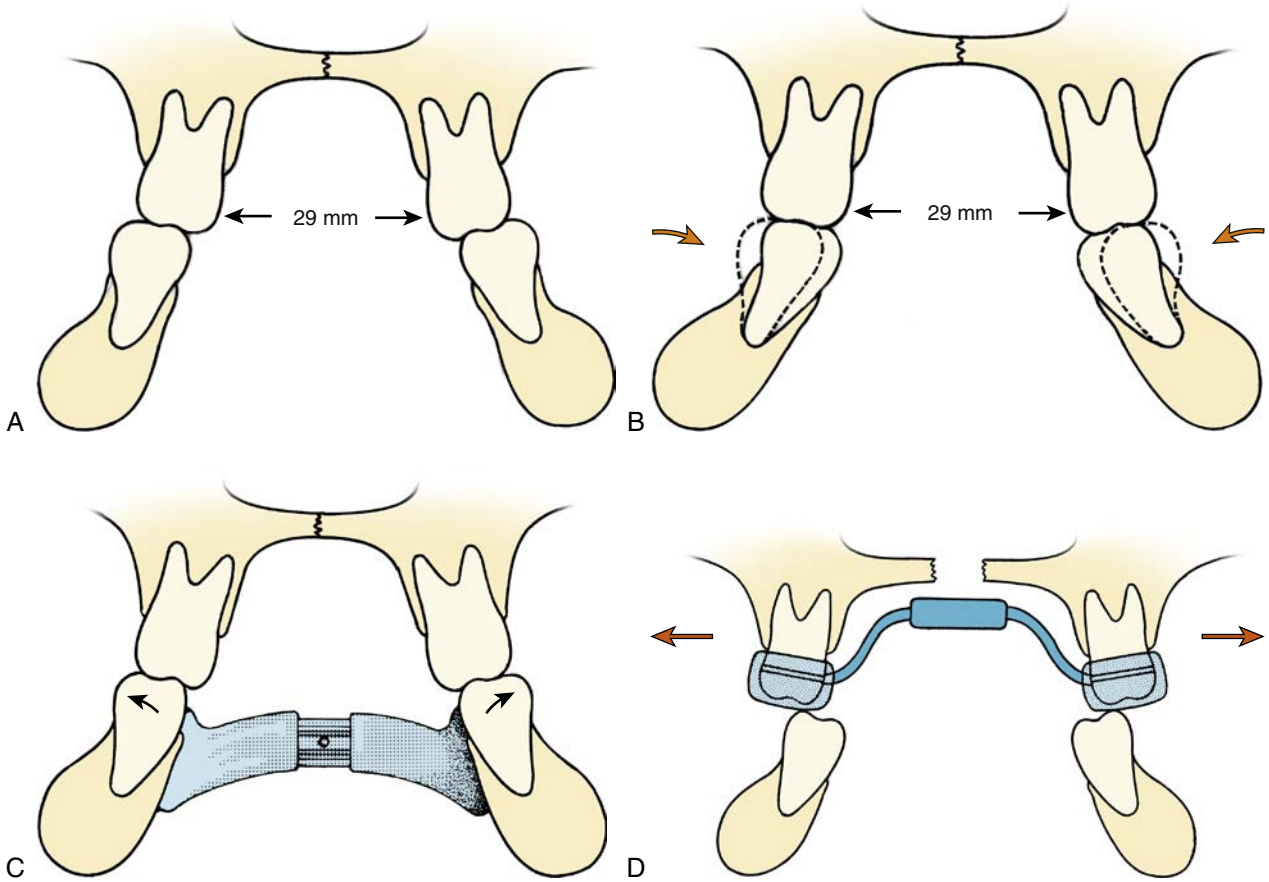


FIGURE 16-25 Frontal cross-sectional views. **A,** Patient with a constricted maxilla, properly uprighted lower posterior dentition, and a bilateral crossbite. **B,** Patient with a similar transpalatal width and with the body of the mandible in the same position as in A. Note the lower posterior teeth are more lingually inclined, camouflaging the maxillary constriction. The uprighting of the lower posterior teeth (i.e., mandibular dental "decompensation") is indicated before rapid maxillary expansion (RME). **C,** The removable lower Schwarz expansion appliance uprights the lower molars, orthodontically producing a tendency toward a posterior crossbite. **D,** RME after mandibular dental decompensation. The upper lingual cusps approximate the lower buccal cusps at the end of expansion. (Adapted from McNamara JA Jr, Brudon WL. *Orthodontics and dentofacial orthopedics*. Ann Arbor, MI: Needham Press; 2001.)

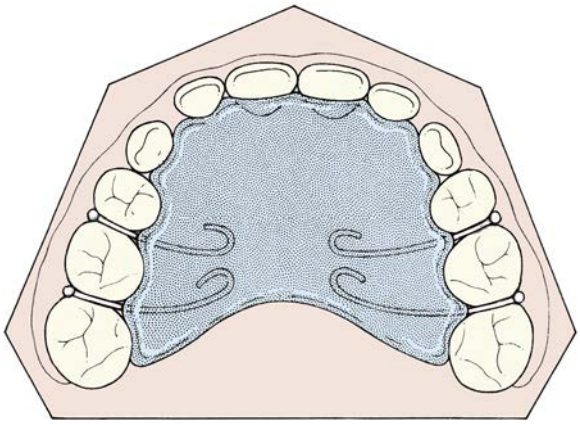


FIGURE 16-26 Maxillary stabilization plate. This appliance usually is worn on a near full-time basis for at least 1 year after rapid maxillary expansion removal. The acrylic of the plate also may serve as a guide plane for erupting canines and premolars. A labial bow, clasps, or both may be added. (Adapted from McNamara JA Jr, Brudon WL. *Orthodontics and dentofacial orthopedics*. Ann Arbor, MI: Needham Press; 2001.)

though maxillary width is the same as in Figure 16-25, *A*, the lower posterior teeth have erupted in a more lingual inclination. The palate appears narrow (in this example, a transpalatal width of 29 mm), and the arches are tapered in form. Mild to moderate lower incisor crowding also is present (not shown). In such a patient, mandibular dental "decompensation" using a removable lower Schwarz appliance often is undertaken. The width and form of the mandibular dental arch are made more ideal before the time that RME is attempted. By decompensating the mandibular dental arch, greater arch expansion of the maxilla can be achieved than when RME is used alone.[58,59]

Simply stated, the purpose of the Schwarz appliance is to produce *orthodontic tipping* of the lower posterior teeth, uprighting these teeth into a more normal inclination (Fig. 16-25, *C*). This movement is unstable if no further treatment is provided to the patient. A tendency toward a posterior crossbite is produced that is similar in many respects to the posterior crossbite shown in Figure 16-25, *A*.

Usually the Schwarz appliance is left in place until the maxillary orthopedic expansion phase is completed (Fig. 16-25, *D*). As described earlier, the maxilla is expanded using a bonded acrylic splint appliance until the upper lingual cusps barely touch the lower buccal cusps. After a 5-month period of RME stabilization, which allows adequate time for the midpalatal suture and the adjacent sutural systems to reorganize and reossify, both appliances are removed, and the patient is given a simple maxillary maintenance plate (Fig. 16-26), with no retention provided in the mandible. In instances of severe anterior malalignment in either arch, fixed appliances may be placed on the incisors to align these teeth, and interim lingual arch retention may be used.

Lip Bumper

The lip bumper (Fig. 16-27) is a removable appliance that also can be used for mandibular dental decompensation.[44,60] The lip bumper is particularly useful in patients who have very tight or tense buccal and labial musculature. The lip bumper lies away from the dentition at the gingival margin of the lower central incisors and shields the teeth from the forces of the adjacent

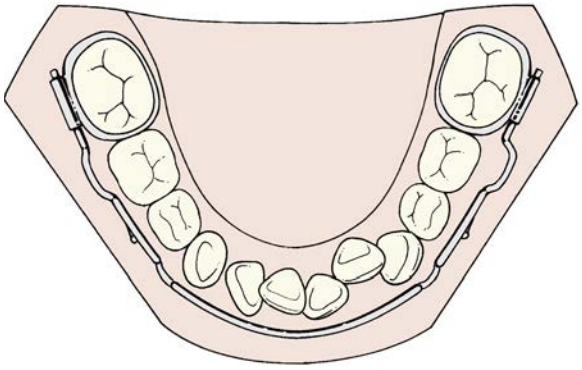


FIGURE 16-27 Occlusal view of a mandibular lip bumper that inserts into buccal tubes on the lower first permanent molar bands. (Adapted from McNamara JA Jr, Brudon WL. *Orthodontics and dentofacial orthopedics*. Ann Arbor, MI: Needham Press; 2001.)

soft tissue. The appliance usually is worn on a full-time basis and may be ligated in place. This appliance not only increases arch length through passive lateral and anterior expansion but also serves to upright the lower molars distally, adding to the available arch-length increase. Patients with lip bumper therapy must be monitored to avoid impacting the erupting second molars.

From a neuromuscular perspective, the lip bumper theoretically creates a more desirable treatment effect than does the Schwarz appliance. (The Schwarz appliance simply produces orthodontic tipping of the teeth through direct force application to the dentition and alveolus.) On the other hand, the lip bumper shields the soft tissue from the dentition, allowing for spontaneous arch expansion as is seen with the Fränkel and other soft tissue shielding appliances. We tend to favor the use of the Schwarz appliance over the lip bumper, however, in most instances because of the predictability of the treatment outcome and the ease of clinical management. Only in patients with very constricted (tense) soft tissue is the lip bumper the appliance of choice.

SPONTANEOUS IMPROVEMENT OF SAGITTAL MALOCCLUSIONS

The major focus of this section of the chapter thus far has been the resolution of intraarch tooth-size/arch-size discrepancy problems. Interestingly, there is another phenomenon that has been a serendipitous finding—"spontaneous" improvement of mild Class II and Class III malocclusions after RME.

Class II Patients

There are many patients in the mixed dentition who not only have intraarch problems but also have a Class II malocclusion or a strong tendency toward a Class II malocclusion. Generally, these patients do not have severe skeletal imbalances but rather may be characterized clinically as having either slight mandibular skeletal retrusion or an orthognathic facial profile with minimal neuromuscular imbalances.

According to the routine protocol described previously, these patients undergo RME with or without prior mandibular dental decompensation. At the time of expander removal, these patients will have a buccal crossbite tendency, with only the lingual cusps of the upper posterior teeth contacting the buccal cusps of the lower posterior teeth (Fig. 16-28, *A*). A maxillary

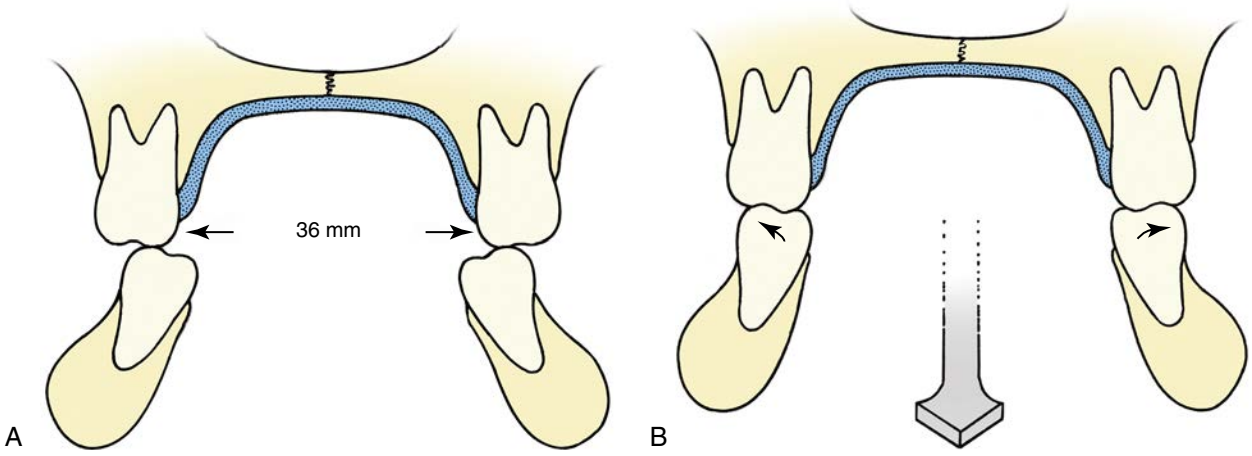


FIGURE 16-28 Frontal cross-sectional view of patient during the post–rapid maxillary expansion period. **A,** The maxilla has been expanded so that the intermaxillary width is 36 mm, as measured between the upper first permanent molars. Note the tendency toward a buccal crossbite bilaterally. **B,** During the postexpansion period, note that the lower dentition has uprighted slightly, and there has been a forward, sagittal movement of the mandible as the patient seeks to find a more stable position in which to occlude. (Adapted from McNamara JA Jr, Brudon WL. *Orthodontics and dentofacial orthopedics*. Ann Arbor, MI: Needham Press; 2001.)

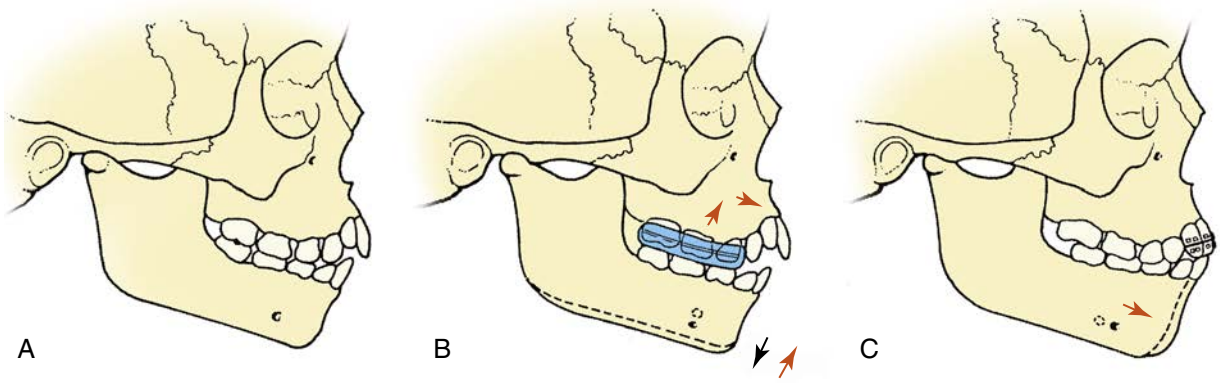


FIGURE 16-29 Sequence of events leading to a spontaneous improvement in the sagittal malocclusion. **A,** Pretreatment. The patient has excessive overjet and an end-to-end molar relationship. **B,** The placement of the appliance immediately creates a downward rotation of the position of the mandible due to the posterior occlusal acrylic. During treatment, an intrusive (and slightly protrusive) force is produced on the skeletal and dental structures of the maxilla. **C,** During the postexpansion period, the upper dental arch has been widened. The lower jaw often is postured forward to achieve a more stable occlusal relationship—a forward functional occlusion. In this illustration, brackets have been placed on the upper anterior teeth to facilitate incisal alignment.

maintenance plate typically is used to stabilize this relationship. Several appointments later, some interesting observations are noted: the tendency toward a buccal crossbite has disappeared (Fig. 16-28, *B*), and the patient now has a significant improvement in molar relationship, sometimes the establishment of a solid Class I sagittal occlusal relationship.

The orthodontist traditionally has viewed a Class II malocclusion as primarily a sagittal and vertical problem. Our experience with the post-RME correction of the Class II problem indicates that many Class II malocclusions also have a strong transverse component. The overexpansion of the maxilla, which subsequently is stabilized through the use of a removable palatal plate, disrupts the occlusion. It appears that the patient becomes more comfortable by positioning his or her lower jaw slightly forward, thus eliminating the tendency toward a buccal crossbite and at the same time improving the overall sagittal occlusal relationship. In many respects, the teeth themselves act as an endogenous functional appliance, encouraging a change in mandibular posture and, ultimately, a change in the maxillomandibular occlusal relationship.

The correction of a Class II tendency patient is illustrated in Figure 16-29. Figure 16-29, *A*, shows the sagittal view of the skeletal and dentoalveolar structures of a Class II tendency patient who has excessive overjet and a narrow maxilla. The placement of a bonded maxillary expansion appliance immediately causes an increase in the vertical dimension of the face

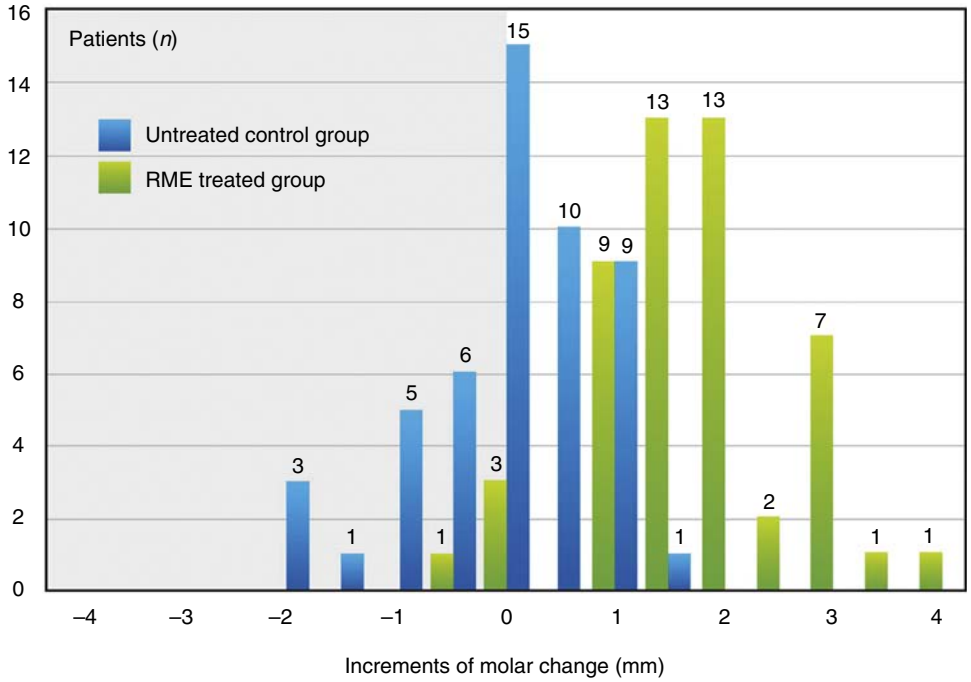


FIGURE 16-30 Prospective clinical study of spontaneous improvement in Class II molar relationship after expansion in the early mixed dentition. Both the treated and control groups had 50 patients or participants. The interval between the two lateral cephalometric films analyzed was 4 years. There was virtually no change in the control subjects, but 92% improved toward Class I in the treated group, nearly 50% by 2 mm or more. *RME,* Rapid maxillary expansion. (From Guest SS, McNamara JA Jr, Baccetti T, Franchi L. Improving Class II malocclusion as a side-effect of rapid maxillary expansion: a prospective clinical study. *Am J Orthod Dentofacial Orthop* 2010;138:582–591.)

because of the posterior occlusal coverage. This change is beneficial in most patients, in that the temporary increase in the vertical dimension prevents extrusion of the posterior teeth during the expansion process.[53] This treatment also may result in an upward and slightly forward displacement (Fig. 16-29, *B*) of the maxilla. (This phenomenon will be discussed subsequently in the discussion of the spontaneous improvement of Class III malocclusion.) During the post-RME period, during which a removable palatal plate is worn (Fig. 16-29, *C*), the mandible is postured forward by the patient because of the overexpansion of the maxilla. Thus, the spontaneous improvement of patients with a tendency toward a Class II malocclusion does not occur during the active expansion period but rather during the time that the maintenance plate is being worn.

Because of the perceived importance of this issue, we have conducted two prospective clinical trials investigating this phenomenon. Guest and coworkers[61] contrasted the treatment results of 50 Class II or end-to-end patients treated with a bonded expander to 50 matched untreated control participants (Fig. 16-30). The analysis of serial cephalometric films taken 4 years apart indicated that the bonded RME had its greatest effects at the occlusal level, specifically producing highly significant improvement of Class II molar relationship and a decrease in overjet. The Class II molar relationship remained virtually unchanged in the control group, but the RME group showed an improved molar relationship of more than 1 mm in more than 90% of the expansion patients and more than 2 mm in almost 50% (see Fig. 16-30). The second study[61] on a larger group of patients (500 RME patients from the McNamara private practice; 188 control participants) revealed similar results.

Thus, this improvement in Class II relations occurs with such frequency that it can be included as part of the overall treatment plan. If the resulting occlusion remains Class II at the time of phase II treatment, then definitive Class II corrective procedures can be initiated.

Class III Patients

The use of a bonded RME appliance also can lead to a spontaneous occlusal improvement in a patient with a tendency toward a Class III malocclusion. At first glance, this phenomenon seems paradoxical, given the previous discussion concerning the spontaneous improvement of Class II tendency problems. The mechanism of Class III correction, however, is distinctly different from that described previously.

An examination of Figure 16-29, *B*, provides some explanation for this phenomenon. The placement of an acrylic splint expander that opens the bite vertically 3 mm not only provides an intrusive force against the maxilla,[53] presumably because of the stretch of the masticatory musculature, but also may produce a slight forward repositioning of the maxilla. A slight forward movement of the maxilla after RME has been documented in both clinical[62] and experimental[63] studies. In addition, the placement of a bonded expander with acrylic coverage of the occlusion helps eliminate a tendency toward a pseudo–Class III malocclusion.

As with the Class II tendency patients described previously, patients in whom a borderline Class III malocclusion exists

usually have a reasonably balanced facial pattern, often with only a slight tendency toward maxillary skeletal retrusion. Obviously, in patients in whom Class III malocclusion persists after expansion, more aggressive types of therapies are indicated, as will be discussed later.

When contrasting the spontaneous improvement of both Class II and Class III tendency patients, it must be emphasized that any spontaneous improvement of a Class III malocclusion usually occurs (if it does occur) during the *active* phase of treatment (within the first 30 or 40 days). The spontaneous correction of Class II malocclusion usually is noted during the *retention* phase, after the bonded expander has been removed and the maintenance plate has been worn for 6 to 12 months. When planning the treatment for a Class III tendency patient, FM hooks may be attached to the expansion appliance to facilitate the use of an FM if that treatment is deemed necessary from the original plan or at a later time.

THE TREATMENT OF CLASS II MALOCCLUSION

A number of treatments are available for correcting Class II malocclusions, including a variety of extraoral traction appliances, arch expansion appliances, extraction procedures, and FJO appliances. Each treatment approach, however, differs in its effect on the skeletal structures of the craniofacial region, sometimes accelerating or limiting the growth of the various craniofacial structures involved. As noted at the beginning of this chapter, timing of treatment usually is most effective during the circumpubertal growth period, the late mixed to permanent dentition.

Components of Class II Malocclusion

Numerous studies have considered the components of Class II malocclusions, with most focusing on patients in the adolescent or adult age range.[64-66] These studies have shown that the term *Class II malocclusion* is not a single diagnostic entity but rather can result from numerous combinations of skeletal and dentoalveolar components.

McNamara[67] has shown that in mixed dentition Class II subjects before treatment, mandibular skeletal retrusion was the most common single characteristic of his large Class II sample ($n = 277$). Substantial variation also was noted in the vertical development of the face, with one-third to half of the sample having increased vertical facial dimensions. The anteroposterior position of the maxilla on average was neutral, with far more instances of maxillary skeletal retrusion than maxillary skeletal protrusion being observed. When measures independent of mandibular position were used for evaluation, the upper incisors of the Class II sample were on average in a normal anteroposterior position, with more instances of maxillary dentoalveolar retrusion than maxillary dentoalveolar protrusion being observed.[67] The lower incisors usually were well positioned anteroposteriorly, but instances of mandibular dental retrusion and protrusion also were noted.

Available Class II Treatment Strategies

After the skeletal and dentoalveolar components of an individual Class II malocclusion are identified, using data gathered from the clinical examination and a radiographic evaluation as well as from study models, the appropriate treatment regimen can be selected. This discussion focuses on the treatment of problems of Class II malocclusion that are primarily skeletally related, with specific emphasis on *maxillary distalization* and *mandibular enhancement* mechanics, two of the most commonly used treatment approaches.

Maxillary Distalization

In patients with a forward positioning of the maxillary dentition relative to the bony base of the maxilla, either extraction protocols (i.e., ultimately removing the upper first premolars) or dentoalveolar distalizing mechanics (e.g., Pendulum/Pendex appliance,[68-70] Distal Jet,[71,72] TAD-secured distalizers) can be used.

Extraoral Traction

Historically, the most common treatment for true maxillary skeletal protrusion has been extraoral traction. Extraoral traction appliances can be divided arbitrarily into two types: facebows and headgears. Whereas facebows attach to tubes on the maxillary first molar bands, a headgear attaches directly to the archwire or to auxiliaries connected to the archwire.

The cervical (low-pull) *facebow* (Fig. 16-31, *A*) is used most frequently in patients with normal or decreased vertical facial dimensions. The inner bow of the facebow is anchored to tubes that are placed on the buccal surface of bands that are attached to the upper first molars. The outer bow is connected to a safety release elastic strap that extends to the cervical region and is anchored against the dorsal aspect of the neck. Usually the outer bow of the facebow lies above the plane of occlusion (e.g., 15 to 20 degrees) so that the force is directed through the center of resistance to prevent distal tipping of the molars during treatment. Numerous clinical studies[28,73,74] have shown that the forward movement of the maxilla can be inhibited through the use of this type of appliance. Cervical traction also can increase the vertical dimension through the extrusion of posterior teeth.

The direction of extraoral force can be altered, depending on the placement of the attached anchoring units. For example, an occipital (high-pull) facebow (Fig. 16-31, *B*) is used in individuals in whom increases in vertical dimension are to be minimized or avoided. The facebow is anchored to an occipital anchoring unit (headcap) to produce a more vertically directed force. As a growth guidance appliance, a high-pull facebow can decrease the vertical development of the maxilla, thereby allowing for autorotation of the mandible and maximizing the horizontal expression of mandibular growth. A facebow also can be anchored simultaneously to a cervical neckstrap and an occipital headcap, a combination often termed a *straight-pull* or *combination facebow* headgear.

The forces produced by extraoral traction also can be attached anteriorly to the archwire through the use of a J-hook *headgear*. Flared upper incisors can be retracted using either a straight-pull (Fig. 16-32, *A*) or a high-pull headgear (Fig. 16-32, *B*) combined with J hooks that are attached to the archwire anteriorly or by using a closing arch supported by headgear. A headgear with J hooks also is used to potentiate archwire mechanics by helping control forces incorporated into the archwire (e.g., torque, intrusion).

The use of the Interlandi-type headgear (Fig. 16-33) provides an additional treatment option with a variable direction of force. J hooks can be applied to the maxillary teeth in a variety of force vectors to retract and intrude the upper incisors (Fig. 16-33, *A*). A similar type of retraction-stabilization of the

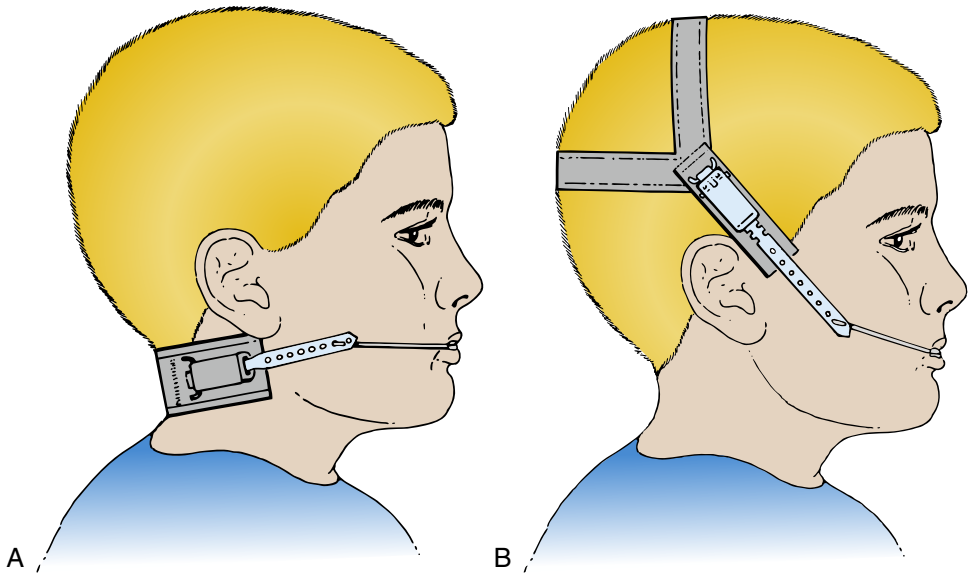


FIGURE 16-31 Extraoral traction. **A,** Low-pull (cervical) facebow with safety connector. **B,** High-pull (occipital) facebow with safety connector. (Adapted from McNamara JA Jr, Brudon WL. *Orthodontics and dentofacial orthopedics*. Ann Arbor, MI: Needham Press; 2001.)

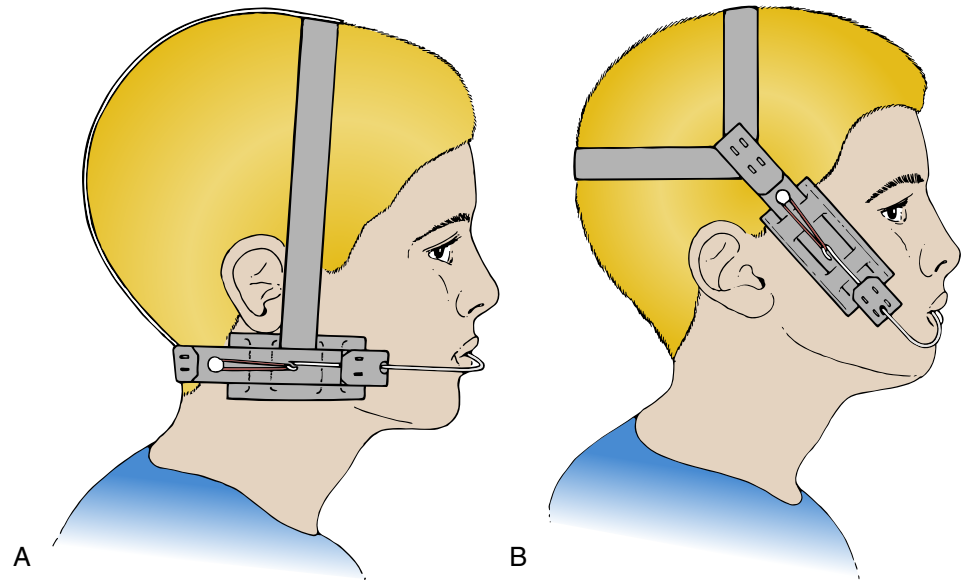


FIGURE 16-32 Headgear. **A,** Straight-pull headgear with J hooks. **B,** High-pull headgear with J hooks. (Adapted from McNamara JA Jr, Brudon WL. *Orthodontics and dentofacial orthopedics*. Ann Arbor, MI: Needham Press; 2001.)

mandibular dental arch also can be achieved. In addition, it is possible to attach to a high-pull headgear to the upper arch and a straight-pull headgear to the lower arch simultaneously (Fig. 16-33, *B*).

Virtually all of the extraoral traction appliances described earlier restrict the normal downward and forward movement of the maxilla and also may help retract the maxillary and mandibular dentitions to differing degrees depending on a nonextraction or premolar extraction plan—and patient cooperation. These types of appliances are indicated in instances of maxillary skeletal protrusion, maxillary dentolaveolar protrusion, and mandibular dentoalveolar protrusion. The direction of force (i.e., low pull, straight pull, high pull) is determined in part by the pretreatment vertical dimensions of the patient and treatment goals.

Maxillary Molar Distalization

The use of distalization mechanics to correct Class II malocclusions is a common treatment modality. A survey by Sinclair[75] found that all responding orthodontists reported use of molar distalization. However, nearly all indicated that patient cooperation was the most significant problem encountered in distalizing maxillary molars. Most traditional approaches to molar distalization, including extraoral traction, Wilson distalizing arches, removable spring appliances, and intermaxillary elastics with sliding jigs, require considerable patient compliance

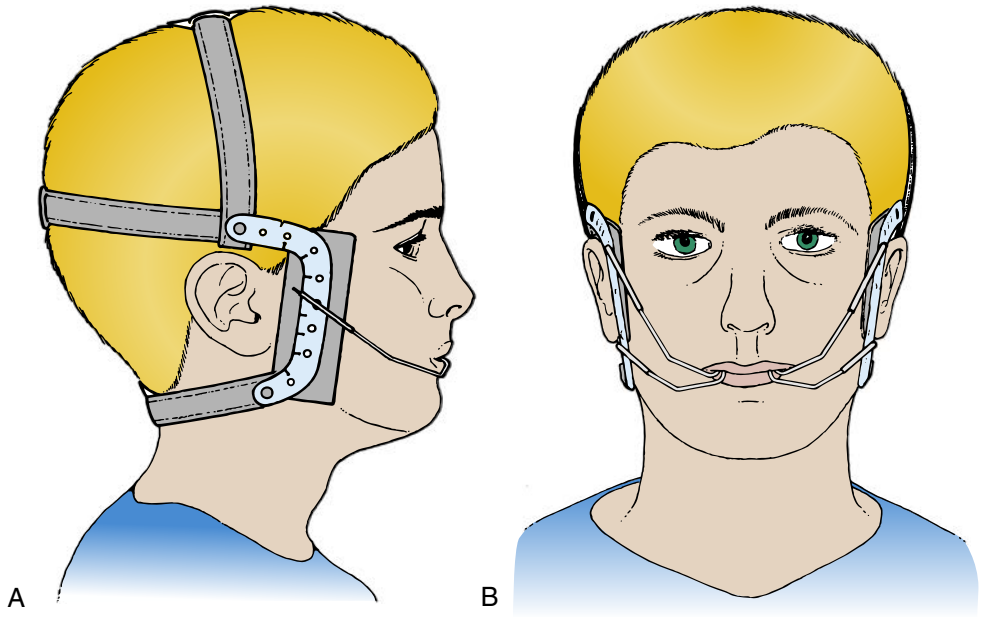


FIGURE 16-33 Interlandi-type headgear. **A,** Single pull to the maxillary dentition. **B,** Attachment of the J hooks to both the maxillary and mandibular dental arches. (Adapted from McNamara JA Jr, Brudon WL. *Orthodontics and dentofacial orthopedics*. Ann Arbor, MI: Needham Press; 2001.)

to be successful. More recently, problems of predicting patient behavior have led many clinicians to devise appliances that minimize reliance on the patient and that are under the control of the clinician. Relying on the patient's willingness to wear an appliance consistently may result in increased treatment time, a change of treatment plans, or both with related uncertainty in attaining treatment goals.

Pendulum and Pendex Appliances

A popular method of molar distalization that requires no direct patient cooperation is the Pendulum appliance system. In 1992, Hilgers[76] described the development of two hybrid appliances, the Pendulum and Pendex.

The *Pendulum appliance* (Fig. 16-34) consists of a large acrylic Nance button that covers the middle part of the palate. The acrylic pad is connected to the dentition by means of occlusal rests that extend from the lateral aspect of the pad and are bonded to the occlusal surfaces of the upper first and second premolars. Posteriorly directed springs, made of 0.032-inch titanium molybdenum alloy (TMA) wire, extend from the distal aspect of the palatal acrylic to form a helical loop near the midline, then extending laterally to insert into lingual sheaths on bands cemented on the upper first molars.

When in a passive state, the springs extend posteriorly, paralleling the midpalatal raphe. When activated and inserted into the lingual sheaths, they produce a distalizing force against the upper first molars that moves the molars distally and medially. Hilgers[76] estimates that these springs deliver approximately 230 g per side to the maxillary molars. The springs also may have adjustment loops that can be manipulated to increase molar expansion, molar rotation, or distal root tip.[77,78]

The design of the *Pendex appliance* (Fig. 16-35) is essentially the same as the Pendulum, except for the addition of a palatal expansion screw in the midline (hence the name "Pendex"). In most instances, we use the Pendex design because of the tendency toward transverse maxillary constriction in patients with Class II malocclusion as well as the trend toward the lingual movement of molars as they are distalized with this type of appliance.

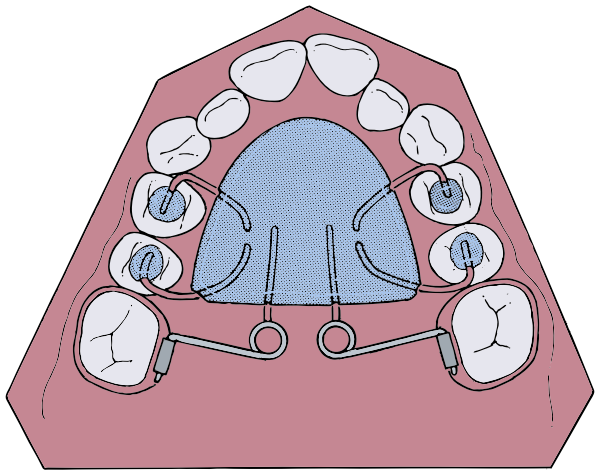


FIGURE 16-34 The Pendulum appliance of Hilgers that has been bonded in place after cementing the molar bands. The distalizing springs are activated after the occlusal rests have been bonded to the premolars by placing the ends of the springs into the lingual sheaths on the upper first molars.

The so-called T-Rex configuration of the Pendex is our design of choice. This design features two wires that extend from the palatal acrylic and are soldered to the lingual aspect of the upper first molars. These wires provide additional stability to the Pendex appliance during the expansion phase of treatment (Fig. 16-36). They are removed when the molar distalization phase is initiated after adequate expansion has been achieved (Fig. 16-37). After molar distalization is complete, the occlusal rests can be removed from the maxillary second

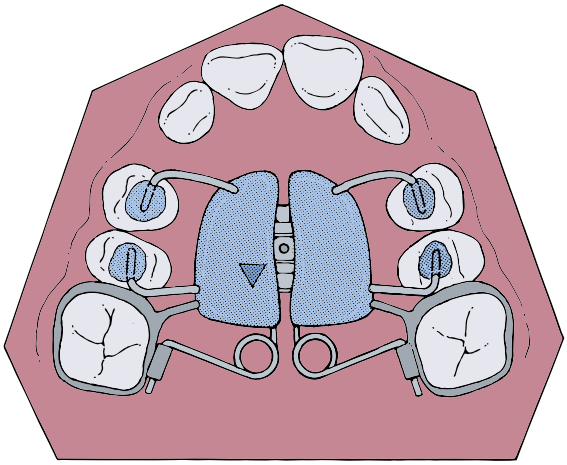


FIGURE 16-35 The "T-Rex" design of the Pendex appliance of Hilgers. Locking wires connect the bands on the upper first molars to the acrylic button. These connecting wires are removed after the desired expansion has been achieved, partially counteracting the medial action of the distalizing molar springs.

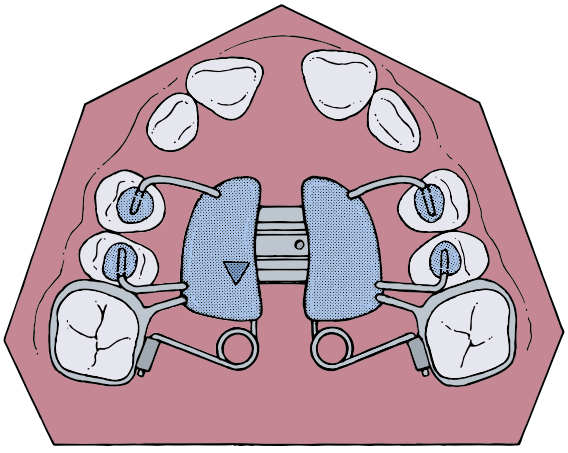


FIGURE 16-36 The "T-Rex" design of the Pendex appliance after expansion. The locking wires still connect the molar bands to the acrylic Nance button.

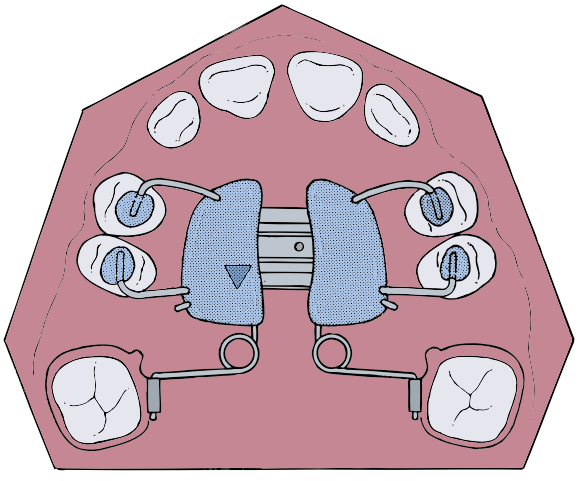


FIGURE 16-37 Full activation of the Pendex appliance. The upper first molars now are in an overcorrected ("super Class I") relationship with the lower first molars.

premolars, allowing for distal migration as a result of the pull of the transseptal fibers (Fig. 16-38).

After appliance removal, a Nance holding arch with a palatal button (Fig. 16-39) is delivered to the patient within the next 24 hours. The Nance holding arch is left in place until proper distalization of the premolars and canines is achieved. Final distalization of the premolars and canines is accomplished by placing brackets on these teeth and using elastomeric chain sequentially to move these teeth distally one at a time per side (i.e., moving "beads on a string"). Anterior space closure can be achieved by way of a retraction utility arch or an anterior closing loop arch. Typically, space closure also is supported by Class II elastics. For further details concerning the clinical management of the appliance, the reader is referred to the McNamara and Brudon text.[1]

Mandibular Enhancement: Functional Jaw Orthopedics

The previous section described several approaches aimed at correcting a Class II molar relationship by moving the maxilla

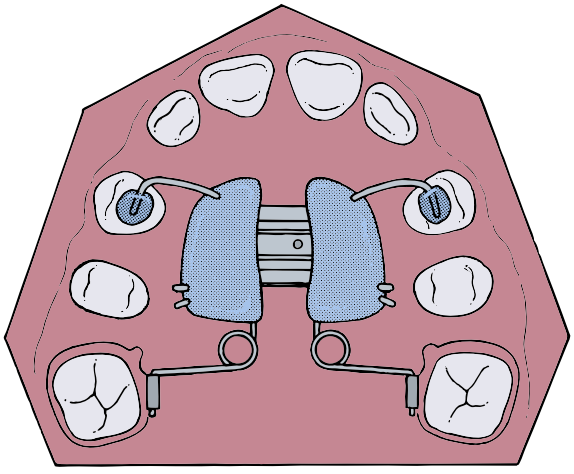


FIGURE 16-38 The spontaneous movement of the upper second premolars distally due to the pull of the transseptal fibers after the occlusal rests on the maxillary second premolars are removed.

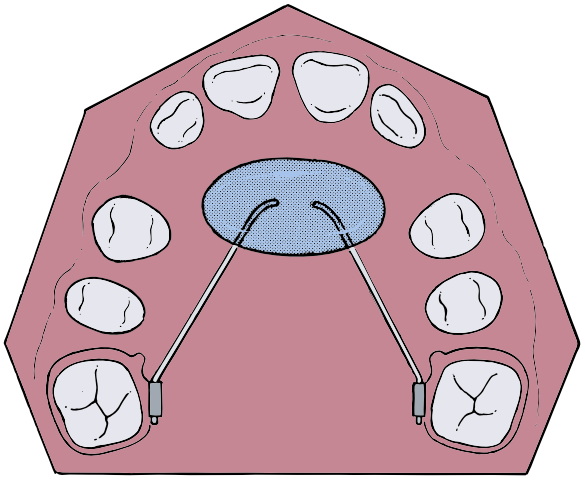


FIGURE 16-39 Placement of a Nance holding arch after molar distalization. The button should be contoured to engage both the anterior slope and superior "flat" portion of the palate.

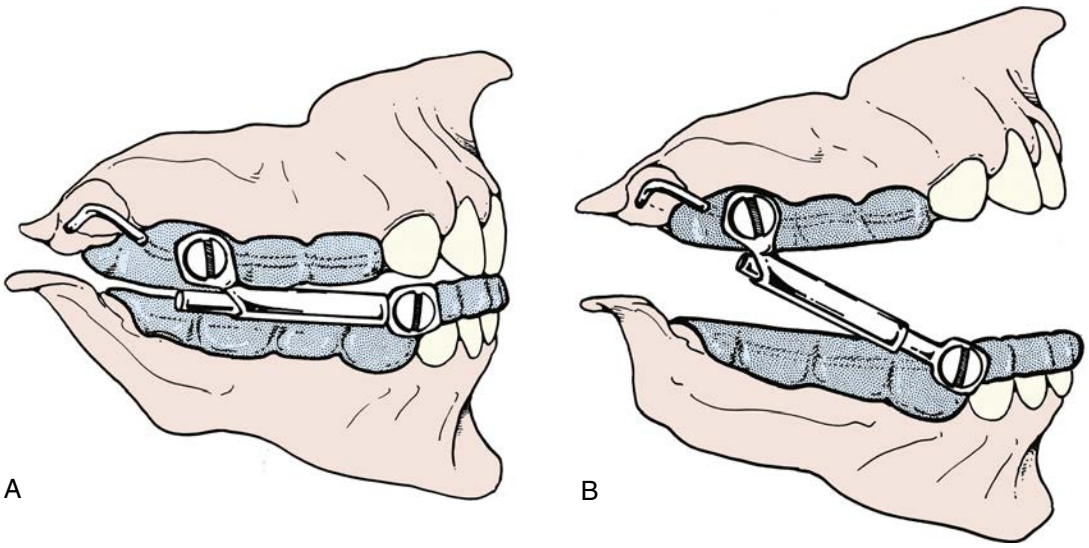


FIGURE 16-40 The Herbst bite-jumping mechanism. In this illustration, the bite-jumping mechanism is secured to the teeth by way of acrylic splints made from 3-mm-thick splint Biocryl. **A,** In occlusion. **B,** Mouth open.

and/or the maxillary posterior teeth distally. A second type of treatment modality aimed at correcting a Class II malocclusion focuses its mechanics on influencing the mandibular dentition and the growth of the mandible. This type of treatment is referred to as *functional jaw orthopedics*, with the intent of treating occlusal problems associated, at least in part, with mandibular skeletal retrusion.

The concept of FJO or advancement of the mandible is not new to dentistry. In 1880, Kingsley[79] wrote of "jumping the bite" and forward posturing the mandible. Yet given its long history, such appliances were used rarely in the United States until the mid-1970s. Over the past 40 years, there has been a gradual evolution of the way in which FJO is used in a contemporary orthodontic practice, especially concerning appliance selection, the timing of intervention, and the need for preorthopedic orthodontic treatment.

Like in most aspects of orthodontics, there are many ways to achieve a similar outcome. This is true of FJO appliances, with their differences in both material and design. The orthodontic literature describes a variety of appliance designs, including the activator,[80] the bionator,[5,81] the function regulator (FR-2) appliance of Fränkel,[42] the Herbst appliance,[1,82] the MARA appliance,[83] and the twin block appliance.[84] Although these appliances may appear very different at first glance, they all work to achieve a Class I occlusion by posturing the mandible forward during a time of growth (see Chapter 35).

It is not the purpose of this chapter section to debate whether FJO appliances do or do not enhance mandibular growth, a topic of great controversy during the past 35 years. The question of whether the mandible can be increased in length in comparison with untreated controls has been addressed in numerous experimental studies, and many clinical studies of various appliances have been conducted as well (see McNamara and Brudon[1] for a discussion of these issues). As mentioned earlier, the bulk of scientific evidence indicates that, in growing individuals, mandibular growth can be enhanced over the short term. For example, one of the more rigorous studies of FJO that analyzed patients treated by Rolf Fränkel showed that the residual increase in mandibular length was on average 3 mm,[85] not 5 to 8 mm (an amount that would be equivalent to a surgical mandibular advancement). The long-term effect on mandibular length remains open to question.

The major question remaining is whether or not the extra growth and development throughout the dentofacial region have clinical relevance. It is our belief that the skeletal adaptations, combined with dentoalveolar changes, can lead to a significant correction of a Class II malocclusion using a variety of FJO treatment modalities. Whereas these questions regarding the existence of extra growth as a result of FJO can be studied and debated further, the clinical effectiveness of the use of these appliances is less debatable. Experience has shown us that by posturing the lower jaw forward into a Class I or an end-on Class III relationship during a time of maximum mandibular growth, the lower jaw grows forward into its postured position without dragging the upper teeth with it. With proper appliance selection, treatment timing, and patient compliance, one may achieve a major orthodontic goal of obtaining a Class I molar and canine relationship and often improved maxillary–mandibular balance.

Appliance Selection

All FJO appliances have one aspect in common: they induce a forward mandibular posturing as part of the overall treatment effect. Presumably, this alteration in the postural activity of the muscles of the craniofacial complex ultimately leads to changes in both skeletal and dental relationships.[78]

Herbst Appliance

An appliance that has proved excellent in the treatment of Class II malocclusions in the permanent dentition is the Herbst appliance (Fig. 16-40), a fixed or removable functional appliance depending on the anchoring system used. The original bite-jumping mechanism was developed by Herbst,[86] and the banded design of the appliance was reintroduced in the late 1970s by Pancherz[82] and refined later by Rogers.[87] Designs that incorporate stainless steel crowns as anchoring mechanisms have been advocated by Dischinger,[88] Smith,[89] and Mayes.[90]

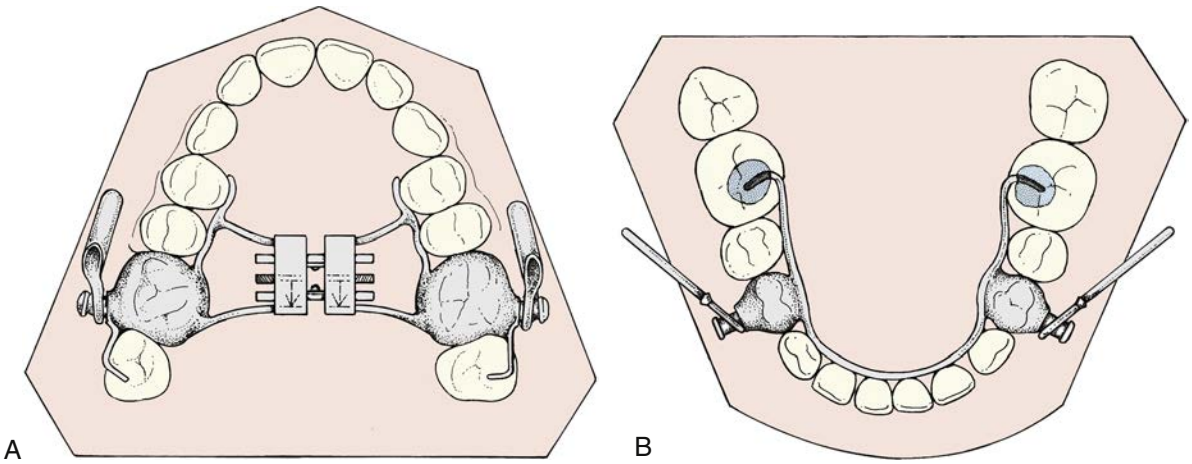


FIGURE 16-41 The stainless steel crown Herbst appliance. **A,** Maxillary portion. Stainless steel crowns are placed on the upper first molars, and a rapid maxillary expansion screw typically is placed in the midpalatal region. **B,** Mandibular portion. Stainless steel crowns are placed on the lower first premolars. A lower lingual wire made from 0.036-inch stainless steel extends from first molar to first molar. Occlusal rests are bonded to the lower first molars bilaterally. There are a variety of designs commercially available, with the clinician cautioned to match desired anteroposterior and vertical effects with treatment goals.

FIGURE 16-42 The banded Herbst provides ease and accuracy of fit, minimal increase in bite opening, and ease of removal. The advancing arm is telescoping (providing for a smaller appliance profile), and the anterior attachments of the lower member allow for increased lateral range of mandibular function. The lingual wire is 0.051 inch, and rests are placed either on the first or second premolars. The "bands" are either occlusal-removed crowns or heavier material bands with reinforcing wires on the occlusal. Archwire tubes are incorporated to control intraarch mechanics.

We have used many types of fixed Herbst appliances over the years, with the current preferred version incorporating stainless steel crowns on the maxillary first molars (Fig. 16-41) and mandibular first premolars (see Fig. 16-41). Our routine prescription also includes an RME screw with lingual wires extending anteriorly to the premolars. A removable acrylic splint design Herbst appliance[91,92] also has been used successfully in our practice for several decades (see Fig. 16-40). The banded Herbst design as described by Rogers[87] (Fig. 16-42) has a number of features that prove helpful. In particular, there is no interference with the occlusion, it is easier to fit precisely, and removal of the appliance is easier.

Clinical studies of the fixed and removable designs of the appliance indicate that both skeletal and dentoalveolar adaptations are produced.[70,93,94] Generally speaking, about 50% of the treatment effect is due to tooth movement, primarily the backward and upward movement of the posterior maxillary dentition. The primarily skeletal treatment effect produced is a short-term increase in mandibular growth (i.e., 2.0 to 2.5 mm greater than normal values).[70,93-95]

There is no question that a Class I molar relationship can be achieved in most growing Class II patients after Herbst appliance treatment. It has been our experience, however, that a Herbst appliance is not the appliance of choice in mixed dentition patients. After having followed patients originally treated in the mixed dentition for several years after Herbst therapy was completed but before the placement of fixed appliances was initiated, we have noted a significant tendency toward a relapse to the original malocclusion. This finding also has been noted by Pancherz,[96] among others.

This observation concerning Herbst patients treated in the mixed dentition may be due, in part, to the lack of direct effect on the orofacial musculature produced by the Herbst appliance (in contrast to the FR-2 appliance) and also may be related to the shape of the deciduous teeth. The posterior deciduous teeth tend to be relatively flat or are lost and thus do not provide the same type of occlusal interdigitation as occurs in the permanent dentition.

Our lack of success using the Herbst appliance in the mixed dentition does not eliminate this appliance from consideration. As mentioned earlier, we initiate regimens in the early mixed dentition to address intraarch space discrepancy problems and postpone functional appliance treatment until after all deciduous teeth are lost and the succeeding teeth are erupted. This delayed intervention is particularly useful in patients who have excessive vertical facial development and steep mandibular plane angles. We have found that by intervening in the early permanent dentition using the Herbst appliance, satisfactory skeletal and dental adaptations have been noted overall. This appliance is used most effectively in patients who do not have profound neuromuscular imbalances.

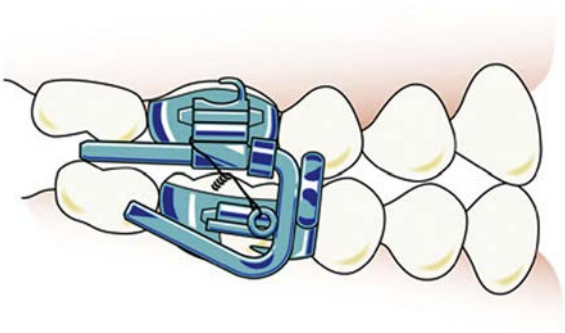


FIGURE 16-43 Mandibular anterior repositioning appliance (MARA). The arm soldered to the lower stainless steel crown guides the lower jaw anteriorly as it articulates with the maxillary arm during closure.

Mandibular Anterior Repositioning Appliance

Another appliance that has gained in popularity for correction of Class II malocclusions is the mandibular anterior repositioning appliance, or MARA.[83] The MARA is a fixed tooth-borne appliance that is fabricated on stainless steel crowns commonly placed over the maxillary and mandibular first permanent molars (Fig. 16-43). It has been used throughout the mixed and early permanent dentition stages, with similar indications as the Herbst appliance described earlier. The MARA acts by prohibiting the patient from closing in a natural, Class II relationship. Upon mandibular closure, the MARA's extension arms interfere, making it so that the mandible must be postured forward in order for full occlusal contact to occur.

The MARA appliance produces treatment effects that generally are similar to those of the Herbst, except for a few differences. Whereas maxillary molar intrusion is a characteristic feature of Herbst use,[93,97] this finding has not been reported with the MARA. Although it has been demonstrated that the MARA and Herbst appliances both produce significant change with respect to controls in the horizontal position of the mandibular incisors, the MARA has been shown to produce less flaring of the lower incisors.[83]

The anteroposterior treatment effect of the MARA is achieved through both skeletal and dental changes. Studies on skeletal changes indicate that the MARA produces increases in mandibular length but exerts negligible skeletal effects on the maxilla. In contrast, dental changes seen are due mainly to the distalization of the maxillary molar, which is said to be about 75% of the total dental correction.[83] Mesial movement of the mandibular molar accounts for approximately 25% of the total dental correction.

Because of the MARA's design, possible undesirable dental movements should be considered. In the sagittal plane, distal rotation of upper molars or mesial rotation (or both) of the lower molars may be observed. These movements may be controlled by incorporation of additional support into the design of the MARA (e.g., a TPA, a fixed expander, and/or a lower lingual holding arch). Because of crowns on both maxillary and mandibular first molars, there is often a transient intrusion of the molars on crown removal that self-corrects quickly. Overall, the appliance works well in correcting Class II problems. The biggest drawback to using the MARA, however, is appliance breakage and initial patient perception of bulk, which can be perceived as a significant problem. To mitigate this issue on patients with small mouths and tight cheeks, one can leave off the second premolar brackets so that the MARA arms may be constructed more closely to the dental arch. Small shields can also be placed to help keep tight cheeks away from the MARA arms.

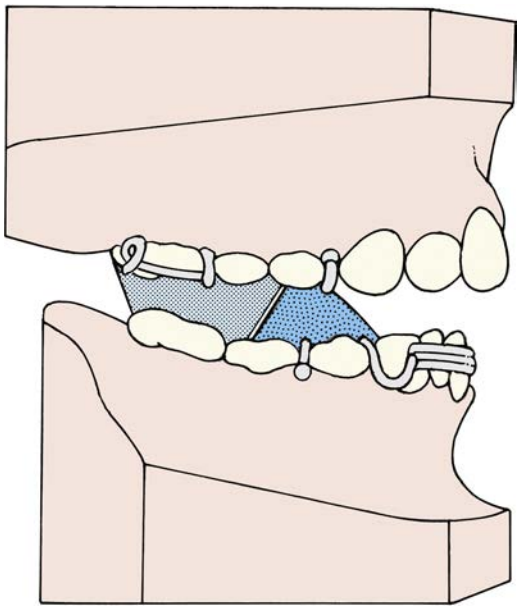


FIGURE 16-44 Sagittal view of the twin block appliance. A labial bow to which clear acrylic has been added increases the anterior retention of the lower appliance, especially during the period of the transitional dentition. The angled interface between maxillary and mandibular appliances guides the mandible into a forward functional position.

The Twin Block Appliance

Our choice of functional appliance selection for mixed dentition patients has changed during the past decade because of our increased clinical experience with the twin block appliance. This appliance, developed more than 30 years ago by Clark,[84] is composed of maxillary and mandibular removable acrylic components that fit tightly against the teeth, alveolus, and adjacent supporting structures (Fig. 16-44). Interproximal clasps are used bilaterally to anchor the maxillary appliance to the first permanent molars and premolars.

The maxillary occlusal view of the twin block appliance is shown in Figure 16-45. We have modified the design of the twin block appliance slightly by adding a second midline screw in the midsagittal region of the appliance. Our experience has shown that in patients in whom significant expansion is desired during twin block treatment, the appliance becomes unstable and too flexible if only one midline screw is used. Each screw is activated once per week (≈0.2 mm) until adequate expansion is attained. Clasps are used to secure the appliance to the first molars.

In the lower arch, Clark[84] has recommended the use of a series of ball clasps that lie in the interproximal areas between the canines and lower incisors. We have modified this design (Fig. 16-46) by placing a labial bow anterior to the lower incisors that has labial acrylic similar to that of a lower spring retainer.[1] In contrast to the spring retainer laboratory setup, however, the positions of the lower incisors are not altered in the work model before appliance construction.

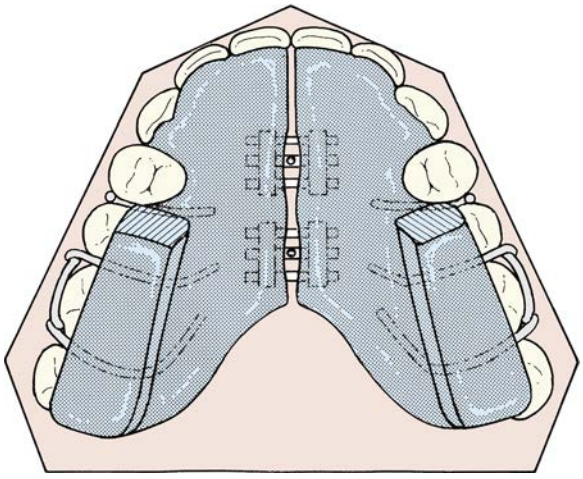


FIGURE 16-45 Maxillary occlusal view of the twin block appliance. Two expansion screws are placed in the midline. Delta clasps are used to secure the appliance to the molars posteriorly, and ball clasps are used to anchor the appliance in the premolar and deciduous molar region.

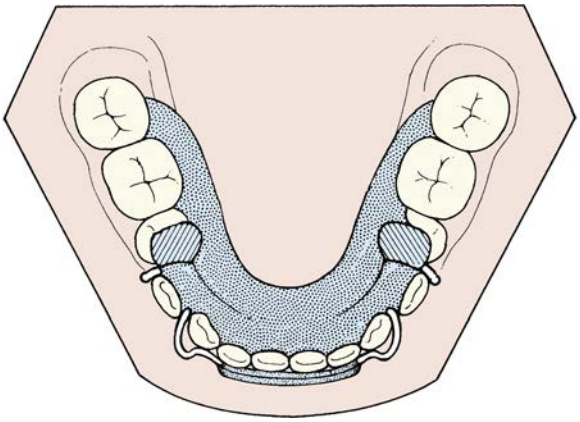


FIGURE 16-46 Mandibular occlusal view of the twin block appliance. Preferred modified design in which the lower lingual acrylic extends posteriorly into the permanent molar region. The lower labial bow with clear acrylic covering extends anteriorly to cover the labial surfaces of the lower anterior teeth. The bite blocks terminate 2 to 3 mm in front of the lower first molar.

The twin block appliance has been shown to produce increases in mandibular length as well as variations in lower anterior facial height.[1,84] The posterior bite blocks of the twin block appliance can be trimmed to facilitate the eruption of the lower posterior teeth in patients with a deep bite and an accentuated curve of Spee. The blocks also can be left untouched to prevent the eruption of the posterior teeth in patients with a tendency toward an anterior open bite, increased lower vertical facial height, or both.

One of the primary reasons why the twin block appliance is indicated in the treatment of Class II malocclusion is a reasonably high level of patient compliance. Because the twin block is composed of two parts, speaking typically is not a problem. The duration of treatment usually is 9 to 12 months followed by nighttime wear of the appliance or by the use of a stabilization plate to allow for the eruption of the canines and premolars into occlusion. Phase II treatment with fixed appliances usually is begun after the transition to the permanent dentition is complete.

Treatment Timing for Class II Malocclusion

As mentioned earlier, one of the major changes that has occurred during the past 35 years has been an alteration in the timing of treatment using functional appliances. An early study by our group[24] indicated that, when comparing two cohorts of patients who were treated with the FR-2 appliance of Fränkel, those patients who began treatment at an average of 11.5 years showed a greater mandibular growth response than did patients beginning treatment at approximately 8.5 years of age. The reason for this increased growth response may be related to the synergistic interaction between a change in function, produced by the functional appliance, and growth hormone and related substances that are in greater quantity during the circumpubertal growth period.

A follow-up study by Franchi and Baccetti[98] that considered CVM stages (CS-1 and CS-2 vs. CS-3 and CS-4) of the same populations also showed substantially more mandibular growth in patients receiving FJO treatment during the circumpubertal growth period than earlier when the treated samples were compared with matched control participants. The interaction between altered function and growth hormone also has been demonstrated in the experimental studies of Petrovic and coworkers,[23] among others. (See Chapter 1 for more detailed description of the growth-related factors.)

In general, the onset of FJO therapy in a mild to moderate Class II patient typically is delayed until the middle or end of the mixed dentition. It is our intention to schedule FJO treatment so that this treatment will be followed immediately by a comprehensive phase of fixed appliance therapy. In patients with a significant overjet and mandibular skeletal retrusion, treatment with the cantilever-type Herbst appliance or twin block appliance may be initiated in the early mixed dentition as a single appliance or as part of a more comprehensive protocol that includes RME before the eruption of the permanent dentition. In instances of significant neuromuscular imbalances, however, the FR-2 appliance is the appliance of choice.

Additional Comments Regarding Class II Treatment

There is no one ideal method of treating all Class II malocclusions. After a thorough clinical examination, a precise analysis of both the radiographic images and the dental casts should be undertaken to identify the components of the malocclusion that make an individual patient unique. After a thorough diagnosis has been established, the clinician can select the appropriate treatment regimen from a wide variety of available treatment modalities.

In recent years, our use of FJO appliances has decreased in frequency compared with 10 to 15 years ago. This change is in response to our experiences using RME during the mixed dentition to improve the underlying transverse discrepancy, observing spontaneous improvement of some Class II problems. Additionally, we have focused on providing our patients with noncompliance-based treatment options. It has been our experience that patient cooperation with removable orthopedic appliances has become more difficult to secure as children's activities remove them from the home and parents provide less appliance wear monitoring. Because true maxillary skeletal protrusion patients are observed relatively infrequently[67] and noncompliance treatments have become

available, extraoral traction is a minimally used treatment option within our patient population.

TREATMENT OF CLASS III MALOCCLUSION

One of the most difficult types of malocclusions to treat is a Class III malocclusion. The occurrence of an end-to-end incisor relationship or a frank anterior crossbite is identified easily by both the family practitioner and the parent as an abnormal occlusal relationship. Thus, it is common for Class III patients to be referred for early treatment. The outcome of various early treatment protocols may or may not be successful, however, depending on the severity of the problem, the familial malocclusion and respiratory histories of the patient, and the age at which treatment is initiated.

Components of Class III Malocclusion

Various authors have estimated that 3% to 5% of the population demonstrates a Class III malocclusion. Government survey data from the National Health and Nutrition Examination Survey (NHANES) also show that the prevalence of extreme reverse overjet (Class III) is three times higher in Asians than in blacks or whites and twice as great in Hispanics (Mexican Americans).[99] Class III patients comprise about 5% of the typical orthodontic patient load in the United States.[100] This type of malocclusion is far more prevalent in other regions of the world, particularly in Pacific Rim countries. Thus, the treatment of Class III problems comprises a significant portion of orthodontic and orthopedic treatment with excellent clinical reports primarily from Japan, Taiwan, and Korea.

Class III malocclusion does not encompass a single diagnostic entity. Rather, it can be due to maxillary skeletal retrusion, mandibular skeletal protrusion, or a combination of the two.[101,102] As with all malocclusions that are considered by the Angle classification system, Class III malocclusions include a variety of skeletal and dental components that may vary from our concept of normal or ideal in all three dimensions.

Available Class III Treatment Strategies

Before discussing early treatment strategies, it is important to review briefly the usual approach to the correction of Class III malocclusions in an adolescent or adult patient. When a patient first is diagnosed as having a Class III malocclusion in the permanent dentition, treatment options are limited, particularly if there is a strong skeletal component to the Class III occlusal relationship. Such treatment usually includes comprehensive orthodontic therapy combined with extractions, orthognathic surgery, or both.

The orthognathic surgical procedure is designed to address the imbalance of the skeletal component (e.g., sagittal split osteotomy or vertical ramus osteotomy to posteriorly reposition the mandible in instances of mandibular prognathism, LeFort I advancement in instances of maxillary skeletal retrusion; procedures in both jaws may be used in instances of severe maxillomandibular skeletal imbalances). In other words, the surgical procedure is designed to correct whatever skeletal imbalances are present. In patients in whom significant skeletal growth is anticipated, the surgical procedure is deferred until the end of the active growth period. Such patients, however, still face potentially adverse psychosocial problems during childhood and the teen years that have been shown to be associated with this type of malocclusion.[103]

The treatment of Class III malocclusion in the primary and mixed dentition can be approached from a slightly different conceptual viewpoint. It is possible to select a treatment protocol that is intended to address the skeletal imbalance in a Class III mixed dentition patient. For example, Fränkel[42,104] recommends the function regulator (FR-3) appliance in patients whose malocclusion is characterized primarily by maxillary skeletal retrusion. On the other hand, the orthopedic chin cup[9] has been used in patients whose malocclusions are characterized primarily by mandibular prognathism, a procedure that has its greatest effect when used in primary and early mixed dentition patients.

The orthopedic FM that has been popularized by Delaire[105] and refined by Petit[106] is the appliance most widely used in the United States today. Each of these treatments has been shown to produce favorable effects in Class III patients, but the long-term outcomes have been variable. Also, there are substantial differences with regard to the speed of correction and in the regions of the craniofacial complex that are affected.

Appliance Selection

A basic axiom of orthodontic treatment is that the treatment approach should be designed to address the specific nature of the skeletal or dentoalveolar imbalance (or both). This axiom is illustrated by the selection of the specific surgical procedure or procedures used in the correction of a Class III malocclusion in an adolescent or adult patient. In patients with a Class II malocclusion, it also is demonstrated by the selective use of extraoral traction in the correction of maxillary prognathism and of FJO in the correction of mandibular retrusion. A seeming exception to this rule may be the interceptive treatment of the developing Class III malocclusion.

The Orthopedic Facial Mask

Of the three mixed dentition treatment strategies discussed earlier, the orthopedic FM (Fig. 16-47) has the widest application and produces the most dramatic results in the shortest period of time. Thus, the orthopedic FM is our customary appliance of choice for most Class III patients seen in the early mixed dentition or late deciduous dentition. The use of this single regimen in most early Class III patients seems arbitrary and paradoxical at first glance, given the various combinations of skeletal and dental components of Class III malocclusions in mixed dentition patients.[101,107] Because intervention using an orthopedic FM is undertaken at such an early age, however, the treatment effects produced by the FM ultimately are incorporated into the future craniofacial growth of the patient that occurs over a long period of time. Importantly, the appliance system affects virtually all areas contributing to a Class III malocclusion (e.g., maxillary skeletal retrusion, maxillary dentoalveolar retrusion, mandibular prognathism, decreased lower anterior facial height) by manipulating force vectors, and thus this treatment protocol can be applied effectively to most developing Class III patients regardless of the specific etiology of the malocclusion.

The orthopedic FM system has three basic components: the FM, a bonded maxillary splint, and elastics. The FM (see Fig. 16-47) is an extraoral device that has been modified by Petit[106] and now is available in various forms commercially. It can also be custom designed as discussed in Chapter 15. In essence, the FM is composed of a forehead pad and a chin pad that are connected by a heavy steel support rod. To this support rod is

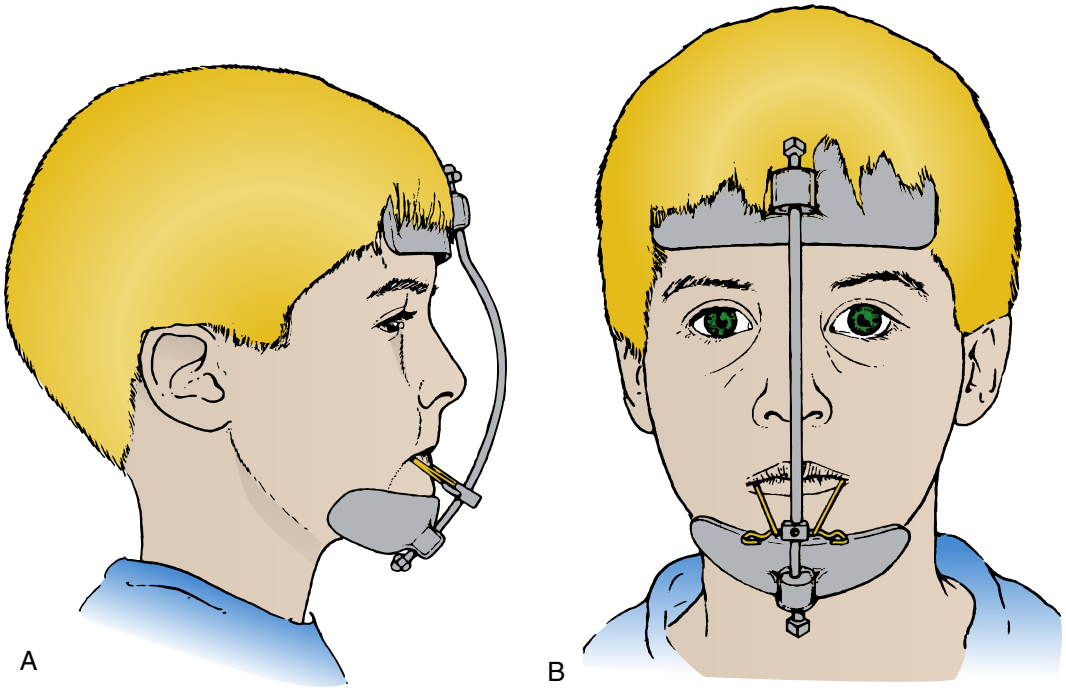


FIGURE 16-47 The orthopedic facial mask of Petit. A, Lateral view. B, Frontal view. This appliance, best used in patients in the early mixed dentition, is worn on a full-time basis for about 6 months, after which it can be worn on a night-time basis as a retention appliance. The elastics are connected to a bonded maxillary splint (see Fig. 16-18), to which have been attached hooks in the upper first deciduous molar region. The angle of elastic pull from the horizontal affects the amount of vertical versus horizontal movement. In overclosed patients, the downward angle is greater, but in patients with excessive vertical height, the pull is more parallel to the Frankfort horizontal plane. The most common is 15 to 20 degrees downward from the Frankfort horizontal plane.

connected a crossbow to which are attached rubber bands to produce a forward and downward elastic traction on the maxilla. The position of the pads and crossbow can be adjusted simply by loosening and tightening set screws within each part of the appliance.

Although Petit[106] has recommended a number of different intraoral devices, both fixed and removable, to which the elastics can be anchored, it is our strong preference to use a bonded maxillary expansion appliance that is similar in design to that discussed previously in the treatment of arch-length discrepancy problems. The major modification in the splint design is the addition of FM hooks in the region of the maxillary deciduous first molar (Fig. 16-48).

Even though the orthopedic FM has been available for more than 100 years, surprisingly few studies have dealt with the treatment effects produced by the FM. Until recently,[108-112] most published studies dealing with FM therapy have been anecdotal in nature.[113-115] It appears that the FM, especially when combined with a rigid maxillary anchorage unit (e.g., a bonded acrylic splint expander) that unlocks the occlusion, can produce one or more of the following treatment effects:

1. Correction of a discrepancy between centric occlusion and centric relation, a shift in occlusal relationship that is immediate and is associated with pseudo–Class III patients
2. Maxillary skeletal protraction, with 1 to 2 mm of forward movement of the maxilla often (but not always) observed
3. Forward movement of the maxillary dentition

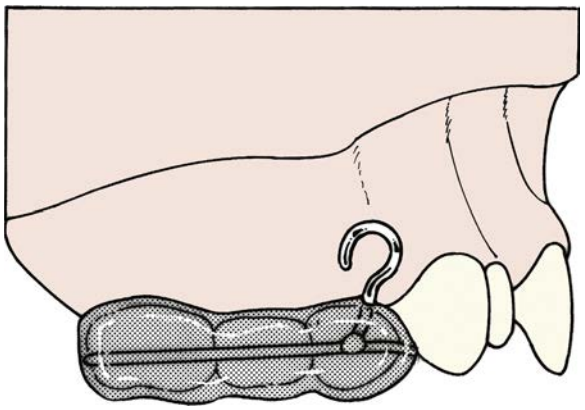


FIGURE 16-48 The bonded maxillary acrylic splint (lateral view). The hooks for the elastics usually are placed adjacent to the upper first deciduous molars. (Adapted from McNamara JA Jr, Brudon WL. *Orthodontics and dentofacial orthopedics*. Ann Arbor, MI: Needham Press; 2001.)

4. Lingual tipping of the lower incisors, particularly in patients with a preexisting anterior crossbite and labial incisor flare
5. Redirection of mandibular growth in a downward and backward direction, resulting in an increase in lower anterior facial height[112,116]

After the decision has been made to use an orthopedic FM, the first step of the appliance therapy is the fabrication and

bonding of the maxillary expander/splint (see McNamara and Brudon[1] concerning the technical details). The expander/splint is activated 0.25 mm once per day at bedtime until the desired increase in maxillary width has been achieved. In patients in whom no increase in transverse dimension is desired, the appliance still is activated for 8 to 10 days to disrupt the maxillary sutural system and to promote maxillary protraction.[1]

After the patient has become accustomed to wearing the maxillary splint, FM treatment is initiated. A sequence of elastics of increasing force (200, 350, 600 g per side) is used during the break-in period until a heavy orthopedic force is delivered to the maxillary complex. Ideally, the FM is worn on a full-time basis (≈20 hours per day) for 4 to 6 months, and then it can be worn on a nighttime basis only for an additional period of time. It usually is unwise to have the splint remain bonded in place for longer than 9 to 12 months because of the potential risk of leakage and subsequent decalcification of the underlying dentition.

The ideal stage of dental development during which to begin FM therapy is at the eruption of the permanent maxillary central incisors. Usually, the lower incisors already have erupted into occlusion. Achieving a positive horizontal and vertical overlap of the incisors during treatment is essential in providing an environment that will help maintain the achieved anteroposterior correction of the original Class III malocclusion. In patients with mild to moderate Class III problems, a positive overjet of 4 to 5 mm is achieved before the time that the FM is discontinued. It is anticipated that there will be some regression of the overjet relationship during the early posttreatment period. Every effort should be made, however, to maintain a positive overbite and overjet relationship throughout the retention period.

After the FM and the RME appliance have been removed, the patient can be retained using a number of appliances, including a simple maintenance plate (see Fig. 16-26), an FR-3 appliance of Fränkel, or a chin cup (or a combination of these). Because the FM usually is used in the early mixed dentition, a substantial amount of time may elapse before the final phase of fixed appliance treatment can be initiated. In some instances, multiple stages of orthopedic intervention may be required; thus, these patients must be monitored until all major facial growth is completed. It is very important to discuss with parents the potential need for long-term orthopedic or orthodontic management for Class III patients, especially those starting treatment in the primary or early mixed dentition. Additionally, the potential for future orthognathic surgery, if growth or treatment response proves adverse, should be reviewed.

The FR-3 Appliance of Fränkel

An intraoral appliance that has been used quite effectively in the treatment of Class III malocclusions in the mixed dentition is the function regulator FR-3 appliance of Fränkel.[42,104,117] Of all the Fränkel appliances, the FR-3 appliance (Fig. 16-49) perhaps is the easiest to manage clinically because there is no substantial postural change produced in the maxillomandibular relationship. As with all of Fränkel's appliances, the base of operation of the FR-3 appliance is the maxillary and mandibular vestibules. The appliance is designed to restrict the forces of the associated soft tissue on the maxillary complex, transmitting these forces through the appliance to the mandible.

A major advantage in using the FR-3 appliance of Fränkel is that it is relatively inconspicuous, especially compared with the orthopedic FM or chin cup. The FR-3 is worn intraorally, and often wearing the appliance actually improves the appearance of the patient by filling out the upper lip region in individuals with substantial maxillary skeletal retrusion. It also helps to reduce forward mandibular posture and overclosure. This appliance is worn easily by the patient. Interestingly, the treatment effects produced by the FR-3 appliance have been shown to be similar to those produced by the orthopedic FM.[42,118]

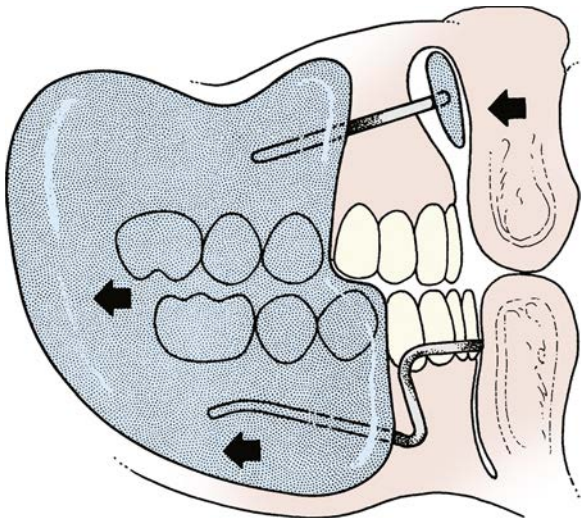


FIGURE 16-49 The FR-3 appliance of Fränkel. The vestibular shields and the upper labial pads shield the maxillary alveolus from the forces of the surrounding soft tissue. These forces are transmitted through the appliance to the mandible, providing a soft tissue generated distalizing force. (Adapted from McNamara JA Jr, Brudon WL. *Orthodontics and dentofacial orthopedics*. Ann Arbor, MI: Needham Press; 2001.)

A major difference between the FR-3 appliance and the orthopedic FM is the duration of treatment. In a routine Class III patient, the orthopedic FM may produce a correction of the malocclusion within the first 6 months after initiating treatment. Normally, 12 to 24 months is necessary to produce a similar response with the FR-3 appliance. It is obvious, however, that the FR-3 appliance has much more of an effect on the associated soft tissue, particularly on any existing hyperactivity in the muscles associated with the maxilla, than does the FM. This appliance was designed by Fränkel, based on the principles of Roux,[119] in that the primary action of the appliance is on the associated soft tissue, hopefully leading to a reprogramming of the central nervous system and a retraining of the craniofacial musculature.

When used as the primary appliance, the FR-3 is worn for about 20 hours per day, with the patients removing the appliance only during such activities as eating and playing contact sports. If the appliance is worn as a retainer after either full-time FR-3 therapy or FM therapy, it usually is worn only during the nighttime hours.

The Orthopedic Chin Cup

The oldest of the orthopedic approaches to the treatment of Class III malocclusion is the chin cup. The effects of this appliance have been investigated thoroughly,[9,120-123] with much of the research conducted on Asian populations because of the higher incidents of Class III malocclusion in these groups.

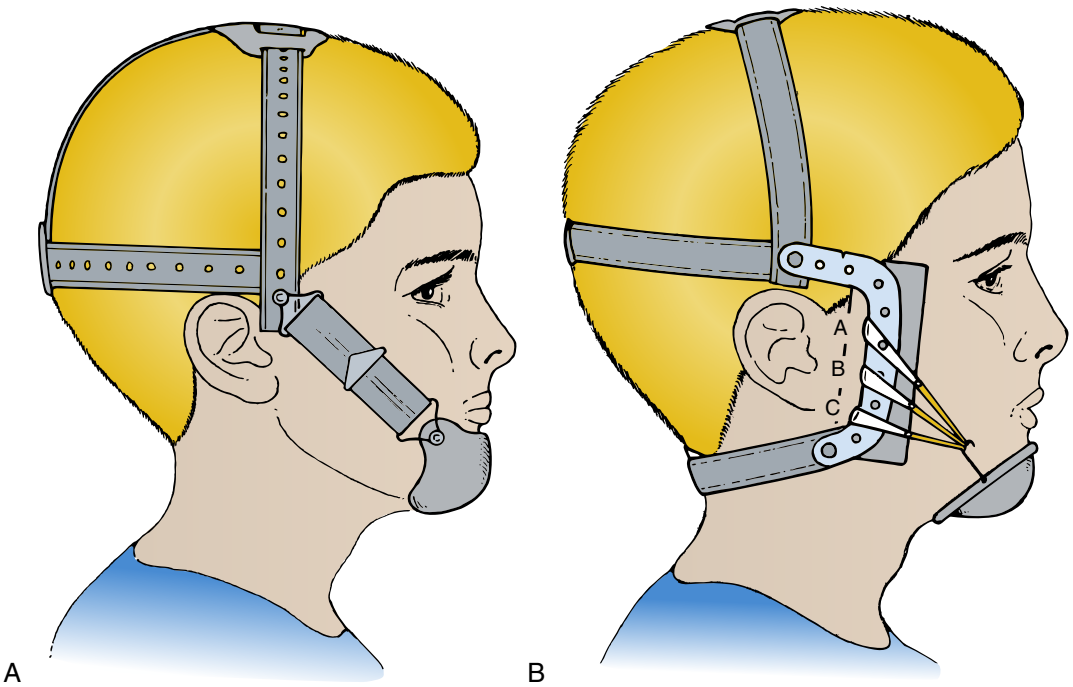


FIGURE 16-50 The occipital-pull chin cup. **A,** Soft elastic appliance with a soft chin cup. The direction of force is determined by the position of the head cap. Patients can use cloth baby diaper material cut in squares inside the cup to provide more comfort. **B,** Hickam-type headgear. Used as anchorage for a hard chin cup. The direction of pull can be adjusted according to the placement of the elastics (A–C). (Adapted from McNamara JA Jr, Brudon WL. *Orthodontics and dentofacial orthopedics*. Ann Arbor, MI: Needham Press; 2001.)

Although a wide variety of chin cup designs are available commercially, in general, these appliances can be divided into two types. The *occipital-pull chin cup* is used in instances of mandibular prognathism, and the *vertical-pull chin cup* (VPCC) is used in patients with steep mandibular plane angles and excessive lower anterior facial height.

The occipital-pull chin cup (Fig. 16-50, *A*) frequently is used in the treatment of Class III malocclusions. This type of chin cup is indicated for use in patients with mild to moderate mandibular prognathism. Success is greatest in patients in the primary and mixed dentition who can bring their incisors close to an edge-to-edge position when in centric relation. This treatment is useful particularly in patients who begin treatment with a short lower anterior facial height because this type of treatment can lead to an increase vertical facial height. If the pull of the chin cup is directed below the condyle, the force of the appliance may lead to a downward and backward rotation of the mandible. If no opening of the mandibular plane angle is desired, the force should be directed through the condyle to help restrict and redirect mandibular growth. The use of a Hickham-type headcap combined with a hard chin cup (Fig. 16-50, *B*) allows for variable vectors of force to be produced on the lower jaw.

If no increase in lower anterior facial height is desired, the VPCC can be used (Fig. 16-51, *A*). Pearson[124,125] has reported that the use of a VPCC can result in a decrease in the mandibular plane angle and the gonial angle and an increase in posterior facial height in comparison with the growth of untreated individuals. This type of extraoral traction can be used not only in individuals who have a Class III malocclusion but also for patients in whom an increase in the anterior vertical dimension is not desired. A study by Schulz and coworkers[126] that compared the VPCC combined with the bonded acrylic splint expander with the bonded expander used alone in high-angle patients indicated that a modest improvement can be obtained in the mandibular plane angle and in lower anterior facial height with the use of the VPCC. They noted, however, that the effect of the vertical orthopedic treatment was observed only during early phase I therapy, not during comprehensive fixed appliance treatment.

It is difficult to create a true vertical pull on the mandible because of the problems encountered in anchoring the appliance cranially. One of the easiest of the vertically directed chin cups to manipulate clinically is shown in Figure 16-51, *A*. A padded band extends coronally and is secured to the posterior part of the head by a cloth strap. A spring mechanism is activated by pulling the tab inferiorly and attaching the tab to a hook on the hard chin cup.

Another type of chin cup that produces a vertical direction of force is shown in Figure 16-51, *B*. This appliance incorporates a cloth headcap that curves around the crown of the head and is secured posteriorly with two horizontal straps. A throat strap also secures the appliances to the head of the patient. This particular design is useful in patients in whom anchorage in the cranial region is difficult to achieve. Either of these designs may be modified further with the construction of a custom chin cup that may be fabricated from acrylic. If customized, the attachment hook may be placed more to the posterior of the cup, closer to the throat angle, providing a more effective vertical direction of pull.

One of the substantive concerns regarding chin cup therapy is whether the growth of the mandible can be retarded through

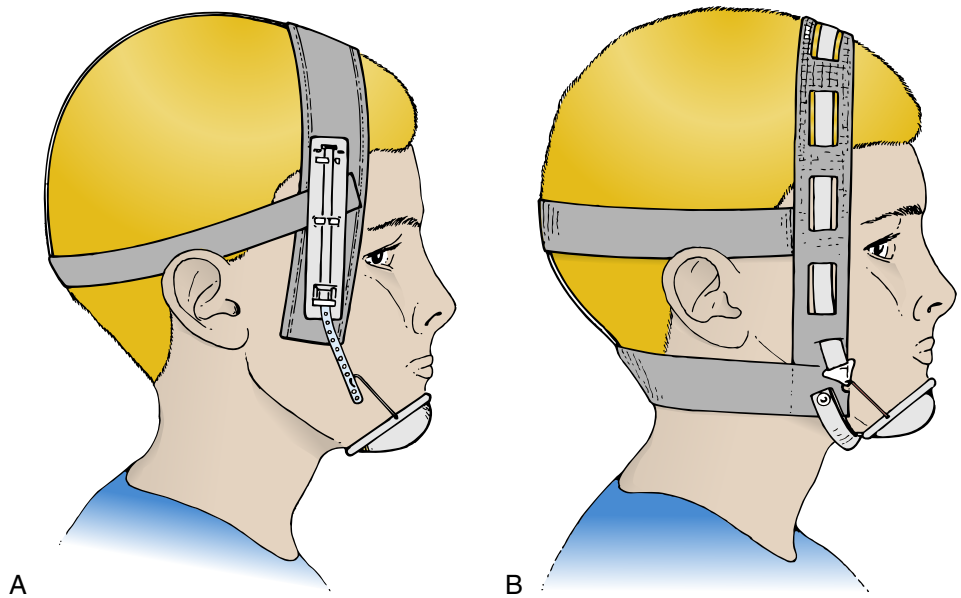


FIGURE 16-51 The vertical-pull chin cup. **A,** Unitek design. A spring force design is used to create a vertical direction of pull. **B,** Summit Orthodontics design. A cloth head cap curves around the crown of the head and is secured posteriorly with two horizontal straps. The force is produced by the stretch of the elastic material. In both of these examples, a hard chin cup is shown. (Adapted from McNamara JA Jr, Brudon WL. *Orthodontics and dentofacial orthopedics*. Ann Arbor, MI: Needham Press; 2001.)

wearing a chin cup. Sakamoto[127] and Wendell and coworkers[128] have noted decreases in mandibular growth during treatment. Wendell and associates, when examining a group of Class III patients treated in the mixed dentition, noted that the mandibular length increases in the treated group were only about two-thirds of those observed in the control group of mixed dentition individuals who received no treatment. Mitani and Fukazawa,[129] however, noted no differences in mandibular length in Class III individuals who began treatment during the adolescent growth period in comparison with control values. In addition, in a recent study of long-term adaptation to the chin cup, Sugawara and Mitani[123] noted that such treatment seldom alters the inherited prognathic characteristics of skeletal Class III profiles over the long term. Changes in the vertical direction of mandibular growth, however, have been noted. L.W. Graber[10] reported that, in a sample of young Class III patients with mandibular prognathism, the predominantly horizontal mandibular growth pattern was redirected more vertically, with changes noted in both the maxilla and mandible. The orthopedic chin cup usually produces an increase in lower anterior facial height while correcting the anteroposterior malrelationship.

It has been our observation that the chin cup works best when used in the primary and early mixed dentition and when the adverse mandibular growth has been mild to moderate in nature. The earlier the problem is addressed, the more successful treatment appears to be. Multiple "stages" of active chin cup home wear are often required to be successful in the case of moderate prognathism. Thus even the "corrected" patients need to be monitored at 4- to 6-month intervals until major growth has ceased. This need for follow-up treatment is to be expected for any orthopedic treatment that is redirecting excessive jaw growth or a severely deficient jaw growth pattern because of the genetic basis for growth and development. As noted earlier, parents must be apprised from the start of treatment that growth guidance may be needed in multiple stages and that the patient must be monitored throughout the growing years.

Bone-Anchored Maxillary Protraction. The most recent addition to the armamentarium of Class III treatment is the bone-anchored maxillary protraction (BAMP) therapy of De Clerck et al.[130] This approach involves the surgical placement of bone plates in the infrazygomatic region of the maxilla and the canine region of the mandible. Class III elastics that are attached to these surgical plates allow the forces produced to be transmitted to the bony bases (Fig. 16-52). De Clerck and coworkers[130-132] have shown dramatic results in young adolescent patients when Class III elastics (150-250 g) are worn full time, in that the force of the elastics is applied directly to the bones rather than on the teeth. Treatment changes are seen in the maxilla as well as the mandible, including remodeling in the temporomandibular joint. Interestingly, the effects of the BAMP therapy closely mimic findings from Class III nonhuman primate studies completed in our laboratory in the 1970s.

There are several limitations to this procedure, including patient age; De Clerck recommends using this approach in patients at least 10 to 11 years of age. The quality of bone is insufficient in younger patients to anchor the bone plates, especially in the maxilla. In addition, the lower permanent canines should be erupted before the lower bone plates are secured mesial to the canines.

Although this technique has an added surgical procedure (and related cost), there are biomechanical and patient management advantages in the technique that reduce the potential adverse effects of other treatment protocols. In addition, the appliance can be worn 22 to 23 hours per day without social liability to the patient. The timing of treatment is critical, balancing dental eruption and bone density maturation with the need to control the adverse Class III growth. Use of this technique at a later stage of growth, though, allows for decreased

FIGURE 16-52 Cone beam computed tomography scan of patient with Bollard plates placed surgically in the infrazygomatic region of the maxilla and the canine region of the mandible. Class III elastics (150–250 mg) are worn bilaterally full time for about 1 year depending on the severity and patient response. (Courtesy of Hugo De Clerck and Lucia Cevidanes.)

time for adverse catch-up Class III growth when treatment is completed.

Additional Comments Regarding Class III Treatment

As discussed previously, the appearance of Class III malocclusion is relatively easy to identify in young patients, yet the treatment of this occlusal problem is fraught with many difficulties. Fortunately, the level of patient cooperation in primary and young mixed dentition patients generally is excellent, and thus satisfactory compliance usually is achieved.

Of the four treatment modalities considered in this section, the orthopedic FM combined with a bonded maxillary splint seems most applicable in growing Class III patients. This type of appliance produces treatment effects in both skeletal and dentoalveolar aspects of the craniofacial complex. Given a young patient, the resolution of the underlying Class III relationship occurs relatively quickly (4 to 6 months), and then the mask can be worn for an additional period as a retainer at night before the bonded maxillary splint is removed.

The FR-3 appliance of Fränkel can be used either as a primary interceptive appliance or as a retainer. This treatment regimen makes the most biologic sense because the primary focus of this therapy is on the soft tissue, particularly the musculature, which in part may have been the etiology of the Class III relationships. The FR-3 appliance is less intrusive to the everyday life of the patient but may take two or three times as much treatment time to achieve correction of the malocclusion. The FR-3 appliance also can be used as a retention appliance after orthopedic FM or chin cup therapy.

Because mandibular growth exceeds maxillary growth during adolescence, early Class III correction may be lost during the teenage years. Indeed, orthopedic appliances may be required during the typical retention stage of treatment, particularly in males, in whom mandibular growth often continues well after fixed orthodontic appliances are removed. It cannot be overly stressed that at the onset of any interceptive treatment the patient and parents should be advised of the possibility of multiple stages of orthopedic intervention as well as the potential need for surgical correction. The wise clinician never makes guarantees regarding the treatment of Class III malocclusion because the outcome of any individual Class III patients is very difficult to estimate. We agree with T.M. Graber,[133] Sakamoto,[127] and Sugawara and coworkers,[9] who advocate the treatment of Class III malocclusion as early as is practical. Primary dentition chin cup treatment may be started to intercede in a developing Class III with subsequent maxillary protraction as the patient matures.

SUMMARY

An attempt has been made in this chapter to synthesize a coherent approach to orthodontic and orthopedic treatment, making available to the orthodontist a variety of early and late treatment protocols. Virtually all orthodontists are well versed in the management of adolescent and adult patients. Many orthodontists, however, are uncomfortable (and perhaps a bit skeptical, given the controversy) about mixed dentition treatment.

Although this latter topic has been addressed in many of the orthodontic texts since the beginning of the past century, mixed dentition treatment generally has been considered as secondary or peripheral to full banded or bonded appliance therapy in the adolescent or adult patient. By taking advantage of many of the newer technologies available, especially the bonded acrylic splint expander, the Herbst appliance, and the Pendex appliance, we have attempted to provide the reader with a conceptual framework on which the selection and timing of various treatment modalities can be based.

A few concluding comments should be made on the basis of our own clinical experiences. Some of these comments are obvious; some are not.

The timing of orthodontic and orthopedic treatment protocols varies with the underlying nature of the malocclusion. Some problems respond well to early intervention, others to late treatment. The selection of specific protocols and when to use them should be based on evidence derived from rigorous prospective and retrospective clinical studies.

Implicit in initiating early treatment is that the overall treatment time of the patients will be extended from the normal 12 to 24 months generally needed for comprehensive treatment of an adolescent patient. Initiating treatment in a patient with mixed dentition, however, does not imply that treatment will be provided continuously from the time of eruption of the permanent incisors until the time that the permanent second molars are aligned with fixed appliances. We have tried to structure our treatment protocols so that typically a concentrated period of early treatment is initiated, generally in the early mixed dentition. There are a defined beginning and ending of the treatment that are known to the patient and to the parents before the protocol is started.

Intermittent observation of the patient during the transition of the dentition is a prime component of early treatment. We generally prefer to see our patients every 4 to 6 months after the first phase of treatment is completed. The appliances used during this time are simple, usually consisting of only a

removable palatal plate typically without a labial wire that is worn full time for at least 1 year. Monitoring the patient on an intermittent basis allows the clinician to take advantage of the transition of the dentition, particularly in the second deciduous molar regions. It also allows observation of an adverse growth spurt and the opportunity to intercede as needed.

Passive holding arches (i.e., TPA, lingual arch) should be placed before the loss of the second deciduous molars in most early treatment patients. Not only will the leeway space be maintained (i.e., on average 5 mm in the mandible, 4 mm in the maxilla), but also maxillary molar rotation and uprighting can be achieved at the same time.

Almost all patients undergoing early treatment will require a final phase of fixed appliances. Usually the treatment time is reduced to 12 to 18 months because the majority of patients undergoing comprehensive therapy will be treated as nonextraction patients with Class I or near Class I molar relationships. Parents must be informed at the start and reminded at the end of mixed dentition treatment that a second stage of orthodontic treatment will be required after permanent teeth erupt.

Initiating early treatment does not imply that all patients treated in the mixed dentition will avoid the extraction of permanent teeth. It has been our experience that even in patients in whom orthopedic expansion protocols are initiated, the extraction of permanent teeth (usually premolars) is necessary in about 10% of these patients. In some instances, orthopedic expansion of the maxilla is initiated to broaden the smile in patients with severe maxillary constriction, and subsequently permanent teeth are extracted as part of the overall treatment protocol.

We have emphasized the importance of treatment timing in Class II malocclusion. In most Class II patients seen in the mixed dentition, we often start early treatment by managing the transverse dimension, with definitive Class II treatment rendered (if necessary) at the time of the circumpubertal growth period (the "spontaneous improvement" in Class II malocclusion is a frequently occurring phenomenon). Only in instances of a socially disabling malocclusion will definitive Class II intervention be undertaken by us in the early mixed dentition.

Early treatment will not eliminate the need for corrective jaw (orthognathic) surgery in all patients with severe skeletal and neuromuscular imbalances. FJO or maxillary distalization can be used to minimize substantially the sagittal maxillomandibular imbalance, but it may be impossible to eliminate this imbalance entirely without compromising the facial aesthetics of the individual. In these instances, orthognathic surgery in combination with fixed appliances is the treatment of choice. The need for orthognathic surgery also is obvious in patients with a Class III malocclusion characterized by significant skeletal imbalances, especially in those with a family history of significant Class III malocclusion. There may well be, however, an important psychological benefit for both child and parent in reducing a malocclusion and providing an "interim" aesthetic smile, knowing that orthognathic surgery for skeletal balance may or will be required after growth has been completed.

Patient compliance usually is excellent in patients treated in the mixed dentition, particularly if the appliance that is selected requires no or minimal patient cooperation other than that usually associated with a routine orthodontic treatment (e.g., good oral hygiene, diet control, and wearing of retainers). By initiating treatment in the mixed dentition, many of the skeletal and dentoalveolar problems associated with malocclusion often are eliminated or reduced substantially, thus lessening the need for prolonged fixed appliance therapy in the adolescent years.

In conclusion, we have attempted to provide an overview of various early and late treatment protocols that may be appropriate for certain orthodontic patients within a given practice. As with all such technologies, each of these protocols should be evaluated with healthy skepticism and should be initiated slowly until the parameters of success and failure are clearly established. The protocols outlined in this chapter have been used routinely by us for four decades and have proved to be satisfactory if approached within a framework of common sense and with a thorough understanding of comprehensive orthodontic biomechanics. Routine fixed appliance therapy is characterized by a series of "individual specific midcourse corrections," and our type of treatment is no different. Observation and diagnosis that will influence treatment are never static.

Finally, it must be stressed that early intervention is not always necessary or appropriate. In some instances, early treatment does not change appreciably the environment of dentofacial development and permanent tooth eruption. In such instances, early treatment may serve only to increase treatment time and cost and may result in a lack of patient cooperation in later years. If every effort is made, however, to time the treatment appropriately so as to maximize the treatment benefit in the shortest period of time and if the implemented treatment protocol has a reasonably predictable duration and outcome, orthodontic and orthopedic intervention can be provided successfully.

ACKNOWLEDGMENTS

The authors recognize the enormous contributions of Dr. Lorenzo Franchi and the late Dr. Tiziano Baccetti (1966–2011) of the University of Florence, whose collaborative work has been cited throughout this chapter. Their insights have influenced our approach to treatment timing through a series of evidence-based clinical studies that have been published jointly with us since the mid-1990s.

The authors thank our long-time friend and collaborator, the late William L. Brudon (1921–2009), associate professor emeritus in the School of Art and the Department of Medical and Biological Illustration in the School of Medicine at the University of Michigan, for producing almost all of the line drawings used in this chapter. The colorizing of the line drawings was provided by Chris Jung.

REFERENCES

1. McNamara JA Jr, Brudon WL. *Orthodontics and Dentofacial Orthopedics*. Ann Arbor, MI: Needham Press; 2001.
2. Freeman CS, McNamara Jr JA, Baccetti T, Franchi L. Treatment effects of the bionator and high-pull facebow combination followed by fixed appliances in patients with increased vertical dimension. *Am J Orthod Dentofacial Orthoped*. 2007;131:184–195.
3. Malta LA, Baccetti T, Franchi L, et al. Long-term dentoskeletal effects and facial profile effects induced by bionator therapy. *Angle Orthod*. 2010;80:10–17.
4. Franchi L, Pavoni Jr Faltin K, et al. Long-term dentoskeletal effects and treatment timing for functional appliances. *Angle Orthod*. 2013;83:334–340.
5. Faltin Jr K, Faltin RM, Baccetti T, et al. Long-term effectiveness and treatment timing for bionator therapy. *Angle Orthod*. 2003;73:221–230.

6. Angelieri F, Cevidanes LHS, Franchi L, et al. Long-term treatment effects of the FR-2 appliance: a prospective evaluation 7 years post-treatment. *Eur J Orthod*. 2014;36:192–199.
7. Pancherz H. Personal communication; 2001.
8. Wieslander L. Long-term effect of treatment with the headgear-Herbst appliance in the early mixed dentition. Stability or relapse? *Am J Orthod Dentofac Orthop*. 1993;104(4):319–329.
9. Sugawara J, Asano T, Endo N, Mitani H. Long-term effects of chincap therapy on skeletal profile in mandibular prognathism. *Am J Orthod Dentofacial Orthop*. 1990;98(2):127–133.
10. Graber LW. The alterability of mandibular growth. In: McNamara Jr JA, ed. *Determinants of Mandibular Form and Growth*. Ann Arbor, MI: Monograph 4, Craniofacial Growth Series, Center for Human Growth and Development, The University of Michigan; 1975.
11. Hellman M. The process of dentition and its effects on occlusion. *Dent Cosmos*. 1923;65:1329–1344.
12. Björk A, Helm S. Prediction of the age of maximum pubertal growth in body height. *Angle Orthod*. 1967;37(2):134–143.
13. Björk A. Variability and age changes in overjet and overbite. *Am J Orthod*. 1953;39:770–801.
14. Nanda RS. The rates of growth of several facial components measured from serial cephalometric roentgenograms. *Am J Orthod*. 1966;41: 658–673.
15. Greulich WW, Pyle SI. *Radiographic Atlas of Skeletal Development of the Hand and Wrist*. Stanford: Stanford University Press; 1959.
16. Hägg U, Taranger J. Menarche and voice changes as indicators of the pubertal growth spurt. *Acta Odontol Scand*. 1980;38:179–186.
17. Lamparski DG. Skeletal Age Assessment Utilizing Cervical Vertebrae. Pittsburgh. Unpublished master's thesis, Department of Orthodontics, The University of Pittsburgh; 1972.
18. Hassel B, Farman AG. Skeletal maturation evaluation using cervical vertebrae [published erratum appears in *Am J Orthod Dentofacial Orthop* 1995;107(6):19a]. *Am J Orthod Dentofacial Orthop*. 1995;107(1):58–66.
19. Lamparski DG, Nanda SK. *Skeletal Age Assessment Utilizing Cervical Vertebrae*. In: McNamara Jr JA, Kelly KA, eds. *Treatment Timing: Orthodontics in Four Dimensions*. Ann Arbor, MI: Monograph 39, Craniofacial Growth Series, Department of Orthodontics and Pediatric Dentistry and Center for Human Growth and Development, The University of Michigan; 2002.
20. Franchi L, Baccetti T, McNamara Jr JA. Mandibular growth as related to cervical vertebral maturation and body height. *Am J Orthod Dentofac Orthop*. 2000;118:335–340.
21. Baccetti T, Franchi L, McNamara Jr JA. The cervical vertebral maturation (CVM) method for the assessment of optimal treatment timing in dentofacial orthopedics. *Semin Orthod*. 2005;11:119–129.
22. Franchi L, Baccetti T, McNamara Jr JA. Post-pubertal assessment of treatment timing for maxillary expansion and protraction therapy followed by fixed appliances. *Am J Orthod Dentofacial Orthoped*. 2004;126:555–568.
23. Petrovic A, Stutzmann JJ, Oudet C. Control process in the postnatal growth of the condylar cartilage. In: McNamara Jr JA, ed. Determinants of *Mandibular Form and Growth*. Ann Arbor, MI: Monograph 4, Craniofacial Growth Series, Center for Human Growth and Development, The University of Michigan; 1975.
24. McNamara Jr JA, Bookstein FL, Shaughnessy TG. Skeletal and dental changes following functional regulator therapy on Class II patients. *Am J Orthod*. 1985;88(2):91–110.
25. Baccetti T, Franchi L, Toth LR, McNamara Jr JA. Treatment timing for twin block therapy. *Am J Orthod Dentofac Orthop*. 2000;118:159–170.
26. Franchi L, Contardo L, Primožič J, Perinetti G. Clinical alteration of mandibular growth: what we know after 40 years. In: McNamara Jr JA, ed. *The 40th Moyers Symposium: Looking Forward ... Looking Back*. Ann Arbor, MI: Monograph 50, Craniofacial Growth Series, Department of Orthodontics and Pediatric Dentistry and Center for Human Growth and Development, The University of Michigan; 2014.
27. Kloehn SJ. Orthodontics—force or persuasion. *Angle Orthod*. 1953;23:56–65.
28. Graber TM. Extra-oral force—facts and fallacies. *Am J Orthod*. 1955;41: 490–505.
29. Moyers RE, van der Linden FPGM, Riolo ML, McNamara Jr JA. Standards of human occlusal development. Ann Arbor, MI: Monograph 5, Craniofacial Growth Series, Center for Human Growth and Development, The University of Michigan; 1976.
30. Gianelly AA. Crowding: timing of treatment. *Angle Orthod*. 1994;64(6):415–418.
31. Zablocki HL, McNamara Jr JA, Baccetti T, Franchi L. The effect of the transpalatal arch during extraction treatment. *Am J Orthod Dentofacial Orthoped*. 2008;133:852–860.
32. Dewel BF. Serial extractions in orthodontics: indications, objections, and treatment procedures. *Int J Orthod*. 1954;40:906–926.
33. Dewel BF. Serial extraction: its limitations and contraindications in orthodontic treatment. *Am J Orthod*. 1967;53(12):904–921.
34. Graber TM. *Orthodontics: Principles and Practices*. Philadelphia: WB Saunders; 1966.
35. Dale JG, Dale HC. Interceptive guidance of the occlusion with emphasis on diagnosis. In: Graber LW, Vanarsdall Jr RL, Vig KL, eds. *Orthodontics: Current Principles and Techniques*. 5th ed. St. Louis: C.V. Mosby; 2011.
36. Proffit WR, Fields HW, Sarver DM. *Contemporary Orthodontics*. 5th ed. St. Louis: C.V. Mosby; 2013.
37. Ringenberg QM. Serial extraction: stop, look, and be certain. *Am J Orthod*. 1964;50:327–336.
38. Merrifield LL, Cross JJ. Directional forces. *Am J Orthod*. 1970;57(5): 435–464.
39. Sung JH, Kyung HM, Bae SM, et al. *Microimplants in Orthodontics*. Daegu: South Korea Dentos; 2006.
40. Little RM, Wallen TR, Riedel RA. Stability and relapse of mandibular anterior alignment-first premolar extraction cases treated by traditional edgewise orthodontics. *Am J Orthod*. 1981;80(4):349–365.
41. Little RM. Stability and relapse of dental arch alignment. *Br J Orthod*. 1990;17(3):235–241.
42. Fränkel R, Fränkel C. *Orofacial Orthopedics with the Function Regulator*. Munich: S. Karger; 1989.
43. Brieden CM, Pangrazio-Kulbersh V, Kulbersh R. Maxillary skeletal and dental change with Fränkel appliance therapy—an implant study. *Angle Orthod*. 1984;54(3):226–232.
44. Bjerregaard J, Bundgaard AM, Melsen B. The effect of the mandibular lip bumper and maxillary bite plate on tooth movement, occlusion and space conditions in the lower dental arch. *Eur J Orthod*. 1980;2(4): 257–265.
45. Haas AJ. Long-term posttreatment evaluation of rapid palatal expansion. *Angle Orthod*. 1980;50(3):189–217.
46. Wertz RA. Skeletal and dental changes accompanying rapid midpalatal suture opening. *Am J Orthod*. 1970;58(1):41–66.
47. Haas AJ. Rapid expansion of the maxillary dental arch and nasal cavity by opening the mid-palatal suture. *Angle Orthod*. 1961;31:73–90.
48. Starnbach H, Bayne D, Cleall J, Subtelny JD. Facioskeletal and dental changes resulting from rapid maxillary expansion. *Angle Orthod*. 1966;36(2):152–164.
49. Howe RP, McNamara Jr JA, O'Connor KA. An examination of dental crowding and its relationship to tooth size and arch dimension. *Am J Orthod*. 1983;83(5):363–373.
50. Spillane LM, McNamara Jr JA. Arch width development relative to initial transpalatal width. *J Dent Res, IADR Abstracts*. 1989;(1538):374.
51. Melsen B. Palatal growth studied on human autopsy material. A histologic microradiographic study. *Am J Orthod*. 1975;68(1):42–54.
52. Melsen B, Melsen F. The postnatal development of the palatomaxillary region studied on human autopsy material. *Am J Orthod*. 1982;82(4):329–342.
53. Wendling LK, McNamara Jr JA, Franchi L, Baccetti T. Short-term skeletal and dental effects of the acrylic splint rapid maxillary expansion appliance. *Angle Orthod*. 2005;75:7–14.
54. Wertz RA. Changes in nasal airflow incident to rapid maxillary expansion. *Angle Orthod*. 1968;38(1):1–11.
55. Fränkel R. Guidance of eruption without extraction. *Trans Europ Orthod Soc*. 1971;47:303–315.
56. Fränkel R. Decrowding during eruption under the screening influence of vestibular shields. *Am J Orthod*. 1974;65(4):372–406.

57. McDougall PD, McNamara Jr JA, Dierkes JM. Arch width development in Class II patients treated with the Fränkel appliance. *Am J Orthod*. 1982;82(1):10–22.
58. Brust EW, McNamara Jr JA. Arch dimensional changes concurrent with expansion in mixed dentition patients. In: Trotman CA, McNamara Jr JA, eds. *Orthodontic Treatment: Outcome and Effectiveness*. Ann Arbor, MI: Monograph 30, Craniofacial Growth Series, Center for Human Growth and Development, The University of Michigan; 1995.
59. O'Grady PW, McNamara Jr JA, Baccetti T, Franchi L. A long-term evaluation of the mandibular Schwarz appliance and the acrylic splint expander in early mixed dentition patients. *Am J Orthod Dentofacial Orthop*. 2006;130:202–213.
60. Cetlin NM, Ten Hoeve A. Nonextraction treatment. *J Clin Orthod*. 1983;17(6):396–413.
61. Guest SS, McNamara Jr JA, Baccetti T, Franchi L. Improving Class II malocclusion as a side-effect of rapid maxillary expansion: a prospective clinical study. *Am J Orthod Dentofacial Orthop*. 2010;138:582–591.
62. Haas AJ. The treatment of maxillary deficiency by opening the mid-palatal suture. *Angle Orthod*. 1965;35:200–217.
63. Dellinger EL. A preliminary study of anterior maxillary displacement. *Am J Orthod*. 1973;63(5):509–516.
64. Wylie WL. The assessment of anteroposterior dysplasia. *Angle Orthod*. 1947;17:97–109.
65. Moyers RE, Riolo ML, Guire KE, et al. Differential diagnosis of Class II malocclusions. Part 1. Facial types associated with Class II malocclusions. *Am J Orthod*. 1980;78(5):477–494.
66. Blair ES. A cephalometric roentgenographic appraisal of the skeletal morphology of Class I, Class II, division 1 and Class II, division 2 (angle) malocclusion. *Angle Orthod*. 1954;24:106–119.
67. McNamara Jr JA. Components of Class II malocclusion in children 8-10 years of age. *Angle Orthod*. 1981;51(3):177–202.
68. Hilgers JJ. The pendulum appliance: an update. *Clin Impressions*. 1993:15–17.
69. Bussick TJ, McNamara Jr JA. Dentoalveolar and skeletal changes associated with the pendulum appliance. *Am J Orthod Dentofacial Orthop*. 2000;117:333–343.
70. Burkhardt DR, McNamara Jr JA, Baccetti T. Maxillary molar distalization or mandibular enhancement: a cephalometric comparison of the pendulum and Herbst appliances. *Am J Orthod Dentofacial Orthop*. 2003;123:108–116.
71. Carano A, Testa M. The distal jet for upper molar distalization. *J Clin Orthod*. 1996;30:374–380.
72. Bowman SJ. Modifications of the distal jet. *J Clin Orthod*. 1998;32: 549–556.
73. Kloehn SJ. Guiding alveolar growth and eruption of teeth to reduce treatment time and produce a more balanced denture and face. *Angle Orthod*. 1947;17:10–23.
74. McNamara Jr JA, Peterson Jr JE, Alexander RG. Three-dimensional diagnosis and management of Class II malocclusion in the mixed dentition. *Semin Orthod*. 1996;2:114–137.
75. Sinclair PM. The reader's corner. *J Clin Orthod*. 1994;28:361–363.
76. Hilgers JJ. The pendulum appliance for Class II non-compliance therapy. *J Clin Orthod*. 1992;26(11):706–714.
77. Byloff FK, Darendeliler MA, Clar E, Darendeliler A. Distal molar movement using the pendulum appliance. Part 2: the effects of maxillary molar root uprighting bends. *Angle Orthod*. 1997;67(4):261–270.
78. Byloff FK, Darendeliler MA. Distal molar movement using the pendulum appliance. Part 1: clinical and radiological evaluation. *Angle Orthodontist*. 1997;67(4):249–260.
79. Kingsley NW. *A Treatise on Oral Deformities as a Branch of Mechanical Surgery*. New York: D. Appleton; 1880.
80. Andresen V. The Norwegian system of gnatho-orthopedics. *Acta Gnathol*. 1936;1:4–36.
81. Balters W. Die technik und übung der allgemeinen und speziellen bionator-therapie. *Quintessenz*. 1964;1:77.
82. Pancherz H. Treatment of Class II malocclusions by jumping the bite with the Herbst appliance. A cephalometric investigation. *Am J Orthod*. 1979;76(4):423–442.
83. Pangrazio-Kulbersh V, Berger JL, Chermak DS, et al. Treatment effects of the mandibular anterior repositioning appliance on patients with Class II malocclusion. *Am J Orthod Dentofacial Orthoped*. 2003;123:286–295.
84. Clark WJ. *Twin Block Functional Therapy*. 3rd ed. New Delhi: Jaypee Bros Medical Publishers; 2015.
85. Freeman DC, McNamara Jr JA, Baccetti T, Franchi L. Long-term treatment effects of the FR-2 appliance of Fränkel. *Am J Orthod Dentofacial Orthoped*. 2008;133:513–524.
86. Herbst E. *Atlas und grundriss der zahnärztlichen orthopädie*. Munich: JF Lehmann Verlag; 1910.
87. Rogers MB. The banded Herbst appliance. *J Clin Orthod*. 2001;35: 494–499.
88. Dischinger TG. Edgewise bioprogressive Herbst appliance. *J Clin Orthod*. 1989;23(9):608–617.
89. Smith JR. Matching the Herbst to the malocclusion. *Clin Impressions*. 1998;7:6–12, 20–23.
90. Mayes JH. The single appointment preattached cantilever bite jumper. *Clin Impressions*. 1996;5:14–17.
91. Howe RP, McNamara Jr JA. Clinical management of the bonded Herbst appliance. *J Clin Orthod*. 1983;17(7):456–463.
92. McNamara Jr JA, Howe RP. Clinical management of the acrylic splint Herbst appliance. *Am J Orthod Dentofacial Orthop*. 1988;94(2):142–149.
93. McNamara Jr JA, Howe RP, Dischinger TG. A comparison of the Herbst and Fränkel appliances in the treatment of Class II malocclusion. *Am J Orthod Dentofacial Orthop*. 1990;98(2):134–144.
94. Pancherz H. The Herbst appliance—its biologic effects and clinical use. *Am J Orthod*. 1985;87(1):1–20.
95. Lai M, McNamara Jr JA. An evaluation of two-phase treatment with the Herbst appliance and preadjusted edgewise therapy. *Semin Orthod*. 1998;4:46–58.
96. Pancherz H. Dentofacial orthopedics in relation to somatic maturation, an analysis of 70 consecutive cases treated with the Herbst appliance. *Am J Orthod*. 1988;88:273–287.
97. Pancherz H. Vertical dentofacial changes during Herbst appliance treatment: a cephalometric investigation. *Swed Dent J Supp*. 1982;15: 189–196.
98. Franchi L, Baccetti T. New emphasis on the role of mandibular skeletal maturity in dentofacial orthopedics. In: McNamara Jr JA, ed. *The Enigma of the Vertical Dimension*. Ann Arbor, MI: Monograph 36, Craniofacial Growth Series, Center for Human Growth and Development, The University of Michigan; 2000.
99. Proffit WR, Fields HW, Moray LJ. Prevalence of malocclusion and orthodontic treatment need in the United States: estimates from the N-HANES III survey. *Int J Adult Orthod Orthogn Surg*. 1998;13:97–106.
100. McNamara Jr JA, McClatchey LM. Unpublished Data; 2015.
101. Guyer EC, Ellis E, McNamara Jr JA, Behrents RG. Components of Class III malocclusion in juveniles and adolescents. *Angle Orthod*. 1986;56(1):7–30.
102. Jacobson A, Evans WG, Preston CB, Sadowsky PL. Mandibular prognathism. *Am J Orthod*. 1974;66(2):140–171.
103. Graber LW. *The Psychological Implications of Malocclusion*. Ann Arbor: Unpublished doctoral thesis, Center for Human Growth and Development, The University of Michigan; 1980.
104. Fränkel R. Maxillary retrusion in Class III and treatment with the Function Corrector III. *Trans Eur Orthod Soc*. 1970:249–259.
105. Delaire J. Confection du masque orthopedique. *Rev Stomat Paris*. 1971;72:579–584.
106. Petit HP. Adaptation following accelerated facial mask therapy. In: McNamara Jr JA, Ribbens KA, Howe RP, eds. *Clinical Alterations of the Growing Face*. Ann Arbor, MI: Monograph 14, Craniofacial Growth Series, Center for Human Growth and Development, The University of Michigan; 1983.
107. Reyes BC, Baccetti T, McNamara Jr JA. An estimate of craniofacial growth in Class III malocclusion. *Angle Orthod*. 2006;78(de c):577–584.
108. Williams MD, Sarver DM, Sadowsky PL, Bradley E. Combined rapid maxillary expansion and protraction facemask in the treatment of Class III malocclusion in growing children: a prospective study. *Semin Orthod*. 1997;3:265–274.

109. Ngan P, Hagg U, Yiu C, Wei H. Treatment response and long-term dentofacial adaptations to maxillary expansion and protraction. *Semin Orthod*. 1997;3:255–264.
110. Baccetti T, McGill JS, Franchi L, et al. Skeletal effects of early treatment of Class III malocclusion with maxillary expansion and face-mask therapy. *Am J Orthod Dentofac Orthop*. 1998;113:333–343.
111. McGill JS, McNamara Jr JA. Treatment and post-treatment effects of rapid maxillary expansion and facial mask therapy. In: McNamara Jr JA, ed. *Growth Modification: What Works, What Doesn't and Why*. Ann Arbor, MI: Monograph 36, Craniofacial Growth Series, Center for Human Growth and Development, University of Michigan; 1999.
112. Westwood PV, McNamara Jr JA, Baccetti T, et al. Long-term effects of early Class III treatment with rapid maxillary expansion and facial mask therapy. *Am J Orthod Dentofacial Orthop*. 2003;123:306–320.
113. McNamara Jr JA. An orthopedic approach to the treatment of Class III malocclusion in young patients. *J Clin Orthod*. 1987;21(9):598–608.
114. Roberts CA, Subtelny JD. Use of the face mask in the treatment of maxillary skeletal retrusion. *Am J Orthod Dentofac Orthop*. 1988;93:388–394.
115. Turley PK. Orthopedic correction of Class III malocclusion with palatal expansion and custom protraction headgear. *J Clin Orthod*. 1988;22(5):314–325.
116. McGill JS. Orthopedic Alterations Induced by Rapid Maxillary Expansion and Face Mask Therapy. Ann Arbor: Unpublished master's thesis, Department of Orthodontics and Pediatric Dentistry, University of Michigan; 1995.
117. McNamara Jr JA, Huge SA. The Functional Regulator (FR-3) of Fränkel. *Am J Orthod*. 1985;88(5):409–424.
118. Kerr WJ, TenHave TR, McNamara Jr JA. A comparison of skeletal and dental changes produced by Function Regulators (FR-2 and FR-3). *Eur J Orthod*. 1989;11(3):235–242.
119. Roux W. *Entwicklungsmechanik der organismen, bd* I und ii. Leipzig: W. Engelmann Verlag W; 1895.
120. Thilander B. Treatment of Angle Class III malocclusion with chin cap. *Trans Eur Orthod Soc*. 1963;39:384–398.
121. Graber LW. Chin cup therapy for mandibular prognathism. *Am J Orthod*. 1977;72(1):23–41.
122. Graber TM, Chung DD, Aoba JT. Dentofacial orthopedics versus orthodontics. *J Am Dent Assoc*. 1967;75(5):1145–1166.
123. Sugawara J, Mitani H. Facial growth of skeletal Class III malocclusions and the effects, limitations, and long-term dentofacial adaptations to chincup therapy. *Semin Orthod*. 1997;3:244–254.
124. Pearson LE. Vertical control in treatment of patients having backward-rotational growth tendencies. *Angle Orthod*. 1978;48(2):132–140.
125. Pearson LE. The management of vertical problems in growing patients. In: McNamara Jr JA, ed. The enigma of the vertical dimension. Ann Arbor, MI: Monograph 36, Craniofacial Growth Series, Center for Human Growth and Development, The University of Michigan; 2000.
126. Schulz SO, McNamara Jr JA, Baccetti T, Franchi L. Treatment effects of bonded RME and vertical pull chin cup followed by fixed appliances in patients with increased vertical dimension. *Am J Orthod Dentofacial Orthop*. 2005;128:326–336.
127. Sakamoto T. Effective timing for the application of orthopedic force in the skeletal Class III malocclusion. *Am J Orthod*. 1981;80(4):411–416.
128. Wendell PD, Nanda R, Sakamoto T, Nakamura S. The effects of chin cup therapy on the mandible: a longitudinal study. *Am J Orthod*. 1985;87(4):265–274.
129. Mitani H, Fukazawa H. Effects of chincap force on the timing and amount of mandibular growth associated with anterior reversed occlusion (Class III malocclusion) during puberty. *Am J Orthod Dentofacial Orthop*. 1986;90(6):454–463.
130. De Clerck HJ, Cornelis MA, Cevidanes LHS, et al. Orthopedic traction of the maxilla with miniplates: a new perspective for treatment of midface deficiency. *J Oral Maxillofac Surg*. 2009;67:2123–2129.
131. De Clerck H, Cevidanes LHS, Baccetti T. Dentofacial effects of bone-anchored maxillary protraction: a controlled study of consecutively treated Class III patients. *Am J Orthod Dentofacial Orthop*. 2010;138(5):577–581.
132. De Clerck H, Timmerman H. Orthopedic changes by bone-anchored intermaxillary elastic traction in Class III growing patients. In: McNamara Jr JA, ed. *The 40th Moyers Symposium: Looking Forward ... Looking Back*. Ann Arbor, MI: Monograph 50, Craniofacial Growth Series, Department of Orthodontics and Pediatric Dentistry and Center for Human Growth and Development, The University of Michigan; 2014:249–261.
133. Graber TM. Extrinsic control factors influencing craniofacial growth. In: McNamara Jr JA, ed. *Determinants of Mandibular Form and Growth*. Ann Arbor, MI: Monograph 4, Craniofacial Growth Series, Center for Human Growth and Development, The University of Michigan; 1976.

17

Contemporary Straight Wire Biomechanics

Antonino G. Secchi and Jorge Ayala Puente

OUTLINE

Straight Wire Appliance Design and Values, 437
Straight Wire Appliance and Self-Ligation, 438
Optimal Bracket Placement, 438
Treatment Mechanics, 439
Stage 1: Leveling and Aligning, 439
Stage 2: Working Stage, 440
Stage 3: Finishing Stage, 445
Future Directions, 445
Summary, 454

It is not only the appliance system you have, but how you use it."

The straight wire appliance (SWA) was developed and introduced by Lawrence Andrews in 1970[1] with the idea of having an orthodontic fixed appliance that would enable the orthodontist to achieve the "six keys" of normal occlusion[2] in the vast majority of cases in an efficient and reliable fashion.

Even though the SWA is 40 years old and has become the most common appliance concept over the past three decades, a review of some of the original concepts on which the SWA was designed and the evolution it has gone through are fundamental to better understand the beauty of this appliance and the treatment mechanics, which we then discuss.

STRAIGHT WIRE APPLIANCE DESIGN AND VALUES

There are a few features that need to be present in an appliance to be considered a true SWA.[3] First, each bracket has to be tooth specific and have built-in torque, tip, in/out, and (for the molars) proper offset. Second, the torque has to be built in the base of the bracket, not in the face, and the tip in the face of the slot. These prerequisites are very important in order to achieve proper alignment of the center of the slot, the center of the base, and the reference point (middle of the clinical crown occlusogingivally along the facial long axis of the crown) for all teeth at the completion of treatment. This is the only way that the desired built-in features can be properly transferred from the bracket to the tooth. Third, the base of the bracket must be contoured mesiodistally and occlusogingivally. This has been referred to as a "compound contour" base, and it allows the bracket to firmly adapt to the convexities of the labial surface of each tooth, helping the orthodontist to achieve an optimal bracket placement.

Although Andrews thought his appliance could be used to treat a large variety of cases, he introduced a series of additional brackets with different degrees of overcorrection to account for undesired tooth movement that would occur specifically when sliding teeth in extraction cases. For example, if a maxillary canine had to be moved distally, because Andrews uses round stainless steel wires to slide teeth through, the canine most likely would tip and rotate distally. Therefore, he introduced more mesial tip and rotation to the canine bracket. Andrews then came out with a line of overcorrected brackets, which he first called *extraction brackets*[3] and then *translation brackets*.[4] Andrews' complete bracket system (standard and translation brackets) was less popular than expected, partly because of the large bracket inventory needed to satisfy his treatment mechanics. However, in the mid-1970s, Ronald H. Roth took the Andrews SWA and combined some of the standard bracket prescription values with some of the overcorrected values found in the translation bracket prescription to create the "Roth setup."[5] The Roth setup became the most popular SWA prescription in the world. Roth realized that because the size of the brackets at that time caused bracket interferences, it was virtually impossible to place each tooth in its final ideal position. Also, he observed that when appliances were removed, teeth would rebound and settle. Therefore, he slightly overcorrected some of the original Andrews values to allow teeth to properly settle in the ideal final position after removal of the appliances. After the Roth prescription, a great number of clinicians came out with small variations to either the Andrews prescription or the Roth prescription. Most of these changes were done for a commercial purpose, to compensate for unknown errors in bracket position, or to suit a particular orthodontist's type of mechanics.

Today, there are a large number of preadjusted appliances wrongly called SWA only because they have built-in torque, tip, and in/out. However, if they are not manufactured with the features specified earlier, the appliance will not transfer the built-in information correctly to the teeth. Therefore, selecting the proper appliance is paramount when using an SWA.

STRAIGHT WIRE APPLIANCE AND SELF-LIGATION

Both of the authors of this chapter use a self-ligating bracket (SLB) system, and, consequently, some of the concepts on mechanics that will be reviewed later take advantage of such appliance systems. Therefore, even though self-ligation is the subject of Chapter 17 in this book and thus is extensively reviewed, it is important to point out a few aspects of this type of appliance that will allow the reader to better understand the information provided in this chapter.

Self-ligating brackets have been classified as "active" or "passive" depending on the behavior of the gate or clip on the archwire. Active SLBs have a clip with a spring effect that exerts pressure on the archwire, pushing it onto the base of the bracket's slot. This pressure is based on the archwire size and/or bracket and archwire configuration (Fig. 17-1). On the other hand, passive SLBs have a gate that passively opens and closes without exerting pressure on the archwire. Passive SLBs also have been described as tubes.[6] Today it is known that active SLBs have some important advantages over passive SLBs. In active SLBs, as stated earlier, one can manage the amount of activity that the clip will have by the size of the archwire. For instance, at the beginning of treatment when less resistance to sliding is desirable and usually the archwire of choice is a small, round thermal-activated wire, both active and passive brackets have shown equal behavior.[7] As treatment progresses, an increased resistance to sliding is desired to achieve proper torque expression. At this stage of treatment, passive SLBs have demonstrated poor behavior compared with active SLBs.[8,9] It is important to remember that the active clip is a very important feature of active SLBs, and therefore the quality of it will determine to some extent how good is the bracket. Substantial differences have been shown in clip performance when some SLBs' clips lose an important percentage of force during treatment. Also, it is important to note that not every active available SLB is a true SWA. The SLB must have all the other features mentioned earlier to be a true SWA.

Optimal Bracket Placement

Assuming we have the right appliance, the next most important factor when working with an SWA is bracket position. This is where the orthodontist's dexterity is of great value. As with techniques that require bending the wire, the quality and precision of each bend will determine to some extent the quality of the final result, as the precision of bracket placement will do it when using an SWA. When using an SWA, you "start finishing" your case the day you place the brackets. This is why an important percentage of problems that orthodontists experience toward the end of active treatment—such as marginal ridge discrepancies, difficulty correcting rotations, lack of root parallelism, and, ultimately, less than ideal tooth position—are caused by incorrect bracket placement. As Andrews described 40 years ago, the brackets should be placed at the FA point. The FA point is the middle of the clinical crown occlusogingivally and mesiodistally, following the long axis of the crown, for each tooth in the mouth (Fig. 17-2).

Because all the brackets are working at the same time through the wire, one misplaced bracket will automatically affect the adjacent brackets. If more than one bracket is misplaced, the problem will increase and become more noticeable as the wire sequence progresses. This issue, if not corrected, can prevent the orthodontist from finishing the case in an optimal and efficient way.

Because we have a limited space in this chapter, we will not describe the specific bracket position for each individual tooth but rather focus on the teeth that usually cause more problems for clinicians. It is important to emphasize the following concepts:

- Andrews demonstrated that trained clinicians are able to place brackets consistently at the FA point without any aids but their own eyes.
- The use of any gauge as an aid to position the brackets is not necessary; in fact, to use any predetermined height from the incisal edge to locate the brackets, as some orthodontists advocate, is wrong and negates the use of the FA point, which is one of the fundamentals of the proper management of the SWA. However, you have to take into account shorter crowns caused by excessive gingival tissue, worn teeth, or fractured teeth that eventually will be restored, so in some of these situations, the brackets will look more incisally or gingivally than they should be.
- To fully level and express the torque, tip, and in/out of each bracket, the slot of the bracket has to be filled, which requires 0.021- × 0.025-inch stainless steel wire. It is important to know that a 0.019- × 0.025-inch stainless steel wire has about 10.5 degrees of play on a 0.022-inch bracket slot.[10] However, this is not the case when using active SLBs, as explained later in the chapter.

FIGURE 17-1 The design features that make a self-ligating bracket active. **A,** Notice the difference in the depth of the occlusal wall of the slot compared with the gingival wall. **B,** This particular active self-ligating bracket (In-Ovation R) wire is smaller than 0.019 inch; the clip is not active. **C,** A wire larger than 0.019 inch pushes out the clip and therefore activates it.

Although the FA point and long axis of clinical crowns are key to bracket position, a few specific considerations facilitate bracket placement on certain teeth, such as the upper and lower canines, upper and lower first molars, and sometimes upper lateral incisors and premolars.

- **Canines:** The long axis of the upper and lower canines, which is also the most convex part of the labial surface, is located more mesial than the true mesiodistal center of the tooth; therefore, the FA point looks a little bit more mesial than the dead center of the tooth. If you err and place the bracket on the center of the crown mesiodistally, the canine will rotate mesially.
- **Molars:** The landmark that Andrews used as the long axis of the clinical crown for the molar is the buccal groove. The FA point then lies along the buccal groove, midway occlusogingivally (see Fig. 17-2). It is important to realize that the center of the tube mesiodistally should be in agreement with the FA point. As some manufacturers have reduced the mesiodistal length of tubes, orthodontists have started positioning tubes too far mesial, resulting in distal overrotation of the molars.
- **Upper lateral incisor:** After the third molars, the upper lateral incisors are the teeth with problems involving size and shape. This makes it difficult to determine the long axis of the crown from the buccal. It is wise to use a mirror to look at the lingual surface of the incisor and then extend the long axis of the clinical crown from the lingual to the buccal.
- **Premolars:** Usually premolars, specifically second premolars, represent a challenge at the time of bonding because of a lack of direct vision. In these cases, it is advisable to look with a mirror from the occlusal and the buccal to locate the FA point and the long axis of the clinical crown.

Both authors believe that indirect bonding can be of great help to better position the brackets, specifically on premolars and molars, decreasing the need for "fine tuning" rebonding toward the end of treatment.

Treatment Mechanics

For didactic purposes, treatment mechanics usually has been divided in different stages, from three to seven depending on authors' preference. Simplicity is of paramount importance when teaching; therefore, all the mechanics to be accomplished in our orthodontic treatments with the SWA can be divided into three stages: stage 1, leveling and alignment; stage 2, working stage; and stage 3, finishing stage.

During each of these stages, there are specific movements of teeth that will occur and specific goals that have to be achieved before continuing to the next stage of treatment. It is important to emphasize that both the treatment outcome and its efficiency will be greatly improved if the orthodontist follows these stages. The following stages of treatment mechanics, with their respective wire sequence, have been tailored for active SLBs, although they can be applied to any SWA.

Stage 1: Leveling and Aligning

Leveling and aligning is a complex process in which all the crowns are moving at the same time and in different directions. As the teeth level and align, reciprocal forces between them develop, which can be of great help to guide the movements to our advantage. Then, when possible, all teeth should be engaged from the beginning to obtain maximum efficiency of tooth movement. Usually at this stage, round small-diameter thermal-activated wires, such as a 0.014 inch for severe crowding or a 0.018 inch for moderate to minimum crowding, are preferred. In cases that required retraction of the incisors, it is recommended to cinch the wire back of the second molar tube or to place crimpable stops to avoid undesirable movement of the wire, causing discomfort to the patient. These round wires can be in place for as long as 8 to 12 weeks before proceeding to the next wire, which usually is a 0.020- × 0.020-inch thermal-activated wire. This wire is a low-deflection thermal-activated wire that works very well as a transitional wire from stage 1 to stage 2. The 0.020- × 0.020-inch wire corrects most of the rotations left by the previously used round wires and provides more stiffness to start leveling the curve of

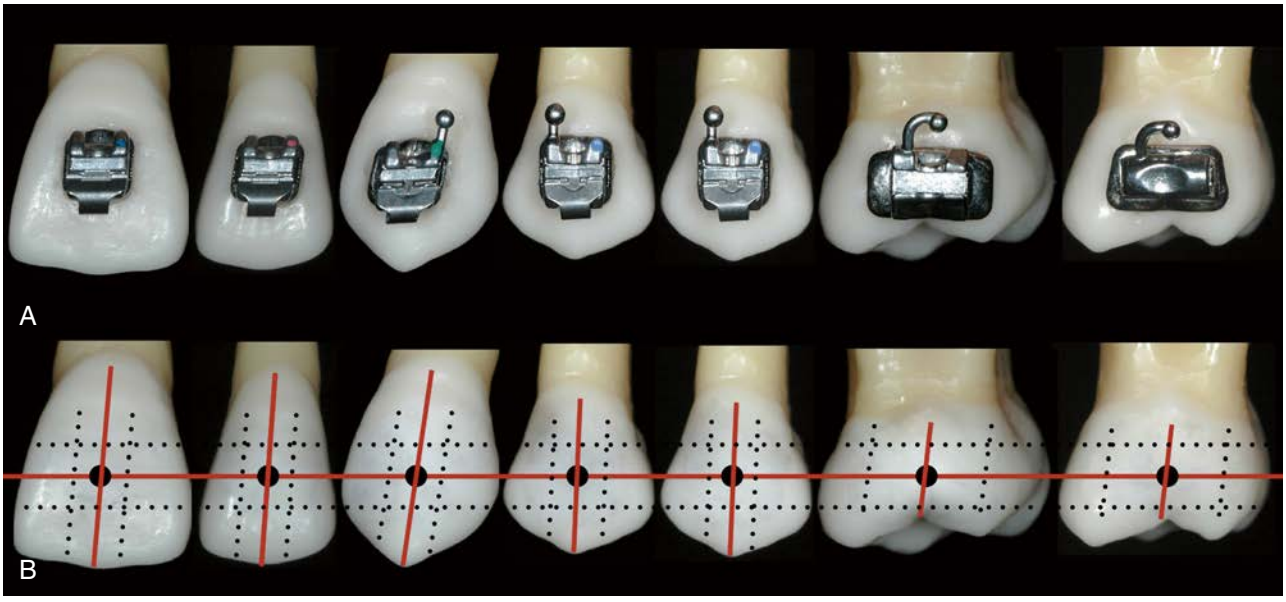


FIGURE 17-2 A, All the brackets aligned along the references described by Andrews, such as the FA point and the long axis of the clinical crowns. In B, the long axis of the clinical crown for the molars is the buccal groove.

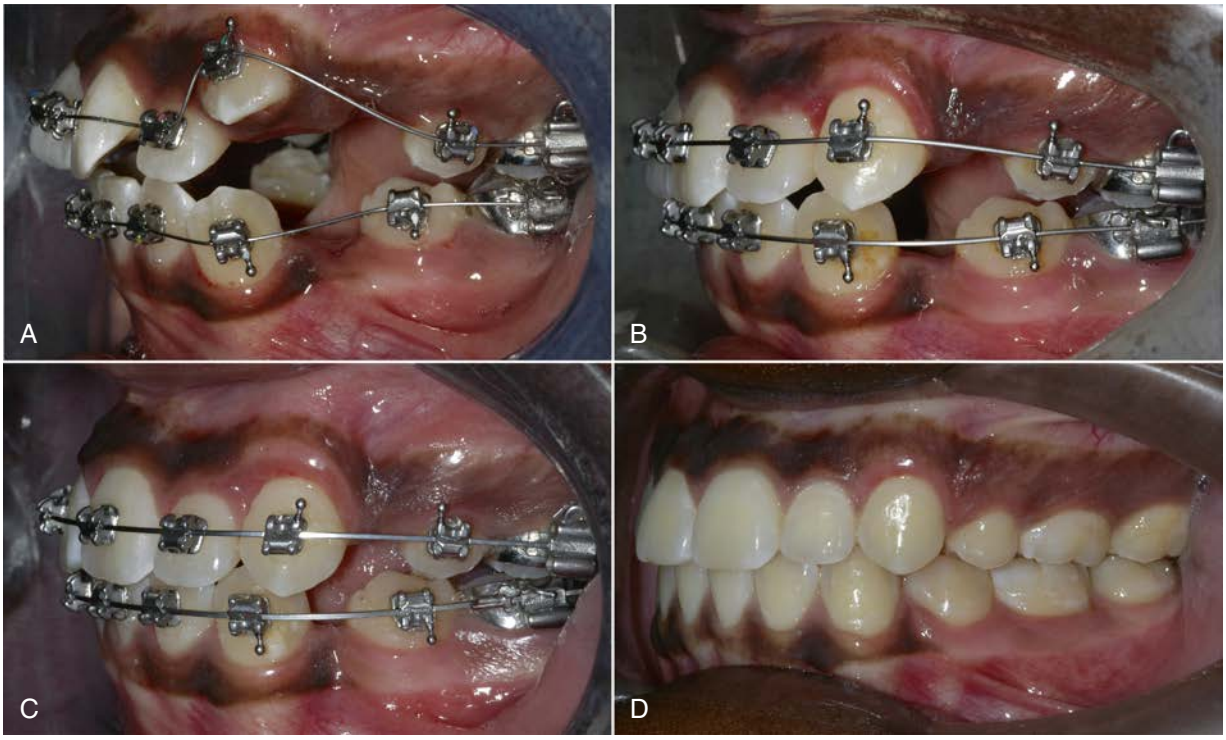


FIGURE 17-3 As the initial alignment occurs, molars upright, and the maxillary and mandibular planes of occlusion become more parallel, helping to retract the incisors and improve the overbite. The wire sequence is very important to control tip, torque, and rotations. Small, round Sentalloy wires such as 0.014 **(A)** and 0.018 **(B)** inch are excellent to control initial alignment; upright incisors, premolars, and molars; and correct major rotations. Bioforce wires such as 0.020- × 0.020-inch **(C)** are ideal to finish with the leveling and aligning stage. This wire finishes correcting the rotations still present after the round wires. It also expresses more crown tipping and starts providing a small amount of torque because its dimension mildly activates the springing clip of the bracket. **D,** Finished case.

Spee and therefore flatten the occlusal plane. It is important to notice that even if you could start treatment with a rectangular or square thermal-activated low-deflection wire, with the assumption of saving time and providing torque from the beginning of treatment, this is absolutely not recommended because it may cause loss of posterior anchorage. This happens for two main reasons: first, the only teeth with positive labial crown torque are the maxillary central and lateral incisors, and second, the mesial crown tip of the maxillary and mandibular canines is rather large. Therefore, if we start treatment resolving the crowding with a rectangular or square wire, we are providing labial crown torque to the maxillary incisors and mesial crown tip to canines, which will increase our anchorage in the front part of the arch, facilitating the loss of anchorage in the posterior part of the arch. This is critical in cases in which the treatment plan calls for maximum retraction of the maxillary or mandibular incisors (or both). In these particular cases, the use of a 0.020-inch thermal-activated wire can be better indicated than the 0.020- × 0.020-inch wire and thus will not provide torque and the tip effect on the canines will be minimal. This allows the molar and premolars to level, align, and upright, which will produce a "lasso" effect on the incisors that will upright and sometimes even retract them (Fig. 17-3).

The 0.020- × 0.020-inch wire will make the clip of the SLB active and thus start delivering torque; nonetheless, its strength is not sufficient to compromise the anchorage that has already been created with the round wires. Usually, after 8 to 10 weeks with the 0.020- × 0.020-inch wire, the stage 1 of leveling and aligning is finished, and in the authors' opinion, it is the first time to evaluate bracket placement and debond or rebond as necessary. Then we are ready to start stage 2, the working stage.

The following are the movements we should expect and goals we should accomplish when leveling and aligning, before starting stage 2:

- Teeth move individually.
- It is mainly crown movement.
- Molars and Premolar (PM) derotate and upright distally.
- Incisors are upright and sometimes even retract.
- Start building posterior anchorage.
- Before proceeding to stage 2, check bracket position (gross errors) and debond or rebond as indicated.

The following are the most common wires and sequence used at stage 1 of treatment:

- Mainly round, small-diameter, superelastic wires (ideal thermal activated)
- Square or rectangular superelastic wires to correct remaining rotations and level the occlusal plane (Tables 17-1 and 17-2)

Stage 2: Working Stage

This stage of treatment is the one on which we will spend more time. At this stage, the maxillary and mandibular arches are coordinated, proper overbite and overjet are achieved, Class II

TABLE 17-1 Types of Wires, Size, and Sequence Suggested for Stage 1 of Treatment Mechanics in Cases with Moderate to Severe Crowding

	SEVERE TO MODERATE CROWDING	
STM1 Type	**Size (inches)**	**Sequence**
Niti thermal activated	0.014	0.014
	0.018	
Niti thermal activated	0.018 × 0.018	0.018
	0.020 × 0.020	
	0.019 × 0.025	0.020 × 0.020

STM, Stage of Treatment Mechanics.

TABLE 17-2 Types of Wires, Size, and Sequence Suggested for Stage 1 of Treatment Mechanics, in Cases with Mild Crowding

	MILD CROWDING	
STM1 Type	**Size (inches)**	**Sequence**
Niti thermal activated	0.014	0.018
	0.018	
Niti thermal activated	0.018 × 0.018	0.020 × 0.020
	0.020 × 0.020	
	0.019 × 0.025	

STM, Stage of Treatment Mechanics.

or Class III are corrected, maxillary and mandibular midlines are aligned, extraction spaces are closed, and maxillary and mandibular occlusal planes are paralleled. Although most of these corrections happen simultaneously, we will describe them separately for didactic reasons so key points can be emphasized.

Arch Coordination. The maxillary and mandibular archwires must be coordinated in order to obtain a stable occlusal intercuspation and proper overjet. In an ideal intercuspation of a Class I, one-tooth to two-teeth occlusal scheme, the palatal cusps of the maxillary molars should intercuspate with the fossae and marginal ridges of mandibular molars, the buccal cusp of the mandibular premolars should intercuspate with the marginal ridges of the maxillary premolars, and the mandibular canines and incisors should intercuspate with marginal ridges of the maxillary canines and incisors. If this occlusal scheme occurs, it will then provide an overjet of 2 to 3 mm all around the arch from second molar to second molar. Then the maxillary archwire must be 2 to 3 mm wider than the mandibular archwire. The archwire coordination is done with the stainless steel wire. Even if they come preformed, the clinician should not rely on it and check them before insertion.

Another important aspect of arch coordination is the effect that it has on the vertical dimension and the sagittal dimension. Arch coordination is a transverse issue. The maxillary teeth should be upright and centered in the alveolar/basal bone and coordinated with the mandibular teeth, which should also be upright and centered in the alveolar/basal bone to obtain a proper intercuspation. Often, this is not the case, and we find maxillary molars buccally inclined, also referred as an *accentuated curve of Wilson*, which can produce contacts between the palatal cusp of maxillary molars and the inclines or even the cusps tip of the mandibular molars. This decreases the overbite

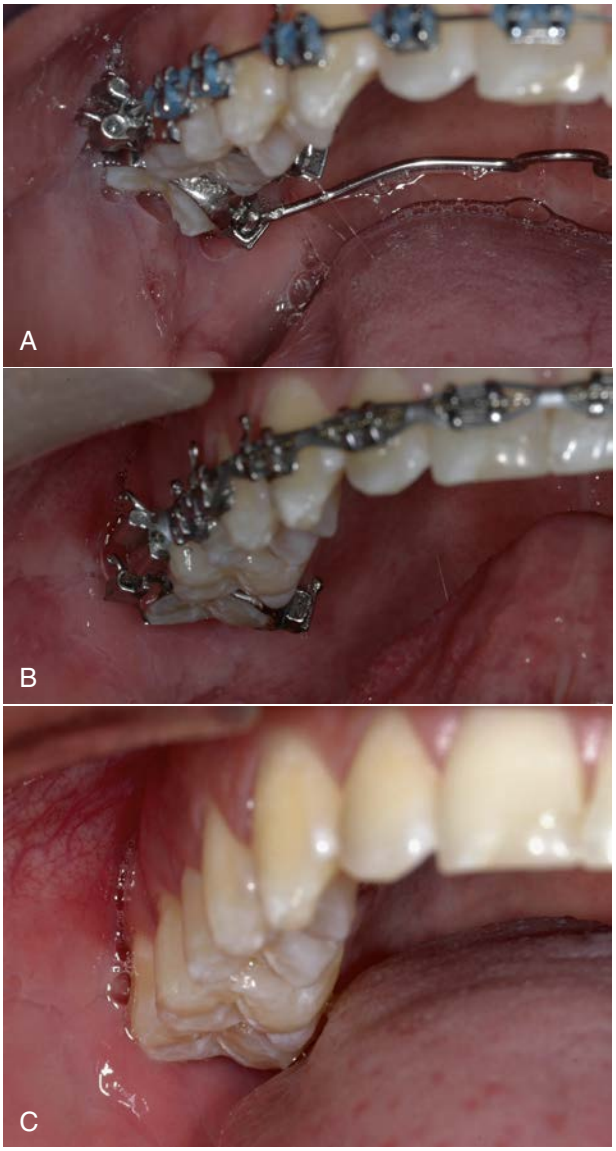


FIGURE 17-4 Asequence of a maxillary second molar severely tipped to the buccal corrected with a transpalatal bar. **A,** Initial. **B,** After correction is done. **C,** Finished case.

and sometimes produces even an open bite (vertical problem), which in turn can produce a downward and backward movement of the mandible (sagittal problem). This phenomenon is due to the lack of palatal crown torque of the maxillary molars. Depending on the amount of palatal crown torque needed for the maxillary molars to level the curve of Wilson, we suggest three solutions:

1. For minor problems with torque, we can wait until the finishing stage when a larger size wire (0.021- × 0.025-inch stainless steel) can be used to fill the slot and deliver more torque to the molars.
2. For moderate problems with torque, we can add palatal crown torque to the working wire.
3. For severe problems with torque, the use of a transpalatal bar (TPB) is suggested. A TPB can be used to easily place and deliver palatal crown torque to maxillary molars (Fig. 17-4).

Overbite and Overjet Correction. An optimal overbite–overjet relationship does not have to be a certain predetermined number of millimeters. More important is the functional relationship

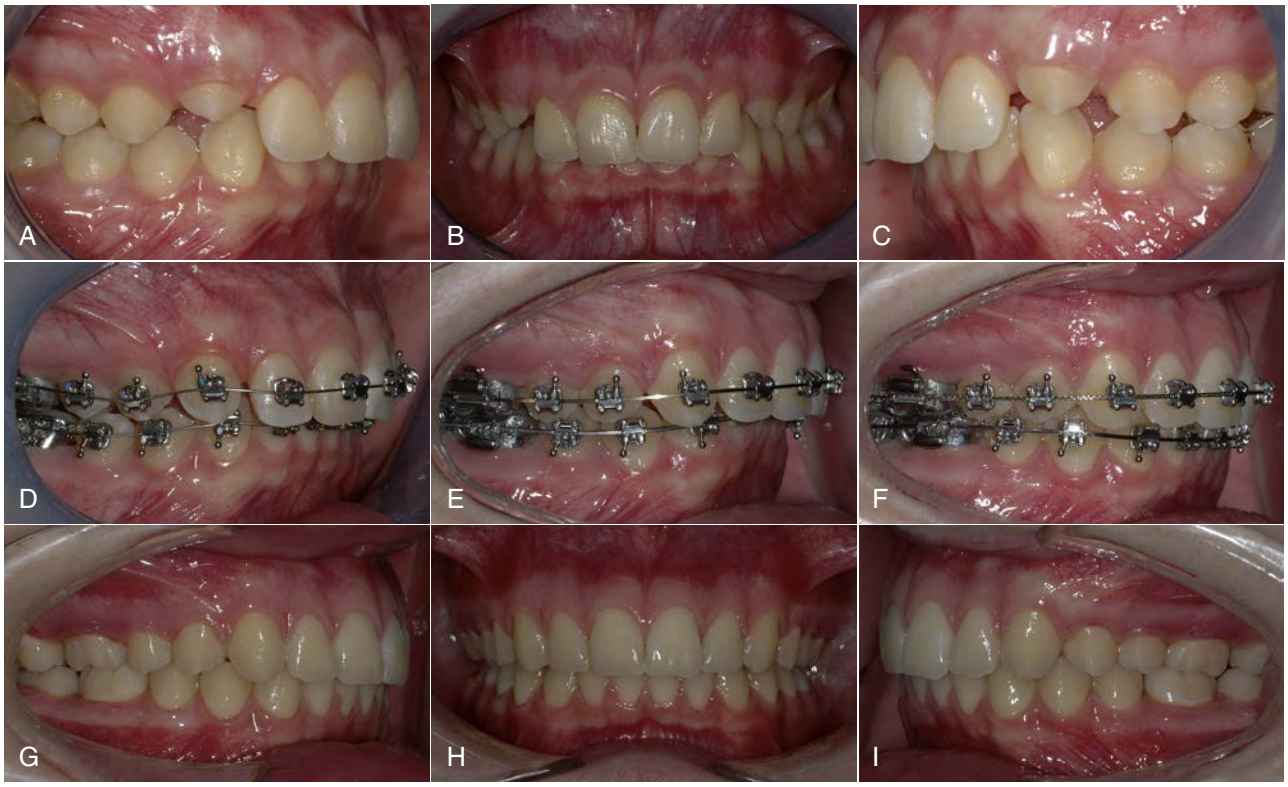


FIGURE 17-5 A deep bite case in which the occlusal plane was flattened with the use of a reverse curve of Spee on a 0.019- × 0.025-inch stainless steel wire. **A–C,** Initial intraoral views. **D,** Initial wire to level and align. **E,** A 0.019- × 0.025-inch stainless steel wire with reverse curve of Spee before correction. **F,** A 0.019- × 0.025-inch stainless steel wire with reverse curve of Spee after correction. **G–I,** Final intraoral views.

they have. This means that the overbite–overjet should be compatible with a mutually protected occlusal scheme and thus allows for a proper anterior guidance in protrusion and lateral excursive movements. Although, as already discussed, the number of millimeters is less important than the function, we find that an optimal overbite is usually around 4 mm and an optimal overjet is 2 to 3 mm. When diagnosing and treatment planning overbite–overjet problems, it is important to take the following key points into consideration: arch space management, position of the mandible in centric relation, and relationship of the upper and lower incisors with the lips. Arch space management is important to understand because the SWA tends to flatten the curve of Spee, which requires space in the arch. If not enough space is available or created, the incisors will procline, increasing the arch perimeter. This incisor proclination will also decrease the overbite and may help, if it only occurs in the lower arch, to decrease the overjet. Flattening the maxillary and mandibular occlusal planes proclining the incisors can be of help in deep bite cases (Fig. 17-5). When the incisors are not allowed to procline, space in the arch must be created. This is specifically important to avoid periodontal problems in cases with thin bone surrounding the incisor area. Advanced diagnostic imaging tools, such as cone-beam computed tomography (CBCT), could be of great help to precisely identify the condition of the bone in this area. Up to 4 to 6 mm can be created with interproximal reduction of teeth, usually done on the incisors and less often the canines and premolars. If more than 6 mm of space is required, extraction of premolars could be indicated.

Another important factor to consider when evaluating overbite–overjet problems is the position of the mandible. Often, differences between a maximum intercuspation (MIC) and centric relation (CR) can produce significant differences in the overbite–overjet relationship. This can be clearly seen in Figure 17-6, in which what looks like a normal overbite–overjet relationship in MIC is an anterior open bite in CR. In this case, as the mandible rotates close in CR, a primary contact found at the second molar keeps the bite open in the anterior, decreasing the overbite and preventing the mandible to achieve a more stable occlusal scheme.

Last, but by no means the least important, is the sagittal and vertical relationship of the maxillary and mandibular incisors with the lips. In an open bite case, should we intrude the molars or extrude the incisors? In a deep bite case, should we intrude the maxillary incisors, the lower, or both? These basic but very important questions can be answered through an understanding of the optimal relationship of the incisors with the lips. According to contemporary a esthetic trends and taking into account the aging process, for adolescents and young adults, the maxillary incisors should have, at rest, an exposure of about 4 mm beyond the most inferior point of the upper lip known as the *upper stomion*. As explained earlier, an optimal functional overbite should be about 4 mm. Now, if we put together the last two concepts, the incisal edge of the lower incisors should be at the same level with the most inferior point of the upper lip. Therefore, any vertical change of the incisors will affect not only the function through changes of the anterior guidance but also the aesthetics through the amount of tooth exposure. These

FIGURE 17-6 An example of a case with a clinically significant discrepancy between a maximum intercuspation (MIC) and centric relation (CR). This case was treated with the aid of mini-implants to control the vertical position of the maxillary first and second molars. **A, B,** Initial intraoral views in MIC. **C, D,** Initial intraoral views in CR. **E, F,** Intraoral view before molar intrusion. **G, H,** Intraoral view after molars have been intruded and open bite closed. **I, J,** Intraoral view with the final wire. **K, L,** Intraoral view after appliances have been removed. **M–O,** Occlusal intraoral views of the maxillary arch before treatment, during treatment with mini-implant, and after treatment.

anterior functional and aesthetic references, explained by Ayala as the "upper stomion concept" (Fig. 17-7), will help the clinician to determine the best strategies to correct overbite–overjet problems and will be of special importance for planning cases involving orthognathic surgery.

Closing Extraction Spaces. Usually after leveling and aligning, the extraction spaces left are smaller than at the beginning of treatment because some of the space has been taken to unravel the initial crowding and to upright the maxillary and mandibular incisors, as described earlier in this chapter. Also, the maxillary and mandibular occlusal planes should be flat or almost flat, and the six anterior teeth should be consolidated into one unit. Then, to efficiently close the remaining spaces, achieving the desired functional and aesthetic goals, we need to determine the anchorage requirement. This will allow us to know which teeth should be moved more mesially or distally

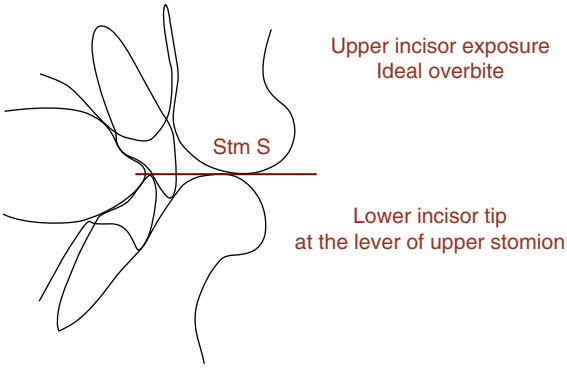


FIGURE 17-7 Diagram of anterior functional and a esthetic references, the "upper stomion concept." *Stm S,* Stage of Treatment Mechanics.

and therefore to choose the appropriate mechanics. We believe that one of the easiest ways to determine the anchorage requirement is to perform a visual treatment objective (VTO). The VTO is a cephalometric exercise in which we modify the patient's cephalometric tracing to achieve the desired end-of-treatment result, and then, by superimposing both tracings, we can visualize the movements that need to occur to obtain that result. The VTO is not a formula or equation that will determine or impose a specific type of treatment but rather an exercise in which we take into account our experience gathered from other similar cases, an estimation of the growth the patient will have during treatment, the patient's biotype and soft tissue characteristic, and so on to more accurately plan treatment in our cases and have a visual representation of it. Thus, after the VTO has been performed, the anchorage requirement can be minimum, medium, or maximum. Before describing each one of these anchorage situations, it is important to indicate the wires and auxiliaries used at this stage. In our mechanics, we use either a double keyhole loop (DKH) or a straight wire with hooks and Niti thermal-activated coils. Both of these types of wires are stainless steel and can be either 0.019- × 0.025-inch or 0.021- × 0.025-inch, depending on the anchorage situation. The Niti thermal-activated coils can be light (100 g), medium (150 g), or heavy (200 g). Also, when the anchorage situation calls for it, we use TPBs and temporary anchorage devices (TADs).

Double Keyhole Loop Activation. The DKH archwire can be activated in two different ways. It can be pulled from the distal of the first or second molar, so as to open the loops 1 mm, and then cinched back to keep the loops open. As the loops close, the teeth come together, closing the space. A different activation method is to open the loops 1 mm and then use a stainless steel ligature to ligate the distal loop to the hook of the first or second molar tube, with sufficient tension to keep the loops open. As the loops tend to close, the ligature will exert force on the molar tube, and the teeth will come together. In either way of activation, changes in wire size and place of activation will determine the type of anchorage obtained.

Niti Thermal-Activated Coil Activation. Niti thermal-activated coils come in three different strengths: 100 g (blue dot), 150 g (yellow dot), and 200 g (red dot). It is the authors' preference to use the 150-g Niti thermal-activated coil. These coils deliver the same force independent of the amount of activation. In our mechanics, we usually crimp a surgical type of hook distal of the canine from which a Niti thermal-activated coil is engaged all the way to the hook of either the first or second molar. If a surgical hook is not available, the Niti thermal-activated coil can be engaged to the hook of the canine's bracket. This situation requires the six front teeth to be tied together with either an elastomeric chain or a stainless steel ligature so they act as a unit.

Minimum Anchorage. On a minimum anchorage situation, molars will be moved mesially to close the remaining extraction spaces. We use a 0.021- × 0.025-inch wire. This wire will express the buccal crown torque of the maxillary incisors and the mesial tip of the canines. In the mandible, this wire will express the mesial tip of the canine. This situation increases the anchorage in the anterior part of the mouth because it would be more difficult to retract or even tip back the anterior teeth while moving the molars forward. It also has been recommended to reduce the size of the wire in the posterior part, specifically the edges, to decrease the anchorage of the posterior teeth. The activation of the DKH or the Niti thermal-activated coils must be done from the first molars. Then, after the first molar has been moved forward as desired, the second molar can be activated and moved forward, too. Often, though, this is not required because the second molars will travel forward as we move the first molars, and then the space remaining between the first and second molars will be very small and easily closed with an elastomeric chain.

Medium Anchorage. This is the most common anchorage situation encountered in our cases. Medium anchorage means that the remaining spaces should be closed reciprocally. For this situation, we use a 0.019- × 0.025-inch wire. The activation of the DKH or Niti thermal-activated coils is done, most of the time, from the first molar. However, it can also be done from the second molars depending on how the case is progressing. The bone and attachment apparatus are not the same for every patient, and therefore, the response to the closing mechanics could differ among cases. Then a clinical examination of the overbite–overjet, canine and molar relationship, and facial aesthetics should be done at each visit to evaluate any changes in activation that may be required. This should not take any extra time because the activation of a DKH or Niti thermal-activated coil is a rather easy procedure.

Maximum Anchorage. In a maximum anchorage situation, most of the remaining space left after leveling and aligning is closed because of distal movement of the anterior teeth. We use a 0.019- × 0.025-inch wire. The DKH or Niti thermal-activated coil is activated from the second molars. Although not frequently required, auxiliaries to enhance posterior anchorage such as TPB, TADs, or extraoral force (headgear) can be used as needed.

Intermaxillary Elastics. *Discretion* is a good word to describe the use of intermaxillary elastics. We use them and like them, but it is important to understand how they are used to avoid problems. We do not use intermaxillary elastics in the following situations:

- Round wires
- Initial leveling and aligning, low-deflection wires
- To a terminal tooth, last tooth in the arch
- In the anterior part of the mouth to close open bites
- In the posterior part of the mouth to correct crossbites
- For an extended period of time

We use intermaxillary elastics in the following situations:

- At the working and finishing stages
- On square or rectangular stainless steel wires
- On the buccal side of the mouth, short Class II or III, or triangular verticals

The three types of intermaxillary elastics we commonly use are 3/16-inch 4 oz, 6 oz, and 8 oz elastics as well as 1/8-inch 4 oz, 6 oz, and 8 oz. Short means, in a Class II, for instance, from the maxillary canine to the mandibular second premolar in a nonextraction case and to the first mandibular molar in an extraction case.

The following are the movements we should expect and goals we should accomplish at the working stage before starting stage 3:

- Movement of group of teeth in all planes of the space: sagittal, vertical, and transverse
- Overjet–overbite correction
- Class II and III correction
- Close all remaining extraction spaces, aligning maxillary and mandibular midlines
- Finish leveling the occlusal plane
- Arch coordination

The following are the most common wires and sequence used at stage 2 of treatment:

- In nonextraction cases, a 0.019- × 0.025-inch stainless steel wire. Reverse curve of Spee can be manually added to the wire if needed
- In extraction cases, either a 0.019- × 0.025-inch or a 0.021- × 0.025-inch stainless steel wire depending on the anchorage requirement, as previously explained (Tables 17-3 and 17-4)

Stage 3: Finishing Stage

At this stage, to place each tooth on its ideal position and flatten the occlusal plane, full bracket expression is desired; thus, a larger wire such as a 0.021- × 0.025-inch or a 0.022- × 0.028-inch stainless steel may be required. In our experience using an active SLB with the clip pushing and sitting the wire onto the slot, often optimal bracket expression is achieved after a 0.019- × 0.025-inch stainless steel wire has been in place for a few months. This is especially true in nonextraction cases with an average curve of Spee. However, in some cases, the size and stiffness of a 0.021- × 0.025-inch or 0.022- × 0.028-inch stainless steel are indicated, such as in cases with a deep curve of Spee, extraction cases that have required an important amount of tooth movement, and cases that required significant labial crown torque of maxillary incisors such as Class III camouflage cases and Class II, Division 2 cases. When the maxillary and mandibular occlusal planes are parallel and all the bracket slots are aligned, bracket position should be carefully checked for minor correction of tooth position, and therefore the second time of debond or rebond should be done. It is also suggested, at this point in treatment, to mount the models in an articulator to better visualize the intercuspation of the posterior teeth, which is very difficult to do clinically. The last wire we use is a stainless steel multibraided 0.021- × 0.025-inch archwire. Although this wire is large enough to fill the slot of the bracket and then maintain the tip, torque, and offset of each tooth, its resilience permits both minor bracket repositioning and "end of treatment" optimal intercuspation. It is important to notice that at this point in treatment, all the appliance interferences should be removed using a finishing carbide burr on a high-speed handpiece. With a thin articular paper, all contacts must be checked. Only tooth–tooth contacts should be allowed. All bracket, tube, or band contacts must be removed to allow proper settling. Vertical triangular 3/16-inch elastics, either 6 oz or 8 oz, are used to achieve proper intercuspation. These vertical elastics should not be used with the braided wire for more than 6 weeks to avoid rolling premolars and molars lingually, which can be detected not from the buccal but rather from the lingual, where premolars and/or molars will not be contacting. Finally, before removing the appliance, a complete assessment of the occlusal end-of-treatment goals should be performed. We strive to finish our cases with a static occlusal scheme compatible with the six keys of optimal occlusion described by Andrews[1] and a dynamic mutually protected occlusal scheme in centric relation described by Roth[11] (Table 17-5).

TABLE 17-3 Types of Wires, Size, and Sequence Suggested for Stage 2 of Treatment Mechanics in Nonextraction Cases

	NONEXTRACTION	
STM2 Type	**Size (inches)**	**Sequence (inches)**
SW stainless steel	0.019 × 0.025	
Niti thermal activated	0.021 × 0.028	0.019 × 0.025
Reverse curve stainless steel	0.019 × 0.025	

STM, Stage of Treatment Mechanics: *SW*, Straight Wire.

TABLE 17-4 Types of Wires, Size, and Sequence Suggested for Stage 2 of Treatment Mechanics in Extraction Cases

	EXTRACTION	
STM2 Type	**Size (inches)**	**Sequence**
SW stainless steel with hooks	0.019 × 0.025	
	0.021 × 0.025	
Or		Depends on anchorage requirement
DKL stainless steel	0.019 × 0.025	
	0.021 × 0.025	

DKL, Double Key Loops; *STM*, Stage of Treatment Mechanics.

TABLE 17-5 Types of Wires, Size, and Sequence Suggested for Stage 3 Treatment Mechanics

STM3 Type	Size (inches)	Sequence
SW stainless steel	0.021 × 0.025	SW stainless steel
	0.021 × 0.028	↓
Braided stainless steel	0.019 × 0.025	Braided stainless steel;
	0.021 × 0.025	either size is fine

SW, Straight Wire; *STM*, Stage of Treatment Mechanics.

FUTURE DIRECTIONS

Although alternative SWA prescriptions for the maxillary anterior teeth such as "high torque" as well as "low torque" have been in existence for many years, it has not been until recently that SWA systems with variable prescriptions and/or a fully customized prescription have gained some traction. The SWA was developed based on measurements taken from the facial surface of each tooth of 120 individuals with an optimal occlusion who never had orthodontic treatment. It is generally known that

tooth anatomy varies among individuals, and therefore, there are variations in the form such as degree of convexity, inclination, length, and width of the facial surface of each tooth. These variations could result in different degrees of optimal torque, tip, offset, and in/out needed for each tooth of each individual. The idea is clear, and it does certainly make sense to individualize the bracket prescription for each patient.[12] Some manufactures have already attempted to customize the bracket prescription based on individual needs, but as of now, it is not practical for the clinician, and it is expensive; therefore, this technology has not increased in popularity at all. However, we think that because of recent advances in digital technology such as low-radiation CBCTs, faster intraoral scanners, and three-dimensional printing, individualized prescriptions will soon be an interesting option to consider for orthodontists.

Another important reason that favors the use of an individualized prescription is the different amount of bone surrounding teeth that we find among our patients. The increased use of CBCTs over the past decade has helped us to better understand the anatomic limitations of tooth movement. Recent studies[13] have shown that a significant percentage of our patients have dehiscence and fenestrations before orthodontic treatment, so special biomechanical considerations need to be taken if buccal tooth movement such as protrusion and arch expansion are required. Also, in cases of canines and molars that are too close to the buccal cortical bone, adjustment of the buccal–lingual inclination (torque) should be done to prevent root damage as well as periodontal problems (dehiscence and fenestrations) that could lead to gingival recession.

CASE STUDY 17-1

A 20-year-old woman presented with an anterior open bite and a unilateral posterior crossbite (Fig. 17-8). The open bite was closed by leveling, aligning, and flattening the maxillary and mandibular occlusal planes. Triangular vertical elastics were used only on the buccal segment at the working and finishing stages. A transpalatal bar attached to the maxillary first molars was used to help with arch coordination and correct the unilateral posterior crossbite. An active self-ligating straight wire appliance (In-Ovation R and C, Dentsply/GAC) was used. Active treatment was for 15 months.

Dr. Antonino G. Secchi

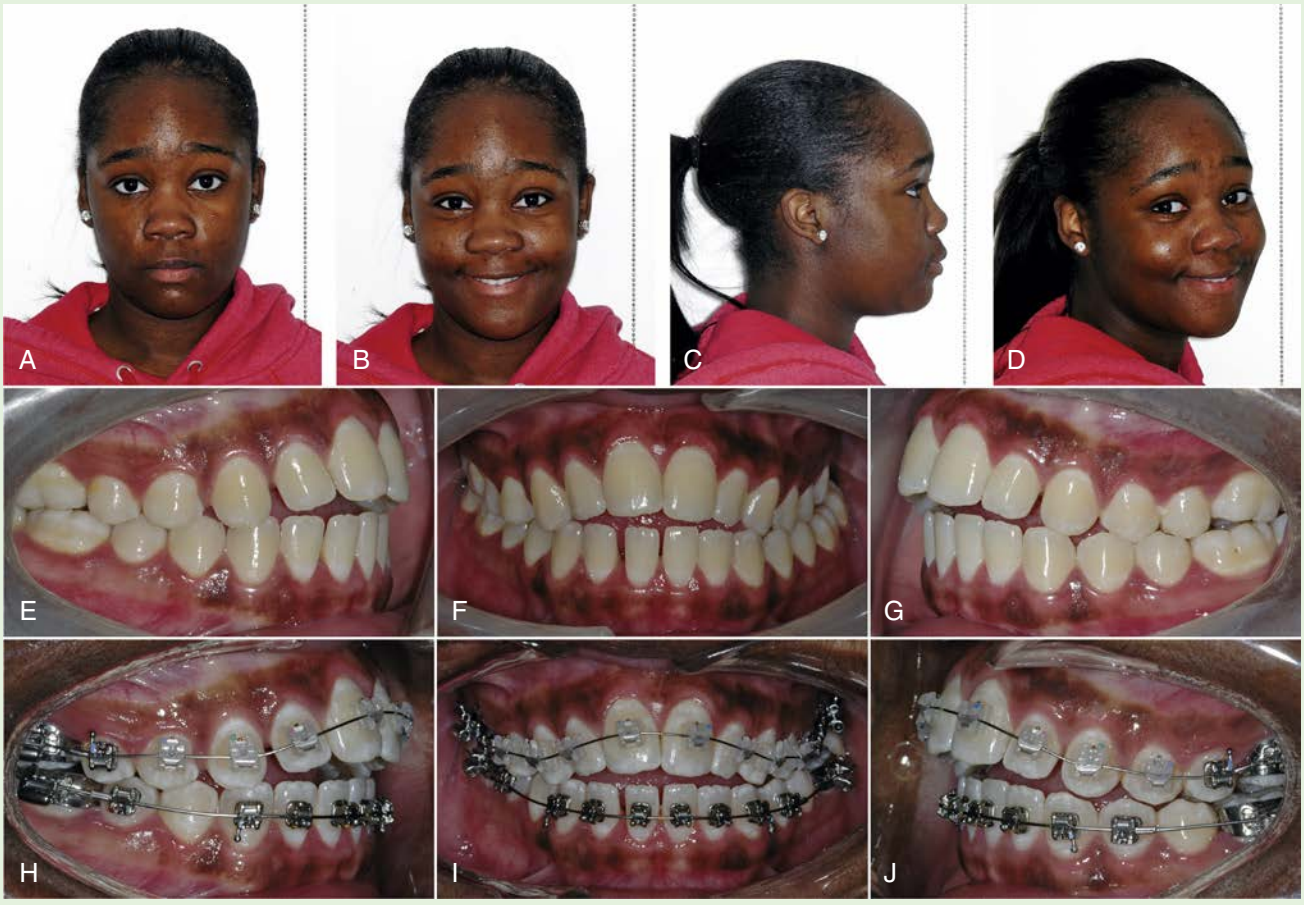


FIGURE 17-8 A–D, Facial photographs before treatment. E–G, Intraoral photographs before treatment. H–J, 0.014-inch Sentalloy initial wires. K–M, 0.020- × 0.020-inch Bioforce wires to finish stage 1 of leveling and aligning. N–P, 0.019- × 0.025-inch stainless steel working wire to flatten the occlusal plane, coordinate arches, and consolidate spaces. The patient starts using triangular vertical elastics.

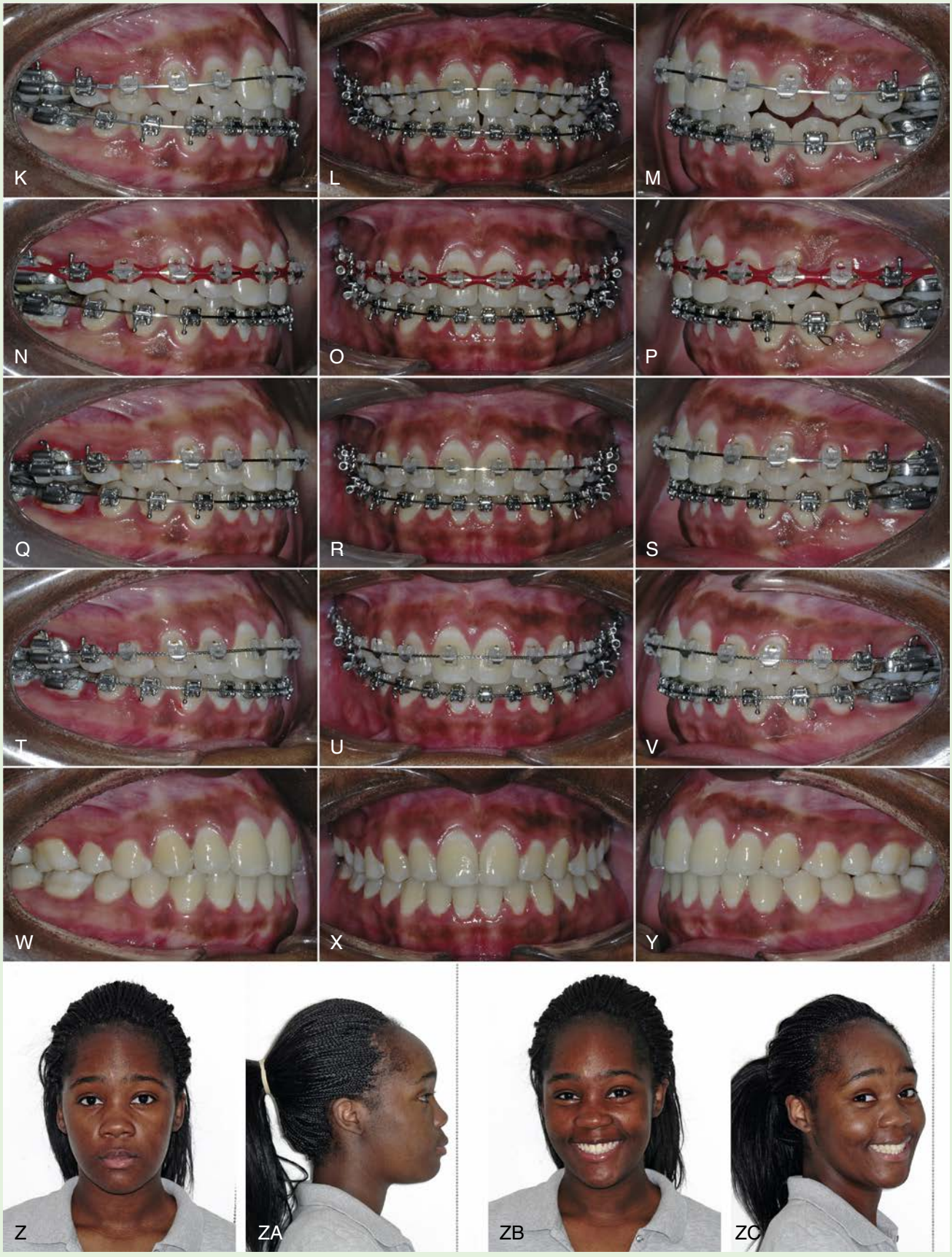


FIGURE 17-8, cont'd **Q–S,** 0.021- × 0.025-inch stainless steel to finalize leveling occlusal plane, continuing with triangular vertical elastics. **T–V,** 0.021- × 0.025-inch stainless steel braided wire, finishing archwire. **W–Y,** Intraoral photographs after treatment. **Z–ZC,** Facial photographs after treatment.

CASE STUDY 17-2

An 11-year-old boy presented with Class II-2 deep bite, crowding, and severe retroinclination of maxillary incisors. After initial leveling and aligning with a 0.014 and 0.020 Niti wires, the Class II was treated with a high-pull headgear (Fig. 17-9). The mandibular plane was flattened with a 0.019- × 0.025-inch stainless steel wire. Proper buccal crown torque was obtained with a 0.021- × 0.025-inch stainless steel wire and final interdigitation with a 0.021- × 0.025-inch stainless steel braided wire and 1/8-inch heavy elastics. During the final stage, mounted models and bracket rebonding were done. A Roth 0.022 straight wire appliance (True Roth, A Company, Sorrento Valley, San Diego, CA) was used. Active treatment was for 28 months.

Dr. Jorge P. Ayala

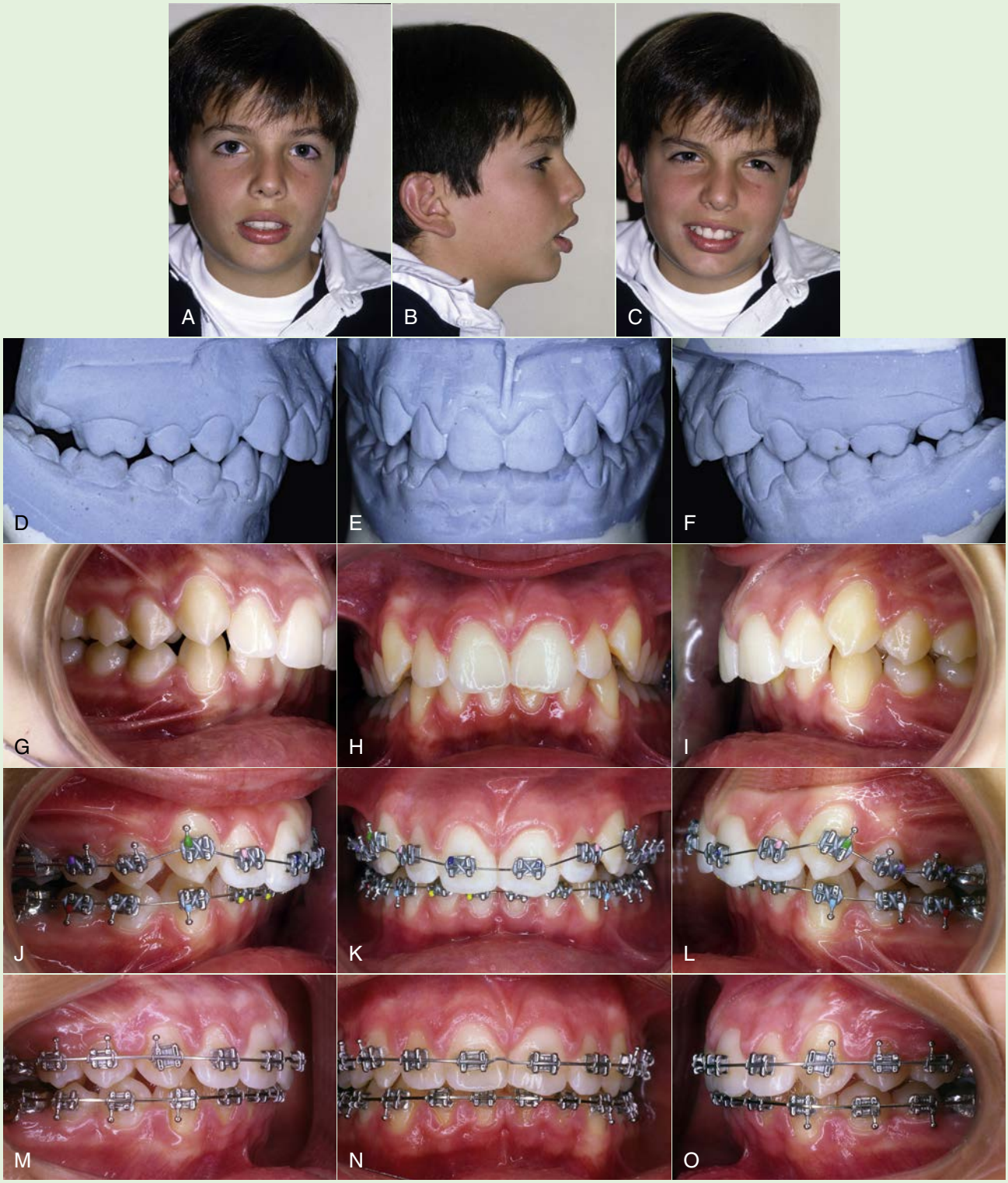


FIGURE 17-9 A–C, Facial photographs before treatment. **D–F,** Mounted models. **G–I,** Intraoral photographs before treatment. **J–L,** 0.014-inch Niti initial wires. **M–O,** 0.019- × 0.025-inch stainless steel wire.

CASE STUDY 17-2—cont'd

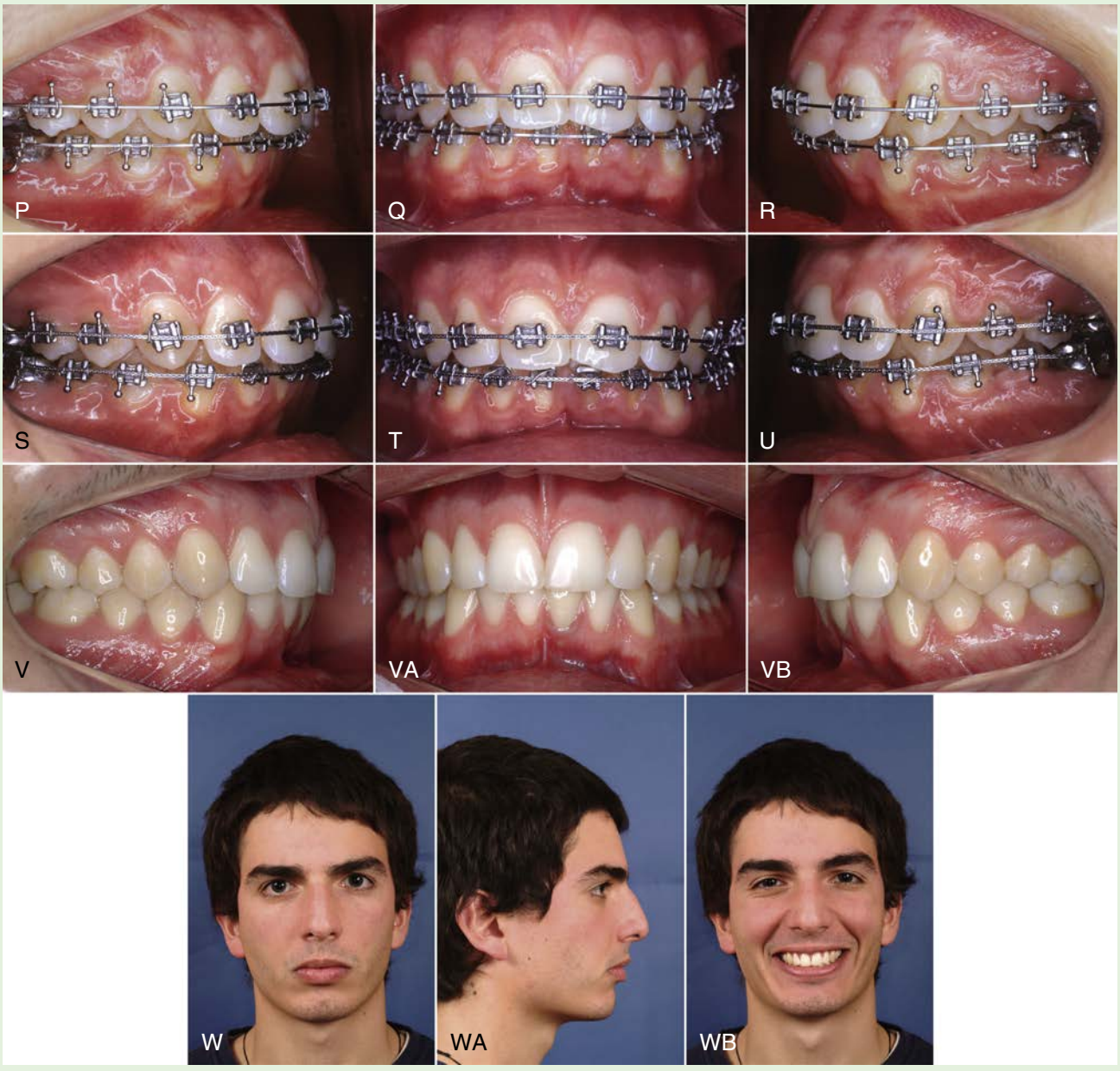


FIGURE 17-9, cont'd P–R, 0.021- × 0.025-inch stainless steel wire for proper buccal crown torque. S–U, Final interdigitation with a 0.021- × 0.025-inch stainless steel braided wire and 1/8-inch heavy elastics. V–VB, Intraoral photographs after treatment. W–WB, Facial photographs after treatment.

CASE STUDY 17-3

A 12-year-old girl presented with a deep bite, Class II canines, crowding, and severe retroclination of maxillary incisors (Fig. 17-10). After initial leveling and aligning, the mandibular occlusal plane was flattened using a 0.019- × 0.025-inch stainless steel wire with reverse curve of Spee. Short Class II elastics were used at the working stage. Proper buccal crown torque to the maxillary incisors was achieved with a 0.021- × 0.025-inch stainless steel wire. An active self-ligating straight wire appliance (In-Ovation R) was used. Active treatment was for 25 months.

Dr. Antonino G. Secchi

FIGURE 17-10 A–D, Facial photographs before treatment. E–G, Intraoral photographs before treatment. H–J, 0.014-inch Sentalloy initial wires only in the maxillary arch. Mandibular arch was bonded after initial alignment of the maxillary arch was completed. K–M, Maxillary 0.019- × 0.025-inch stainless steel wire and mandibular 0.014-inch Sentalloy initial wire. Notice the mandibular severe curve of Spee. N–P, Mandibular arch with a 0.019- × 0.025-inch stainless steel wire with reverse curve of Spee. At this point patient was asked to use short Class II 6 oz.

CASE STUDY 17-3—cont'd

FIGURE 17-10, cont'd **Q–S,** Maxillary 0.021- × 0.025-inch stainless steel wire to express buccal crown torque of incisors. Notice flattening of mandibular occlusal plane due to reverse curve of Spee. **T–V,** 0.021- × 0.025-inch stainless steel braided wire, finishing archwire. **W–Y,** Intraoral photographs after treatment. **Z–ZC,** Facial photographs after treatment.

CASE STUDY 17-4

An 18-year-old male, Class I dental with crowding came to our clinic to continue a treatment started in another country (Fig. 17-11). During the initial alignment with cinched-back 0.014-inch Sentalloy wires, the extraction spaces were reduced and the incisors passively retracted. The second wire was a 0.018- × 0.025-inch NeoSentalloy in the mandibular arch and a 0.020-inch Sentalloy in the maxillary arch. Spaces were closed with minimum anchorage using a 0.019- × 0.025-inch Double Key Loops (DKL) reduced in the posterior segment. Finishing was obtained with a 0.021- × 0.025-inch stainless steel wire followed by a 0.021- × 0.025-inch stainless steel braided wire and triangular 1/8-inch heavy elastics. During the final stage, mounted models and bracket rebonding were done. The case was treated with a 0.022 straight wire appliance (Mini-twin Dentsply/GAC). Active treatment was for 24 months.

Dr. Jorge P. Ayala

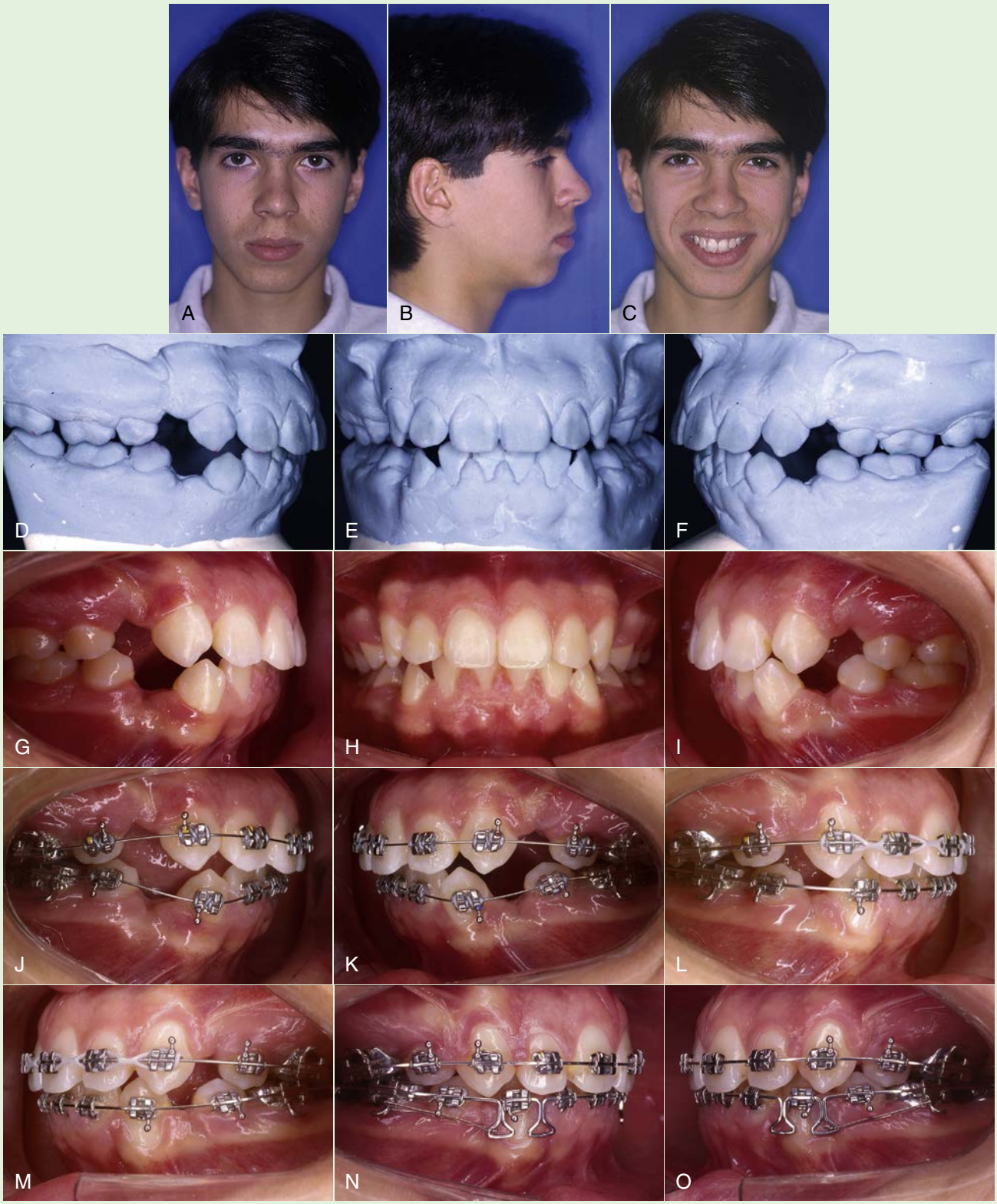


FIGURE 17-11 **A–C,** Facial photographs before treatment. **D–F,** Mounted models. **G–I,** Intraoral photographs before treatment. **J–K,** Cinched-back 0.014-inch Sentalloy initial wires. **L–M,** 0.018- × 0.025-inch NeoSentalloy in the mandibular arch and a 0.020-inch Sentalloy in the maxillary arch.

CASE STUDY 17-4—cont'd

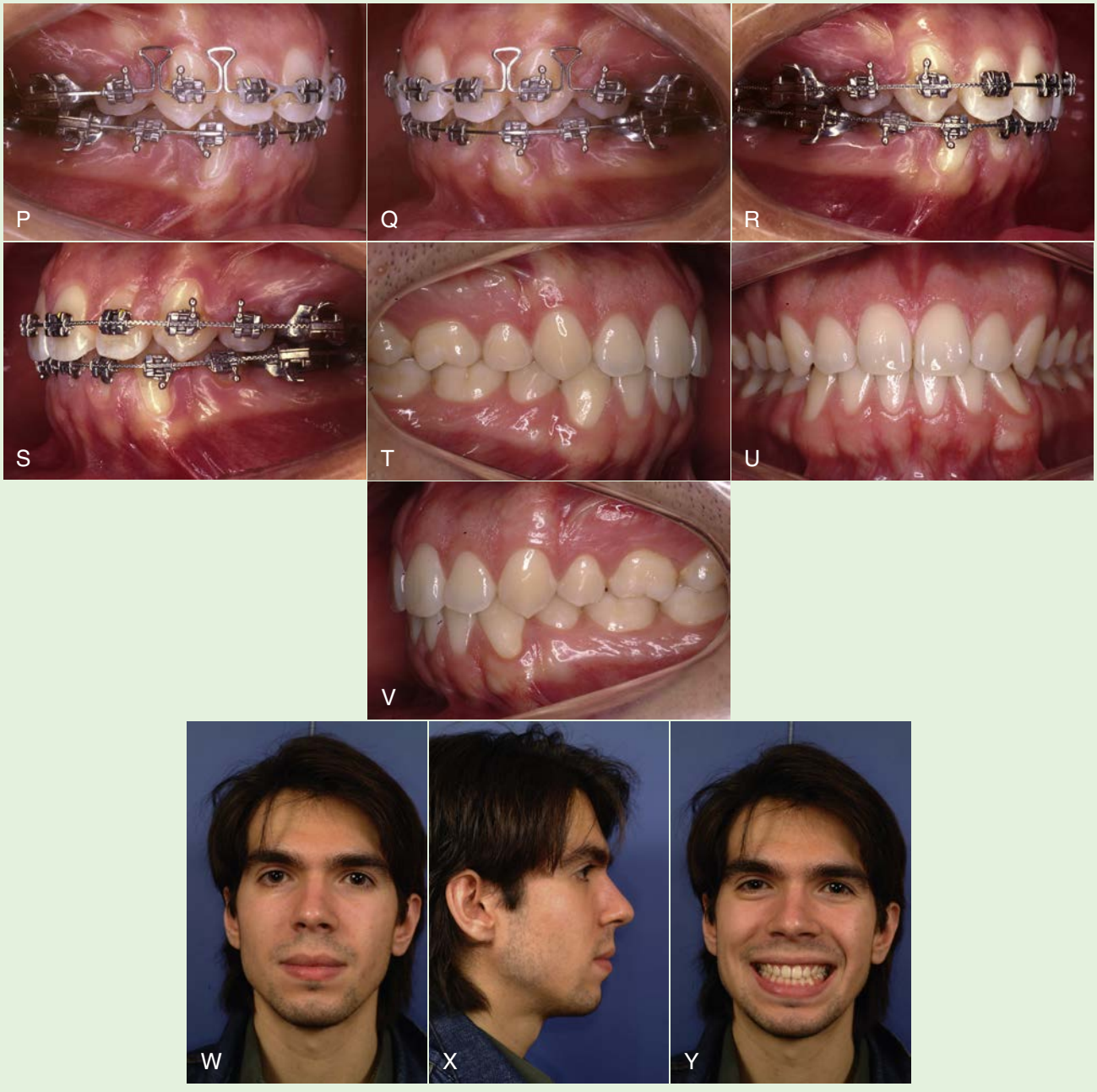


FIGURE 17-11, cont'd **N–Q,** 0.019- × 0.025-inch DKL to close spaces with minimum anchorage. **R–S,** Final 0.021- × 0.025-inch stainless steel braided wire and triangular 1/8-inch heavy elastics. **T–V,** Intraoral photographs after treatment. **W–Y,** Facial photographs after treatment.

SUMMARY

The advantages of the SWA are unquestionable. At 40 years after its introduction to our specialty, it remains the most popular orthodontic appliance used in the world. But today, at the beginning of the 21st century, the challenge is to integrate the SWA with recent changes in bracket design such as self-ligation and technologically advanced low-deflection thermal-activated archwires to provide orthodontists with a state-of-the-art appliance system that can deliver, through a practical, efficient, and reliable biomechanical system, excellent results for a wide range of dentofacial problems. The objective of this chapter is to delineate some of the basic principles of the SWA, emphasize the importance of optimal bracket placement, and provide readers with the framework of a simple, but complete, biomechanics tailored for active SLBs.

REFERENCES

1. Andrews LF. Six keys to normal occlusion. *Am J Orthod*. 1972;62:296–309.
2. Andrews LF. The straight wire appliance origin, controversy, commentary. *J Clin Orthod*. 1976;10(2):99–114.
3. Andrews LF. The straight wire appliance explained and compared. *J Clin Orthod*. 1976;10(3):174–195.
4. Andrews LF. Fully programmed translation brackets. In: Andrews LF, ed. *Straight Wire: the Concept and Appliance*. San Diego, CA: LA Wells; 1989.
5. Roth RH. Treatment mechanics for the straight wire appliance. In: Graber TM, Swain BF, eds. *Orthodontics: Current Principles and Techniques*. St. Louis: Mosby; 1985.
6. Damon DH. Treatment of the face with biocompatible orthodontics. In: Graber TM, Vanarsdall RL, Vig KW, eds. *Orthodontics: Current Principles and Techniques*. St. Louis: Mosby; 2005.
7. Pandis N, Polychronopoulou A, Eliades T. Active or passive self-ligating brackets? A randomized controlled trial of comparative efficiency in resolving maxillary anterior crowding in adolescents. *Am J Orthod Dentofac Orthop*. 2010;137:12.e1–12.e6.
8. Badawi HM, Toogood RW, Carey JP, et al. Torque expression of self-ligating brackets. *Am J Orthod Dentofac Orthop*. 2008;133:721–728.
9. Stefanos S, Secchi AG, Coby G, et al. Friction between various self-ligating brackets and archwire couples during sliding mechanics. *Am J Orthod Dentofac Orthop*. 2010;138:463–467.
10. Meyer M, Nelson G. Preadjusted edgewise appliances: theory and practice. *Am J Orthod*. 1978;73(5):485–498.
11. Roth RH. Functional occlusion for the orthodontist. *J Clin Orthod*. 1981;15(1):32–40, 44–51.
12. Ventureira CP. Prescripción variable en ortodoncia: lo que todo ortodoncista debería conocer. *Rev Esp Ortod*. 2010;40(9):24.
13. Evangelista K, de Faria KV, Bumann A, et al. Dehiscence and fenestration in patients with Class I and Class II Division 1 malocclusion assessed with cone-beam computed tomography. *Am J Orthod Dentofacial Orthop*. 2010;138:133.e1–133.e7.

18

Nonextraction Treatment

Robert L. Vanarsdall, Jr., and Raffaele Spena

OUTLINE

The Original Cetlin Approach, 455
Phase Sequences, 456
Phase I: Space-Gaining Phase, 456
Upper Arch, 456
Palatal Bar, 456
Extraoral Force, 462
Clinical Management of Headgear, 462
The Removable Distalizing Plate, 464
Clinical Management of the Distalizing Plate, 464
Lower Arch, 466
Lip Bumper, 466
Class III Mechanics, 468
Phase II: Space-Utilization Phase, 468
Evolution of the Technique, 468
Reduction of Patient's Compliance, 468
Increase of Anterior Anchorage, 470
Reduction of Molar Resistance to Distalizing Forces, 470
Improvement of Appliance Efficiency, 479
Conclusions, 479

The decision of whether or not to extract permanent teeth in planning the treatment of an orthodontic case is a crucial moment for the clinician, especially when facing a borderline case. The education, experience, and skill of the clinician on one side and the requests or the limitations imposed by the patient on the other side may greatly influence the final solution and, eventually, the outcome.

Clinical research has so far failed to demonstrate that nonextraction treatment is better than extraction treatment (or vice versa). No difference in influencing the final dental and facial aesthetic result, no difference as far as the final functional result, and no difference in final stability have been established because of the great variability and unpredictability from patient to patient. Dogmas have been proposed, but most have proved to be inadequate.

The nonextraction approach proposed by Norman Cetlin has long been used by several clinicians with excellent clinical results. In the past few years, some modifications have been proposed in order to overcome the problems related to the headgear appliance and the distalizing plate, trying to reduce the level of patient compliance required.

New procedures and techniques have been adopted to improve control of the dental anchorage and movement, including mini-implants to ensure skeletal anchorage for both dental and orthopedic modifications, alveolar corticotomy to facilitate dental movement, active self-ligating brackets with new prescriptions associated to thermal NiTi wires trying to increase control of tooth movement in Class I, II, and III mechanics and reduce the level of applied force and friction produced.

This chapter is divided into two parts. In the first part, the original nonextraction approach is described. In the second part, modifications and implementations of the technique with the adoption of new appliances and protocols are presented.

THE ORIGINAL CETLIN APPROACH

In its original version, the Cetlin nonextraction approach was divided into two phases: (1) an initial space-gaining phase and (2) a final space-utilization phase. Sagittal and transverse corrections and major tooth movements were accomplished in the first phase, and full bracketing and final detailing were achieved in the second phase.

The objectives of the first, or space-gaining, phase were to increase space and change the original malocclusion into a "super Class I" malocclusion with ample spaces in both the upper and lower arches. Molar rotation, inclination, and crossbite were corrected, and the curve of Spee was leveled. In the second, or space-utilization, phase, Andrews' six keys to normal occlusion[1] were obtained, with some modifications as described. Overbite, overjet, and all dental malpositions were corrected. Spaces were closed, and a good anchorage was established during the entire treatment period. The final objective was a mutually protected occlusion with canine and incisal guidance.

This approach is most suitable for treating Class I and II malocclusions with upper and lower crowding. The best patients are those who are growing and who are in the late mixed dentition stage (i.e., right before the exfoliation of second deciduous molars). This stage offers several advantages:

- The leeway or "E" space is still available.
- Facial growth is at its peak.
- Because good cooperation is an essential factor, more can be obtained before the patient becomes socially and academically active.

With proper timing, treatment could be completed in 18 to 30 months, depending on how rapidly teeth erupt and skeletal changes occur. It should be noted that the procedures presented

in this system are viable and acceptable for both children and adults. A lesser and slower response generally is expected in adults, but the final result can be equally satisfactory.

Gaining space in the upper and lower arch is the key phase of any nonextraction treatment because crowding is very often found. The objectives of this initial part are (1) correction of molar inclination, rotation, and crossbite; (2) overcorrection of molar relationship; and (3) generalized spacing in both the upper and lower arch.

Space may be gained in the arch in several modalities. If we exclude surgical modalities (e.g., distraction osteogenesis, surgical assisted maxillary expansion) that have specific and limited indications, there are 10 ways to create space in an arch:

1. Distolateral rotation of mesially rotated and constricted upper molars (found in most of Class I and II malocclusions with crowding)
2. Distalization of upper molars (in both Class I and II malocclusions)
3. Distal and buccal uprighting of lower molars (as leveling of the curve of Spee occurs)
4. Distal rotation of ovoid premolars
5. Maintenance of the leeway space
6. Modification of the arch width (orthopedic/orthodontic transverse increase in the maxilla; orthodontic transverse increase in the mandible)
7. Modification of the arch form (an initial constricted, tapered arch form needs to be changed in the same way a rotated tooth needs to be corrected)
8. Selective stripping on trapezoid teeth or teeth with interproximal fillings or teeth with abnormal anatomy. In these cases, the Bolton ratio must be carefully evaluated.
9. Incisor repositioning (depending on periodontal, aesthetic, skeletal, and dental features of the patient)
10. Extraction of teeth (the last option)

Phase Sequences

The most commonly used treatment sequence in the space-gaining phase was as follows:

- Upper arch
- Crossbite correction, as well as rotation, distalization, and torque of upper molars with one or more palatal bars (PBs)
- Molar distalization
- Creation of a super Class I molar relationship
- Molar anchorage to allow spontaneous distal drift of premolars and canines
- Lower arch
- Insertion of a lip bumper on the first or second lower molars
- Achievement of an upright and rotated position of lower molars
- Constant reactivation of lip bumper to permit spontaneous lateral dentoalveolar growth and leveling of curve of Spee
- Use of Class III elastics from overcorrected upper molars to canine hooks on lip bumper to enhance uprighting force on lower molars

The most commonly used treatment sequence in the space-utilization phase was as follows:

- Upper arch
- Creation of a posterior anchorage unit
- Leveling and alignment of the upper arch
- Closure of residual spaces
- Detailing of the arch and occlusion
- Lower arch
- Correction of rotations and dental malpositions
- Complete leveling of curve of Spee
- Closure of residual spaces
- Detailing of the arch and occlusion

PHASE I: SPACE-GAINING PHASE

Upper Arch

Space was gained in the upper arch through the use of three appliances: the PB, the headgear, and the removable distalizing plate. The PB helped to correct maxillary constriction, rotation, distalization, and torque of both first and second upper molars; it also controlled them vertically. The extraoral forces (cervical or occipital, depending on the skeletal pattern) were used to control the roots and the vertical dimension of the upper molars.[2] The removable Cetlin plate, with two distalizing springs against the first molars, tipped the crowns distally while maintaining a good control of anterior anchorage. The combined actions of extraoral force and the plate created a two-force system that allowed bodily distal movement of the upper molars.

Space was gained in the sagittal dimension as well as in the transverse dimension. The premolars and canines followed the molars in a more distal and lateral position. The entire upper arch widened and spontaneously changed its form. The overjet often decreased because the upper front teeth tipped lingually in a better position and/or the mandible, released from a constricted upper arch, repositioned and grew forward.

Palatal Bar

Cetlin and TenHoeve[3] have modified the Goshgarian's anchorage appliance to make it a removable tooth-moving appliance. The PB is made of a 0.036-inch stainless steel (spring temper) wire, doubled back at the ends to be inserted in a 0.036- × 0.072-inch horizontal lingual sheath (Fig. 18-1, *A*). The PB incorporates a small "U-shaped" Coffin loop (Fig. 18-2), which is positioned generally toward the mesial for two reasons: (1) to make the PB more comfortable and (2) to improve vertical upper molar control because of forces exerted by the tongue during speech and swallowing anterior to the center of resistance (CR) of molars, an effect on upper molars only when the PB is distant from the palate and low in the oral cavity.

Clinical Use of the Palatal Bar. The PB is used on both first and second permanent molars. Second molars should be banded and moved with the PB as soon as possible to facilitate distal movement of the first molars.

Forces applied with a PB must be light and in one direction. For example, rotation and torque should not be attempted at the same time. Overactivation leads to soreness, mobility, destruction of the lamina dura, and periodontal breakdown and does not produce results more quickly. Reactivation is required approximately every 6 weeks. Terminals must always be checked to ensure that they are passive to their tubes before additional force is added.

Palatal bars must be tied. This can be done with an elastic or chain from the hook of the sheath to the curved end of the bar. Ligatures prevent the PB from being dislodged or, worse, swallowed.

Palatal bars can be used for the following purposes:

- Distalization
- Rotation
- Expansion or constriction

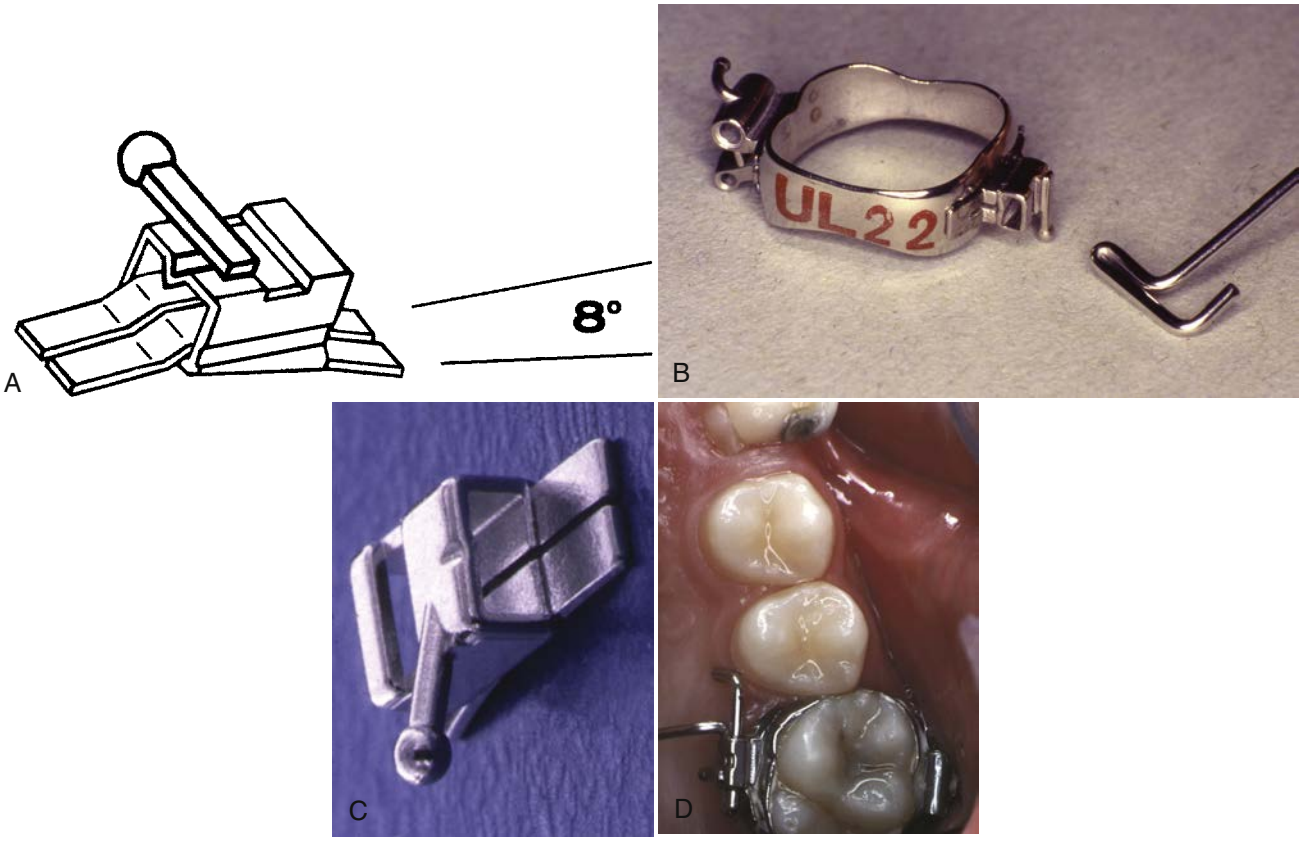


FIGURE 18-1 A, A lingual sheath holds the palatal bar (8-degree medial offset). B, The lingual sheath is welded to the maxillary first molar band; a gingival hook is used. C, A close-up distal view of the lingual sheath. D, A maxillary left molar with a palatal bar held in place with an elastic attached to gingival hooks.

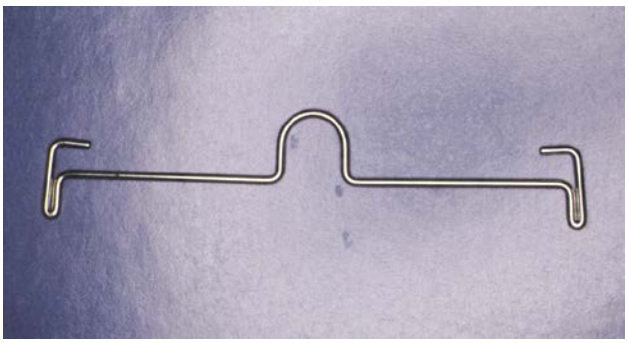


FIGURE 18-2 A transpalatal bar with a U-loop positioned generally toward the mesial.

- Vertical control
- Torque
- Anchorage increase

Distalization. Distalization is the only unilateral activation of the PB. It can be used in two clinical situations: (1) when, in a malocclusion, there is a Class II molar relationship on one side and the other side is normal or (2) when both upper molars must be distalized and the patient does not want to wear headgear.

In the first situation, rotation on the Class I side is offset with a headgear with an inner bow that has a toe-out on this side or an edgewise wire extended at least to the controlateral premolar (Figs. 18-3 to 18-8).

Rotation. L.F. Andrews[1] (see Chapter 14) has observed that molar relationships in untreated normal patients are defined by three contacts between the upper molars and lower molars: (1) the mesiobuccal cusp of upper first molars occludes with the mesiobuccal groove of lower first molars, (2) the distal marginal ridge of upper first molars contacts the mesial marginal ridge of lower second molars, and (3) the palatal cusp of upper first molars occludes with the central fossa of lower first molars. Most Class I and Class II malocclusions present mesiolingually rotated and constricted upper molars. When the upper first molars are rotated, several problems may occur:

1. The relationship between the upper first molars and the lower molars on the buccal side is different from that on the palatal side. Molar relationships always look worse in the buccal view. If the palatal cusps still seat in the central fossa, the faulty molar relationship is easier to correct; however, if occlusion occurs mesial to the central fossa, the Class II molar relationship is more difficult to solve.
2. The upper first molar distal surface faces buccally, and second molars tend to erupt laterally.
3. If the upper molars are banded, archwires or headgears may be difficult to insert.

An upper first molar measures approximately 10 mm from mesial to distal. From its mesiobuccal corner to its distopalatal corner, it measures 13 mm. For this reason, distal rotation of upper molars may gain as much as 3 mm of space per side. Furthermore, several false Class II relationships (i.e., those in which

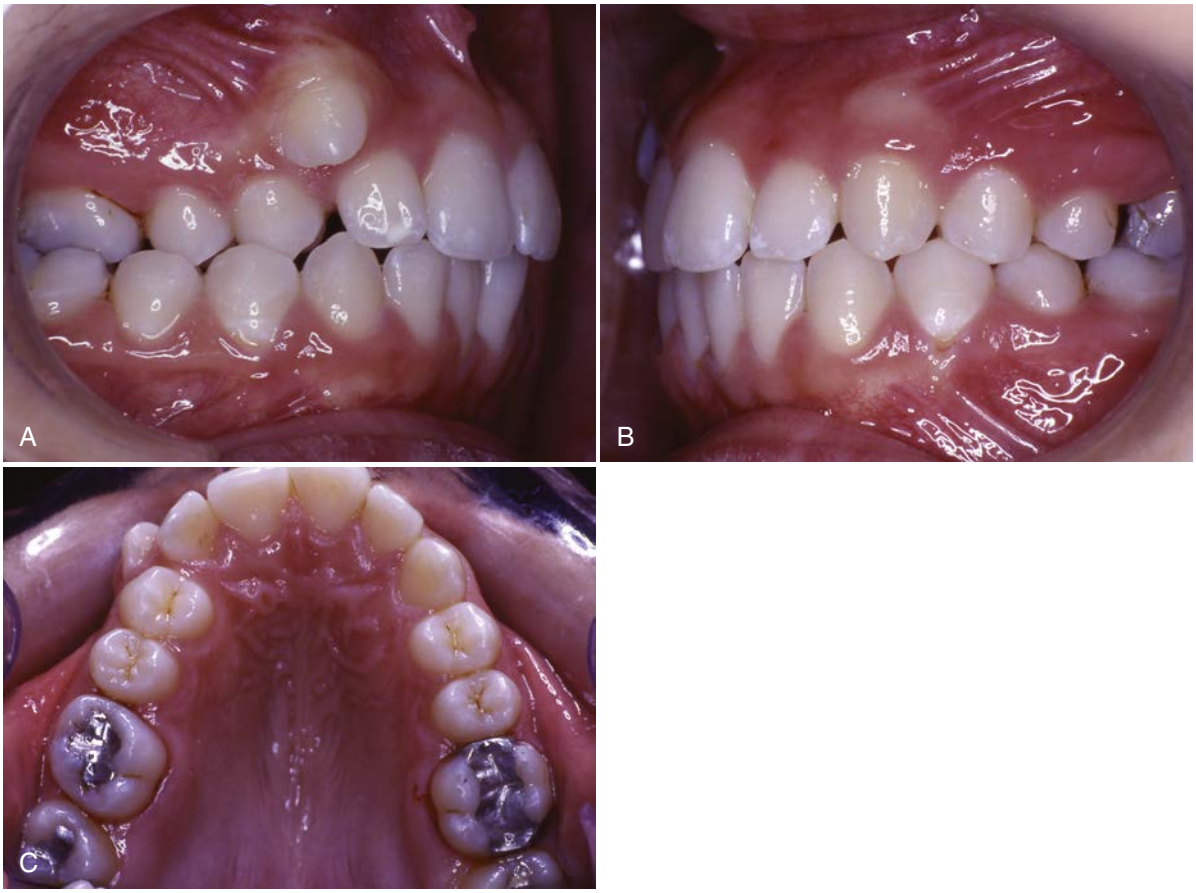


FIGURE 18-3 A–C, A malocclusion with Class II molar relationship on the right side and Class I molar relationship on the left side. A unilateral crossbite is present on the right side.

the palatal cusp of the upper molar sits in the central fossa of the lower molars) may be resolved simply by rotating the upper molars distally.

Upper second molars are usually triangular. Rotating these molars helps gaining additional space in the upper arch and facilitates distalization of the first molar. They therefore should be banded and moved as soon as possible. If needed, an extraoral force should be added on the first molars as soon as it can be easily inserted.

As a final objective, whereas the upper first molars occupy a wider part of the arch, the upper second molars are more lingually placed. For this reason, the activation of the PB on the first molars is different from the activation on the second molars. The premolars usually follow molars laterally in a distal direction because of the transseptal fibers. It should take 3 to 4 months to rotate the upper molars completely.

Expansion or Constriction. The PB can solve transverse problems such as crossbite and lateral overjet. These corrections should be made before rotating or distalizing the molars. Activation should be in the range of 1.0 to 1.5 mm of expansion or constriction per side and per activation until the problem has been corrected. If bodily movement is required, buccal-root torque (when expanding) or lingual-root torque (when constricting) must be added.

To correct a unilateral crossbite, a vertical elastic (to maintain occlusion on the normal side) and a cross elastic (to increase lateral movement of the abnormal side) are added to the expanded PB (Fig. 18-9). Premolars usually follow upper molars in the lateral movement, thus spontaneously correcting their position. In young patients, expansion with a PB may cause the palatal suture to open.

Vertical Control: Intrusion. Vertical control of upper molars is a peculiar aspect of this nonextraction mechanics. Most nonextraction approaches deal nicely with molar distalization but afford little or no vertical and transverse control. Control is needed in almost any case, but it is mandatory if nonextraction treatment is to be used in cases with a dental or skeletal open bite tendency.

The PB, alone or with a high-pull headgear, can be used to take advantage of the intrusive force exerted by the tongue during chewing, swallowing, and speech. The PB is kept low in the oral cavity, 4 or 5 mm away from the palatal vault. To increase the surface on which the tongue will be acting, two extra loops (Fig. 18-10) or an acrylic button can be added to the Coffin loop; this makes the PB more comfortable and effective.

By controlling molar eruption, or even more by intruding the molars, the orthodontist can correct or prevent vertical problems, obtain an upward and forward rotation of the mandible, and correct sagittal problems. In mixed dentition, the effect can be enhanced by grinding the occlusal of the primary molars (Fig. 18-11) as the upper permanent molars are kept away from the occlusion.

In a study conducted at the University of Ferrara, 13 male and 12 female patients, with an average age of 7.3 years (range, 6.7 to 8.5 years), with an anterior open bite due to various causes (i.e., thumb sucking, oral breathing, tongue thrust, and so on)

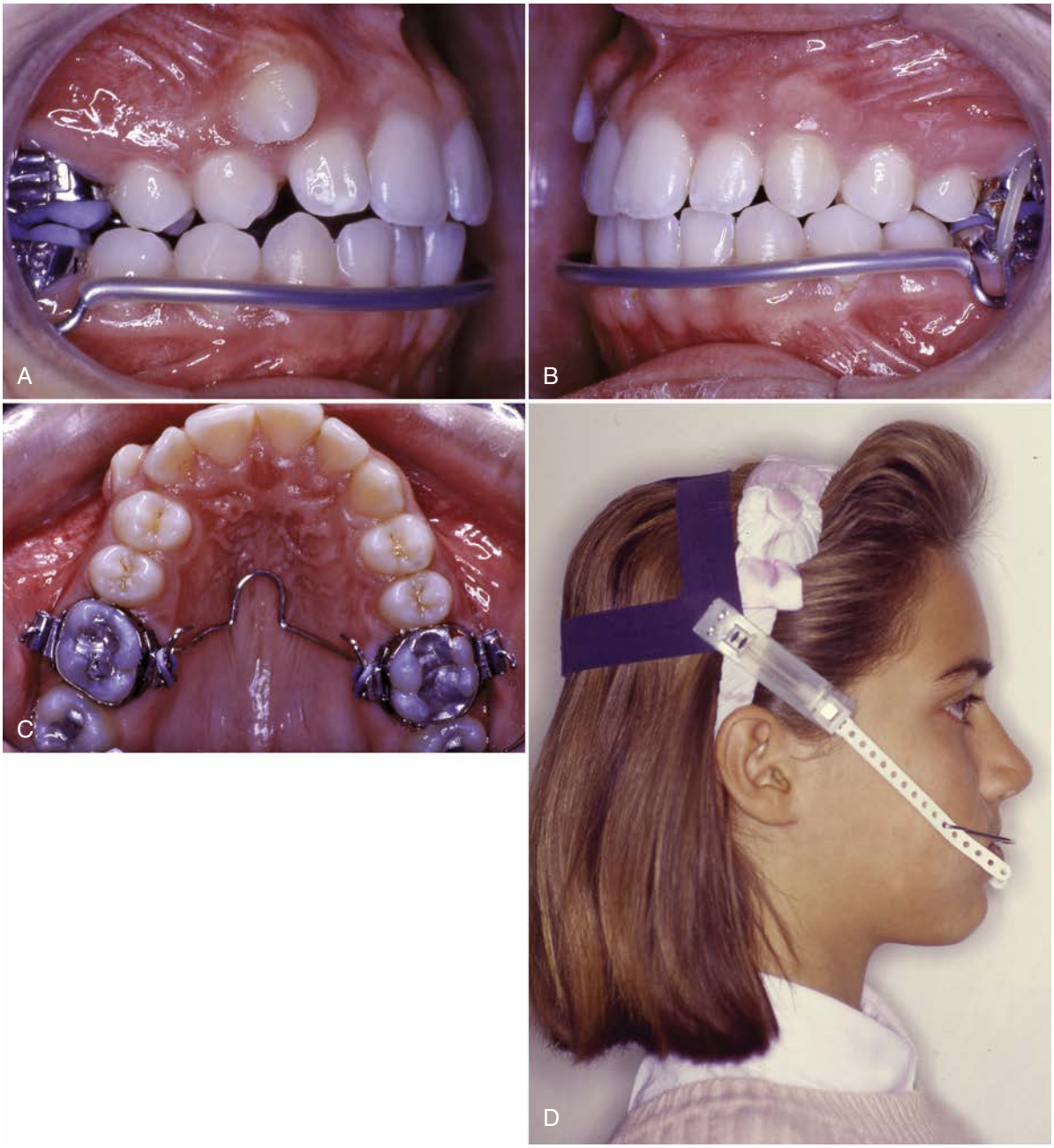


FIGURE 18-4 A–D, An expanded palatal bar with distal force on the right side is inserted. The lateral force is offset on the left side with a vertical elastic, and the rotational force is offset on the left side with the extraoral force.

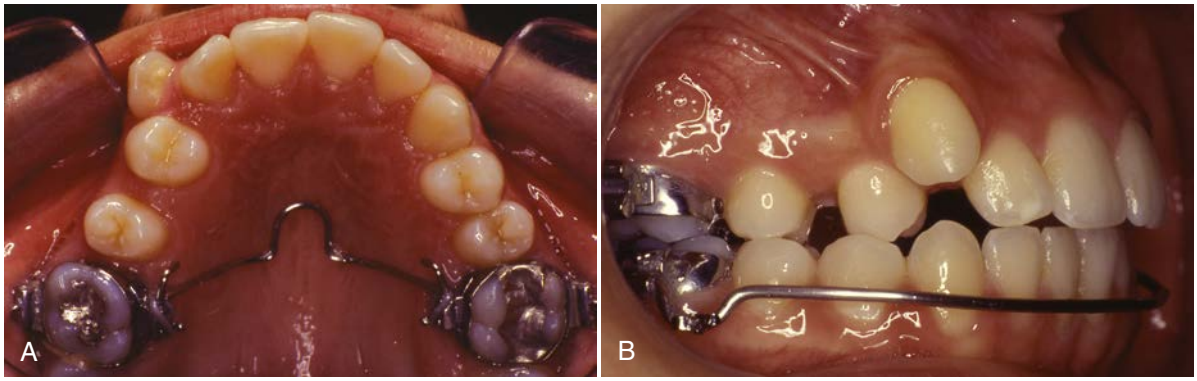


FIGURE 18-5 A, After a few activations, the upper right molar has been distalized and moved laterally to correct the Class II relationship and the crossbite. B, In the second situation, the palatal bar is activated first on one side and then on the other to obtain as much distalization as possible without the use of headgear or distalizing plate (see Figs. 18-7 to 18-9).

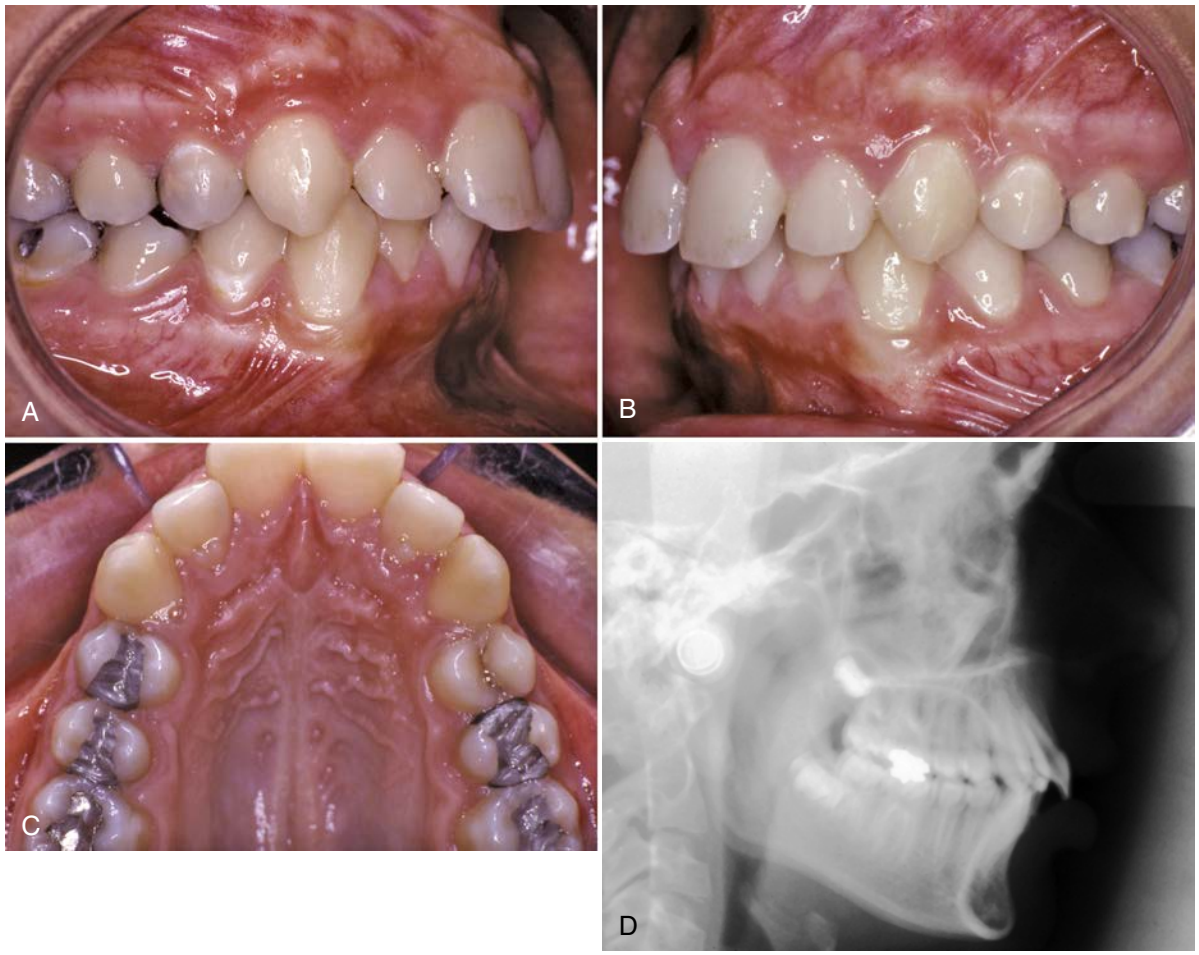


FIGURE 18-6 A–C, A malocclusion with Class I molar relationship, bimaxillary protrusion and crowding, and increased overjet and overbite (D).

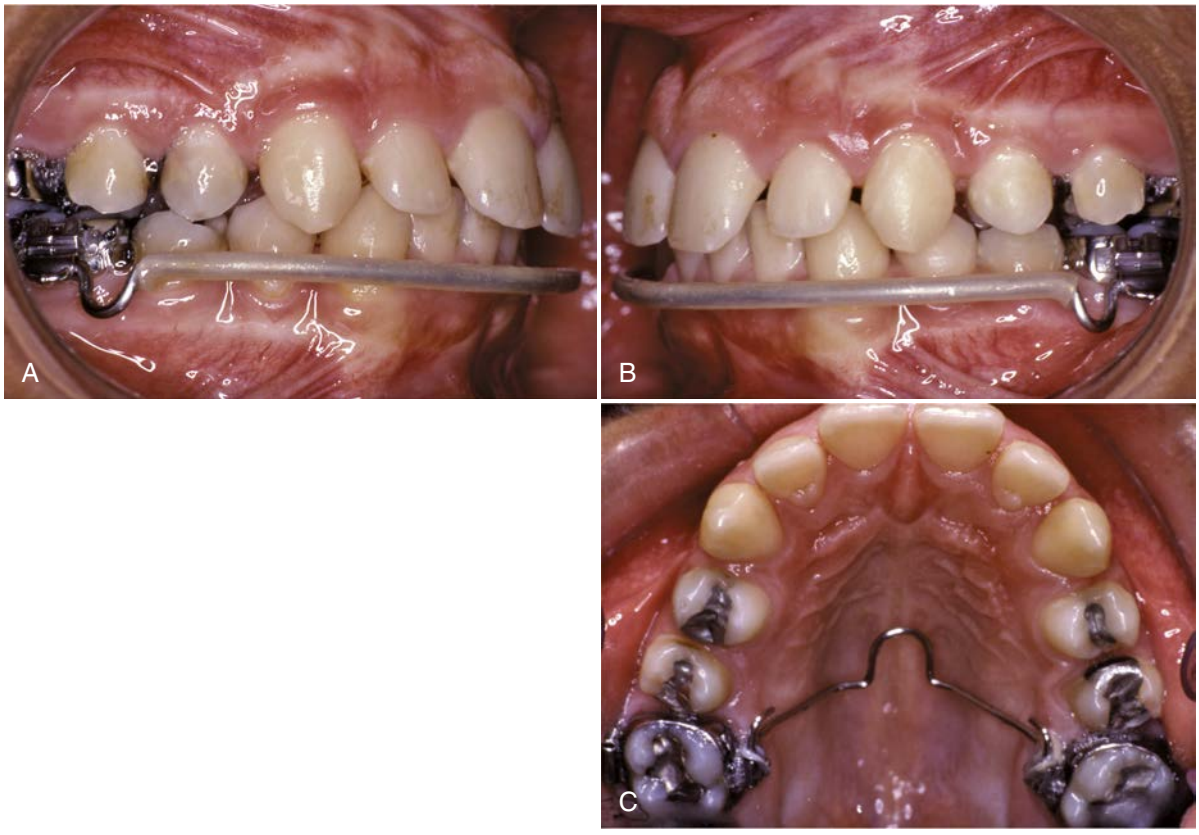


FIGURE 18-7 A–C, A palatal bar with distal force first on one side and then on the other has been used. Note the changes in the sagittal as well as in the transverse dimension.

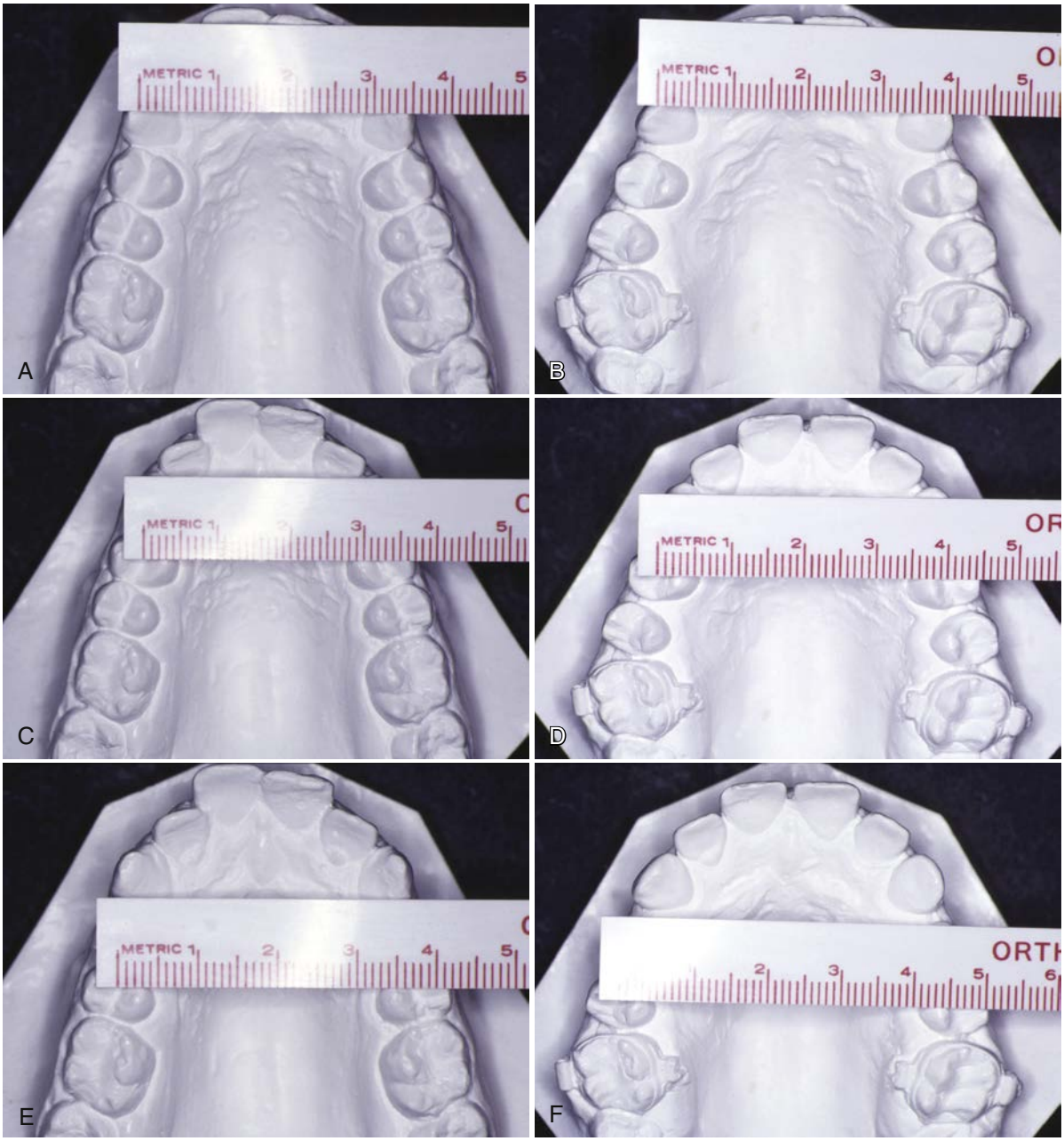


FIGURE 18-8 A–F, Molar, second premolar, first premolar, and canine widths before and after the distalization of the upper first molars.

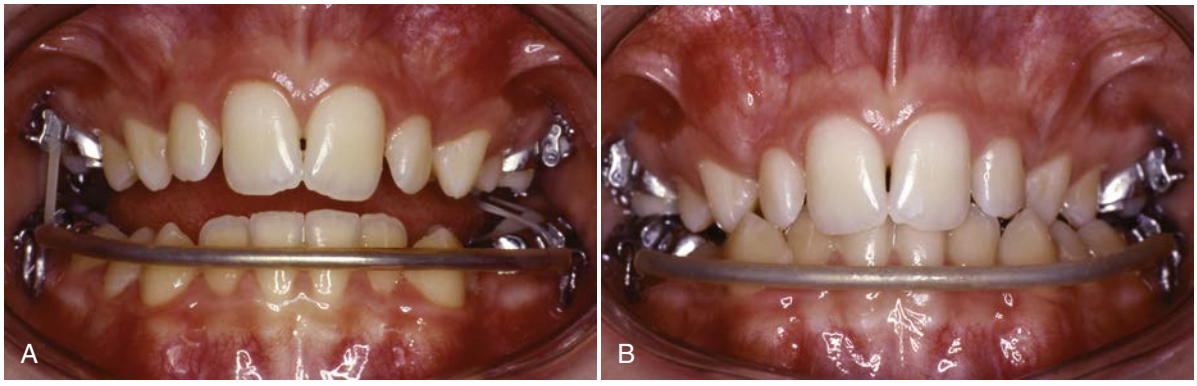


FIGURE 18-9 A, View of a transpalatal bar used to correct unilateral crossbite. Normally, a vertical elastic is used to maintain occlusion, and a cross-tooth elastic is used on the crossbite side to increase lateral movement of the upper molar. B, After correction of the crossbite.

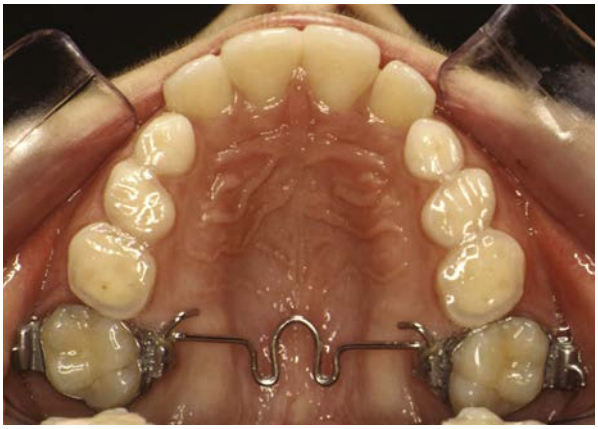


FIGURE 18-10 Occlusal view of two extra loops that can be added to the Coffin loop on a low palatal bar for comfort.

FIGURE 18-11 In mixed dentition, the orthodontist can grind the primary molars to prevent molar eruption.

and a skeletal divergence were treated with a low PB, a high-pull headgear and progressive grinding of the primary molars to reestablish an occlusal contact between the upper and lower first molars. Treatment was carried out until the anterior open bite was closed (Figs. 18-12 and 18-13).

At this time, treatment was discontinued, and patients were reevaluated. Pretreatment and posttreatment cephalometric mean values revealed a reduction of both sagittal and vertical skeletal problems. Functional improvements were observed in all subjects. Space was gained in both the upper and lower arches in all cases. Changes during the interceptive treatment helped in a subsequent definitive phase II treatment to finish the cases without extracting teeth (Tables 18-1 and 18-2).

Torque. Several mechanics tend to extrude or to make more prominent molar palatal cusps (i.e., high-pull headgear, expansion, intrusion arches). Other mechanisms tend to extrude buccal cusps (i.e., cervical headgear, constriction). The PB allows an excellent control of upper molar torque. Buccal root torque is more often required because it helps keep palatal cusps high and away from occlusal interference (Fig. 18-14). This third order bend tends to extrude the upper molars. If control of the extrusive component is needed, the PB should be kept low and/or a high-pull headgear could be added to the upper molars.

Anchorage. After space has been created, control of the upper molars is crucial. In this biomechanical sequence, three-dimensional control is achieved with the BP alone or with the use of extraoral forces.

Extraoral Force

In this nonextraction approach, extraoral forces were used during most of the treatment. During the space-gaining phase, headgear was applied with the PBs and eventually with the removable plate to distalize upper molars in a bodily fashion. During the second phase, when the spaces were used to finish up the case, extraoral forces helped to maintain posterior anchorage. The two types of extraoral forces were:

1. The occipital force (high-pull headgear) used in all those cases where molar vertical control was important (i.e., malocclusions with dental open bite tendencies, hyperdivergent skeletal patterns, and biomechanical systems that tended to extrude upper molars). When used with the Cetlin plate, the high-pull headgear allowed a backward and upward bodily movement of the molars (Fig. 18-15, *A* and *B*).
2. The cervical force (low-pull headgear) used in cases involving a dental deep bite and a skeletal hypodivergent pattern or, generally, when vertical control of the upper molars was not crucial. When this appliance was combined to the removable plate, the molars were moved bodily backward and downward (Fig. 18-16, *A* and *B*). Forces of occlusion might limit the amount of extrusion, and favorable condylar growth could compensate for molar extrusion.

Clinical Management of Headgear

Extraoral forces were applied to upper first permanent molars as soon as the molars had been rotated with PBs, when facebows could be easily inserted into molar tubes. When headgear was used together with a PB, the inner bows had to be passive to molar tubes. When the headgear was used alone, the inner bows of the high-pull device should have been slightly constricted, but those of the low-pull device should have been expanded to prevent the upper molars from rolling out or in.

Facebows had to lie on lower lip. In this way, when traction units were applied, the facebow lay right at the level of labial rim and was comfortable for the patient (Fig. 18-17). If the facebow had two canine hooks on its inner bows, a light elastic (L10) could be applied to fit on the labial screen of the Cetlin plate to increase anterior anchorage.

Extraoral forces had to be about 150*g* per side to exert orthodontic and not orthopedic effects. The devices had to be worn at least 12 to 14 hours a day, and at each appointment, the headgear had to be checked to ensure that it was applying the desired force system. The facebow had to be kept a safe distance from the upper front teeth and the screen of the plate. For the patient's safety, the neck pad and the occipital cap had to have breakaway traction units.

The Class II relationship was corrected by moving the maxillary teeth distally until the patient closed in a super-, or overcorrected, Class I occlusion and mandibular forward growth had occurred. The maxillary molars were distalized initially, and the premolars and anterior teeth were treated later. Distalization was accomplished by the combination of (1) headgear and a PB or (2) headgear and the Cetlin Distalizing Plate.

Distalization was accompanied by rotation or overrotation of the molars, which significantly improved intercuspation (this technique is described in the following section).

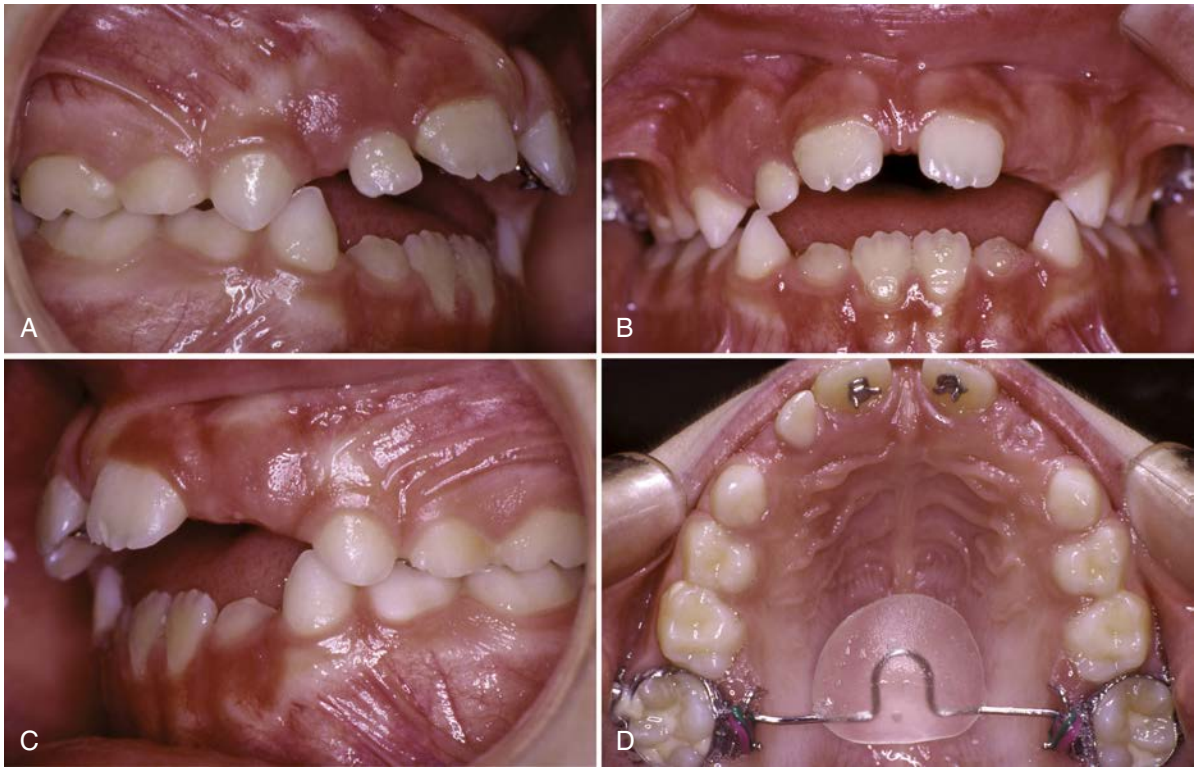


FIGURE 18-12 A–D, Treatment of the open bite in early mixed dentition. A low palatal bar with an acrylic pad around the Coffin loop to enhance upper first permanent molars' vertical control. Lingual cleats have been bonded on the lingual surface of the upper incisors to discourage thumb sucking and as a reminder for tongue positioning during swallowing and speech.

FIGURE 18-13 A–D, Bite closed after treatment with palatal bar, high-pull headgear, and grinding of the deciduous molars and canines.

TABLE 18-1 Pretreatment and Posttreatment Average Cephalometric Values Found in the Female Group

Female	Pretreatment Mean ± SD	Posttreatment Mean ± SD
Sagittal		
SNA	82 ± 3.92 degrees	81.31 ± 3.73 degrees
SNB	76.92 ± 5.0 degrees	77.62 ± 4.35 degrees
ANB	5.08 ± 2.32 degrees	3.69 ± 2.02 degrees†
MDB body/ACB	0.83 ± 0.17	0.97 ± 0.04
Vertical		
SN/ANS-PNS	8 ± 3.46 degrees	8.92 ± 3.04 degrees
ANS-PNS/GoGn	32.38 ± 3.23 degrees	30.54 ± 2.60 degrees*
SN/GoGn	40.38 ± 4.11 degrees	39.38 ± 4.07 degrees*
PFH/AFH	0.61 ± 0.05	0.61 ± 0.04*
SN/ArGo	87.62 ± 5.33 degrees	89.23 ± 5.31 degrees
Gonial angle	134.77 ± 3.00 degrees	133.85 ± 2.58 degrees
LAFH	67.38 ± 2.60 mm	67.69 ± 2.32 mm
Dentobasal		
1/ANS-PNS	115.38 ± 8.66 degrees	111.85 ± 4.29 degrees
1/GoGn	93.92 ± 9.78 degrees	97.23 ± 12.21 degrees
Dental		
Overjet	4.92 ± 3.05 mm	3.27 ± 2.16 mm
Overbite	(–)4.19 ± 2.63 mm	1.5 ± 1.22 mm*
Interincisal	119.15 ± 11.17 degrees	124.31 ± 7.62 degrees

SD, Standard deviation.
*Wilcoxon analysis with statistical significance of $p < 0.01$.
†Wilcoxon analysis with statistical significance of $p < 0.05$.

TABLE 18-2 Pretreatment and Posttreatment Average Cephalometric Values Found in the Male Group

Male	Pretreatment Mean ± SD	Posttreatment Mean ± SD
Sagittal		
SNA	81.91 ± 3.98 degrees	82.33 ± 2.93 degrees
SNB	77.41 ± 4.79 degrees	79.75 ± 3.55 degrees*
ANB	4.5 ± 1.51 degrees	2.58 ± 0.99 degrees*
MDB body/ACB	0.84 ± 0.16	0.96 ± 0.05
Vertical		
SN/ANS-PNS	8.25 ± 3.36 degrees	9 ± 2.34 degrees
ANS-PNS/GoGn	31.33 ± 3.11 degrees	29.58 ± 3.12 degrees*
SN/GoGn	39.58 ± 4.58 degrees	38.58 ± 4.14 degrees*
PFH/AFH	0.61 ± 0.05	0.62 ± 0.05
SN/ArGo	86.58 ± 4.1 degrees	87.42 ± 4.19 degrees
Gonial angle	132.16 ± 3.37 degrees	131.33 ± 2.74 degrees
LAFH	65.33 ± 3.31 mm	65.25 ± 2.38 mm
Dentobasal		
1/ANS-PNS	117 ± 7.43 degrees	112.25 ± 4.71 degrees
1/GoGn	94.5 ± 10.3 degrees	94.25 ± 9.59 degrees
Dental		
Overjet	4.96 ± 2.72 mm	2.63 ± 1.58 mm
Overbite	(–)4.5 ± 2.15 mm	1.63 ± 0.57* mm
Interincisal	118.33 ± 10.97 degrees	123.83 ± 7.59 degrees

SD, Standard deviation.
*Wilcoxon analysis with statistical significance of $p < 0.01$.

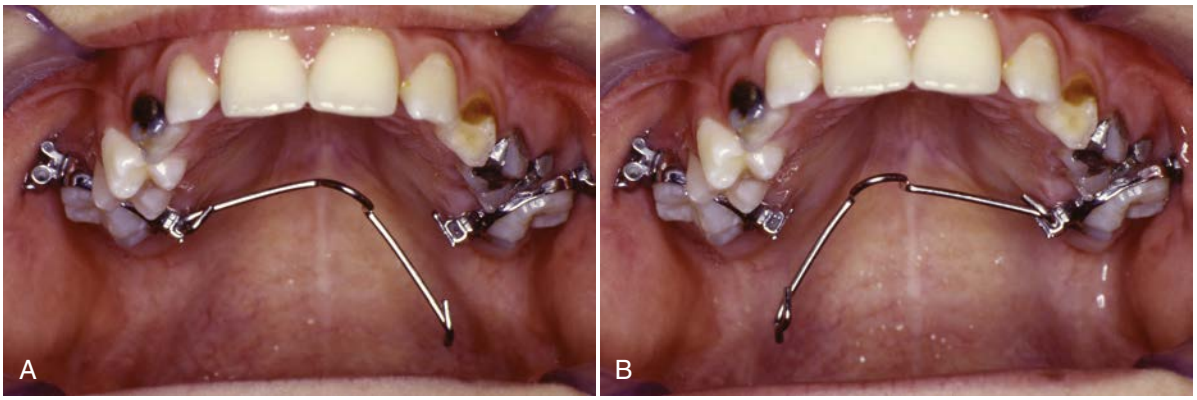


FIGURE 18-14 **A,** To achieve palatal root torque, both terminals are twisted so that when inserted into the right molar, the left (or opposite) terminal stands low to its sheath. **B,** When the transpalatal bar is inserted into the opposite side, it stands low to the right side.

The Removable Distalizing Plate

The removable distalizing plate was used to continue distalization of the upper molars when a "super Class I" relationship could not be obtained using only PBs and headgear. This plate was designed (Fig. 18-18) to apply a gentle, constant force of approximately 30*g* to the upper first permanent molars with minimal reaction on the upper front teeth. However, its forces might tend to incline molar crowns distally and extrude the molars. For this reason, it always had to be used in conjunction with an extraoral force to control molar roots, ensure vertical control, and thus obtain the desired distal bodily movement of the molars.

Clinical Management of the Distalizing Plate

The distalizing plate had to be worn 24 hours a day except for meals and hygiene. It always had to be used with the extraoral force to control molar roots and obtain a distal bodily movement. The springs had to be very lightly activated. An activation of 2.0 to 2.5 mm per side gave approximately 30*g* of distal force either bilaterally or adjusted on alternate sides, as it was done with the PB. Greater activation only increased the chance of molar tipping and loss of anterior anchorage, resulting in worsening of the overjet. Excessive spring force could dislodge the plate or cause it to fit loosely, exerting little distalizing force. The arm of the spring had never to

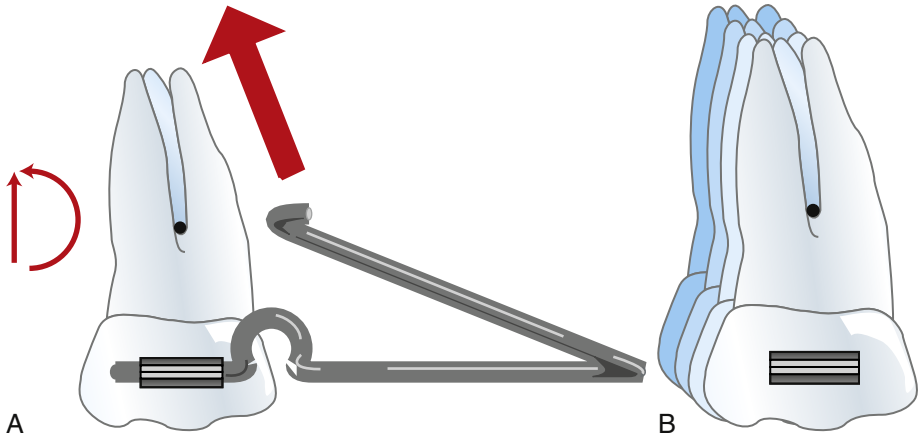


FIGURE 18-15 A, When the high pull is applied to upper first molars by means of a facebow with the outer bow the same length as the inner bow, the point of force application and the line of force lie above the center of resistance of the upper molar. B, When used with the Cetlin plate, the high-pull headgear allows a backward and upward bodily movement of the molars.

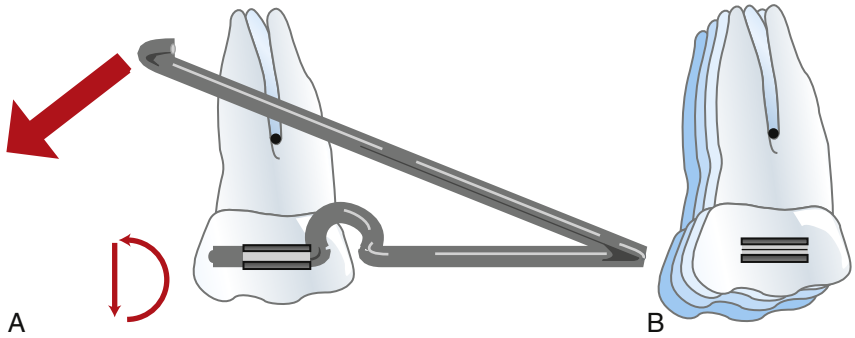


FIGURE 18-16 A, In cervical gear traction force the point of force application and the line of force lie above the center of resistance of the upper molar. B, When this appliance is combined to a removable plate, the molars are moved bodily backward and downward.

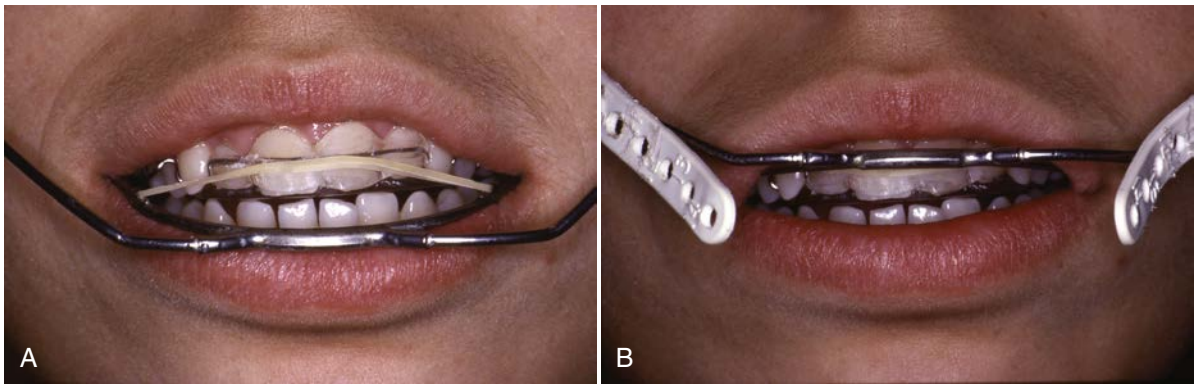


FIGURE 18-17 A, Facebow adjusted to lie on the lower lip. An elastic extends from the inner headgear bow to the labial aspect of the removable appliance to increase anterior anchorage. B, When traction is applied, the facebow goes to the level of the labial rim, which is more comfortable for the patient.

be bent buccally around the molars because this prevented spontaneous lateral movement of the molars. The anterior bite plane had to articulate with as many lower front teeth as possible and provide a small disclusion of posterior teeth (Fig. 18-19, *A* and *B*). It was not to be included in open-bite or hyperdivergent cases.

The acrylic of the plate had never to limit the distal movement of the molars and second premolars. After the first permanent molars had been brought to a super Class I relationship, the Adams clasps were removed, and the acrylic palatal to the canines and first premolars was trimmed to allow spontaneous distal movement of these teeth through the action of the transeptal fibers, with no strain on the anchorage. The patient had to continue to wear the plate with springs that were passive against the molars. Retention was provided by the labial shield, which was relined with cold-cure acrylic (Fig. 18-19, D).

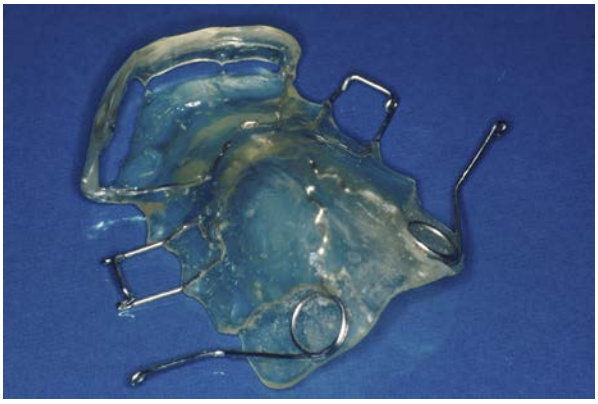


FIGURE 18-18 The removable distalizing plate: anterior biteplate, molar distalizing springs, Adams clasps for the first premolars or deciduous molars, and labial screen, which covers the incisors.

To increase sagittal control of the upper front teeth, a groove was cut into the labial shield to engage a light anterior elastic attached to the inner bow of the headgear.

Lower Arch

Space gaining in the lower arch was accomplished with the lip bumper.

The objectives of the first phase of Cetlin's nonextraction approach in the lower arch were accomplishing rotation and upright positioning of the lower molars, leveling of the curve of Spee, encouraging lateral growth of the arch, and creating space to align all the teeth.[3]

Lip Bumper

The lip bumper is a fixed functional orthodontic appliance. It works by altering the equilibrium between cheeks, lips, and

FIGURE 18-19 A–E, Class II malocclusion with the upper canine blocked out of the arch. Distalization of the upper molars with a palatal bar, followed by the Cetlin distalizing plate and a cervical headgear. After having distalized the molars, the plate is left in place to prevent the upper molars from moving forward. A palatal bar is reinserted to allow spontaneous drift of all the teeth anterior to the distalized molars.

tongue and by transmitting forces from perioral muscles to the molars where it is applied (Fig. 18-20, *A–C*). The lip bumper has been used for various purposes:

- Molar anchorage[4]
- Therapy of habits[5,6]
- Space gaining in the lower arch.[7,8] The differences in results, which were published in the orthodontic literature,[9,10] probably are related to the fact that several types of lip bumpers are available, and they can be used in various ways.[11–13] Cetlin and TenHoeve[3] have described a lip bumper that is easy to use and very effective. All three purposes listed previously can be obtained with it. If used for an appropriate length of time, this lip bumper can help gain an incredible amount of space in the lower arch while maintaining good control of the molars and incisors (Fig. 18-20, *D* and *E*).

Characteristics of the Lip Bumper. The lip bumper was essentially composed of a 0.045-inch stainless steel wire that runs in the lower vestibule from molar to molar between teeth and lip and cheeks inserted in 0.045-inch molar tubes with 4-degree mesial offset to facilitate insertion.

Cetlin[3] has described two different lip bumpers. One type, which has been used since the early days of his practice, is custom made for each patient. It has two loops at the molar level that allow modifications of the appliance during therapy. The second type is preformed and available in different sizes (Fig. 18-21). It has four loops, two at the molar level and two at the canine level. The two additional loops give a better shielding effect in the canine region and allow the use of Class III elastics in more severe cases.

Fitting the Lip Bumper. The lip bumper must keep cheeks and lip away from the lower dentoalveolar area, and this shielding effect must be verified at each appointment. The lip bumper should not exert any expansion or contraction on the molars. It must be easy for both the clinician and the patient to insert and remove. As the arches anterior to the molars respond to the reshaping and widening of the lip bumper, they take on a wider natural arch form.

Guidelines are given next for obtaining an optimal adaptation of the appliance:

1. **Transverse position:** The wire must be 2.0 mm from the lower canines and 3.0 to 4.0 mm from the premolars. Protection of the canine area is crucial, and the four-looped bumper definitely is more effective.
2. **Sagittal position:** The lip bumper should not be more than 1.0 to 2.0 mm away from the labial surface of the lower incisors. The position offers good support of the lower lip for the anterior seal without rendering the appliance uncomfortable.
3. **Vertical position:** In the lateral segments, the wire must be positioned generally at the middle third of premolar and canine crowns. In the severest cases, in which good vertical control is necessary, the bumper can be adapted to rest deeper in the vestibule. The cheeks override the bumper

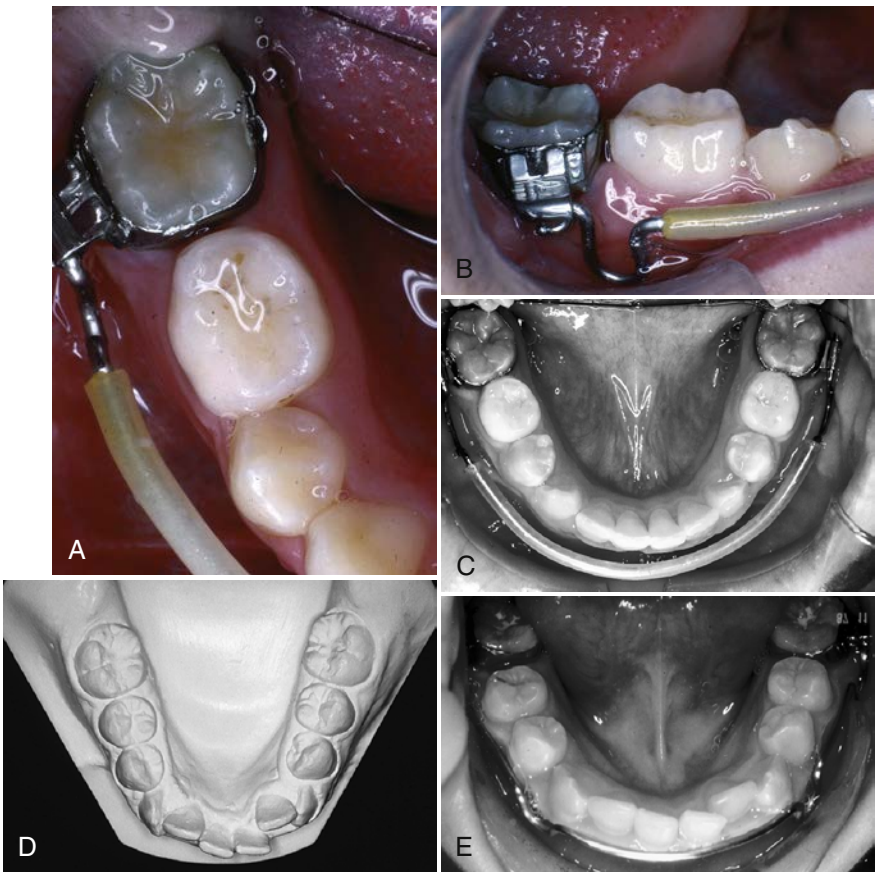


FIGURE 18-20 A, Occlusal view of lower right molar and lip bumper. B, Buccal view of lower right molar with lip bumper in place. C, Occlusal view of lip bumper placement in the mandibular arch. D, The mandibular arch before treatment. E, The same lower arch after 11 months of lip bumper wear. Note the space gained from dentoalveolar widening and reduction of incisor crowding.

during function, producing an intrusive force on the lower molars. In the anterior region, depending on the overbite, the bumper can be positioned at three different levels with respect to the incisor crowns.

- **Incisal edge:** This position usually is used during the initial phase of treatment. It helps to upright mesially inclined molars because the lower lip tends to lift the anterior part of the bumper, creating a long lever effect on the molars.
- **Middle third:** This is the position to use when a shielding effect on the incisors is desired. The lower lip is kept away from the teeth, altering the equilibrium in favor of the tongue. The incisors slowly translate labially.
- **Gingival level:** This level is used when the orthodontist does not want to alter the equilibrium between centripetal and centrifugal forces. Because the incisors are still under the lower lip action, they maintain their position. The lip bumper must be kept very close to the incisors.

Activating the Lip Bumper. After space has been obtained in the lower arch and bonding of the lower arch has been planned in a few appointments, the bumper can be activated to correct the rotation of the lower molars. A slight lingual bend is placed in one terminal (or both, if necessary), adding approximately 1.0 mm of expansion to counteract the lower molar tendency to tip lingually.

In a few circumstances, the appliance can be expanded if the lower molars are lingually inclined. The activation should not exceed 1.0 to 1.5 mm per side.

Clinical Management of the Lip Bumper. The appliance must be worn 24 hours a day and should be removed only for meals and hygiene. Although patient adaptation may not be optimal during the first month, the appliance should be worn as much as possible during that time. The two-looped bumper has the shrink tubing that gradually discolors as the patient wears the device. If cooperation is still a problem after a few months, the appliance can be tied to molar hooks with an elastic chain.

If the appliance has been well fitted, a red line can be seen on the inside of the cheeks and the lower lip where the wire runs. If the lip bumper is too distant from the teeth, ulcers may appear. In such cases, the appliance is removed for a day or two, lesions heal, and treatment is restarted with an appliance that runs closer to the teeth. At each appointment, the bumper is checked to ensure that it is still passive on the molars and that it maintains the desired distance from the teeth. This prevents undesirable mechanical expansive forces on the molars. At the end of the space-gaining phase, use of the bumper is discontinued. If it is needed for anchorage problems, it is readapted so that it does not contact brackets, while not being too procumbent.

FIGURE 18-21 Preformed lip bumper with four loops: two adjustment loops mesial to each molar and two in the canine area. The additional loops provide a better shielding effect in the canine area and can be used for Class III elastics in more severe cases. The custom-made bumper (shown in Fig. 18-20, *C*) has only two loops (mesial to each molar).

Class III Mechanics

If the upper arch has been overcorrected and the upper molars are in super Class I relationship but space is still needed in the lower arch, the lip bumper's action can be enhanced by the use of light (2–3 oz per side) Class III elastics that are driven from the upper first molars to the bumper's hooks at the canine level. Anchorage on the upper molar can be controlled with a PB, an extraoral force, or both. If lower second molars are present, it is better to bond them and put a sectional between the first and second molars; otherwise, a discrepancy may result between marginal ridges.

PHASE II: SPACE-UTILIZATION PHASE

Intrusion and retraction of the upper incisors concluded the nonextraction mechanics of Cetlin's approach. Depending on the inclination of the upper front teeth, the appropriate system was used to correct overjet and overbite without risking loss of molar anchorage and achieved buccal correction.

EVOLUTION OF THE TECHNIQUE

The Cetlin mechanics, as it has been just described, had some limitations because good cooperation of the patient for most of the treatment was required, and the use of removable appliances has some inherent inefficiency. Problems have been encountered mainly with the use of (1) the distalizing plate that needed full-time wear to obtain a bodily distalization of the upper permanent molars as well as a continuous disclusion between the two arches to achieve mandibular growth, leveling of the lower curve of Spee, and lateral dentoalveolar growth in both the upper and lower molar and premolar area and (2) the headgear that, even if it has always been considered the home appliance to be worn afternoon and night as much as possible, has often been a difficult-to-accept appliance.

Modifications have become necessary to overcome these problems of compliance. Clinical research has been carried out in four major directions:

1. Reduction of patient's compliance
2. Increase of the anterior anchorage
3. Reduction of molar resistance to distalizing force
4. Improvement of the archwire–bracket interaction

Reduction of Patient's Compliance

In the early 1990s, several distalizing intraoral appliances were described in the literature. Among them, the Pendulum Appliance presented by Dr. Hilgers seemed to fit perfectly in the Cetlin mechanics. It has replaced the Cetlin Distalizing Plate and, partly, the headgear.

The original Cetlin mechanics has not been dramatically changed. The use of the PB as the initial appliance in the upper arch has been kept for several reasons:

1. Correction of the first permanent molar position (rotation, torque, and so on) is more easily achieved by the use of a PB and no anterior anchorage is required.
2. Starting molar movement with a PB and then applying distalizing forces with a pendulum may give better anchorage control and make the pendulum more effective.

The pendulum as described by Hilgers has been slightly modified to better satisfy the therapeutic needs. The original appliance is anchored on all four premolars. In this way, the second premolars are not free to move distally and laterally together with the first permanent molars as with the Cetlin Distalizing Plate. For this reason, anterior anchorage has been limited to the first premolars. The applied force to distalize molars has been notably reduced to the level of the force produced by the Cetlin Distalizing Plate. An uprighting and expansion force has been added as described by Byloff et al. In this way, molars are distalized in a more bodily fashion, and the second premolars are free to move distolaterally together with the first molars thanks to the transseptal fibers as in the original Cetlin mechanics (Fig. 18-22).

The sequence of treatment has gradually changed in the following way:

- Crossbite correction, as well as rotation, distalization, and torque of upper molars with one or more PBs
- Molar distalization with the modified pendulum appliance
- Creation of a super Class I molar relationship

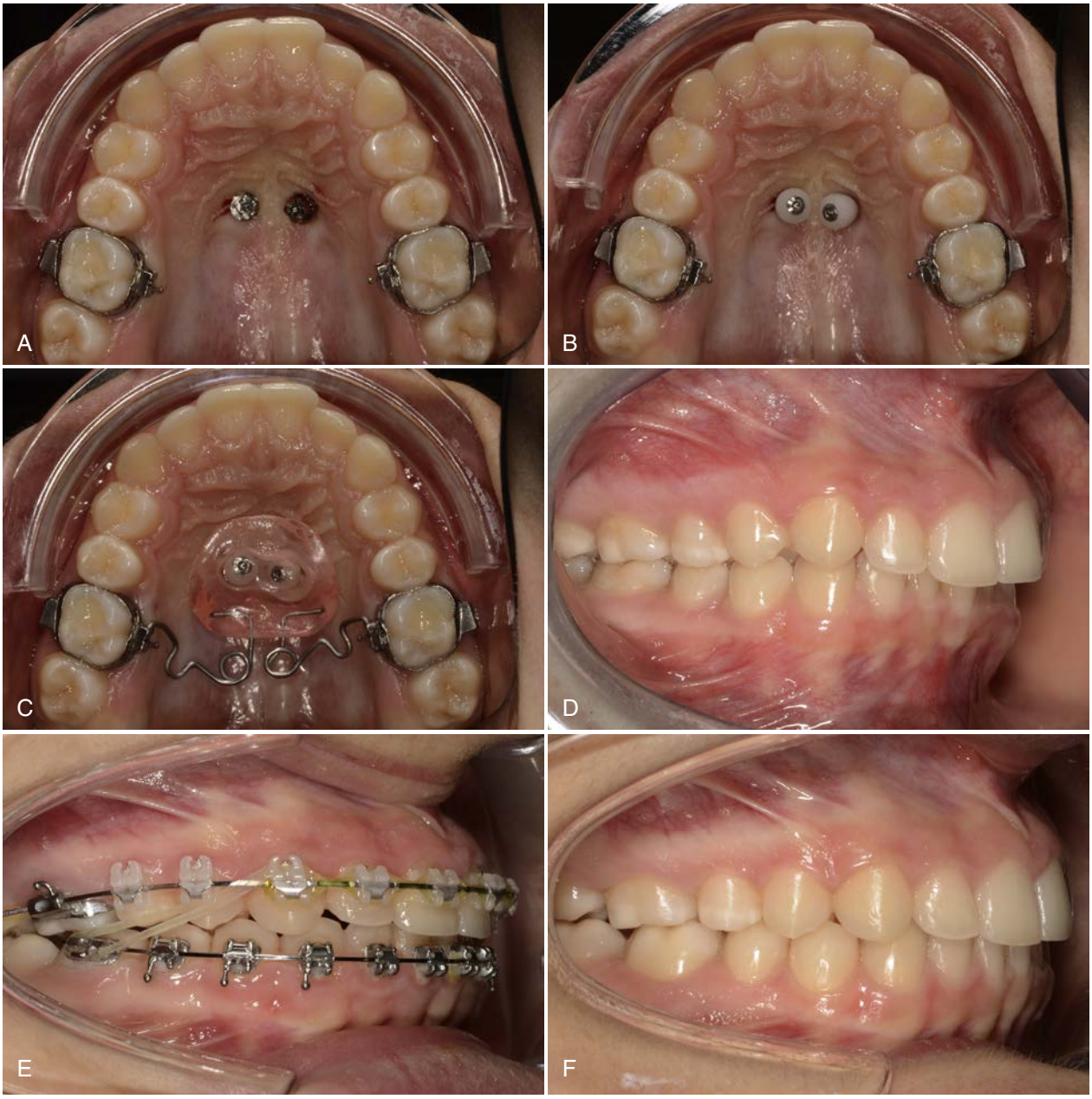


FIGURE 18-22 Pendulum with skeletal anchorage. A case in which molars were distalized with this type of appliance. **A–C,** Occlusal view of the Nance button screwed on the temporary anchorage devices, allowing easy insertion and removal for reactivation. **D–F,** Sagittal view of initial, progress, and final treatment.

- Molar anchorage and control with a PB with a "biscuit-like acrylic button" (Fig. 18-23) to upright and intrude molars and to allow spontaneous distal drift of premolars and canines

Several other similar appliances and procedures to distalize upper molars can be found in the current literature. They could be easily and effectively integrated in the Cetlin's nonextraction treatment of both growing and nongrowing patients.

Increase of Anterior Anchorage

The latest version of the Pendulum appliance is with two miniscrews placed in the palate in the first premolar region. The Nance button supporting the distalizing springs embeds two silicon caps that fit perfectly on the heads of the miniscrews. The Pendulum is placed and fixed onto the miniscrews with two small screws. In this way, it can be easily removed for spring reactivations. Skeletal anchorage helps with excellent control of the anterior teeth, allows spontaneous distal drift of all the premolars and canines, and allows earlier bonding of teeth for final positioning (Fig. 18-24).

Reduction of Molar Resistance to Distalizing Forces

Corticotomy has long been used in orthodontic treatment to accelerate dental movement and improve its efficacy and to reduce the appearance of undesired phenomena such as root resorption, loss of vitality, and relapse of the corrections carried out. In 2001, Wilcko et al.[14] published a case report in which corticotomy was used in conjunction with resorbable alloplastic grafts of demineralized freeze-dried bone to increase the amount of alveolar bone, regenerate the bone in the zone of dehiscence and fenestration, and avoid gingival recession resulting from expansion of the arches. These authors found that the fast orthodontic tooth movement was not due to repositioning of single tooth-bone units, as believed by Kole and Suya,[15] but to a cascade of physiologic events (area of transitory secondary osteoporosis and greatly reduced bone density) described by Frost as regional accelerated phenomena (RAP).

The surgical-orthodontic protocol, subsequently patented as Periodontally Accelerated Osteogenic Orthodontics (PAOO), claims as effects (1) accelerated tooth movement with reduction of the total treatment time, (2) osteogenic modifications with transportation of the bony matrix and final improvement of hard and soft tissue support of the teeth treated orthodontically, and (3) increase of the short- and long-term stability of the orthodontic treatment.

A large number of studies and case reports have been produced trying to give scientific evidence to these claims. As of today, six aspects seem to be important for effective and realistic use of alveolar corticotomy in an orthodontic treatment.

1. **Corticotomy has limited effect in time.** It seems that we can rely on a maximum 4 to 6 months of accelerated bone metabolism after decortication. For this reason, tooth movement may be accelerated only for a limited part of the therapy. Different aspects must be taken into account when we try to evaluate orthodontic treatment time.[16-19] A randomized clinical trial in which similar patients are treated with or

Text continued on page 478

FIGURE 18-23 Class II, division 1 malocclusion in adult patient treated with segmental corticotomy performed on both buccal and palatal sides limited to the upper first and second molars. Molars were distalized with NiTi coils compressed between first molars and second premolars with no anterior anchorage. **A–D,** Initial intraoral pictures.

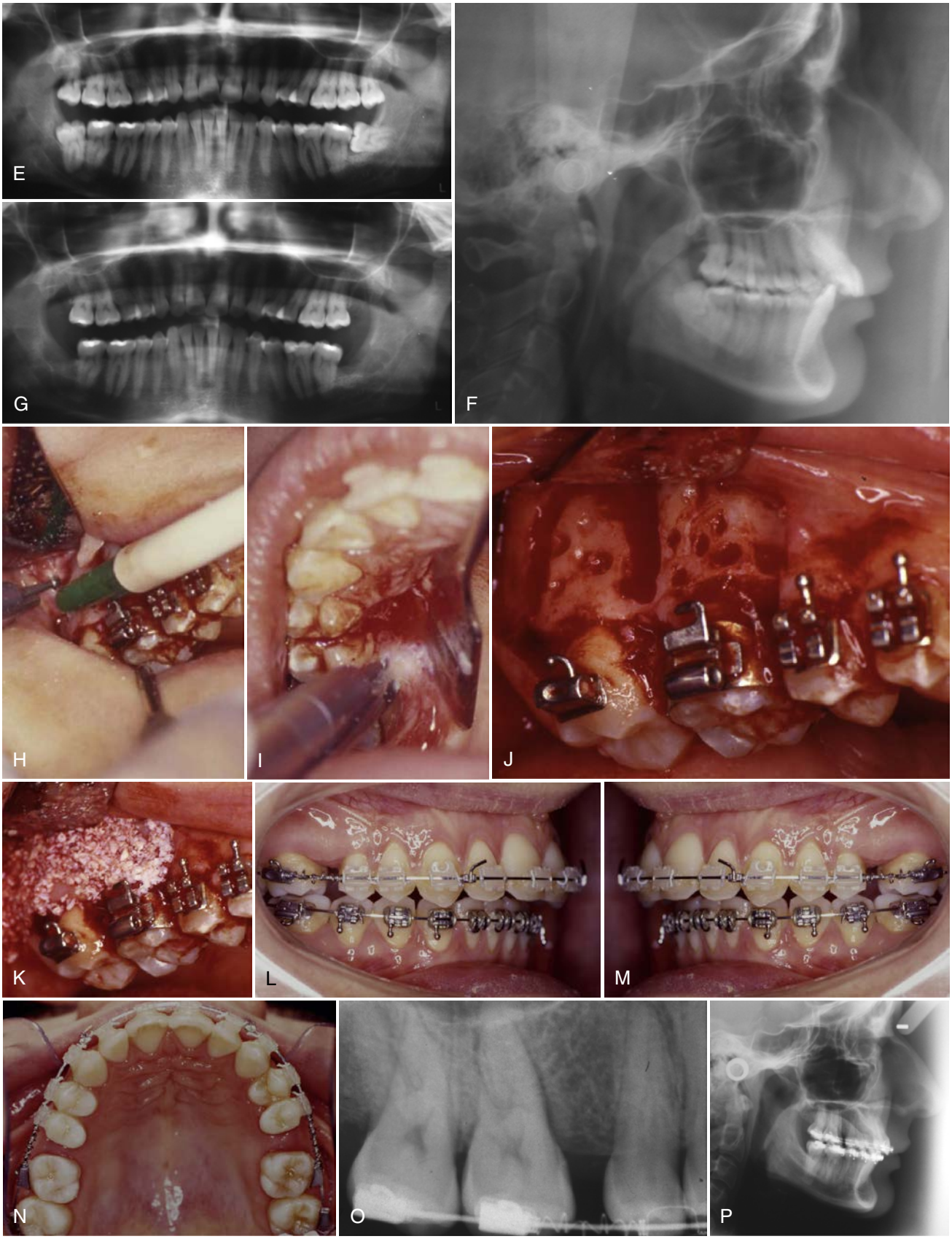
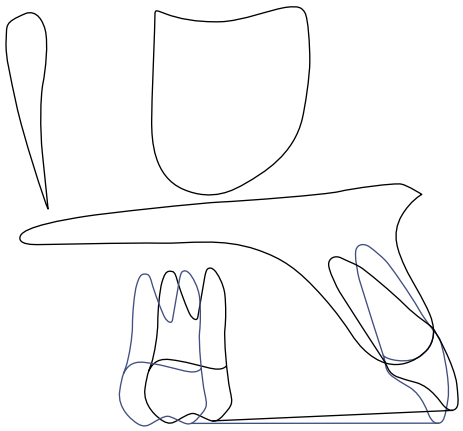
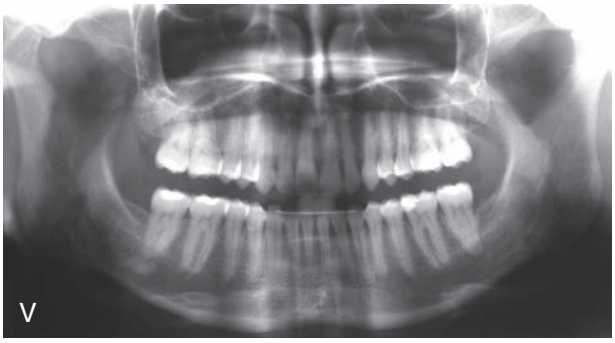


FIGURE 18-23, cont'd E–G, Radiographs. H–K, Surgical images of segmental corticotomy and bone grafting performed on both buccal and palatal sides. L–P, Progress pictures and radiographs.





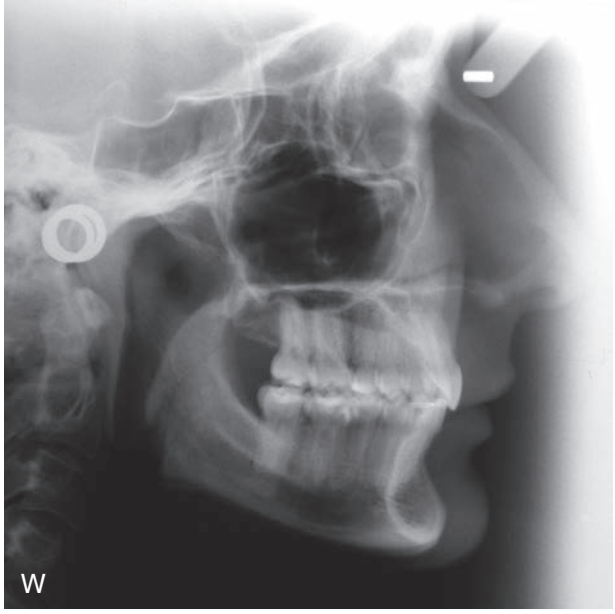


FIGURE 18-23, cont'd **Q,** Tracing superimposition before and after treatment. Note distalization of molars. **R–W,** Final records.

FIGURE 18-24 Adult patient with a Class II, division 1 malocclusion with anterior open bite and unilateral crossbite. Skeletal divergence and maxillary constriction were associated. **A–I,** Initial records of the case. **J–N,** The patient refused orthognathic surgery and accepted treatment with alveolar corticotomy extended from second molar to second molar. Extensive bone and soft tissue grafting mixed with growth factors were associated. Ten days after the surgery, with the sutures still in place, upper and lower arches were bonded with the CCO System, and the first 0.016 NiTi wires were inserted. **O–S,** Treatment was carried out with a sequence of 0.020 × 0.020 Bioforce–0.019 × 0.025 stainless steel–0.021 × 0.025 stainless steel archwires. No miniscrews or posterior anchorages were used. **T–V,** Treatment progress. **W–ZC**, Patient at the end of treatment. **ZD–ZH,** Final radiographs and cone-beam computed tomography scans. **ZI,** Superimposition of pre- and post tracings.

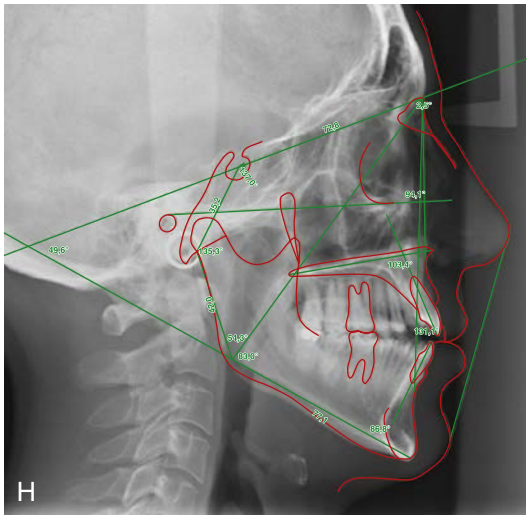


FIGURE 18-24, cont'd

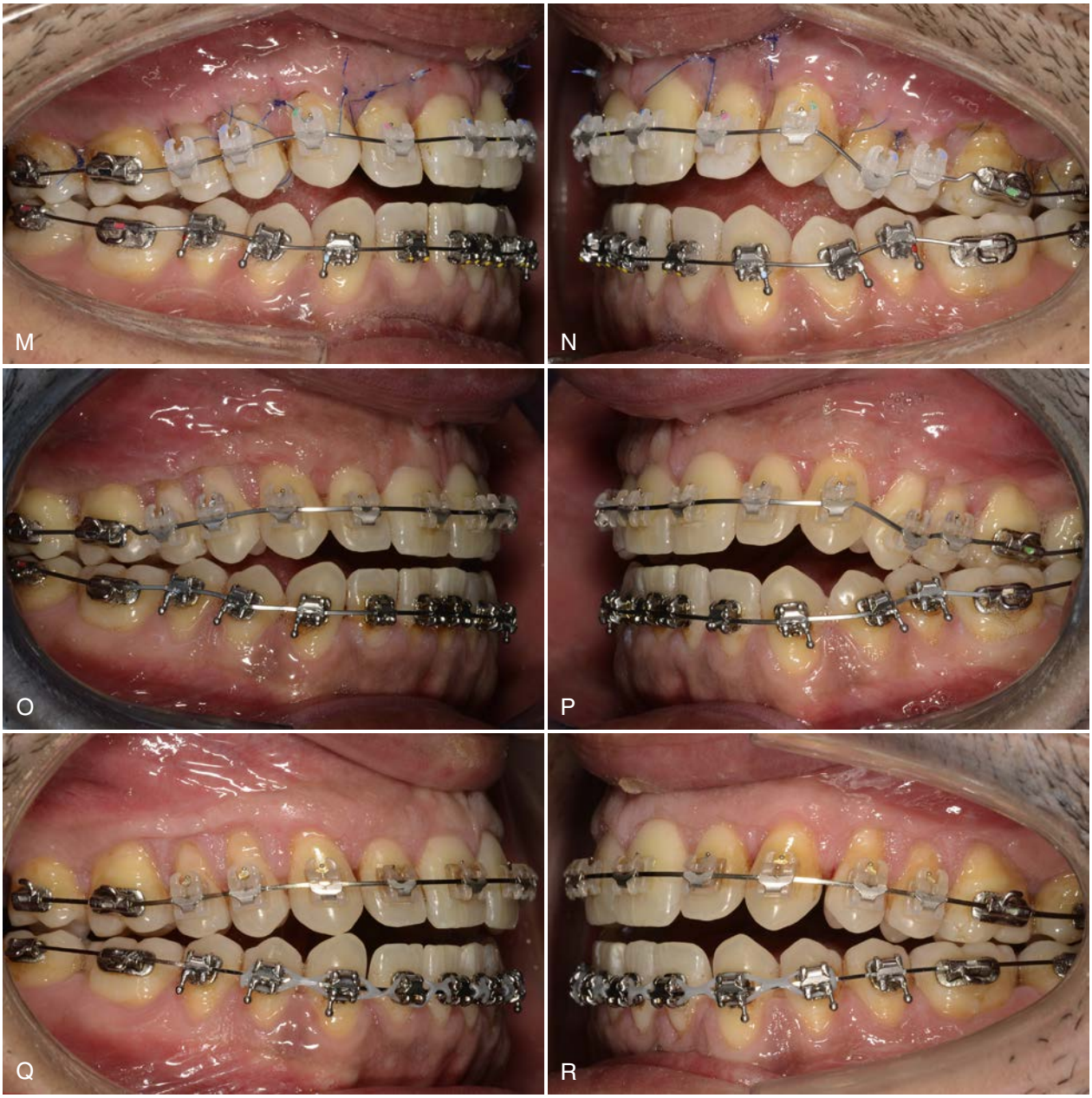


FIGURE 18-24, cont'd

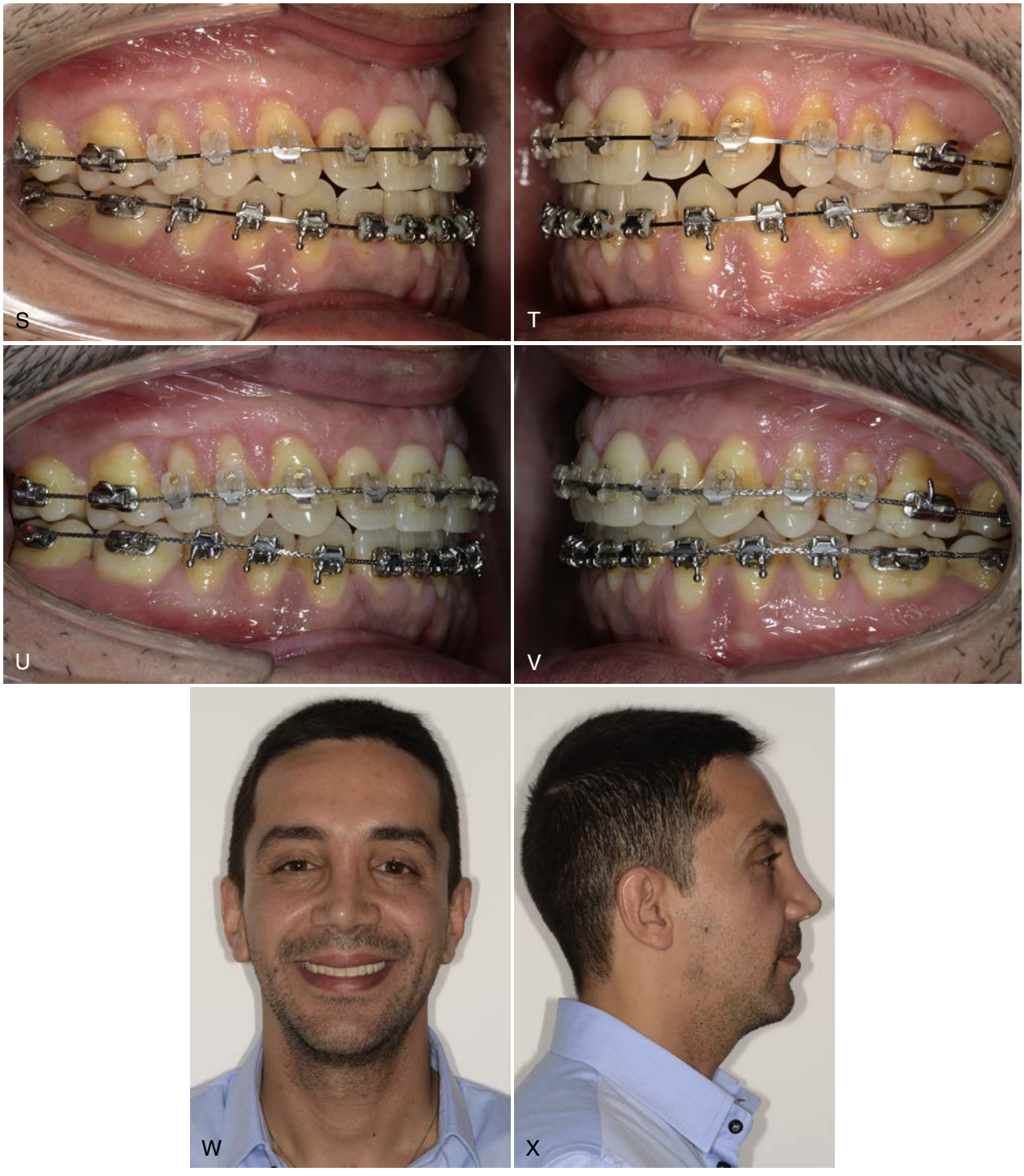


FIGURE 18-24, cont'd

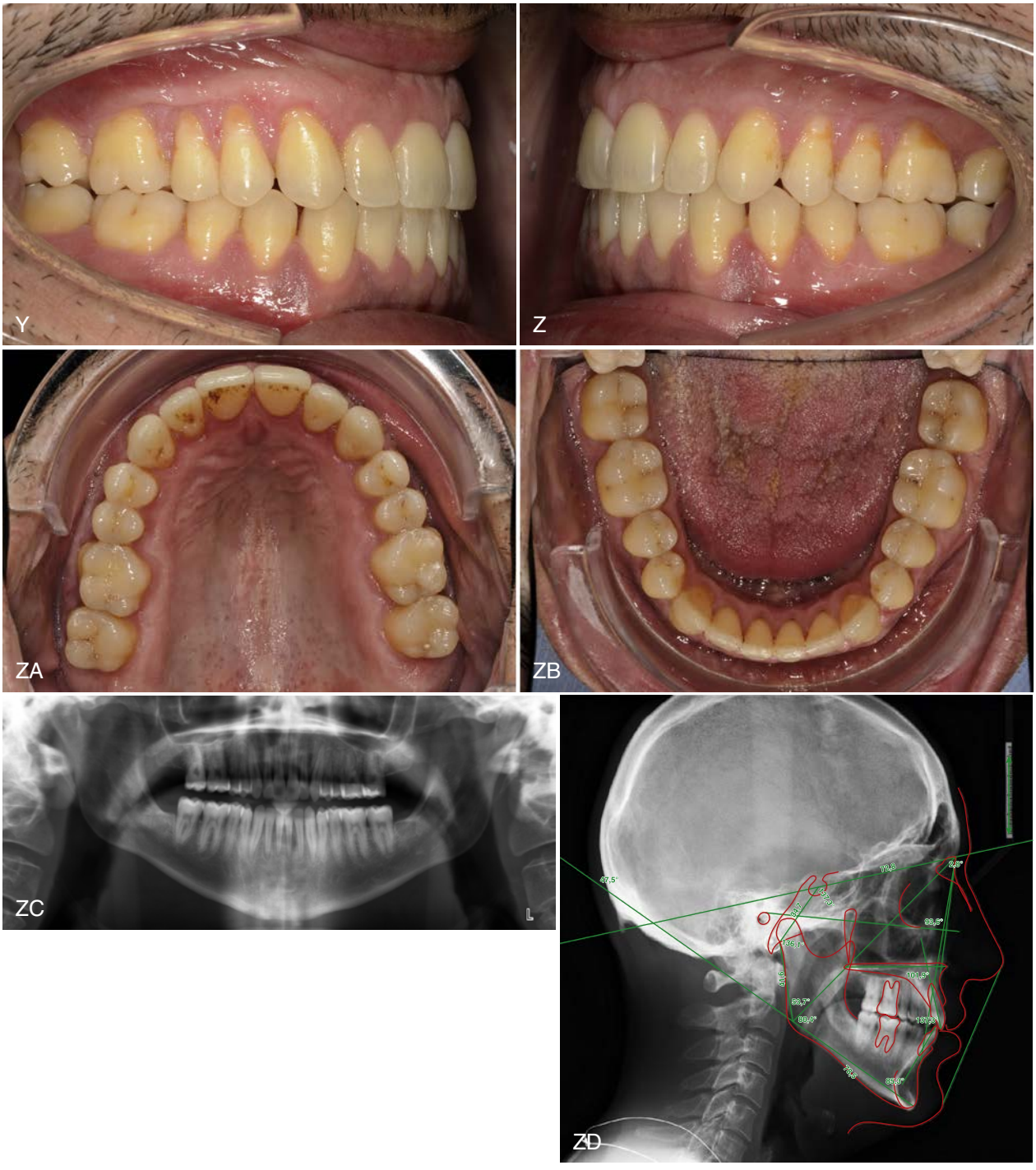
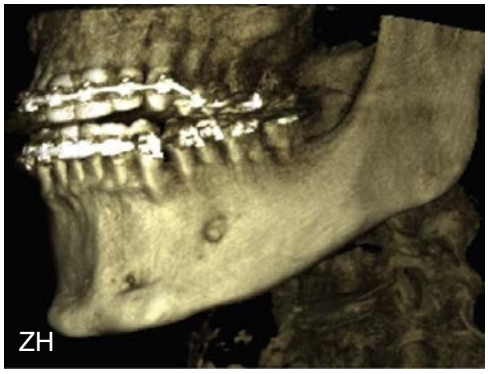
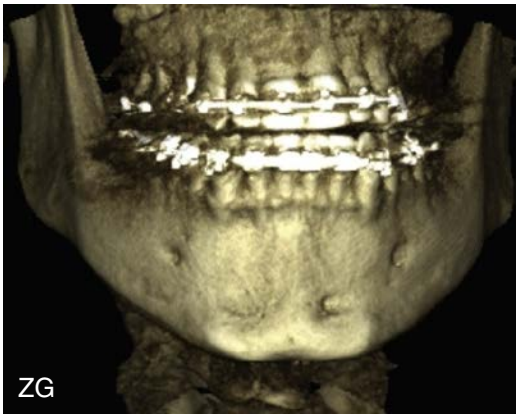


FIGURE 18-24, cont'd





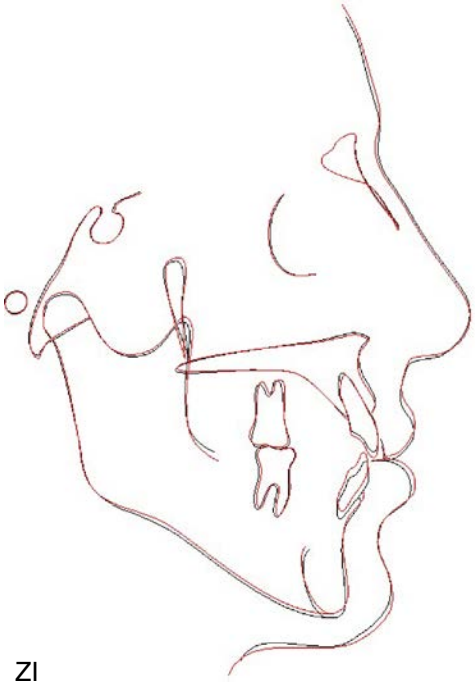


FIGURE 18-24, cont'd

without corticotomy using the same appliance and finished with the same quality of treatment result would be the only way to give the final word on this matter, but it has not been done yet.

2. **Corticotomy has limited effect in space.** It seems that the changes in bone metabolism caused by decortication are well correlated to the amount of the insult (the greater the insult, the greater the reaction), but they are restricted to the decorticated area.[20] This aspect is very important from an orthodontic point of view because it may affect the clinical and biomechanical effects of corticotomy on Orthodontic Tooth Movement (OTM): when moving multirooted teeth and when bodily translating a tooth, the outcome may not be as expected. This may be a limitation for surgeries that do not extend the surgical insult around the entire radicular area (both buccal and lingual) of the teeth to move.
3. **Corticotomy facilitates tooth movement.** Clinical research has been focused almost entirely on acceleration of tooth movement. It is the idea of the authors that corticotomies are mainly able to facilitate biomechanically complex orthodontic movements. Spena et al.,[21,22] in two studies made on a total of 12 adult patients with Class II malocclusions treated with distalization of the upper molars, showed how upper molars could be bodily distalized with simple buccal

mechanics and no anterior anchorage. Corticotomy was performed only on the teeth to move, thus reducing their resistance to distal forces and the anterior anchorage needs. Periodontally Facilitated Orthodontics (PFO) is preferred to PAOO to describe the advantages of alveolar corticotomies.

4. **Corticotomy needs proper surgical management.** Open flap surgery is preferred in most cases. The design of the flap should provide clear access to the area of decortication and a tension-free flap closure to provide optimal coverage of the decorticated area, the grafted material, and allow final soft tissue healing. Decortication of the alveolar bone is preferably performed with calibrated piezo blades and possibly extended around the entire root area of the teeth to move. Interproximal vertical cuts from 2 mm below the alveolar crest to 1 to 2 mm beyond the root apices are associated to horizontal cuts in the supra-apical area. The depth of the cuts is approximately 2 to 3 mm, just slightly extended into the medullary bone. Grafting is not always needed and may include hard tissue, soft tissue, or both. Thin gingival biotype, gingival recessions, dehiscences, fenestrations, and tooth movements that may cause periodontal problems are just some of the clinical situations that call for grafting with corticotomy.
5. **Corticotomy needs proper orthodontic management.** Orthodontic treatment associated with alveolar corticotomy may be carried out with any fixed or removable appliances. It is our choice to combine PFO procedures with fixed active self-ligating appliances (Inovation) with the new prescription of the Complete Clinical Orthodontics (CCO) System. The management and wire changes are similar to any orthodontic case. No initial heavy force is necessary. There is no rule as far as timing of the bonding: in some cases, appliances are placed at the suture removal, but in other cases (e.g., when distalizing upper molars or repositioning impacted teeth), they are placed several months before corticotomy. The enhanced tooth movement deriving from the RAP reaction is obtained when needed. The major difference is that after the periodontal surgery, the visits are every 2 weeks instead of the usual 6 to 8 weeks. During the RAP reaction, teeth will move faster and more easily, and what we usually see happen in 6 to 8 weeks will happen in less time. The idea is to get as much as possible from these metabolic changes after alveolar surgery, knowing that at some point, tooth movement will go back to a normal pace. PFO may be easily associated to skeletal anchorage devices. Whereas temporary anchorage devices are used to increase anchorage, corticotomies are to used reduce anchorage.
6. **Corticotomy has to have precise indications.** Corticotomy is not for every patient. Alveolar decortication should not be combined to orthodontic treatments with the only objective of accelerating OTM and reducing treatment time. Corticotomies should be used to facilitate biomechanically complex orthodontic movements, reducing their risks of periodontal and root damage and future instability.

Improvement of Appliance Efficiency

In the previous nonextraction sequence, after maxillary molars had been distalized, a generally long period followed to wait for spontaneous drift of premolars and canines. No apparent active treatment was carried out. Bonding of the arch was contraindicated because friction of brackets and archwires and ligatures would have jeopardized the spontaneous movement of teeth in an improved position and would have caused anchorage loss. If indicated, only intrusion of the maxillary incisors was initiated to remove the last obstacle to mandibular repositioning and growth.

The new self-ligating brackets combined with the light small wires seem to overcome these problems and allow bonding of the arches earlier without compromising spontaneous changes. At present, the self-ligating brackets are the best way to standardize force application, wire seating, friction deriving from the "fourth wall," and everyday clinical maneuvers when changing wires.

The new prescriptions for the active self-ligating brackets (Inovation) incorporated into the CCO System of Dr. Secchi, together with an efficient sequence of initial round thermal NiTi archwires, followed by square and rectangular thermal NiTi to complete correction of rotations, start expressing torque, and allow easy insertion of the stiff working archwires as the 0.019- × 0.025-inch and 0.021- × 0.025-inch stainless steel, allow better three-dimensional control of both anterior and posterior teeth during the treatment and finalization of nonextraction cases.

Two cases in which alveolar corticotomy with proper nonextraction treatment has been combined are shown in Figures18-23 and 18-24.

CONCLUSIONS

Nonextraction treatment mechanics as described by Cetlin has been proved to be a successful way of treating Class I and II malocclusions with crowding. The original mechanics needed to be updated to overcome problems related to patient cooperation and to take advantage of the new appliances and methods. Clinical research will continue to improve the efficacy of this unique therapeutic approach.

REFERENCES

1. Andrews LF. The six keys to normal occlusion. *Am J Orthod*. 1972;62:296–309.
2. Greenspan RA. Reference charts for controlled extraoral force application to maxillary molars. *Am J Orthod*. 1970;58:486–491.
3. Cetlin NM, Ten Hoeve A. Nonextraction treatment. *J Clin Orthod*. 1983;17:396–413.
4. Bergersen EO. A cephalometric study of the clinical use of the mandibular lip bumper. *Am J Orthod*. 1972;61:578–602.
5. Denholtz M. A method of harnessing lip pressure to move teeth. *J Am Soc Study Orthod*. 1963;1:16–35.
6. Graber TM, Neumann B. The use of muscle forces by simple orthodontic appliances. In: Graber TM, Neumann B, eds. *Removable Orthodontic Appliances*. Philadelphia: WB Saunders; 1984:80–84.
7. Bjerregaard J, Bundgaard A, Melsen B. The effect of the mandibular lip bumper and maxillary bite plane on tooth movement, occlusion and space condition in the lower dental arch. *Eur J Orthod*. 1980;2:257–265.
8. Ghafari J. A lip activated appliance in early orthodontic treatment. *J Am Dent Assoc*. 1985;111:771–774.
9. Frankel R, Muller M, Falck F. The uprighting effect of the Frankel appliance on the mandibular canines and premolars during eruption. *Am J Orthod*. 1987;92:109–116.
10. Nevant CT, Buschang PH, Alexander RG, Steffen JM. Lip bumper therapy for gaining arch length. *Am J Orthod*. 1991;100:330–336.
11. Korn M, Shapiro E. Flexible lip bumpers for arch development. *J Clin Orthod*. 1994;28:43–49.
12. Sakuda M, Ishizawa M. Study of the lip bumper. *J Dent Res*. 1970;49:677.
13. Werner SP, Shivapuja PK, Harris EF. Skeletodental changes in the adolescent accruing from use of the lip bumper. *Angle Orthod*. 1994;64:13–22.

14. Wilcko WM, Wilcko MT, Bouquot JE, Ferguson DJ. Rapid orthodontics with alveolar reshaping: two case reports of decrowding. *Int J Periodont Rest Dent*. 2001;21:9–19.
15. Suya H. Corticotomy in orthodontics. In: Hosl E, Baldauf A, eds. *Mechanical and Biological Basics in Orthodontic Therapy*. Heidelberg: Huthig Buch Verlag; 1991:207–226.
16. Vig PS, Weintraub JA, Brown C, Kowalski CJ. The duration of orthodontic treatment with and without extractions: a pilot study of five selected practices. *Am J Orthod Dentofac Orthop*. 1990;97:45–51.
17. Beckwith FR, Ackermann RJ, Cobb CM, Tira DE. An evaluation of factors affecting duration of orthodontic treatment. *Am J Orthod Dentofac Orthop*. 1999;115:439–447.
18. Skidmore KJ, Brook KJ, Thomson WM, Harding WJ. Factors affecting treatment time in orthodontic patients. *Am J Orthod Dentofac Orthop*. 2006;129:230–238.
19. Mavreas D, Athanasiou AE. Factors affecting the duration of orthodontic treatment: a systematic review. *Eur J Orthod*. 2008;30:386–395.
20. Sanjideh PA, Rossouw PE, Campbell PM, et al. Tooth movements in foxhounds after one or two alveolar corticotomies. *Eur J Orthod*. 2010;32:106–113.
21. Spena R, Caiazzo A, Gracco A, Siciliani G. The use of segmental corticotomy to enhance molar distalization. *J Clin Orthod*. 2007;16:693–699.
22. Spena R, Turatti G. Distalizacion molar superior y ortodoncia acelerada periodontalmente. *Rev Esp Ortod*. 2011;41:167–174

SUGGESTED READING

Burket LW. Consideration of extraction in orthodontic treatment. *Penn Dent J*. 1932. May.
Burstone CJ. Deep overbite correction by intrusion. *Am J Orthod*. 1977;72:1–22.
Cetlin NM. La filosofia della terapia ortodontica senza estrazioni. *Quad SIDO*. 1984:17.
Dake ML, Sinclair PM. A comparison of the Ricketts and Tweed-type arch leveling techniques. *Am J Orthod*. 1989;95:72–78.
DeVincenzo JP, Winn MW. Maxillary intrusion and facial growth. *Angle Orthod*. 1987;57(4):279–289.
Fontana M, Cozzani M, Mutinelli S, et al. Maxillary molar distalization therapy in adult patients: a multicentre study. *Ortho Craniofac Res*. 2015;18(4):221–231.
Frankel R. Decrowding during eruption under the screening influence of vestibular shields. *Am J Orthod*. 1974;65:372–406.
Giannelly A. One-phase versus two-phase treatment. *Am J Orthod*. 1995;108:556–559.
Haas AJ. Palatal expansion: just the beginning of dentofacial orthopedics. *Am J Orthod*. 1970;57:219–255.
Hilgers JJ. The pendulum appliance for Class II non-compliance therapy. *J Clin Orthod*. 1992;26:706.
Kloehn SJ. Orthodontics: force or persuasion. *Angle Orthod*. 1953;23:56–65.
Little RM, Riedel RA, Artun J. An evaluation of changes in mandibular anterior alignment from 10-20 years post retention. *Am J Orthod*. 1988;93:423–428.
Moorrees CF, Gron AM, Lebret LM, et al. Growth studies of dentition: a review. *Am J Orthod*. 1969;55:600–616.
Roth RH. Functional occlusion for the orthodontist. *J Clin Orthod*. 1981;15:32–51. 100–123, 174–198, 246–265.
Secchi AG. Complete Clinical Orthodontics. The Course Manual. *DoctorOs, Ariesdue*. 2012.
Secchi AG. Complete clinical orthodontics. Treatment mechanics: part 1 & 2. *Orthod Pract*. 2012;4:28–35.
Spena R. *The clinical use of the lip bumper in mixed and adult dentition*. St. Louis: Roundtable Breakfast, AAO Annual Session; 1992.
Spena R. *How to change archform and gain space in the upper arch with the use of palatal bars*. Toronto: Roundtable Breakfast, AAO Annual Session; 1993.
Spena R. *Cetlin: punti chiave della filosofia e della tecnica*. Ottobre: Schede di aggiornamento, SIDO; 1994.
Spena R. Il trattamento ortodontico senza estrazioni: possibilità e limiti. *Quad SIDO*. 1994;1:13–16.
Spena R, Caiazzo A, Gracco A, Siciliani G. The use of segmental corticotomy to enhance molar distalization. *J Clin Orthod*. 2007;16:693–699.
Spena R, Turatti G. Distalizacion molar superior y ortodoncia acelerada periodontalmente. *Rev Esp Ortod*. 2011;41:167–174
Spena R, Garganese D. Periodontally Facilitated Orthodontics – A new perspective on alveolar corticotomy. *Inf Orthod Kieferorthop*. 2015;47:1–25.
Swain BF. Straight-wire design strategies: five-year evaluation of the Roth modification of the Andrews Straight Wire appliance. In: Graber LW, ed. *Orthodontics: State of the Art*. St. Louis: CV Mosby; 1986:279–298.
Swain BF. Overcorrection, fiberotomy, reapproximation: getting them together for stability. *NESO Newslett*. 1982.
TenHoeve A, Mulie RM. The effect of anteroposterior incisor repositioning on the palatal cortex as studied with laminagraphy. *J Clin Orthod*. 1976;10:804–822.
TenHoeve A. Palatal bar and lip bumper in non extraction treatment. *J Clin Orthod*. 1985;19:272.
TenHoeve A, Cetlin NM. *Non-extraction. Long term results. JV Mershon Memorial Lecture*. Toronto: AAO Annual Session; 1993.
Vanarsdall RM. *Diagnosis and treatment of transverse discrepancy. JV Mershon Memorial lecture*. St. Louis: AAO Annual Session; 1992.
Vanarsdall RM, Secchi AG, Ghung CH, Katz SH. Mandibular basal structure response to lip bumper treatment in the transverse dimension. *Angle Orthod*. 2004;74:473.
Wilcko MT, Wilcko WM, Bissada NF. An evidence-based analysis of periodontally accelerated orthodontic and osteogenic techniques: a synthesis of scientific perspectives. *Semin Orthod*. 2008;14:305–316.

19

Standard Edgewise: Tweed-Merrifield Philosophy, Diagnosis, Treatment Planning, and Force Systems

James L. Vaden, Herbert A. Klontz, and Jack G. Dale

Nothing worthwhile ever departs.

OUTLINE

Historical Perspective, 481
The Angle System, 481
The Edgewise Appliance, 482
Charles H. Tweed, 482
Levern Merrifield, 484
Diagnostic Concepts, 484
Treatment Concepts, 484
Dimensions of the Dentition, 484
Differential Diagnosis, 485
Facial Disharmony, 485
Z Angle, 486
Frankfort Mandibular Incisor Angle, 486
Cranial Disharmony, 488
Skeletal Analysis Values, 488
Craniofacial Analysis, 490
Dental Disharmony, 492
Total Dentition Space Analysis, 492
Anterior Space Analysis, 492
Midarch Space Analysis, 492
Posterior Space Analysis, 493
Differential Diagnostic Analysis System, 493
Tweed-Merrifield Edgewise Appliance, 493
Brackets and Tubes, 493
Archwires, 493
First-, Second-, and Third-Order Bends and Their Interaction, 493
First-Order Bends, 493
Second-Order Bends, 494
Third-Order Bends, 494
Auxiliaries, 495
Variations of the Appliance, 495
Treatment with the Tweed-Merrifield Edgewise Appliance, 495
Sequential Appliance Placement, 495
Sequential Tooth Movement, 495
Sequential Mandibular Anchorage Preparation, 495
Directional Force, 495
Timing of Treatment, 496
Steps of Treatment, 496
Denture Preparation, 496
Denture Correction, 497
Sequential Mandibular Anchorage Preparation, 498
Class II Force System, 498
Orthodontic Correction of the Class II Dental Relationship, 499
Denture Completion, 500
Denture Recovery, 501
Summary, 502

HISTORICAL PERSPECTIVE

An obsession for order motivated Edward Hartley Angle (Fig. 19-1) to create, in 1888, the Angle system. This system ultimately resulted in the introduction of the edgewise multibanded appliance 5 years before Angle's death, which has been the progenitor of all modern appliances.

THE ANGLE SYSTEM

Edward Angle, after graduation from dental school in 1878 and before his introduction of the Angle system in 1888, experienced many technical problems and frustrations in patient treatment that motivated and inspired him to develop a standard appliance.[1,2] He believed that an orthodontic appliance must have five properties:

1. **Simplicity:** It must push, pull, and rotate teeth.
2. **Stability:** It must be fixed to the teeth.
3. **Efficiency:** It must be based on Newton's third law and anchorage.
4. **Delicacy:** It must be accepted by the tissues, and it must not cause inflammation and soreness.
5. **Inconspicuousness:** It must be aesthetically acceptable.

Angle designed a standard appliance composed of a specific number of basic components (Fig. 19-2). He had these

FIGURE 19-1 Edward H. Angle.

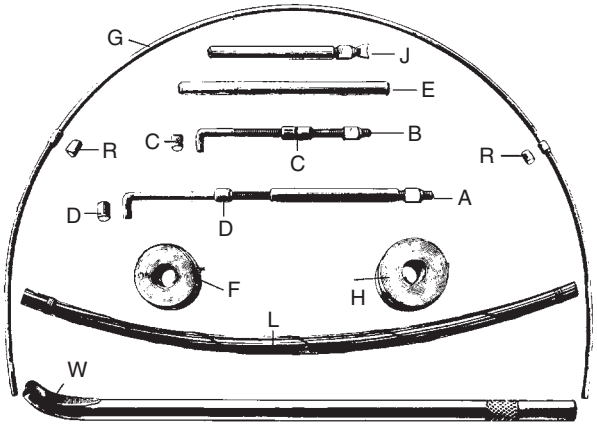


FIGURE 19-2 The basic components of Angle's standard appliance, illustrating traction screws (A, B), attachment tubes (C, D), jackscrews (E, J), lever wires (L), band material (F, H), archwire (G), wrench (W), and archwire lock (R). (From Angle EH: *Treatment of Malocclusion of the Teeth*. Philadelphia: SS White; 1907.)

components mass produced so that they could be assembled into a simple, stable, efficient, delicate, and inconspicuous treatment device, without difficulty, in less time and with minimal pain and discomfort to the patient. This universal application enabled practitioners to treat more patients at a higher level of excellence and at less cost than they had done previously. In effect, it was the beginning of a relationship among manufacturers, suppliers, and orthodontists; it was the Angle system.

THE EDGEWISE APPLIANCE

Two years before he died, with knowledge born from experience and gained from his other appliance inventions, Angle set out to devise an appliance that would not only overcome past difficulties but also would have a greater chance than did its

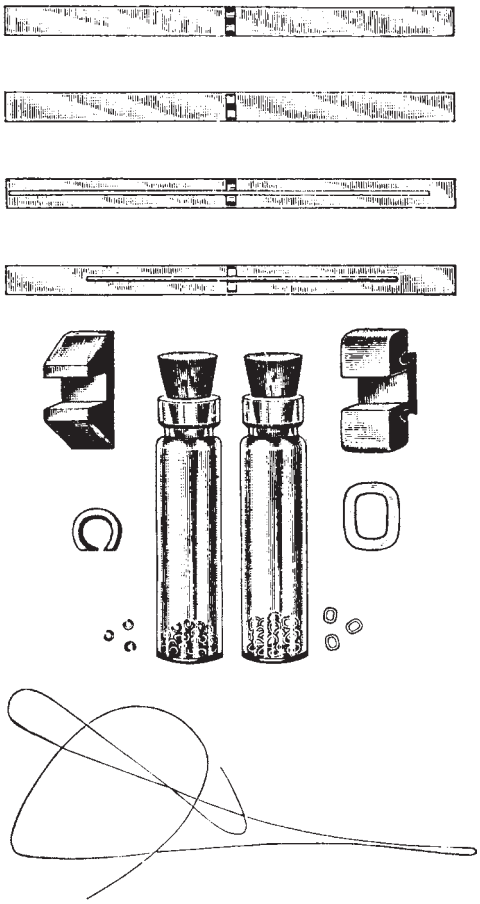


FIGURE 19-3 Original edgewise brackets.

predecessors of treating to "ideals." He changed the form of the brackets by locating the slot in the center and placing it in a horizontal plane instead of a vertical plane. The archwire was held in position first by a brass ligature and later by a delicate stainless steel ligature. The new edgewise bracket consisted of a rectangular box with three walls within the bracket, 0.022 × 0.028 inch in dimension. The bracket slot opened horizontally (Fig. 19-3). This new design provided more accuracy and thus a more efficient torquing mechanism.

Because Angle introduced the edgewise bracket only 2 years before he died, he had little time to teach its manipulation, develop it further, and improve its use—and he knew it.

CHARLES H. TWEED

When Charles H. Tweed graduated from an improvised Angle course given by George Hahn in 1928, he was 33 years old, and Angle was 73 (Fig. 19-4). Angle was bitterly disappointed by the reception that had been accorded the edgewise appliance. He was infuriated and bitter about the modifications that were being made by several of his graduates (e.g., Spencer Adkinson). To him, it was obvious that something had to be done if the edgewise appliance was to survive intact.

Angle decided that an article describing the appliance must be published in *Dental Cosmos*. He asked Tweed to help him with the article because Tweed had just finished the Angle "course" and because he admired and respected Tweed's ability.

FIGURE 19-4 Charles H. Tweed.

For 7 weeks, they worked together and in the process became close friends. During this time, Angle advised Tweed that he could never master the edgewise appliance unless he limited his practice solely to its use. And so with the completion of the article for *Dental Cosmos*, Charles Tweed returned to Arizona and established in Phoenix what was probably the first pure edgewise specialty practice in the United States.

For the next 2 years, the two men worked together closely. Tweed treatment planned and treated his patients, and Angle acted as his advisor. Angle was pleased with Tweed's treatment and was instrumental in getting Tweed on several programs. During these 2 years, in a series of more than 100 letters that are now housed in the Tweed Memorial Center Library, Angle urged his young disciple to carry out two vital requests: (1) to dedicate his life to the development of the edgewise appliance and (2) to make every effort to establish orthodontics as a specialty within the dental profession.

Tweed followed Angle's advice. First, he instigated the passing of the first orthodontic specialty law in the United States. He did this by canvassing patients, persuading dentists, influencing and arousing politicians, speaking at meetings, having petitions signed, and even taking patients before the legislature. In short, it was a one-man blitz. His untiring and relentless efforts were successful, and in 1929, the Arizona legislature passed the first law limiting the practice of orthodontics to specialists. Tweed received Certificate No. 1 in Arizona and became the first certified specialist in orthodontics in the United States.

In 1932, Tweed published his first article in the *Angle Orthodontist*. It was titled "Reports of Cases Treated with Edgewise Arch Mechanism."[3] Tweed held to Angle's firm conviction that one must never extract teeth. This conviction lasted for 4 years.

The facial aesthetics Tweed began to observe in his patients was discouraging to him, so discouraging in fact that he almost gave up orthodontic practice. He knew he had the appliance, and he knew he had the ability, but his results were aesthetically unsatisfactory and unstable. He devoted the next 4 years of his life to the study of his successes and failures. During this 4-year period, he made a most important observation: upright mandibular incisors frequently were related to both posttreatment facial balance and stability of the treated dentition. To position mandibular incisors upright, he concluded that one must, in many instances, extract teeth and prepare anchorage. He selected his failures, extracted four first premolar teeth, and retreated the patients. He did this without charging a fee.

In 1936, Tweed delivered a paper on the extraction of teeth for orthodontic malocclusion correction to the membership of the Angle Society and subsequently published it.[4,5] "Mother" Angle, the editor of the *Angle Orthodontist* and a member of the Angle Society, refused to attend the lecture. George Hahn, the man who went out of his way to create the opportunity for Tweed to take the Angle course, criticized him severely. Angle disciples considered Charles Tweed to be a traitor to the greatest man orthodontics had ever known. Tweed was crushed by the response, but he returned home determined to continue his research.

He worked even harder than before. By 1940, he had produced case reports, with four sets of records, of 100 consecutively treated patients who were first treated with nonextraction and later with extraction. He managed to get himself on the program of the next meeting of the Angle Society in Chicago, where he would present a paper and display his case reports.

Dr. Robert Strang, one of Angle's students in the early years, described the event this way:[6]

> *I noted that Dr. Charles Tweed was scheduled to be on the program of the meeting in Chicago. I planned to be there with the objective of lacing into him for violating Dr. Angle's sacred principle of non-extraction in treatment.*
>
> *Previous to reading his paper, Dr. Tweed had placed on tables before and after casts and photographs of one hundred consecutively treated patients. The results in all of these one hundred patients were magnificent and beyond criticism.*
>
> *Dr. Tweed read his well-written and illustrated paper. He explained his objective of keeping the teeth over basal bone, which made it necessary to extract teeth in many patients; however, it did produce stable results. Then he sat down. There was no applause. The room filled with shouted demands from the floor. For at least an hour, Charlie got the worst tongue-lashing that you can possibly imagine and not one word of praise for the beautiful results of treatment. Here was a student of Dr. Angle's violating the most fixed and rigid rule in his instruction—never extract teeth.*
>
> *During all this vicious attack, my mind took a complete turnover. I could visualize nothing but that marvelous exhibit of treated cases. Not one individual in the room had complimented the essayist. They were all ripping him to pieces for extracting teeth. Finally, I obtained the floor and complimented and defended him to the best of my ability. When I sat down, I, too, took a tongue-lashing that compared very favorably with the one Charlie had just received. Subsequently, I took his course, and practiced, taught and published his techniques in my textbook.*

Tweed's many contributions to the specialty established a benchmark in orthodontic thought and treatment. Most notable among his many contributions were the following:

1. He emphasized four objectives of orthodontic treatment: aesthetics, health, function, and stability, with emphasis and concern for balance and harmony of the lower face.

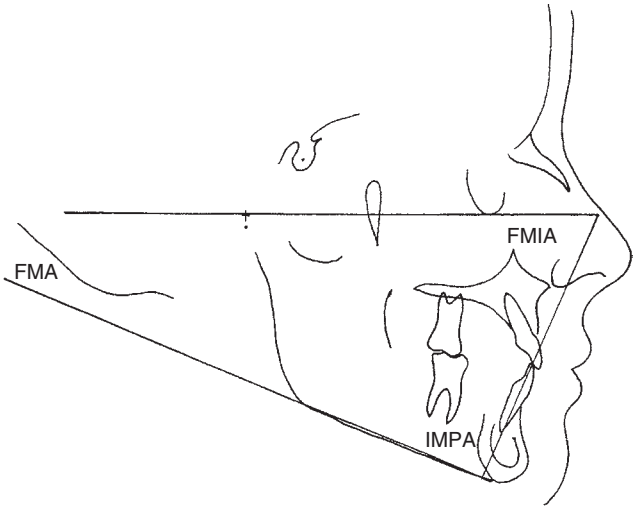


FIGURE 19-5 Tweed's diagnostic facial triangle. *FMA,* Frankfort mandibular plane angle; *FMIA,* Frankfort mandibular incisor axis angle; *IMPA,* lower incisor mandibular plane angle.

FIGURE 19-6 Levern Merrifield.

2. He developed the concept of positioning teeth over basal bone with emphasis on the mandibular incisors.[7]
3. He made the extraction of teeth for orthodontic correction acceptable and popularized the extraction of the first premolars.[8]
4. He enhanced the clinical application of cephalometrics.
5. He developed the diagnostic facial triangle[9] in order to make cephalometrics a diagnostic tool and a guide in treatment and in the evaluation of treatment results (Fig. 19-5).
6. He developed a concept of orderly treatment procedures and introduced anchorage preparation as a major step in treatment.[10]
7. He developed a fundamentally sound and consistent preorthodontic guidance program that popularized serial extraction of primary and, later, permanent teeth.

In addition to his many clinical contributions to the specialty, Tweed gave guidance, inspiration, and leadership to more orthodontists in the world than anyone else of his time. Because of Charles Tweed's skill and determination, the edgewise appliance became universally popular, and the practice of clinical orthodontics became a health service requested by the public.

Angle gave orthodontics the edgewise bracket, but Tweed gave the specialty the appliance. Tweed was considered the premier edgewise orthodontist of his day. Many who admired his results wished to learn his techniques. The orthodontic world journeyed to Tucson, Arizona, to take Tweed's course and learn his method of treatment with the edgewise appliance. The Tweed philosophy was born.

LEVERN MERRIFIELD

In 1960, Tweed selected one of his most outstanding students, Levern Merrifield, from Ponca City, Oklahoma, to continue his work on the edgewise appliance (Fig. 19-6). Tweed asked Merrifield to join him and be the codirector of his course. Merrifield took Tweed's course in 1953 and became a member of Tweed's staff in 1955. He became the course director at the time of Tweed's death in 1970. Merrifield devoted the remaining 45 years of his life to the study of orthodontic diagnosis and the use of the edgewise appliance. Merrifield's contributions have been disseminated and popularized. They include the following:

Diagnostic Concepts

1. The fundamental concept of dimensions of the dentition[11]
2. Dimensions of the lower face[12]
3. Total space analysis[13]
4. Guidelines for space management decisions to achieve the following:
   a. Facilitate maximum orthodontic correction of a malocclusion.
   b. Define areas of skeletal, facial, and dental disharmony.[14]

Treatment Concepts

1. Directional force control during treatment[15]
2. Sequential tooth movement
3. Sequential mandibular anchorage preparation[16]
4. The organization of treatment into four orderly steps that have specific objectives
   Merrifield's innovations in diagnosis and treatment planning and his experience with the edgewise appliance have augmented Tweed's contributions and concepts to give modern orthodontists a more accurate, reliable, precise, efficient, and practical protocol of diagnosis, treatment planning, and treatment. Adherence to this protocol allows the clinician to do the following:
   a. Define objectives for the face, the dentition, and the skeletal pattern.
   b. Properly diagnose the malocclusion and treatment plan the malocclusion correction.
   c. Use the edgewise appliance to reach predetermined objectives efficiently.

DIMENSIONS OF THE DENTITION

The clinical practice of orthodontics has always been based on the various dimensions of the dentition: height, width, and length (vertical, transverse, and sagittal). These dimensions allow the teeth to be moved in six directions: mesially, distally, facially, lingually, intrusively, and extrusively. All of these

movements, which are accomplished routinely with orthodontic appliances, are limited and restricted by the physical environment of bone, muscle, and soft tissue, all of which exert an influence on the teeth and jaws.

Since the beginning of the orthodontic specialty, an effort has been made to determine the extreme limits of this environment. Each engineering change in appliance fabrication appears to bring about a new challenge to the physical limitations of the dentition's environment. Dimensions of the dentition includes four basic premises, *provided that the musculature is normal:*

Premise 1: An anterior limit exists. The teeth must not be placed forward, off basal bone. If the teeth are too far forward, all the objectives of treatment are compromised.

Premise 2: A posterior limit exists. Teeth can be positioned and/or impacted into the area behind the mandibular first molar in the mandibular arch, even as they can be moved too far forward off basal bone.

Premise 3: A lateral limit exists. If the teeth are moved buccally into the masseter and buccinator muscles, relapse is likely to result over the long term.

Premise 4: A vertical limit exists. Vertical expansion is disastrous to facial balance and harmony in the sagittal plane, except in deep-bite cases.

In summary, orthodontists must recognize the limitations of the dental environment and design treatment to conform to these dimensions when normal muscle balance exists.

DIFFERENTIAL DIAGNOSIS

Merrifield, in his effort to establish a sound diagnostic basis for directional force treatment with multibanded mechanotherapy, introduced diagnostic analyses that allow clinicians to determine (1) whether and when extractions are necessary and (2) if extractions are indicated, which teeth should be removed. His work enables the clinician to arrive at a differential diagnosis[17,18] instead of treating all extraction problems by the removal of four first premolars, as did Dr. Tweed. Merrifield's diagnostic philosophy can be outlined as follows:

1. Recognize and treat within the dimensions of the dentition. This means nonexpansion of malocclusions when normal muscular balance exists.
2. Recognize the dimensions of the lower face and treat for maximum facial harmony and balance.
3. Recognize and understand the skeletal pattern. Diagnose and treat in harmony with normal growth and developmental patterns, and optimize the less than normal pattern. After the major areas of disharmony are identified, all necessary and practical means should be expended to correct the problem.

Facial Disharmony

A study of the face and its balance or lack of balance must be the first concern during a differential diagnosis. The clinician must have an intuitive concept of a balanced face. Essentially, three factors influence facial balance or lack thereof: (1) the positions of the teeth, (2) the skeletal pattern, and (3) the soft tissue thickness.

Facial balance is influenced by protrusion and crowding of the teeth. Protruded teeth cause facial imbalance. The lips are supported by the maxillary incisor teeth. The upper lip rests on the upper two thirds of the labial surface of the maxillary incisors, and the lower lip is supported by the lower one third of the labial surface of the maxillary incisors; thus, lip protrusion is a reflection of the degree of maxillary incisor protrusion. Maxillary incisor position is related directly, of course, to the position of the mandibular incisors.

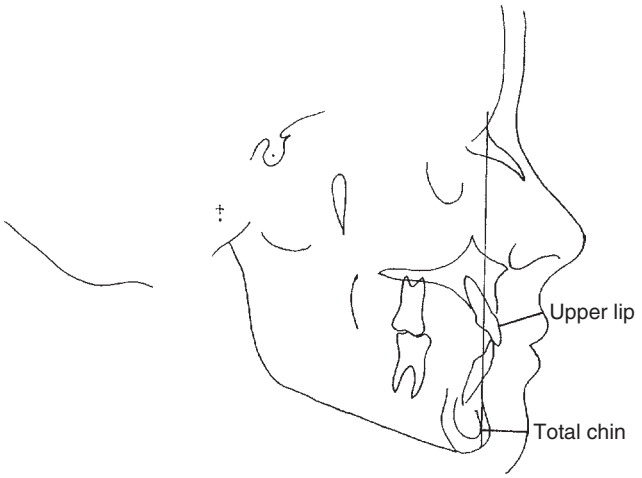


FIGURE 19-7 Upper lip equals total chin. Upper lip thickness should equal total chin thickness in a balanced face.

Facial disharmonies can often be the result of abnormal skeletal relationships. The clinician must understand the skeletal pattern and have the ability to compensate for abnormal skeletal relationships by changing the positions of the teeth.[19] The Frankfort mandibular plane angle (FMA) is a skeletal angular value that is crucial in differential diagnosis. Dental compensation for a patient with a high FMA requires additional uprighting of mandibular incisors. Lower facial balance for the patient with a high FMA can be improved dramatically by using this knowledge.

Conversely, dental compensation for a patient with a low FMA requires less mandibular incisor uprighting. Decisions regarding tooth position objectives must be made after a thorough study of the skeletal pattern.

Facial disharmonies that are not the result of skeletal or dental distortion are generally the result of poor soft tissue distribution.[20,21] Poor soft tissue distribution needs to be identified during differential diagnosis so that dental compensations can be planned. The millimetric measurements of total chin thickness and upper lip thickness are essential components in any study of facial balance. Upper lip thickness is measured from the greatest curvature of the labial surface of the maxillary central incisor to the vermilion border of the upper lip (Fig. 19-7). Total chin thickness is measured horizontally from the NB (Nasion-pt B) line extended to soft tissue pogonion. Total chin thickness should equal upper lip thickness. If total chin thickness is less than upper lip thickness, the anterior teeth must be positioned more upright to facilitate a more balanced facial profile because lip retraction follows tooth retraction.

Careful consideration of the positions of the teeth, the skeletal pattern, and the soft tissue overlay will give crucial information about the face and enable the clinician to determine whether dental compensations will improve facial balance. Before initiating tooth movement, the clinician must clearly understand its impact on the overlying soft tissue.[22]

The profile line and its relationship to facial structures and to the Frankfort plane can be used to give the orthodontic practitioner an idea of lip procumbency. If the profile line lies outside

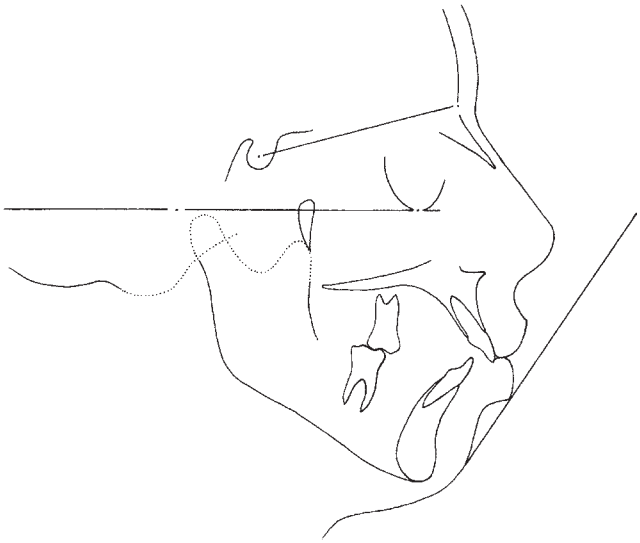


FIGURE 19-8 Profile line drawn on a protrusive face.

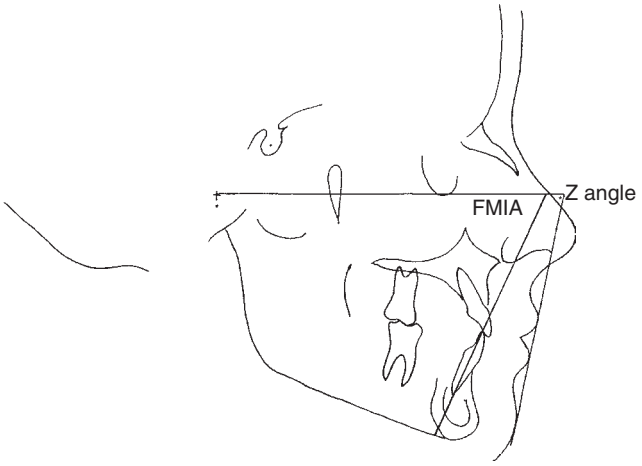


FIGURE 19-10 The Z angle and Frankfort mandibular incisor axis angle (FMIA). Tweed used the FMIA as an indicator of facial balance. Merrifield's Z angle quantifies balance of the lower face.

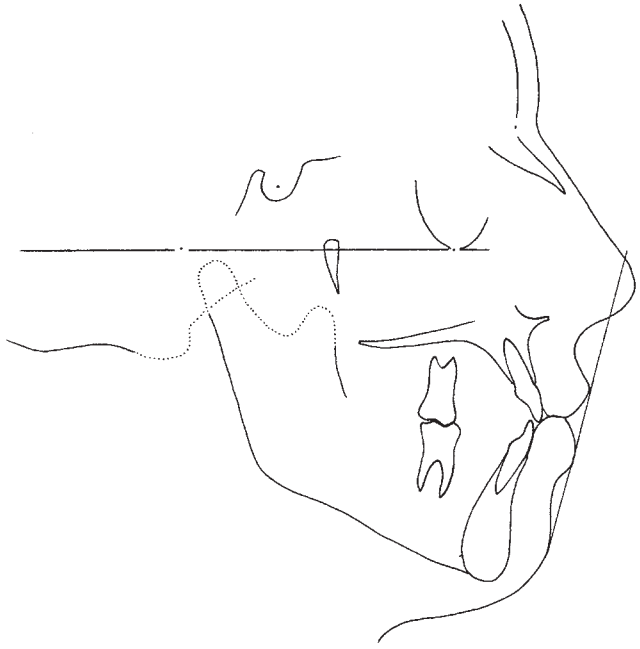


FIGURE 19-9 Profile line drawn on a facial profile that exhibits balance and harmony.

the nose, a protrusion exists (Fig. 19-8). When facial balance is present, the ideal relationship of profile line is to be tangent to the chin and the vermilion border of both lips, and it should lie in the anterior one third of the nose (Fig. 19-9). For centuries, the premise has been that this type of relationship of the profile line to the lips, chin, and nose reflects a pleasing and balanced appearance.

Similarly, on frontal view, the face should be balanced. The vermilion border of the lower lip should bisect the distance between the bottom of the chin and the ala of the nose. The vermilion border of the upper lip also should bisect the distance from the vermilion border of the lower lip to the ala of the nose. These relationships are universally accepted orthodontic standards for facial balance and harmony.

Several cephalometric angles quantify facial balance. Two that have been found to be useful are the Z angle and the Frankfort mandibular incisor axis angle (FMIA) (Fig. 19-10).

Z Angle

The chin/lip soft tissue profile line relationship to the Frankfort horizontal plane quantifies facial balance (see Fig. 19-10). The normal range is 70 to 80 degrees.[12] The ideal value is 75 to 78 degrees, depending on age and gender. This angle was developed to define facial aesthetics further and is an adjunct to the FMIA. The Z angle is more indicative of the soft tissue profile than the FMIA and is responsive to maxillary incisor position. Maxillary incisor retraction of 4 mm allows 4 mm of lower lip retraction and about 3 mm of upper lip response. Horizontal mandibular repositioning also affects this value. Vertical facial height increase, either anterior or posterior, can influence the Z angle.

The Z angle quantifies the combined abnormalities in the values of the FMA, FMIA, and soft tissue thickness because all have a direct bearing on facial balance. The Z angle gives immediate guidance relative to anterior tooth repositioning. If the patient has a normal FMA of 25 degrees, a normal FMIA of 68 degrees, and good soft tissue overlay distribution, the Z angle value should be about 78 degrees.

Frankfort Mandibular Incisor Angle

Tweed established a standard of 68 degrees for individuals with an FMA of 22 to 28 degrees. The standard should be 65 degrees if the FMA is 30 degrees or more, and the FMIA will increase if the FMA is lower. Tweed believed that this value was significant in establishing balance and harmony of the lower face (see Fig. 19-10).

The records of one patient shown in Fig. 19-11 graphically illustrate the facial aesthetics "issue" in orthodontics. The pretreatment facial photographs (Fig. 19-11) confirm an acceptable face. The casts (Fig. 19-12) reflect mild crowding and a very moderate Class II occlusion. The pretreatment cephalometric tracing (Fig. 19-13) shows skeletal and dental values that are "almost" normal.

The patient was treated for 18 months without regard for facial aesthetics (Fig. 19-14). The progress cephalometric

FIGURE 19-11 Pretreatment facial photographs.

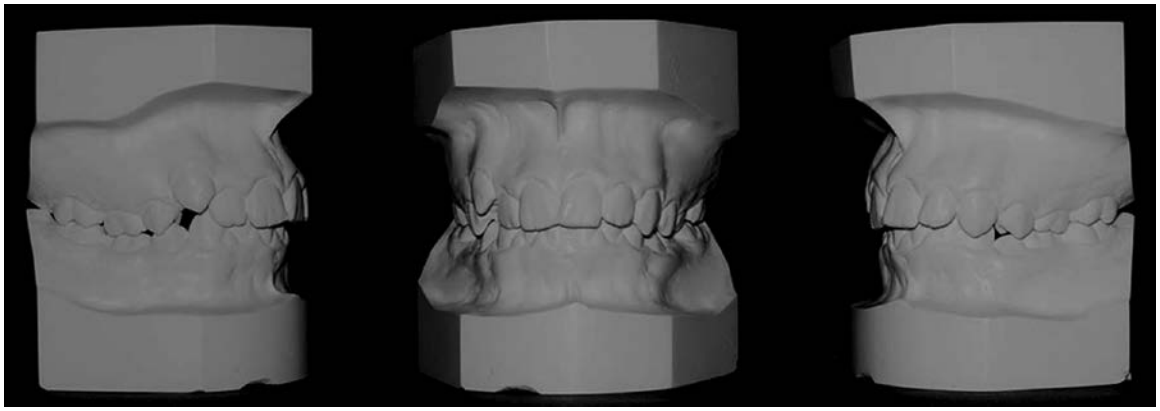
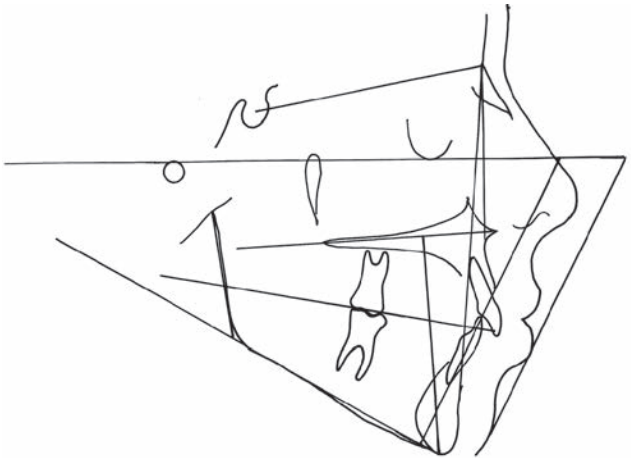


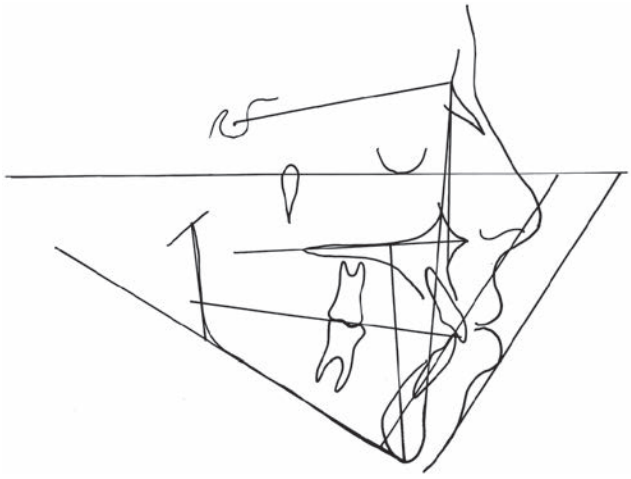
FIGURE 19-12 Pretreatment casts.



FMIA	64	OCC	9
FMA	30	Z	63
IMPA	86	UL	14 mm
SNA	78	TC	12 mm
SNB	75	PFH	41 mm
ANB	3	AFH	67 mm
AO-BO	0 mm	Index	0.61

FIGURE 19-13 Pretreatment cephalometric tracing. *AFH,* Anterior facial height; *ANB,* subspinale-nasion-supramentale angle; *AO-BO,* Subspinale (Pt. A) perpendicular to occlusal plane Supramentale (Pt B) perpendicular to occlusal plane; *FMA,* Frankfort mandibular plane angle; *FMIA,* Frankfort mandibular incisor axis angle; *IMPA,* incisor mandibular plane angle; *OCC,* The angle made by occlusal plane when it intersects Frankfort; *PFH,* posterior facial height; *SNA,* sella-nasion-subspinale angle; *SNB,* sella-nasion-supramentale angle; *TC,* total chin thickness; *UL,* upper lip thickness; *Z,* angle is the inferior angle the profile line makes with Frankfort horizontal plane.

FIGURE 19-14 Facial photographs: 18 months of treatment.



FMIA	53	OCC	7
FMA	32	Z	56
IMPA	95	UL	14 mm
SNA	78	TC	10 mm
SNB	74	PFH	43 mm
ANB	4	AFH	72 mm
AO-BO	2 mm	Index	0.59

FIGURE 19-15 Progress cephalometric tracing. See Figure 19-13 for abbreviations.

tracing (Fig. 19-15) illustrates mandibular incisor flaring and loss of vertical dimension control. Compare the pretreatment photos with the "progress" photographs (Fig. 19-16). Note the respective profile lines.

The patient's treatment plan was altered. Premolars were removed. The posttreatment facial photographs (Fig. 19-17) show a balanced face. The posttreatment casts (Fig. 19-18) illustrate a well-interdigitated occlusion. The posttreatment cephalometric tracing (Fig. 19-19) confirms mandibular incisors that are now in a proper position over basal bone. The pretreatment, "progress," and posttreatment facial photographs (Fig. 19-20) illustrate the Tweed-Merrifield concept of treatment planning for maximum balance and harmony of the lower face.

Cranial Disharmony

An analytic assessment of the skeletal pattern is an integral part of any diagnosis. A careful cranial analysis must include an understanding of the following information, which can be gleaned from a cephalogram (Figs. 19-21 and 19-22).

Skeletal Analysis Values

Frankfort Mandibular Plane Angle. The FMA is probably the most significant value for skeletal analysis because it defines the direction of lower facial growth in the horizontal and vertical dimensions. The standard or normal range of 22 to 28 degrees for this value reflects a skeletal pattern with normal growth direction. An FMA greater than the normal range indicates excessive vertical growth direction, and an FMA less than the normal range indicates deficient vertical growth.

Incisor Mandibular Plane Angle. The incisor mandibular plane angle (IMPA) defines the axial inclination of the mandibular incisors in relation to the mandibular plane. The IMPA is a good guide to use in maintaining or positioning these teeth in their relationship to basal bone. The standard of 88 degrees indicates an upright mandibular incisor position that with a normal FMA will ensure optimal balance and harmony of the lower face. If the FMA is above normal, the orthodontist must dentally compensate with more uprighting of the mandibular incisors. If the FMA is below the normal range, compensation can be made by leaving the mandibular incisors in their pretreatment position or, in rare instances, by positioning them more to the labial. Labial inclination of the mandibular incisors is generally limited to their original inclination if the patient has normal muscular balance.

Sella-Nasion-Subspinale Angle (SNA). The sella-nasion-subspinale (SNA) angle indicates the relative horizontal position of the maxilla to cranial base. The range at the termination of growth is 80 to 84 degrees for a white population sample.

Sella-Nasion-Supramentale Angle. The sella-nasion-supramentale (SNB) angle expresses the horizontal relationship of the mandible to the cranial base. A range of 78 to 82 degrees indicates a normal horizontal mandibular position. If the value is less than 74 degrees and a large maxillomandibular discrepancy exists, orthognathic surgery should be considered as an adjunct to orthodontic treatment. The same concern should be accorded to a value of more than 84 degrees.

Subspinale-Nasion-Supramentale Angle. The normal range for the subspinale-nasion-supramentale (ANB) angle is 1 to 5 degrees. This value expresses a very "treatable" horizontal

FIGURE 19-16 Pretreatment photographs with the "progress" photographs.

FIGURE 19-17 Posttreatment facial photographs.

relationship of the maxilla to the mandible. Class II malocclusions become proportionally more difficult with higher ANB angles. A patient with an ANB greater than 10 degrees will usually require surgery as an adjunct to proper treatment. The negative ANB angle is even more indicative of facial disproportion in the horizontal dimension. An ANB angle of −3 degrees or more, when the mandible is in its centric relation, indicates a possibility for the need of surgical assistance in Class III malocclusion correction.

Subspinale (Pt. A) perpendicular to occlusal plane; supramentale (pt. B) perpendicular to occlusal plane. Orthodontic treatment becomes more difficult when the AO-BO is outside the normal range of 0 to 4 mm. The AO-BO changes in direct proportion to the occlusal plane angle.

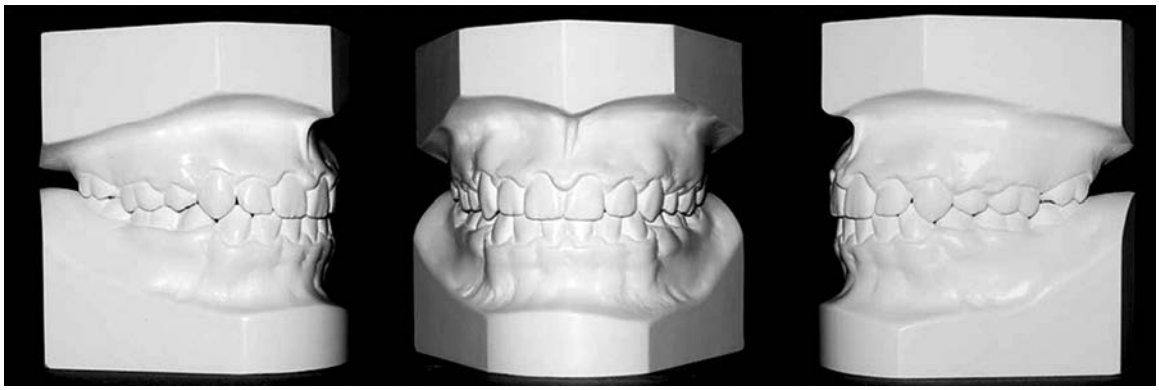
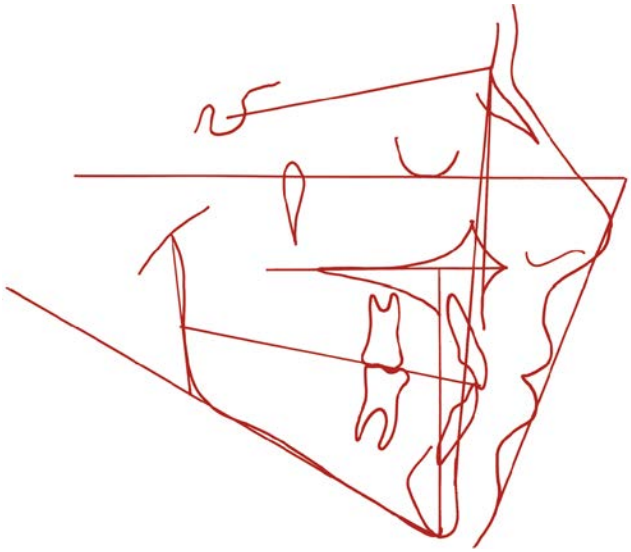


FIGURE 19-18 Posttreatment casts.



FMIA	66	OCC	9
FMA	30	Z	71
IMPA	84	UL	17 mm
SNA	78	TC	12 mm
SNB	76	PFH	47 mm
ANB	2	AFH	74 mm
AO-BO	–1 mm	Index	0.63

FIGURE 19-19 Posttreatment cephalometric tracing. See Figure 19-13 for abbreviations.

Occlusal Plane. The occlusal plane angle expresses a dentoskeletal relationship of the occlusal plane to the Frankfort horizontal plane. A range of 8 to 12 degrees is normal, with variations of about 2 degrees between males and females. The angle for females averages about 9 degrees and for males about 11 degrees. Values greater or less than the normal range indicate more difficulty in treatment. During most orthodontic malocclusion corrections, the original value should be maintained or decreased. An increase in the occlusal plane angle during treatment indicates a loss of vertical control. An increase is usually unstable because the occlusal plane is determined by muscular balance, primarily the muscles of mastication. The occlusal plane angle frequently returns to its pretreatment value when it is increased. The result is a detrimental relapse of the "corrected" interdental relationship.

Posterior Facial Height. Posterior facial height (PFH) is a linear millimetric measurement of ramus height from articulare to the mandibular plane tangent to the posterior border of the ramus. The value is related to facial form, vertically and horizontally. The relationship of PFH to anterior facial height determines the FMA and lower facial proportion. In a growing child with a Class II malocclusion, ramal growth change and its relationship to anterior facial height in proportion and in volume are crucial.

Anterior Facial Height. Anterior facial height (AFH) is a linear millimetric measurement of the vertical distance between the palatal plane and menton. The line is drawn perpendicular to the palatal plane. A value of about 65 mm for a 12-year-old individual indicates a normal anterior facial height. This vertical value must be monitored carefully if it is 5 mm more or less than normal. During Class II malocclusion correction, limiting an increase in AFH is essential and can be accomplished by controlling maxillary and mandibular molar extrusion in conjunction with an anterior "high-pull" force on the maxilla.

Facial Height Index. André Horn[23] studied the relationship of AFH to PFH. After developing the facial height index, he found that normal PFH is 69% of AFH. The normal range of PFH to AFH is 0.65 to 0.75. If the value is less than or greater than this range, the malocclusion is more complex, and the orthodontist will encounter more difficulty in correction. An index of 0.80 is severe and indicates a "low FMA" malocclusion caused by too much posterior ramal growth or too little anterior growth. As the index approaches 0.60, the skeletal pattern demonstrates too little posterior height or too much anterior height.

Facial Height Change Ratio. Radziminski,[24] Gebeck and Merrifield,[25,26] Issacson,[27] Pearson,[28,29] and Schudy[30] have described the important relationship between vertical dimension control and successful treatment of Class II malocclusions. After an evaluation of successfully and unsuccessfully treated Class II malocclusions, Merrifield and Gebeck[26,31,32] concluded that successfully treated patients exhibited favorable mandibular changes. These changes occurred in part because AFH was controlled while PFH increased. Unsuccessful treatment results were more likely to occur in patients in whom an increase was observed in AFH but not in PFH.

Craniofacial Analysis

For a period of about 15 years, until his untimely death in June 1993, Jim Gramling of Jonesboro, Arkansas, was the research director for the Charles H. Tweed Foundation. During those years, Gramling compiled a large sample of successfully and unsuccessfully treated Class II malocclusions.[33,34]

FIGURE 19-20 Pretreatment, "progress," and posttreatment facial photographs.

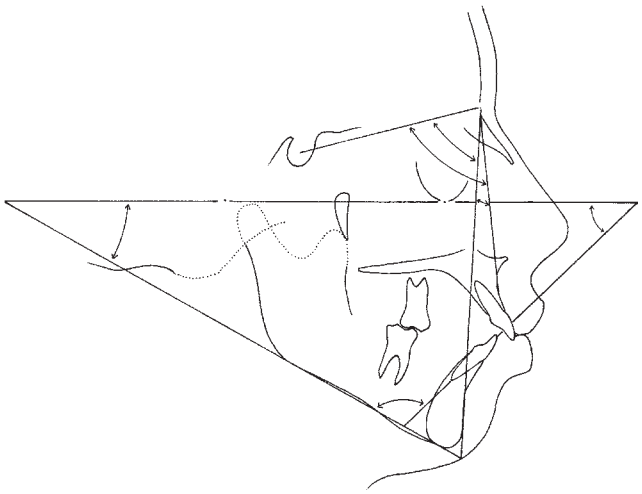


FIGURE 19-21 Cephalometric tracing with all values and planes.

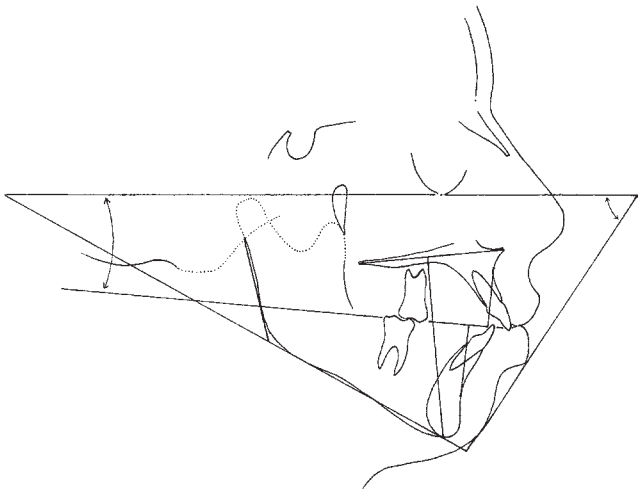


FIGURE 19-22 Cephalometric tracing with all values and planes.

TABLE 19-1 The Probability Index Variables with Statistically Computed Difficulty Factors*

Cephalometric Angle	Variation
FMA	5 points
ANB	15 points
FMIA	2 points
Occlusal plane	3 points
SNB	5 points

*This was Gramling's initial attempt at a probability index.
ANB, Subspinale-nasion-supramentale angle; *FMA,* Frankfort mandibular plane angle; *FMIA,* Frankfort mandibular incisor axis angle; *SNB,* sella-nasion-supramentale angle.

From the background of evidence gathered from these studies, Gramling formulated a probability index[35] for three specific purposes:

1. To augment diagnostic procedures
2. To guide treatment procedures
3. To predict possible treatment success or failure

Gramling statistically established a difficulty factor and assigned a specific number of points to each variable (Table 19-1).

In 1989, Gramling studied a different sample of 40 successful and 40 unsuccessful Class II malocclusion corrections.[36] After this study, he changed the "successful" FMA range of 18 to 35 degrees to 22 to 28 degrees (Table 19-2).

Using information from the previously described clinical research, the craniofacial analysis (Table 19-3), an integral component of the differential diagnostic analysis system, was developed. Each of the six cephalometric values used has been determined to have significant merit. In determining the difficulty of correction, the areas were weighted, taking into consideration the necessary diagnostic decisions and the complexity and importance of treatment management.

The FMA, the AFH/PFH ratio, and the occlusal plane to Frankfort angle are significant when used as a group. These values make up the vertical component of the craniofacial analysis. The vertical skeletal pattern can be a problem of excessive AFH in the presence of a decreased PFH, or conversely, a problem of excessive PFH and a decreased AFH. If facial height, anterior or posterior, is out of proper proportion, correction of the

TABLE 19-2 The Probability Index after the Pretreatment Range for the Frankfort Mandibular Plane Angle Was Adjusted Downward to 22 to 28 Degrees

	Point Value	Cephalometric Value	Probability Index
FMA 22–28 degrees	5	—	—
ANB ≤6 degrees	15	—	—
FMIA ≥60 degrees	2	—	—
Occlusal plane ≤7 degrees	3	—	—
SNB ≥80 degrees	5	—	—
Total			—

ANB, Subspinale-nasion-supramentale angle; *FMA,* Frankfort mandibular plane angle; *FMIA,* Frankfort mandibular incisor axis angle; *SNB,* sella-nasion-supramentale angle.

TABLE 19-3 Craniofacial Analysis

Normal Range	Cephalometric Value	Difficulty Factor	Difficulty
FMA 22–28 degrees	—	5	—
ANB 1–5 degrees	—	15	—
Z angle 70–80 degrees	—	2	—
Occlusal plane 8–12 degrees	—	3	—
SNB 78–82 degrees	—	5	—
FHI (PFH-AFH) 0.65–0.75	—	3	—
Craniofacial difficulty total			—

AFH, Anterior facial height; *ANB,* subspinale-nasion-supramentale angle; *FHI,* facial height index, FHI = AFH/PFH; *FMA,* Frankfort mandibular plane angle; *FMIA,* Frankfort mandibular incisor axis angle; *PFH,* posterior facial height; *SNB,* sella-nasion-supramentale angle.

malocclusion is more difficult, and one must take great care with treatment procedures so that the vertical disharmonies do not significantly worsen.

The horizontal skeletal component of the craniofacial analysis is composed of the SNB and the ANB. A high ANB caused by a low SNB makes the horizontal skeletal disharmony much more difficult to manage than if the high ANB is caused by an excessive SNA. The low SNB requires a treatment compromise or, if a more ideal result is desired, orthognathic surgery.

The previously described Z angle value is the only nonskeletal measurement in the craniofacial analysis. The Z angle was included because it is a facial reflector of craniofacial imbalance.

Dental Disharmony

Total Dentition Space Analysis

Along with a consideration of the face and the skeletal pattern, the orthodontist must consider the dentition (Table 19-4). Total space analysis as described by Merrifield[13] is divided into three parts: anterior, midarch, and posterior. This division is made for two reasons: (1) simplicity in identifying the area of space deficit or space surplus and (2) accuracy in differential diagnosis.

Anterior Space Analysis

Anterior space analysis includes the measurement in millimeters of the space available in the mandibular arch from canine

TABLE 19-4 Total Space Analysis

Area	Value	Difficulty Factor	Difficulty
Anterior			
Tooth arch discrepancy	—	1.5	—
Head film discrepancy	—	1.0	—
Total	—		—
Midarch			
Total arch discrepancy	—		—
Curve of Spee	—		—
Total	—		—
Occlusal disharmony (Class II or Class III)	—	2	—
Posterior			
Tooth arch discrepancy	—		
Expected increase (–)	—		
Total	—	0.5	—
Space analysis total	—	Space analysis difficulty total	—

to canine and a measurement of the mesiodistal dimension of each of the six anterior teeth. The difference is referred to as a surplus or deficit. Tweed's cephalometric discrepancy is used to further analyze this area. The cephalometric discrepancy is defined as the amount of space required to upright the mandibular incisors for optimum facial balance. This value is added to the anterior space measurement.

The sum of the anterior tooth arch surplus or deficit and the cephalometric discrepancy is referred to as the anterior discrepancy. Each of the values in the anterior discrepancy calculation has been given a difficulty factor so that an anterior space analysis difficulty value can be calculated.

Midarch Space Analysis

The midarch area includes the mandibular first molars and the first and second premolars. The midarch is an important area of the dentition. Careful analysis of this area may show mesially inclined first molars, rotations, spaces, a deep curve of Spee, crossbites, missing teeth, habit abnormality, blocked-out teeth, tooth size discrepancies, and occlusal disharmonies. Careful measurement of the space from the distal of the canine to the distal of the first molar should be recorded as available midarch space. An equally accurate measurement of the mesiodistal width of the first premolar, the second premolar, and the first molar must also be recorded. To this value is added the space required to level the curve of Spee.[37] From these measurements, the orthodontist can determine the space deficit or surplus in this area.

Occlusal disharmony, a Class II or III buccal segment relationship, although not a part of the actual midarch space analysis, must be measured because an occlusal disharmony adds a great deal to the difficulty of correction of any malocclusion and requires careful treatment strategy and space management.

Occlusal disharmony is best measured by articulating the casts and by using the maxillary first premolar cusp and its relationship to the mandibular first premolar–second premolar embrasure as a reference. Measure mesially or distally from the maxillary first premolar buccal cusp to the embrasure between the mandibular first and second premolars. This measurement is made on both sides and is then averaged to determine the

occlusal disharmony. The difficulty factor for occlusal disharmony is 2, so the averaged disharmony is doubled and added to the midarch difficulty because it has to be corrected by moving teeth that are in the midarch area of the dentition.

Posterior Space Analysis

The posterior dentition area is of great importance. The dentition has a posterior limit. Regardless of age, this posterior limit appears to be the anterior border of the ramus. The required space in the posterior space analysis is the mesiodistal width of the second molars and the third molars in the mandibular arch. The available space is more difficult to ascertain in an immature patient. It is a linear measurement in millimeters of the space distal to the mandibular first molars. The measurement is made from the distal of the mandibular first molar to the anterior border of the ramus along the occlusal plane. The posterior limit is recognized as being 2 to 3 mm distal to the anterior border of the ramus because of the lingual shelf that exists to accommodate the mandibular molars. However, teeth on the lingual shelf are not generally in good functional occlusion.

An estimate of posterior arch length increase based on age and gender is added to this value. Certain unpredictable variables must be considered in estimating the increase in posterior available space. These variables are the following:

1. Rate of mesioocclusal migration of the mandibular first molar
2. Rate of resorption of the anterior border of the ramus
3. Time of cessation of molar migration
4. Time of cessation of ramus resorption
5. Gender
6. Age

A review of the literature[38-40] reveals that the consensus from researchers suggests that 3 mm of increase per year occurs in the posterior dentition area until age 14 years for girls and age 16 years for boys. This is an increase of 1.5 mm per year on each side of the arch after the complete eruption of the first molars. In a mature patient (girls beyond 15 years and boys beyond 16 years), a measurement from the distal of the first molar to the anterior border of the ramus at the occlusal plane is a valuable determination of the space available in the posterior area. This measurement is important in diagnosis and treatment because it gives the clinician the ability to know whether a surplus or deficit of space exists in this area.

To create a posterior discrepancy while making adjustments in either the midarch or anterior area is not prudent. Equally imprudent is to not use a posterior space surplus to help alleviate midarch and anterior deficits. The most easily recognizable symptom of a posterior space deficit in a young patient is the late eruption of the second molars. If space is not available for these teeth by the age of their normal eruption, it should be obvious that a posterior space problem exists. The posterior space analysis surplus or deficit has been given a low difficulty factor of 0.5 because a posterior space deficit can be easily resolved with third molar removal.

Differential Diagnostic Analysis System

The two diagnostic tools that have been described, the craniofacial analysis and the total dentition space analysis, used together, make up the differential diagnostic analysis system (Table 19-5). Use of this diagnostic methodology will significantly improve the clinician's ability to diagnose, plan, and execute treatment. The sum of the craniofacial difficulty and the total dentition space analysis difficulty is called the *total difficulty*. This value gives the clinician a quantitative method of evaluating the difficulty of correction for each malocclusion. The analysis identifies the specific areas of major disharmony (i.e., facial, skeletal, and dental) and gives guidance for treatment strategy.

Other factors such as habit evaluation, joint health, muscle balance, dental or skeletal malrelationships, and other cephalometric values must be duly noted, evaluated by the orthodontist, and factored into any diagnosis. The orthodontist must also evaluate the patient's motivation and desire for correction of the malocclusion. The range of values for the total difficulty that have been found to be most appropriate when malocclusion correction difficulty is studied are as follows: mild, 0 to 60; moderate, 60 to 120; and severe, more than 120.

TWEED-MERRIFIELD EDGEWISE APPLIANCE

Brackets and Tubes

An appliance is an instrument used to achieve orthodontic goals. As Angle stressed, an appliance must have certain characteristics: simplicity, efficiency, and comfort. An appliance must also be hygienic and aesthetic and, above all, have a wide range of versatility. The neutral 0.022 slot edgewise appliance consists of posterior bands and anterior mesh pads with single, double-width 0.022 brackets on the six anterior teeth; intermediate single-width brackets on premolar bands; twin brackets on first molar bands; and heavy edgewise 0.022 tubes with mesial hooks on second molar bands (Fig. 19-23).

All bands have lingual cleats attached. Lingual cleats increase versatility and are especially necessary to correct and control rotations. Each of the brackets and tubes is placed at a right angle to the long axis of the tooth. The brackets are positioned precisely in relation to the incisal edges of the incisor teeth and the cusps of the remaining teeth. No tip, torque, or variations in thickness are present in the bracket. A slot size of 0.022 allows the clinician to use a multiplicity of archwire dimensions.

Archwires

Resilient edgewise archwire is used with the Tweed-Merrifield 0.022-inch edgewise appliance. The dimensions (in inches) of the wire commonly used are 0.017 × 0.022, 0.018 × 0.025, 0.019 × 0.025, 0.020 × 0.025, and 0.0215 × 0.028. These wire dimensions give a great range of versatility with the 0.022 × 0.028 bracket slot and allow the sequential application of forces as needed for various treatment objectives. The objective is to enhance tooth movement and control with the proper edgewise archwire at the appropriate time.

First-, Second-, and Third-Order Bends and Their Interaction

Knowledge of the action, interaction, and reaction of teeth to bends in the archwire is crucial to the use of any orthodontic appliance. Such knowledge is fundamental and dramatically affects clinical results.

First-Order Bends

The action and reaction of first-order bends affect expansion or contraction. These actions are monitored easily and are routinely used to move individual teeth. The interaction of the bends can affect the third-order position of the teeth if expansion forces are used.

TABLE 19-5 Differential Diagnostic Analysis System

CRANIOFACIAL ANALYSIS			
Normal Range	**Cephalometric Value**	**Difficulty Factor**	**Difficulty**
FMA 22–28 degrees	—	5	—
ANB 1–5 degrees	—	15	—
Z angle 70–80 degrees	—	2	—
Occlusal plane 8–12 degrees	—	3	—
SNB 78–82 degrees	—	5	—
FHI (PFH-AFH) 0.65–0.75	—	3	—
Craniofacial difficulty total			—
TOTAL SPACE ANALYSIS			
	Value	**Difficulty Factor**	**Difficulty**
Anterior			
Tooth arch discrepancy	—	1.5	—
Head film discrepancy	—	1.0	—
Total	—		
Midarch	—		
Tooth arch discrepancy	—	1.0	—
Curve of Spee	—	1.0	—
Total	—		
Occlusal disharmony (Class II or Class III)	—	2.0	—
Posterior			
Tooth arch discrepancy	—		
Expected increase (–)	—		
Total	—	0.5	—
Space analysis total	—	Space analysis difficulty total	—
Craniofacial difficulty total	—		
Space analysis difficulty total	—		
Total difficulty	—		

AFH, Anterior facial height; *ANB,* subspinale-nasion-supramentale angle; *FHI,* facial height index, FHI = AFH/PFH ; *FMA,* Frankfort mandibular plane angle; *PFH,* posterior facial height; *SNB,* sella-nasion-supramentale angle.

FIGURE 19-23 Tweed-Merrifield edgewise appliance. The appliance is composed of neutral 0.022-inch edgewise slots with double-width brackets on the six anterior teeth, intermediate single-width brackets on the premolar bands, twin brackets on the first molars, and 0.022-inch tubes with mesial hooks on the second molars.

Second-Order Bends

Second-order bends in the posterior segment of the mandibular archwire are antagonistic to the teeth in the anterior segment. Without excellent directional control and a careful application of these second-order forces in a sequential manner, vertical control of the anterior teeth will be lost.

Second-order bends in the posterior segment of the mandibular archwire also negatively affect the third-order position of the mandibular anterior teeth. Therefore, the mandibular anterior teeth generally require lingual crown torque in the archwire because posterior second-order tipping bends apply labial crown torque force to the incisors. This fact must be given careful consideration in archwire fabrication and force application.

In the maxillary arch, second-order bends (an exaggerated curve of Spee) in the posterior segments are generally desirable or complementary to the teeth in the anterior segment. The reaction to the tipping forces intrudes the maxillary incisors and gives a lingual root torque effect to these teeth. This is generally positive or complementary to treatment objectives.

Third-Order Bends

Third-order bend reaction in the mandibular archwire is complementary to all the teeth if properly placed. The objective is to

have some degree of lingual crown torque on all the mandibular teeth. The posterior and anterior segments work together in action, reaction, and interaction. The ideal third-order bends (lingual crown torque) in the mandibular archwire are as follows: incisors, 7 degrees; canines and first premolars, 12 degrees; and second premolars and molars, 20 degrees.

Conversely, third-order bends in the maxillary archwire are antagonistic. The anterior segment needs no torque (0 degrees), or it needs lingual root torque, and the posterior segment needs lingual crown torque: canines and first premolars (7 degrees) and second premolars and molars (12 degrees). Application of active third-order force simultaneously in segments with opposite third-order requirements is not wise. In the maxillary arch, applying active third-order bends sequentially and in only one direction at any given time is prudent.

Auxiliaries

The auxiliaries routinely used with the Tweed-Merrifield edgewise force system are elastics and directionally oriented headgear, primarily the high-pull J-hook headgear. Patient compliance with auxiliaries is imperative.

Variations of the Appliance

Many variations of the edgewise appliance have been introduced in the past 30 years. Most notable of the variations is the "straight wire" appliance (SWA) introduced in 1972 by Larry Andrews.[41] The SWA incorporates first-, second-, and third-order bends into the bracket. The theory behind this approach is that these bends will not have to be placed in the archwire. Another variation in the standard appliance is a decrease in slot size from 0.022 to 0.018 inch and even to 0.016 inch. Various orthodontic suppliers market numerous variations of the SWA with different tips and torques to suit the individual operator's desires. Other modifications have been extensively described by Burstone,[42] Lindquist,[43] and Roth.[44]

TREATMENT WITH THE TWEED-MERRIFIELD EDGEWISE APPLIANCE

Any treatment protocol must complement a diagnostic philosophy. Using Tweed's treatment concepts as a foundation, Merrifield developed force systems that simplify the use of the edgewise appliance. For example, Tweed used 12 sets of archwires during the treatment of each patient. Today, with the modern edgewise appliance, only three to five sets of archwires are used. Merrifield's sequential directional force technology[45-47] is simple, straightforward, and fundamentally sound. From the era of Tweed and into the era of Merrifield, the key to quality treatment with the edgewise appliance continues to be directionally controlled precision archwire manipulation. Essentially, five concepts compose the treatment philosophy: (1) sequential appliance placement; (2) sequential or individual tooth movement (or both); (3) sequential mandibular anchorage preparation; (4) directional forces, including control of the vertical dimension to enhance a favorable mandible to maxilla spatial change; and (5) proper timing of treatment.

Sequential Appliance Placement

The application of the appliance to the patient is important. In a first premolar extraction patient, the second molars and the second premolars are banded. Initially, the first molars are left unbanded. The central incisors, lateral incisors, and canines are bonded. Anterior teeth that are malaligned are not ligated to the archwire or are ligated passively. This method of sequential appliance placement is less traumatic to the patient and is easier and less time consuming for the orthodontist. The method allows much greater efficiency in the action of the archwire during the first months of treatment because it gives the posterior segment of the archwire much longer interbracket length. This length creates a power storage that accomplishes second molar movement more rapidly. Sequential appliance placement also gives the orthodontist the opportunity to insert a wire of larger dimension that is less subject to occlusal or bracket engagement distortion.

After the banded and bracketed teeth respond to the forces of the archwire and auxiliaries, the first molars are banded. The maxillary first molars are banded after one appointment. The mandibular first molars are banded after the second appointment.

Sequential Tooth Movement

Tooth movement is sequential. It is not the en masse movement that was introduced by Tweed. Individual teeth are moved rapidly and with precision because they are moved singly or in small units.

Sequential Mandibular Anchorage Preparation

Tweed attempted, with varying degrees of success, to prepare mandibular anchorage with Class III elastics. All the compensation bends were placed in the archwire at one time. Sequelae of this force system were often labially flared and intruded mandibular incisors. Sequential mandibular anchorage preparation, developed by Merrifield, is a system that allows mandibular anchorage to be prepared quickly and easily by tipping only two teeth at a time to their anchorage prepared position. This system uses high-pull headgear and anterior vertical elastics rather than Class III elastics for support. Unlike the en masse anchorage of the Tweed era, movement is controlled, sequential, and precise.

Directional Force

The hallmark of modern Tweed-Merrifield edgewise treatment is the use of directional force systems to move the teeth. Directional forces can be defined as controlled forces that place the teeth in the most harmonious relationship with their environment.

To use a force system that controls the mandibular posterior teeth and the maxillary anterior teeth is crucial. The resultant vector of all forces should be upward and forward to enhance the opportunity for a favorable skeletal change, particularly during dentoalveolar protrusion Class II malocclusion correction (Fig. 19-24). An upward and forward force system requires that the mandibular incisor be upright over basal bone so that the maxillary incisor can be moved properly (Fig. 19-25). For the upward and forward force system to be a reality, vertical control is crucial. To control the vertical dimension, the clinician must control the mandibular plane, palatal plane, and occlusal plane. If point B drops down and back, the face becomes lengthened, the mandibular incisor is tipped forward off basal bone, and the maxillary incisor drops down and back instead of being moved to a proper functional and aesthetic position (Fig. 19-26). The unfortunate result of point B dropping down and back is a patient with a lengthened face, a gummy smile, incompetent lips, and a more recessive chin.

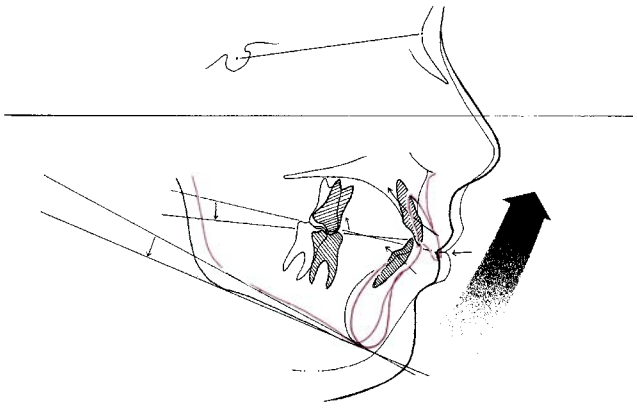


FIGURE 19-24 Upward and forward force system.

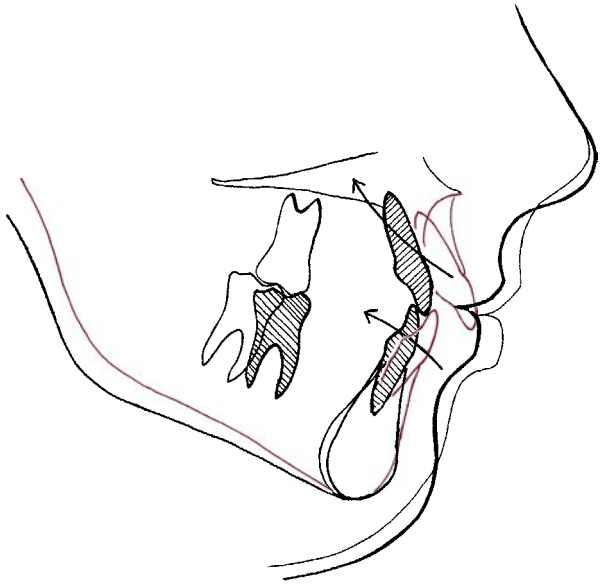


FIGURE 19-25 Upright mandibular incisor and maxillary incisor moved up and back.

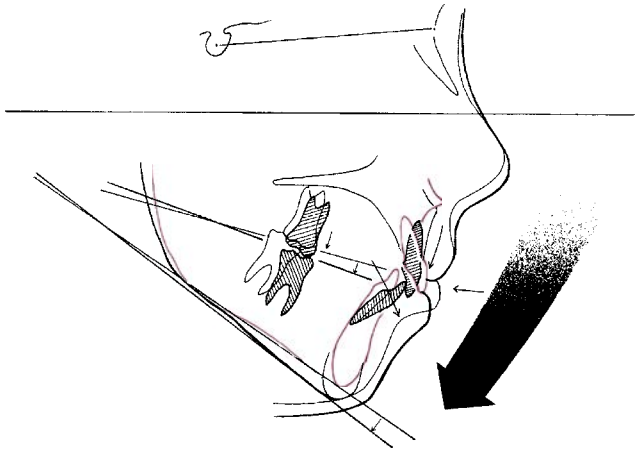


FIGURE 19-26 Downward and backward force system.

Timing of Treatment

The timing of treatment is an integral part of the philosophy. Treatment should be initiated at the time when treatment objectives can be accomplished most readily. This may mean interceptive treatment in the mixed dentition, selected extractions in the mixed dentition, or waiting for permanent second molar eruption before initiating active treatment. Diagnostic discretion is the determinant.

STEPS OF TREATMENT

Tweed-Merrifield edgewise directional force treatment can be organized into four distinct steps: denture preparation, denture correction, denture completion, and denture recovery. During each step of treatment, certain objectives must be attained.

Denture Preparation

Denture preparation prepares the malocclusion for correction. Objectives include the following:

1. Leveling
2. Individual tooth movement and rotation correction
3. Retraction of maxillary and mandibular canines

The denture preparation step of treatment takes about 6 months. One mandibular archwire and one maxillary archwire are used to complete this step.

The teeth of the original malocclusion are sequentially banded and bonded (Fig. 19-27). After the placement of the appliance, an 0.018- × 0.025-inch resilient mandibular archwire and a 0.017- × 0.022-inch resilient maxillary archwire are inserted. The omega loop stops are flush against the second molar tubes in each arch. The mandibular second molar receives an effective distal tip that will upright its mesial inclination. In the maxillary arch, a 20-degree tip is placed in the wire distal to the omega loop stop to maintain the distal inclination of the second molar. The objective in each respective arch is to maintain the maxillary molar in its distally tipped position and to begin to distally tip the mandibular second molar enough to level it into the arch.

A second premolar offset bend is placed mesial to the second premolar bracket in each archwire. The purpose of this bend is to prevent the canines from expanding labially as they are retracted with the headgear. The third-order bends in each

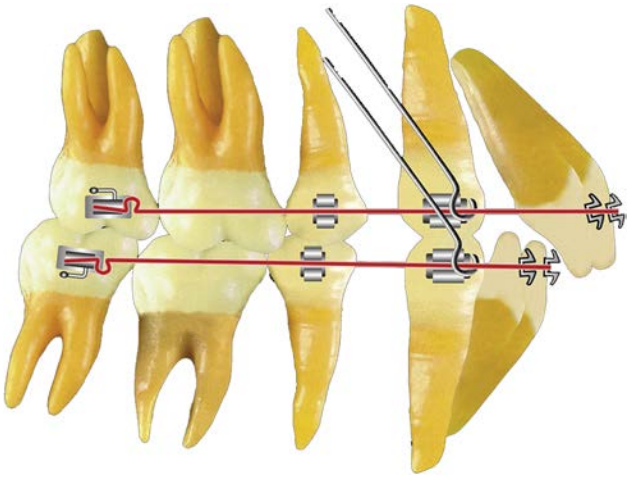


FIGURE 19-27 Denture preparation: Initial archwires consist of a 0.017- × 0.022-inch resilient maxillary archwire and a 0.018- × 0.025-inch resilient mandibular archwire.

archwire are ideal. High-pull J-hook headgear is used to retract maxillary and mandibular canines. After the first month of treatment, the maxillary first molars are banded, and after the second month of treatment, the mandibular first molars are banded. As the canines retract and the arches are leveled, the lateral incisors are ligated, and power chain force to aid canine retraction can be used (Fig. 19-28).

One must remember that at each visit during denture preparation, the archwires are removed; carefully coordinated; checked for proper first-, second-, and third-order bends; and religated. Canine retraction is continued with power chain and headgear force. At the end of the denture preparation stage of treatment, the dentition should be level, the canines should be retracted, all rotations should be corrected, and the mandibular second molars should be level (Fig. 19-29).

Denture Correction

The second step of treatment is called denture correction. During denture correction, the spaces are closed with maxillary and mandibular closing loop archwires. Vertical support to the maxillary arch is achieved with J-hook headgear attached to hooks soldered to the maxillary archwire between the maxillary central and lateral incisors. Vertical support of the mandibular anterior teeth is accomplished with anterior vertical elastics. The mandibular archwire is a 0.019- × 0.025-inch working archwire with 7.0-mm vertical loops distal to the lateral incisor brackets. The 0.020- × 0.025-inch maxillary archwire has 7.5-mm vertical loops distal to the lateral incisor brackets. In both arches, the omega loop stops are immediately distal to the brackets of the first molars (Fig. 19-30). The maxillary archwire is coordinated with the mandibular archwire. The archwires are activated each month until all space is closed. At the end of space closure (Fig. 19-31), the curve of occlusion in the maxillary arch should have been maintained, and the mandibular

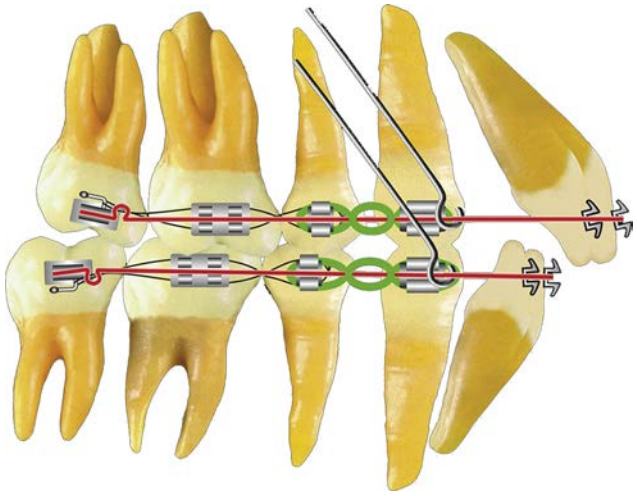


FIGURE 19-28 Denture preparation: The canines are retracted with a J-hook headgear during denture preparation.

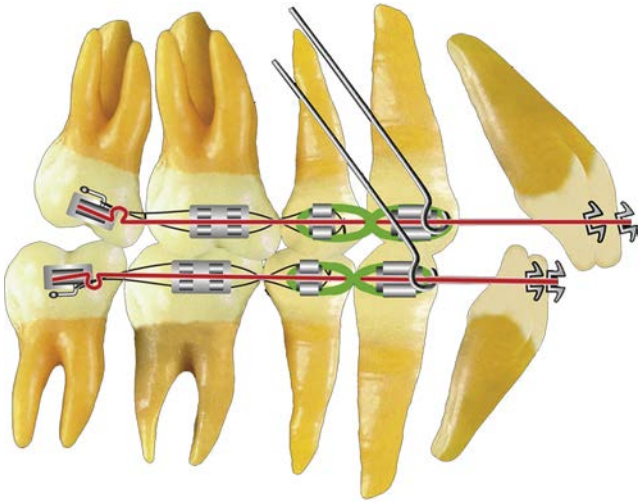


FIGURE 19-29 Denture preparation: At the end of denture preparation the arches are level, rotations are corrected, and the canines are retracted.

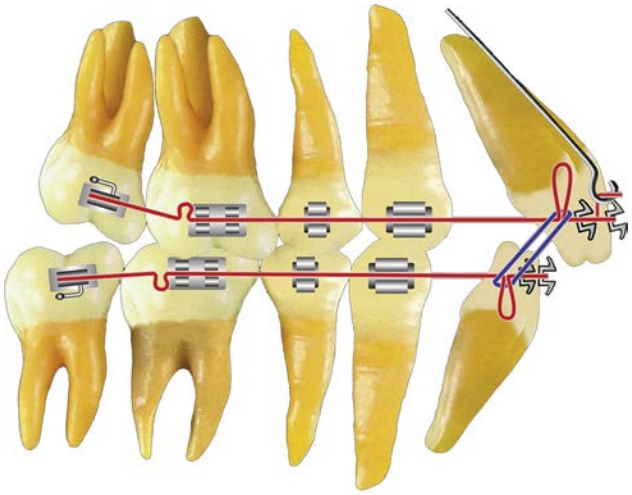


FIGURE 19-30 Denture correction: Maxillary and mandibular closing loops are used to close the space mesial to the distalized canines.

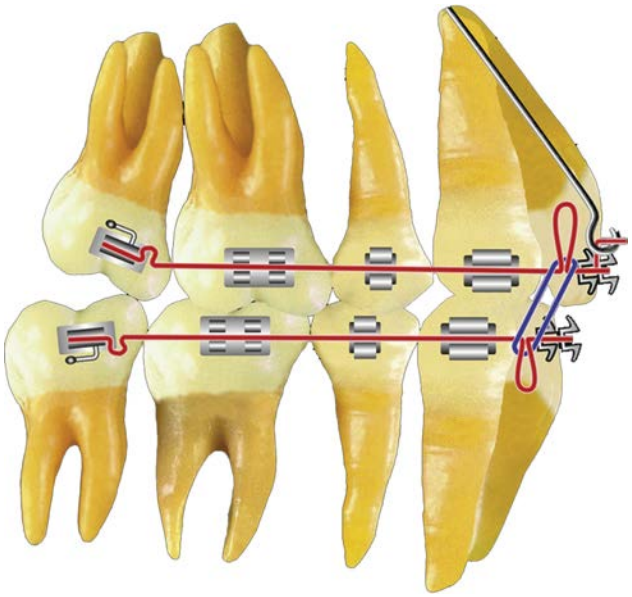


FIGURE 19-31 Space closure. The arches are level, and all spaces are totally closed.

arch should be completely level. The dentition is now ready for mandibular anchorage preparation. This step positions teeth in the mandibular midarch and posterior areas into axial inclinations that will allow final coordination with the maxillary teeth for normal functional occlusion.

Sequential Mandibular Anchorage Preparation

Sequential mandibular anchorage preparation is based on the concept of sequential tooth movement. The archwire produces an active force on only two teeth while remaining passive to the other teeth in the arch. Therefore, the remaining teeth act as stabilizing or anchorage units as two teeth are tipped. The method is referred to as the "10-2" (10 teeth vs. 2 teeth) anchorage system, and it allows a quickly controlled response without serious adverse reaction. The anchorage preparation system is supported by anterior vertical elastics attached to spurs that are soldered distal and gingival to the mandibular lateral incisors. The elastics are hooked to the closing loops of the maxillary archwire. They are supported by a high-pull headgear that is attached to hooks soldered to the maxillary archwire.

After closing the mandibular space, the mandibular arch must be level. At this time, the first step of sequential mandibular anchorage preparation, second molar anchorage, is initiated. Another 0.019- × 0.025-inch archwire with the omega loop stops bent flush against the second molar tubes is fabricated. First- and third-order bends are ideal. Gingival spurs for anterior vertical elastics are soldered distal to the lateral incisors.

To tip the mandibular second molars to an anchorage prepared position, a 15-degree tip is placed distal to the omega loop stop. The second molar is tipped to an anchorage prepared position. It should have a distal inclination of 10 degrees to 15 degrees, which can be verified with a readout (Fig. 19-32, *A*).[48]

After the second molar has been tipped, the first molar is tipped to its anchorage prepared position by placing a 10-degree distal tip 1 mm mesial to the first molar bracket. When this first molar tip is placed in the archwire, a compensating bend that maintains the 15-degree second molar inclination must be placed mesial to the omega loop stop (Fig. 19-32, *B*).

The archwire is now passive to the second molar and crosses the twin brackets of the first molar at a 10-degree bias. The second molars are now part of the 10 stabilizing units, and the first molars are the two teeth that receive the action of the directional forces and the archwire. After 1 month, the archwire is removed, and a readout should show a 5- to 8-degree distal inclination of the first molars. The second molars should continue to read out at 15 degrees.

The denture correction step of treatment should now be complete for the Class I malocclusion. The objectives of the denture correction step are (1) complete space closure in both arches, (2) sequential anchorage preparation in the mandibular arch, (3) an enhanced curve of occlusion in the maxillary arch, and (4) a Class I intercuspation of the canines and premolars. The mesiobuccal cusp of the maxillary first molar should fit into the mesiobuccal groove of the mandibular first molar. The distal cusps of these teeth should be discluded, as should the second molars.

Class II Force System

For patients with an "end-on" Class II dental relationship of the buccal segments at the conclusion of space closure, a new force system must be used to complete the denture correction stage of treatment. A careful study of the cusp relationships will determine the force system required. Making a final diagnostic decision for Class II correction is usually based on (1) the ANB relationship, (2) a maxillary posterior space analysis, and (3) patient cooperation. The following guidelines are used:

1. If the maxillary third molars are missing or if the ANB is 5 degrees or less and the patient is cooperative, the system to be described will accomplish the best result. If the third molars are present and are approaching eruption, they should be removed to facilitate distal movement of the maxillary teeth.
2. If a cooperative patient has (a) a mild Class II dental relationship, (b) a normal vertical skeletal pattern (FMA of ≤28

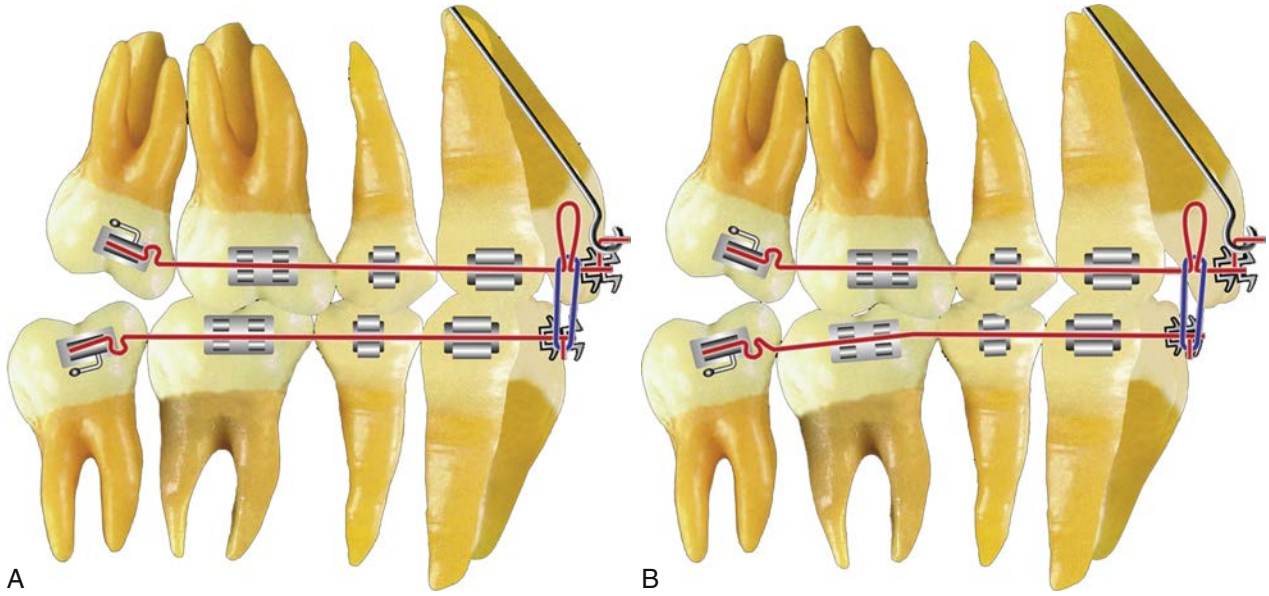


FIGURE 19-32 Mandibular anchorage preparation. A, 10-2-7. The second molar is tipped to its anchorage prepared position. B, 10-2-6. The first molar is tipped to its anchorage prepared position.

degrees), (c) an ANB of 5 to 8 degrees, and (d) normally erupting maxillary third molars, the extraction of maxillary second molars is most advantageous. The force system to be described is used to distalize the maxillary dentition into the second molar extraction space.

3. If (a) the ANB is approaching 10 degrees, (b) maxillary third molars are present, (c) there is a full step Class II molar occlusion, or (d) the patient's motivation is questionable, the first molars may be considered for removal after the maxillary and mandibular first premolar has been closed. Surgical correction may also be considered. Facial balance and harmony "projections" after correction with either molar extraction or orthognathic surgery should be considered carefully before making either decision.

The Class II force system cannot be used unless compliance requirements are strictly followed by the patient. If one attempts to use the Class II force system without cooperation, the maxillary anterior teeth will be pushed forward off basal bone.

Orthodontic Correction of the Class II Dental Relationship

At the end of sequential mandibular anchorage preparation, a mandibular 0.0215- × 0.028-inch stabilizing archwire is fabricated. Ideal first-, second-, and third-order bends are incorporated into the archwire. The omega loop stop must be 0.5 mm short of the molar tubes, and the wire must be passive to all the brackets. Gingival spurs are soldered distal to the mandibular lateral incisors. The wire is seated and ligated, and the terminal molar is cinched tightly to the loop stop.

A 0.020- × 0.025-inch maxillary archwire with 7.5-mm closed helical bulbous loops bent flush against the second molar tubes is fabricated. The helix is wound to the lingual during fabrication. This archwire has ideal first- and second-order bends. The molar segment has 7 degrees of progressive lingual crown torque. A gingival spur is attached to the archwire immediately distal to the maxillary second premolar bracket. Gingival high-pull headgear hooks are soldered distal to the central incisors. Class II "lay on" hooks with a gingival extension for anterior vertical elastics are soldered distal to the lateral incisors. Before archwire insertion, the closed helical bulbous loops are opened 1 mm on each side. The anterior segment of the archwire will be 1 mm labial to the maxillary incisor brackets. The archwire is ligated in place. Class II elastics are worn from the hooks on the mandibular second molar tubes to the Class II hooks on the maxillary archwire. Anterior vertical elastics are worn from the spurs on the mandibular archwire to the gingival extension hooks on the maxillary archwire. The high-pull headgear is worn on the maxillary headgear hooks (Fig. 19-33, *A*).

This force system is used to sequentially move the maxillary second molars distally. At each appointment, the mandibular archwire is removed and checked, and the helical bulbous loops are activated 1 mm. The activation of the maxillary archwire is repeated until the second molars have a Class I dental relationship (Fig. 19-33, *B*). When the Class I relationship of the second molars has been established, a closed coil spring is "wound" distal to the second premolar spur and compressed between the spur and the first molar bracket when the maxillary archwire is inserted. (The coil spring length should be 1.5 times the space between the second premolar and the first molar brackets.) An elastic chain is stretched from the second molar to the distal bracket of the first molar. The spring and the elastic chain create a distal force on the maxillary first molar. Additionally, a Class II elastic is worn continuously from the mandibular second molar hook to the Class II hook on the maxillary archwire. An anterior vertical elastic is worn 12 hours each day (Fig. 19-34). The high-pull headgear is worn 14 hours per day on the spurs soldered to the maxillary archwire. This is an efficient force system for first molar distalization (Fig. 19-35).

After the first molars have been moved distally into an overcorrected Class I dental relationship, the spur that was attached distal to the second premolar bracket is removed. The coil spring is moved mesially so that it is compressed between the lay-on hook and the canine bracket. Subsequently, the maxillary second premolars and the maxillary canines are moved distally with elastic chain and headgear force (Fig. 19-36). Four months

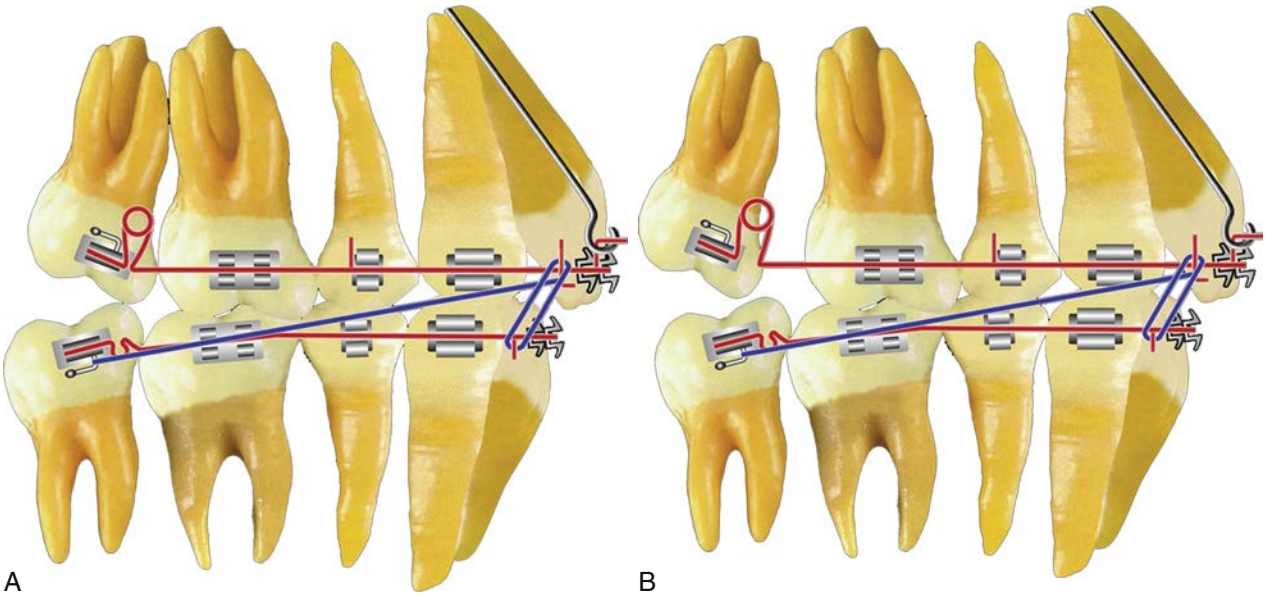


FIGURE 19-33 Denture correction: Class II force system. Maxillary second molar distalization. **A,** Step 1. A helical bulbous loop is placed against the maxillary second molar. **B,** Step 2. The helical bulbous loop pushes the maxillary molar distally to a Class I relationship.

of treatment with monthly reactivation should position the posterior teeth and the maxillary canine in an overtreated Class I relationship. This system will not strain the mandibular arch if the anterior vertical elastics are worn and if sufficient space is available in the maxillary posterior denture area.

After overcorrection of the Class II dental relationship, a 0.020- × 0.025-inch maxillary archwire with 7.5-mm closing loops distal to the lateral incisors is fabricated. This archwire has ideal first-, second-, and third-order bends. Gingival headgear hooks are soldered distal to the central incisors (Fig. 19-37). The closing loops are opened 1 mm per visit by cinching the omega loop stops to the molar tube. Class II elastics, anterior vertical elastics, and the maxillary high-pull headgear are used. After all the maxillary space is closed, the denture correction step of treatment has been completed. The dentition is ready for the next step of treatment—denture completion.

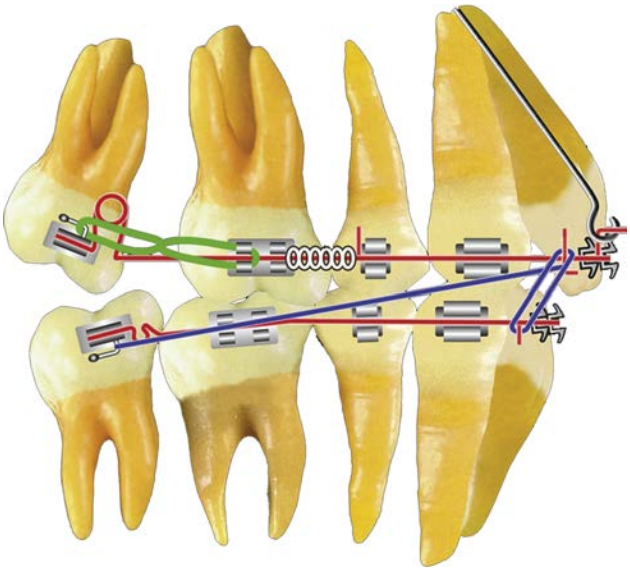


FIGURE 19-34 Denture correction: Class II force system. A coil spring is trapped mesial to the first molar.

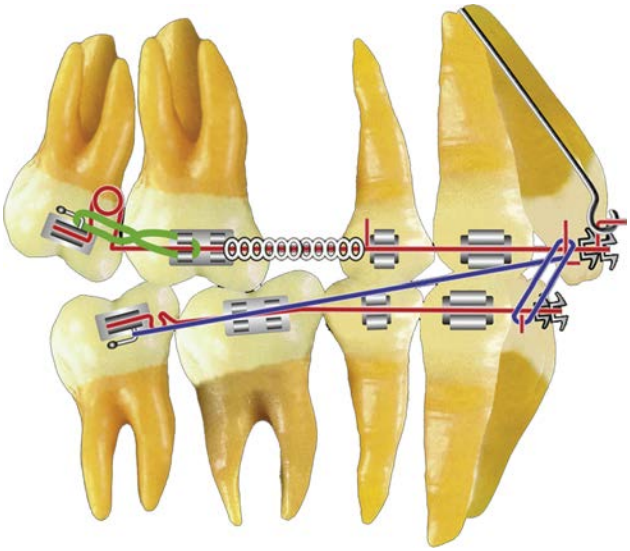


FIGURE 19-35 Denture correction: Class II force system. Maxillary first molar distalization.

Denture Completion

The third step of treatment is identified as denture completion. Ideal first-, second-, and third-order bends are placed in finishing mandibular and maxillary 0.0215- × 0.028-inch resilient archwires. The mandibular archwire duplicates the previously used mandibular stabilizing archwire. The maxillary archwire has artistic bends and hooks for the high-pull headgear, anterior vertical elastics, and Class II elastics. Supplemental hooks for vertical elastics are soldered as needed (Fig. 19-38).

The forces used during denture completion are based on a careful study of the arrangement of each tooth in each arch. The orthodontist must also study the relationship of one arch to the other and the relationship of the arches to their environment. The orthodontist makes necessary first-, second-, and third-order adjustments in each archwire as needed. A progress cephalogram and tracing

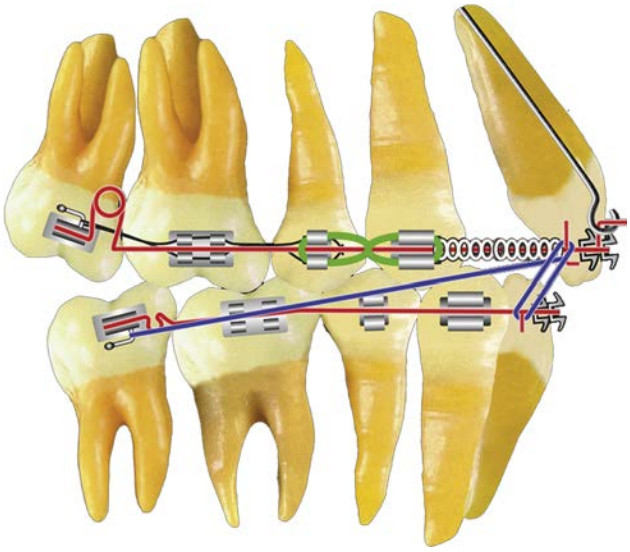


FIGURE 19-36 Denture correction: Class II force system. Maxillary second premolar and maxillary canine distalization. After molar distalization the premolars and canines are distalized.

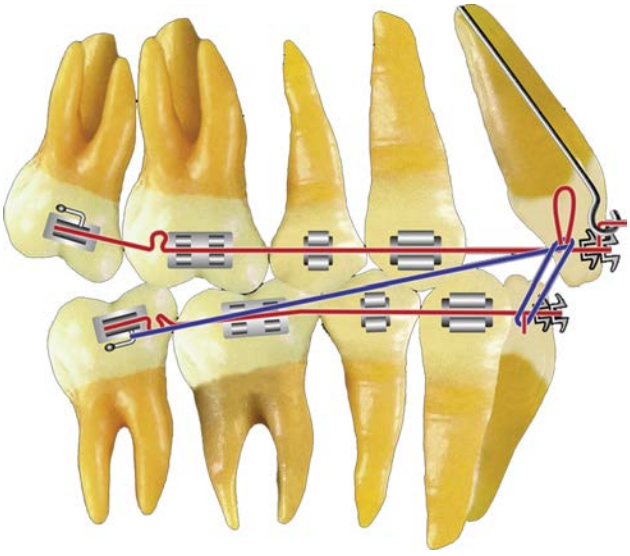


FIGURE 19-37 Denture correction: Class II force system. Maxillary anterior space closure. A 0.020- × 0.025-inch maxillary closing loop archwire is used to close the maxillary anterior space.

can be evaluated to determine the final mandibular incisor position and any minor control of the palatal, occlusal, and mandibular planes that may be needed. Study of the cephalogram may also reveal to the clinician the requirement for lingual root torque in the maxillary incisors. Visual clinical observations permit evaluation of the lip line, the maxillary incisor relationship, and the amount of cusp seating and artistic positioning of the incisors that is necessary.

Denture completion can be considered as minitreatment of the malocclusion. During this treatment step, the orthodontist uses the forces that are necessary until the original malocclusion is overcorrected.

Denture Recovery

An ideal occlusion will be present only after all treatment mechanics are discontinued and uninhibited function and other environmental influences active in the posttreatment period stabilize and finalize the position of the total dentition. When all appliances are removed and the retainers are placed, a most crucial "recovery" phase occurs. During this recovery period, the forces involved are those of the surrounding environment, primarily the muscles and the periodontium. If mechanical corrective procedures barely achieve normal relationships of the teeth, relapse is inevitable. Any change is likely to be away from ideal occlusion toward malocclusion. Recovery, based on a concept of overcorrection, is predicated on clinical experience and research.

The posttreatment occlusion, which is carefully planned, sometimes referred to as Tweed occlusion but properly identified as transitional occlusion (Fig. 19-39), is characterized by disclusion of the second molars. The mesiolingual cusp of the maxillary first molar is seated into the central fossa of the mandibular first molar with the mesial inclined plane of the mesial cusp of the maxillary first molar contacting the distal inclined plane of the mesial cusp of the mandibular first molar. This arrangement allows the muscles of mastication to effect the greatest force on the "primary chewing table" in the midarch area. The slightly intruded distally inclined maxillary and mandibular second molars now can "reerupt" to a healthy functional occlusion without trauma or premature contact. Because of overtreatment of Class I and Class II deep-bite patients, the anterior teeth are positioned in an end-to-end relationship with no overbite or overjet. This relationship, however, is transitory and will adjust rapidly to an ideal overjet and overbite relationship (Fig. 19-40).

The correction of two malocclusions will be illustrated. These patients were treated with the Tweed-Merrifield edgewise appliance. The reason for showing the records of these patients is to illustrate that all treatment objectives can be routinely attained in the treatment of different types of malocclusions if the force

FIGURE 19-39 Transitional occlusion: The occlusion must be overtreated. The anterior teeth should be edge to edge. The canines, second premolars, and first molars should have a solid Class I dental relationship. The second molars should be tipped out of occlusion.

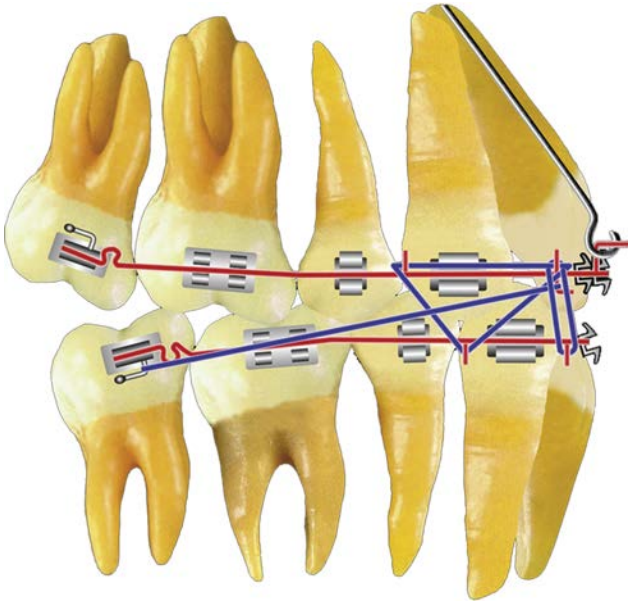


FIGURE 19-38 Denture completion: Maxillary and mandibular stabilizing archwires, along with the proper elastics and headgear force, are used to complete the orthodontic treatment.

FIGURE 19-40 Final occlusion is characterized by the teeth settling into their most efficient, healthy, and stable positions.

system that has been presented is used. The diagnosis of the first malocclusion is described in detail to illustrate the use of the craniofacial analysis and the total dentition space analysis for making the differential diagnosis from which the treatment plan is formulated. This first malocclusion was a protruded Class II, division I subdivision malocclusion with facial imbalance. The force system used to correct the malocclusion was the Class II force system previously described in this chapter.

SUMMARY

The standard edgewise appliance has endured the test of time. Angle was determined to use it to correct malocclusions while preserving the "full complement of teeth." Angle collaborated with Charles H. Tweed, who, after countless failures, introduced the extraction of four first premolars and anchorage preparation to produce facial balance. The use of the appliance evolved with Levern Merrifield over many more years. He introduced the following: (1) differential diagnosis, which led to the removal of the teeth that would best produce balance, harmony, and facial proportion within the cranial–facial complex; (2) directional force technology; and (3) sequential archwire manipulation. The edgewise appliance is a precise instrument for the routine correction of major malocclusions. Although the Tweed-Merrifield edgewise appliance is the direct descendent of the appliance invented in 1928 by Edward H. Angle, it is used with a totally different philosophy of treatment. A consistent effort is being continuously directed toward its further sophistication. The edgewise appliance has stood the test of time and will be used in the future by many more generations of orthodontists.

Acknowledgment

The images that illustrate the force systems that are described in this chapter were created by Sergio Cardiel Rios, Morelia, Michoacan, Mexico.

CASE REPORTS: MALOCCLUSION CORRECTION WITH THE EDGEWISE APPLIANCE

Patient 1: NY

This 12-year-old boy shows a malocclusion with facial, dental, and craniofacial disharmony.

Profile and full face photographs (Fig. 19-41) illustrate the protrusion and the facial imbalance. The casts (Fig. 19-42) exhibit an Angle Class II end-to-end occlusion on the right, a deep impinging overbite, and flared and protrusive maxillary and mandibular incisors. The pretreatment cephalogram and its tracing (Fig. 19-43) illustrate an ANB of 7 degrees and an AO-BO

FIGURE 19-41 Pretreatment facial photographs.

FIGURE 19-42 Pretreatment casts.

of 14 mm, both of which are indicators of the severity of the malocclusion. The FMIA of 58 degrees and the Z angle of 63 degrees reflect the protrusive face. Mandibular incisors are flared. The differential diagnostic analysis system was used to give some objectivity to the patient's skeletal, dental, and facial problems. The craniofacial analysis shows a craniofacial difficulty total of 71.0. The SNB is lower than normal. The ANB is larger than normal. The facial protrusion needs to be corrected. The total dentition space analysis reflects the protrusion in the anterior dentition area with a cephalometric correction discrepancy factor of 9.6, a curve of Spee of 2.5 mm, and a 5-mm Class II occlusal issue. The total anterior discrepancy difficulty was 14.1 mm, the midarch and the occlusal disharmony discrepancy was 12.5 mm, and the posterior discrepancy was 9 mm. The space analysis difficulty was 35.6 mm. Total dentition difficulty was 106.6 (Fig. 19-44).

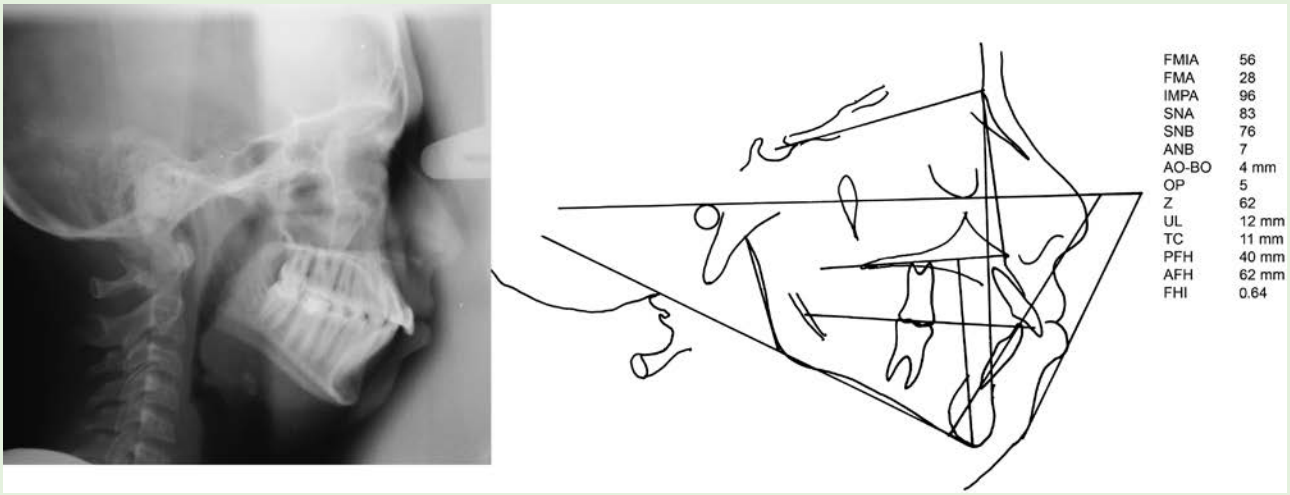


FIGURE 19-43 Pretreatment cephalogram and cephalometric tracing. See Figure 19-13 for abbreviations.

Cranial Facial Analysis

Normal Range	Cephalometric Value	Difficulty Factor	Difficulty
FMA 22–28	28	5	0
ANB 1–5	7	15	30
Z angle 70–80	62	2	16
Occusal plane 8–12	4	3	12
SNB 78–82	76	5	10
PFH/AFH 0.65–0.75	0.64	3	3
	Craniofacial Difficulty Total		**71**

Total Space Analysis

Anterior	Value	Difficulty Factor	Difficulty
Tooth arch discrepancy	3.0	1.5	4.5
Headfilm discrepancy	9.6	1.0	9.6
Total	12.6		14.1
Midarch			
Tooth arch discrepancy	0.0	1.0	0.0
Curve of Spee	2.5	1.0	2.5
Total	2.5		2.5
Horizontal occlusal disharmony (Class II or III)	5.0	2.0	10.0
Posterior			
Tooth arch discrepancy	20.0		
(-)Expected increase	12.0		
Total	18.0	.5	9.0
Space analysis total	**35.6**	**Space Analysis Difficulty total**	35.6

Craniofacial difficulty total	**71.0**
Space analysis difficulty total	**35.6**
Total difficulty	**106.6**

Difficulty Index: mild, 0 to 60; moderate, 60 to 120; severe, 120+.

FIGURE 19-44 Total dentition difficulty analysis. See Figure 19-13 for abbreviations.

Continued

CASE REPORTS: MALOCCLUSION CORRECTION WITH THE EDGEWISE APPLIANCE—cont'd

Because of facial and dental protrusion and the immediate need to upright the mandibular incisors, maxillary and mandibular first premolars were extracted. The patient was banded and bonded according to the protocol that has been described. The force system that has been described was used. The patient's malocclusion correction was completed with 22 months of active appliance therapy. The patient's posttreatment facial photographs (Fig. 19-45), when compared with the pretreatment photographs, exhibit a significant and positive change in facial balance. The protrusion no longer exists, and the patient has a pleasing profile. The posttreatment casts (Fig. 19-46) exhibit control of the dentition and creation of an ideal Angle Class I dental relationship on the right side. Some distal tipping of the mandibular molars should be noted. These teeth have been tipped out of occlusion because of the anchorage preparation that enhanced vertical control during the Class II elastic wear. Arch form has been maintained. The posttreatment cephalogram and tracing (Fig. 19-47) illustrate changes in many of the cephalometric values. FMIA increased from 56 degrees to 69 degrees. IMPA decreased from 96 degrees to 84 degrees. The Z angle increased from 63 degrees to 78 degrees. These values are clear indications that the goals for facial balance and harmony were met. Superimposition tracings (Fig. 19-48) illustrate the control of the maxillary and mandibular molars, uprighting of the mandibular incisors, and the upward and backward movement of the maxillary incisors. The direction of the spatial change of the mandible in relation to the maxilla was downward and forward. This downward and forward change contributed greatly to the improved facial balance and harmony. The smiling photographs of the patient (Fig. 19-49) confirm intrusion of the incisors and a considerably better smile with less gingival display.

Patient 2: CL

A patient whose treatment plan requires the extraction of maxillary first premolars and mandibular second premolars generally has the following characteristics: (1) Balance and harmony of the lower face (Fig. 19-50) is within reasonable limits, or there is only a mild facial imbalance. In other words, these patients do not generally exhibit a great degree of unbalance of the lower face. (2) The dentition (Fig. 19-51) is most often a Class II dental relationship and mild mandibular and/or maxillary anterior crowding. Many of these patients have a protrusion of the maxillary anterior teeth. (3) The cephalometric examination of the skeletal pattern most often reveals a moderate mandibular plane angle, mandibular incisors that are "reasonably upright" over basal bone, and a discrepancy in the

FIGURE 19-45 Posttreatment facial photographs.

FIGURE 19-46 Posttreatment casts (transitional occlusion).

CASE REPORTS: MALOCCLUSION CORRECTION WITH THE EDGEWISE APPLIANCE—cont'd

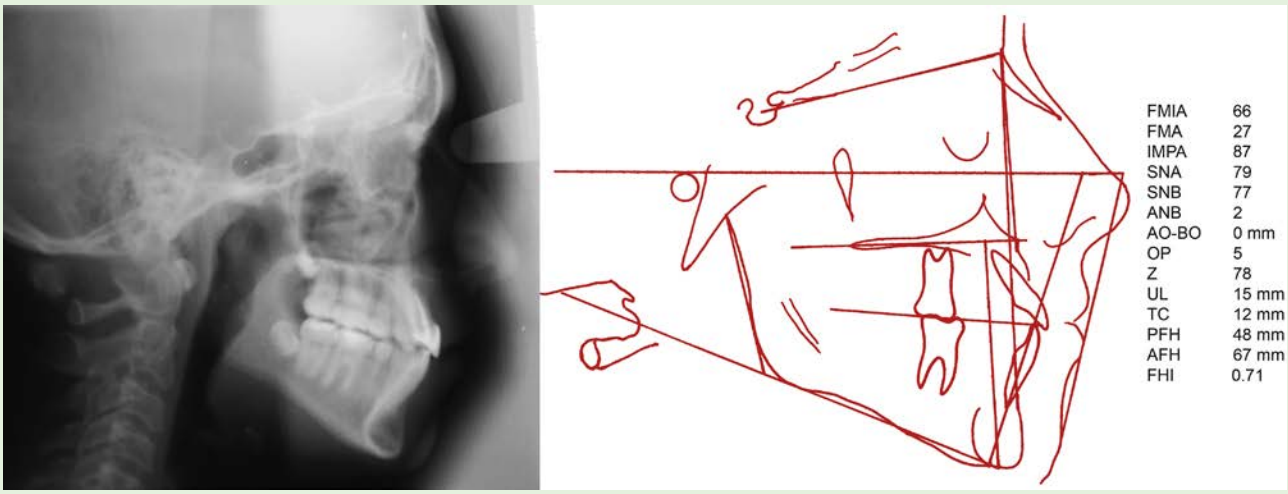


FIGURE 19-47 Posttreatment cephalogram and cephalometric tracing. See Figure 19-13 for abbreviations.

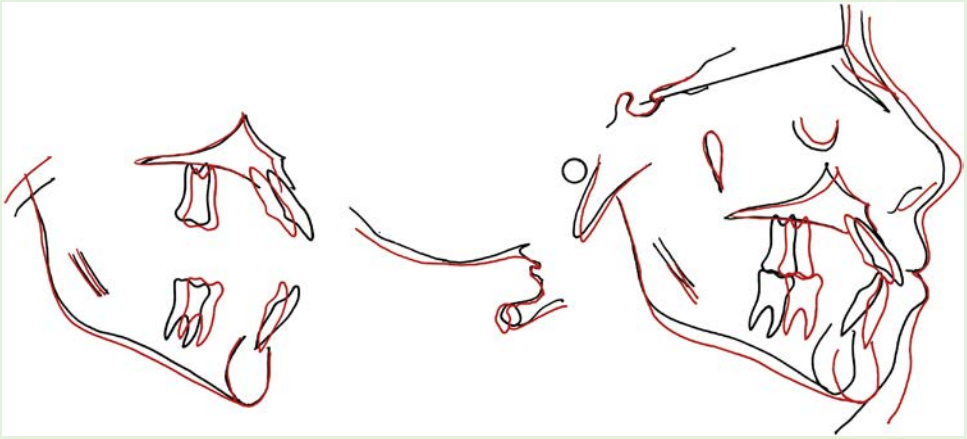


FIGURE 19-48 Pretreatment–posttreatment superimpostitions.

FIGURE 19-49 Pretreatment–posttreatment smiling photographs.

Continued

CASE REPORTS: MALOCCLUSION CORRECTION WITH THE EDGEWISE APPLIANCE—cont'd

FIGURE 19-50 Pretreatment facial photographs.

FIGURE 19-51 Pretreatment casts.

anteroposterior relationship of the mandible to the maxilla. In our example, patient CL has an ANB of 7 degrees, an FMIA of 61 degrees, and an IMPA of 97 degrees (Fig. 19-52). These are values that one would expect for patients who "fit" into this facial, skeletal, and dental pattern.

When making the differential diagnosis, it is important to understand the relationship of the mandible to the maxilla and the relationship of the teeth to the face. In this particular example, the FMIA, Tweed's "facial angle," must be increased if the patient is to have improved balance and harmony in the lower face. Patient CL was treated with the extraction of the maxillary first premolars and the mandibular second premolars. The mandibular incisors were uprighted, the posterior occlusion was corrected by mesial movement of the mandibular posterior teeth, and crowding was ameliorated.

The mesialization of the mandibular first molar is accomplished with a variation of the mandibular archwires used during denture preparation (Fig. 19-53, *A–D*). This system is based on control of the mandibular anterior and posterior teeth as the second premolar extraction space is closed. As the mandibular first molar is protracted, the second molar drifts mesially. When all mandibular space is closed, the second molar is banded, mandibular anchorage is prepared, and treatment is finished with the archwire sequences that have been previously described.

The posttreatment facial photographs (Fig. 19-54) illustrate improvement in balance and harmony of the lower face. The chin is not as recessive. There is a nice curl in the maxillary lip and less eversion of the lower lip. The pretreatment–posttreatment casts (Fig. 19-55) confirm correction of the posterior occlusion and crowding amelioration. The posttreatment cephalogram and cephalometric tracing (Fig. 19-56) confirm mild uprighting of the mandibular incisors, an increase in FMIA from 61 degrees to 66 degrees, maintenance of the mandibular plane angle, and reduction of the ANB from 7 degrees to 3 degrees because of retraction of point A. The superimpositions (Fig. 19-57) confirm a nice spatial change of the mandible in its relationship to the maxilla, vertical control of the molars, mild uprighting of the mandibular incisors, and intrusion and retraction of the maxillary incisors.

The correction of two distinctly different types of malocclusions has been illustrated. Both of these malocclusions were recently treated. The intent of showing NY's records is to illustrate the fact that all treatment objectives can be routinely obtained in the treatment of a difficult malocclusion if the force system that has been presented is used. The treatment planning of NY's malocclusion is described in detail to illustrate the use of the craniofacial analysis and the total dentition space analysis. This malocclusion was an Angles Class II, division I, subdivision, with facial imbalance. The Class II force system, which has been described in this chapter, was used on the patient's right side after all premolar extraction spaces had been closed. Because this patient was recently treated, his casts reflect the transitional occlusion that will settle into the final occlusion.

The second case report is presented to illustrate the carefully planned correction of a malocclusion that was treatment planned for the extraction of the maxillary first and mandibular second premolars. The treatment goal was to improve the mild facial imbalance and to correct the dental disharmony. The Tweed-Merrifield force system that was used to protract the mandibular first molars was described and illustrated.

CASE REPORTS: MALOCCLUSION CORRECTION WITH THE EDGEWISE APPLIANCE—cont'd

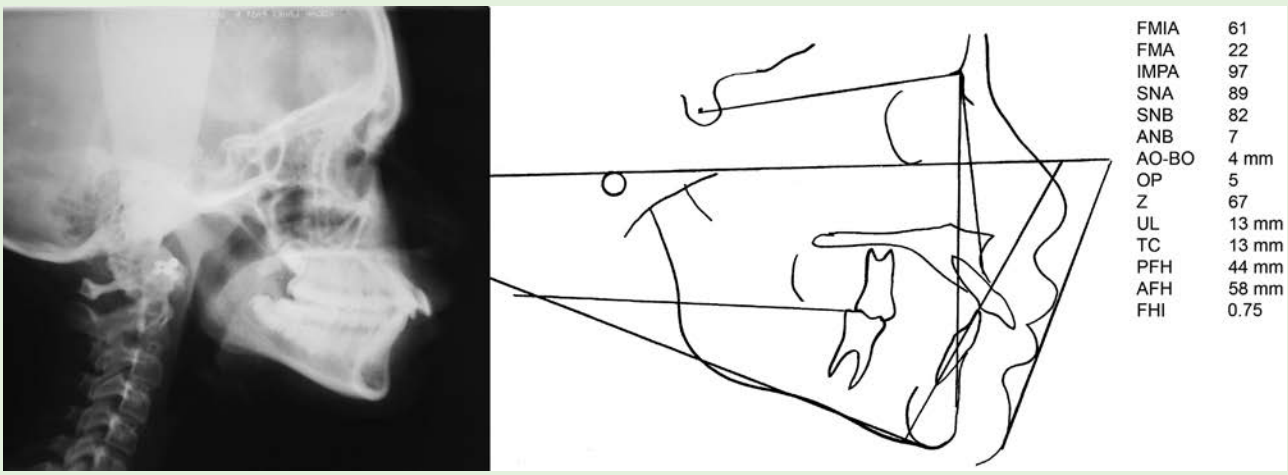


FIGURE 19-52 Pretreatment cephalogram and cephalometric tracing. See Figure 19-13 for abbreviations.

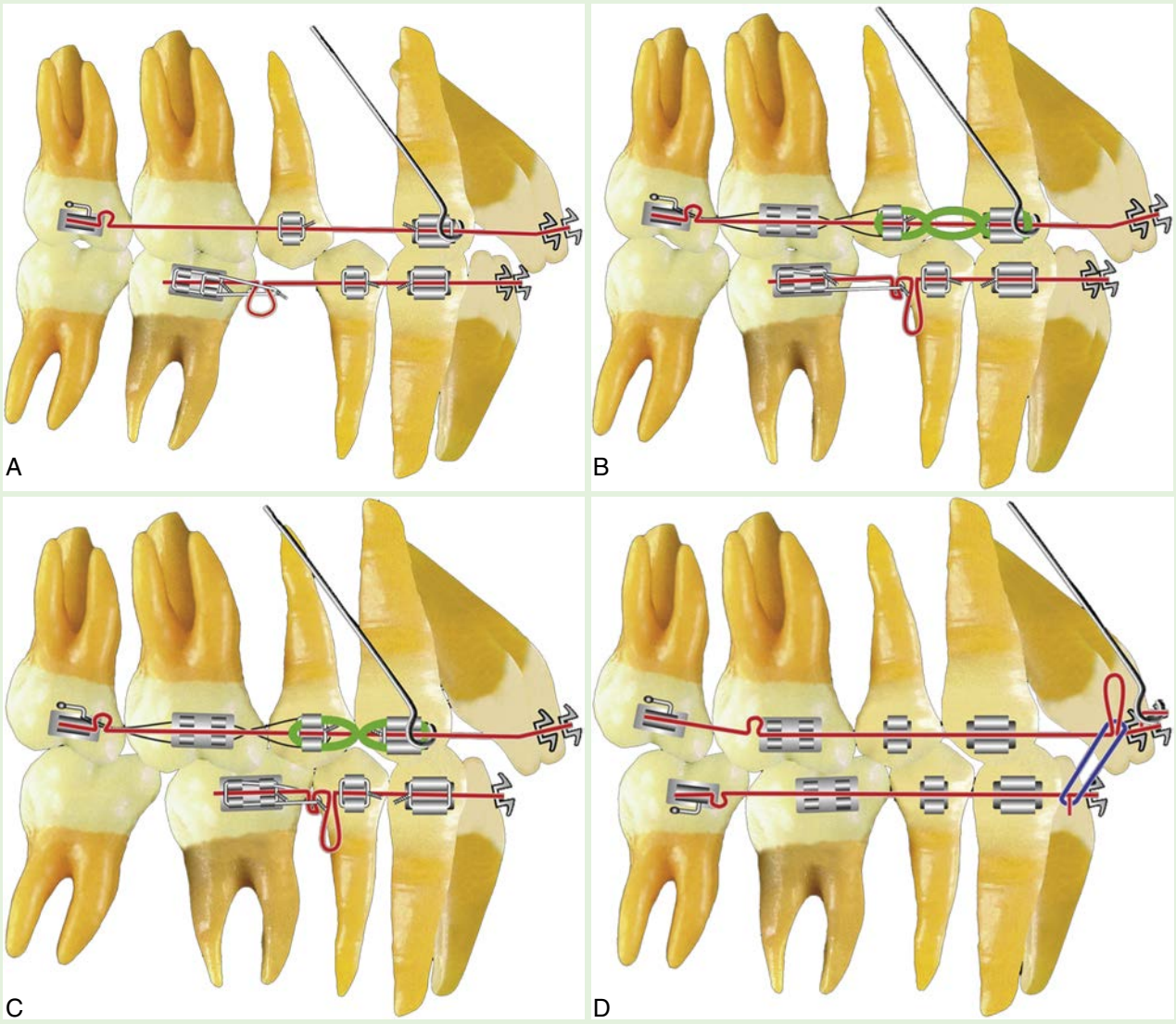


FIGURE 19-53 **A,** Initial archwires for a maxillary first, mandibular second premolar extraction patient. **B,** Mandibular first molar protraction archwire. **C,** The maxillary canine has been retracted; the mandibular first molar has been protracted. **D,** The mandibular second molar is banded, and mandibular anchorage is prepared.

Continued

CASE REPORTS: MALOCCLUSION CORRECTION WITH THE EDGEWISE APPLIANCE—cont'd

FIGURE 19-54 Posttreatment facial photographs.

FIGURE 19-55 Posttreatment casts (transitional occlusion).

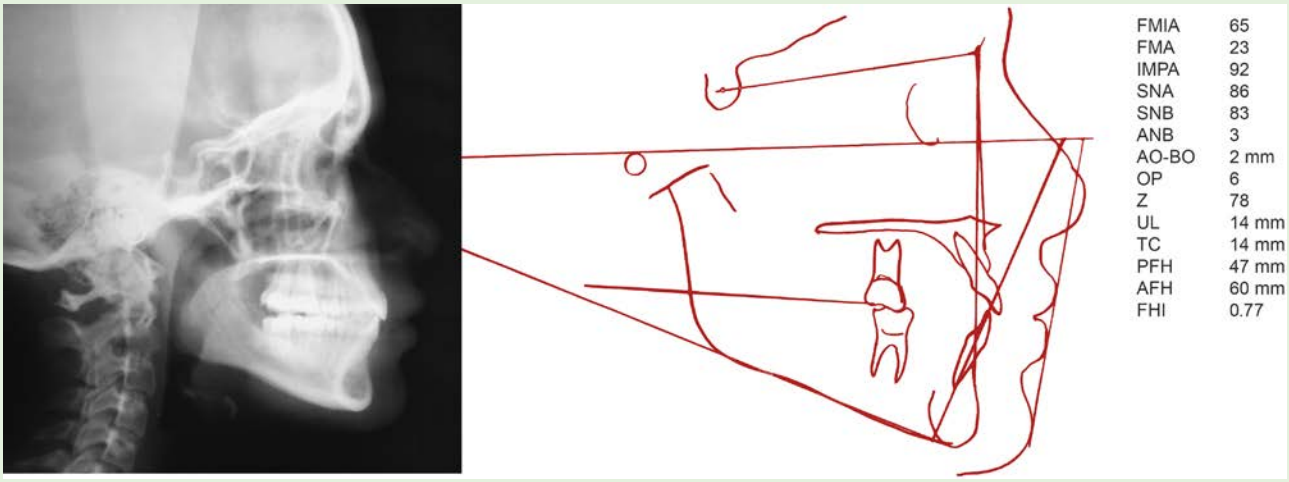


FIGURE 19-56 Posttreatment cephalogram and tracing. See Figure 19-13 for abbreviations.

CASE REPORTS: MALOCCLUSION CORRECTION WITH THE EDGEWISE APPLIANCE—cont'd

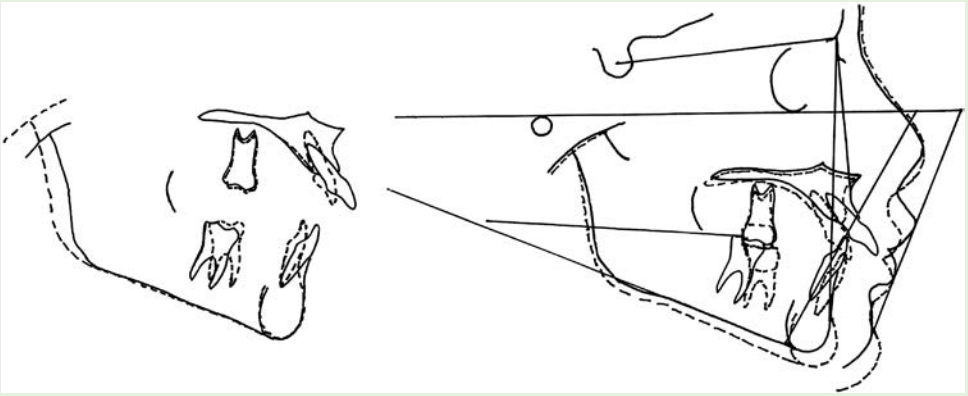


FIGURE 19-57 Pretreatment–posttreatment superimpositions.

REFERENCES

1. Angle EH. *The Treatment of Malocclusion of the Teeth and Fractures of the Maxillae*. 6th ed. Philadelphia: SS White; 1900.
2. Angle EH. *The Treatment of Malocclusion of the Teeth*. 7th ed. Philadelphia: SS White; 1907.
3. Tweed CH. Reports of cases treated with the edgewise arch mechanism. *Angle Orthod*. 1932;2:236.
4. Tweed CH. The application of the principles of the edgewise arch in the treatment of Class II, Division 1, part I. *Angle Orthod*. 1936;6(3):198–208.
5. Tweed CH. The application of the principles of the edgewise arch in the treatment of Class II, Division 1, part II. *Angle Orthod*. 1936;6(4):255–257.
6. Strang RH. Highlights of sixty-four years in orthodontics. *Angle Orthod*. 1974:44.
7. Tweed CH. A philosophy of orthodontic treatment. *Am J Orthod Oral Surg*. 1945;31:74.
8. Tweed CH. Indications for the extraction of teeth in orthodontic procedures. *Am J Orthod Oral Surg*. 1944;30:405.
9. Tweed CH. The Frankfort mandibular incisor angle (FMIA) in orthodontic diagnosis, treatment planning and prognosis. *Am J Orthod Oral Surg*. 1954;24:121.
10. Tweed CH. *Clinical Orthodontics*. Vols. 1 and 2. St. Louis: Mosby; 1966.
11. Merrifield LL. The dimensions of the denture: back to basics. *Am J Orthod Dentofac Orthop*. 1994;106:535.
12. Merrifield LL. The profile line as an aid in critically evaluating facial esthetics. *Am J Orthod*. 1966;11:804.
13. Merrifield LL. Differential diagnosis with total space analysis. *J Charles H Tweed Int Found*. 1978;6:10.
14. Merrifield LL. *Identification and Classification of Orthodontic and Orthognathic Disharmonies, unpublished lecture*. Rio de Janeiro: Brazilian Society of Orthodontics; November 20, 1997.
15. Merrifield LL, Cross JJ. Directional force. *Am J Orthod*. 1970;57:435.
16. Merrifield LL. The systems of directional force. *J Charles H Tweed Int Found*. 1982;10:15.
17. Merrifield LL. Differential diagnosis. *Semin Orthod*. 1996;2:241.
18. Merrifield LL, Klontz HA, Vaden JL. Differential diagnostic analysis systems. *Am J Orthod Dentofac Orthop*. 1994;106:641.
19. Bishara SE, Hession TJ, Peterson LC. Longitudinal soft tissue profile changes. *Am J Orthod*. 1985;88:209.
20. Burstone CJ. The integumental contour and extension patterns. *Angle Orthod*. 1950;29:93.
21. Burstone CJ. Lip posture and its significance in treatment planning. *Am J Orthod*. 1967;53:262.
22. Johnston L. Nothing personal. *Newsletter of the Great Lakes Association of Orthodontists*. 1997;33:3.
23. Horn A. Facial height index. *Am J Orthod Dentofac Orthop*. 1992;102(2):180.
24. Radziminski G. The control of horizontal planes in Class II treatment. *J Charles H Tweed Int Found*. 1987;15:125.
25. Gebeck TR, Merrifield LL. Orthodontic diagnosis and treatment analysis: concepts and values, part I. *Am J Orthod Dentofac Orthop*. 1995;107(4):434.
26. Merrifield LL, Gebeck TR. Analysis: concepts and values, part II. *J Charles H Tweed Int Found*. 1989;17:49.
27. Isaacson RJ. The geometry of facial growth and its effect on the dental occlusion and facial form. *J Charles H Tweed Int Found*. 1981;9:21.
28. Pearson LE. Vertical control in treatment of patients having backward rotational growth tendencies. *Angle Orthod*. 1978;43:132.
29. Pearson LE. Vertical control in fully-banded orthodontic treatment. *Angle Orthod*. 1986;56:205.
30. Schudy FF. Sound biological concepts in orthodontics. *Am J Orthod*. 1973;63:376.
31. Gebeck TR, Merrifield LL. Analysis: concepts and values, part I. *J Charles H Tweed Int Found*. 1989;17:19.
32. Merrifield LL, Gebeck TR. Orthodontic diagnosis and treatment analysis: concepts and values, part II. *Am J Orthod Dentofac Orthop*. 1995;107:541.
33. Gramling JF. A cephalometric appraisal of the results of orthodontic treatment on fifty-five unsuccessfully corrected difficult Class II malocclusions. *J Charles H Tweed Int Found*. 1987;15:112.
34. Gramling JF. A cephalometric appraisal of the results of orthodontic treatment on one hundred fifty successfully corrected difficult Class II malocclusions. *J Charles H Tweed Int Found*. 1987;15:102.
35. Gramling JF. The probability index. *J Charles H Tweed Int Found*. 1989;17:82.
36. Gramling JF. The probability index. *Am J Orthod Dentofac Orthop*. 1995;107:165.
37. Baldridge D. *Leveling the Curve of Spee: Its Effect on Mandibular Arch Length, unpublished master's thesis*. Memphis: University of Tennessee; June 1960.
38. Bjork A, Gensen E, Palling M. Mandibular growth and third molar impaction. *Eur Orthod Soc Trans*. 1956:164.
39. Ledyard BC. A study of the mandibular third molar area. *Am J Orthod*. 1953;39:366.
40. Richardson ME. Development of the lower third molar from ten to fifteen years. *Angle Orthod*. 1973;43:191.
41. Andrews LF. The six keys to normal occlusion. *Am J Orthod*. 1972;62(3):296.
42. Burstone CJ. Application of bioengineering to clinical orthodontics. In: Graber TM, Vanarsdall RL, eds. *Orthodontics: Current Principles and Techniques*. 2nd ed. St. Louis: Mosby; 1994.
43. Lindquist JT. The edgewise appliance. In: Graber TM, Vanarsdall RL, eds. *Orthodontics: Current Principles and Techniques*. 2nd ed. St. Louis: Mosby; 1994.

44. Roth RH. Treatment mechanics for the straight wire appliance. In: Graber TM, Swain B, eds. *Orthodontics: Current Principles and Techniques*. 1st ed. St. Louis: Mosby; 1985.
45. Klontz HA. Tweed-Merrifield sequential directional force treatment. *Semin Orthod*. 1986;2:254.
46. Merrifield LL. Edgewise sequential directional force technology. *J Charles H Tweed Int Found*. 1986;14:22.
47. Vaden JL, Klontz HK, Dale JG. The Tweed Merrifield edgewise appliance. In: Graber TM, Vanarsdall RL, eds. *Orthodontics: Current Principles and Techniques*. 3rd ed. St. Louis: Mosby; 2000.
48. Klontz HA. *Readout. J Charles H Tweed Int Found*. 1985;13:53.

20

Biomechanical Considerations with Temporary Anchorage Devices

Jong Suk Lee, Jung Kook Kim, and Young-Chel Park

OUTLINE

Characteristics and Clinical Significance of Temporary Anchorage Devices, 511
Characteristics of Temporary Anchorage Device Mechanics, 511
Rigid Anchorage, 511
Intrusive Mechanics, 511
High-Efficiency Mechanics, 511
Clinical Significance of Temporary Anchorage Device Mechanics, 513
Easy and Simple Anchorage Preparation, 513
Increased Treatment Efficiency, 513
Expansion of the Range of Orthodontic Mechanotherapy, 515
Shifting from a Mechanics-Centered Approach, 516
Considerations for Temporary Anchorage Device Mechanics, 516
Temporary Anchorage Device Stability, 516
Temporary Anchorage Device Positioning, 516
Load-Bearing Capacity of the Temporary Anchorage Device, 517
Biomechanical Considerations, 517
Biological Considerations, 517
Side Effects Related to Temporary Anchorage Device Mechanics and Their Management, 519

Clinical and Biomechanical Applications of Temporary Anchorage Devices, 519
General Principles in Biomechanical Application of Temporary Anchorage Device Mechanics, 519
Establishment of an Individualized and Optimal Treatment Plan, 519
Selection of a Temporary Anchorage Device System and Insertion Site, 519
Treatment Strategy, 522
Mechanotherapy Design, 522
Molar Intrusion, 524
Decision Making, 524
Biomechanics, 525
Treatment Mechanics, 526
Molar Distalization, 528
Decision Making, 528
Biomechanics, 528
Molar Protraction, 528
Decision Making, 528
Biomechanics, 541
Anterior Retraction in Extraction Treatment, 541
Decision Making, 541
Biomechanics, 541

CHARACTERISTICS AND CLINICAL SIGNIFICANCE OF TEMPORARY ANCHORAGE DEVICES

Mechanics using temporary anchorage devices (TADs) follow general biomechanical principles; however, several characteristic features are specific to TAD mechanics when compared with conventional mechanics. These features not only simplify treatment with conventional techniques and increase efficiency, but they also make treatment of seemingly impossible cases with challenging anchorage preparation feasible. A good understanding of TAD mechanics with the proper biomechanical treatment principles can minimize side effects while maximizing the efficiency of TAD mechanics.

Characteristics of Temporary Anchorage Device Mechanics

Characteristics of TAD mechanics can be divided into three categories: mechanics using rigid anchorage, intrusive mechanics, and high-efficiency mechanics.

Rigid Anchorage

When the TAD achieves bony support via a stable osseous interface, immobile rigid anchorage to the orthodontic load can be supplied within physiologic thresholds.[1,2] This means that the use of a TAD can easily secure rigid anchorage without any additional preparation of the dentition while relieving the limitations of anchorage found with conventional orthodontic mechanotherapy.

Intrusive Mechanics

Conventional mechanics essentially consist of characteristics of extrusive mechanics.[3] Conversely, the TAD is generally located apical to the brackets, and, in this location, TAD mechanics are advantageous in achieving intrusive mechanics (Fig. 20-1).[4–6]

High-Efficiency Mechanics

TAD mechanics generally use a single force without moments, which is very efficient for tooth movement[6] (see Fig. 20-1). The line of action, point of application, and direction of

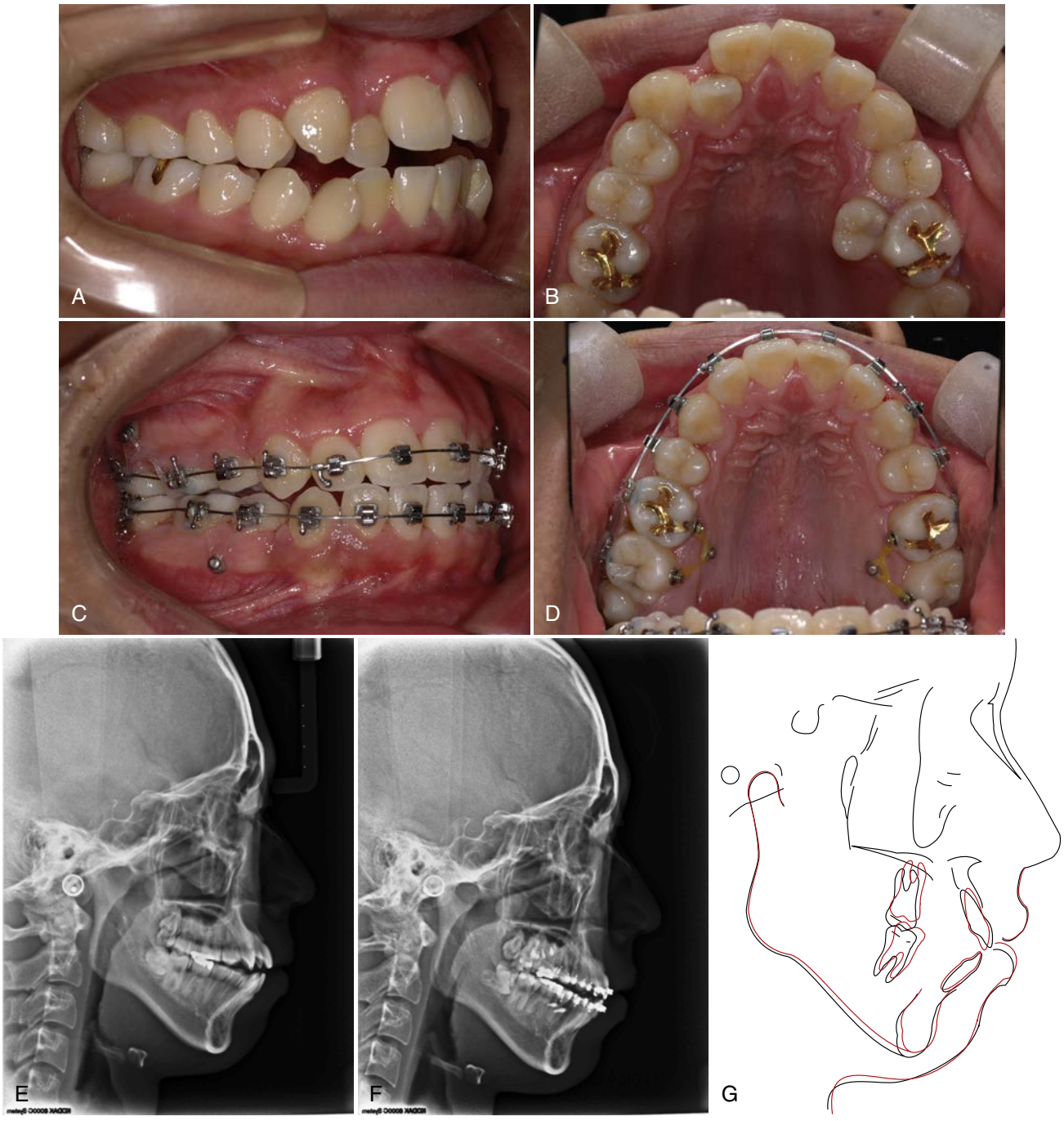


FIGURE 20-1 Buccal and palatal temporary anchorage devices (TADs) were placed, and two single continuous forces without moments were applied for efficient molar intrusion. Cephalometric superimposition shows intruded upper molars and autorotation of the mandible. **A, B,** Intraoral views before treatment. **C, D,** Intraoral views after 3 months of maxillary molar intrusion. **E,** Cephalometric radiograph before treatment. **F,** Cephalometric radiograph after 3 months of molar intrusion. **G,** Cephalometric superimposition.

FIGURE 20-2 The mesially angulated second molar was uprighted and protracted to the first molar position with a temporary anchorage device (TAD) and bonding of sectional orthodontic attachments. The third molar was guided to erupt into the second molar position. **A,** Intraoral view before treatment. **B,** A single TAD was placed between the canine and the premolar and splinted to the first premolar. The first and second premolars and the second molar were bonded and common tied to prevent distal uprighting of the second molar. **C,** The second molar was uprighted and mesially protracted to the first molar position by root movement. **D,** The third molar was guided into the second molar position. **E,** Panoramic radiograph before treatment visualizes the residual roots of the first molar (*black arrow*). **F,** Panoramic radiograph after treatment.

force can be designed for efficient tooth movement by controlling the location of TAD placement.

Clinical Significance of Temporary Anchorage Device Mechanics

The characteristics of TAD mechanics contribute to the following four features of clinical treatment (Case Studies 20-1 through 20-4).

Easy and Simple Anchorage Preparation

Orthodontic anchorage can be easily prepared using TADs, regardless of the condition of the dentition. Treatment mechanics can also become simpler than conventional mechanics. For example, designing mechanics for asymmetric tooth movement is comparatively convenient because the teeth are not providing the anchorage.[7–10] Furthermore, adjunctive treatment of tooth loss or impacted teeth can be addressed with the use of TADs and sectional orthodontic attachments (Figs. 20-2 and 20-3).

Increased Treatment Efficiency

As a result of the rigid anchorage supplied by the TAD, orthodontic treatment can be more easily and efficiently conducted.[11–21] Moreover, mechanotherapy can be designed using a treatment objective–centered approach, as opposed to a mechanics-centered approach, which frees orthodontic mechanotherapy from the biomechanical limitations of anchorage.

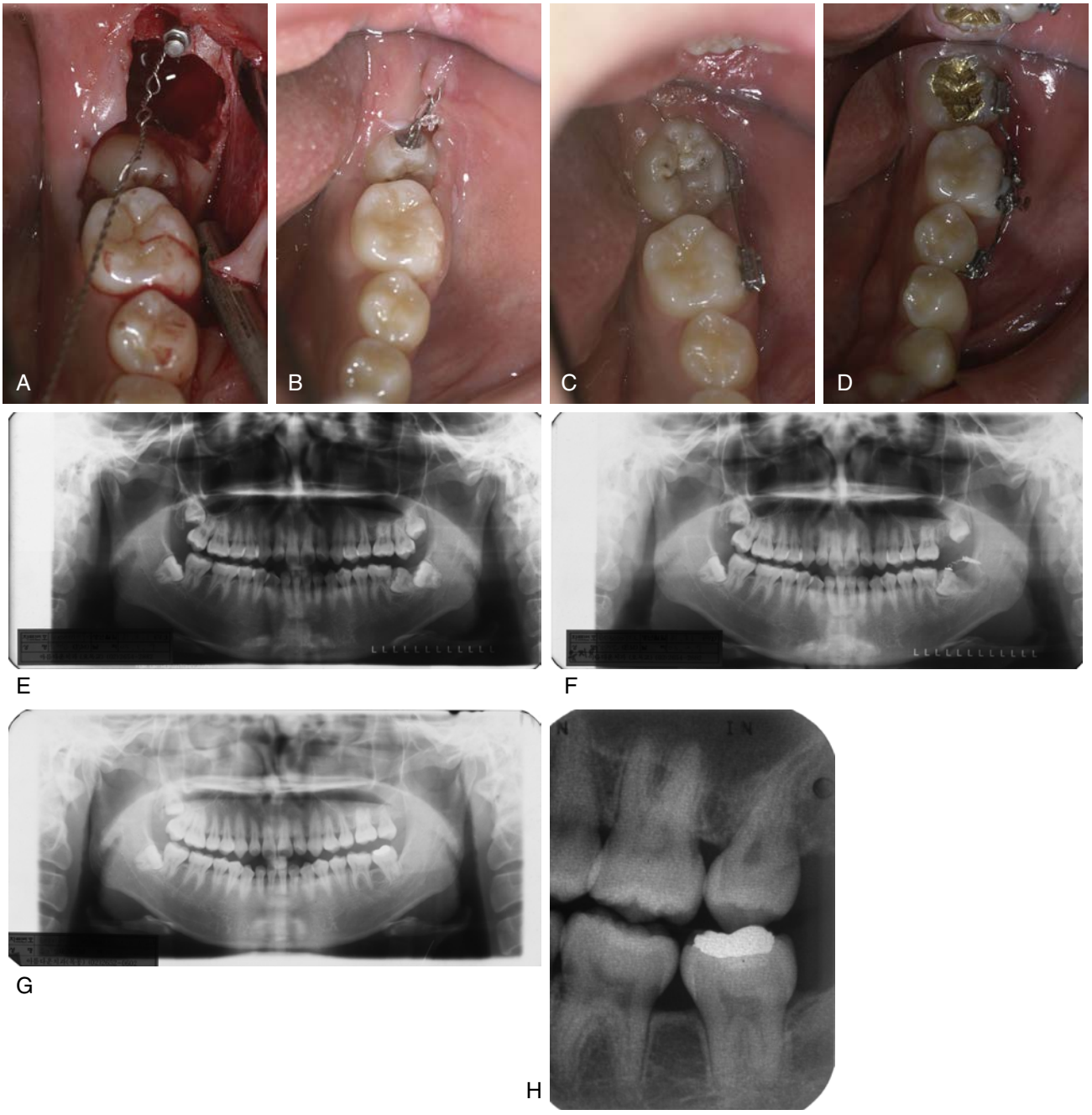


FIGURE 20-3 A 22-year-old female patient had an impacted second molar blocked by the third molar. When the third molar was extracted, a temporary anchorage device (TAD) was simultaneously placed in the retromolar area. The maxillary second molar was extracted as a result of supereruption, and guided eruption of the third molar into the second molar position was planned. The mandibular second molar was guided to erupt to the proper position, and a tube was bonded on the second molar to level and align. The alveolar bone level of this second molar was in good condition. **A,** Intraoral view shows the extraction of the third molar and TAD placement. **B**, Intraoral view at the start of molar uprighting. **C,** Attachments were bonded on the first and second molars for leveling. **D**, Intraoral view after molar uprighting. **E,** Panoramic radiograph before treatment. **F,** Panoramic radiograph while the molar was uprighted with the TAD. **G,** Panoramic radiograph at the completion of treatment. **H,** Bitewing radiograph shows a healthy bone level between the molars.

Molar distalization becomes simpler and more predictable, even in adult patients, and can be a very useful option for the treatment of cases with moderate crowding or for camouflage treatment of anteroposterior skeletal discrepancies.[22–30] Furthermore, all of the anterior and posterior teeth can be moved at the same time using rigid anchorage (Fig. 20-4).

Expansion of the Range of Orthodontic Mechanotherapy

TAD mechanics can expand the range of orthodontic mechanotherapy. One of the most significant changes is the potential for intrusion of the posterior teeth.[6,8–10,31–46] By intrusion of the entire dentition or intrusion of the posterior teeth, nonsurgical orthodontic mechanotherapy can change the occlusal plane and the position of the chin point, similar to that seen

FIGURE 20-4 A 22-year-old male patient's chief complaint is protrusive lips and a retrusive chin. After premolar extractions, anteroposterior and vertical disharmonies were improved by anterior retraction and molar intrusion. The chin position was also altered through molar intrusion and subsequent autorotation of the mandible. The duration of active treatment was 31 months. **A,** Frontal facial view before treatment. **B,** Lateral facial view before treatment. **C,** Intraoral view before treatment. **D,** Frontal facial view at the completion of treatment. **E,** Lateral facial view at the completion of treatment. **F,** Intraoral view at the completion of active treatment. Overcorrection of molar intrusion was performed. **G–I,** Intraoral views during active treatment. **J,** Panoramic radiograph before treatment. **K,** Panoramic radiograph at the completion of treatment.

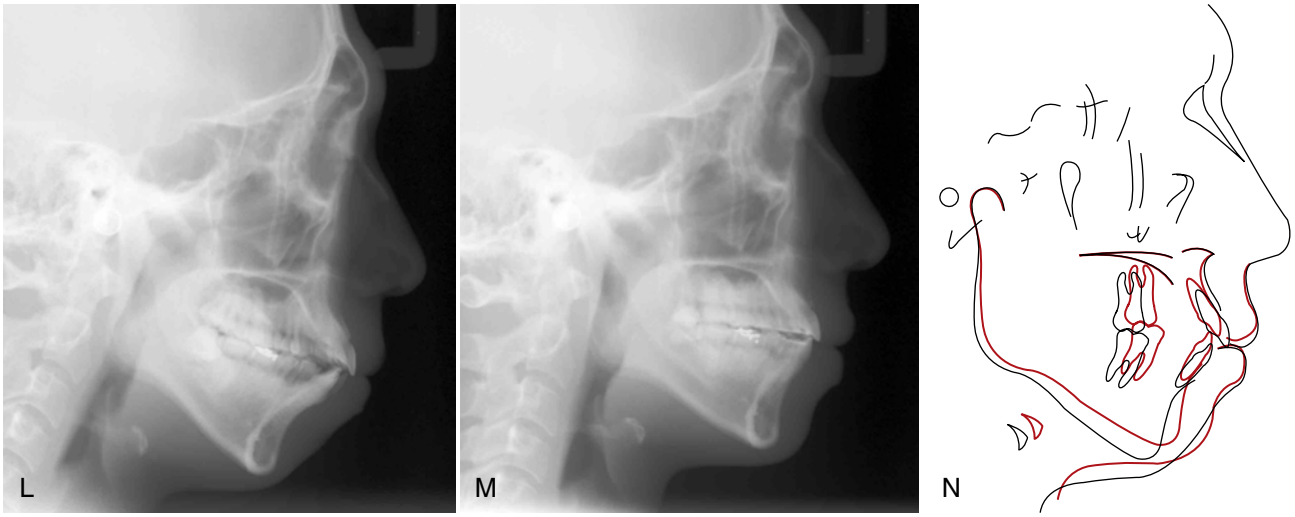


FIGURE 20-4, cont'd L, Cephalometric radiograph before treatment. M, Cephalometric radiograph at the completion of treatment. N, Superimposition of pretreatment and posttreatment cephalometric radiographs.

by surgical repositioning of the maxilla (see Fig. 20-4).* The stability of molar intrusion with TADs is clinically acceptable if proper treatment protocols are followed.[6,32,38,39]

Shifting from a Mechanics-Centered Approach

Although anchorage preparation has become easier and simpler with TADs and the range of orthodontic therapy has broadened, with this has come the need to consider several additional factors for treatment. To put it more simply, deciding between extraction and nonextraction treatment has been the key with conventional mechanics in the past. However, further considerations, such as whether to intrude teeth, have become necessary with TAD mechanics. Furthermore, with the shift from two-dimensional tooth movements to three-dimensional tooth movements, the sheer amount of information that the orthodontist must process has tremendously increased. The *mechanics* portion of biomechanics has been the primary limiting factor in anchorage control in conventional treatment, whereas the *bio* portion has become the primary limiting factor in treatment using TAD mechanics.

The importance of establishing a diagnosis and treatment plan based on an understanding and reflection of the physiologic functions of the stomatognathic system, including the temporomandibular joint (TMJ) and masticatory muscles, is growing ever more important.[47,48] For greater amounts of tooth movement, a thorough understanding of the biologic side effects, such as root resorption or attachment loss, has gained importance as well.[49–52] Extra precautions need to be taken with regard to understanding and considering the functional aspects of more extensive tooth movements to prevent and manage biologic problems properly. Placing the TAD in the exact desired position with minimal side effects and maximum stability requires as much skill of the clinician as precise bracket positioning.[53,54] Accurate placement, referral of TAD placement, and all related patient communications are also issues that the clinician needs to weigh before treatment.

Considerations for Temporary Anchorage Device Mechanics

TAD mechanics are useful in solving mechanical problems, but they have restrictions when addressing the biological limitations encountered with mechanotherapy.

Temporary Anchorage Device Stability

TAD mechanics is entirely based on the stability of the TAD. The success rate of TADs is greater than 80%, which is clinically acceptable.[55–64] Loosening of a TAD is not clinically uncommon.[65] The more favorable alternative when loosening occurs is to modify the location of TAD placement. However, if the location of the TAD cannot be compromised, a 3- to 6-month waiting period is essential for cortical bone formation before replacing the TAD in the same location.[66–68] In cases of repeated failure, an alteration of the treatment plan may be required. The patient should be thoroughly informed in detail about such changes to the treatment plan before treatment with the TAD is initiated.

Temporary Anchorage Device Positioning

Selecting a TAD position is extremely important to the design of TAD mechanics. In some instances, TADs cannot be placed in the desired position because of limitations of the anatomic structures and accessibility.[69–74] Considering the fact that substantial individual variations exist among patients, the clinician must perform single measurements in specific anatomic sites for TAD placement to prevent potential damage to anatomic structures, to minimize the risk of failure, and to obtain the most favorable clinical results.[69,71,74] Cone-beam computed tomography (CBCT) imaging is very useful in helping achieve these goals.[64]

Moreover, TADs placement in interradicular areas may restrict tooth movement of adjacent teeth because of the lack

* References 6, 21, 31–34, 36, 38–40, 42–46.

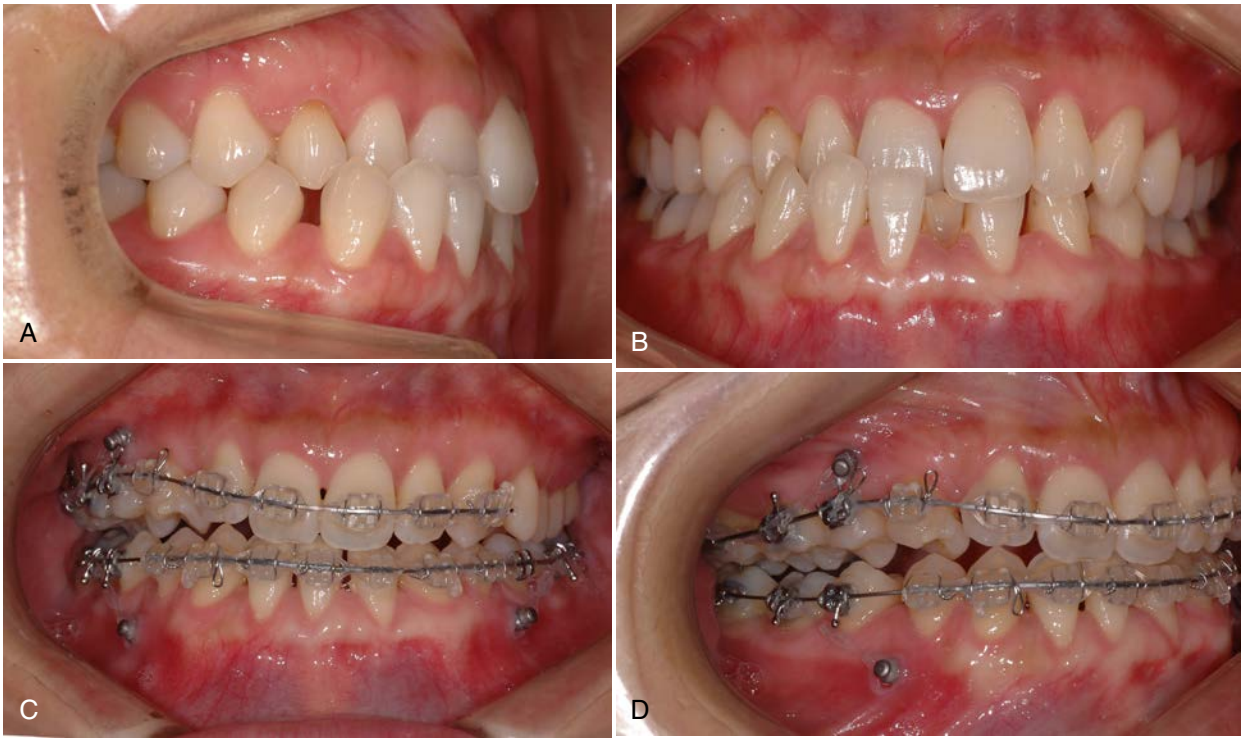


FIGURE 20-5 Orthodontic force was applied to protract the molar unilaterally from the temporary anchorage devices (TAD) to close the space of the upper right deciduous canine. However, an open bite developed as a result of mesial tipping of the molar, as well as the intrusive force vector in the premolar area. Using lever-arm mechanics, intermaxillary elastics can prevent such side effects and can solve similar problems. **A, B,** Intraoral views before treatment. **C, D,** Intraoral views during treatment. Occlusal canting developed.

of space between the roots.[70–74] The mechanics design can compensate for such restrictions.[75,76]

Load-Bearing Capacity of the Temporary Anchorage Device

The orthodontic load-bearing capacity is closely related to the size and biocompatibility (i.e., bonding strength at the implant-bone interface) of the TAD.[77–79] According to a finite-element model (FEM) analysis study, a miniscrew-type TAD made of titanium alloy can withstand approximately 200 to 400 g of orthodontic force, depending on the bone condition and diameter of the TAD.[6] However, splinting two TADs or placing extra TADs can allow for heavier forces to be applied.[80,81] The use of wider and longer TADs may also be helpful.[6,82–84]

Biomechanical Considerations

The TAD itself can provide favorable orthodontic anchorage but cannot offer an ideal force system for all types of tooth movements. Rigid anchorage is only one of the contributing factors to ideal treatment. However, rigid anchorage alone does not guarantee successful tooth movement; anchorage loss and unwanted side effects can result, even with TAD mechanics. For example, the intrusive force vector of TAD mechanics can produce unforeseen side effects with conventional mechanics (Figs. 20-5 and 20-6). These consequences are very difficult to correct. Therefore removing or controlling unwanted force vectors from the TAD is necessary in all three dimensions of space.

Biological Considerations

TAD mechanics move teeth using the same principles as conventional mechanotherapy and must be used with consideration paid to its biological limitations. Tooth movement should take place within the periodontal complex,[85–89] as with all mechanotherapy.

The ability of TADs to address vertical discrepancies nonsurgically has resulted in the need for greater precautions for biological considerations. For example, molar intrusion using TADs can affect treatment progress and maintenance of a decrease in the overall vertical dimension. When protracting molars into spaces left by missing or extracted teeth, especially in the mandible, more attention to the periodontal management of the molars being protracted, including attachment loss, is necessary.[90,91] Although TADs have resolved many of the problems concerning the *mechanics* portion of biomechanics, the importance of managing problems related to the *bio* aspect have become greater.

Although research and clinical trials have been reported,[92–108] a clear protocol of the orthopedic effects to the basal bone using TAD mechanics is not yet established. Further studies are needed to clarify the orthopedic effects of TAD mechanics. With regard to transverse orthopedic expansion, several studies examining the expansion of the lateral envelope of movement of the posterior teeth have been conducted,* but no definite conclusions have been established.

* References 94, 99, 100, 103–106, 108.

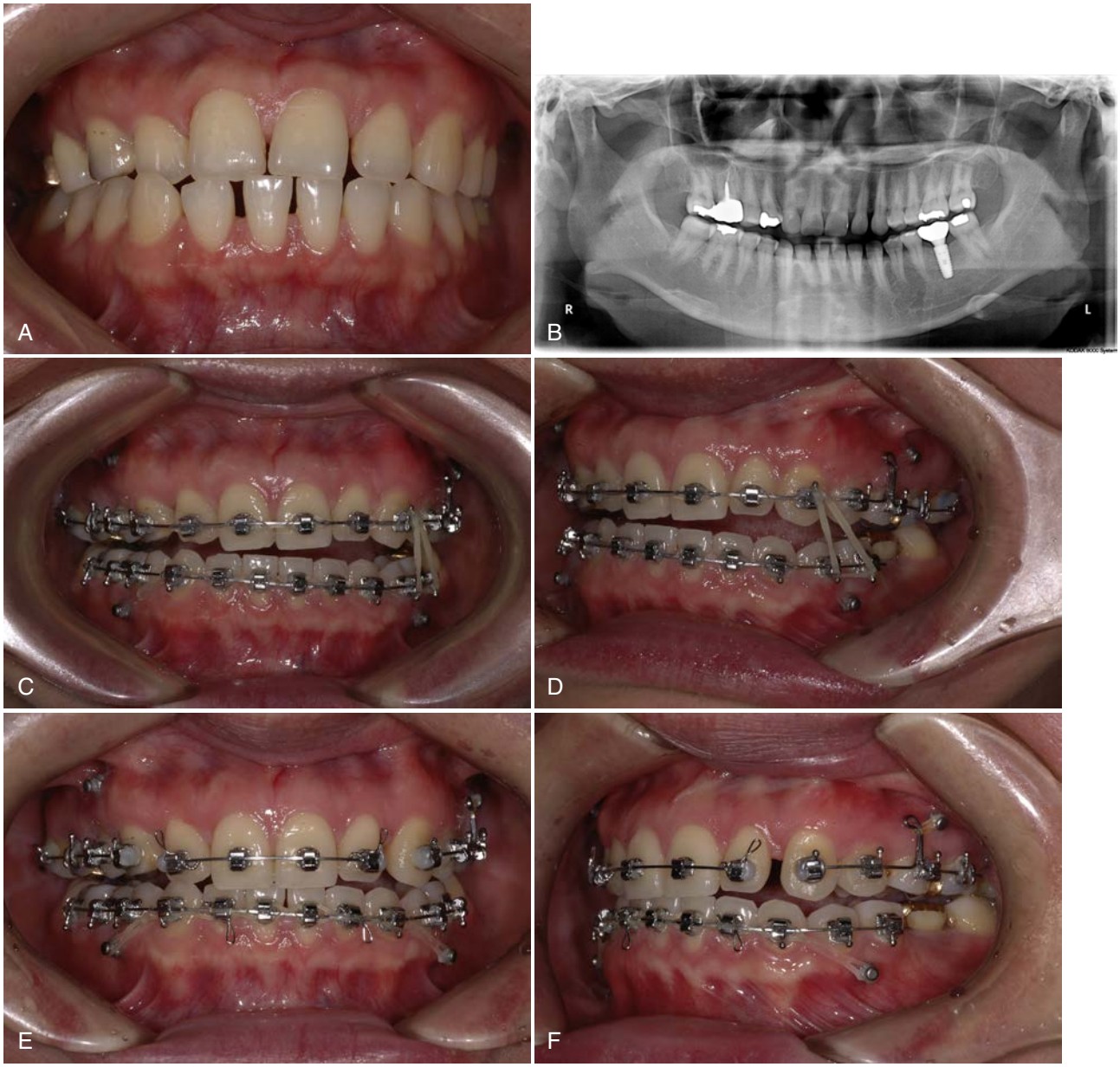


FIGURE 20-6 Bilateral, symmetric retraction, and intrusive forces were applied, but occlusal canting developed as a result of the different anchorage values on either side—that is, more intrusion developed in the left anterior area because the left first molar prosthodontic implant was not included in the full bonding. Using intermaxillary elastics, canting was improved. Conventional extrusive mechanics are useful to compensate for the disadvantages of the intrusive components of temporary anchorage device (TAD) mechanics. **A,** Intraoral views before treatment. **B,** Panoramic radiograph before treatment. **C, D,** Intraoral views during treatment. Occlusal canting developed. **E, F,** Intraoral views during the canting correction.

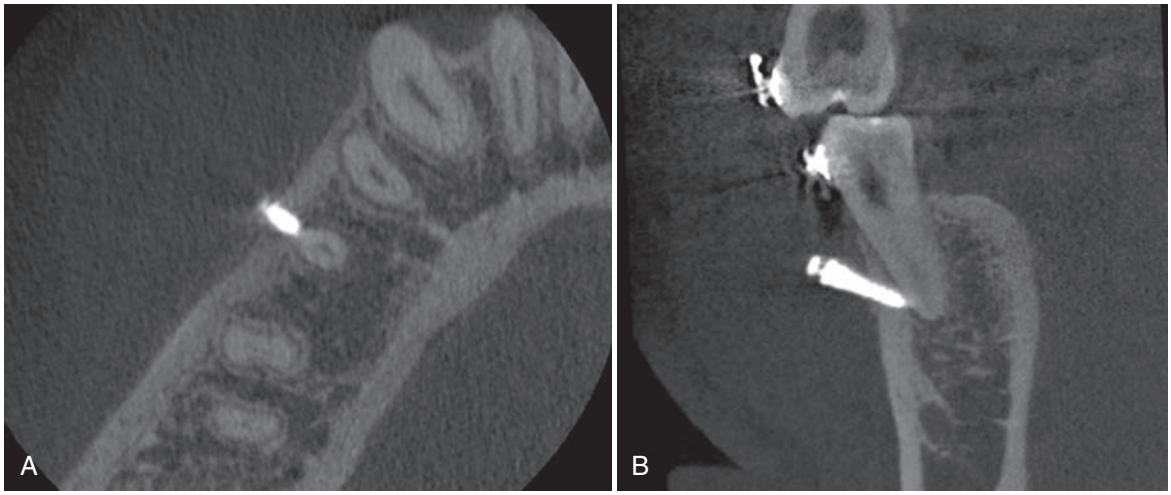


FIGURE 20-7 The most important thing to remember in root injury prevention is that root injuries can and do occur. If even a hint of doubt is present, then the entire procedure should be double and triple checked. During the insertion process, if the temporary anchorage device (TAD) makes contact with the root, then the patient will likely experience an abrupt pain. If the patient suddenly reports pain, then the insertion procedure should be stopped and the location of the implant reconfirmed. **A,** The best way to check the location is with three-dimensional cone-beam computed tomography (CBCT) imaging. Complaints of pain from patients should not be regarded lightly, as they have the potential for becoming sources of legal conflicts in the future. **B,** Contact of the TAD tip with the apex of the root was confirmed, and the location of TAD placement was modified on the basis of the examination results of three-dimensional CBCT imaging.

Side Effects Related to Temporary Anchorage Device Mechanics and Their Management

Unexpected iatrogenic side effects such as root injuries and penetration into the nasal cavity or maxillary sinus may occur during the surgical placement of the TAD.[109–111] Root injuries are reversible in many cases[112–120]; however, a crack to a root or root fracture is considered irreversible.[6,121,122] Remembering that root injuries are always possible is of the utmost importance in preventing iatrogenic injury. In addition, if an injury is suspected, then the clinician should halt the procedure and carefully double check the entire placement process (Fig. 20-7). Adherence to proper surgical protocols can prevent iatrogenic injuries to anatomic structures.[6,123]

Root injuries that occur near the apex may not require any particular treatment (Fig. 20-8). Furthermore, root injury without attachment loss can be managed to some extent with endodontic treatment or apical surgery (Fig. 20-9).[124–128] If the maxillary sinus is healthy, then simple penetration to the sinus under proper aseptic principles does not pose a significant problem (Fig. 20-10).[110]

TAD mechanics can expand the envelope of discrepancies of tooth movement but may also contribute some negative aspects.[50,51,87] Side effects that are not common to conventional mechanics, which are related to intrusive mechanics, can develop. Additionally, TAD mechanics may worsen the conventional side effects of orthodontic treatment. Root resorption and periodontal problems, in particular, may occur as a result of large amounts of tooth movement using rigid anchorage. Side effects resulting from misdiagnosis and overtreatment should also be avoided.

TAD mechanics can increase the expectation level of patients and may cause further dissatisfaction in a subjective patient. For satisfactory and successful treatment, the operator should engage in thorough communications with the patient regarding the effectiveness and limitations of the TAD.

CLINICAL AND BIOMECHANICAL APPLICATIONS OF TEMPORARY ANCHORAGE DEVICES

General Principles in Biomechanical Application of Temporary Anchorage Device Mechanics

Establishment of an Individualized and Optimal Treatment Plan

An individualized treatment plan should be determined by collecting an adequate database of information with regard to the patient and by interviewing the patient and any persons concerned.[129] Cost-benefit analyses should also be considered when deciding treatment options.

Selection of a Temporary Anchorage Device System and Insertion Site

Various TAD systems are available on the dental market,[130,131] as well as numerous reported clinical applications. A specific TAD system and insertion site should be selected according to the individual treatment plan.

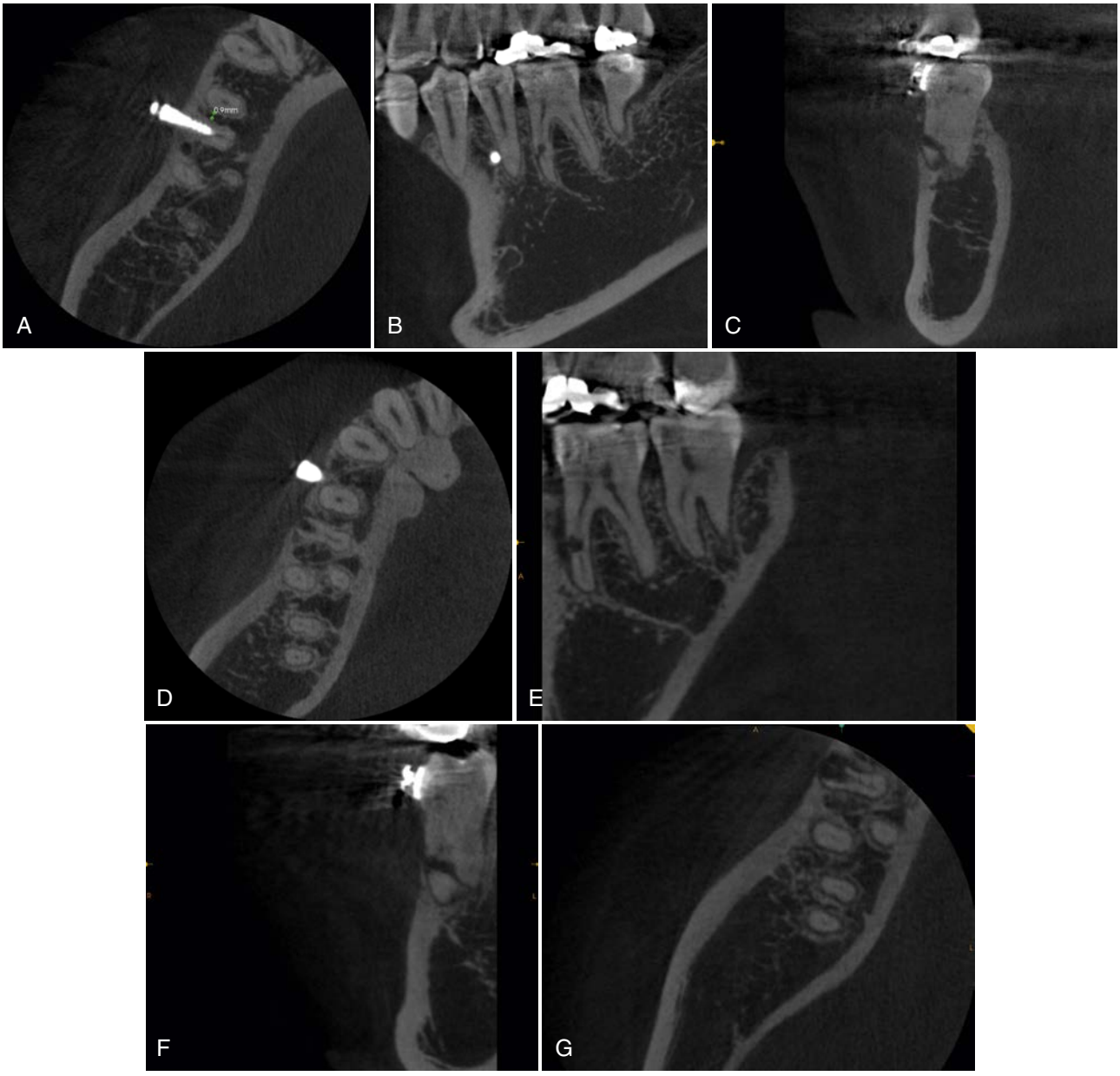


FIGURE 20-8 Root injuries in the vicinity of the apex can be addressed by ensuring that the affected tooth is not subject to secondary trauma, which can be accomplished with occlusal adjustments. If properly handled, then root injury near the apex will not cause any particular problems, even without the aid of root canal treatment. **A,** During temporary anchorage device (TAD) insertion, a distinct breaking sound was heard. Confirmation with three-dimensional cone-beam computed tomography (CBCT) imaging revealed that the TAD had been placed in the mesial root of the first molar. **B–D,** Antibiotics were prescribed for 5 days after the removal of the TAD. At the 1-week check-up appointment, no additional treatment, such as root canal, was administered because no symptoms were present. After occlusal adjustment and a 3-month observation period, orthodontic treatment was continued. **E–G,** After the completion of treatment, the patient was monitored for 2 years. The affected tooth remained vital, and no particular pathologic signs developed.

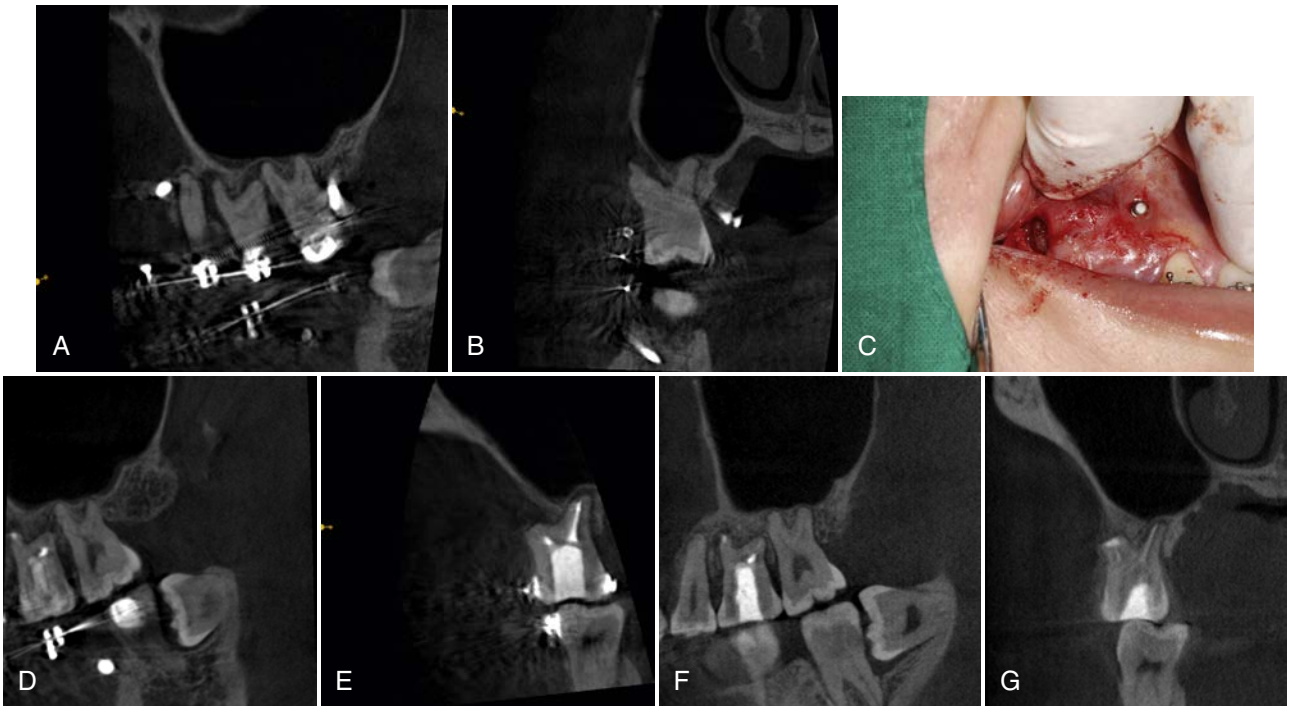


FIGURE 20-9 Injuries to the root or adjacent periodontal tissues can be efficiently managed using root canal treatment or apicoectomy procedures, depending on the extent of damage and location. **A, B,** An examination was performed with three-dimensional cone-beam computed tomography (CBCT) imaging when continuous pus discharge was observed after temporary anchorage device (TAD) removal. Root injury and consequent surrounding bone loss were observed; furcation involvement was also noted. **C,** When the operative field for apical surgery was secured, a hole was observed in the distal root, presumably attributable to the TAD. **D, E,** Three-dimensional CBCT image 1 month after root resection. Preventing secondary trauma with occlusal adjustment is very important for favorable healing. **F, G,** Normal lamina dura was observed in radiographs 12 months after the root resection was performed. No abnormal signs or symptoms were present.

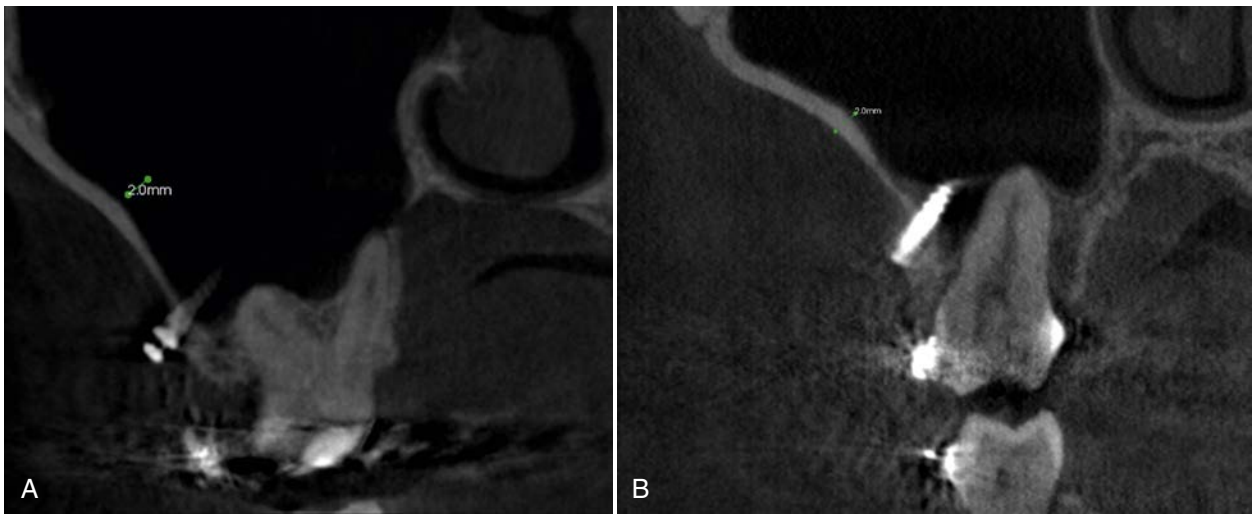


FIGURE 20-10 Even temporary anchorage devices (TADs) placed in thin bone can provide orthodontic anchorage that can withstand approximately 150 g of orthodontic force. **A,** No feeling of resistance was detected while placing the TAD; therefore a radiographic examination with three-dimensional cone-beam computed tomography (CBCT) was performed. A computed tomography (CT) image, taken just after placement, shows that the thickness of the supporting cortical bone is 0.7 mm, which is comparatively thin. The image also shows that the TAD has penetrated the maxillary sinus. **B,** A CT image taken 14 months after TAD placement and application of orthodontic force shows no particular signs of inflammation of the maxillary sinus. No new bone formation was observed. Approximately 150 g of orthodontic force was applied using a nickel-titanium (NiTi) coil spring, and no mobility or movement of the TAD was observed.

Anatomic factors. Proper cortical bone thickness is a key factor in gaining sufficient primary stability (mechanical stabilization from cortical bone immediately after implantation), and thus adequate cortical bone is required for early stability and favorable healing.[132–134] However, even if the TAD is placed in thinner cortical bone, a certain amount of primary stability can be obtained, and this stability is capable of withstanding approximately 150 g of orthodontic force (see Fig. 20-10).[64,135] Sufficient support from cortical bone is important in gaining stability; however, primary stability itself does not entirely account for the success of the TAD, although it is an important contributing factor. The management of forces attributable to function or parafunction, which are significantly stronger and more continuous than orthodontic forces, is also important.

Edentulous areas may have low bone quality, sometimes attributable to atrophy. In these areas, bone probing is necessary after anesthesia to check the quality of cortical bone.

Although not always necessary for TAD maintenance,[56,61,136] attached gingiva is more favorable, compared with the oral mucosa. However, stability may be compromised if the TAD is irritated by the oral mucosa and can lead to unfavorable conditions.

TAD placement in areas where significant stress is applied should be avoided when possible. For example, stability of a TAD near the mandibular first molar can be compromised[55,60] as a result of masticatory stress. Good accessibility during surgical placement is advantageous in achieving primary stability. The risk of irreversible injury to important anatomic structures should be minimized. Furthermore, the TAD, itself, should not be an obstacle for planned tooth movements.

Biomechanical factors. The TAD should be placed in a biomechanically suitable position for planned tooth movements. Moreover, the TAD position must be predominantly favorable for the primary target tooth.

Clinical factor. Pain and discomfort during or after surgical placement of TADs are clinically acceptable.[137–140] Furthermore, the TAD should be placed in areas that result in minimal discomfort for the patient during treatment.

Treatment Strategy

First, to achieve treatment objectives efficiently, a strategy should be planned (Fig. 20-11) according to the TAD type to compensate for any respective disadvantages. The priority of tooth movement should be decided before instituting a plan to move the target tooth. In other words, the teeth to be moved and the establishment of an anchorage unit at each stage of treatment should be identified before movement begins.

Mechanotherapy Design

To obtain the desired tooth movement according to the treatment strategy, mechanics with an optimized orthodontic force system should be designed (Fig. 20-12). During this process, two things need to be considered: (1) how to produce tooth movement and (2) how to control this movement.

With regard to producing tooth movement, the operator needs to determine what kind of orthodontic force system will be used. The force system of the mechanics at the start of treatment and any changes to this force system that come about when the tooth is moving are important and are related to mechanical efficiency and to the speed of tooth movement. This determination is especially imperative in difficult types of tooth movements.

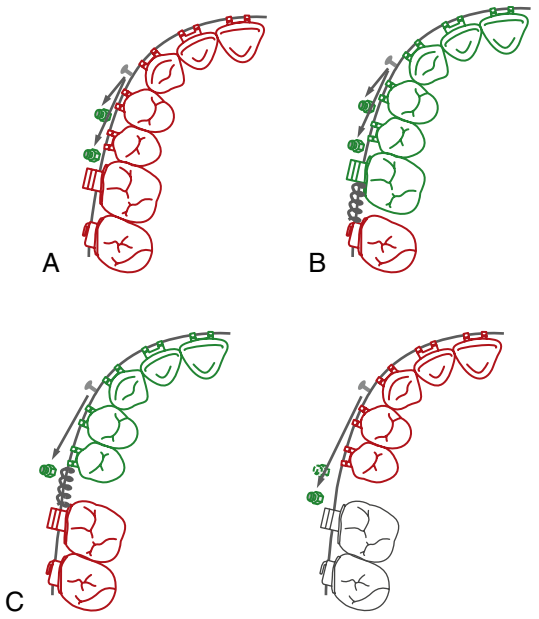


FIGURE 20-11 There are two methods for molar distalization: the entire dentition can be distalized **(A)** or the second molars can be distally moved first with the rest of the dentition following **(B, C)**. When the entire dentition is distalized, temporary anchorage devices (TADs) alone provide anchorage for movement of all the teeth. On the other hand, when only the molars are distally moved first, the rest of the dentition and the TADs both provide anchorage. Treatment mechanics may be simpler and the treatment time shorter when distalizing the entire dentition at once. However, treatment predictability also lessens as more teeth are involved. Conversely, when separately distalizing the molars, tooth movement is more predictable because of the increased anchorage and the movement of fewer teeth. Furthermore, distalization of more than one-half a cusp width can be gained when a second TAD is placed after the first segment has been distalized with the first TAD **(C)**. This method aids in overcoming the restricted interradicular space found between the first and second molars. (*Green* indicates an anchorage unit; *red* indicates the unit to be moved.)

The operator should also decide how to control the teeth three-dimensionally during treatment. Unwanted movements can occur as treatment progresses, even in an ideally designed force system. For successful application of TAD mechanics, proper monitoring and three-dimensional adjustments should be made on tooth movement (Case Study 20-1). There are several different ways to make such modifications: the use of a single force with or without an additional TAD, the use of brackets and wires, and the combination of both.[6]

More specifically, three types of mechanics can be used (Fig. 20-13): force-driven mechanics, which use a single force; shape-driven mechanics, which use the shapes of the archwires engaged in the brackets; and a combination of the former two types of mechanics.[141]

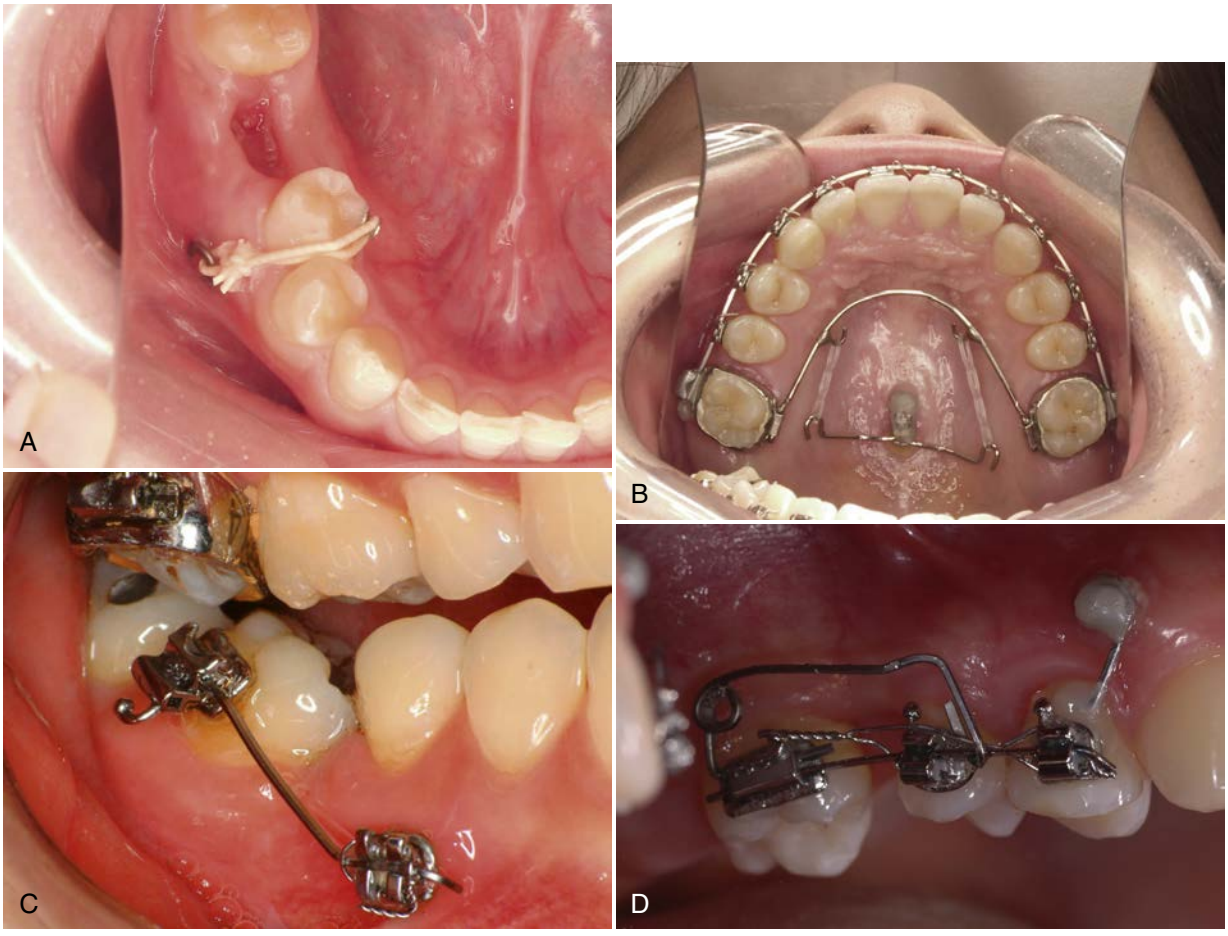


FIGURE 20-12 Diverse methods using temporary anchorage devices (TADs). **A,** *Direct application of a single force.* When using a single force, precise calibration is possible. Moreover, the whole force system does not significantly change even as the tooth is moved. To control the tooth movement, the line of action should be adjusted. **B,** *Indirect application of a single force.* Using attachments on TADs, the line of action can be controlled. **C,** *Direct application of a force and moment.* If wires can be engaged to TADs, TADs can produce not only a force but also a moment. When the wire attached to the TAD is engaged into the bracket slot on the opposite side, it becomes a statically indeterminate force system, and this force system cannot be precisely predicted. When the tooth is moved, the total force system will be altered as well. Additionally, complex use of a TAD can negatively affect its stability. **D,** *Indirect application of a force and moment.* The combination of a TAD and tooth can be considered a total anchor unit. It can provide three-dimensional anchorage, but little movement of the anchorage unit occurs. When using this unit for an indirect application, the operator has to consider that the tooth, which has a periodontal ligament (PDL), can be moved, but the stable TAD with an osseous interface cannot be moved. This means that if the TAD is splinted with a wire of higher stiffness to a tooth receiving heavier occlusal forces, a detrimental effect to the stability of the TAD will occur.

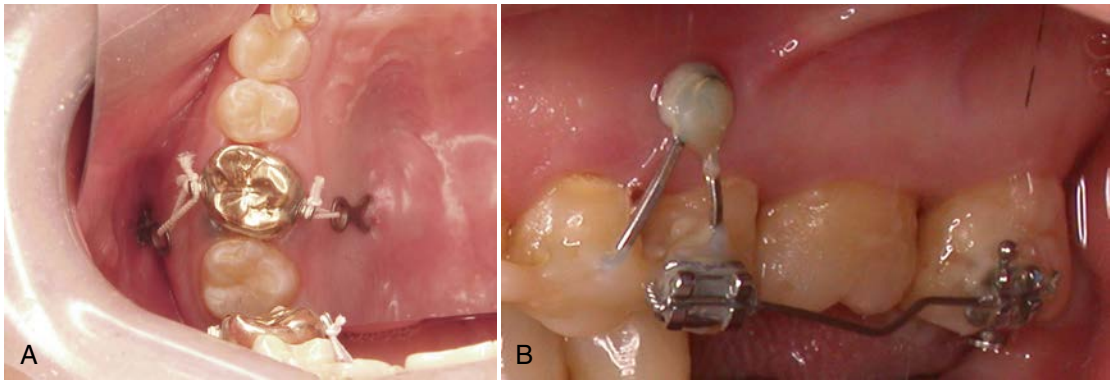


FIGURE 20-13 Molar intrusion can be achieved by force-driven mechanics **(A)** or shape-driven mechanics **(B)**. **A,** Force-driven mechanics use only single forces without moments. **B,** Shape-driven mechanics use continuous archwires, which are engaged into the brackets. As a consequence, forces and moments are produced but cannot be calculated chairside.

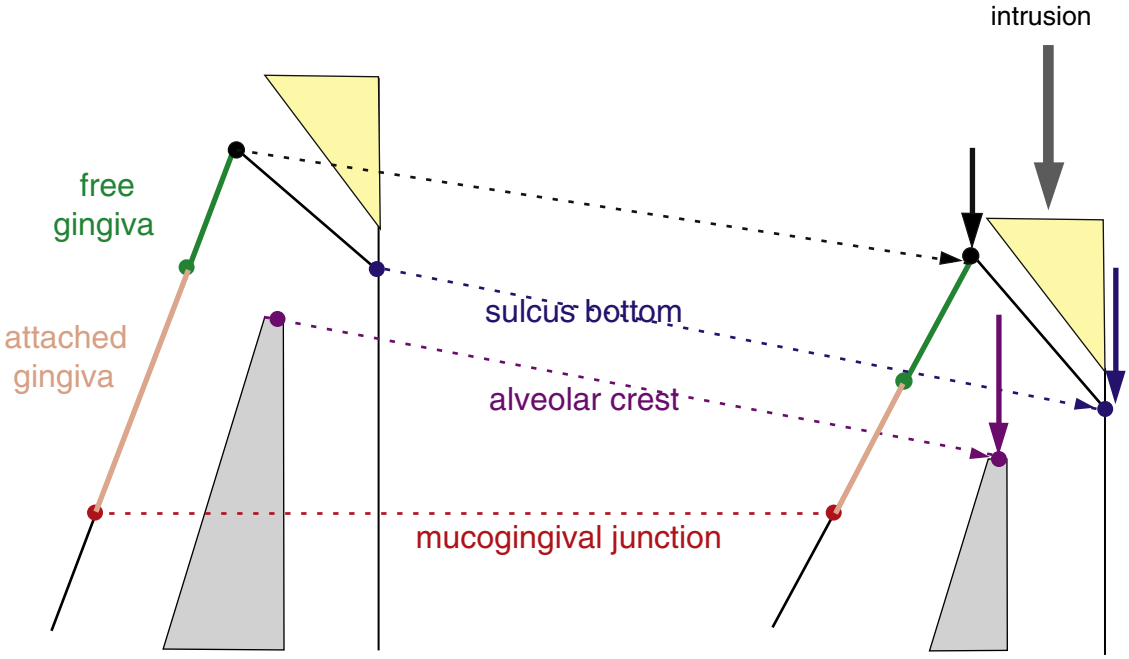


FIGURE 20-14 Local effects of molar intrusion. As the molars are intruded, the alveolar bone crest and free gingival margin will eventually move together if proper oral hygiene control is maintained. However, the mucogingival junction does not change; therefore the width of the attached gingiva decreases.

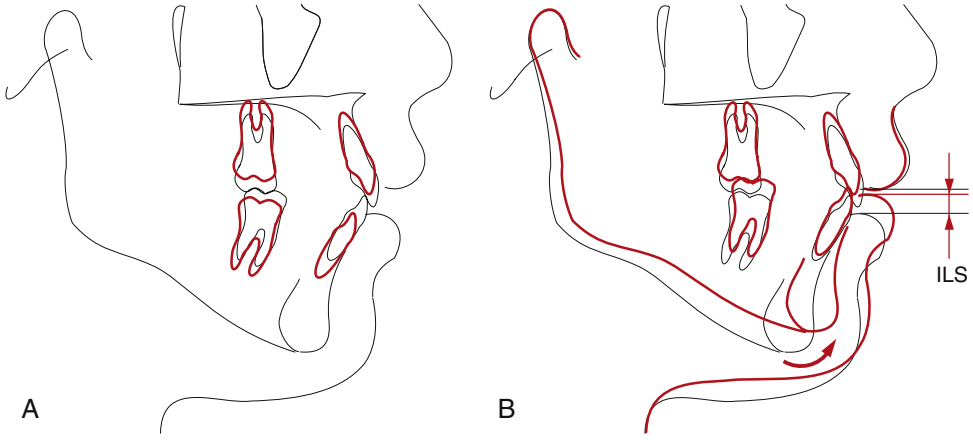


FIGURE 20-15 General effects of molar intrusion. After molar intrusion (**A**), the mandible rotates around the horizontal condylar axis to align itself to maintain interocclusal rest space. Consequently, the chin moves upward and forward, and the interlabial space (ILS) at rest decreases (**B**).

Force-driven mechanics have a statistically determinate force system, whereas shape-driven mechanics have a statistically indeterminate force system. From the standpoint of efficiency, force-driven mechanics are more advantageous because the force system can be precisely designed and does not significantly change, even with tooth movement.

The force system of shape-driven mechanics cannot be precisely designed, and it significantly changes with tooth movement because it is a statistically indeterminate force system. Therefore shape-driven mechanics are not efficient in cases with difficult types of tooth movements, such as molar intrusion. However, shape-driven mechanics are more effective in detailed and clinical adjustments of tooth positions. The combination of both types of mechanics takes advantage of the strengths that are exhibited by each.

Molar Intrusion

Decision Making

When considering the effects of molar intrusion* (Figs. 20-14 and 20-15) and deciding whether a molar should be intruded, three major factors should be evaluated.

Local factors. The intermaxillary occlusal relationship should be considered. The condition of the alveolar bone and attached gingiva should also be evaluated.

* References 6, 31–46, 129, 142–157.

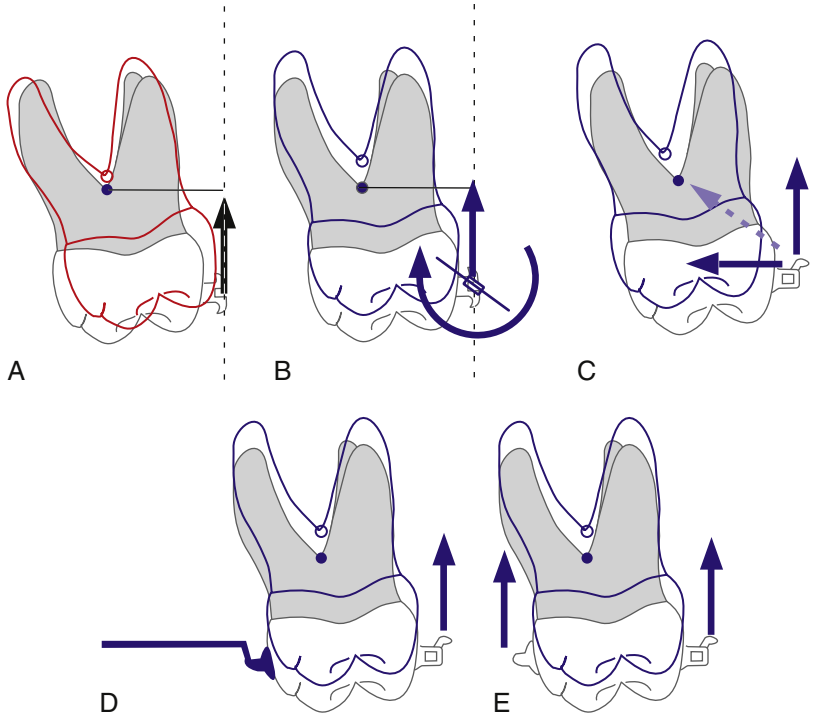


FIGURE 20-16 Posterior torque and arch form control during molar intrusion. Posterior torque and arch form (buccolingual positioning) control are related. **A,** Buccal intrusive forces away from the center of resistance cause buccal tipping and arch expansion. **B,** Lingual crown torque can be used to offset the tendency of buccal tipping for bodily movement. However, the precise amount of moment (palatal crown torque) needed is difficult to calculate. Theoretically, even if palatal crown torque is precisely applied, slight tooth movements can generate changes to the force system, rendering it biomechanically inefficient. To apply lingual crown torque, the archwire can be torqued or brackets with sufficient lingual crown torque can be used. **C,** In the case of using buccal intrusive forces, a constriction force can be applied to reduce the tendency for buccal tipping. The degree of constriction force should be similar to that of the intrusion force, but this force system is difficult to control precisely. **D,** An active or passive transpalatal archwire (TPA) or lingual archwire (LA) is effective for controlling torque and arch form. However, these appliances may be uncomfortable for patients while lowering the rate of tooth movement as well. **E,** Labial and lingual combined intrusion forces are most effective for torque control. This system can also control the arch form.

General factors. In addition to occlusal relationships, facial and smile aesthetics should be assessed. To reduce lower facial height, the upper and lower dentition should be controlled at the same time. If only one arch is intruded, then unwanted extrusion of the posterior teeth occurs in the opposing arch.[6,38,39,44]

Factors for stability. Stability of molar intrusion and anterior facial height reduction can be achieved by orthodontic overcorrection.[6,44,146] Anterior open bites can be corrected through surgery as well; however, surgical correction does not always guarantee stability.[158,159] For retention of an anterior open bite correction, functional improvement of the musculature after treatment is essential.* Tongue thrust during swallowing should be especially controlled for increased stability.[6,160,162,163]

Management of problems related to TMJ function and the masticatory muscles is also important for stability.[47,48] The mandible can rotate posteroinferiorly in the presence of a temporomandibular disorder (TMD), night teeth clenching, or bruxism, and, as a result, open bites can relapse.

Biomechanics

Mechanical efficiency. Molar intrusion is one of the most difficult tooth movements to achieve. Therefore mechanical efficiency is very important in the design of molar intrusion mechanics; that is, force-driven mechanics should be included in the mechanical design, considering its efficiency and predictability (Fig. 20-16). Approximately 80 to 100 g of intrusive force per molar can achieve 0.3 to 0.4 mm of intrusion per month.[31,35,36] Orthodontically induced root resorption is not clinically significant after the application of such intrusive forces, and no clinically critical side

* References 6, 38, 39, 44, 47, 48, 160–164.

FIGURE 20-17 Biomechanical efficiency of posterior intrusion. As seen with anterior intrusion, the use of a single force (e.g., force-driven mechanics) for posterior intrusion is effective and efficient, as opposed to the use of simply the brackets and wires. Intrusion can also be more quickly achieved with a single force. A single force, however, is not effective for controlling arch form, tooth axis, inclination of the occlusal plane, and detailed adjustments. A continuous arch, which is a statically determinate force system, is advantageous for controlling the arch form, tooth axis, and individual tooth positions yet disadvantageous from the viewpoint of efficiency. If a combination of the two force systems is used, then the disadvantages of each system are mutually compensated. For maxillary molar intrusion, force-driven mechanics (e.g., single force) were used to increase efficiency on the palatal side, and shape-driven mechanics (archwire with compensating curve) were used on the labial side to make detailed adjustments. **A, B,** Intraoral views before treatment. **C, D,** Intraoral views after 3 months of molar intrusion with buccal and palatal temporary anchorage devices (TADs) and a continuous archwire.

effects, such as attachment loss, are expected if trauma from occlusion (TFO) is adjusted and if the clinical situation is carefully monitored.

Three-dimensional control. Mechanics for posterior intrusion should be designed to achieve three-dimensional control of the molar, and the molar must be monitored in all dimensions during movement (Figs. 20-17 through 20-19; see also Fig. 20-16).[6] More specifically, rotations, tipping, torque, mesiodistal positioning, and inferosuperior positioning of the tooth all need to be controlled. Arch form, inclination of the occlusal plane, and the frontal occlusal plane should also be evaluated.

As previously mentioned, three-dimensional control can be managed in three ways: the use of a single force from the TAD, the use of brackets and archwires, and a combination of both of these methods (see Fig. 20-19). The use of a single force generated from a TAD in an appropriate position (i.e., force-driven mechanics) is more effective for gross control.

Treatment Mechanics

Maxillary molar intrusion. Palatal root control is important for upper molar intrusion because the center of resistance of the maxillary molar is located on the palatal side.[165] A palatal intrusion force is very effective for palatal root control and for an increase in biomechanical efficiency. However, palatal intrusive forces have a tendency to constrict the arch form and should be taken into consideration before applying these forces.

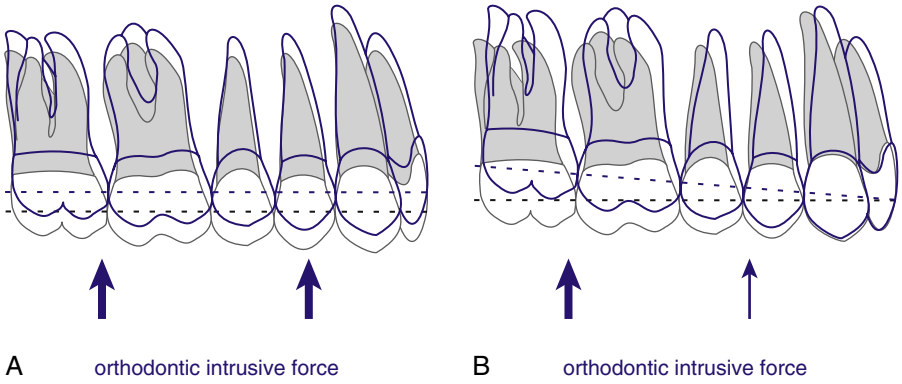


FIGURE 20-18 Control from a lateral view. Clinically, control of the inclination of the occlusal plane is one of the most important considerations in posterior intrusion, and, more specifically, the intrusion of the maxillary second molar is key. Occlusal plane inclination is related to molar axis control. A, To maintain the inclination of the occlusal plane, the premolars and anterior teeth should also be intruded approximately the same amount as the molars. This is especially indicated in the correction of a gummy smile or long face. B, The second molars should be intruded more than the premolars if the occlusal plane is to be steepened, especially in the correction of open bites. A steepening of the occlusal plane is difficult to achieve. Note the change in the inclination of the posterior occlusal plane and the changes in the axes of the individual posterior teeth, which suggests that axis control is related to occlusal plane control. Furthermore, the individual posterior teeth should be tipped back to aid in a steepening of the occlusal plane.

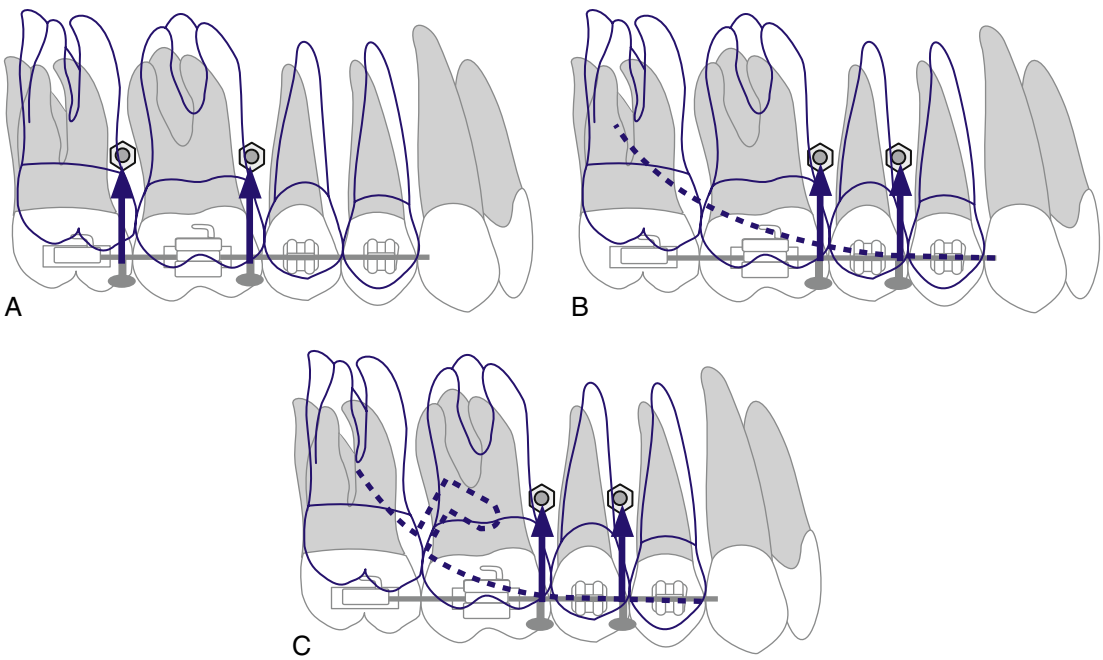


FIGURE 20-19 Second-order control. A single force generated from the temporary anchorage device (TAD) near the second molar is effective for intrusion of the second molar (A). TADs may not always be ideally positioned, but the mechanical design can compensate for such limitations in placement. For example, tip back bends and/or step down bends (B) or L-loops (C) can be used to increase efficiency.

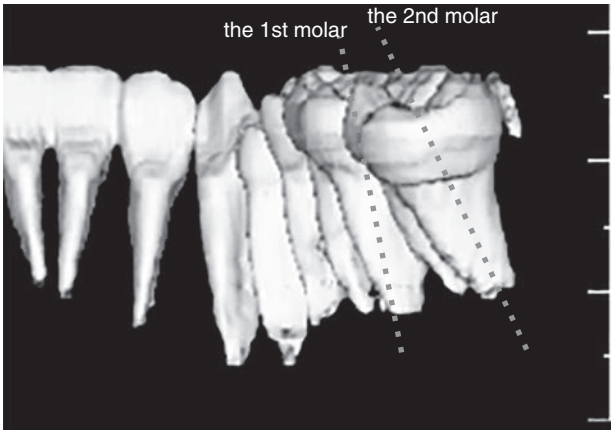


FIGURE 20-20 Root inclination of the mandibular molar. From a lingual view, three-dimensional computed tomography (CT) reconstruction view shows that the lingual inclination of the roots of the posterior teeth increases from the premolars to molars. The mandibular second molar is tipped more lingually than the mandibular first molar.

Mandibular molar intrusion. Mandibular molar intrusion is different from maxillary molar intrusion; biologically, the mandible comprises harder and denser bone, contributing to a slower bone turnover rate. Clinically, the success rate of a miniscrew-type TAD placed between the mandibular molars may be lower than a TAD positioned between the maxillary molars[55,60] because of masticatory stresses. The mandibular lingual area is especially difficult for TAD insertion because of low accessibility attributable to the tongue.

However, lingual intrusive forces are less of a necessity in mandibular molar intrusion than in maxillary molar intrusion because buccal intrusive forces in the mandible produce less buccal tipping, which is due to the fact that more lingual inclination exists in the mandibular molars, compared with the maxillary molars (Fig. 20-20). Considering these obstacles, control of the second molars should be a priority from the very beginning.

Yet when unilateral mandibular molar intrusion or posterior intrusion in conjunction with arch constriction is required, lingual miniscrew-type TADs prove to be very useful (Fig. 20-21). When the mandibular second molar is distally tipped, the interradicular space between the first and second molars is very limited, necessitating special precautions when placing TADs in this area. Additionally, because of low accessibility and thick lingual cortical bone, the risk for TAD fracture in the posterior lingual area is high.

Molar Distalization

Decision Making

- Required space: If more than 3 mm of space per side is required to achieve the treatment objectives, then premolar extraction may be preferable from the standpoint of treatment efficiency.
- Hard tissue conditions: There must be enough space for distalization. Second or third molar extraction should be considered before distalization to secure adequate space (Fig. 20-22).
- Soft tissue conditions: A clinically acceptable amount of attached gingiva must be present after distalization, especially on the distobuccal aspect of the molar.

Biomechanics

Mechanical efficiency. Distalization forces need to be efficiently applied to the molar as opposed to the other teeth. The distalization forces can be applied en masse or singly to each tooth.

In the maxilla, TADs placed on the palatal side can directly apply distalization forces to the molar. Moreover, these TADs can also control the mesiodistal axis of the molar through manipulation of the line of action (Figs. 20-23 through 20-25).

Although adjacent teeth may limit mesiodistal tooth movement, buccal interradicular miniscrew-type TADs are very useful in molar distalization because of the ease of placement and simple application during treatment (Figs. 20-26 through 20-28). With a properly positioned TAD, 3 mm of distal movement per side can be achieved, after which the initial TAD can be removed and a new TAD placed to gain further distalization.

Three-dimensional control. As with molar intrusion, three-dimensional control of the molar is important in molar distalization when using TAD mechanics (Figs. 20-29 through 20-34). Thus the mechanics should be designed to manage the three-dimensional position of the molar. Once again, three methods will achieve this control: the use of a single force from the TAD, the use of brackets and archwires, and a combination of both systems.

Notably, in molar distalization, special attention to vertical control is needed because distalization can inadvertently cause an increase in the overall vertical dimension.

En masse distalization. All of the anterior and posterior teeth can be distalized at the same time using rigid anchorage. The same principles are applied for full dentition distalization as for single tooth movement. Considerations for mechanical efficiency and three-dimensional control are necessary as well. With regard to three-dimensional control, the center of resistance of the total dentition can be estimated.[166] Theoretically, if the force is applied through the center of resistance of the whole dentition, then translation of the entire dentition will be achieved. Clinically, however, the tendency for the teeth to move individually as opposed to entirely is great (Fig. 20-35). Furthermore, if the dentition moves as a whole body and does not allow for tipping of individual teeth, then movement will be very slow. Even in en masse distalization, the key factor is molar control. If the molars are three-dimensionally well controlled, then moving the remaining teeth is comparatively easy. En masse movement is indicated when intrusion is needed in addition to distalization.

Molar Protraction

Decision Making

TADs can provide stable anchorage for molar protraction.[167–170] However, molar protraction toward an edentulous area can be more affected by biologic conditions than by biomechanics.[90,91,171,172]

When the first molar or second premolar is missing, protraction in the maxilla is somewhat predictable. However, in the mandible, various individual differences exist. According to research conducted by Roberts and colleagues,[169] the rate of molar traction can be as low as 0.2 mm per month. Extra caution should also be taken when moving teeth into edentulous areas in the mandible because severe periodontal attachment loss can be induced during protraction, depending on the condition of the alveolar bone.[90,91,172]

Text continues on page 541

FIGURE 20-21 A 25-year-old female patient reported chief complaints of protrusion, an anterior open bite, long face, and missing molars. Vertical disharmony was improved by anterior and posterior intrusion, as was the chin position attributable to molar intrusion and subsequent autorotation of the mandible. **A–C,** Intraoral view before treatment. **D–F,** Intraoral view after intrusion. Lingual temporary anchorage devices (TADs) between the mandibular molars were used to control the mandibular molars vertically and anteroposteriorly.

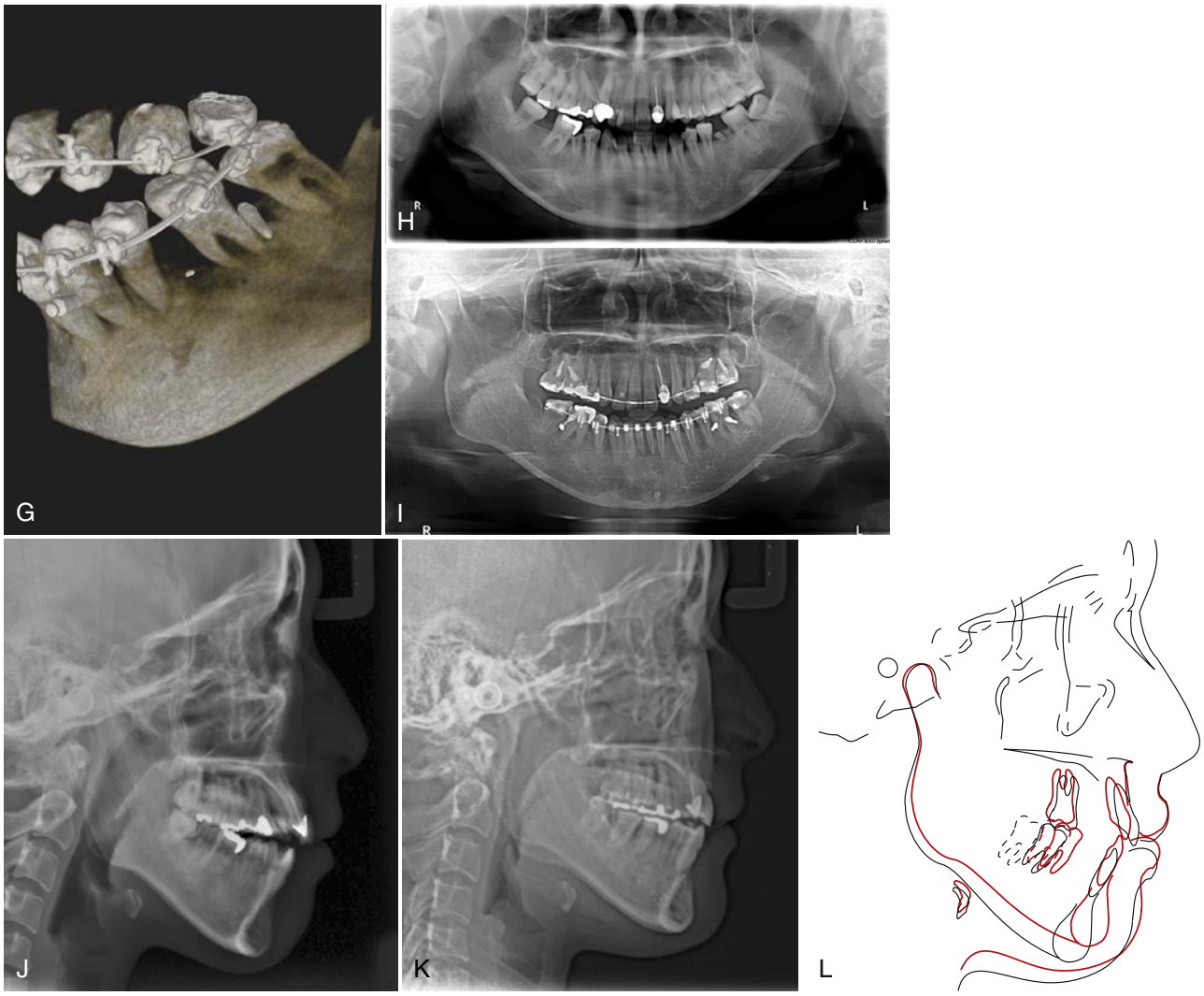


FIGURE 20-21, cont'd **G,** A three-dimensional cone-beam computed tomography (CBCT) image shows buccal and lingual TAD placement. Anterior buccal TADs were used to protract the posterior teeth, and lingual TADs between the molars were used to increase the efficiency of molar intrusion and to control the arch form. **H,** Panoramic radiograph before treatment. **I,** Panoramic radiograph at the completion of treatment. **J,** Cephalometric radiograph before treatment. **K,** Cephalometric radiograph at the completion of treatment. **L,** Superimposition of pretreatment and posttreatment cephalomatric radiographs. The solid line refers to the mandibular right first molar, and the dotted line indicates the mandibular left second molar.

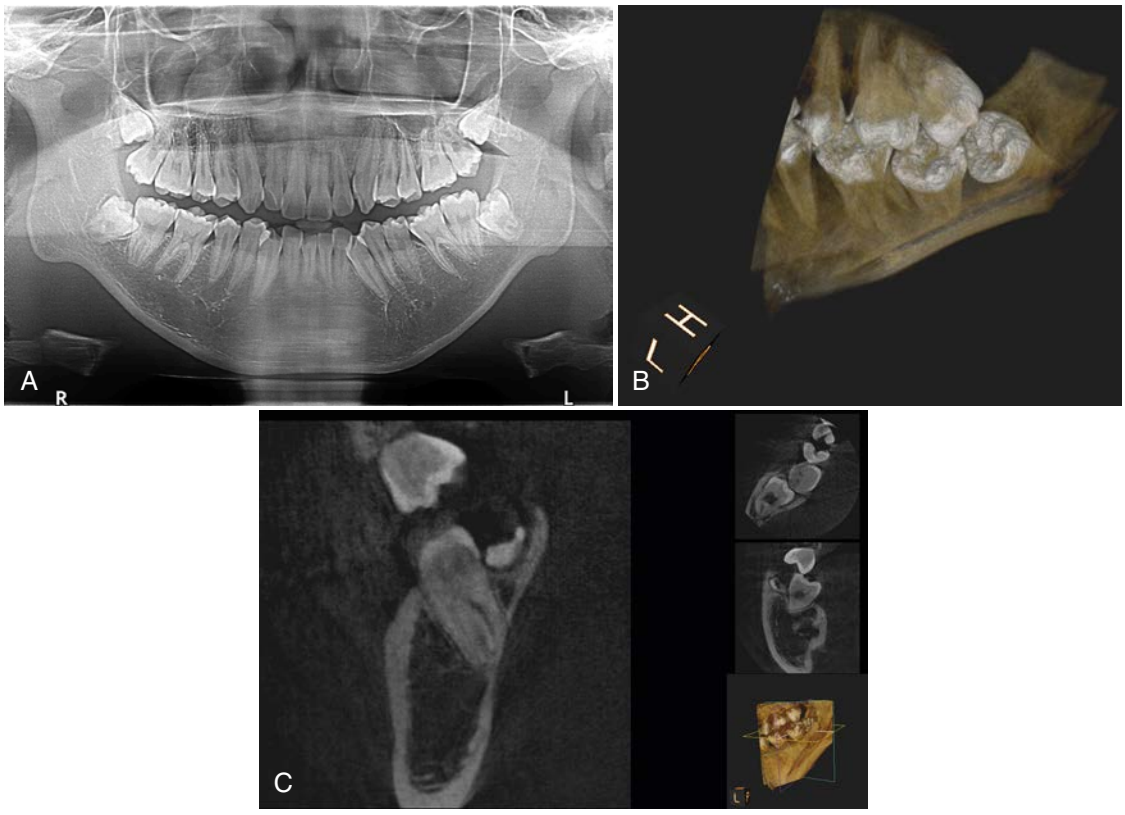


FIGURE 20-22 Even when the mandibular third molars are extracted, sufficient space to distalize the second molars cannot always be secured because of the three-dimensional morphologic structure of the mandible, which can cause the third molars to be located buccal to the mandibular second molars. **A,** Securing a moderate amount of space in the mandible was necessary to treat this 25-year-old male patient. The panoramic radiograph reveals an impacted right mandibular third molar. **B,** The three-dimensional view from the upper inner side shows the root tip of the mandibular second molar touching the inferior border of the mandible. **C,** A sagittal view at the level of the root tip of the mandibular second molar also shows contact between the root tip of the mandibular second molar and cortical bone. There is no space for distalization of the second molar.

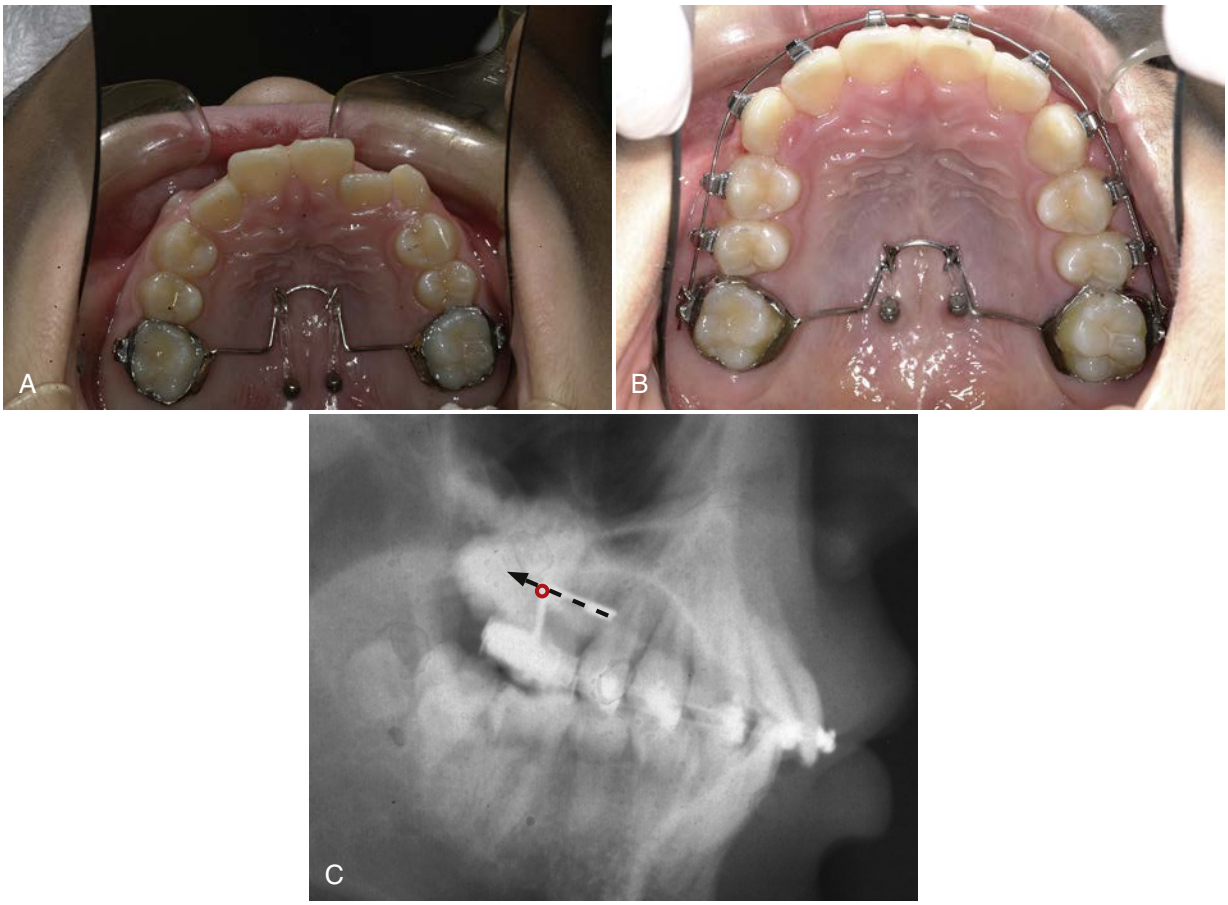


FIGURE 20-23 With a shallow palatal vault, mechanics, which consist of temporary anchorage devices (TADs) in the midpalatal suture area and a transpalatal arch, are simple and effective. With this anatomic structure, distalization forces from TADs travel through the center of resistance of the molar, which results in distalization by bodily movement. In this clinical case, the patient was in the middle of a growing period. Thus parasagittal TADs were placed, as opposed to midsagittal TADs, because palatal suture growth was not completed. **A,** Intraoral view before treatment. **B,** Intraoral view during treatment. **C,** Cephalometric radiograph during molar distalization. Distalization force (*black arrow*) travels through the center of resistance of the molar (*red circle*).

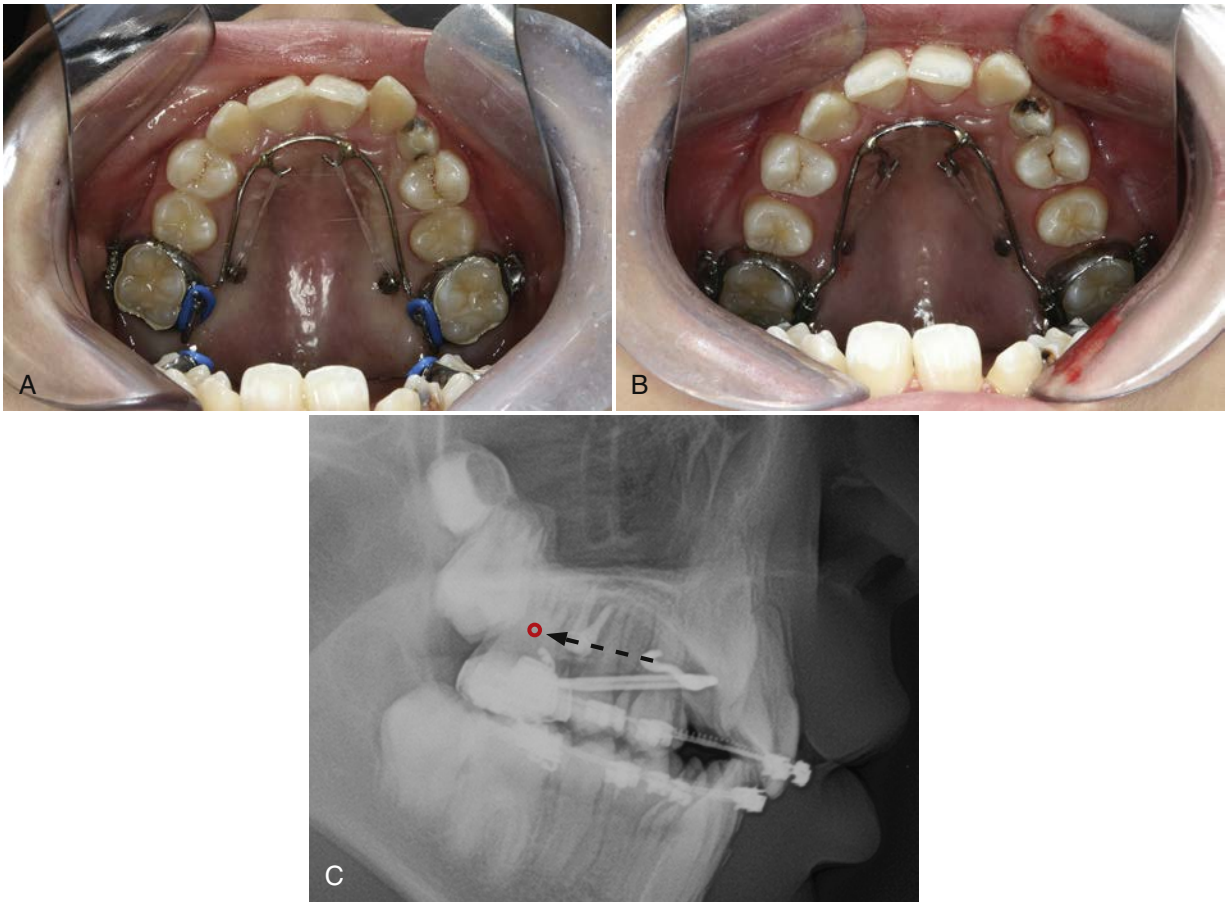


FIGURE 20-24 By modulating the line of action, distalization by bodily movement can be produced. With deep palatal vaults, temporary anchorage devices (TADs) in the palatal interdental area and a transpalatal arch can produce distalization forces that travel through the center of resistance of the molars. **A,** Intraoral view before molar distalization. **B,** Intraoral view after 5 months of molar distalization. **C,** Cephalometric radiograph before during molar distalization. Distalization force (*black arrow*) travels through the center of resistance of the molar (*red circle*). When compared with the case in Figure 20-23, the line of action moves occlusally, attributable to TAD positioning.

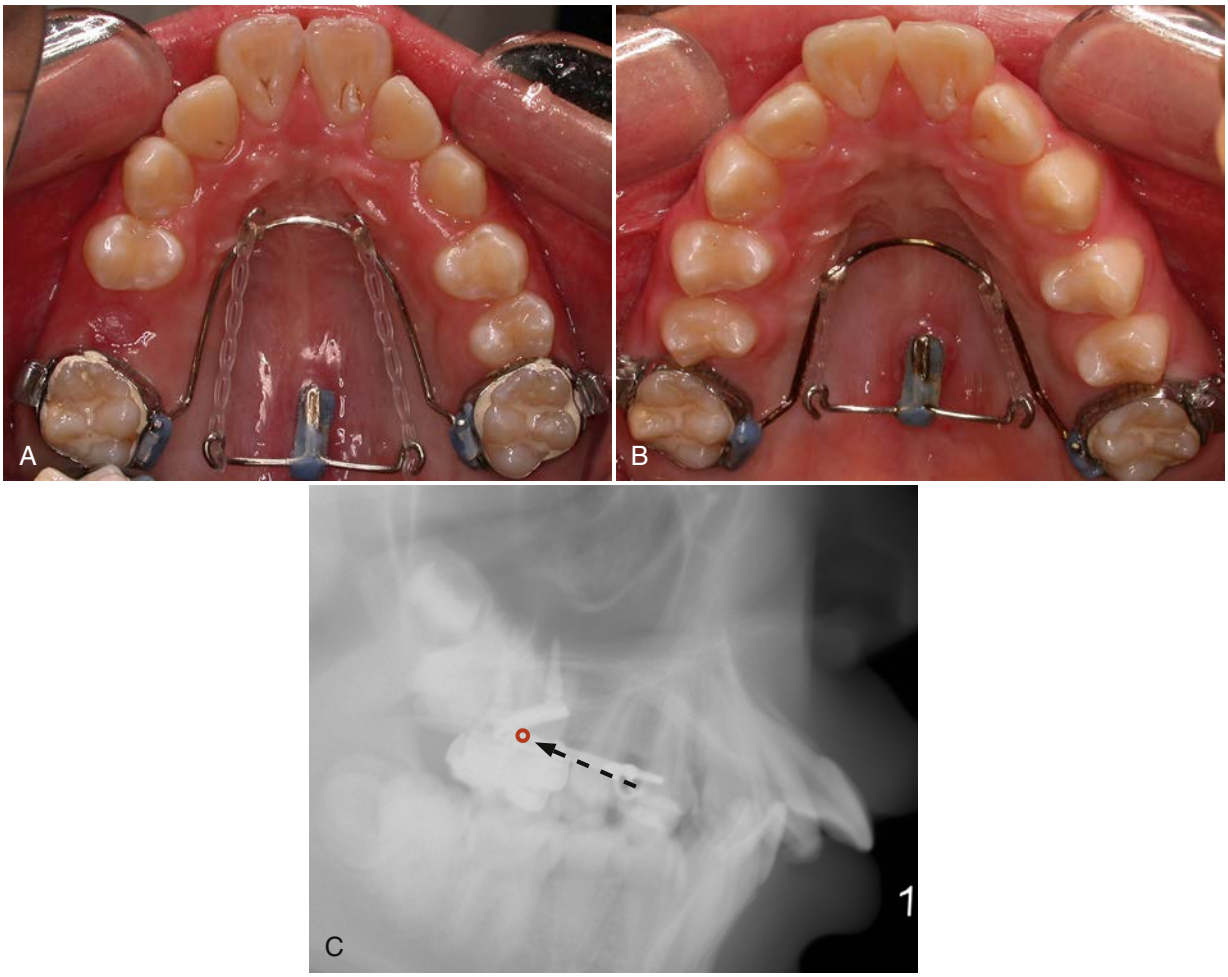


FIGURE 20-25 With deep palatal vaults, temporary anchorage devices (TADs) in the midpalatal suture area with attachments can modulate the line of action to produce distalization forces (**C,** *black arrow*) that travel through the center of resistance of the molars (**C,** *red circle*). If the distalization force is directly coming from the midpalatal TADs, as in this clinical case, then the line of action passes more apically (**C,** *black arrow*) than the center of resistance of the molar. **A,** Intraoral view before distalization. **B,** Intraoral view during molar distalization. **C,** Cephalometric radiograph taken during molar distalization.

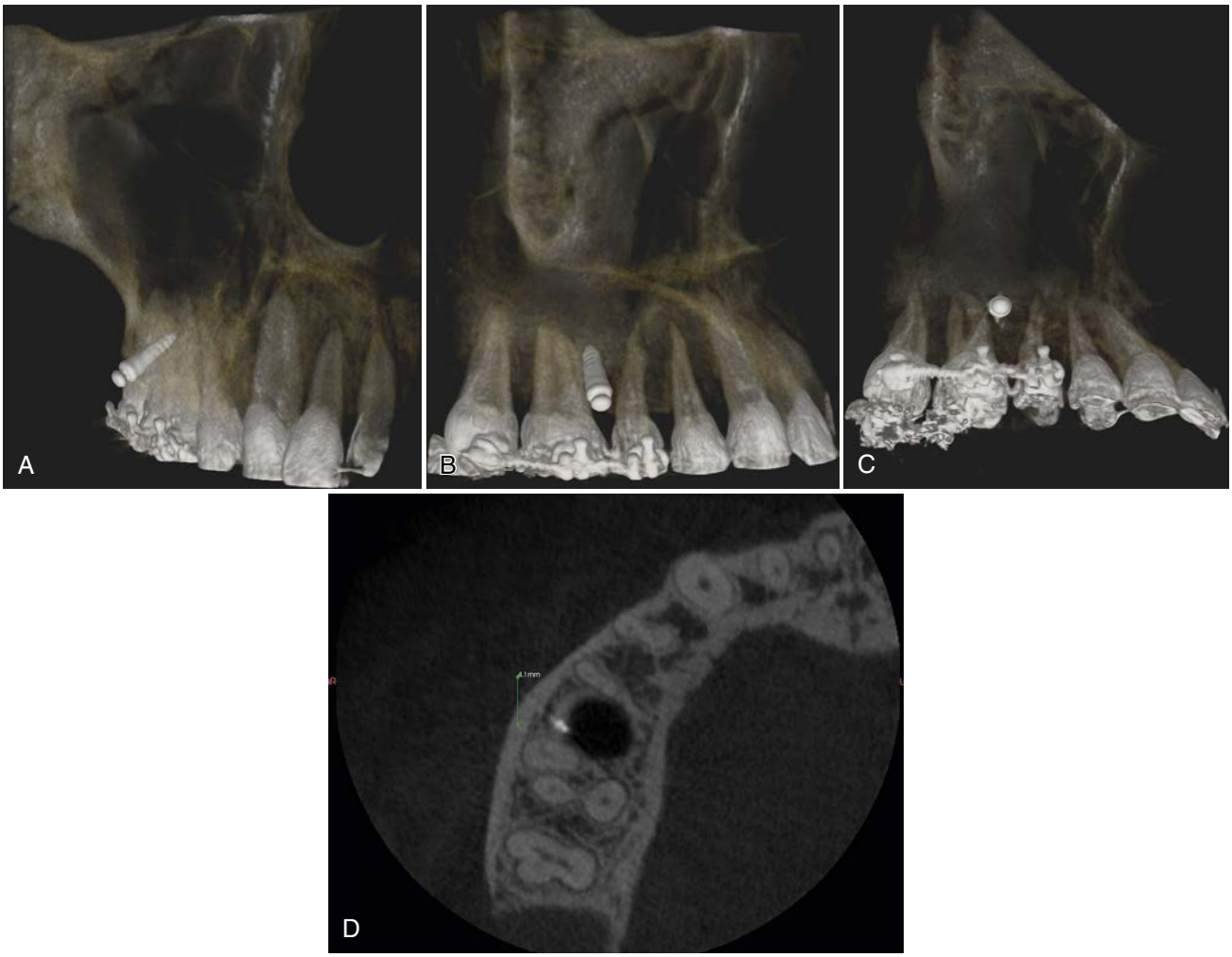


FIGURE 20-26 Buccal alveolar bone can provide enough space for a half-cusp width of distalization if the temporary anchorage device (TAD) is properly placed. However, narrow interradicular widths cannot provide enough space for mesiodistal movement. Angular placement of the TAD to the occlusal plane can take advantage of a wider buccal space. **A,** A three-dimensional view from the right side. The TAD was placed at an angle near the first molar for distalization. **B,** A three-dimensional view from the occlusal plane. **C,** A three-dimensional frontal oblique view. **D,** An axial view at the level of the tip of the TAD shows that the second premolar can move distally at least 4.0 mm.

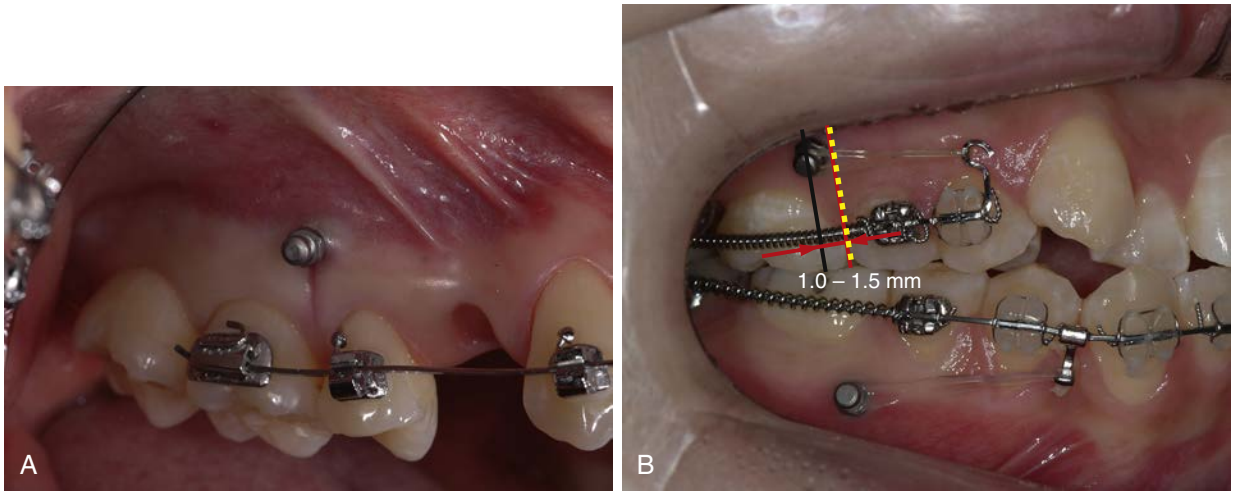


FIGURE 20-27 Off-center distal placement of the temporary anchorage device (TAD) is important in molar distalization. However, the protocol for the prevention of root injury should be followed to minimize the possibility of root injury. Normal insertion site in the buccal alveolus: **A,** The orthodontic TAD is usually placed on the midline between the adjacent teeth where it intersects with the mucogingival junction (MGJ). **B,** The TAD is placed 1.0 to 1.5 mm distal from the midline (*yellow dotted line*) because molar distalization is planned.

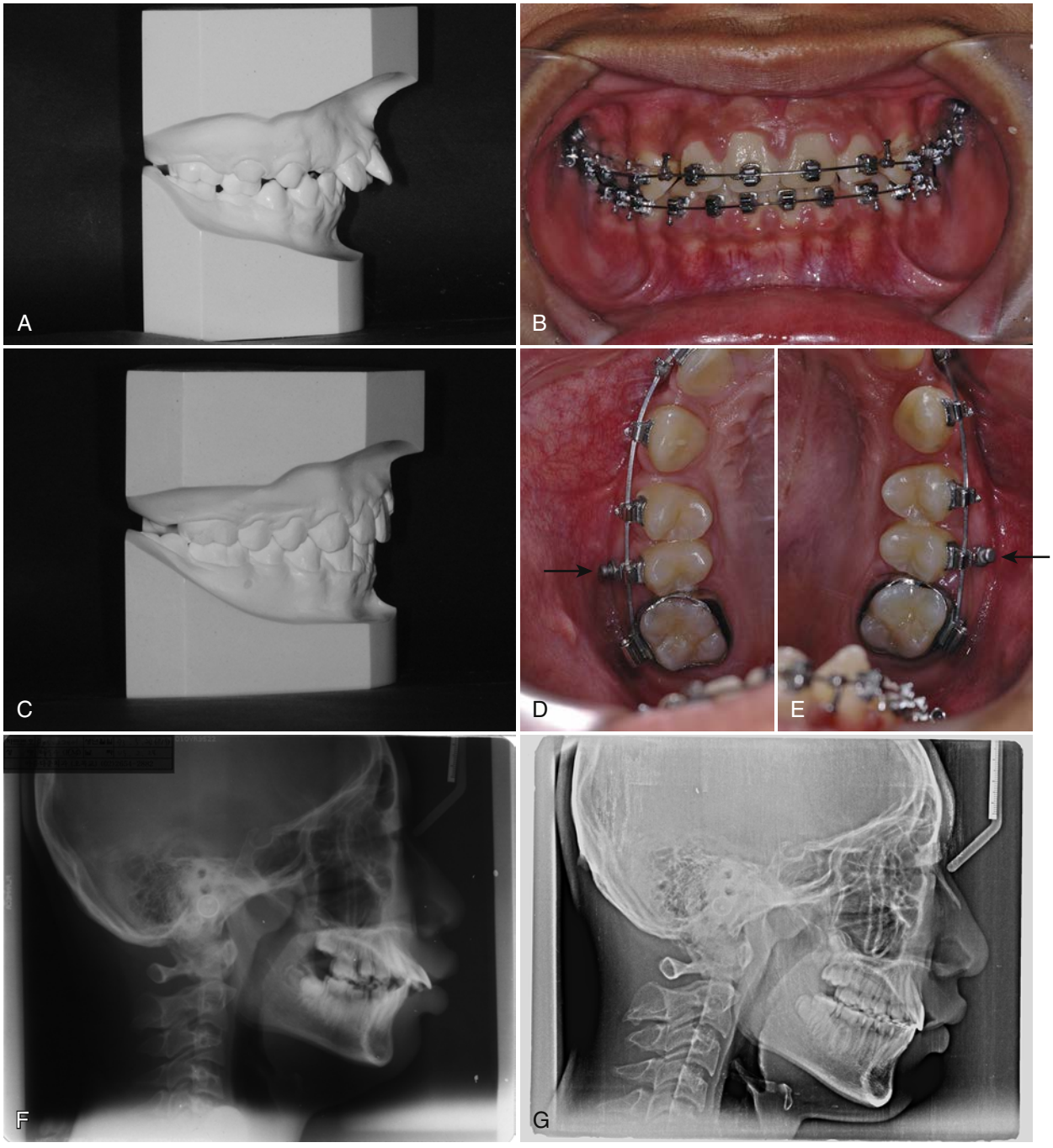


FIGURE 20-28 The patient was a 12-year-old boy whose chief complaint was protrusion. Severe overjet and a Class II canine and molar relationship were corrected by molar distalization using temporary anchorage devices (TADs) placed in the buccal interdental areas. Initial positions of the TADs at the beginning of treatment were close to the first molars. After distalization, the TADs appeared to be on the same line as the roots of the second premolars **(D, E)**. If placed with proper angulation to make use of the buccal space and placed more distally rather than in the middle of the interproximal space, then buccal TADs are useful in molar distalization. **A,** View of occlusal relationship before treatment. **B,** Intraoral view during molar distalization. **C,** View of occlusal relationship at the completion of active treatment. **D, E,** Intraoral view during treatment. Buccal TADs are in line with the second premolars (*black arrows*). **F,** Cephalometric radiograph before treatment. **G,** Cephalometric radiograph at the completion of active treatment.

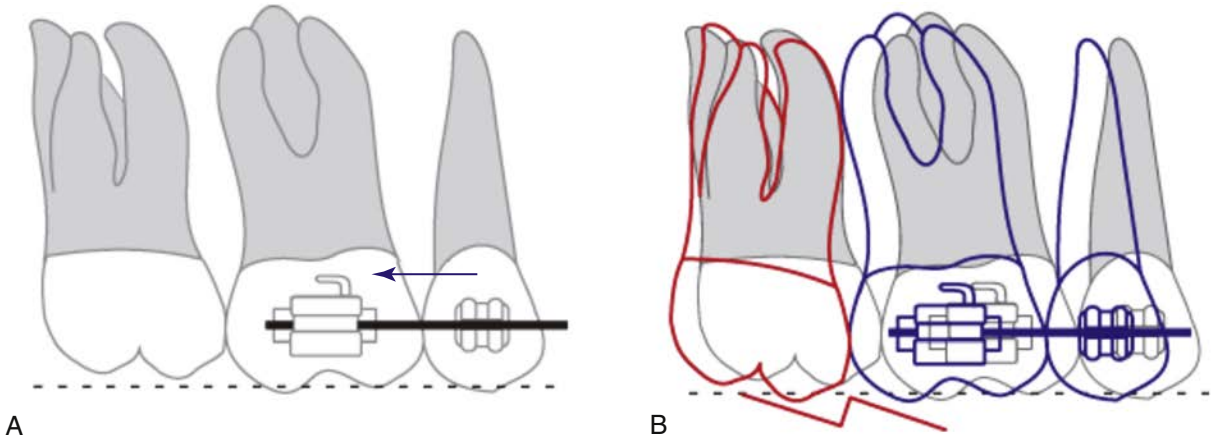


FIGURE 20-29 Mesiodistal axis control. **A, B,** If the second molar is not included, then it may tip back as a result of the distal movement of the first molar; marginal ridge discrepancies can result. The second molar should be simultaneously controlled whenever possible during molar distalization to prevent any vertical discrepancies. **C,** Even with the strap-up including the second molar, bodily distalization of the second molar is not easy to attain because tipping can easily occur. A clinical sign of tipping is the elevation of the second molar mesial marginal ridge (*green arrow*). To prevent distal tipping while distalizing, the use of wires of adequate stiffness is also a practical consideration.

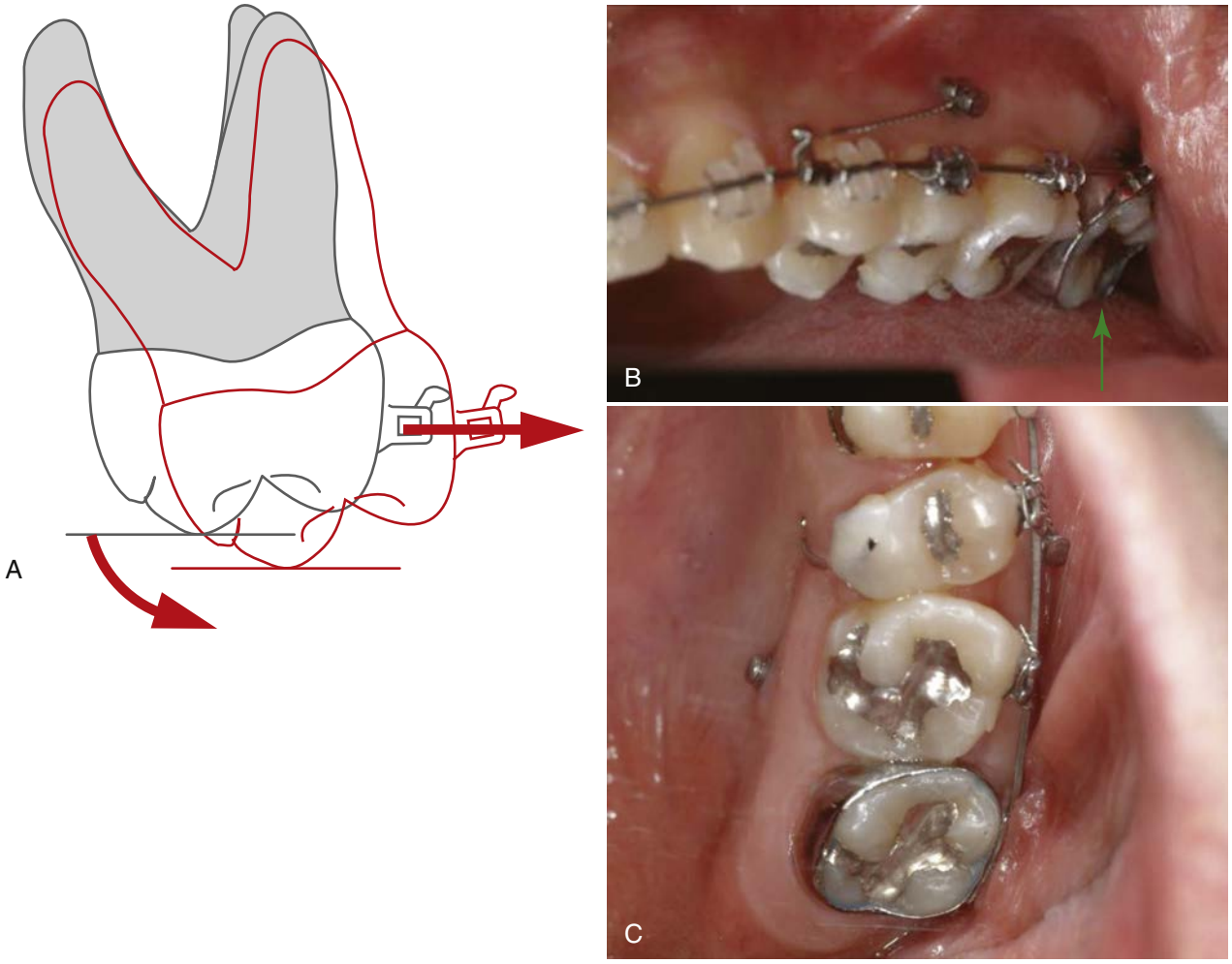


FIGURE 20-30 Arch form (buccolingual positioning) and torque control. During distalization, the maxillary second molar can easily tip buccally, whereas the mandibular second molar can easily tip lingually, attributable to the respective buccolingual inclinations of these molars. In addition, posterior torque control and arch form (buccolingual positioning) control are related. If the second molar is tipped to the buccal, then the palatal cusps inferiorly fall and the torque of the second molar worsens. A slight toe-in bend may aid in controlling the buccolingual positioning of the second molar and, as a result, may also be useful in controlling torque. **A,** Schematic illustration of arch form changes and the relationship between buccolingual position and torque. **B, C,** An intraoral view after molar distalization. The second molar torque worsened because of buccal tipping (*green arrow*).

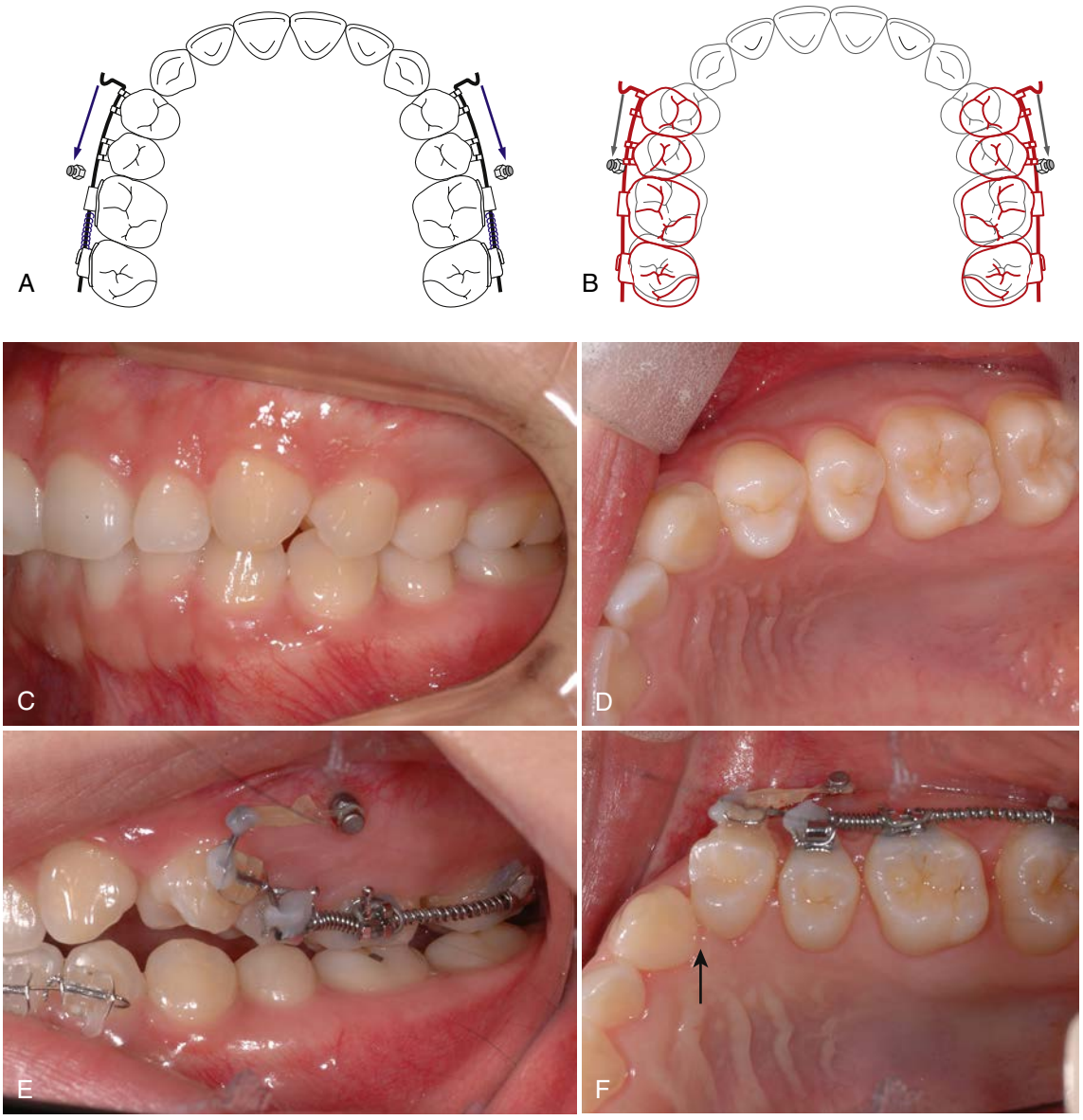


FIGURE 20-31 Arch form control. Sectional mechanics without cross-arch splinting are not adequate for arch form control because of the tendency for mesial outrotation of the posterior segment. **A, B,** Schematic illustrations demonstrate the rotation of the buccal segment by distalizing forces. **C, D,** Intraoral views before molar distalization. **E, F,** Intraoral views during molar distalization demonstrate the mesial outrotation (*black arrow*).

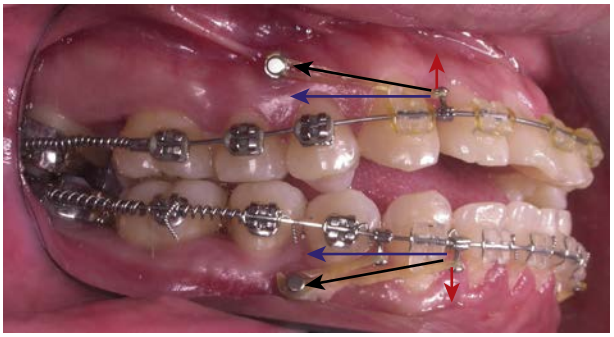


FIGURE 20-32 Vertical control: Distalization forces may have intrusive force vectors because of geometric positions of the temporary anchorage devices (TADs). Intrusive force vectors, in turn, can cause unwanted intrusion, such as anterior bite opening or occlusal plane canting, both of which are difficult to correct once developed. Therefore such intrusive force vectors should be controlled. The use of lever arms is one way to eliminate intrusive force vectors effectively (*black arrows*, retraction force; *purple arrows*, distalizing force vector; *red arrows*, intrusive force vector).

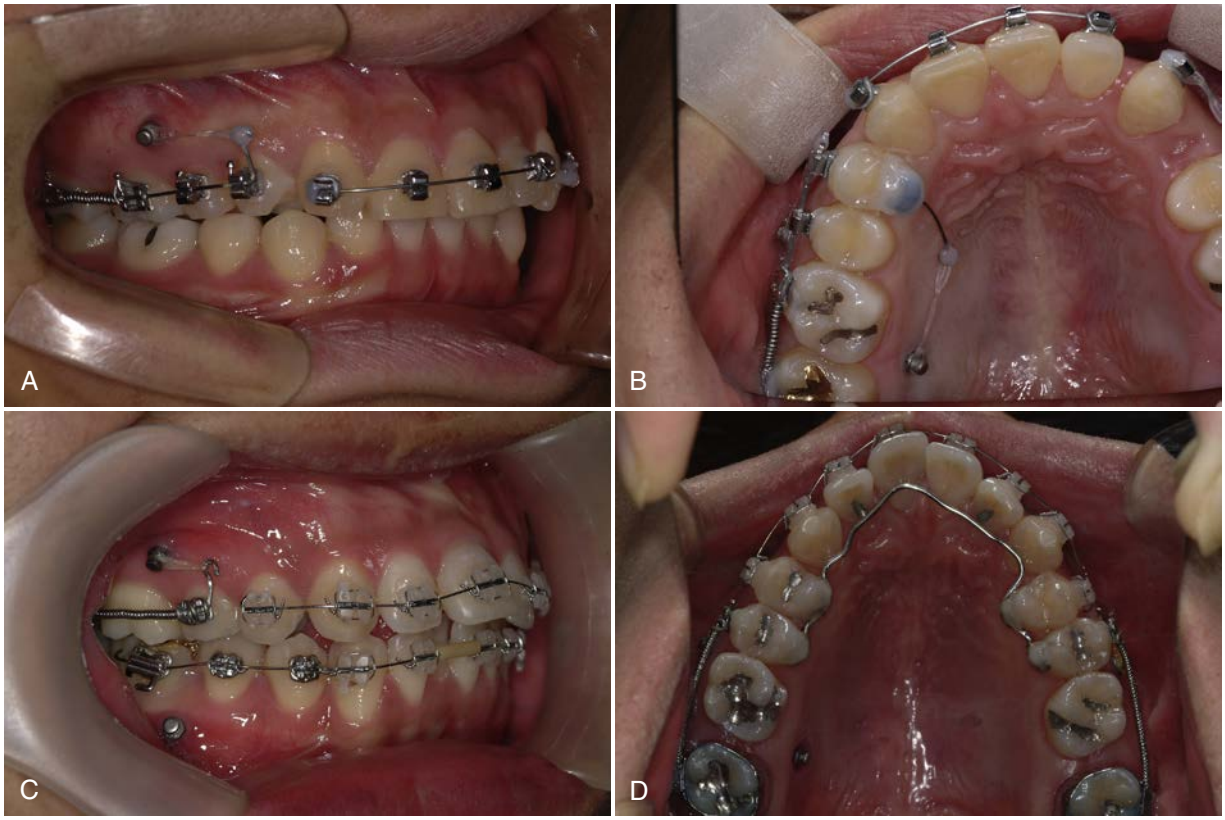


FIGURE 20-33 Arch form and vertical control. **A, B,** Lever arms are used for vertical control. Simultaneously applying buccal and palatal distalizing forces is also effective for arch form control and is useful for asymmetric distalization. Lever arms are used for vertical control **(C)**, and cross-arch splinting is used for arch form control **(D)**.

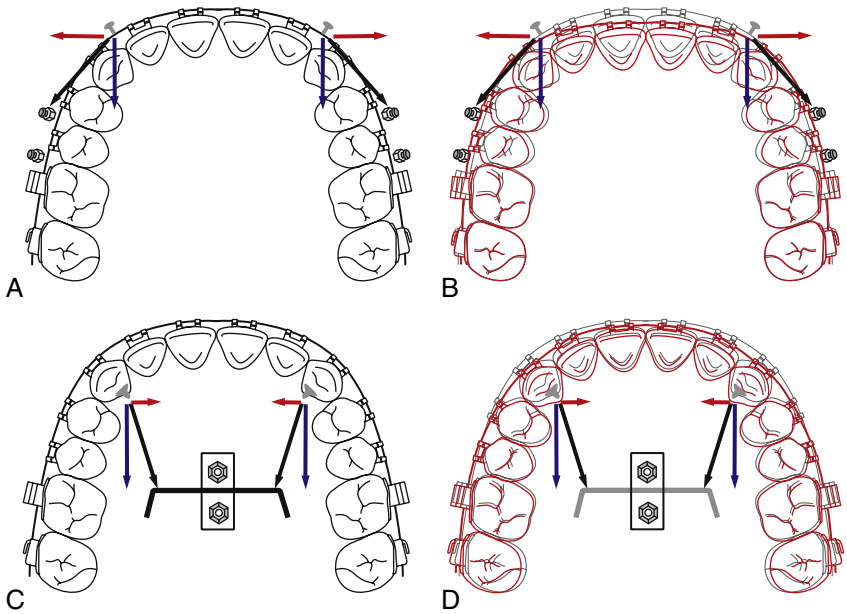


FIGURE 20-34 According to the geometric positions of temporary anchorage devices (TADs), distalizing forces have a horizontal force vector. In addition to distal movement, buccolingual tipping is more likely to occur than distal movement because the sum of the circumcemental area of the molar roots is greater than the sum of the anterior teeth. These horizontal force vectors can lead to arch expansion **(A, B)** or constriction **(C, D)**.

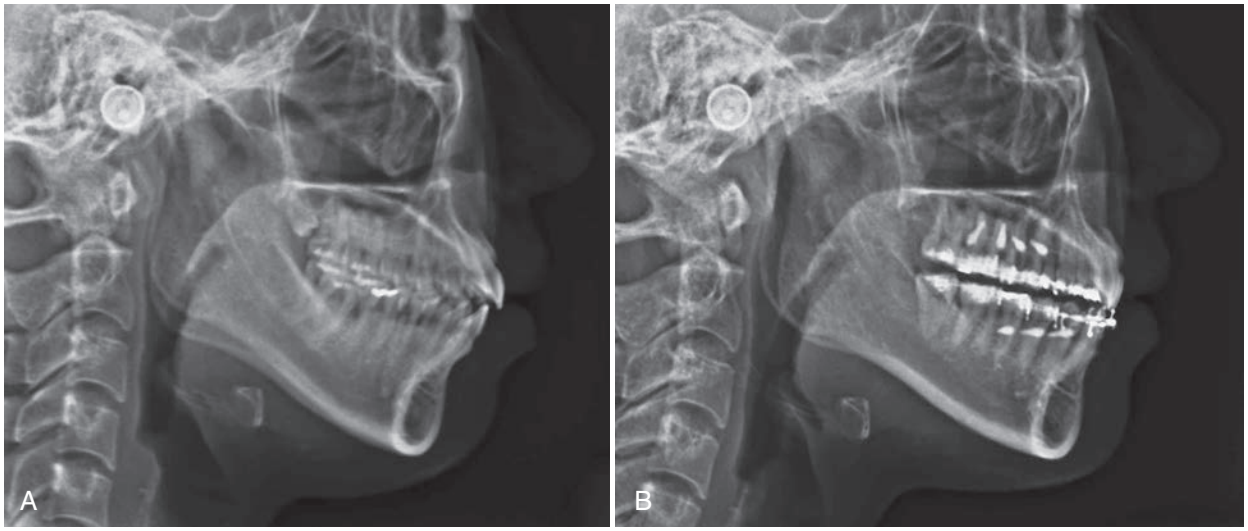


FIGURE 20-35 The entire dentition was distalized to correct protrusion. Although twin brackets were used, distal tip back of the individual teeth was observed. **A,** Cephalometric radiograph before treatment. **B,** Cephalometric radiograph after molar distalization.

Therefore, to prevent loss of attachment, the following precautions should be considered when protraction into an edentulous area is planned, especially in the mandible. Evaluating the periodontal condition is necessary in the area where protraction is planned. Vertical and transverse bone quantity influences the periodontal prognosis. Low alveolar bone levels and narrow alveolar bone result in a greater chance of attachment loss. An inadequate amount of attached gingiva has a greater chance of attachment loss as well. The patient's chronologic and dental ages should be also considered as growing children and patients with incomplete third molar development have a lower chance of attachment loss. The number of roots and the shape of the roots of the tooth to be protracted, in addition to the thickness of the surrounding cortical bone, influence the level of difficulty of the case.

Biomechanics

Mechanically, three-dimensional control is also important for successful protraction (Fig. 20-36 through Fig. 20-38).

Biologically, additional considerations may be necessary during mandibular second molar protraction after extracting the first molar.[173] Removal of a tooth results in both horizontal and vertical changes to the dimensions of the hard and soft tissues. Considering the slow speed of tooth movement observed during molar protraction, hemisection and then sequential extraction of the first molar, in conjunction with second molar protraction, should be considered (Fig. 20-39).[174]

Anterior Retraction in Extraction Treatment

A unique feature of TAD mechanics in premolar extraction cases is the ability to adjust the anteroposterior position of the anterior teeth and molars. Moreover, TAD mechanics can simultaneously address vertical disharmony anterior retraction (see Fig. 20-4).*

Decision Making

First of all, the amount of anterior retraction and the type of tooth movement that will be used to retract the anterior teeth must be determined. If the anterior teeth are excessively retracted, then smile aesthetics can, in fact, be negatively affected. Adequate alveolar space is also necessary for bodily retraction of the roots of the anterior teeth. Verification of the amount of alveolar bone is necessary during the treatment planning process. When a large amount of retraction is planned, the risk for root resorption and periodontal attachment loss is higher[50,51,87]; therefore risk management is important. In addition, precaution is greatly needed to avoid overretraction and overintrusion of the anterior teeth when using rigid anchorage.

Biomechanics

Biomechanically speaking, no significant difference exists between conventional mechanics and TAD mechanics with regard to anterior retraction. The general principles in extraction treatment with conventional edgewise techniques are also important in extraction treatment with mini-implants. A proper force system should be designed, and monitoring this system is especially important, as it is with conventional mechanics.

The TAD can supply rigid anchorage for maximum anterior retraction and anterior torque adjustment. Moreover, TADs are generally placed apically, making it advantageous to control the line of action for canine axis and torque control during retraction (Fig. 20-40).

To achieve successful anterior retraction, canine axis control and anterior torque control are imperative, even with TAD mechanics. Canine lingual tipping, which is loss of canine torque, should be closely followed in cases with large amounts of anterior retraction (Fig. 20-41).

When the retraction force is directly generated and only from the TAD, further considerations are needed (Fig. 20-42 and Fig. 20-43).[15,20,21,175–177] For example, canine control becomes more critical. Additionally, the intrusive force vector of TAD mechanics is available for vertical control of the anterior teeth; however, when left improperly monitored, side effects such as occlusal plane canting can occur.

One should remember that the TAD itself is not controlling the canine axis and anterior torque.

Text continues on page 546

* References 11, 15, 20, 21, 175–177.

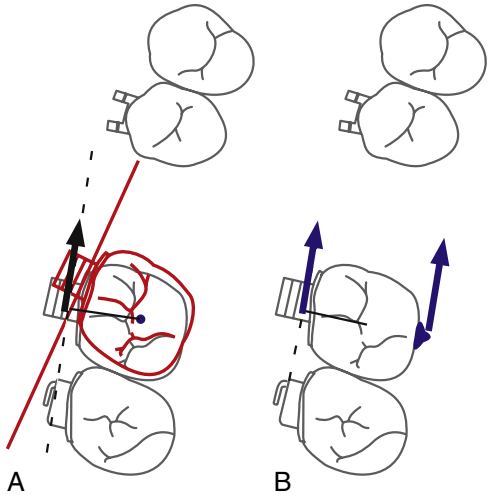


FIGURE 20-36 Rotation control: **A,** Buccal protraction forces (*black arrow*) produce a moment of mesial inrotation (red line). **B,** Combination of buccal and lingual protraction forces (*blue arrow*) is a simple and effective way to offset this mesial inrotation tendency.

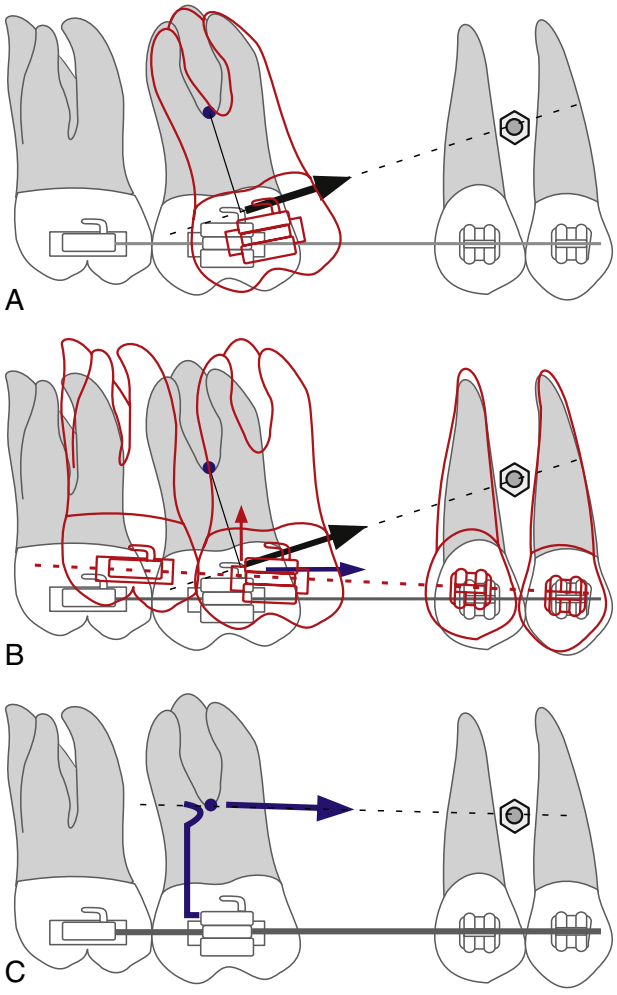


FIGURE 20-37 Molar axis and vertical control from a lateral view. **A,** Protraction forces (*black arrow*) away from the center of resistance (black dot) cause an inclination for mesial tipping. **B,** Generally, temporary anchorage devices (TADs) are apically placed, and as a result, protraction forces (*black arrow*) have intrusive force vectors (*red arrow*). If intrusion occurs on only one side, then occlusal canting can develop, which is very difficult to correct. **C,** A lever arm (blue wire) engaged in an auxiliary tube on the first molar is effective in vertical control.

FIGURE 20-38 The patient was an 18-year-old girl, whose problem was the severely mutilated upper first molar. Because of prolonged inflammation, little alveolar bone was left in the upper first molar area. Using temporary anchorage devices (TADs), the second molar was protracted after extraction of the first molar, and the third molar erupted to its proper position. The periodontal tissue of the protracted second molar was in good condition. Indirect application is also effective in vertical control, and this same application can also provide stable anchorage in cases of unilateral molar protraction. **A, B,** Intraoral views at the start of treatment. **C, D,** Intraoral views at the completion of treatment.

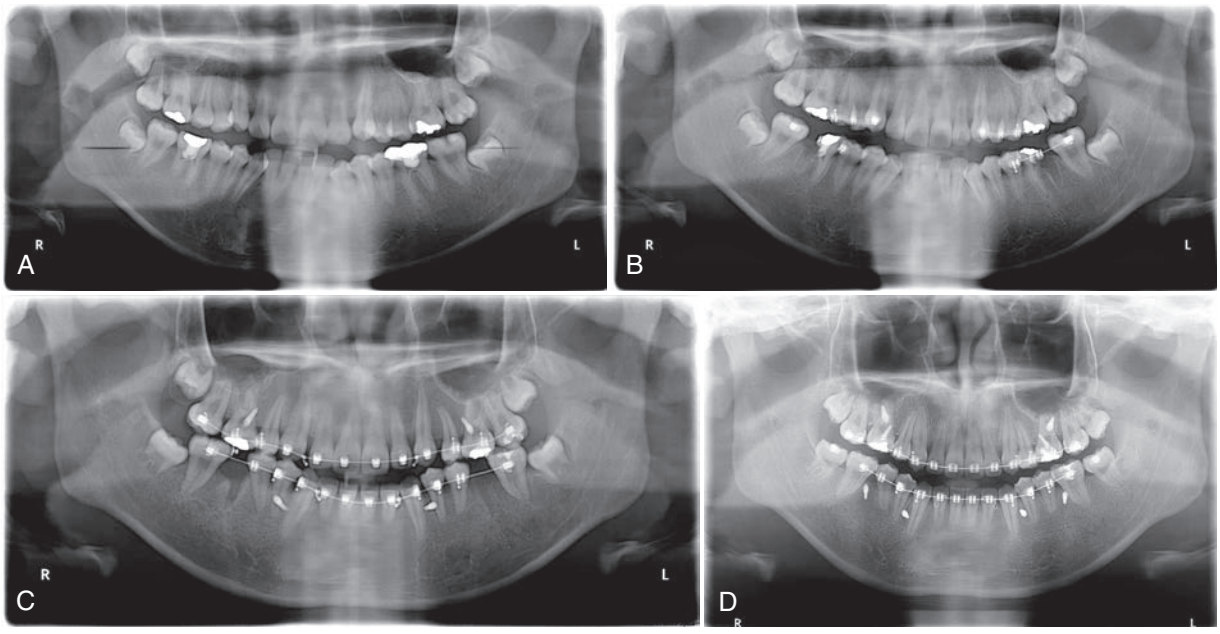


FIGURE 20-39 For the orthodontic treatment of this 18-year-old female patient, extraction of the premolars was needed. However, the treatment plan was established to include extraction of the mandibular first molars instead, as they were compromised with multiple pulpotomy procedures and mesial protraction of the third molars. When a first molar is extracted, a large amount of space is left behind, requiring prolonged treatment time to close. Considering alveolar bone loss after extraction, consecutive extractions after hemisection of the first molar were decided upon to minimize the risk of delayed tooth movement attributable to narrowing of the alveolar ridge. **A,** Panoramic radiograph before treatment. **B,** Panoramic radiograph after hemisection and extraction of the distal portion of the first molar. **C,** Panoramic radiograph after extraction of the mesial portion of the first molar. **D,** Panoramic radiograph after space closure.

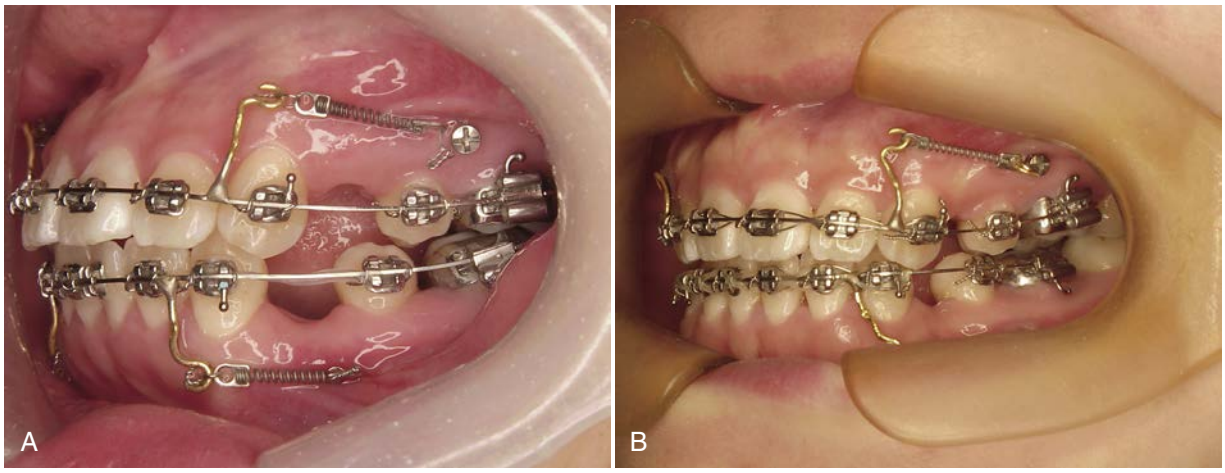


FIGURE 20-40 The line of action can be apically moved by using long lever arms for anterior torque control during retraction. **A,** Intraoral view at the start of anterior retraction. **B,** Intraoral view during anterior retraction.

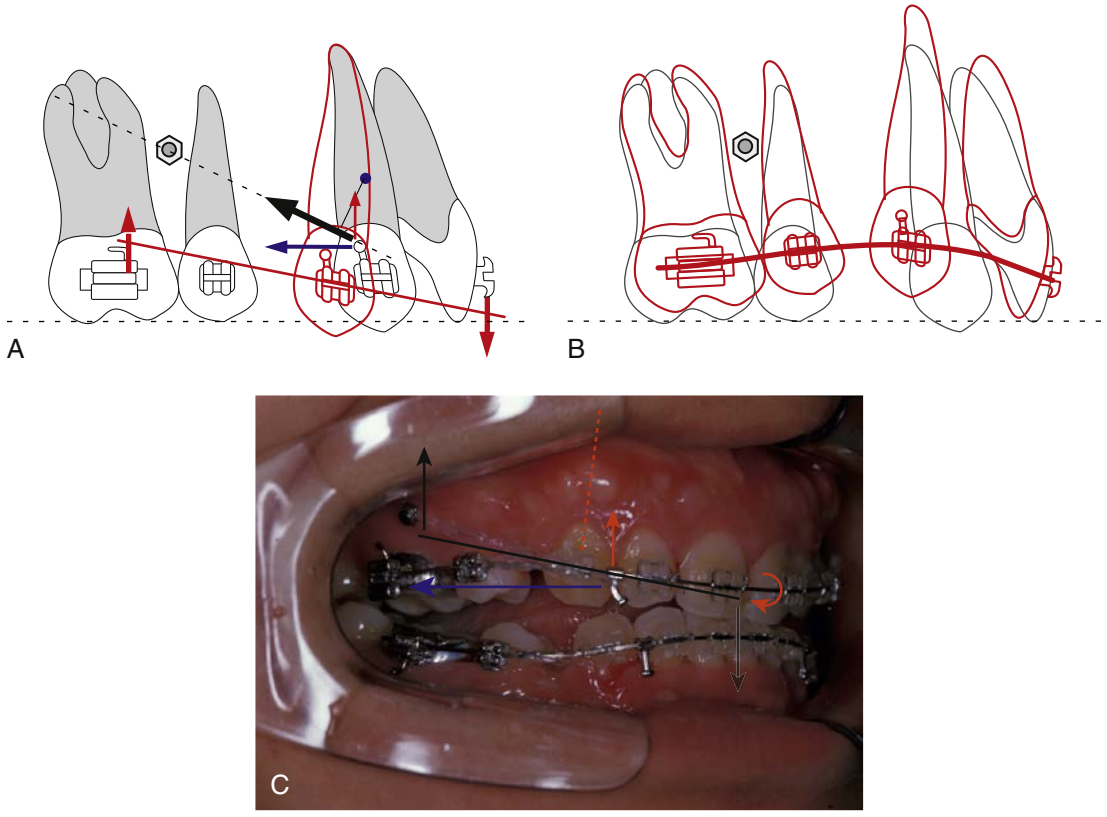


FIGURE 20-41 Distal tipping of the canine causes deflection of the archwire **(A)**. As a consequence, an extrusive force develops in the anterior teeth and anterior torque worsens. Moreover, as a result of the deflection (twisting) of the archwire, lingual crown torque is produced in the anterior teeth, which induces loss of anterior torque. The posterior segments are intruded because of deflection of the primary archwire, which is caused by distal tipping of the canine and the intrusive force vector from the retraction force **(B, C)**.

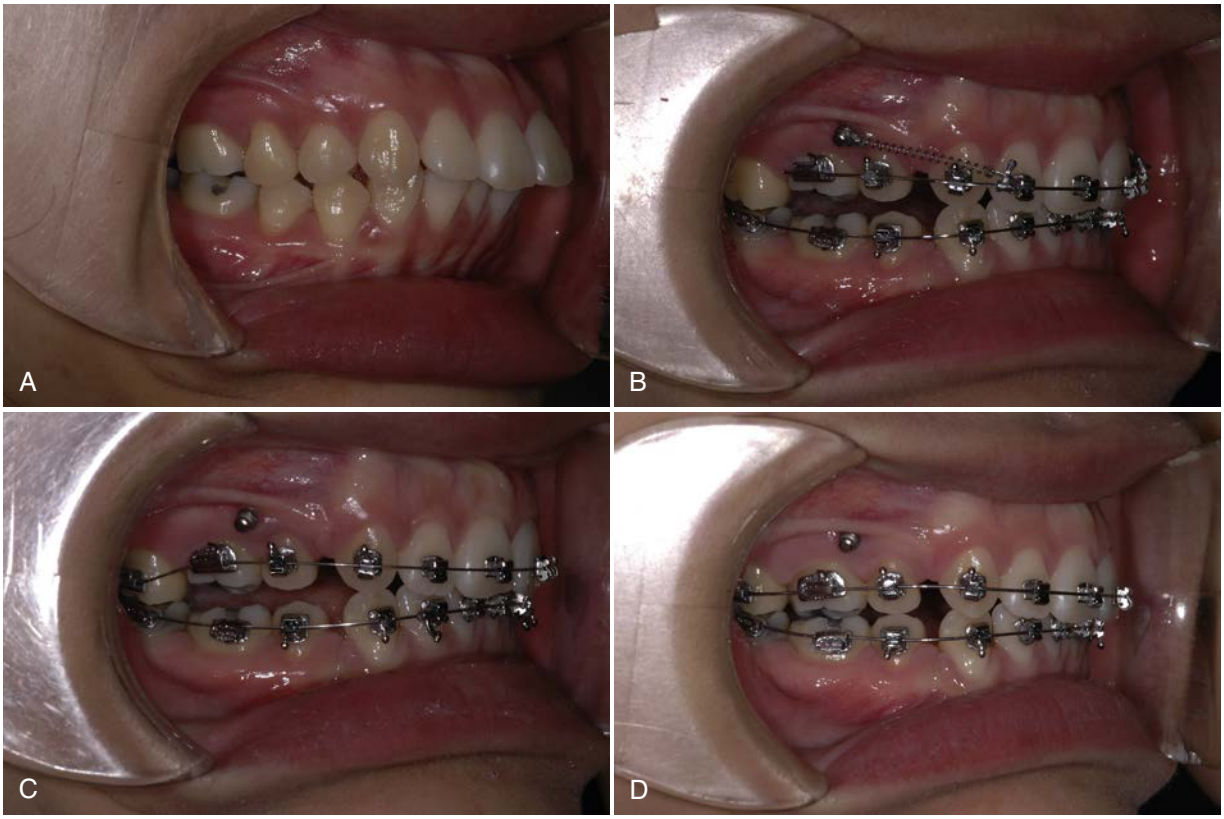


FIGURE 20-42 Canine axis control and control of the intrusive force vector are important in preventing posterior bite opening. A posterior bite opening was corrected by canine axis control and wire engagement into the second molar. **A,** Intraoral view before treatment. **B,** Intraoral view during anterior retraction, demonstrating a posterior bite opening. **C,** An attachment was bonded to the second molar. A leveling archwire was placed, and the retraction force was removed. **D,** Intraoral view during anterior retraction, demonstrating an improvement in the posterior bite.

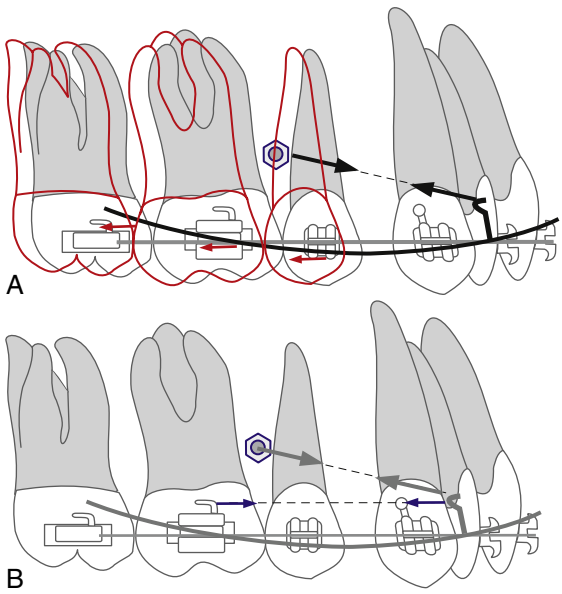


FIGURE 20-43 When a temporary anchorage device (TAD) is used for anterior retraction, particularly with a curved main archwire, the retraction force can be delivered to the posterior teeth by friction of the archwire. As a consequence, the posterior teeth can be distalized **(A)**. This distal movement of the posterior teeth can be monitored by checking the intermaxillary occlusal relationship. Application of a light force to the molar is useful in maintaining molar position **(B)**.

CASE STUDY 20-1 Treatment of Congenitally Missing Teeth: Molar Protraction Using Asymmetric Mechanics

Jung Kook Kim

The patient is a 20-year-old woman whose chief complaints are mobile primary molars, anterior crowding, and minor protrusion. Prosthodontic treatment after extraction of the primary molars is an option, but the patient desires a conservative approach and also wishes to address the anterior crowding. Therefore protraction of the maxillary left and mandibular right molars with temporary anchorage devices (TADs) is planned to close the spaces left by the congenitally missing premolars and to relieve the anterior crowding. The presence of the mandibular right third molar allows for proper occlusion with the maxillary right second molar after protraction of the mandibular right second molar.

For satisfactory treatment results and facial aesthetics, overretraction of the anterior teeth has to be prevented, and each molar needs to be anteroposteriorly controlled using asymmetric mechanics.

In an effort to protract the molars while controlling their anteroposterior positions, continuous arch mechanics and lever arm mechanics are used (Fig. 20-44, *C* and *D*). The mechanics consist of 0.022-inch slot SPEED brackets (SPEED System Orthodontics, Ontario, Canada), buccal Orlus TADs (1.8 mm in diameter and 7.0 mm in length in the maxilla, 1.6 mm in diameter and 7.0 mm in length in the mandible) (Masel Orthodontics, Bristol, PA) and 0.017- × 0.022-inch SPEED stainless-steel wires. An average compensating curve (reverse curve of Spee) is placed in the wires to prevent mesial angulation of the molars during protraction. Furthermore, constriction bends are used to prevent arch widening.

Lever arms are placed on the molars to apply protraction forces near the center of resistance to prevent mesial tilting and unwanted intrusive forces that could lead to occlusal canting.

The second molars are included during full bonding, first to aid order rotation control of the first molars and then to maintain arch form.

The design of the mechanics is important, but the three-dimensional monitoring of the factors that contribute to the design is more critical. For example, if first-order rotation and arch form control are insufficient, then the mandibular second molar will buccally tilt and the buccal overjet will be shallow or a crossbite may develop. If a shallow overjet or crossbite results after the protraction force is reduced and the arch form is adjusted, then components that control first-order rotation and arch form, such as lingual attachments and lingual force vectors, can be added to the design of the mechanics.

Active treatment to achieve the desired results is completed after 24 months. Radiographic examination shows that all of the molars are vertically and anteroposteriorly well controlled (Fig. 20-44, *M–R*). Furthermore, although pneumatization of the maxillary sinuses is present, it does not present an obstacle in the protraction of the maxillary left molars.

Fixed retainers extending from first premolar to first premolar are used in the maxilla and mandible. In addition, a maxillary circumferential retainer is worn at night.

The results are well maintained 1 year after treatment.

CASE STUDY 20-1 Treatment of Congenitally Missing Teeth: Molar Protraction Using Asymmetric Mechanics—cont'd

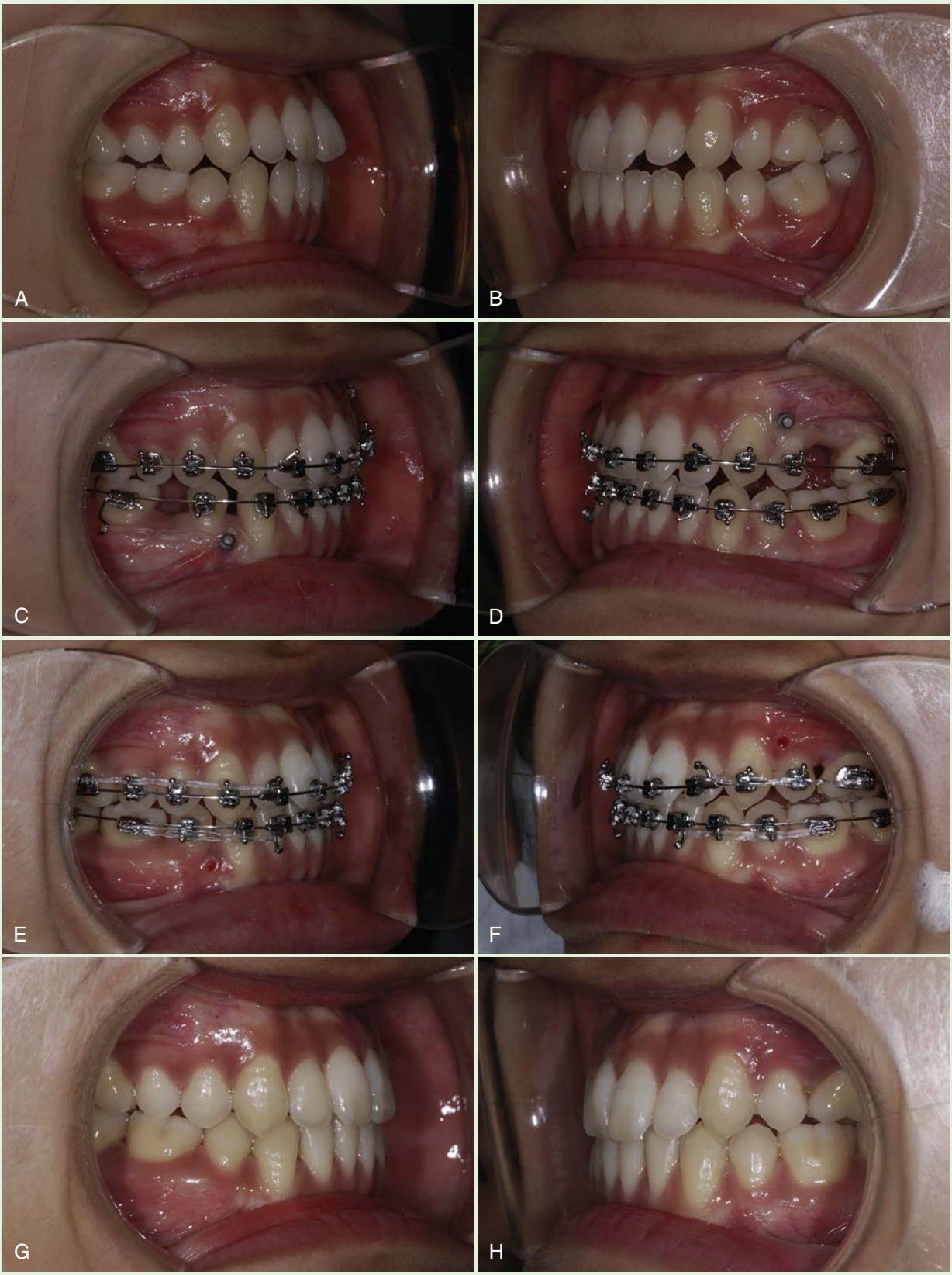


FIGURE 20-44 A, B, Intraoral views before treatment. C, D, Intraoral views during treatment. E, F, Intraoral views after molar protraction. G, H, Intraoral views at the completion of active treatment.

Continued

CASE STUDY 20-1 Treatment of Congenitally Missing Teeth: Molar Protraction Using Asymmetric Mechanics—cont'd

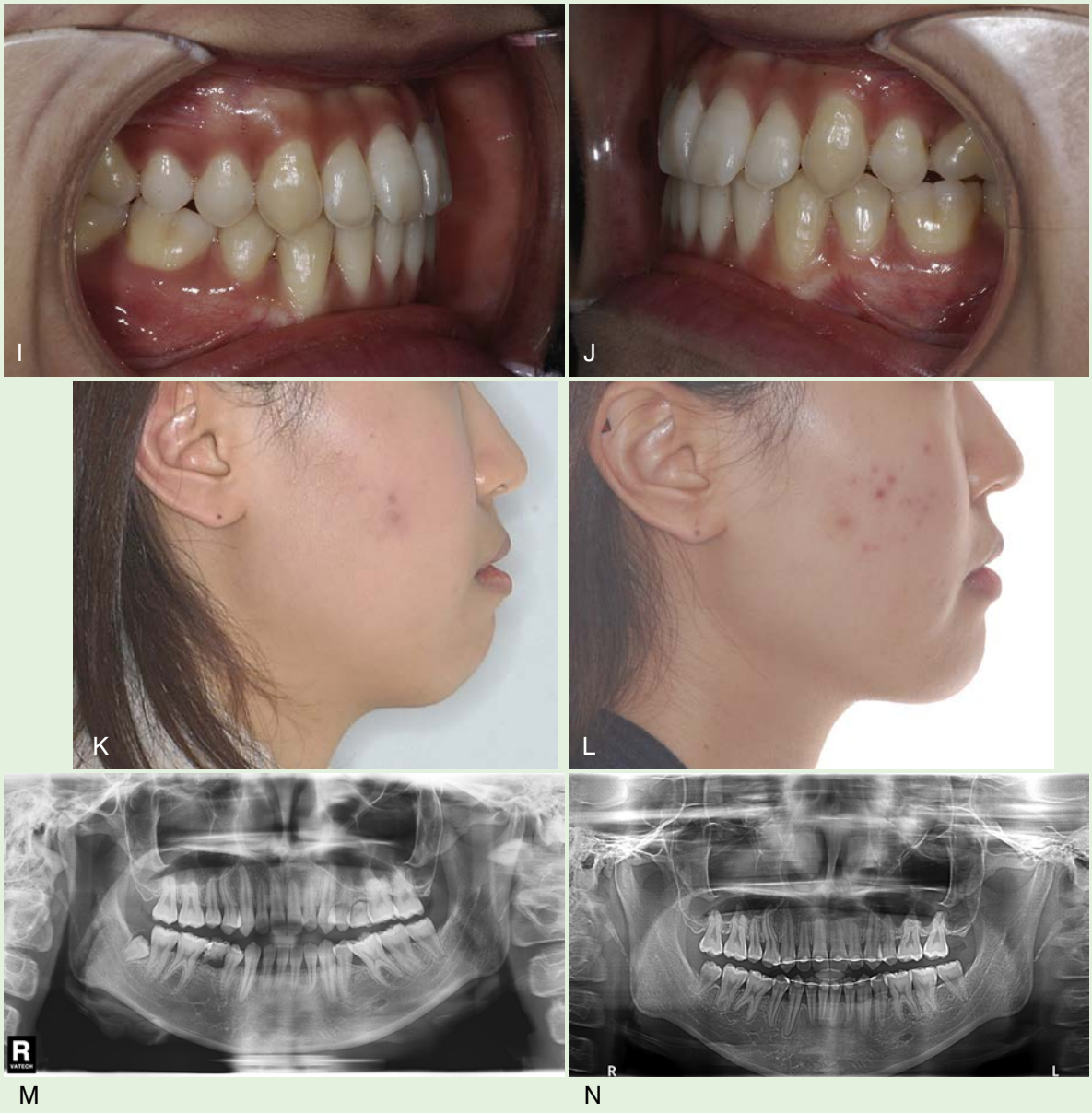
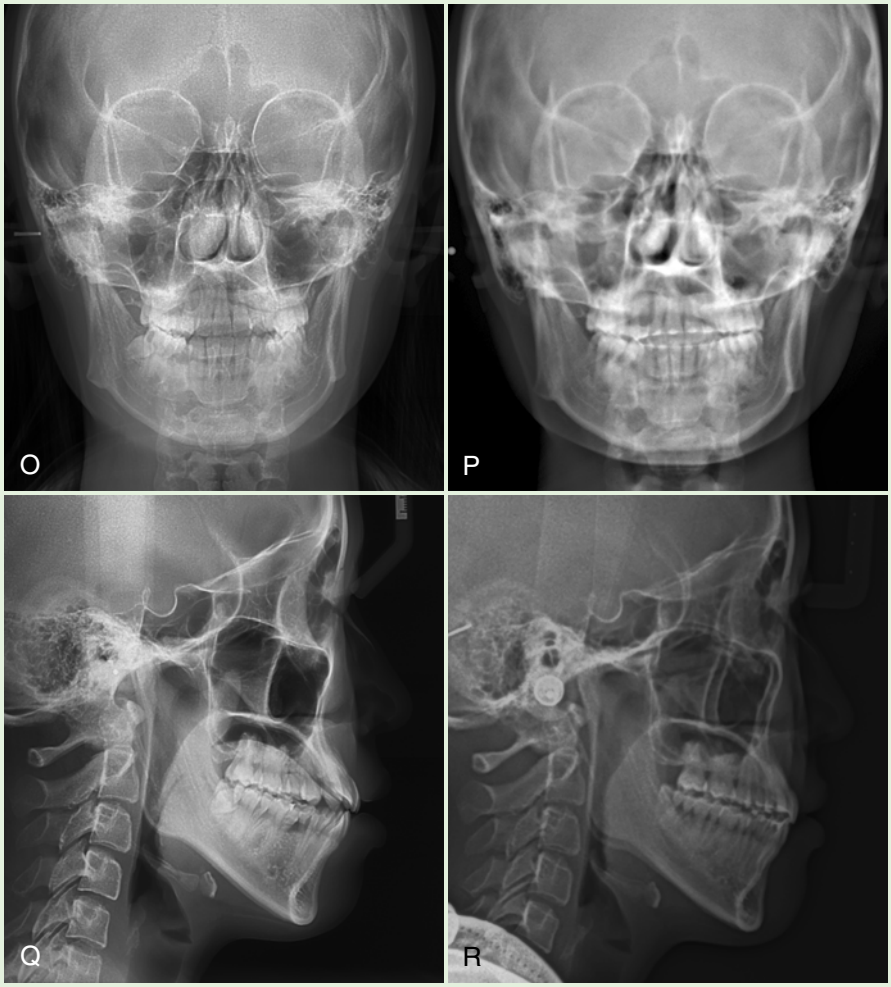


FIGURE 20-44, cont'd **I, J,** Intraoral views 10 months after the completion of active treatment. **K,** Profile view before treatment. **L,** Profile view at the completion of active treatment. **M,** Panoramic radiograph before treatment. **N,** Panoramic radiograph at the completion of active treatment.

CASE STUDY 20-1 Treatment of Congenitally Missing Teeth: Molar Protraction Using Asymmetric Mechanics—cont'd



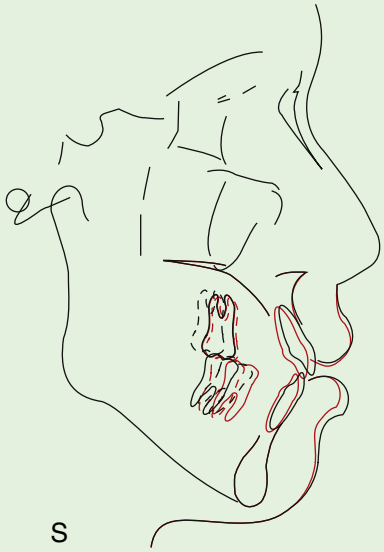


FIGURE 20-44, cont'd O, Posteroanterior cephalometric radiograph before treatment. P, Posteroanterior cephalometric radiograph at the completion of active treatment. Q, Cephalometric radiograph before treatment. R, Cephalometric radiograph at the completion of active treatment. S, Cephalometric superimpositions. The solid line refers to the mandibular right first molar, and the dotted line indicates the mandibular left first molar.

Continued

CASE STUDY 20-2 Nonsurgical Correction of Anterior Open Bite and Vertical Excess: Retraction and Intrusion of the Maxillary and Mandibular Dentitions

Jong Suk Lee

The patient is a 28-year-old woman whose chief complaints are lip incompetence and an anterior open bite. She exhibits the typical features of a long-faced patient: a long lower third of the face, lip incompetence, extreme mentalis strain upon lip closure, and a recessive chin. Cephalometric analysis confirms vertical excess with a flat occlusal plane. Surgical correction is an option, but the patient desires a nonsurgical approach. Therefore intrusion of the maxillary and mandibular dentitions with temporary anchorage devices (TADs) is planned to correct the anterior open bite and vertical excess and to improve the facial aesthetics.

First of all, the posterior segments are vertically controlled to establish an anterior occlusion for functional rehabilitation. For this purpose, the maxillary posterior teeth are intruded, but no orthodontic forces are applied to the maxillary anterior teeth at this time. Buccal and palatal Orlus TADs (1.8 mm in diameter and 8.0 mm in length) (Masel Orthodontics, Bristol, PA) are used to control the maxillary molars three dimensionally during intrusion. The mandibular teeth are vertically controlled with Orlus TADs (1.8 mm in diameter and 8.0 mm in length) to prevent extrusion (Fig. 20-45, *B* and *C*).

After an anterior occlusion is established, the anterior and posterior segments are vertically intruded further to improve the disharmony between the soft and hard tissues caused by vertical excess.

In an effort to distalize and intrude the entire dentition, continuous arch mechanics are used. The mechanics consist of continuous arch mechanics with 0.017- × 0.025-inch titanium molybdenum alloy (TMA) wires (Ormco Corporation, Orange, CA) with tip back bends to intrude the molars and to control occlusal plane inclination in addition to the TADs. In the maxilla, palatal TADs are used to increase treatment efficiency and to control arch form and occlusal plane inclination. In the mandible, a constriction bend is applied to control arch form.

The duration of active treatment is 16 months. The entire dentition is distalized and intruded. The cephalometric superimposition shows that the chin point has moved upward and forward (Fig. 20-45, *P*). The anterior facial height is decreased by 6 mm, and the mandible is rotated counterclockwise 4.3 degrees.

Fixed retainers extending from first premolar to first premolar are used in the maxilla and mandible. Functional rehabilitation, such as tongue function, is achieved, and instruction about the importance of controlling habits for temporomandibular joint (TMJ) health is thoroughly reviewed with the patient.

A maxillary circumferential retainer with tongue cribs is also worn at night to control tongue posture.

At the 3-year posttreatment follow-up, the results are well maintained.

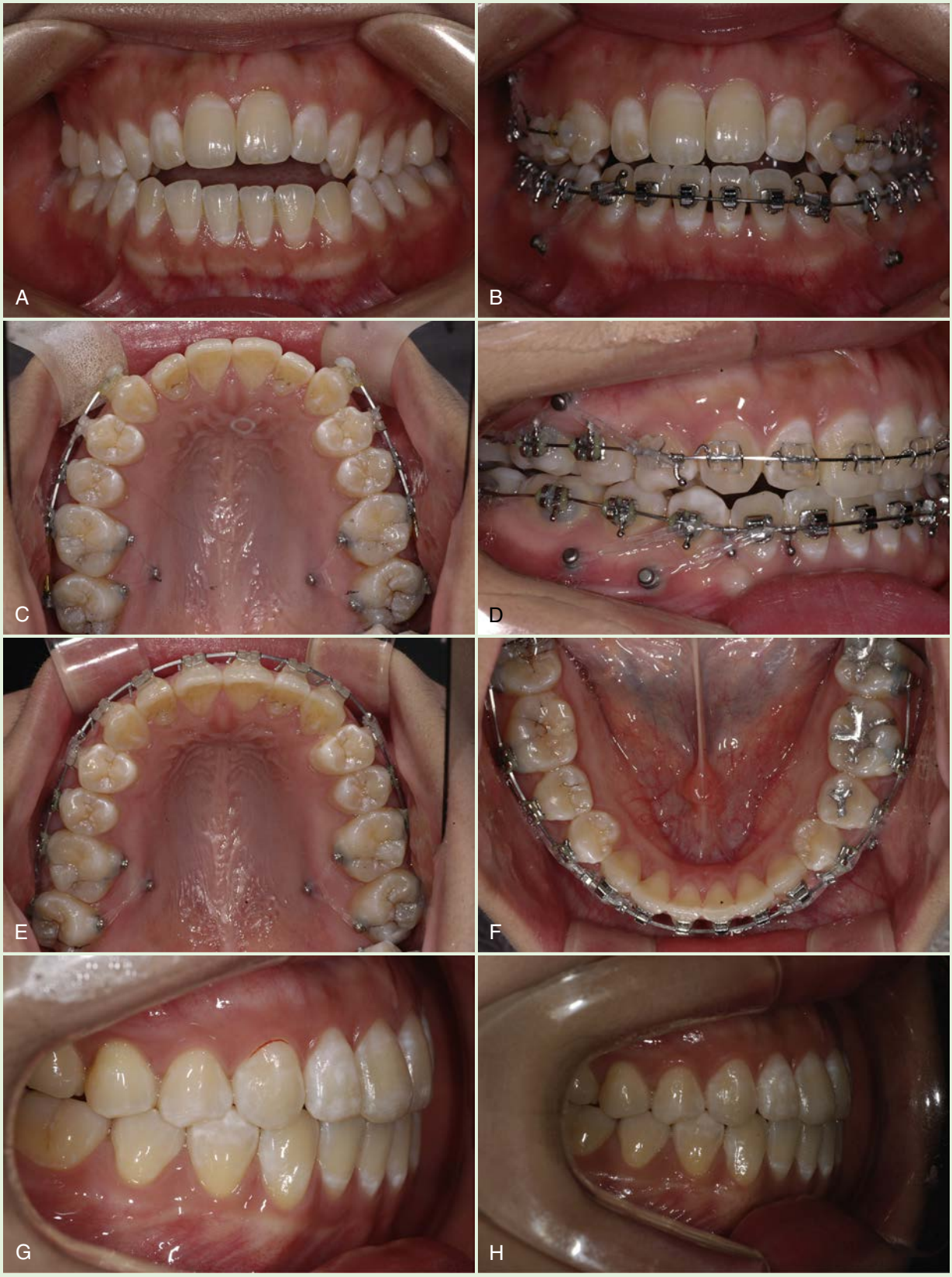


FIGURE 20-45 A, Intraoral views before treatment. B, C, Intraoral views, demonstrating maxillary molar intrusion mechanics. D–F, Intraoral views after 12 months of treatment, demonstrating the mechanics for intrusion of the entire dentition. G, Intraoral views at the completion of active treatment. H, Intraoral views 3 years after the completion of active treatment.

Continued

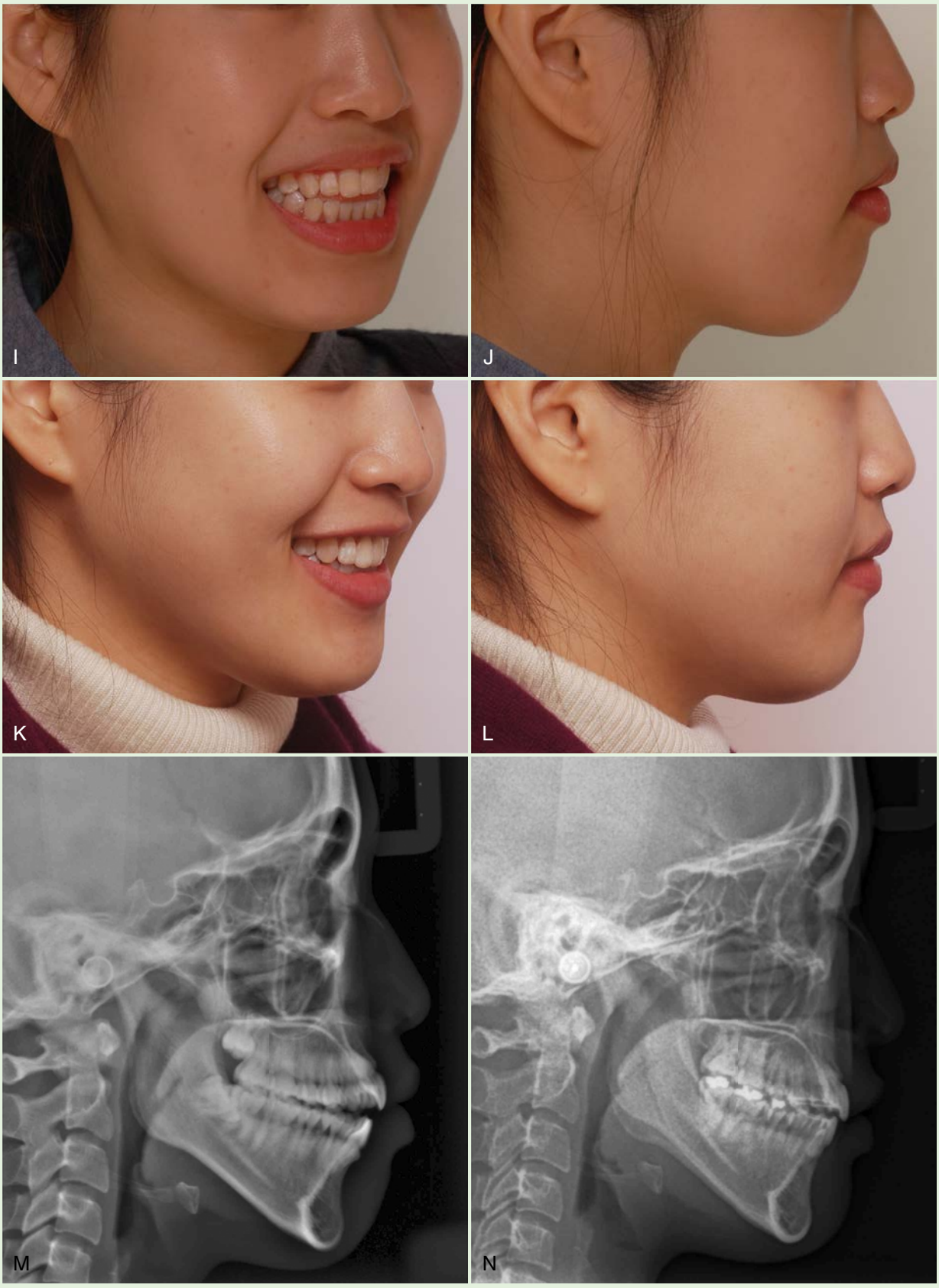
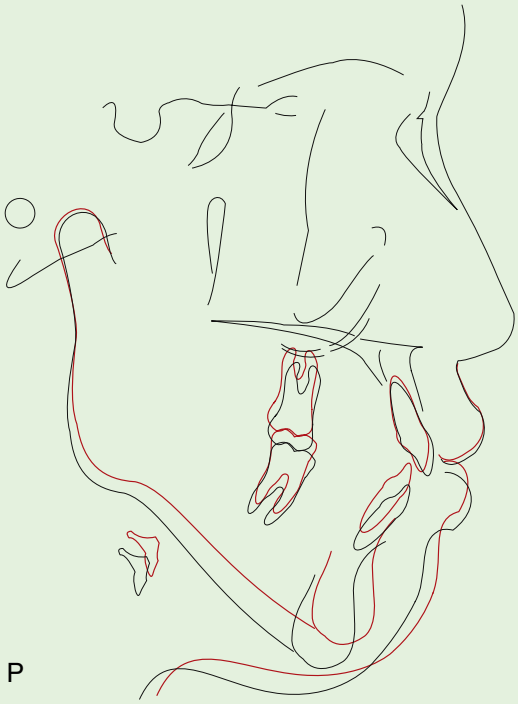


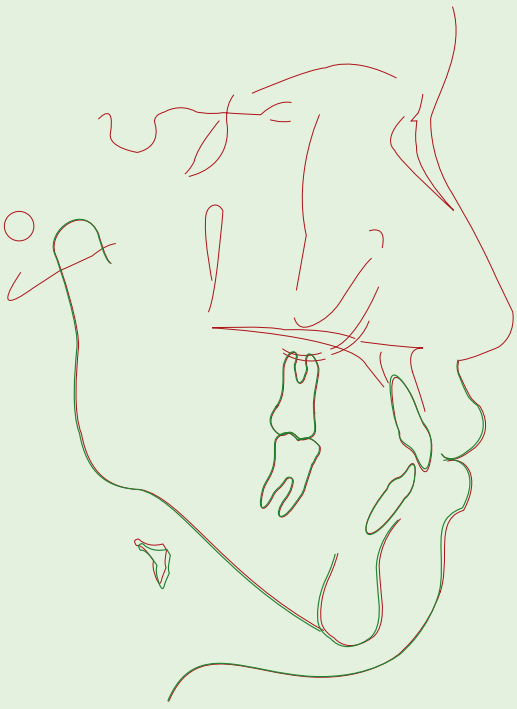
FIGURE 20-45, cont'd I, J, Facial views before treatment. K, L, Facial views at the completion of active treatment. M, Cephalometric radiograph before treatment. N, Cephalometric radiograph at the completion of active treatment.

CASE STUDY 20-2 Nonsurgical Correction of Anterior Open Bite and Vertical Excess: Retraction and Intrusion of the Maxillary and Mandibular Dentitions—cont'd

O



P



Q

FIGURE 20-45, cont'd O, Cephalometric radiograph 3 years after completion of active treatment. P, Superimposition of pretreatment and posttreatment cephalomatric radiographs. Q, Cephalometric superimposition after treatment and at 3-year retention follow-up.

Continued

CASE STUDY 20-3 Nonsurgical Correction of Vertical Excess: Retraction and Intrusion of Maxillary and Mandibular Dentition

Jong Suk Lee

The patient is a 28-year-old woman whose chief complaint is protrusion of the lips, even after orthodontic treatment with three premolar extractions. She exhibits an acceptable occlusion and a nice posed smile but has the typical features of a long-faced patient: long lower third of the face, lip incompetence, extreme mentalis strain upon lip closure, and a recessive chin. Cephalometric analysis confirms anterior vertical excess with a flat occlusal plane. Surgical correction may have been an option, but the patient desires a nonsurgical approach. Therefore maxillary and mandibular molar intrusion and distalization with orthodontic mini-implants are planned to correct the protrusion and vertical excess and to improve the facial aesthetics.

In an effort to distalize and intrude the entire dentition, continuous arch mechanics are used. To provide the proper space for the lower dentition, lower third molars are extracted. The mechanics consisted of buccal Orlus temporary anchorage devices (TADs) (1.8 mm in diameter and 7.0 mm in length in the maxilla, 1.6 mm in diameter and 7.0 mm in length in the mandible) (Masel Orthodontics, Bristol, PA) and 0.017- × 0.025-inch titanium molybdenum alloy (TMA) wire (Ormco Corporation, Orange, CA) with a tip back bend to intrude the molars and control occlusal plane inclination. A constriction bend is also applied to control arch form (Fig. 20-46, *G-I*). Palatal or lingual TADs are not used.

Active treatment to the desired position is completed after 20 months. The entire dentition is distalized and intruded with only the use of buccal

FIGURE 20-46 **A–C,** Facial views before treatment. **D–F,** Intraoral views before treatment.

CASE STUDY 20-3 Nonsurgical Correction of Vertical Excess: Retraction and Intrusion of Maxillary and Mandibular Dentition—cont'd

TADs. The cephalometric superimposition shows that the upper and lower anteriors are retracted and that the chin point has moved upward and forward (Fig. 20-46, *W*).

Fixed retainers extending from first premolar to first premolar are used in the maxilla and mandible. A maxillary circumferential retainer is also worn at night.

At 18 months' posttreatment follow-up, the results are well maintained (Fig. 20-46, *P–R*).

FIGURE 20-46, cont'd G–I, Intraoral views during treatment. J–L, Facial views at the completion of active treatment.

Continued

CASE STUDY 20-3 Nonsurgical Correction of Vertical Excess: Retraction and Intrusion of Maxillary and Mandibular Dentition—cont'd

FIGURE 20-46, cont'd M–O, Intraoral views at the completion of active treatment. P–R, Intraoral views 18 months after the completion of active treatment.

CASE STUDY 20-3 Nonsurgical Correction of Vertical Excess: Retraction and Intrusion of Maxillary and Mandibular Dentition—cont'd

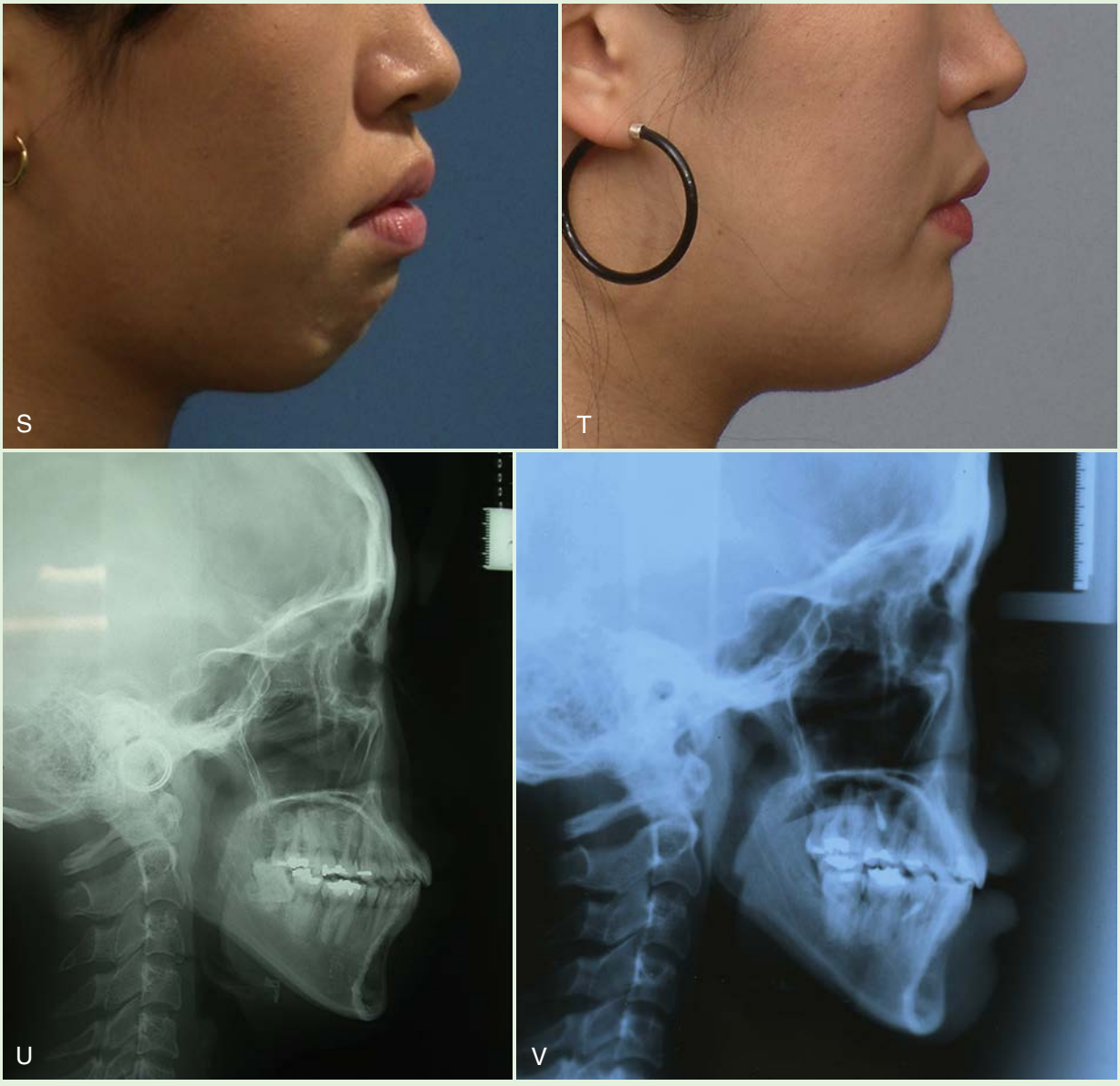


FIGURE 20-46, cont'd S, Profile view before treatment. T, Profile view at the completion of active treatment. U, Cephalometric radiograph before treatment. V, Cephalometric radiograph at the completion of active treatment.

Continued

CASE STUDY 20-3 Nonsurgical Correction of Vertical Excess: Retraction and Intrusion of Maxillary and Mandibular Dentition—cont'd

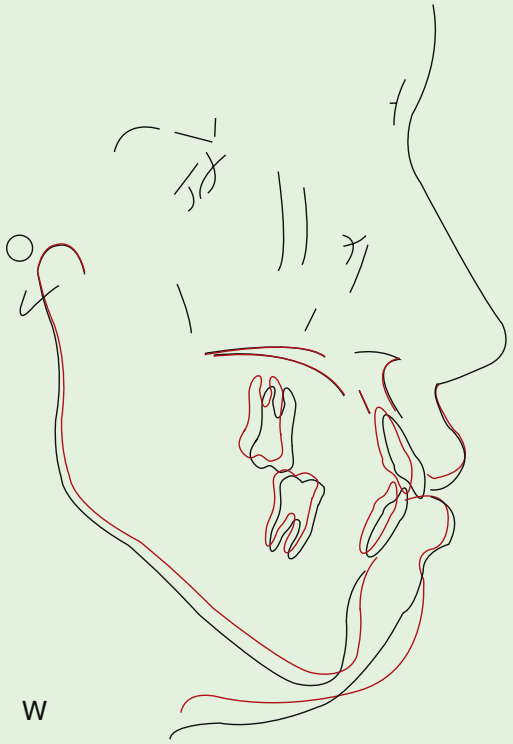


FIGURE 20-46, cont'd W, Cephalometric superimposition.

CASE STUDY 20-4 Correction of Occlusal Cant and Midline: Maxillary Molar Intrusion with Mandibular Molar Extrusion

Young-Chel Park

The patient is a 22-year-old man whose chief complaint is "my orthodontic treatment was finished 2 months ago, but my chin has slanted to one side after treatment."

His presentation includes temporomandibular joint (TMJ) symptoms of intermittent pain and clicking on the left TMJ, and the upper left first premolar has been extracted. Clinically, the patient's maxillary dentition is canted downward on the right side. The patient also exhibits upper and lower midline deviations of approximately 2 mm to the left from the facial midline at maximum intercuspation (Fig. 20-47, *F*). A significant lateral shift is noted from centric relation to maximum intercuspation, and the lower dental midline coincides with the facial midline in centric relation (Fig. 20-47, *I*). Facial examination reveals that the chin deviates to the left with canted lips at maximum intercuspation, but this deviation is alleviated once the mandible is in the centric relation position.

Radiographic examination confirms that the mandibular asymmetry is due to a significant lateral shift from centric relation to maximum intercuspation. The upper right first molar is 3.3 mm lower than the upper left first molar (Fig. 20-47, *K*).

There are two possible options to correct such canting. The first is surgical correction of the maxilla, and the second is nonsurgical correction using temporary anchorage devices (TADs) to intrude the maxillary molars. The patient opts for the nonsurgical treatment plan.

The treatment plan calls for intrusion and distalization of the upper right molars to correct maxillary canting, as well as the midline deviation to guide the mandible to the centric relation position.

Five TADs, 1.8 mm in diameter and 7.0 mm in length, are placed in the buccal and palatal areas (Fig. 20-47, *M-P*).

After 10 months of treatment, the upper right molars are intruded and distalized. The canting of the occlusal plane is improved, and the mandible is also guided into centric relation (Fig. 20-47, *Q* and *R*).

After 12 months of treatment, one Orlus TAD, 1.6 mm in diameter and 7.0 mm in length (Masel Orthodontics, Bristol, PA), is placed on the right buccal slope of the mandible. Then an extrusion spring, made of 0.016- × 0.022-inch titanium molybdenum alloy (TMA) wire (Ormco Corporation, Orange, CA), is positioned to extrude the lower right posterior teeth. The spring is connected and bonded to the TADs and is then applied to the bracket bases to produce an extrusive force (Fig. 20-47, *S* and *T*).

At 18 months after the start of treatment, the appliances are removed and fixed retainers are used (Fig. 20-48, *A–F*). Additionally, an active retainer using TADs in the maxillary arch is worn at night (Fig. 20-48, *H–J*).

The occlusal and mandibular planes have rotated 6.2 degrees and 7.5 degrees, respectively.

Menton, therefore, has moved 6.4 mm to the right, and the facial asymmetry has improved (Fig. 20-48, *G*).

At a follow-up examination 27 months after the end of treatment, the results are well maintained (Fig. 20-48, *K–M*).

Continued

CASE STUDY 20-4 Correction of Occlusal Cant and Midline: Maxillary Molar Intrusion with Mandibular Molar Extrusion—cont'd

FIGURE 20-47 A–C, Extraoral views before treatment. D, The patient exhibits occlusal plane canting with a tongue blade. E–G, Intraoral views before treatment in centric occlusion.

CASE STUDY 20-4 Correction of Occlusal Cant and Midline: Maxillary Molar Intrusion with Mandibular Molar Extrusion—cont'd

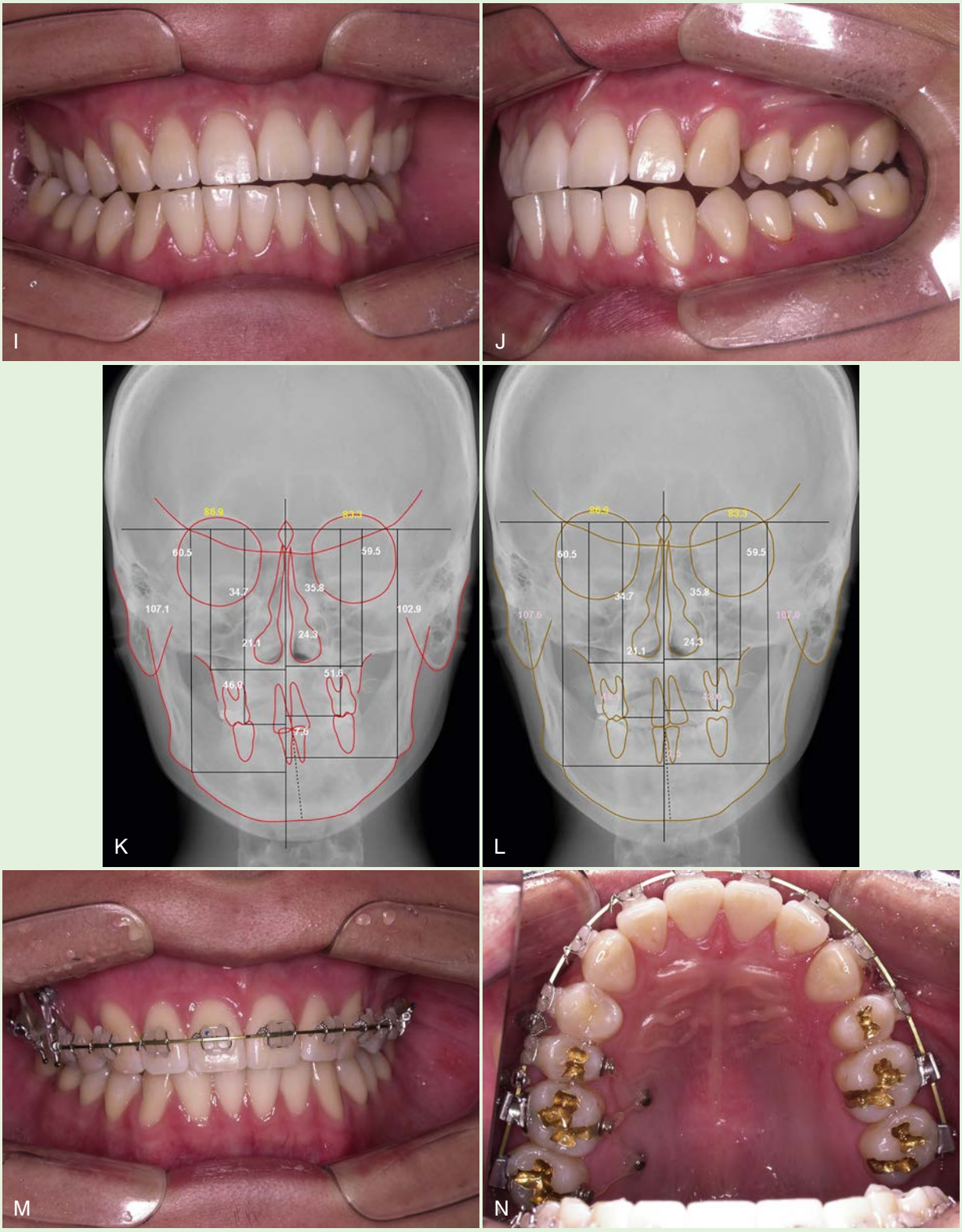


FIGURE 20-47, cont'd H–J, Intraoral views before treatment in centric relation. K, Posteroanterior cephalometric radiograph in centric occlusion. L, Posteroanterior cephalometric radiograph in centric relation.

Continued

CASE STUDY 20-4 Correction of Occlusal Cant and Midline: Maxillary Molar Intrusion with Mandibular Molar Extrusion—cont'd

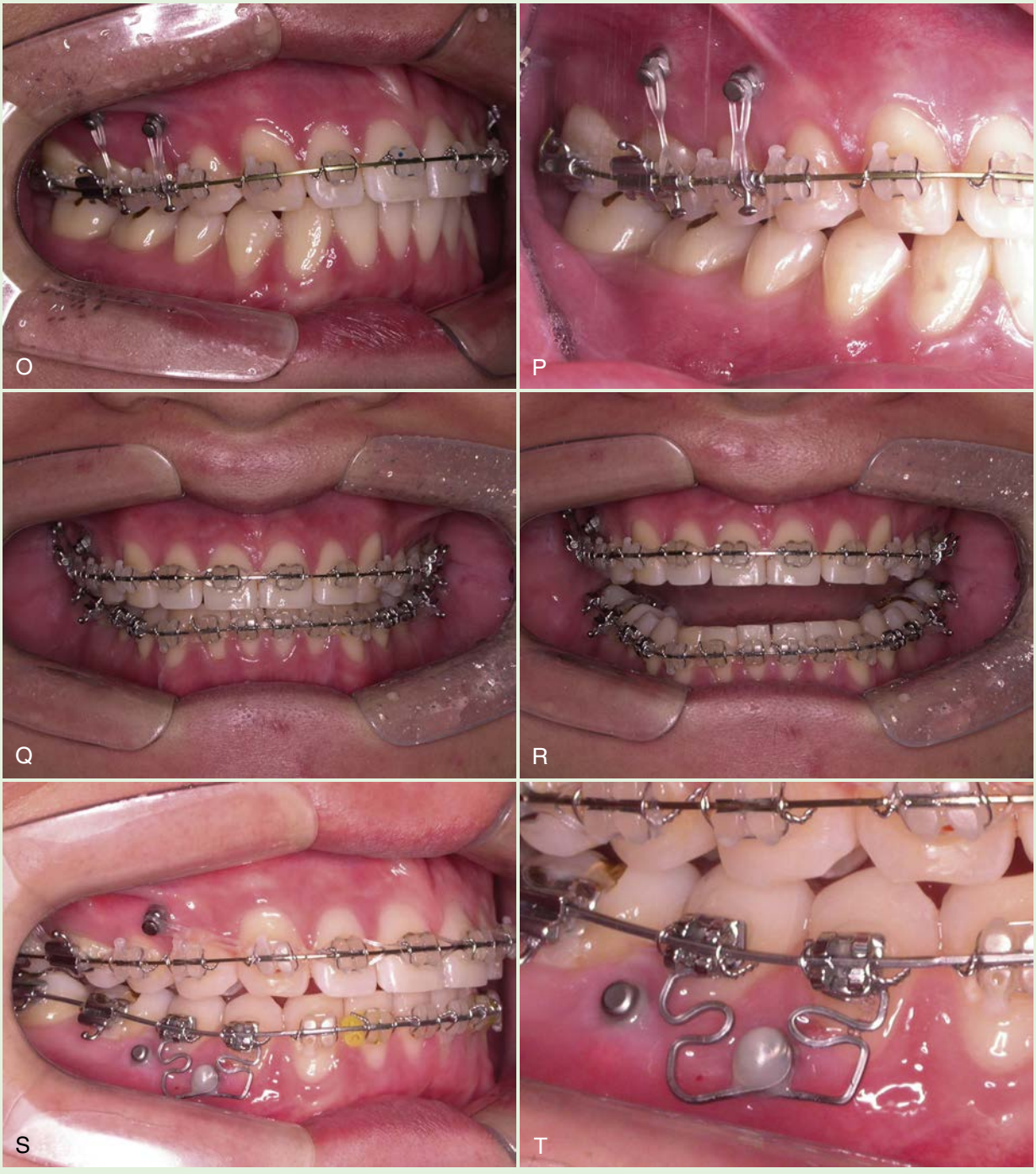


FIGURE 20-47, cont'd M–P, During treatment, the maxillary working archwire and temporary anchorage devices (TADs) are placed, and intrusion of the upper right molars is initiated. Q, R, After 12 months of treatment, the right maxillary molars are intruded and the differential interocclusal space is shown. S, T, Intraoral views after 12 months of treatment, demonstrating mandibular TADs and extrusion spring.

CASE STUDY 20-4 Correction of Occlusal Cant and Midline: Maxillary Molar Intrusion with Mandibular Molar Extrusion—cont'd

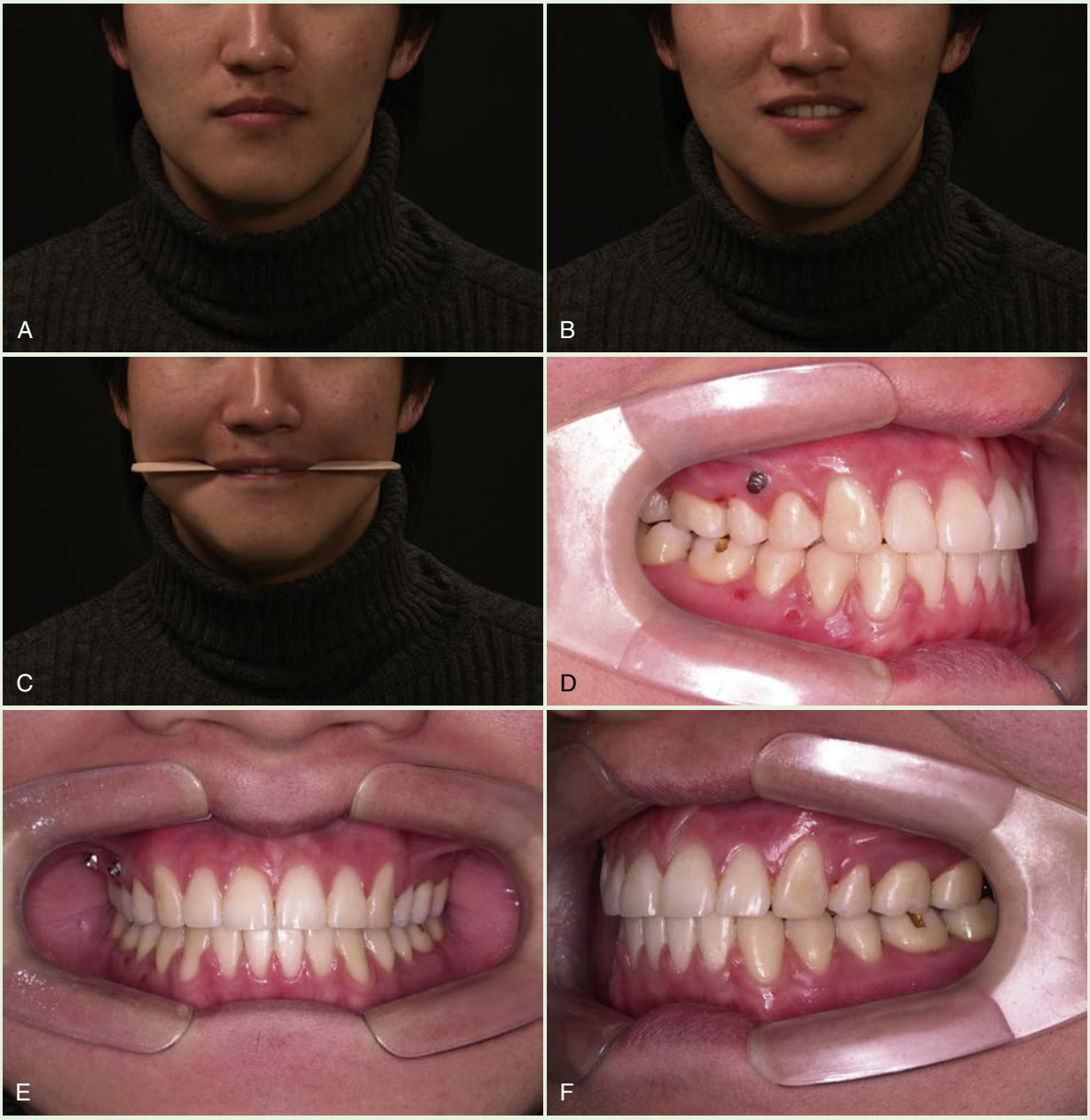


FIGURE 20-48 A, B, Extraoral views at the completion of treatment. C, A flat occlusal plane is shown with a tongue blade. D–F, Intraoral views at the completion of treatment.

Continued

CASE STUDY 20-4 Correction of Occlusal Cant and Midline: Maxillary Molar Intrusion with Mandibular Molar Extrusion—cont'd

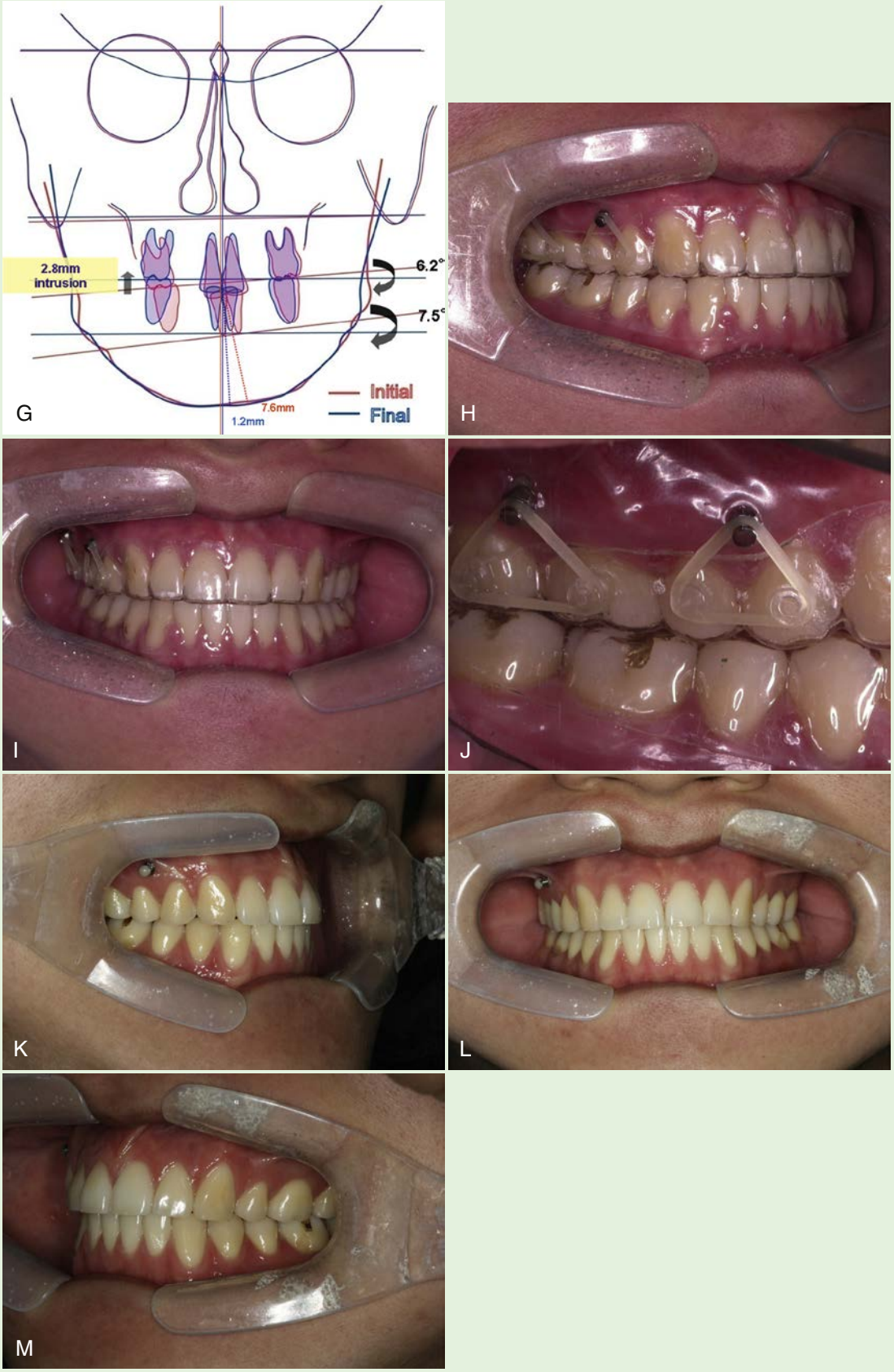


FIGURE 20-48, cont'd G, Superimposition of posteroanterior cephalometric radiographs. H–J, Intraoral views with active retainers. K–M, Intraoral views at 27 months' retention.

REFERENCES

1. Hsieh YD, Su CM, Yang YH, et al. Evaluation on the movement of endosseous titanium implants under continuous orthodontic forces: an experimental study in the dog. *Clin Oral Implants Res*. 2008;19:618–623.
2. Roberts WE. Bone dynamics of osseointegration, ankylosis, and tooth movement. *J Indiana Dent Assoc*. 1999;78:24–32.
3. Melsen B. Limitations in adult orthodontics. In: Melsen B, ed. *Current Controversies in Orthodontics*. Chicago: Quintessence Publishing; 1991:147–180.
4. Janssen KI, Raghoebar GM, Vissink A, Sandham A. Skeletal anchorage in orthodontics—a review of various systems in animal and human studies. *Int J Oral Maxillofac Implants*. 2008;23:75–88.
5. Wehrbein H, Göllner P. Skeletal anchorage in orthodontics—basics and clinical application. *J Orofac Orthop*. 2007;68:443–461.
6. Lee JS, Kim JK, Park YC, Vanarsdall Jr RL. *Applications of Orthodontic Mini-Implants*. Chicago: Quintessence Publishing; 2007.
7. Chung KR, Kim SH, Choo H, et al. Distalization of the mandibular dentition with mini-implants to correct a Class III malocclusion with a midline deviation. *Am J Orthod Dentofacial Orthop*. 2010;137:135–146.
8. Jeon YJ, Kim YH, Son WS, Hans MG. Correction of a canted occlusal plane with miniscrews in a patient with facial asymmetry. *Am J Orthod Dentofacial Orthop*. 2006;130:244–252.
9. Ko DI, Lim SH, Kim KW. Treatment of occlusal plane canting using miniscrew anchorage. *World J Orthod*. 2006;7:269–278.
10. Takano-Yamamoto T, Kuroda S. Titanium screw anchorage for correction of canted occlusal plane in patients with facial asymmetry. *Am J Orthod Dentofacial Orthop*. 2007;132:237–242.
11. Deguchi T, Murakami T, Kuroda S, et al. Comparison of the intrusion effects on the maxillary incisors between implant anchorage and J-hook headgear. *Am J Orthod Dentofacial Orthop*. 2008;133:654–660.
12. Feldmann I, Bondemark L. Anchorage capacity of osseointegrated and conventional anchorage systems: a randomized controlled trial. *Am J Orthod Dentofacial Orthop*. 2008;133:339.e19–339.e28.
13. Kaya B, Arman A, Uçkan S, Yazici AC. Comparison of the zygoma anchorage system with cervical headgear in buccal segment distalization. *Eur J Orthod*. 2009;31:417–424.
14. Kuroda S, Yamada K, Deguchi T, et al. Class II malocclusion treated with miniscrew anchorage: comparison with traditional orthodontic mechanics outcomes. *Am J Orthod Dentofacial Orthop*. 2009;135:302–309.
15. Lai EH, Yao CC, Chang JZ, et al. Three-dimensional dental model analysis of treatment outcomes for protrusive maxillary dentition: comparison of headgear, miniscrew, and miniplate skeletal anchorage. *Am J Orthod Dentofacial Orthop*. 2008;134:636–645.
16. Park HS, Yoon DY, Park CS, Jeoung SH. Treatment effects and anchorage potential of sliding mechanics with titanium screws compared with the Tweed-Merrifield technique. *Am J Orthod Dentofacial Orthop*. 2008;133:593–600.
17. Polat-Özsoy Ö, Arman-Özçırpıcı A, Veziroğlu F, Çetinşahin A. Comparison of the intrusive effects of miniscrews and utility arches. *Am J Orthod Dentofacial Orthop*. 2011;139:526–532.
18. Polat-Ozsoy O, Kircelli BH, Arman-Ozçirpici A, et al. Pendulum appliances with 2 anchorage designs: conventional anchorage vs bone anchorage. *Am J Orthod Dentofacial Orthop*. 2008;133:e9–339.e17.
19. Senışık NE, Türkkahraman H. Treatment effects of intrusion arches and mini-implant systems in deepbite patients. *Am J Orthod Dentofacial Orthop*. 2012;141:723–733.
20. Upadhyay M, Yadav S, Patil S. Mini-implant anchorage for en-masse retraction of maxillary anterior teeth: a clinical cephalometric study. *Am J Orthod Dentofacial Orthop*. 2008;134:803–810.
21. Yao CC, Lai EH, Chang JZ, et al. Comparison of treatment outcomes between skeletal anchorage and extraoral anchorage in adults with maxillary dentoalveolar protrusion. *Am J Orthod Dentofacial Orthop*. 2008;134:615–624.
22. Cornelis MA, De Clerck HJ. Maxillary molar distalization with miniplates assessed on digital models: a prospective clinical trial. *Am J Orthod Dentofacial Orthop*. 2007;132:373–377.
23. Escobar SA, Tellez PA, Moncada CA, et al. Distalization of maxillary molars with the bone-supported pendulum: a clinical study. *Am J Orthod Dentofacial Orthop*. 2007;131:545–549.
24. Fudalej P, Antoszewska J. Are orthodontic distalizers reinforced with the temporary skeletal anchorage devices effective? *Am J Orthod Dentofacial Orthop*. 2011;139:722–729.
25. Gelgor IE, Karaman AI, Buyukyilmaz T. Comparison of 2 distalization systems supported by intraosseous screws. *Am J Orthod Dentofacial Orthop*. 2007;131:e1–e8.
26. Oh YH, Park HS, Kwon TG. Treatment effects of microimplant-aided sliding mechanics on distal retraction of posterior teeth. *Am J Orthod Dentofacial Orthop*. 2011;139:470–481.
27. Onçağ G, Seçkin O, Dinçer B, Arikan F. Osseointegrated implants with pendulum springs for maxillary molar distalization: a cephalometric study. *Am J Orthod Dentofacial Orthop*. 2007;131:16–26.
28. Sugawara J, Daimaruya T, Umemori M, et al. Distal movement of mandibular molars in adult patients with the skeletal anchorage system. *Am J Orthod Dentofacial Orthop*. 2004;125:130–138.
29. Sugawara J, Kanzaki R, Takahashi I, et al. Distal movement of maxillary molars in nongrowing patients with the skeletal anchorage system. *Am J Orthod Dentofacial Orthop*. 2006;129:723–733.
30. Yamada K, Kuroda S, Deguchi T, Takano-Yamamoto T. Distal movement of maxillary molars using miniscrew anchorage in the buccal interradicular region. *Angle Orthod*. 2009;79:78–84.
31. Akan S, Kocadereli I, Aktas A, Taşar F. Effects of maxillary molar intrusion with zygomatic anchorage on the stomatognathic system in anterior open bite patients. *Eur J Orthod*. 2013;35:93–102.
32. Deguchi T, Kurosaka H, Oikawa H, et al. Comparison of orthodontic treatment outcomes in adults with skeletal open bite between conventional edgewise treatment and implant-anchored orthodontics. *Am J Orthod Dentofacial Orthop*. 2011;139(4 Suppl):S60–S68.
33. Erverdi N, Keles A, Nanda R. The use of skeletal anchorage in open bite treatment: a cephalometric evaluation. *Angle Orthod*. 2004; 74:381–390.
34. Hashimoto T, Fukunaga T, Kuroda S, et al. Mandibular deviation and canted maxillary occlusal plane treated with miniscrews and intraoral vertical ramus osteotomy: functional and morphologic changes. *Am Orthod Dentofacial Orthop*. 2009;136:868–877.
35. Heravi F, Bayani S, Madani AS, et al. Intrusion of supra-erupted molars using miniscrews: clinical success and root resorption. *Am J Orthod Dentofacial Orthop*. 2011;139(4 Suppl):S170–S175.
36. Kaku M, Koseki H, Kawazoe A, et al. Treatment of a case of skeletal class II malocclusion with temporomandibular joint disorder using miniscrew anchorage. *Cranio*. 2011;29:155–163.
37. Komori R, Deguchi T, Tomizuka R, Takano-Yamamodo T. The use of miniscrew as orthodontic anchorage in correction of maxillary protrusion with occlusal cant, spaced arch, and midline deviation without surgery. *Orthodontics (Chic.)*. 2013;14:e156–e167.
38. Kuroda S, Katayama A, Takano-Yamamoto T. Severe anterior open-bite case treated using titanium screw anchorage. *Angle Orthod*. 2004;74: 558–567.
39. Kuroda S, Sugawara Y, Tamamura N, Takano-Yamamoto T. Anterior open bite with temporomandibular disorder treated with titanium screw anchorage: evaluation of morphological and functional improvement. *Am J Orthod Dentofacial Orthop*. 2007;131:550–560.
40. Park HS, Kim JY, Kwon TG. Occlusal plane change after intrusion of maxillary posterior teeth by microimplants to avoid maxillary surgery with skeletal Class III orthognathic surgery. *Am J Orthod Dentofacial Orthop*. 2010;138:631–640.
41. Lee JS, Kim DH, Park YC, et al. The efficient use of midpalatal miniscrew implants. *Angle Orthod*. 2004;74:711–714.
42. Ma J, Wang L, Zhang W, et al. Comparative evaluation of micro-implant and headgear anchorage used with a pre-adjusted appliance system. *Eur J Orthod*. 2008;30:283–287.
43. Sherwood KH, Burch JG, Thompson WJ. Closing anterior open bites by intruding molars with titanium miniplate anchorage. *Am J Orthod Dentofacial Orthop*. 2002;122:593–600.

44. Sugawara J, Baik UB, Umemori M, et al. Treatment and posttreatment dentoalveolar changes following intrusion of mandibular molars with application of a skeletal anchorage system (SAS) for open bite correction. *Int J Adult Orthodon Orthognath Surg*. 2002;17:243–253.
45. Xun C, Zeng X, Wang X. Microscrew anchorage in skeletal anterior open-bite treatment. *Angle Orthod*. 2007;77:47–56.
46. Yao CC, Lee JJ, Chen HY, et al. Maxillary molar intrusion with fixed appliances and mini-implant anchorage studied in three dimensions. *Angle Orthod*. 2005;75:754–760.
47. Byun ES, Ahn SJ, Kim TW. Relationship between internal derangement of the temporomandibular joint and dentofacial morphology in women with anterior open bite. *Am J Orthod Dentofacial Orthop*. 2005;128:87–95.
48. Hur YK, Ko MY, Ahn YW. The relationship between anterior disc displacement without reduction and development of anterior openbite. *Korean J Oral Medicine*. 2007;32:293–303.
49. Chen Y, Hong L, Wang CL, et al. Effect of large incisor retraction on upper airway morphology in adult bimaxillary protrusion patients. *Angle Orthod*. 2012;82:964–970.
50. Guo QY, Zhang SJ, Liu H, et al. Three-dimensional evaluation of upper anterior alveolar bone dehiscence after incisor retraction and intrusion in adult patients with bimaxillary protrusion malocclusion. *J Zhejiang Univ Sci B*. 2011;12:990–997.
51. Liou EJ, Chang PM. Apical root resorption in orthodontic patients with en-masse maxillary anterior retraction and intrusion with miniscrews. *Am J Orthod Dentofacial Orthop*. 2010;137:207–212.
52. Sarikaya S, Haydar B, Ciğer S, Ariyürek M. Changes in alveolar bone thickness due to retraction of anterior teeth. *Am J Orthod Dentofacial Orthop*. 2002;122:15–26.
53. Kim YH, Yang SM, Kim S, et al. Midpalatal miniscrews for orthodontic anchorage: factors affecting clinical success. *Am J Orthod Dentofacial Orthop*. 2010;137:66–72.
54. Lim HJ, Choi YJ, Evans CA, Hwang HS. Predictors of initial stability of orthodontic miniscrew implants. *Eur J Orthod*. 2011;33:528–532.
55. Chen YJ, Chang HH, Huang CY, et al. A retrospective analysis of the failure rate of three different orthodontic skeletal anchorage systems. *Clin Oral Implants Res*. 2007;18:768–775.
56. Chen YJ, Chang HH, Lin HY, et al. Stability of miniplates and miniscrews used for orthodontic anchorage: experience with 492 temporary anchorage devices. *Clin Oral Implants Res*. 2008;19:1188–1196.
57. Cornelis MA, Scheffler NR, Mahy P, et al. Modified miniplates for temporary skeletal anchorage in orthodontics: placement and removal surgeries. *J Oral Maxillofac Surg*. 2008;66:1439–1445.
58. Kuroda S, Sugawara Y, Deguchi T, et al. Clinical use of miniscrew implants as orthodontic anchorage: success rates and postoperative discomfort. *Am J Orthod Dentofacial Orthop*. 2007;131:9–15.
59. Männchen R, Schätzle M. Success rate of palatal orthodontic implants: a prospective longitudinal study. *Clin Oral Implants Res*. 2008;19:665–669.
60. Moon CH, Lee DG, Lee HS, et al. Factors associated with the success rate of orthodontic miniscrews placed in the upper and lower posterior buccal region. *Angle Orthod*. 2008;78:101–106.
61. Papageorgiou SN, Zogakis IP, Papadopoulos MA. Failure rates and associated risk factors of orthodontic miniscrew implants: a meta-analysis. *Am J Orthod Dentofacial Orthop*. 2012;142:577–595.e7.
62. Park HS, Jeong SH, Kwon OW. Factors affecting the clinical success of screw implants used as orthodontic anchorage. *Am J Orthod Dentofacial Orthop*. 2006;130:18–25.
63. Schätzle M, Männchen R, Zwahlen M, Lang NP. Survival and failure rates of orthodontic temporary anchorage devices: a systematic review. *Clin Oral Implants Res*. 2009;20:1351–1359.
64. Watanabe H, Deguchi T, Hasegawa M, et al. Orthodontic miniscrew failure rate and root proximity, insertion angle, bone contact length, and bone density. *Orthod Craniofac Res*. 2013;16:44–55.
65. Hyde JD, King GJ, Greenlee GM, et al. Survey of orthodontists' attitudes and experiences regarding miniscrew implants. *J Clin Orthod*. 2010;44:481–486.
66. Berglundh T, Abrahamsson I, Lang NP, Lindhe J. De novo alveolar bone formation adjacent to endosseous implants. *Clin Oral Implants Res*. 2003;14:251–262.
67. Campbell TM, Wong WT, Mackie EJ. Establishment of a model of cortical bone repair in mice. *Calcif Tissue Int*. 2003;73:49–55.
68. Tanaka M, Sakai A, Uchida S, et al. Prostaglandin E2 receptor (EP4) selective agonist (ONO-4819.CD) accelerates bone repair of femoral cortex after drill-hole injury associated with local upregulation of bone turnover in mature rats. *Bone*. 2004;34:940–948.
69. Baumgaertel S. Quantitative investigation of palatal bone depth and cortical bone thickness for mini-implant placement in adults. *Am J Orthod Dentofacial Orthop*. 2009;136:104–108.
70. Kim SH, Yoon HG, Choi YS, et al. Evaluation of interdental space of the maxillary posterior area for orthodontic mini-implants with cone-beam computed tomography. *Am J Orthod Dentofacial Orthop*. 2009;135:635–641.
71. Laursen MG, Melsen B, Cattaneo PM. An evaluation of insertion sites for mini-implants: a micro-CT study of human autopsy material. *Angle Orthod*. 2013;83:222–229.
72. Lee KJ, Joo E, Kim KD, et al. Computed tomographic analysis of tooth-bearing alveolar bone for orthodontic miniscrew placement. *Am J Orthod Dentofacial Orthop*. 2009;135:486–494.
73. Shinohara A, Motoyoshi M, Uchida Y, Shimizu N. Root proximity and inclination of orthodontic mini-implants after placement: cone-beam computed tomography evaluation. *Am J Orthod Dentofacial Orthop*. 2013;144:50–56.
74. Silvestrini Biavati A, Tecco S, Migliorati M, et al. Three-dimensional tomographic mapping related to primary stability and structural miniscrew characteristics. *Orthod Craniofac Res*. 2011;14:88–99.
75. Chung KR, Choo H, Kim SH, Ngan P. Timely relocation of mini-implants for uninterrupted full-arch distalization. *Am J Orthod Dentofacial Orthop*. 2010;138:839–849.
76. Doshi UH, Jamwal RS, Bhad WA. Distalization of molars using two stage mini-implants—a case report. *J Orthod*. 2011;38:55–63.
77. Moroni A, Toksvig-Larsen S, Maltarello MC, et al. A comparison of hydroxyapatite coated, titanium coated and uncoated tapered external fixation pins. An in vivo study in sheep. *J Bone Joint Surg Am*. 1998;80:547–554.
78. Schätzle M, Männchen R, Balbach U, et al. Stability change of chemically modified sandblasted/acid-etched titanium palatal implants. A randomized-controlled clinical trial. *Clin Oral Implants Res*. 2009;20:489–495.
79. Skripitz R, Aspenberg P. Tensile bond between bone and titanium: a reappraisal of osseointegration. *Acta Orthop Scand*. 1998;69:315–319.
80. Leung MT, Rabie AB, Wong RW. Stability of connected mini-implants and miniplates for skeletal anchorage in orthodontics. *Eur J Orthod*. 2008;30:483–489.
81. Holberg C, Winterhalder P, Holberg N. Direct versus indirect loading of orthodontic miniscrew implants—an FEM analysis. *Clin Oral Investig*. 2013;17:1821–1827.
82. Duaibis R, Kusnoto B, Natarajan R, et al. Factors affecting stresses in cortical bone around miniscrew implants: a three-dimensional finite element study. *Angle Orthod*. 2012;82:875–880.
83. Liu TC, Chang CH, Wong TY, Liu JK. Finite element analysis of miniscrew implants used for orthodontic anchorage. *Am J Orthod Dentofacial Orthop*. 2012;141:468–476.
84. Lee JS. Contact non-linear finite element model analysis of immediately-loaded orthodontic mini implant. PhD thesis. Seoul, Korea: Yonsei University; 2005.
85. Engelking G, Zachrisson BU. Effects of incisor repositioning on monkey periodontium after expansion through the cortical plate. *Am J Orthod*. 1982;82:23–32.
86. Handelman CS. The anterior alveolus: its importance in limiting orthodontic treatment and its influence on the occurrence of iatrogenic sequelae. *Angle Orthod*. 1996;66:95–109.
87. Mimura H. Treatment of severe bimaxillary protrusion with miniscrew anchorage: treatment and complications. *Aust Orthod J*. 2008;24:156–163.
88. Steiner GG, Pearson JK, Ainamo J. Changes of the marginal periodontium as a result of labial tooth movement in monkeys. *J Periodontol*. 1981;52:314–320.
89. Wehrbein H, Bauer W, Diedrich P. Mandibular incisors, alveolar bone, and symphysis after orthodontic treatment. A retrospective study. *Am J Orthod Dentofacial Orthop*. 1996;110:239–246.
90. Hom BM, Turley PK. The effects of space closure of the mandibular first molar area in adults. *Am J Orthod*. 1984;85:457–469.

91. Stepovich ML. A clinical study on closing edentulous spaces in the mandible. *Angle Orthod*. 1979;49:227–233.
92. Aslan BI, Kucukkaraca E, Turkoz C, Dincer M. Treatment effects of the Forsus Fatigue Resistant Device used with miniscrew anchorage. *Angle Orthod*. 2014;84:76–87.
93. Buschang PH, Carrillo R, Rossouw PE. Orthopedic correction of growing hyperdivergent, retrognathic patients with miniscrew implants. *J Oral Maxillofac Surg*. 2011;69:754–762.
94. Cortese A, Savastano M, Savastano G, et al. Maxillary constriction treated by a new palatal distractor device: surgical and occlusal evaluations of 10 patients. *J Craniofac Surg*. 2010;21:339–343.
95. De Clerck H, Cevidanes L, Baccetti T. Dentofacial effects of bone-anchored maxillary protraction: a controlled study of consecutively treated Class III patients. *Am J Orthod Dentofacial Orthop*. 2010;138:577–581.
96. De Pauw GA, Dermaut L, De Bruyn H, Johansson C. Stability of implants as anchorage for orthopedic traction. *Angle Orthod*. 1999;69:401–407.
97. Feng X, Li J, Li Y, et al. Effectiveness of TAD-anchored maxillary protraction in late mixed dentition. *Angle Orthod*. 2012;82:1107–1114.
98. Ge YS, Liu J, Chen L, et al. Dentofacial effects of two facemask therapies for maxillary protraction. *Angle Orthod*. 2012;82:1083–1091.
99. Hansen L, Tausche E, Hietschold V, et al. Skeletally-anchored rapid maxillary expansion using the Dresden Distractor. *J Orofac Orthop*. 2007;68:148–158.
100. Harzer W, Schneider M, Gedrange T. Rapid maxillary expansion with palatal anchorage of the Hyrax expansion screw—pilot study with case presentation. *J Orofac Orthop*. 2004;65:419–424.
101. Hong H, Ngan P, Han G, et al. Use of onplants as stable anchorage for facemask treatment: a case report. *Angle Orthod*. 2005;75:453–460.
102. Kircelli BH, Pektaş ZO, Uçkan S. Orthopedic protraction with skeletal anchorage in a patient with maxillary hypoplasia and hypodontia. *Angle Orthod*. 2006;76:156–163.
103. Lagravère MO, Carey J, Heo G, et al. Transverse, vertical, and anteroposterior changes from bone-anchored maxillary expansion vs traditional rapid maxillary expansion: a randomized clinical trial. *Am J Orthod Dentofacial Orthop*. 2010;137:304.e1–304.e12.
104. Koudstaal MJ, Wolvius EB, Schulten AJ, et al. Stability, tipping and relapse of bone-borne versus tooth-borne surgically assisted rapid maxillary expansion; a prospective randomized patient trial. *Int J Oral Maxillofac Surg*. 2009;38:308–315.
105. Parr JA, Garetto LP, Wohlford ME, et al. Sutural expansion using rigidly integrated endosseous implants: an experimental study in rabbits. *Angle Orthod*. 1997;67:283–290.
106. Ramieri GA, Spada MC, Austa M, et al. Transverse maxillary distraction with a bone-anchored appliance: dento-periodontal effects and clinical and radiological results. *Int J Oral Maxillofac Surg*. 2005;34:357–363.
107. Sar C, Arman-Özçırpıcı A, Uçkan S, Yazici AC. Comparative evaluation of maxillary protraction with or without skeletal anchorage. *Am J Orthod Dentofacial Orthop*. 2011;139:636–649.
108. Zandi M, Miresmaeili A, Heidari A. Short-term skeletal and dental changes following bone-borne versus tooth-borne surgically assisted rapid maxillary expansion: a randomized clinical trial study. *J Craniomaxillofac Surg*. 2014;42:1190–1195.
109. Crismani AG, Bernhart T, Tangl S, et al. Nasal cavity perforation by palatal implants: false-positive records on the lateral cephalogram. *Int J Oral Maxillofac Implants*. 2005;20:267–273.
110. Kim GT, Kim SH, Choi YS, et al. Cone-beam computed tomography evaluation of orthodontic miniplate anchoring screws in the posterior maxilla. *Am J Orthod Dentofacial Orthop*. 2009;136:628.e1–628.e10.
111. Kravitz ND, Kusnoto B. Risks and complications of orthodontic miniscrews. *Am J Orthod Dentofacial Orthop*. 2007;131(4 Suppl):S43–S51.
112. Alves Jr M, Baratieri C, Mattos CT, et al. Root repair after contact with mini-implants: systematic review of the literature. *Eur J Orthod*. 2013;35:491–499.
113. Brisceno CE, Rossouw PE, Carrillo R, et al. Healing of the roots and surrounding structures after intentional damage with miniscrew implants. *Am J Orthod Dentofacial Orthop*. 2009;135:292–301.
114. Chen YH, Chang HH, Chen YJ, et al. Root contact during insertion of miniscrews for orthodontic anchorage increases the failure rate: an animal study. *Clin Oral Implants Res*. 2008;19:99–106.
115. Cobum DG, Kennedy DW, Hodder SC. Complications with intermaxillary fixation screws in the management of fractured mandibles. *Br J Oral Maxillofac Surg*. 2002;40:241–243.
116. Dao V, Renjen R, Prasad HS. Cementum, pulp, periodontal ligament, and bone response after direct injury with orthodontic anchorage screws: a histomorphologic study in an animal model. *J Oral Maxillofac Surg*. 2009;67:2440–2445.
117. Fabbroni G, Aabed S, Mizen K, Starr DG. Transalveolar screws and the incidence of dental damage: a prospective study. *Int J Oral Maxillofac Surg*. 2004;33:442–446.
118. Kadioglu O, Büyükyilmaz T, Zachrisson BU, Maino BG. Contact damage to root surfaces of premolars touching miniscrews during orthodontic treatment. *Am J Orthod Dentofacial Orthop*. 2008;134:353–360.
119. Kang YG, Kim JY, Lee YJ, et al. Stability of mini-screws invading the dental roots and their impact on the paradental tissues in beagles. *Angle Orthod*. 2009;79:248–255.
120. Renjen R, Maganzini AL, Rohrer MD, et al. Root and pulp response after intentional injury from miniscrew placement. *Am J Orthod Dentofacial Orthop*. 2009;136:708–714.
121. Alves Jr M, Baratieri C, Araújo MT, et al. Root damage associated with intermaxillary screws: a systematic review. *Int J Oral Maxillofac Surg*. 2012;41:1445–1450.
122. Lee YK, Kim JW, Baek SH, et al. Root and bone response to the proximity of a mini-implant under orthodontic loading. *Angle Orthod*. 2010; 80:452–458.
123. Wilmes B, Su YY, Sadigh L, Drescher D. Pre-drilling force and insertion torques during orthodontic mini-implant insertion in relation to root contact. *J Orofac Orthop*. 2008;69:51–58.
124. Er K, Bayram M, Taşdemir T. Root canal treatment of a periradicular lesion caused by unintentional root damage after orthodontic miniscrew placement: a case report. *Int Endod J*. 2011;44:1170–1175.
125. Hwang YC, Hwang HS. Surgical repair of root perforation caused by an orthodontic miniscrew implant. *Am J Orthod Dentofacial Orthop*. 2011;139:407–411.
126. Kim H, Kim TW. Histologic evaluation of root-surface healing after root contact or approximation during placement of mini-implants. *Am J Orthod Dentofacial Orthop*. 2011;139:752–760.
127. Lim G, Kim KD, Park W, et al. Endodontic and surgical treatment of root damage caused by orthodontic miniscrew placement. *J Endod*. 2013;39:1073–1077.
128. McCabe P, Kavanagh C. Root perforation associated with the use of a miniscrew implant used for orthodontic anchorage: a case report. *Int Endod J*. 2012;45:678–688.
129. Proffit WR, White RP, Sarver DM. *Contemporary Treatment of Dentofacial Deformity*. St. Louis: Mosby; 2003.
130. Jolley TH, Chung CH. Peak torque values at fracture of orthodontic miniscrews. *J Clin Orthod*. 2007;41:326–328.
131. Wilmes B, Ottenstreuer S, Su YY, Drescher D. Impact of implant design on primary stability of orthodontic mini-implants. *J Orofac Orthop*. 2008;69:42–50.
132. Chaddad K, Ferreira AF, Geurs N, Reddy MS. Influence of surface characteristics on survival rates of mini-implants. *Angle Orthod*. 2008;78: 107–113.
133. Motoyoshi M, Yoshida T, Ono A, Shimizu N. Effect of cortical bone thickness and implant placement torque on stability of orthodontic mini-implants. *Int J Oral Maxillofac Implants*. 2007;22:779–784.
134. Okazaki J, Komasa Y, Sakai D, et al. A torque removal study on the primary stability of orthodontic titanium screw mini-implants in the cortical bone of dog femurs. *Int J Oral Maxillofac Surg*. 2008;37:647–650.
135. Jung YR, Kim SC, Kang KH, et al. Placement angle effects on the success rate of orthodontic microimplants and other factors with cone-beam computed tomography. *Am J Orthod Dentofacial Orthop*. 2013;143:173–181.
136. Lim HJ, Eun CS, Cho JH, et al. Factors associated with initial stability of miniscrews for orthodontic treatment. *Am J Orthod Dentofacial Orthop*. 2009;136:236–242.
137. Cornelis MA, Scheffler NR, Nyssen-Behets C, et al. Patients' and orthodontists' perceptions of miniplates used for temporary skeletal anchorage: a prospective study. *Am J Orthod Dentofacial Orthop*. 2008;133:18–24.

138. Feldmann I, List T, Feldmann H, Bondemark L. Pain intensity and discomfort following surgical placement of orthodontic anchoring units and premolar extraction: a randomized controlled trial. *Angle Orthod*. 2007;77:578–585.
139. Lehnen S, McDonald F, Bourauel C, Baxmann M. Patient expectations, acceptance and preferences in treatment with orthodontic mini-implants. A randomly controlled study. Part I: insertion techniques. *J Orofac Orthop*. 2011;72:93–102.
140. Lehnen S, McDonald F, Bourauel C, et al. Expectations, acceptance and preferences of patients in treatment with orthodontic mini-implants: part II: implant removal. *J Orofac Orthop*. 2011;72:214–222.
141. Burstone CJ. Biomechanical rationale of orthodontic therapy. In: Melsen B, ed. *Current Controversies in Orthodontics*. Chicago: Quintessence Publishing Co; 1991:131–146.
142. Ari-Demirkaya A, Masry MA, Erverdi N. Apical root resorption of maxillary first molars after intrusion with zygomatic skeletal anchorage. *Angle Orthod*. 2005;75:761–767.
143. Baek MS, Choi YJ, Yu HS, et al. Long-term stability of anterior open-bite treatment by intrusion of maxillary posterior teeth. *Am J Orthod Dentofacial Orthop*. 2010;138:396.e1–396.e9; discussion 396–398.
144. Carrillo R, Buschang PH, Opperman LA, et al. Segmental intrusion with mini-screw implant anchorage: a radiographic evaluation. *Am J Orthod Dentofacial Orthop*. 2007;132:576.e1–576.e6.
145. Carrillo R, Rossouw PE, Franco PF, et al. Intrusion of multiradicular teeth and related root resorption with mini-screw implant anchorage: a radiographic evaluation. *Am J Orthod Dentofacial Orthop*. 2007;132:647–655.
146. Choi YJ, Kim KH, Lee KJ, et al. Radiographic evaluations of molar intrusion and changes with or without retention in rats. *Angle Orthod*. 2011;81:389–396.
147. Daimaruya T, Nagasaka H, Umemori M, et al. The influences of molar intrusion on the inferior alveolar neurovascular bundle and root using the skeletal anchorage system in dogs. *Angle Orthod*. 2001;71:60–70.
148. Daimaruya T, Takahashi I, Nagasaka H, et al. Effects of maxillary molar intrusion on the nasal floor and tooth root using the skeletal anchorage system in dogs. *Angle Orthod*. 2003;73:158–166.
149. Kanzaki R, Daimaruya T, Takahashi I, et al. Remodeling of alveolar bone crest after molar intrusion with skeletal anchorage system in dogs. *Am J Orthod Dentofacial Orthop*. 2007;131:343–351.
150. Konno Y, Daimaruya T, Iikubo M, et al. Morphologic and hemodynamic analysis of dental pulp in dogs after molar intrusion with the skeletal anchorage system. *Am J Orthod Dentofacial Orthop*. 2007;132:199–207.
151. Li W, Chen F, Zhang F, et al. Volumetric measurement of root resorption following molar mini-screw implant intrusion using cone beam computed tomography. *PLoS One*. 2013;8(4):e60962.
152. Proffit WR, Fields Jr HW, Sarver DM. *Contemporary Orthodontics*. 4th ed. St. Louis: Mosby; 2007.
153. Proffit WR, Phillips C, Turvey TA. Stability following superior repositioning of the maxilla by LeFort I osteotomy. *Am J Orthod Dentofacial Orthop*. 1987;92:151–161.
154. Ramirez-Echave JI, Buschang PH, Carrillo R, et al. Histologic evaluation of root response to intrusion in mandibular teeth in beagle dogs. *Am J Orthod Dentofacial Orthop*. 2011;139:60–69.
155. Veberiene R, Smailiene D, Danielyte J, et al. Effects of intrusive force on selected determinants of pulp vitality. *Angle Orthod*. 2009;79:1114–1118.
156. Wessberg GA, O'Ryan FS, Washburn MC, Epker BN. Neuromuscular adaptation to surgical superior repositioning of the maxilla. *J Maxillofac Surg*. 1981;9:117–122.
157. Wessberg GA, Washburn MC, LaBanc JP, Epker BN. Autorotation of the mandible: effect of surgical superior repositioning of the maxilla on mandibular resting posture. *Am J Orthod*. 1982;81:465–472.
158. Denison TF, Kokich VG, Shapiro PA. Stability of maxillary surgery in openbite versus nonopenbite malocclusions. *Angle Orthod*. 1989;59:5–10.
159. Maia FA, Janson G, Barros SE, et al. Long-term stability of surgical-orthodontic open-bite correction. *Am J Orthod Dentofacial Orthop*. 2010;138:254.e1–254.e10.
160. Celli D, Gasperoni E, Deli R. Long-term outcome in a patient with a dento-skeletal open-bite malocclusion treated without extraction. *World J Orthod*. 2007;8:344–356.
161. Kawamura M, Nojima K, Nishii Y, Yamaguchi H. A cineradiographic study of deglutitive tongue movement in patients with anterior open bite. *Bull Tokyo Dent Coll*. 2003;44:133–139.
162. Huang GJ. Long-term stability of anterior open-bite therapy: a review. *Semin Orthod*. 2002;8:162–172.
163. Huang GJ, Justus R, Kennedy DB, Kokich VG. Stability of anterior open-bite treated with crib therapy. *Angle Orthod*. 1990;60:17–24.
164. Yamaguchi H, Sueishi K. Malocclusion associated with abnormal posture. *Bull Tokyo Dent Coll*. 2003;44:43–54.
165. Katona TR. An engineering analysis of dental occlusion principles. *Am J Orthod Dentofacial Orthop*. 2009;135:696.e1–696.e8.
166. Jeong GM, Sung SJ, Lee KJ, et al. Finite-element investigation of the center of resistance of the maxillary dentition. *Korean J Orthod*. 2009;39:83–94. Available at http://dx.doi.org/10.4041/kjod.2009.39.2.83.
167. Lee KJ, Joo E, Yu HS, Park YC. Restoration of an alveolar bone defect caused by an ankylosed mandibular molar by root movement of the adjacent tooth with miniscrew implants. *Am J Orthod Dentofacial Orthop*. 2009;136:440–449. http://dx.doi.org/10.1016/j.ajodo.2007.05.028.
168. Nagaraj K, Upadhyay M, Yadav S. Titanium screw anchorage for protraction of mandibular second molars into first molar extraction sites. *Am J Orthod Dentofacial Orthop*. 2008;134:583–591.
169. Roberts WE, Arbuckle GR, Analoui M. Rate of mesial translation of mandibular molars using implant-anchored mechanics. *Angle Orthod*. 1996;66:331–338.
170. Roberts WE, Marshall KJ, Mozsary PG. Rigid endosseus implant utilized as anchorage to protract molars and close an atrophic extraction site. *Angle Orthod*. 1990;60:135–152.
171. Gündüz E, Rodriguez-Torres C, Gahleitner A, et al. Bone regeneration by bodily tooth movement: dental computed tomography examination of a patient. *Am J Orthod Dentofacial Orthop*. 2004;125:100–106.
172. Vanarsdall RL, Secchi AG. Periodontal-orthodontic interrelationships. In: Graber TM, Vanarsdall RL, Vig KWL, eds. *Orthodontics: Current Principles and Techniques*. 4th ed. St. Louis: Mosby; 2005.
173. Tan WL, Wong TL, Wong MC, Lang NP. A systematic review of post-extractional alveolar hard and soft tissue dimensional changes in humans. *Clin Oral Implants Res*. 2012;23(Suppl 5):1–21.
174. Northway W. Hemisection: one large step toward management of congenitally missing lower second premolars. *Angle Orthod*. 2004;74:792–799.
175. Upadhyay M, Yadav S, Nagaraj K, Patil S. Treatment effects of mini-implants for en-masse retraction of anterior teeth in bialveolar dental protrusion patients: a randomized controlled trial. *Am J Orthod Dentofacial Orthop*. 2008;134:18–29.e1. http://dx.doi.org/10.1016/j.ajodo.2007.03.025.
176. Kojima Y, Kawamura J, Fukui H. Finite element analysis of the effect of force directions on tooth movement in extraction space closure with miniscrew sliding mechanics. *Am J Orthod Dentofacial Orthop*. 2012;142:501–508.
177. Lee KJ, Park YC, Hwang CJ, et al. Displacement pattern of the maxillary arch depending on miniscrew position in sliding mechanics. *Am J Orthod Dentofacial Orthop*. 2011;140:224–232. http://dx.doi.org/10.1016/j.ajodo.2010.05.020.

21

Adult Interdisciplinary Therapy: Diagnosis and Treatment

Robert L. Vanarsdall, Jr., and David R. Musich

OUTLINE

Current Status of Adult Interdisciplinary Therapy, 569
Goals of Adult Interdisciplinary Therapy, 570
Ideal Orthodontic Treatment Goals and the Adult Patient, 570
Individualized Adult Interdisciplinary Orthodontic Treatment Objectives, 571
Additional Adult Treatment Objectives, 571
Diagnosis of Adult Orthodontic Conditions, 583
Skeletal Differential Diagnosis, 583
Periodontal Differential Diagnosis, 586
Temporomandibular Joint Differential Diagnosis, 589
Clinical Management of the Interdisciplinary Adult Therapy Patient, 589
Biomechanical Considerations, 591
Control of Occlusion, 591
Periodontal Management During the Orthodontic Tooth Movement, 594
Significance of Tooth Mobility, 594
Periodontal Preparation of Adults before Orthodontic Therapy, 595
Behavioral Management Orthodontist and Staff Preparation for Adult Interdisciplinary Patient Management, 595
Sequence of Adult Interdisciplinary Therapy, 596
Evaluation of the Skeletal Component of the Malocclusion, 596
Periodontal Preparation, 597
Dentoalveolar Collapse: Orthodontic and Restorative Considerations in the Adult Interdisciplinary Patient, 597
Application of Principles, 600
Conclusion for Adult Interdisciplinary Therapy Patients with Missing Teeth and Dentoalveolar Collapse, 602
Evaluation before Debonding or Debanding, 602
Coordination of Debonding or Debanding with Other Treatment Providers, 606
Stability and Retention Individualized for the Interdisciplinary Adult Therapy Patient, 606
Communicating with Team Members, 613
Risk Management and the Adult Interdisciplinary Therapy Patient, 613

CURRENT STATUS OF ADULT INTERDISCIPLINARY THERAPY

In the past several years a major reorientation of orthodontic thinking has occurred regarding adult patients. The following list cites several reasons for the increased interest by orthodontists in adults as patients, as well as several reasons for increased interest shown by adults in orthodontic treatment.

In 1971, Lindegaard et al.[1] stated that three main factors determine which problems (including adult interdisciplinary conditions) could be treated from both a medical and an orthodontic point of view:

1. A disease or an abnormality must be present.
2. The need for treatment should be determined by the clinical gravity of the disorder, the available resources for orthodontic care, the prognosis for successful treatment, and the priority for orthodontic care based on personal and professional judgment.
3. The patient must have a strong desire for treatment.

In this context, the past 40 years have seen a major change in orthodontic practices. Changed lifestyles and patient awareness have led to a continued increased demand for adult orthodontic treatment, and multidisciplinary (more appropriately called interdisciplinary) dental therapy has allowed better management of the more complicated and unique requirements of the adult patient population. Much of the improved technology and its application have greatly improved the quality of care and treatment prognosis for our adult patients.[2]

Department chairs of several teaching institutions participating in a round table discussion concerning the future of orthodontics[3] made significant statements regarding adult orthodontic therapy. At this conference 40 years ago (1976), Reidel and Dougherty most accurately predicted the status of adult orthodontic treatment today. Reidel was supportive regarding the future of adult therapy, adding that clinicians should not forget adjunctive orthodontic services provided by periodontists and restorative dentists. Dougherty claimed that "orthodontics is total discipline and it makes no difference whether the patient is young or old."[3] Demographics of the American population further dictate that orthodontists provide services sought by "aging baby boomers."

The Population Reference Bureau, a nonprofit demographic study group in Washington, D.C., has predicted that by 2025, Americans older than 65 years of age will outnumber teenagers by more than 2:1. According to the U.S. Census Bureau, by

2030, the median age is expected to be 41 years. By 2050, it is likely that as many as one in four Americans will be older than 65 years. Many demographers consider these projections to be very conservative; by some estimates, the median age will eventually reach 50 years (Fig. 21-1). Thus, the demographic considerations demonstrated here and illustrated in Figure 21-2 emphasize the importance of orthodontists developing the skills necessary to manage the increasing number of interdisciplinary adult orthodontic patients.

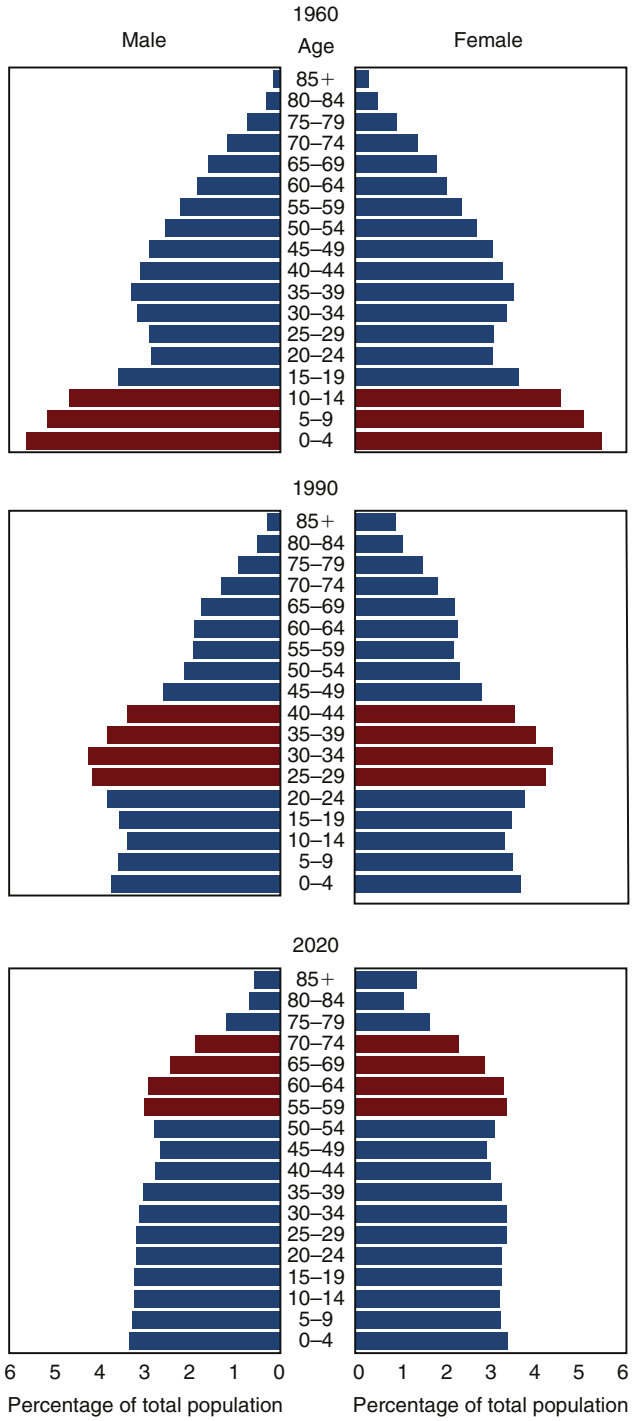


FIGURE 21-1 Percent of U.S. population by age and sex: 1960, 1990, and projections for 2020. Note the graphic illustration of the aging of the U.S. population. The implications for providing optimal interdisciplinary dental therapy to provide the best quality of life for our aging population are quite clear. (Source: U.S. Bureau of the Census.)

In addition to recent improvements in treatment techniques and changes in treatment philosophies, important statistical reasons explain why orthodontists have become more involved in the management of adult patients (see Fig. 21-2).[4] Practices that grew reported a higher percentage of adult patients than those that did not grow. The treatment of adults was rated significantly higher as a practice-building method by growing practices than by declining practices. As the volume (percentage) of adult patients increased in orthodontic practices, the skills required of orthodontists changed. Musich's 1986 paper demonstrated the scope of treatment planning considerations[5] (Fig. 21-3). Of the almost 1400 consecutively examined adults in Musich's study, about 70% to 75% of the sample required multidisciplinary management to attain optimal treatment outcomes.

When treating adults, the orthodontist needs to be prepared to do the following:

1. Determine which cases require orthognathic surgical management and which ones require incisor reangulation to camouflage the skeletal base discrepancy
2. Diagnose different stages of periodontal disease and the associated risk factors
3. Diagnose temporomandibular joint (TMJ) dysfunction (TMD) before, during, or after tooth movement
4. Work cooperatively with a team of other specialists to give the patient the best outcome. It is to be noted that the orthodontist must frequently take a leadership role in both the diagnosis and the treatment planning of adult interdisciplinary therapy (AIT) patients.

GOALS OF ADULT INTERDISCIPLINARY THERAPY

Ideal Orthodontic Treatment Goals and the Adult Patient

In 1972, Andrews described the six keys to normal occlusion, and this description of orthodontic treatment objectives is still the standard by which orthodontic treatment results are measured. More recently, the American Board of Orthodontics (AJODO) sought to quantify further optimal treatment (November 1998, AJODO).[6]

However, adult patients have many preexisting conditions that are not seen in the adolescent population, including tooth loss, severe skeletal dysplasias, periodontal disease, and various forms of TMD. Frequently, the preexisting conditions that are present in an adult patient interfere with the achievement of orthodontists' general idealized goals. In such adult cases, an attempt to achieve ideal tooth positions that are feasible only in dentitions with a Class I skeletal relationship may be considered overtreatment. This is not to say that the orthodontic therapy provided is any less precise; rather, it suggests a need to customize orthodontic treatment for the individual patient so that the achievement of any one goal (perhaps facial aesthetics) does not undermine a less obvious but equally important functional need.

Problem-oriented synthesis of the dental needs of each case will help determine specific treatment objectives that must be established before the orthodontic treatment plan can be determined. Beginning treatment without knowing the specific goals for the individual patient or with unrealistic goals can lead to treatment failure.

In addition to goal clarification, adult patients desire treatment efficiency, convenience in appointment times, and good communication with other health care providers. Both the examination and consultation should allow for two-way communication so the orthodontist and the patient understand the treatment process. Unlike the typical adolescent, an adult may exhibit rapid (within 2 to 3 months) periodontal breakdown and bone loss. Therefore, adult therapy requires the establishment of goals and efficient mechanotherapy so that completion occurs as expeditiously as possible. It is necessary to inform adult patients of the need to keep all of their appointments and to optimize their compliance with periodontal maintenance.

Individualized Adult Interdisciplinary Orthodontic Treatment Objectives

The generally applied orthodontic treatment goals of (1) favorable dentofacial aesthetics, (2) effective stomatognathic system function, (3) short- and long-term stability, and (4) a static and dynamic Class I occlusion often may not be realistic or necessary for all adult patients. Treatment in which general goals are not achieved is not necessarily a compromised result; rather, the mechanotherapy should satisfy the objective of providing the minimal dental manipulation appropriate for the individual patient. Many Class I occlusal goals can be considered overtreatment for patients who also require restorative dentistry, prosthetics, plastic surgery, and other multidisciplinary dentofacial corrections. Box 21-1 contains a list of additional goals particularly useful for adult problems requiring tooth replacement but not requiring surgical skeletal correction.

Additional Adult Treatment Objectives

1. **Parallelism of abutment teeth.** The abutment teeth must be placed parallel with the other teeth to permit insertion of multiple unit replacements and allow for restorations that involve both the anterior and posterior teeth. A restoration will have a better prognosis if the abutment teeth are parallel before tooth preparation[7] because that position does not require excess cutting or devitalization during abutment preparation and allows for a better periodontal response. For full-arch splints, the posterior teeth should be reasonably parallel to anterior abutments. Parallel abutments allow for better restorative retention and help prevent cement washout and caries (Fig. 21-4).
2. **Most favorable distribution of teeth.** The teeth should be distributed evenly for replacement of fixed and removable prostheses in the individual arches (Fig. 21-5). In addition, they should be positioned so that occlusion of natural teeth can be established bilaterally between arches.[8]
3. **Redistribution of occlusal and incisal forces.** Cases with significant bone loss (60%–70%) require that occlusal forces be directed vertically along or on the long axis of the roots to maintain the occlusal vertical dimension (Fig. 21-6). When the posterior teeth are missing, the anterior teeth can be positioned to allow for more axially directed transfer of force and can then be reshaped to function as posterior teeth (supporting the vertical dimension).[8]
4. **Adequate embrasure space and proper root position.** This allows for better periodontal health, especially when the placement of restorations is necessary (Fig. 21-7). The anatomic relation of the roots is important in the pathogenesis of periodontal disease,[9] interproximal cleaning, and placement of restorative materials.[10]
5. **Acceptable occlusal plane and potential for incisal guidance at satisfactory vertical relationship.** To establish the acceptable occlusal plane for a mutilated dentition exhibiting bite collapse, the Hawley bite place (Fig. 21-8) is inserted with the platform of the anterior plane adjusted at a right angle to the long axis of the lower incisors.[11] This allows a centric relation at an acceptable vertical relationship. Even with extended incisolingual platforms in Class II patterns, patients can usually speak well. However, if the vertical dimension is excessive, they will whistle involuntarily and complain of muscle fatigue in the morning, or the acrylic will develop etched lines or wear streaks. When properly adjusted at the correct vertical height, the bite plane will allow simultaneous bilateral neuromuscular activity. It is important that no contact occurs between the posterior teeth during excursive movements and no interferences occur between anterior teeth while the bite plane is in place. Such interferences will prevent the patient from demonstrating simultaneous bilateral neuromuscular activity and correct location of centric relation.

The curve of Spee should be mild to flat bilaterally. This is difficult to achieve if supraerupted molars are present. However, the most extruded posterior segment will be the ruling factor in determining the potential for an orthodontic solution at an acceptable vertical. Adult molars with amalgam restorations and normal pulpal recession and mixed constrictions

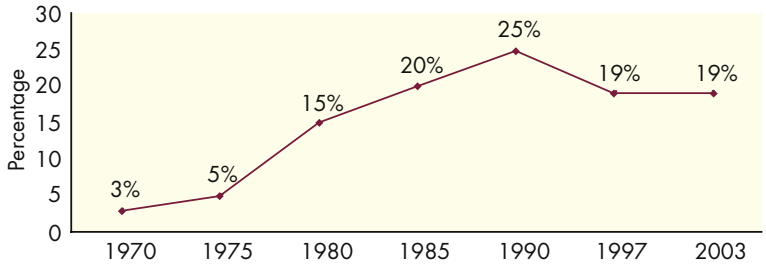
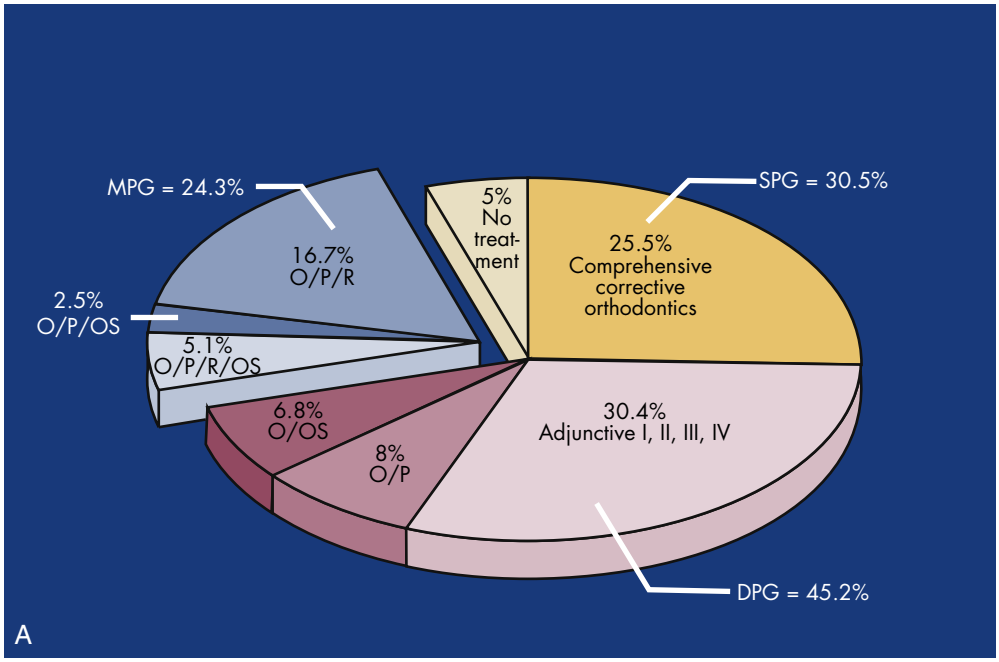


FIGURE 21-2 This graph illustrates an 800% increase of adult patients in the United States receiving orthodontic treatment from 1970 to 2003. The median percentage of patients identified as adults in orthodontic practices seems to have leveled off or even decreased as a percentage of the total number of patients in braces. The actual number of adults in orthodontic therapy probably has increased and has the potential to increase even more as more sophisticated and aesthetic methods of providing care are used. (From Keim RG, Gottleib EL, Nelson AH, et al: 2003 JCO orthodontic practice study. 1. Trends, *J Clin Orthod* 2003;37:545–553.)



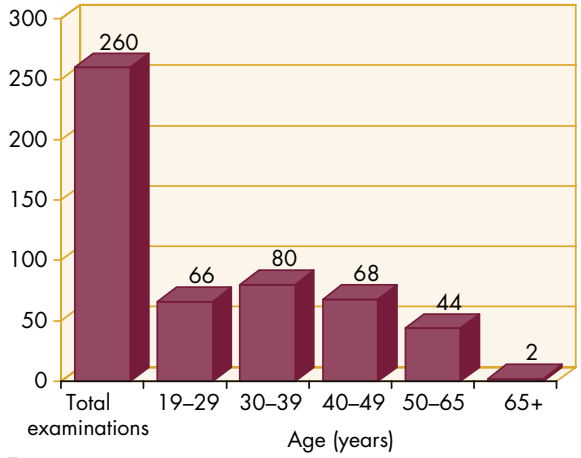


FIGURE 21-3 **A,** A summary diagram of findings from Musich's study[5] of 1370 consecutively examined adults: 30.5%, solo provider group (SPG)—25.5% required conventional corrective orthodontics; 45.2%, dual provider group (DPG)—within this group two primary providers were required to complete the treatment (orthodontist/restorative dentist [O/R], 30.4%; orthodontist/periodontist [O/P], 8.0%; orthodontist/oral surgeon [O/OS], 6.8%); and 24.3%, multiple provider group (MPG). Note that 5% required no orthodontic treatment and 65% required dual or multiple provider therapy. **B,** Graph showing number of adults examined by author according to age. Note that 90% of adults were examined between the ages of 19 and 49 years of age.

Adult Patients Redefined: 19 to 80 Years

Age (yr)	Abbreviated Social Factors
19–29	Many still in transition to adult level of independence
30–39	Pressure, pregnancy (70% of adults seeking orthodontic care are women), stress, money, temporomandibular joint
40–49	Time for self (before it is too late)
50–65	Use of insurance, prepare for retirement, other health issues ("stay younger longer")
66–80	Preserve and restore, major role of interdisciplinary therapy, including implants

Graph based on a recommended division of adult patients by decade of life, which may factor into treatment decisions.

often can be occlusally reduced 2 to 4 mm and still allow for placement of restorations without the need for devitalization. With the aid of heavy musculature, molars may be intruded 1 to 2 mm in treatment. The unilateral orthodontic treatment of an accentuated occlusal plane should be avoided; one side cannot be left extruded. For the extruded posterior segments, temporary anchorage devices (TADs) are available to help level the occlusal plane and to provide proper dental relationships for the restorative phase of therapy (see Chapter 20).

BOX 21-1 Additional Orthodontic Treatment Goals in Adult Treatment

- Parallelism of abutment teeth
- Most favorable distribution of teeth
- Redistribution of occlusal and incisal forces
- Adequate embrasure space and proper tooth position
- Acceptable occlusal plane and potential for incisal guidance at satisfactory vertical dimension
- Adequate occlusal landmark relationships
- Better lip competency and support
- Improved crown-to-root ratio
- Improvement or self-correction of mucogingival and osseous defects
- Improved self-maintenance of periodontal health
- Aesthetic and functional improvement
- Best outcome with least risk (using minimally invasive approach)

1. **Adequate occlusal landmark relationships.** As previously described for adult patients, the transverse dimension is the most difficult to correct and maintain orthodontically, the sagittal next, and the vertical least. However, when teeth are to be restored, they must be positioned to achieve acceptable buccolingual landmarks. Posterior teeth in crossbite that are not corrected through a surgical procedure should be positioned so that the maxillary buccal cusps contact the lower central fossae with the crossover for incisal guidance in the premolar area or the canine positions. Figure 21-9 illustrates the achievement of favorable occlusal landmark relationships in a patient who has a true skeletal imbalance both in the transverse plane and the sagittal plane without incorporation of surgical procedures. The sagittal correction occurred through incisor reangulation, and the transverse

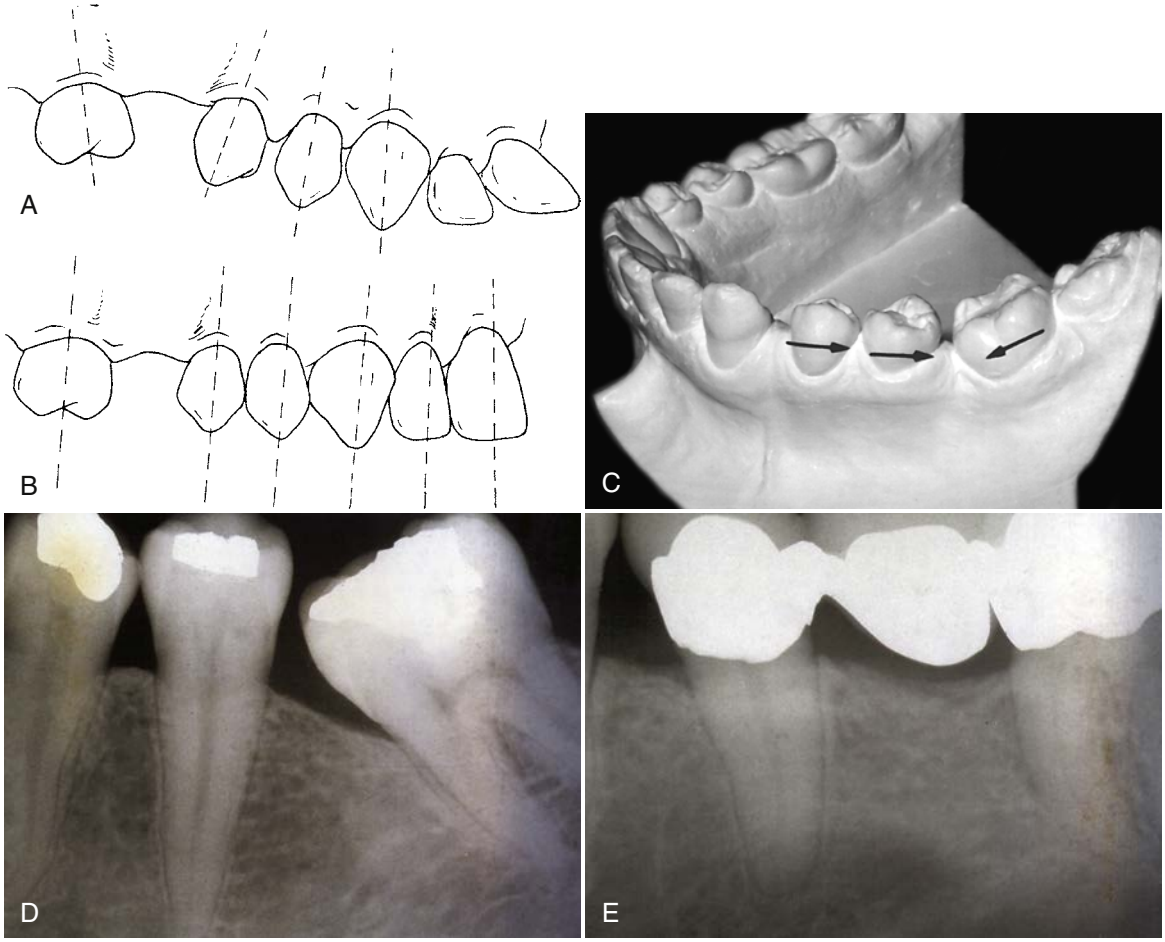


FIGURE 21-4 Clinical example of adult treatment objective 1: parallelism of abutment teeth. **A,** Drifting and flaring of the maxillary teeth caused by mutilation, periodontal disease, and occlusal and muscular forces. **B,** Orthodontic preparation. This should produce parallelism of the anterior and posterior segments before restorative dentistry that involves full arch stabilization. **C,** Lower model of collapsed arch with missing no. 19. **D,** Radiograph of tilted molar. **E,** Molar uprighted and restoration placed with roots parallel. Orthodontic reangulation of teeth adjacent to missing teeth is an important goal to facilitate long-term periodontal health and the aesthetics of the replacement tooth in posterior and anterior portions of the mouth.

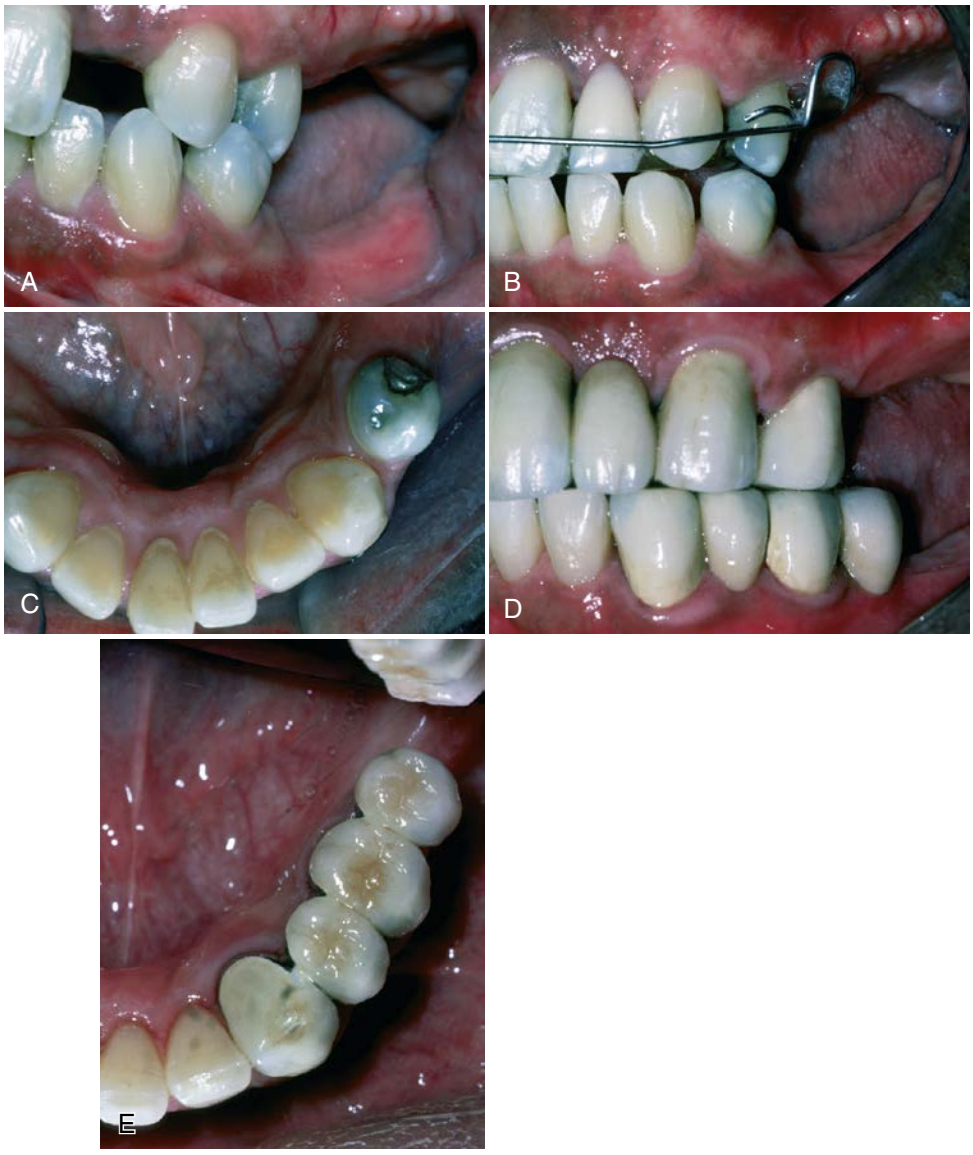


FIGURE 21-5 Clinical example of adult treatment objective 2: most favorable distribution of teeth. **A,** First premolars, the only teeth present in the left maxillary and mandibular quadrants of a 56-year-old man. **B,** A combination of fixed and removable appliances was used to move the mandibular first premolar one pontic space distally in the left quadrant. **C,** Preoperative. Note the lower left first premolar adjacent to the canine. **D,** Final restoration. Distal movement of the first premolar abutment created a pontic space between the canine and the distal premolar abutment. **E,** Better distribution of abutments allowed for this lower left four-unit restoration, preventing the need for a distal extension partial denture or implants.

was restored in a "functional" crossbite described earlier, resulting in a stable outcome.

2. **Better lip competency and support.** Many adults have long upper lips that preclude significant maxillary retraction. In cases requiring anterior restorations, retraction is recommended to achieve lip competency while maintaining lip support. The restoration then can be shaped to provide incisal guidance on the canines or by 1- or 2-mm palatal extension of the incisors. Incisors extended more than 1 or 2 mm palatally cause constant palatal soft tissue irritation. In some Class II, division 1 cases (when orthognathic surgery is rejected), the lower incisors can be advanced into a more procumbent position than the usual orthodontic norm to establish incisal guidance. With the aid of bilateral posterior flared positions (incisor mandibular plane angle [IMPA] of 105–120 degrees). In some Class III patients as well, the maxillary incisors can be kept in stable relation (even though more flared than normal) with posterior restorations. Inadequate support may create a change of anteroposterior and vertical position of the upper lip and increase wrinkling. This often makes the face seem prematurely aged and is a major aesthetic concern of adults, especially women, who are usually anxious about changes of the upper lip (Fig. 21-10).
3. **Improved crown-to-root ratio.** In adult patients who have lost bone on individual teeth, the length of the clinical crown

FIGURE 21-6 Clinical example of adult treatment objective 3: redistribution of occlusal and incisal forces. **A,** No natural tooth stops in a 45-year-old woman. The initial tooth contact in centric relation was between the mandibular first premolar and the maxillary second premolar. **B,** Anteriorly, the mandible fits within the maxillary arch. **C,** No tooth contact on the left side. **D,** Soft tissue indentations indicate the location of lower incisor contact with the palate. **E,** Severe maxillary protrusion. A Hawley bite plane was used to locate centric relation at the acceptable vertical. **F,** After maxillary and mandibular alignment, a splint was placed before maxillary segmental osteotomy. The osteotomy positioned the maxillary canines axially to contact the lower dentition bilaterally. **G,** After surgery, occlusal platforms placed on the maxillary canines support the vertical dimension. **H,** Three years post treatment. **I,** Lower anteriors bonded with composite resin as a form of retention. **J**, Pretreatment. **K,** Three years post treatment. **L,** Pretreatment cephalogram. Acceptable vertical dimension. **M,** One year post treatment.

can be reduced with the high-speed handpiece; as the tooth is erupted orthodontically (the same amount of bone will remain on the clinical root), the ratio of crown to root will be improved (Fig. 21-11).[12]

4. **Improvement or correction of mucogingival and osseous defects.** Proper repositioning of prominent teeth in the arch will improve gingival topography (Fig. 21-12). In adolescents, the brackets are placed to level marginal ridges and

FIGURE 21-7 Clinical example of adult treatment objective 4: adequate embrasure space and proper root position. **A–E,** Pretreatment intraoral photographs showing compromised embrasures and altered root position of lower second molars preventing proper restorations. **F–J,** Posttreatment intraoral photographs showing corrected crown and root position. Note no. 7 rotation correction and lower anterior spacing correction. **K,** Pretreatment radiographs. Note rotated no. 7 and no. 10 and tilted nos. 18 and 31. **L,** Long-term panoramic radiograph. Note the continued stability of nos. 18, 19, 30, and 31 and the previously rotated no. 7 at 7 years after orthodontic treatment. **M,** Long-term cephalogram. Note the continued stability of the posterior occlusion 7 years after orthodontic treatment.

FIGURE 21-8 Clinical example of adult treatment objective 5: acceptable occlusal plane and potential for incisal guidance at satisfactory vertical dimension. **A,** No occlusal stops bilaterally in a 61-year-old patient. The lower right premolar had only soft tissue attachment. **B,** Lower canines tipped lingually and mobile. **C,** Preoperative. **D,** Upper and lower removable appliances were placed to support the vertical height and move each lower canine labially over its basal support. **E,** After the lower canines were positioned axially, the restorative dentist (Dan Casullo, Philadelphia) placed a provisional restoration. A platform then was added to the upper appliance (to determine the satisfactory vertical), and the maxillary incisors were aligned. **F,** Seven years postoperative. **G,** Final restoration. **H,** Twenty-year follow-up of patient with advanced attachment loss.

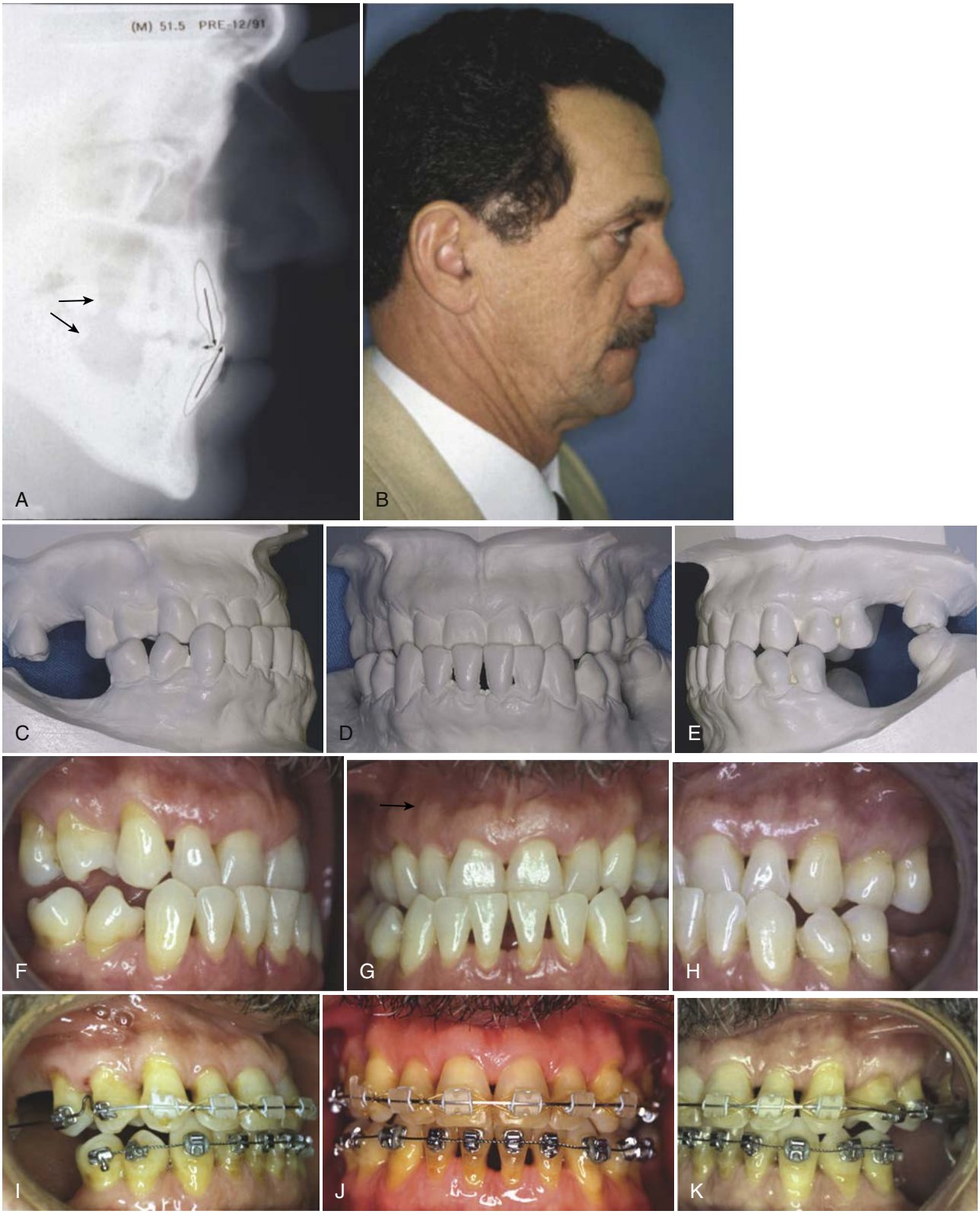


FIGURE 21-9 A-B, Pre-treatment cephalometric radiograph and profile picture showing Class III with anterior crossbite of a 51:6 male. C-E, Pre-treatment study models of the patient for whom jaw surgery was the only option recommended by several surgeons and other orthodontists. F-H, Pre-treatment intra-oral photographs of same patient, showing evidence of mild functional shift. Arrow at the abundant attached gingival tissue in maxillary anterior, indicating adequate tissue available for incisor re-angulation and camouflage therapy for this patient. I-K, Intra-oral photographs illustrating favorable response to initial stages of therapy.

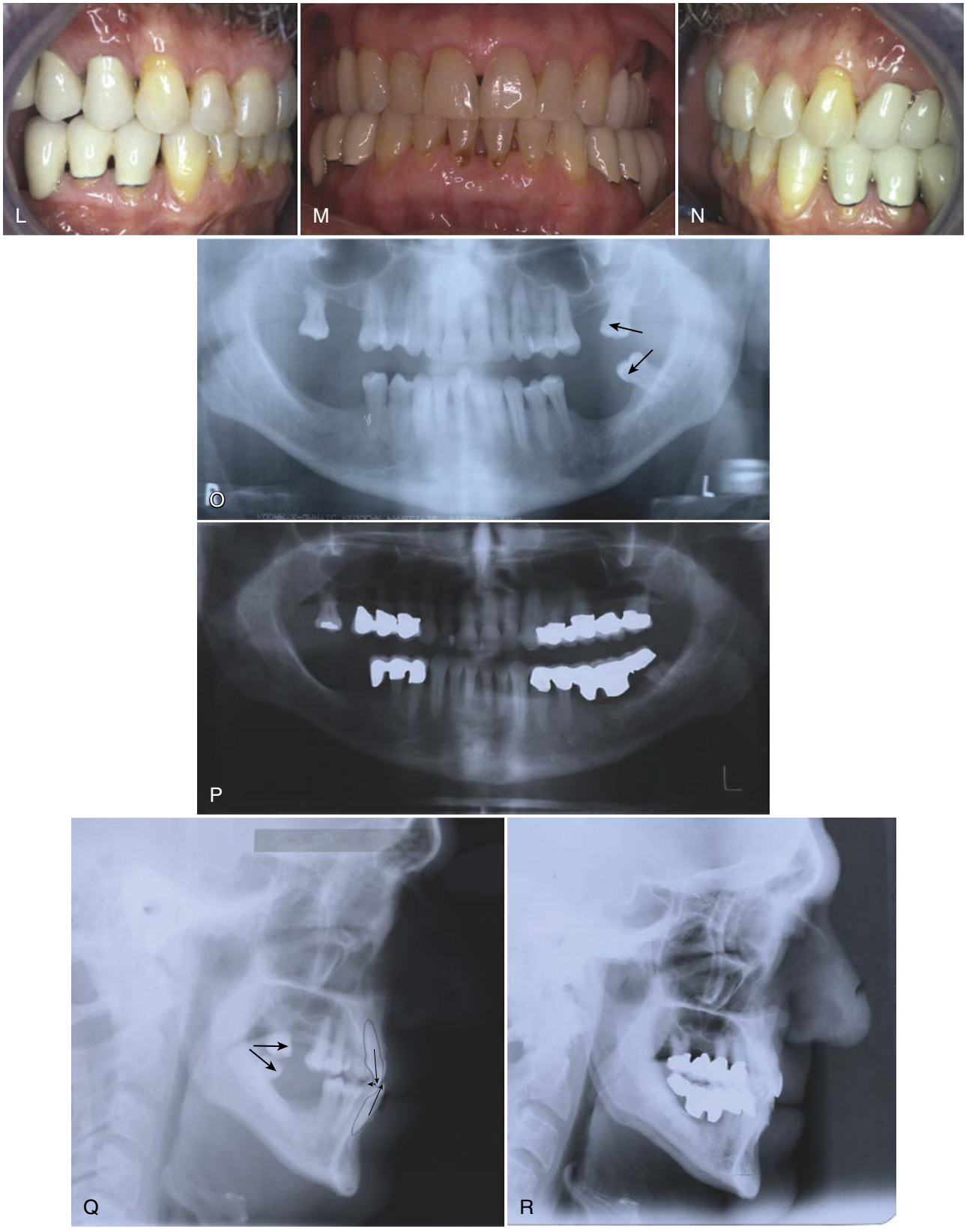


FIGURE 21-9, cont'd L-N, Intra-oral photographs with posterior restoration complete. Posterior segments left in cross-bite. O, Pre-treatment panoramic radiograph showing loss of several posterior teeth and bone loss. P, Post-orthodontic and restorative radiograph showing restoration of posterior occlusion. Q, Pre-treatment cephalometric radiograph showing Class III with anterior crossbite and loss of posterior vertical support due to extensive tooth loss. Class III relationship has a mild skeletal component and a moderate dental component with a functional shift that increases the severity of the Class III appearance. R, Post-treatment cephalometric radiograph with corrected anterior relationship and restored posterior occlusion.

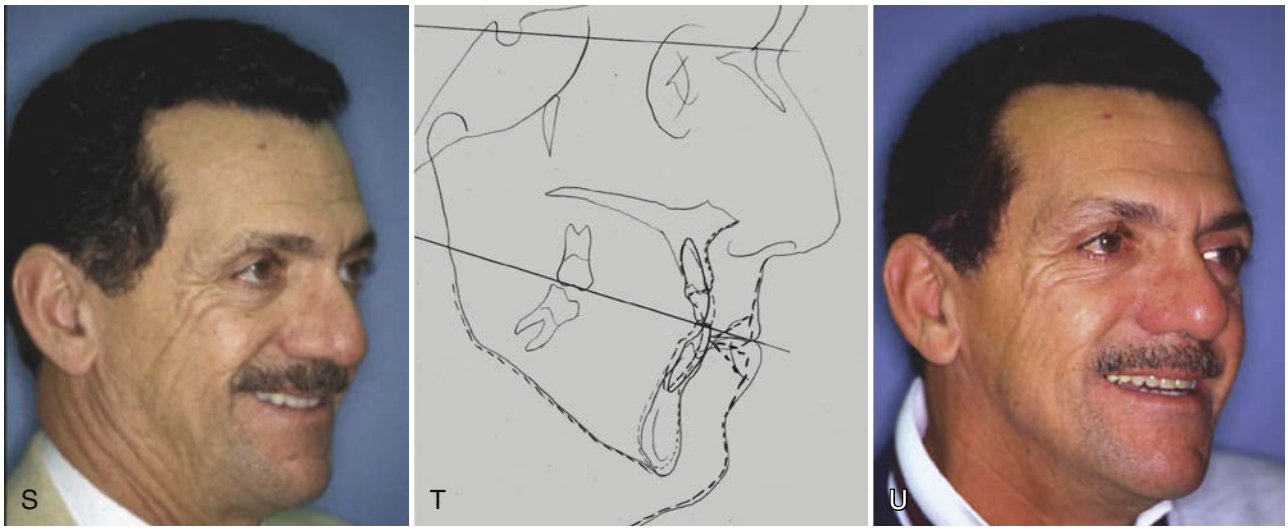


FIGURE 21-9, cont'd S, Pre-treatment of 51:6 year old male with long-standing Class III malocclusion. Several specialist had recommended jaw surgery to resolve this problem. Jaw surgery would have been "over-treatment" for this patient. T, Pre and Post treatment cephalometric supra-impositions improved facial balance due to camouflage therapy and the elimination of the functional shift. U, Post-treatment of patient with very favorable achievement of Objective #6 (Adequate Occlusal Landmark Relationships) through non-surgical management of his Class III malocclusion.

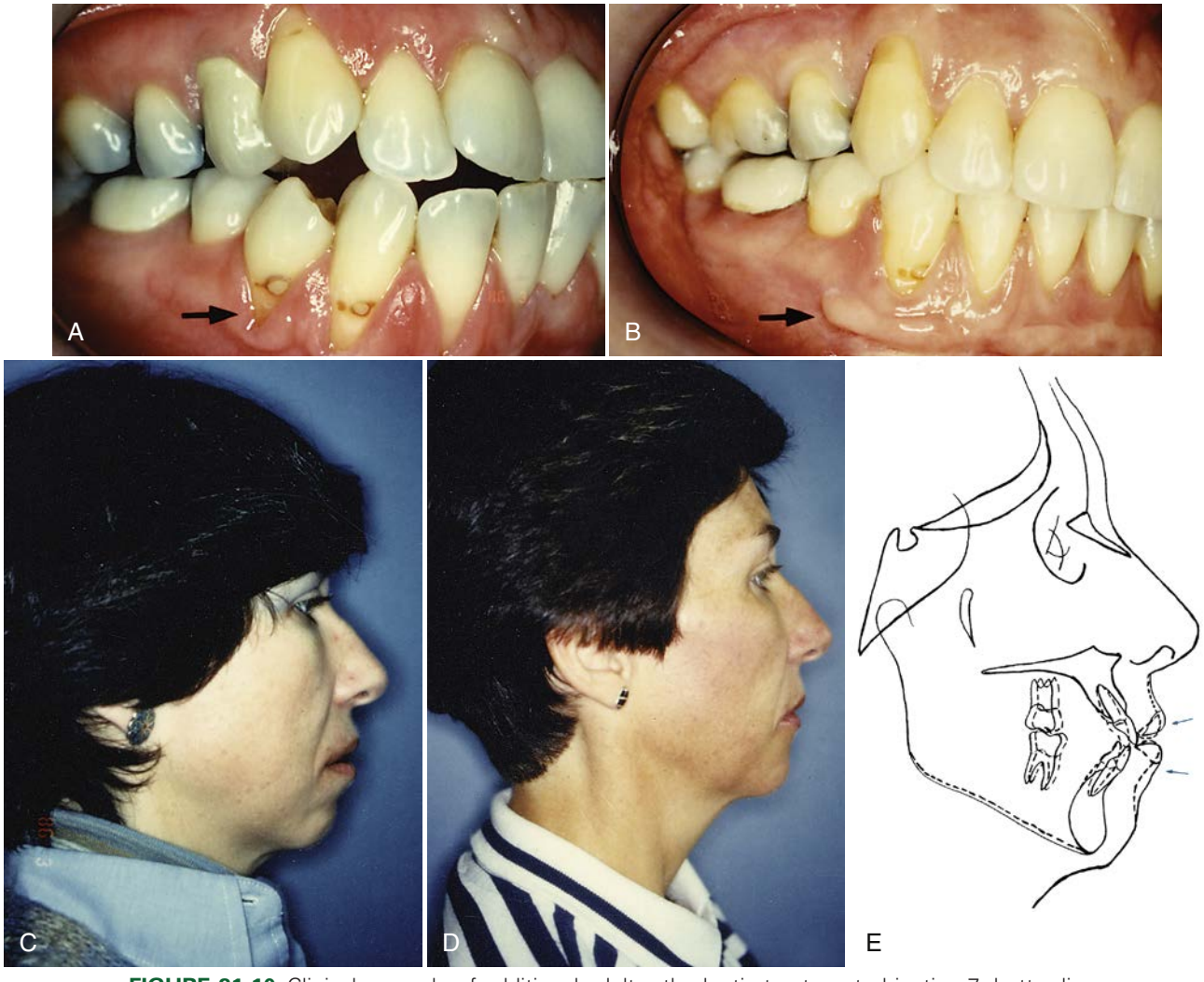


FIGURE 21-10 Clinical example of additional adult orthodontic treatment objective 7: better lip competency and support. A, Pretreatment right occlusion illustrating open bite, protrusive upper and lower incisors with crowding and gingival recession (*arrow*). B, Posttreatment right occlusion illustrating correction of preexisting malocclusion and incisor retraction with extraction of nos. 5,12, 21, and 28 and correction of gingival recession (*arrow*). C, Pretreatment profile showing lip incompetence caused by dentoalveolar protrusion. D, Posttreatment profile showing improved lip competence and relaxed mentalis. E, Cephalometric superimposition showing corrected incisor protrusion and subsequent lip position improvement (*arrows*).

FIGURE 21-11 Clinical example of adult treatment objective 8: improved crown-to-root ratio. **A,** Before correction of an incisor crossbite. Note the normal gingival position on the labially displaced lower left lateral incisor in a 14-year-old patient. After lower incisor alignment the gingival margins were confluent. **B,** Narrow zone of attached gingiva in a 61-year-old patient. As the lower incisors were retracted and allowed to erupt, more gingiva was created incisogingivally. **C,** Woman with large osseous defect on the mesial of the lower left premolar. Note the significant probing depth. **D,** Preoperative radiograph of intrabony defect. **E,** Before orthodontic movement, guided tissue regeneration was used to create new attachment, and then eruption was used to eliminate any remaining osseous defect. Note nonresorbable membrane that was placed over the defect, which was allowed to heal, and was removed 8 weeks later. **F,** The tooth was extruded to resolve the intrabony defect. **G,** Buccal view of the preoperative intrabony defect. **H,** Buccal view of the new bone after the tooth was extruded and prepared for a restoration. **I,** Lingual view of the preoperative intrabony defect. **J,** Lingual view of the new bone after regeneration and tooth movement. **K,** Provisional restoration. The osseous defect has been corrected and the crown-to-root ratio has been improved. **L,** Radiographic appearance of the premolar. (**C–J,** Courtesy of Eric Saacks, Bondi Junction, Australia.)

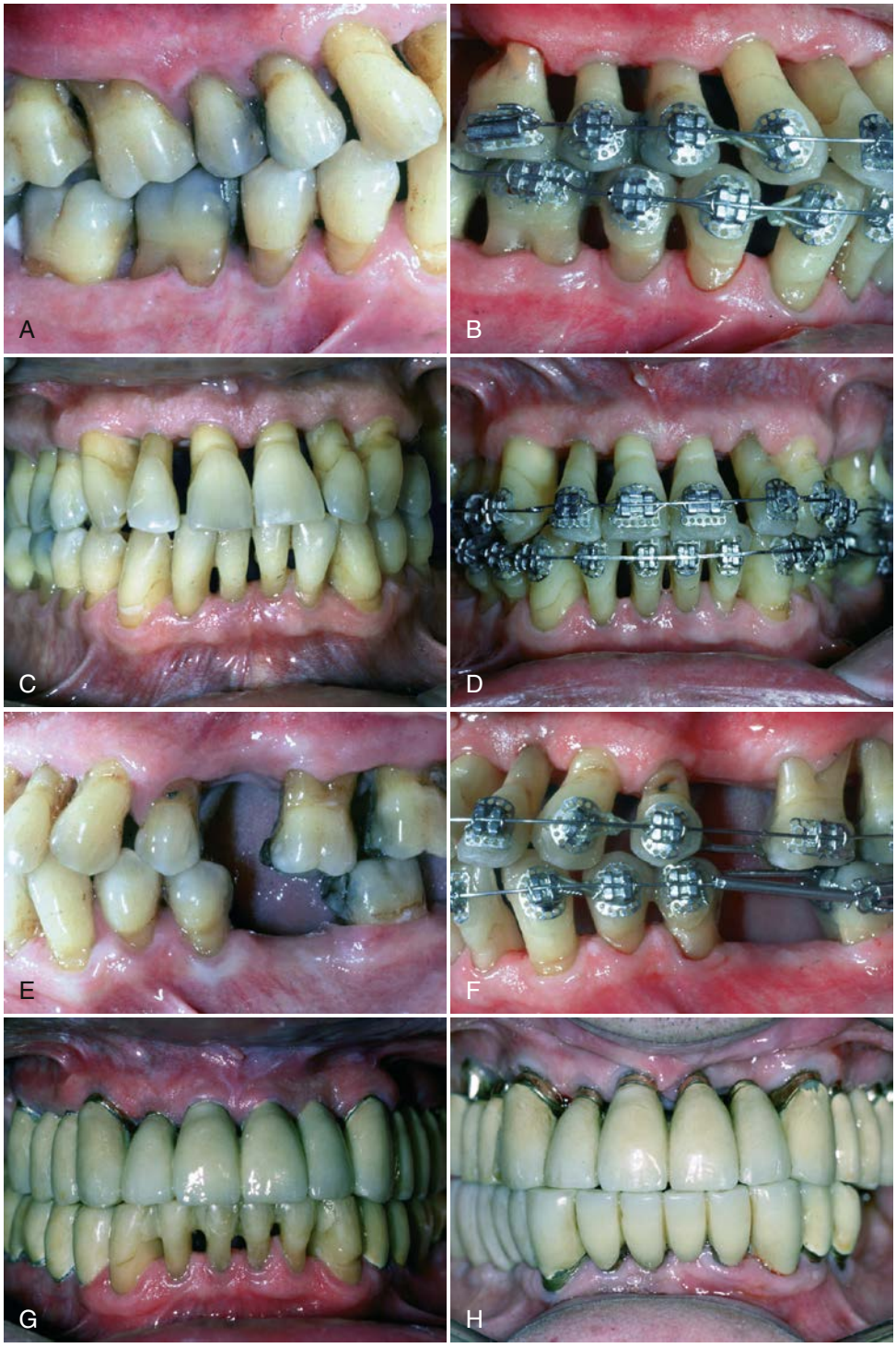


FIGURE 21-12 Clinical example of adult treatment objective 9: improvement or correction of the mucogingival and osseous defects; and adult treatment objective 10: better self-maintenance of periodontal health. **A, C, E,** Gingival form after control of inflammation and occlusal therapy with a bite plane in a 58-year-old man. In patients with posterior bite collapse, the posterior teeth are disarticulated with a Hawley bite plane appliance during scaling and root planing. **B, D, F,** Ahexial positioning of the teeth. Note the changes in gingival form. (The topography improved as the bite collapse was corrected and the teeth were properly positioned.) In addition, controlling gingival inflammation is easier after better tooth position has been established. **G,** Two years post treatment. **H,** Twenty-two-year follow-up. The patient has maintained his natural teeth.

cusp tips. In adults, the goal should be to level the crestal bone between adjacent cementoenamel junctions. It has been demonstrated that the need for osseous and mucogingival surgery may be diminished by favorable changes of the osseous and soft tissue topography during tooth movement. Therefore, attachments should be placed on individual teeth to allow the leveling of the attachment apparatus. This creates more physiologic osseous architecture with the potential to correct certain osseous defects.[13] During leveling stages, any teeth that have erupted above the occlusal plane should be grossly reduced occlusally. Also, continuous adjustment should be done to prevent the patient from contacting individual posterior teeth prematurely and causing occlusal trauma.

5. **Better self-maintenance of periodontal health.** The location of the gingival margin is determined by the axial inclination and alignment of the tooth. Clinically, it appears that improved self-maintenance of periodontal health occurs with proper tooth position.[14–16] This can be seen in adult patients as a result of correction of bite collapse and accelerated mesial drift (see Fig. 21-12).

Patients who need weekly periodontal maintenance during the initial leveling phases of therapy may require less frequent scaling and root planning as periodontal status improves. Poor tooth position and improper tooth preparation before irreversible restorative dentistry are causative factors that may contribute to periodontal disease. For better periodontal health on an individual pattern basis, teeth should be positioned properly over their basal bone support. In the nonsurgical management of skeletal Class III and Class II malocclusions, a delicate balance exists between periodontally desirable tooth positions and achievement of other nonsurgical treatment objectives.

1. **Aesthetic and functional improvement.** As stated previously, a plan should provide acceptable dentofacial aesthetics and allow for improved muscle function, normal speech, and masticatory improvements. This is possible when a therapeutic occlusion is provided that enables the anterior teeth to function as disarticulators and the posterior teeth to support the vertical dimension (Fig. 21-13).[11]
2. **Best outcome with least risk (using minimally invasive approach).** As technology increases therapeutic options, it is more incumbent on treatment providers to assess the advantages and disadvantages of each treatment option available. For example, certain borderline extraction situations can benefit from judicious therapeutic diagnosis that allows the providers and the patient to achieve a result equal to the outcome if extraction therapy was part of the treatment plan (Fig. 21-14).

DIAGNOSIS OF ADULT ORTHODONTIC CONDITIONS

Adult patients present numerous and demanding problems requiring both more specialized data pertinent to the patient's dental and orthodontic problem and a highly skilled mechanism to interpret it. The problem-oriented medical record (POMR) of Weed[17] takes the following into account:

1. Tremendous growth in medical knowledge
2. Resulting increase in specialization
3. Fragmentation of knowledge that has followed this increase
4. Consequential impossibility of relying on memory for adequate patient care

The needs that prompted development of the POMR have also influenced dentistry, particularly adult orthodontics. Figure 21-15 is a synthesized description of the steps necessary to include all variables into the adult patient's treatment planning process. The format and mechanism used for interpreting problems into orthodontic dental records are important steps in improving the practitioner's understanding of the adult patient and in providing treatment measures that can optimize the overall treatment result. Problem-oriented dental records significantly aid in making the appropriate diagnosis because they require that a problem list be developed to manage each problem.[16,17] This is an important improvement over the morphologically oriented diagnostic method that dates from the introduction of Angle's original classification of malocclusion.

A coordinated, logical approach is crucial to successful adult treatment. The following diagnostic steps are recommended.

Skeletal Differential Diagnosis

The correct skeletal differential diagnosis is the key responsibility of the orthodontic member of the interdisciplinary team. Although the lateral cephalogram has been relied on for the skeletal diagnosis in the anteroposterior and vertical planes, the frontal or posteroanterior (PA) cephalogram (Fig. 21-16) has not been routinely used by orthodontists to make an appropriate three-dimensional (3D) diagnosis. Recent advances in 3D radiographic imaging make the skeletal assessment in all planes of space more efficient and predictable. These 3D images allow the clinician to make both a thorough diagnosis and to use improved imaging for patient education. (See Chapter 11 of this textbook for examples of diagnosis enhanced by 3D imaging.) It should be noted that in a recent study of adult patients who sought orthodontic retreatment, it was reported that about 20% of retreatment adults have a maxillomandibular skeletal discrepancy of 6 mm or greater in the transverse dimension that requires jaw surgery (usually surgically assisted rapid palatal expansion [SARPE]) to achieve an optimal treatment outcome[5] (see Fig. 21-16).

Diagnoses in the anteroposterior and vertical planes are typically completed using a variety of cephalometric analyses,[19–25] and newer computerized software programs allow cephalograms to be digitized and analyzed, allowing an abundant amount of numeric information about the patient's skeletal jaw relations. Frequently, the analyses generate confusing and, at times, conflicting information regarding differential diagnosis of the patient's conditions.

In 1979, Jacobson presented the use of a simplified approach to aid in the diagnosis of skeletal disproportions that might be evident on a patient's lateral cephalogram.[26] This excellent report demonstrated the value of using normative, composite templates to aid in diagnosis and treatment planning. Since that time, two of America's most respected department chairs made statements that validated the use of templates to aid in differential diagnosis. In 1987, Lysle Johnston made the following straightforward statement:

> *Many clinicians stop tracing cephalograms at about the time their practices start to get busy. Ideally, a descriptive analysis should consist only of those measurements that are needed to illuminate the clinically significant idiosyncrasies of the patient at hand. Template analysis may seem an ideal solution.*[27]

Proffit pointed out in his 1991 textbook on surgical orthodontics that "the template may appear to be somewhat less scientific than a table of cephalometric measurements with

FIGURE 21-13 Clinical example of additional adult orthodontic treatment objective 11: aesthetic and functional improvement. **A–C,** Pretreatment intraoral photographs showing diminished occlusal function because of significant anterior open bite and posterior crossbite. At initial examination, patient had severe temporomandibular joint and myofascial pain because she was forced to "fulcrum" with second molar occlusion. **D–F,** Posttreatment intraoral photographs showing correction of malocclusion through a treatment sequence that included (*1*) splint therapy to improve comfort, (*2*) gingival grafting of lower anterior to protect from further recession, (*3*) orthodontic decompensation and alignment to prepare for jaw surgery, (*4*) orthognathic surgery with maxillary posterior impaction and mandibular auto rotation with mandibular setback, (*5*) completion of orthodontic treatment, and (*6*) stabilization with ongoing nightguard therapy. **G,** Pretreatment profile photograph. **H,** Posttreatment profile photograph illustrating favorable aesthetic improvement that accompanied functional improvement. **I,** Cephalometric superimposition showing overall dental, basal bone, and soft tissue changes that accompanied the interdisciplinary treatment plan. Note that temporomandibular joint and myofascial pain and headaches diminished as functional malocclusal relationships of the masticatory system were corrected.

standard deviations, but the template is a *visual analog* of a table and is just as valid." Proffit also states:

> *What a template does is place the emphasis on the analysis itself; that is, deciding what the distortions are, rather than on an intermediate measurement step that too often becomes an end in itself rather than just a means to an end.*[28]

For the past 30 years, one of the authors of this chapter has used a variation of the Jacobson template method of

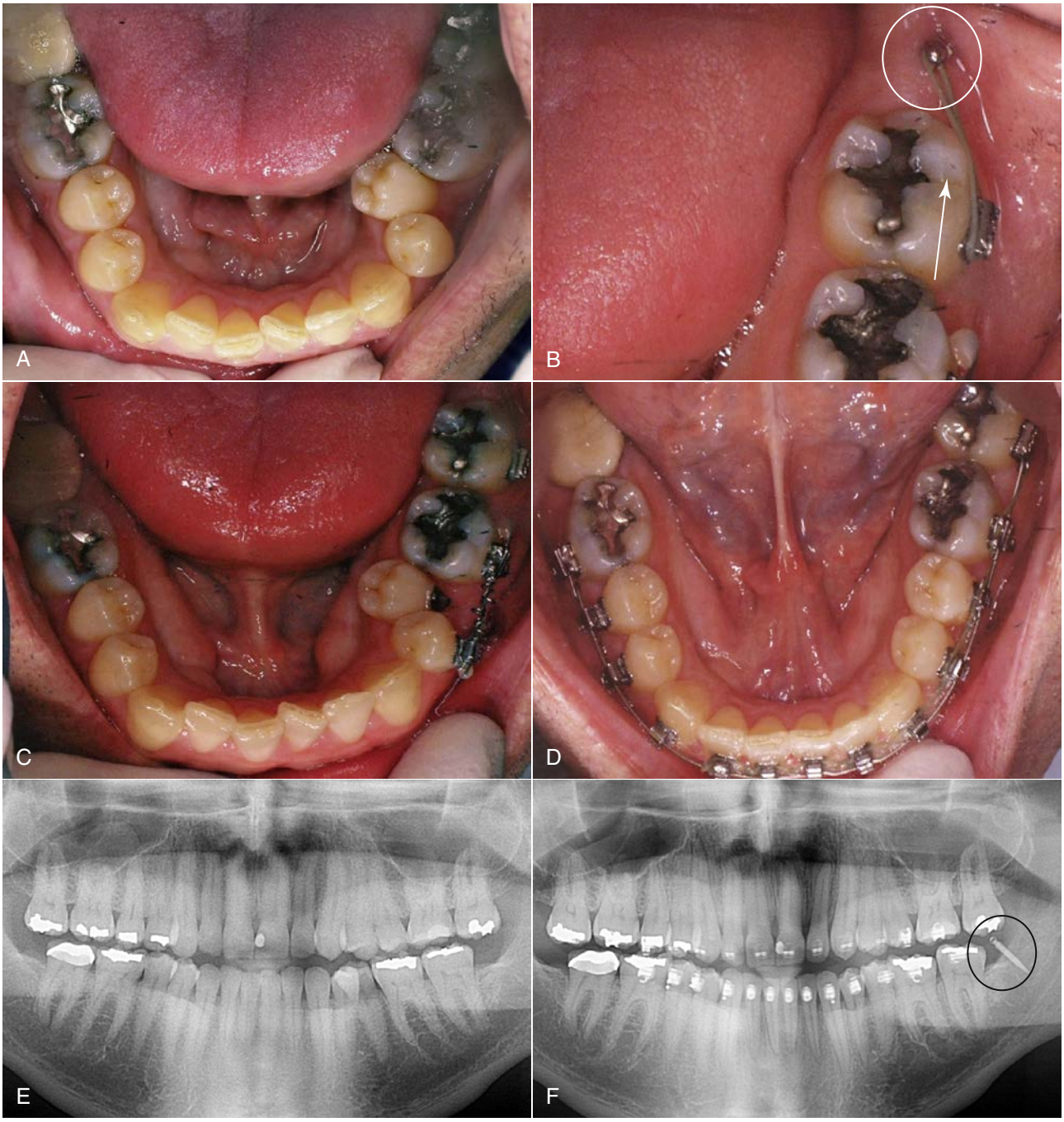


FIGURE 21-14 A, Intraoral occlusal photographic view of mandibular arch showing mild anterior crowding and collapse of the arch in the area of no. 20. One could assume that this arch collapse was a result of early loss of "k" without appropriate space maintenance and subsequent mesial drift of the posterior teeth in that area, disturbing the eruption path of no. 20. **B,** Close-up occlusal view of that area illustrating a minimally invasive approach using temporary anchorage devices (TADs) to allow posterior movement (reversal of arch collapse) of Nos. 19 and 18 to allow uprighting of #20 and improved axially positioning for better long-term periodontal health in that area (*white circle* at TAD and *white arrow* showing direction of movement). **C,** Lower occlusal view after 4 months of appliance activation. A small amount of room is becoming available for future movement of #20. **D,** Lower occlusal view after 12 months of appliance activation. Mechanotherapy included incorporation of TAD for distal anchorage and open coil spring at area of no. 20, and light enamel contouring created adequate space for no. 20 and preserved a healthy, natural dentition. **E,** Pretreatment panoramic radiograph illustrating the root proximity in the area of the collapse arch at no. 20. **F,** Progress radiograph illustrating TAD (*circle*) with favorable molar repositioning caused by mechanotherapy described in **D**.

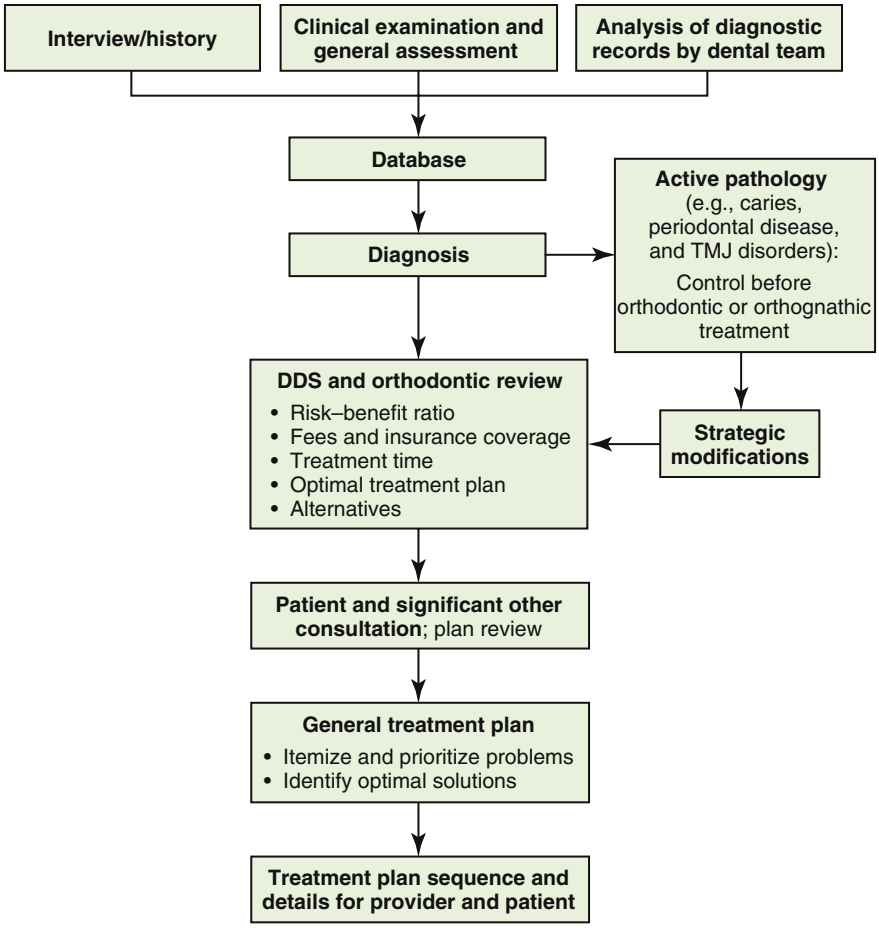


FIGURE 21-15 Treatment pathway for adult patients illustrating a *problem-oriented approach* and the decisions that are required to present and to accomplish an optimal treatment plan. *TMJ,* Temporomandibular joint.

superimposition; its use has been described as *template-guided diagnosis and treatment planning* (see Figs. 21-21 to 21-26). The method described in Chapter 23 of the current text incorporates the following concepts:

1. Standardized templates from the Bolton study provide a composite tracing of proportionate soft and hard tissue that can be of diagnostic value when compared with the patient's cephalometric tracing (for adults, the 18-year-old standard is used).
2. This superimposition technique acknowledges that the size of the patient's nose is a key factor in actual visualized, profile perceptions (e.g., a patient with a large nose and a moderate mandibular deficiency may appear to have a severe mandibular deficiency when viewed in profile because of nasal dominance).
3. The template method described and illustrated thoroughly in Chapter 23 is very "staff and patient friendly" because it allows rapid visualization of the skeletal discrepancy, which helps patients understand why surgical intervention may be needed in a variety of situations.
4. This method is called *template-guided diagnosis and treatment planning* because it provides an excellent orientation for the clinician to make decisions that will help guide the planning of both the skeletal and dental corrections "in the direction of balanced hard and soft tissue facial proportions" (Fig. 21-17).

Periodontal Differential Diagnosis

Periodontally involved or compromised patients who have experienced shifting migration, extrusion, or flaring or lost teeth can benefit from orthodontics designed to correct local causative factors, predisposing malpositions, and certain bony and periodontal pockets.[29,30] Clinical evidence in periodontics shows overwhelmingly that changing local environmental factors can improve periodontal health and reduce the frequency of long-term periodontal maintenance. Orthodontics is one of the most dramatic means available to modify local factors and site specificity of the disease process.

Patients who have already been affected by periodontal disease and have lost significant tooth support are at risk for recurrent episodes of active disease; this group is especially susceptible because of their past history. In the United States, 80% of older adults have experienced some degree of periodontitis, and 95% of older adults have moderate to severe periodontal disease. Movement of teeth for periodontally susceptible or previously compromised patients in the presence of inflammation can result in increased loss of attachment, irreversible crestal bone loss, or both. Fortunately, research and clinical studies have shown that dentitions with a history of periodontitis or teeth with reduced attachment height can be moved without significant loss of attachment.[31] Identification of the susceptible sites or areas and control of the inflammatory lesion are crucial to

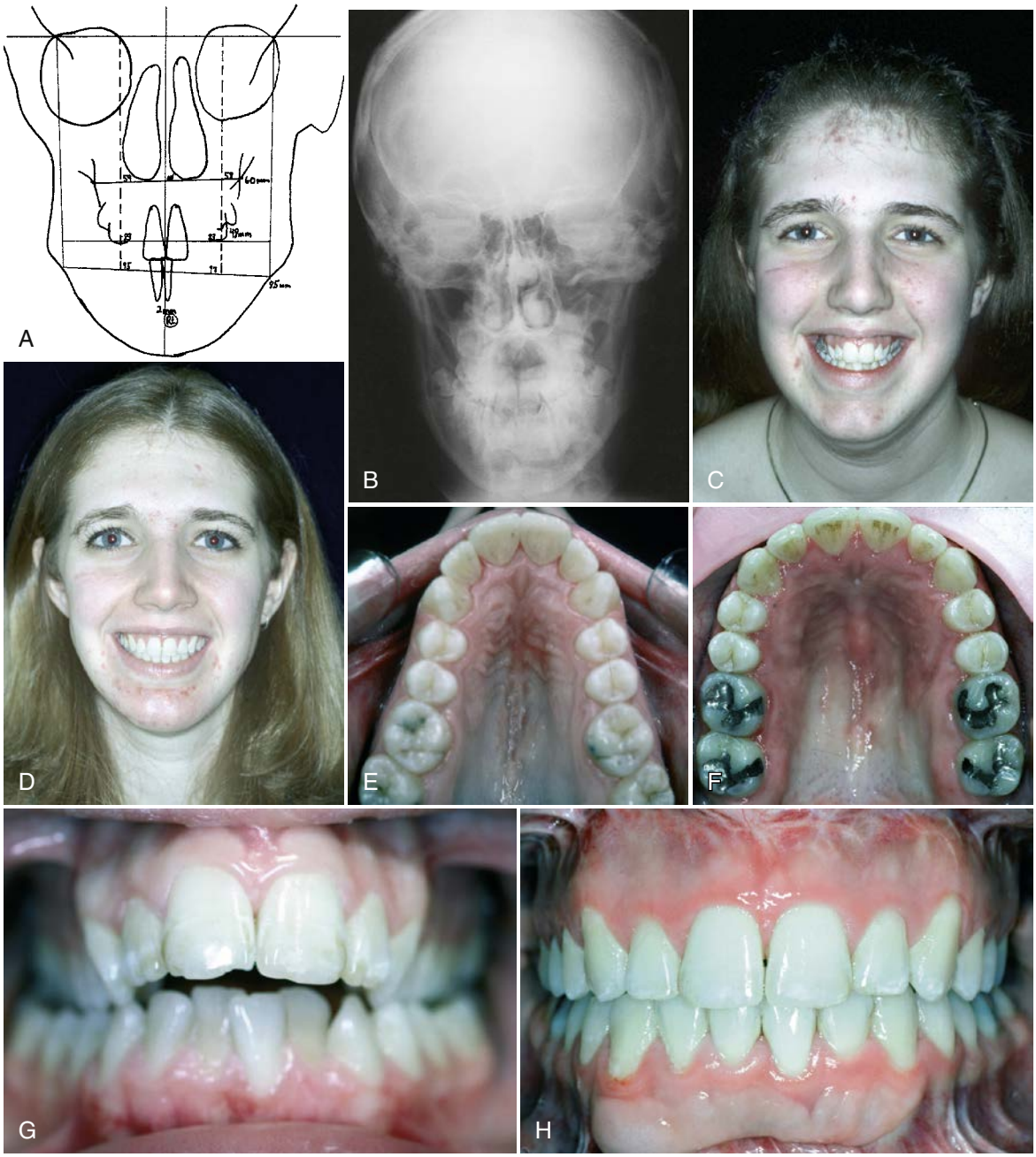


FIGURE 21-16 **A,** Rocky Mountain transverse analysis indicates a mandibular width (AG to AG) of 85 mm and a maxillary width (MX to MX) of 60 mm. The maxillomandibular difference is 25 mm, and the normal for this patient is 18 mm. Therefore, the expected/actual difference is 7 mm. Surgically assisted rapid palatal expansion may be considered a form of osteodistraction and has been found to be stable if large amounts (>7 mm) of expansion are required. **B,** Pretreatment posteroanterior cephalogram. **C–H,** Class II, Division 1, 14-year-old white female patient (skeletal age, 17 years), with an A point–nasion–B point angle of 7 degrees, a high mandibular plane, a severe transverse skeletal discrepancy with a differential index of 7.6 mm because of a wide mandible and a narrow maxilla, bilateral posterior crossbites, and 10-mm overjet. **C,** Note the large negative space at the corners of the mouth on the pretreatment photograph. **D,** Note the increased buccal tooth visibility and elimination of the negative space after a surgically assisted rapid maxillary expansion to correct the transverse dimension and achieve a natural or broader arch form. **E,** Pretreatment maxillary arch. **F,** Posttreatment natural or broader arch form. **G,** Pretreatment anterior view. **H,** Post treatment. A second-stage surgery consisted of Le Fort 1 osteotomy with 5 mm of maxillary impaction allowing for mandibular autorotation and a 7-mm advancement genioplasty for profile improvement. Surgically assisted maxillary expansion has been shown to be more stable than two or more segments of Le Fort 1 osteotomy with expansion. The treatment strategy addressed the type and magnitude of the transverse skeletal deficiency, vertical maxillary excess, growth status, dentofacial aesthetics, stability factors, and periodontal health.

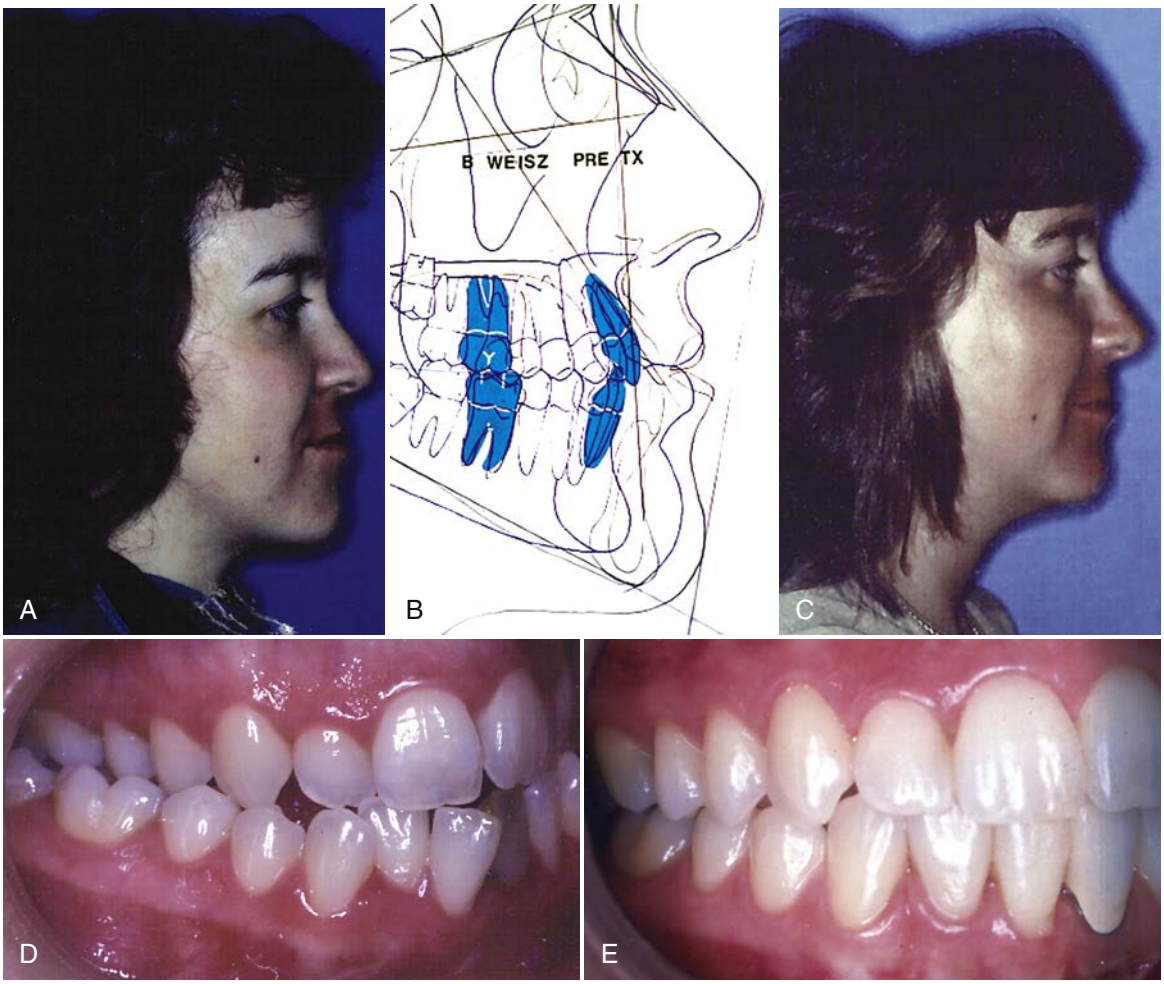


FIGURE 21-17 Differential diagnosis of Class III problem using Bolton template technique to guide the diagnosis and treatment plan. **A,** Pretreatment profile of a Class III patient before orthodontic/surgical treatment. **B,** Bolton template (*blue*) superimposed on the patient tracing: nasal tip with the nasal tip and forehead parallel. **C,** Profile after orthodontics and surgery. **D**, Right occlusion before orthodontic decompensation and surgery. **E,** Right occlusion after orthodontic decompensation and surgery.

successful therapy. All patients, however, will have some degree of inflammation, and it is important to ensure that periodontitis remains stable or quiescent throughout orthodontic treatment.

To manage the periodontal issues in adult treatment effectively, the orthodontist must make an accurate assessment of the patient's potential for bone loss or gingival recession during orthodontic tooth movement. A common problem occurs when the orthodontist assumes that the general dentist will provide skilled inflammation control for a patient with incipient periodontal disease. After months of tooth movement and clenching or grinding instigated by movement interferences, dental radiographs may reveal significant bone loss. Consequently, regaining control of periodontal inflammation is harder than controlling it from the beginning. The orthodontist needs to monitor every adult case closely and collaborate with the periodontal specialist to properly treat adult patients. The orthodontist must bear in mind that periodontal disease continues to be a large percentage of dental malpractice claims. The basis for the litigation appears to originate from a number of sources:

- Poor case selection
- Poor office procedure in insisting that prospective patients be periodontally stable
- Patient reluctance for or refusal of periodontal checkups
- Dentists' acquiescence in the patient's neglectful behavior
- Ill-defined protocol between orthodontist and general dentist (or periodontist)[32]

Therefore, appropriate management of several factors is needed to prevent negative periodontal sequelae during orthodontic treatment:

1. Awareness and vigilance of the orthodontist and the staff must be heightened.
2. Awareness and vigilance of the patient must be frequently reinforced.
3. Awareness of risk factors related to periodontal breakdown must be understood by and reviewed with the patient (see Chapters 22 and 30).
   - General factors
   - Family history of premature tooth loss (indication of immune system deficiency in resistance to chronic bacterial infection associated with periodontal disease)
   - General health status and evidence of chronic diseases (e.g., diabetes)
   - Nutritional status
   - Current stress factors
   - Periodontal biotype[33]
   - Life stage of women[34]

- Local factors
- Tooth alignment (e.g., marginal ridge, cementoenamel junction relationship)
- Plaque indices
- Occlusal loading
- Crown-to-root ratio
- Grinding, clenching habits (parafunctional activity)
- Restorative status

One of the most overlooked considerations in the management of adult patients is the impact of patient susceptibility to periodontal disease coupled with the preexisting habit of smoking. Until 2003, many orthodontists' health history did not include a question on the adult medical history form asking if the patient used tobacco products and a follow-up question about the frequency of use. In 1996, the American Academy of Periodontists published a position paper titled "Tobacco Use and the Periodontal Patient."[35] In this well-researched paper, the American Academy of Periodontists reference 97 published papers that verify the following:

> *Clinical and epidemiological studies support the concept that tobacco use is an important variable affecting the prevalence and progression of periodontal diseases, such as adult periodontitis, refractory periodontitis, and ANUG [acute necrotizing ulcerative gingivitis]. Several studies have demonstrated that the severity of periodontal disease appears to be related to the duration of the tobacco use, smoking status, and amount of daily tobacco intake.*[35]

If there is no health history question regarding tobacco use and no follow-up in which the patient is informed of the risk of accelerated bone loss and a poor prognosis for long-term health and function, both the patient and the providers could have an undesirable outcome—the patient could because a large investment in his or her dental treatment has high probability of failure, and the providers could because they could be named in legal action for lack of diagnosis and lack of informed consent. The added risk of orthodontic appliances reducing oral hygiene capacity and the "jiggling trauma" of changing tooth positions in a mature neuromuscular environment could lead to rapid periodontal breakdown and blame placed on the orthodontist for "moving the teeth too fast," which hypothetically may lead to expensive restorative procedures and an angry patient.

Temporomandibular Joint Differential Diagnosis

Because the signs and symptoms of TMD often increase in frequency and severity during adult treatment, it is imperative that orthodontists be familiar with their diagnostic and treatment parameters (see Chapter 7). A study by Howard shows that the majority of 3428 TMD patients were between the ages of 15 and 45 years (mean age, 32.9 years).[36] However, it is important to note that "craniomandibular disorders are self-limiting, or they fluctuate over time as suggested by declining incidence with age."[37] Unfortunately, knowledge regarding the natural history or course of TMD is limited.[38,39]

In a prevalence study, Schiffman and Friction found that the group of patients whom they treated for TMD problems were divided into several types:[40]

Muscle disorders	23%
Joint disorders	19%
Muscle–joint combinations	27%
Normal	31%

It is important to note that prevalence data frequently overstate the clinical significance of the problem because many patients have mild signs that may be transitory and are better left untreated. The orthodontist treating adults would be wise to have a separate TMD questionnaire to supplement other health history information as part of the initial evaluation process. The goals of such a questionnaire would be to answer the following questions:

1. What type of TMD does the patient have? Muscle, joint, combination, psychosomatic? Where is most of the problem located?
2. Is there pain? What is the degree of pain? What is the frequency of pain? Is the pain chronic or acute? "Chronic pain syndromes are defined as persistent pain that lasts more than 6 months with significant associated behavioral and psychosocial factors."[41]
3. Has there been previous treatment for this problem? What treatment? How long? How effective? Any medication?
4. What is the patient's understanding of his or her condition? Does he or she need a program designed to educate?
5. Is there an occlusal factor that may be exacerbating the problem? Are there occlusal habits that are known or apparent?

The differential diagnosis may not be established until there are more assessments, records, and imaging. However, the orthodontist must have a clear perspective of diagnostic possibilities. An excellent discussion of TMDs is summarized in Chapter 13.

> *Each of these five factors [symptoms, conditions of the dentition, systemic health, aesthetics, and finances] needs to be considered before an appropriate occlusal treatment plan can be developed. It is important to realize that the priority of the factors may be different for the patient and for the therapist. When symptoms are not severe, finances and aesthetics will often be more important concerns of the patient. At the same time, however, the dentist may believe that the condition of the dentition is more important. In any case, the patient's concerns must always remain foremost in the development of a successful treatment plan.*[42]

Although there is no established cookbook for 100% reliability in the management of temporomandibular disorders, Dr. Okeson has presented a series of diagnostic algorithms of appropriate steps in the management of a variety of TMDs.[42]

In summary, an orthodontist treating adults plays a key role in diagnosing a skeletal problem that may require surgery, a periodontal condition that may worsen as a result of tooth movement, and a TMD that requires its own differential diagnosis and plan. Diagnostic steps described previously provide an overview to lead the orthodontist and the team of dental providers to a comprehensive treatment plan that has the highest probability of achieving the desired goals of treatment (Fig. 21-18).

CLINICAL MANAGEMENT OF THE INTERDISCIPLINARY ADULT THERAPY PATIENT

Before the initiation of treatment for the IAT patient, there are several skills that are required beyond those commonly used in the orthodontic treatment of child and adolescent patients. Although not every adult patient requires each of the considerations listed next, treatment will progress more easily if the concepts in the section are understood and the orthodontist

FIGURE 21-18 A–C, Pretreatment intraoral photographs illustrating previous temporomandibular joint surgery, condyle remodeling, and anterior open bite. D–F, Posttreatment intraoral photographs illustrating improved function and bite closure using orthodontics to prepare for surgery, orthognathic surgery, and long-term splint therapy. G, H, Pretreatment facial photographs. I, J, Posttreatment facial photographs show minimal facial change because of patient's desire to proceed with single-jaw surgery instead of double-jaw surgery. K, L, Pretreatment panoramic radiograph and cephalogram. M, N, Posttreatment panoramic radiograph and cephalogram. Note that surgery resolved open bite with Le Fort surgery only. Mandibular advancement surgery would have been ideal for profile change but was not used to prioritize reduced risk to fragile and remodeled condyles. *SNB,* Sella-nasion-supramentale angle.

is already skilled at providing the necessary strategy at the appropriate time.

Biomechanical Considerations

Control of Occlusion

There are three crucial ways to control occlusal forces during appliance therapy: disarticulation or disclusion of teeth moved, selective grinding with a high-speed drill, and modification of mechanotherapy for a periodontally susceptible or compromised individual.

Disarticulation. The Hawley bite plane is used for disarticulation (and for diagnosis of TMD patients), to establish centric relation at the acceptable vertical dimension, and as necessary throughout orthodontic treatment to prevent excessive tooth mobility. During the leveling stages, the bite plane is used (in conjunction with posterior sectional archwires) to allow teeth to move free of occlusal forces. The appliance is worn at all times except while the patient is eating or sleeping. A significant biomechanical advantage of disarticulation (or disclusion) in an adult is that it allows mesially inclined tooth crowns to tip distally to an upright position with only slight mesial movement of their root apices, thereby creating space in the mandible distal to the canines for alignment of crowded lower anterior teeth. If the posterior teeth are not disarticulated, occlusal forces may cause the buccal segment roots to move mesially so that no space is gained distal to the canines during uprighting. Note also that uprighting of posterior teeth, free of occlusal forces, requires much less time and reduces the hazard of mesial root movement and root resorption. The use of disarticulation appliances is particularly important when treating patients who have bone loss or short facial height with strong masseter activity or patients with evidence of posterior bite collapse requiring molar uprighting (Fig. 21-19).

Selective Grinding. The typical adult malocclusion is characterized by posterior teeth that have moved and tipped mesially (accentuated mesial drift) and bite collapse. There is less wear on the mesial marginal ridges than on the distal marginal ridges of mesially inclined posterior teeth. After these teeth start to upright, the mesial marginal ridges need to be reshaped so that the occlusal table will be perpendicular to the long axis of the tooth. Many teeth need to be extruded to correct osseous defects and level the crestal bone but must not be allowed to remain in premature contact after eruption has occurred.

Therefore, at each appointment, the bite plane is removed, and selective grinding is done as necessary to ensure simultaneous bilateral contact with the posterior teeth when in centric relation. After a malocclusion involving mesially inclined posterior teeth has been uprighted, the posterior segments will not be stable unless selective grinding is used to reshape the occlusal surfaces along with any required operative and restorative dentistry to ensure simultaneous bilateral contact along the long axis when in centric relations. The objectives of selective grinding then are to allow leveling of the attachment to crestal bone and leveling of the marginal ridges to achieve simultaneous bilateral occlusion.

Modification of Mechanotherapy. With traditional multibonded techniques in non–periodontally involved patients, both arches are usually bonded, and full continuous archwires are placed. In adult patients (for reasons stated previously), the authors delay appliance placement on both the maxillary and mandibular anterior segments. This approach is well accepted by adults, who invariably prefer no brackets on the anterior teeth during the initial 9 to 12 months that it takes for posterior segments to be axially aligned and for transverse correction to be made. The objective is to set up the posterior occlusion before placing appliances on the anterior segments—these are moved last. After posterior teeth are aligned and axially positioned, the bite plane is removed, and the upper and lower anterior teeth are bonded. Space closure is begun and anterior alignment is completed. In Class II patients in whom upper premolars may need to be extracted, the extractions can be delayed, and then maxillary canines can be immediately retracted into the extraction site, minimizing the time for maxillary space closure. It is more difficult to traumatize posterior segments by parafunctional habits after the teeth have been axially positioned. However, if significant bruxism and clenching during stressful periods cause increased mobility, an immediate impression should be taken for a bite plane. The patient should be given a bite plane for disarticulation and referred for inflammatory control (scaling and root planning) as well. After mobility patterns have been reduced, mechanotherapy can be continued.

Removable Appliances. Within the removable appliance group are appliances designed for both diagnostic purposes and tooth movement. The diagnostic appliances are generally bite planes and splints. The acrylic splints are acrylic and wire bite planes with several features that are helpful, depending on the patient's problem. Some of the ways in which these appliances prove useful follow:

- For neuromuscular deprogramming[25,41–43]
- As therapeutic adjuncts to reduce joint inflammation, pain, and parafunction[7,15,44–46]
- As intermediaries during corrective orthodontic therapy to avoid transitory occlusal trauma or treatment-induced TMJ symptoms[22,47–50]

Removable appliances thus serve the patient as a crutch during specific treatment stages. Some patients whose problems cannot be therapeutically resolved without surgery instead will choose to wear a bite plane splint permanently to help maintain a symptom-free joint. Although tooth-movement appliances are in a separate group, on some occasions the appliance can be diagnostic initially and then therapeutic after the diagnostic phase has been completed.

The authors use the following appliances for adults:

- The sagittal appliance for distalizing buccal segments or individual teeth, thereby reducing the need for extraoral anchorage (headgear). This type of appliance with a spring-loaded jackscrew is particularly useful for patients who have had maxillary mesial buccal segment drift and thus require distal movement to create or to regain space. Frequently, a bite plane is used with this appliance to open the bite and allow more rapid distal tooth movement without occlusal or intercuspal interference.
- The slow palatal expander (Schwartz plate) for mild dental transverse discrepancy cases. This appliance has the potential for the addition of an anterior or posterior bite plane to minimize the resistance of occlusal or intercuspal interference during the occlusal correction. Also, bite planes on a palatal expander can deprogram an existing mandibular functional shift. However, skeletal transverse problems in adults resist treatment with this type of appliance and usually require surgical intervention for a stable correction. The most reliable and stable procedure for correction of maxillary skeletal transverse problems is the SARPE procedure.[51]

- The phase I appliances (placed before full appliances) used for reducing the length of time the patient must wear fixed appliances, for testing patient acceptance of the treatment plan, and for initiating tipping movements that may be more efficiently handled by a removable appliance. Orthodontists using phase I removable appliances appreciate the rapid tooth movement initiated by them. In addition, the use of this type of appliance frequently provides early insight into the patient's degree of motivation that would otherwise be unattainable without a much longer treatment commitment.
- Bite plane therapy is used in periodontally susceptible or already compromised bite collapse cases (and for diagnostic aspects in the treatment of TMD patients) and for disarticulation to find the centric relationship at the acceptable vertical dimension. During this phase of treatment, an evaluation can be made of the patient's response to the removal of adverse local factors, helping the clinician determine not only the prognosis but also the best therapy for the patient.[48,52]

Preorthodontic inflammation and occlusal control can be managed by a general dentist or the periodontist, but an orthodontist should monitor this phase and evaluate patient response over time before initiating the orthodontic phase of treatment. In addition, obviously, all caries control should have been completed, overhanging margins corrected, overbulked contacts reduced, irregular marginal ridges reshaped, and endodontic procedures completed.

Fixed Appliances. Accurate predictable interarch and intraarch tooth positioning in most adults requires the use of fixed orthodontic appliances. With continued advancement in bracket design featuring prescribed torque and other angulations, orthodontists now have the capability of more precise tooth movement with shorter periods of therapy.[53] Advancements in boding techniques, new compositions, and types of fixed

FIGURE 21-19 **A–C,** Pretreatment intraoral photographs showing Class II malocclusion with a 9-mm overjet. This patient was initially treatment planned for mandibular advancement jaw surgery before a second opinion with the recommended nonsurgical correction illustrated. **D–F,** Posttreatment intraoral photographs with occlusal correction through reversal of posterior bite collapse through early extraction of no. 30. **G,** Periapical radiograph showing collapsed area from loss of no. 30 about age of 10 years because of "deep caries." This resulted in no. 31 migrating mesially and nos. 29, 28, and 27 erupting distally and worsening the Class II posterior relationship.

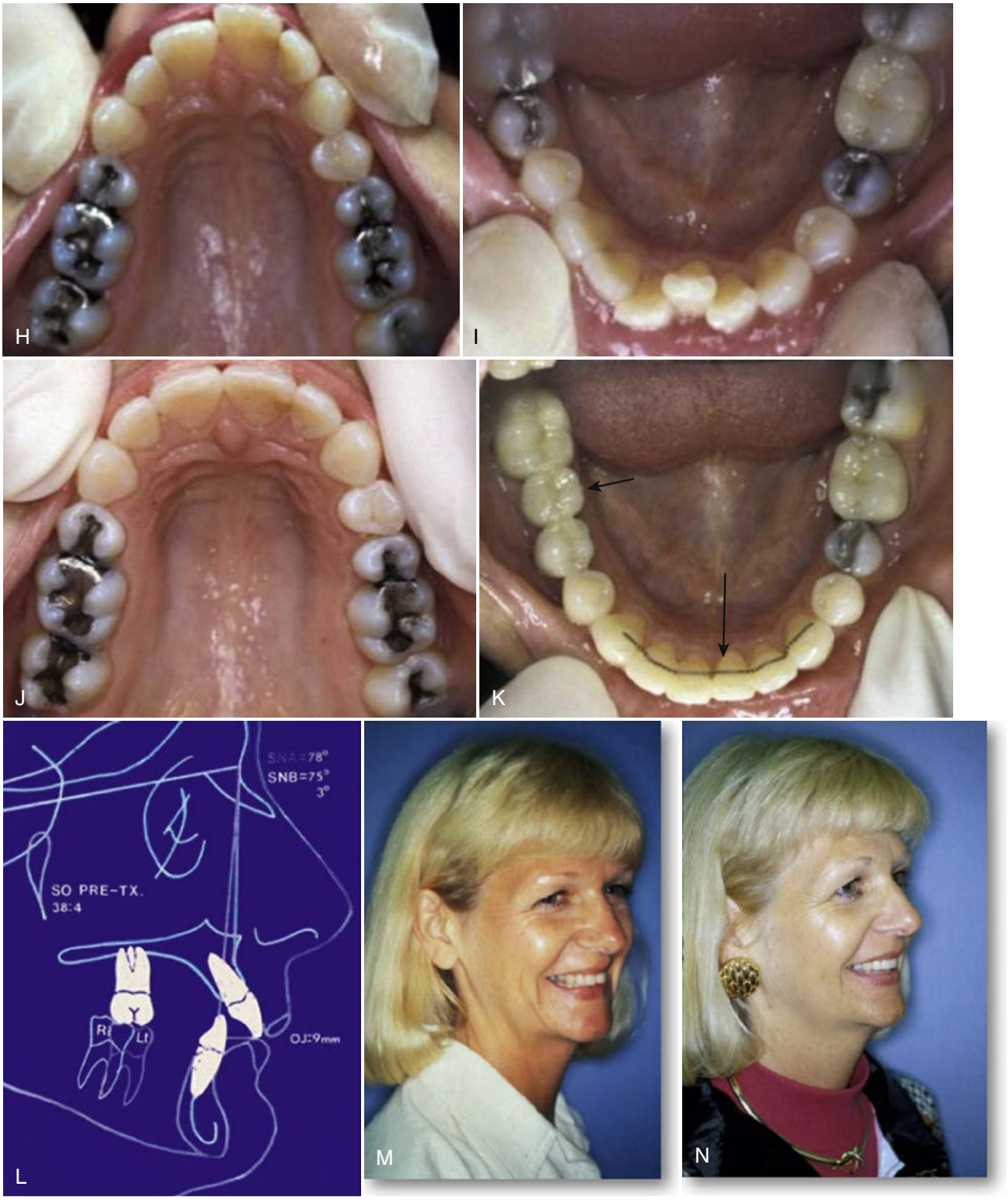


FIGURE 21-19, cont'd H, I, Pretreatment intraoral occlusal photographs illustrating narrowed maxillary and mandibular arch. J, K, Posttreatment intraoral occlusal photographs. The *arrows* in *K* illustrate the important stabilization features of the correction of the long-standing bite collapse—replacement of no. 30 and bonded lingual arch to stabilize lower incisors and support lower lip position. L, Pretreatment cephalometric tracing illustrating that the Class II problem is more dental than it is skeletal. M, Pretreatment ¾ smiling photograph showing lower lip "entrapment" forcing the incisors to move lingually over time. N, Posttreatment ¾ smiling photograph showing lower lip now supported by new, stabilized lower incisor position. Lower incisors were moved forward as part of the posterior bite collapse reversal as room was created for missing no. 30 restoration.

appliances have become available, thereby adding to the orthodontist's armamentarium of mechanotherapy options. Pretorqued and preangulated stainless steel labially affixed appliances are described elsewhere in this text and are generally regarded as the most predictable appliances for accomplishing desired tooth movements in all three planes of space. However, plastic brackets, porcelain brackets, mini-brackets such as the Hanson SPEED appliance, self-ligating brackets (see Chapter 24), and lingual or invisible appliances (see Chapters 25 and 26) have additional appeal to adult patients because of their more cosmetic appearance.

Although plastic and porcelain brackets have cosmetic advantages, they also present significant mechanical disadvantages for certain types of tooth positioning problems. From an analysis of the patient's records, the orthodontist must determine whether the problem is treatable (i.e., treatment goals achievable) within the limitations imposed by the use of cosmetic appliance choices. Recent advances have been made regarding the effectiveness of a progressive series of plastic aligners made from simulated movements on computer-generated models. (See Chapter 26 for a thorough review of aligner therapy and its application for adult patients.) These removable aligners using computer-simulated movements to guide the progressive application of desired forces can be effectively used in a variety of adult treatment situations. The initial choice of appliances can be altered during treatment, but it is wise nonetheless to provide for an alternative treatment plan (and appliances if needed) that will accomplish the treatment goals efficiently and predictably, especially when treating adults whose expectations of treatment may be extremely high.

Use of Temporary Anchorage Devices. Miniscrew implants for orthodontic anchorage have become a useful clinical tool that can simplify orthodontic treatment. Tooth movement that has been considered beyond ordinary mechanotherapy (e.g., asymmetric extractions, bimaxillary protrusions, molar intrusions, distalization, mesialization) can be achieved with minimal patient cooperation with the use of miniscrews (see Chapter 20).

Orthognathic surgery may not be needed in certain open bite and bimaxillary protrusion cases. Patients may be presented with an option other than orthognathic surgery. Chapter 20 in this textbook has excellent clinical examples of the effectiveness of TADs in the management of complex dentoalveolar and borderline surgical skeletal conditions.

Occlusal Control. Occlusal control during the leveling phase of treatment in advanced cases can be accomplished with the aid of the Hawley bite plane and posterior sectional arches (see Fig. 21-8). To prevent excessive forces from bruxism and clenching, it may also be necessary to insert bite planes during stressful periods throughout the course of orthodontic care.[45] Fortunately, after the posterior segments have been axially positioned and the transverse correction made, it is more difficult for the patient to traumatize posterior segments by parafunctional habits.

Periodontal Accelerated Osteogenic Orthodontics. The periodontally accelerated osteogenic orthodontic (PAOO) technique has been suggested to enhance orthodontic treatment by acceleration of treatment, better stability, and improvement of the periodontium.[54] Drs. Blasi and Pavlin illustrate several examples of the effective use of minimally invasive approaches to accelerate tooth movement. It has been observed that the duration of appliance wear can be particularly important to adult orthodontic patients (see Chapter 31).

Periodontal Management During the Orthodontic Tooth Movement

Patients who have already been affected by periodontal disease and have lost significant tooth support are at risk for recurrent episodes of active disease; this group is especially susceptible because of their past history. In the United States, 80% of adults have experienced some degree of periodontitis, and 95% of older adults have moderate to severe periodontal disease.[55] Movement of teeth for periodontally susceptible or previously compromised patients in the presence of inflammation can result in increased loss of attachment, irreversible crestal bone loss, or both. Fortunately, research and clinical studies have shown that dentitions with a history of periodontitis or teeth with reduced attachment height can be moved without significant loss of attachment.[56] Identification of the susceptible sites or areas and control of the inflammatory lesion are crucial to successful therapy. All patients, however, will have some degree of inflammation, and it is important to ensure that periodontitis remains stable or quiescent throughout orthodontic treatment. Additional important considerations on the periodontal–orthodontic interface are described by Drs. Vanarsdall, Blasi, and Secchi in Chapter 22 of this textbook.

Significance of Tooth Mobility

Many experimental studies have demonstrated that traumatic lesions in the presence of a healthy periodontium do not initiate loss of attachment or periodontitis. Investigations using beagles[57] and monkeys[58] have improved our understanding of the relationship between inflammation and occlusal trauma.

Studies in the beagle by Svanberg have shown that trauma from occlusion that had created increased tooth mobility can also cause increased vascularity, increased vascular permeability, and increased osteoclastic activity during a traumatic phase lasting approximately 60 days.[59,60] After a stable, permanent hypermobility was established, vascularity and osteoclastic activity returned to normal. These findings led Svanberg to suggest that only through measurements taken at intervals can one determine the existence of progressively increasing mobility that may be diagnosed as trauma from occlusion. Jiggling occlusal forces have been found to aggravate active periodontitis, accelerate loss of connective tissue attachment, and possibly diminish gain in reattachment after periodontal treatment.[61,62] A study evaluating the association between trauma from occlusion and severity of periodontitis concluded that teeth with increased mobility have greater pocket depths and less bone support than teeth without these findings.[63] A University of Pennsylvania study evaluated initial alignment and level of teeth and also determined the effect of probing depths on healthy teeth.[32] The results indicated that test teeth in this study did not show significant change (i.e., the increase in the plaque or sulcular bleeding indices between the initial appointment and the appointment at which maximal tooth mobility was recorded) to ensure that any increase in probing depth could not be caused by increased periodontal inflammation. Eleven patients served as their own controls. The test teeth that became mobile demonstrated an increase in probing depth by an average of 1.09 ± 0.53 mm between the initial appointment and the appointment at which the maximal tooth mobility value was recorded. Therefore, it was concluded that during orthodontic alignment and leveling, teeth with significant increase in mobility will also have an increase in probing depth and that severely rotated teeth will have a greater increase in mobility.

The findings of this preliminary study indicate that increased tooth mobility has a detrimental effect on the periodontium and that increased probing depth indicates an increased periodontal risk such as an increase in the patient's susceptibility to periodontal disease. Therefore, subgingival microbiota should be removed during periods of increased mobility, and appropriate steps should be taken to prevent further tooth mobility in patients who have already demonstrated periodontal susceptibility.

Periodontal Preparation of Adults before Orthodontic Therapy

Successful adult orthodontic treatment for many patients will depend on the periodontal preparation before treatment and the maintenance of periodontal health throughout all phases of mechanotherapy. Gingival inflammation must be eliminated by acceptable oral hygiene measures and removal of accretions on the teeth (usually by scaling and root planning). The occlusion also must be controlled during periods of stress and severe bruxism throughout orthodontic treatment so that occlusal trauma[49,64] and excessive tooth mobility will be prevented.[65] The Hawley bite plane is used for disarticulation as necessary throughout treatment (see Fig. 21-8).

Smoking Cessation. Oral health providers can have a significant impact on patient tobacco cessation. Established tobacco intervention protocols, such as those advocated by the National Cancer Institute, can be easily applied in the clinical setting. Providers should apply the 6 As:

1. Ask—Systematically identify all tobacco users at every visit.
2. Advise—Strongly urge tobacco users to quit. Advice should be clear, direct, and personalized for each patient.
3. Assess—Determine willingness to make a quit attempt.
4. Assist—Aid the patient in quitting. Tell the patient to remove tobacco products from his or her environment and to avoid situational cues, such as alcohol consumption or socializing with active smokers, that may facilitate unplanned relapse.
5. Arrange—Schedule follow-up contact. Follow-up contact should occur soon after the quit date, preferably during the first week. A second follow-up contact is recommended within the first month. Schedule further follow-up contacts as indicated.
6. Actions during follow-up—Congratulate success (if tobacco reuse has occurred, review circumstances and elicit recommitment to total abstinence). Remind the patient that a lapse can be used as a learning experience. Identify problems already encountered, and anticipate challenges in the immediate future. Assess pharmacotherapy use and problems. Consider use or referral to more intensive treatment.[54,66–72]

Gingival Tissue. The orthodontist should make a clinical distinction between thin, delicate gingival tissue and normal or thickened tissue that is in the labiolingual plane.[73,74] The thin, friable tissue is more prone to undergo recession during orthodontics than normal or thick tissue (see Chapter 22).

In addition, if a minimal zone of attached gingival exists, particularly on abutment teeth, it is prudent to place free gingival grafts to help control inflammation before orthodontic treatment begins. It is important to note that the orthodontist controls the timing of referral for grafting procedures. The prognosis for successful gingival grafts and prevention of bone loss is better if these are done before any recession has taken place.

Osseous Surgery. As a rule, orthodontics should logically precede definitive osseous surgery. The optimal approach is as follows: (1) complete the orthodontic therapy, (2) establish a stable occlusion, and (3) wait a minimum of 6 months before requesting the periodontist to intervene for definitive periodontal procedures.

Inflammation Control. Several visits with a periodontist or hygienist will not suffice to prepare a periodontally compromised adult properly for orthodontic correction. Meticulous root surface preparation and curettage are required. In patients with significant bone loss, furcation involvement, and severe pocket depths, it may be necessary for the periodontist to perform open-flap procedures[75] for removal of diseased gingival tissue and proper root surface preparation before orthodontic therapy. Better visualization and access may allow deeper pocket areas and tortuous root configurations to be root planed properly. Before open-flap debridement in cases with advanced periodontal disease, control of adverse occlusal factors using the Hawley bite plane is essential.

Gingival bleeding provides the most reliable index for determining the presence of clinically significant gingivitis,[76] and before placement of appliances, no significant gingival bleeding should occur on gentle probing.

Healthy tissue does not bleed when a thin Michigan probe is gently inserted into the sulcus. To keep the patient free of inflammation during treatment, root planning and curettage should be scheduled as frequently as necessary to prevent significant bleeding. Also, to assess disease activity, the tendency to bleed should be evaluated at each appointment. If this inflammation control is not rigidly adhered to, irreversible bone loss will inevitably result in the periodontally susceptible patient.

Behavioral Management Orthodontist and Staff Preparation for Adult Interdisciplinary Patient Management

Adult patients present an ongoing challenge before, during, and after the prescribed treatment. To meet this challenge and improve the quality of care for each patient, orthodontists treating adults should integrate several steps in the areas of behavioral and clinical management into their professional activities.[77]

1. **Advanced continuing education courses**

Because a larger percentage of adult patients (1) have conditions that require interdisciplinary therapy and (2) require treatment in areas of the emerging knowledge, it is helpful to seek continuing education that provides an opportunity to update the orthodontic clinician's knowledge and skills in the specialty areas of periodontics, orthognathic surgery, TMD management, and restorative dentistry. In addition, opportunities exist to play an efficient role in the delivery of care.

2. **Refined consultation techniques**

Methods of consultation or case presentation vary from orthodontist to orthodontist.[78,79] When reviewing records with adults, particularly patients who require interdisciplinary dental therapy (60%–70%), it is valuable to use a consultation format designed to inform and educate. For patients with tooth size discrepancies, borderline or atypical extraction problems, restorative uncertainties, or some unique surgical situations, using a diagnostic setup on mounted models is usually informative and sometimes quite revealing to both the orthodontist and the patient.

For cephalometric analysis of a patient with a skeletal disharmony, the Broadbent-Bolton transparencies or the Jacobson

templates are helpful. They have proved to be graphic tools that enable the orthodontist to translate cephalometric data into a visually understandable reference for the patient who is considering orthodontics and orthognathic surgery (see Figs. 21-21 and 21-27).

Also, the chart enables orthodontic/orthognathic patients to consider anticipated changes resulting from combined therapy in a more informed way. A patient with skeletal disharmony, particularly one with a borderline skeletal-dental problem, requires a clearer explanation regarding therapeutic alternatives than do many other patients.[80,81]

Educating and informing the patient about the presence and significance of moderate or advanced periodontal disease can be a delicate process if the referring dentist has not discussed this with the patient.[82–84]

Frequently, the orthodontist's hygienist may have made the patient aware of the existence of this problem at the initial examination. The orthodontist will then be in a better position to refer the patient to a periodontist for needed therapy. A recent publication provides excellent schematics that help to educate patients.[85]

Other innovative consultation devices (i.e., a before and after photographic bulletin board, videotaped interviews with similarly treated adults) can greatly enhance the patient's knowledge and confidence. Grateful patients who have successfully undergone complex treatment will often provide a reassuring person-to-person consultation with a prospective patient regarding their own experiences with a proposed treatment modality. This is a frequently used educational procedure for patients who are considering orthognathic surgery. A patient who has already received the projected care is uniquely qualified to fill any knowledge voids, especially in areas of family care and inconvenience, work interference, rate of recovery, and pain and discomfort. Finally, adult patients who are about to undergo multidisciplinary therapy seem to appreciate dual or multi-specialist consultation so that all questions and information exchanges can be addressed on one occasion, thereby increasing patient trust regarding a shared level of agreement and permitting unanimous approval and understanding of upcoming treatment.[37] Dual or multi-specialist consultations also aid in organizing the timing and sequencing of steps needed to complete the planned therapy.

3. **Appliance modifications for adult treatment to reduce concern about appearance**[6,74,86]

Numerous appliances are available to provide for tooth movement within a biologically sound range. Two basic appliances, removable and fixed, are widely used. Within the categories are a multitude of choices, and it is logical that each orthodontist will use the one that is both comfortable for the patient and reliable in accomplishing the desired treatment goals.[87]

Key elements of diagnosis and patient preparation have been discussed in this chapter thus far. Incorporation of the skeletal diagnosis, periodontal assessment, and TMJ management lead to the treatment plan and to patient education. After these steps have been taken, the adult interdisciplinary team and the patient should be ready to identify the specific sequence of steps that need to be taken to create a balanced occlusion, with healthy supporting tissue, harmonious facial proportions, and restorations to repair damaged or lost teeth.

SEQUENCE OF ADULT INTERDISCIPLINARY THERAPY

As has been discussed, the diagnostic considerations are more complex for the AIT patient, and the treatment planning decisions vary greatly between adults and their adolescent counterparts. Many points discussed in the previous two sections may seem self-evident to the practicing orthodontist, but although they may be self-evident, it must be emphasized that it is the orthodontist who is frequently placed in the position of treatment plan director. For example, decisions regarding extraction or nonextraction and surgery to correct or tooth movement to camouflage skeletal problems are key orthodontic responsibilities. Along with these decisions, the orthodontist, as treatment plan director, must establish the sequence of implementing the treatment plan—who does what when and how the orthodontist communicates the sequence and the rationale of the sequence to the patient and to the other providers for their input and modification. The written sequence then becomes the blueprint for the restoration of the skeletally and dentally imbalanced, diseased, mutilated, or worn stomathognathic system.

An important advantage of categorizing patients into provider groups is in the area of patient education, particularly as it relates to sequence of treatment. After the diagnostic process, the practitioners involved must be precise in outlining the steps of treatment and the rationale for these steps to their patients. Patients under dual provider and multiple provider care must also be educated to the plateaus of treatment progress and how these relate to their well-being.[5]

1. Active disease
2. Disease arrested
3. Disease controlled
4. Contributing structural malrelationships corrected
5. Periodontal defects corrected
6. Restorative and reconstructive dentistry completed
7. Optimal oral health maintained

Failure to adequately educate patients in this area will result in high rates of noncompliance and potential litigious action. Informed consent is essential.

Evaluation of the Skeletal Component of the Malocclusion

One of the first questions that must be asked to determine the sequence of AIT is, "Does the skeletal problem require surgical intervention to create a stable correction?" Patients with skeletal imbalances who plan to undergo orthognathic procedures require our utmost attention and care (see Chapter 23). Although they have the potential to receive the greatest benefits from combined therapy, they also are exposed to the greatest risk. Each step in this outline (see Box 23-2) is essential for consistent success and quality outcomes. Note: Step 16 in the sequence, posttreatment (6–12 months) records, is often skipped. However, it is crucial for refinement and improvement of future team efforts. This step can become an important in-house approach to the team's continuing education protocol.

Interdisciplinary adult patients require treatment sequencing information, as outlined below, so that standardized communication of the treatment plan is available to the patient and all providers.

The key elements are as follows (see Fig. 21-15):

- Orthodontic diagnosis
- List of dental problems

- Sequence of treatment to manage problems
- Rating of severity
- Rating of prognosis with treatment (include a discussion of limiting factors)
- Addressing patient concerns

Periodontal Preparation

Does the periodontal problem require intervention to create a stable correction? One of the most frequently overlooked aspects of orthodontic treatment for adults is the patient's level of periodontal health before the placement of fixed orthodontic appliances. Frequently, the orthodontist is responsible for determining the level of periodontal health so that thorough measures can be undertaken to stop the advancement of periodontal destruction during tooth movement.

For the orthodontist, the periodontal health of adult patients may be classified in one of three categories: incipient periodontal disease, moderate periodontal disease, or advanced periodontal disease. Each level of periodontal disease requires different therapy with consistent monitoring throughout fixed mechanotherapy to ensure the disease is arrested and inflammation is controlled.[76,88] Careful compliance with these steps will help ensure no loss of the supporting alveolar bone during orthodontic treatment.

Although various types of procedures have been recommended to control periodontal disease, it should be remembered that conservative approaches are effective in the treatment of cooperative patients with periodontal disease, and no orthodontic appliances should be applied to patients who are undergoing periodontal treatment. Adult orthodontic patients generally have 12 to 30 months of fixed appliance therapy, and these appliances interfere with convenient oral physiotherapy with the additional potential negative effects of iatrogenic transitional occlusal trauma induced during certain states of orthodontic tooth movement.

Before beginning tooth movement, inflammation should be reduced by root planning and curettage. As pointed out in Chapter 22, it may be necessary to perform open-flap curettage for direct visualization. All members of the interdisciplinary dental team must accept responsibility for adequate oral physiotherapy and mechanical maintenance of gingival health in deep pocket areas. This is achieved by root planning, tooth polishing, and curettage throughout tooth movement as frequently as necessary to keep the periodontium free of significant inflammation. Bleeding on gentle instrumentation or problem is the most reliable clinical indication of inflammation. If, during curettage, blood pools in the gingival margin surrounding a tooth, too much inflammation will be present to perform tooth movement for a periodontally susceptible patient. The frequency of visits during tooth movement will be determined not by orthodontic adjustments but by the necessity to control soft tissue inflammation during appliance therapy. This could become a biweekly basis in the 60- to 70-year-old patient. A periodontally susceptible patient should never be placed on an automatic 2- to 3-month recall program for removal of subgingival microbiota, as has been suggested in the literature.[89] Periodontally susceptible patients who are undergoing orthodontic treatment may have very rapid repopulation of the gingival bacteria and require appointment-to-appointment monitoring.

Periodontally involved or compromised patients who have experienced shifting migration, extrusion, flaring, or lost teeth can benefit from orthodontics designed to correct local causative factors, predisposing malpositions, and certain bony and periodontal pockets.[29] Clinical evidence in periodontics shows overwhelmingly that changing local environmental factors can improve periodontal health and reduce the frequency of long-term periodontal maintenance. Orthodontics is one of the most dramatic means available to modify local factors and site specificity of the disease process.

DENTOALVEOLAR COLLAPSE: ORTHODONTIC AND RESTORATIVE CONSIDERATIONS IN THE ADULT INTERDISCIPLINARY PATIENT

Because alveolar and basal bone are fundamental to successful oral health, it is not surprising that any new findings regarding bone healing and bone remodeling would be important to dental specialists. This interest becomes particularly true with an increased number of aging patients who seek oral rehabilitation (Fig. 21-20). The recent study by Osterberg and Carlsson[90] found that as fixed partial denture technology improved, so also did patients' desire for implant restoration as part of oral rehabilitation. Before the reported success and acceptance of the Branemark system of dental implants in the 1980s, oral rehabilitation was a very complicated process incorporating the principles of perioprosthetics that were well documented by Morton Amsterdam in 1974.[11]

In 1974, when very sophisticated perioprosthetics were required to provide fixed restorations to avoid removable dentures, dentists were reminded of the role that orthodontics can play in optimizing the restorative outcome. "It should be noted that when an orthodontic malocclusion also exists in the presence of disease, more frequently than not, it will be necessary to correct or modify those functional aspects of the malocclusion which may be acting as contributing factors to the disease process."[11]

However, current treatment planning strategies for patients with missing teeth and secondary dentoalveolar collapse (DAC) frequently neglect to incorporate important occlusal improvements and orthodontic biomechanical components of orthodontics to the treatment plan. Bone remodeling advantages made possible by tooth movement are often bypassed. For example, in Misch's recently published 1088-page textbook on implant dentistry, there are only two pages that discuss the value of orthodontics in cases of DAC, and these pages discuss orthodontics only as an aid for implant site development (accelerated eruption before extraction and implant replacement).[91]

Many dental providers encourage expedited treatment plans for patients with missing teeth, and as a result, key orthodontic considerations have been undervalued and therefore generate minimal attention to those providing expertise on implants and their restoration. The key considerations that are often overlooked in oral rehabilitation procedures are:

1. **Updated understanding of principles of bone physiology (originally Wolff's law), particularly as it relates to the alveolar bone:**

"There are no mathematical optimization rules for bone architecture; there is just a biological regulatory process, producing a structure adapted to mechanical demands by the nature of its characteristics, adequate for evolutionary endurance."[92] In this regard, Atwood documented the degree of bone

FIGURE 21-20 **A,** A youthful appearance has a well-defined mandibular line and good definition between the face and neck. **B,** An aged face has a less-defined mandibular line and lack of definition between the face and neck. **C,** Pretreatment profile photograph of a Class II mandibular deficiency patient treated with camouflage. The patient declineda mandibular advancement procedure that was recommended. **D,** Ten-year postorthodontic camouflage occlusal correction. Note the degree of facial aging, related to the mandibular deficiency, that occurred in the 10-year follow-up period. (**A** and **B** from Koury ME, Epker BN. The aged face: the facial manifestations of aging. *Int J Adult Orthod Orthognath Surg* 1991;6(2):81–95.)

loss that can occur, over time with tooth loss in the anterior segment of the mandible (Fig. 21-22).

2. **Epigenetic components of alveolar development and maintenance (functional matrix hypothesis [FMH] of Moss)**

"More precisely, the FMH claims that epigenetic, extraskeletal factors and processes are the prior, proximate, extrinsic, and primary cause of all adaptive, secondary responses of skeletal tissues and organs. It follows that the responses of the skeletal unit (bone and cartilage) cells and tissues are not directly regulated by informational content of the intrinsic skeletal cell genome per se. Rather, this additional, extrinsic, epigenetic information is created by functional matrix operations."

3. **Tooth movement–induced alveolar remodeling**[12,93] According to the equilibrium theory of tooth position of Weinstein with new concepts of orthodontic tooth movement must be taken into consideration over time:

Conclusion 1. Forces exerted on the crown of the tooth by the surrounding soft tissue may be sufficient to cause tooth movement in the same manner as that produced by orthodontic appliances.

Conclusion 3: Differential forces, even when they are of small magnitude, if applied over a considerable period of time can cause important changes in tooth position (DAC).[94]

Masella and Meister's work coupled with Weinstein et al.'s work in 1963 brings us to a clearer understanding of the orthodontist's role as bioengineer:

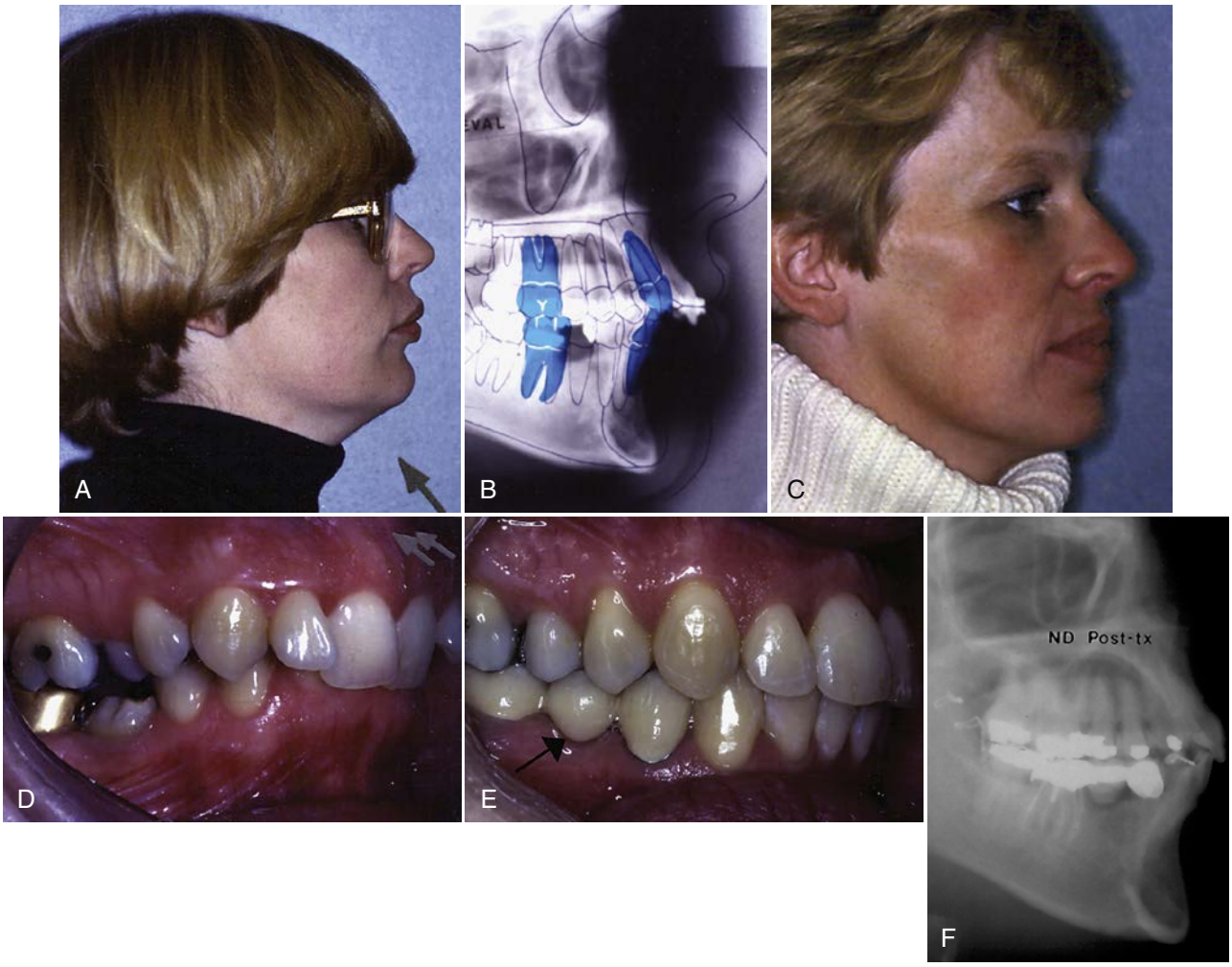


FIGURE 21-21 **A,** Profile photograph taken after preliminary leveling (same as cephalogram in **B**). Evidence of maxillary dentoalveolar protrusion and mandibular deficiency is apparent. Two different treatment plans make sense: the one chosen will depend on what goals are prioritized. The *arrow* illustrates "youthful" throat–chin soft tissue contour. **B,** Cephalogram after 4 months of incisor reangulation (decompensation) to allow reassessment of treatment plan using Bolton template to guide decision regarding extraction of the upper first premolars and camouflage of moderate mandibular deficiency or "slow aging" of the lower third of the face by correction of mandibular deficiency with mandibular advancement. **C,** Profile photograph taken after orthodontic treatment and mandibular advancement surgery. Although a camouflage treatment plan would have worked, the patient indicated the importance of chin position improvement. The Bolton template superimposition guided the treatment plan and indicated that mandibular advancement would be appropriate based on landmark comparison to the proportionate facial guidelines of the superimposed template. **D,** Pretreatment right occlusion illustrating anterior crowding; retroinclined maxillary incisors; deep bite; retained deciduous right mandibular second molar; and a Class II, Division 2 malocclusion. **E,** Posttreatment comparing right occlusion with **D.** The patient desired to position her chin more forward to avoid the potential "aging effects of chin deficiency," which the patient observed as her mother aged. **F,** The patient chose a conventional bridge to replace the missing lower right second premolar shown with an *arrow* in **E.**

Adaptive biochemical response to applied orthodontic force is a highly sophisticated process. Many layers of networked reactions occur in and around periodontal ligament and alveolar bone cells that change mechanical force into molecular events (signal transduction) and orthodontic tooth movement (OTM). Osteoblasts and osteoclasts are sensitive environment-to-genome-to-environment communicators, capable of restoring system homeostasis disturbed by orthodontic mechanics. Five micro-environments are altered by orthodontic force: extracellular matrix, cell membrane, cytoskeleton, nuclear protein matrix, and genome. Gene activation (or suppression) is the point at which input becomes output, and further changes occur in all 5 environments. Hundreds of genes and thousands of proteins participate in OTM.[95]

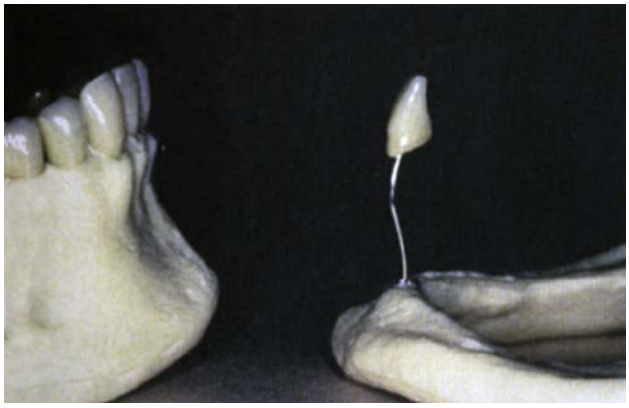


FIGURE 21-22 Alveolar bone requires the function of the dentition to avoid progressive atrophy. This vivid photograph illustrates how much alveolar bone can be lost over time when there is no dentition and no function. Preserving the dentition and the alveolar process are key responsibilities of general dentists and orthodontists.

Because dentists frequently refer patients with missing teeth to the orthodontist for treatment planning, the orthodontist becomes the "gatekeeper" in the following:

1. Determining whether the collapse will be reversed or camouflaged (see Fig. 21-23 and Boxes 21-2 and 21-3)
2. Setting up the spacing for tooth or root replacement
3. Aiding in the provision of temporary tooth replacement

Application of Principles

Understanding the physiologic principles of alveolar bone remodeling and the role that tooth movement can play in guiding bone preservation and regeneration can alter the choices made when patients proceed through the treatment planning process. Prevention of alveolar collapse (AC) through alveolar bone preservation and reversal of AC through alveolar bone regeneration apply to patients in the deciduous dentition, mixed dentition, and adult dentition. Often the orthodontist is the key person in the treatment plan for patients with missing teeth and should consider the implications of AC when developing the treatment plan.

FIGURE 21-23 In patients with congenitally missing teeth, orthodontists frequently have to decide whether the "collapse" into the area of congenital absence should be reversed and stabilized with implants or camouflaged with substitution of adjacent teeth. **A–C,** Pretreatment intraoral photographs of a 14-year-old young woman who has congenital absence of nos. 7 and 10.

FIGURE 21-23, cont'd D–F, Posttreatment intraoral photographs of a 16.5-year-old young woman from A to C in whom the decision was made to "reverse" the anterior arch collapse and to move the canines into their correct anatomic and functional positions. G, Pretreatment smile showing unaesthetic spacing in the areas of the missing lateral incisors. H, Posttreatment smile illustrating dental, occlusal, and alveolar stabilization after implant replacement with crowns for teeth nos. 7 and 10. Favorable aesthetic balance was achieved with the decision to reverse the dentoalveolar collapse. I, Posttreatment panoramic radiograph illustrating properly positioned implants allowing long-term health and function.

BOX 21-2 Characteristics of Dentoalveolar Collapse

- Missing tooth or teeth
- Migration of adjacent teeth into area of missing tooth or teeth
- Migration of adjacent teeth is in horizontal, transverse, or vertical plane.
- Time impacts degree of tooth migration.
- Alveolar changes (atrophy) related to the missing teeth are progressive until new equilibrium positions are achieved with accompanying bone loading.

BOX 21-3 Etiology of Dentoalveolar Collapse

Collapse secondary to:

- Early loss of deciduous teeth (especially c's and e's—this may not be addressed until adult treatment)
- Delayed exfoliation of deciduous molars
- Ankylosis of deciduous molars
- Missing first molars
- Missing second premolars
- Missing upper anteriors (congenital absence or trauma)
- Missing lower anteriors (congenital absence or trauma)
- Unilateral or bilateral cleft repair
- Cancer and bone resections

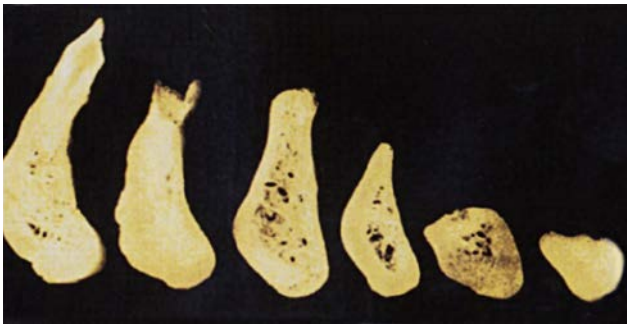


FIGURE 21-24 Atwood's work illustrates the significant alveolar atrophy that accompanies the absence of teeth and roots in the anterior portion of the mandible. This documentation should be a constant reminder of the responsibility of all members of the dental profession to preserve the natural dentition or replace teeth that are lost with implants as soon as is possible.

The following cases illustrate the application of the principles of alveolar preservation and collapse reversal for varying age groups. Figures 21-24 through 21-26 demonstrate how components of DAC impact the many aspects of the malocclusion and frequently require a treatment plan to include implants and restoration to stabilize DAC reversal.

Conclusion for Adult Interdisciplinary Therapy Patients with Missing Teeth and Dentoalveolar Collapse

Patients with a diagnosis of AC due to tooth loss or congenital absence should be notified of the etiology of this aspect of their malocclusion and guided to the most predictable methods of successful oral rehabilitation. Dental teams will benefit from bringing each specialty's unique perspective into treatment planning for patients who present with characteristics of DAC. Most DAC cases require:

1. Etiologic recognition
2. Early collapse reversal through orthodontic measures
3. Alveolar bone stabilization through implant placement when facial growth is complete
4. Followed shortly thereafter with occlusal stabilization through restoration

Although this seems to be a simple concept to understand and to apply, the range of situations that are part of the AC "umbrella" of conditions has, to this point, been undocumented in the orthodontic literature. As recent as 1996, a survey published in the *Journal of Clinical Orthodontics* presented their findings related to three conditions that frequently occur when there is AC:

1. "When questioned about their preferred approach to manage patients with congenitally missing maxillary laterals, nearly 80% of orthodontists preferred substitution of canines for the missing laterals."[72] Current studies indicate a 2.2% incidence of missing lateral incisors.[96]
2. When questioned about their preference for congenitally missing lower second premolars, the majority chose forward movement of molars.[72] Current studies indicate a 3.4% incidence of second premolar agenesis with lack of a single premolar being most frequent; absence of three premolars occurs least frequently.[96]
3. Again, for the condition of congenital absence of a lower incisor, the majority of surveyed orthodontists preferred to accept the occlusal discrepancy and deep bite to space opening.[72]

Implant reliability and interdisciplinary interaction have improved the awareness of the orthodontists to the implant option when teeth are missing. In assessing the cases illustrated in the Applications of Principles section (see Figs. 21-24 through 21-26), the concept of AC is better understood as an etiologic factor complicating the severity of many features of a malocclusion. It is the authors' opinion that as the short- and long-term impacts of DAC on the occlusion and the alveolar bone become better understood, dentistry will be better able to provide optimal care by achieving the goals of occlusal stability, dental and facial aesthetics, and bone preservation.

The issues related to sequencing treatment for patients with skeletal problems, susceptibility to periodontal disease, TMDs, and DAC with restorative requirements add to the list of intraprovider planning requirements. Clearly, improved technology, patient education, and interdisciplinary teamwork allow dental providers to achieve outstanding outcomes and achieve treatment goals of function, aesthetics, stability, and health through the use of reliable methods that thereby reestablish longevity to the "mutilated" stomatognathic system.

Evaluation before Debonding or Debanding

The orthodontist should consider the following before debonding or debanding:

1. **Root parallelism:** The orthodontist must verify this by panoramic, periapical radiographs or a cone-beam scan (for select situations.) This assessment can be done during progress records about three-fourths of the way through treatment.
2. **Coincidence of centric relation and habitual occlusion:** This can be done through clinical assessment with the aid of deprogramming appliances.

FIGURE 21-25 **A,** Pretreatment photograph of the anterior dentition of a young adult who had congenital absence of nos. 13, 20, and 29. The missing posterior teeth and the subsequent extraction of deciduous "K and T" resulted in progressive dentoalveolar collapse of the posterior and anterior portion of her dentition. **B,** Posttreatment of the anterior dentition after 2 years of orthodontic treatment to reverse the posterior and anterior collapse and open the space for the missing lower second premolars. Current dental therapy would use implants to replace the missing premolars; 20 years ago, the collapse reversal was stabilized with bridges on the right and left posterior. **C,** Pretreatment panoramic radiograph showing the missing premolars—nos. 13, 20, and 29. Overeruption of the upper and lower incisors can be seen as well. **D,** Pretreatment ¾ posed smile photograph illustration of the retroinclination of the incisors secondary to the progressive dentoalveolar collapse and overeruption. **E,** Posttreatment ¾ posed smile photograph illustrating improved aesthetics of reangled incisors as the arch collapse was reversed and the interincisal angle improved.

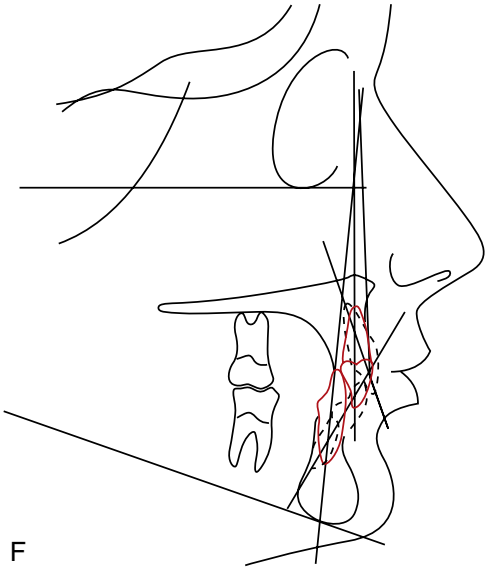


FIGURE 21-25, cont'd F, Cephalometric superimposition illustrating the improved interincisal angle. The pretreatment interincisal angle was 176 degrees, and the posttreatment interincisal angle was 137 degrees.

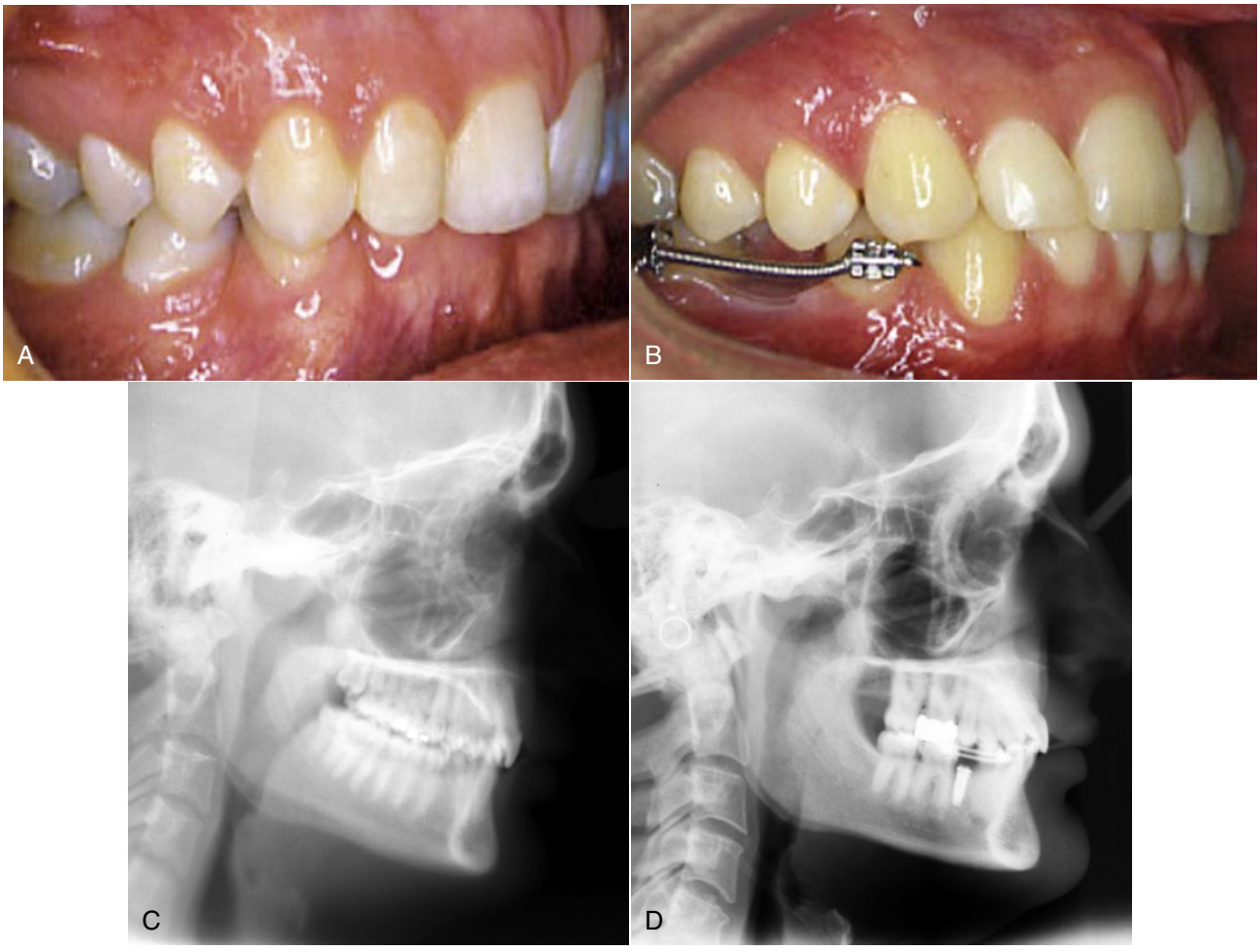


FIGURE 21-26 A, Pretreatment side view of right posterior occlusion showing a Class II canine relationship but with a Class I molar relationship. The upper incisors are very upright, and the bite is very deep with apparent lack of the second premolar. B, Postorthodontic side view of the right posterior occlusion after orthodontics to reverse the dentoalveolar collapse, reangle the incisors, "open the bite," and prepare for posterior restoration to stabilize anterior and posterior occlusion. C, Pretreatment view of lateral cephalogram illustrating the "collapsed" dentoalveolar relationship with very upright incisors with minimal vertical "stops." D, Postorthodontic interincisal relationship corrected with a normal interincisal angle, which will provide long-term vertical stability in the area of the incisors.

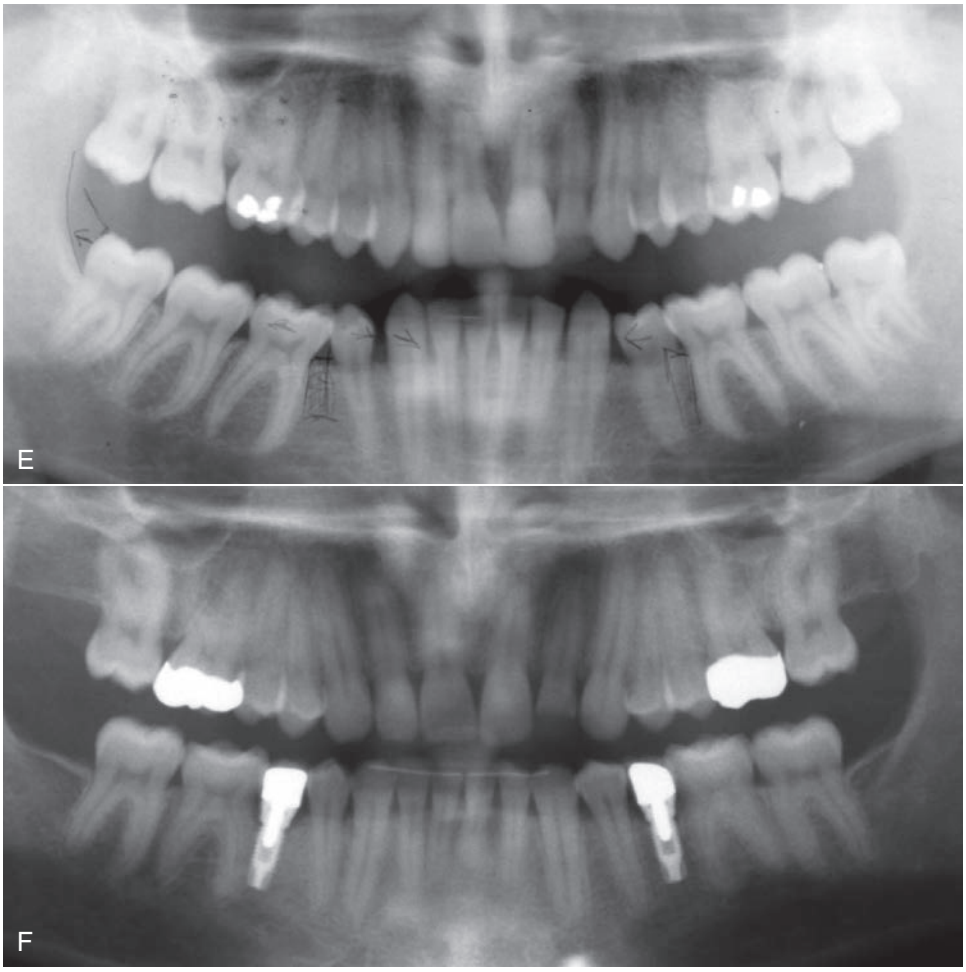


FIGURE 21-26, cont'd E, Pretreatment panoramic radiograph showing the congenital absence of the lower second premolars. The third molars are present, and some of the third molars are only partially erupted. The third molars require removal to allow collapse reversal and occlusal correction with space reopening in areas of nos. 20 and 29. This will also allow proper replacement of the missing second premolars. F, Posttreatment panoramic radiograph showing the space opened at nos. 20 and 29 with implants placed to preserve the alveolar housing and crowns placed to preserve the corrected occlusion.

3. **Incisal guidance:** The orthodontist should evaluate this clinically, in some cases, with mounted study models.
4. **Joint symptoms:** These should be assessed clinically and appropriate treatment or referral provided as needed.
5. **Excursive movements:** The orthodontist examines these clinically and augments examination in some cases with mounted models.
6. **Patient input:** The orthodontist should encourage patients to discuss existing dentofacial aesthetics before appliance removal. Many patients point out concerns that may be correctable with minor modifications in mechanotherapy.
7. **Reaffirmation of restorative commitment:** The orthodontist should review this commitment with the patient and discuss it with the restorative dentist and the periodontist. The timing of the restorative procedures and the coordination of restorative treatment with the orthodontic retention schedule are very important.
8. **Reevaluation of periodontal considerations:** The orthodontist reevaluates clinically with radiographs; the orthodontist should also check for mobility and fremitus, perform probing, and make a careful gingival tissue assessment.
9. **Reevaluation of anterior and posterior tooth size discrepancies:** The orthodontist informs the patient and the general dentist of additional measures that may be necessary to resolve tooth size discrepancies without adversely altering the posterior occlusal relationships or incisal guidance (i.e., acid-etched add-on technique).
10. **Anticipated retention problems:** The orthodontist should make plans and provisions and should enter them on the patient's chart if retention problems are anticipated so that problem-solving steps can be recalled easily in the future as indicated.
11. **Reassessment of the characteristics of the original malocclusion:** This is helpful to determine the specific anatomic retention needs of the case (i.e., skeletal or dental deep bite situations, open bite, or overcorrection of rotations) (see Fig. 21-27).

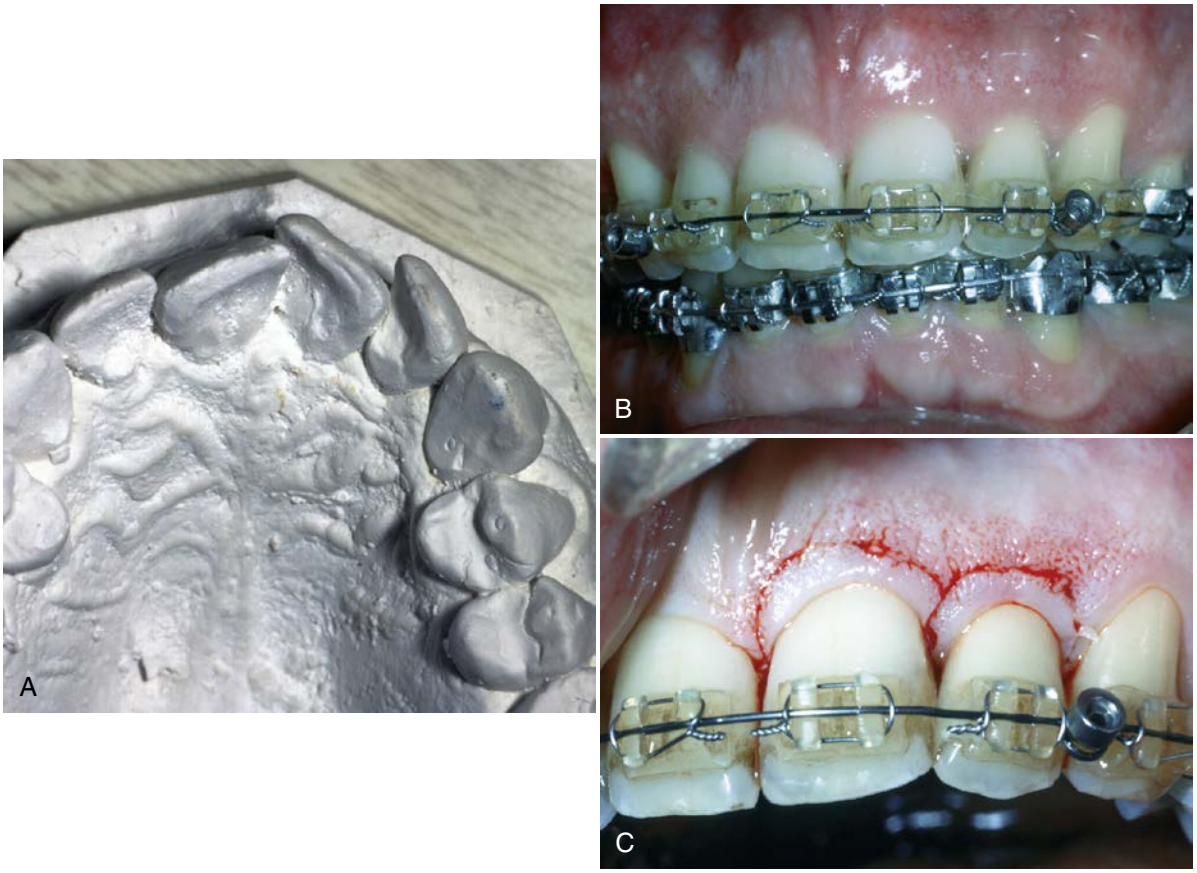


FIGURE 21-27 A, Before maxillary left central and lateral incisor rotations. B, Incisors correctly aligned. C, Before appliance removal; a circumferential fiberotomy was performed to enhance posttreatment stability.

Coordination of Debonding or Debanding with Other Treatment Providers

The orthodontist should also consider the following to coordinate debonding and debanding with other treatment providers:

1. **Posttreatment radiographs**
2. **Periodontal reevaluation and treatment**
3. **Restorative treatment and retention considerations:** Frequently, this may include the use of stabilizing sectional appliances that can be removed by the restorative dentist before placement of restorations. Removable appliances may be used for temporary tooth replacements when the patient is missing anterior teeth and there is an aesthetic concern.
4. **Duration of the retention period:** The retention period will vary according to the case type, overall restorative treatment plan, orthodontic result, and various causative factors (which again must be customized to the individual patient). Generally, the retention period for adults is longer. The weaning period from removable retainers also takes place over a longer time. To enhance the overall stability of treatment, selective grinding for centric and anterior disclusion during excursive movements is essential. In the case of significant (≥2.5 mm) centric relation to centric occlusion discrepancy, occlusal adjustment is required. A Hawley appliance with an anterior bite plane helps eliminate muscle dysfunction and facilitates selective grinding to achieve a stable centric relation position.

STABILITY AND RETENTION INDIVIDUALIZED FOR THE INTERDISCIPLINARY ADULT THERAPY PATIENT

Treating a Class I relationship in teenage patients in which the skeletal bases are normal is usually satisfactory and often quite stable. Unfortunately, these are not the clinical circumstances presented by most adult patients. Frequently, adults have skeletal Class II problems that are more appropriately treated with extraction in the upper arch only; the outcome is finished with a Class II molar relationship that usually requires occlusal adjustment by selective grinding that may or may not be done. A tooth size discrepancy is created by removing maxillary first premolars only, and this approach has been shown to be less stable than extraction in both arches. Also, the effects of the periodontium in creating instability in posttreatment orthodontic cases are well documented.[2] Picton and Moss have shown that the effects of transseptal fibers on the teeth with a constant and mesial migration of the dentition throughout life could cause incisor irregularities and crowding[97] (see Fig. 21-27). Horowitz and Hixon stated in their 1969 article that "the significant point is that orthodontic therapy may temporarily alter the course of continuous physiological changes and possibly for a time can even reverse them. However, following mechanotherapy and a period of retention restraint, the developmental maturation process resumes."[97]

In addition, there has been the apical base school through the history of orthodontics (discussed in Chapter 22). Holdaway cautioned that facial harmony could be achieved as long as the apical base did not exceed a range permitting compensating dental adjustments.[98] Apical base discrepancies are important limiting factors in adult treatment unless orthognathic procedures are accepted by the patient so that these discrepancies can be corrected rather than camouflaged. (See Fig. 21-9 for an example of a camouflaged Class III apical base patient and Fig. 21-1 for an effectively camouflaged Class II apical base discrepancy.)

Figure 21-28 illustrates a high mandibular plane angle Class II adult male patient who has multiple characteristics related to his malocclusion, including crowding of upper and lower arch, overjet, open bite, and mild lip incompetence with an accompanying mandibular apical base deficiency. After a review of different treatment options, this man decided to pursue an interdisciplinary treatment plan that would address both the skeletal and dental concerns identified. Through the use of presurgical orthodontics and maxilla-mandibular surgery with an occlusal plane change, this patient's treatment achieved an optimal outcome with high probability of long-term stability.

Figure 21-29 demonstrates the skeletal limitations of orthodontic treatment, particularly in adult patients. The sagittal limitations, without treating the skeletal discrepancy through surgical procedures, with a normal vertical relationship would be approximately a Class II subspinale-nasion-supramentale (ANB) angle of 7 or 8 degrees (this would exclude bimaxillary protrusion; see Fig. 21-1), and in the Class III skeletal problems, an ANB of –3 degrees or WITS of less than 8 mm usually requires restorative procedures or orthognathic surgery to achieve compensated anterior tooth stability. (See Fig. 21-9 for a good Class III example of this point.) With patterns that involve vertical open bite, excessive face height, and associated muscle problems, it is common with the use of vertical elastics to encounter more root-blunting resorption. Clinicians are well aware of the problems of Class II elastics in high-angle cases, tending to tip the occlusal plane, moving the lower dentition forward, and creating periodontal problems that result in discrepancies in centric relation and habitual occlusion. Patients with open bites who do not accept an orthognathic solution can now be treated with the use of TADs that have been illustrated in Chapter 20. These devices allow the intrusion of posterior teeth, mandibular autorotation and bite closure without the adverse side effects associated with the extrusion of the incisors and the need for Class II elastics.

The familiar long face problem exhibits excessive eruption of maxillary teeth, maxillary transverse constrictions, excessive overjet, anterior open bite, and associated mouth breathing. The patient shown in Figure 21-30, with a long face and a gingival smile, who elected to go through orthodontic treatment first, could only have been improved to look as he does on the right through the use of the Le Fort osteotomy technique.

In adult treatment, common pitfalls leading to instability are as follows:

1. Tendency to extract premolars in borderline cases and extracting in the lower arch as well, when the mandibular dentition is more distally placed
2. With vertical excess problems of lip incompetency, extracting upper premolars only
3. Attempting to close excessive extraction spaces when the problem should be treatment planned for alignment of the teeth and using prosthetic replacements to replace teeth rather than attempting space closure.

A significant report was made by Casko and Shepherd[99] in *The Angle Orthodontist* in 1984; the authors evaluated 79 untreated adults with ideal Class I dental occlusions. None of the patients subjectively was thought to have poor or unacceptable profiles. Casko and Shepherd discovered that the cephalometric values for the skeletal discrepancies in this group were far beyond mean figures often used as treatment goals. Figure 21-31 illustrates the envelope of discrepancy in the sagittal and vertical dimensions and can be used as a guide when treatment planning adult patients. As well, cases must be evaluated in the transverse dimension for transverse skeletal discrepancy, also seen in Figure 21-31; this can best be done at the present time with the use of the PA radiograph. A significant skeletal discrepancy in a transverse dimension diagnosed by looking only at the dentition and attempting to use archwires to laterally expand the teeth on a constricted maxillary apical base frequently creates buccal gingival recession as one of the side effects of camouflaging patients with transverse dysplasias. Clinicians must develop an envelope of discrepancy concept for the transverse dimension, as well as for the sagittal and vertical dimension (see Fig. 21-31). In transverse skeletal problems in adults, treatment options are to leave the patient in crossbite, use restorative dentistry to change the contour of the posterior teeth to achieve a more normal dental buccolingual landmark relationship, or correct the skeletal pattern by surgically assisted palatal expansion. Camouflaging the transverse skeletal deficiency by only moving the teeth may cause periodontal problems, mainly buccal gingival recession and instability of the occlusal scheme. The effective surgical procedure to use was reported in August 1976.[100]

In the transverse dimension, the clinician may elect to camouflage the skeletal discrepancy by tipping the teeth and creating occlusal instability and periodontal problems; if a surgical procedure is used, the expansion appliance should have acrylic that covers the palate. With an effective surgical procedure, tissue-borne appliances do not create excessive pressure on the palatal mucosa and will not create vascular ischemia by pressure against the palatal tissue. Tooth-borne appliances alone, however, tend to allow for more dental tipping than is seen with appliances that have acrylic on the palate. Therefore, Hyrax-type appliances are not recommended. The case in Figure 21-32 illustrates significant dental tipping that occurred as a result of using that appliance. A more effective appliance is the Haas-type appliance. The surgical procedure involves raising the mucoperiosteal tissue in the buccal vestibule bilaterally in the maxilla; a horizontal osteotomy is made through the lateral wall of the maxilla 4 to 5 mm, superior to the posterior teeth. In addition, to facilitate the separation of the maxilla, an osteotome is used between the central incisors to separate the palate. Usually the procedure is a day surgery procedure and does not require the patient to stay overnight in the hospital.

For Class II orthodontic patients, extraction in the maxillary arch only is necessary to effectively camouflage skeletal deformities, vertical growth patterns, and bimaxillary protrusions. Figures 21-1, 21-9, and 21-29, *A* and *B,* show examples of limitations that exist in adult treatment when orthognathic procedures are not accepted by the patient or when there is no insurance coverage. Another example of limiting factors in Class II camouflage patients is the need to graft the lower anterior to allow more lower incisor proclination to achieve incisor guidance. In the case shown in Figure 21-29, *A* and *B,* the patient also demanded that all appliances be removed before complete space closure; a

FIGURE 21-28 A–C, Pretreatment intraoral photographs. D–F, Posttreatment intraoral photographs. G, Pretreatment profile. H, Posttreatment profile showing improved facial balance and elimination of the appearance of mandibular deficiency.

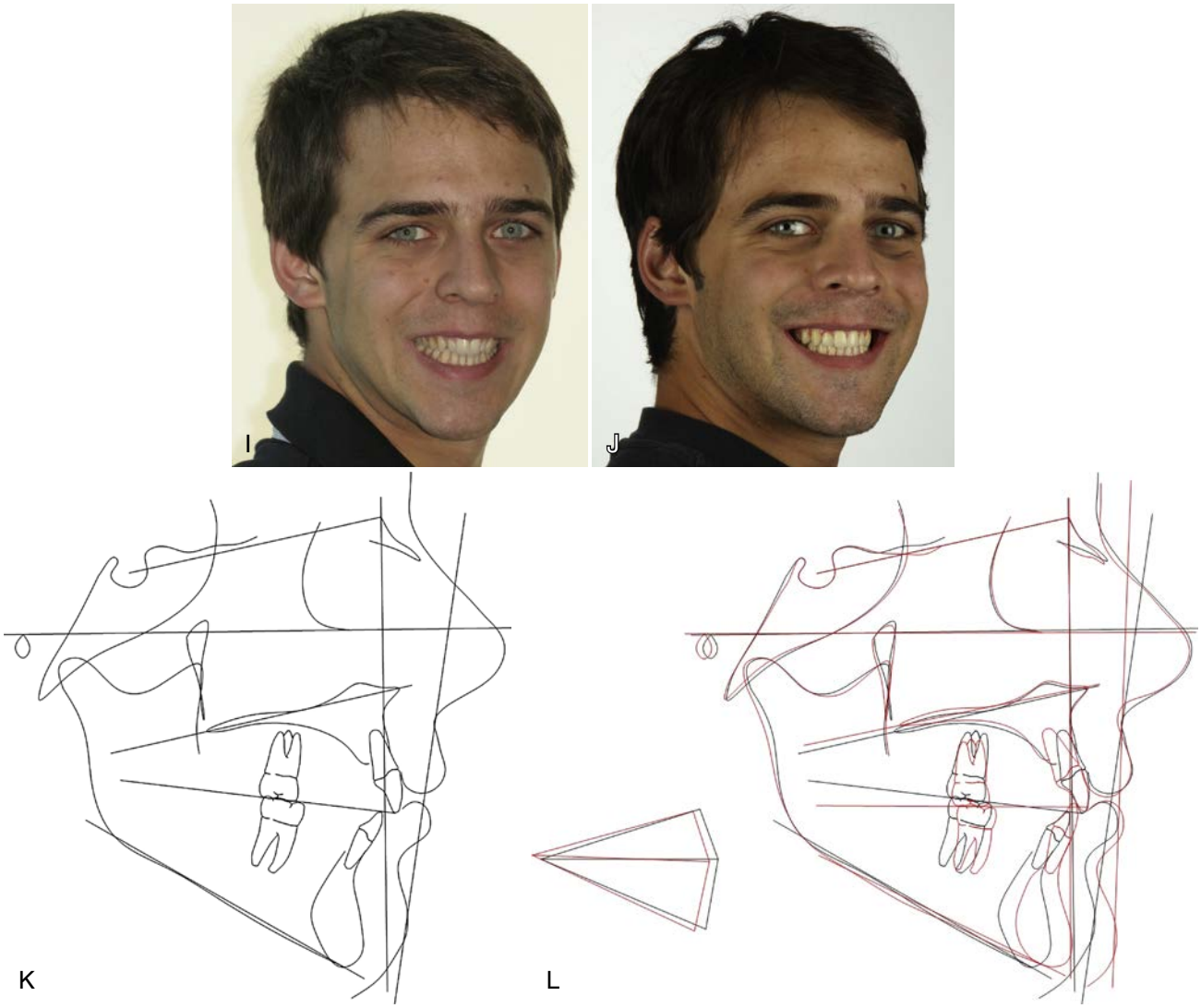


FIGURE 21-28, cont'd I, Pretreatment ¾ smile. J, Posttreatment smile illustrating the achievement of favorable facial balance. K, Pretreatment cephalometric tracing. L, Pre- and posttreatment cephalometric supraimposition with posttreatment outcome. illustrated in *red*. (Courtesy of *Orthodontics* by Dr. Lorenz Moser & Dr. Ute-Schneider-Moser, Italy. Surgery by Prof. Mirco Raffaini, Italy.)

retainer was bonded to the lower, canine to canine. The lower right molar had a large amalgam filling and was crowned so that mesiodistal contact (arch integrity) was maintained through the second molar (Fig. 21-33). An operative restoration was placed on the patient's left side, and "immediate" removable retainers were inserted. An impression was taken for the upper arch with instructions for the laboratory to carve off all the brackets, and at the time the case was debonded, the retainer was placed immediately because of the muscle problem (tongue thrust). The patient was given two retainers for the maxillary arch. One was worn during the day (a regular removable retainer) for an indefinite period of time until it could be left out during the day. The termination of daytime wear was indicated when the patient felt no pressure on the teeth at the insertion of the night crib appliance. The day appliance may be required for as long as 1 year, but the night crib is used indefinitely. The crib appliance at night prevents the tongue from being placed between the anterior teeth, which would allow for eruption of molars and a return of the open bite. The tongue appliance that is given to all patients with a forward-resting tongue posture problem is illustrated in Figure 21-34.

Patients who exhibit a tongue placement habit can also be recognized by their forward tongue placement during speech. Retention requires special consideration for posttreatment stability, as indicated in Figure 21-33.

The forward-resting tongue posture problem is significant, particularly for an adult patient who has maintained the habit of placing his or her tongue between the anterior segment beyond the adolescent years. In an article published in *The Angle Orthodontist* in 1989, Denison, Kokich, and Shapiro[101] divided the sample population into three groups based on the degree of pretreatment overbite in the open bite subsample; results show clearly that the three subsamples tested responded differently during the posttreatment interval. Among the subsample, 42.9% showed a significant increase in facial height, eruption of maxillary molars, and decrease in overbite; 26.6%

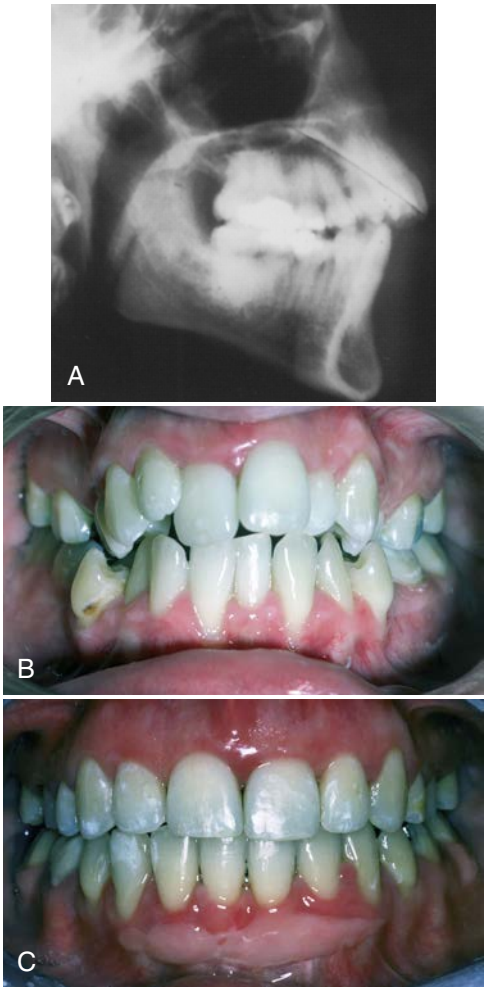


FIGURE 21-29 A, Pretreatment cephalometric film reveals an anterior open bite and overjet. B, Pretreatment alignment. C, Posttreatment alignment.

of the open bite sample and 16.7% of the overlap subsamples showed a significant increase in facial height, significant eruption of posterior teeth, and no change in overbite. The incisal contact sample had no significant posttreatment changes.

Among the possible reasons for posttreatment instability and one of the factors not taken into consideration in retention with this study population was the unconscious forward placement of the tongue, and a crib at night only could have prevented the open bite relapse. Open bite that recurred in 42.9% of subjects with pretreatment open bite could have been reduced with the use of a tongue crib retainer at night. As stated earlier, patients commonly place their tongues between the anterior segments, allowing vertical eruption to occur at the molars and resulting in recurrence of the dental open bite.

For skeletal Class II patients, it may be necessary to advance the lower incisors (compensation is placed in the mandibular dentition and not in the maxillary incisors) and place tooth material bilaterally in the posterior segments of the mandible, as seen in Figure 21-35, where two restorations were placed in the mandible. In Class III cases, orthodontists may compensate the maxillary incisors for the Class III skeletal pattern by adding tooth material in the maxillary posterior segments bilaterally to maintain a more procumbent maxillary incisor without allowing the upper incisors to be placed into permanent trauma or fremitus because of the Class III skeletal pattern.

Figure 21-36 exhibits a Class III case in which the maxillary centrals are moved to the midline: two bridges were placed in the maxillary posterior segments, allowing the anterior teeth to be stable without putting permanent trauma on the upper anterior teeth. To remove the appliance and merely insert an upper and lower removable retainer would not ensure stability. Placing two retainers without reshaping old restorations that have been carved up in the preorthodontic occlusion would not achieve stability. When tooth size discrepancies exist or when teeth are taken out, it may be necessary to reshape remaining teeth with bonding material. It is much easier to establish intimate occlusion and stability for a patient who does not have

FIGURE 21-30 A, Long-faced patient who initially refused surgery and after 1.5 years of orthodontics requested to be reevaluated for a maxillary impaction. B, Appearance after orthognathic surgery. Note the excellent lip competency.

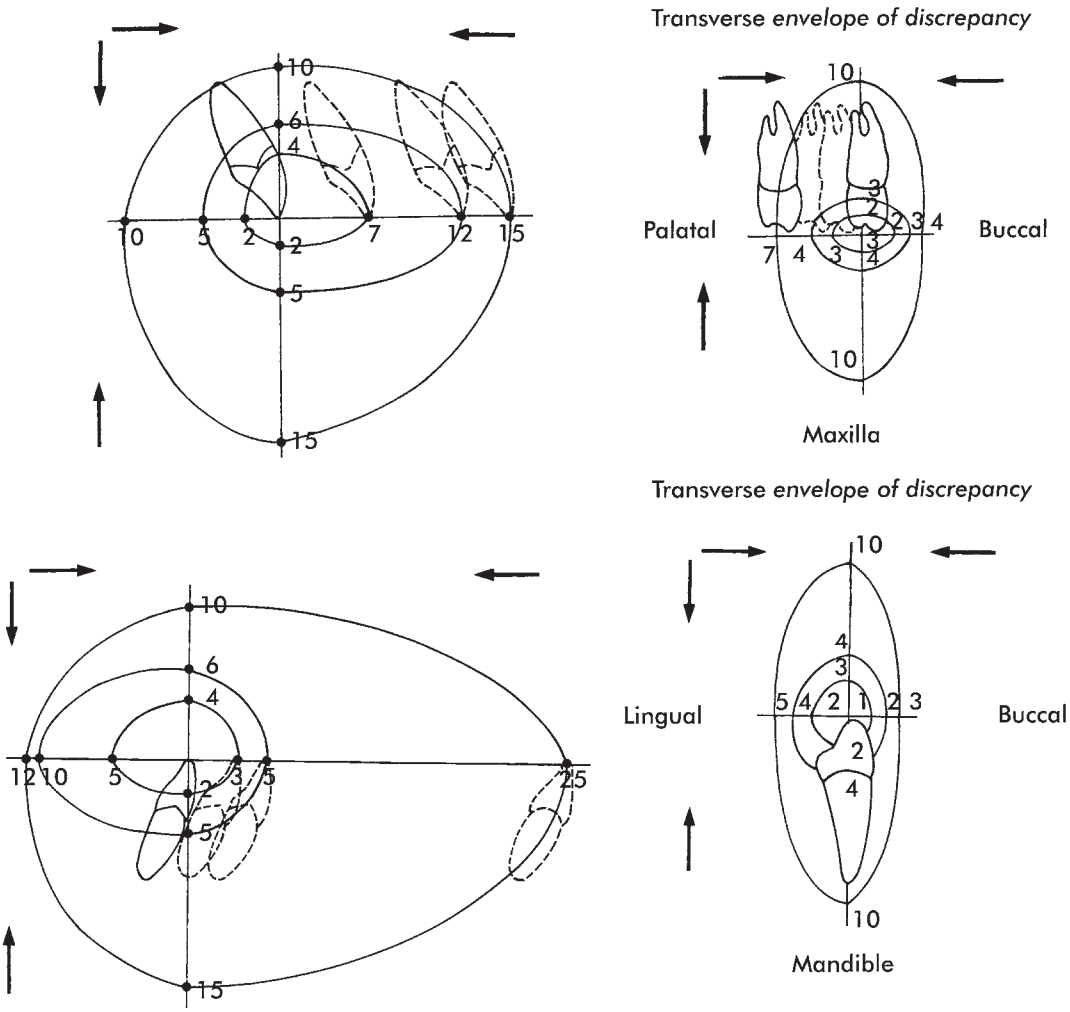


FIGURE 21-31 The envelope of discrepancy for the maxillary and mandibular arches in three planes of space. The ideal position of the upper and lower incisor in the anteroposterior (AP) and vertical planes is shown in the center of the incisor diagrams. The millimeters of change required to retract a protruding incisor, move forward a posteriorly positioned incisor, or extrude or intrude an incorrectly vertically positioned incisor are shown along the horizontal and vertical axes, respectively. The limits of orthodontic tooth movement alone are represented by the inner envelope, possible changes in incisor position from combined orthopedic and orthodontic treatment in growing individuals are shown by the middle envelope, and the limits of change with combined orthodontic and surgical treatment are shown by the outer envelope. The inner envelope for the upper arch suggests that maxillary incisors can be brought back a maximum of 7 mm by orthodontic tooth movement alone to correct protrusion but can be moved forward only 2 mm. The limit for retraction is established by the lingual cortical plate and is observed in the short term; the limit for forward movement is established by the lip and is observed in long-term stability or relapse. Upper incisors can be extruded 4 mm and depressed 2 mm, with the limits being observed in long-term stability rather than as limits on initial tooth movement. The envelopes of discrepancy for the transverse dimension in the premolar areas are much smaller than those for incisors in the AP plane. The transverse dimension can be crucial to long-term stability, periodontal health, and frontal dentofacial aesthetics. The orthodontic and surgical envelopes can be viewed separately for the upper and lower arches, but the growth modification envelope is the same for both: 5 mm of growth modification in the AP plane to correct Class II malocclusion is the maximum that should be anticipated, whether occlusion is achieved by acceleration of mandibular growth or restriction of maxillary growth. The outer envelope suggests that 10 mm is the limit for surgical maxillary advancement or downward movement, although the maxilla can be retracted or moved up as much as 15 mm; the mandible can be surgically set back 25 mm but can be advanced only 12 mm. These numbers are merely guidelines and may underestimate or overestimate the possibilities for any given patient; however, they help place the potential of the three major treatment modalities in perspective.

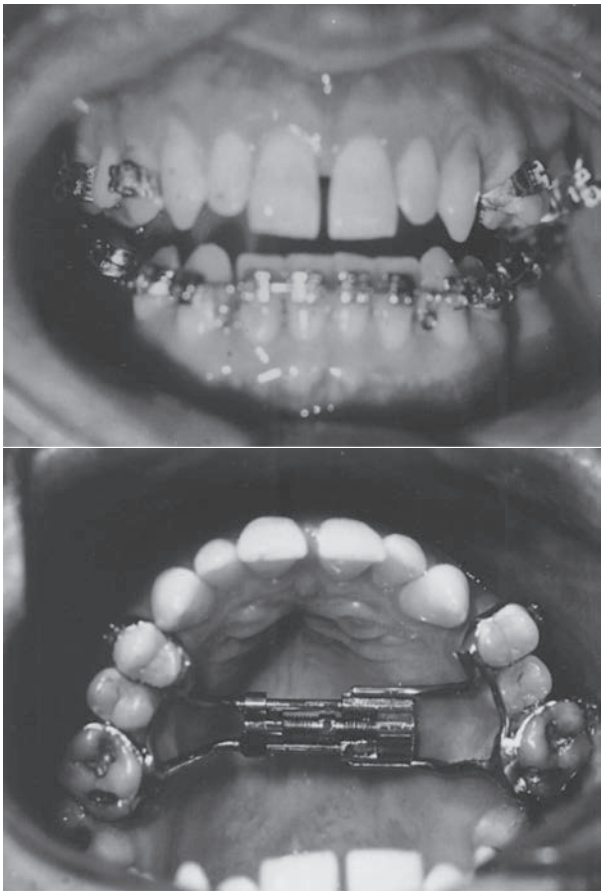


FIGURE 21-32 Frontal and occlusal view of severe dental tipping with the Hyrox-type palatal expander.

old fillings and restorations. Patients with old crowns that were carved to a previous malocclusion or large restorations and amalgams that need to be reshaped also require occlusal adjustment by selective grinding to create a definitive occlusion that coordinates maximal intercuspation and central relation.

Adaptations that occur with aging have been well described by Behrents.[102] The adult orthodontic patient will need to have indefinite retention in the mandibular anteriors. The Sillman collection at the University of Pennsylvania features long-term records of Class I individuals (who received minimal or no treatment) who had well-aligned incisors at age 13 years but had crowding at 22 years of age, together with normal dentitional compensation of aging. Prerestorative space or pontic areas that are created in treatment require indefinite retention, and the forward tongue position, particularly in adults, requires a crib to be worn at night for an indefinite time.

Three important situations demand indefinite retention:

1. Patients who have generalized spacing, in which the arches are large and the tooth structure is not sufficient to close all the space
2. In circumstances of lip competency, the objective should be to transfer the space to the posterior segments and add tooth material in the posterior areas to achieve arch integrity. With adult patients, the object is to keep the restorative dentistry in the posterior segments and have natural tooth material in the anterior.
3. With tooth size discrepancies in the anterior area, it may be necessary to correct the tooth material problem where the tooth size discrepancy exists. If the discrepancy is in the anterior teeth, bonding and reshaping of the anterior group are mandatory; if the discrepancy is in the posterior teeth, the solution lies in treating the posterior midline and advancing the canine and premolar teeth mesially, shifting the space to the posterior segments.

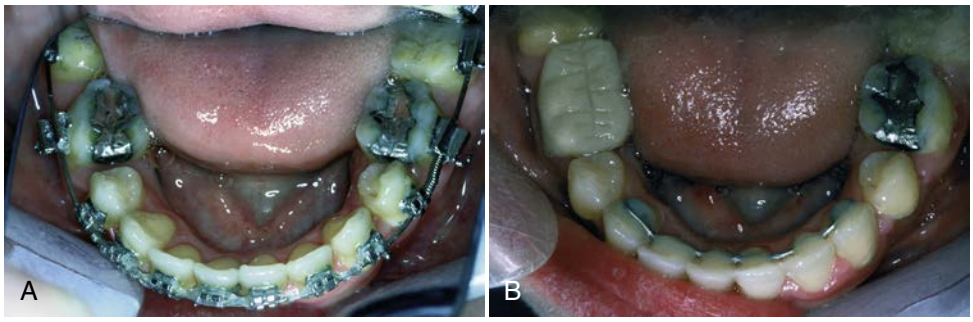


FIGURE 21-33 Same patient as in Figures 21-28 and 21-29. **A,** Note the spaces mesial and distal to the lower right molar when the patient demanded that appliances be removed. **B,** The large mandibular molar provisional was placed at the time appliances were removed.

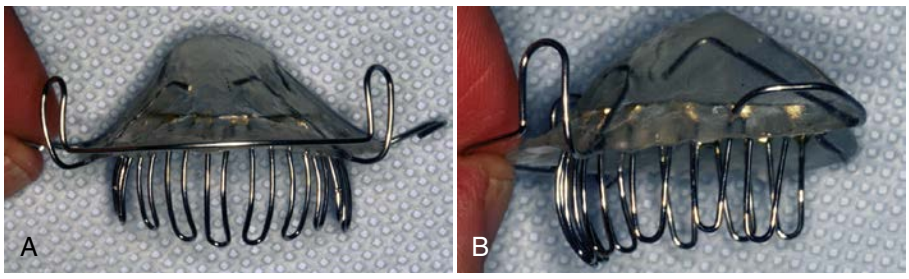


FIGURE 21-34 **A,** Anterior view of the tongue crib that is used indefinitely as a retainer. **B,** The appliance has a labial bow with ring clasps, and the crib extends back to the second molar area to the lingual mucogingival junction of the mandible when the patient is in maximal intercuspation.

FIGURE 21-35 **A,** Left lateral view of a 59-year-old Class II adult. **B,** Lower incisors, canines, and first premolars were advanced to obtain a Class I dental relationship and incisal guidance. **C,** Mandibular arch after pontic space was created in lower left quadrant between the premolars and before a pontic space had been created between the mandibular right premolars. **D,** Postrestorative occlusal view with the bridges having been placed bilaterally, allowing the more procumbent incisors to remain stable. **E,** The compensated mandibular incisors remain stable 20 years after treatment.

Again, it is recommended that the orthodontist refer to the basic concepts of retention clarified in Chapter 33. Those concepts and research findings can guide clinicians in further integrating a retention approach that is optimal for each patient.

Communicating with Team Members

Preorthodontic inflammation and occlusal control can be managed by the general dentist or the periodontist, but the orthodontist should monitor this phase and evaluate patient response over time before initiating the orthodontic phase of treatment. In addition, obviously, all caries control should have been completed, overhanging margins corrected, overbulked contacts reduced, irregular marginal ridges reshaped, and endodontic procedures completed. Consequently, adult interdisciplinary dental therapy requires treatment sequencing information so that standardized communication of the treatment plan is available to the patient and all providers.

RISK MANAGEMENT AND THE ADULT INTERDISCIPLINARY THERAPY PATIENT

According to recent statistics from the American Association of Orthodontists Council on Insurance, adult patients account for 50% of malpractice claims. Because adult patients account for fewer than 20% of the orthodontic patients, it would seem that each time orthodontists treat an adult versus a child, they are

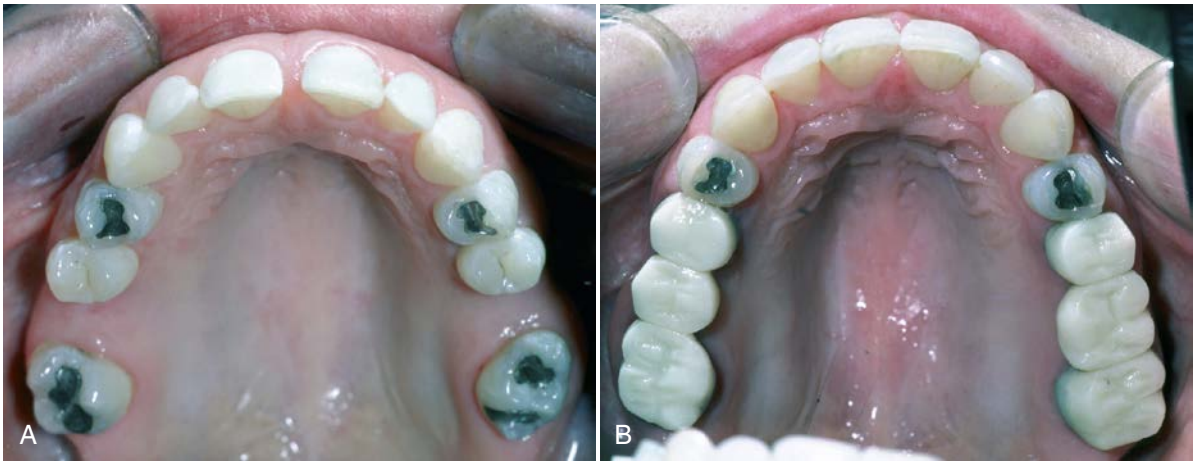


FIGURE 21-36 A, Occlusal view of the maxillary arch in a Class III patient who is missing posterior teeth bilaterally. If a Class III patient has an A point–nasion–B point greater than –3 degrees, space can be created bilaterally in the maxillary posterior areas, and bonding material can be added mesiodistally to achieve arch integrity and maintain more procumbent maxillary incisor stability without occlusal trauma. B, The incisors are advanced and centered on the midline, and restorations have been placed bilaterally in posterior areas.

four times more likely to be named in some form of malpractice claim. Is the increased risk simply because the cases are more complicated? Is the increase in risk because adult patient expectations are so much higher than those of the adolescent patient or parent of the adolescent? Or is the problem simply a lack of communication, as several articles have suggested?[78,83]

Because almost 80% of adult patients require interdisciplinary treatment planning and treatment execution, the role of communication among providers and with the patient cannot be emphasized enough. In Roblee's text, there is an important discussion of the role of communication:[57]

> *Extensive communication between team members is crucial to the success of Interdisciplinary Therapy . . . lack of communication or improper communication between team members is frequently the most common source of breakdown of therapy and of the team. The various team members must have a common purpose with the same objectives, as well as common knowledge that allows them to communicate intelligently and effectively with one another.*[57]
>
> *If an orthodontic clinician plans on emphasizing more availability of adult treatment to their community, they would be wise to form an interdisciplinary study group of key members of an interdisciplinary team (IDT)—periodontist, oral and maxillofacial surgeon, restorative dentist, endodontist, psychologist, physical therapist, and other professionals who might be appropriate for team interaction.*[57]

As the outline of this AIT chapter indicates, the IDT members should clarify their own concepts and the dental team's concept of:

- Goals of treatment
- Diagnosis of AIT problems
- Sequence of treating various conditions
- Stabilization and retention

In addition, the team's approach to communicating the plan of treatment to each of the providers and to the patient needs to be very clear. Many offices that function as part of an IDT have assigned staff as treatment coordinators to assist in the communication process because IDT patients frequently require occasional updates so they are kept appraised of treatment progress.

The authors suggest that a *disciplined* approach to patient communication be part of the treatment protocol for each patient. Four key conferences should be considered fundamental to adult interdisciplinary patient management, and with each of these conferences, it is advisable to have a written report to summarize what was said for future reference if needed.

1. **Treatment conference report** (Fig. 21-37): The report shown is a summary of the treatment plan that was discussed and presented to the patient. It should include the diagnosis, a problem list, a plan of treatment in sequential order (including proposed future consultations), a list of providers for each procedure, a statement of the prognosis, a list of potential treatment limitations, and a statement of patient concerns. This report is frequently generated by the orthodontist member of the team in collaboration with other key team members.
2. **Progress report** (Fig. 21-38): After about 12 to 14 months of orthodontic treatment, photographs and a panoramic radiograph (periapical radiographs, cephalogram, and study models are elective) should be taken and a progress report generated. This is used to evaluate tooth response, periodontal health, root positioning for restorative considerations, and potential treatment idiosyncrasies. Time should then be scheduled to provide an opportunity to sit quietly with the adult patient (other providers if indicated) to advise him or her of the progress of treatment, time remaining in treatment, and any problems or changes in the treatment plan that now needs to be considered. These few minutes of the orthodontist's time are greatly appreciated by the adult interdisciplinary patient and his or her restorative dentist. In addition, the time taken for a **formal progress update** may provide the patient and the referring dentist with the necessary positive reinforcement that is needed to reduce the stress of a long, complicated, and expensive treatment plan.
3. **Stabilization or retention report** (Fig. 21-39): After removal of fixed appliances, a retention conference is scheduled, and

TREATMENT CONFERENCE REPORT

TO: Dr. **RE:** **(Age:)** **DATE:**

I. Classification and diagnostic description of malocclusion:

II. List of dental problems: (mild / moderate / severe)
*
*
*
*

III. Treatment sequence recommended at this time: **Provider:**
1.

2.

3.

4.

IV. Overall problem severity: (0-10 scale, with 10 being most severe) ___ /10

V. Limiting factors in achieving "text book ideal" result:
1.
2.
3.

VI. Patient concerns and current plan of action:
1.
2.
3.

Treatment Time: Treatment Start Date:

cc:

FIGURE 21-37 Sample treatment report for a patient requiring interdisciplinary therapy.

a written report is generated so that the adult patient can review the treatment and the treatment outcome. This is an important opportunity to discuss any treatment limitations (e.g., skeletal imbalances, missed appointments, compliance issues) and any negative sequelae (e.g., root resorption, gingival recession or bone loss). On a positive note, the authors have found that giving the adult patient a copy of before-and-after photographs is a well-appreciated good-will gesture. The photographs also illustrate to the patient the positive changes that have occurred with treatment. Also, this provides the orthodontist with a good opportunity to reorient the patient to the additional periodontal and restorative procedures that are needed. Any patient concerns should be noted and appropriately managed during the retention phase of treatment. (See Figs. 21-33 and 21-34 with special attention to the graphs of the patient's emotional "highs and lows" during their interdisciplinary treatment.)

4. **Treatment completion** (Fig. 21-40): Approximately 12 to 18 months after the removal of appliances, a final evaluation of the patient and the stability of the correction should be done. Again, any problems or additional treatment steps can be discussed. This is a good time for a formal review of future retention requirements that need to be considered. It is helpful to advise them of your office's availability for further retainer maintenance and what type of fees would be charged for new retainers, office visits, and bonded retainer maintenance.

Progress Note

To: Dr. _____ Date: _____

Re: _____ Age: _____

Your patient was seen in our office today for an appointment/mini-conference to discuss: (❑ **Parent Present**)

❑ **Progress panoramic radiograph (see enclosed x-ray) findings:**
- ❑ Decay or Pathology _____
- ❑ Shape of Condyles and Symmetry _____
- ❑ Root Resorption _____
- ❑ Root Alignment/CEJ _____
- ❑ Third molars _____

(We typically make recommendations regarding third molar status during the retention phase)

❑ **Growth response to treatment:**
- ❑ Not a factor in this patients treatment

Additional records to evaluate including:
- ❑ Cephalogram ❑ Hand/Wrist Film
- ❑ Cephalogram/Superimposition to evaluate "Growth Treatment Response Vector"

Comments: _____

_____

_____

❑ **Compliance with treatment requirements:**

Appointments:	Excellent ___	Good ___	Fair ___	Poor ___
Oral Hygiene:	Excellent ___	Good ___	Fair ___	Poor ___
Cooperation with appliance instruction:	Excellent ___	Good ___	Fair ___	Poor ___

Additional Comments: _____

❑ **Treatment completion timeline:**
- ❑ Treatment is on time (Brace removal estimated – Spring/Summer/Fall/Winter, _____.)
- ❑ Treatment delayed due to _____

(New estimated debanding month is: _____)

If there are any questions or suggestions, please call

Doctor

FIGURE 21-38 Progress report used on all orthodontic treatment patients to update patient and providers of treatment plan progress. This is particularly important for adult interdisciplinary dental therapy patients.

RETENTION/STABILIZATION CONFERENCE REPORT

TO:_____ DATE:_____

RE:_____

Fixed orthodontic appliances were removed on_____; and_____has been referred back to you with the following observations and recommendations:

I. RETENTION PHASE:

Estimated Retention Time:_____

Appliances Prescribed:

Maxillary:_____

Mandibular:_____

Frequency of Retention Recall:_____

II. FUTURE TREATMENT CONSIDERATIONS:

Your patient,_____, has been advised to make an appointment with you to be evaluated for the following:

- ☐ Routine post-orthodontic examination and caries check:_____
- ☐ Cleaning/fluoride:_____
- ☐ Radiographic survey (as you see fit):_____
- ☐ Periodontal Evaluation:_____
- ☐ Restorative Treatment:_____
- ☐ Equilibration Needed:_____
- ☐ Future Extractions:_____
- ☒ Other: JAW GROWTH- for girls who are 15 and younger and boys who are 21 and younger, studies have shown that 5-8% of persons in this age group experience "late stage growth changes" that may affect the bite.

III. TREATMENT APPRAISAL:

Description of original problem and treatment objectives:_____

Limitations imposed by complexity of orthodontic problem:_____

Limitations imposed by patient cooperation:_____

Overall assessment and comments:_____

If there are any questions regarding this report, please call me, at your earliest convenience.

Doctor

FIGURE 21-39 Retention and stabilization conference report used after adult interdisciplinary dental therapy to review treatment plan and future periodontal and restorative needs. Many times interdisciplinary treatment patients forget the sequence of who is doing what next. This is a great time to show orthodontic outcome and reinforce future treatment steps.

SUMMARY

This chapter has described the diagnosis, planning, sequencing, and stabilizing involved in the interdisciplinary management of complicated adult patient care. Advances in technology have provided IDTs with new methods of reliably managing the restoration of the stomatognathic system. In a high percentage of adult patients, interdisciplinary interaction among providers is critical. Therefore, a disciplined communication approach (provided to each patient) is required for each AIT patient. The more thorough the communication among providers and with the patient, the more likely it is that an optimal outcome will be achieved.

Orthodontic Completion Summary

To Dr: _____ Date of Report: _____

Re: _____ Appliance Removal Date: _____

Retention Phase: _____ to _____

Now that your braces have been removed and the supervised retention phase of treatment is completed, we want to release you from our care back to the long term care of your general dentist. As far as your orthodontic correction is concerned, your teeth have shown good stability while we have observed you during the retention period. Due to the dynamic nature of your mouth (i.e. speech, chewing, breathing, swallowing, and the possibility of clenching and grinding habits) your teeth will most likely continue to shift a little as times goes by. This is nature's way of allowing your teeth to achieve your mouth's "position of equilibrium".

OUR RECOMMENDATIONS:

The following recommendations require your attention:

- ❑ *A decision about your third molars* (wisdom teeth) will have to be made in the future. Your general dentist will monitor their development and advise you if extraction is appropriate. Comments: _____
- ❑ *Bonded Retainers* need to be checked by your dentist periodically - if loose, they need to be rebonded. We can do this or your dentist can do this. However, if the retainer is not rebonded, decay may occur. NOTE: If a bonded retainer is removed, expect some minor shifting due to "adult stage" lower incisor crowding.
- ❑ *Removable Retainers* - it's a good idea to wear them periodically to be sure that your teeth have stabilized.
- ❑ *Jaw Growth* - for girls who are 15 and younger and boys who are 21 and younger, studies have shown that 5-8% of persons in this age group experience "late stage growth changes" that may affect the bite. If you feel this happening, please call us so we can determine if an additional phase of treatment is needed.

ADDITIONAL TREATMENT:

Charges for future treatment that you may request are:

- ❑ *Office Visit:*
  $40.00 - $80.00.
- ❑ *Diagnostic Records:*
  Fees at prevailing office rates.
- ❑ *Orthodontic Re-treatment:*
  The fee for this is variable depending on the problem and the underlying etiology of the need for additional treatment.
- ❑ *Therapy for Temporomandibular Joint (TMJ) Problems:*
  Many patients who grew up with malocclusions also have a weakened ligament apparatus controlling TMJ function. Many factors like stress, trauma, clenching, grinding, and gum chewing can cause the jaw joint to make noise or become painful. The fee for any additional treatment in this area will be based on the severity of the problem and the amount of treatment which is needed to resolve the problem.

Thank you for taking the time to review this material. We will miss seeing you, but we hope that you will continue to make healthy choices and, thereby, maintain your optimal health in the future.

_____
Date

_____
Patient/Responsible Party

_____
Doctor

FIGURE 21-40 Completion conference report used 12 to 18 months after appliance removal for the orthodontist to complete the communication process with the patient and providers and to bring closure to the treatment retention phase. Without "formal closure," the patient may drop in any time for ongoing treatment that may not be part of the initial orthodontic treatment plan.

Acknowledgments

The authors express the deepest appreciation to the restorative dentists, periodontists, and oral and maxillofacial surgeons and their staff members who make interdisciplinary therapy for our patients possible and successful. Special appreciation goes to our staff, whose diligence and professionalism have helped each patient through the intricacies of interdisciplinary dentofacial therapy. Also, special thanks to Elizabeth Barrett, Beth McFarland, and Melissa Carstens for their time and assistance with the preparation of this chapter; without their outstanding professional input, this chapter would not have been possible.

REFERENCES

1. Lindegård B, Lindegård L, Carlson M, Larsson S. Need and demand for orthodontic treatment. *Tandlaegebladet*. 1971;75:1198.
2. Alexander RG, Sinclair PM, Goates LJ. Differential diagnosis and treatment planning for the adult nonsurgical orthodontic patient. *Am J Orthod Dentofacial Orthop*. 1986;89:95–112.
3. Brandt S. The future of orthodontics. *J Clin Orthod*. 1976;10:668.
4. Gorman JC. Treatment with lingual appliances: the alternatives for adult patients. *J Adult Orthodont Orthognath Surg*. 1987;3:131.
5. Musich DR. Assessment and description of the treatment needs of adult patients evaluated for orthodontic therapy, parts I–III. *Int J Adult Orthodon Orthognath Surg*. 1986;1:55.
6. Casko JS, Vaden JL, Kokich VG, et al. ABO: objective grading system for dental casts and panoramic radiographs. *Am J Orthod Dentofac Orthop*. 1998;114:589.
7. Epker BN, Fish LC. *Dentofacial Deformities*. St. Louis: Mosby; 1986.
8. Amsterdam M, Abrams L. Periodontal prosthesis in periodontal therapy. In: Goldman HM, Cohen DW, eds. *Periodontal Therapy*. 6th ed. St. Louis: Mosby; 1980.
9. Ramfjord SP, Ash MM. *Periodontology and Periodontics*. Philadelphia: WB Saunders; 1979.
10. Nevins M. Interproximal periodontal disease: the embrasure as an etiologic factor. *Int J Periodont Restor Dent*. 1982;6:9.
11. Amsterdam M. Periodontal prosthesis: twenty-five years in retrospect. *Alpha Omegan*. 1974;67:9–51.
12. Ingber JS. Forced eruption. I. A method of treating one and two wall infrabony osseous defects: rationale and case report. *J Periodontol*. 1974;45:199.
13. Uysal A, Kansu H, Akhan O, et al. Comparison of ultrasonography with magnetic resonance imaging in the diagnosis of temporomandibular joint internal derangements: a preliminary investigation. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2002;94(1):115–121.
14. Miller TE. Orthodontic therapy for the restorative patient. I. The biomechanical aspects. *J Prosthet Dent*. 1989;61:268.
15. Morgan DH, Hall WP, Vamvas SJ. *Disease of the Temporomandibular Apparatus: A Multidisciplinary Approach*. St. Louis: Mosby; 1977.
16. Musich DR. Problem oriented record. In: Graber TM, Swain BF, eds. *Current Orthodontic Concepts and Techniques*. 2nd ed. Philadelphia: WB Saunders; 1975.
17. Weed LL. *Medical Records, Medical Education, and Patient Care: The Problem Oriented Record as a Basic Tool*. Cleveland: Case Western Reserve Press; 1969.
18. Arnett GW, Bergman RT. Facial keys to orthodontic diagnosis and treatment planning, Part II. *Am J Orthod Dentofac Orthop*. 1993;103:395–411.
19. Broadbent Sr BH, Broadbent Jr BH, Golden WH. *Bolton Standards of Dentofacial Developmental Growth*. St. Louis: CV Mosby; 1975.
20. Nevins M. The interdental embrasure and interproximal periodontal disease. In: Nevins M, Mellonig JT, eds. *Periodontal Therapy: Clinical Approaches and Evidence of Success*. Vol. 1. Carol Stream, IL: Quintessence; 1998.
21. Shulman J. Teaching patients how to stop bruxing habits. *J Am Dent Assoc*. 2001;132:1275–1277.
22. Smalley WM. Clinical and laboratory procedures for implant anchorage in partially edentulous dentitions. In: Higuchi KW, ed. *Orthodontic Applications of Osseointegrated Implants*. Chicago: Quintessence; 2000.
23. Spear FM. Restorative considerations in combined orthodontic implant therapy. In: Higuchi KW, ed. *Orthodontic Applications of Osseointegrated Implants*. Chicago: Quintessence; 2000.
24. Williamson EH. JCO interviews: Dr Eugene Williamson on occlusion and TMJ dysfunction, Part I. *J Clin Orthod*. 1981;14:333.
25. Jacobson A. The proportionate template as a diagnostic aid. *Am J Orthod*. 1979;75:156.
26. Johnston L. Template analysis. *J Clin Orthod*. 1987;21:585.
27. Proffit WR, White RP. *Surgical Orthodontics Treatment*. St. Louis: Mosby; 1991.
28. Vanarsdall RL. Uprighting the inclined mandibular molar in preparation for restorative treatment. Continuing Dental Education Series. Vol. 1, no 2. Philadelphia: University of Pennsylvania Press; 1977.
29. Vanarsdall RL. Orthodontics, provisional restorations and appliances. *Dent Clin North Am*. 1989;33(3):479.
30. Sherwood KH, Burch JG, Thompson WJ. Closing anterior open bites by intruding molars with titanium miniplate anchorage. *Am J Orthod Dentofac Orthop*. 2002;122:593–600.
31. Ackerman JL. The challenge of adult orthodontics. *J Clin Orthod*. 1978;12:43.
32. Olsson M, Lindhe J, Marinella CP. On the relationship between crown form and clinical features of the gingiva in adolescents. *J Clin Periodontol*. 1993;20:570–577.
33. Folkers SA, Weine FS, Weissman DP. Periodontal disease in the life states of women. *Compend Contin Educ Dent*. 1992;13:852.
34. Ryder MI. Position paper: tobacco use and the periodontal patient. *J Periodontol*. Jan 1996;67:51–56.
35. Holmes TH, Rahe RH. Social readjustment rating scale. *J Psychosom Res*. 1967;11:213.
36. Howard JA. Temporomandibular joint disorders, facial pain and dental problems of performing artists. In: Stalof R, Brandfonbrenner A, Leberman R, eds. *Textbook of Performing Arts Medicine*. New York: Raven Press; 1990.
37. Nickerson JW, Boering G. Natural course of osteoarthrosis as it relates to internal derangement of the temporomandibular joint. *Oral Maxillofac Surg Clin North Am*. 1989;1:27.
38. Rasmussen OC. Clinical findings during the course of temporomandibular arthropathy. *Scand J Dent Res*. 1981;89:285.
39. Schiffman E, Fricton JR. Epidemiology of TMJ and craniofacial pain. In: Fricton JR, Kroening RJ, Hataway KM, eds. *TMJ and Craniofacial Pain: Diagnosis and Management*. St. Louis: Ishiyaku Euro America; 1988.
40. McNeill C, ed. *Craniomandibular Disorders: Guidelines for Evaluation, Diagnosis, and Management*. Chicago: Quintessence; 1990.
41. Okeson JP. *Management of Temporomandibular Disorders and Occlusion*. St. Louis: Mosby; 2003.
42. Roth RH. Functional occlusion for the orthodontist. *J Clin Orthod*. 1981;15:1.
43. Alpern MC, Nubelle DG, Wharton MC. TMJ diagnosis and treatment in multidisc environments and follow-up study. *Angle Orthod*. 1988;58:101.
44. Greco PM, Vanarsdall RL, Levrini M, et al. An evaluation of anterior temporal and masseter activity in appliance therapy. *Angle Orthod*. 1999;69(2):141–146.
45. Mair LH. Understanding wear in dentistry. *Compendium*. 1999;20:19–30.
46. Grant DA, Stern IB, Everett FG. Periodontics. In: *Tradition of Orban and Gottlieb*. 5th ed. St. Louis: Mosby; 1979.
47. Levitt HL. Modification of appliance design for the adult mutilated dentition. *J Adult Orthod Orthognath Surg*. 1988;1:9.
48. Linde J, Svanberg G. Influence of trauma from occlusion on progression of experimental periodontitis in the beagle dog. *J Clin Periodontol*. 1974;1:3.
49. Shulman J. Teaching patients how to stop bruxing habits. *J Am Dent Assoc*. 2001;132:1275–1277.

50. Betts NJ, et al. Diagnosis and treatment of transverse maxillary deficiency. *Int J Adult Orthod on Orthognath Surg*. 1995;102:75.
51. Levitt HL. Adult orthodontics. *J Clin Orthod*. 1971;5:1130.
52. Melsen B. Adult orthodontics: factors differentiating the selection of biomechanics in growing and adult individuals. *Int J Adult Orthodon Orthognath Surg*. 1988;3:167.
53. Ahrens DG, Shapria Y, Kuftinec M. An approach to rotational relapse. *Am J Orthod*. 1981;80:83.
54. National Institute of Dental Research. *Oral Health of United States Adults, The National Survey of Oral Health in US Employed Adults and Seniors: 1985–86*, publication No. DHHS (NIH)87-2868. Bethesda, MD: National Institute of Dental Research; 1987.
55. Thilander B. Orthodontic treatment in dentitions with reduced periodontal support. *Rev Belge Med Dent*. 1982;37:119.
56. Roblee RD. *Interdisciplinary Dentofacial Therapy: A Comprehensive Approach to Optimal Patient Care*. Chicago: Quintessence; 1994.
57. Polson AM, Meiner SW, Zander HA. Trauma and progression of marginal periodontitis in squirrel monkeys. III. Adaptation of interproximal alveolar bone to repetitive injury. *J Periodont Res*. 1976;12:279.
58. Svanberg G. Effect of trauma from occlusion on the periodontium. In: McNamara Jr JA, ed. *Malocclusion and the Periodontium.* Craniofacial Growth Series, Monograph 15. Ann Arbor: Center for Human Growth and Development, University of Michigan; 1983.
59. Steiner CC. Cephalometrics in clinical practice. *Angle Orthod*. 1959;29:8.
60. Epker BN, Fish LC. *Dentofacial Deformities*. St. Louis: 1986.
61. Linde J. *Textbook of Clinical Periodontology*. 2nd ed. Copenhagen: Munksgaard; 1989.
62. Philstrom BL, et al. Association between signs of trauma from occlusion and periodontitis. *J Periodontol*. 1986;57:1.
63. Vanarsdall RL. Tooth movement as an adjunct to periodontal therapy. In: Genco R, Goldman H, Cohen DW, eds. *Contemporary Periodontics*. St. Louis: Mosby; 1990.
64. Thompson JR. The individuality of the patient in facial skeletal growth, part 2. *Am J Orthod Dentofac Orthop*. 1994;105:117–127.
65. Baker F, et al. Health risks associated with cigar smoking. *JAMA*. 2000;284(6):735–740.
66. Burgan S. The role of tobacco use in periodontal diseases: a literature review. *Gen Dent*. 1997;45(5):440–460.
67. Dale LC, Hurt RD, Hays JT. Drug therapy to aid in smoking cessation, tips on maximizing patients chances for success. *Postgrad Med*. 1998;104(6):75–84.
68. Eberman KM, Patten CA, Dale LC. Counseling patients to quit smoking, what to say, when to say it, and how to use your time to advantage. *Postgrad Med*. 1998;1–4(6):89–94.
69. Greene JC. Tobacco use, prevention and cessation: introduction to the special section. *J Dent Educ*. 2002;66(9):1049–1050.
70. Setness PA. Smoking and chewing in young people, the power of prevention is in our hands. *Postgrad Med*. 1997;101(3):13–18.
71. Sinclair PM. The reader's corner. *J Clin Orthod*. 1996;30(4):227–229.
72. Blazi S, Vanarsdall RL. Correlation of Visual Assessment and Instrumental Recording of the Thickness of Attached Gingiva, unpublished research. Philadelphia: University of Pennsylvania; 1984.
73. Geiger A, Hirschfeld L. *Minor Tooth Movement in General Practice*. 3rd ed. St. Louis: Mosby; 1974.
74. Ammons WF, Smith H. Flap curettage: rationale, techniques, and expectations. *Dent Clin North Am*. 1976;20:215.
75. Greenstein G, Canton J, Polson AM. Histologic characteristics associated with bleeding after probing and visual signs of inflammation. *J Periodontol*. 1981;52:420.
76. Machen DE. Developing protocols for adult patients. *Am J Orthod Dentofac Orthop*. 1990;98:476.
77. Baird JF, Kiyak HA. The uninformed orthodontic patient and parent: treatment outcomes. *Am J Orthod Dentofac Orthop*. 2003;124:212–215.
78. Lazaridou-Terzoudi T, Kiyak HA, Moore R, et al. Long-term assessment of psychological outcomes of orthognathic surgery. *J Oral Maxillofac Surg*. 2003;61:545.
79. Broder HL, Phillips C, Kaminetzky S. Issues in decision making: should I have orthognathic surgery? *Semin Orthod*. 2000;6(4):249–258.
80. Cassidy Jr DW, Herbosa EG, Rotskoff KS, et al. A comparison of surgery and orthodontics in "borderline" adults with Class II, Division 1 malocclusions. *Am J Orthod Dentofac Orthop*. 1993;104:455–470.
81. Berns JM. *What Is Periodontal Disease?* Chicago: Quintessence; 1982.
82. Christensen GJ. Educating patients: a new necessity. *J Am Dent Assoc*. 1993;124:86–87.
83. Sfikas PM. Informed consent and the law. *J Am Dent Assoc*. 1998;29: 1471–1473.
84. Christen AG, Klein JA. *Tobacco and Your Oral Health*. Chicago: Quintessence; 1997.
85. Burt BA. Dental practice in the aging society. In: Carlson DS, ed. *Orthodontics in an Aging Society,* Craniofacial Growth Series: Monograph 22. Ann Arbor: Center for Human Growth and Development, University of Michigan; 1989.
86. Kvam E. Traumatic ulcers and pain in adults during orthodontic treatment. *Commun Dent Oral Epidemiol*. 1989;17:154.
87. Griffiths CS, Abby M. Effects of malalignment of teeth in the anterior segments on plaque accumulation. *J Clin Periodont*. 1981;8:481.
88. Boyd RL, et al. Periodontal implications of orthodontic treatment in adults to reduced or normal periodontal tissues versus adolescents. *Am J Orthod Dentofac Orthop*. 1989;96:191.
89. Osterberg T, Carlsson GE. Dental state, prosthodontic treatment and chewing ability—a study of five cohorts of 70-year-old subjects. *J Oral Rehabil*. 2007;34:8.
90. Misch CE. *Contemporary Implant Dentistry*. 3rd ed. St. Louis: Elsevier Mosby; 2008.
91. Huiskes R. If bone is the answer, then what is the question? *J Anat*. 2000;197(Pt 2):145–156.
92. Brown IS. Effects of orthodontic therapy on periodontal pockets. I. Clinical findings. *J Periodontol*. 1973;44:742.
93. Weinstein S, Haack DC, Morris LY, et al. On an equilibrium theory of tooth position. *Angle Orthod*. 1963;33:1–26.
94. Masella RS, Meister M. Current concepts in the biology of orthodontic tooth movement. *Am J Orthodont Dentofac Orthop*. 2006;129(4):458–468.
95. Picton DCA, Moss JP. The part played by the transseptal fibre system in experimental approximal drift of the cheek teeth of monkeys (*Macaca iris*). *Arch Oral Biol*. 1973;18:669.
96. Horowitz S, Hixon EH. Physiologic recovery following orthodontic treatment. *Am J Orthod*. 1969;55(1):1–4.
97. Holdaway RA. Changes in relationship of points A and B during orthodontic treatment. *Am J Orthod*. 1956;42:176.
98. Casko JS, Shepherd WB. Dental and skeletal variation within the range of normal. *Angle Orthod*. 1984;54:5–17.
99. Kennedy J, et al. Osteotomy as an adjunct to rapid maxillary expansion. *Am J Orthod*. 1976;70:123.
100. Denison T, Kokich VG, Shapiro PA. Stability of maxillary surgery in openbite vs. non-openbite malocclusion. *Angle Orthod*. 1989;59(1):5–10.
101. Behrents RG. *A Treatise on the Continuum of Growth in the Aging Craniofacial Skeleton*. Ann Arbor: University of Michigan; 1984.

22

Periodontal–Orthodontic Interrelationships

Robert L. Vanarsdall, Jr., Ignacio Blasi, Jr., and Antonino G. Secchi

OUTLINE

Types of Inflammatory Diseases, 623
Gingivitis: The Reversible Lesion, 623
Periodontitis: The Irreversible or Destructive Lesion, 623
High-Risk Factors, 624
Gingival Recession, 626
Biotype, 626
Transverse Dimension, 626
Bone-Anchored Rapid Palatal Expansion, 629
Early Treatment, 633
Tissue Response to Certain Types of Tooth Movements, 634
Extrusion (Eruption), 634
Intrusion, 636
Tipping, 637
Bodily Movement into a Defect, 637
Mucogingival Considerations, 640
Mandibular Midline Frenum, 640
Maxillary Midline Frenum, 640
Gingival Hyperplasia, 642
Gingival Retention and Aesthetic Considerations, 642
Fiberotomy, 644
Mouth Breathing, 644
Considerations with Ectopically Positioned and Unerupted Teeth, 645
Surgical Technique, 647
Maxillary Palatal Impactions, 647
Second Premolar and Second Molar Impactions, 650
Incisor Impactions, 650
Ankylosis and External Root Resorption, 650
Alveolar Decortication and Augmentation Grafting, 661
Background, 661
Alveolar Bone Biological Response (Regional Acceleratory Phenomenon), 661
Surgical Technique, 662
Decortication, 662
Particulate Grafting, 662
Patient Management, 663
Treatment Indications and Limitations, 663
Summary, 665

As orthodontists enter the 21st century, the adoption of evidence-based health care[1] and the invention of new preventive strategies are primary goals. Recent studies have provided compelling evidence of the importance of these two objectives.

Orthodontics is the most conservative and predictable treatment to improve many of the local etiologic factors that contribute to periodontal diseases and periodontal breakdown.

For example, statistically significant periodontal differences have been demonstrated in patients with crossbite, excessive overjet, and crowding compared with members of a control group.[2] Another investigation has shown that alveolar bone height in 19-year-old men was reduced in regions of severe malocclusion (8 mm of overjet) compared with the corresponding regions in healthy men who had near ideal occlusion.[3] A recent study evaluated the relationship between the irregularity of teeth and the incidence of periodontal disease; the results indicated that overlapping of incisors is related directly to gingivitis and that this relationship could not be explained simply by an effect on oral hygiene.[4] In a similar investigation involving 15 adults, subgingival bacterial samples were taken from anterior crowded and uncrowded teeth, and control samples were taken interproximally between two aligned teeth on the contralateral side within the same sextant.[5] Statistically, the pathologic levels of bacterial cells were significantly higher in the anterior crowded dentition than in the anterior well-aligned dentition. Furthermore, as crowding increased, the number of subgingival microorganisms increased at a predictable rate. This report offers strong evidence that orthodontics can promote periodontal health and even can prevent periodontal disease. Previous reports tended to conclude that malalignment of teeth could contribute to the establishment of periodontal disease only when poor oral hygiene was a factor.[6] Periodontal inflammatory response is considered to be a reaction to the local microbial biofilm. However, the biofilm may not be the determinant of the progression of the disease to periodontitis.[7] The concept of periodontitis occurs when the periodontal tissues provide an ecologic environment for opportunistic bacteria to exist and flourish.[8]

The short- and long-term successful outcomes of orthodontic treatment are influenced by the patient's periodontal status before, during, and after active orthodontic therapy. The long-term prognosis of the natural dentition depends to a significant extent on optimal responses and systemic resistance and on the patient's predisposition to the different clinical forms of

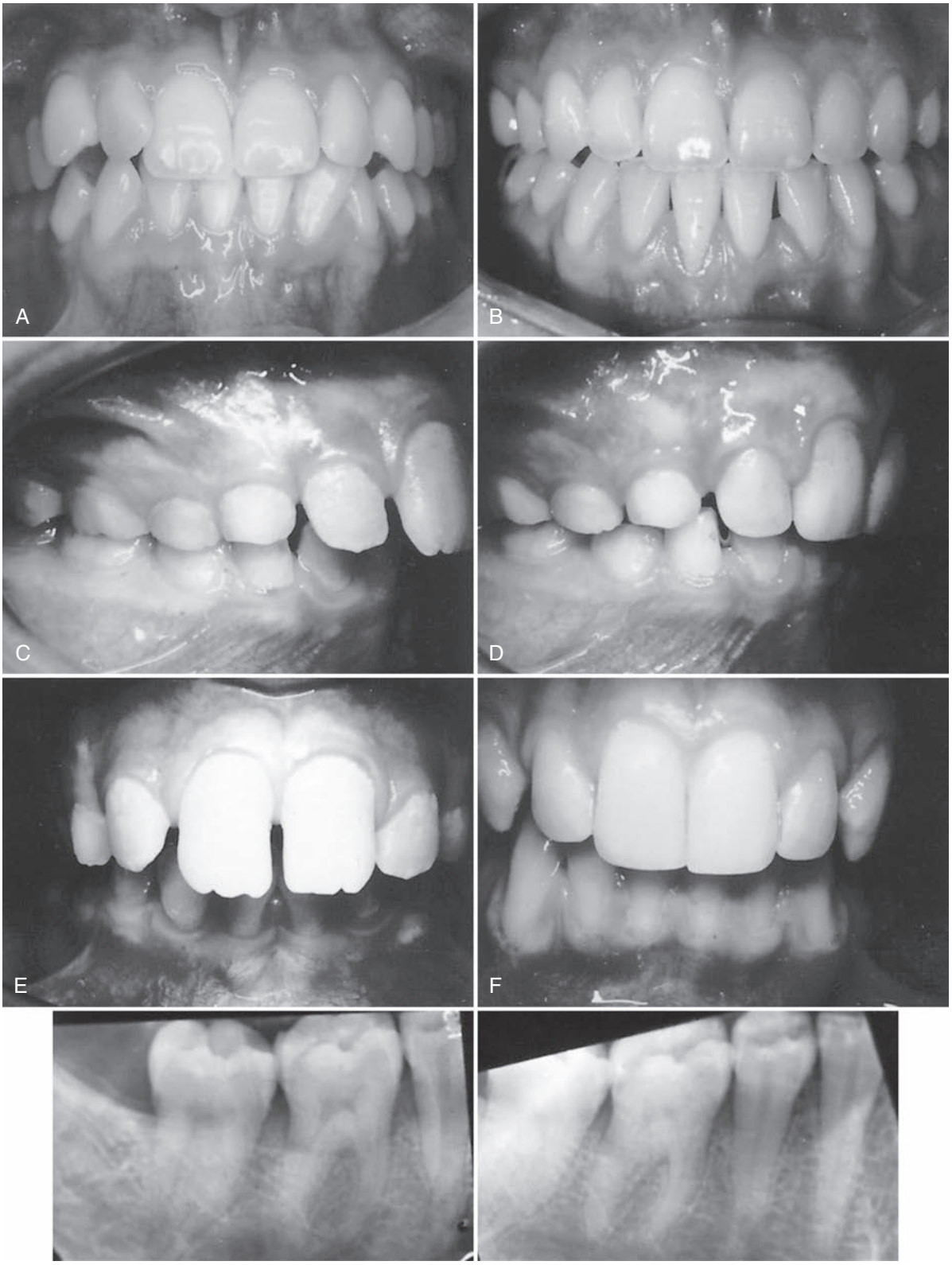
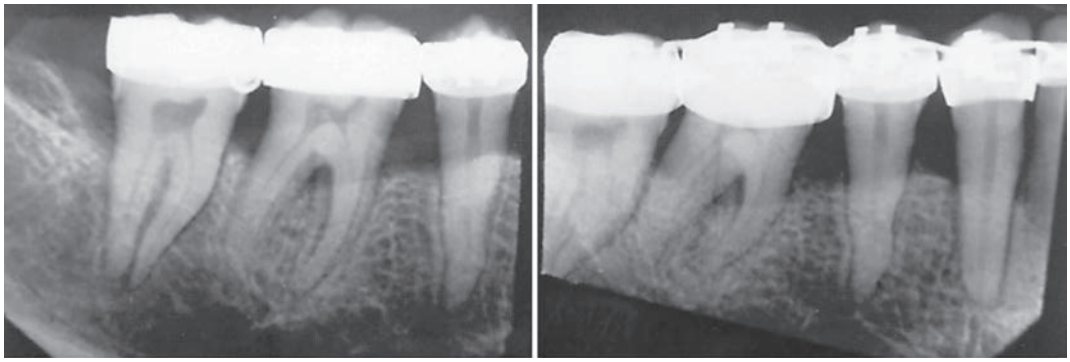


FIGURE 22-1 **A,** A 12-year-old girl before orthodontic treatment. **B,** After removal of the appliances. Note the gingival recession, labial root exposure, and interproximal space under contacts in the mandibular incisor area. **C,** Highly susceptible 9-year-old boy with thin, friable tissue before phase I therapy involving a headgear. **D,** Note the good cooperation, resulting in distalization of the maxillary dentition. **E,** Anterior view before placement of orthodontic appliances. **F,** Anterior view at the end of phase I shows excessive exposure of the anatomic crowns and beginning root exposure on all lower anterior teeth. **G,** Periapical radiographs of a woman's teeth taken before placement of orthodontic appliances.



H

FIGURE 22-1, cont'd H, Several months later, progress films are taken because of excessive tooth mobility. Note the radiographic evidence of accelerated bone loss.

periodontal diseases. Periodontal pathogenesis is a multifactorial process, and the orthodontist must recognize the clinical forms of inflammatory periodontal diseases. At the 1989 World Workshop in Clinical Periodontics, a classification system was established for the various types of periodontal infections (microbial combinations) even though these disorders still may be difficult to distinguish by their clinical features.[9] The need for a revised classification system for periodontal diseases was emphasized during the 1996 World Workshop in Periodontics.[10] In 1999, the International Workshop for a Classification of Periodontal Diseases and Conditions agreed on a new classification.[11] Classification systems should always be considered "a work in progress." The systems should be adapted as more is known about the basic characteristics of the diseases and conditions.[12]

No matter how talented the orthodontist, a magnificent orthodontic correction can be destroyed by failure to recognize periodontal susceptibility (Fig. 22-1). Therefore, identifying periodontally susceptible patients is critical for the outcome of treatment.

TYPES OF INFLAMMATORY DISEASES

Gingivitis: The Reversible Lesion

The accumulation of microorganisms around teeth can cause gingival redness, bleeding, and edema; changes in gingival morphology; reduced tissue adaptation to the teeth; an increase in the flow of gingival crevicular fluid; and other clinical signs of inflammation.[13,14] Obviously, mechanical removal of plaque can reduce chronic gingivitis, but many orthodontic patients may not be motivated to remove the plaque; therefore, plaque removal must be done by the referring dental office or through other professional services such as scaling and thorough tooth cleaning during orthodontic treatment. Patients whose compliance is poor with a manual toothbrush may be more willing to use an electric device (e.g., Interplak [Conair, Stamford, CT], Rota-dent [Professional Dental Technologies, Batesville, AR], Oral-B plaque remover [Boston, MA]) that can improve oral hygiene.[15]

The removal of supragingival bacteria has been shown to have an inhibiting effect on the formation of subgingival plaque. One of the more effective antiplaque, antigingivitis agents is chlorhexidine digluconate; however, the orthodontist must monitor the side effects of reversible staining, tissue response, and taste alterations. Exaggerated gingival overgrowth and response to plaque have been reported with hormonal changes in pregnancy, with puberty, during the menstrual cycle, and in response to steroid therapy and birth control medications.[16]

Gingivitis (without attachment loss) has been classified as having initial, early, and established stages.[17] The initial lesion develops 1 to 2 days after plaque is allowed to remain on the teeth. The established lesion develops weeks later, when inflammation has advanced to proliferation of epithelium into the connective tissue. Only the established lesion can be observed as clinical gingivitis. The important point is that alveolar bone loss has not yet occurred, and one can hope that the lesion can be prevented from spreading into the supporting tissues. Therefore, determining the appropriate plaque control interval for the patient is crucial to prevent destructive alveolar bone loss. Pseudo-pockets or gingival overgrowth or enlargement of the gingival margin and papilla, whether they are drug induced (e.g., phenytoin or cyclosporine) or primarily plaque related, are exacerbated by poor oral hygiene. Constant professional reinforcement is necessary.

Periodontitis: The Irreversible or Destructive Lesion

Although gingival inflammation may be a prerequisite, the actual mechanisms responsible for conversion of gingivitis to periodontitis are still being debated.[18] All forms of periodontitis are marked by loss of connective tissue attachment and usually gingival inflammation.

Chronic periodontitis is the most common form of periodontitis. One of every two American adults aged 30 years and older has periodontal disease, according to recent findings from the Centers for Disease Control and Prevention. Around 47.2%, or 64.7 million American adults, have mild, moderate, or severe periodontitis, the more advanced form of periodontal disease. In adults 65 years of age and older, prevalence rates increase to 70.1%.[19] The organisms most often reported that have been associated with adult periodontitis are the ones associated with the red complex, which include *Porphyromonas gingivalis*, *Tannerella forsythensis*, and *Treponema denticola*. These bacteria are usually found together in periodontal pockets, suggesting that they may cause destruction of the periodontal tissue in a cooperative manner.[20]

Chronic periodontitis results in inflammation within the supporting tissues of the teeth, progressive attachment, and bone loss. It is characterized by the formation of pockets or recession of the gingiva (or both). It is prevalent in adults but

can also occur in children and adolescents. Conventionally, this form of periodontitis has been characterized as a slowly progressive disease. However, data indicate that some patients may experience short periods of rapid progression.[11] Good plaque control as well as removal of bacteria and calculus deposits from the subgingival environment by mechanical instrumentation constitutes an effective approach to reduce signs of inflammation in most cases of chronic periodontitis.

Aggressive periodontitis, by definition, causes rapid destruction of the periodontal attachment apparatus and the supporting alveolar bone. It can present in a localized or generalized form. It occurs in patients who are otherwise clinically healthy (Fig. 22-2). The primary features to diagnose the disease include a history of familial aggregation with rapid attachment loss and bone destruction. Secondary features include elevated proportions of *Aggregatibacter actinomycetemcomitans* and, in some far East populations, *Porphyromonas gingivalis*; phagocyte abnormalities; hyper-responsive macrophage phenotype, including elevated production of prostaglandin E2 (PGE2) and interleukin-1β (IL-1β) in response to bacterial endotoxins; and progression of attachment loss and bone loss may be self-arresting. There is compelling evidence that adjunctive antibiotic treatment frequently results in a more favorable clinical response than mechanical therapy alone. Indeed, several authors have reported success in the treatment of aggressive periodontitis using antibiotics as adjuncts to standard therapy.[11,21,22]

Periodontitis also can be associated with **systemic diseases** such as familial and cyclic neutropenia, Down syndrome, Papillon-Lefèvre syndrome, hypophosphatasia, and leukemias.[11,23]

Necrotizing periodontal diseases is an infection characterized by necrosis of gingival tissues, periodontal ligament (PDL), and alveolar bone. The major consistent symptoms noted are pain and bleeding, but low-grade fever and halitosis (bad breath) may also be noted. These lesions are most commonly observed in individuals with systemic conditions such as HIV infection, malnutrition, and immunosuppression. Treatment can be successful if begun early and if there is no predisposing systemic illness. Acute-phase emergency treatment may include pain relief, debridement of necrotic gum tissue, antiseptic mouth rinses, thorough regular dental cleaning and flossing, oral antibiotics if it is severe or there are signs of systemic infection, and antifungal agents for immunosuppressed patients treated with antibiotics. Also, it is important to treat any predisposing illness or trigger (e.g., smoking). Finally, surgical correction should be done of any remaining defects such as craters with gingivectomy and gingivoplasty. It is essential to maintain good dental hygiene and good health to reduce the risk of recurrence. However, where there is a predisposing condition such as AIDS, recurrence or relapse is common.[24,25]

The 1989 classification did not include a section on periodontal abscesses and a connection between periodontitis and endodontic lesions, nor a section on developmental or acquired deformities and conditions. This was remedied by the addition of these lesions the International Workshop for a Classification of Periodontal Diseases and Conditions held in 1999.[26]

In general, one should recognize that children can develop severe forms of periodontitis, but the prevalence of destruction is much less than in adults. Severe bone loss in a child or adolescent also may be an early sign of systemic disease and may indicate the need for medical evaluation.

As is described in this chapter, orthodontic patients may be at greater risk of attachment loss after teeth have become mobile because of tooth movement (see Fig. 22-1, *G* and *H*). The orthodontist must recognize and control clinical signs of inflammation and tooth mobility during treatment to prevent extensive bone loss. Identification of occlusal trauma should be done clinically by identifying fremitus and increased tooth mobility and radiologically by the widened PDL and bone loss.[27,28] The signs of occlusal trauma are reversible. The trauma must be eliminated and teeth protected from the adverse effects of plaque-induced inflammation to allow healing of the attachment apparatus.[29] Periodic monitoring of the periodontal status with probing, microbiologic assessment by immunologic assays, DNA probes, and culturing, as well as clinical findings are useful in determining scaling intervals and detecting potential sites of increased risk of attachment loss. These methods may be used to assess the end point of the effectiveness of scaling and root planing before orthodontic treatment to ensure that no putative pathogens remain. Genetic studies offer potential benefit in identifying high-risk individuals,[30] such as those with neutrophil chemotactic abnormalities who are family members of patients with aggressive periodontitis; such individuals are 10 times more likely to develop periodontal disease than most family members.

HIGH-RISK FACTORS

The orthodontist must assess each patient for periodontal factors that may place the patient at higher risk of developing periodontal disease during orthodontic therapy.

A significant dental history could include unsuccessful orthodontic treatment at an earlier age. A patient with a history of previous periodontitis[31] obviously is more susceptible to the disease. Although predicting which sites may progress from gingival inflammation to periodontitis is difficult, those who have had the disease previously are more vulnerable to further bone loss. In fact, in individuals who have had previous bone loss, gingivitis may be a greater threat for further bone loss.[32] No one should begin orthodontic treatment if active destructive sites are present, and a person who has had periodontal disease before should be monitored more closely to prevent new bursts of active sites that may result in rapid bone loss. A small percentage of adolescents (10%) and a much larger group of susceptible adults (50%) must be treated differently. Other risk factors include gingival bleeding from probing; tooth mobility; and thin, friable gingival tissue. This chapter discusses each of these factors. In addition, tobacco use and diabetes mellitus have proved to be risk factors for a higher prevalence of periodontal disease.[33–35]

The orthodontist should record a list of periodontal risk factors for any patient who seeks orthodontic treatment. Risk factors are important, for example, in identifying patients who will need a special management approach with individualized periodontal recall intervals. The orthodontist must control for risk factors before beginning orthodontic treatment so that periodontal problems are less likely to develop. Stress, diabetes mellitus, tobacco smoking, osteoporosis, and genetic predisposition are examples of disease modifiers.[36] Genetic testing could be used as part of risk assessment for the development of future disease, and the patient can be informed of the greater risk of developing disease.[30,37]

FIGURE 22-2 A 14-year-old patient seeking orthodontic treatment. Upon clinical and radiographic examination, the patient was diagnosed with localized aggressive periodontitis and referred for phase I periodontal treatment. **A, B,** Intraoral photographs before treatment. **C,** Note probing depth of 11 mm on the affected area. **D,** Periapical film showing an angular bone defect. The patient was treated with scaling and root planing and adjunctive antibiotic therapy as phase I. Then extrusion during orthodontic treatment was completed. **E, F,** Probing depth of 3 mm after treatment and periapical film showing healing of the angular crest defect. **G, H,** Final intraoral pictures after orthodontic treatment. (Case treated by Drs. Blasi and Bockow.)

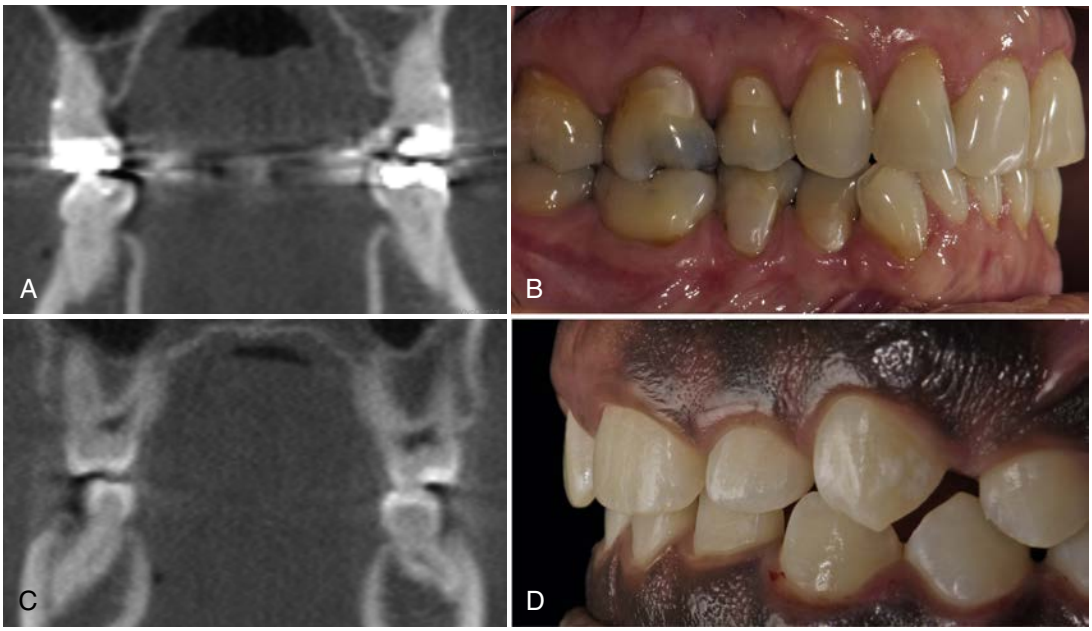


FIGURE 22-3 A, Coronal cone-beam computed tomography cut of a treated patient with a thin hard tissue biotype; note the minimum width of the cortical plate. B, The gingival of the same patient presents with an average soft tissue biotype. C, The thick bone tissue biotype correlates to the thick soft tissue biotype (D); the patient presents with altered passive eruption (APE).

The clinician must identify susceptible patients and develop strategies to prevent loss of attachment and gingival recession.

Gingival Recession

Gingival recession is not uncommon in young adults and could be related to past orthodontic treatment.[38] A recent study by Remeka et al.[39] concluded that the prevalence of gingival recessions steadily increases after orthodontic treatment. They observed that there was a continuous increase in gingival recessions after orthodontic treatment with full fix appliances from 7% at end of treatment to 20% at 2 years after treatment and to 38% at 5 years after treatment. The same group concluded that orthodontic treatment and the retention phase may be risk factors for the development of labial gingival recessions.[40]

Orthodontic movement of teeth should be carefully made within the alveolar housing. A failure to a correct diagnosis could make orthodontics an etiologic factor to gingival recession.

Biotype

The periodontal tissue and its clinical appearance may differ from subject to subject.[41] The tissue thickness on the faciopalatal dimension has been described as a biotype.[42–48] The identification of the hard and soft tissue biotype is important in clinical practice since differences in gingival and osseous architecture have been shown to exhibit a significant impact on maintaining the integrity of the periodontal tissues during treatment (Fig. 22-3).

Subjects with thick-flat biotype have wider and voluminous gingiva and slightly scalloped or flat gingival margin with short and wide teeth; the contact areas between the teeth are large and more apically located, and the interdental papillae are short. On the other hand, a person with a thin-scalloped biotype has a narrow zone of gingiva, highly scalloped gingival margin, long and slender teeth with tapered crown form, and interdental contact areas that are located incisally.[49,50] In the presence of inflammation, a flat periodontium results in pocket depth; a scalloped periodontium results in gingival recession.[41]

Gingival biotype can be evaluated by direct visual assessment only,[50,51] recognizing the washboard effect, visual assessment with visual periodontal probe through the tissue,[52] and direct measurement with gingiva perforation.[53–55]

Although the hard tissue and bone biotype could be evaluated with cone-beam computed tomography (CBCT) (see Fig. 22-3, *A* and *C*), not yet with 100% accuracy,[56,57] the only diagnostic method to accurately determine the hard tissue biotype is direct visualization with an open flap. On the other hand, bone sounding of each individual tooth remains the gold standard in the evaluation of alveolar crest position.

The periodontal biotype is partially related to the dimensions of alveolar process, the shape of the teeth and tooth eruption, rotations, inclinations or other malpositions.[58–60]

Even though subjects with a thin-scalloped gingival biotype are more likely to have thin, hard tissue architecture, variations occur among individuals.[58] Patients with thin or thick gingival tissue might present with thin or thick bone tissue (see Fig. 22-3).

The biotype of the soft and hard tissue may play a critical role in the outcome of treatment.[61] A thin-scalloped biotype has a higher prevalence of gingival recession.[41,62,63] Therefore, an accurate diagnosis and special attention should be given to hard and soft tissue biotype when treatment planning and before proceeding with orthodontic treatment to achieve predictable outcomes.

Transverse Dimension

A risk marker is a characteristic that identifies a patient who is likely to have periodontal disease. In this regard, the transverse

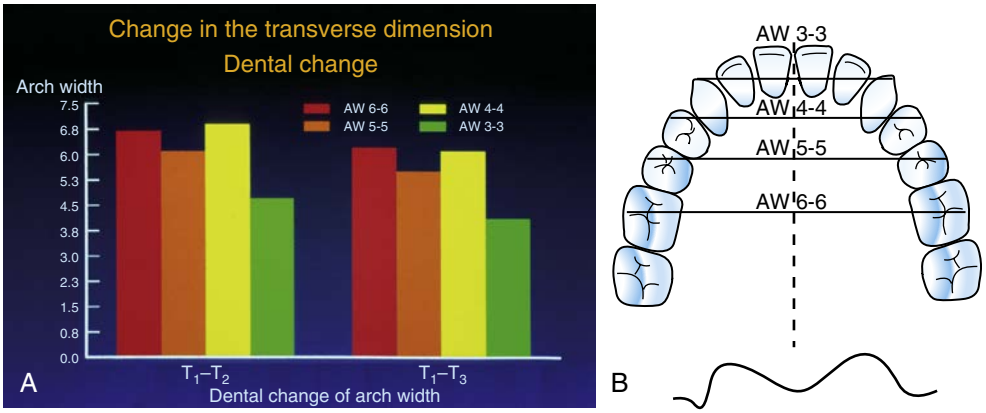


FIGURE 22-4 **A,** A histogram of maxillary dental change, with rapid palatal expansion from T_1 (before expansion) to T_2 (completion of expansion). The second group of bars represents the expansion that was maintained until T_3 (the final period, 10 years after expansion). **B,** Diagram showing the measurement of maxillary arch dental width with electronic digital calipers for each tooth in the 55 expanded cases. *AW,* Arch width.

skeletal pattern may be the most crucial evaluation in gauging the potential for facial gingival recession.

In a study on patients from two private orthodontic practices conducted at the University of Pennsylvania, 55 patients (ages 8–13 years at the time of treatment with a Haas expander) were recalled 8 to 10 years after rapid palatal expander (10–10.5 mm over 3 weeks) and compared with 30 control patients (matched for age) from the same practices who were evaluated for stability and clinical crown height after edgewise nonexpansion.[64] The patients were assessed from models, Kodachrome slides, and posteroanterior (PA) cephalograms made before and at the end of orthodontic treatment; they were recalled 10 years later for examination and new records. The study indicated that of the expanded cases, although the results were stable (Fig. 22-4), 11 (20%) showed unusual facial gingival recession on one or more teeth compared with only 6% gingival recession in the nonexpanded control patients (Fig. 22-5). Skeletally, 9 of the 11 patients who had undergone expansion and experienced facial gingival recession had maxillary transverse deficiency and mandibular transverse excess, and the other 2 patients had excessively large mandibles (Fig. 22-6). Therefore, in an adolescent patient with a small maxilla and a large mandible, a potential exists to move teeth beyond the envelope of the alveolar process, predisposing the patient to gingival recession. The envelope of discrepancy for the transverse dimension is much more crucial from a stability and periodontal standpoint (see Chapter 21).

Another study[65] evaluated posttreatment gingival recession in three groups of individuals. At 7 to 10 years after nonextraction treatment, 75 patients treated with RPE therapy (group 1), 22 patients (from the same practice) treated with standard edgewise therapy only (group 2), and an untreated control group (mean age, 17.2 years; group 3) were evaluated for cemental exposure, and PA cephalograms were evaluated to determine whether a transverse skeletal discrepancy existed between the maxilla and mandible for all subjects. The graph (see Fig. 22-6, *H*) shows transverse discrepancy (width of the mandible minus width of the maxilla) larger than the normal value (Rocky Mountain normal for age) expressed as a negative number. Twenty percent of all three groups experienced gingival recession. Individuals in each group who exhibited a negative

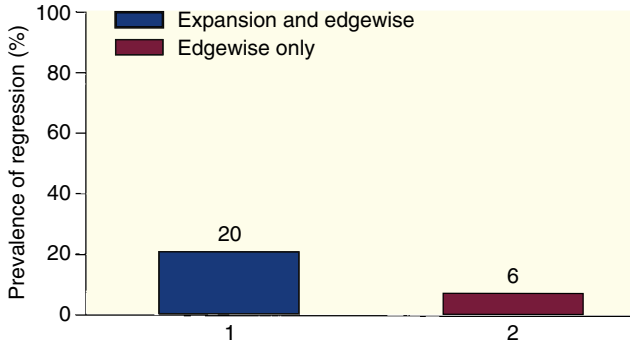


FIGURE 22-5 About 20% (11 of 55) of the expanded cases showed gingival recession compared with only 6% in the edgewise-only treatment group. The hypothesis that recession is distributed evenly across expanded and edgewise groups was rejected ($p = 0.0025$).

skeletal differential greater than 5 mm (from the norm value) experienced gingival recession. The individuals with a normal transverse skeletal differential in all three groups did not experience recession. Therefore, the transverse skeletal severity is a critical risk marker for identifying patients susceptible to gingival recession and periodontal disease. This is important diagnostic information that can result in a treatment plan that may improve future periodontal health for patients in whom the transverse skeletal differential can be corrected.

Untreated susceptible adults with significant transverse skeletal discrepancy show advanced stages of destructive periodontal disease. Hundreds of adult orthodontic retreatment patients have achieved excellent stability from surgically assisted RPE to correct transverse skeletal discrepancy. Growth in the transverse dimension slows severely by 15 years of age and has been reported to be essentially complete.[66,67] Therefore, early orthodontic treatment in the deciduous or early mixed dentition is ideal for correcting transverse skeletal abnormalities orthopedically when growth is significantly active and palatal separation is most effective. It is important to identify the patient's skeletal age to determine whether

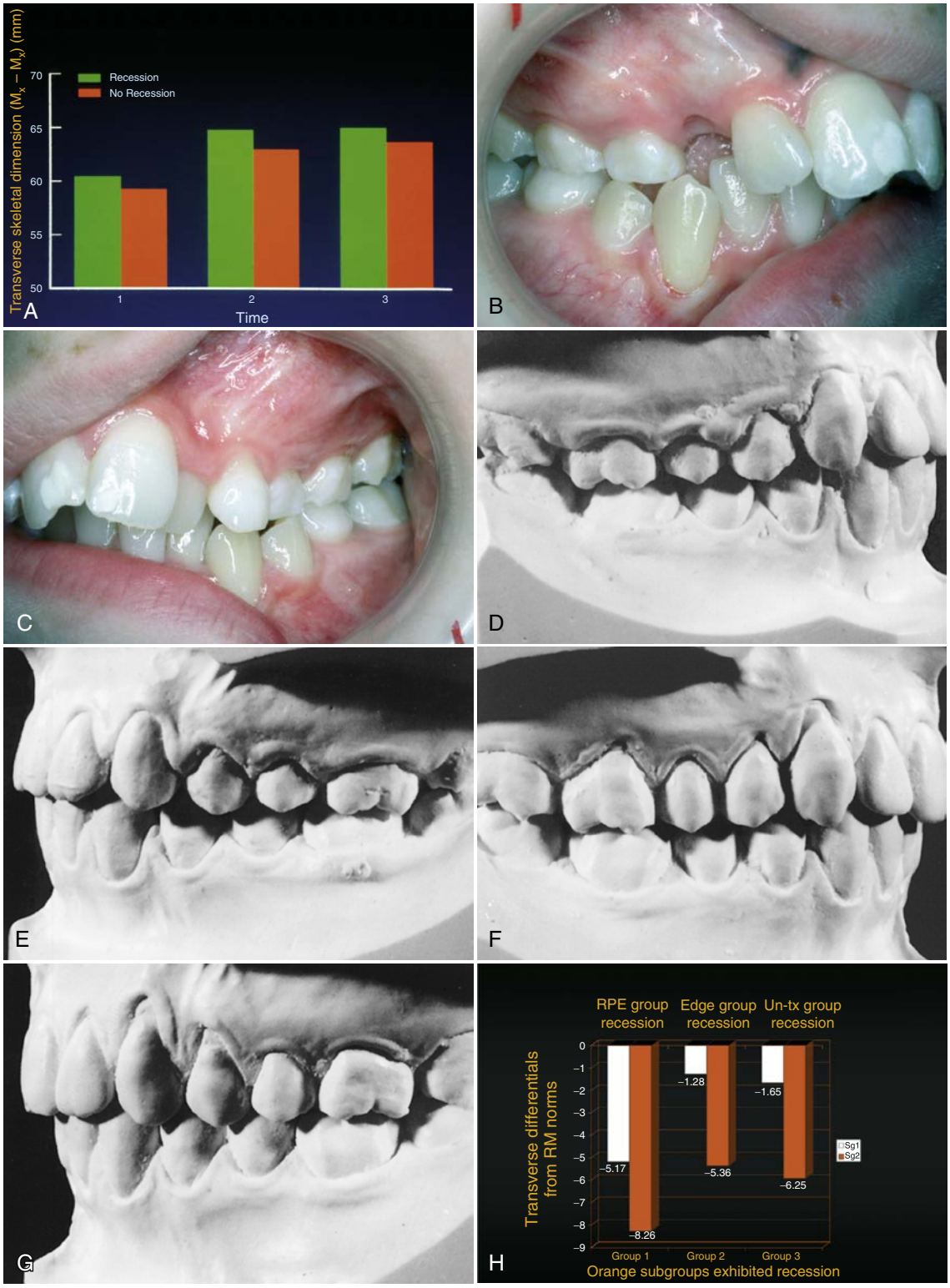


FIGURE 22-6 **A,** From the histogram, note that all 55 of the expanded patients at T_1 had a maxillary (Mx–Mx) transpalatal dimension of about 60 mm (deficient) and were expanded at the bony base 4 to 5 mm at T_2 and at T_3 (final period). The basal skeletal increase remained stable and increased with time only 0.33 mm. Little difference was seen in the maxillary skeletal dimension between the patients who showed recession (11 [*dark screen*]) and the patients who did not (44 [*lighter screen*]). The relationship between the maxillary and mandibular widths was a crucial factor in the evaluation, and this proportional evaluation identified the 11 individuals with problems. **B, C,** Buccal views of a 9.4-year-old girl (Class I, Division 1 condition) with a 9-mm narrow maxilla and an excessively wide mandible in the transverse dimension. Observe that there are no crossbites. **D, E,** Models made when appliance was removed. **F, G,** Models made 10.8 years after completion of treatment. Note the gingival recession in the maxillary buccal segments observed years after the appliances had been removed. **H,** This graph represents the mean differentials (difference between Ricketts Mx–Mx and Ag–Ag measurements in width) for Rocky Mountain norm values in the rapid palatal expansion group 1, orthodontic-only group 2, and control group 3. *White bars* represent the norm-recession individuals, mean differential, and *orange bars* indicate the mean value found for individuals with gingival recession. *Orange* subgroups exhibited recession. All groups were found to have individuals with gingival recession, but recession occurred in those with severe transverse skeletal discrepancy greater than 5 mm.

FIGURE 22-7 A, A 12-year-old girl before rapid maxillary expansion. B, After completion of active expansion. Note the midline diastema and tissue level on the left first premolar. C, Note the tissue level on the maxillary left first premolar before expansion. D, Several weeks after expansion. Note the 3 to 4 mm of recession on the first premolar, which has been reported as an initial complication with rapid palatal expansion treatment.

or not orthopedic expansion would be effective on correcting the skeletal maxillary base. Expansion of buccal segments with fixed appliances has limitations and tends to be unstable, regressing toward pretreatment widths.[68]

Orthopedic expansion of the palate has been studied with the use of implants. This study confirmed 50% dental movement and 50% skeletal movement in young children. In adolescents, however, only 35% of the movement was skeletal, and 65% was dental.[69] Therefore, as the young patient grows older, dental tipping becomes greater, which puts the teeth at higher risk of gingival recession (Fig. 22-7, *A* and *B*). Lindhe[70] has stated that "[when] during orthodontic treatment a tooth is moved through the envelope of the alveolar process . . . at sites with thin and inflamed gingiva [there is] a risk that gingival recession may occur." The labial plate of bone in the maxilla is thin on the facial surfaces of the teeth (Fig. 22-8, *A*). The buccal plate in the mandible is thin in the coronal thirds from the first molar area, moving anteriorly, and is considerably thinner in the premolar/incisor areas (Fig. 22-8, *A* and *C*).

Garib et al.[71] compared the periodontal effects of RPE with tooth-tissue-borne (Haas-type expander) and tooth-borne (Hyrax) using computed tomography. They observed that both expanders reduced the bone buccal plate thickness of the maxillary posterior teeth and induced bone dehiscences on the anchorage teeth buccal aspect (Fig. 22-9). The tooth-borne (Hyrax) expander produced more reduction of first premolar buccal alveolar crest level than did the tooth-tissue-borne expander. Dental expansion and alveolar bending predispose to recession and dental instability if treated beyond the limits of the envelope of discrepancy. Therefore, proper diagnosis and proper selection of the type of appliance should be made.

Bone-Anchored Rapid Palatal Expansion

Temporary anchorage devices (TADs) have raised orthodontics to a completly different level of excellence, expanding the treatment limits, even in apparently impossible cases.

In our 2012 study[72] on patients treated, by one operator alone, with a bone-anchored expander (TAD type), 11 patients (11.3–17 years) were evaluated after expansion and compared with a group of 24 patients[73] (7.8–12.8 years) treated with a bonded tooth-tissue-borne expander (bonded type) from a different practice. The hand–wrist radiograph was used to establish treatment response of the needed correction. The patients were assessed from CBCT data taken before and right after the expansion was completed (Fig. 22-10).

A *t*-test was conducted and found that there were statistically significant differences, with a $p < 0.05$ between the mean of maxillary basal bone change at the first molars of both groups. Furthermore, the percent of the mean screw expansion associated with the width of the palatal expansion at the first molar was calculated. The mean value of the basal expansion with the bonded RPE was 40.65%, and the mean value of the expansion with the TAD RPE was 65.04%. It can be concluded that the large difference in the magnitude of this difference in the efficiency of the expansion was due to the direct effects of the expansion on the palate itself and not the surrounding molars

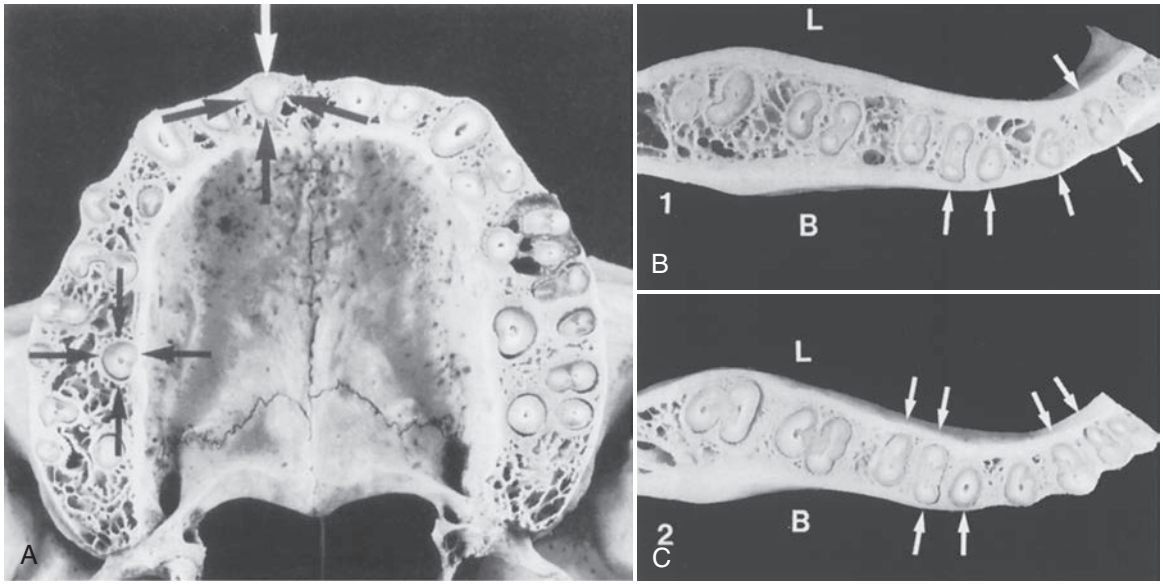


FIGURE 22-8 **A,** Cross-section through the maxillary alveolar process at the midroot level of the teeth, showing the extreme thinness of the buccal bone in the maxillary arch. **B,** and **C,** Cross-section through the mandibular alveolar process at the coronal and apical thirds of the roots. The art illustrates the thin labial bone on the mandibular teeth in the crestal two thirds. *B,* Buccal; *L,* labial. (From Lindhe J. Textbook of clinical periodontology. Copenhagen: Munksgaard; 1989.)

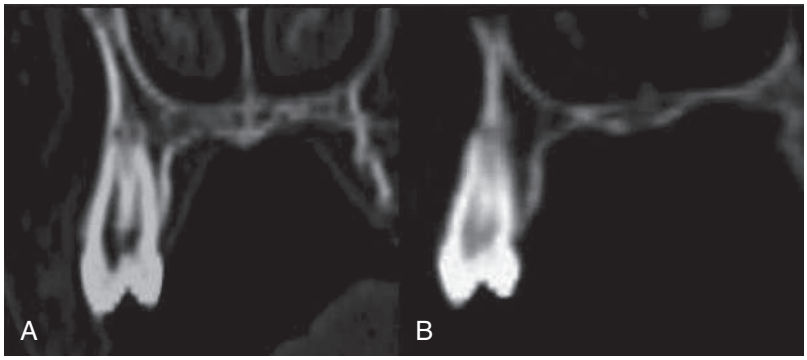


FIGURE 22-9 Patient treated beyond the limits of the envelope of discrepancy. Note the dehiscence created by a tooth-born (Hyrax) rapid palatal expander. **A,** Pretreatment computed tomography (CT) scan of the right first premolar. **B,** Post-RPE CT. (From Garib DG, Henriques JF, Janson G, et al. Periodontal effects of rapid maxillary expansion with tooth-tissue-borne and tooth-borne expanders: a computed tomography evaluation. *Am J Orthod Dentofacial Orthop* 2006;129(6):749–758.)

of the maxillary arch where the bonded tooth-tissue-borne RPE device ordinarily engages (Fig. 22-11).

This interpretation is also strongly supported by the substantial intermolar tipping angle effect of the bonded TX, which resulted in a mean 11.7 standard deviation (SD) ± 3.05-degree difference over the course of TX and the near absence of any mean effect 0.2 SD ± 3.47-degree difference in the case of the TAD TX group. In this case, a *t*-test of the differences between the two groups demonstrated a highly significant difference at $p < 0.00001$ (Fig. 22-12).

Comparing the bone-anchored to the bonded tooth-tissue-borne expander treatment, expansion efficacy demonstrated in both significant skeletal changes. However, the bone-anchored devices achieved (25%) significantly more skeletal change (Mx–Mx) without dental compensation than did the bonded tooth-tissue-borne device. More maxillary expansion was noted with the bone-anchored versus the bonded tooth-tissue-borne expander, and a highly statistically significant difference in the molar tipping.

With all the emphasis on evidence-based orthodontics, Lagravère et al.[74] reported in a CBCT randomized clinical trial (patients were 14.05–14.54 years old) that bone-anchored maxillary expanders and traditional (Hyrax) rapid maxillary expanders showed similar results. The tooth-borne group exhibited more first premolar expansion than the bone-anchored appliance, and both exhibited significant increase in

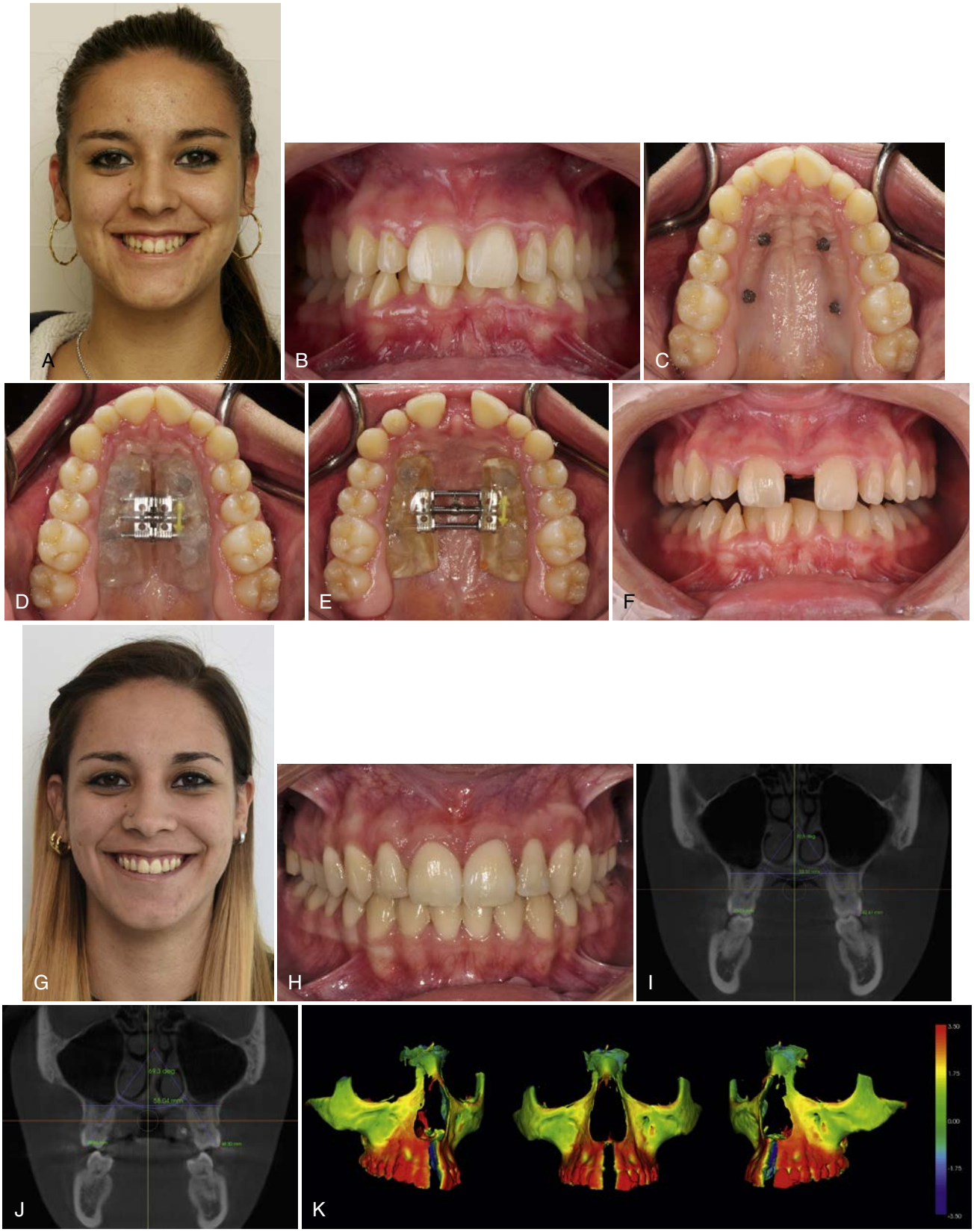


FIGURE 22-10 **A, B,** A 16-year-old girl before rapid maxillary expansion with a bone-anchored expander. Note the posterior negative spaces. **C,** Four temporary anchorage devices (TADs) were inserted between the canine and first premolar and between the second premolar and first molar. **D,** Rapid palatal expander (RPE) cemented 1 week after TAD insertion. **E, F,** After completion of active expansion. Note the midline diastema and minimum dental tipping. **G, H,** Final treatment outcome 18 months of treatment duration. Peg laterals were restored, and diastema was closed to the patient's facial midline. Note that the posterior negative spaces were eliminated. **I, J,** Coronal cone-beam computed tomography cuts at the level of the first molar. Note the molar inclination compensating for the narrow maxilla. The comparison of before (**I**) RPE and after (**J**) RPE exhibits 5.68 mm of skeletal expansion achieved at the Mx level of the maxilla and a negative (−1.2 degrees) dental tipping caused by a purely skeletal expansion. **K,** Treatment evaluation in three dimensions after RPE. Note the skeletal change on the color-mapping superimposition.

crown inclinations. They found that dental expansion was greater than skeletal expansion. However, they only evaluated dental objectives and focused on the dental correction of posterior crossbites. There were no skeletal objectives and/or evaluation of the skeletal transverse dimension changes at the basal bone level. In addition, they used only two TADs for their appliance design, while it has been reported that the use of four TADs on the palatal slopes makes a better force distribution.[75]

Our findings have been substantiated by Lin et al.[76] They evaluated and compared the effects of a tooth-borne expander (Hyrax; $n = 13$; age = 17.4 ± 3.4 years) and a bone-borne expander ($n = 15$; age = 18.1 ± 4.4 years). The bone-borne expander group produced more skeletal expansion except in the first premolar area that exhibited alveolar tipping. The Hyrax group had more buccal tipping of the dentition and alveolar process, except in the area of the second molar, and significant adverse buccal dehiscences at the first premolar level. The authors concluded that the bone-borne expanders produced greater orthopedic changes and fewer dentoalveolar tipping than the Hyrax expander.

Skeletal anchorage should permit orthopedic change without the adverse dental changes by applying force directly to the maxillary bone.[77a] It has been reported that the skeletal-anchored RPE produced less molar tipping than the tooth-tissue-borne RPE.[72,76]

The skeletal anchorage could be reserved for moderate to severe skeletal discrepancies, skeletally mature individuals, periodontally involved cases, or patients with missing teeth.

The palatal skeletal change that is predictably possible remains unknown, and future research is needed. Even though there is also a need to determine the skeletal age barriers of the bone-anchored RPE, it is clear that the envelope of discrepancy has been increased to include older patients (Fig. 22-13).

The keys to diagnosis and treatment of the transverse dimension are skeletal age that determines the response to

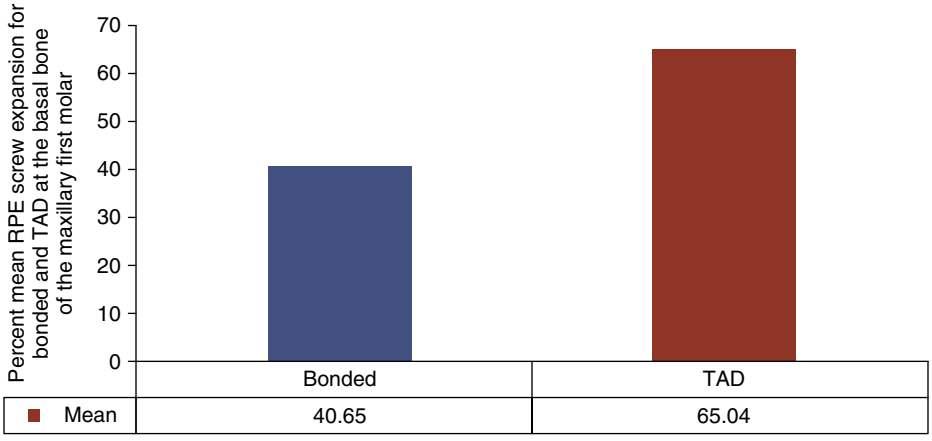


FIGURE 22-11 Histogram showing percentage of the basal bone expansion of the mean jackscrew opening at the levels of the first permanent molars. The mean value of the basal expansion with the bonded rapid palatal expander (RPE) was 40.65%, and the mean value of the expansion with the bone-anchored RPE was 65.04%.

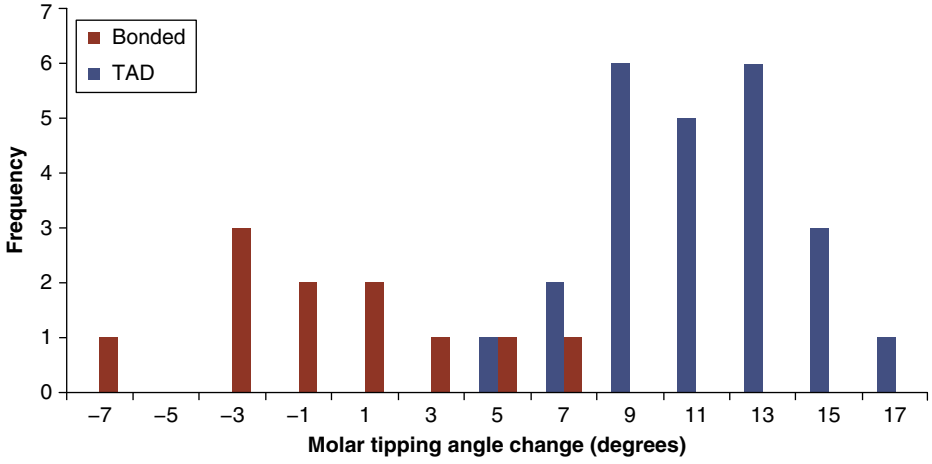


FIGURE 22-12 Comparison of the bonded tooth-tissue-born rapid palatal expander (RPE) and bone-anchored RPE intermolar tipping angle change. A *t*-test of the differences between the two groups exhibited a highly significant difference ($p < 0.00001$). *TAD,* Temporary anchorage device.

treatment, the severity of the discrepancy of the skeletal pattern (the quantitative difference between the maxillary and mandibular basal bone and not the dental relationship), and the selection of the appliance (Haas, 20% basal change; bonded RPE, 41% basal change; TAD RPE, 65% basal change) (Fig. 22-14).

Facial movement of the teeth into thin tissues has been tested in monkeys. Extensive bodily movement of incisors in a labial direction through the alveolar bone (Fig. 22-15) resulted in small apical displacement of the gingival margin, which appeared to be thinned by the tooth movement, and reduction of the alveolar bone height.[77] Therefore, less tooth movement and greater orthopedic change in the transverse dimension could be obtained by a bone-anchored RPE or surgical adjunct, which has not been shown to create the adverse gingival changes seen with orthopedic expansion in severe or skeletal transverse maxillary deficiency problems. The decision to use a surgical adjunct is based on comparison of the maxillomandibular proportions with individual normal variations. The transverse skeletal discrepancy is corrected (1) to prevent periodontal problems; (2) to achieve greater dental and skeletal stability (Fig. 22-16, *A–C*); and (3) to improve dentofacial aesthetics by eliminating or improving lateral negative space, which accompanies maxillary transverse deficiency (Fig. 22-16, *D–G*).

EARLY TREATMENT

A transverse correction may have more periodontal significance than an AP correction. In addition to changing skeletal characteristics, studies have shown that posterior airway space increased (Fig. 22-17), and there has been a considerable decrease in obstructive sleep apnea after expansion procedures of the maxilla as well.[78,79]

Rapid maxillary expansion is the orthopedic treatment used to correct the skeletal transverse problem in children.[80] Proper orthopedic treatment with skeletal landmarks as goals to correct the skeletal transverse dimension does not required overcorrection. The mandible can be adjusted using a mandibular lip bumper appliance in conjunction with a maxillary RPE.[81] Through the interaction of the tongue and lip bumper, spontaneous decrowding of the lower incisors occurs as space is gained by growth and widening. The lip bumper allows for dentoalveolar widening and reshaping, which are induced without direct attachments to the teeth except contact at the molar tubes. For most practitioners, diagnosis of the transverse dimension is

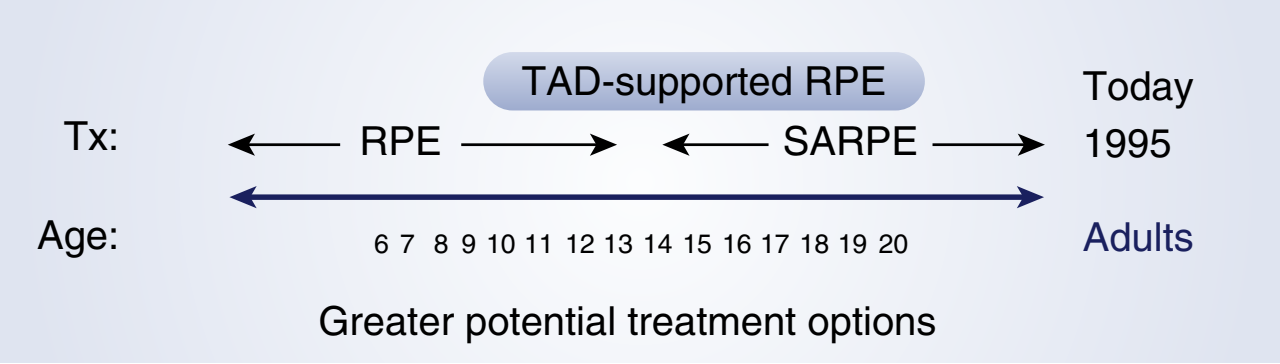


FIGURE 22-13 Treatment envelope of the transverse skeletal dimension. Today, although there is a need to determine the skeletal age limits of the bone-anchored rapid palatal expander, the envelope of discrepancy has been increased to treat older patients without surgically assisted rapid palatal expansion (SARPE).

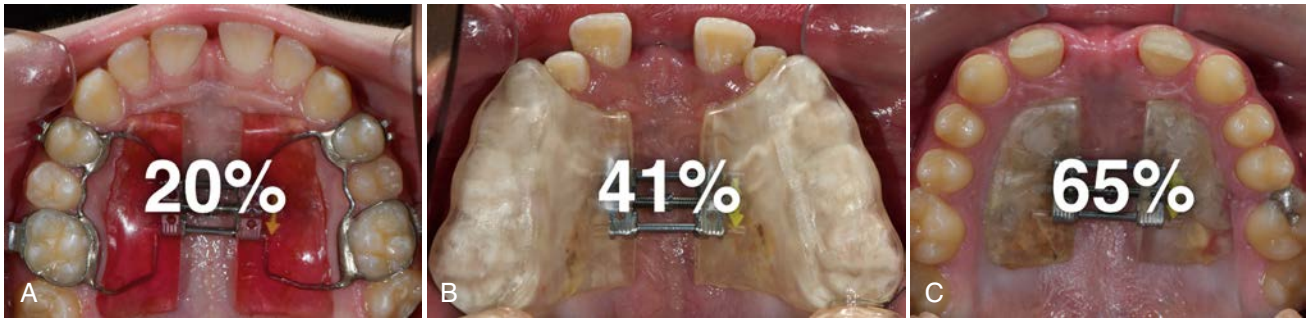


FIGURE 22-14 Selection of appliance is important depending on the skeletal correction needed (transverse skeletal discrepancy between the mandible and maxilla) and the skeletal age. **A,** Haas, 20% basal change. **B,** Bonded rapid palatal expander (RPE), 41% basal change. **C,** bone-anchored RPE, 65% basal change of the mean jackscrew opening at the levels of the first permanent molars.

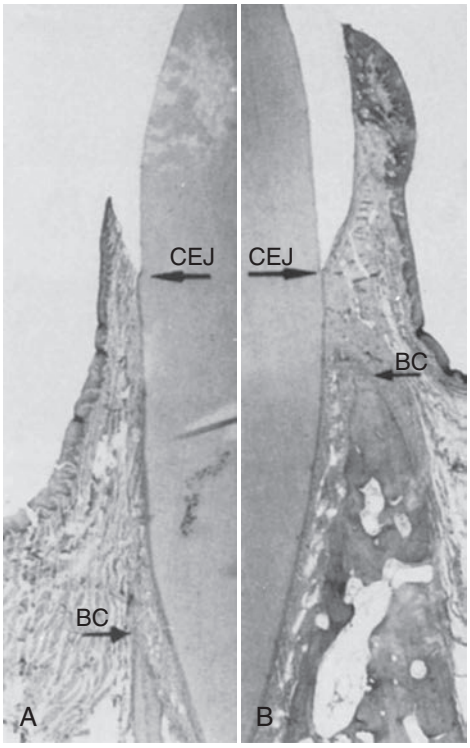


FIGURE 22-15 Histologic specimens demonstrating reduced alveolar bone height at an incisor that has been bodily moved in a labial direction (**A**) and normal alveolar bone height at an unmoved control tooth (**B**). Note that the connective tissue attachment has been maintained; however, the free gingival tissue on the labially displaced incisor (**A**) has been reduced in height. *BC,* Bone crest; *CEJ,* cement-enamel junction. (From Lindhe J. *Textbook of Clinical Periodontology*. Copenhagen: Munksgaard; 1989.)

based on the clinical appearance of the teeth without properly assessing the skeletal width of both the maxilla and the mandible. It has been established that no tooth in the mandible correlates with the skeletal width, and only the maxillary first molar correlates with the skeletal width of the maxilla.[82] Furthermore, clinical and visual evaluation of the maxillary width are not diagnositic.[83] Compensatory buccal or lingual inclination of the maxillary or mandibular teeth (or both) usually masks the underlying transverse skeletal discrepancy. Therefore, the presence of crowding, crossbite, or constricted arches does not necessarily indicate a transverse skeletal problem or the severity of the discrepancy.

Established skeletal landmarks should be used to diagnose the skeletal width of the maxilla and mandible. In the past, we have emphasized the used of the PA cephalogram to diagnose the skeletal width or determine the severity of the problem. Figure 22-18 illustrates the landmarks and norms developed by Rickkets in 1969.[84] The severity of the transverse discrepancy, age, and the appliance are the most important factors in determining appropriate treatment and prognosis. To achieve optimal skeletal correction, the Haas, bonded occlusal, or bone pin expanders that use palatal contour for anchorage and produce orthopedic response, minimizing adverse dental tipping, are recommended. As the patient matures, the skeletal expansion obtained can be less than one-third of the expansion produced at the level of the jackscrew. A post-expansion PA cephalogram or CBCT view is useful to evaluate the amount of skeletal correction achieved.

TISSUE RESPONSE TO CERTAIN TYPES OF TOOTH MOVEMENTS

Age-related changes occur in the skeleton and alveolar bone, and adults show a greater lag, or delayed response, to mechanical force, than that seen in younger patients.[85] However, teeth move equally well in adults and children, and no evidence indicates that they move more slowly in adults. In a healthy periodontium, regardless of the direction in which a tooth is moved, the bone around the tooth remodels without damage to the supporting tissues. The bone should follow the tooth in changes of position; this principle is used to create favorable alveolar changes in patients with periodontal defects. Significant evidence shows that drawing mesially inclined molars upright reduces pocket depth and improves altered bony morphology[60,86] (Fig. 22-19). The bone on the mesial erupts as the molar tips distally. Every dentist has seen teeth erupt, and as a tooth moves occlusally, the healthy alveolar process moves with it (Fig. 22-20, *A*). With chronic inflammation, as a tooth erupts, the alveolar bone is lost, and the tooth appears to extrude from the periodontal tissues (Fig. 22-20, *B*). When molars are uprighted, the connective tissue attachment on the mesial aspect of the molar to the crestal bone creates tension and allows for remodeling of the bone (see Fig. 22-19, *C* and *D*).

Clinically, ranges of force exist that are biologically acceptable to the periodontium. One should remember that because tooth movement is primarily a phenomenon induced by the PDL, identical forces do not put the same stress on the supporting tissues of different teeth. The root length and configuration, the quantity of bone support, the point of force application, and the center of rotation come into play to determine stress areas in the PDL. To prevent tissue damage, one must consider areas of maximal stress that might occur in the PDL. The risk of bone loss is higher if inflamed connective tissue is located apical to crestal alveolar bone,[87] and certain types of force may aggravate the progression of inflammatory periodontal disease.[88] Periodontal disease in a PDL under stress has been shown in rats (between M1 and M2), where infectious inflammatory infiltration spreads from the epithelial aspect into the transseptal ligament (Fig. 22-21, *A*). These animals showed complete destruction of the transseptal ligament within 28 days.[89]

To prevent aggravated loss of attachment or tissue damage, therefore, determining maximal stress areas, which can occur crestally or in the PDL, is crucial. Although most tooth movement involves combinations of movements, clinicians tend to view movement more simply in terms of extrusion (eruption), intrusion, tipping, translation, and torque.

Extrusion (Eruption)

Extrusion, or eruption, of a tooth or several teeth, along with reduction of the clinical crown height, has been reported to reduce infrabony defects and decrease pocket depth.[90,91] Single-tooth eruption should be distinguished from overbite correction and control of the vertical dimension during routine orthodontic correction. Extrusion of an individual tooth is used specifically for correction of isolated periodontal osseous lesions. Studies have shown that eruption in the presence of gingival inflammation

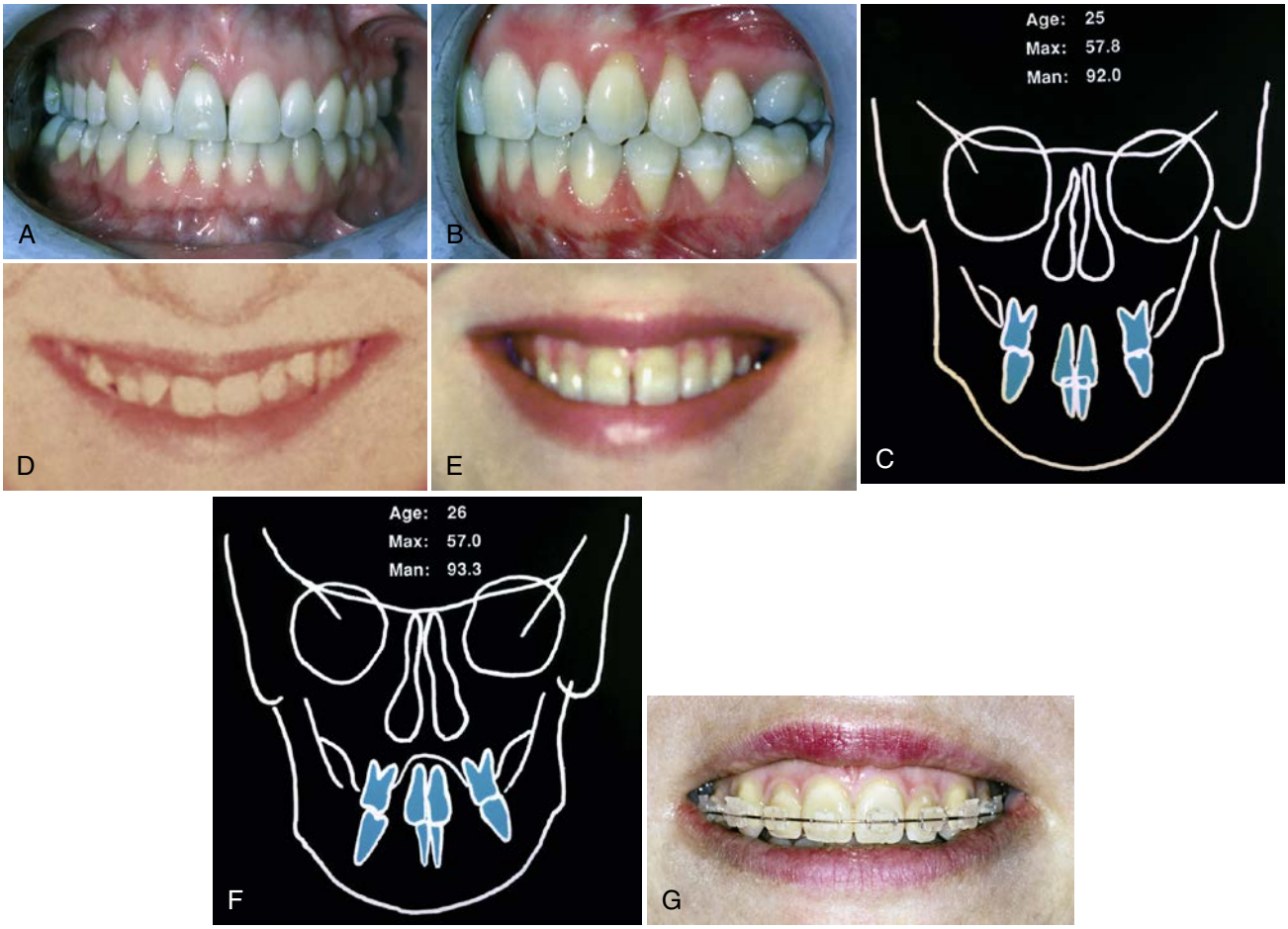


FIGURE 22-16 A case in which orthodontic treatment was done and the transverse skeletal discrepancy was camouflaged. **A,** Posttreatment right lateral view. The case is unstable and has significant gingival recession in buccal segments and poor dental aesthetics with negative space between the cheeks and the buccal segments. **B,** Gingival recession on the maxillary and mandibular premolars. **C,** The skeletal pattern is normal in the sagittal and vertical dimensions. On the posteroanterior (PA) cephalometric tracing, note the maxillary and mandibular widths. The Mx-to-Mx maxillary distance (57.8 mm) and the AG-to-AG mandibular distance (92.0) can be evaluated from the PA cephalometric radiograph. **D,** A 15-year-old patient with a severe Class III condition and normal transverse skeletal dimension; with a full smile, there is no negative space. **E,** Note the large negative spaces in the corners of the buccal segments of a 26-year-old woman with transverse maxillary deficiency and mandibular excess. **F,** Posteroanterior cephalometric tracing. Note the maxillary and mandibular widths. **G,** Negative space is eliminated by surgically assisted rapid palatal expansion performed to correct the transverse skeletal dysplasia. Excess gingiva was removed with the gingival retention procedure at appliance removal.

reduces bleeding on probing, decreases pocket depth, and even causes the formation of new bone at the alveolar crest as teeth erupt, with no occlusal factor present and while controls remain unchanged.[92] Eruption or uprighting of molars without scaling and root planing in human patients has been shown to reduce the number of pathogenic bacteria.[93] During clinical treatment, however, inflammation always should be controlled to ensure that the supracrestal connective tissue remains healthy and that the crestal alveolar bone height remains at its original level.

In the double-blind molar uprighting study done at the University of Pennsylvania,[93] bacterial samples were taken from the mesial pockets of molars to be uprighted (experimental tooth) and from the contralateral mesially inclined molar that served as the control in each subject. Indirect immunofluorescence was used to identify *Bacteroides forsythus, B. gingivalis, B. intermedius,* and *Actinobacillus actinomycetemcomitans*. During the study, no scaling, root planing, or subgingival inflammatory control was used. This study revealed that in all experimental sites that showed these microorganisms at the time of bonding, the number had diminished significantly by the end of treatment. However, Hawley bite planes were worn by all patients to disarticulate the molars during molar uprighting. In a similar study of 10 patients over a 12-week period, in which the effects of molar uprighting on the microflora of human adults were assessed using DNA probes, the number of *Bacteroides* pathogens was found to decrease at the experimental site with uprighting.[94]

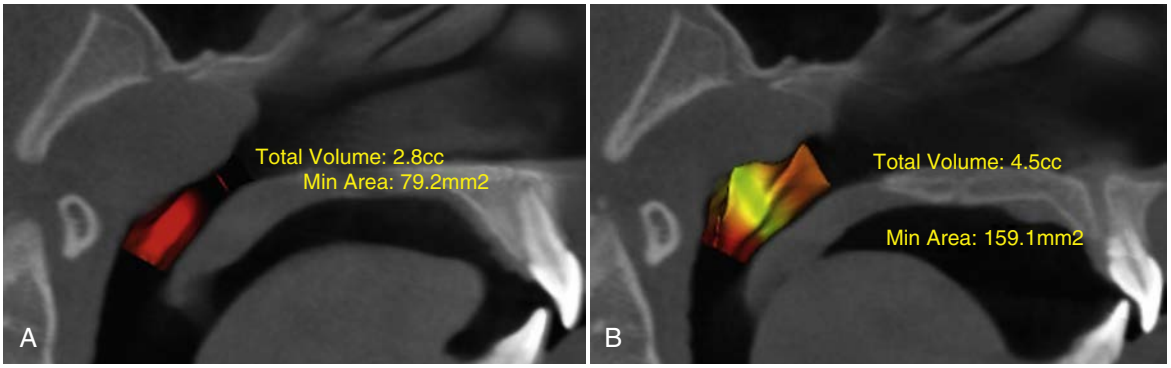


FIGURE 22-17 The skeletal transverse correction increases the posterior airway space. Furthermore, it could decrease the severity of the symptoms of sleep breathing disorders in patients. This patient's cone-beam computed tomography scan demonstrates the changes on the posterior airway space in a three-dimensional volumetric measurement. **A,** A 9-year-old patient before expansion with a bonded rapid palatal expander (RPE) showing a minimum area of constriction of 79.2 mm^2 and a volume of 2.8 cc of the posterior airway space. **B,** After RPE, note the changes on the minimum area of constriction (159.1 mm^2) as well as the volume (4.5 cc) of the airway posterior space.

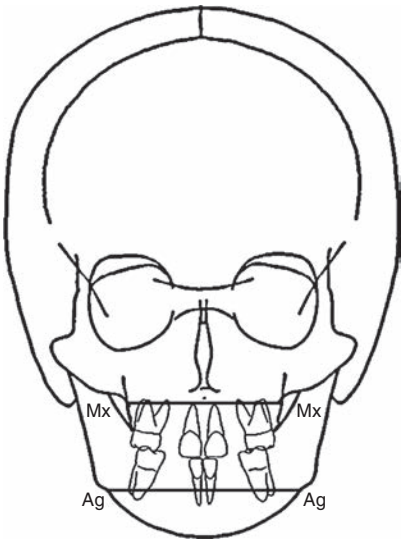


FIGURE 22-18 Posteroanterior cephalogram tracing with skeletal landmarks used to evaluate the transverse dimension, the width of maxilla and mandible. The maxillare (Mx), or J point, is located at the depth of concavity of the lateral maxillae contours, where the maxilla intersects the zygomatic buttress. *Mx–Mx,* distance between left and right Mx (mm) that represents the skeletal width of the maxilla. Antegonion (Ag), or antegonial notch of mandible is defined as the innermost height of the contour along the curved outline of the inferior mandibular border, low and medial to gonial angle. *Ag-Ag,* distance between left and right Ag (mm); represents skeletal width of the mandible.

Eruption of a tooth is the least hazardous type of movement to solve osseous morphologic defects on individual teeth created by periodontal disease or tooth fractures (Fig. 22-22). Forced eruption has been used to enhance soft and hard tissue dimensions.[95] Extrusion of anterior segments in skeletal open-bite patterns with muscular problems has shown shortening of the roots. If teeth have normal bone support or if bone loss has been horizontal (the alveolar crests and the cement-enamel junctions [CEJs] are level), then movement should involve intrusion or extrusion as necessary to correct the orthodontic deformity.

Intrusion

Conflicting evidence has been reported regarding the benefits of intrusion of individual teeth. One study has reported that intrusion of individual teeth did not result in the development of pockets.[96] This same investigator had reported earlier that intrusion in monkeys induced increased new attachment levels after flap operations to excise pocket epithelium and place an experimental notch on the root.[97] In periodontal research, dogs and monkeys are well known to show new attachment during normal healing after surgical flap procedures without tooth movement (Fig. 22-23), and this has been described.[98] Others have observed that intrusion may result in root resorption, pulpal disturbances, and incomplete root formation in younger individuals.[99] Clinicians have cautioned that intrusion of anterior teeth during leveling of the occlusal plane to correct overbite can deepen infrabony defects (Fig. 22-24) on individual teeth.[100] These conflicting reports indicate that intrusion can be a more hazardous type of movement; because the force is concentrated at the apex, root resorption has been a well-known sequela, and light forces have been recommended (see Chapters 3 and 4).

Intrusion has been reported to alter CEJ and angular crest relationships and to create only epithelial attachment roots; therefore, a periodontally susceptible patient is at greater risk of future periodontal breakdown (see Fig. 22-24, *B* and *C*). Tooth movement, when properly executed, improves periodontal conditions and is beneficial to periodontal health (Fig. 22-25). Whenever intrusion forces are applied, the mucogingival junction remains unchanged, but there can be a reduced amount of attached gingiva (Fig. 22-26). Extrusion is much more predictable than intrusion in accomplishing this purpose. Whenever the treatment objective is new connective tissue attachment or periodontal regeneration to restore lost supporting periodontal tissues, guided tissue regeneration (e.g., barrier membranes) is the predictable way to manipulate cells that lead to new attachment. Guided tissue regeneration procedures always should

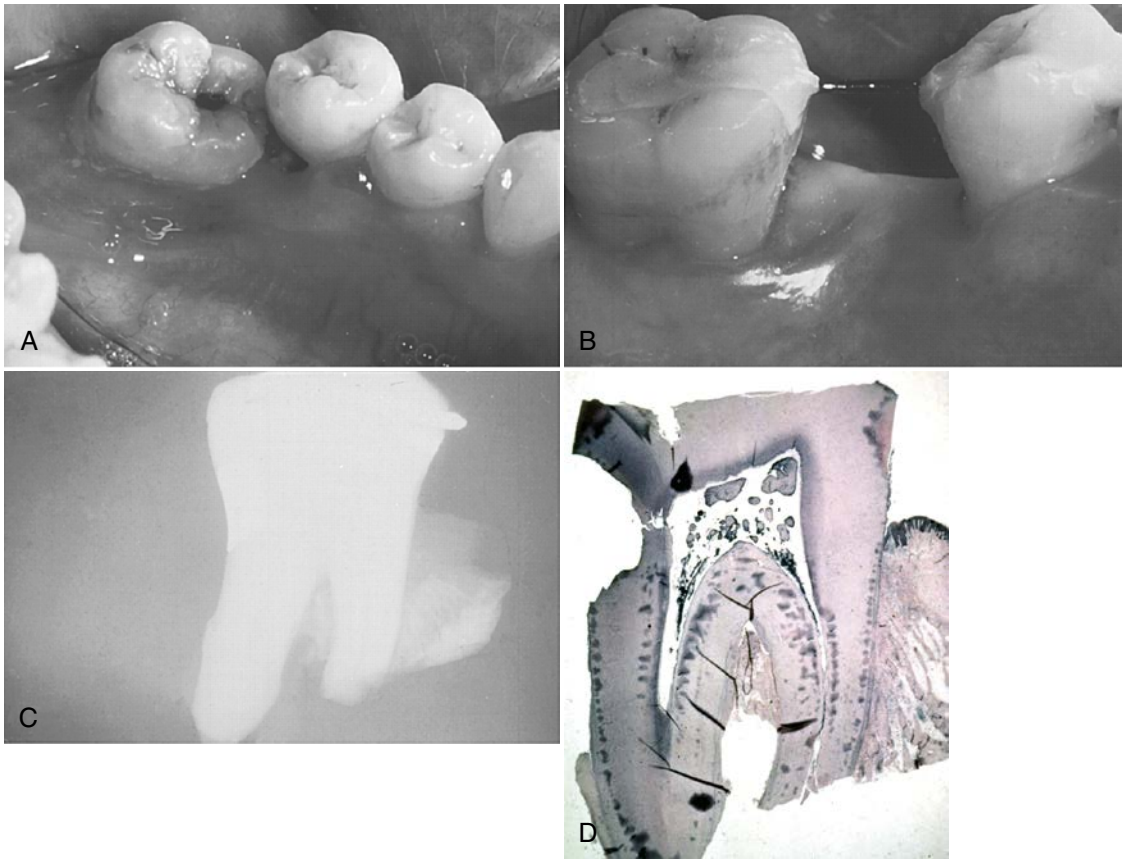


FIGURE 22-19 A, Mesially inclined molar before being uprighted. B, Reduction of mesial soft tissue pocket depth. The extrusive component of uprighting the molar allows for eversion of the pocket epithelium. C, Radiograph of a human clinical case after a tooth has been uprighted. D, Histologic slide of the tooth. Note the new bony spicules on the mesial where the alveolar crest has remodeled from the tension of the connective tissue attachment. No reattachment or increase in new attachment is evident. (**A, B,** From Brown IS. Effect of orthodontic therapy on periodontal defects. *J Periodontol* 1973;47:742; **C, D,** courtesy of I.S. Brown, Philadelphia.)

precede orthodontic tooth movement and should be part of the initial therapy before active orthodontic treatment is begun.

Tipping

When a single force is applied to the crown of the tooth, the tooth can rotate around its center of resistance (for an incisor, about the midpoint of the root), and compression (pressure) is increased at the crest and at the root apex. In this case, half of the PDL has the potential to receive high pressure from essentially light force.

Experiments in beagles demonstrated that with tipping and intruding movements, forces were capable of causing conversion of a gingival lesion to a lesion associated with attachment loss.[101] In tipping movements, the force should be light and the area should be kept clean to prevent the formation of angular bony defects.

Bodily Movement into a Defect

Movement into infrabony defects has been suggested to result in healing and regeneration of the attachment apparatus.[102] In addition, periodontists have believed that if a wide osseous defect is adjacent to a tooth and the tooth were moved to narrow the defect, better healing potential may be possible. Unfortunately, it has been shown that if infrabony defects are created in the lower incisor area of rhesus monkeys (undergoing good plaque control) and the tooth then is moved into and through the original defect, a long epithelial attachment to the roots is created, with no new attachment apparent.[103] The results, however, indicated that even though movement into infrabony periodontal defects did not result in regeneration of attachment, no further loss of connective tissue attachment occurred.

The most recent study performed in four beagles found that angular bony defects were created, and plaque was allowed to accumulate. Teeth were translated into inflamed, infrabony pockets, and additional attachment loss occurred on the teeth moved into the infrabony pockets.[104] Therefore, the conclusion was that bodily tooth movement may increase the rate of destruction of the connective tissue attachment of teeth with inflamed, infrabony pockets.

At the University of Pennsylvania, teeth have been moved into defects, with the patients' contralateral side serving as the control (Fig. 22-27). Radiographs revealed that loss of attachment occurred when a tooth was moved into a defect in an edentulous area (see Fig. 22-27, *D* and *E*). The tooth can move away from a defect and, with sufficient eruption, a bony defect can be reduced or eliminated; this usually is the treatment of choice to improve osseous architecture.

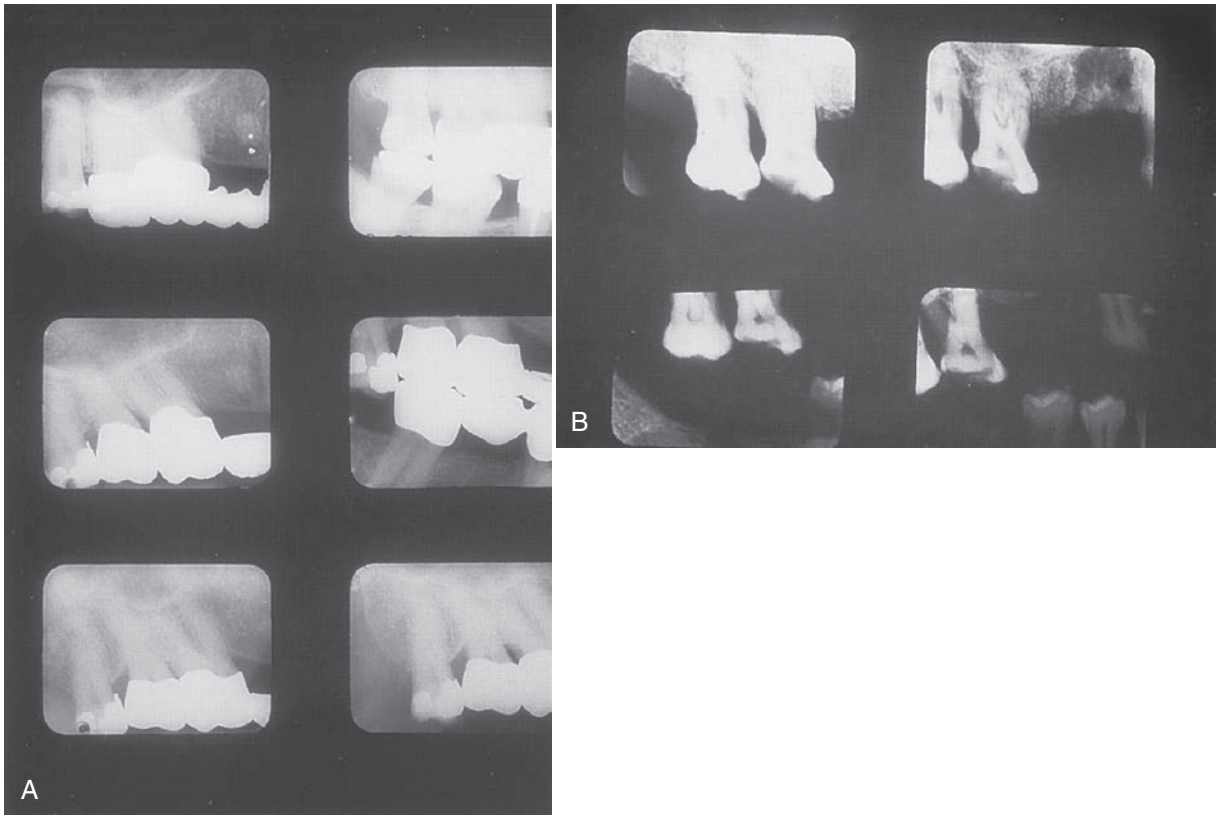


FIGURE 22-20 Periapical films taken over an 18-year span. **A,** The maxillary third molar erupts naturally. The area was kept clean, and the bone followed the tooth. **B,** Radiograph of unopposed tooth erupting in the presence of periodontal disease. The bone does not follow the tooth. (Courtesy of Morton Amsterdam, Philadelphia.)

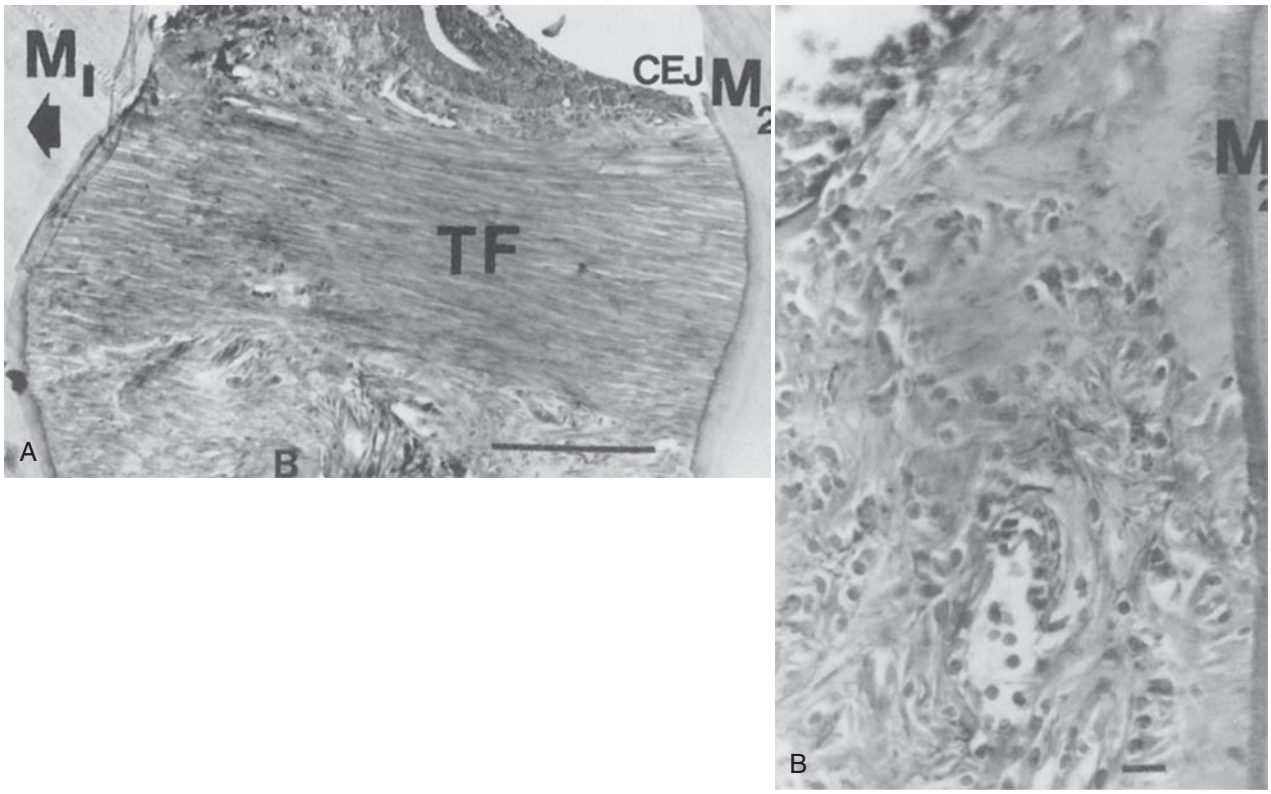


FIGURE 22-21 **A,** Area of tension. The first molar, M_1, moved in the direction indicated by the arrow for 28 days. Stretched transseptal fibers are detached from the alveolar bone crest. **B,** Area of tension mesial to the second molar, M_2. Periodontal disease with inflammatory infiltration and breakdown of transseptal fibers resulting from impaction of food caused by loss of approximal contact. *CEJ,* Cement-enamel junction; *TF,* transseptal fibers. (From Norton LA, Burstone CJ, eds. *The Biology of Tooth Movement*. Boca Raton, FL: CRC Press; 1989.)

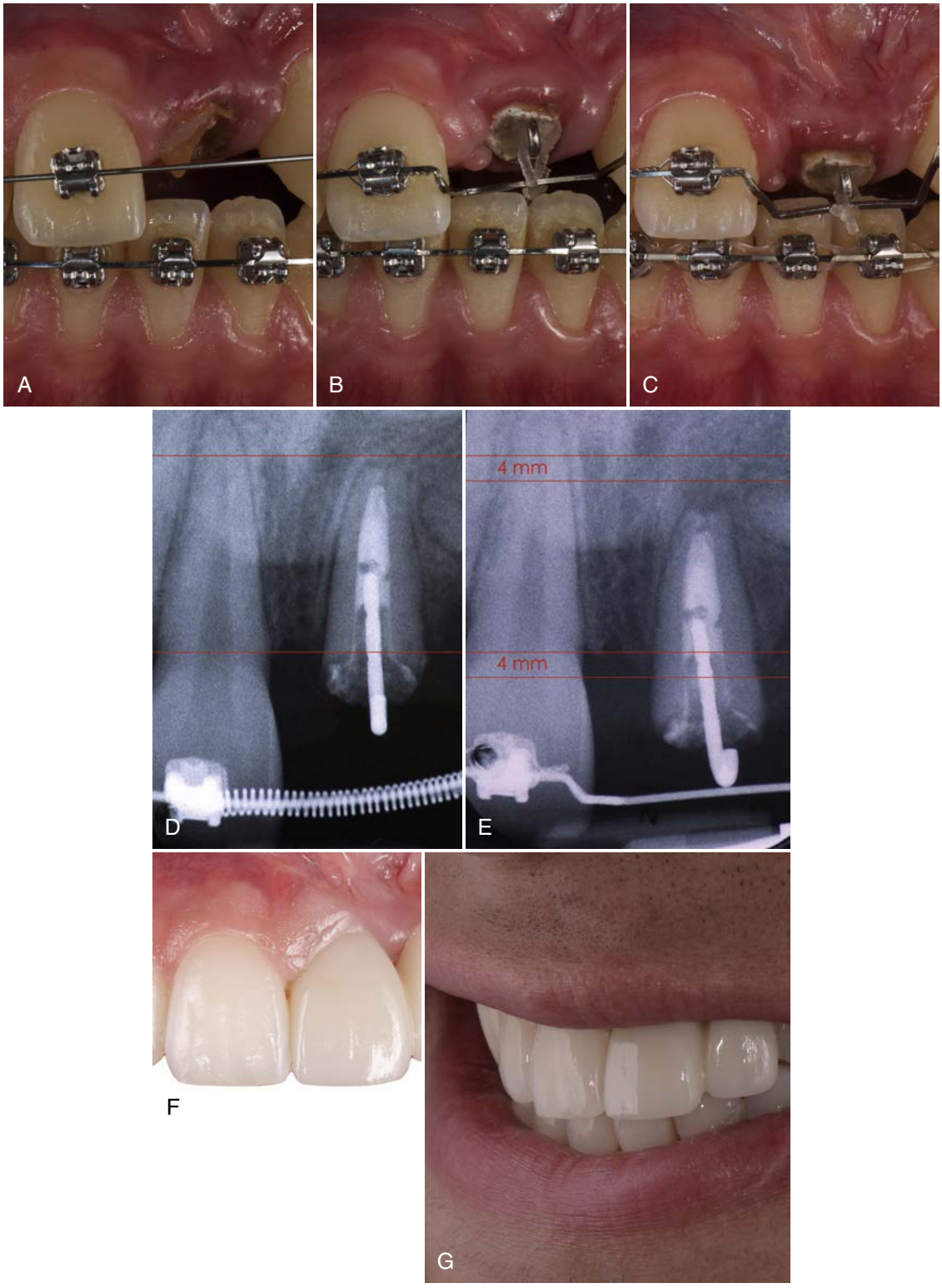


FIGURE 22-22 A 12-year-old patient with dental trauma. **A,** Fractured central incisors with an intact root. **B, C,** Forced eruption of the endodontically treated root with a custom-made post temporarily cemented. A stiff stainless steel wire was use as a base to avoid distortion of the maxillary arch while the force was applied. **D, E,** Radiographically, 4 mm of extrusion were obtained. Note the crestal bone that followed the movement. **F, G,** Long-term temporary restoration for a periodontal susceptible and growing patient.

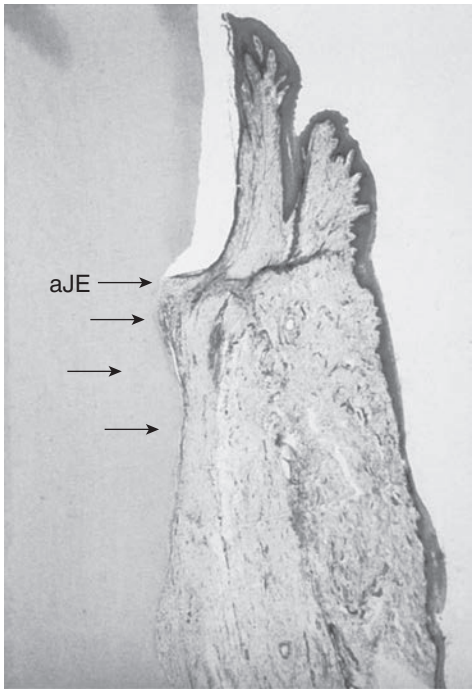


FIGURE 22-23 Normal healing after flap surgery in a dog (without tooth movement). A new connective tissue attachment has formed and extends coronally from the apical border of the notch prepared at the surgically created bone crest to the apical termination of the apical junctional epithelium (aJE). (Courtesy of J Lindhe, Göteborg, Sweden.)

A study involving closure of edentulous spaces in the mandible was done on a group of patients ranging in age from 11 to 17 years, and the results were compared with those from a group of patients aged 23 to 46 years.[105] The results indicated that the older adults had more loss of crestal bone and greater root resorption than the younger patients.

MUCOGINGIVAL CONSIDERATIONS

During tooth movement, the periodontal tissues should maintain a stable relationship around the cervical area of the tooth. An adequate amount of attached gingiva is necessary for gingival health and to allow appliances (functional or orthopedic) to deliver orthodontic treatment without causing bone loss and gingival recession. Clinical experience and animal studies clearly have established that more pronounced, clinically recognizable inflammation occurs in regions with a lack of attached gingiva (Fig. 22-28, *A*) than in areas with a wider zone of attached gingiva (Fig. 22-28, *B*). Histologically, the teeth lacking gingiva were thinner in the buccolingual dimension than were those with a wide zone of attached gingiva; however, investigators reported that inflammatory cell infiltrate and its apical extrusion (degree of inflammation) were similar.[106,107] Two studies have indicated that as teeth are moved labially and tension is created on the marginal tissue, the thickness of the gingival tissue on the pressure side becomes important.[108] With labial bodily movement, incisors showed apical displacement of the gingival margin, but no loss of connective tissue attachment was apparent where there were no signs of inflammation. Where inflammation was present, loss of connective tissue attachment occurred.[60] Therefore, if the tooth movement is expected to result in a reduction of soft tissue thickness and an alveolar bone dehiscence may have occurred in the presence of inflammation, gingival recession is a risk. Obviously, all orthodontic cases involve gingival inflammation, and bodily facial movement can predispose the patient to gingival recession; a gingival graft can be used to prevent this.

Soft tissue graft used for gingival augmentation is performed before recession occurs. It is considered to be less traumatic and highly predictable, and it prevents recession during orthodontics. However, many periodontists still believe that with younger patients, a wait-and-see attitude is acceptable because so many predictable ways of correcting recession have become available. Root exposure is most likely to be progressive in a younger patient because labial bone loss may be impossible to correct; it is essential to prevent labial bone loss. For this reason, clinical judgment and animal studies support the need to create a thicker gingiva that can withstand the inflammatory insult better during tooth movement.

Thin, delicate tissue is far more prone to exhibit recession during orthodontics than is normal or thick tissue. If a minimal zone of attached gingiva or thin tissue exists (Fig. 22-29), particularly on abutment teeth, a soft tissue graft that enhances the type of tissue around the tooth helps control inflammation; this should be done before orthodontic movement is begun. The orthodontist also should evaluate differences in alveolar housing. That a thin, soft tissue is associated with a thin labiolingual osseous support is not necessarily true. All combinations are seen, such as thick, soft tissue with a thin labial plate of bone. To change the labial osseous thickness (especially the thin type) that is characteristic of the individual patient is difficult; however, to improve the soft tissue with a soft tissue graft is not difficult or traumatic. Obviously, the decision concerning prophylactic periodontal procedures must be made with consideration, among other things, for growth and development, tooth position, type and direction of anticipated tooth movement, oral physiotherapy, integrity of the mucogingival junction, tissue type, inflammation, muscle pull, frenum attachment, mucogingival and osseous defects, anticipated tissue changes, and profile considerations (Box 22-1).

Critical evaluation of the periodontium before orthodontic treatment begins can prevent, minimize, or at least avoid aggravation of an existing periodontal condition. Other areas that frequently involve conflicting periodontal management are frenum considerations, gingival hyperplasia, mouth breathing, and ectopically positioned teeth.

Mandibular Midline Frenum

When a frenum is associated with a mucogingival problem, it most often relates to an inadequate zone of attached gingiva. For this reason, the use of frenectomy to correct mucogingival problems is considered obsolete. The high frenum insertion contributes to movement of the marginal gingiva where the keratinized tissue has been lost or detached or where mechanical trauma exists. This problem is most prevalent in the lower anterior area.

Maxillary Midline Frenum

In the past, frenectomy has been recommended in the maxillary midline for young children on the grounds that the midline diastema is caused by the maxillary labial frenum. Many believe

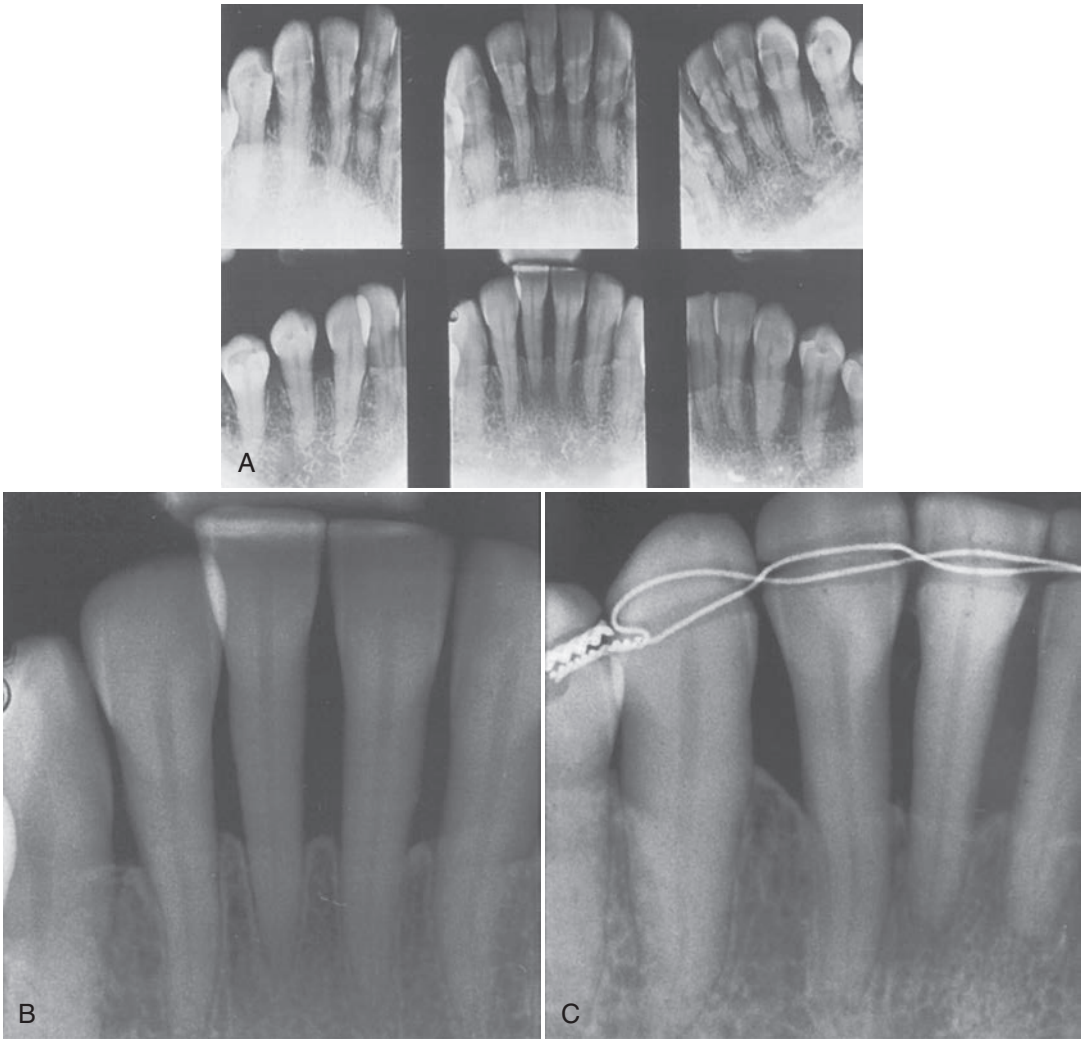


FIGURE 22-24 **A,** Radiographs of lower anterior teeth showing loss of bone before scaling. Lower radiographs were taken after scaling. Note that bone loss is horizontal (level alveolar crests) between the canine and incisor groups. The ideal way to reduce the overbite would have been to reduce the crowns with a high-speed bur and improve the crown-to-root ratio, keeping the bone level between adjacent teeth. Instead, the teeth were banded according to the incisal edge and were intruded. **B,** Preorthodontic periapical radiograph of lower anterior teeth before intrusion. Note the level alveolar crest between the canine and the lateral incisors. **C,** Periapical radiograph of lower anterior area after intrusion. Note that the cement-enamel junction is no longer level between the canine and the lateral incisors; however, a large angular crest has been created between those two incisors. An angular crest has been described as a predilected site for periodontal breakdown. (From Marks MH, Corn H, eds. *Atlas of Adult Orthodontics*. Philadelphia: Lea & Febiger; 1989.)

that this frenum prevents mesial migration of the maxillary central incisors and that it should be removed before orthodontic therapy is begun. Others have suggested that removal of the frenum allows the space to be closed more easily orthodontically. However, the practitioner must remember that a physiologic space normally is present between the maxillary central incisors until eruption of the canines in the adolescent dentition. In addition, a frenectomy may cause scar tissue that could prevent orthodontic space closure. With large diastemas (6–8 mm) in the early transitional dentition, a frenectomy usually is recommended to facilitate space closure, regain space at the midline, and prevent ectopic eruption of the lateral incisors or canines. These interceptive early treatment problems require complete orthodontic supervision, usually additional mechanotherapy, and several stages of treatment.

A U- or V-shaped radiographic appearance of the interproximal bone between the maxillary central incisors is a diagnostic key to the persistent midline diastema (Fig. 22-30). This radiograph of the mature midline suture with firm teeth before orthodontic treatment indicates that relapse will follow even excellent orthodontic treatment (i.e., no occlusal discrepancies, muscular habits, or problems; no tooth-size discrepancies; and ideal axial inclinations, overbite, and overjet). The patient should be informed before orthodontic treatment of the need for indefinite retention with bonding of the central incisors after treatment to prevent return of the maxillary midline diastema.

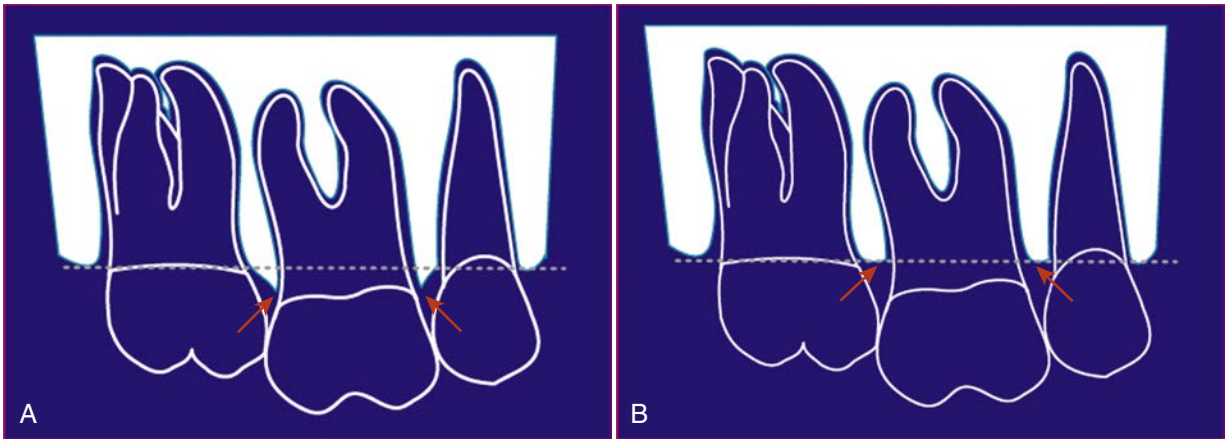


FIGURE 22-25 It is important to carefully evaluate the periodontal condition of the tooth to be intruded. **A,** Diagram of a healthy periodontium. The bone followed the tooth during extrusion. Intrusion mechanics are indicated, as the crestal bone will level after orthodontic movement. **B,** This diagram shows a periodontally compromised situation in which the bone has not followed the tooth during extrusion. If intrusion forces are applied, an angular crest defect may be created. Therefore, intrusion is not indicated in this situation.

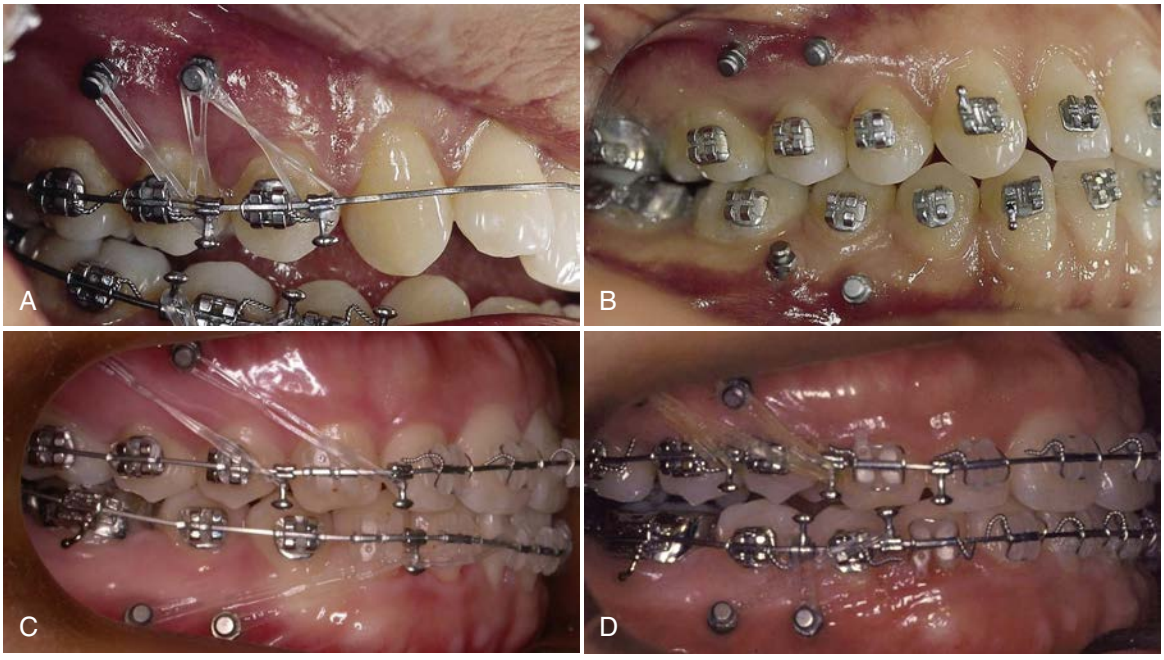


FIGURE 22-26 Intraoral images of two patients with healthy gingival tissues that underwent posterior intrusion. **A, C,** Before intrusion. **B, D,** After intrusion is completed. Note that the mucogingival junction remains unchanged, but there is a reduced amount of attached gingiva.

Generally, surgical removal of a maxillary labial frenum should be delayed until after orthodontic treatment unless the tissue prevents space closure or becomes painful and traumatized (Fig. 22-31). Removal may be indicated after treatment to change irreversible hyperplastic tissue to normal gingival form and to enhance posttreatment stability. This is particularly helpful on incisors during phase I of early treatment problems.

Gingival Hyperplasia

Mild gingival changes associated with orthodontic appliances seem to be transitory, and the periodontal tissues sustain little permanent damage.[109] Gingival hyperplasia usually resolves itself or responds to plaque removal, curettage, or both. If the gingival tissue or enlargement interferes with tooth movement, however, it must be removed surgically. Otherwise, waiting until the appliances are removed to correct surgically abnormal gingival form[110] and using the procedure to enhance posttreatment stability are preferable.

Gingival Retention and Aesthetic Considerations

Often tissue shows an exaggerated response to local factors, and gingival reflection with internal bevel incision (gingivectomy) can be done to promote stability and achieve optimal aesthetics and gingival topography (see Chapter 21). In

FIGURE 22-27 **A,** A 37-year-old man with bilaterally missing mandibular first molars. Lower right preoperative (test side) view of side with defect in the edentulous area. **B,** Lower right edentulous ridge area with tissue reflected. Note the defect mesial to the second molar. **C,** Appliance placement on the right side to begin space closure. **D,** Preorthodontic periapical radiograph. Note the mesial attachment level radiographically. **E,** Postorthodontic periapical radiograph. Note the evidence of bone loss on the mesial of the second molar during space closure. **F,** Lower left preorthodontic view of the side without a vertical defect in the ridge. **G,** Tissue reflected in the lower left (control side). Note that no defect is seen in the osseous ridge mesial to the second molar. **H,** Appliance placement on the left side to begin space closure. **I,** Periapical radiograph of the lower left side before space closure. Note the mesial attachment on the second molar. **J,** Periapical radiograph after space closure. No radiographic evidence of bone loss appears on the mesial of the second molar on the control side.

adults with altered passive eruption (APE), the gingival tissue fails to recede, and the patient thinks that the teeth are short (see Figures 22-3, *D*, 22-30, and 22-32). Many such patients have thick buccal alveolar bone that must be thinned by minor osteoplasty; if necessary, the clinician should establish a normal 1.5-mm relationship between the osseous crest and the CEJ. This prevents the return of the tissue incisally on the anatomic crown during healing. The operator should not do internal bevel gingivectomy and gingival reflection procedures on the labial aspect of anterior teeth that have thin gingival tissue. Also, the clinician should reflect tissue on bell-shaped anterior teeth only on the lingual or palatal aspect. The interdental tissue in these two instances may not heal back to the level of the contact points, creating an unaesthetic result. Spaces below the contact points are disturbing to the patient.

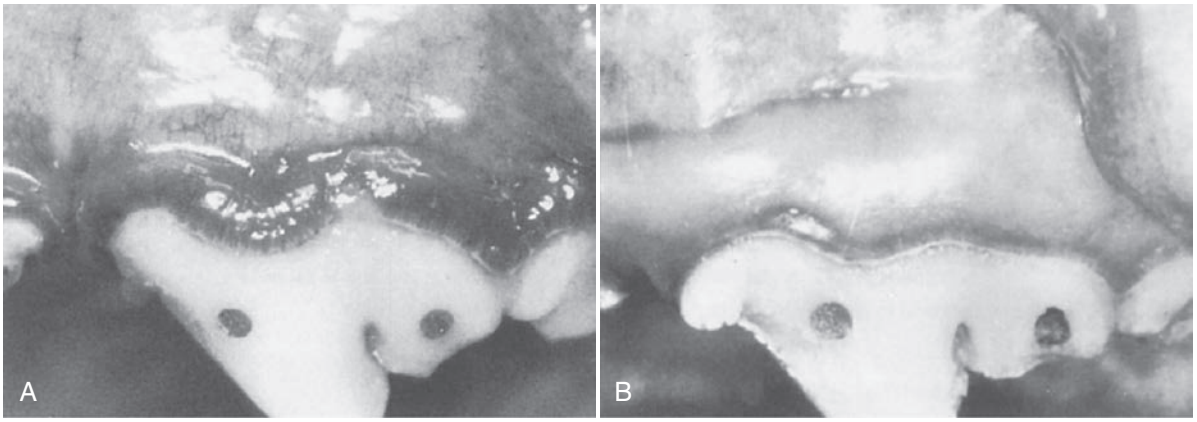


FIGURE 22-28 Teeth in a dog after 40 days of plaque accumulation. The clinical signs of inflammation are more pronounced at the site with the narrow band of gingiva (**A**) than at the site with a wide zone of attached gingiva (**B**). The vascular system is believed to have been more readily visible in its thinner units that lack gingiva. (From Lindhe J. *Textbook of Clinical Periodontology*. Copenhagen: Munksgaard; 1989.)

FIGURE 22-29 **A,** Inadequate attached gingiva on the lower central incisors of an 8-year-old girl. The signs of inflammation are pronounced, especially on the left central side; a free graft should be placed before early treatment protocols are begun. **B,** Donor tissue from the palate in place. **C,** Postoperative result before treatment was begun.

BOX 22-1 Diagnostic Considerations in Case Selection for Grafting before Orthodontic Treatment

- Growth and development
- Tooth position
- Oral physiotherapy
- Types of hard and soft tissues
- Inflammation
- Integrity of the mucogingival junction
- Mucogingival and osseous defects
- Type and direction of anticipated tooth movement
- Tissue changes associated with tooth movement
- Profile considerations
- Mechanotherapy to be used
- Patient cooperation

Fiberotomy

Fiberotomy[111,112] should be done after any preorthodontically rotated teeth have been corrected, especially maxillary and mandibular anterior teeth (e.g., maxillary lateral incisors with Class II, division 2 problems). The procedure should be done before debonding after mild overcorrection (3–5 degrees). The overcorrection is removed 1 week after the surgical procedure and before impressions for retainers are taken (Fig. 22-33). Internal bevel gingivectomy or labiolingual flap reflections with interproximal sutures (see Chapter 21) enhance alignment and reduce labiolingual and vertical dental relapse. These procedures should be done before fixed appliances are removed.

Mouth Breathing

A significant problem in orthodontic patients is the added periodontal insult of mouth breathing. The drying effect on the exposed tissue in susceptible patients is associated with enlarged, erythematous labial gingivae, particularly in the maxillary and mandibular anterior regions. With a short upper lip, a demarcation line usually can be seen where the lip contacts the labial tissue (Fig. 22-34). Mouth breathers usually have dry, cracked lips as well. Although orthodontic retraction of anterior segments may help provide a better lip seal, extraoral appliances and lip bumpers exacerbate the problem and even may cause mouth breathing in a normal patient. A patient who shows symptoms of an inability to breathe properly (tongue posture; enlarged adenoid tissue; narrow, high palatal vault; allergies) should be referred for evaluation for nasal obstruction and adenoid tissue. Although the plaque index is not significantly higher in mouth breathers, an increase in the gingival index has been reported.[113] This increased inflammation should be

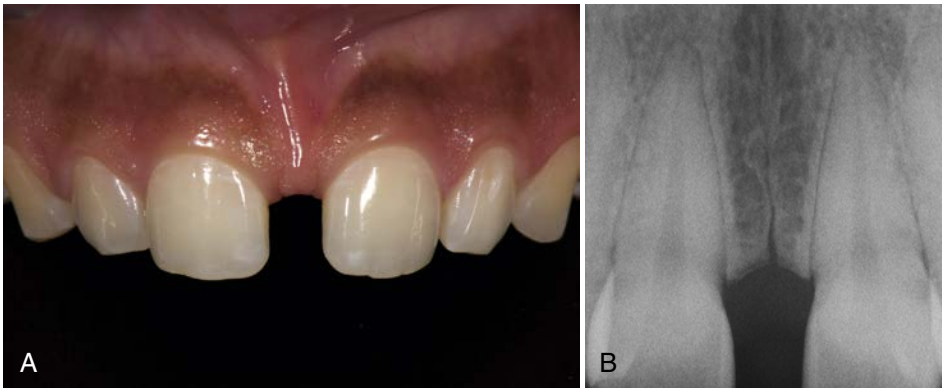


FIGURE 22-30 A, Clinical appearance of a persistent midline diastema in a 22-year-old woman. B, Radiographic appearance of midline interdental bone before orthodontic treatment, showing a V-shaped suture, which is the diagnostic key to potential relapse.

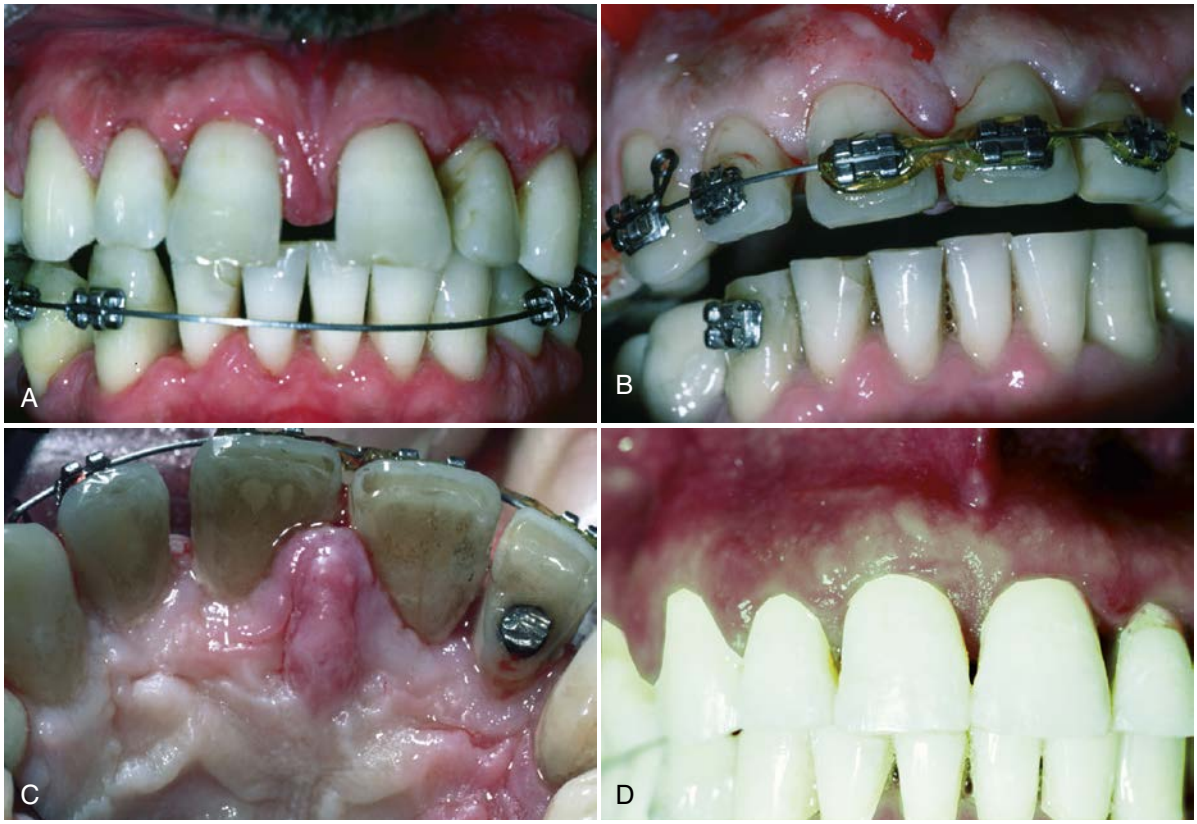


FIGURE 22-31 A, Preorthodontic diastema of a 40-year-old man with significant periodontal disease and loss of posterior support. B, C, During closure of the midline diastema, the soft tissue became enlarged and sore and prevented complete space closure. A frenectomy was performed to remove the frenum, which allowed completion of tooth movement. D, Midline area after completion of tooth movement and reestablishment of posterior support.

reduced to a minimum before fixed appliances are placed, and this usually is accomplished by scaling and curettage.

CONSIDERATIONS WITH ECTOPICALLY POSITIONED AND UNERUPTED TEETH

Many orthodontic patients have teeth that have not penetrated the oral mucosa or that will not erupt. Certain teeth demonstrate eruption that has been delayed significantly beyond the time when normal dental eruption for a particular patient should have occurred. Clinical orthodontists who have treated cases involving unerupted teeth have faced the problems of devitalization, reexposure or reuncovering of a tooth, ankylosis, external root resorption, and injury to adjacent teeth when an unerupted tooth has been surgically uncovered from the wrong side of the alveolar ridge. The marginal bone loss, gingival recession, and sensitivity problems that occur after the roots have been exposed are

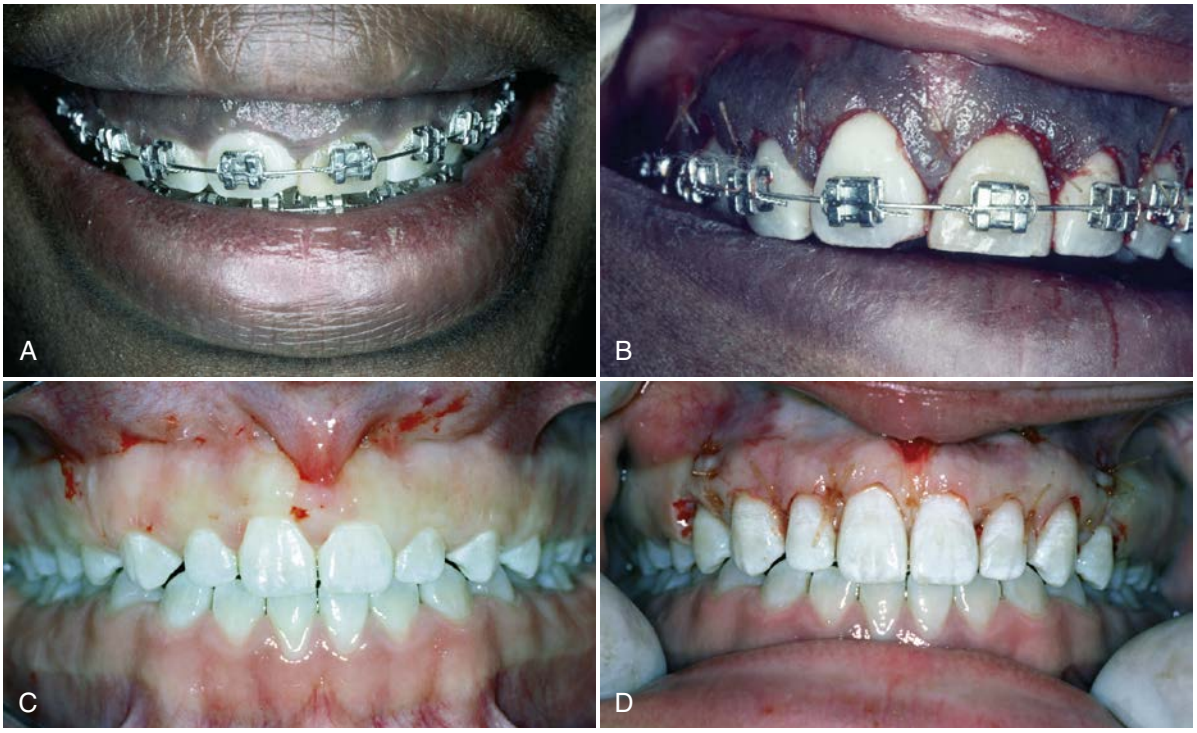


FIGURE 22-32 **A,** A 37-year-old woman who complained of short teeth. The woman has a normal vertical dimension and upper lip length. **B,** Note the normal-sized anatomic crowns at the time an internal bevel gingivectomy was performed for aesthetics and stability. **C,** A 14-year-old girl with short clinical crowns. **D,** After gingival tissue removal.

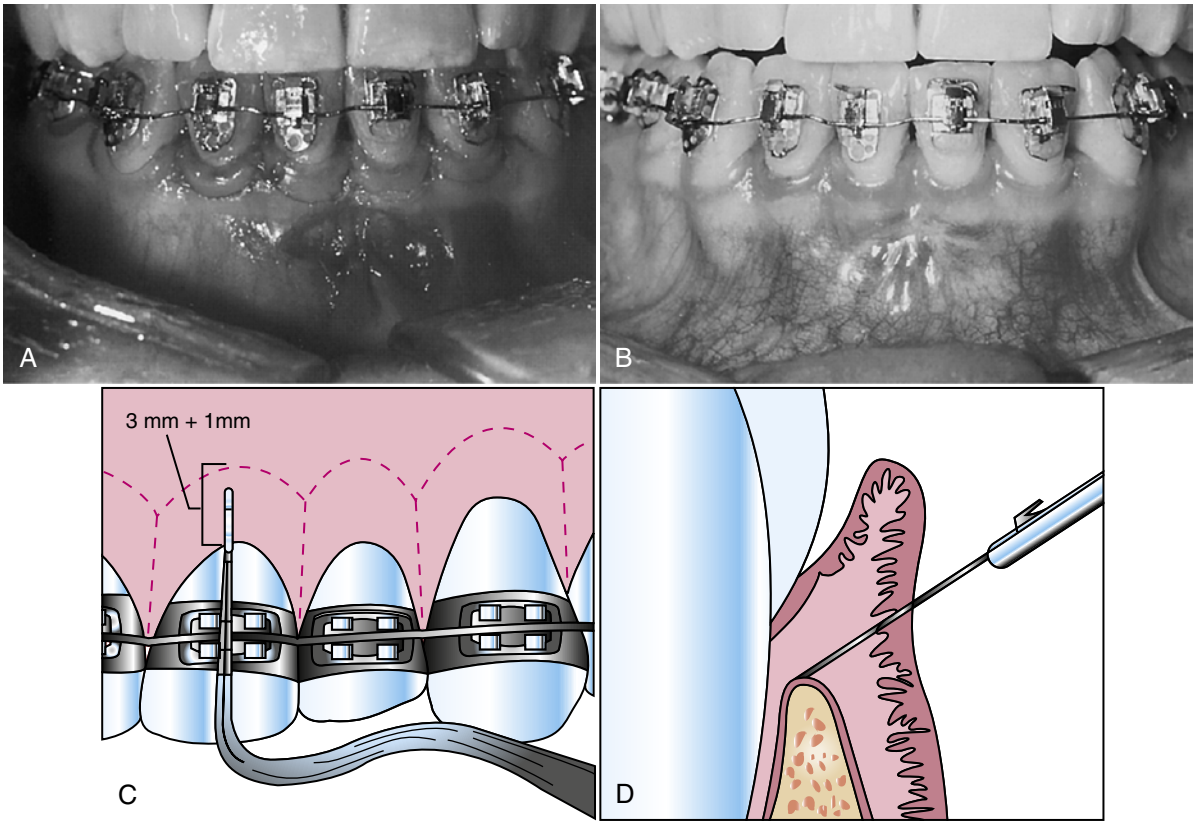


FIGURE 22-33 **A,** Mild overcorrections have been done for the mandibular incisors. Sounding-type incisions are made through the incisal papilla to bone and are angled toward the tooth. The interproximal incisions are joined labially in an attempt to sound out the osseous crest. Incisions are made on the lingual aspects as well. In this case, a mandibular frenum was released so that the tissue would not be retracted on the labial aspect of the central incisors. **B,** Two weeks after surgery; overcorrections will be removed. **C,** Representation of probing to establish bleeding points. **D,** Diagram of sounding incision made at a 45-degree angle to the long axis of the tooth. (From Ahrens DG, Shapira Y, Kuftinec MM. An approach to rotational relapse. *Am J Orthod* 1981;80:83. With permission from the American Association of Orthodontists.)

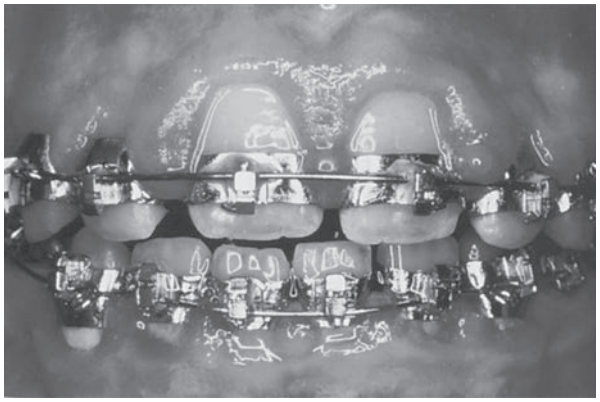


FIGURE 22-34 Exaggerated response to mouth breathing. Note the lip line over the central incisors, where the lip covers the labial gingiva. Enlarged gingival tissue harbors dentobacterial plaque, and the deeper pseudo-pocket acts as a reservoir for retention of subgingival plaque.

complications that invariably result in prolonged treatment time, aesthetic deformities, and in many cases, loss of teeth (Fig. 22-35, *A–E*). Most of these problems (i.e., reexposure, gingival recession, and bone loss) can be prevented. Proper management of the periodontal tissues is crucial in preventing loss of attachment. For example, one should not use electrosurgery or lasers to uncover teeth. These techniques have attracted considerable attention recently, but they should be used strictly to remove the overlying tissue; excision of the surrounding tissue on the unerupted tooth leaves inadequate keratinized tissue.

Vermette et al.[114] found no difference in periodontal attachment levels between control teeth and teeth uncovered by an apically repositioned flap or a closed surgical technique. However, they reported that teeth uncovered by apically repositioned grafts had more gingival scarring, were less aesthetic, and experienced intrusive relapse more often than did a group of teeth subject to closed eruption or flap replacement. These findings were not in agreement with the study by Kim et al.[115] on labially apically repositioned flaps, in which it was concluded that ectopically positioned maxillary canines treated by apically positioned flap techniques had satisfactory results for periodontal, radiographic, aesthetic, and endodontic outcomes compared with orthodontically moved control teeth. Periodontally, the control teeth had normal values, and there were no significant differences in plaque index, gingival index, pocket depth, width of attached gingiva, and clinical crown length between the treated teeth and control teeth. Aesthetically, there were no differences in recession, scarring, and texture of gingiva. Radiographically, control teeth had normal crestal and bone support values. Furthermore, reflecting a surgical flap, bonding an attachment with a gold chain, and suturing the flap over the crown always prolongs the treatment time, requires additional surgical procedures, diminishes control of tooth movement, and may cause adverse periodontal responses (Fig. 22-35, *F* and *G*). Localization and the exact position of the tooth must be known before the tooth is uncovered surgically. Palpation, evaluation of the teeth in the arch, CBCT, panoramic radiographs, periapical views, and occlusal or cephalometric evidence can be helpful.

Labial impactions should not be uncovered unless keratinized tissue is placed on the tooth. It should not have a free gingival graft placed on it; free gingival grafts do not survive on enamel.

Surgical Technique

The preferred surgical procedure is primarily an apically or laterally positioned pedicle graft[116] (Fig. 22-36). The maxillary incisors can be done nicely from an orthodontic standpoint. When uncovering a tooth, the orthodontist should create a space in the arch before uncovering the tooth if the tooth is on the labial aspect of the arch. The edentulous space created in the arch provides a tissue area to act as a donor site so that an adequate zone of attached gingiva can be taken for the partial thickness of the apically or laterally positioned graft. If space is unavailable between the teeth in the arch, the clinician may place a free graft at the mucogingival junction and convert it into a pedicle graft to be placed on the unerupted tooth. Whenever the tissue for a particular patient is so thin that it cannot be dissected away as a partial-thickness graft, one can place a free graft at the mucogingival junction. Grafts should be partial thickness; the orthodontist should express this preference to the periodontist or oral surgeon because full-thickness grafts are thick and unaesthetic.

Large dental follicles often occur, especially on maxillary canines in 9- or 10-year-old children, before the teeth have erupted from the palate. When these enlarged dental follicles are resorbing hard tooth structure in the arch (i.e., the roots of teeth), they must be uncovered (Fig. 22-37). Even though the follicles may not have more than 50% root formation at that point, the clinician should uncover the canine and place a bonded attachment. The orthodontist should move the canine into contact with the palatal mucosa, take off the attachment, and allow the root to continue to develop on the canine. If the canine is not uncovered and brought into contact with the oral cavity, the enlarged follicle usually continues to resorb the roots of the incisor teeth. If the palatal tissue is allowed to recover, it begins to resorb the tooth structure again.

The question often arises whether a tooth will erupt into the arch. The real key is the incisal edge; if the incisal edge is headed away from the occlusal plane or if the tooth is upside down, it will not erupt into the oral cavity. If a tooth is erupting slowly or if it is holding up treatment time, it should be surgically uncovered and orthodontically positioned into the arch. The most difficult canine is the canine that erupts between the roots of a premolar, particularly a maxillary first premolar with a bifurcated root (Fig. 22-38). The orthodontist should evaluate the periapical radiograph closely for this type of premolar root form. When the canine erupts and lodges between the roots of a bifurcated first premolar, it is difficult to uncover without getting into the apices of the premolar. This is true whether the canine is uncovered from the palatal or labial side of the arch. In this situation, the premolar would have to be extracted to salvage the maxillary canine (see Fig. 22-38, *E* and *F*). Labial impactions are much less difficult to manage if a soft tissue grafting procedure is used (Fig. 22-39).

Maxillary Palatal Impactions

With maxillary palatally positioned canines, which occur three times more often than labially malposed canines, the problem is different. Because the palate is all masticatory mucosa, a graft is not placed on the teeth. To uncover these teeth, the orthodontist reflects the palatal tissue, places a window, and replaces the tissue over the palate (Fig. 22-40). A periodontal dressing is

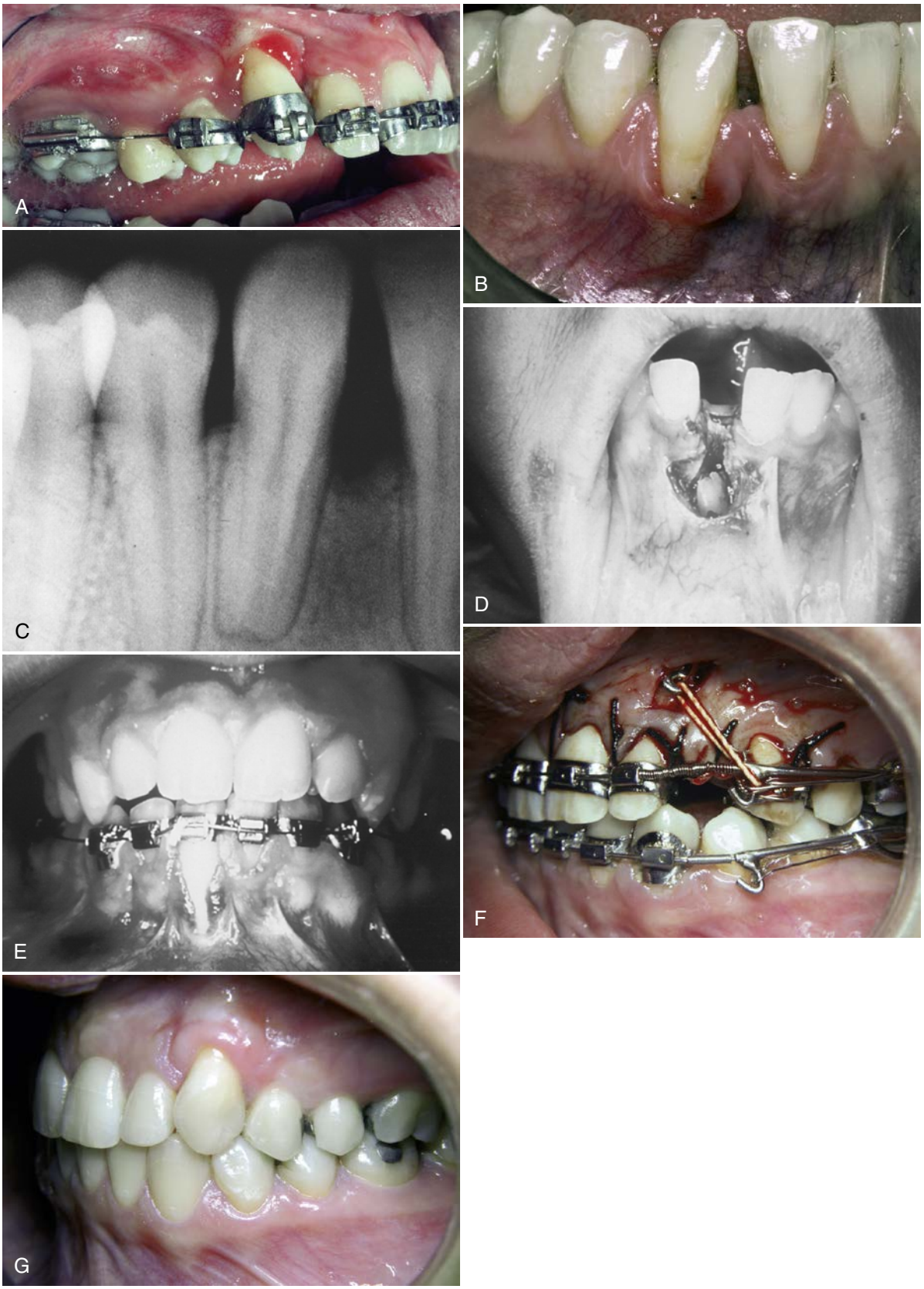


FIGURE 22-35 **A,** A typical aesthetic and recession problem on a maxillary canine that was surgically uncovered without keratinized tissue being placed on the tooth. **B,** Clinical appearance of a previously unerupted and surgically uncovered mandibular canine. Note the absence of attached gingiva and the gingival recession; the tooth is an aesthetic concern to the patient. **C,** A periapical radiograph reveals marginal bone loss of one-third of the alveolar support and root blunting. These complications could have been prevented. **D,** Surgical uncovering involved extraction of the primary tooth and removal of all overlying soft and hard tissue. The surgery did not provide for attached gingiva over the labial of the tooth. **E,** An undesirable response, with recession and loss of attachment as the tooth is guided into the arch. **F,** The maxillary canine was uncovered, an attachment was placed, and the flap was sutured back into place. An elastic was used, with the objective of guiding the tooth into the zone of keratinized tissue. **G,** When a tooth is covered over with soft tissue, it is difficult to guide into the zone of keratinized tissue. Note the root exposure and recession after the tooth has been positioned in the arch with a flap replacement procedure.

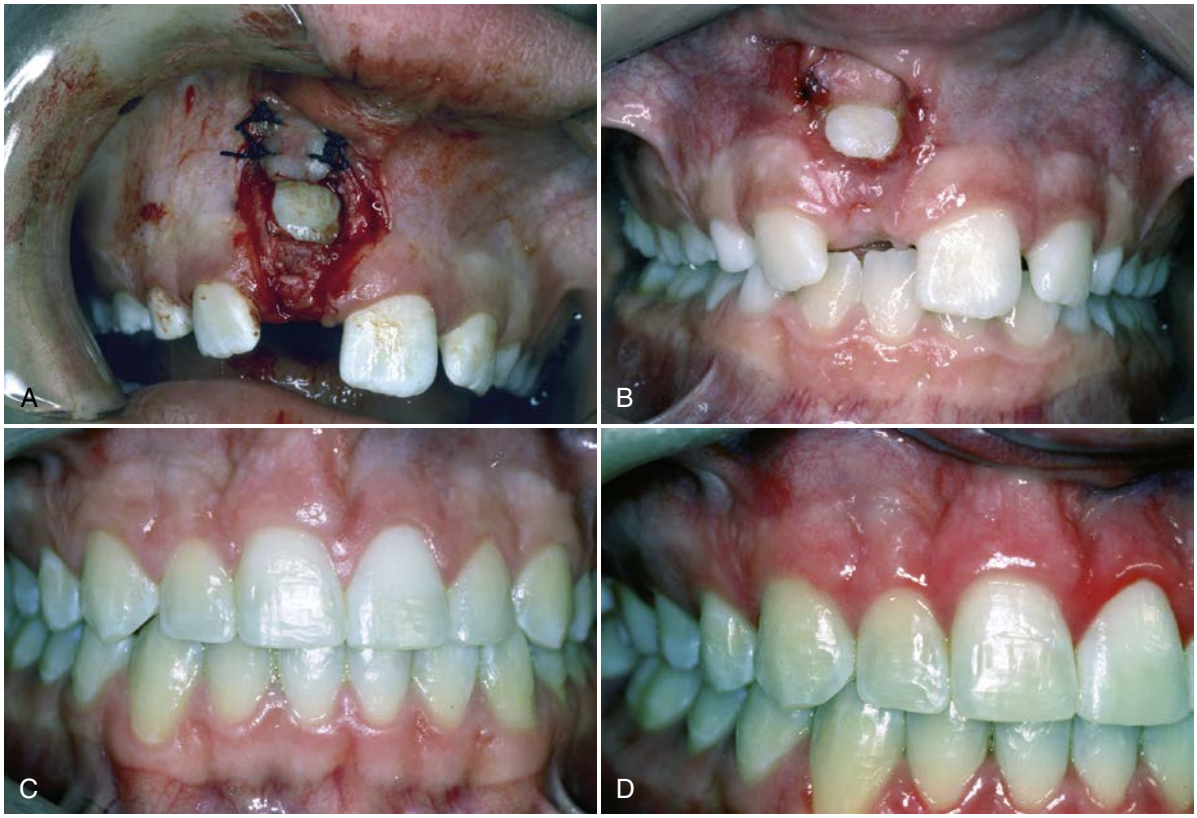


FIGURE 22-36 **A,** A right central incisor was uncovered surgically, and an apically positioned partial-thickness pedicle graft was placed on the cervical area of the anatomic crown. **B,** Several months later. Note that normal eruption has occurred without the use of an appliance. **C,** From the anterior view several years after orthodontic treatment, the gingiva has the same appearance on both central incisors. Detecting that the tooth was grafted is impossible, and normal periodontal support is present. **D,** Right lateral view of the incisor 10 years after surgery.

kept in place for 7 to 10 days, and then the orthodontist places a bonded attachment and begins tooth movement. It is important to postpone the bonded attachment, to allow the periodontal tissues (hemidesmosomal attachment) to heal around the CEJ and protect it from the acid etch during bonding, which might damage the PDL cells of the tooth side and induce ankylosis.

A major orthodontic principle that applies to palatal canines is the importance of using a sufficiently large base arch (at least an 0.018-inch or a rectangular stainless steel base arch). The base arch must be large enough to prevent deflection of teeth in the maxillary arch into the opposing occlusion each time the patient closes the mouth. Devitalization of lateral incisors often occurs when a flexible, multistranded base arch or a light nitinol base arch is used; when the palatally impacted tooth is activated over a long period, it deflects teeth (particularly maxillary lateral incisors) into the opposing occlusion. Lateral incisors also can be devitalized by occlusal trauma. In adults, the primary teeth are left in place until the palatally displaced tooth has been moved close to the arch; the primary tooth then is taken out. After removing the primary tooth, the orthodontist places an acrylic pontic on the archwire for aesthetics.

The orthodontist must evaluate palatally impacted teeth constantly for bleeding around the crown and active bleeding around the tooth; ideally, this is done with a curette. If excess bleeding is noted, the orthodontist should refer the patient back to the general dental office or to the periodontal office for periodic scaling and curettage to remove bacteria from the subgingival area. Bacterial buildup occurs because of the depth of the palatal tissue around unerupted teeth. Otherwise, a small chance exists of loss of attachment on a palatally impacted tooth. At each visit, the orthodontist should check any tooth that has been uncovered surgically for excessive mobility. In many instances, the dental follicle can grow aggressively around the erupting tooth that has been uncovered and can displace or detach the graft from the crown on labial impactions. The follicle also can cause bone loss around the entire tooth as the tooth is moved into the arch.

If excessive mobility is observed, the orthodontist must refer the patient to the periodontist to find out whether bone is following the erupting tooth. An x-ray examination should be done to determine whether osteoid tissue is present and to ensure that the periodontal attachment is following the tooth as it is guided into the arch. To aid eruption of a deeply positioned palatal impaction, a 0.012- or 0.014-inch stainless steel wire should be twisted until taut directly across the palate from the canine or premolar area to the premolar area on the opposite side of the arch. The authors do not recommend the use of auxiliary springs because these springs tend to overextrude the canine. The orthodontist uses an explorer to tighten the wire with a Spanish windlass technique and then places the attachment on the palatally positioned tooth and ties a power thread from a tooth to the wire that goes across the palate. The force

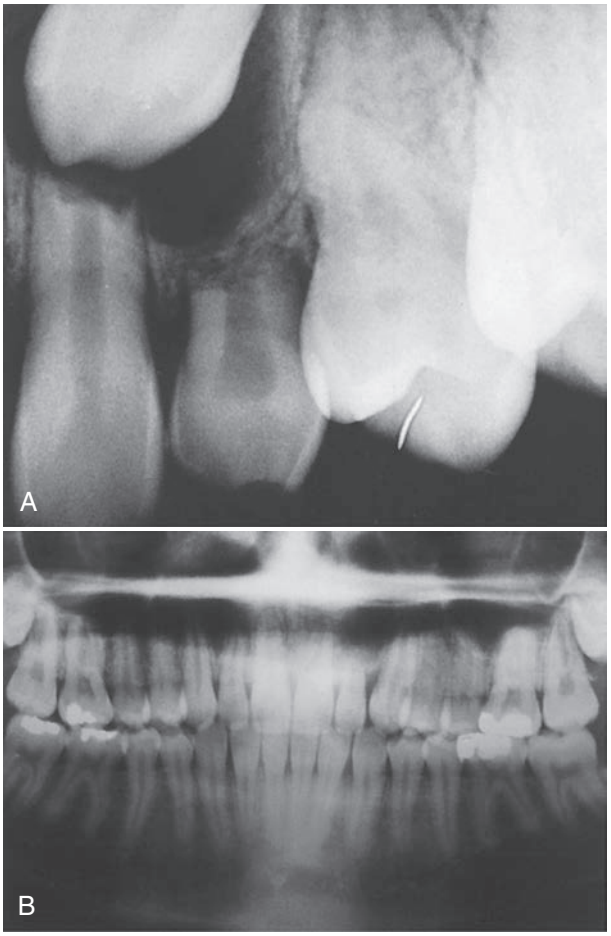


FIGURE 22-37 A, Periapical radiograph shows more than 50% resorption of the root of a lateral incisor. The crown of the canine had to be uncovered surgically and kept in contact with the oral cavity. B, Panoramic radiograph shows no further resorption of the lateral root in the finished case.

is directed so as to erupt the tooth into the palate out of the palatal soft tissues (Fig. 22-41). The orthodontist then rotates the tooth and moves it directly into the arch. If palatal tissue recovers the tooth and the tooth erupts through the palatal tissue into the arch, treatment time is prolonged by many months. Therefore, the tooth should be erupted within 7 to 10 days after the surgical procedure and should be erupted down out of the thick tissue and brought to the level of the palatal contour. At this point, the orthodontist can determine whether the tooth is backward and needs to be rotated; rotational movement should be initiated immediately (even in the center of the palate) as the tooth is moved toward its proper position in the arch.

Second Premolar and Second Molar Impactions

After the canines, the second premolar is the tooth that most often requires surgical uncovering. As with mandibular premolars, apically repositioned grafts are necessary. If the tooth is located on the lingual, the graft is placed on the lingual only. If the tooth is in the center of the arch (an occlusal radiograph is needed to locate these teeth before they are uncovered), an apically positioned graft on the facial and on the lingual is required (Fig. 22-42). Ideally, the orthodontist should give the surgeon as much assistance as possible in determining the location of teeth, as well as any special information about the surgical uncovering. With the mandibular molars, one also must do a buccal and a lingual graft when these teeth are located in the middle of the ridge. The osseous surgery must allow the graft to be placed onto the enamel on the buccal and lingual aspects of the anatomic crown.

Incisor Impactions

Less often, a mandibular incisor (or several mandibular incisors) may need to be uncovered. With mandibular incisors, a labial and a lingual graft must be done, and these grafts must be extended beyond the line angle of each crown (Fig. 22-43). If the grafts are not placed beyond the line angle of the unerupted tooth, a mucogingival defect may be created on the line angle, whether it is on the lingual or the facial aspect, even when teeth are in the center of the ridge.

As persons get older, the teeth tend to become ankylosed, particularly as an individual reaches the 30s and 40s; radiographs can reveal this complication. A radiograph of a tooth that appears to fade into the bone indicates ankylosis (Fig. 22-44). Teeth with this radiographic appearance should be considered hopeless, and no attempt should be made to bring them into the oral cavity. All unerupted teeth cannot be salvaged, and the prognosis may be poor. Certain impacted teeth, even the canines, should be extracted. However, the number of successfully treated unerupted teeth can be increased by proper management of the tissues attached to these teeth. Protecting the marginal integrity of the attachment apparatus and preventing an apical shift of the dentogingival junction result in fewer aesthetic deformities and a more favorable long-term prognosis; these goals can be accomplished most effectively by using a pedicle graft, as has been described.

Ankylosis and External Root Resorption

In addition to surgical procedures and orthodontic management of unerupted teeth, impacted teeth present other, more perplexing problems.[117] Most clinicians have been confronted with ankylosis of deciduous and permanent molars in the primary and transitional dentition. Some permanent teeth that have been exposed to the oral cavity are ankylosed. The most common cause of ankylosis generally is believed to be trauma to a tooth. External root resorption commonly is associated with tooth replantation. Many reports have discussed the possible causative factors associated with ankylosis.[118–121]

All too often a clinical orthodontist has ordered extraction of teeth with the expectation of bringing an unerupted tooth into the arch. The oral surgeon exposes the unerupted tooth, and later the orthodontist finds that the tooth will not move. After repeated attempts at luxation, with ankylosis returning, the surgeon removes the tooth. The statement "One in the mouth is worth two in the gums" may seem to apply.

A high incidence of tooth ankylosis and external root resorption reportedly has been associated with impacted teeth that have been uncovered and ligated with dead soft stainless steel at the CEJ area[122,123] (Fig. 22-45). This procedure,[124] first reported in 1942, was extensively used before bonding.[125,126] Use of the wire loop has been called the "simplest and most often used means of attachment . . . of an unerupted tooth."[127]

However, to tighten a wire placed in the cervical third of an impacted tooth and not impinge on the normal CEJ–alveolar bone relationship requires an extreme amount of surgical

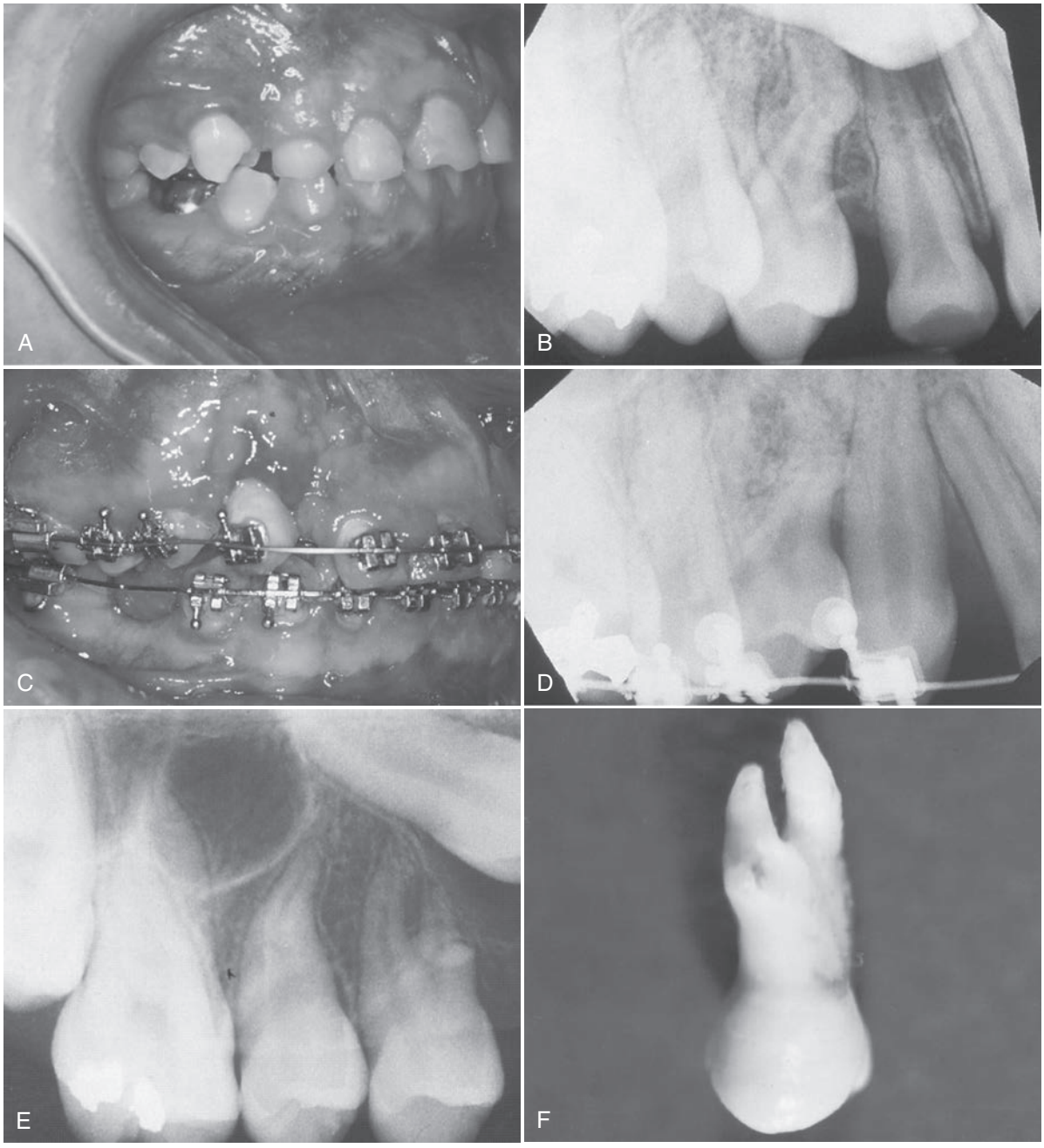


FIGURE 22-38 A, Right clinical view of a primary canine still in position before appliances were placed. B, On this preoperative periapical radiograph, note the bifurcated first premolar root with the maxillary right canine lodged between the roots. C, Several years later, the tooth has been guided anteriorly from the labial into the arch. This is an adolescent boy whose oral hygiene, despite constant reinforcement, remained inadequate; note the significant gingival hyperplasia. D, Progress periapical film indicates possible loss of attachment, although the probing depth is minimal. E, A young girl with an unerupted maxillary right canine. A pretreatment periapical radiograph shows a bifurcated first premolar. This root form can complicate and prolong treatment. F, The premolar was extracted.

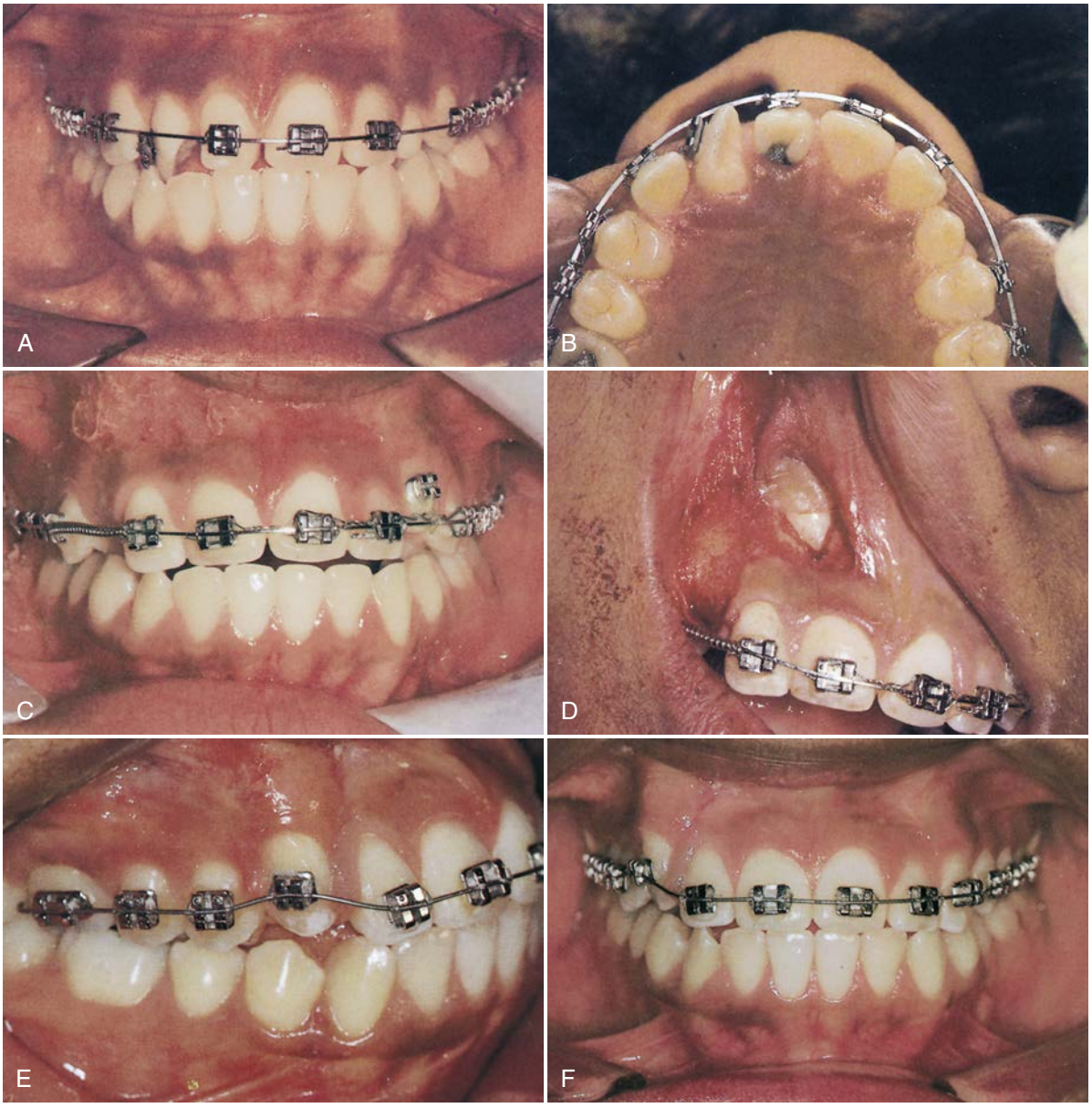


FIGURE 22-39 **A,** A young woman with a supernumerary incisor between the two central incisors. **B,** Palatal view of the supernumerary incisor to the right of the midline. **C,** The supernumerary tooth was removed, and the right central and lateral incisors were positioned properly. **D,** The maxillary right canine was uncovered surgically, and a pedicle graft was placed on the labial. **E,** Note the tissue health as the tooth is moved into position. **F,** Anterior view of the maxillary right canine.

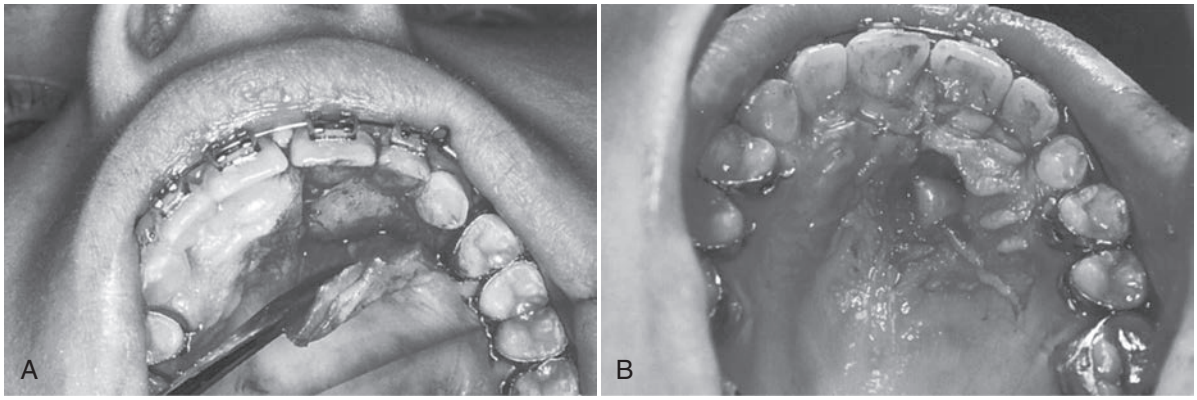


FIGURE 22-40 **A,** An ectopically positioned palatal canine rarely can be palpated. The palatal soft tissue must be reflected, and osseous surgery is required to enter the follicular space. Inadequate exposure not only prolongs treatment but also may result in injury to the tooth because of the inadequate field of vision. **B,** The palatal tissue is put back in position, and a soft tissue window is created in the orientation of the long axis of the anatomic crown.

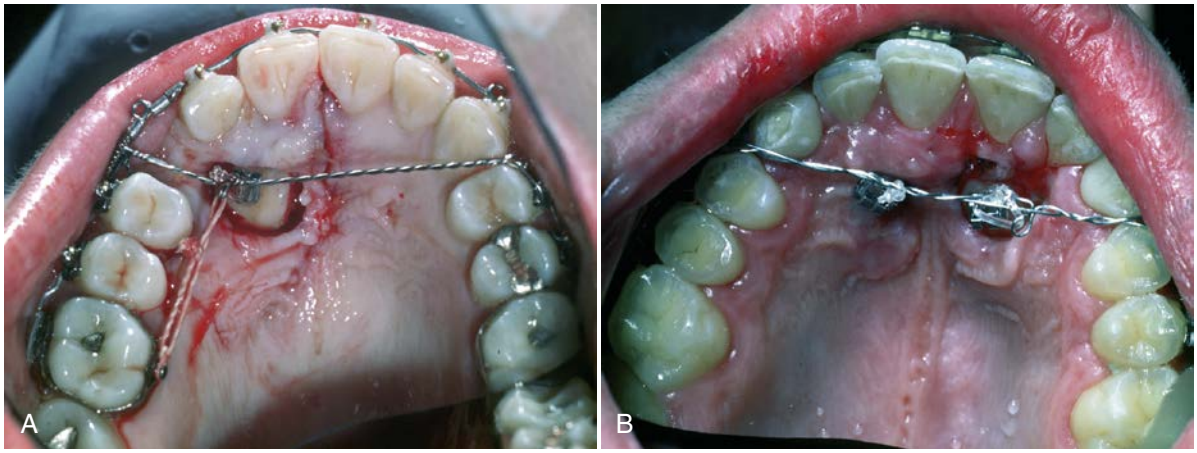


FIGURE 22-41 **A,** A 0.014-inch dead soft stainless steel wire is placed across the palate, with thread used to erupt the canine distally toward the tongue. **B,** A 0.012-inch stainless steel wire is used to tie elastomeric thread to each canine to erupt both teeth to the level of the palatal vault. They then can be guided toward the arch.

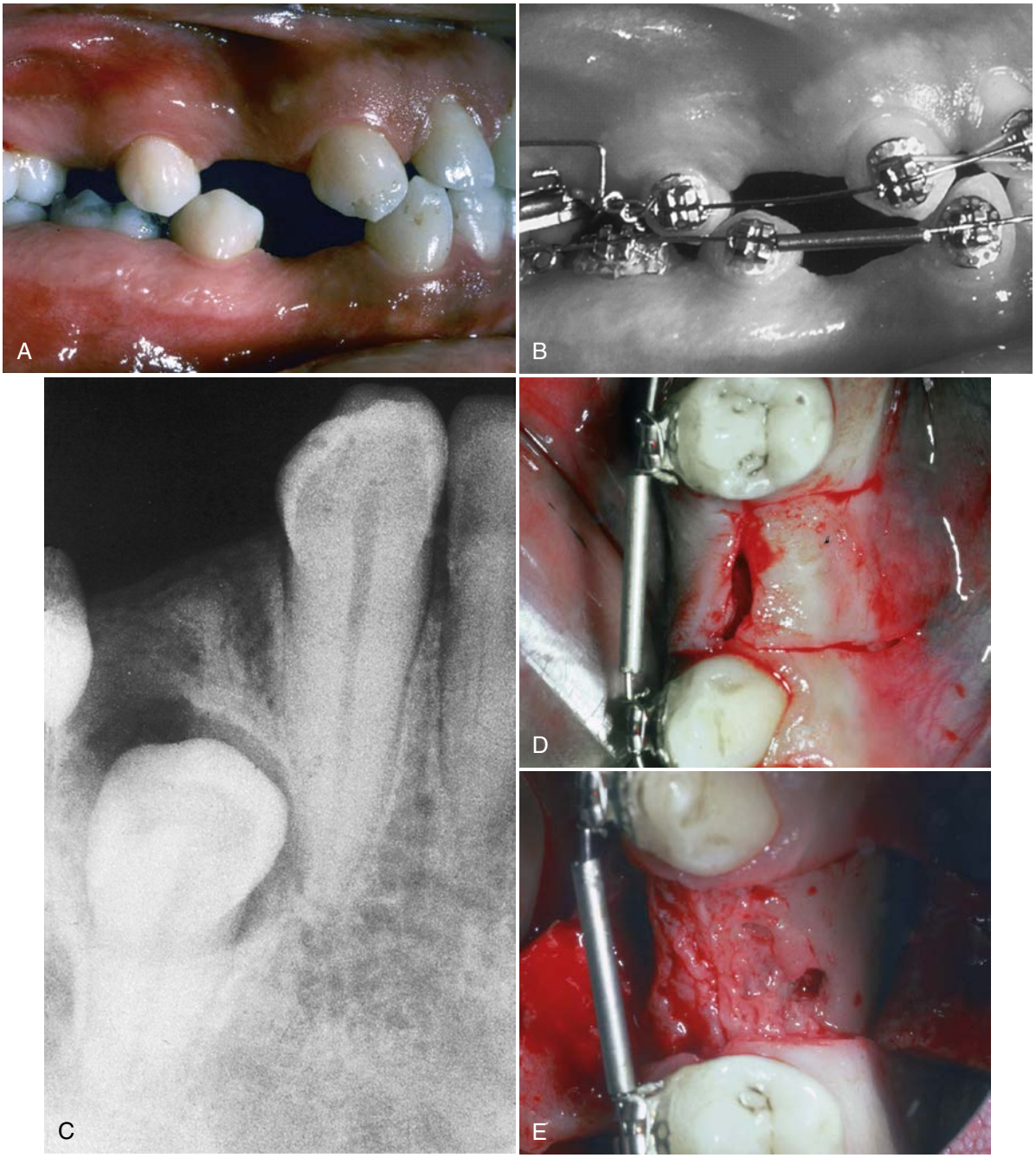


FIGURE 22-42 **A,** A 37-year-old woman who needs alignment in the maxillary right posterior segment before restorative dentistry. The lower right first premolar is impacted and ankylosed. The patient was advised to have the tooth extracted, but she elected to have orthodontic treatment. **B,** Appliances were placed to align the maxillary arch and allow immediate activation of orthodontic force on the lower premolar after it had been luxated. **C,** Preoperative radiograph of the ankylosed first premolar before it was uncovered. **D,** Initial releasing incisions made to mobilize a pedicle graft for the lingual and the facial of the unerupted premolar. **E,** With the tissue reflected, one can see that the premolar is covered completely by bone.

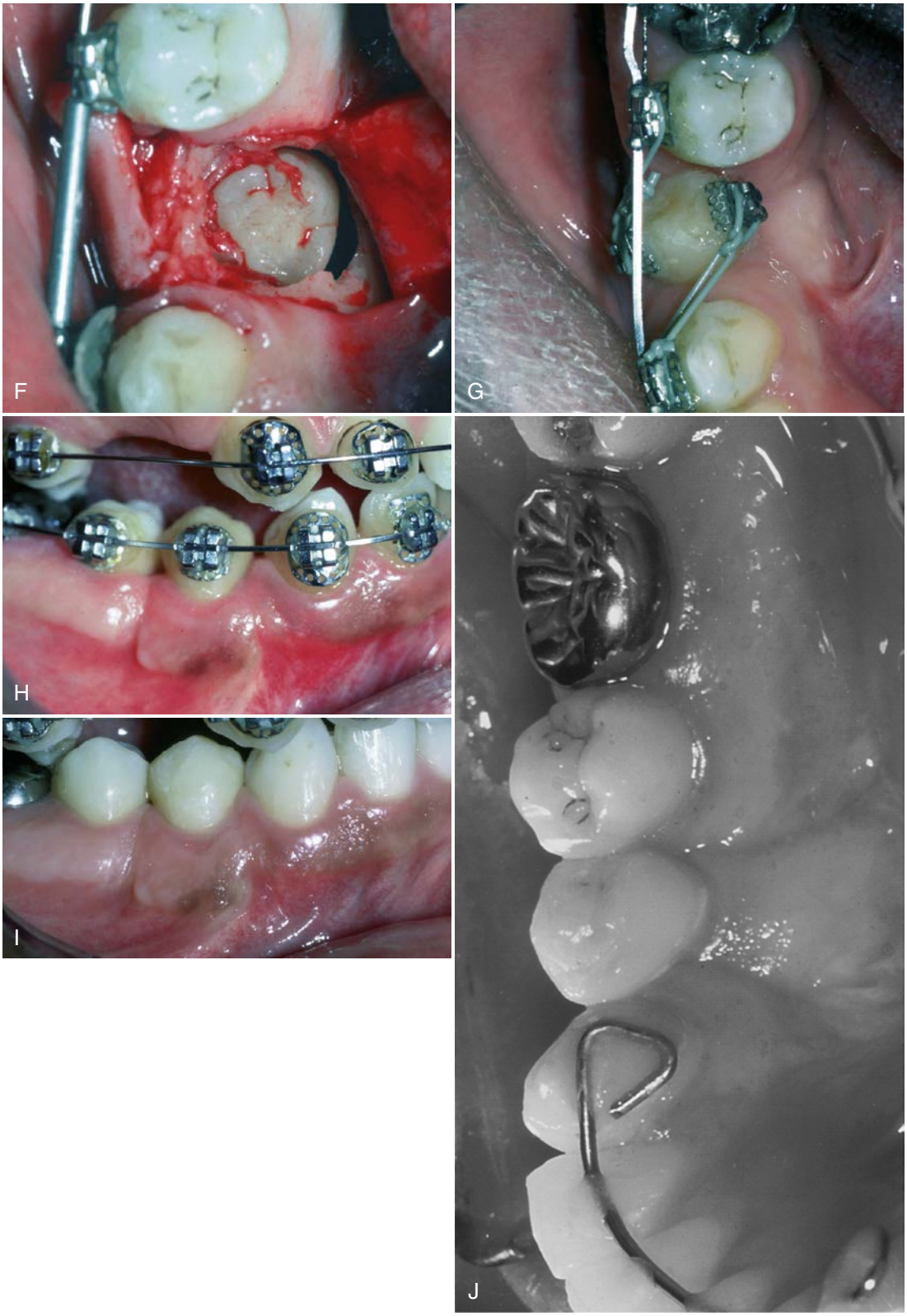


FIGURE 22-42, cont'd **F,** Osseous surgery is performed to uncover the tooth, which is located toward the lingual. The tooth is luxated carefully (moved occlusogingivally) until the area of fusion has been broken. A bonded attachment is placed (bonding should be done only on an ankylosed tooth at the time of surgery), and power thread must be activated within 24 hours and reactivated every 10 days, or the tooth will reankylose. **G,** Two points of attachment are needed to rotate the premolar effectively. **H,** Note that as the tooth moves toward the occlusal plane, the facial gingiva moves with it. **I,** One year after the tooth was positioned in the arch, it had not reankylosed. **J,** On the lingual aspect, note that more gingiva was created as the tooth was erupted. When the tissue attaches to the bone and the tooth (as eruption occurs), more gingiva is created. (From Hösl E, Zachrisson BU, Baldauf A, eds. *Orthodontics and Periodontics*. Chicago: Quintessence; 1985.)

FIGURE 22-43 **A,** An 11-year-old girl in whom the mandibular left central and lateral incisors would not erupt. **B,** Preoperative radiograph of the unerupted mandibular incisors. **C,** Initial incisions made to establish individual pedicle grafts for each incisor on the labial and lingual aspects of the two teeth. **D,** The mandibular arch was bonded and archwire was placed before the surgical procedure. Individual apically positioned pedicle grafts were placed on the labial surface of each incisor. **E,** Occlusal view showing the apically positioned pedicle grafts that were placed on the lingual aspect of each mandibular incisor. The margins of the grafts were placed on the enamel coronal to the cementoenamel junction area. **F,** Note that the incisors with grafts attached to the enamel on the left central and lateral incisors show normal gingival contour. (From Schatz JP, Joho J, eds. *Minor Surgery and Orthodontics*. Chicago: Quintessence; 1992.)

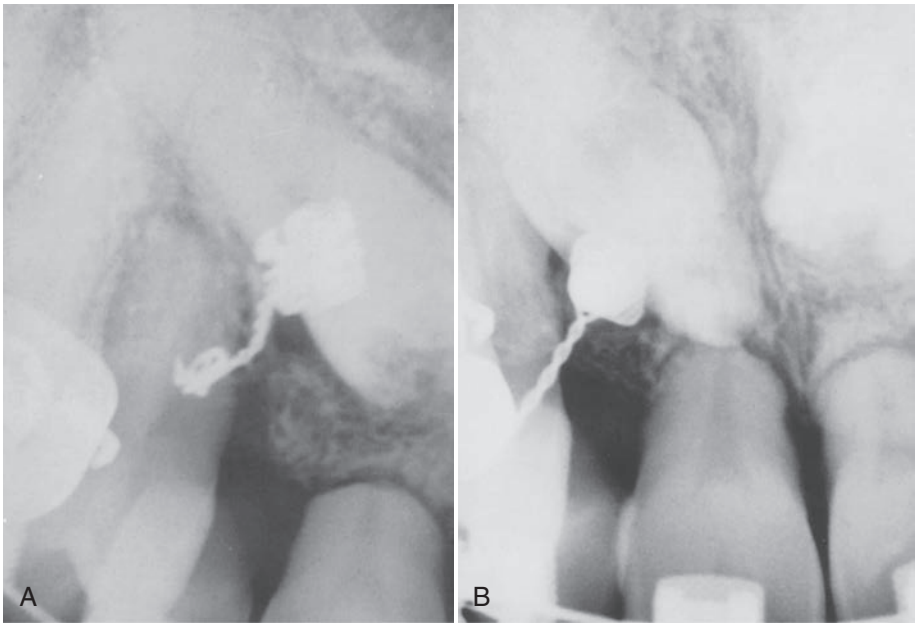


FIGURE 22-44 A, Periapical radiograph of a 50-year-old woman; the root is not well defined, and a periodontal ligament space is not evident on the unerupted canine teeth. The pulp also appears to be obliterated. B, Both canine crowns seem to fade or blend into the bone. Note the resorption areas on the anatomic crowns of both canines and the bonded incisors. The canines are ankylosed and cannot be moved into the arch.

finesse. This form of attachment can cause permanent loss of crestal bone at the time the tooth is ligated. In addition, the wire may damage the tooth and act as an irritant on the tooth side of the PDL space; this could result in injury or create an environment (in a susceptible patient) prone to ankylosis (see Fig. 22-45, *C–I*). Recent studies in periodontal wound healing have clarified the biologic response that allows replacement resorption and ankylosis. The wire can damage the cells on the tooth side of the PDL and prevent apical downgrowth of epithelium. The epithelium can protect the root against resorption and ankylosis. Granulation tissue derived from the bone against the root is known to induce resorption and ankylosis. Ankylosis, however, always occurs at the crestal area where the wire is positioned. One hypothesis is that the circumferential destruction of the PDL extending from the cementum to the bone may be a necessary prerequisite for ankylosis.[5] Several researchers have suggested that ankylosis can occur because of the repopulation of the PDL from the adjacent bone marrow.[128,129]

The mechanism of injury and ankylosis are becoming better understood;[130] however, patients with this problem predictably show one common denominator: all demonstrate rapid healing clinically. In periodontics, supracrestal external root resorption has been reported to occur when fresh hip marrow with cancellous bone was used as osseous autograft material. The wire loop also may allow inflammation to be established in the area of resorption. The replacement resorption may be arrested by removal of the bone, or the tooth may become extensively resorbed at the crestal area.

Similar to the lasso wire technique, the gold chain (close technique) has a high incidence of tooth ankylosis, in addition to pain in every activation (the soft tissue grows in between the chain), prolonged treatment time, additional surgical procedures, multiple debondings, lack of visibility to change direction of pull, and persistence of the inflammatory dental follicle, which may all cause periodontal damage (Fig. 22-46).[131]

Usually, when teeth that show external root resorption are exposed (if one assumes that ankylosis has been allowed to exist for a short period), only a small area (12 mm) is involved. The bone can be removed with a chisel, and the tooth can be orthodontically positioned into the arch, with continuous force being placed the same day the tooth is uncovered surgically and is freed up (Fig. 22-47). If the force is allowed to dissipate for any length of time, the tooth may become reankylosed. After the tooth has been freed from the bone by osseous surgery or luxation (so that the tooth has a vertical component to the mobility), the force must be reactivated every 10 days.

Many teeth that are ankylosed do not have crestal external root resorption or crestal ankylosis that can be seen on tissue reflection, thus allowing the ankylosed spot to be removed surgically. These teeth can be freed merely by luxation, and an immediate continuous force can be used to position the tooth orthodontically into the arch. Multirooted molars or canines with dilacerated roots have a poorer prognosis for movement after luxation than normally shaped, single-rooted teeth. Because of crestal replacement or resorption (or ankylosis), tooth extraction may be prevented by osseous surgery to remove the bone attached to the tooth followed by orthodontic movement into the arch and reestablishment of the normal anatomic crown for the tooth using restorative dentistry.

One should understand that all ectopically positioned or ankylosed teeth cannot be treated successfully. However, a higher percentage of success can be achieved with unerupted teeth by attending to normal development, supporting tissue, atraumatic surgery, bonded attachments, control of gingival inflammation, and use of minimal orthodontic forces.

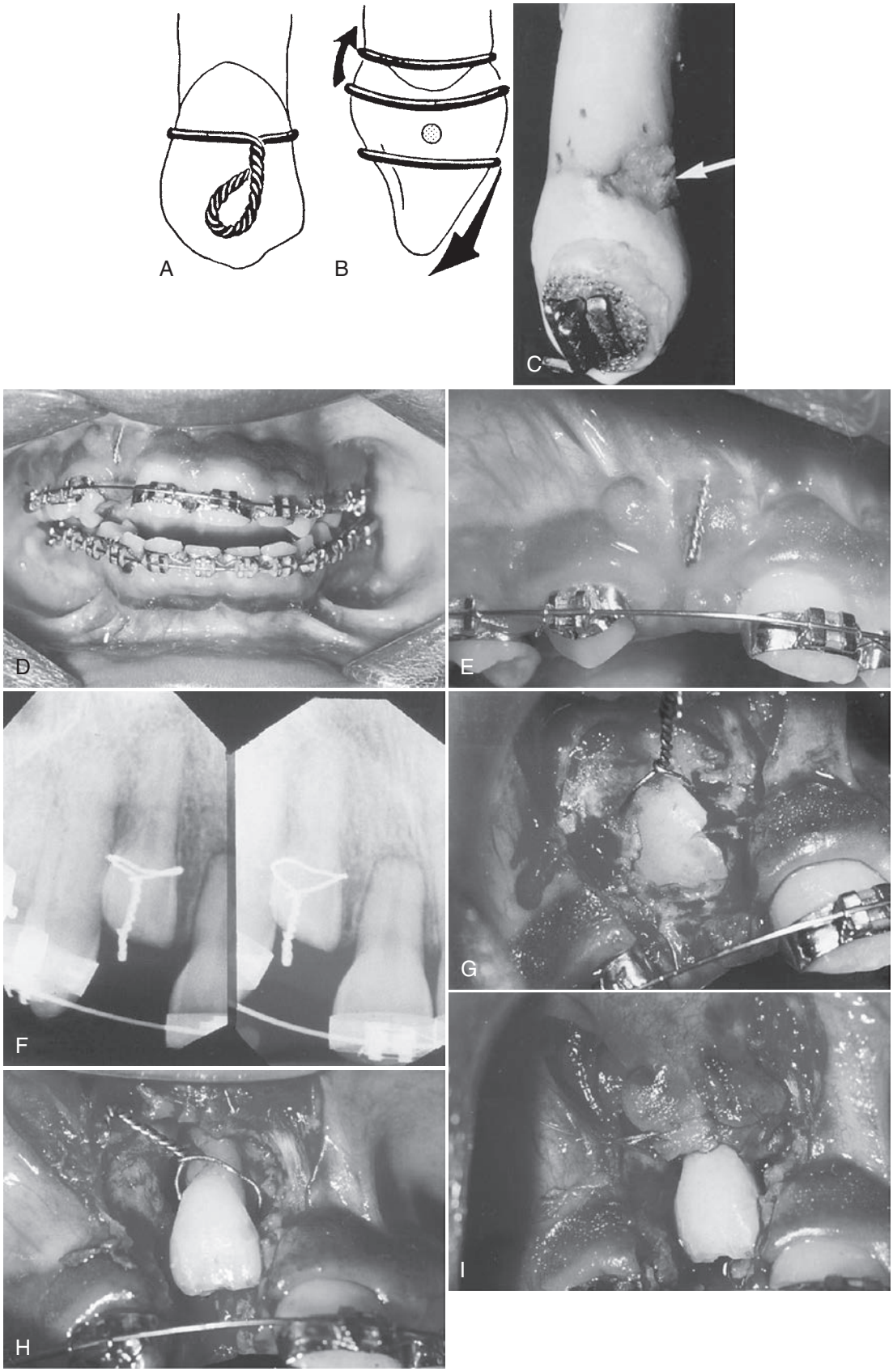


FIGURE 22-45 **A,** Diagrammatically, the ligature tied in the area of the cementoenamel junction (if it were to stay above the contact area) will move incisally and come off the tooth. **B,** If the tooth is ligated below the contact area, the ligature will tend to move apically and impinge on the cement-enamel (CEJ) area, damaging the root surface. **C,** An extracted canine that was uncovered surgically and ligated with a wire loop. The bone invading the root had spread from the CEJ area into the root and the anatomic crown. **D,** In the case of this 16-year-old girl, the lateral incisor was uncovered, and a wire looped attachment was placed by the surgeon; ankylosis occurred. The ankylosed lateral incisor caused an open bite by intruding the maxillary arch as wire changes continued. **E,** Wire that penetrated the alveolar mucosa was tied to the base arch by elastomeric thread. **F,** Note the radiographic appearance of bone invading the root apical to the wire loop. **G,** On reflection of the soft tissue, note the bone over the wire loop and invading the tooth. **H,** Osseous surgery was performed to create a sound tooth structure, and all bone attached to the root was removed. **I,** A pedicle graft was placed on the tooth, which was salvaged.

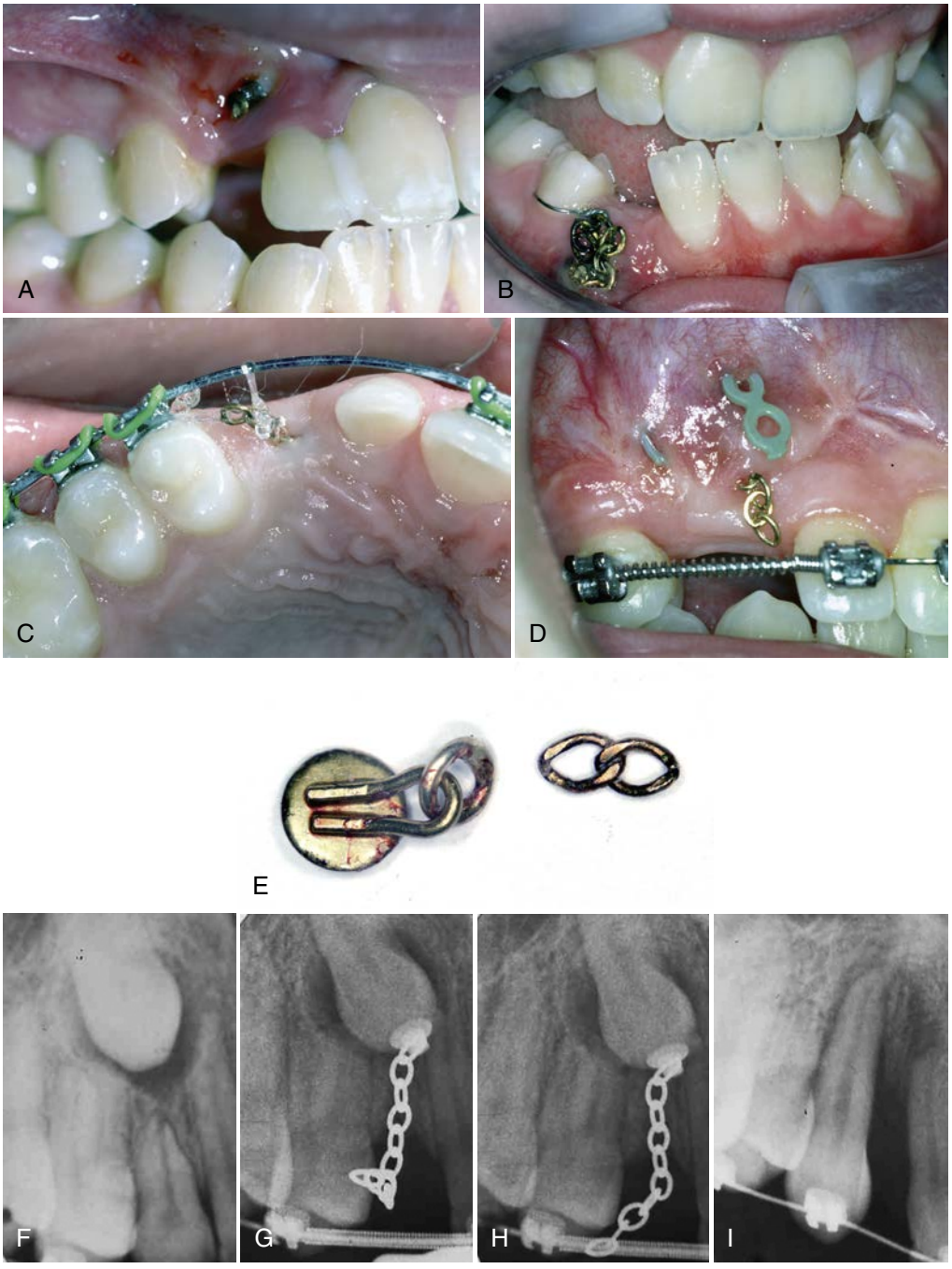


FIGURE 22-46 The gold chain and closed eruption has been a misleading technique for many clinicians. **A–E,** Problem cases with no arch preparation and arch distortion caused by forces applied (**A, B**), diminished control over the direction of pull (**C**), additional surgical interventions (**D**), and multiple debondings (**E**). Prolonged treatment time and the persistence of the inflammatory follicle are additional problems of this technique that may cause periodontal damage. **F,** Initial periapical film of an impacted cuspid with an existent follicle that (**G, H**) was exposed with a closed technique and a gold chain. **I,** One year and 3 months later, the cuspid was brought into the arch. Note the damage on the adjacent tooth and the severe bone loss.

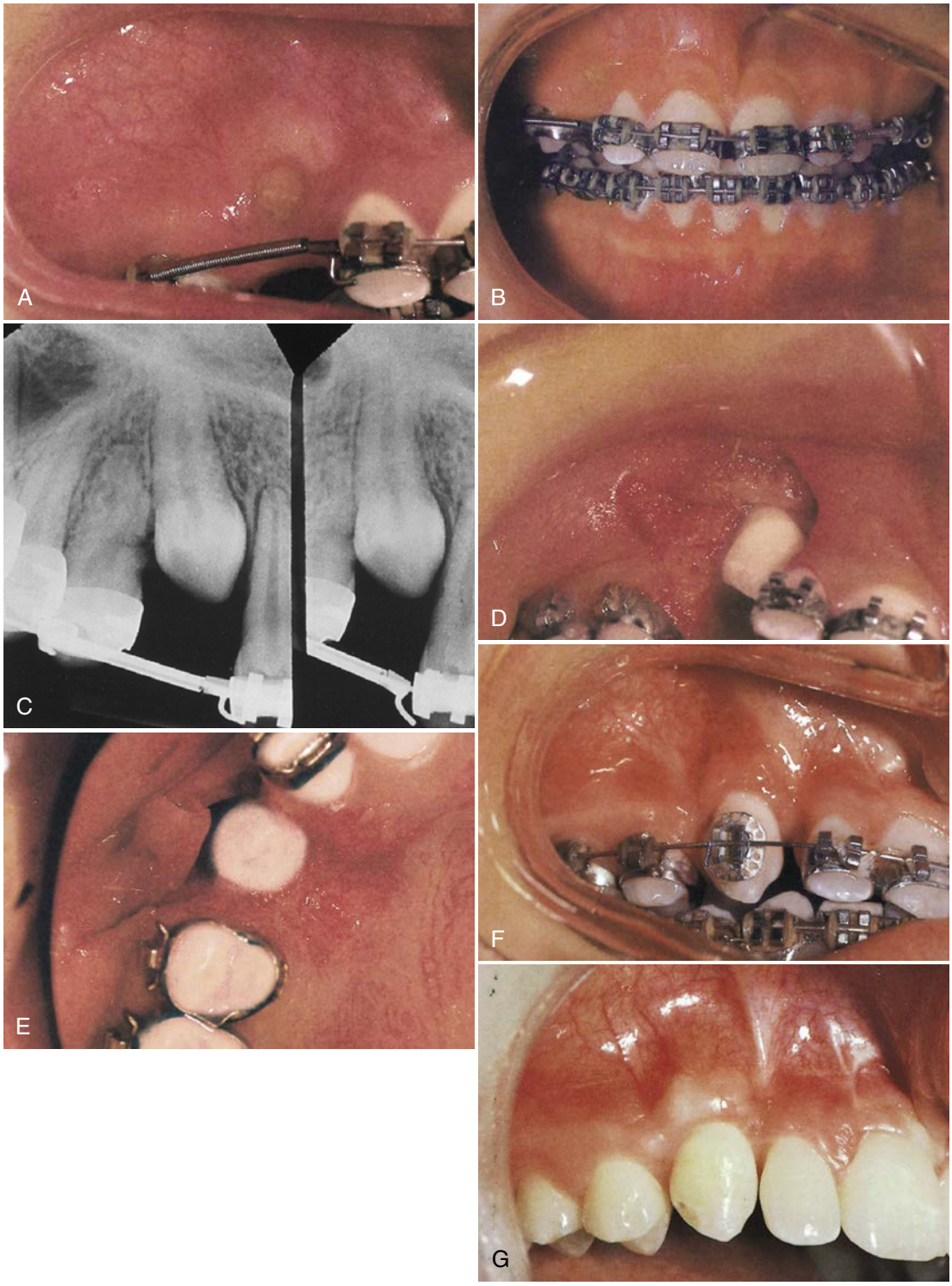


FIGURE 22-47 **A,** Note the labial aspect of a partly exposed maxillary right canine that had been uncovered on three previous occasions. **B,** Anterior view after the arch had been intruded and leveled again. **C,** Periapical radiograph made when the tooth was uncovered. **D,** An apically positioned graft was placed on the labial. **E,** A small area of spot ankylosis was found and removed on the palatal aspect, and the tooth was moved into the arch. **F,** The tooth has been in the arch for 2 months after the surgical procedure. **G,** Appliances were removed, and the tooth was stable.

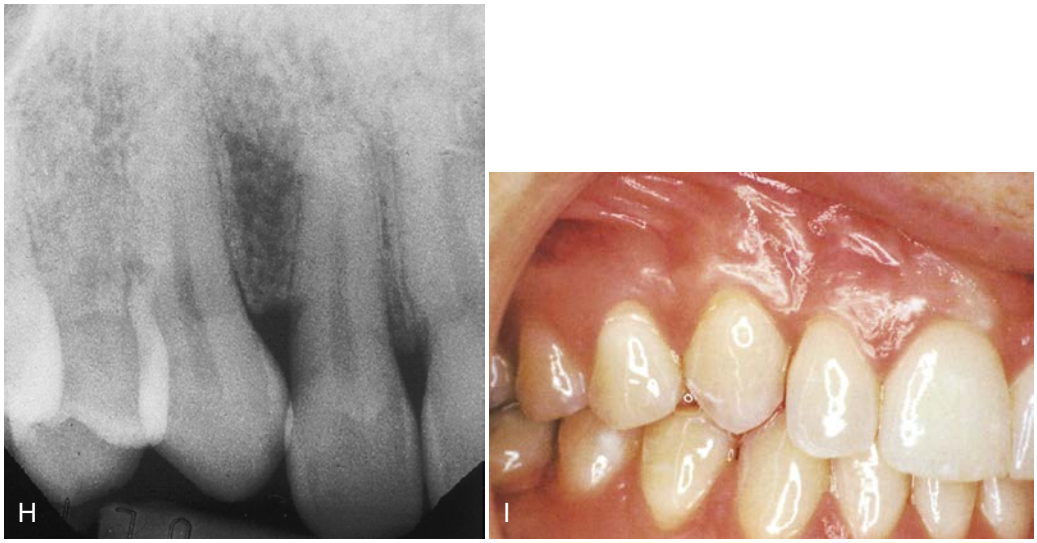


FIGURE 22-47, cont'd H, Periapical radiograph made 18 years later. I, Eighteen-year follow-up finds excellent function of the tooth.

ALVEOLAR DECORTICATION AND AUGMENTATION GRAFTING

The thickness of the alveolar bone delineates the limits of the tooth movement. Exceeding these boundaries may produce adverse consequences for the periodontal tissues. Moreover, bone resorption occurs in the direction of tooth movement, so the reduced volume of the cortical bone, sometimes with minimal thickness and sometimes nonexistent, is a risk factor for loss of attachment.[132] Reducing the cortical alveolar bone may increase the incidence of dehiscences and fenestrations, which are the most common alveolar defects. An alveolar dehiscence represents the lack of facial or lingual cortical plates, which exposes the root surface; an alveolar fenestration is a circumscribed defect of the cortical plate that exposes the underlying root surface without affecting the marginal bone.[133–135] In general, whereas fenestrations are commonly observed in the maxilla (maxillary first molar), dehiscences are more predominant in the mandibular arch (mandibular canine).[133,134,136] In the mandible, the bone becomes thinner in the anterior region.[137] Huynh-Ba et al.[138] evaluated socket bone wall dimensions of extracted teeth and reported that only a minority (6.5%) of the maxillary teeth, including incisors, canine, and premolars, had a buccal bone wall width of 2 mm or more. For the anterior sites (canine to canine), the mean width of the buccal bony wall was 0.8 mm. For the posterior (premolar) sites, it was 1.1 mm.

Clinically, gingival recession is always accompanied by alveolar bone dehiscences.[139] The incidence of dehiscence is positively correlated with thin alveolar bone.[140] The direction of movement, the frequency and magnitude of orthodontic forces, and the volume and anatomic integrity of periodontal tissues also influence the occurrence of dehiscence and fenestration during orthodontic treatment.[141]

Dental arch expansion and incisor buccal-lingual movement are the most critical orthodontic movements.[142] Based on the amount of orthodontic movement and the initial morphology of the alveolar bone, orthodontics may result in alveolar defects.

Therefore, an alveolar bone morphology determination[143] and the need for the surgical procedure are keys to avoid these complications. Alveolar decortication in combination with bone grafting would be indicated to resolve existing dehiscences or fenestration and possible combination with orthodontic treatment. If excessive labiolingual movement is needed, grafting material should be considered.

Background

The alveolar decortication–assisted orthodontic treatment is an established and efficient technique that has been extensively studied in the past decade. It is defined as a surgical procedure that helps orthodontic tooth movement by stimulation of bone metabolism caused by controlled surgical damage. It is done to induce a state of increased tissue turnover and a transient osteopenia, which are followed by a faster rate of orthodontic tooth movement.

The application of corticotomy surgery to correct malocclusion was first described in 1892 by L.C. Bryan.[144] But it was Heinrich Köle[145] in 1959 who reintroduced alveolar corticotomy to resolve malocclusion. He combined interdental alveolar corticotomy surgery with a through-and-through osteotomy apical to the teeth. In 1975, Duker[146] found in a study on beagle dogs treated with vertical corticotomies and horizontal osteotomies that approximately 4 mm of movement occurred in an 8- to 20-day period on the maxillary incisor area with minimal changes in the marginal mucosa. Suya[147] also believed that the tooth movement occurred by movement of the independent bony blocks created during the surgery. In 2001, Wilcko et al.[148] described alveolar decortication with augmentation grafting combined with orthodontic treatment to accelerate tooth movement and to provide increased alveolar volume. They speculated that the dynamics of physiologic tooth movement in patients who underwent decortication might be due to a demineralization–remineralization process rather than bony block movement. They suggested that this process would manifest as a part of "the regional acceleratory phenomenon" (RAP).

Alveolar Bone Biological Response (Regional Acceleratory Phenomenon)

The orthopedist Herald Frost documented that surgical wounding of osseous tissue results in a restructuring activity adjacent to the site where the surgery was performed. He named this cascade of physiologic healing events "the regional acceleratory phenomenon."[149,150]

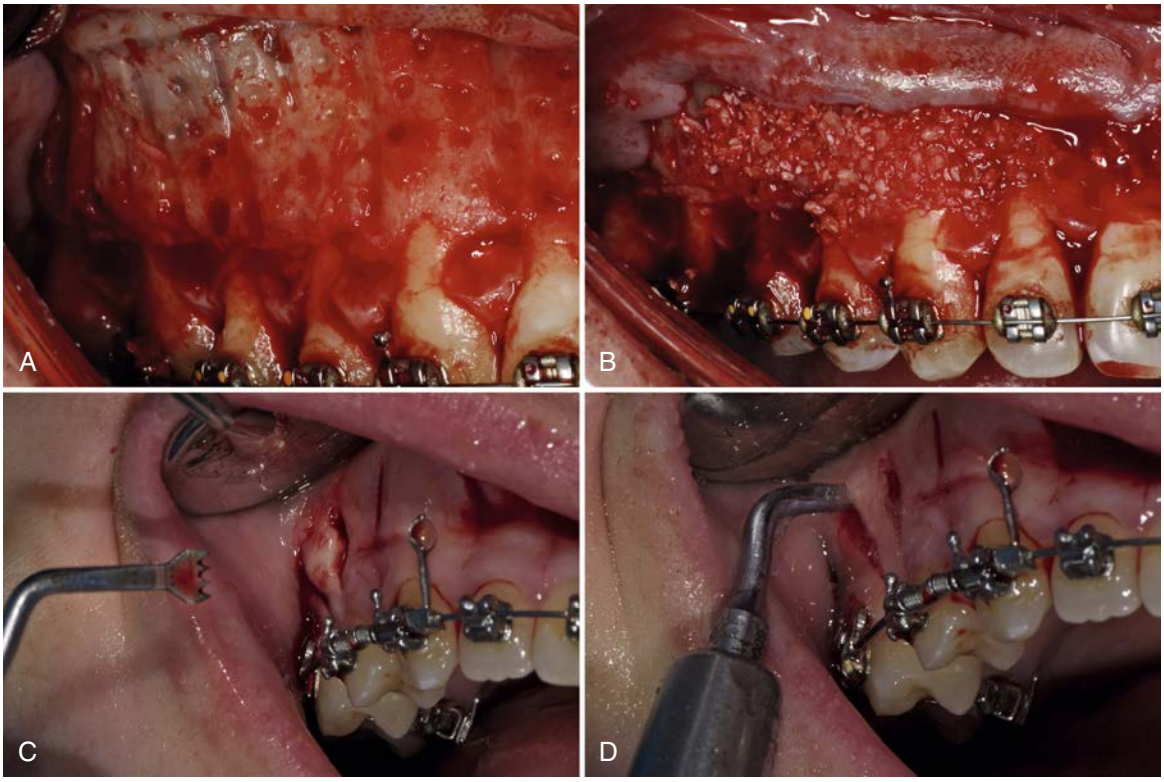


FIGURE 22-48 A, Surgical technique. Note the surgical damage to the cortical plate to induce the regional acceleratory phenomenon. B, Particulate bone grafting. C, D, A modified flapless technique, with vertical incisions and piezoincision. (Case treated by Drs. Blasi and Dhyllon.)

RAP is a local response of tissues to noxious stimuli by which the rate of remodeling in the region of a bone defect exceeds the ordinary tissue activity to potentiate tissue healing and local tissue defense reactions.[151] This response differs in duration, size, and intensity with the extent of the stimulus. The duration of RAP depends on the type of tissue and usually consists around 4 months in human bone. This phenomenon produces bone healing to occur 10 to 50 times faster than regular bone turnover.[152]

The healing phases of RAP have been studied in rat tibias. After a burr hole defect, there is an initial phase of maximally stimulated bone formation followed by a phase of predominant resorption. The first osteogenic phase consists of woven bone formation. It takes place in the periosteal area and then covers the medullary bone, attaining its maximal thickness on day 7. This cortical bridge of woven bone provides mechanical stability of bone subsequently injury. From day 7, whereas most of the medullary bone undergoes resorption, the woven bone in the cortical area begins to be replaced to lamellar bone. It was also found that restoration of a bone defect leads to a systemic acceleratory phenomenon (SAP) of osteogenesis caused by systemic release of humoral factors.[152] In human long bones, after surgical injury, RAP initiates within a few days, usually peaks at 1 to 2 months, and may take from 6 to 24 months to subside completely.[150] RAP causes osteopenia in healthy tissues, but the volume of bone matrix remains constant.[149,152,153] It is hypothesized that small perforations of the buccal cortical plate of the maxilla would lead to the expression of inflammatory cytokines. These inflammatory cytokines have been related with the acceleration of bone remodeling process and therefore the rate of tooth movement.[154] Studies of knockout mice deficient for tumor necrosis factor–alpha receptors revealed reduced tooth movement in response to orthodontic forces.[155] Previous reports have shown that anti-inflammatory medication can decrease the rate of tooth movement.[156] Orthodontic force application alone triggers mild RAP activity. But when tooth movement is combined with selective decortication, RAP is maximized.

Surgical Technique

Decortication

The goal of decortication is to initiate RAP response. It is performed around the teeth planned for movement, with bone cuts made using a round bur or piezoelectric knife.[157] Bone is injured with a surgical bur, taking into account the areas where the cortical bone is thick rather than satisfying any particular preconceived pattern. Typically, it is done between the root prominences where a vertical groove is placed. The groove extends 2 to 3 mm below the crest of the bone to a point 2 mm beyond the apices of the roots. Then vertical corticotomies are connected with the circular-shaped corticotomy (Fig. 22-48).

Particulate Grafting

The combination of corticotomy with an alveolar graft was introduced by Wilcko et al.[148,158] and is referred to as accelerated osteogenic orthodontics (AOO) or periodontally accelerated osteogenic orthodontics (PAOO).[159–161] Wilcko et al. asserted that bone grafting of the labial and lingual cortical bones would increase the range of possible tooth movements, enhance alveolar bone volume, provide a more structurally stable periodontium, and gain stability of orthodontic treatment.[162] It is surmised that the stability found in the mandibular anterior segment may be due to increased cortical bone thickness.[163] Furthermore, there is the assumption that after performing a flap surgery and the decortications, there is a substantial tissue memory loss.[164]

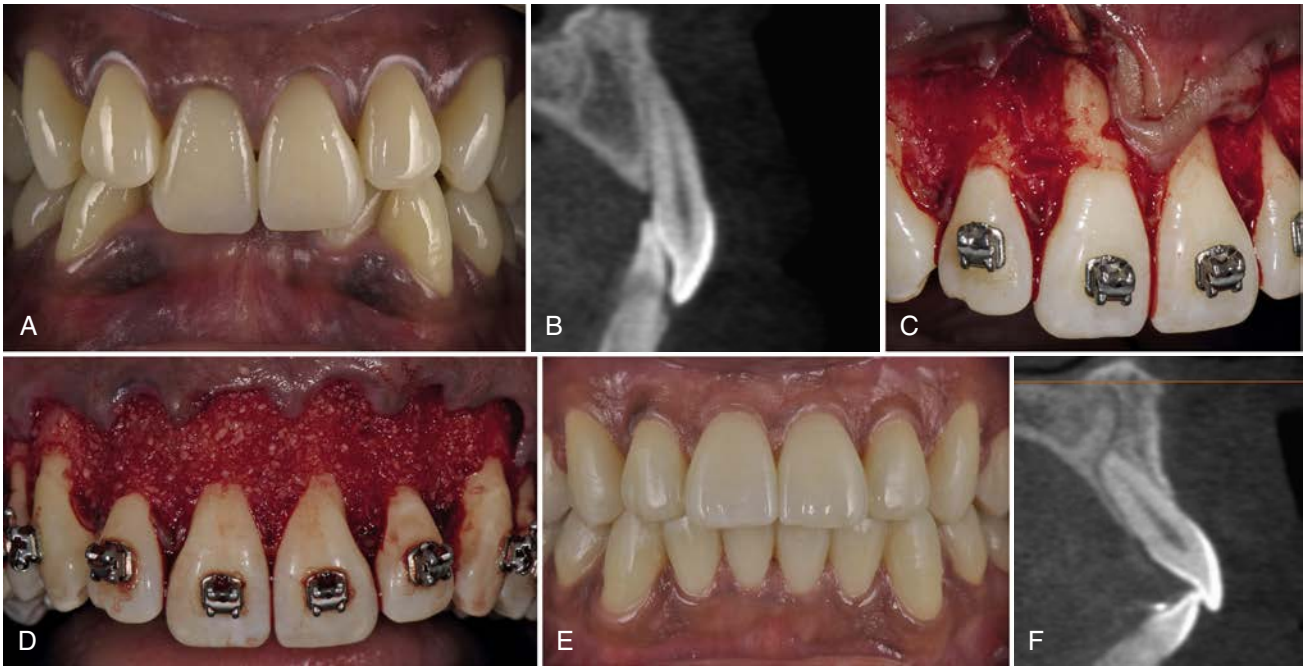


FIGURE 22-49 **A,** A 25-year-old man with a deep bite, retroclined upper incisors, and average biotype. **B,** Cone-beam computed tomography (CBCT) sagittal cut demonstrating inclination of upper incisor and dehiscence. The patient was diagnosed and treatment planned by the orthodontist; referred to a periodontist for decortication to initiate the regional acceleratory phenomenon and achieve a predictable correction with decrease of anchorage; and to perform bone grafting augmentation to modify the alveolar bone width and cover the dehiscence. **C,** Open flap view of the area of interest. Note the lack of buccal plate with full exposure of the root demonstrating a true positive finding on the CBCT. **D,** Labial augmentation bone grafting on the anterior segment. **E,** Finished case with reduced risk of loss of attachment. **F,** Posttreatment CBCT sagittal cut of the upper incisor. Note the presence of bone on the facial aspect of the incisor. (Case treated by Drs. Blasi, Boucher, and Evans.)

In a study with five beagle dogs by Choi et al.,[165] new bone formation was found at the buccal surface in an augmented corticotomy site. Another study by Araujo et al.[166] using five beagle dogs demonstrated that it was possible to move a tooth into an area of an alveolar ridge that 3 months previously had been augmented with a biomaterial. It was also demonstrated that 12 months after grafting, Bio-OssA particles were not present in the area of tension site where the tooth movement was but were present in small amounts in a zone where the pressure side was. No objective data exist comparing one grafting material with another in terms of superiority. The most commonly used materials are deproteinized bovine bone, autogenous bone, decalcified freeze-dried bone allograft, or a combination of these (see Fig. 22-48, *B*).[159] Use of platelet-rich plasma or calcium sulfate increases the stability of the graft material.[159] Antibiotics can be mixed with bone graft. Wilcko et al.[148,158,167,168] recommended the use of a mix of demineralized freeze-dried bone and bovine bone wetted with a clindamycin phosphate solution. Alveolar augmentation with demineralized freeze-dried bone allograft (DFDBA)/xenograft (bovine bone) or alloplastic graft (bioactive glass) was described by Wilcko et al.[148] to cover any fenestrations and dehiscences and increase bony support for teeth with corticotomy-assisted orthodontic tooth movement. The author noticed that particles of xenograft were not fully incorporated at 15 months of reentry; however, these particles were simply wiped off with a piece of gauze, revealing a layer of bone with a marbleized appearance. Additionally, the fenestration buccal of the root of the first premolar was covered.[148] Later, in two cases reports, it was noticed that the dehiscence areas were completely filled in with bone shown in histology of bone biopsy.[160] Furthermore, a recent study reported that augmented corticotomy using nonautogenous grafts facilitated tooth movement without fenestrations and accelerated new bone formation.[169]

There are case reports that describe predictability of combination with ridge augmentation procedures, facilitating force eruption procedures and in combination with soft tissue procedures for the treatment of gingival recessions.[170] Some authors report less invasive techniques with the use of a tunneling technique with piezoincision (see Fig. 22-48, *C*).

Patient Management

Activation of appliances should be done every 2 weeks.

The technique allows the clinician to change the patient biotype (described earlier in this chapter) of the patient. Alveolar decortication and augmentation grafting might be indicated in thin-scalloped biotypes, especially if teeth might be moved past the anatomic boundaries or basal bone.

Treatment Indications and Limitations

Unfortunately, there is a misunderstanding of the benefits of this procedure because of the emphasis placed on speed of movement rather than **enhancing the periodontium** and expanding limits of the treatment (Fig. 22-49). Shortening the treatment plan should not be an indication; rather, it

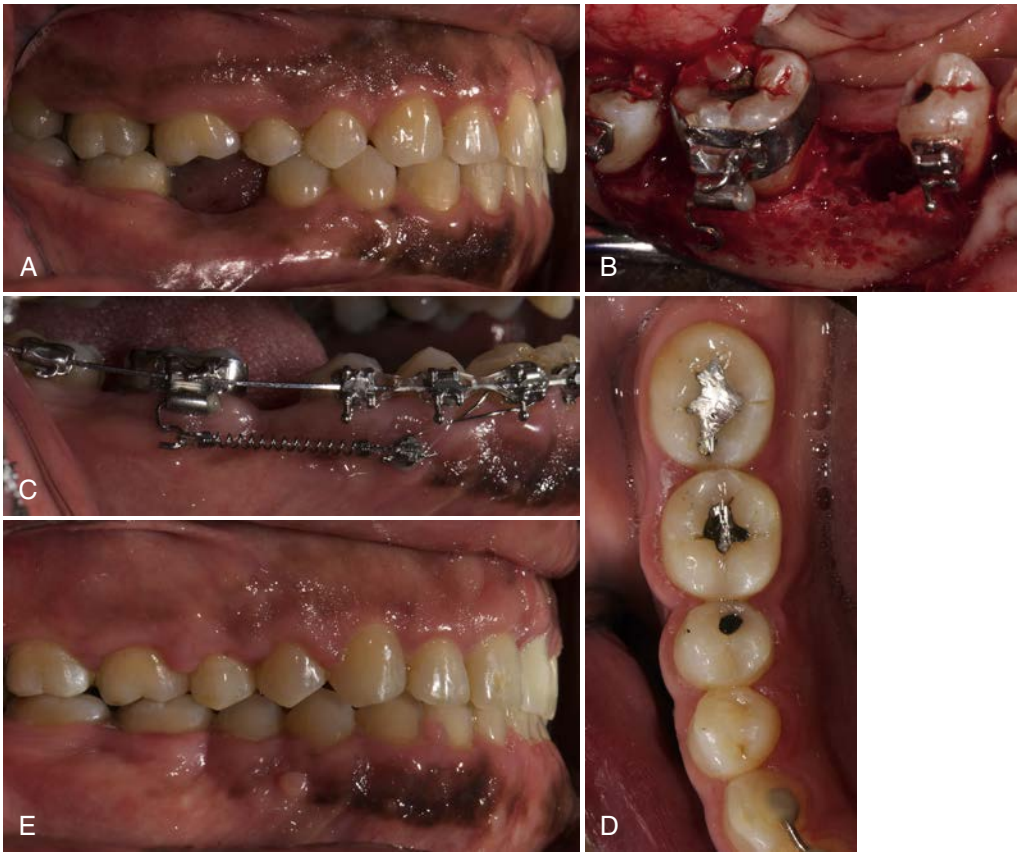


FIGURE 22-50 **A,** A 56-year-old woman before orthodontic treatment. The patient had a history of dental extraction. Upon radiologic evaluation, there was noted a radicular rest left on the molar extraction site, and it was decided to perform decortication at the time of the surgical extraction. **B,** Decortication of cortical plate to facilitate closure of the 11-mm edentulous space. **C,** Activation of force applied with a closed coil. A temporary anchorage device was used for maximum protraction. A passive stainless steel ligature was tied to the cuspid to maintain the position of the anterior segment. **D, E,** Finished case with full protraction of second and third molars into first and second molar position. Note that the Class I dental relationship was maintained. (Case treated by Drs. Blasi and Tanna.)

should be seen as a beneficial side effect (see Chapter 31). It is critical to make a differential diagnosis between the severity of the skeletal and dental components for proper case selection.

It has been reported that the major resistance to the forces acting in orthodontic treatment comes from the cortical bone.[171] Therefore, alveolar decortication allows reducing anchorage, decreasing the resistance of the cortical bone, and achieving difficult tooth movements, movements that were considered to be impossible to achieve in the past.

The envelope of discrepancy for the maxillary and mandibular arches in three planes of space, described in Chapter 21, indicates the limits of tooth movement. Orthodontic treatment is limited by the scope of the alveolar bone, and additional surgical procedures are needed if the limits have to be surpassed. Therefore, an alveolar decortication and augmentation grafting surgical approach increases the alveolar bone volume and expands the anatomic alveolar limits on which teeth can be moved. The quantified limits have to be yet determined. It is important to remember that this approach only changes the alveolar bone, not the basal structures. It does not substitute for or equal the benefits of an orthopedic expansion and changes on the maxillary basal bone described in this chapter. The decortication procedures combined with orthodontic treatment could be indicated for mild transverse discrepancies (only for changes of arch form). The camouflage treatment of skeletal discrepancies in three planes of space could be expanded with PAOO. However, for severe skeletal discrepancies, orthognathic surgery remains the best or a combination of both (orthognathic surgery and PAOO).[172]

PAOO is indicated to resolve anterior crowding, close extraction or edentulous space (Fig. 22-50), change the shape of the alveolus (expansion, asymmetric or mutilated cases), forward movement of lower anterior teeth, intrusion, correction of open bites, unilateral case types, decrease or change anchorage (see Chapter 18 for change of anchorage), and correct dehiscence and fenestrations. The advantages of this procedure are that it enhances the periodontum, increases stability, increases the scope of alveolar change, leads to less root resorption, reduces treatment time, and reduces the risk of periodontal breakdown. Its potential disadvantages include that it is a surgical procedure (invasive and aggressive, surgical complications

(discomfort, loss of bone, and so on), increased treatment cost, and inconvenience (appointments every 2 weeks).

Alveolar decortication and augmentation grafting is a legitimate modality that provides enhanced treatment outcomes and accelerated leveling and alignment with reduced risk of loss of attachment.

SUMMARY

No attempt has been made in this chapter to provide a complete discussion of orthodontic and periodontal interrelationships. The intent has been to highlight frequent problem areas. Gingival retention procedures, management of adult patients, and correction of periodontal problems are discussed in Chapter 21. Research information and documented periodontal problems—gathered from clinical observation, clinical trials, animal research, and retrospective analysis—are sufficient to allow the formulation of clinical guidelines for the achievement of orthodontic objectives in problem cases; the ultimate objective is that the patient will be better off for having received treatment. Periodontal problems have not been eliminated for all patients, but significant progress has been made toward prevention of many adverse responses.

About 10% of adolescents, as well as patients with special problems and adult patients, remain periodontal challenges for the future. For orthodontists, the prerequisite for providing effective treatment is a periodontal knowledge base sufficient to prevent periodontal injury.

REFERENCES

1. Newman MG. Improved clinical decision making using the evidence-based approach. *Ann Periodontol*. 1996;1(1), i–ix.
2. Helm S, Petersen PE. Causal relation between malocclusion and periodontal health. *Acta Odontol Scand*. 1989;47(4):223–228.
3. Bjornaas T, Rygh P, Boe OE. Severe Overjet and overbite reduced alveolar bone height in 19-year-old men. *Am J Orthod Dentofacial Orthop*. 1994;106(2):139–145.
4. Ashley FP, Usiskin LA, Wilson RF, et al. The relationship between irregularity of the incisor teeth, plaque, and gingivitis: a study in a group of schoolchildren aged 11-14 years. *Eur J Orthod*. 1998;20(1):65–72.
5. Baldinger J, Chung CH, Vanarsdall RL. *The Presence of Periopathogenic Organisms in Crowded Versus Aligned Teeth in the Anterior Adult Dentition*. Thesis. Philadelphia: University of Pennsylvania; 1998.
6. Davies TM, Shaw WC, Worthington HV, et al. The effect of orthodontic treatment on plaque and gingivitis. *Am J Orthod Dentofacial Orthop*. 1991;99(2):155–161.
7. Offenbacher S. Periodontal diseases: pathogenesis. *Ann Periodontol*. 1996;1(1):821–878.
8. Kornman KS, Van Dyke TE. Bringing light to the heat: "inflammation and periodontal diseases: a reappraisal." *J Periodontol*. 2008;79(8):1313.
9. American Academy of Periodontology. Consensus Report. Discussion section I. In: Nevins M, Becker W, Kornman K, eds. *Proceedings of the World Workshop in Clinical Periodontics*. Chicago: American Academy of Periodontology; 1989, pp I-23-I-32.
10. Zambon JJ. Periodontal diseases: microbial factors. *Ann Periodontol*. 1996;1(1):879–925.
11. Armitage GC. Development of a classification system for periodontal diseases and conditions. *Ann Periodontol*. 1999;4(1):1–6.
12. Armitage GC. AAP Centennial Commentary: theme 3: evolution and application of classification systems for periodontal diseases—a retrospective commentary. *J Periodontol*. 2014;85(3):369–371.
13. Cimasoni G. Crevicular fluid updated. *Monogr Oral Sci*. 1983;12:III–VII. 1–152.
14. Greenstein G. The role of bleeding upon probing in the diagnosis of periodontal disease. A literature review. *J Periodontol*. 1984;55(12):684–688.
15. Heintze SD. *Oral Health for the Orthodontic Patient*. Chicago: Quintessence; 1999.
16. Sooriyamoorthy M, Gower DB. Hormonal influences on gingival tissue: relationship to periodontal disease. *J Clin Periodontol*. 1989;16(4):201–208.
17. Page RC, Schroeder HE. Pathogenesis of inflammatory periodontal disease. A summary of current work. *Lab Invest*. 1976;34(3):235–249.
18. Socransky SS. Microbiology of periodontal disease—present status and future considerations. *J Periodontol*. 1977;48(9):497–504.
19. Eke PI, Dye BA, Wei L, et al. Prevalence of periodontitis in adults in the United States: 2009 and 2010. *J Dent Res*. 2012;91(10):914–920.
20. Socransky SS, Haffajee AD, Cugini MA, et al. Microbial complexes in subgingival plaque. *J Clin Periodontol*. 1998;25(2):134–144.
21. Moreira RM, Feres-Filho EJ. Comparison between full-mouth scaling and root planing and quadrant-wise basic therapy of aggressive periodontitis: 6-month clinical results. *J Periodontol*. 2007;78(9):1683–1688.
22. Yek EC, Cintan S, Topcuoglu N, et al. Efficacy of amoxicillin and metronidazole combination for the management of generalized aggressive periodontitis. *J Periodontol*. 2010;81(7):964–974.
23. Leggott PJ, Robertson PB, Greenspan D, et al. Oral manifestation of primary and acquired immunodeficiency diseases in children. *Pediatr Dent*. 1987;9(2):98–104.
24. Bermejo-Fenoll A, Sanchez-Perez A. Necrotising periodontal diseases. *Med Oral Patol Oral Cir Bucal*. 2004;9(suppl):114–119. 108–114.
25. Cobb CM, Ferguson BL, Keselyak NT, et al. A TEM/SEM study of the microbial plaque overlying the necrotic gingival papillae of HIV-seropositive, necrotizing ulcerative periodontitis. *J Periodontal Res*. 2003;38(2):147–155.
26. Armitage GC. Development of a classification system for periodontal diseases and conditions. *Northwest Dent*. 2000;79(6):31–35.
27. Gher ME. Changing concepts. The effects of occlusion on periodontitis. *Dent Clin North Am*. 1998;42(2):285–299.
28. Serio FG, Hawley CE. Periodontal trauma and mobility. Diagnosis and treatment planning. *Dent Clin North Am*. 1999;43(1):37–44.
29. Polson AM, Meitner SW, Zander HA. Trauma and progression of marginal periodontitis in squirrel monkeys. IV Reversibility of bone loss due to trauma alone and trauma superimposed upon periodontitis. *J Periodontal Res*. 1976;11(5):290–298.
30. Schenkein HA. Inheritance as a determinant of susceptibility for periodontitis. *J Dent Educ*. 1998;62(10):840–851.
31. Grbic JT, Lamster IB, Celenti RS, et al. Risk indicators for future clinical attachment loss in adult periodontitis. Patient variables. *J Periodontol*. 1991;62(5):322–329.
32. Loe H, Anerud A, Boysen H, et al. Natural history of periodontal disease in man. Rapid, moderate and no loss of attachment in Sri Lankan laborers 14 to 46 years of age. *J Clin Periodontol*. 1986;13(5):431–445.
33. Grossi SG, Zambon JJ, Ho AW, et al. Assessment of risk for periodontal disease. I. Risk indicators for attachment loss. *J Periodontol*. 1994;65(3):260–267.
34. Grossi SG, Genco RJ, Machtei EE, et al. Assessment of risk for periodontal disease. II. Risk indicators for alveolar bone loss. *J Periodontol*. 1995;66(1):23–29.
35. Genco RJ, Loe H. The role of systemic conditions and disorders in periodontal disease. *Periodontol*. 2000;1993(2):98–116.
36. Page RC, Beck JD. Risk assessment for periodontal diseases. *Int Dent J*. 1997;47(2):61–87.
37. Offenbacher S, Collins JG, Arnold RR. New Clinical diagnostic strategies based on pathogenesis of disease. *J Periodontal Res*. 1993;28(6 Pt 2):523–535.
38. Slutzkey S, Levin L. Gingival recession in young adults: occurrence, severity, and relationship to past orthodontic treatment and oral piercing. *Am J Orthod Dentofacial Orthop*. 2008;134(5):652–656.
39. Renkema AM, Fudalej PS, Renkema A, et al. Development of labial gingival recessions in orthodontically treated patients. *Am J Orthod Dentofacial Orthop*. 2013;143(2):206–212.
40. Renkema AM, Fudalej PS, Renkema AA, et al. Gingival labial recessions in orthodontically treated and untreated individuals: a case-control study. *J Clin Periodontol*. 2013;40(6):631–637.
41. Olsson M, Lindhe J. Periodontal characteristics in individuals with varying form of the upper central incisors. *J Clin Periodontol*. 1991;18(1):78–82.

42. Kois JC. Predictable single-tooth peri-implant esthetics: five diagnostic keys. *Compend Contin Educ Dent*. 2004;25(11):895–896, 898, 900 passim; quiz 906–897.
43. Kan JY, Rungcharassaeng K, Morimoto T, et al. Facial gingival tissue stability after connective tissue graft with single immediate tooth replacement in the esthetic zone: consecutive case report. *J Oral Maxillofac Surg*. 2009;67(11 suppl):40–48.
44. Kan JY, Rungcharassaeng K. Site development for anterior single implant esthetics: the dentulous site. *Compend Contin Educ Dent*. 2001;22(3): 221–226, 228, 230–221; quiz 232.
45. Evans CD, Chen ST. Esthetic outcomes of immediate implant placements. *Clin Oral Implants Res*. 2008;19(1):73–80.
46. De Rouck T, Eghbali R, Collys K, et al. The gingival biotype revisited: transparency of the periodontal probe through the gingival margin as a method to discriminate thin from thick gingiva. *J Clin Periodontol*. 2009;36(5):428–433.
47. Melsen B, Allais D. Factors of importance for the development of dehiscences during labial movement of mandibular incisors: a retrospective study of adult orthodontic patients. *Am J Orthod Dentofacial Orthop*. 2005;127(5):552–561; quiz 625.
48. Kao RT, Fagan MC, Conte GJ. Thick vs. thin gingival biotypes: a key determinant in treatment planning for dental implants. *J Calif Dent Assoc*. 2008;36(3):193–198.
49. Weisgold AS. Contours of the full crown restoration. *Alpha Omegan*. 1977;70(3):77–89.
50. Seibert J, Lindhe J. Esthetics and periodontal therapy. In: Lindhe J, ed. *Textbook of Clinical Periodontology*. 2nd ed. Copenhagen: Munksgaard; 1989:477–514.
51. Ochsenbein C, Ross S. A reevaluation of osseous surgery. *Dent Clin North Am*. 1969;13(1):87–102.
52. Kan JY, Morimoto T, Rungcharassaeng K, et al. Gingival biotype assessment in the esthetic zone: visual versus direct measurement. *Int J Periodontics Restorative Dent*. 2010;30(3):237–243.
53. Claffey N, Shanley D. Relationship of gingival thickness and bleeding to loss of probing attachment in shallow sites following nonsurgical periodontal therapy. *J Clin Periodontol*. 1986;13(7):654–657.
54. Goaslind GD, Robertson PB, Mahan CJ, et al. Thickness of facial gingiva. *J Periodontol*. 1977;48(12):768–771.
55. Kydd WL, Daly CH, Wheeler 3rd JB. The thickness measurement of masticatory mucosa in vivo. *Int Dent J*. 1971;21(4):430–441.
56. Leung CC, Palomo L, Griffith R, et al. Accuracy and reliability of cone-beam computed tomography for measuring alveolar bone height and detecting bony dehiscences and fenestrations. *Am J Orthod Dentofacial Orthop*. 2010;137(4 Suppl):S109–S119.
57. Molen AD. Considerations in the use of cone-beam computed tomography for buccal bone measurements. *Am J Orthod Dentofacial Orthop*. 2010;137(4 Suppl):S130–S135.
58. Cook DR, Mealey BL, Verrett RG, et al. Relationship between clinical periodontal biotype and labial plate thickness: an in vivo study. *Int J Periodontics Restorative Dent*. 2011;31(4):345–354.
59. Daly CH, Wheeler 3rd JB. The use of ultra-sonic thickness measurement in the clinical evaluation of the oral soft tissues. *Int Dent J*. 1971;21(4):418–429.
60. Wennstrom JL, Lindhe J, Sinclair F, et al. Some periodontal tissue reactions to orthodontic tooth movement in monkeys. *J Clin Periodontol*. 1987;14(3):121–129.
61. Lindhe J, Wennström JL, Berglundh T. The mucosa at teeth and implants. In: Lindhe J, Lang NP, Karring T, eds. *Clinical Periodontology and Implant Dentistry*. 5th ed. Blackwell: Munksgaard; 2008:69–85.
62. Anderegg CR, Metzler DG, Nicoll BK. Gingiva thickness in guided tissue regeneration and associated recession at facial furcation defects. *J Periodontol*. 1995;66(5):397–402.
63. Koke U, Sander C, Heinecke A, et al. A possible influence of gingival dimensions on attachment loss and gingival recession following placement of artificial crowns. *Int J Periodontics Restorative Dent*. 2003;23(5):439–445.
64. Vanarsdall R, Herberger T. *Rapid Palatal Expansion: Long-Term Stability and Periodontal Implications*. Thesis. Philadelphia: University of Pennsylvania; 1987.
65. Anzilotti C, Vanarsdall R, Balakrishnan M. *Expansion and Evaluation of Post-Retention Gingival Recession* Thesis. Philadelphia: University of Pennsylvania; 2002.
66. Korn EL, Baumrind S. Transverse development of the human jaws between the ages of 8.5 and 15.5 years, studied longitudinally with use of implants. *J Dent Res*. 1990;69(6):1298–1306.
67. Bjork A, Skieller V. Growth of the maxilla in three dimensions as revealed radiographically by the implant method. *Br J Orthod*. 1977;4(2):53–64.
68. Riedel RA, Brandt S. Dr. Richard A. Riedel on retention and relapse. *J Clin Orthod*. 1976;10(6):454–472.
69. Krebs A. Midpalatal suture expansion studies by the implant method over a seven-year period. *Rep Congr Eur Orthod Soc*. 1964;40:131–142.
70. Lindhe J. *Clinical Periodontology*. 2nd ed. Copenhagen: Munksgaard; 1989.
71. Garib DG, Henriques JF, Janson G, et al. Periodontal effects of rapid maxillary expansion with tooth-tissue-borne and tooth-borne expanders: a computed tomography evaluation. *Am J Orthod Dentofacial Orthop*. 2006;129(6):749–758.
72. Blasi I, Vanarsdall RL. *Rapid Maxillary Expansion with Skeletal Anchorage vs Bonded Tooth/Tissue Born Expanders. A Comparison Utilizing CBCT*. Thesis. Philadelphia: University of Pennsylvania; 2012.
73. Christie KF, Boucher N, Chung CH. Effects of bonded rapid palatal expansion on the transverse dimensions of the maxilla: a cone-beam computed tomography study. *Am J Orthod Dentofacial Orthop*. 2010; 137(4 Suppl):S79–S85.
74. Lagravere MO, Carey J, Heo G, et al. Transverse, vertical, and anteroposterior changes from bone-anchored maxillary expansion vs traditional rapid maxillary expansion: a randomized clinical trial. *Am J Orthod Dentofacial Orthop*. 2010;137(3):304, e301–312; discussion 304–305.
75. Lee HK, Bayome M, Ahn CS, et al. Stress distribution and displacement by different bone-borne palatal expanders with micro-implants: a three-dimensional finite-element analysis. *Eur J Orthod*. 2014;36(5):531–540.
76. Lin L, Ahn HW, Kim SJ, et al. Tooth-borne vs bone-borne rapid maxillary expanders in late adolescence. *Angle Orthod*. 2015;85(2):253–262.
77. Reitan K. The reaction as related to age factor. *Dent Rec*. 1954;74:271.
77a. Vanarsdall RL, Blasi I, Evans M, Kocian P. Rapid Maxillary Expansion with Skeletal Anchorage Vs. Bonded Tooth/Tissue Born Expanders: A Case Report Comparison Utilizing CBCT. *RMO Clinical Review*. 2012;1(1):18–22.
78. Villa MP, Rizzoli A, Miano S, et al. Efficacy of rapid maxillary expansion in children with obstructive sleep apnea syndrome: 36 months of follow-up. *Sleep Breath*. 2011;15(2):179–184.
79. Wriedt S, Kunkel M, Zentner A, et al. Surgically assisted rapid palatal expansion. an acoustic rhinometric, morphometric and sonographic investigation. *J Orofac Orthop*. 2001;62(2):107–115.
80. Secchi AG, Wadenya R. Early orthodontic diagnosis and correction of transverse skeletal problems. *N Y State Dent J*. 2009;75(1):47–50.
81. Vanarsdall Jr RL, Secchi AG, Chung CH, et al. Mandibular basal structure response to lip bumper treatment in the transverse dimension. *Angle Orthod*. 2004;74(4):473–479.
82. Pedetta F. *Correlation Between Maxillay Pa Ceph and Intraoral Molar Width Measurements* Thesis. Philadelphia: University of Pennsylvania; 1993.
83. Betts NJ, Lisenby WC. Normal adult transverse jaw values obtained using standardized posteroanterior cephalometrics [abstract]. *J Dent Res*. 1994;73:298.
84. Ricketts RM. *Introducing Computerized Cephalometrics*. Denver: Rocky Mountain Data Systems, Inc, March, 1969.
85. Brown IS. The effect of orthodontic therapy on certain types of periodontal defects. I. Clinical findings. *J Periodontol*. 1973;44(12):742–756.
86. Wise RJ, Kramer GM. Predetermination of osseous changes associated with uprighting tipped molars by probing. *Int J Periodontics Restorative Dent*. 1983;3(1):68–81.
87. Waerhaug J. The infrabony pocket and its relationship to trauma from occlusion and subgingival plaque. *J Periodontol*. 1979;50(7):355–365.
88. Lindhe J, Svanberg G. Influence of trauma from occlusion on progression of experimental periodontitis in the beagle dog. *J Clin Periodontol*. 1974;1(1):3–14.
89. Rygh P. The periodontal ligament under stress. In: Norton LA, Burtone CJ, eds. *The Biology of Tooth Movement*. Boca Raton, FL: CRC Press; 1989, pp. 9–28.

90. van Venrooy JR, Yukna RA. Orthodontic extrusion of single-rooted teeth affected with advanced periodontal disease. *Am J Orthod*. 1985;87(1):67–74.
91. Ingber JS. Forced eruption. I. A method of treating isolated one and two wall infrabony osseous defects: rationale and case report. *J Periodontol*. 1974;45(4):199–206.
92. Van Venrooy JR, Vanarsdall RL. Tooth eruption: correlation of histologic and radiographic findings in the animal model with clinical and radiographic findings in humans. *Int J Adult Orthodon Orthognath Surg*. 1987;2(4):235–247.
93. Vanarsdall R, Hamlin J. *The Effects of Molar Uprighting on the Microbiological Flora of the Periodontium*. Thesis. Philadelphia: University of Pennsylvania; 1987.
94. Morenzi V. *Assessment of the Effects of Orthodontic Molar Uprighting on the Microflora of Human Adult Periodontal Tissues Using Recombinant DNA Technology*. Thesis. Philadelphia: University of Pennsylvania; 1989.
95. Salama H, Salama M. The role of orthodontic extrusive remodeling in the enhancement of soft and hard tissue profiles prior to implant placement: a systematic approach to the management of extraction site defects. *Int J Periodontics Restorative Dent*. 1993;13(4):312–333.
96. Melsen B, Agerbaek N, Markenstam G. Intrusion of incisors in adult patients with marginal bone loss. *Am J Orthod Dentofacial Orthop*. 1989;96(3):232–241.
97. Melsen B. Tissue reaction following application of extrusive and intrusive forces to teeth in adult monkeys. *Am J Orthod*. 1986;89(6):469–475.
98. Vanarsdall RL. [Reaction of the periodontal tissues to orthodontic movement]. *Orthod Fr*. 1986;57(Pt 2):421–433.
99. Stenvik A, Mjor IA. Pulp and dentine reactions to experimental tooth intrusion. A histologic study of the initial changes. *Am J Orthod*. 1970;57(4):370–385.
100. Marks MH, Corn H. *Atlas of Adult Orthodontics: Functional and Esthetic Enhancement*. Philadelphia: Lea & Febiger; 1989.
101. Ericsson I, Thilander B, Lindhe J, et al. The effect of orthodontic tilting movements on the periodontal tissues of infected and non-infected dentitions in dogs. *J Clin Periodontol*. 1977;4(4):278–293.
102. Geraci TF. Orthodontic movement of teeth into artificially produced infrabony defects in the rhesus monkey. A histological report. *J Periodontol*. 1973;44:116.
103. Polson A, Caton J, Polson AP, et al. Periodontal response after tooth movement into intrabony defects. *J Periodontol*. 1984;55(4):197–202.
104. Wennstrom JL, Stokland BL, Nyman S, et al. Periodontal tissue response to orthodontic movement of teeth with infrabony pockets. *Am J Orthod Dentofacial Orthop*. 1993;103(4):313–319.
105. Stepovich ML. A clinical study on closing edentulous spaces in the mandible. *Angle Orthod*. 1979;49(4):227–233.
106. Wennstrom J, Lindhe J. Plaque-induced gingival inflammation in the absence of attached gingiva in dogs. *J Clin Periodontol*. 1983;10(3):266–276.
107. Wennstrom J, Lindhe J. Role of attached gingiva for maintenance of periodontal health. healing following excisional and grafting procedures in dogs. *J Clin Periodontol*. 1983;10(2):206–221.
108. Steiner GG, Pearson JK, Ainamo J. Changes of the marginal periodontium as a result of labial tooth movement in monkeys. *J Periodontol*. 1981;52(6):314–320.
109. Geiger A. Gingival response to orthodontic treatment. In: McNamara JA, Ribbens KA, eds. *Malocclusion and the Periodontium*. Monograph 15, Craniofacial Growth Series. Ann Arbor, MI: Center for Human Growth and Development, The University of Michigan; 1984.
110. Vanarsdall R. Periodontal considerations in corrective orthodontics. In: Clark JW, ed. *Clinical Dentistry*. Vol. 2. Hagerstown, MD: Harper & Row; 1978.
111. Edwards JG. A surgical procedure to eliminate rotational relapse. *Am J Orthod*. 1970;57(1):35–46.
112. Ahrens DG, Shapira Y, Kuftinec MM. An approach to rotational relapse. *Am J Orthod*. 1981;80(1):83–91.
113. Jacobson L. Mouthbreathing and gingivitis. 1. Gingival conditions in children with epipharyngeal adenoids. *J Periodontal Res*. 1973;8(5):269–277.
114. Vermette ME, Kokich VG, Kennedy DB. Uncovering labially impacted teeth: apically positioned flap and closed-eruption techniques. *Angle Orthod*. 1995;65(1):23–32; discussion 33.
115. Kim S-J, Vanarsdall RL, Polson A, et al. Periodontal outcomes on unerupted maxillary canines after apically positioned flaps [abstract]. *J Dent Res*. 2007:85.
116. Vanarsdall RL, Corn H. Soft-tissue management of labially positioned unerupted teeth. *Am J Orthod*. 1977;72(1):53–64.
117. Vanarsdall RL. Periodontal problems associated with orthodontic treatment. In: H B, ed. *Orthodontics: The State of the Art*. Philadelphia: University of Pennsylvania Press; 1981.
118. Biederman W. The ankylosed tooth. *Dent Clin North Am*. 1964;July:493–508.
119. Biederman W. The problem of the ankylosed tooth. *Dent Clin North Am*. 1968:409–424.
120. Kronfeld R. Bony union (ankylosis) of roots and alveolus. In: *Histopathology of the Teeth and Their Surrounding Structures*. 2nd ed. Philadelphia: Lea & Febiger; 1939:284–285.
121. Noyes FB. "Submerging" deciduous molars. *Angle Orthod*. 1932;(2):77.
122. Vanarsdall RL. *Periodontal Problems Associated with Orthodontic Treatment. Annual Conference on Orthodontics*. Philadelphia: University of Pennsylvania; May 1978.
123. Vanarsdall RL. *Management of ankylosed teeth. Special Interest Clinics, Annual Session of the American Association of Orthodontists*; May 5, 1979. Washington, D.C.
124. Leslie SW. A new method of treating unerupted teeth by means of surgery and orthodontia. *J Can Dent Assoc*. 1942;8:364.
125. Goodsell JF. Surgical exposure and orthodontic guidance of the impacted tooth. *Dent Clin North Am*. 1979;23(3):385–392.
126. Alling 3rd CC. Impacted canine teeth. *Dent Clin North Am*. 1979;23(3):439–450.
127. Buchanan G. Surgical intervention as an aid to orthodontics. *Ann Aust Coll Dent Surg*. 1967:70–77.
128. Line SE, Polson AM, Zander HA. Relationship between periodontal injury, selective cell repopulation and ankylosis. *J Periodontol*. 1974;45(10):725–730.
129. Melcher AH. Repair of wounds in the periodontium of the rat. Influence of periodontal ligament on osteogenesis. *Arch Oral Biol*. 1970;15(12):1183–1204.
130. Karring T, Nyman S, Lindhe J, et al. Potentials for root resorption during periodontal wound healing. *J Clin Periodontol*. 1984;11(1):41–52.
131. Vanarsdall RL. Efficient management of unerupted teeth: a time-tested treatment modality. *Semin Orthod*. 2010;16(3):212–221.
132. Handelman CS. The anterior alveolus: its importance in limiting orthodontic treatment and its influence on the occurrence of iatrogenic sequelae. *Angle Orthod*. 1996;66(2):95–109; discussion 109–110.
133. Elliott JR, Bowers GM. Alveolar dehiscence and fenestrations. *Periodontics*. 1963;1:245–248.
134. Edel A. Alveolar bone fenestrations and dehiscences in dry Bedouin jaws. *J Clin Periodontol*. 1981;8(6):491–499.
135. Abdelmalek RG, Bissada NF. Incidence and distribution of alveolar bony dehiscence and fenestration in dry human Egyptian jaws. *J Periodontol*. 1973;44(9):586–588.
136. Rupprecht RD, Horning GM, Nicoll BK, et al. Prevalence of dehiscences and fenestrations in modern American skulls. *J Periodontol*. 2001;72(6):722–729.
137. Swasty D, Lee JS, Huang JC, et al. Anthropometric analysis of the human mandibular cortical bone as assessed by cone-beam computed tomography. *J Oral Maxillofac Surg*. 2009;67(3):491–500.
138. Huynh-Ba G, Pjetursson BE, Sanz M, et al. Analysis of the socket bone wall dimensions in the upper maxilla in relation to immediate implant placement. *Clin Oral Implants Res*. 2010;21(1):37–42.
139. Richman C. Is gingival recession a consequence of an orthodontic tooth size and/or tooth position discrepancy? "A paradigm shift." *Compend Contin Educ Dent*. 2011;32(4):e73–79.
140. Siriwat PP, Jarabak JR. Malocclusion and facial morphology: is there a relationship? An epidemiologic study. *Angle Orthod*. 1985;55(2):127–138.
141. Wehrbein H, Bauer W, Diedrich P. Mandibular incisors, alveolar bone, and symphysis after orthodontic treatment. a retrospective study. *Am J Orthod Dentofacial Orthop*. 1996;110(3):239–246.
142. Fuhrmann R. Three-dimensional interpretation of periodontal lesions and remodeling during orthodontic treatment. *Part III. J Orofac Orthop*. 1996;57(4):224–237.

143. Evangelista K, Vasconcelos Kde F, Bumann A, et al. Dehiscence and fenestration in patients with Class I and Class II Division 1 malocclusion assessed with cone-beam computed tomography. *Am J Orthod Dentofacial Orthop*. 2010;138(2):133, e131–137; discussion 133–135.
144. Guilford SH. *Orthodontia or Malposition of the Human Teeth; Its Prevention and Remedy*. Philadelphia: Press of T.C. Davis and Sons; 1898.
145. Kole H. Surgical operations on the alveolar ridge to correct occlusal abnormalities. *Oral Surg Oral Med Oral Pathol*. 1959;12(5):515–529. concl.
146. Duker J. Experimental animal research into segmental alveolar movement after corticotomy. *J Maxillofac Surg*. 1975;3(2):81–84.
147. Suya H. Corticotomy in orthodontics. In: Hosl E, Baldauf A, eds. *Mechanical and Biological Basis in Orthodontic Therapy*. Heidelberg: Huthig Buch Verlag; 1991:207–226.
148. Wilcko WM, Wilcko T, Bouquot JE, et al. Rapid orthodontics with alveolar reshaping: two case reports of decrowding. *Int J Periodontics Restorative Dent*. 2001;21(1):9–19.
149. Frost HM. The biology of fracture healing. An overview for clinicians. Part I. *Clin Orthop Relat Res*. 1989;248:283–293.
150. Frost HM. The biology of fracture healing. an overview for clinicians. Part II. *Clin Orthop Relat Res*. 1989;248:294–309.
151. Frost HM. The regional acceleratory phenomenon: a review. *Henry Ford Hosp Med J*. 1983;31(1):3–9.
152. Schilling T, Muller M, Minne HW, et al. Influence of inflammation-mediated osteopenia on the regional acceleratory phenomenon and the systemic acceleratory phenomenon during healing of a bone defect in the rat. *Calcif Tissue Int*. 1998;63(2):160–166.
153. Generson RM, Porter JM, Zell A, et al. Combined surgical and orthodontic management of anterior open bite using corticotomy. *J Oral Surg*. 1978;36(3):216–219.
154. Teixeira CC, Khoo E, Tran J, et al. Cytokine expression and accelerated tooth movement. *J Dent Res*. 2010;89(10):1135–1141.
155. Yoshimatsu M, Shibata Y, Kitaura H, et al. Experimental model of tooth movement by orthodontic force in mice and its application to tumor necrosis factor receptor-deficient mice. *J Bone Miner Metab*. 2006;24(1):20–27.
156. Arias OR, Marquez-Orozco MC. Aspirin, acetaminophen, and ibuprofen: their effects on orthodontic tooth movement. *Am J Orthod Dentofacial Orthop*. 2006;130(3):364–370.
157. Hoogeveen EJ, Jansma J, Ren Y. Surgically facilitated orthodontic treatment: a systematic review. *Am J Orthod Dentofacial Orthop*. 2014;145(4 Suppl):S51–S64.
158. Wilcko WM, Wilcko MT, Bouquot JE, et al. Accelerated orthodontics with alveolar reshaping. *J Orthod Pract*. 2000;10:63–70.
159. Murphy KG, Wilcko MT, Wilcko WM, et al. Periodontal accelerated osteogenic orthodontics: a description of the surgical technique. *J Oral Maxillofac Surg*. 2009;67(10):2160–2166.
160. Wilcko MT, Wilcko WM, Pulver JJ, et al. Accelerated osteogenic orthodontics technique: a 1-stage surgically facilitated rapid orthodontic technique with alveolar augmentation. *J Oral Maxillofac Surg*. 2009;67(10):2149–2159.
161. Amit G, Jps K, Pankaj B, et al. Periodontally accelerated osteogenic orthodontics (PAOO): a review. *J Clin Exp Dent*. 2012;4(5):e292–296.
162. Makki L, Ferguson DJ, Wilcko MT, et al. Mandibular irregularity index stability following alveolar corticotomy and grafting: a 10-year preliminary study: mandibular irregularity index stability. *Angle Orthod*. 2015;85(5):743–749.
163. Rothe LE, Bollen AM, Little RM, et al. Trabecular and cortical bone as risk factors for orthodontic relapse. *Am J Orthod Dentofacial Orthop*. 2006;130(4):476–484.
164. Ferguson DJ, Makki L, Stapelberg R, et al. Stability of the mandibular dental arch following periodontally accelerated osteogenic orthodontics therapy: preliminary studies. *Semin Orthod*. 2014;20(3):239–246.
165. Choi HJ, Lee DY, Kim TW. Dynamics of alloplastic bone grafts on an early stage of corticotomy-facilitated orthodontic tooth movement in beagle dogs. *Biomed Res Int*. 2014;2014:417541.
166. Araujo MG, Carmagnola D, Berglundh T, et al. Orthodontic movement in bone defects augmented with bio-oss. An experimental study in dogs. *J Clin Periodontol*. 2001;28(1):73–80.
167. Wilcko MW, Ferguson DJ, Bouquot JE, et al. Rapid orthodontic decrowding with alveolar augmentation: case report. *World J Orthod*. 2003;4:197–205.
168. Wilcko MT, Wilko WM, Bissada NF. An evidence-based analysis of periodontally accelerated orthodontic and osteogenic techniques: a synthesis of scientific perspective. *Semin Orthod*. 2008;14:305–316.
169. Lee KB, Lee DY, Ahn HW, et al. Tooth movement out of the bony wall using augmented corticotomy with nonautogenous graft materials for bone regeneration. *Biomed Res Int*. 2014;2014:347508.
170. Alghamdi AS. Corticotomy facilitated orthodontics: review of a technique. *Saudi Dent J*. 2010;22(1):1–5.
171. Köle H. Corticalisschwächung Zur Unterstützung Bei Der Kieferorthopädischen Behandlung Fortschr. *Kiefer- u. Gesichtschir*. 1958;4:208.
172. Kim SH, Kim I, Jeong DM, et al. Corticotomy-assisted decompensation for augmentation of the mandibular anterior ridge. *Am J Orthod Dentofacial Orthop*. 2011;140(5):720–731.

23

Orthodontic Aspects of Orthognathic Surgery

David R. Musich and Peter Chemello

OUTLINE

Background, 669
Goals, 669
Need Versus Demand, 672
Orthodontic Retreatment and Skeletal Malocclusions, 673
Technologic Advances in Orthognathic Surgery, 676
Medical Advances, 676
Surgical Advances, 677
Skeletal Fixation, 684
Patient Treatment, 691
Diagnostic Considerations, 691
Diagnosing Class II Mandibular Deficiency and Class III Mandibular Excess, 695
Treatment Planning Options, 695
Indications for Camouflage Treatment, 703
Contraindications to Camouflage Treatment, 705
Upper and Lower Jaw Skeletal–Dental Problem, 705
Therapeutic Diagnosis of Borderline Cases, 705
Key Considerations of the Integrated Treatment Plan, 706
Treatment Timing, 708
Treatment Timing for Maxillary Deficiency Patients, 708
Treatment Timing for Mandibular Excess Patients, 708
Treatment Timing for Mandibular Deficiency Patients, 711
Considerations in the Management of Facial Asymmetry, 711
Sequence of Treatment for Orthognathic Patients, 711
Importance of the Sequence, 712
Step 1: Multiple Provider Team Selection (Interdisciplinary Therapy), 712
Step 2: Goal Clarification, 712
Steps 3 Through 16, 712
Step 17: Treatment Experience, 712
Applied Concepts for Specific Types of Cases, 712
Optimizing Stability, 713
Orthodontic Considerations, 714
Stability, 714
Surgical Considerations, 714
Risks of Orthodontic–Orthognathic Surgery, 720
Summary, 725

BACKGROUND

The intrinsic genetic factors and extrinsic epigenetic factors affecting facial development are extremely complex, often leading to dental and skeletal imbalances that require interdisciplinary treatment plans to achieve a functional balance. The functional matrix hypothesis of Moss describes the many complex elements of facial development that must integrate to create the final form and function of the stomatognathic system.[1–3]

The development of the greatest amount of a patient's stomatognathic system occurs during the first 14 to 18 years of life, and its development occurs in the transverse, vertical, and sagittal dimensions.[4] When there are growth imbalances in the early stages of facial growth and development, the orthodontist takes the lead, makes a differential diagnosis and treatment planning decision, and decides on an appropriate treatment plan; however, as the patient nears the end of his or her growth cycle and when there is evidence that the skeletal imbalance exceeds orthodontic or orthopedic capacity for correction, patient education and referral for surgical assessment become the next steps in the management of the patient's imbalanced masticatory system. This chapter focuses on current concepts of the teamwork necessary to diagnose and to treatment plan complex skeletal malocclusions.

Goals

Orthodontists and oral and maxillofacial surgeons must individualize the goals of treatment for each adult patient through an interview process that takes place as part of the history and clinical examination and is continued when the records are reviewed in the treatment planning conference. Although achieving functional balance of the intricate stomatognathic system is generally the primary goal of therapy, aesthetic improvement is a natural component of balancing the bones and dentition that make up the chewing system. When treating patients with complex imbalances, we strive to achieve **f**unctional balance using **r**eliable methods that achieve **r**ealistic, **a**esthetic outcomes that are **e**conomically feasible. In addition, the orthodontist's or surgeon's teamwork also strives to provide a **s**table, **s**atisfactory relationship that enhances the overall orofacial **h**ealth of the patient. The acronym FRESH (Box 23-1) has been useful as a guide to describe the major goals of orthodontic–orthognathic treatment to patients considering this option.[5–8]

Although treatment goals among orthodontists and surgeons are generally consistent, many differences exist in the specific meanings of terms such as *stability, facial balance, aesthetics,* and even *function*.[34] For example, does the term *stability* always

BOX 23-1 Orthodontic–Orthognathic Treatment Objectives Summarized by the Acronym FRESH

Function—The fundamental goal of improving occlusal relationship is paramount; it is desirable to establish optimal gnathologic parameters for the stomatognathic system (incisal guidance, canine rise, balanced, bilateral posterior occlusal support) and temporomandibular joint (TMJ) health and TMJ stability without symptoms.[9,10]

Reliable—Use of methods of treatment that have been scientifically verified to be highly successful in achieving correction of skeletal imbalances and that are of lower risks than other available procedures for the improvement of a given condition.[11–13] Recent examples of techniques that have proven their reliability are (1) rigid fixation approaches[14] (2) jaw surgery to resolve sleep apnea,[15,16] and (3) use of microscrews as anchorage to improve the range of nonsurgical treatment.[17]

Realistic—The plan of treatment has a high probability of correcting the patient's chief concern and offers the best option to achieve an outcome with a high probability of meeting both the patient's and doctors' expectations.[18–21]

Aesthetics—Issues of aesthetics relate to both dental and facial appearance; facial aesthetics requires assessment of both the profile and frontal view to achieve soft tissue balance as well optimal symmetry. The facial balance should be present both statically and dynamically in speech and during facial expression, especially when the patient is smiling. The role of the "aging process of the face" should also be integrated into the treatment planning process, especially when considering camouflage options.[8,22–24]

Economic—The cost of the proposed treatment does require consideration because most patients have a budget for their medical and dental needs. With limited insurance for orthodontic care and reducing coverage for orthognathic surgical procedures, financial considerations frequently interfere with the optimal combined orthognathic–orthodontic treatment plan.[25]

Stability—A key part of the treatment outcome is ending with jaw and tooth positions that are reasonably stable. There are several variables that require consideration to achieve a stable outcome and there are many adult orthodontic problems that require fixed retention to achieve stability, especially of the lower and upper incisors,[26,27] (also see Chapter 27).

Satisfaction—Achieving both patient and provider satisfaction is important. This usually occurs when there is open discussion, before treatment, about the goals that both the doctor and patient are striving to achieve in this individual situation. This includes a frank discussion from the doctor about the "down side" or "limits" of the accepted plan. This is particularly true in cases that have a skeletal diagnosis and are being considered for camouflage therapy when skeletal correction is more ideal. The role of visualized treatment objectives and computerized predictions can assist the patient and the providers.[17,28,29]

Health—Includes periodontally healthy tissues, Temporomandibular Joint (TMJ) health, comfort and function, and emotional health. With complicated interdisciplinary treatments, it is important that the Interdisciplinary Dentofacial Teams (IDT) teams work well together and are clear on the combined goals of therapy to provide optimal outcomes in these challenging cases. Treatment plans must always strive to preserve the healthy aspects of the case while improving aspects that are diseased or nonfunctional.[30–33]

mean "without continual retention"? Is long-term mechanical retention to create stability acceptable, as suggested by Zachrisson and Buyukilmaz (see Chapter 27)? Who is the judge of aesthetics: the orthodontist, the surgeon, the spouse or significant other, the parent, or the patient? Each of these important persons may differently judge the aesthetics of the profile, the smile, the tooth angulation, and the chin (see Chapters 9 and 10). In additional recent studies, Montini et al. found variation in perception of aesthetic improvement of profiles: "Data was gathered to see if Class II, mandibular deficient patients who had surgery had a more pleasing profile based on assessment by lay persons, orthodontists, and surgeons. Statistical results showed esthetic improvement was perceived for 13/14 silhouetted pairs of profiles. In some cases there were differences in perceived changes as judged by the different judges."[35]

In addition to this problem of a lack of uniform definition or understanding of the terms used for accepted treatment objectives, the clinician often must prioritize those objectives with the patient. For example, a patient may have a moderate chewing problem and incipient myofascial imbalance caused by a lack of incisal function and a mild skeletal open bite (mandibular deficiency with maxillary protrusion) (Fig. 23-1). The ideal treatment plan is orthodontic treatment and jaw surgery to achieve stability, function, and facial balance; a secondary plan may be the extraction of upper first premolars to close the bite. The functional goal is prioritized, but stability (extraction space rebound), vertical excess, and facial balance (change in the nasolabial angle) are compromised (deprioritized). Therefore, two prerequisites for achieving treatment goals and a successful outcome are:

1. A clear understanding of the patient's chief concerns and *the patient's* treatment goals, documented, clarified, and illustrated through simulation and diagnostic setup
2. A clear explanation by the orthodontist of the treatment goals that can be achieved

This is especially true for patients with skeletal jaw malrelationships.[18] In Chapter 8 of this text, Proffit and Nguyen properly describe the process that most clearly allows clarification of goals (see Figs. 8-7 and 8-8). The team, led by the orthodontist and the oral surgeon, carefully develops a problem list, which is reviewed with the patient; this provides an opportunity to discuss the treatment options for achieving the desired outcomes. The orthodontist informs the patient and family, and then all must agree on a plan that they believe will provide the best possible outcome with the least exposure to risk. The plan of action cannot be finalized until the other members of the provider team have contributed to the treatment plan and sequence. Careful integration of each team member's perspective helps to prevent last-minute surprises—for example, Which jaw is having the surgery? How much movement is needed? and Is two-jaw surgery recommended instead of one-jaw surgery? Such unexpected changes can undermine the outcome and the patient's trust.

Current treatment planning approaches include significant patient participation requiring more of the practitioner's time and, at a humanistic level, more emphasis on educating the patient and less emphasis on arbitrary directives—for example, "Go to the surgeon for jaw surgery." From a risk management perspective, the steps in Proffit's problem-oriented approach (see Figs. 8-7 and 8-8), if well documented, create a record that can assist the clinician if the patient subsequently initiates legal action. Machen[36] clarified that risk management is an important consideration when the orthodontist embarks on treatment using an alternative plan. As described here, the orthodontist started treatment with the plan of mandibular advancement for the Class II patient. During treatment, the patient decided against the recommended jaw surgery and proceeded to have camouflage therapy with the upper first premolars extracted. During protrusion reduction and extraction space closure, the patient developed temporomandibular joint (TMJ) symptoms and eventually sued the orthodontist.

FIGURE 23-1 A–C, Pretreatment intraoral photographs showing Class II malocclusion (10.5-mm overjet). D–F, Long-term postorthodontic intraoral photographs 12 years after treatment completion showing good stability of orthodontic correction. G, Pretreatment facial smile photograph. H, Cephalometric superimposition of pretreatment tracing and posttreatment tracing showing successful Class II camouflage treatment. I, Long-term postorthodontic facial smile photograph 12 years after treatment completion. J, Pretreatment lateral cephalogram radiograph. K, Bolton template–guided diagnosis showing maxillary skeletal and incisor protrusion and mandibular deficiency. Moderate skeletal imbalances in which both jaws contribute to dental protrusion are generally good candidates for camouflage treatment. L, Long-term postorthodontic lateral cephalogram radiograph 12 years after treatment completion.

In evaluating this case, several factors appear to have been deemed important by the jury in determining that the practitioner fulfilled his obligations to the patient. First, the practitioner performed a complete evaluation and suggested a course of treatment designed to correct the patient's problem. Second, an alternative plan was made available that was clearly identified as a compromise. Third, the potential risks and complications associated with the alternative treatment were presented to, discussed with, and acknowledged by the patient. And, finally, a paper trail existed confirming each of the above-mentioned steps.

Following the steps referenced in Figures 8-7 and 8-8 may seem time consuming, but doing so will prove invaluable because the technologic capabilities now available for changing the form and function of the stomatognathic system are profound and require judicious care in their use (Fig. 23-2). With orthognathic surgical insurance coverage unavailable in many situations, clarification of the treatment plan options with an explanation of the risks and benefits is more critical than ever.

NEED VERSUS DEMAND

Stomatognathic system imbalances are generally not life threatening, thus placing their need for treatment into a category of "elective." However, because imbalances in the stomatognathic system can have a major negative impact on one's masticatory function, muscle comfort, dental health, facial aesthetics, and self-esteem, proper treatment is important to each individual patient.

A recently published study from Finland found that there was a significant oral health impact among patients with severe malocclusions. Those with severe malocclusions perceived a significant oral impact "fairly often" or "very often," which was seven times higher than found among adult Finns in the National Health 2000 survey.[37] In addition, through a series of psychological tests, Frejman et al. found that adult patients with severe Class II or Class III skeletal imbalances had lower self-esteem

FIGURE 23-2 A, Presurgical profile photograph showing severe mandibular and chin deficiency. B, Postsurgical profile photograph showing correction of mandibular and chin deficiency. C, Superimposition of lateral cephalogram tracing (*solid lines* represent pretreatment appearance; *dashed lines* represent posttreatment appearance). D–F, Presurgical intraoral photographs showing severe malocclusion. G–I, Posttreatment intraoral photographs showing occlusal correction through orthodontics and jaw surgery.

compared with adults without the skeletal imbalances.[38] It is probable that the lower self-esteem is a motivating factor for patients to seek out a solution to their skeletal imbalance through orthognathic surgery. Johnston et al. assessed self-perception in 162 patients who required orthodontics and orthognathic surgery and compared their findings with a control group without skeletal imbalances.[39] With questionnaires that used a visual analog scale, they found that the Class II and Class III patients seeking orthognathic surgery were significantly more unhappy with their dental and facial appearance.

Because orthodontic–orthognathic treatments of skeletal dysplasias are in most cases elective procedures, further discussion of need and demand considerations is warranted. Proffit and White[40] helped to clarify this discussion with the following statement: "The indication for surgical-orthodontic treatment is that a skeletal or dentoalveolar deformity is so severe that the magnitude of the problem lies outside the envelope of possible correction by orthodontics alone." An arbitrary "envelope of discrepancy" (see Fig. 8-4) was developed to illustrate the concept of limitations of various treatment modalities.[40] To further develop this concept, Squire et al.[41] identified the following nonsurgical limits of certain malocclusion types. They found that the following were not considered treatable by the 28 orthodontists surveyed[41]:

1. Positive overjet greater than 8 mm
2. Negative overjet of –4 mm or more
3. Maxillary transverse discrepancy greater than 3 mm

Orthodontic Retreatment and Skeletal Malocclusions

The main problem with many orthodontic patients who required retreatment appeared to be significant differential growth that led to Class II open bites, maxillomandibular transverse discrepancies with posterior crossbites, or Class III type occlusal problems[42] (Box 23-2). As specialists, orthodontists know that these are difficult growth patterns to manage. Although many of these malocclusions can be corrected with orthopedics and good compliance, many cannot. Therefore, the orthodontist is obliged to diagnose in all three planes of space, to discuss therapeutic modifiability of skeletal imbalances openly, and to present the possibility of a surgical phase if compliance or the growth response is inadequate to achieve an optimal occlusal result with acceptable facial balance.[43] In many of these cases, cephalometric reassessment of therapeutic response is important.

It becomes critical in these cases to communicate more clearly about treatment progress, treatment outcome, and the risks of instability. To refine communication systems so that trained administrative and clinical staff can step into the "treatment coordinator" role at any patient appointment to discuss the various stages of treatment with the patient or parent is advantageous. The communication system should provide a standardized office protocol for the conference and report process with patients and parents and the family dentist, pediatric dentist, or periodontist. Within the recommended communication process are four conference reports that are recommended

BOX 23-2 Sequence of Treatment for Patients with Significant Dentofacial Imbalances

1. Multiple provider team selection
   - Orthodontist
   - Periodontist
   - Restorative dentist
   - Oral surgeon
   - Psychologist
2. Goal clarification for team members
   - Functional occlusion
   - Reliable methods
   - Stability
   - Health
   - Aesthetics
3. Clinical awareness of dentofacial deformity
   - Self, general dentist, friend
4. General assessment of patient
   - A good candidate?
   - Periodontal status
5. Evaluation of preliminary records by the dental team
   - Splint therapy for diagnosis of temporomandibular dysfunction, if needed
6. Completion of diagnostic records
   - May include lifestyle assessment by clinical psychologist
   - May require more sophisticated temporomandibular joint studies
7. Multidisciplinary review of dentofacial problem based on patient records
8. Explanation to patient of available treatment options
   - Optimal treatment plan
   - Alternatives
9. Consultation with the patient and significant other by dental team providers
   - Risk-to-benefit ratio
   - Fees and insurance coverage
   - Treatment time
   - Other concerns
10. Patient acceptance of treatment plan
11. Comprehensive orthodontic treatment (usually for 8–18 months before surgery)
    - Orthodontic movement to decompensate tooth positions
    - Coordination of arches in anticipation of surgical repositioning
    - Alignment of teeth and correction of rotations
12. Presurgical reevaluation records
    - Complete records
    - Prediction tracing (detailed movements planned)
    - Model surgery (reviewed by orthodontist and surgeon)
    - Determination of specific fixation needs
    - Patient–spouse review with orthodontist and surgeon
    - Reevaluation by psychologist
13. Orthognathic surgery
    - Simplest procedure or procedures to achieve professional goals and satisfy the patient's needs
14. Postsurgical period
    - Early: 2 to 3 weeks of fixation
    - Late: 3 to 8 weeks of fixation
    - Reevaluation of surgical procedure
    - Meeting between patient and psychologist for update and support
15. Evaluation of surgical stability
    - Orthodontic finishing, occlusal adjustments, and retention procedures
    - Optimal continuation of contact with psychologist
16. Posttreatment records
    - 1 year posttreatment
    - Reevaluation with dental team, especially oral surgeon and psychologist
17. Treatment experience (used in management of future cases)
    - Positive reinforcement
    - Problem solving
    - Reevaluation of degree of success in achieving goals
    - Treatment evaluations by patient and all providers

FIGURE 23-3 A–C, Patient at age 9 years, 11 months. Intraoral photographs before any orthodontic treatment. D–F, Patient at age 13 years, 1 month. Intraoral photographs after phase I expansion with Class III intraoral traction. G–I, Patient at age 14 years, 11 months. Intraoral photographs illustrating bite change secondary to adolescent growth spurt showing greater Class III jaw imbalance with increasing asymmetry to right. J–L, Patient at age 17 years, 4 months. Intraoral photographs after orthodontic arch preparation and maxillomandibular jaw surgery to resolve the maxillary deficiency and mandibular excess with asymmetry.

and serve as "milestones" in the treatment and communication process:

1. **Treatment planning conference report:** to finalize the treatment planning process and document treatment goals
2. **Progress conference report:** sent to family dentist with panoramic radiographs and any other appropriate growth evaluation records
3. **Stabilization or retention conference report:** to discuss treatment successes and treatment shortcomings and to highlight patients at high risk for needed additional treatment
4. **Treatment completion conference report:** to review treatment results and prepare patient for postretention requirements

This formal, predictable conference system provides information that patients, parents, and dentists appreciate. More important, the conference system is a mechanism that minimizes miscommunication, particularly in complicated cases that without proper documentation could lead to adverse legal action. This communication system is particularly relevant in skeletal imbalance cases in which the orthodontic plan may be "heroic" and only achievable with excellent growth response and excellent cooperation. This process also "conditions" the patient and parent to the possible need for retreatment or more advanced treatment, including braces and jaw surgery.[44]

The patient in Figure 23-3 illustrates a patient for whom orthodontic and orthopedic therapy was initially attempted, but her mandibular growth excess exceeded the capacity to orthopedically modify the maxilla's growth to create a balanced

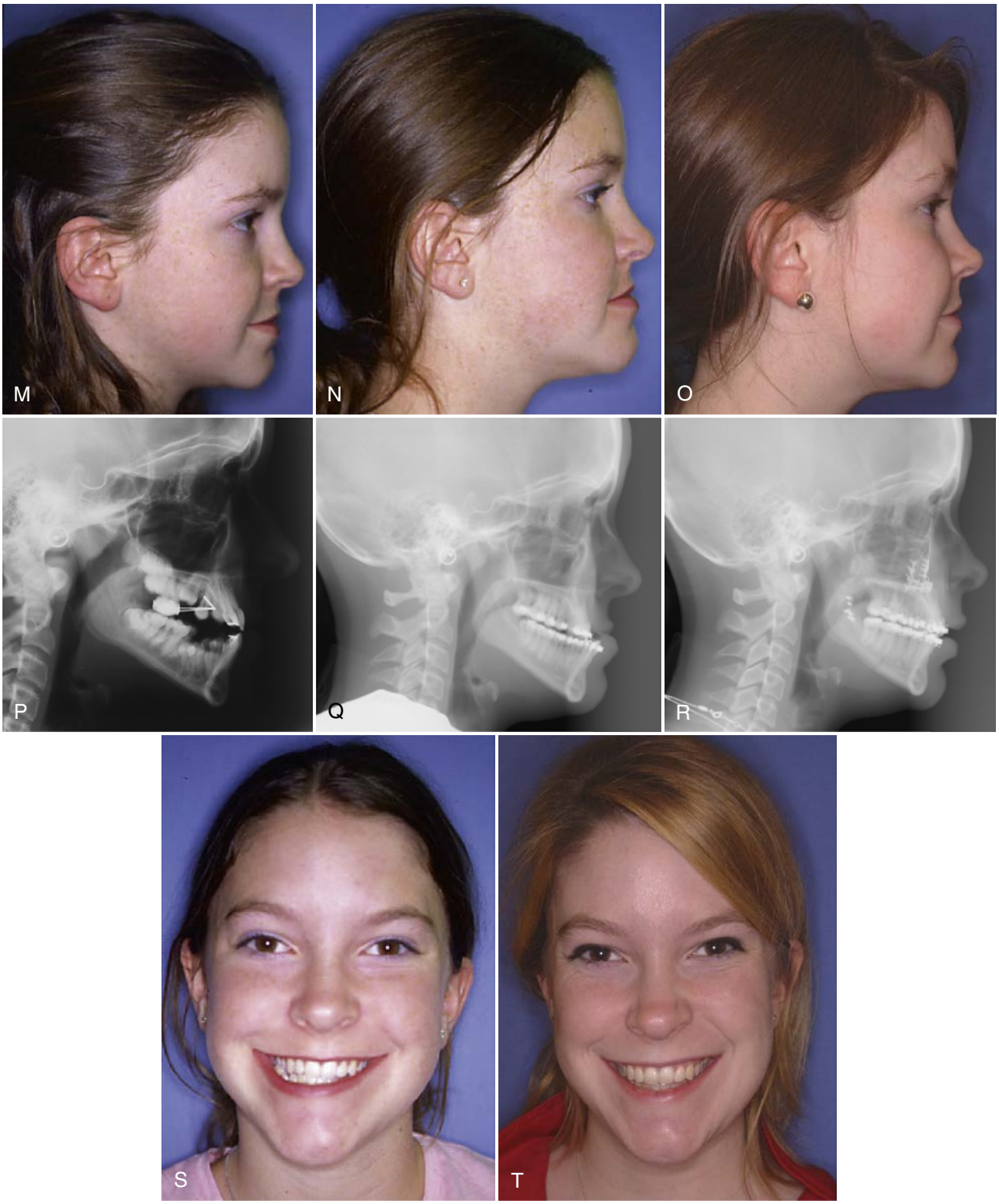


FIGURE 23-3, cont'd M and P, Profile facial view, lateral view of cephalogram at age 9 years, 1 month, respectively. N and Q, Profile view, lateral view of cephalogram at age 16 years, 1 month, respectively. O and R, Profile view, lateral view of cephalogram at age 17 years, 4 months, respectively. S, Frontal facial smile photograph at age 14 years, 8 months, before initiating presurgical orthodontic treatment. T, Frontal facial smile photograph at age 17 years, 4 months, after orthodontics and maxillomandibular surgery. (Surgery by Dr. Peter Chemello, Arlington Heights, IL.)

occlusion. The preferred treatment was then to wait for growth cessation and provide a surgical solution. The number of patients needing and demanding orthodontic–orthognathic correction is increasing with population increases and awareness (Tables 23-1 to 23-3). The following list identifies the categories of patients for whom orthodontists and oral and maxillofacial surgeons will continue to provide advanced management through the refined techniques of orthodontics and orthopedics and, in many cases, orthognathic surgery:

- Genetic growth imbalances beyond the orthodontic–orthopedic range of correction (see Tables 23-1 to 23-3)
- Facial trauma causing growth alterations

TABLE 23-1 Estimated Prevalence of Mandibular Deficiency Severe Enough to Indicate Surgery

	Percentage	Number
Prevalence of skeletal Class II malocclusion	10*	22.5
At appropriate age for surgical treatment	68	4,625,000
Severe enough to warrant surgery	5	731,250
Mandibular advancement	70	510,000
Maxillary setback	10	67,500
Both	20	145,000
New patients added to population yearly†	0.5	21,250

*Assuming a U.S. population of 325 million.
†Assuming 4.25 million live births per year.

TABLE 23-2 Estimated Prevalence of Class III Problems Severe Enough to Indicate Surgery

	Percentage	Number
Prevalence of skeletal Class III malocclusion	0.6*	1.35 million
At appropriate age for surgical treatment	65	877,500
Severe enough to warrant surgery	33	290,000
Mandibular setback	45	130,000
Maxillary advancement	35	101,000
Both	20	58,000
New patients added to population yearly†	0.2	8500

*Assuming a U.S. population of 325 million.
†Assuming 4.25 million live births per year.

TABLE 23-3 Estimated Prevalence of Long-Face Problems Severe Enough to Indicate Surgery

	Percentage	Number
Prevalence of severe anterior open bite	0.6*	1.35 million
At appropriate age for surgical treatment	65	877,500
Severe enough to warrant surgery (superior repositioning of the maxilla)	25	220,000
New patients added to the population yearly†	0.2	6375

*Assuming a U.S. population of 325 million.
†Assuming 4.25 million live births per year.

- Orthodontic retreatment problems that grew outside the range of conventional orthodontic correction
- Patients with sleep apnea disorders that require orthognathic procedures to completely resolve

TECHNOLOGIC ADVANCES IN ORTHOGNATHIC SURGERY

Advances in medical management and surgical techniques have improved the predictability of favorable outcomes and improved functional and aesthetic results for patients undergoing orthognathic surgery. This section presents a brief discussion of medical advances that includes anesthetic technique and edema, nausea, and infection control. Surgical advances are discussed and include surgical procedures and treatments as well as fixation techniques.

Medical Advances

Hypotensive anesthesia has been well documented to decrease blood loss during orthognathic surgery.[45] The technique is sufficiently flexible that a variety of medications can be used to achieve the desired effect. The determination of which medications are used is based on the patient's medical history and the anesthesiologist for the case.[46] Hypotensive anesthesia allows the surgeon to perform complicated orthognathic procedures in highly vascular areas with minimal blood loss.[47,48] This technique has reduced the need for blood transfusion, and the lower blood loss has been shown to be a significant factor for a decreased length of stay in the hospital.[49]

Edema control is essential to allow a quick return to normal function. Measures for controlling edema include (1) minimizing surgical trauma, (2) shortening operating time, (3) maintaining careful hemostasis, and (4) ensuring pharmacologic control of the inflammation process. Pharmacologic control is achieved primarily through the use of corticosteroids. Although corticosteroids can affect leukocyte function adversely, short-term use shows minimal negative effect. Commonly used corticosteroids include methylprednisolone and Decadron.

Nausea is the most common postoperative complication after orthognathic surgery and general anesthesia.[49] Nausea can lead to an increase in hospital stay as well as medical issues such as dehydration. Nausea can also lead to poor patient satisfaction. Silva et al.[50] found that the following factors are associated with an increased risk of postoperative nausea and vomiting:

1. Female gender
2. Young patients (15–25 years old)
3. Nonsmoking status
4. Presence of predisposing factors (prior history of nausea or vomiting or motion sickness, vertigo, migraine headaches)
5. Use of volatile general anesthetics
6. Maxillary surgery
7. Postoperative pain level in the postanesthesia care unit
8. Use of postoperative analgesic opioid drugs

Infection is an uncommon occurrence after orthognathic procedures.[51] The incidence of infection is decreased with antibiotic use preoperatively and postoperatively.[52] However, when infection does develop, it usually is a local inflammation that responds well to incision and drainage and systemic antibiotics. Prophylactic antibiotics are still used; commonly 1 million units of penicillin are administered intravenously just

before surgery and again every 6 hours for 2 days after surgery. Cefazolin (Kefzol), clindamycin, and amoxicillin–clavulanate can be used as alternatives to penicillin G.

Surgical Advances

Oral and maxillofacial surgeons have studied the effects of a variety of surgical techniques over the past 20 years. The following criteria generally are considered in determining which technique best serves the patient's needs:

- Safety of the technique
- Reduced morbidity (neurologic and vascular)
- Stability of the procedure
- Enhanced rate of healing
- Reduced surgical time under anesthesia

Figures 23-3 to 23-13 are schematic illustrations correlated with treated patients for some of the more common and considered conventional surgical procedures currently

Text continued on page 682

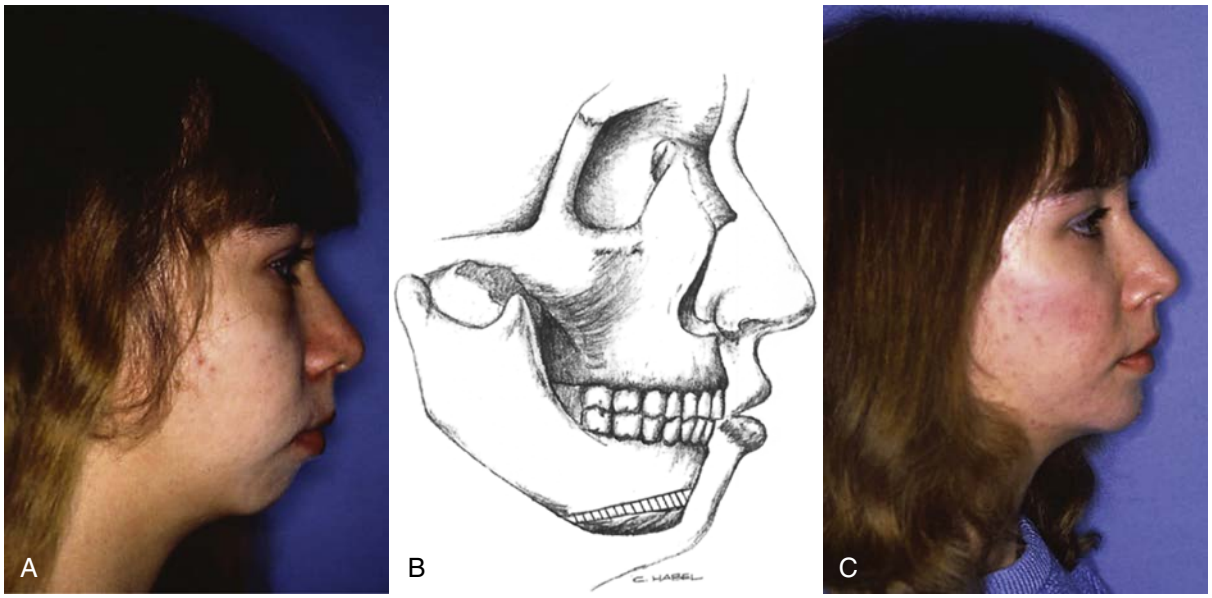


FIGURE 23-4 A, Horizontal deficiency and vertical chin excess before treatment. B, Schematic of surgical procedure: vertical reduction with anterior advancement of the chin. C, Orthodontic–surgical outcome: results after combination therapy (i.e., orthodontics and chin surgery). (Surgery by M. Steichen, Arlington Heights, IL.)

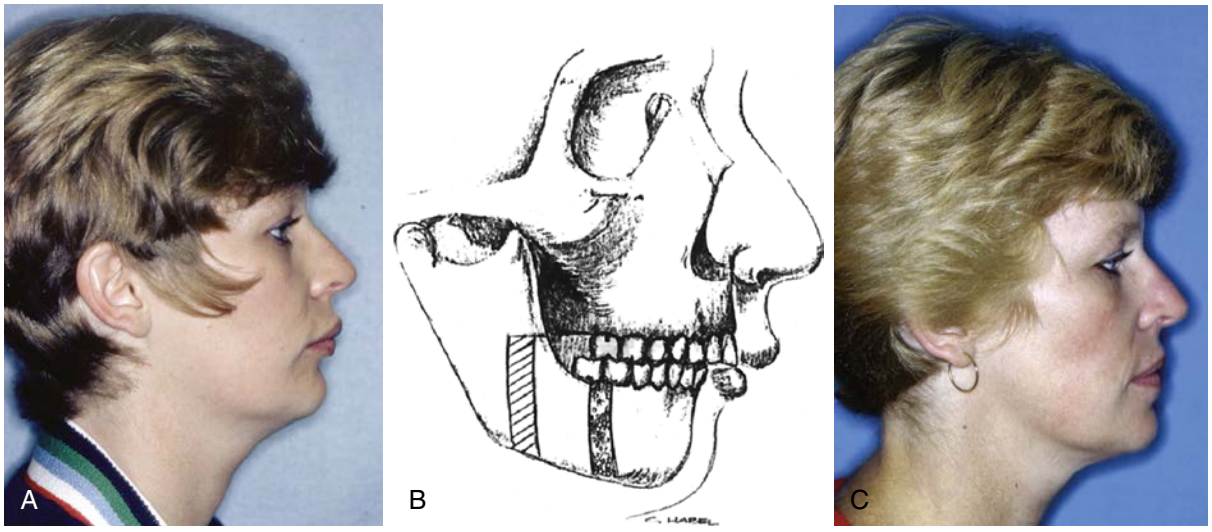


FIGURE 23-5 A, Class II mandibular deficiency before treatment. B, Schematic of surgical procedure: sagittal split osteotomy with advancement. C, Orthodontic–surgical outcome: results after combination therapy. (Surgery by A. Frer, Schaumburg, IL.)

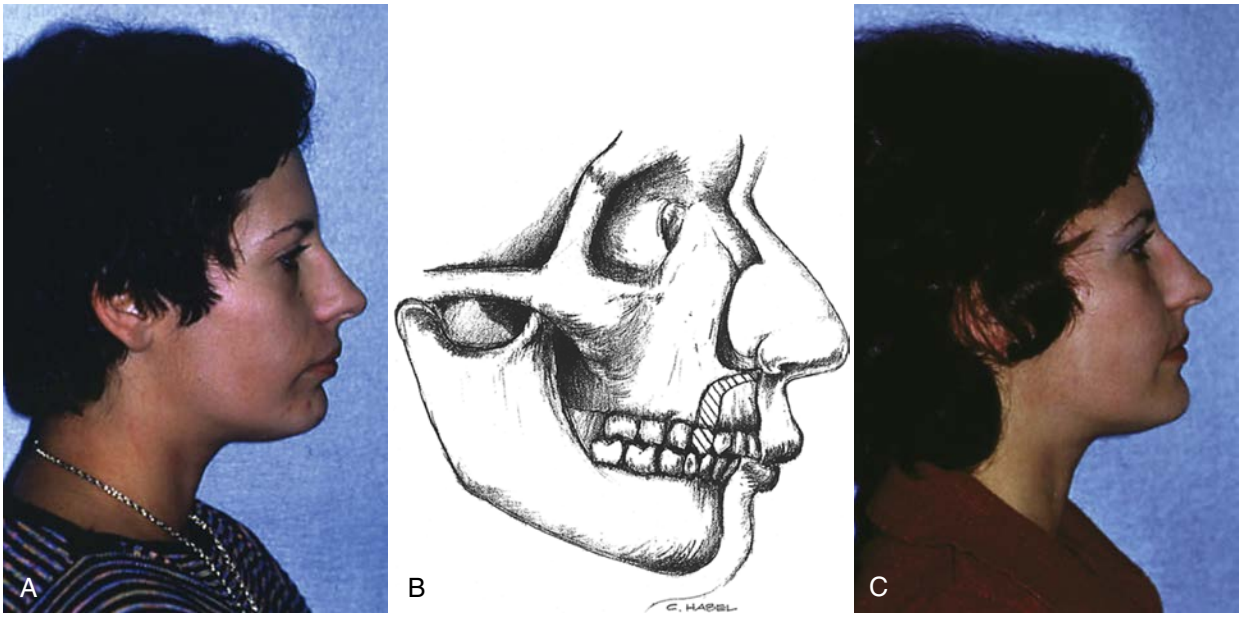


FIGURE 23-6 A, Class II maxillary protrusion before treatment. B, Schematic of surgical procedure: segmental setback of the maxillary anterior teeth. C, Orthodontic–surgical outcome: results after combination therapy. (Surgery by A. Frer, Schaumburg, IL.)

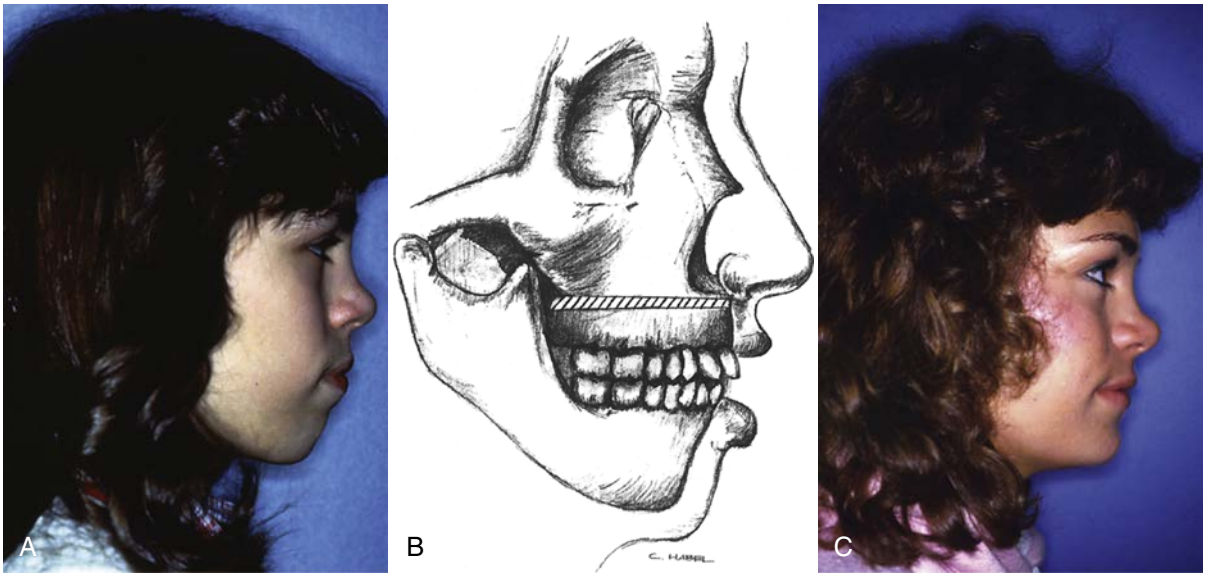


FIGURE 23-7 A, Excess vertical growth of the maxilla and down and back rotation of the mandible before treatment. The Le Fort osteotomy has a number of modifications, has proved to be safe, and has a high degree of stability. Excess vertical maxillary growth is one problem that is readily solved with a Le Fort procedure. B, Schematic of surgical procedure. Le Fort osteotomy with maxillary impaction to impact the maxilla and an advancement genioplasty identical to that depicted in Figure 23-4, *B*, was performed. C, Orthodontic–surgical outcome: results after combination therapy. (Surgery by P.T. Akers, Glenview, IL.)

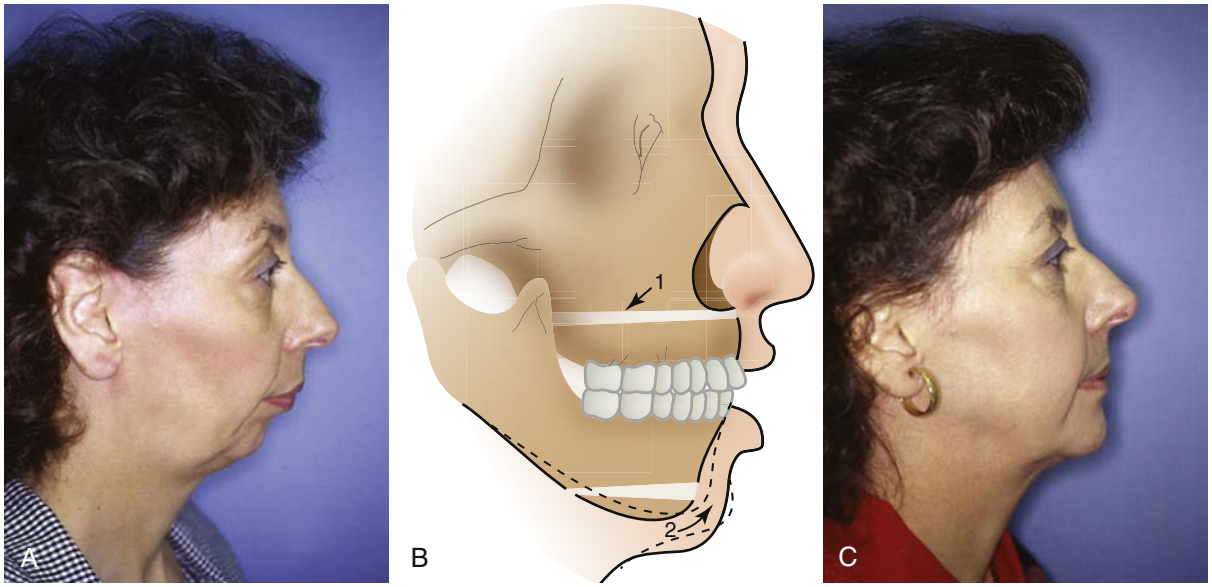


FIGURE 23-8 **A,** Class II mandibular deficiency type III (high mandibular plane angle) before treatment. **B,** Schematic of surgical procedure: maxillary open-bite osteotomy (*arrow 1*), mandibular osteotomy (*arrow 2*), and osteotomy of chin (*arrow 3*). **C,** Orthodontic–surgical outcome: results after combination therapy. (Surgery by P.T. Akers and L. McCarthy, Glenview, IL.)

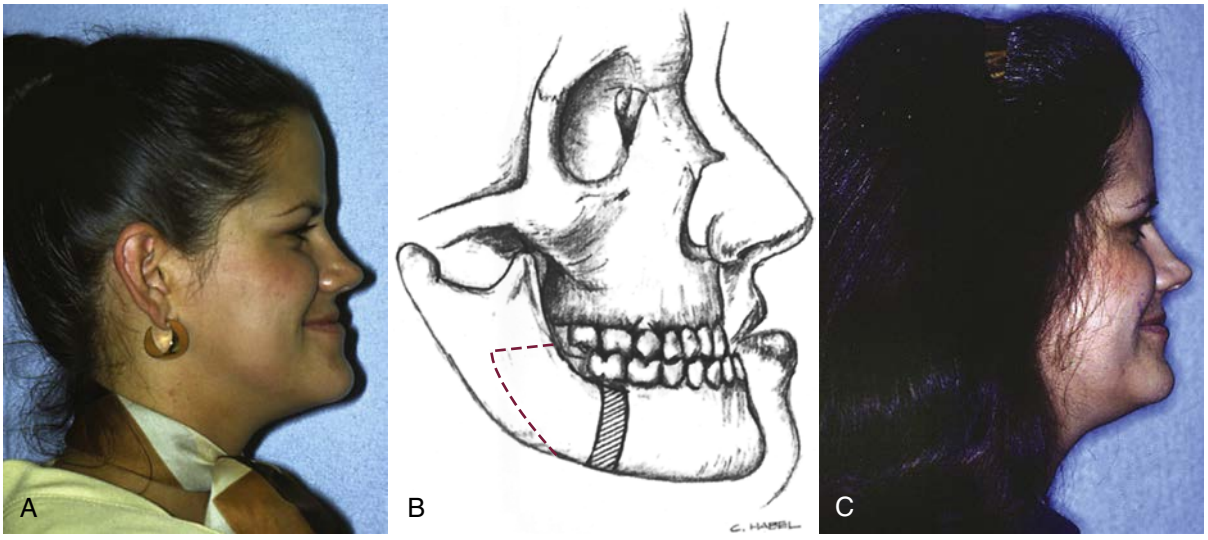


FIGURE 23-9 **A,** Class III mandibular excess before treatment. **B,** Schematic of surgical procedure: sagittal split osteotomy with setback (shown). Subcondylar vertical osteotomy (not shown). **C,** Orthodontic–surgical outcome: results after combination therapy. (Surgery by G. Doerfler, Glen Ellyn, IL.)

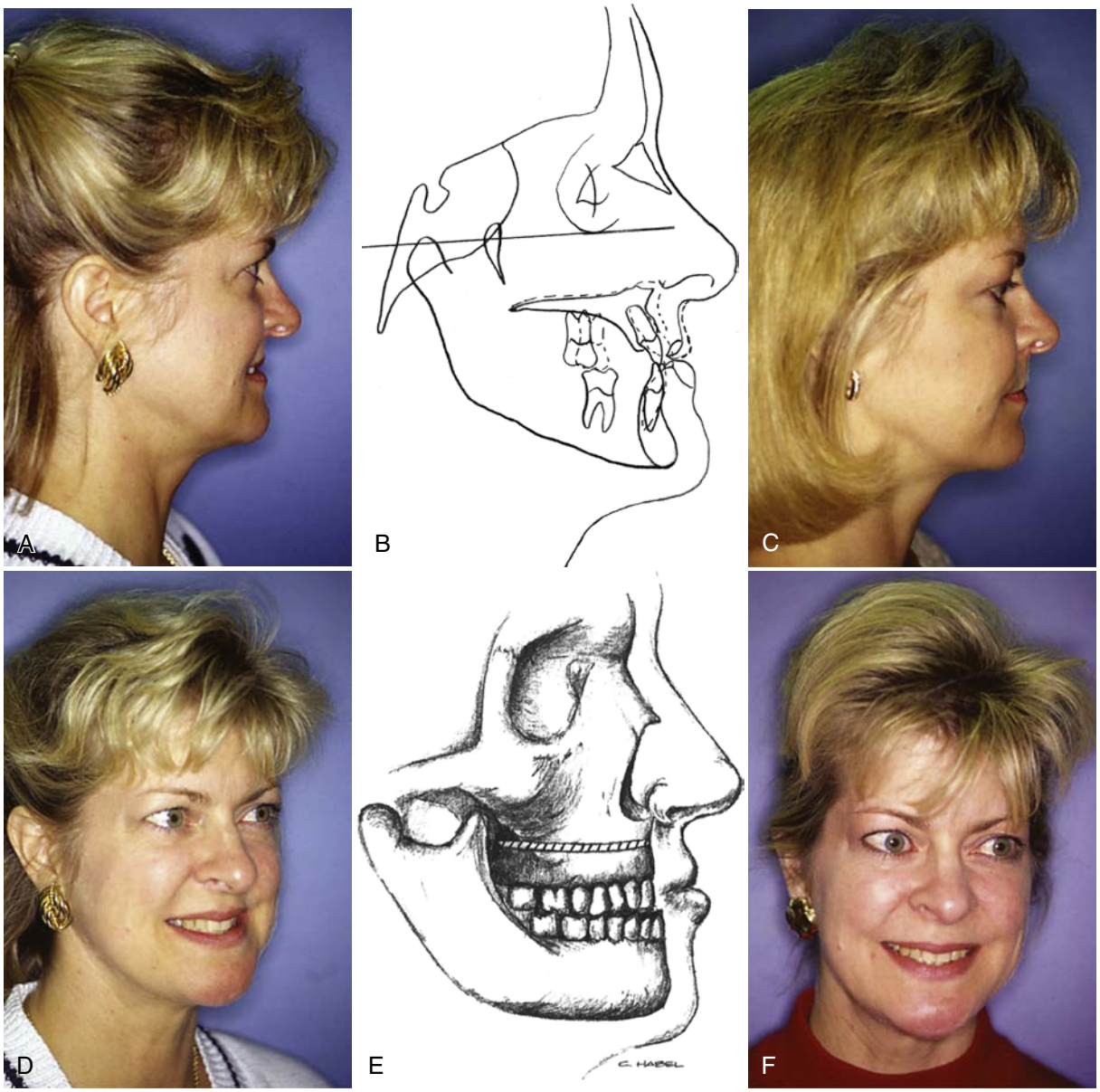


FIGURE 23-10 A, Class III maxillary horizontal deficiency before treatment. B, Cephalometric superimposition showing pretreatment and posttreatment results. C, Orthodontic–surgical outcome: results after combination therapy. Note reversal of facial aging because upper lip is more supported as a result of maxillary advancement. D, Pretreatment facial smile showing Class III maxillary horizontal deficiency. E, Schematic of surgical procedure: Le Fort osteotomy with maxillary advancement. F, Posttreatment facial smile showing corrected skeletal deficiency. (Surgery by Dr. Peter Chemello, Arlington Heights, IL.)

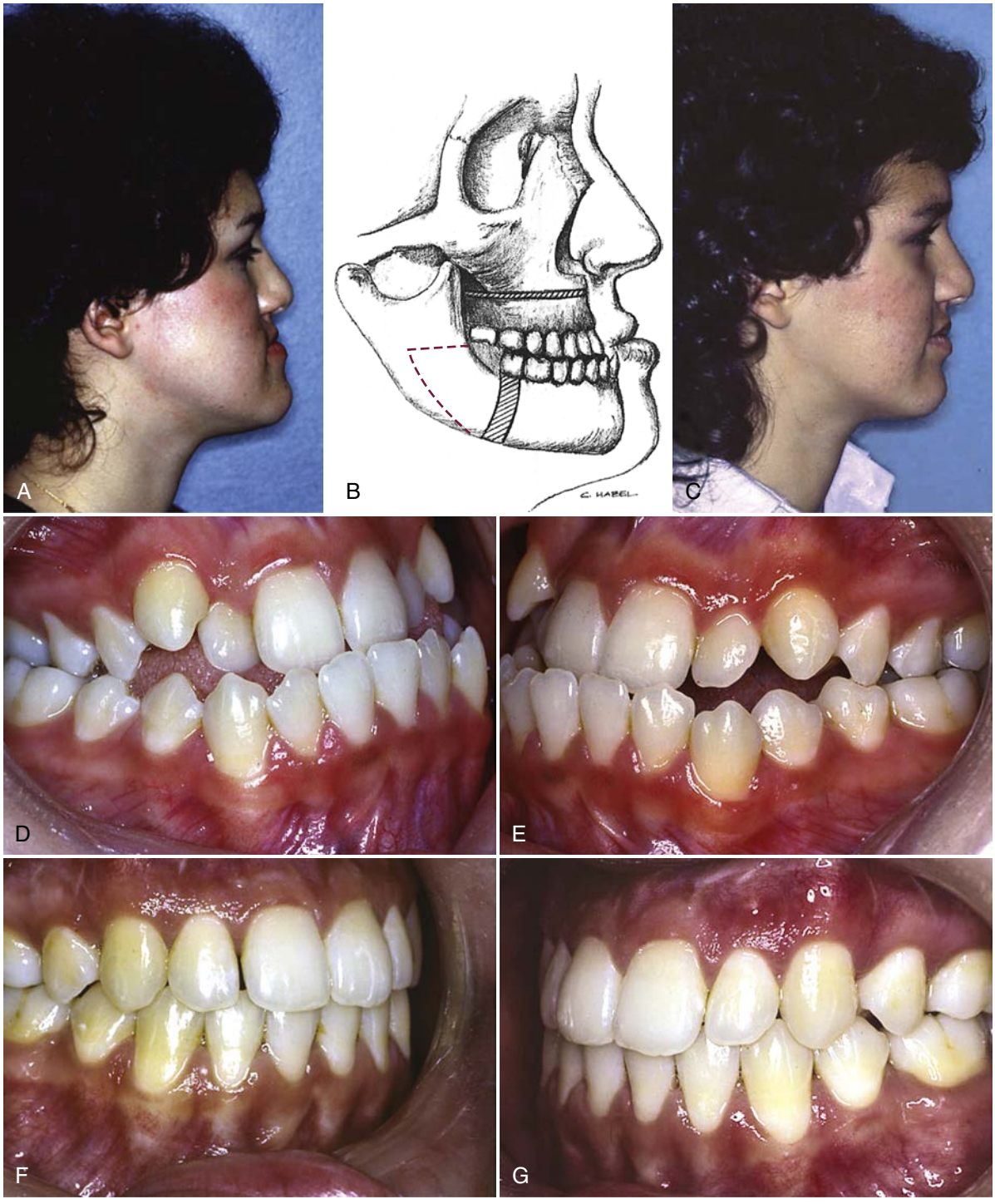


FIGURE 23-11 **A,** Class III maxillary deficiency and mandibular excess before treatment. **B,** Schematic of surgical procedure: Le Fort osteotomy of the maxilla to advance or impact (or both) and sagittal split osteotomy of the mandible to set back. **C,** Orthodontic–surgical outcome: results after combination therapy. **D** and **E,** Pretreatment intraoral photographs illustrating severe Class III malocclusion. **F** and **G,** Posttreatment intraoral photographs after extraction of nos. 4, 13, 20, and 29; orthodontic alignment and decompensation; and maxillary advancement and mandibular setback. (Surgery by A. Frer, Schaumburg, IL.)

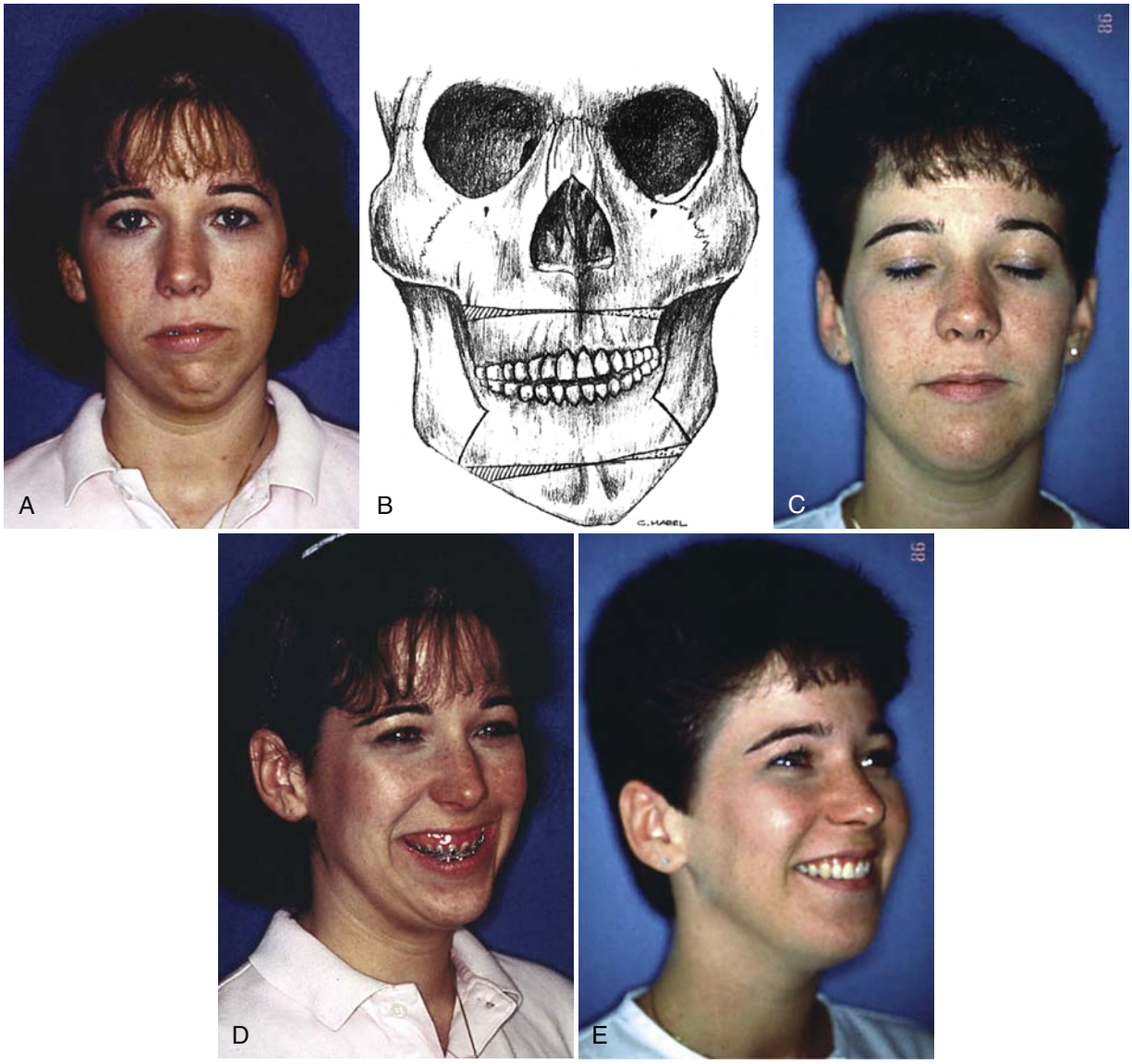
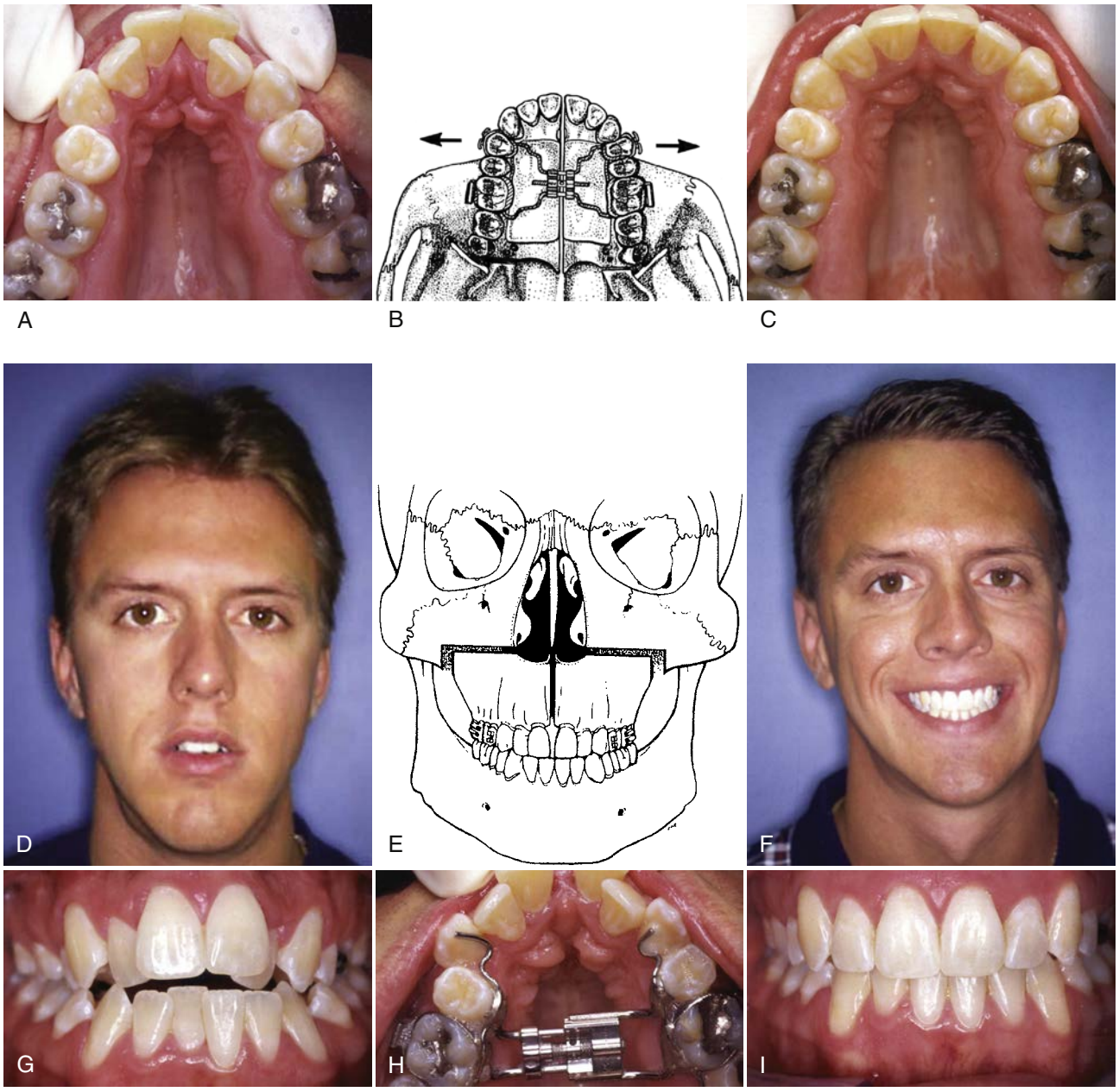


FIGURE 23-12 A, Presurgical frontal photograph of a patient showing a moderate degree of facial asymmetry. B, Frontal view of a differential Le Fort procedure of the maxilla and sagittal split rotation of the mandible and a differential genioplasty to resolve a facial asymmetry problem. C, Results after combination therapy. D, Presurgical smile showing vertical maxillary excess. E, Postsurgical smile showing improved aesthetics. (Surgery by L. Lagrottia, Schaumburg, IL.)

used to correct skeletal malocclusions. Wolford changed a key approach when he presented the benefits of occlusal plane changes in orthognathic surgery.[53] Since that time, orthognathic procedures with occlusal plane change components have become more common and have received more attention because of their benefits in treatment planning. This technique of occlusal plane change allows the surgeons to address both the functional and aesthetic concerns of a patient.[54] For example, classic orthognathic surgery would not move the occlusal plane in a counterclockwise direction to minimize the chance of postsurgical relapse. However, in long-term studies, it has been shown that occlusal plane changes in the counterclockwise direction are indeed stable.[55,56] The use of this technique allows for optimal function as well as profile enhancement with desirable lower third of face and chin repositioning. One of the more recent surgical technique modifications used by surgeons using the occlusal change approach to manage skeletal corrections is the design of the mandibular sagittal split (Fig. 23-14). Incorporating proper orthodontic preparation with the surgical technique of occlusal plane change creates outstanding functional and esthetic results as illustrated in the patients treated by the surgical-orthodontic teams in northern Italy (Figs. 23-15 and 23-16).

Occlusal plane changes are stable in patients with normal TMJ function. However, it has been shown that patients with articular disc displacement who underwent counterclockwise movements exhibit more of a tendency to relapse in both the occlusal plane change area and horizontally at B point and menton.[57]



Treatment Milestones for Patients Requiring Surgically Assisted Rapid Palatal Expansion

Diagosis	Evaluation (1–4 Months)	Initial Treatment Steps (3 Months)	Orthodontic Correction (18–38 Months)	Stabilize (12–18 Months)
With appropriate orthodontic study models and radiographs	By orthodontist and surgeon • Discuss procedure • Discuss risks and benefits • Evaluate insurance issues • Timing of procedures • Dental decompensation of lower arch • Preventive care of general dentist	1. Place expander (1 week before surgery)—Orthodontist 2. Outpatient surgery—oral surgeon 3. At 5 days, initialize widening—oral surgeon 4. Check in 1 week—orthodontist 5. At approximately 14 days, place upper braces (RPE remains for 4–8 months)—orthodontist	1. Level and align arches 2. Correct crowding and spacing 3. If needed, second-phase jaw surgery for bite correction 4. Complete orthodontics	Orthodontist • Bonded maxillary or mandibular retainers (or both) • Removable maxillary retainer Reevaluate for restorative needs with general dentist

FIGURE 23-13 A, D, and G, Maxillary transverse deficiency before treatment. B, E, and H, Schematic of surgical procedure: surgically assisted rapid maxillary expansion; a secondary surgical procedure (Le Fort osteotomy) was used to close the anterior open bite (shown in Figure 23-7, *B*). C, F, and I, Orthodontic–surgical outcome: results after combination therapy. J, Milestones involved in a typical surgically assisted rapid palatal expansion treatment plan include the diagnosis using orthodontic study records, evaluation by the orthodontist and surgeon, initial treatment steps involving rapid palatal expansion (RPE) surgery, orthodontic correction with full-bonded orthodontics, and stabilization with bonded or removable retainers (or both types of retainers). (Surgery by M. Stohle, Glenview, IL.)

Another important surgical technique modification that has been presented in the literature recently is called *surgically facilitated orthodontic therapy* (SFOT).[58] This new technique, involving single- or multiple-tooth osteotomies combined with the principles of distraction osteogenesis, facilitates rapid changes in the alveolar position of the dentition (Fig. 23-17). The SFOT concept builds on the corticotomy technique that allows rapid tooth movement because of the bone demineralization caused by the regional acceleratory phenomenon. The technique can be considered for borderline surgical cases instead of orthognathic procedures, especially when a restorative component is required. It has the advantage of increasing the adult alveolar bone volume in both the anteroposterior (AP) and transverse directions. For a properly selected case, the economic advantage may be of great value with declining insurance coverage for certain orthognathic procedures. Case selection and outcomes can be seen in the treatments presented by Roblee et al.[58] Complications associated with the technique have been minor and uncommon but include gingival recession, devitalized teeth, and ankylosis of teeth.[59]

Coscia et al. studied corticotomy plus bone grafting to aid presurgical decompensation of lower incisors for 14 skeletal Class III patients.[60] They found that that the corticotomy plus bone grafting procedures allowed patients in their study to experience shorter decompensation time, to proceed more with less risk of typical periodontal complications, and to complete decompensation with or without significant labial bone loss using cone-beam computed tomography (CBCT) comparisons.

Skeletal Fixation

Immobilization of bone fracture sites is a necessity for proper healing of the bone. The acceptance of the technologic advances in orthognathic surgery by patients and the orthodontic profession has been influenced greatly by recent changes in fixation techniques. For many years, interdental fixation with arch bars was the method of choice for orthognathic surgery patients. As presurgical orthodontic treatment became state of the art in the late 1960s, arch bars were replaced by interdental fixation with modified orthodontic appliances and by surgical archwires with soldered hooks. Because of the limitations imposed by periodontally compromised teeth, short-rooted teeth, mutilated dentition, and double-jaw procedures, more stable forms of fixation using the skeletal bases were required.

Skeletal fixation used suspension wires from stable bone such as the infraorbital rim, the zygomatic arch, or the anterior nasal spine to the mobilized maxilla. The fixation period would last for at least 6 weeks. Postsurgical depression was common, and the patient's interaction with the outside world was severely hampered. Weight loss, loss of appetite, TMJ pain, and facial muscle pain also became common concerns. Rigid fixation was introduced in 1980 in the oral and maxillofacial surgery literature. This technique helped eliminate one of the major hurdles for many patients considering jaw surgery, jaw immobilization. This advantage allows (1) a normal ability to communicate, (2) a more normal diet, (3) less time away from work, (4) less sense of being handicapped or of feeling stigmatized, and (5) better oral hygiene during healing. Rigid fixation techniques use malleable plates, which are contoured to fit over a bony osteotomy and are fixated using screws. These fixated plates hold the maxilla or mandible in the new position while healing.

Studies have shown that rigid fixation has acceptable but variable relapse rates depending on the procedure.[61–67] Factors that affect stability in the mandible include magnitude

FIGURE 23-14 A–C, Intraoral photographs before any orthodontic treatment; note the degree of anterior open bite and arch constriction. D–F, Intraoral photographs after orthodontic treatment and orthognathic surgery.

Continued

of movement; proper seating of condyles, soft tissue, and muscles; the mandibular plane angle (a low angle has greater vertical relapse; a high angle has greater horizontal relapse); remaining growth; preoperative age; and hyperdivergent facial pattern.[68,69] Maxillary inferior positioning surgery is one of the more unstable orthognathic procedures.[70] Issues with rigid fixation include relapse, condylar resorption, and infection of plates or screws. Condylar position devices have been tried at length to help minimize relapse potential. Many different devices and techniques are available, but Gerressen et al.[71] found that their use did not lead to an increase in skeletal stability compared with the manual technique of seating the condyles in the fossa. The benefits of rigid fixation include the previously mentioned patient issues as well as less pain as a result of less micromovement at the osteotomy site afforded by rigid versus nonrigid fixation. There have been concerns that rigid fixation may increase the risk of temporomandibular disorders, but a study by Nemeth et al.[72] showed no difference in the incidence of these disorders whether rigid or wire fixation was used.

Along with the advances that have developed with three-dimensional (3D) imaging, surgeons now have the capacity to use computer-aided design and computer-aided manufacturing (CAD/CAM) splints as they virtually treatment plan their

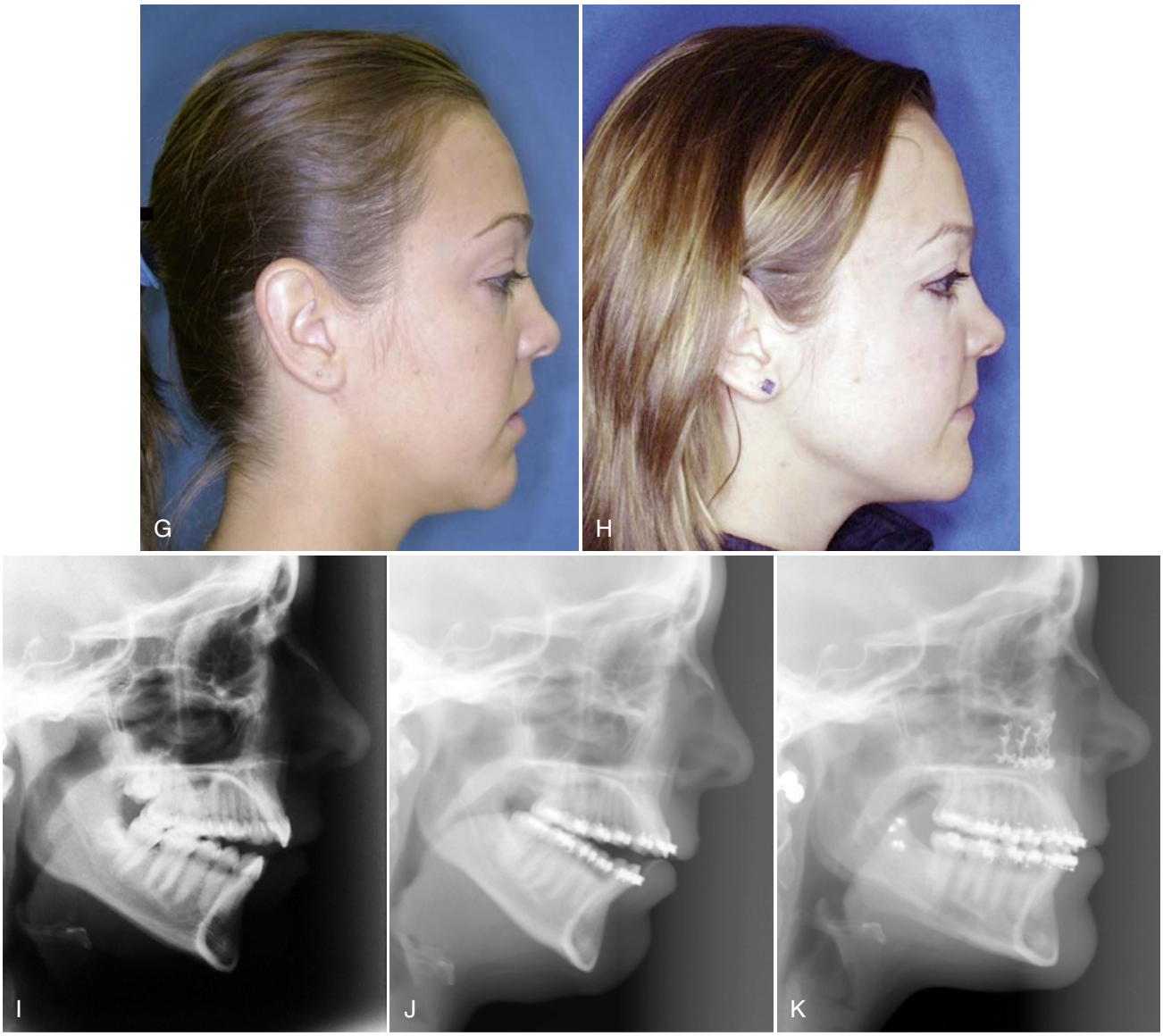


FIGURE 23-14, cont'd **G,** Profile before treatment. **H,** Profile after orthodontics and maxillomandibular surgery with occlusal plane change surgical modification. **I–K,** Cephalograms: pretreatment, presurgical after orthodontic preparation, and postsurgical, respectively. Postsurgical image (**K**) shows a significant amount of mandibular rotation and occlusal plane change to close bite and to optimize facial balance. (Surgery by Dr. Peter Chemello, Arlington Heights, IL.)

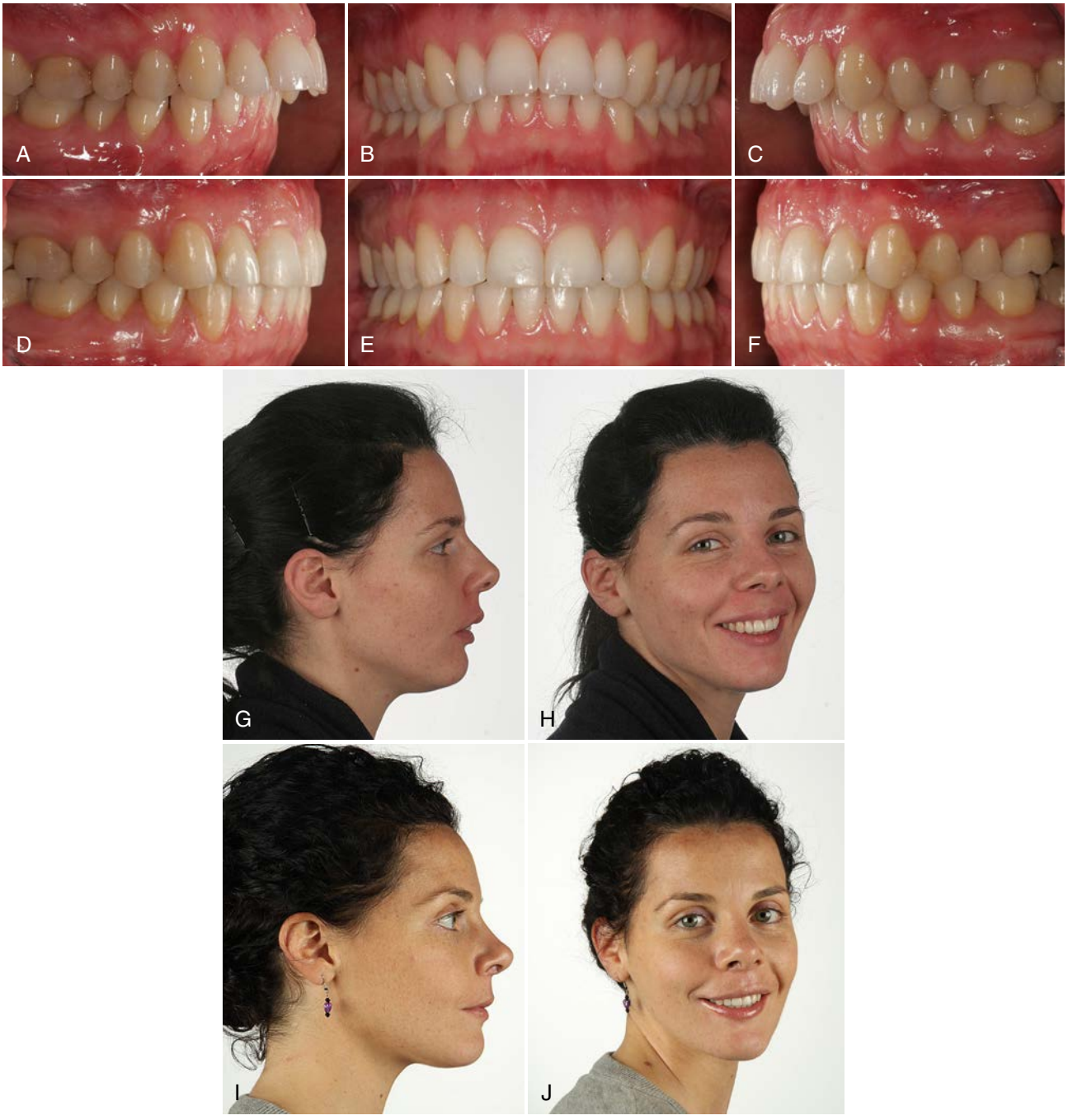
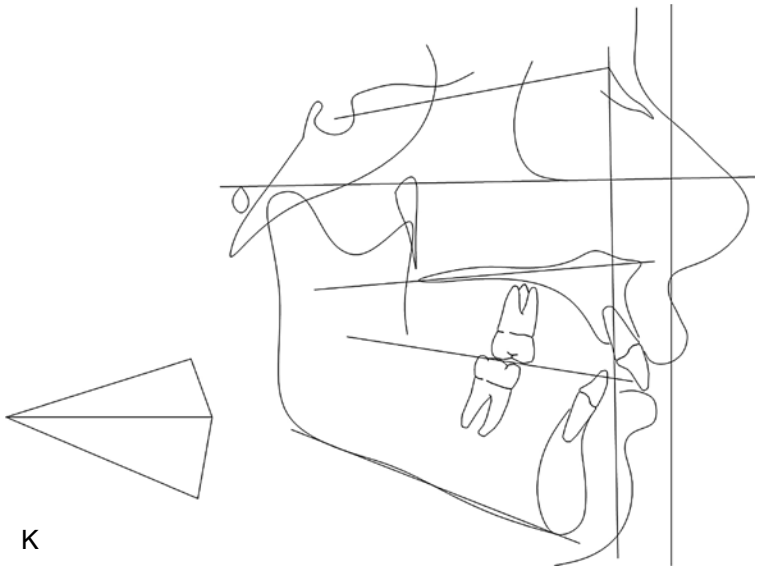
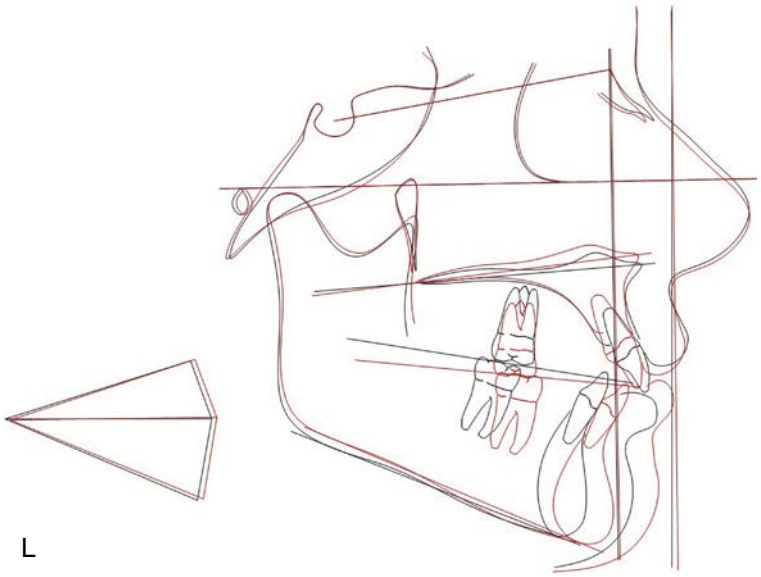


FIGURE 23-15 **A-C,** Intraoral photographs before orthodontic treatment; note degree of overjet secondary to mandibular deficiency and dental protrusion. **D-F,** Intraoral photographs after orthodontic treatment and orthognathic surgery. (Orthodontic treatment by Drs. Ute Moser and Lorenz Moser from Italy and surgery by Dr. Ugo Baciliero) **G-H,** Pre-treatment facial photographs **I-J,** Post-treatment facial photographs

Continued



K



L

FIGURE 23-15, cont'd K, Pre-treatment cephalometric tracing L, Pre & Post-treatment cephalometric superimposition on SN; black tracing is pre-treatment and red tracing are post-treatment. Note the positive impact of the occlusal plane change to optimize facial balance.

orthognathic cases and in their fixation splint design. The accuracy of this technique was tested by Zinser et al.[73] In their 2013 prospective, observational study, three techniques were assessed for 30 patients who underwent bimaxillary osteotomy:

- Approach A (8 patients): virtual treatment planning with surgical planning transfer using CAD/CAM splints
- Approach B (10 patients): approach using a surgical waferless navigation system
- Approach C (12 patients): classic approach using a semi-adjustable articulator and acrylic intermaxillary occlusal splints

This study determined that the highest precision for the virtual treatment occurred when the CAD/CAM splints were used. In addition, Zinser et al.[73] found that the CAD/CAM splint group was the only approach that maintained the condyles in their central position in the temporomandibular fossa.

Some surgeons continue to use previously successful skeletal fixation in orthognathic surgery. Use of the rigid fixation technique will continue to be less than 100% until more long-term data are reported that clarify any possible latent negative effects for mandibular surgery. Examples of several fixation techniques are shown in the postsurgical cephalograms in Figure 23-18. The patient's choice obviously favors rigid fixation methods for all types of jaw surgeries as long as stability and safety are not sacrificed. During the past several years, the most common material being used for rigid fixation has been titanium screws and plates. Some patients require removal of the titanium plates

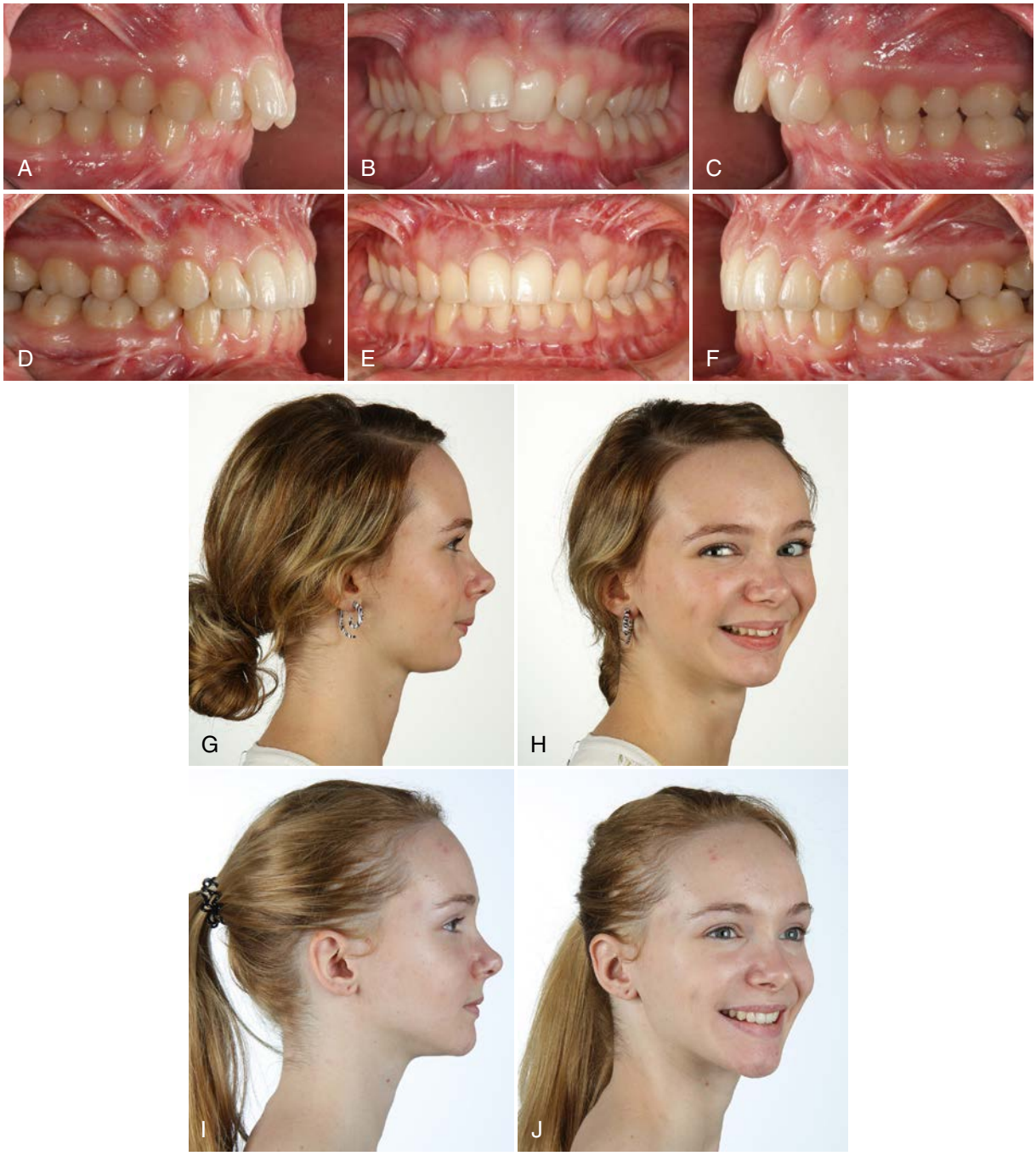
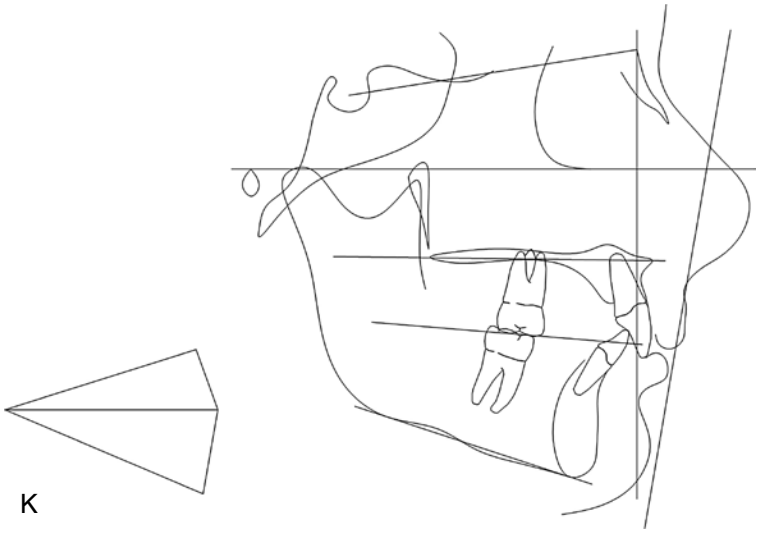


FIGURE 23-16 A-C, Intraoral photographs before orthodontic treatment; note degree of overbite. D-F, Intraoral photographs after orthodontic treatment and orthognathic surgery. Note the favorable vertical changes as a result of treatment. The removal of lower 1st premolars was necessary to decompensate the lower incisors and to optimize the degree of mandibular advancement for the best facial balance. (Orthodontic treatment by Drs. Ute Moser and Lorenz Moser from Italy and surgery by Dr. Mirco Raffiani from Italy) G-H, Pre-treatment facial photographs I-J, Post-treatment facial photographs



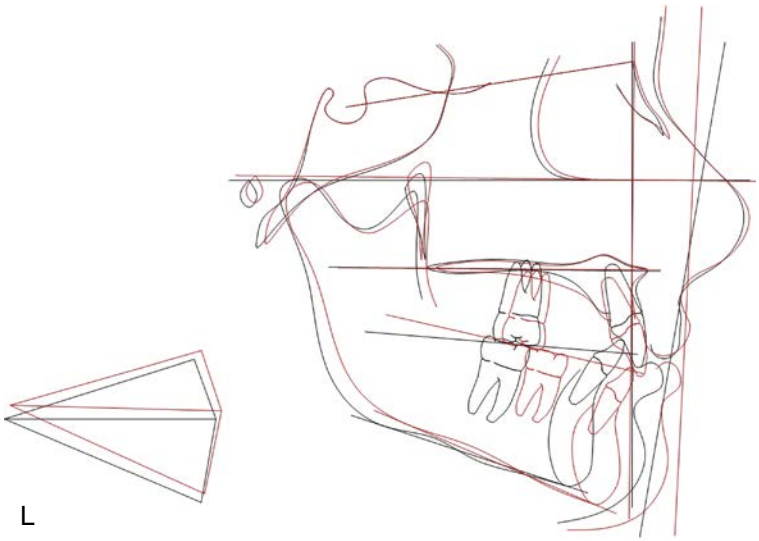


FIGURE 23-16, cont'd K, Pre-treatment cephalometric tracing L, Pre & Post-treatment cephalometric superimposition on SN; black tracing is pre-treatment and red tracing is post-treatment. Note the positive impact of the occlusal plane change to enhance the facial balance.

because of "cold" sensation, loosening, and even infection. In 2010, Ahn et al.[74] conducted a retrospective study comparing titanium plates and resorbable fixation plates in a group of 272 orthognathic patients. Although the authors believed the success rate was 98%, both groups did have complications: 8.6% for the titanium group and 18.3% for the resorbable plate group. Some of the complications revolved around relapse of the resorbable plate group who started with open bites.

The rate at which chewing capacity, range of motion, and functional comfort return is affected greatly by the type of surgery, the type of fixation, and the quality of postoperative management. Studies have shown that functional improvement occurs faster if physical therapy is added to the postsurgical regimen.[75] Figure 23-19 presents the basic flowchart for jaw rehabilitation used by the surgical team at the University of North Carolina School of Dental and Hospital Medicine.[76]

Figures 23-4 to 23-13 are schematic illustrations of some of the more common surgical procedures currently used to correct skeletal malocclusions. Figure 23-14 illustrates the positive effect of including the "occlusal plane change" technique to the orthognathic procedure. With occlusal plane change as part of the double-jaw procedure, the open bite can be closed and corrected.

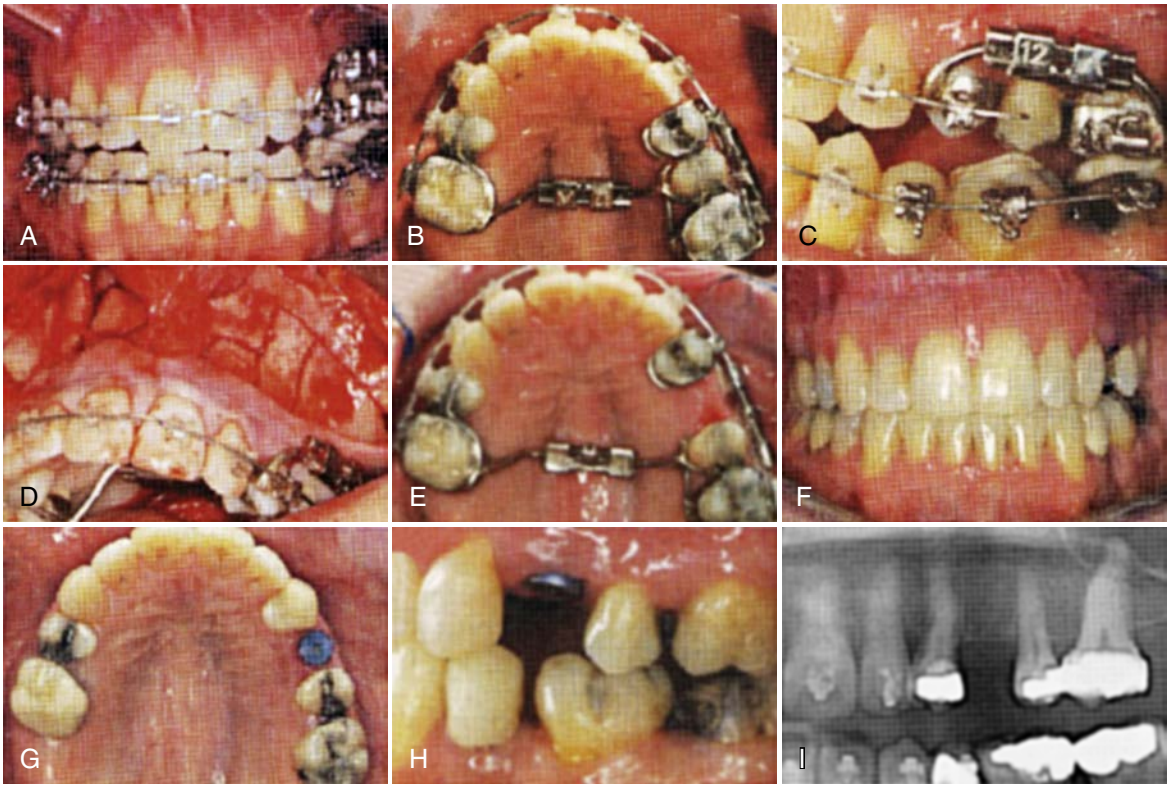


FIGURE 23-17 **A–C,** An osteotomy in surgically facilitated orthodontic treatment (SFOT) consists of cuts through both the cortical and medullary bone and typically indicates the creation of one or more dento-osseous segments. Shown here is a 50-year-old woman concerned about her appearance and bite. A jackscrew appliance was placed after 6 months of orthodontic alignment. **D–F,** The patient was missing no. 11 and had anteroposterior and transverse maxillary deficiencies with anterior and left posterior crossbites. The bite was opened with glass ionomer cement. Single-tooth osteotomies were performed on nos. 6 to 12, and multiple osteotomies were completed on nos. 3 to 4, 7 to 10, and 13 to 14. **G,** Corticotomies were also performed on nos. 7 to 10 segment. **H,** Jackscrews were activated 0.5 mm/day after a 5-day latency period. Note the pure translation of segments without tipping. Result 8 months after surgery. **I,** Tooth no. 12 was converted to a canine, and an implant was placed in regenerate bone. Note the amount of alveoloskeletal correction and increase in volume of maxillary arch.

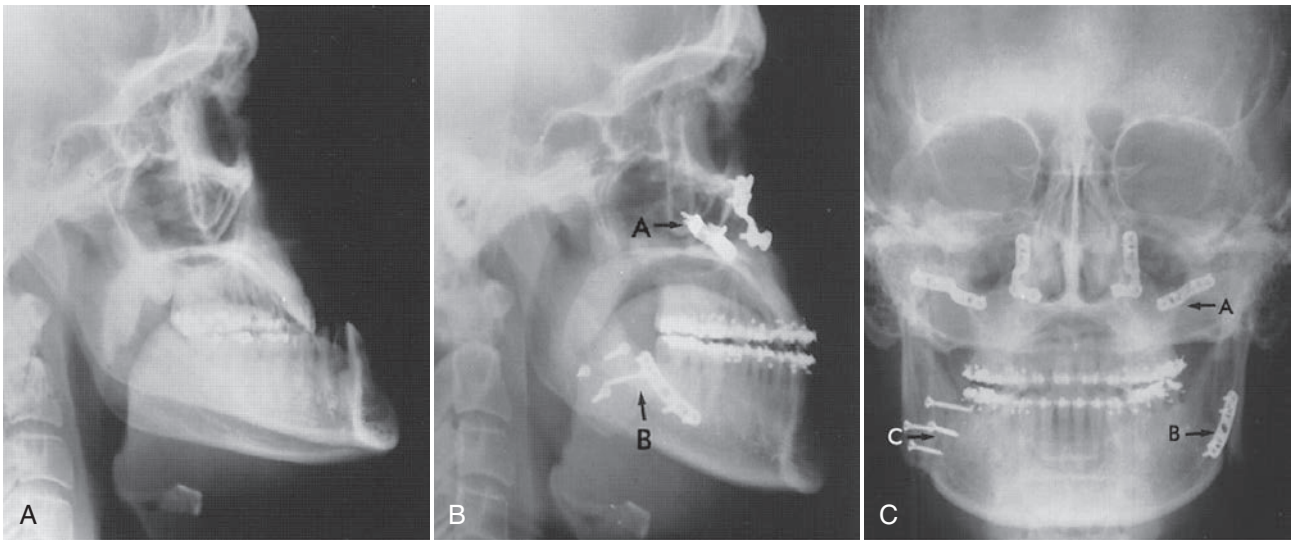


FIGURE 23-18 **A,** Pretreatment lateral cephalogram of a patient (age 33 years, 10 months) with a severe maxillary deficiency and mandibular excess. **B,** Postsurgical lateral cephalogram showing advancement of the maxilla and mandibular setback. *Arrow A*: Rigid fixation plates stabilizing the maxillary Le Fort advancement; *arrow B*: rigid fixation screws and plate stabilizing the sagittal split mandibular setback. **C,** Postsurgical frontal cephalogram. *Arrow A*: Maxillary fixation plates; *arrow B*: mandibular fixation screws; *arrow C*: mandibular fixation plate. This patient delayed surgery for 9 years to avoid 8 weeks of intermaxillary fixation and the downtime from work. Five days after his double-jaw surgery, he returned to work. Stabilization was with rigid fixation devices only.

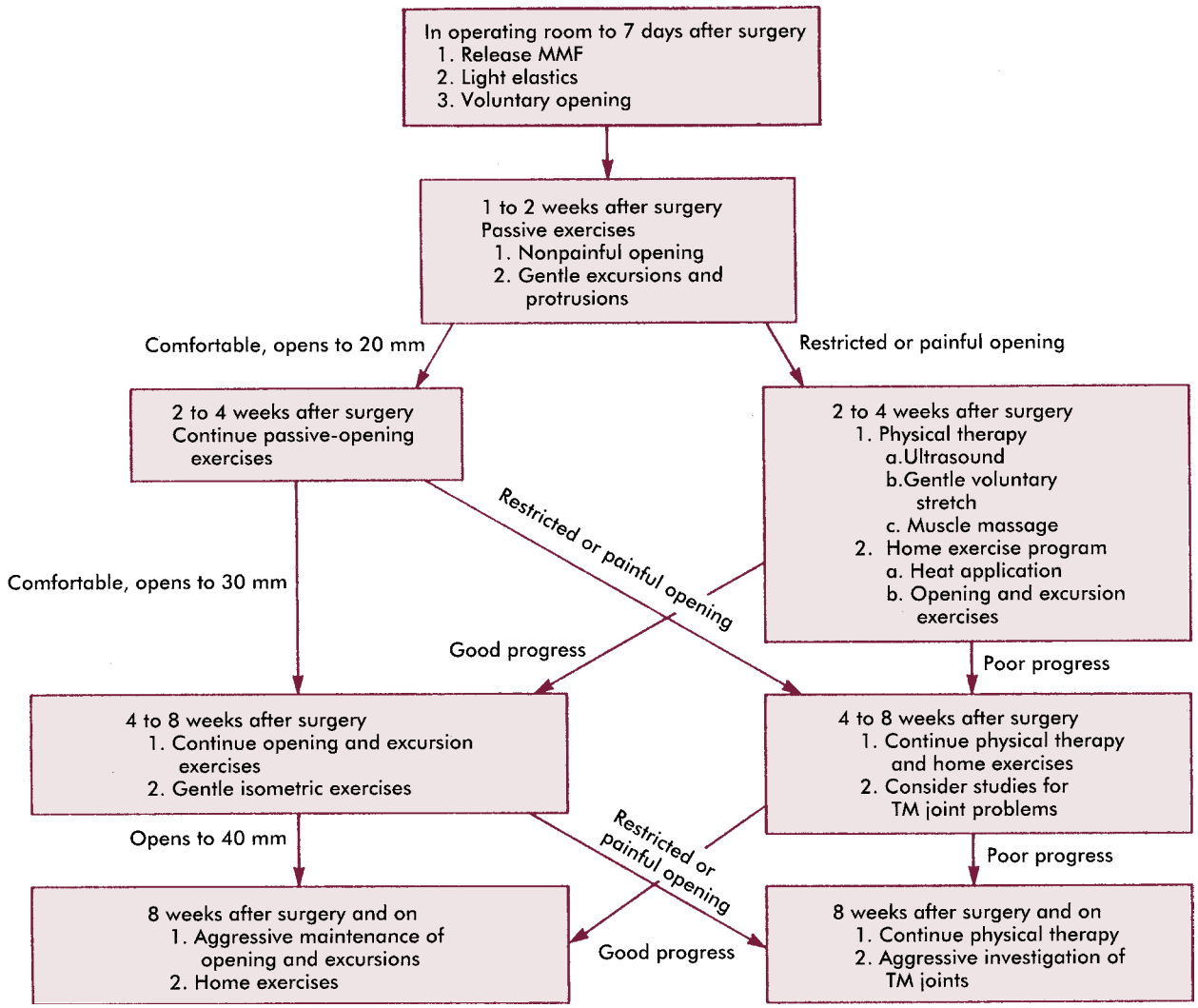


FIGURE 23-19 Flowchart for jaw rehabilitation. *MMF*, Maxillomandibular fixation; *TM*, temporomandibular.

PATIENT TREATMENT

See Box 23-3.

Diagnostic Considerations

Cone-beam computed tomography has become increasingly available and gives a 3D view of the patient's facial structure.[77–85] Information gained is used for treatment planning and gaining a better preoperative understanding of a jaw deformity in all three planes of space. Cephalometric analyses are being developed to bring information to the operating room (Fig. 23-20).[81] Surgical splints can be prefabricated based on data from the CT scan after the maxilla or mandible (or both) has been virtually moved.[82,84] Stereolithographic models based on scans can help increase intraoperative accuracy.[83] These models are extremely useful in fully uncovering a patient's deformity so that proper planning will occur to produce an optimal result (Figs. 23-21 and 23-22). In addition, these stereolithographic models are very useful for patient education regarding the needed surgical procedures to solve the skeletal aspect of the malocclusion.

For most patients, orthodontic treatment decisions have a significant elective component. Although the patient is examined for any pathologic conditions and medical problems, the orthodontist generally focuses on diagnostic considerations of the structural imbalances (Table 23-4). Three fundamental factors in the diagnostic process are (1) the standardization of high-quality records, (2) consistency of record analysis, and (3) clarity and integration of diagnostic thought by the members of the orthognathic team.

Franchi et al.[86] indicated that cephalometric "floating norms" be considered in understanding the diagnosis of skeletal imbalances: "The use of cephalometric floating norms may be helpful for diagnosis and treatment planning in orthognathic surgery and dentofacial orthopedics." Cephalometric analyses using a template method (Jacobson)[87] are graphically able to qualify (demonstrate which jaw is not in balance) and quantify (demonstrate the degree to which each jaw's dental and skeletal components contribute to the imbalance).

Superimposition of the Broadbent/Bolton template, similar in concept to the Jacobson approach, has proved to be a simple,

BOX 23-3 Characteristics of Adult Patients Seeking Orthodontic Retreatment

A. Distribution of orthodontic conditions identified in study of 100 patients seeking retreatment (most serious problem prioritized in tabulations)
   1. Skeletal problems (42%): Class II, open bites, Class III, and transverse maxillary deficiency
   2. Lower incisor (25%): unacceptable alignment
   3. Upper incisors (21%): unacceptable alignment
      - Central incisor problems
      - Lateral incisor problems
   4. Other (12%): temporomandibular joint, trauma, and periodontal issues

B. Distribution of skeletal problems identified in study of 100 patients seeking retreatment
   - Class II, high angle: 20
   - Class II, medium angle: 3
   - Class III, several types: 15
   - Class I, high angle: 3
   - Class I, maxillary transverse deficiency: 1

C. Case characteristics of skeletal patients from retreatment study
   - Age (range): 15 to 59 years
   - Age (mean): 30.12 years
   - Number of males: 13 (31%)
   - Number of females: 29 (69%)

D. Acceptance rate of skeletal cases identified in retreatment study
   - Accept braces and surgical plan: 29 of 42 (69%)
   - Accept orthodontics only; leave bite uncorrected: 4 of 42 (10%)
   - Did not accept additional treatment: 9 of 42 (21%)

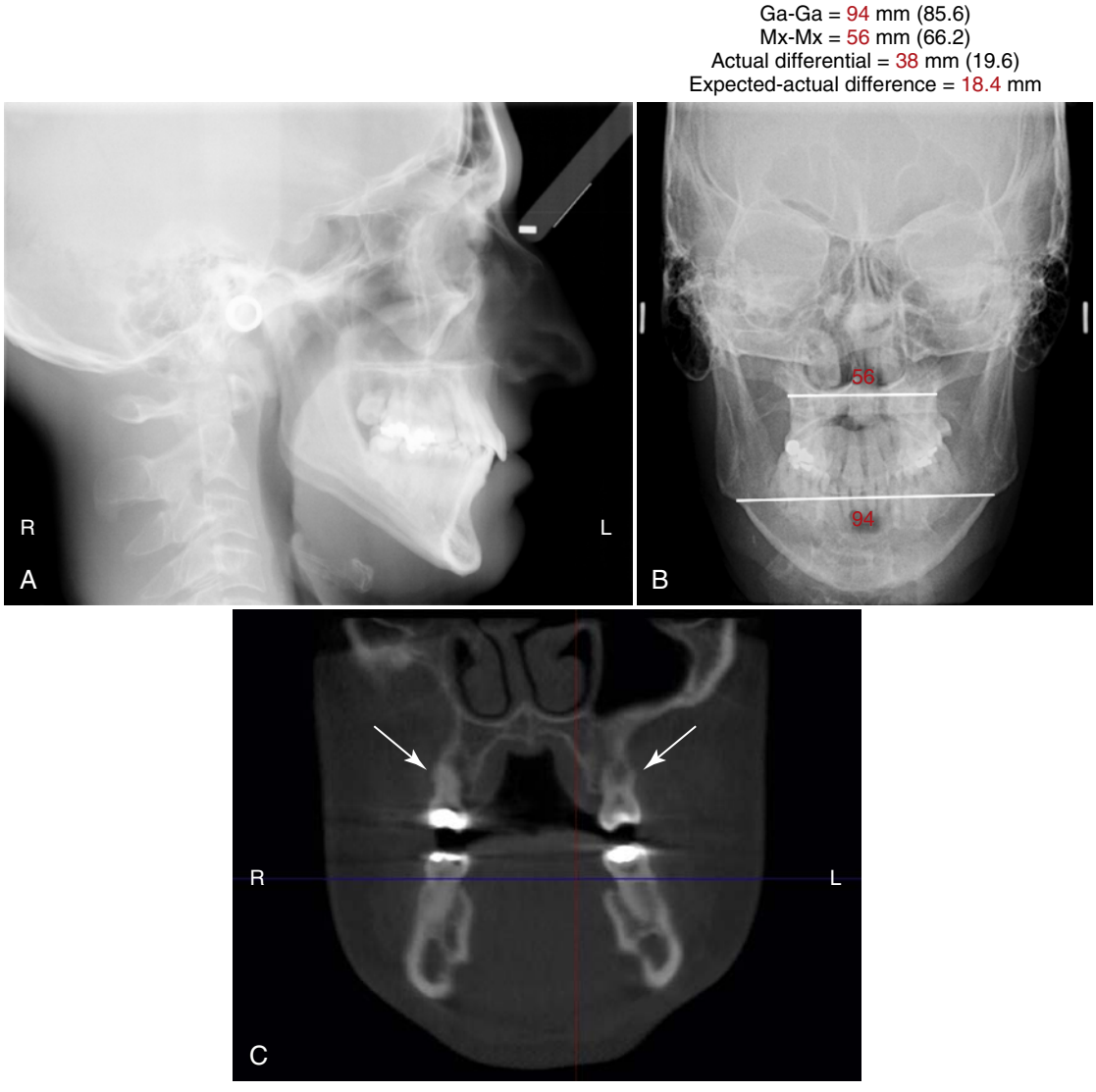


FIGURE 23-20 A, Lateral cephalogram illustrates antero-posterior dento-skeletal diagnostic landmarks. B, Postero-anterior (Frontal) cephalogram illustrates the transverse diagnostic landmarks. C, Postero-anterior (Frontal) Cone Beam image provides additional diagnostic information to manage width discrepancies of the maxilla and mandible.

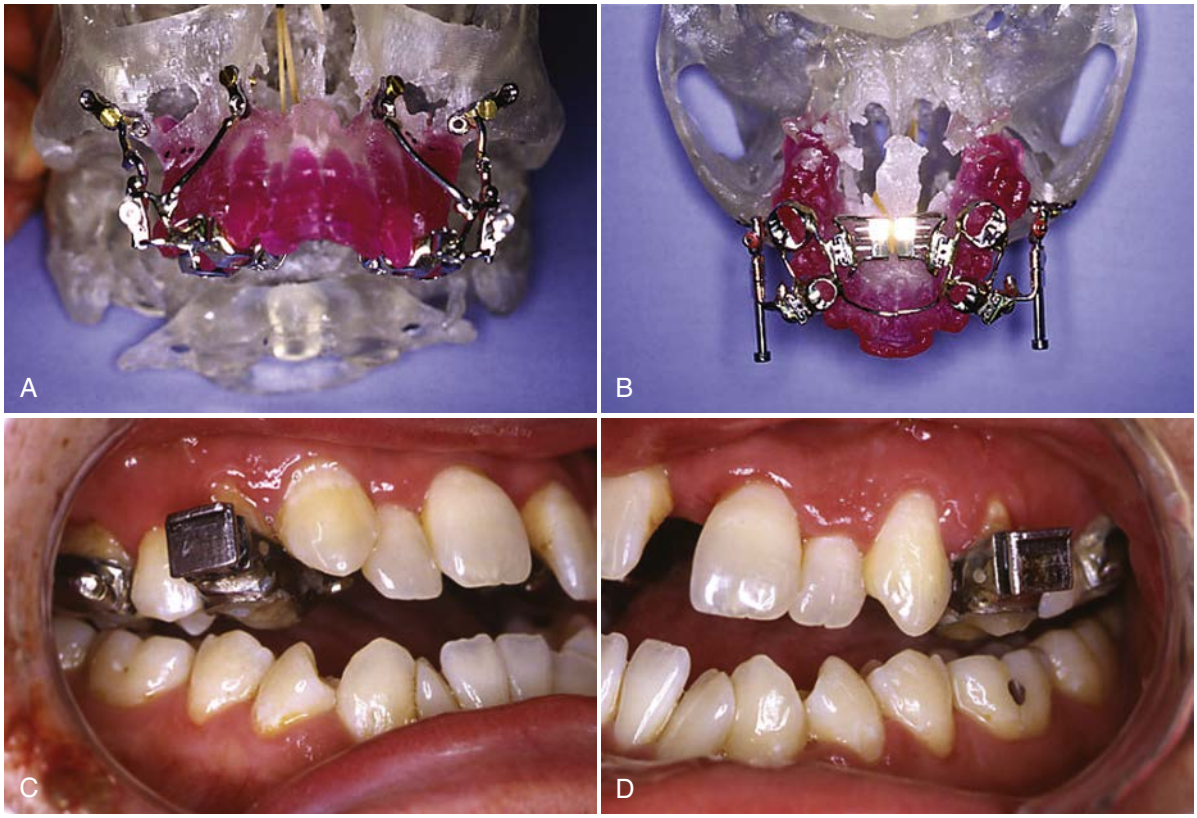


FIGURE 23-21 A and B, Stereolythic plastic model of patient's maxillary anatomy used to create custom two-dimensional distraction appliance (transverse expander and anteroposterior advancement). C and D, Intraoral view of appliance after 2 weeks of "distraction activation."

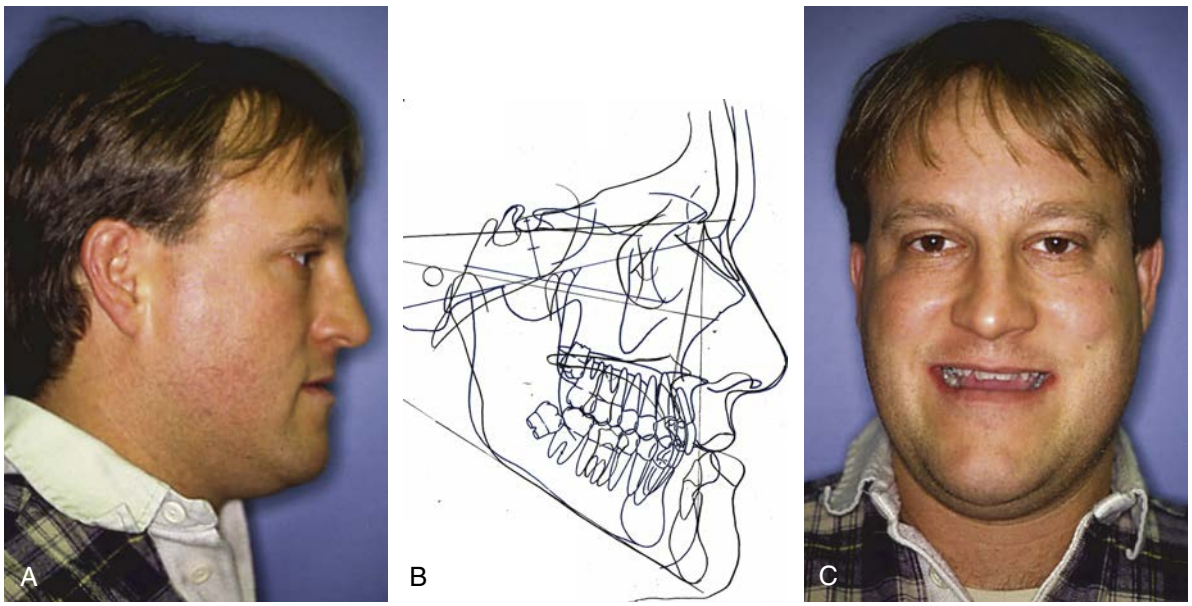


FIGURE 23-22 A, Pretreatment profile photograph. B, Patient tracing with Bolton template used to guide direction and amount of skeletal and dental changes needed for correction. C, Pretreatment facial photograph.

FIGURE 23-22, cont'd D, Posttreatment profile photograph. E, Superimposition of lateral cephalogram tracing (*solid lines* represent pretreatment appearance; *dashed lines* represent posttreatment appearance). F, Posttreatment facial photograph. G–I, Pretreatment intraoral photographs showing severe skeletal Class III malocclusion, crowding, anterior and posterior crossbites, and anterior open bite. J–L, Posttreatment intraoral photographs showing Class III correction after (*1*) phase I distraction surgery to resolve maxillary transverse deficiency and to reduce anteroposterior (AP) maxillary deficiency, (*2*) orthodontic treatment for about 22 months to decompensate dental malpositions, and (*3*) phase II jaw surgery to advance maxilla to complete the AP correction and to set mandible back and correct mandibular asymmetry and excess.

quick, and reliable tool that could be used in consultation with the treating oral surgeon and with patients to demonstrate the direction and approximate amount of surgery needed to correct the skeletal disharmony (Figs. 23-23 to 23-29). Proffit has discussed the use of template analysis in the surgical textbook he wrote with White.[40] Proffit pointed out that the template may appear to be less scientific than a table of cephalometric measurements with standard deviations, but he also noted that "the template is a visual analog of a table and is just as valid." Proffit also explains, "What the template does is place the emphasis on the analysis itself; that is, deciding what the distortions are, rather than on an intermediate measurement step that too often becomes an end in itself rather than just a means to an end."

The authors have found that the template approach to cephalometric analysis allows an assessment of skeletal landmarks compared with age-matched norms. Use of nasal superimposition fully integrates the nose into cephalometric assessment and allows the clinician to assess soft tissue compensation for skeletal imbalance. The authors have found this approach to

TABLE 23-4 Medical Problems Significant for Surgical-Orthodontic Treatment*

Condition	Significance	Comments
Diabetes mellitus	Susceptible to periodontal breakdown during orthodontic treatment; decreased resistance to infection; poor wound healing	Maintenance of excellent control is necessary.
Hyperthyroidism	Increased metabolic rate; tendency to osteoporosis	Infection may create crisis; psychological instability is possible.
Adrenal insufficiency	Decreased stress tolerance; delayed healing	Knowledge of steroid dosage is important.
Pregnancy	Major hormonal changes; increased susceptibility to periodontal breakdown	Defer surgical treatment; limited orthodontic treatment is acceptable, but careful periodontal monitoring is required.
Rheumatic heart disease, other heart disease	Susceptible to endocarditis	Antibiotic coverage is required for invasive procedures (in orthodontics, for banding but not for bonding or routine adjustments).
Bleeding disorders (e.g., hemophilia, von Willebrand disease, and thrombocytopenia)	Susceptible to bleeding	Replace missing clotting factors before surgery (in orthodontics, bonded attachments instead of bands and avoidance of aspirin and related drugs for pain control).[106]
Sickle cell anemia	Susceptible to sickle cell crisis and bone loss	Patients are not good candidates for general anesthesia; therefore, they usually are not considered for orthognathic surgery.
Allergy-immune problems	Excessive reaction to drugs and other antigens	In rare cases, nickel content in stainless steel orthodontic appliances causes problem.
Rheumatoid arthritis	Episodic involvement of multiple joints and possible destruction of temporomandibular joint (TMJ)	Manipulation of TMJ tends to exacerbate problem; avoid functional appliances, Class II elastics, and mandibular advancement.
Osteoarthritis	Progressive involvement of multiple joints, possibly including TMJ, with increasing age	Orthodontics or orthognathic surgery has little effect for better or worse on involved TMJ.
Behavioral disorders	Depends on degree of control; patients often taking potent medications	Drug effects may slow orthodontic tooth movement; bizarre reactions to surgery are possible.

*In all instances, surgical-orthodontic treatment should be undertaken only after these and related conditions are under medical control.
Adapted from Proffit WR, White RP. *Surgical-Orthodontic Treatment*. St. Louis: Mosby; 1990.

be excellent for diagnosis and treatment planning for skeletal dysplasias. In its application, it should be considered *template-guided diagnosis and treatment planning* and is illustrated in Figures 23-23 to 23-29.

In addition to diagnosing the AP and vertical skeletal components of the patient's disharmony, it is also essential to make an accurate diagnosis of the contributing transverse skeletal component. Until 3D diagnostic parameters are clearly established (see Chapter 11), the analysis of frontal radiographs continues to be an effective approach to identify true transverse skeletal discrepancies that may require surgical management (Fig. 23-30).

Diagnosing Class II Mandibular Deficiency and Class III Mandibular Excess

The authors use the expanded version of the Angle classification system to create a more accurate perspective to diagnostic descriptions (Class II mandibular deficiency—low Mandibular Plane (MPl) angle as illustrated in Figure 23-23, or Class III mandibular excess—high angle as illustrated in Fig. 23-28). These descriptions are then integrated with the diagnostic steps of quantification and a rating of severity to illustrate diagnostic and treatment differences of the basic types of problems that arise with mandibular deficiency and mandibular excess (see Figs. 23-23 to 23-28). In each of these figures, note that the Bolton template superimposition is used to show the skeletal deviations from the template ideal. The process of differential diagnosis of the structural problem, then, includes the following steps:

1. **Classification:** The condition is assigned to a category of Class I, II, or III. (Angle's traditional system of classification still is important and can help with codification for insurance purposes.)
2. **Qualification:** The problem location is determined (i.e., Is the problem in the upper jaw only? The lower jaw only? Is it all skeletal, or part skeletal and part dental?).
3. **Quantification**: The extent to which each jaw contributes to the problem is determined by percentage or in millimeters.
   - Severity: A scale of 0 to 10 provides a rating of the severity of AP, transverse, and vertical problems, as well as of tooth alignment, dental pathologic conditions, TMJ status, psychosocial factors, and the patient's degree of motivation. Although each problem could be rated individually, the concept of using one number to rate the whole constellation of problems helps the patient understand, in a relative way, the severity of the current condition. Whereas a rating of 0 means that conditions are ideal or that no problems exist, a rating of 10 indicates a severe dentoskeletal imbalance with possible pathologic sequelae. This number also can be used to project the prognosis for the outcome of the various treatment options under consideration.
4. **Prediction of the prognosis:** (This may vary depending on the treatment plan chosen.) Again using the visual analog scale (0–10), it is helpful to predict the improvement of the patient's condition based on the treatment plan. For example, a patient with a transverse problem who accepts surgical expansion may improve from severity of 7 of 10 to 1 of 10 with surgery but only improve to a 5 of 10 without surgery.

Treatment Planning Options

With the safety of jaw surgery techniques improving as a result of modifications in surgical approaches and with anesthetic procedures becoming more sophisticated, orthognathic surgery offers the orthodontist and the patient a range of treatment planning options. When a patient has a problem in which

Text continued on page 703

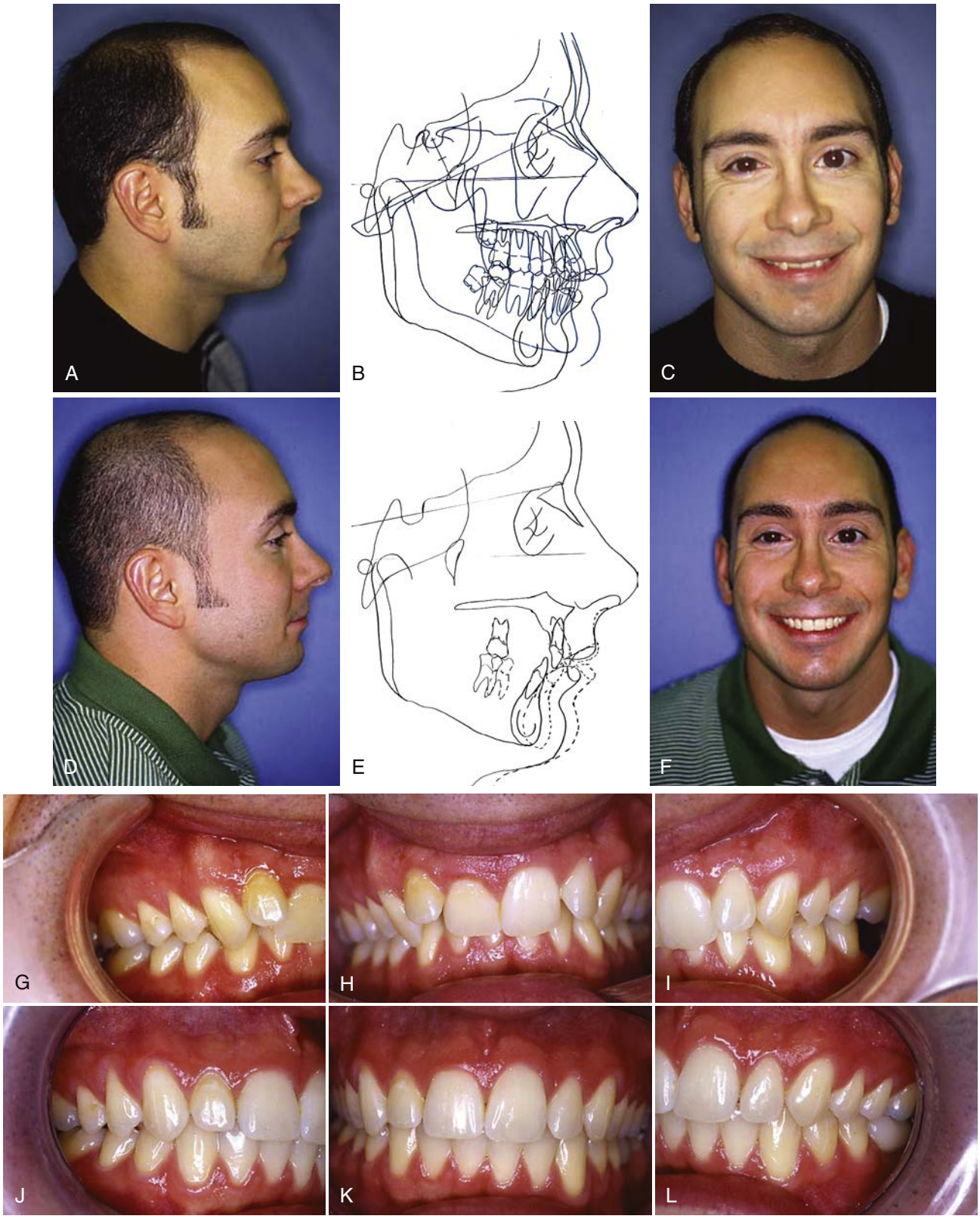


FIGURE 23-23 Patient: TH; age 27 years, 10 months; male. Classification: Class II, Division 2, deep bite, and bite collapse on left. Qualification: Mandibular anteroposterior deficiency and vertical deficiency. Quantification: 100% mandibular skeletal deficiency; mandibular vertical deficiency; moderate horizontal excess in chin area camouflages the skeletal deficiency of the mandible. Severity: 8 of 10, with 10 being most severe and 0 being perfect. Prognosis: 1 of 10 with comprehensive orthodontics and jaw surgery. **A,** Pretreatment profile photograph. **B,** Bolton template–guided diagnosis and treatment plan. **C,** Pretreatment facial photograph. **D,** Posttreatment profile photograph. **E,** Cephalometric superimposition. **F,** Posttreatment smile photograph. **G–I,** Intraoral pretreatment photographs showing Class II malocclusion. **J–L,** Intraoral posttreatment photographs showing optimal correction with replacement of missing no. 19.

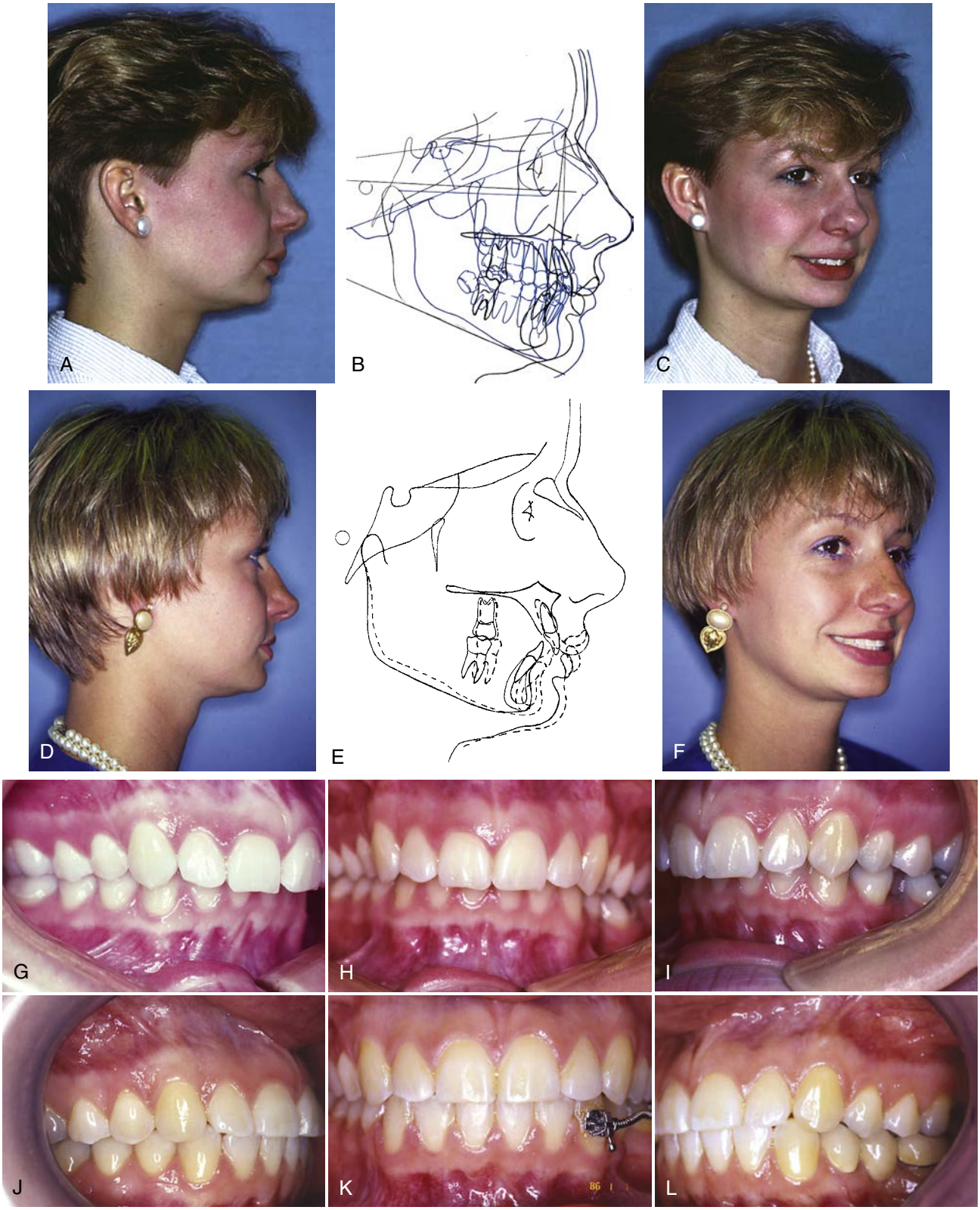


FIGURE 23-24 Patient: JT; age 17 years, 11 months; female. Classification: Class II, Division 1. Qualification: Mandibular deficiency type II. Quantification: 100% mandibular deficiency with normal maxillary skeletal and dental position as seen through template analysis (14C). Severity: 7 of 10. Prognosis: 0.5 of 10 with comprehensive orthodontics and jaw surgery. **A,** Pretreatment profile photograph showing evidence of mandibular deficiency. **B,** Bolton template–guided diagnosis and treatment plan used template superimposition that shows good maxilla position and horizontal and vertical mandibular deficiency. **C,** Pretreatment facial photograph. **D,** Posttreatment profile photograph showing change as a result of orthodontic treatment and mandibular advancement. **E,** Cephalometric superimposition showing corrected mandibular deficiency. **F,** Posttreatment smile photograph. **G–I,** Intraoral pretreatment photographs showing Class II malocclusion, bite collapse, and deep bite. **J–L,** Intraoral posttreatment photographs showing correction with bridge in **L** replacing no. 19.

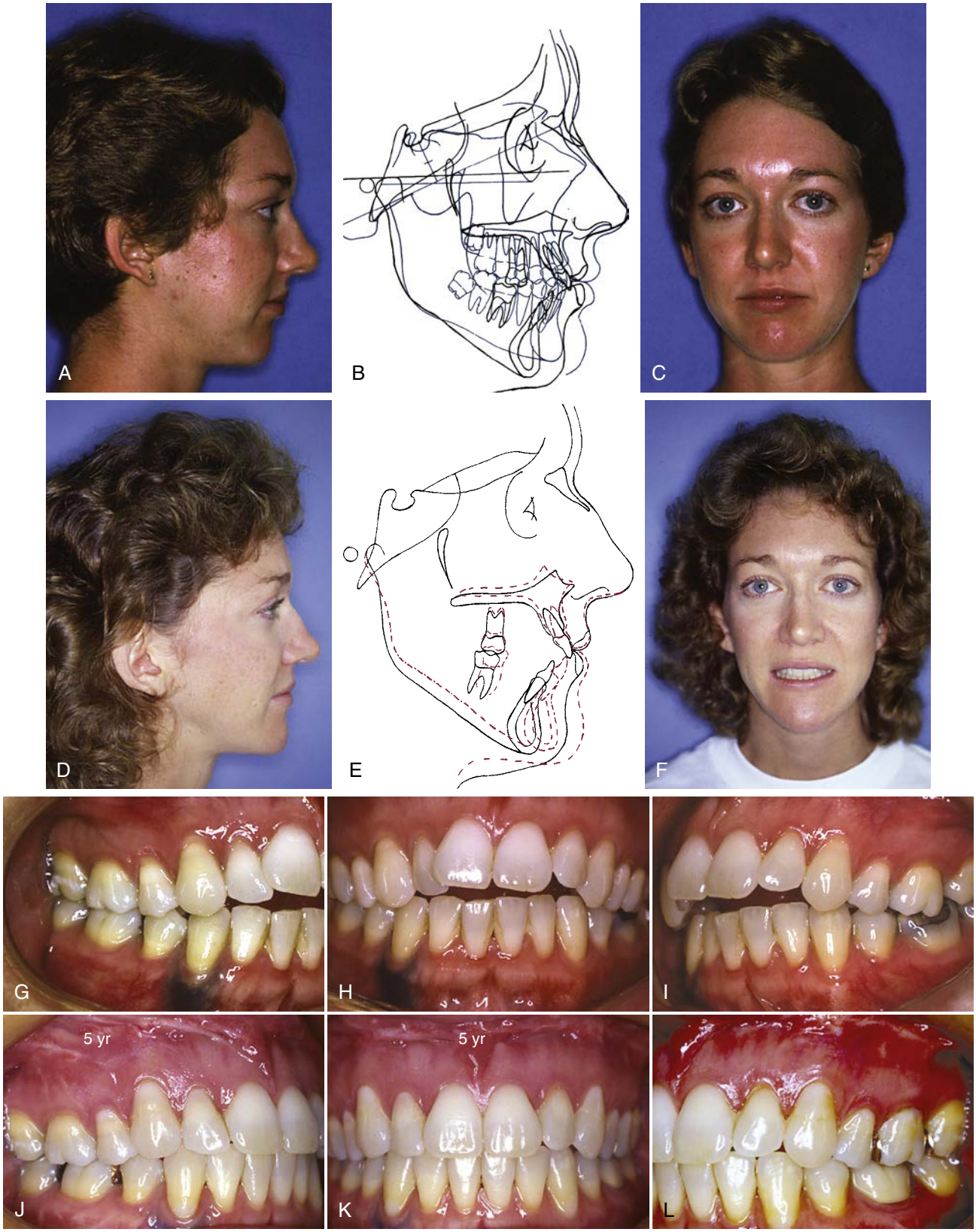


FIGURE 23-25 Patient: LG; age 26 years, 4 months; female. Classification: Class II, Division 1. Qualification: Mandibular deficiency type III with open bite. Quantification: Maxillary posterior vertical excess in 100% mandibular deficiency. Severity: 8 of 10 (patient also has temporomandibular joint pain and disk displacement with reduction). Prognosis: Temporomandibular (reevaluate) occlusal function, 1 of 10; favorable prognosis with comprehensive orthodontic treatment and jaw surgery. **A,** Pretreatment profile photograph. **B,** Bolton template–guided diagnosis and treatment plan. **C,** Pretreatment facial photograph. **D,** Posttreatment profile photograph. **E,** Cephalometric superimposition. **F,** Posttreatment smile photograph. **G–I,** Intraoral pretreatment photographs showing malocclusion. **J–L,** Intraoral posttreatment photographs showing optimal correction.

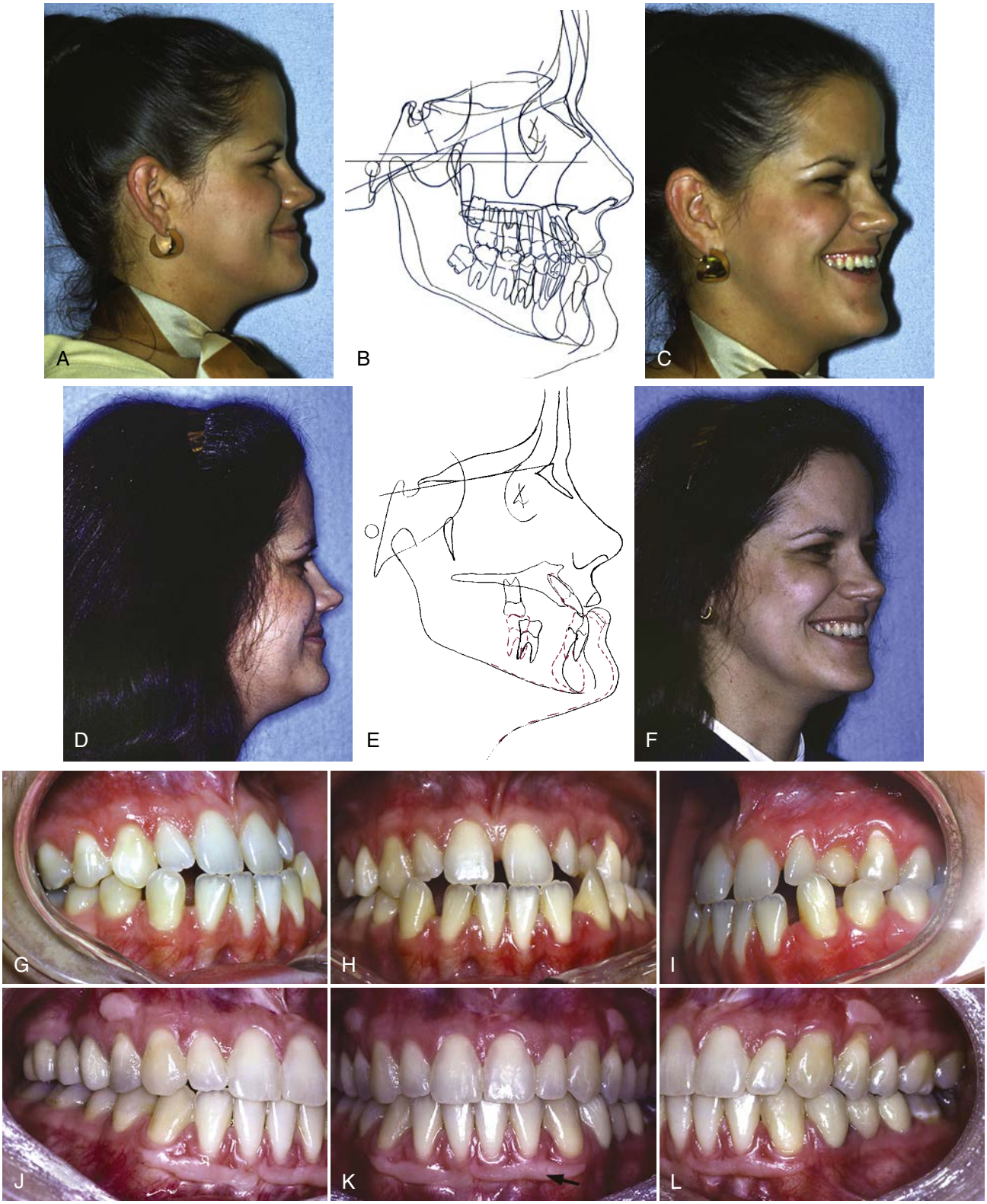


FIGURE 23-26 Patient: PM; age 24 years, 3 months; female. Classification: Class III mandibular excess type I (low mandibular plane angle). Qualification: Mandibular skeletal excess with significant maxillary dentoalveolar compensation and relative protrusion. Quantification: 100% mandibular excess. Severity: 8.5 of 10; severe skeletal problem; impaction of no. 11, dental spacing, and mucogingival deficiency. Prognosis: 2 of 10; prognosis is estimated at 2 because of the impacted canine and other dental problems that require management; the patient has a good attitude for handling complicating factors in treatment. **A,** Pretreatment profile photograph. **B,** Bolton template–guided diagnosis and treatment plan. **C,** Pretreatment facial photograph. **D,** Posttreatment profile photograph. **E,** Cephalometric superimposition. **F,** Posttreatment smile photograph. **G–I,** Intraoral pretreatment photographs showing malocclusion. **J–L,** Intraoral posttreatment photographs showing optimal correction. Note an extra premolar was added between no. 20 and no. 21 to aid in dental decompensation and to stabilize excess lower spacing.

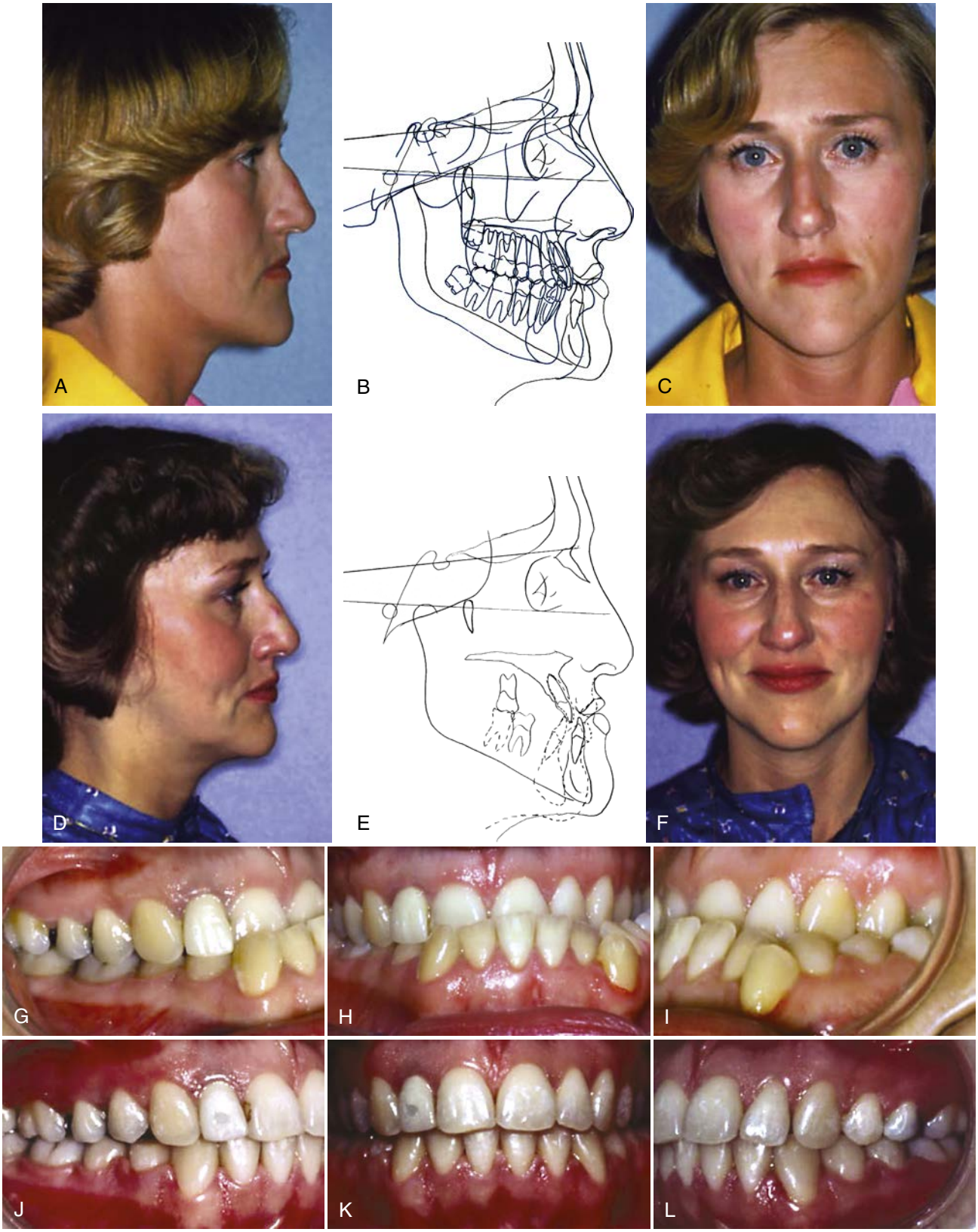


FIGURE 23-27 Patient: EF; age 30 years, 8 months; female. Classification: Class III skeletal condition. Qualification: Mandibular excess type II (moderate mandibular plane angle). Quantification: 100% mandibular excess. Severity: 7 of 10 skeletal severity and long-standing dental compensation of the lower incisors. Prognosis: 2 of 10; the prognosis is good with full orthodontic treatment and jaw surgery (chin excess and nasal form may require cosmetic procedure for ideal outcome). **A,** Pretreatment profile photograph. **B,** Bolton template–guided diagnosis and treatment plan. **C,** Pretreatment facial photograph. **D,** Posttreatment profile photograph. **E,** Cephalometric superimposition. **F,** Posttreatment smile photograph. **G–I,** Intraoral pretreatment photographs showing malocclusion. **J–L,** Intraoral posttreatment photographs showing optimal correction.

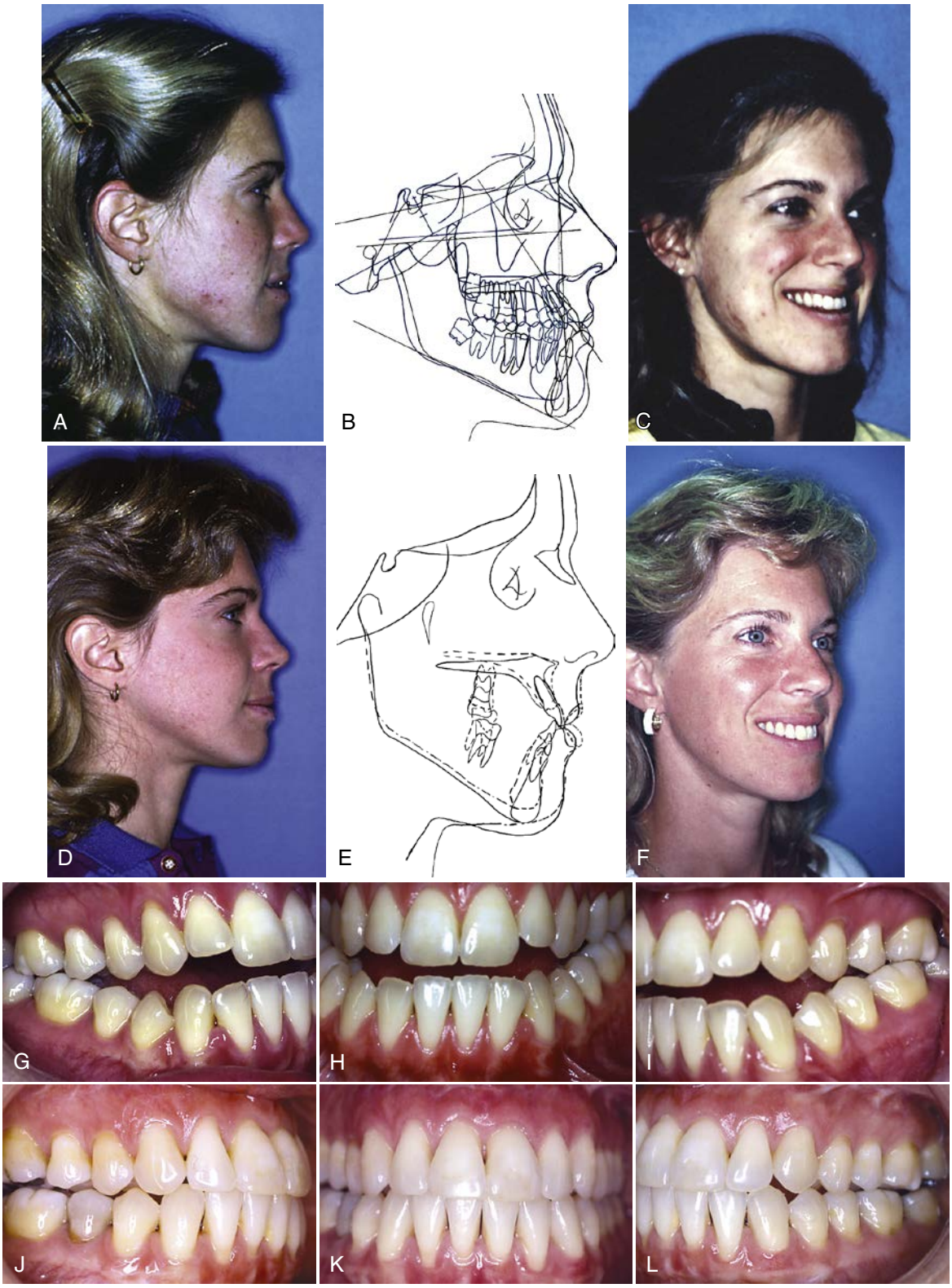


FIGURE 23-28 Patient: AL; age 25 years, 6 months, female. Classification: Class III skeletal condition with mild alignment problems and no incisor occlusion. Qualification: Mandibular excess with a high mandibular plane angle. Quantification: Mandibular horizontal excess with maxillary posterior vertical excess and an anterior open bite+. Severity: 8.5 of 10; the condition is a multifaceted problem complicated by muscle and joint pain and severe gingival deficiency in the lower anterior and upper posterior areas. Prognosis: 2 of 10; significant improvement can be achieved with successful interdisciplinary therapy using proper temporomandibular dysfunction management, orthodontic and surgical treatment, and periodontal grafting procedures. This patient was highly motivated and complied with the provider team's interdisciplinary treatment recommendations. Her condition responded well to the therapy, as can be seen in the posttreatment photographs. **A,** Pretreatment profile photograph. **B,** Bolton template–guided diagnosis and treatment plan. **C,** Pretreatment facial photograph. **D,** Posttreatment profile photograph. **E,** Cephalometric superimposition. **F,** Posttreatment smile photograph. **G–I,** Intraoral pretreatment photographs showing malocclusion. **J–L,** Intraoral posttreatment photographs showing optimal correction.

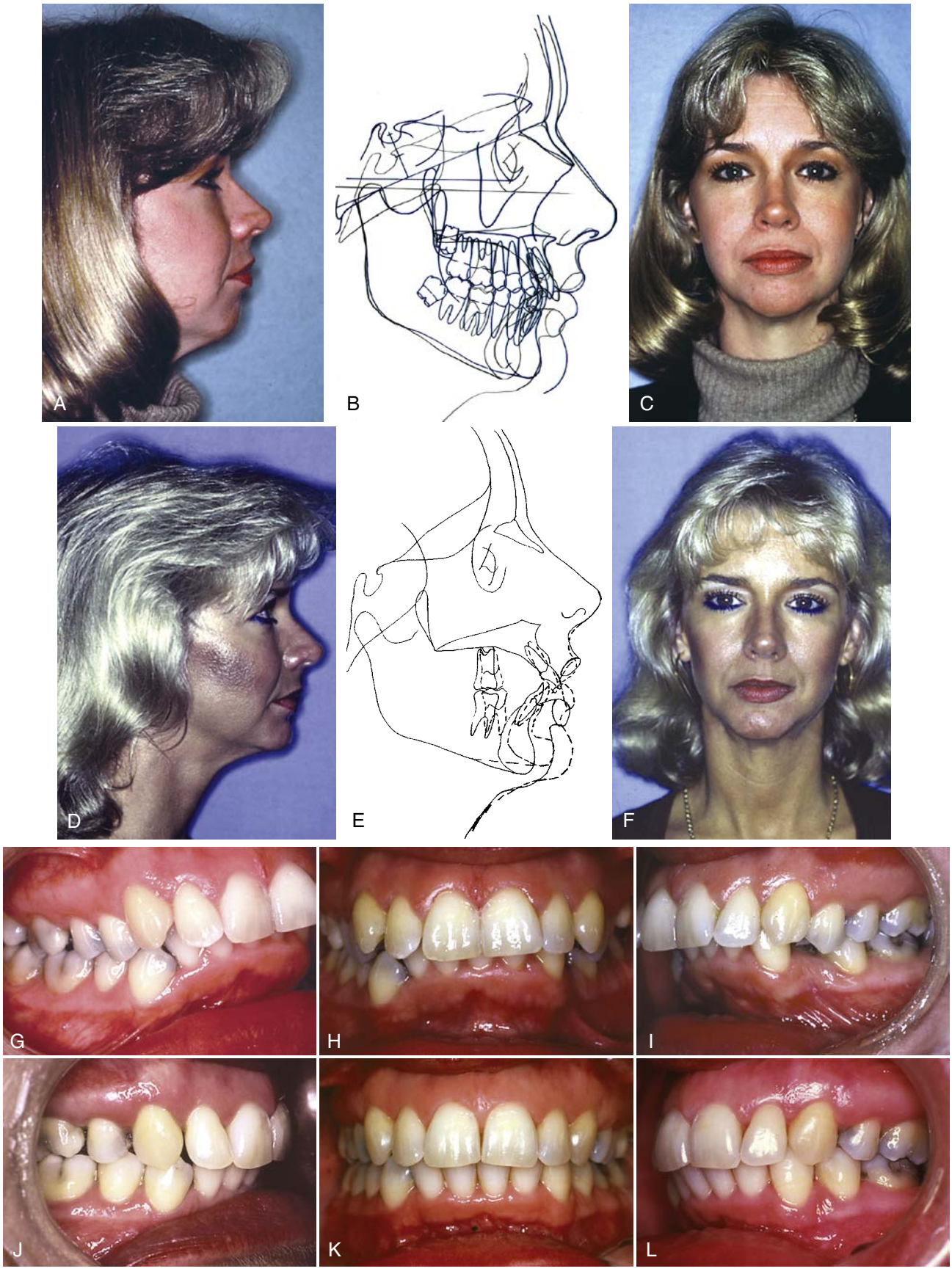


FIGURE 23-29 A, Pretreatment profile photograph. Note protrusive lip and minimal throat and chin definition. B, Patient tracing with Bolton template indicates maxillary dentoalveolar protrusion and mandibular deficiency with chin deficiency. C, Pretreatment facial photograph. D, Posttreatment profile photograph showing improved throat and chin definition because of soft tissue "stretch" from successful chin implant. E, Superimposition of lateral cephalogram tracing showing dental retraction of upper incisors (camouflage skeletal Class II) and improved hard and soft tissue in chin area. F, Posttreatment facial photograph. G–I, Pretreatment intraoral photographs. J–L, Posttreatment intraoral photographs.

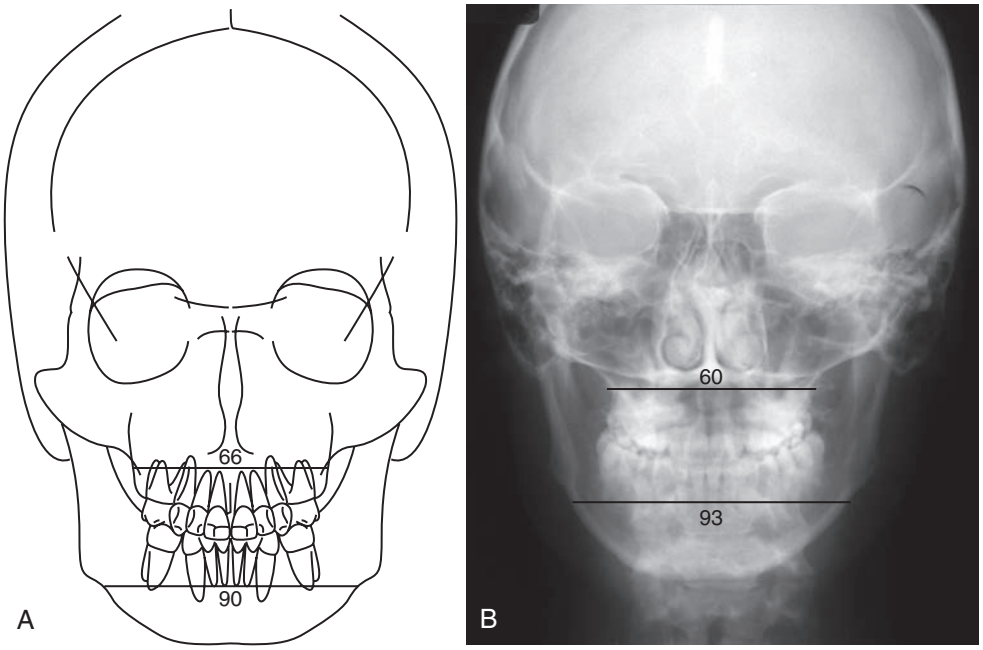


FIGURE 23-30 A, Composite frontal tracing of Bolton standard for adults. The normal maxillary width is 66 mm, and the mandibular width is 90 mm, as measured from 18 adults from 5000 who were determined to be the standard (normal) with a maxillomandibular differential of 24 mm. B, Frontal cephalogram of a woman who required orthodontic retreatment because of inadequate correction of maxillary–skeletal transverse deficiency. This patient had a maxillomandibular differential of 9 mm and required surgical expansion in conjunction with her orthodontic therapy.

both the maxilla and mandible are major components of the problem, both jaws may require surgery. If the patient accepts the optimal treatment plan and when the result achieved matches the patient's, orthodontist's, and maxillofacial surgeon's expectations, optimal outcomes result (see Figs. 23-8, 23-11, and 23-12).

Treatment planning options for cases with *skeletal (basal bone discrepancy) components* affecting the malocclusion include:

- Mild skeletal factors: Comprehensive orthodontics is generally effective.
- Moderate skeletal factors (with growth): comprehensive orthodontics + orthopedics + camouflage with the need to periodically reevaluate the response while growth proceeds[88]
- Moderate skeletal factors (without growth): comprehensive orthodontics + camouflage + skeletal anchorage to amplify the camouflage effort[89]
- Moderate to severe skeletal factors: comprehensive orthodontics + one-jaw surgery
- Severe skeletal imbalance: comprehensive orthodontics + one- or two-jaw surgery; considerations for two-stage orthognathics if transverse discrepancy is also severe
- Moderate skeletal (without growth) and moderate to severe skeletal factors with dentoalveolar components: may be candidates for SFOT, as discussed previously in this chapter and presented by Roblee et al.[58] It is to be noted that a recent systematic review of the literature on the topic of corticotomy assisted tooth by Hoogeveen et al.[90] revealed:
  - There is need for more well-designed studies on this topic because only 18 of 510 articles met the criteria for inclusion in the systematic review.
  - Corticotomy-facilitated orthodontics temporarily enhanced the rate of tooth movement.
  - There were minimal complications.

Indications for Camouflage Treatment

To illustrate the treatment planning process further, one should be able to answer this question: What makes a borderline orthodontic–surgical case into a successful camouflage treatment? Craniofacial characteristics make up only part of several important considerations in determining the effectiveness of camouflage treatment (see Figs. 23-1 and 23-31 and Box 23-4).

Other important considerations include the following:

1. Moderate basal bone discrepancy as determined by conventional cephalometric standards
2. Double-jaw involvement in the discrepancy; that is, 50% of the skeletal problem is due to maxillary AP excess, and the remaining 50% of the AP problem is related to mandibular deficiency (preferably with some chin contour). This allows the appearance of a more balanced soft tissue profile in the presence of a large AP discrepancy, thereby allowing more latitude for dental compensations.[91] One effective mechanism to assess this discrepancy quantitatively is by comparison of the patient's cephalogram with the Bolton template using nasal superimposition and Frankfurt horizontal (FH) parallel to the patient's FH (see Fig. 23-1).
3. Adequate alveolar bone and gingival tissue for incisor reangulation: Sometimes gingival grafting procedures are indicated to allow for the compensatory movements, and this is usually acceptable to achieve proper incisor guidance.
4. Patient acceptance of the possibility of long-term lingual arch bonded to mandibular anterior teeth

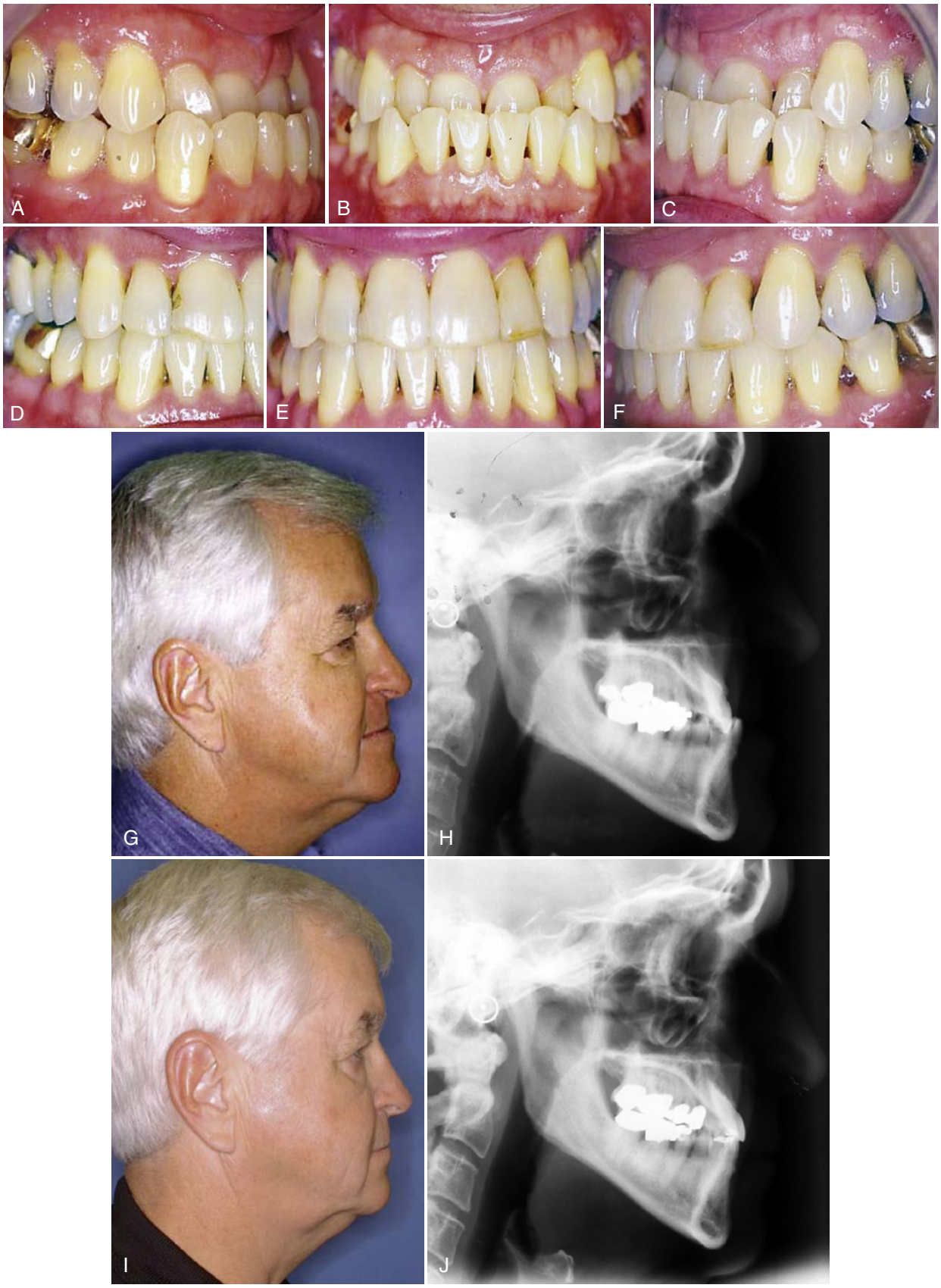


FIGURE 23-31 A–C, Intraoral photographs before any orthodontic treatment. Note the degree of anterior crossbite in this 62-year-old man. This functional imbalance is now causing tooth fracture and accelerated wear. D–F, Intraoral photographs after orthodontic treatment. Principles of therapeutic diagnosis were applied to determine if nonsurgical, or camouflage, treatment could be incorporated to solve this long-standing bite problem. Although a significant skeletal imbalance was present, the skeletal Class III problem was made worse because of incisal prematurity that forced the patient to shift his mandible forward 4 mm. G and H, Pretreatment profile photograph and pretreatment lateral cephalogram, respectively. I and J, Posttreatment profile photograph and posttreatment lateral cephalogram, respectively.

BOX 23-4 Camouflage Checklist for Class II Patients

- Skeletal malrelationship with Wits differential +4 mm or greater
- Bolton template superimposition (as described in this chapter)
- With almost equal portions of the Wits differential being contributed by maxillary basal (measured by "A" point) excess and mandibular deficiency (measured by "B" point) compared to the template. When the skeletal differential is present because of each jaw playing a part, as it relates to the nose, camouflage can more easily occur without having deleterious effects on soft tissue aesthetics. In other words, after upper premolar extractions, facial balance continues to be proportionate.
- If the cephalogram shows a Wits differential of +7, for example, and the template shows that the differential is all because of mandibular deficiency, camouflage therapy may help reduce the overjet, but it will result in overretraction of the upper incisors, require a great deal of incisal root torque (possible root resorption), and accelerate the aging of the face.
- Moderate upper incisor crowding and minimal lower incisor crowding also helps to justify camouflage because correcting the anterior alignment limits the degree of retraction and there is less impact on negative soft tissue changes of the upper lip.
- Patient acceptance of the possibility of long-term bonded lingual arch to lower anteriors
- If slightly more of the mandibular deficiency (Class II) is in the mandible, then to preserve upper lip support, the lower incisors may have to be compensated (proclined) more. In such cases, the amount of attached gingival of the lower anterior segment needs to be assessed. If the tissue is thin or deficient, gingival grafting may be needed to avoid gingival recession during proclination. In addition, the long-term stability of the lower anterior segment may require a lifetime bonded lower lingual arch.
- Another consideration is the aesthetic balance of the chin. Is there enough chin to create facial balance? Will proclination of the lower incisors reduce the effective chin to an undesirable aesthetic position? Should advancement genioplasty be considered and discussed with the patient? How much less risk does the advancement genioplasty carry than a mandibular advancement in this case?
- Stability of the outcome should also be included in the considerations of which treatment plan would serve the patient the best. With the above considerations, nongrowing Class II, Division 1 patients with maxillary dentoalveolar protrusion, moderate maxillary crowding, and minimal mandibular deficiency are good candidates for successful camouflage therapy with upper premolar extractions.

In the successful planning for camouflage treatment, the foregoing factors determined by template-guided diagnosis and evaluation help to define the orthodontist's treatment planning limits without surgical intervention and give the patient an opportunity to consider available treatment alternatives.

Contraindications to Camouflage Treatment

When treatment planning moderate skeletal imbalances, one should be aware, however, that not all borderline cases can accommodate camouflage treatment planning. In general, camouflage therapy is contraindicated when single-jaw imbalances are severe; that is, a patient demonstrates, on clinical evaluation and cephalometric measurement confirmation, that the total skeletal Class II malocclusion is secondary to mandibular deficiency (see Figs. 23-2 and 23-23 to 23-25). Although an incisal relationship may be achieved with camouflage treatment, the resulting profile would be imbalanced; the upper incisors would be excessively retroinclined and the lower incisors excessively proclined to achieve incisal contact. The skeletal Class II case in which there are both maxillary and mandibular deficiencies (relative to nasal superimposition) is another case type for which camouflage treatment planning should be avoided. This case type is seldom presented in the literature unless it is referenced to sleep apnea in which both maxillary and mandibular surgery is medically indicated.[15,69]

Upper and Lower Jaw Skeletal–Dental Problem

The orthodontist should consider the following three steps in addressing a skeletal–dental problem of the upper and lower jaws:

1. Discuss treatment options: conventional and expanded options, including skeletal anchorage[92] to enhance dentoalveolar changes as well as SFOT.[58]
2. Analyze anticipated outcomes.
3. Consider the tradeoffs that may result from non-orthognathic surgery management.

Therapeutic Diagnosis of Borderline Cases

Because of the range of treatment plan options, the concept of therapeutic diagnosis is even more important today than when it was first introduced. Ackerman and Proffit clarify this diagnostic approach in the following quotation:

> *Therapeutic diagnosis is not a substitute for established procedures, nor should it become a cover for fuzzy thinking and diagnosis. Where uncertainty exists despite a careful diagnostic evaluation, there is danger in formulating a rigid treatment plan. Systematic evaluation of the initial response to orthodontic treatment can help a great deal in making the difficult diagnostic and treatment planning decisions, especially as concerns the basic question of extraction or nonextraction. Borderline cases in which the treatment response should be considered before one decides to extract are more common than many diagnostic systems indicate. It is strength, not weakness, to recognize true uncertainty.*[93]

The process calls for the use of therapeutic aids indicated in Table 23-5. Borderline surgical–nonsurgical cases often require initial therapy followed by appropriate new records and reevaluation to allow a thorough review of treatment compromises if the surgical approach is not undertaken. A case may be a definite surgical case but borderline in terms of the number of osteotomies. Achieving a practical, safe objective rather than a hypothetical ideal is important. In essence, borderline surgical cases have moderate skeletal malrelationships that affect both jaws equally; an example is Class II maxillary protrusion and mandibular deficiency (see Fig. 23-29). In such cases, dental camouflage rather than surgical correction may be considered appropriate. Another example of a borderline case is a situation in which a surgical plan has been selected and a decision must be made between two-jaw and one-jaw surgery. For example, a Class III mandibular excess and vertical maxillary excess with a moderate mandibular asymmetry may achieve the best result with double-jaw surgery. An alternative procedure with one-jaw surgery would leave the vertical excess uncorrected. Sometimes the added risks of a double-jaw procedure, in relation to the minor gains anticipated, make a single osteotomy the procedure of choice. Occasionally, a patient will present with a situation that appears to require two-stage surgery (stage I for the skeletal transverse discrepancy and stage II for the AP and

TABLE 23-5 Therapeutic Aids

Appliance	Used to Assess	Duration
Bite splint	Neuromuscular habits and deprogramming muscles of mastication	1–6 months; reevaluate new cephalogram and mounted study models
Superior repositioning splint High-pull headgear orthopedic effect limited to growing patient	1. Degree of patient cooperation 2. Orthopedic response of Class II with vertical maxillary growth	6–12 months (possibility of continuation)
DeLaire face mask	1. Orthopedic response in Class III with mild to moderate maxillary deficiency 2. Maxillary horizontal growth acceleration	6–12 months (possibility of continuation)
Chin cup	1. Orthopedic response in Class III with mild mandibular excess 2. Restraint or redirection of mandibular growth, primarily in early years 3. Reprogramming of jaw muscles with pseudoskeletal Class III problems	6–24 months
Fixed appliances to level and align (also test compliance with cervical or high-pull headgear)	1. Combination maxillary protrusion and mandibular deficiency (Class II, Division 2 type) 2. Upper lip contour and option between upper premolar extraction and mandibular advancement	6 months

vertical problem). The patient illustrated in Figure 23-32 was treatment planned as a two-stage surgical patient, but after the surgically assisted rapid palatal expansion (SARPE) procedure, her open bite continued to improve to the point that we agreed to camouflage the Class II open bite with conventional orthodontic treatment.

Key Considerations of the Integrated Treatment Plan

After both the orthodontist and the surgeon have determined that a patient is ready for surgery, the surgeon will create a surgical prediction tracing or surgical treatment objective (STO) and model surgery.[53] There are now many computer programs that allow the surgeon to digitize a cephalometric radiograph and create a prediction tracing. These programs allow movements in any direction by entering the amount of movement at a given cephalometric point. Rustemeyer et al.[94] evaluated the accuracy of two-dimensional predictive software with the actual outcomes for 54 patients who underwent bilateral sagittal split ramus osteotomy (BSSRO) with or without a Le Fort procedure. Their findings indicated that there is some variance between the predicted outcome and the actual result, but they thought that the software was relatively accurate. Another method is to trace a cephalometric radiograph and create tracings on acetate paper to predict the surgical movement. The benefit of this method is that it provides a visual road map that can be used during the surgery as a guide to the desired result. A sample case is provided with the steps involved in creating the STO (see Fig. 23-14).

Step-by-step instructions are presented in Box 23-5. Using the patient's cephalometric tracing and simulating the surgical movements, this method shows the skeletal movement anticipated by moving the skeletal structures to normal positions. The magnitude of movement is shown at the reference lines both in the AP and vertical directions. The soft tissue is drawn and is variable, but most studies indicate that the AP movement of the upper lip is 50% of the skeletal movement.[54]

The image from 3D capture software shows no significant difference between the lateral cephalometric images when superimposed[78] (see Chapter 11).

A common occurrence in daily practice for orthodontists is to guide their patients toward a surgical treatment plan or away from a surgical treatment plan. This guidance depends on the orthodontist's training, personal experiences, professional experiences, and diagnostic data along with a clear awareness of the variable goals of treatment based on individual patient treatment requirements. In addition, the surgical or nonsurgical treatment planning decision often is made with the patient's own process of gathering detailed treatment information; a careful determination of the patient's own goals for comprehensive treatment; and, in many cases, on the availability of insurance coverage to assist in the fees for surgical aspects of treatment. As a result of this multilayered decision-making process, the orthodontist must define goals with the patient and establish a plan that may include several well-thought-out options and not just present a plan to the patient.

Borderline orthodontic cases provide orthodontists with excellent examples of the benefits of setting mutually defined treatment goals. The strategy for treating borderline orthodontic cases with camouflage therapy is to create dentoalveolar changes that will compensate for a skeletal base imbalance. Although clinical orthodontists commonly use this approach in treatment planning, there is a limit to how much dentoalveolar change can compensate for an imbalanced skeletal structure. To discuss the limits of camouflage properly, one must define the goals of treatment clearly. Because orthodontic treatment is elective, the orthodontist and the patient must visualize the goals of treatment in the same way. The orthodontist, for example, may have a clear concept of goals for treatment as taught during the orthodontic graduate program. However, orthodontic providers must individualize the treatment goals for each adult surgical patient through a detailed interview process that starts at the initial examination appointment and continues through the review of the diagnostic records at the treatment planning conference with the patient.

The treatment planning process for patients with malocclusions plays a pivotal role in guiding the patient to a treatment that will satisfy his or her short-term concerns and long-term functional requirements and establish reasonable stability. Although the three steps listed previously seem simple, in several specific treatment planning modalities, orthodontists must strive to gain expertise to achieve maximal effectiveness. With each of these modalities, the orthodontist should be able to answer the following pertinent questions:

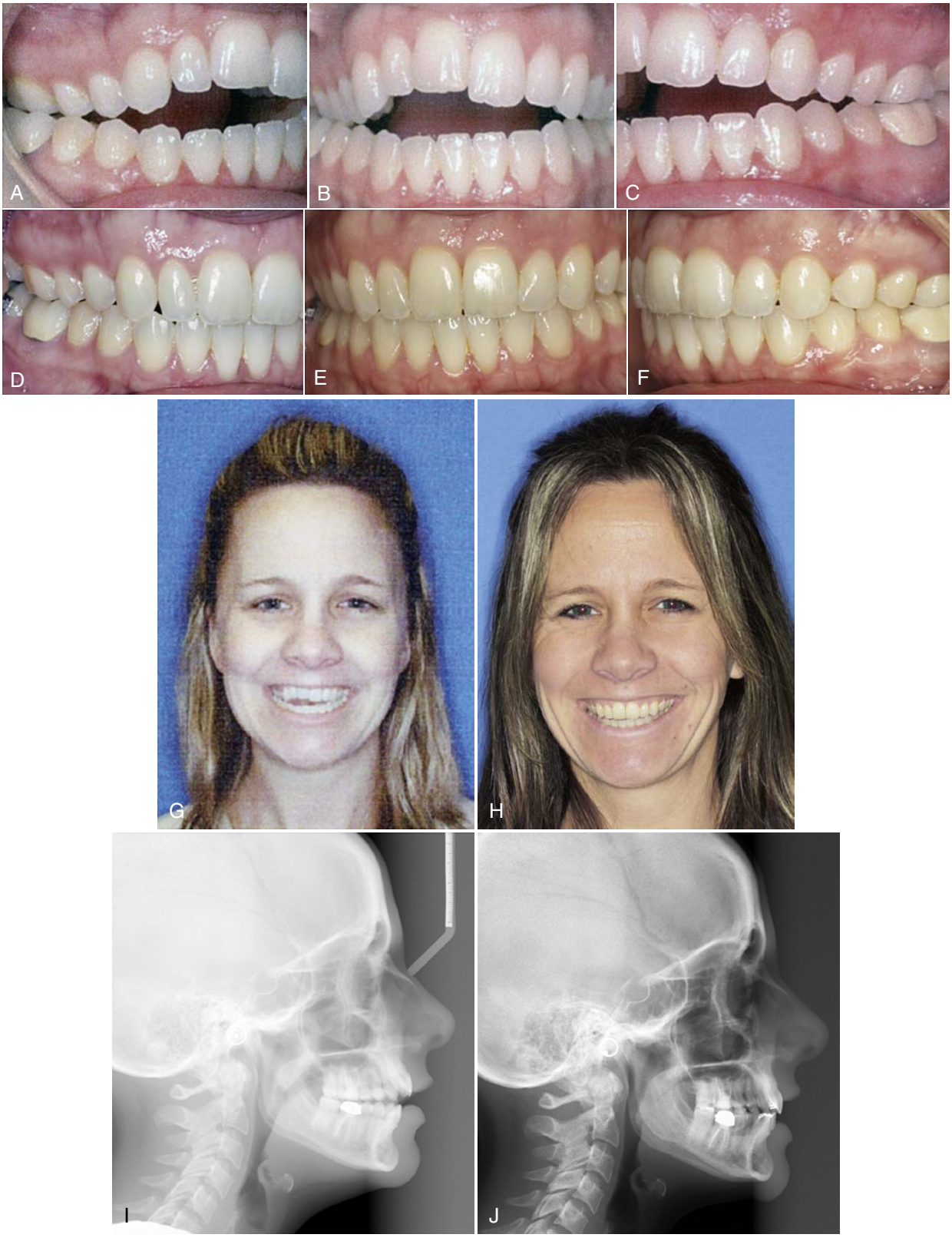


FIGURE 23-32 A–C, Intraoral photographs before any orthodontic treatment; note the degree of anterior open bite and arch constriction with posterior crossbite. Incisor position is consistent with an ankylosed no. 8. Previous trauma to the area of nos. 8 and 9 was ruled out, and the results of percussion test for ankylosis of nos. 8 and 9 were negative. D–F, Intraoral photographs after orthodontic treatment and orthognathic surgery. This patient was originally treatment planned for two-stage surgery. She responded sufficiently well to stage I surgery of surgically assisted rapid palatal expansion procedure and orthodontic movement that the patient, the surgeon, and the orthodontist agreed to complete treatment without the second-stage Le Fort procedure that was anticipated. G and H, Frontal facial photographs showing pretreatment smile and posttreatment smile, respectively. I and J, Lateral cephalograms illustrating pretreatment and posttreatment smile, respectively. Initially, incisor position showed that the incisor could be erupted to allow more incisal display. Long-term stability and soft tissue (tongue) adaptation is being monitored. Long-term tongue crib may be indicated. (Surgery by Dr. Peter Chemello, Arlington Heights, IL.)

BOX 23-5 Orthognathic–Orthodontic Treatment Milestones for the Typical Orthognathic Surgical Patient

Evaluation (2–3 mo)
- Orthodontic records
- Diagnosis and treatment plan
- Surgical consultation

Presurgical Orthodontics (6–18 mo)
- Presurgical alignment
- Presurgical records
- Presurgical reconsult with surgeon

Surgical Treatment
- Orthodontic placement of surgical wires or hooks
- Surgical correction
- Postsurgical records and reevaluation

Postsurgical Orthodontics (6–12 mo)
- Postsurgical tooth alignment to finalize bite correction
- To complete arch coordination

Retention and Observation (12–18 mo)
- Observe tooth "settling"
- Monitor retainers
- Bite adjustment as needed

1. **Clarification and individualization of treatment plan for patients with skeletal malocclusion**
   - Is it imperative that every patient be guided to a treatment plan exemplifying a textbook ideal result?
   - When will a "practical," nonsurgical result suffice?
   - Whose concept of facial aesthetics and balance takes precedence: that of the patient, the orthodontist, the surgeon, the parent(s), or the significant other?
   - Is it acceptable to achieve stability with mechanical approaches (e.g., lifetime bonded retainers, lifetime removable retainers, or both)?
2. **Differential diagnosis**
   - What is a borderline surgical case? What are the risks if a patient with a severe skeletal problem does not accept surgery?
   - At what point does a stomatognathic system skeletal imbalance change from a nonsurgical problem to a surgical problem?
   - Is the surgical–nonsurgical decision made by the patient, the orthodontist, the surgeon, or the insurance company?
3. **Extractions**
   - Will extraction of teeth be effective for orthodontically camouflaging the malocclusion?
   - Is extraction of premolars needed to decompensate a condition before surgery?
   - Is extraction of premolars being planned for alignment purposes only?
   - Should molar or premolar extraction be planned to maximize autorotation of the mandible?
   - What happens when several of these extraction considerations are applicable to one patient?
   - Should the extraction decision be made even though the final treatment plan has not been defined?
4. **Is there a clear cephalometric measure of an "acceptable compromise"?**[40]
   - Can patients with a +5 degree or greater A point–nasion–B point (ANB) difference or a −3 degree ANB difference have an acceptable result without surgery? If so, under what conditions?
   - Should the concept of acceptable compromise be redefined with the understanding of new surgical capabilities?
5. **Visualized treatment objectives**
   - Can cephalometric predictions be achieved routinely?
   - Do patients have an overly optimistic perception of the outcome?[17,32]
   - Will computerized 3D predictions be an integral part of visual treatment objectives in the future (see Chapter 8)?[8,88]
   - Is computerized imaging a treatment planning tool, a marketing tool, or both?[78,95]
   - Are a patient's presurgical expectations a predictor of postsurgical satisfaction?
6. **Patient expectations**[8,22,29,96–98]
   - What percentage of surgical-orthodontic patients eventually indicate that they are satisfied with the treatment outcome?
   - Are immediate postsurgical patients prone to be less satisfied than expected? If so, why?
   - Will the planned procedures affect facial aging?

Treatment Timing

The orthodontist generally is the key person in deciding the appropriate time to initiate treatment plans that fall into one of the following categories:

1. Orthodontic only
2. Orthopedic and orthodontic (growth modification)
3. Orthodontic and surgical
4. Independent arch alignment with nighttime splint therapy to provide interim occlusal "balance"

General concepts that are accepted for specific treatment problems are reviewed in this section. The principles presented must be applied to each individual patient.

Treatment Timing for Maxillary Deficiency Patients

Deficiency of the maxilla with AP and transverse components is managed best in the mixed dentition stage. The orthopedic effect is predictable, and the early treatment staging enhances the environment for future harmonious growth. The early permanent dentition stage is not too late to plan treatment for many patients with maxillary deficiency, but the stability of the results may be less than if the patient were treated earlier (see Chapter 16).

Treatment Timing for Mandibular Excess Patients

Patients with a family history of moderate to severe mandibular excess and who demonstrate growth patterns coincident with mandibular growth imbalance are treated best when growth is complete, as verified by wrist films and head-plate superimposition (Fig. 23-33).[99]

An important note is that some patients have mild mandibular excess, mild maxillary deficiency, and a functional forward shift of the mandible (Fig. 23-34). The combination of these factors creates the clinical appearance of a severe Class III mandibular excess pattern. Accurate recording of mandibular position, early orthopedics, and some compensatory camouflage (compensate upper incisor labially and lower incisor lingually)

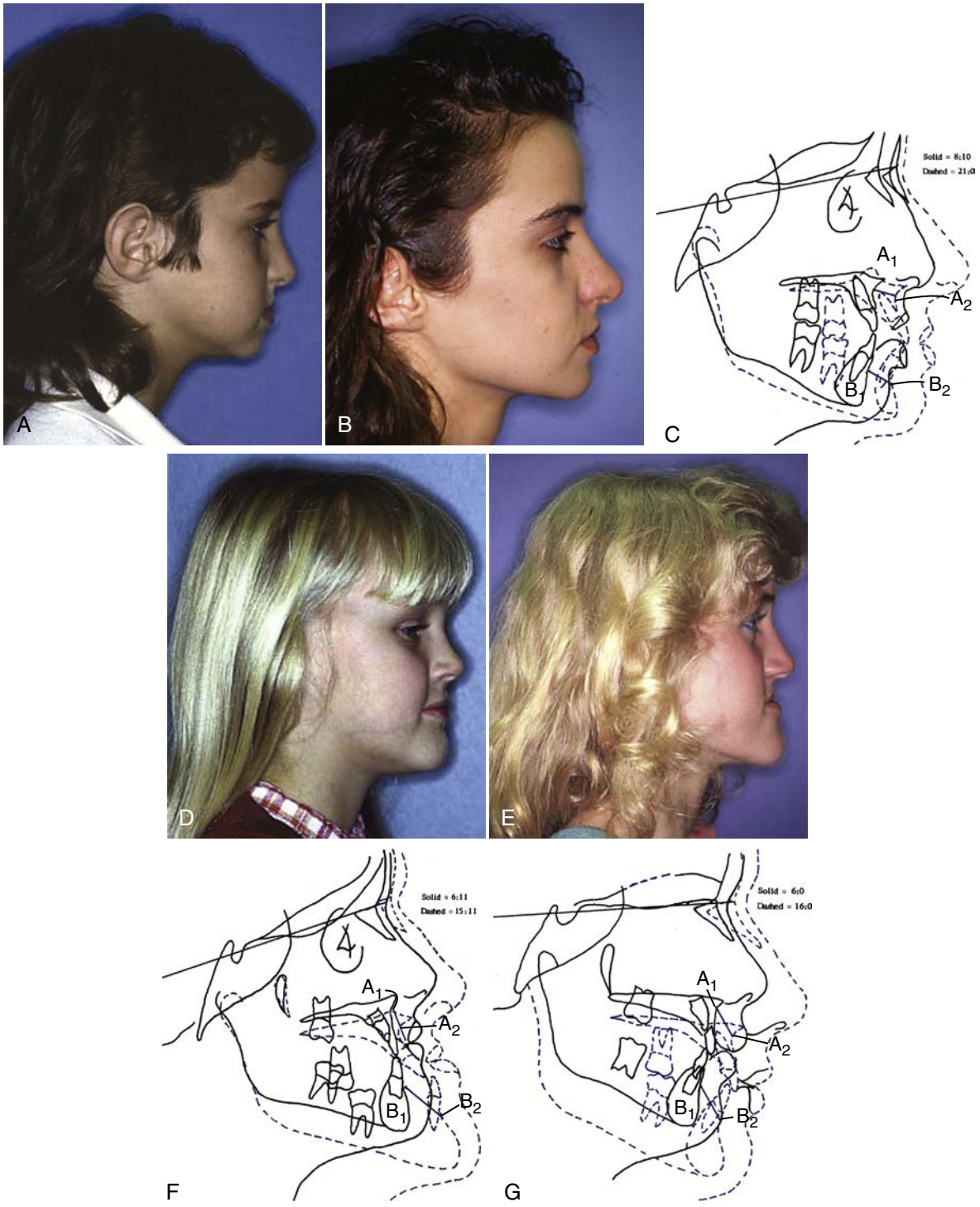


FIGURE 23-33 **A,** Pretreatment profile photograph of an 8-year-old patient who had full Class III malocclusion with anterior crossbite. She was treated with two-stage Class III orthopedics and orthodontics. **B,** Profile of same patient at age 21 years. Acceptable facial balance was achieved without surgical intervention. **C,** Cephalometric superimposition. Note favorable response because of early correction and favorable maxillary (*A1–A2*) and mandibular (*B1–B2*) growth vectors. **D,** Pretreatment profile photograph of 7-year-old girl who had a developing Class III problem. She was treated with Class III orthopedics. **E,** Profile at age 16 years showing unfavorable pattern of maxillary and mandibular growth. **F,** Cephalometric superimposition. Note the unfavorable response with orthopedic attempt at early correction and unfavorable maxillary (*A1–A2*) and mandibular (*B1–B2*) growth vectors. **G,** Cephalometric superimposition of Bolton template for the 6- to 16-year-old standard, illustrating maxillary and mandibular growth vectors for maxilla and mandible in patients with optimal occlusions and having proportionate facial growth.

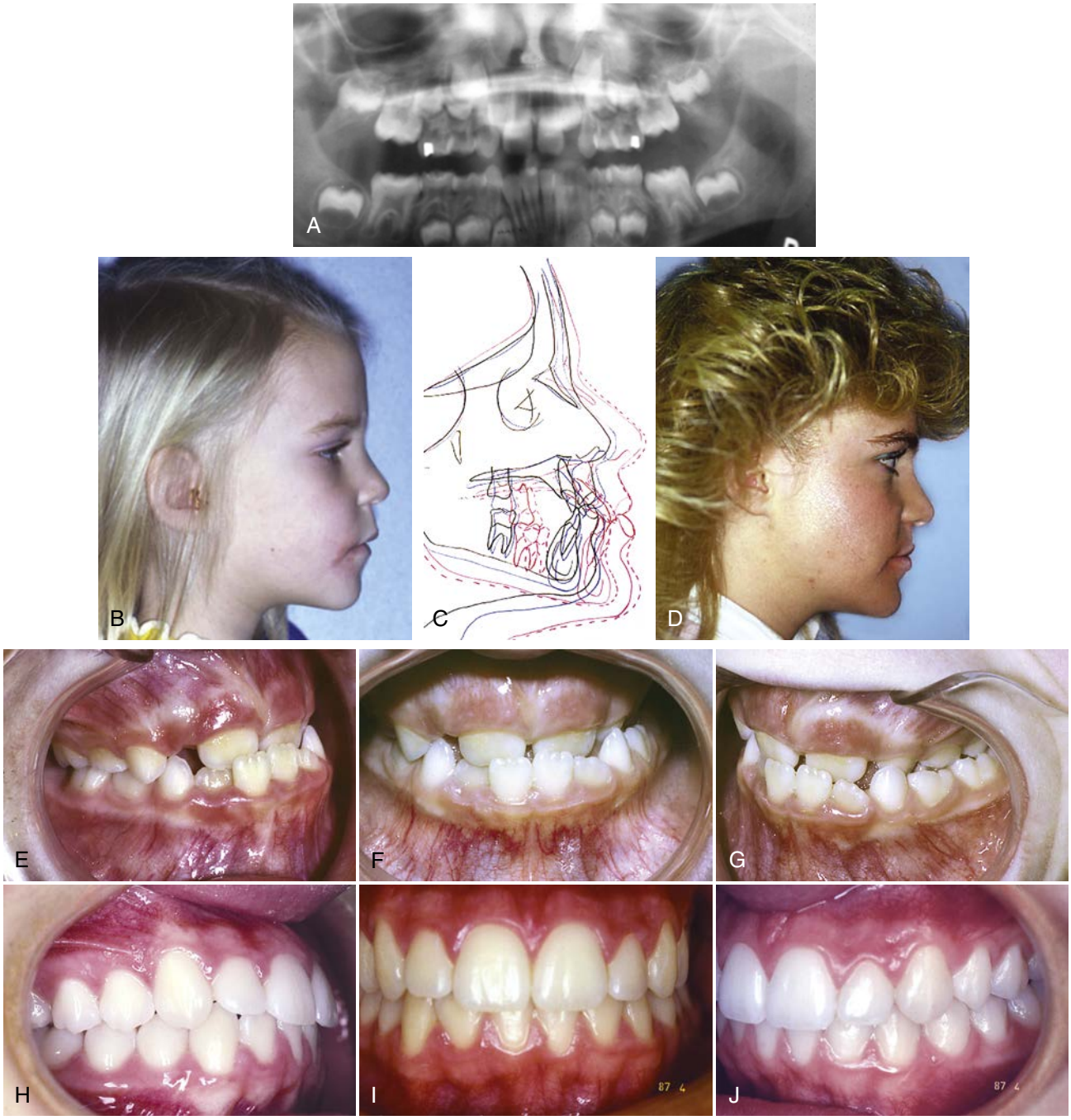


FIGURE 23-34 **A,** Pretreatment panoramic radiograph showing severe maxillary crowding. **B,** Pretreatment facial photograph at 6 years of age. **C,** Cephalometric tracing illustrating skeletal relationship at 6 years old (*solid black*), 8.5 years old (*solid blue*), 16 years old (*solid red*), and 18 years old (*dashed red*). **D,** Facial photograph taken 3 years after treatment during the retention period. **E–G,** Pretreatment intraoral photographs of a 6-year-old girl with the appearance of a significant Class III problem. A functional shift of the mandible to the left and forward has occurred, which makes the problem look worse than it is. **H–J,** Posttreatment photographs illustrating corrected Class III malocclusion. This borderline Class III surgical problem was able to be treated nonsurgically because of early intervention.

BOX 23-6 Phase I Orthodontic Milestones for Patients with Skeletal Components to Their Malocclusion (Ages 4–12 Years)

Evaluation (1–3 mo)
- Orthodontic records
- Diagnosis and treatment plan

Appliances in Place (12–18 mo)
- Growth and treatment response monitored (patient cooperation is essential)
- Remind parents and patients of the importance of compliance

Observation (6–12 mo)
- If favorable progress, go to stabilization program
- Evaluate growth treatment response vectors (GTRV)

Periodic Recall (18–24 mo)
- Continue growth guidance and monitor dental development
- Reevaluate GTRV

Phase II
- Evaluate for new problems
- Full braces to complete bite correction
- Additional orthopedics possible
- If poor response—wait and plan orthognathics

may allow satisfactory nonsurgical management. This type of patient benefits from therapeutic diagnosis and periodic reevaluation of growth. One must take care not to overtreat these patients orthodontically through camouflage if the mandibular excess growth continues, however. Orthodontic overtreatment can lead to an unstable result, traumatic incisor occlusion, incisor mobility, and gingival recession (Box 23-6, Phase I, Orthodontic Milestones).

Assessment of the growth treatment response vector (GTRV) (see Fig. 23-33) is an important tool to be used by an orthodontist when trying to determine if a Class III mandibular excess and Class III maxillary deficiency patient is growing outside the range of nonsurgical correction.[100,101]

Treatment Timing for Mandibular Deficiency Patients

The degree of mandibular deficiency (mild, moderate, or severe) and the type of the deficiency (type I [low angle], type II [medium angle], or type III [high angle]) are factors in deciding the timing of treatment.

Most orthodontists attempt conservative (nonsurgical) measures in the 14- to 16-year-old age group to limit maxillary horizontal growth and enhance mandibular growth. The mandibular growth rate slows earlier in girls than in boys. Therefore, for girls who have severe cases of pure mandibular deficiency or mandibular deficiency with asymmetry, surgery can be recommended along with orthodontic treatment in this age group.[102,103]

In boys, there is evidence of protracted mandibular growth until age 21 to 23 years. Although a mandibular deficiency will not self-correct, the maxillomandibular growth differential can be used with directional force orthopedics to improve significantly many low and medium mandibular plane angles to the point that surgery is not necessary.

With moderate or severe type III mandibular deficiency, boys and girls usually require orthodontics and surgery to achieve facial balance, stability, and good function. The timing of surgical intervention in these cases ultimately depends on (1) growth factors, (2) emotional maturity, (3) self-image considerations, (4) school and work plans, (5) insurance factors, and (6) the patient's and family's perception of relative need.[17,96,104]

Considerations in the Management of Facial Asymmetry

One of the more challenging treatment planning decisions that orthodontists face is the management of facial asymmetry. Key questions must be addressed early in the analysis of the case:

1. **Is the developing asymmetry a result of trauma?** A careful history must be taken. An excellent discussion of acute care of a condylar fracture can be found in Proffit and White's textbook.[40]
2. **Is the asymmetry affecting the maxilla and the mandible?** Is it getting worse with time? Panoral radiographs and serial posteroanterior cephalograms, as well as a submental vertex radiograph, are needed to aid in this assessment.
3. **Would functional appliance therapy be effective in this case?** Some authors have shown good improvement using a functional appliance approach (see Chapter 14). Severe cases of hemifacial microsomia, however, may require orthopedic treatment to prepare for staged surgery to correct the growth imbalance.
4. **What is the appropriate timing for surgical intervention?** Early consultation with a surgeon familiar with the range of considerations in asymmetry cases is desirable. Periodic functional and emotional assessment also is desirable in preparing the patient for the corrective steps. Orthopedic measures can be taken, but orthodontists need to avoid patient burnout and disappointment if further surgery will be needed, especially in cases of moderate and severe asymmetry (see Fig. 23-12). A recent case report (2009) illustrates the effective use of miniscrew anchorage to intrude the overerupted maxillary molars in a case of mandibular asymmetry. The use of bone screw anchorage to level the compensatory cant of the maxillary molars allowed the patient to be treated with just mandibular surgery.[105]

In patients who have condylar hypertrophy, a scintigraphy analysis (isotope uptake) can help to determine the appropriate timing based on hot spot analysis. Waiting until the hypertrophy burns itself out is the most judicious way to proceed in these cases.

It is important to remember that facial asymmetries can have skeletal manifestations that need to be understood in treatment planning. In an asymmetric face, the body of the mandible may be vertically larger than the opposite side. Although the dentition can be leveled via orthognathic surgery, there still may be a residual facial asymmetry because of a size discrepancy in the mandible. The patient needs to be aware of this discrepancy so that decisions can be made to address the problem. Unilateral disc displacement without reduction (UDDN) may display altered craniofacial morphology and needs to be assessed, especially in growing patients.[106]

SEQUENCE OF TREATMENT FOR ORTHOGNATHIC PATIENTS

Although many believe that the sequence of treatment starts with the interview and examination of patients with skeletal jaw imbalance, the most important steps take place long before the surgeon or orthodontist meets the patient. The feedback

loop sequence described in Box 23-2 has 17 significant steps that provide the patient and doctors with an excellent means of achieving the desired treatment outcome.

Importance of the Sequence

In the sequence of treatment for patients with major dentofacial imbalances (see Box 23-2), steps 1, 2, and 17 are most often underemphasized. Because of this deficiency in private practice, these steps of the treatment sequence are reviewed to emphasize the reasons for their importance.

Step 1: Multiple Provider Team Selection (Interdisciplinary Therapy)

The dentist will refer patients with skeletal malocclusion to an orthodontist if the dentist is confident that the patients will receive appropriate state-of-the-art care. The orthodontist must select surgeons who are experienced and well trained in the spectrum of orthognathic procedures. A periodontist, psychologist, and physical therapist may become key team members in selected cases. An additional consideration is the desirability to continue to educate the orthodontic staff who will be on the "front line" of questions from potential orthognathic patients. It might even be advisable to invite an oral surgeon to an orthodontist's office to discuss critical communication aspects of patient care for surgical cases.[107]

Step 2: Goal Clarification

The members of the provider team should share similar concepts of treatment goals. Functional occlusal goals, stability of outcome, and dental and facial aesthetic goals can mean different things to different professionals. Periodic group meetings oriented to individual case analysis offer an opportunity for team members to discuss their concepts of the treatment goals that apply to the individual patient (see Box 23-1). For example, Arnett and Gunson discuss how their *systematic* approach to assess and treatment plan orthognathic cases facilitates the orthodontic preparation for surgery. Their article emphasizes the importance of manipulation of the occlusal plane to achieve the most desirable result.[108]

Steps 3 Through 16

Steps 3 through 16 are self-explanatory and commonly performed by provider groups. By following the general concepts stated in each step, the orthodontist automatically spends time with the patient and can address most of the patient's pre- and postsurgical concerns.

The orthodontist can explain the sequence of treatment to the patient and the general dentist through the use of a treatment conference report.

Orthodontists and their staffs must be reminded constantly, through appropriate chart designation, of patients who have orthognathic treatment plans. These patients often are subject to increased anxiety and have extra questions that need to be addressed to maintain their confidence and trust. At the presurgical consultation (step 12), the orthodontist and the surgeon must listen to the patient and determine the patient's physical, dental, and emotional readiness for surgery.[17,28,29,96]

The surgeon should present a clear review of the surgical procedure and the risks of surgery. The surgeon and the orthodontist must remember that most orthognathic patients have much to learn about their options, the recommended procedures, and the incumbent risks. The current trend to have a "one-step consultation" for orthodontic patients generally does not apply to orthognathic patients. One must respect the individual patient's "learning curve" requirements and be supportive by providing needed information and patience.

Several authors have done numerous studies assessing patients' responses to current ortho-surgical treatment modalities.[17,28,29,96,98] A thorough review of their work is necessary to help orthodontists and their staff members appreciate the range of feelings that orthognathic patients report before and after surgical procedures. Table 23-6 describes the psychological considerations during the various treatment steps for a patient who is having jaw surgery.

Step 17: Treatment Experience

Step 17 is seldom incorporated in the routine policy sequence of private orthodontic and surgical practices. To gain the most from this step, all team members should review postsurgical records and discuss the positive and negative aspects of the overall treatment plan, the sequence of treatment, and provider interaction. Each completed case provides an opportunity for providers to consider feedback from one another and the patient. This step is the most effective continuing education program in which a practitioner can participate. Having each orthognathic patient complete a posttreatment questionnaire also provides additional feedback and is an integral part of practice maturation.

APPLIED CONCEPTS FOR SPECIFIC TYPES OF CASES

Certain concepts that have been discussed in this chapter are integrated and applied to case types previously introduced in the differential diagnosis section. A summary of the concepts that are fundamental to consistent excellence in the management of skeletal disharmonies is as follows:

- *Treatment goals*: These are defined and agreed on in principle by the providers (see Box 23-1).
- *Technique advancements*: These are used to enhance the safety of all orthognathic procedures.[26,52,55,100,101]
- *Current capacity to alter dentofacial structures:* Because the potential degree of change is so great, the responsibility of the surgeon and the orthodontist in planning treatment also is great. Psychosocial and emotional balance may be at risk.[96,109]
- *Differential diagnosis process*: The differential diagnosis process generates a problem list and treatment planning options. The so-called textbook ideal result should be considered and discussed with all patients; however, not all patients have the resources for state-of-the-art treatment.
- *Treatment planning*: Treatment planning therefore creates alternatives. The good, better, best approach to patient education yields understanding and trust.
- *Borderline cases*: Borderline cases (e.g., Class II with moderate lower jaw deficiency) can benefit from therapeutic diagnosis with initial leveling and alignment. Facial balance then can be assessed with the mandible postured forward. Computer and video imaging also can be used to teach patients about their options and potential outcomes.[79,110]
- *Standardized sequencing*: Following the 17 steps presented in Box 23-2 ensures a well-educated patient and a well-prepared treatment team.

TABLE 23-6 Psychological Considerations in Clinical Interaction

Treatment Stage	Steps in Psychological Management
Initial assessment	Explore motives for treatment and expectations from treatment in detail. Why treatment? Why now instead of last or next year?
	Consider using an auxiliary with a warm personality to do at least part of the interview. Patients often are intimidated by the doctor and may not reveal their true concerns.
	Be careful not to create unrealistic expectations: it is better not to discuss the specifics of treatment procedures before the diagnostic workup has been completed, but a broad outline including the possibility of surgery should be presented.
Orthodontic consultation	The spouse, a family member, or a close friend should attend if at all possible, but be careful about invasion of patient's privacy.
	Begin with a discussion of the patient's problems; be sure the doctor and patient agree as to what is most important; then describe how the problems might be solved, beginning with the most important problem and presenting the alternatives.
	Encourage—indeed, insist on—an early appointment with the surgeon if surgical treatment may be needed.
Surgical consultation	Review patient's records with same general approach as the orthodontist, emphasizing the problems and their possible solutions.
	Discuss the functional and aesthetic benefits of surgery in that order. To tell patients they will have functional benefits and aesthetic changes is better than the reverse. Functional changes nearly always are appreciated; aesthetic changes may not be.
	Provide more detail about the surgical experience, to the extent the patient has questions, but keep the discussion relatively general. Many details can wait until just before surgery. (Some patients may want a complete discussion.)
	Consider using patient education materials at this stage, such as booklets, videocassettes, and videodiscs.
	Offer to help with insurance preauthorization.
Presurgical treatment	Evaluate the patient's personality characteristics and psychological stability in more detail. Focus on neuroticism, degree of external locus of control, introversion in males, mood states (particularly depression and current major life events), and tendency for patient to be a vigilant coper.
Immediate surgery	Review the planned surgery in detail, but take the patient's psychological profile into account. Patients who expect the worst are more likely to experience it.
	Discuss with family members and close friends the importance of their psychological and emotional support postoperatively. Be sure significant others are prepared for changes in the patient's facial appearance so that they do not express shock or dismay.
Immediate postoperative period	Expect a period of mood swings and negative emotions, which usually peak at about 2 weeks. Reassure the patient, family, and friends that these emotions are normal and will soon disappear.
	A visit from the orthodontist and perhaps flowers or a care package of easy-to-eat foods keep the patient from feeling forgotten.
Postoperative orthodontics	Negative emotions and mood swings are more likely to be seen by the orthodontist in patients who return for finishing orthodontics. Reassurance and psychological support may be needed.
	Remember that a decrease in satisfaction and facial body image occurs if active treatment takes more than 6 months postoperatively. If treatment has not been completed by this time, progress should be reviewed with the patient, and an anticipated completion date should be discussed.

From Kiyak HA, Bell R. Psychosocial considerations in surgery and orthodontics. In: Proffit WR, White RP, eds. *Surgical Orthodontic Treatment.* St. Louis:, Mosby; 1990.

An important part of orthognathic treatment planning involves surgical prediction. Hard tissue changes are easy to predict; however, soft tissue changes are less predictable (see Chapter 11). In Table 23-7, a variety of general guidelines are used to predict soft tissue changes in the lateral cephalometric view. However, with current surgical treatments, the most difficult soft tissue predictions to make are those that deal with changes in the frontal view. The patients in Figures 23-23 and 23-25 illustrate the outcome of treatment using the concepts described in this chapter.

The patient in Figure 23-23 has a classic skeletal Class II mandibular deficiency type I, low mandibular plane angle, condition (see template assessment in Fig. 23-23, *B*). The patient in Figure 23-25 has a classic skeletal Class II mandibular deficiency type III, high mandibular plane angle (see template assessment in Fig. 23-25, *B*).

Figures 23-27 and 23-29 illustrate the treatment outcomes of three types of Class III skeletal mandibular excesses. The patient in Figure 23-27 represents a classic patient whose skeletal problem can be described as Class III mandibular excess type II (see the template superimposition in Fig. 23-27, *B*). Complete surgical correction of the mandibular excess depended on lower incisor decompensation from pretreatment incisor to mandibular plane angle of 77 degrees to presurgical angulation of 89 degrees.

The patient in Figure 23-28 demonstrates a classic example of mandibular excess type III (high mandibular plane with open bite). Generally, patients with high mandibular plane angles and open bites require a maxillary Le Fort procedure with impaction and a mandibular procedure. Maxillary and mandibular procedures were performed on this patient. The patient in Figure 23-35 illustrates a well-treated patient who was not fully satisfied with her treatment (see Fig. 23-35, *F*). Notice the improved response (see Fig. 23-36, *K*) to treatment, using more current techniques of rigid fixation, in the patient who had a similar complicated skeletal problem (Fig. 23-36).

OPTIMIZING STABILITY

Of the treatment goals discussed in the chapter introduction, orthodontists and surgeons are alike in their quest to recommend and perform procedures that give their patients lasting stability.[111–113] The stability of orthognathic procedures has been one of the most studied aspects of the techniques in use.[26,42,114–118] Stability is a key goal of orthodontic treatment and orthognathic treatment; lack of stability is considered a complication, particularly as it relates to the surgical aspect of the correction. To discuss the general concept of stability, one must consider several factors necessary to achieve stability (see Chapter 33).

TABLE 23-7 Soft Tissue Changes Related to Specific Surgical Movements

Treatment	Soft Tissue Change	Notes
Anteroposterior movement of incisors: maxillary or mandibular, forward or back, surgical or orthodontic	60%–70% of incisor movement Minimal unless jaw rotates	1–5
Vertical movement of incisors	Soft tissue: chin 1:1 with bone; lower lip 60%–70% with incisor	6
Mandibular advancement	Nose: slight elevation of tip	7, 8
Maxillary advancement	Base of upper lip: 20% of point A Upper lip: 60% of incisor protraction, shortens 1 to 2 mm	
Mandibular setback	Chin: 1:1 Lip: 60%	5
Maxillary setback	Nose: no effect Base of upper lip: 20% of point A Upper lip: 60% of incisor Advancement of lower lip: variable; may move back	3
Mandibular setback plus maxillary advancement	Changes similar to a combination of the two procedures separately	
Maxillary superior repositioning	Nose: usually no effect Upper lip: shortens 1 to 2 mm Lower lip: rotates 1:1 with mandible	7
Mandibular advancement plus maxillary superior repositioning	Chin: 1:1 Lower lip: 70% of incisor Upper lip: shortens 1–2 mm 80% of any incisor advancement Nose: slight elevation of tip	
Mandibular inferior border repositioning	Soft tissue forward: 60%–70% bone Chin: Up—1:1 with bone Back—50% bone Laterally—60% bone Down—?	

1. Little difference occurred with surgery or orthodontics.
2. If both upper and lower incisors are retracted (bimaxillary protrusion), lip movement stops when lips come into contact.
3. Lip shortens 1 to 2 mm with vestibular incision (more if surgical technique is poor).
4. Lip rotates with mandible 1:1.
5. If face height increases, lip may uncurl and lengthen.
6. If lip uncurls, it will go forward less.
7. Nose change is usually temporary.
8. Less soft tissue change occurs after cleft lip repair.

Data from Jensen AC, Sinclair PM, Wolford IM. Soft tissue changes associated with double-jaw surgery, *Am J Orthod Dentofac Orthop* 1992;101(3):266.

Orthodontic Considerations

1. Differential diagnosis[119]
2. Growth factors and the timing of the referral[12,120]
3. TMJ stability when planning the treatment[121]
   - Centric relation–habitual occlusion discrepancies[10,122]
   - Asymmetric growers
4. Dental stability
   - Crown-to-root ratio is important (periodontal health must be maintained).
   - Temporary crowns can be a source of instability during intradental fixation.
5. Presurgical orthodontic preparation to reduce dental compensations

Stability

- Orthodontic considerations
- Dental stability
- Presurgical orthodontic treatment to eliminate dental compensations
- Surgical considerations

Surgical Considerations

1. Treatment plan correlated with diagnosis
   - Surgery on which jaw
   - Single- or double-jaw procedures, especially for open bite and Class III situations[113]: Al-Delayme et al. assessed two Class III groups to compare the stability of two surgical procedures. One group exhibited mandibular setback only and the other with double-jaw surgery (maxillary advancement and mandibular setback). The authors found that patients who had double-jaw surgery were much more stable; the mandible-only group had about 44% relapse, and the lower incisors compensated by retroinclining 4 degrees to compensate for the mandibular AP relapse.
2. Design of osteotomy
   - Maintenance of blood supply and achievement of surgical objectives[44,47,123]
3. Type of fixation
   - Dental, skeletal, rigid, semirigid, or combination
4. Muscles of mastication and blood supply
   - In a sagittal split of the mandible, one is advised not to strip the pterygomasseteric attachment to avoid ischemia, delayed healing, and unusual nerve complications.
5. Type of mandibular rotation
   - Downward, backward movement is considered more stable than upward, forward movement.
   - Recent research has verified that occlusal plane change surgery and counterclockwise mandibular rotation can be very stable.[55]
6. The stability decreases as the magnitude of surgical correction increases, especially in the following:
   - Maxillary vertical deficiencies
   - Mandibular horizontal deficiencies

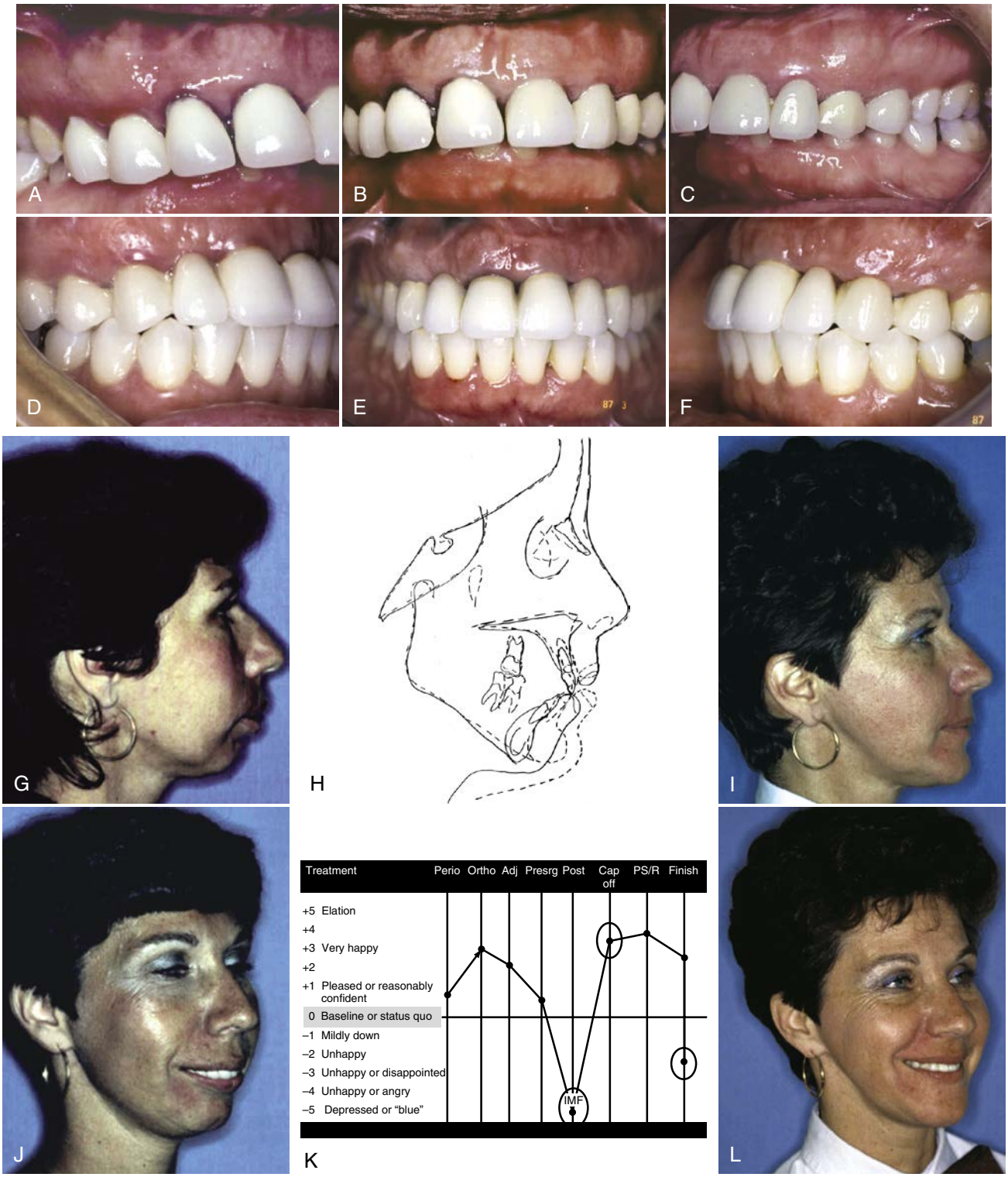


FIGURE 23-35 A–C, Pretreatment intraoral photographs. D–F, Posttreatment photographs. G, Pretreatment profile. H, Cephalometric superimposition; *solid line* indicates pretreatment contours, and *dashed line* indicates results after treatment. Treatment included the following: Le Fort osteotomy with 5 mm of impaction and movement of A point 2 mm distally Mandibular autorotation and advancement; B point came forward 13 mm Vertical reduction and advancement genioplasty; pogonion came forward 16 mm I, Posttreatment profile. J, Pretreatment three-quarter view, smiling. K, Graph showing the patient's attitude changes during the program of state-of-the-art comprehensive dentistry. Her posttreatment negative feelings were related to residual temporomandibular joint discomfort and to the belief that she had not been informed fully about some of the downsides of the multidisciplinary treatment. *Adj,* adjustments; *Ortho,* orthodontics; *Perio,* periodontics; *Post,* post-surgery; *Presrg,* pre-surgical; *PS/R,* post surgical restorative. L, Posttreatment three-quarter view, smiling. (Surgeryby Dr. Peter Chemello, Arlington Heights,, IL.)

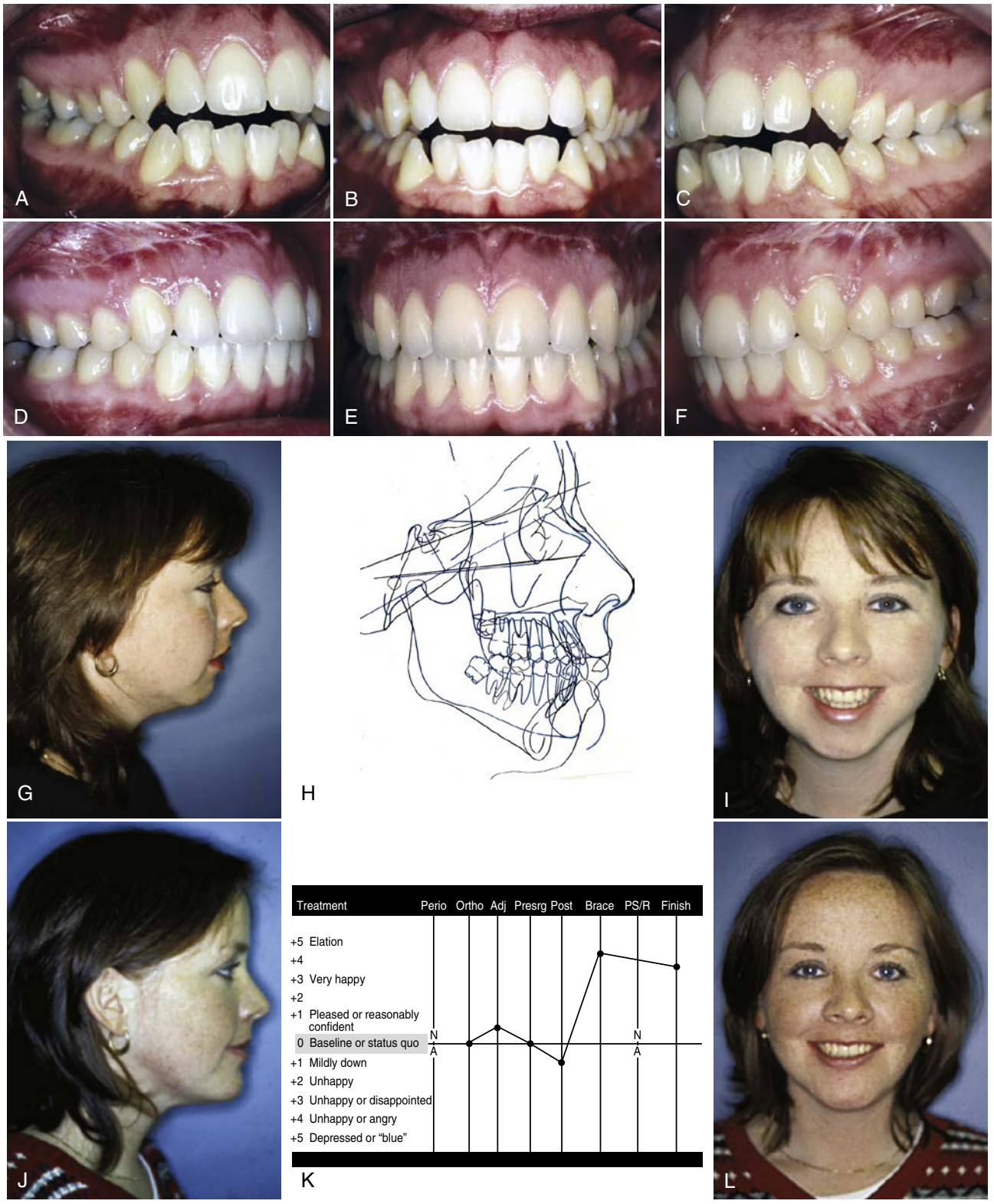


FIGURE 23-36 A–C, Pretreatment intraoral photographs. D–F, Posttreatment photographs. G, Pretreatment profile. H, Bolton template–guided diagnosis. Treatment included the following: Presurgical upper and lower arch alignment to prepare for skeletal correction indicated in Figure 23-34, *H*, Le Fort 1 osteotomy with rigid fixation—anterior raised 5 mm; posterior raised 2 mm Mandibular advancement via bilateral sagittal split osteotomies with rigid fixation—advanced 10 mm Alloplastic chin implant—6-mm implant placed. I, Pretreatment facial smile. J, Posttreatment file. K, Chart showing patient's attitude changes during the interdisciplinary treatment plan. Note the differences in charts depicted in Figures 23-33, *K*, and 23-34, *K*, reflecting posttreatment "quality of life improvement" as a result of rigid fixation rather than IMF used for the patient in Figure 23-33. *Adj,* adjustments; *Ortho,* orthodontics; *Perio,* periodontics; *Post,* post-surgery; *Presrg,* pre-surgical; *PS/R,* post surgical restorative. L, Posttreatment facial smile. (Surgery by Dr. Peter Chemello, Arlington Heights, Illinois.)

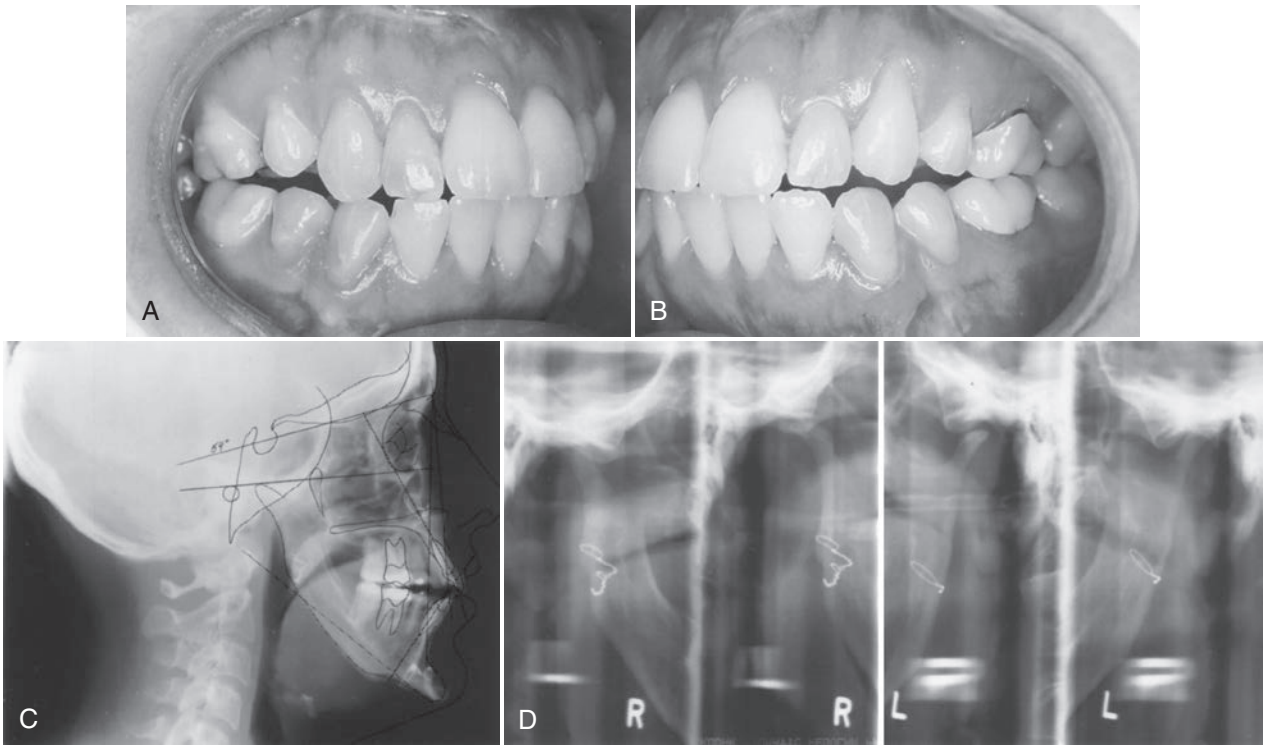


FIGURE 23-37 **A,** Right posterior occlusion in a woman (age 35 years, 10 months) after two orthodontic treatments and two separate orthognathic procedures. **B,** Lateral posterior occlusion after two orthodontic treatments and two separate orthognathic procedures. **C,** Lateral cephalogram after two separate orthodontic and surgical treatments. Note the lack of incisal contact. The patient stated that the "open bite is progressively getting worse." **D,** The right condyle shows degenerative changes, and the left condyle shows degenerative changes similar to those described by Arnett and Tamborello. (From Proffit WR, Phillips C, Turvey TA. Stability after mandibular setback: mandible-only versus 2 jaw surgery. *J Oral Maxillofac Surg* 2012;70(7):e408–e414.)

7. Stability of the condylar position
   Stable condylar position applies to presurgical assessment, surgical control of the proximal segment, and the patient's postsurgical adaptation to the new neuromuscular and functional environment. In the early postsurgical stage of recovery, patients are frequently concerned about the lack of mobility of the mandible. In 2014, Ueki et al.[122] assessed the border movements of the mandible following Class III surgical correction. Their sample consisted of 73 patients; 41 underwent a sagittal split ramus osteotomy (SSRO), and 32 had SSRO and a Le Fort I osteotomy. For 52 of the patients, mandibular movements were measured with the Myotronics measurement system before and then 6 months after surgery. For 21 of the patients, the measurements were delayed for 18 months. Based on the findings of this paper, patients who had mandibular setback with or without a Le Fort I procedure did have hypomobility at the 6-month measurement stage. However, those evaluated after 18 months demonstrated that the measured border movements returned to pretreatment levels.

   A second important consideration related to surgical orthodontic outcomes is the long-term stability of the patient's condyle. Gunson et al.[57] have described the condition of idiopathic female condylar resorption, which may be responsible for late condylar changes (e.g., cheerleader's syndrome, avascular necrosis, or condylosis). These findings led the authors to hypothesize that some women are prone to condylar resorption that occurs after increased joint load and may be related to oral contraceptive pill use, which is well tolerated by most women (Fig. 23-37).[124]
8. Neuromuscular adaptation
   The surgical procedures that change the skeletal structures also change long-standing neuromuscular patterns of the stomatognathic system; achieving a balance or an equilibrium of the skeletal structures, dental structures, and muscles is the ultimate biologic challenge for the orthognathic team. Although the procedures performed today are based scientifically on a variety of working hypotheses, the relapse problems that still occur are reminders that some of the treatment provided may have empirical components.

Planning treatment for orthognathic cases can aid the stability of the treatment outcome through the following:

1. Proper *differential diagnosis* of patients who would most benefit from combined therapy: The classic example is the Class III grower. The orthodontist must determine through differential diagnostic studies and growth forecasting whether the patient has a mandibular excess condition. In borderline situations, an early phase of treatment can test, through therapeutic diagnosis, for skeletal modifiability and functional shifts. In other cases, surgery clearly is needed. However, the orthodontist still

may resolve the maxillary transverse deficiency and the anterior alignment during phase I while anticipating the second phase of braces at ages 16 to 23 years and surgery when facial growth is complete. The orthodontist plays a major role in guiding these cases to completion. For example, heroic orthodontic efforts to treat skeletal Class II problems nonsurgically for a protracted period in camouflage therapy may overcompensate the teeth and interfere with the ultimate need to provide corrective surgery later.[125] "The efficacy data of 34 Class II subjects who underwent surgical-orthodontic treatment with mandibular advancement and rigid fixation demonstrated that presurgical orthodontic treatment often does not fully decompensate the incisors, which limits the surgical outcome."[118]

This is also true of moderate to severe mandibular deficiency, high mandibular plane angle (downward and backward) growth patterns. Early introduction of the concept of combined therapy (orthodontics and surgery) conditions parents to the necessity of surgical correction and begins the psychological preparation of the patient. One must take care to use terminology that does not frighten or stigmatize young patients. The authors recommend conducting preliminary surgical consultations with the parents only for patients younger than age 16 years. This allows the parents to assimilate the surgical information and make the final decision as to the desirability of including the adolescent in the surgical discussion at the current time.

2. *Growth factors and the timing of the referral*: Orthodontists know that posttreatment growth is a destabilizing factor in orthodontic treatment results. In a study by the author, 42 of 100 adult patients (42%) who required retreatment did so because of growth changes that moderately or severely altered the original orthodontic correction.[44]

   If the differential growth is minimal, the occlusal change can be handled by 4 to 8 months of orthodontic retreatment. However, if growth undermines an orthodontic correction, the patient may not easily accept retreatment by means of surgery.

   To avoid growth-induced complications, one should consider the following guidelines:

   a. For moderate and severe Class III mandibular excess patients, surgery should be delayed until standardized cephalograms demonstrate completion of mandibular growth. In boys and young men, this could be as late as 22 to 24 years of age.
   b. For patients with asymmetry and unilateral hypertrophy, surgery should be delayed until bone scans demonstrate cessation of growth activity of the hypertrophic condyle.[126]
   c. In selected patients with excessive vertical growth (Class I and Class II), the maxillary Le Fort procedure can be considered before growth is complete when psychosocial factors dictate.[96] The effect of this procedure on the future growth of the nasal cartilage is not understood fully. However, a risk exists that premature double-jaw surgery for mandibular deficiency, high mandibular plane angle type III patients will be unstable if the mandible persists in a downward and backward growth mode and reopens the bite.[127]
   d. For patients with maxillary transverse deficiency, orthopedic expansion may be effective for most girls up to 14 years of age and for many boys up to 16 years of age. Successful orthopedic expansions have been reported in both genders at older ages, especially with surgical assistance. The predictability of nonsurgical orthopedic success declines as the patient's age increases.[28,29,116]
   e. Psychosocial needs may supersede growth factors when the patient has a severe growth imbalance. Self-esteem considerations during adolescence deserve concern, and two-stage sequential surgery may be desirable for patients whose psychological and social development is affected by their jaw growth imbalance[128–130] (see Fig. 23-12).
   f. The timing of the referral to the maxillofacial surgeon and the initial patient education process are important responsibilities of the orthodontist. The orthodontist should try to determine the patient's level of awareness about the growth imbalance. If a patient is unaware that the condition may require surgical adjuncts, the education process may take longer and require more than one consultation with the orthodontist to create sufficient understanding to pursue the consultation.

3. *TMJ stability*: The orthodontic literature emphasizes the importance of planning treatment based on a reproducible condylar–fossa relationship.[131,132]

   This concept is all the more true when diagnosing and planning treatment for orthognathic patients. The orthodontist's skill in diagnosing and documenting functional shift problems in the occlusion is important. Without such skill, a mandibular functional shift may make a case look better than it is, causing a treatment plan to fail because of under-treatment. Or at the other extreme, it may make a case look worse than it is (e.g., Class III skeletal problem amplified by a functional shift) because of functional forward shift, causing treatment plan failure by overtreatment (i.e., insisting on surgery when the case can be managed with conventional treatment only) (see Fig. 23-31).

   The time to diagnose mandibular shifts is before treatment is initiated and is accomplished through the use of superior repositioning deprogramming splints, leaf gauge condylar seating, or careful manipulation of the mandible.[10] If such discrepancies are not detected before appliance placement but show up during treatment, the clinician has an obligation to document and inform the patient of the diagnostic data arising during treatment and advise the patient of any recommended change in the plan. The patient then must decide whether to pursue or reject the surgical plan. Undetected functional shifts in maxillary vertical excess and mandibular deficiency cases can lead to a treatment plan that calls for Le Fort impaction only. The operating room, with the patient under general anesthesia, is not the place for the surgeon to revise the treatment plan from a one-jaw procedure to a double-jaw procedure. Clarification and documentation of centric relation mandibular position should be done before the treatment plan is finalized.[13,26,133] Other TMJ stability concerns are addressed in the Risks section of this chapter.

4. *Dental unit stability*: One aspect of dental instability may be an unfavorable crown-to-root ratio, which occurs after bone loss from periodontal disease or as a result of short roots, whether genetic or acquired (Fig. 23-38). The orthodontist should be able to detect tooth mobility problems and advise the surgeon of the need for skeletal rather than interdental fixation.

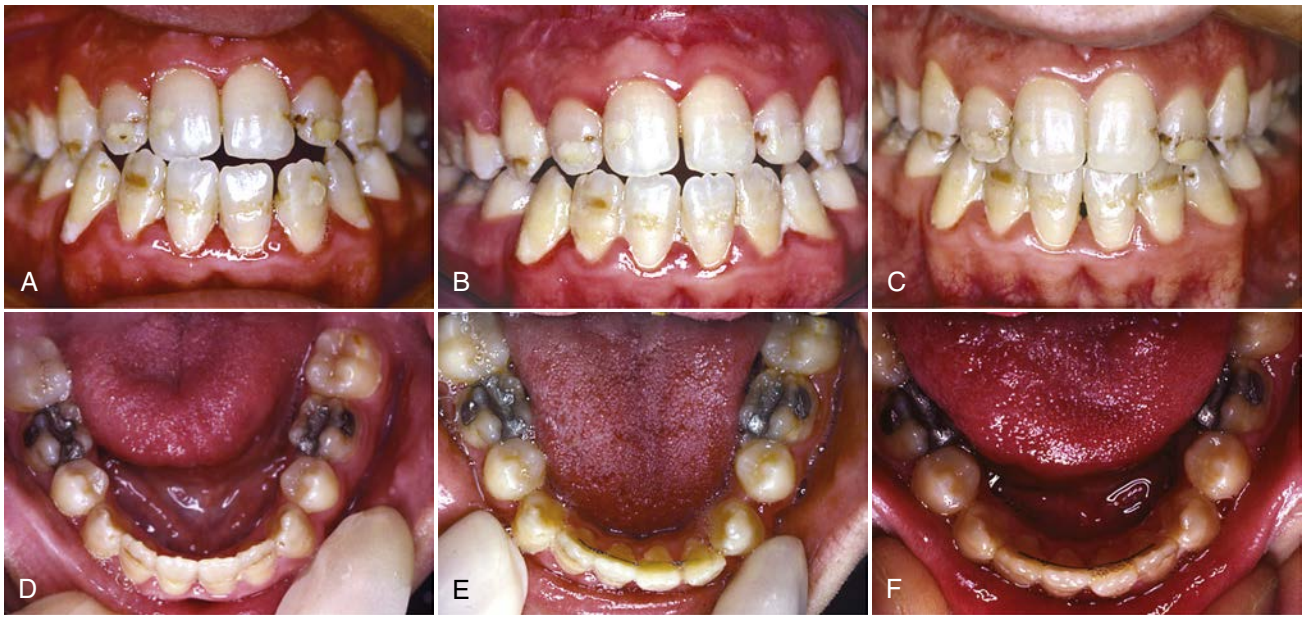


FIGURE 23-38 **A,** Anterior occlusion after braces and maxillary and mandibular surgery on a 14.5-year-old Class III patient. **B,** Anterior occlusion after 1.5 years of retention. Note lower incisor displacement. **C,** Anterior occlusion after phase II orthodontics; the patient refused additional jaw surgery. **D,** Lower incisors after braces and maxillary and mandibular surgery on a 14.5-year-old patient. **E,** Lower incisor displacement after 1.5 years of retention with bonded retainer. Note the tongue size. **F,** Lower incisors after phase II orthodontics and tongue reduction.

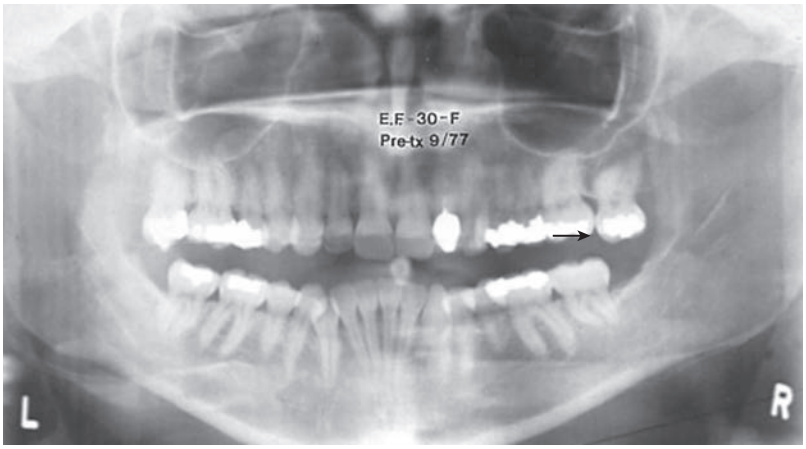


FIGURE 23-39 Pretreatment panoral radiograph showing several problems arising from mandibular excess (this patient's condition was shown in Figure 23-25). Note the supererupted right second molar (*arrow*).

Another form of dental instability features temporary crowns cemented as part of a periorestorative program. Brackets break easily from these crowns, and the crowns dislodge easily from the teeth. For these reasons, interdental fixation in a patient with temporary crowns is not advisable.

5. *Presurgical orthodontic preparation*: Arch leveling should be inclusive of second molars. In mandibular excess cases, the upper second molar frequently is supraerupted (Fig. 23-39), and in mandibular deficiency cases, the lower second molar may be supraerupted. These distal teeth, if not level vertically, can interfere with proper occlusal seating at the time of surgery, creating condylar distraction and instability. In addition, the lower incisors may be supraerupted in a skeletal Class II or III problem and require leveling with intrusion to prepare for the surgical phase. Certain surgical treatment plans may call for segmental intrusion, and the arch may require segmental leveling to satisfy the stability and aesthetic requirements of the case. Continuous archwires may cause instability through unwanted incisor extrusion (in an open bite case), causing less skeletal correction of the open bite and dental relapse.
6. *Considerations of pre- and postsurgical airway function* (see Chapter 12)
With current knowledge regarding obstructive sleep apnea, both the orthodontist and surgeon must assess the patient's presurgical sleep tendencies. If the patient has characteristics

of obstructive sleep apnea, surgical procedures that have a narrowing effect on the airway (e.g., mandibular setback for Class III patients) should also include maxillary advancement to aid in the correction of the Class III skeletal imbalance.[134,135] Aydemir and colleagues confirmed that the type of surgery on Class III patients can impact the airway. They assessed 48 patients with Class III malocclusions. Maxillary advancements were performed on 9 subjects, 7 subjects had mandibular setbacks, and 32 subjects had double-jaw procedures.[136] They found that the superior pharyngeal space and inferior pharyngeal space decreased after mandibular setback.
In the bimaxillary and maxillary advancement group, the palatopharyngeal space was significantly increased.

Additional prospective research on airway issues related to Class III jaw surgery has been reported by Uesugi et al.[135] In their study, 40 mandibular prognathic subjects were treated with orthodontics and orthognathic surgery: 22 patients had single-jaw mandibular setback surgical procedures; the other group of 18 had a double-jaw procedure with mandibular setback and a Le Fort I maxillary advancement surgical procedure. This was a CBCT study that included 16 control participants with normal occlusions and who had CBCT on two separate days for comparison. This study assessed pre- and postsurgical apnea-hypopnea index (AHI), morphologic differences in airway anatomy, and pharyngeal airway volume. Although Uesugi and colleagues found that there was a decrease in pharyngeal volume for patients having mandibular setback surgery, *they did not find an increase in AHI.* This well-done study and findings of are useful for assessing and planning orthognathic surgery for Class III patients.

As treatments are planned, the eight key responsibilities of the surgeon to maximize the stability of the orthognathic outcome (presented earlier in this section of the chapter) require periodic review with the surgeon.

RISKS OF ORTHODONTIC–ORTHOGNATHIC SURGERY

The risks to which patients are exposed during routine orthodontic therapy are minimal compared with the complications that may occur during orthognathic procedures. Practicing orthodontists must understand all the differential diagnostic considerations of the patient with skeletal imbalances and should also be well aware of the risk factors associated with the orthognathic procedures recommended. As the range of corrective procedures has widened, the reported negative sequelae have increased.

In the previous section of this chapter, the undesirable risk factors of instability were discussed. This section focuses on a variety of other complications that have been associated with orthognathic procedures (i.e., risk management). Complications are not common in orthognathic surgery, but they do occur. Several studies have shown complication rates.[49,50,76,137] Box 23-7 summarizes the complications recorded in the studies referenced.

There is a broad range of complications related to orthognathics that can be divided into the following categories: A, physical well-being; B, stomatognathic function; C, emotional well-being; and D, oral health sequelae.

BOX 23-7 Common Complications in Orthodontic–Orthognathic Surgery

Most Common Complications with Maxillary Surgery[21]

- Nausea or vomiting—40.08%
- Infection rate (acute or chronic)—7.4%
- Anatomic complications including deviated nasal septum—2.65%, nonunion at osteotomy gap—1.0%
- Extensive bleeding requiring blood transfusion—11%
- Ischemic complications causing aseptic necrosis of the alveolus—0.2%, gingival recession—0.8%
- Insufficient fixation—0.5% (most common in patients with clefts, craniofacial dysplasias, or vascular anomalies)

Most Common Complications with Mandibular Surgery

- Neurosensory deficit, mild—32%
- Disturbing—3%
- Infection rate—7.4%

A. Physical well-being
1. Excessive blood loss[76,137,138]
2. Neurologic injury (anesthesia, paresthesia)[138]
   a. Hypoesthesia (decreased sensation)
   b. Hyperpathia (increased sensation)
   c. Dysesthesia (altered sensation that is painful)
3. Allergic reaction to anesthetic, antibiotic, or anti-inflammatory drugs
4. Postoperative infections[76,139]
   a. Surgical site
   b. Skeletal fixation wires
   c. Rigid fixation plates[140]

   Current infection prevention techniques are discussed by Spaey and colleagues.[52] In addition, orthodontists can aid surgeons in reducing the potential iatrogenic incidence of nidi of infection by using soldered or slide-on surgical hooks, well-secured brackets, and stainless steel ligatures (not A-lastics because their force decays rapidly, and they are not radiopaque; if A-lastic ligatures inadvertently fall into the surgical site, they are difficult to locate and remove). Figure 23-40 shows the postsurgical migration of a molar bracket that broke off the left first molar during surgery. Fortunately, no infection developed.
5. Bone fractures (Fig. 23-41)

   Reports exist in the literature of unfavorable surgical splits of the mandible, including buccal plate fracture associated with impacted third molar teeth that were extracted at the time of the mandibular osteotomy. When sagittal split procedures are planned for mandibular deficiency or mandibular excess, it is prudent to remove third molars 6 to 12 months before the osteotomy to avoid the risk of unfavorable fractures. When unfavorable splits occur, the stability of the procedure is jeopardized, and the likelihood of neuropathies and facial deformities is greater. If an unfavorable split occurs, the surgeon must decide whether to proceed with the surgery or repair the fracture and redo the surgery at a later time.
6. Delayed healing and nonhealing

   Delayed healing and nonhealing occur most often when unexpected microtrauma or macrotrauma occurs in the surgical and postsurgical periods. Intraoperative complications such as those discussed previously and including unfavorable splits in the sagittal split or unfavorable

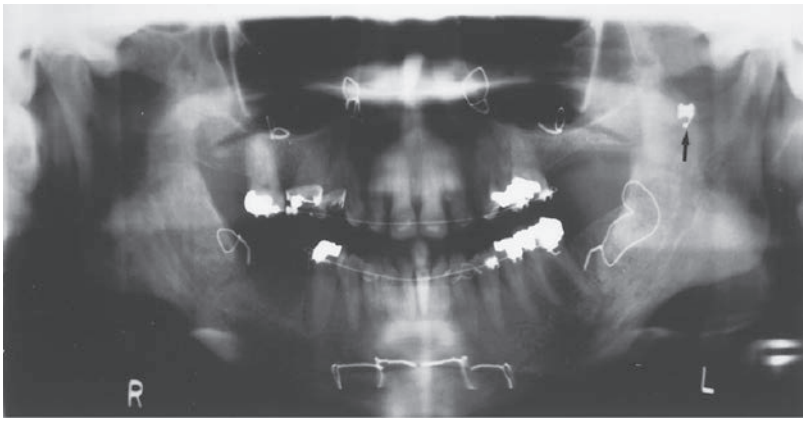


FIGURE 23-40 Postsurgical panoral radiograph showing molar bracket (*arrow*) that broke off during surgery. The bracket has migrated to the coronoid notch.

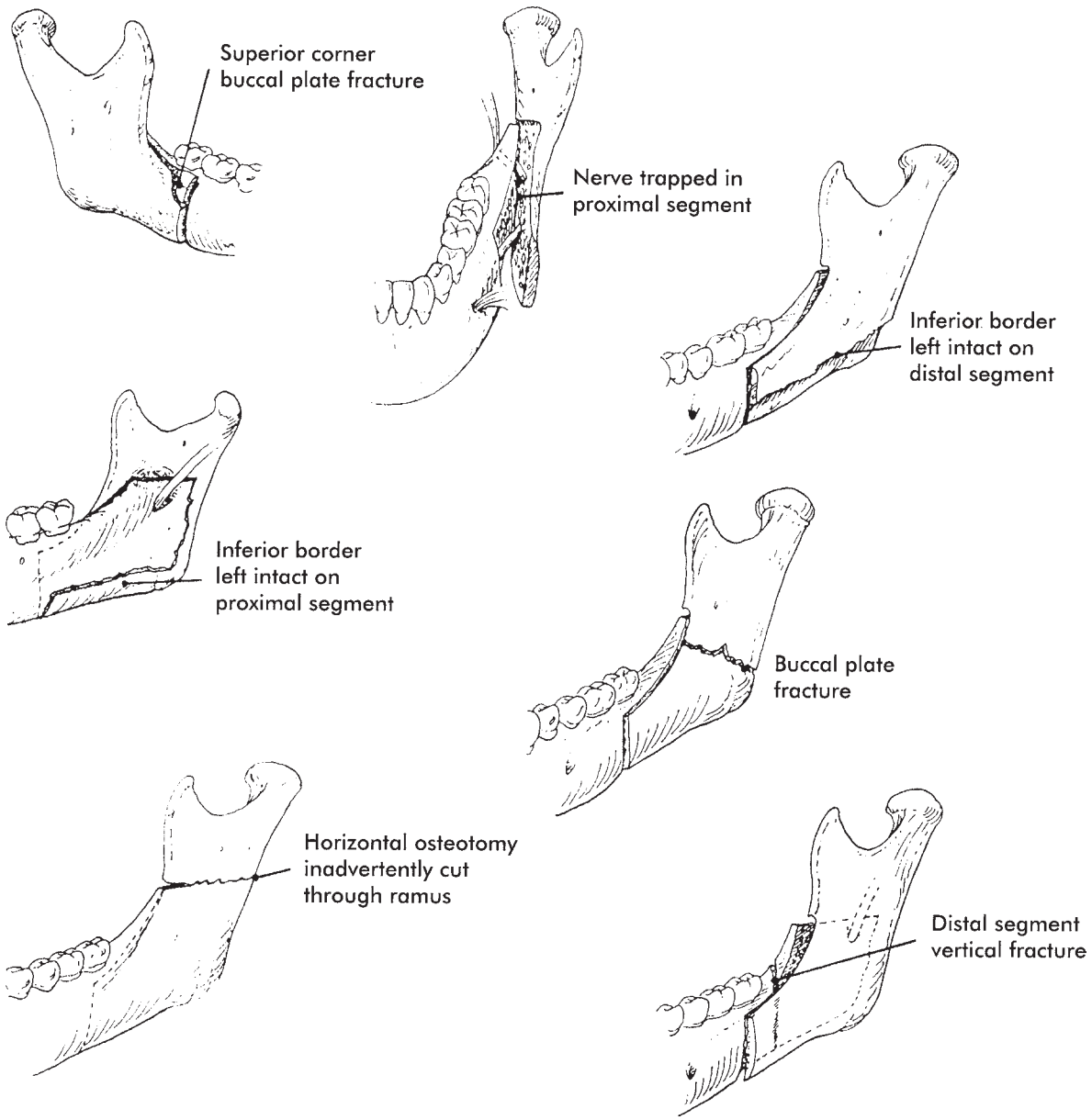


FIGURE 23-41 Common types of unfavorable splits. (From Sinn DP, Ghali GE. Management of intraoperative complications in orthognathic surgery. *Oral Maxillofac Clin North Am* 1990;2(4):872.)

downfracture in the Le Fort osteotomy can result in incomplete bone healing. An inaccurate preoperative medical assessment (see Table 23-5) or postoperative surgical traumatic occlusion also can result in poor bone healing and fibrous unions.

7. Secondary surgery
   A small percentage of orthognathic patients must return to the operating room for secondary surgery because of unexpected surgical outcomes. Some return before release from the hospital after the first surgery; others return months or years later (Fig. 23-42). The most common reasons for secondary surgery include the following:
   - Early relapse
   - Posthealing relapse
   - Unfavorable split
   - Patient dissatisfaction with the aesthetic result (in the early years of the Le Fort procedure, the maxilla was impacted beyond an aesthetic smile line in some patients)

   Overimpaction of the maxilla can occur for several reasons:
   - Removal of an excessive amount of bone from the lateral walls
   - Presence of thin, concave maxillary bone that may collapse on loading
   - Use of overly tight interosseous suspension wires that tend to pull the maxilla superiorly
   - Excessive masticatory function that overrides the stability of the bony interfaces
   - Lack of bony contact
   - Inappropriate treatment planning

   Correction of overimpaction cannot be achieved short of surgically repositioning the maxilla inferiorly and returning the dentoalveolar portion to its original position. This would require interpositional bone grafting and rigid internal fixation and possibly concomitant mandibular surgery.[138]
8. Sinus complications
   Several authors who have studied postsurgical patients have found that a higher percentage than expected reported sinus problems after Le Fort procedures.[141,142] Other authors have indicated that postsurgical sinus disease is no greater than that in the general population.[138] Postsurgically, the maxillary sinus function may have been altered such that it interferes with proper drainage, causing sinus-type headaches, or sinuses may congest more frequently, causing interference with nasal breathing. Although this complication may seem relatively minor, it can have an adverse effect on the quality of life of patients who must deal with chronic sinus flare-ups.

B. Stomatognathic system function
1. Decreased efficiency and discomfort during chewing
   Two common causes of postsurgical occlusal inefficiency are the following:
   - Posterior open bites caused by overseating of condyles during mandibular surgery
   - Excessive superior positioning of the posterior maxilla, especially when posterior vertical stops are inadequate in the area of the osteotomy
2. Temporomandibular dysfunction, discomfort, and pain
   In some cases, orthognathic surgery is justified because it has the potential to reduce temporomandibular dysfunction symptoms. Karabouta and Martis[143] reported that temporomandibular dysfunction symptoms declined from 40% to 11% of the sample. However, 4% of previously asymptomatic patients developed temporomandibular dysfunction symptoms after surgery. In another study Kerstens et al.[144] and Kim et al.[145] found a 66% reduction in temporomandibular dysfunction symptoms; they also found that 11.5% of preoperatively asymptomatic patients experienced symptoms after surgery. Many of these patients may have had latent temporomandibular dysfunction problems that were activated by the altered joint loading after jaw surgery. Management requires a team effort and appropriate care, depending on the nature of the problem. In a recent systematic review of 1776 articles, Lindenmeyer et al.[146] found 23 articles seeking to establish a therapeutic role of orthognathic surgery in relation to TMD issues. After assessing the data in these articles, the authors were unable to establish a positive or negative role of the orthognathic procedures causing or "curing" TMD issues.
3. Limitation in range of motion
   A program of physical therapy is prescribed for most orthognathic patients shortly after the release of fixation. If mandibular hypomobility is present after jaw surgery, it could have an intracapsular or extracapsular cause. Intracapsular problems are characterized by a sharp pain localized in the TMJs. Extracapsular problems are characterized by less intense, dull pain that is generalized and diffuse and may or may not be aggravated by mandibular surgery; often the pain is alleviated by biting.

   A prospective study of 55 orthognathic patients by Aragon et al.[147] found that the maximal incisal opening (MIO) decreased in most of these patients after surgery. The percentage of MIO decrease depended on the surgery performed

Procedure	Percentage of Maximal Incisal Opening Reduction
Sagittal split osteotomy (SSO)	29% (mean)
Vertical subcondylar osteotomy (VSO)	10% (mean)
Le Fort I	2% (mean)
Le Fort I and SSO	28% (mean)
Le Fort I and VSO	9% (mean)

C. Emotional well-being
1. Adaptation to facial appearance change
   In the authors' experience, negative emotional responses seldom occur with single-jaw mandibular advancement or mandibular setback procedures because these procedures mainly affect the patient's profile, and therefore the patient does not observe these changes readily. Changes that occur with single-jaw Le Fort procedures or Le Fort procedures with mandibular advancement or setback alter the frontal facial appearance immediately, and the patient must adapt to the new look.[5] Although it has been determined that appearance change is not the primary motivating factor to pursue orthognathic surgery, it remains a key factor in the choice to pursue jaw surgery. After evaluating a retrospective questionnaire of 500 patients who underwent orthognathic surgery, Proothi et al.[148] determined that the primary motivating factor for patients to pursue jaw surgery was "bite improvement." Additional motivating factors in their study were appearance, pain, smile, and speech.

FIGURE 23-42 A–C, Intraoral photographs during evaluation for exposure to third orthodontic and surgical treatment plan. Instability resulted from previous treatment, which included orthodontics and four premolar extractions followed by double-jaw surgery. Instability of the initial surgical result and temporomandibular joint pain created a need for a second surgical procedure with the same orthodontist and surgeon. D–F, Intraoral photographs after orthodontic preparation and third surgical treatment with a new orthodontist and surgeon. Because of previous dental instability, permanent, fixed retention was incorporated in her treatment. G and H, Profile photograph before and after surgery, respectively. I–K, Lateral cephalograms: initial, presurgical (after decompensation), and postsurgical, respectively. Vertical chin reduction and advancement were considered, but the patient declined this procedure.

Esperao et al.[149] recently assessed the broader topic of quality of life issues in three groups of patients who were in different stages of surgical-orthodontic treatment: 20 patients who were in consultation phase of surgical-orthodontic treatment, 70 patients who were in presurgical orthodontic treatment, and 27 patients who had completed presurgical and orthognathic treatment but were still in postsurgical orthodontic treatment. The 14 questions from the Oral Health Impact Profile (OHIP) were used to compare the oral health impact profile of the three different groups. The authors found that those in the consultation phase and those in the presurgical phase were 6.48 and 3.14 times, respectively, more likely to experience a negative impact of their oral condition compared with patients in the postsurgical phase.

2. Self-esteem changes caused by unexpected facial changes[17,28,29,32,96,97,148,149]

   In Jacobson's study.[150] most patients (80%) said that orthodontic-orthognathic treatment influenced their lives positively, and 4% said that treatment had a negative influence on social activities. The remaining 16% were neutral about the impact of surgery on their lives. In their study of 55 patients, Kiyak et al.[28] found that at the 9-month postoperative interval, the patients' self-esteem had declined significantly from an early postsurgical period. The orthodontist and staff who follow these patients after surgery must be aware of the potential for emotional highs and lows during the postsurgical period. Having a psychologist available who is aware of the needs of orthognathic patients and who is able to intercept significant patient emotional problems is imperative.

3. Lack of preparation for changes in interpersonal relationships

   As the surgical technology has improved to allow more stable, functional results, so too has the capacity to alter a person's appearance and identity. Most orthognathic patients are women between the ages of 20 and 40 years. Many are married and have husbands and young children. When this person returns home from the hospital after a Le Fort osteotomy and genioplasty, she is swollen and black and blue (bruised), and she may be in fixation with limited speech and eating capacities. If the immediate family members in the support group are not prepared fully for her to return looking so different, significant stress and disruption of normal family functions will result. As one patient in the study by Kiyak et al.[28] commented, "I would not recommend this surgery to anyone without counseling."

   Patients who have had the surgery have many good ideas about ways to enhance the recovery process. Another patient in the study said the following:

Much more time is needed to prepare the patient with facts and information. I was unprepared for the bleeding and earaches, leaving the hospital so soon—less than 24 hours after surgery. I lost more weight than expected and really suffered psychologically and felt little support during the fixation period. Maybe the patients could be invited to form a mutual support group.[28]

Bhamrah et al.[151] assessed 1912 randomly selected Internet posts from an online orthognathic discussion forum to determine effectiveness of this mechanism as a support resource and information resource. The study used five major themes as topics for study:

a. Reasons for undergoing orthognathic treatment
b. Pre- and postsurgical treatment stages, including orthodontics
c. Surgery including postsurgical complications and difficulties
d. Expected and actual end-of-treatment changes
e. Seeking and sharing information

Bhamrah et al. found that Internet forums can be used to provide curious patients with additional information about orthognathic treatment, and the forums can be a source of support and emotional encouragement. The authors' findings provide practical insights into the mindsets of their patients going through the preparation and process of pursuing orthognathic surgery.

In a 2011 study, Oland et al.[152] provided orthognathic teams with additional insights into posttreatment patient satisfaction of surgical orthodontic patients. From the 118 patients who were studied via a pre- and posttreatment questionnaire that assessed pretreatment motivation and posttreatment satisfaction, it seemed that the degree of posttreatment satisfaction was correlated to pretreatment motivation:

Those who were motivated by oral functional motives before treatment had lower post-treatment satisfaction than those who were motivated by a change in appearance. Additionally, the more fulfilled a patient's motive for appearance change, the greater were the measurable improvements in self-concept and social interaction.[152]

4. Depression

   Postsurgical depression has received little attention in the orthognathic literature and only recently has been discussed as a legitimate complication of jaw surgery.[153] Generally, dentists are not trained properly to handle depression medically, even if it is likely to be a transitory state of mind for orthognathic patients. However, the orthodontic members of the health care team have the duty to recognize the symptoms of depression, discuss them with the patient and family, refer the patient when necessary, and interact with the psychologist to aid in the patient's recovery. In addition, the orthodontist should try to be aware of other stressful events in the patient's life that may add to the anxiety level during the 2 to 3 years these patients are followed.

   Although the current literature discusses the postsurgical sequelae of depression, the authors' experience is that patients with rigid fixation show rapid psychological rebound and fewer signs of depression than patients 5 to 10 years ago whose jaw surgery required intermaxillary fixation for 6 to 10 weeks.

D. Oral health complications and sequelae

1. Bone loss[150]

   Although this complication is not reported frequently, the exacerbation of existing periodontitis is a potentially serious problem. Patients with mild, moderate, or advanced periodontal disease require strict supervision and management by a specialist before orthognathic procedures. Patients with moderate or advanced periodontal disease are at high risk for further periodontal breakdown after orthognathic surgery because of ensuing dietary changes, interference with oral hygiene (depending on the mode of fixation), and transitory occlusal trauma.

2. Gingival deficiency with recession
   During the treatment planning stage, orthodontists need to consider the degree of decompensation of the lower incisors as they prepare the patient for jaw surgery. Frequently, in Class III patients, for example, lower incisor decompensation requires gingival grafting before tooth movement to avoid gingival recession (see Figs. 23-26 and 23-28).
3. Pulpal changes
   The osteotomies that are performed, particularly Le Fort I procedures and segmentals, often approximate root apices. Occasionally, after an osteotomy, the affected tooth starts to change color and lose its vitality, and eventually it requires a root canal. The likelihood of loss of vitality is minimal, but it does occur. Careful preparation by the orthodontist and planning by the surgeon should eliminate this annoying complication.
4. Tooth loss
   Tooth loss is an uncommon sequela, but it does warrant brief discussion. A cohort study published in 2012 by Williams et al.[154] assessed complications after SARPE. This retrospective study evaluated 120 patients who had a SARPE procedure at the Kaiser Permanente Oakland Medical Center. The average age of the patients was 29 years, and the sample was half male and half female. Although most orthodontists and surgeons consider the SARPE procedure to have minimal complications, this study determined that 41 of the 120 patients assessed had at least one complication. Most of the complications were minor and were resolved with local measures. There were, however, 15% who required additional unanticipated treatment. The most common problem was symmetric or inadequate expansion. This occurred in 13% of the sample. The most common dental or periodontal problem involved the maxillary central incisor. There were reports of discoloration, gingival recession, and periodontal bone loss that occurred in 15% of the patients in this study. The authors also reported "catastrophic" midline bony defects with the loss of both central incisors. These data are important informed consent information for both the surgeon and the orthodontist.

   Tooth loss may also occur because of inadequate apical root space (4 mm is the desired amount of separation between apices). Proper preparation of the area to be segmented reduces the probability of tooth loss sequelae. Although this discussion of complications associated with orthognathic surgery is far from complete, it should give the orthodontist an awareness of some of the more common postsurgical sequelae that must be managed in cooperation with the oral surgeon for the overall well-being of the patient. Orthodontists who refer patients for orthognathic surgery should review the informed consent policy of the surgeon's office so that the orthodontist and staff can help their patients understand any aspects that are unclear. The routine surgical practice in the United States is to review potential negative sequelae with the patient before surgery. This is part of the informed consent procedures and is appreciated greatly by patients.[18]

SUMMARY

This chapter began with a discussion of treatment goals and the technologic advances that have helped orthodontic-surgical teams enhance the lives of many patients who were born with facial skeletal disharmonies. The chapter intentionally concluded with a discussion of complications. New surgical technology has broadened the scope of orthodontists' treatment capability and at the same time has expanded their responsibilities. In his very informative book *Second Opinions*, Jerome Groopman[155] provides a valuable insight from his medical experience to orthodontists and surgeons. He states:

> *In a predictable world, clinical decision making would be a well-defined, scientific exercise with set methods for diagnosis and treatment. The unfortunate truth is that this is not possible. People adapt differently, physically and emotionally, to each illness (condition) and react in varying ways to a given therapy. This means that diagnosis and treatment cannot be strictly bound by generic recipes, but must be made individual, to be consistent with the particular clinical and psychological characteristics of the person.*

As the orthodontic specialty and the surgical specialty continue to study factors that reduce risk and enhance success, the surgeon and the orthodontist have the responsibility to integrate many important variables to provide optimal patient care.

Acknowledgments

With great appreciation to my staff and the oral and maxillofacial surgeons who have made these dramatic changes possible for our patients. Also, tremendous appreciation to Elizabeth Barrett, Beth McFarland, and Melissa Carstens for their time and assistance in editing the text and organizing the figures for this chapter.

REFERENCES

1. Moss ML. The functional matrix hypothesis revisited. *Am J Orthod*. 1997;112:8–11.
2. Moss ML. The functional matrix hypothesis revisited. *Am J Orthod*. 1997;112:221–226.
3. Moss ML. The functional matrix hypothesis revisited. *Am J Orthod*. 1997;112:338–342.
4. Enlow DH. *The Human Face: an Account of the Postnatal Growth and Development of the Craniofacial Skeleton*. New York: Harper & Row Publishers; 1968.
5. Bollen AM, Cunha-Cruz J, Hujoel PP. Secular trends in preadult orthodontic care in the United States: 1942–2002. *Am J Orthod Dentofacial Orthop*. 2007;132(5):579–585.
6. Rocha NS, Cavalcantr JR, de Oliveira e Silva ED, et al. Patient's perception of improvement after surgical assisted maxillary expansion (SAME): pilot study. *Med Oral Pathol Oral Cir Buccal*. 2008;13(12):E783–E787.
7. Sabri R. Orthodontic objectives in orthognathic surgery: state of the art today. *World J Orthod*. 2006;72(2):177–191.
8. Selber JC, Rosen HM. Aesthetics of facial skeletal surgery. *Clin Plast Surg*. 2007;34(3):437–445.
9. Dellavia C, Ghislanzoni LTH, Peretta R. Occlusal morphology 1 year after orthodontic and surgical-orthodontic therapy. *Angle Orthod*. 2008;78(1):25–31.
10. Williamson EH. Occlusal concepts in orthodontic diagnosis and treatment. In: Johnston LE, ed. *New Vistas in Orthodontics*. Philadelphia: Lea & Febiger; 1985.

11. Kuroda S, Katayama A, Takano-Yamamoto T. Severe anterior open-bite case treated using titanium screw anchorage. *Angle Orthod*. 2004;74(4):558–567.
12. Weaver N, Glover K, Major P, et al. Age limitation on provision of orthopedic therapy and orthognathic surgery. *Am J Orthod Dentofacial Orthop*. 1998;113(2):156–164.
13. Xun C, Zeng X, Wang X. Microscrew anchorage in skeletal anterior open-bite treatment. *Angle Orthod*. 2007;77(1):47–56.
14. Berger JL, Pangrazio-Kulbersh V, George C, Kaczynski R. Long-term comparison of treatment outcome and stability of Class II patients treated with functional appliances versus bilateral sagittal split ramus osteotomy. *Am J Orthod Dentofacial Orthop*. 2005;127(4):451–464.
15. Barrera JE, Powell NB, Riley RW. Facial skeletal surgery in the management of adult obstructive sleep apnea syndrome. *Clin Plast Surg*. 2007;34(3):565–573.
16. Schendel SA, Powell NB. Surgical orthognathic management of sleep apnea. *J Craniofac Surg*. 2007;18(4):902–911.
17. Broder HL, Phillips C, Kaminetzky S. Issues in decision making: Should I have orthognathic surgery? *Semin Orthod*. 2000;6(4):249–258.
18. Brons S, Becking AG, Tuinzing DB. Value of informed consent in surgical orthodontics. *J Oral Maxillofac Surg*. 2009;67(5):1021–1025.
19. Wheeler PW. Risk preclusion. *Am J Orthod Dentofacial Orthop*. 1992;101(2):194–195.
20. Widmann G, Stoffner R, Bale R. Errors and error management in image-guided craniomaxillofacial surgery. *Oral Surg Oral Med Oral Pathol Oral Radio Endod*. 2009;107(5):701–715.
21. Williams AC, Shah H, Sandy JR, Travess HC. Patients' motivations for treatment and their experiences of orthodontic preparation for orthognathic surgery. *J Orthod*. 2005;32(3):191–202.
22. Koury ME, Epker BN. The aged face: the facial manifestations of aging. *Int J Adult Orthodon Orthognath Surg*. 1991;6(2):81–95.
23. Sforza C, Laino A, D'Allessio R, et al. Soft-tissue facial characteristics of attractive and normal adolescent boys and girls. *Angle Orthod*. 2008;78(5):799–807.
24. Wong NK, Kassim AA, Foong KW. Analysis of esthetic smiles by using computer vision techniques. *Am J Orthod Dentofacial Orthop*. 2005;128(3):404–411.
25. Kumar S, Williams AC, Ireland AJ, Sandy JR. Orthognathic cases: what are the surgical costs? *Eur J Orthod*. 2008;30(1):31–39.
26. Bailey LJ, Cevidanes LH, Proffit WR. Stability and predictability of orthognathic surgery. *Am J Orthod Dentofacial Orthop*. 2004;126(3):273–277.
27. Bailey LJ, Dover AJ, Proffit WR. Long-term soft tissue changes after orthodontic and surgical corrections of skeletal Class III malocclusions. *Angle Orthod*. 2007;77(3):389–396.
28. Kiyak HA, Bell R. Psychological considerations in surgery and orthodontics. In: Proffit WR, White PR, eds. *Surgical Orthodontic Treatment*. St. Louis: Mosby; 1991.
29. Kitahara T, Nakasima A, Kurahara S, et al. Hard and soft tissue stability of orthognathic surgery. *Angle Orthod*. 2009;79(1):158–165.
30. Bays RA, Bouloux GF. Complications of orthognathic surgery. *Oral Maxillofac Surg Clin North Am*. 2003;15(2):229–242.
31. Bellucci CC, Kapp-Simon KA. Psychological considerations in orthognathic surgery. *Clin Plast Surg*. 2007;34(3):e11–e16.
32. Phillips C, Bennet ME, Broder HL. Dentofacial disharmony: psychological status of patients seeking treatment consultation. *Angle Orthod*. 1998;68:547–556.
33. Phillips C, Blakey 3rd G, Jaskolka M. Recovery after orthognathic surgery: short-term health-related quality of life outcomes. *J Oral Maxillofac Surg*. 2008;66(10):2110–2115.
34. Truitt J, Strauss RA, Best A. Centric relation: a survey study to determine whether a consensus exists between oral and maxillofacial surgeons and orthodontists. *J Oral Maxillofac Surg*. 2009;67(5):1058–1061.
35. Montini RW, McGorray SP, Wheeler TT, et al. Perceptions of orthognathic surgery patient's change in profile. *Angle Orthod*. 2007;77(1):5–11.
36. Machen DE. Legal aspects of orthodontic practice. *Am J Orthod Dentofacial Orthop*. 1991;99:91.
37. Rusanen J, Lahti S, et al. Quality of life in patients with severe malocclusion before treatment. *Eur J Orthod*. 2010;32(February):43–48.
38. Frejman MW, Vargas IA, Rösing CK, Closs LQ. Dentofacial deformities are associated with lower degrees of self-esteem and higher impact on oral health-related quality of life: results from an observations study involving adults. *J Oral Maxillofac Surg*. 2013;71(4):763–767.
39. Johnston C, Hunt O, et al. Self-perception of dentofacial attractiveness among patients requiring orthognathic surgery. *Angl Orthod*. 2010;80(3):361–366.
40. Proffit WR, White Jr RP. Who needs surgical-orthodontic treatment? *Int J Adult Orthodon Orthognath Surg*. 1990;5(2):82–89.
41. Squire D, Best AM, Lindauer SJ, et al. Determining the limits of orthodontic treatment of overbite, overjet, and transverse discrepancy: a pilot study. *Am J Orthod Dentofacial Orthop*. 2006;129(6):804–808.
42. Orminston JP, Huang GJ, et al. Retrospective analysis of long-term stable and unstable treatment outcomes. *Am J Orthod Dentofacial Orthop*. 2005;128(5):568–574.
43. Yoshida K, Rivera GA, Matsuo N, et al. Long-term prognosis of BSSO mandibular relapse and its relation to different facial types. *Angle Orthod*. 2000;70:220–226.
44. Musich DR. Lessons learned from patient re-treatment: an analysis of 200 patients who had orthodontics as children and who sought re-treatment as adults. *AAO Webinar Series*. 2013.
45. Shepherd J. Hypotensive anaesthesia and blood loss in orthognathic surgery. *Evid Based Dent*. 2004;5(1):16.
46. Rodrigo C. Anesthetic considerations for orthognathic surgery with evaluation of difficult intubation and technique for hypotensive anesthesia. *Anesth Prog*. 2000;47(4):151–156.
47. Ueki K, Marukawa K, Shimada M, et al. The assessment of blood loss in orthognathic surgery for prognathia. *J Oral Maxillofac Surg*. 2005;63(3):350–354.
48. Zellin G, Rasmusson L, Pålsson J, et al. Evaluation of hemorrhage depressors on blood loss during orthognathic surgery, a retrospective study. *J Oral Maxillofac Surg*. 2004;62(6):662–666.
49. Huamán ET, Juvet LM, Nastri A, et al. Changing patterns of hospital length of stay after orthognathic surgery. *J Oral Maxillofac Surg*. 2008;66(3):492–497.
50. Silva AC, O'Ryan F, Poor DB. Postoperative nausea and vomiting (PONV) after orthognathic surgery: a retrospective study and literature review. *J Oral Maxillofac Surg*. 2006;64(9):1385–1397.
51. Chow LK, Singh B, Chiu WK, et al. Prevalence of postoperative complications after orthognathic surgery: a 15 year review. *J Oral Maxillofac Surg*. 2007;65(5):984–992.
52. Spaey YJ, Bettens RM, Mommaerts MY, et al. A prospective study on infectious complications in orthognathic surgery. *J Craniomaxillofac Surg*. 2005;33(1):24–29.
53. Wolford LM, Chemello PD, Hilliard FW. Occlusal plane alteration in orthognathic surgery. *J Oral Maxillofac Surg*. 1992;51:730–740.
54. Wolford LM, Hilliard FW, Dugan DJ. *A Systematic Approach to the Prediction Tracing*. St. Louis: Mosby; 1985:54–74.
55. Chemello PD, Wolford LM, Buschang PH. Occlusal plane alteration in orthognathic surgery—Part II: long-term stability of results. *Am J Orthod Dentofacial Orthop*. 1994;106(4):434–440.
56. Reyneke JP, Bryant RS, Suuronen R, et al. Postoperative skeletal stability following clockwise and counter-clockwise rotation of the maxillomandibular complex compared to conventional orthognathic treatment. *Br J Oral Maxillofac Surg*. 2007;45(1):56–64.
57. Gunson MJ, Arnett GW, Formby B, et al. Oral contraceptive pill use and abnormal menstrual cycles in women with severe condylar resorption: a case for low serum 17B-estradiol as a major factor in progressive condylar resorption. *Am J Orthod Dentofacial Orthop*. 2009;135(6):772–779.
58. Roblee RD, Bolding SL, Landers JM. Surgically facilitated orthodontic therapy: a new tool for optimal interdisciplinary results. *Compendium*. 2009;30(5):264–275.
59. Resnick CM, Kaban LB, Troulis MJ. Minimally invasive orthognathic surgery. *Fac Plast Surg*. 2009;25(1):49–62.
60. Coscia G, Coscia V, et al. Augmented corticotomy combined with accelerated orthodontic forces in Class III orthognathic patients: morphologic aspects of the mandibular anterior ridge with cone-beam computer tomography. *J Oral Maxillofac Surg*. 2013;71(10):1760.e1–1760.e9.

61. Hoffman GR, Brennan PA. The skeletal stability of one-piece Le Fort I osteotomy to advance the maxilla; part 2. The influence of uncontrollable clinical variables. *Br J Oral Maxillofac Surg*. 2004;42(3):226–230.
62. Joss CU, Vassalli IM. Stability after bilateral sagittal split osteotomy advancement surgery with rigid internal fixation: a systemic review. *J Oral Maxillofac Surg*. 2009;67(2):301–313.
63. Joss CU, Thüer UW. Stability of the hard and soft tissue profile after mandibular advancement in sagittal split osteotomies: a longitudinal and long-term follow up study. *Eur J Orthod*. 2008;30(1):16–23.
64. Dolce C, Hatch JP, Van Sickels JE, et al. Rigid versus wire fixation for mandibular advancement: skeletal and dental changes after 5 years. *Am J Orthod Dentofacial Orthop*. 2002;121(6):610–619.
65. Costa F, Robiony M, Sembronio S, et al. Stability of skeletal Class III malocclusion after combined maxillary and mandibular procedures. *Int J Adult Orthodon Orthognath Surg*. 2001;16(3):179–192.
66. Joss CU, Vassalli IM. Stability after bilateral sagittal split osteotomy setback surgery with rigid internal fixation: a systematic review. *J Oral Maxillofac Surg*. 2008;66(8):1634–1643.
67. Joss CU, Thüer UW. Stability of hard tissue profile after mandibular setback in sagittal split osteotomies: a longitudinal and long-term follow-up study. *Eur J Orthod*. 2008;30(4):352–358.
68. Egermark I, Blomqvist JE, Cromvik U, et al. Temporomandibular dysfunction in patients treated with orthodontics in combination with orthognathic surgery. *Eur J Orthod*. 2000;22(5):537–544.
69. Conley RS, Legan HL. Correction of severe obstructive sleep apnea with bimaxillary transverse distraction osteogenesis and maxillomandibular advancement. *Am J Orthod Dentofacial Orthop*. 2006;129(2):283–292.
70. Costa F, Robiony M, Politi M. Stability of Le Fort I osteotomy in maxillary inferior repositioning: review of the literature. *Int J Adult Orthodon Orthognath Surg*. 2000;15(3):197–204.
71. Gerressen M, Stockbrink G, Smeets R, et al. Skeletal stability following bilateral sagittal split osteotomy (BSSO) with and without condylar positioning device. *J Oral Maxillofac Surg*. 2007;65(7):1297–1302.
72. Nemeth DZ, Rodrigues-Garcia RC, Sakai S, et al. Bilateral sagittal split osteotomy and temporomandibular disorders: rigid fixation versus wire fixation. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2000;89(1):29–34.
73. Zinser MJ, Sailer HF, et al. A paradigm shift in orthognathic surgery? A comparison of navigation, computer-aided designed/computer-aided manufacture splints, and "classic" intermaxillary splints to surgical transfer of virtual orthognathic planning. *J Oral Maxillofac Surg*. 2013;71(12):2151.21–2151.e21.
74. Ahn Y-S, Kim S-G, et al. Comparative study between resorbable and nonresorbable plates in orthognathic surgery. *J Oral Maxillofac Surg*. 2010;68(2):287–292.
75. Phillips C, Essick G, Preisser JS, et al. Sensory retraining after orthognathic surgery: effect on patients' perception of altered sensation. *J Oral Maxillofac Surg*. 2000;65(6):1162–1173.
76. Kramer FJ, Baethge C, Swennen G, et al. Intra- and perioperative complications of the LeFort I osteotomy: a prospective evaluation of 1000 patients. *J Craniofac Surg*. 2004;15(6):971–977.
77. Caloss R, Atkins K, Stella JP. Three-dimensional imaging for virtual assessment and treatment simulation in orthognathic surgery. *Oral Maxillofac Surg Clin North Am*. 2007;19(3):287–309.
78. Alves PV, Bolognese AM, Zhao L. Three-dimensional computerized orthognathic surgical treatment planning. *Clin Plast Surg*. 2007;34(3):427–436.
79. Kusnoto B. Two-dimensional cephalometry and computerized orthognathic surgical treatment planning. *Clin Plast Surg*. 2007;34(3):417–426.
80. Papadonpoulos MA, Lazaridou-Terzoudi T, Oland J, et al. Comparison of soft and hard tissue profiles of orthognathic surgery patients treated recently and 20 years earlier. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2009;108(1):e8–e13.
81. Olszewski R, Villamil MB, Trevisan DG, et al. Towards an integrated system for planning and assisting maxillofacial orthognathic surgery. *Comput Methods Programs Biomed*. 2008;91(1):13–21.
82. Metzger MC, Hohlweg-Majert B, Schwarz U, et al. Manufacturing splints for orthognathic surgery using a three-dimensional printer. *Oral Surg Oral Med Oral Pathol Oral Radio Endod*. 2008;105(2):e1–e7.
83. Mavili ME, Canter HI, Saglam-Aydinatay B, et al. Use of three-dimensional medical modeling methods for precise planning of orthognathic surgery. *J Craniofac Surg*. 2007;18(4):740–747.
84. Swennen GR, Barth EL, Eulzer C, et al. The use of a new 3D splint and double CT scan procedure to obtain an accurate anatomic virtual augmented model of the skull. *Int J Oral Maxillofac Surg*. 2007;36(2):146–153.
85. Noguchi N, Tsuji M, Shigematsu M, et al. An orthognathic simulation system integrating teeth, jaw and face data using 3D cephalometry. *Int J Oral Maxillofac Surg*. 2007;36(7):640–645.
86. Franchi L, Baccetti T, McNamara JA. Cephalometric floating norms for North American adults. *Angle Orthod*. 1998;68:497–502.
87. Jacobson A. Orthognathic diagnosis using the proportional template. *Oral Surg*. 1980;38:820.
88. DeFigueiredo MA, Siqueira DF, Bommarito S, et al. Early tooth extraction in the treatment of anterior open bite in hyperdivergent patients. *World J Orthod*. 2007;8(3):249–260.
89. Sugawara J, Kanzaki R, Takahasi I, et al. Distal movement of maxillary molars in nongrowing patients with the skeletal anchorage system. *Am J Orthod Dentofacial Orthop*. 2006;129(6):723–733.
90. Hoogeveen EJ, Jansma J, Ren Y. Surgically facilitated orthodontic treatment: a systematic review. *Am J Orthod Dentofacial Orthop*. 2014;145(4):S51–S54.
91. Conley RS, Jernigan C. Soft tissue changes after upper premolar extraction in Class II camouflage therapy. *Angle Orthod*. 2006;76(1):59–65.
92. Chung KR, Kim SH, Lee BS. Speedy surgical-orthodontic treatment with temporary anchorage devices as an alternative to orthognathic surgery. *Am J Orthod Dentofacial Orthop*. 2009;135(6):787–798.
93. Ackerman JL, Proffit WR. Treatment response as an aid to diagnosis and treatment planning. *Am J Orthod*. 1970;57(5):490–496.
94. Rustemeyer J, Groddeck A, et al. The accuracy of two-dimensional planning for routine orthognathic surgery. *Br J Oral Maxillofac Surg*. 2010;48(6):271–275.
95. Incrapera A, Kau CH, English JD, et al. Soft tissue images from cephalograms compared with those from a 3-D surface acquisition system. *Angle Orthod*. 2010;80:58–64.
96. De Sousa A. Psychological issues in oral and maxillofacial reconstructive surgery. *Br J Oral Maxillofac Surg*. 2008;46(8):661–664.
97. Kim SJ, Kim MR, Shin SW, et al. Evaluation on the psychosocial status of orthognathic surgery patients. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2009;108(6):828–832.
98. Kiyak HA, McNeill RW, West RA. The emotional impact of orthognathic surgery and conventional orthodontics. *Am J Orthod*. 1985;88:224–234.
99. Schuster G, Lux CJ, Stellzig-Eisenhauer A. Children with Class III malocclusion: development of multivariate statistical models to predict future need for orthognathic surgery. *Angle Orthod*. 2003;73:136–145.
100. Moon YM, Ahn SJ, Chang YI. Cephalometric predictors of long-term stability in the early treatment of Class III malocclusion. *Angle Orthod*. 2005;75(5):747–775.
101. Stellzig-Eisenhauer A, Lux CJ, Schuster G. Treatment decision in adult patients with Class III malocclusion. Orthodontic therapy or orthognathic surgery? *Am J Orthod Dentofacial Orthop*. 2002;122:27–28.
102. Chaiyongsirisern A, BakrRabie A, Wong RWK. Stepwise advancement Herbst appliance versus mandibular sagittal split osteotomy. *Angle Orthod*. 2009;79(6):1084–1089.
103. Pancherz H, Ruf S, Erbe C, et al. The mechanism of Class II correction in surgical orthodontic treatment of adult Class II, Division 1 malocclusions. *Angle Orthod*. 2004;74(6):800–809.
104. Bellucci CC, Kapp-Simon KA. Psychological considerations in orthognathic surgery. *Clin Plast Surg*. 2007;34(3):e11–e16.
105. Gurel HG, Memili B, et al. Long-term effects of rapid maxillary expansion followed by fixed appliances. *Angle Orthod*. 2010;80(1):5–9.
106. Gidarakou IK, Tallents RH, Kyrkanides S, et al. Comparison of skeletal and dental morphology in asymptomatic volunteers and symptomatic patients with unilateral disk displacement without reduction. *Angle Orthod*. 2003;73:121–127.
107. Levin RP. Developing staff potential. *J Am Dent Assoc*. 2010;141(5):582–583.
108. Arnett GW, Gunson MJ. Esthetic treatment planning for orthognathic surgery. *J Clin Orthod*. 2010;44:196–200.

109. Pavone I, Rispoli A, Acocella A, et al. Psychological impact of self-image dissatisfaction after orthognathic surgery: a case report. *World J Orthod*. 2005;6(2):141–148.
110. Lin J, Gu Y. Lower second molar extraction in correction of severe skeletal Class III malocclusion. *Angle Orthod*. 2006;76(2):217–225.
111. Zuroff J, Chen S-H, et al. Orthodontic treatment of anterior open-bite malocclusion: stability 10 years postretention. *Am J Orthod Dentofacial Orthop*. 2010;137(3):302.e1–302.e8.
112. Proffit WR, Phillips C, Turvey TA. Stability after mandibular setback: mandible-only versus 2-Jaw Surgery. *J Oral Maxillofac Surg*. 2012;70(7):e408–e414.
113. Al-Delayme R, AL-Khen M, et al. Skeletal and dental relapses after skeletal Class III deformity correction surgery: single-jaw versus double-jaw procedures. *Oral Surg Oral Med Oral Pathol Oral Radiol*. 2013;115(4l):466–472.
114. Bailey LJ, Phillips C, Proffit WR. Long-term outcome of surgical Class III correction as a function of age at surgery. *Am J Orthod Dentofacial Orthop*. 2008;133(3):365–370.
115. Wolford LM, Chemello PD, Hilliard FW. Occlusal plane alteration in orthognathic surgery. *J Oral Maxillofac Surg*. 1993;51(7):730–740; discussion 740–741.
116. Ayse T, Atac A, Karasu HA. Surgically assisted rapid maxillary expansion compared with orthopedic rapid maxillary expansion. *Angle Orthod*. 2006;76(3):353–359.
117. Eggensperger N, Smolka W, Rahal A, et al. Skeletal relapse after mandibular advancement and setback in single-jaw surgery. *J Oral Maxillofac Surg*. 2004;62(12):1486–1496.
118. Potts B, Shanker S, Fields HW, et al. Dental and skeletal changes associated with Class II surgical-orthodontic treatment. *Am J Orthod Dentofacial Orthop*. 2009;135(5):566.e1–566.e7.
119. Wolford LM, Chemello PD, Hilliard F. Occlusal plane alteration in orthognathic surgery: Part I: Effects of function and esthetics. *Am J Orthod Dentofacial Orthop*. 1994;106(3):304–316.
120. Wolford LM, Karras SC, Mehra P. Considerations for orthognathic surgery during growth, part 1: mandibular deformities. *Am J Orthod Dentofacial Orthop*. 2001;119(2):95–101.
121. Ueki K, Marukawa K, Hashiba Y, et al. Assessment of the relationship between the recovery of maximum mandibular opening and the maxillomandibular fixation period after orthognathic surgery. *J Oral Maxillofac Surg*. 2008;66(3):486–491.
122. Ueki K, Marukawa K, Moroi A, et al. Changes in border movement of the mandible in skeletal Class III before and after orthognathic surgery. *Int J Oral Maxillofac Surg*. 2014;43(2):213–216.
123. Bottger S, Streckbein P, Hartmann B, et al. Retrospective analysis of autologous blood use in bimaxillary repositioning osteotomy surgery: a quality improvement study. *Transfusion*. 2009;49(8):1747–1753.
124. Arnett GW, Tamborello JA. Progressive Class II development: female idiopathic condyle resorption. *Oral Maxillofac Surg Clin North Am*. 1990;2(4):699–716.
125. Mihalik CA, Proffit WR, Phillips C. Long-term follow-up of Class II adults treated with orthodontic camouflage: a comparison with orthognathic surgery outcomes. *Am J Orthod Dentofac Orthop*. 2003;123(3):266–278.
126. Jackson TH, Mitroff Sr, et al. Face symmetry assessment abilities: clinical implications for diagnosing asymmetry. *Am J Orthod Dentofacial Orthop*. 2013;144:663–671.
127. Sankey WL, Buschang PH, English J, et al. Early treatment of vertical skeletal dysplasia: the hyperdivergent phenotype. *Am J Orthod Dentofacial Orthop*. 2000;118(3):317–327.
128. Mojdehi M, Buschang PH, English JD, et al. Postsurgical growth changes in the mandible of adolescents with vertical maxillary growth patterns. *Am J Orthod Dentofacial Orthop*. 2001;119(2):106–116.
129. Williams DM, Bentley R, Cobourne MT, et al. Psychological characteristics of women who require orthognathic surgery: comparison with untreated controls. *Br J Oral Maxillofac Surg*. 2009;47(3):191–195.
130. Bjork A. Facial growth in man studied with the aid of metallic implants. *Acta Odontol Scand*. 1955;13:9–34.
131. Kawakami M, Yamamoto K, Inoue T, et al. Disk position and temporomandibular joint structure associated with mandibular setback in mandibular asymmetry patients. *Angle Orthod*. 2009;79(3):521–527.
132. Kersey ML, Nebbe B, Major PW. Temporomandibular joint morphology changes with mandibular advancement surgery and rigid internal fixation: a systematic literature review. *Angle Orthod*. 2003;73:79–85.
133. Reil RB. Computer planning and intraoperative navigation in craniomaxillofacial surgery. *Oral Maxillofac Surg Clin North Am*. 2010;22(1):135–166.
134. Hwang S, Chung CJ, et al. Changes of hyoid, tongue, and pharyngeal airway after mandibular setback surgery by intraoral vertical ramus osteotomy. *Angle Orthod*. 2010;80(3):302–308.
135. Uesugi T, Kobayashi T, et al. Effects of orthognathic surgery on pharyngeal airway and respiratory function during sleep in patients with mandibular prognathism. *Int J Oral Maxillofac Surg*. 2014;43(9):1082–1090.
136. Adydemir H, Memikoglu U, Karasu H. Pharyngeal airway space, hyoid bone position and head posture after orthognathic surgery in Class III patients. *Angel Orthod*. 2012;82(11):993–1000.
137. Patel PK, Morris DE, Gassman A. Complications of orthognathic surgery. *Craniofac Surg*. 2007;18(4):975–985.
138. Panula K, Finne K, Oikarinen K. Incidence of complications and problems related to orthognathic surgery: a review of 655 patients. *J Oral Maxillofac Surg*. 2001;59(10):1128–1136.
139. Tay AB, Poon CY, Teh LY. Immediate repair of transected inferior alveolar nerves in sagittal split osteotomies. *J Oral Maxillofac Surg*. 2008;66(12):2476–2481.
140. Park YC, Lee HA, Choi NC, et al. Open bite correction by intrusion of posterior teeth with miniscrews. *Angle Orthod*. 2008;78(4):699–710.
141. Eggensperger NM, Lieger O, Thüer U, et al. Soft tissue profile changes following mandibular advancement and setback surgery and average of 12 years postoperatively. *J Oral Maxillofac Surg*. 2007;65(11):2301–2310.
142. Valstar MH, Baas EM, Te Rijdt JP, et al. Maxillary sinus recovery and nasal ventilation after LeFort I osteotomy; a prospective clinical, endoscopic, functional and radiographic evaluation. *Int J Oral Maxillofac Surg*. 2013;42:1431–1436.
143. Karabouta I, Martis C. The TMJ dysfunction syndrome before and after sagittal split osteotomy of the rami. *J Maxillofac Surg*. 1985;13:185.
144. Kerstens HC, Tuinzing DB, VanderKwast WA. Temporomandibular joint symptoms in orthognathic surgery. *J Oral Surg*. 1989;17:215.
145. Kim M-R, Grabber TM, Viana MA. Orthodontics and temporomandibular disorder: a meta-analysis. *Am J Orthod Dentofacial Orthop*. 2002;121:438–446.
146. Lindenmeyer A1, Sutcliffe P, Eghtessad M, et al. Oral and maxillofacial surgery and chronic painful temporomandibular disorders—a systematic review. *J Oral Maxillofac Surg*. 2010;68(11):2755–2764.
147. Aragon SB, et al. The effects of orthognathic surgery on mandibular range of motion. *J Oral Maxillofac Surg*. 1985;43:938.
148. Proothi M, Drew SJ, Sachs SA. Motivating factors for patients undergoing orthognathic surgery evaluation. *J Oral Maxillofac Surg*. 2010;68:1555–1559.
149. Esperao PTG, deOliveira BH, et al. Oral health related quality of life in orthognathic surgery patients. *Am J Orthod Dentofacial Orthop*. 2010;137:790–795.
150. Jacobson A. Psychological aspects of dentofacial esthetics and orthognathic surgery. *Angle Orthod*. 1984;54:18.
151. Bhamrah G, Ahmad S, NiMhurchadha S. Internet discussion forums, an information and support resource for orthognathic patients. *Am J Orthod Dentofacial Orthop*. 2015;147:89–96.
152. Oland J, Jensen J, et al. Motives for surgical-orthodontic treatment and effect of treatment on psychosocial well-being and satisfaction: a prospective study of 118 patients. *J Oral Maxillofac Surg*. 2011;69:104–113.
153. Nicodemo D, Pereira MD, Ferreira LM. Self-esteem and depression in patients presenting angle class III malocclusion submitted for orthognathic surgery. *Med Oral Pathol Oral Cir Bucal*. 2008;13(1):E48–E51.
154. Williams B, Currimbhoy S, et al. Complications following surgically assisted rapid palatal expansion: a retrospective cohort study. *J Oral Maxillofac Surg*. 2012;70:2394–2402.
155. Groopman J. *Second Opinions: Eight Clinical Dramas of Decision Making on the Front Lines of Medicine*. New York: Penguin Books; 2000.

24

Self-Ligating Bracket Biomechanics

Nigel Harradine

OUTLINE

The Historical Context of Self-Ligation, 729
Stainless Steel Ligatures, 729
Elastomeric Ligatures, 729
Begg Pins, 730
Self-Ligation, 731
Factors That Hindered the Adoption of Self-Ligation, 731
Psychological Factors and Adoption of Self-Ligation, 735
Aesthetic Self-Ligating Brackets, 735
Proposed Core Advantages of Self-Ligating Brackets, 736
Speed of Archwire Insertion and Removal, 736
Secure Archwire Engagement, 736
Friction and Self-Ligation, 736
Friction with Active Archwires: Thick, Stiff Wires, 736
Friction with Active Archwires: Thin, Flexible Wires, 737
Friction in Vivo: Occlusal and Masticatory Forces, 738
Secure Ligation and Low Friction as a Combination of Properties, 739
The Clinical Significance of Low Friction, 739
Active Clips and Passive Slides, 740
Thin Aligning Wires Smaller Than 0.018 Inch in Diameter, 740
Wires Larger Than 0.018 Inch in Diameter, 741
Thick Rectangular Wires, 741
Aging of Spring Clips, 741
Active or Passive: Conclusion, 741
Alignment, 741
Friction, 741
Robustness and Ease of Use, 741
Self-Ligation and Treatment Efficiency, 741
Possible Reasons for Some Retrospective Studies Finding an Efficiency Advantage for Self-Ligation, 741
Possible Reasons for Randomized Controlled Trials Finding No Efficiency Advantage for Self-Ligation, 742
A Suggested Treatment Protocol for Future Randomized Controlled Trials, 742
Conclusions, 742
Self-Ligation and Patient Comfort, 742
Self-Ligation and Effectiveness of Torque Control, 742
Further Current Hypotheses About Self-Ligation, 743
Force Levels With Self-Ligation and Conventional Ligation, 746
Does Self-Ligation Reduce the Need for Extractions?, 746
Crowding, 746
Practical Consequences for Clinical Technique, 756
Choice of Self-Ligating Bracket, 756
Practical Tips in Light of the Evidence, 756
Summary, 757

THE HISTORICAL CONTEXT OF SELF-LIGATION

In the context of the current popularity of self-ligation, the continuing lack of consensus about its clinical benefits, and the plethora of types of self-ligating brackets, it is helpful to review the origins and motivations for development of this form of ligation. The vast majority of fixed orthodontic appliances have stored tooth-moving forces in archwires that are deformed within their elastic limit. For this force to be transmitted to a tooth, wires need a form of connection to the bracket. This connection has for many years been referred to as *ligation* because the early forms of connections were most frequently a type of ligature, for example, silk ligatures. The more recent forms of connections between bracket and archwire have all retained this title of ligation. *Elastomeric ligatures* and *self-ligating brackets* are firmly established orthodontic terms.

Stainless Steel Ligatures

When stainless steel became available, it was universally adopted as the method of ligation. Stainless steel ligatures have several beneficial inherent qualities. They are cheap, robust, and essentially free from deformation and degradation, and to an extent, they can be applied tightly or loosely to the archwire. They also permit ligation of the archwire at a distance from the bracket. This distant ligation is particularly useful if the appliance tends to use high forces from the archwires because this high force prevents sensible full archwire engagement with significantly irregular teeth. Despite these good qualities and their widespread use over many decades, wire ligatures have substantial drawbacks, and the most immediately apparent of these is the length of time required to place and remove the ligatures. One typical study[1] found that an additional 11 minutes was required to remove and replace two archwires if wire ligatures were used rather than elastomeric ligatures. Additional potential hazards include those arising from puncture wounds from the ligature ends and trauma to the patients' mucosa if the ligature end becomes displaced.

Elastomeric Ligatures

Elastomeric ligatures became available in the late 1960s and rapidly became the most common means of ligation, almost

entirely because of the greatly reduced time required to place and remove them compared with steel wire ligatures. It was also easier to learn the skills required to place these ligatures, so new clinicians and staff greatly preferred elastomerics. Intermaxillary elastics had been employed since the late 19th century, pioneered by well-known orthodontists such as Calvin S. Case and H.A. Baker. Initially, these elastic bands were made from natural rubber, but production of elastomeric chains and ligatures followed the ability to produce synthetic elastics from polyester or polyether urethanes. The ease of use and speed of placement of elastomeric ligatures did, however, lead to other definite disadvantages being generally overlooked although readily apparent. Elastomerics frequently fail to fully engage an archwire when full engagement is intended. Twin brackets with the ability to "figure of 8" the elastomerics are a significant help in this respect but at the cost of greatly increased friction, which is discussed later in this chapter. A paper by Khambay et al.[2] quantified the potential seating forces with wire and elastic ligatures and clearly showed the much higher archwire seating forces available with tight wire ligatures. A second and well-documented drawback with elastomerics is the substantial degradation of their mechanical properties in the oral environment. A comprehensive literature review of elastomeric chains[3] gave a good account of the relevant data, and a more recent article[4] discusses the underlying reasons and clinical significance of this loss of mechanical properties. Typically, elastomeric chains and ligatures suffer more than 50% degradation in force in the first 24 hours[5] when tested under in vitro experimental environments. The higher temperature in the mouth, enzymatic activity, and lipid absorption by polyurethanes are all cited as in vivo sources of force relaxation. This leads to the well-known potential for elastomeric ligatures to fail to achieve or to maintain full archwire engagement in the bracket slot.

Figure 24-1 shows the familiar loss of rotational control of canines during space closure. Twin brackets with the ability to "figure of 8" the elastomerics are a significant help in this respect but certainly not a complete answer. A further factor of potential clinical importance is the variability in mechanical properties of elastomerics. This is well described by Lam et al.,[6] who reported substantial variation in the range and tensile strength of elastomerics from different manufacturers and for different colors of elastomeric from the same manufacturer. Last, there is a large body of literature to demonstrate the much higher friction between bracket and archwire with elastomeric ligation compared with wire ligatures. Interestingly, this had been proposed as a factor of clinical significance more than 30 years ago[7] but was largely disregarded until more recently. The potential importance of friction and its relation to forms of ligation are discussed in more detail later in this chapter. The great popularity of elastomeric ligation in the past 40 years was achieved despite these substantial deficiencies in relation to wire ligatures. Speed and ease of use were the overriding assets of elastomerics, and it is no surprise that the strongest motivation behind the early efforts to produce a satisfactory self-ligating bracket was a desire to have all the benefits of wire ligation but in addition have a system that is quick and easy to use.

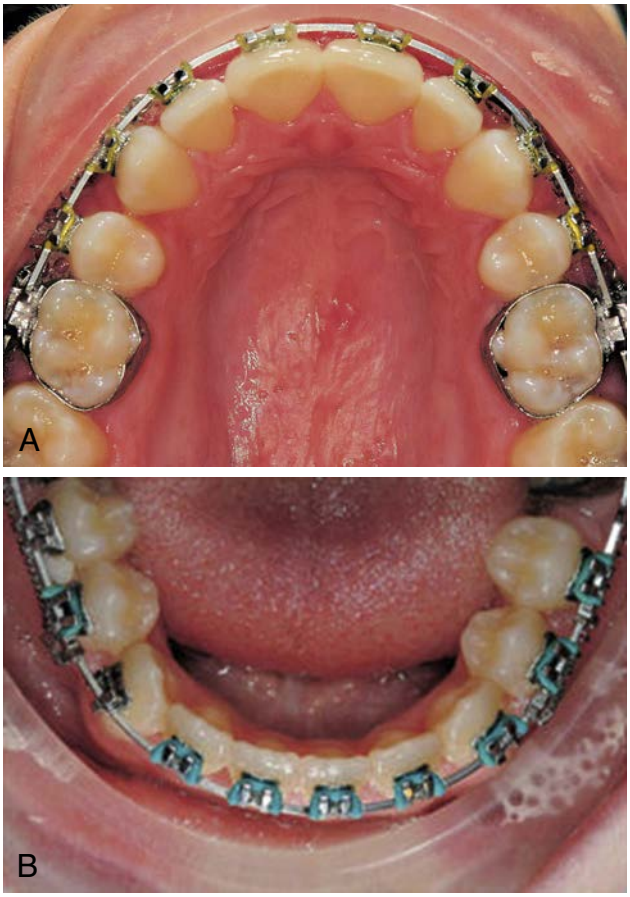


FIGURE 24-1 A, Loss of archwire control on upper canines with elastomeric ligation. B, Loss of elastomeric ligature causing loss of control of lower canine.

Begg Pins

In the 1950s, Raymond Begg, a former pupil of Edward Angle, developed his light-wire technique using Angle's ribbon arch brackets with round wire archwires.[8] A key feature of the technique was the use of brass pins as the method of ligation. These pins constituted the fourth (gingival) wall of the bracket slot and formed a rigid metal wall analogous in some ways to that of a molar tube or a self-ligating bracket. The pins were designed with shoulders to keep from binding the archwire in the early alignment stages and without shoulders as "hook pins" to hold the archwire in a more precise vertical position when thicker wires and auxiliaries were added later in the treatment. This author used many such pins, being trained simultaneously in Begg and edgewise mechanics during his initial specialist training. Begg pins had none of the disadvantages of elastomeric rings and were probably more rapid to place and remove than wire ligatures. These pins cannot be assessed in complete isolation from the rest of the Begg technique, but in relation to self-ligation, it is well worth noting the reputation that the Begg technique acquired for rapid early alignment and the effectiveness of lighter forces when there was no friction to be overcome from tight engagement with elastomerics. As a footnote in orthodontic history, it should be recalled that self-ligating Begg brackets were produced in the 1970s and were used by this author on a number of cases. They had an inbuilt pin that was rotated into position over the archwire with the intention being to further simplify and speed the process of ligation. Interestingly, when the Tip-Edge appliance was developed to be a successor to the Begg technique, it abandoned the metal, low-friction form of ligation that Begg pins represented and reverted to elastomerics.

Self-Ligation

Self-ligating brackets by definition do not require an elastic or wire ligature but have an inbuilt mechanism that can be opened and closed to secure the archwire. In the overwhelming majority of designs, this mechanism is some form of metal labial face to the bracket slot, which is opened and closed with an instrument or fingertip. Brackets of this type have existed for a surprisingly long time in orthodontics, the Russell Lock edgewise attachment being described by Stolzenberg[9] in 1935. This was by modern standards a very primitive mechanism consisting of a labial grub screw to retain the archwire. Since that time, many designs have become commercially available. Self-ligating orthodontic brackets have a relatively long history, but their development should be viewed against the background of an almost universal use of elastomeric ligatures despite the known advantages of wire ligatures and—in a different context—of brass Begg pins. Elastomeric ligation gives unreliable archwire control, high friction, and perhaps an added oral hygiene challenge. Wire ligation is better in every respect but is very slow and highly inconsistent in its force application,[10] and the wire ends can cause trauma to patient and operator. Orthodontists accommodated these shortcomings for several decades. Self-ligation has always offered the potential for very substantial improvements in relation to all of these drawbacks, but for many years remained the choice of a small minority of clinicians. The 21st century has seen a dramatic acceleration of bracket development, with at least 32 new bracket types becoming available. An overview of the status of self-ligation earlier in the current century[11] summarizes the situation at that time. The early part of this century also saw a proliferation of the advocated advantages of self-ligation and, more recently, a much greater research effort to gather the necessary related evidence. Before examining these claims and the associated evidence, it is helpful to look at the reasons why it took so long for self-ligation to attract this degree of attention.

FACTORS THAT HINDERED THE ADOPTION OF SELF-LIGATION

A contributory historical factor in this respect has been the significant imperfections in bracket design and performance. These imperfections have varied with different bracket designs. The author of this chapter has used 15 different types of self-ligating brackets and is therefore in a position to speak from some experience as well as from first principles. An ideal method of ligation should deliver ligation that is rapid and secure and provides low resistance to tooth movement relative to the archwire. Specific requirements for a self-ligating bracket are that:

- It is very easy to open and close with low forces applied to the teeth during these procedures and with all archwire sizes and materials.
- It never opens inadvertently, allowing loss of tooth control.
- It has a ligating mechanism that never jams or breaks or distorts or changes in its performance through the treatment period.
- It has a positively held open clip or slide position, so that the clip or slide does not obstruct the view of the bracket slot or the actual placement of the archwire.
- It is tolerant of a reasonable excess of composite material without obstructing the clip or slide mechanism.
- It is not significantly affected by buildup of calculus.

FIGURE 24-2 Edgelok bracket. An early passive self-ligating bracket.

- It permits easy attachment and removal of all the usual auxiliary components of an appliance such as elastomeric chain, under-tie ligatures, and laceback ligatures without interfering with the self-ligating clip or slide.
- It permits easy placement and removal of hooks and posts and possibly other auxiliaries on the brackets. With the security of self-ligation, the use of elastics or other traction directly to a bracket is much more frequently appropriate than with conventional ligation.
- It has a suitably narrow mesiodistal dimension to take advantage of the secure archwire engagement and permit large interbracket spans and hence lower force levels and a longer range of action.
- It has the performance expected of all orthodontic brackets in terms of bond strength, accuracy of slot dimensions, and smoothness of contour.

Many brackets have been less than satisfactory in several of these requirements, and this can be illustrated with a few better known examples.

- Edgelok brackets[12] (Ormco Corporation, Orange, CA) (Fig. 24-2) were the first self-ligating bracket to be produced in significant quantities. Disadvantages included inadequate rotational control, bulkiness, and some inconvenience with opening and closing the slide, and they were never widely adopted.
- The well-known Speed brackets[13] (Fig. 24-3) have remained in successful production since 1980. This testifies to the inherent soundness of many of the original design features. However, early Speed brackets (Fig. 24-4) were handicapped by clips that could too easily be displaced or distorted. These drawbacks have since been successfully addressed by improvements in the bracket body and in the clip itself, but combined with the inherent unfamiliarity for clinicians of a bracket with no tie wings, these aspects probably hindered the wider popularity of Speed in previous years.

FIGURE 24-3 Speed bracket with clip-restraining slot, labial clip-opening aperture, and slot preadjustment.

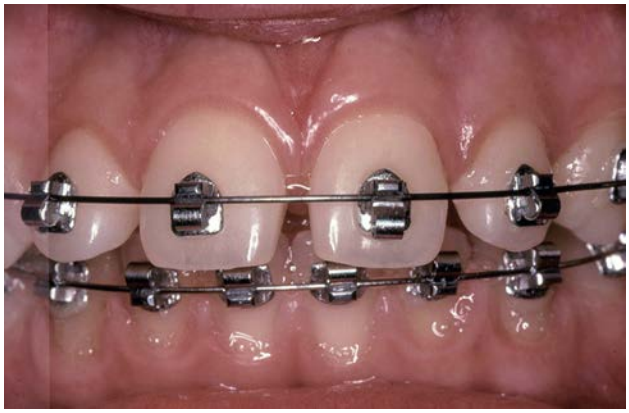


FIGURE 24-4 A case treated with early Speed brackets in 1982. There is no restraining slot preventing clip displacement and no straight-wire preadjustment in the bracket.

- Activa brackets[14] (Fig. 24-5) ("A" Company, San Diego, CA) had a rotating slide that therefore gave a concave inner radius to the labial surface of the slot. This curvature increased the effective slot depth with small diameter wires, diminishing labiolingual alignment with such wires. The slide was retained on the mesial and distal ends of the slot, and this made for a wider than average bracket, which reduced the interbracket span with the consequent disadvantages, and the slide was not robust. The absence of tie wings was an additional nuisance when placing the elastomeric chain, and the unfamiliar shape of the early bonding base made bracket positioning more difficult. Finally, a combination of the design features substantially reduced bond strength. Despite these substantial drawbacks, cases could be successfully treated that demonstrated the now-familiar potential advantages of self-ligation, but the deficiencies of the design ensured that they were only adopted by a minority of enthusiasts.

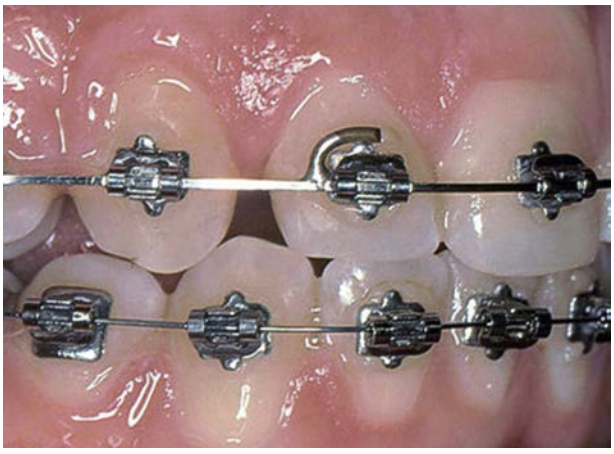


FIGURE 24-5 Activa brackets in 1990, illustrating the undesirable bracket width that reduces interbracket span, the concavity of the rotating clip, the lack of tie wings, and the unorthodox bonding base.

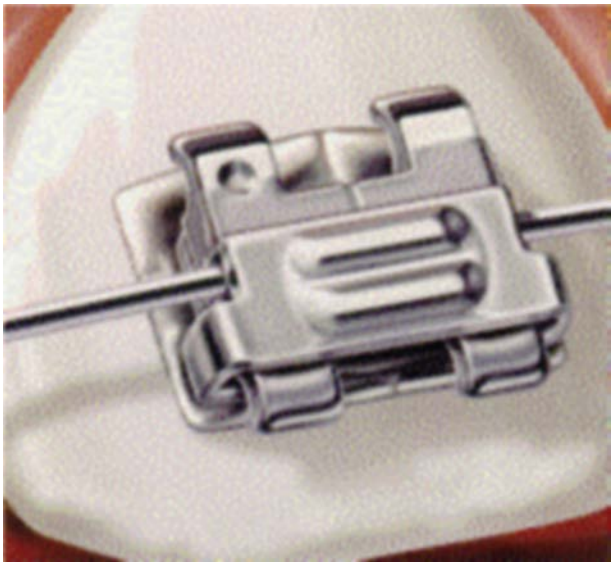


FIGURE 24-6 Damon SL bracket 1996, showing the wrap-around slide which was prone to fracture and loss.

- Damon SL brackets[15,16] (Fig. 24-6) ("A" Company) became available in the mid-1990s and had a slide that wrapped around the labial face of the bracket.

These brackets were a definite step forward but suffered two significant problems—the slides sometimes opened inadvertently because of the play of the slide around the exterior of the bracket, and they were prone to breakage because of work hardening on the angles of the slide during manufacture. One study[17] quantified these problems. In 25 consecutive cases in treatment for more than 1 year, 31 slides broke, and 11 inadvertently opened between visits. This is compared with 15 broken and lost elastomeric ligatures in 25 consecutively treated cases with conventional brackets, so the difference in ligation fragility was not enormous, but when a clinician has paid extra

FIGURE 24-7 Damon 2 bracket released in 2000, showing the more protected and more rigid slide that was produced by metal injection molding as was the bracket body.

for a novel bracket design and the main design feature is not highly robust and is susceptible to inexpert handling from inexperienced operators, it has a significant negative effect on widespread adoption of that bracket. Nevertheless, these brackets generated a substantial increase in the appreciation of the potential of self-ligation.

- Damon 2 brackets (Fig. 24-7) (Ormco Corporation, Orange, CA) were introduced to address the imperfections of Damon SL.

They retained the same vertical slide action and U-shaped spring to control opening and closing but placed the slide within the shelter of the tie wings. Combined with the introduction of metal injection molding manufacture, which permits closer tolerances, these developments almost completely eliminated inadvertent slide opening or slide breakage and led to a further acceleration in the use of self-ligation. However, the brackets were not immediately and consistently easy to open, and this aspect of functionality is very important to new users. Also, it was possible for the slide to be in a half-open position, hindering archwire removal or placement.

- Damon 3 brackets (Fig. 24-8) (Ormco Corporation) had a different location and action of the retaining spring, and this produced a very easy and secure mechanism for opening and closing.
- In addition, Damon 3 brackets were semiaesthetic. However, early Damon 3 production brackets had three very significant problems: a high rate of bond failure, separation of the metal from the reinforced resin components, and fractured resin tie wings. These three problems all received fairly rapid investigation and improvement but illustrate that it continues to be a significant challenge for manufacturers to extrapolate from the experience with prototype brackets in the hands of skilled enthusiasts to subsequent full-scale production and the use by relative novices. The subsequently launched all-metal Damon D3 MX bracket (Fig. 24-9) and more recent Damon Q bracket (Fig. 24-10) have clearly benefited from previous manufacturing difficulties and from further clinical experience and expertise. As with other brackets such as Speed and In-Ovation (GAC International, Bohemia, NY), these later Damon brackets also feature a slot for drop-in hooks, which was mentioned above in the list of ideal requirements.

FIGURE 24-8 Damon 3 semiaesthetic bracket showing the improved slide mechanism and the junction of metal and resin components, which was initially prone to separation.

FIGURE 24-9 Damon MX brackets showing the vertical auxiliary slot that permits placement and removal of drop-in hooks in this bracket and in the Damon Q. Drop-in hooks are very useful for placement of early intermaxillary elastics.

In-Ovation (Fig. 24-11) became available in 2002 and has more recently been called In-Ovation R and is very similar to the Speed bracket in conception and design but of a twin configuration with tie wings. Both of these additional features probably contributed to a greater acceptability of these brackets to new users. In 2002, smaller brackets for the anterior teeth became technically possible and available as In-Ovation R (Reduced, referring to the reduced bracket width), and this narrower width was certainly desirable in terms of greater interbracket span. Several self-ligating brackets have become smaller with development. This has been enabled by advances in manufacturing technology but is also driven by an appreciation that the

FIGURE 24-10 Damon Q bracket showing the additional horizontal slot for auxiliary archwires, a feature of several self-ligating brackets, including In-Ovation, Quick, and Speed.

FIGURE 24-11 An early In-Ovation bracket showing the tie wings that distinguished this bracket from Speed brackets.

secure ligation provided by a self-ligating bracket requires less bracket width to give good rotational control, within reason. In-Ovation is certainly a successful design, but some relatively minor disadvantages in relation to the list of ideal requirements can be experienced with the several examples of this type of bracket that use a spring clip that moves vertically behind the archwire slot. Some such brackets are a bit difficult to open, and this is more common in the lower arch, where the gingival end of the spring clip is difficult to visualize. Excess composite at the gingival aspect of brackets in the lower arch can be difficult to see and may also hinder opening. Similarly, lacebacks, under ties, and elastomerics placed behind the archwire are competing for space with the bracket clip. Both Speed and In-Ovation R as well as the similar and the more recent Quick brackets (Fig. 24-12) (Forestadent Bernhard Foerster GmbH, Pforzheim, Germany) have aimed to address some aspects of this potential difficulty by providing a labial hole or notch in the clip in which a probe or similar instrument can be inserted to open the clip.

The need to acquire the expertise of opening an unfamiliar bracket can dishearten new users of self-ligating brackets, and these more recent refinements of the method of opening are a definite advance in this respect. These refinements are also typical of the incremental improvement of self-ligating brackets that can take place without being appreciated by clinicians who have experienced difficulties with earlier production examples and consequently discontinued their use.

- SmartClip (3M, St. Paul, MN). This bracket (Fig. 24-13) is distinctive in retaining the wire through two C-shaped spring clips on either side of the bracket slot. The pressure required to insert or remove an archwire is therefore not applied directly to a clip or slide but to the archwire, which in turn applies the force to deflect the clips and thus permits archwire insertion or removal. This mechanism therefore has to cope with providing easy insertion and removal through the jaws of the clips but must also prevent inadvertent loss of ligation for both small, flexible archwires and large, stiff archwires.

FIGURE 24-12 Forestadent Quick bracket showing the design features shared by many active self-ligating brackets.

FIGURE 24-13 3M Unitek SmartClip bracket showing the distinctive external spring clips with their jaws through which archwires of all sizes and stiffness must ideally pass very easily and comfortably during archwire changes but never between patient visits.

This is an inherent conflict in requirements and one that is difficult to balance satisfactorily. Other spring clips, such as on the Speed and System R brackets with their vertical action, have a rigid bracket component to assist the spring in resisting a loss of ligation and are opened and closed vertically and independently of forces from the archwire. It became apparent with wider clinical use that the force required for insertion and removal of thick stainless steel wires from SmartClip brackets was uncomfortably high. More recent modifications have been aimed at addressing this difficulty by lowering the effective stiffness of the spring clips.

These examples all illustrate the difficulties that have been experienced by manufacturers aiming to meet the requirements of an ideal ligation system. The resulting imperfections in bracket design undoubtedly slowed the adoption of self-ligation systems by clinicians in previous years. Current self-ligation designs have benefited greatly from previous clinical experience and from advances in the available production techniques such as metal-injection molding, laser forming, and computer-aided design and computer-aided manufacturing technology. Although there remain current self-ligating brackets that do not succeed to a sufficient degree in meeting the listed characteristics of an ideal bracket, there are several that perform very well. Technical shortcomings in bracket design and functionality are no longer a reason to avoid self-ligation.

Psychological Factors and Adoption of Self-Ligation

Psychological factors are possibly of equal importance in their influence on the adoption of new technology or new ideas. Most of us have as part of our mindset an innate conservatism and a desire to stay mentally and technically within our comfort zone. We learn a skill that minimizes the deficiencies of our equipment, and then we stick with it because we are comfortable. The more skilled we become in a technique (e.g., bending complex archwires), the more unimpressed by and resistant to a new development (e.g., the straight-wire appliance), which reduces the need for this skill or demands different skills. We also make treatment plans that consciously or otherwise reflect the strengths and weaknesses of our current armamentarium. For example, an anchorage-demanding technique inherently leads to more extractions and more anchorage reinforcement as part of our plans. A second and related factor that previously hindered the rate of adoption of self-ligation has been a lack of widespread appreciation—and indeed of optimal scientific investigation—of what low-friction, secure archwire engagement and light forces can achieve in combination. This chapter examines the evidence for these potential benefits.

A further factor currently—and understandably—influencing the adoption of self-ligation has been the lack of support from clinical trials of some of the more enthusiastic claims made by originators of self-ligating brackets. This sequence of possibly optimistic claims followed by a lack of research support and consequent rejection by some of any and all merits in an appliance or technique is a common feature in most clinical disciplines. In orthodontics, another example is the reaction to functional appliances in the light of the lack of evidence of significant growth modification effect, disregarding the very useful dentoalveolar effects. Finally, the generally higher price of self-ligating brackets has acted as a deterrent despite evidence of reduced chairside time, which would offset some or all of the additional cost. It is perhaps important for clinicians to bear such factors in mind when forming their own reaction to any innovation.

AESTHETIC SELF-LIGATING BRACKETS

There have been several approaches to production of a more aesthetic self-ligating bracket. First, there are lingual self-ligating brackets. There are at least three lingual self-ligating brackets currently available. Forestadent (Bernhard Foerster GmbH, Pforzheim, Germany) has its lingual system, sometimes referred to as the Philippe bracket.[18] The ligation mechanism involves deforming two retaining wings with a Weingart plier to close and a spatula to open. This mechanism requires considerable care not to damage the enamel if an instrument slips. Also, the wings can be hard to open, which can cause detachment of the bracket. Adenta (Adenta GmbH, Gliching, Germany) produces the Evolution bracket, which is essentially a lingual version of the Time bracket produced by the same company; the same applies to In-Ovation L from GAC. Incognito appliances (LingualCare, Dallas, TX) have more recently added an accessory self-retaining slot on their incisor brackets. Ligation is inherently more difficult with lingual appliances, so an easy form of self-ligation clip or slide that can deliver the advantages of security and low friction are equally or even more valuable in the situation when the interbracket spans are inherently smaller. Combining a successful self-ligation mechanism with the particular lingual demands of low-profile, easy archwire insertion, inbuilt bite ramps on some teeth, and narrow bracket width is a demanding task. Further development is needed on this side of the teeth.

On the labial surface, Oyster (Gestenco, Gothenburg, Sweden) and OPAL Ultradent, South Jordan, UT 84095) and Damon3 (partially) were resin brackets. These had the disadvantages of all-resin polymer brackets and were not successful. Ceramic brackets with metal clips Clarity SL (3M Unitek) and In-Ovation C (GAC) have been more successful, and recently,

completely ceramic brackets (Damon Clear and Damon Clear2 (Ormco) have taken aesthetic self-ligating brackets to what is probably an entirely satisfactory level of aesthetics and mechanical performance. It is still possible that the optimal combination of self-ligation and aesthetics will come from a breakthrough in the technology for coating metal brackets.

PROPOSED CORE ADVANTAGES OF SELF-LIGATING BRACKETS

When considering the sometimes confusing advantages that have been proposed for self-ligation, it is helpful to divide these into the proposed core advantages and, in a second category, the various proposed consequent advantages that may possibly derive from this core. In the past 2 decades, a consensus has emerged on the potential core advantages, which can be summarized as:

- Faster archwire removal and ligation
- More certain full archwire engagement
- Less or no chairside assistance for ligation
- Low friction between bracket and archwire

Speed of Archwire Insertion and Removal

The principal motive when developing the earlier self-ligating brackets was to speed the process of ligation. A paper by Maijer and Smith[19] demonstrated a fourfold reduction in ligation time with Speed brackets compared with wire ligation of conventional brackets. Shivapuja and Berger[1] have shown similar results but also that the advantages compared with elastomeric ligation are less dramatic (≈1 minute per set of archwires). Voudouris[20] also reported a fourfold reduction in archwire removal or ligation time with prototype Interactwin brackets. A study by Harradine[17] found statistically significant but clinically much more modest savings in ligation or religation time with Damon SL, an average of 24 seconds per archwire removal and replacement. It should, however, be remembered that archwire "ligation" using self-ligating brackets does not require a chairside assistant to speed the process because self-ligating brackets require no passing of elastomeric or wire ligatures to the operator during ligation.

Turnbull and Birnie[21] investigated the difference in time taken to open and close brackets for different archwire groups with Orthos and Damon 2 brackets. The authors found time savings slightly greater than those of Harradine, with Damon SL brackets being 1 second per bracket for opening brackets and 2 seconds per bracket for closing brackets. It was twice as quick to close Damon 2 brackets as to ligate conventional Orthos brackets. For both bracket systems, the time taken to ligate and unligate archwires decreased with increasing archwire size and correspondingly better tooth alignment. The Damon 2 bracket was not the easiest of self-ligating brackets to open and close, and it is very probable that the more recent bracket types from that manufacturer would show much greater savings in time for archwire changes. The increased speed of archwire changes may not seem immediately compelling, but with brackets being ever-easier to open and close, a time saving of 1 to 2 minutes per archwire change is probably clinically significant. In looking at such studies, it should be remembered that archwire changes with self-ligating brackets were being done by a single-handed operator and were compared with four-handed changing of elastomeric ligatures. It is perhaps significant that there have been no more recent studies of this aspect of bracket performance; this probably reflects the current general acceptance that the evidence that self-ligation is more rapid is sufficiently strong.

Secure Archwire Engagement

Figure 24-1 illustrates the loss of full archwire engagement, which is commonly seen with elastomeric ligation. An inbuilt metal face to the bracket slot has the potential to ensure full archwire engagement. In the past, several self-ligating designs did not sufficiently fulfill that potential, but many self-ligating brackets now provide reliably secure ligation. A study by Mezomo[22] was one of many to show that the speed of tooth movement was no greater with self-ligation, but it also found that rotational control of the canine being retracted was better on the canines with a self-ligating bracket. A further consideration arises from the thoughtful study by Pandis et al.[23] that reveals that it cannot be taken for granted with at least one bracket type with an active flexible clip and that the designed mechanical properties of some self-ligation mechanisms will be sustained throughout treatment.

Friction and Self-Ligation

This is a well-researched area but one that remains open to misunderstanding. Very low friction was clearly demonstrated and quantified in many studies in the early 1990s for both Activa and Speed brackets and indeed Edgelok. More recent representative studies include one by Kapur et al.[24] that found that with nickel-titanium (NiTi) wires, the friction per bracket was 41 *g* with conventional ligation and 15 *g* with Damon brackets; with stainless steel wires, these values were 61 *g* and only 3.6 *g*, respectively. Thomas et al.[25] confirmed extremely low friction with Damon brackets compared with both conventional preadjusted and Tip-Edge brackets, but Time brackets with their active clip produced lesser but still substantial reductions in friction with larger diameter wires. Pizzoni et al.[26] similarly reported that the passive Damon brackets showed lower friction than Speed brackets, which in turn had less friction than conventional brackets. Earlier work that had concluded that each elastomeric placed in an "O" configuration produces an average of 50 *g* of frictional force per tooth was supported by Khambay et al.[2] using a method that gave zero friction for Damon 2 brackets and found mean frictional forces ranging between 43 and 98 *g* (0.43 and 0.98 cN) for various elastomeric–archwire combinations. All of these laboratory studies investigated the situation with essentially passive archwires in well-aligned brackets, so although this was a sensible starting point for investigating friction and does indeed accurately quantify the friction, it is a very imperfect and incomplete model of an actual clinical situation.

Friction with Active Archwires: Thick, Stiff Wires

When archwires are active, the total resistance to sliding (RS) is composed of FR (frictional resistance) + binding (BI) + (potentially) notching (NO). Several studies in the 1990s concluded that if the wires were active, the RS [It was defined above] with self-ligating brackets was not statistically significantly less than with conventionally ligated brackets. More recent studies are more sophisticated in terms of modeling the clinical situation and more perceptive in their assessment and interpretation of the clinical significance. In the investigation of RS on thick, rigid archwires, four excellent papers by Thorstenson and Kusy remain the best yardstick. Thorstenson and Kusy[27] examined the effects of varying active tip (angulation) of a 0.018- × 0.025-inch stainless steel wire on the RS. They found that angulation beyond the angle at

which the archwire first contacts the diagonally opposite corners of the bracket slot—at which point, binding begins—causes a similar rate of rise in RS for self-ligating (Damon SL) and conventional brackets. The greater the angle of tip, the smaller the percentage of total RS was attributable to friction (FR) because the friction remained constant while the binding linearly increased. So although at all degrees of tip, the Damon brackets produced less RS, for these authors, the key to correct interpretation of their study was the calculation, when clinically advisable forces were applied, of the realistic limiting angle of tipping at which the forces in that stiff wire would upright the tooth as part of a tip-and-upright walking of the bracket along the archwire. For Thorstenson and Kusy, that realistic angle of tip was calculated to be 6 degrees with a 0.018- × 0.025-inch stainless steel wire, at which angle the reduction in RS was from 140 to 80 *g*— that is, 60 *g* per tooth— and this value is very probably of clinical significance. At much higher experimental angles, the increase in binding "drowned out" this 60-*g* difference. In their words, "at low angles, the binding contribution to RS is small. As the angle increases, binding overwhelms friction and the overall effects of the ligation type and method decrease." It is important to note that the percentage contribution of friction to RS is very low with self-ligating brackets precisely because the friction is low. However, the difference in RS compared with conventional ligation remains at 60 *g* according to this research.

The second paper[28] compared different self-ligating brackets for RS with active angulations. It quantifies a little more closely the lower RS with passive self-ligation and points out that low resistance to tooth movement can lead to unanticipated movement as is discussed later in this chapter. The third and fourth papers[29,30] examined the same factors with wires of different sizes and in the dry state and in conventionally ligated brackets with novel design features aimed at reducing RS. The increase in friction when larger wires deflect the clips in active self-ligating brackets is quantified, and the scanning electron micrographs of the different brackets show very clearly the relationship between small and large wires and active clips and passive slides. More recently, Pliska et al.[31] also assessed tipping moments in relation to RS in thick (0.019- × 0.025-inch) wires with self-ligating and conventional brackets. They too found that with "low" forces of 200 *g*, there was a 40-*g* reduction in RS per bracket with self-ligating brackets but that this difference was lost as the applied forces increased to 400 *g*. These investigations are consistent in indicating that although binding provides the majority of RS (and indeed is essential for the force of the wire to be transmitted), the greatly reduced friction with self-ligation contributes a reduction of 40 to 60 *g* per tooth, and at appropriately low levels of force application, this is likely to represent a clinically significant reduction.

Friction with Active Archwires: Thin, Flexible Wires

The relationship between friction and RS has also been extensively researched in thin, flexible wires. Matarese et al. (2008)[32] is an early but sound study of RS in self-ligating brackets with such wires. Using the simple setup of a variably offset bracket (Damon 2 in this instance) and a straight section of wire, this study showed a substantial reduction in RS compared with conventional elastomeric ligation. More recently, Heo and Baek (2011)[33] found similar reductions in RS with self-ligation using a greatly more sophisticated laboratory apparatus that incorporated teeth of realistic anatomic size and shape, periodontal ligaments of a material of similar mechanical properties, and a full dental arch. The differences varied with different aligning wires and brackets, but when modeling a 3-mm vertical displacement of a canine, the RS ranged from 2700 to 3800 cN with conventional ligation and 390 to 850 cN with self-ligation. These differences seem to be of potentially greater clinical significance than those found in thick, stiff archwires.

Baccetti (2009)[34] perceptively researched the important corollary to this difference in RS. He clearly demonstrated that the increased RS with conventional ligation is accompanied by a corresponding reduction of force available to align the displaced tooth. This effect was such that at a 4-mm displacement of a bracket, no residual aligning force remained on the displaced tooth. Of course, Newton's laws tell us that the total force in the system is the same with both types of brackets, but the distribution of force is markedly different if the teeth are significantly irregular, and it can be inferred that the pattern of tooth movement would be correspondingly different. In fact, this observation had been made 8 years earlier by Thorstenson and Kusy[27] in relation to thick archwires. "The self-ligating bracket allows more of the applied force to be used for sliding than does the conventional bracket." Other workers, including Franchi et al. (2009)[35] investigating buccally displaced teeth and Petersen et al. (2009)[36] investigating lingually displaced teeth, have demonstrated the same marked and unfavorable alteration in force distribution with different patterns of tooth irregularity with conventionally ligated brackets.

The group at the University of Alberta has carried out an even more sophisticated experimental modeling of forces and moments[37] (Fig. 24-14). This has shown a similar markedly different force and moment distribution when comparing self-ligating and conventionally ligated brackets for a vertically displaced canine. With conventional ligation, the reciprocal forces on either side of the high canine were not only much more widely spread around the arch but also had a much larger labial/buccal component. These results fit with the conventional clinical wisdom that advises against full engagement of markedly displaced teeth on the grounds that the very irregular teeth may not move much but that previously well-aligned

FIGURE 24-14 The Orthodontic Simulator (OSIM) developed at the University of Alberta. Multiaxis force and moment transducers are attached to all 14 teeth. Micrometer screws permit quantified and incremental movement in the vertical and horizontal planes and simultaneous recording of forces and moments in all three planes. (By kind permission of Dr. Hisham Badawi.)

teeth may move adversely, for example, producing labial flaring of the anteriors in this instance. Figure 24-15 shows a clinical example of self-ligation in the high canine scenario that was researched in the paper by Fok et al.[37] It shows the effective movement of the canine with no detectable adverse tooth movement from reciprocal forces that would be anticipated with conventional ligation and that was indicated by the striking results of the research by the Alberta group.[37] All of these investigations have demonstrated that engaging significantly displaced teeth in self-ligating brackets gives greater desirable force on the displaced teeth and less unwanted reciprocal forces on the other teeth.

Such work strongly supports the view that even though ligation is only one source of the RS, self-ligation can reduce this resistance to a clinically significant extent and that this has consequences on force distribution. A systematic review of this question by Ehsani et al.[38] concluded that the case is proven for lower RS with self-ligation with round wires but not for the larger rectangular wires or with active archwires where more evidence is still required. An interesting facet of the study selection for this review is that two of the seminal papers by Thorstenson and Kusy referred to earlier were not mentioned. The conclusions of that review may have been overtaken by some of the more recent research summarized here. The most appropriate response to these research findings in terms of their clinical application is discussed later in this chapter.

Friction in Vivo: Occlusal and Masticatory Forces

A further potentially confounding factor has been investigated in studies by Braun et al.[39] and O'Reilly et al.,[40] who found that various vibrations and displacements of a test jig (to mimic intraoral masticatory forces) can substantially reduce the frictional component of RS with conventional ligation. This is a valid line of inquiry and an interesting finding, but the question then arises as to how accurately these laboratory studies mimic intraoral masticatory "jiggling" forces. An impressive study by Iwasaki et al.[41] used an intraoral device to produce a combination of tipping and ligation forces and measured the effect of chewing gum on the resulting RS. They concluded, "These results refute the hypothesis that masticatory forces consistently and predictably decrease friction." It could also be proposed that any beneficial effects of masticatory forces might apply equally to conventional and to self-ligation and therefore maintain the RS differential between these types of brackets. Clinically—and rather ironically—the low friction with self-ligating brackets seems very evident from the need to place a stop on all archwires to prevent the much greater tendency for the archwire to slide through the brackets and traumatize the mucosa distally. Measurement of the variables that influence in vivo friction will remain a challenge, but progress is being made. Although there is still ample scope for more and better studies of this potentially complex field, current evidence supports the view

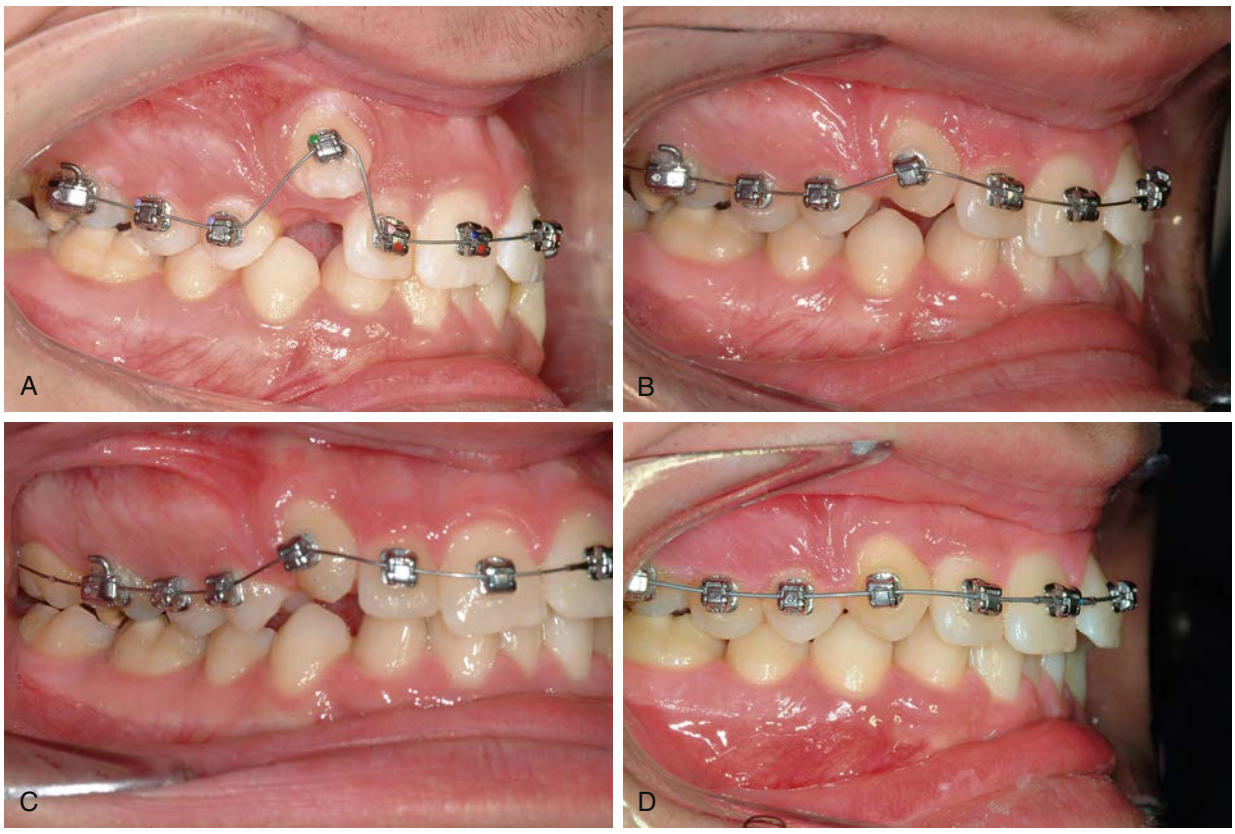


FIGURE 24-15 Full ligation of a markedly irregular canine tooth with self-ligating brackets results in the movements that laboratory research predicts. **A,** A fully engaged 0.014-inch nickel–titanium (NT) wire. **B**, **C,** The next visit. **D,** Engagement of a 0.014- × 0.025-inch NT at the following visit. Low friction (and hence lower resistance to sliding) ensures that sufficient extrusive force remains to align the canine while the reciprocal force causes very little incisor extrusion and the excess wire escapes distally with no detectable incisor proclination.

that RS in vivo is lowered to a clinically very significant extent with self-ligation, especially with passive self-ligation.

Secure Ligation and Low Friction as a Combination of Properties

Other bracket types—most notably, Begg brackets—have low friction by virtue of an extremely loose fit between a round archwire and a very narrow bracket, but this is at the cost of making full control of tooth position correspondingly more difficult. Some brackets with an edgewise slot have incorporated shoulders to distance the elastomeric from the archwire and thus reduce friction, but this type of design also produces reduced friction at the expense of reduced control because the shoulders that hold the ligature away from the archwire increase the slot depth and reduce the tension in the elastomeric. This reduces the control of rotations or of labiolingual tooth position. Elastomeric rings cannot provide and sustain sufficient force to maintain the archwire fully in the slot without also pressing actively on the archwire to an extent that increases friction. Comparison with a molar tube is helpful in this context because such an attachment is in essence a passive self-ligating bracket with the slide permanently closed. If a convertible molar tube is converted to a bracket by removal of the slot cap or straps, an elastomeric or even a wire ligature can prove very ineffective at preventing rotation of the tooth if it is moved along the wire or used as a source of intermaxillary traction. These ligation methods simultaneously increase friction while attempting to retain full archwire engagement. The challenge of simultaneously combining low friction and good control was very nicely illustrated in an article by Matasa[42] and is reproduced here with his kind permission (Fig. 24-16).

Matasa investigated "low-friction" conventional brackets. He showed that all steps to reduce friction in the design of such brackets that involved distancing the elastomeric from the archwire produced a consequent reduction of tooth control. The investigation into three such "low-friction" bracket types by Thorstenson and Kusy[30] also found no evidence that bumps in the floor of the archwire slots reduced RS. With tie-wing brackets and conventional ligation, a reduction in friction is usually at the cost of deterioration in control. The combination of very low-friction and very secure full archwire engagement in an edgewise-type slot is currently only possible with self-ligating brackets (or with molar tubes) and is likely to be the source of the most beneficial effects of such brackets. This combination enables a tooth to be slid along an archwire with lower and more predictable net forces and yet under good control with almost none of the undesirable rotation of the tooth resulting from a deformable or degradable mode of ligation such as an elastomeric.

The Clinical Significance of Low Friction

Friction between the archwire and bracket must be overcome for the majority of tooth movements to occur. Such movements include vertical leveling, buccolingual alignment, rotation, correction of angulation, opening of space, and any space closure with sliding mechanics. All of these tooth movements involve movement of the bracket relative to the archwire. Frictional forces arising from the method of ligation are one source of the resistance to this relative movement, and although its relative contribution to RS decreases with increasing archwire activation, the research summarized earlier indicates that a clinically significant difference remains with both thick and thin archwires. Correspondingly higher forces must therefore be applied to overcome this resistance, and this has two related potential effects that would be expected to inhibit or change tooth movement. First, the net effective force is much harder to assess and is more likely to be undesirably higher than levels best suited to create the optimal histologic response. Second, the binding forces are correspondingly higher both between bracket and wire and also at the contacts between irregular adjacent teeth. These binding forces also inhibit the required relative movement between bracket and wire. Only a few tooth movements such as space closure with closing loops placed in the space, expansion of a well-aligned arch, and torque (inclination) changes are not potentially influenced by a low-friction method of ligation. One situation in which the combination of low friction and secure full engagement would theoretically be expected to be particularly useful is in the alignment of very irregular teeth and especially the resolution of severe rotations when the capacity of the wire to slide through the brackets of the rotated and adjacent teeth significantly facilitates derotation. This relationship between friction and derotation has been classically described and quantified by Koenig and Burstone[43] in which the potential adverse forces from friction were shown to be very large. Low friction should theoretically therefore permit more rapid alignment while the secure bracket engagement with self-ligation permits full engagement with severely displaced teeth and full control while the brackets move relative to the archwire.

Figure 24-17 shows the ability of self-ligation to fully engage and maintain engagement and control of an archwire in a very rotated tooth, while the low friction enables release of binding as the archwire slides through all the brackets in the arch. Modern

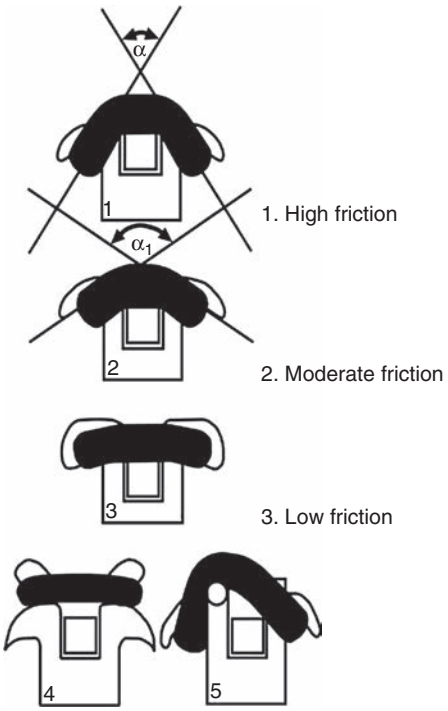


FIGURE 24-16 Illustration showing the trade-off between low friction and good archwire control with conventional ligation. (From Matasa CG: Brackets' shape influences friction, Orthod Materials Insider 13: 2-5, 2001)

FIGURE 24-17 A–C, Three consecutive visits illustrating the effectiveness of rotational alignment that results from the combination of low friction and good archwire engagement with self-ligation.

low-modulus wires with their high elastic limit would be expected to substantially enhance our ability to harness these benefits.

ACTIVE CLIPS AND PASSIVE SLIDES

The choice of design is an issue that has attracted assertive debate.[44] It is therefore worth a fairly detailed consideration.

Speed, In-Ovation, and Forestadent Quick brackets are examples of a number of brackets that have a spring clip, which encroaches on the slot from the labial aspect, potentially generating an additional force on the tooth. With some of the force being stored in the deflection of the spring clip, these brackets are referred to as active clips. In contrast, passive brackets have a slide that closes to create a rigid labial surface to the slot with no intention or ability to invade the slot and store force by deflection of a metal clip. Damon, SmartClip, Lancer Pacific Praxis Glide, Class One/Ortho Organisers Carrière LX, American Orthodontics Vision LP, Ortho Technology Lotus, and OrthoClassic Axis are examples of passive systems. Some literature has occasionally contained statements to the effect that the term “passive” is inappropriate because there must be force between the bracket and the wire for teeth to move. This is an unhelpful and possible disingenuous interpretation of the word passive when used in this context, and the term “passive” is entirely comprehensible when defined as above, meaning a bracket analogous to a molar tube.

An active clip can store some of the applied force in the clip as well as in the wire. The intended benefit is that in general terms, a given wire will have its range of labiolingual and possibly torqueing action extended and produce more alignment than would a passive slide with the same wire. This needs more detailed consideration. It is perhaps helpful to think of the situation with three different wire sizes.

Thin Aligning Wires Smaller Than 0.018 Inch in Diameter

The potentially active clip will be passive and irrelevant unless the tooth (or part of the tooth if it is rotated) is sufficiently lingually placed in relation to a neighboring tooth that the wire touches the clip. In this situation, a higher total force is usually applied to the tooth compared with a passive slide. Even if there is no significant clip deflection, there is still a force on the wire that would not exist with a passive clip because the active clip effectively reduces the slot depth from 0.028 inch to approximately 0.018 inch, either immediately—if the clip is not deflected—or as the wire goes passive if it is deflected. For teeth that were initially placed lingual to their neighbors, the active clip can bring the tooth more labially (up to a maximum of $0.028 - 0.018 = 0.010$ inch) with a given wire. These figures are slightly complicated by the fact that the active clip does not reduce the slot depth to the same extent over the whole height of the slot; all active clips impinge into the slot more at the gingival end than at the occlusal. This is well visualized in the illustrations in Thorstenson and Kusy.[28] This asymmetry would make a difference with small-diameter wires depending on the relative vertical positions of neighboring teeth. The effect of having an active clip at this early stage of treatment can be thought of as having a potentially shallower bracket slot. This will frequently produce higher forces and correspondingly higher friction with a given wire but a potential maximum extra 0.010 inch of labial movement of some teeth for a given small-diameter wire. This figure is approximate for the reasons given earlier.

Wires Larger Than 0.018 Inch in Diameter

The active clip will place a continuous lingually directed force on the wire even when the wire itself has gone passive. On teeth that are in whole or in part lingual to a neighboring tooth, the active clip will again bring the tooth (or part of the tooth if rotated) to a fractionally more labial position than would have been the case with a passive slide. The maximum difference will be the difference between the labiolingual dimension of the wire and 0.028 inch. For a typical 0.016- × 0.022-inch intermediate wire, this would give a maximum difference of 0.006 inch. 0.016- × 0.025-inch or 0.014- × 0.025-inch NiTi wires are recommended as the intermediate aligning wire for passive brackets, and this wire reduces this potential difference to a clinically insignificant 0.003 inch. Lingually placed teeth would have a higher initial force with an active clip and wires of this intermediate size. With an active clip, an active lingually directed friction force will remain on the wire even when it is passive.

Thick Rectangular Wires

An active clip will probably make a labiolingual difference in tooth position of 0.003 inch or less, which is clinically very small. The suggestion that continued lingually directed force on the wire from an active clip (or from a conventional ligature) will cause additional torque from an undersized wire is interesting and is addressed later in this chapter. In relation to active clips or passive slides, a representative study is by Major et al.,[45] who demonstrated that the lingually directed force from an active clip does not contribute to torqueing capacity by reducing the "slop" or "play" angle at which sufficient force is generated to influence third-order tooth position.

Aging of Spring Clips

Finally, there are the important questions of robustness, security of ligation and ease of use. Is a clip, which is designed to flex, more prone to breakage or permanent deformation or to inadvertent opening or closing? This question has not yet been investigated sufficiently, but one pertinent piece of work has been conducted by Pandis et al.,[46] who retrieved spring clips from Speed and In-Ovation R brackets after treatment and compared the stiffness and range of action of these spring clips with those of unused spring clips. The two types of brackets had spring clips of very different initial stiffness and differed in their performance during treatment. The Speed clips changed insignificantly in their performance, but the In-Ovation clips lost an average of 50% of their stiffness during the treatment. This change in properties is sufficient that it may well have biomechanical consequences of clinical significance.

Active or Passive: Conclusion

Alignment

It is probable that with an active clip, initial alignment is more complete for a wire of given size to an extent that is potentially clinically useful. With modern low-modulus wires, it is possible to sequentially insert thicker wires into a bracket with a passive slide and arrive at the largest desired archwire size after a similar number of visits.

Friction

Overall, an active clip will generate higher archwire forces and higher resistance to tooth movement. The increased clearance between a given wire and a passive slide will generate lower forces and may facilitate dissipation of the adverse binding forces and the ability of teeth to push each other aside as they align. This may also lead to qualitative differences in the direction and amount of tooth movement, or the differences in friction may be too small to have a clinical effect. The evidence is insufficient on this question.

Robustness and Ease of Use

These are factors of almost overriding importance compared with the active versus passive issue per se. In any specific bracket, these factors are frequently related to the type of clip or slide, whether active or passive.

SELF-LIGATION AND TREATMENT EFFICIENCY

In the light of substantial experimental evidence that self-ligation reduces RS and alters the ratio of favorable to unfavorable forces, an important area of study has focused on whether these differences can be shown to translate into more efficient or more effective treatment. Principally, these have focused on speed of treatment, number of patient visits, and final occlusal irregularity as assessed by the PAR score. The hypothesis is that lower RS enables more effective relative movement between archwire and bracket at lower levels of applied force and hence more rapid tooth movement, and the reliable and full bracket engagement prevents the waste of time regaining tooth control. Some published retrospective case control studies[17,47] have indeed found greater treatment efficiency, but another study[48] did find a reduction in patient visits but no reduction in treatment duration. In contrast, the more recent randomized controlled trials (RCTs)[49–57] have almost all failed to show any such effect, although one[57] did find an average reduction of 50 minutes in chairside time with self-ligation. This contrast requires some scrutiny.

Possible Reasons for Some Retrospective Studies Finding an Efficiency Advantage for Self-Ligation

In the retrospective studies that did find an efficiency advantage for self-ligation:

- The groups may not have been adequately matched for type and complexity.
- Some other factor may have been confounding the comparison with an RCT in which only the bracket is different (e.g., a different policy on extractions, archwire sequence, or appointment interval with the different brackets).
- The case mix may have been unusual (e.g., more complex cases than average).

With regard to matching of cases, in one of these studies,[17] great care was taken to match the patients in terms of age, type of malocclusion, extraction pattern, and initial PAR score. Regarding case mix, this may well be a factor because it is probable that any actual advantages of self-ligation would be more pronounced in certain cases. The cases in one paper[17] were substantially more complex than average, reflecting the particular tertiary referral setting of that study.

A significant potential confounding factor would be a concurrent change of practice as well as change of bracket. A clear potential instance would be a change to prescribing fewer extractions when also adopting self-ligating brackets. This was not the case in one of these papers,[17] but this does raise an interesting point about experimental design. Assuming for a moment that self-ligation, through a reduced RS, facilitates the alignment of adjacent crowded teeth, it can be argued that it is only sensible to take advantage of a lessening of one of the several reasons for

extraction and to change the pattern and extent of extractions. Similar reasoning can be applied to choice and use of archwires. If particular wires or treatment intervals are more suitable with self-ligation (which in certain circumstances, they are), is it sound scientific investigation to insist on keeping these the same for both bracket types? Although cases should surely be well matched, there is a tenable argument that aspects of treatment mechanics should play to the perceived strengths of each type of bracket. However, the problem with varying more than just the bracket between the groups studied is, of course, that it is hard to attribute any differences that are found to a specific factor. This dilemma is discussed further in the next section.

Possible Reasons for Randomized Controlled Trials Finding No Efficiency Advantage for Self-Ligation

Similar considerations apply to the design RCTs, which have almost all failed to find a difference in treatment efficiency.

- As discussed earlier, factors such as case mix, appointment interval, archwire sequence, and biomechanics may not have been optimized for the self-ligating brackets but have been chosen to be identical regardless of bracket type and may in fact be more suited to treatment with conventional ligation.
- When a difference in treatment for the two groups was permitted,[57] the self-ligating group was treated with 12-week intervals throughout and the conventional group with 6-week intervals. This was certainly defendable in that, at the time of planning the study, the manufacturers' written advice was indeed for 12-week intervals with their self-ligating brackets. It is now self-evident that this was a misguided extrapolation of a sensible proposal, namely, that because research has indicated that more full engagement of early aligning wires in substantially irregular arches is more appropriate with self-ligation, it follows that a longer first appointment interval is sensible in that situation. It is not logical to extrapolate from that to propose that a much longer appointment interval should be used in all cases and throughout treatment. The result in this study was that although the self-ligation cases required an average of 50 minutes less chairside time, the treatments took slightly longer than for the conventionally ligated group. It is surprising that the difference in treatment length was not larger. These are findings of interest but are not testing the central question about treatment efficiency when the current best assessment of the optimal use of self-ligation is used.
- RCTs of novel orthodontic appliance systems are susceptible to the involvement of clinicians who are unfamiliar with one of the appliances. This may be a factor in some of these studies.
- The study on closure of extraction spaces[54] primarily involved the archwire sliding only through molar tubes, which therefore applied equally in both groups. There have been several such studies that are clearly not suited to testing the demonstrated biomechanical differences of self-ligation.
- The NiTi dilemma refers to the fact that it is almost universally accepted that NiTi wires are superior for tooth alignment compared with their stainless steel predecessors. However, none of the studies that investigated that hypothesis ever demonstrated its truth. Similarly, none of the relevant studies ever demonstrated that straight-wire appliances were superior to plain edgewise, but the former are overwhelmingly preferred for reasons that are regarded by clinicians as being self-evident and in no need of the highest order of scientific proof.

A Suggested Treatment Protocol for Future Randomized Controlled Trials

With regard to the choice of patients, archwires, appointment intervals, extraction patterns, extent of bracket engagement, and timing of such biomechanics as intermaxillary elastics, a suggestion would be to insist on these being the same for each group in an RCT, but the protocol chosen should be that which the evidence suggests is most appropriate to take advantage of self-ligation rather than that which has evolved over many decades to best suit conventional ligation.

Conclusions

Although there has been much good research, the question of treatment efficiency remains incompletely resolved. The current evidence strongly suggests that there is not a substantial, blanket enhancement of treatment efficiency that is generally applicable in all cases and situations, however the brackets are used. Perhaps there is no efficiency advantage. The laboratory research does, however, suggest that if investigators and funding persist (and they did not with NiTi wires or the straight-wire appliance) and the best design of study is chosen as outlined earlier, then it may be shown that in some malocclusions treated with particular brackets and wires and treatment intervals, self-ligation is more efficient. It would be surprising if those fairly frequent cases in which conventional ligation allows a loss of tooth control (such as in Fig. 24-1) do not take longer or more chairside time to treat. Currently, the only clearly substantiated gain in efficiency results from faster ligation and the lack of need for assistance with ligation. A summary of opposing slants on the current evidence can be found in the two point–counterpoint articles[58,59] commissioned by the *American Journal of Orthodontics* and *Dentofacial Orthopedics* to take those opposing stances.

SELF-LIGATION AND PATIENT COMFORT

The hypothesis that self-ligation may be less painful is based on the assumption that forces on the teeth will be lower. This is not necessarily the case, being particularly sensitive to the clinician's choice of archwire. There have been a number of studies investigating the hypothesis that self-ligation produces less patient discomfort than conventional brackets and ligation, and their findings have varied.[60,61] However, a systematic review[62] concluded that the evidence to date did not support a reduced level of pain with self-ligation. A very interesting paper by Yamaguchi et al.[63] examined the question not by self-reported discomfort levels but via an objective marker of the inflammation and associated pain resulting from orthodontic forces. They measured levels of the neuropeptidase substance P in gingival crevicular fluid. They found that treatment with Damon brackets significantly lowered the levels of this marker of pain and inflammation compared with conventional ligation at 24 hours after archwire placement. Force levels are discussed later in this chapter.

SELF-LIGATION AND EFFECTIVENESS OF TORQUE CONTROL

The hypothesis has been advanced that torque control is more difficult with self-ligating brackets and perhaps particularly with passive brackets. This proposal is based on the belief that the labiolingual forces between the base of the bracket and a ligature system are a significant additional source of force couple, adding to the couple between the upper and lower bracket walls

that exists with all brackets. Several good studies have been carried out to assess the contribution of ligation force to torque effectiveness. Major et al.[64] and Brauchli et al.[65] found that the additional force from the active clips of Speed and In-Ovation brackets did not provide any effective additional torque force. Sifakakis (2014)[66] found that elastomeric ligatures also added no clinically effective force even immediately after placement, and this finding was reinforced by Al Fakir et al. (2014).[67] The situation with wire ligatures is a little more nuanced. Al Fakir found that if "tight" wire ligatures were added to conventional or Damon Q brackets when no active torque was present and then the brackets twisted to create torque, then the "slop" or play before torque forces became effective was reduced from 15 degrees to 9 degrees, indicating some added torque control from tight wire ligatures. However, if wire ligatures were added to brackets already producing active torque, then the torque "slop" or play was not reduced. These are helpful findings. Elastomeric ligatures add nothing to torque delivery, and tight wire ligatures add a little under some circumstances.

FURTHER CURRENT HYPOTHESES ABOUT SELF-LIGATION

The core advantages of faster ligation, better archwire control, and lower RS can and should continue to benefit from scientific study on points of detail, but they can at this stage be safely regarded as sufficiently proven. The further hypotheses concerning more efficient treatment, more comfortable treatment, and healthier treatment are all receiving appropriate investigation but remain unproven hypotheses. There remains a third category of proposed advantages that can be described as proposed qualitative differences in tooth alignment. Many clinicians who use self-ligating brackets believe that when combined with light round wires, they facilitate the alignment of crowded teeth. However, some clinicians have additionally proposed that the teeth align in a qualitatively different manner, producing less incisor proclination and more lateral expansion than conventional ligation. In this respect, the following qualitative differences have been proposed with self-ligation:

- Less incisor proclination for alignment of a given amount of crowding
- Wider arches, which may be more aesthetic
- Wider arches that have better preserved bone levels and consequent periodontal health than those resulting from more rapid and forceful expansion
- Wider arches that may be more stable
- Less need for extractions
- Easier Class 2 correction through a "lip bumper" effect

These proposals are far from established. Many of them have been set out and illustrated with case examples and medical computed tomography scans.[68] A well-known case example (Fig. 24-18), which helps to illustrate several of these proposals, is reproduced by kind permission of Dr. Dwight Damon. In this Class 2, Division 2 malocclusion, the very crowded arches have been aligned without extractions. The expectation would be that this would result in very pronounced incisor proclination. In fact, the incisors moved labially less than 3 mm, but the posterior arch expansion was much greater than might have been anticipated (Fig. 24-19).

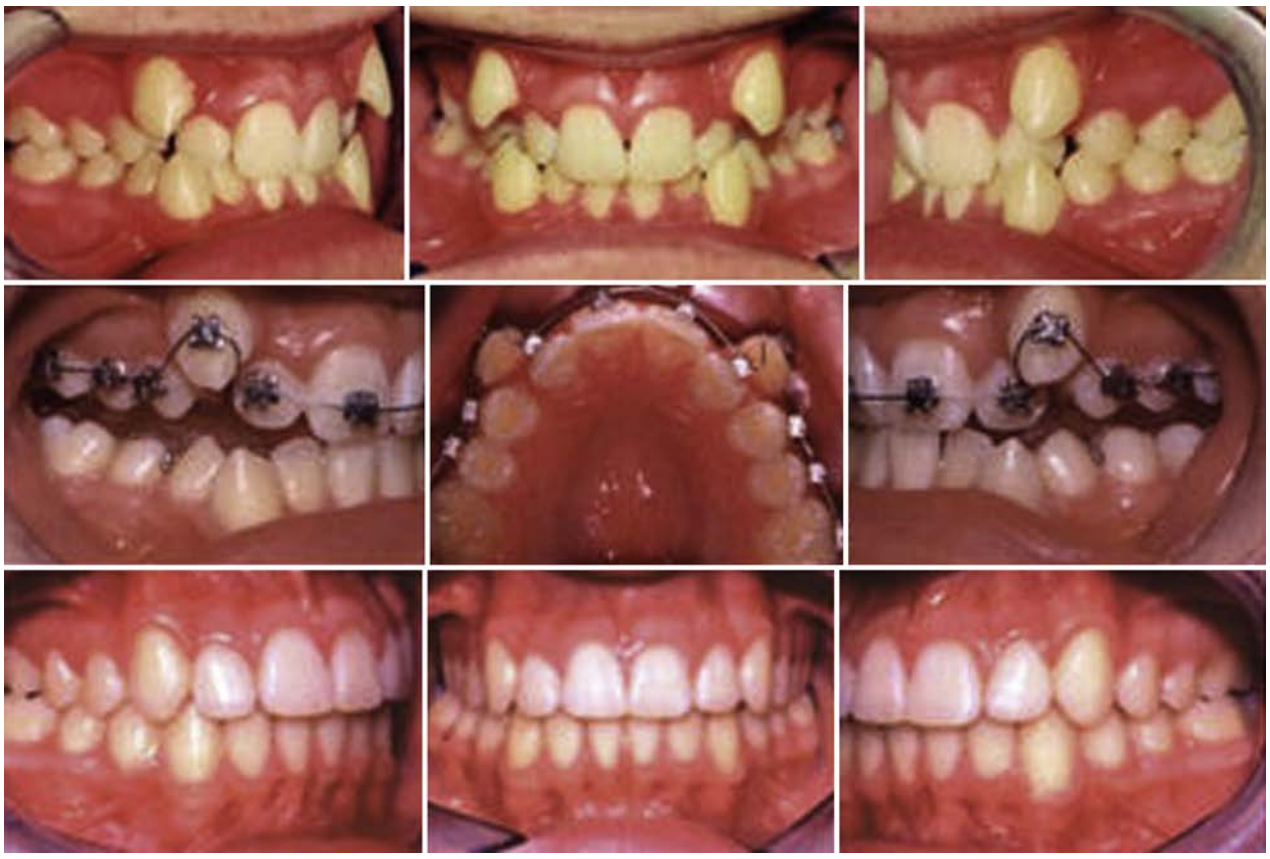


FIGURE 24-18 Class 2, Division 2 malocclusion treated with Damon 2 passive self-ligating brackets and without extractions. The buccally placed canines and palatally inclined premolars and molars at the start of treatment are evident. (By kind permission of Dr. Damon.)

This pattern of tooth movement has been attributed by some clinicians to a qualitatively different interaction of forces. In particular, it is suggested that the applied forces are so low that the lips can compete with orthodontic forces and restrain labial movement of the teeth, sometimes referred to as "the lip bumper effect." Additionally, it has been proposed that the tongue position may alter in response to this expansion and possibly assist in the tooth movement or in its subsequent stability. The author of this chapter suggests that the expansion in this case example may well be entirely explicable by the balance of forces between the teeth and particularly the lateral forces arising from the buccally placed canines and acting on palatally inclined premolars, but the ratio of proclination to expansion still remains surprisingly low in the light of experience with conventional ligation. The relationship between expansion and proclination is discussed further later, but first it is helpful to look at two consecutive visits in a case reproduced by kind permission of Dr. David Birnie (Fig. 24-20).

It can be seen in Figure 24-20 that the incisors have extravagantly (although temporarily in this instance) proclined. A major factor must surely be the high RS produced by the figure-of-8 elastomeric ligatures on the premolars that has prevented the excess wire sliding distally as the canines moved buccally. The extensive evidence reviewed above concerning friction and self-ligation indicates that self-ligation does not necessarily result in lower forces but can generate a higher percentage of desirable force and a lower percentage of unwanted forces, and this difference may potentially alter the resulting tooth positions. In the case in Figure 24-20, the unwanted force is the high and persistent proclining force on the incisors that has arisen because of the friction from the elastomeric premolar ligatures. This is in marked contrast to the behavior of the upper incisors in Figure 24-15, where full engagement of a markedly displaced canine has resulted in good alignment with no detectable incisor proclination. Similarly, Figure 24-21 shows alignment in a Class 3 case who subsequently at an older age underwent the planned orthognathic surgery.

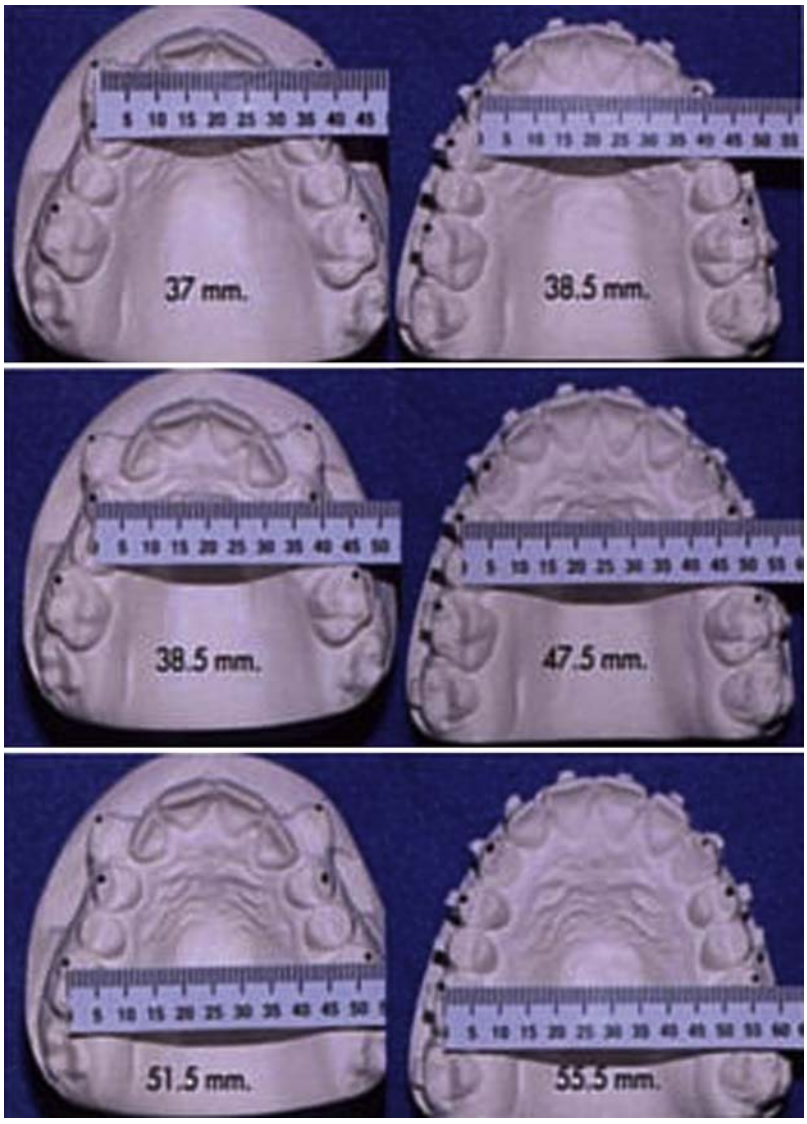


FIGURE 24-19 Arch width changes during treatment of the case in Figure 24-18. There is minimal expansion of the canines but substantial uprighting and expansion of the premolars and molars. This should be distinguished from expansion of posterior teeth, which are already at an upright buccopalatal inclination.

FIGURE 24-20 Proclination of upper incisors between consecutive appointments associated with figure-of-8 elastomeric ligation on all teeth except the palatal canines. 0.014-inch archwire.

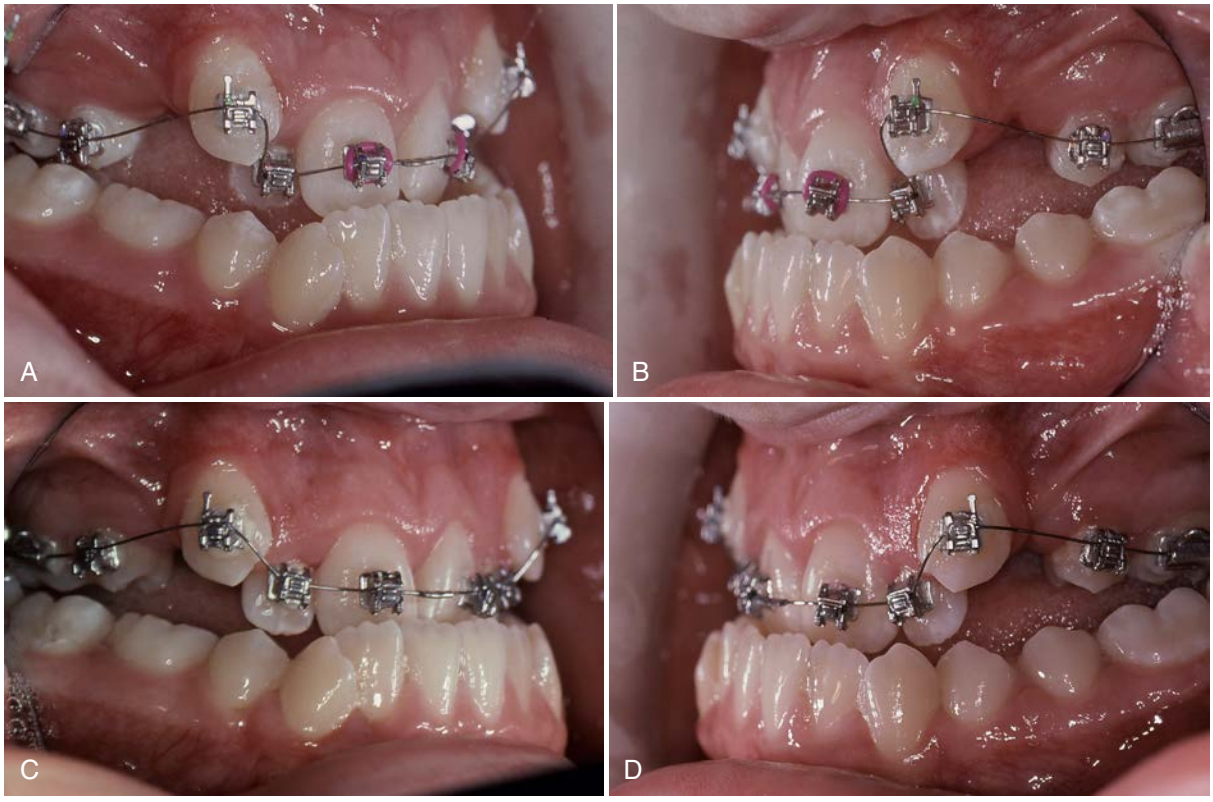


FIGURE 24-21 Three consecutive visits at 8-week intervals. First visit (**A**, **B**) Damon 2 passive brackets with initial 0.012-inch wires were left unchanged at the second visit (**C**, **D**) and replaced by 0.014- × 0.025-inch wires at the third visit (**E**, **F**). The lack of significant central incisor intrusion or proclination is evident. The elastomeric ligatures on some teeth were for decoration at the patient's request and were deliberately placed on teeth anterior to the crowding where no sliding of the archwire relative to the bracket was required. (The author no longer receives requests for this decoration!)

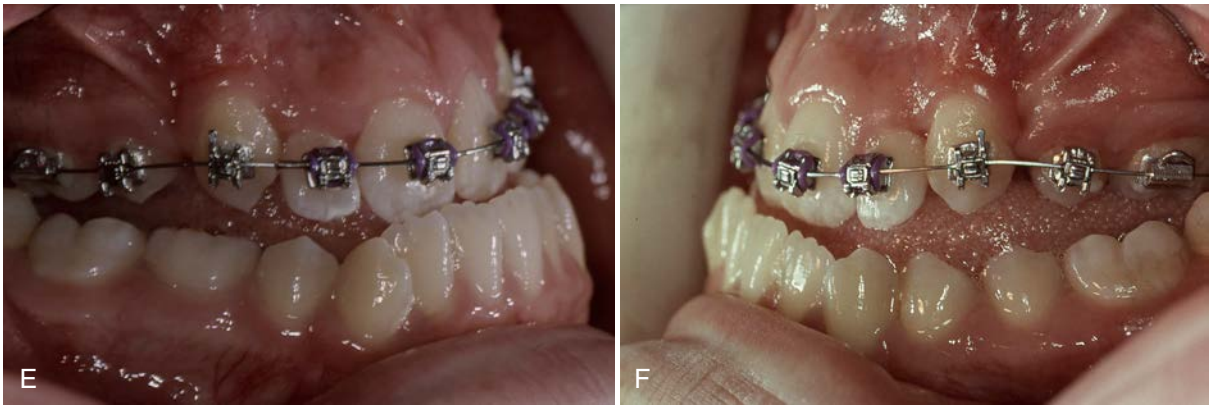


FIGURE 24-21, cont'd

The intention in aligning the upper arch was to do so without significantly proclining the upper incisors and hence compensating for the Class 3 skeletal pattern. The untreated lower incisors act as a reference to demonstrate that there has indeed been very little upper incisor proclination. The initial proclining forces on the incisors must have been very significant and indeed all the higher for the full engagement that self-ligation has enabled and maintained on the lateral incisors. It is suggested that the ability of the wire to release from the binding in the canine and the premolar as soon as tooth movement begins has enabled the alignment to occur with the wire sliding distally rather than by incisor proclination. The hypothesis of a qualitative difference in the pattern of tooth movement would seem to have enough credibility to justify further investigation, although the proposal that the differences arise from interaction with the soft tissues seems less likely and has very little research support.

FORCE LEVELS WITH SELF-LIGATION AND CONVENTIONAL LIGATION

Self-ligation has been described as a "low-force technique." The potential for measurement and comparison of these forces and moments has been potentially advanced to a very substantial degree by the development such systems as used by Heo and Baek and Fok et al., which can model a large range of situations mentioned earlier in the section discussing friction. What clearly emerges from these investigations is that the use of self-ligation does not inherently reduce forces. Indeed, the forces placed on displaced teeth (with any given wire) are higher than would be the case with conventional ligation because less force is dissipated through increased RS on the adjacent teeth. Rather, these studies show that that the distribution of forces is favorably changed and that lighter forces will (if the clinician applies them) be more effective because less force is dissipated. For example, the higher percentage of desirable force that can result from self-ligation is neatly and simply shown in the excellent study by Baccetti et al.[34] The study shows that with conventional ligation, a tooth displaced 3 mm vertically from the line of the arch with a 0.012-inch wire has only 50 *g* of net aligning force available compared with more than 90 *g* with the several types of self-ligating brackets that were tested. With 4.5 mm of displacement, there is zero remaining force available for alignment of the displaced tooth with conventional brackets, but more than 80 *g* remains with self-ligating brackets despite the binding at each corner of the brackets.

DOES SELF-LIGATION REDUCE THE NEED FOR EXTRACTIONS?

In many minds, self-ligation has become associated with an emphasis on nonextraction. A scrutiny of this claim benefits from some dissection of the indications for extraction, which can be categorized as follows:

- Space for alignment of crowded teeth
- Profile improvement
- Interarch relationship
- Correction of overjet
- Correction of overbite

Crowding

The combination of low friction and good archwire control that self-ligation provides would theoretically be expected to facilitate the alignment of crowded arches without extractions, if that is desired. This biomechanical reason for extraction should therefore be correspondingly reduced. It should, however, be recalled that despite the strong evidence for lower friction and good control, studies to date have not confirmed a generic increase in speed of alignment. This may be because the cases in such studies have not a sample with substantial crowding treated nonextraction. The case study of patient AG treated by Dr. David Birnie demonstrates that severe crowding can be accommodated without extraction if that is the desired goal in a specific instance.

CASE STUDY 24-1 A SEVERELY CROWDED LOWER ARCH TREATED WITHOUT EXTRACTION

Patient AG presented at age 14 years, 0 months with moderate upper arch crowding and severe lower arch crowding. His facial appearance was prepubescent; he did not seem to have entered his pubertal growth spurt, and it was thought that significant nasal tip and chin growth would occur during his teenage years. He had a left unilateral crossbite. The lower right central incisor had a long clinical crown (Fig. 24-22). When looking at the severe lower arch crowding, it should be noted that he still retained lower deciduous second molars and that the first premolars were in a Class 1 relationship. All permanent teeth were present on the dental pantomogram except for the upper third molars. His oral hygiene was good but still capable of improvement. Treatment was initiated with light 0.014-inch wires and lightly activated coil spring with the most ectopic instanding teeth being lightly engaged from an early stage to act as a restraint on overall incisor proclination.

Figure 24-23 shows treatment progress after 7 months of treatment. Alignment has almost been achieved in the upper arch, but there is still insufficient space for the lower left lateral incisor. The lower deciduous molars are still present. No crossbite correction has yet taken place; note that the right second molars, which are just visible, are not in crossbite. Figure 24-24 shows the situation 15 months into treatment. Alignment within the arches has been achieved, but crossbite correction has not yet taken place. Figure 24-25 was taken 25 months into treatment. The second molars were bonded 16 months into treatment, and crossbite correction then occurred spontaneously without the need for auxiliary appliances or cross elastics. After stainless steel archwires were placed, the upper archwire was expanded and the lower contracted to ensure maximal correction. Figure 24-26 shows the posttreatment cephalometric radiograph and the superimpositions. There has been no significant change in the skeletal pattern. The lower incisors have proclined by 10 degrees with approximately 3 mm of labial movement, and the upper incisors are at their pretreatment position and inclination. Despite the lack of upper incisor retraction, the nasolabial angle has become more obtuse, and the upper lip is substantially farther from the E line, reflecting the substantial nasal growth. The arch widths show increases in varying amounts. The 10-mm increase in the lower intercanine width reflects the inevitable increase if all such crowded incisors are to be accommodated. The increase in the lower intercanine width would have had to be substantial even had premolars been extracted and the canines moved distally as well as buccally. The upper arch expansion additionally reflects the correction of the posterior crossbites. Figure 24-27 was taken 3 years after the end of active treatment. The clinical crown heights of the lower incisors have equalized and continue to show no recession. In addition to the fixed retainers, the patient wears vacuum-formed retainers at night. Despite the significant arch expansion, there is no undesirable buccal inclination of the upper incisor crowns.

FIGURE 24-22 A–H, Pretreatment records of AG, age 14 years but still prepubertal.

Continued

CASE STUDY 24-1 A SEVERELY CROWDED LOWER ARCH TREATED WITHOUT EXTRACTION—cont'd

This case illustrates the ability of passive self-ligation with light wires and gentle activation to accommodate extremely crowded teeth without extractions and perhaps with less incisor proclination that might have been anticipated. The periodontium appears to be remaining healthy. With regard to stability, almost all clinicians would place a fixed lower lingual retainer in this situation however the teeth had been aligned. A clinician with different treatment planning priorities may equally have chosen to accommodate the crowded teeth and correct the overjet by means of extractions. This would have had different consequences for the width and prominence of the dentition within the adult face. The proposal is that without the biomechanical properties of self-ligating brackets, a nonextraction treatment plan would have been much less practicable and therefore not an available option.

Profile

If research confirms that self-ligation can align teeth with less incisor proclination and relatively more lateral expansion, then this would reduce the need for extractions to prevent or correct a profile that is too full. At present, this remains an interesting and plausible hypothesis.

FIGURE 24-23 A–E, Progress records after 7 months of treatment with light aligning wires.

CASE STUDY 24-1 A SEVERELY CROWDED LOWER ARCH TREATED WITHOUT EXTRACTION—cont'd

FIGURE 24-24 A–E, Progress records for AG after 15 months. Alignment almost achieved, but torque and crossbite corrections are still required.

Continued

CASE STUDY 24-1 A SEVERELY CROWDED LOWER ARCH TREATED WITHOUT EXTRACTION—cont'd

FIGURE 24-25 A–E, AG after 25 months of treatment.

CASE STUDY 24-1 A SEVERELY CROWDED LOWER ARCH TREATED WITHOUT EXTRACTION—cont'd

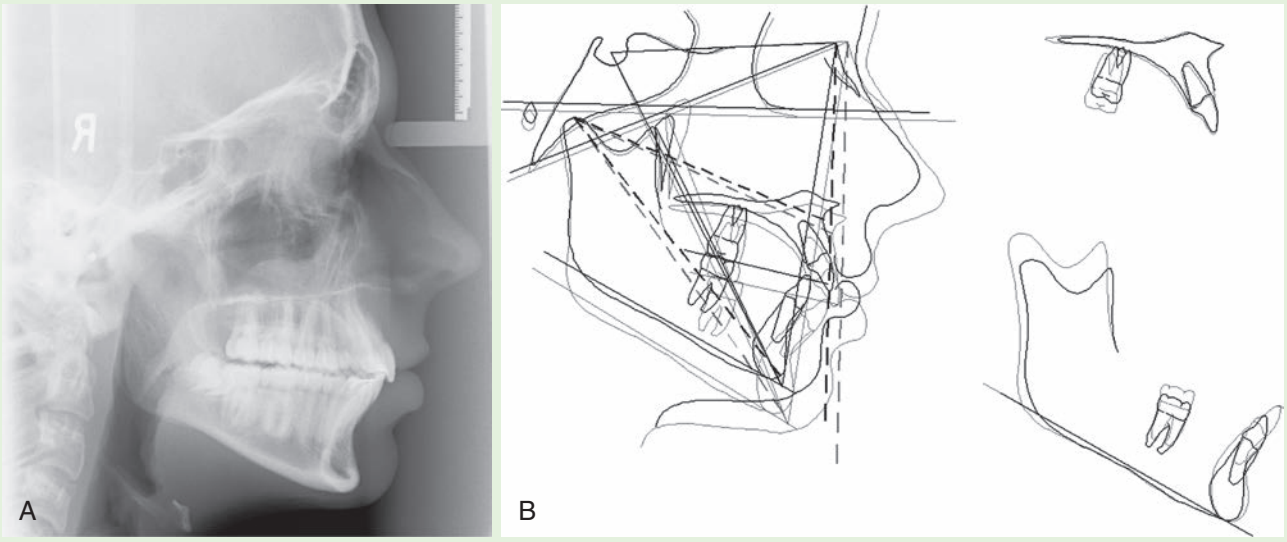


FIGURE 24-26 A, B, Posttreatment cephalometric radiograph and cephalometric superimpositions.

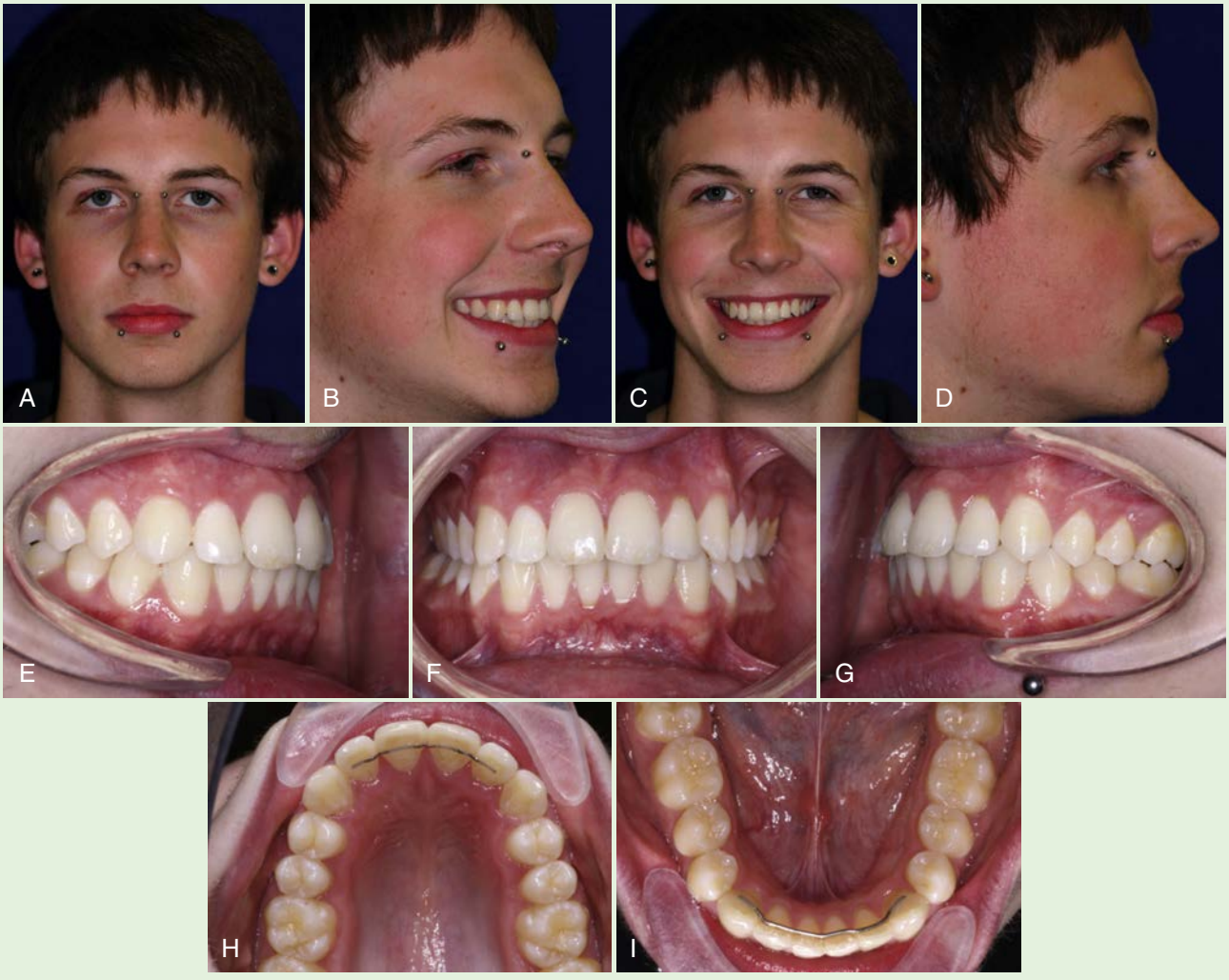


FIGURE 24-27 A–I, Records 3 years after the end of active treatment.

CASE STUDY 24-2 INTERARCH RELATIONSHIP

Self-ligation in itself does not, in the opinion of this author, reduce the potential role for extractions to reduce an overjet. It may, however, help prevent the creation of an unwanted overjet as illustrated through the cases in Figures 24-20 and 24-21. With regard to establishing a positive overbite, it is equally possible that a reduction in incisor proclination for a given amount of tooth alignment may reduce the need for extractions to maintain a positive overbite, but there will always be cases in which any such effect will be insufficient to eliminate the need for extractions. Such a case is illustrated in Case MG (Figs. 24-28 to 24-32) in which self-ligation probably facilitated the alignment of very crowded teeth but in which extractions were still included in order to help achieve the incisor relationship.

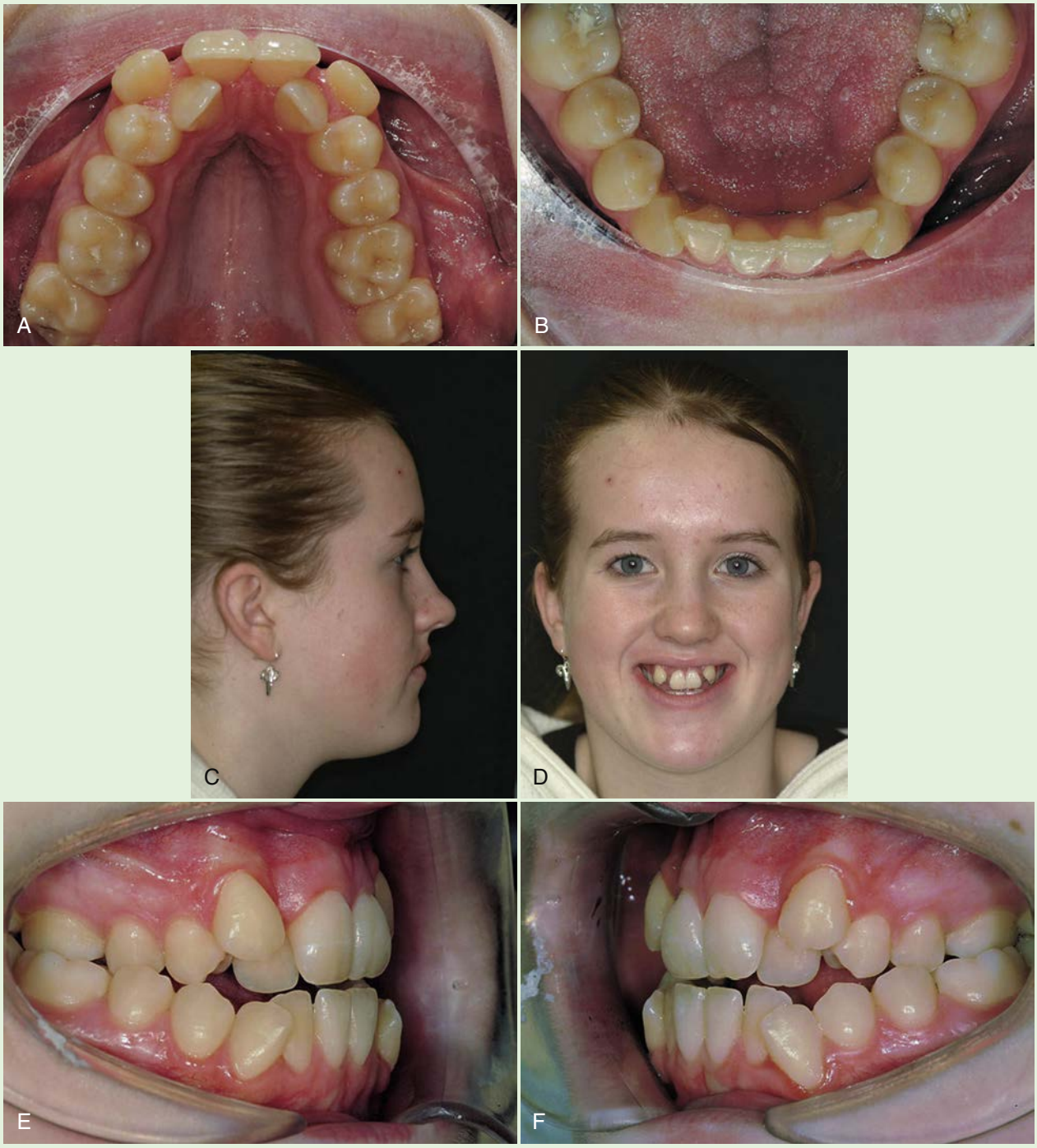


FIGURE 24-28 A–F, Pretreatment records of case MG. The crowding could have been resolved nonextraction (see case AG), but the Class 3 incisor relationship and in particular the lack of overbite strongly influenced the decision to extract.

CASE STUDY 24-2 INTERARCH RELATIONSHIP—cont'd

FIGURE 24-29 A–E, Case MG with first archwires. Upper 0.013-inch and lower 0.014-inch wires. Damon 2 brackets.

Continued

CASE STUDY 24-2 INTERARCH RELATIONSHIP—cont'd

FIGURE 24-30 A to D show sequential visits, and E shows the situation after two further visits. The inclusion of the palatally placed upper lateral incisors from the start has restrained the upper incisor proclination more than the lower despite the greater crowding in the upper arch.

CASE STUDY 24-2 INTERARCH RELATIONSHIP—cont'd

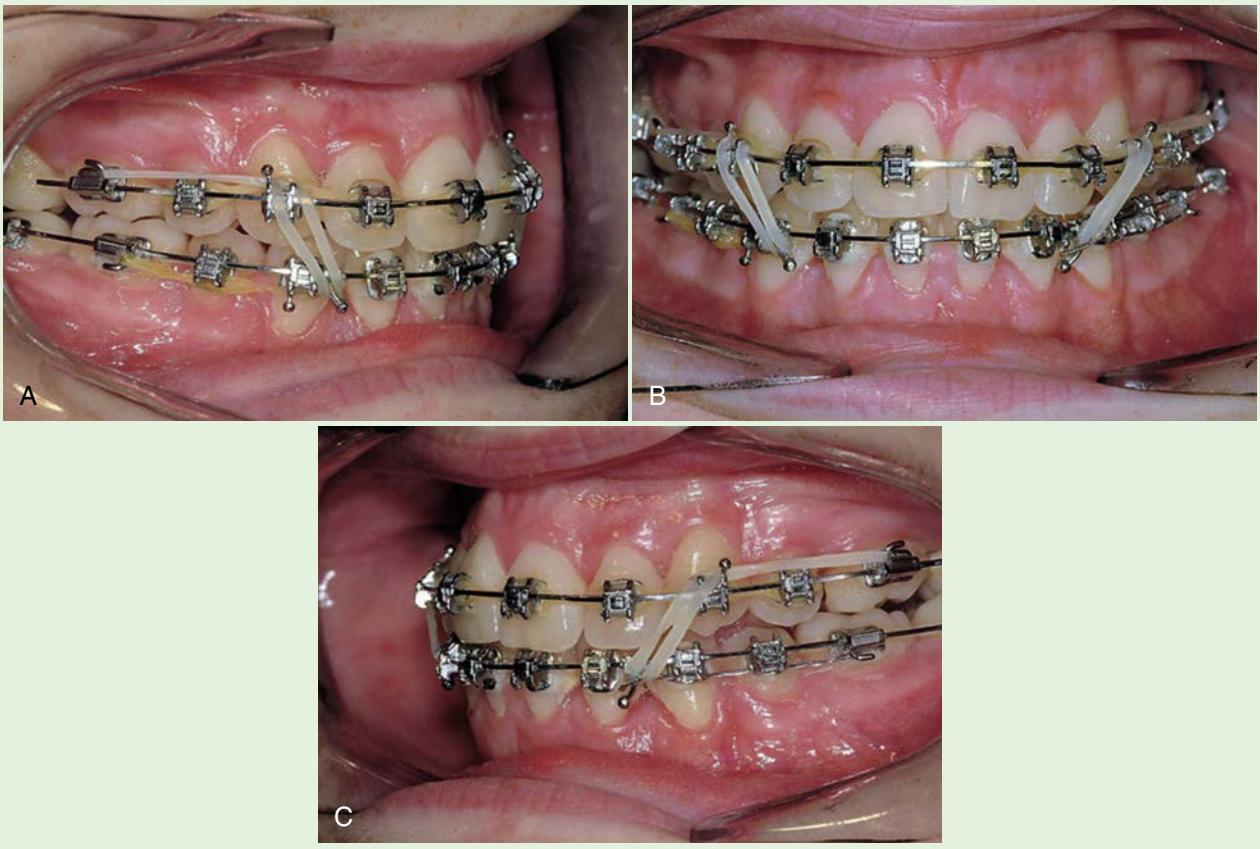


FIGURE 24-31 A–C, 0.019- × 0.025-inch beta titanium wires to detail tooth positions, especially to place buccal root torque on the upper lateral incisors. The selective use of low torque brackets on those two teeth would have reduced or eliminated the need for that torque correction.

Continued

CASE STUDY 24-2 INTERARCH RELATIONSHIP—cont'd

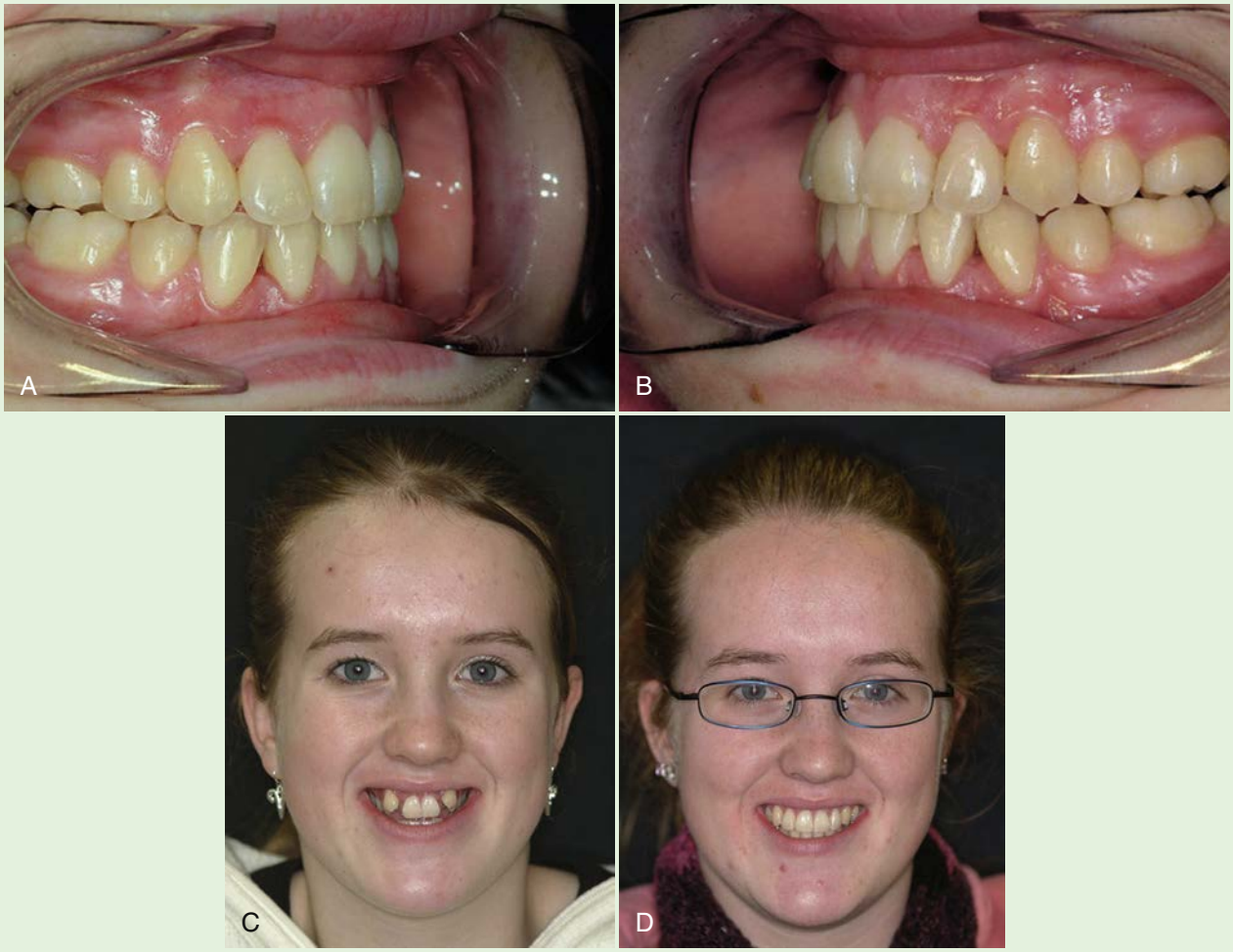


FIGURE 24-32 A, B, The occlusal relations at the end of active treatment. C, D, The change in smile aesthetics.

PRACTICAL CONSEQUENCES FOR CLINICAL TECHNIQUE

Choice of Self-Ligating Bracket

A bracket type should be chosen that most closely meets the list of desirable properties for an ideal self-ligating bracket, listed earlier in the section on factors that may have hindered the adoption of self-ligation. Although a large number of designs are available, some come much closer to fully meeting this list than others. Particular value should be placed on a bracket that is very easy and comfortable to open and close and yet can reliably engage and retain archwires of the full range of diameter and stiffness. A good view of the degree of engagement of the wire in the slot before and during attempted slide or clip closure is a big advantage in preventing excessive and ineffective forces being placed on the teeth during attempted archwire insertion. One significant and recommended feature is the presence of an auxiliary slot permitting elastics to be placed directly to a bracket even with a light aligning wire. This aspect of biomechanics is discussed below.

Practical Tips in Light of the Evidence

In view of the currently established properties of self-ligation as evidenced by the investigations discussed earlier, it seems logical to recommend the following differences in clinical technique in comparison with conventional ligation.

- Because archwire changes are faster and easier,[19–21] take advantage of this to more routinely remove, check, and replace archwires. This can be useful in checking the remaining activation and any deformation of aligning wires and can facilitate oral hygiene instruction during the appointment.
- In view of the much lower friction and hence RS,[27,38] be rigorous in placing stops or hooks on the archwire to prevent distal wire pokes. Archwires with preloaded crimpable stops or presoldered hooks are the most efficient method, but stops can be purchased separately. An upper arch will require two such stops because of the larger anterior interbracket spans and the need to place stops anterior to any crowding to permit distal movement of an aligning wire unhindered by the stop(s).

- Because the low friction means higher net forces on displaced teeth[34] aligning wires must be of smaller diameter or higher transformation temperature to ensure sufficiently low aligning force. In a 0.022-inch slot, initial wires should rarely be larger than 0.014 inch and frequently smaller than that. An important second advantage of thin initial wires is that the greater space between archwire and bracket facilitates the relative movement between bracket and archwire and hence between adjacent teeth that is necessary for teeth to align, especially if space is not made available by extraction.
- In view of the research into force levels and distribution (e.g., Baccetti[34] and Fok[37]), it is sensible and advantageous to engage more displaced teeth at the start of treatment rather than partial and progressive engagement as has traditionally been advocated. As with all such features, this change can be extrapolated too far. Even self-ligating brackets can behave like conventional ligation if the wire deflections to engage the displaced tooth are too severe.
- When planning extractions, the easier alignment of irregular teeth with self-ligation without extraction spaces should be one of the factors in the decision. This may appropriately lead to more therapeutic diagnosis in relation to extractions. If after arch alignment, extractions are judged to be necessary, little time will have been lost because of the properties of self-ligation during alignment. If extractions prove not to be necessary, then this irreversible step will have been avoided.
- After teeth are aligned, there is then no advantage in scheduling appointments less frequently than with conventional ligation. This only prolongs treatment.
- Especially with passive ligation, it is important that intermediate aligning archwires have a buccolingual dimension that is as large as the subsequent stainless steel archwire. The full and secure archwire engagement has the corollary that incomplete archwire engagement cannot inadvertently occur and persist as it can with conventional ligation. So a 0.014- × 0.025-inch wire is significantly preferable to a 0.016- × 0.022-inch as an intermediate wire, ensuring easier progression to full engagement of a stiffer wire with a 0.025-inch buccolingual dimension.
- "Begg mechanics with straight-wire control": In view of the secure archwire engagement, it is possible to be much less wary of applying light traction directly to a tooth rather than to a hook on the archwire. Such a tooth will experience very little loss of rotational control. Light (2-oz) elastics can confidently be placed on 0.014-inch archwires. The greater effectiveness of light forces will increase the chances of a favorable tooth movement while the lighter applied force, the better force distribution, and the guaranteed secure archwire engagement reduce the traditionally anticipated adverse side effects of such biomechanics. This represents a change from a very sequential form of biomechanics in which all the hard work awaits the arrival of "working archwires" to a more simultaneous approach, which is analogous to that previously used with Begg brackets. Begg brackets were another low-friction bracket that greatly enhanced the effectiveness of light forces but with much less tooth control. Figures 24-33 and 24-34 show this approach in action.
- Disclusion via anterior bite ramps or posterior additions of glass ionomer are potentially more beneficial with self-ligation because lighter forces are being expected to achieve tooth movement, and the scope for occlusal interference is correspondingly greater.
- Anticipate greater benefit from the combination of low friction and good control in cases in which individual teeth require sliding along the archwire. This is a particular feature of the treatment of hypodontia cases.

SUMMARY

Self-ligating brackets are a highly active area of technical development, thought-provoking clinical practice, and research. They have been commercially available for almost 30 years but only in the past decade has their use spread beyond a minority of enthusiasts. This proliferation of types of self-ligating brackets and the large increase in their clinical use have been sparked partly by very significant advances in bracket design and manufacture. Brackets are now available that are very reliable and very easy to use. Of equal importance has been a rapid growth in the evidence for and appreciation of the combined advantages of secure full ligation, lower RS, and speed of archwire ligation and removal. The shortcomings of the previously dominant elastomeric ligation have come to be generally appreciated, and orthodontists who are understandably more skeptical about some of the claims made for self-ligation are nevertheless using these brackets and enjoying the speed of archwire changes, the cleanliness of appliances that lack elastomerics, the effectiveness of light archwires with low friction, and the security of ligation and resulting tooth control. However, beyond these core advantages, much remains to be securely supported by research. In particular, the evidence to date has failed to support the hypothesis of faster treatment, with only reduced chairside time being a demonstrated efficiency. Equally, hypotheses that torquing or finishing performance is deficient with self-ligation also lack supporting evidence.

The question of a greatly reduced need for extractions is of real interest and is stimulated by some impressive case presentations but currently lacks hard data. First principles concerning low friction and good archwire control support the clinical impression that the need for extractions purely to provide space for arch alignment may therefore be reduced. An encouraging trend is the recent development and use of much more sophisticated force measurement systems and more realistic simulations of in vivo effects. An important conclusion from such research is the concept that self-ligation does not automatically reduce forces applied to teeth but does enable much lighter applied forces to be effective at moving teeth by substantially altering the ratio between desirably light tooth-moving forces and the unwanted reciprocal forces. At the least, self-ligation is now established as a neat, rapid, and very reliable form of ligation. It does not of necessity change a clinician's treatment goals or mechanics, but it can fairly be said to facilitate a wider choice in these respects. The next few years will resolve many of the questions discussed in this chapter, but it is beyond doubt that the questions are stimulating and of very significant consequence for the care of our patients.

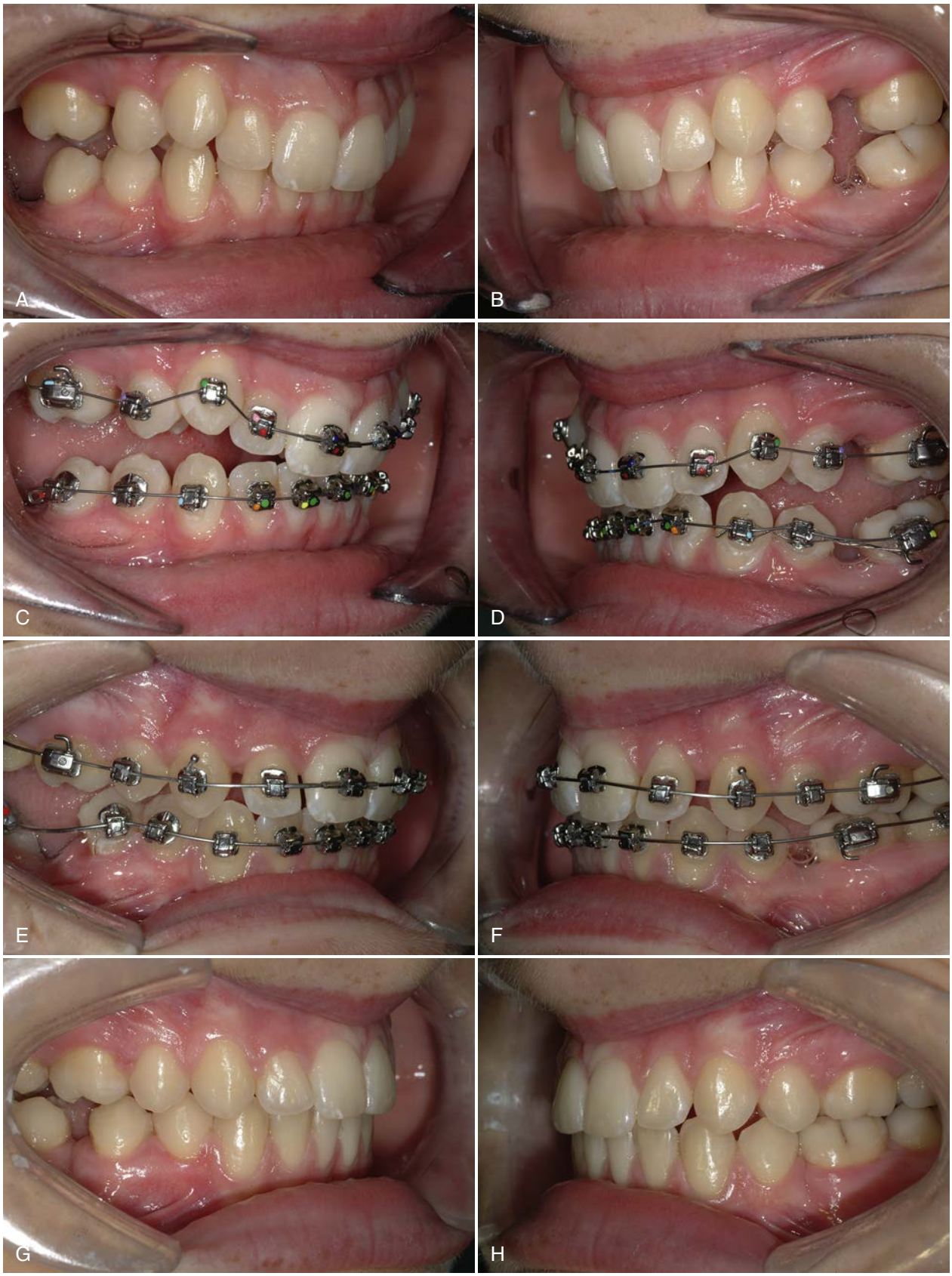


FIGURE 24-33 **A, B,** A case with congenital absence of teeth no. 15, 25, and 35 and extraction of ankylosed no. 46. Early 50 *g* (TP green) Class II elastics were applied to 0.014-inch and then 0.014- × 0.025-inch archwires. **C, D,** Day of bond-up and placement of anterior bite ramps. Class II elastics were applied to drop-in hooks on upper canines. **E, F,** Visit 3. Class I canines. Overbite corrected. Note the drop-in hooks placed in the vertical bracket slot for application of the elastics. Class II elastic traction was stopped at this visit and the drop-in hooks removed. **G, H,** End of 14 months of active treatment. Some space was left in the lower right quadrant to enable good occlusion with UR7.

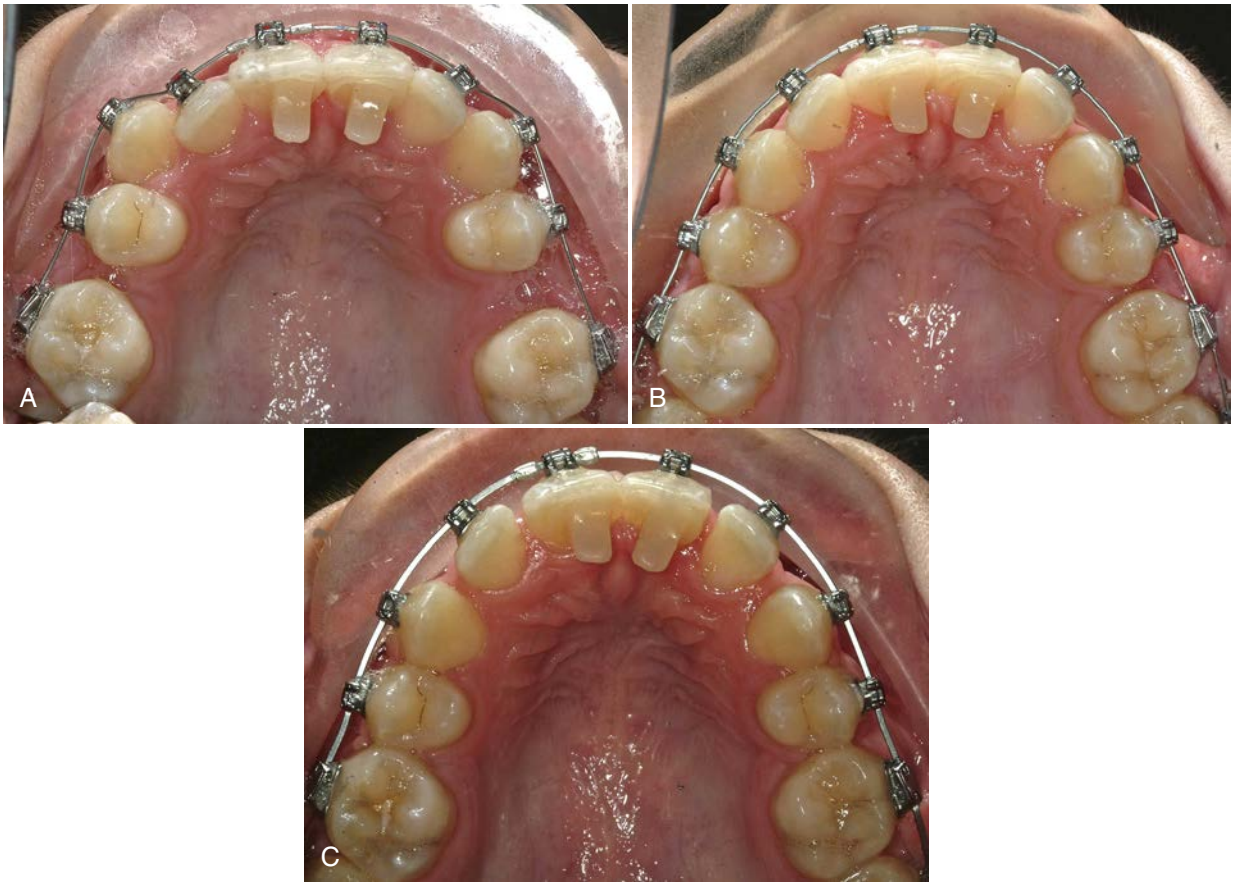


FIGURE 24-34 The same case as Figure 24-33. Traction applied to drop-in hooks on the canines achieves good tooth movement and maintains good rotational control. **A,** Day of bond-up and start of Class II traction on 0.014-inch wires. **B,** Next visit 10 weeks later. 0.014- × 0.025-inch archwire then placed. **C,** Visit 3, which was 7 weeks later. Class II elastics stopped and drop-in hooks removed.

REFERENCES

1. Shivapuja PK, Berger J. A comparative study of conventional ligation and self-ligation bracket systems. *Am J Orthod Dentofac Orthop*. 1994;106:472–480.
2. Khambay B, Millett D, Mc Hugh S. Evaluation of methods of archwire ligation on frictional resistance. *Eur J Orthod*. 2004;26:327–332.
3. Baty DL, Storie DJ, von Fraunhofer JA. Synthetic elastomeric chains: a literature review. *Am J Orthod Dentofac Orthop*. 1994;105:536–542.
4. Eliades T, Bourauel C. Intraoral aging of orthodontic materials: the picture we miss and its clinical relevance. *Am J Orthod Dentofac Orthop*. 2005;127:403–412.
5. Taloumis LJ, Smith TM, Hondrum SO, Lorton L. Force decay and deformation of orthodontic elastomeric ligatures. *Am J Orthod Dentofac Orthop*. 1997;111:1–11.
6. Lam TV, Freer TJ, Brockhurst PJ, Podlich HM. Strength decay of elastomeric ligatures. *J Orthod*. 2002;29:37–42.
7. Thurow RC. Letter: elastic ligatures, binding forces, and anchorage taxation. *Am J Orthod*. 1975;67:694.
8. Begg PR, Kesling PC. *Begg Orthodontic Theory and Technique*. 3rd ed. Philadelphia: WB Saunders; 1977.
9. Stolzenberg J. The Russell attachment and its improved advantages. *Int J Orthod and Dent Children*. 1935;21:837–840.
10. Iwasaki LR, Beatty MW, Randall CJ, Nickel JC. Clinical ligation forces and intraoral friction during sliding on a stainless steel archwire. *Am J Orthod Dentofac Orthop*. 2003;123:408–415.
11. Harradine NWT. Self-ligating brackets: where are we now? *J Orthod*. 2003;30:262–273.
12. Wildman AJ. Round table—the Edgelok bracket. *J Clin Orthod*. 1972;6:613–623.
13. Hanson GH. The SPEED system: a report on the development of a new edgewise appliance. *Am J Orthod Dentofac Orthop*. 1980;78:243–265.
14. Harradine NWT, Birnie DJ. The clinical use of Activa self-ligating brackets. *Am J Orthod Dentofac Orthop*. 1996;109:319–328.
15. Damon DH. The Damon low friction bracket: a biologically compatible straight-wire system. *J Clin Orthod*. 1998;32:670–680.
16. Damon DH. The rationale, evolution and clinical application of the self-ligating bracket. *Clin Orthod Res*. 1998;1:52–61.
17. Harradine NWT. Self-ligating brackets and treatment efficiency. *Clin Orthod Res*. 2001;4:220–227.
18. Macchi A, Tagliabue A, Levrini L, Trezzi G. Philippe self-ligating lingual brackets. *J Clin Orthod*. 2002;36:42–45.
19. Maijer R, Smith DC. Time saving with self-ligating brackets. *J Clin Orthod*. 1990;24:29–31.
20. Voudouris JC. Interactive edgewise mechanisms: form and function comparison with conventional edgewise brackets. *Am J Orthod Dentofac Orthop*. 1997;111:119–140.
21. Turnbull NR, Birnie DJ. Treatment efficiency of conventional versus self-ligating brackets: the effects of archwire size & material. *Am J Orthod Dentofac Orthop*. 2007;131:395–399.
22. Mezomo M, de Lima ES, Macedo de Menezes L, et al. Maxillary canine retraction with self-ligating and conventional brackets. *Angle Orthod*. 2001;81:292–297.
23. Pandis N, Bourauel C, Eliades T. Changes in the stiffness of the ligating mechanism in retrieved active self-ligating brackets. *Am J Orthod Dentofac Orthop*. 2007;132:834–837.

24. Kapur R, Sinha PK, Nanda RS. Frictional resistance of the Damon SL bracket. *J Clin Orthod*. 1998;32:485–489.
25. Thomas S, Birnie DJ, Sherriff M. A comparative in vitro study of the frictional characteristics of two types of self-ligating brackets and two types of preadjusted edgewise brackets tied with elastomeric ligatures. *Eur J Orthod*. 1998;20:589–596.
26. Pizzoni L, Raunholt G, Melsen B. Frictional forces related to self-ligating brackets. *Eur J Orthod*. 1998;20:283–291.
27. Thorstenson BS, Kusy RP. Resistance to sliding of self-ligating brackets versus conventional stainless steel twin brackets with second-order angulation in the dry and wet (saliva) states. *Am J Orthod Dentofac Orthop*. 2001;120:361–370.
28. Thorstenson BS, Kusy RP. Comparison of resistance to sliding between different self-ligating brackets with second-order angulation in the dry and saliva states. *Am J Orthod Dentofac Orthop*. 2002;121:472–782.
29. Thorstenson BS, Kusy RP. Effect of archwire size and material on the resistance to sliding of self-ligating brackets with second-order angulation in the dry state. *Am J Orthod Dentofac Orthop*. 2002;122:295–305.
30. Thorstenson BS, Kusy RP. Effects of ligation type and method on the resistance to sliding of novel orthodontic brackets with second-order angulation in the wet and dry state. *Angle Orthod*. 2003;73:418–430.
31. Pliska BT, Beyer JP, Larson BE. A comparison of resistance to sliding of self-ligating brackets under an increasing applied moment. *Angle Orthod*. 2011;81:794–799.
32. Matarese G, Nucera R, Militi A, et al. Evaluation of frictional forces during dental alignment: an experimental model with 3 nonleveled brackets. *Am J Orthod Dentofac Orthop*. 2008;133:708–715.
33. Heo W, Baek SH. Friction properties according to vertical and horizontal tooth displacement and bracket type during initial leveling and alignment. *Angle Orthod*. 2011;81:653–661.
34. Baccetti T, Franchi L, Camporesi M, et al. Forces produced by different nonconventional bracket or ligature systems during alignment of apically displaced teeth. *Angle Orthod*. 2009;79:533–539.
35. Franchi L, Bacetti T, Camporesi M, Giuntini Z. Forces released by nonconventional bracket or ligature systems during alignment of buccally displaced teeth. *Am J Orthod Dentofac Orthop*. 2009;136:316.e1–e6.
36. Peterson A, Rosenstein S, Kim KB, Israel H. Force decay of elastomerics ligatures: influence on unloading force compared to self-ligation. *Angle Orthod*. 2009;79:934–938.
37. Fok J, Toogood RW, Badawi H, et al. Analysis of maxillary arch force/couple systems for a simulated high canine malocclusion: part 1. Passive ligation. *Angle Orthod*. 2001;81:953–959.
38. Ehsani S, Mandich MA, El-Bialy TH, Flores-Mir C. Frictional resistance in self-ligating orthodontic brackets and conventionally ligated brackets. *Angle Orthod*. 2009;79:592–601.
39. Braun S, Bluestein M, Moore BK, Benson G. Friction in perspective. *Am J Orthod Dentofac Orthop*. 1999;115:619–627.
40. O'Reilly D, Dowling PA, Lagerstrom L, Swartz ML. An ex-vivo investigation into the effect of bracket displacement on resistance to sliding. *Br J Orthod*. 1999;26:219–227.
41. Iwasaki LR, Beatty MW, Randall CJ, Nickel JC. Clinical ligation forces and intraoral friction during sliding on a stainless steel archwire. *Am J Orthod Dentofac Orthop*. 2003;123:408–415.
42. Matasa CG. Brackets' shape influences friction. *Orthod Materials Insider*. 2001;13:2–5.
43. Koenig HA, Burstone CJ. Force systems from an ideal arch—large deflection considerations. *Angle Orthod*. 1989;59:11–16.
44. Rinchuse DJ, Miles PG. Self-ligating brackets: present and future (2007). *Am J Orthod Dentofac Orthop*. 2007;132:216–222.
45. Major TW, Carey JP, Nobes DS, et al. Measurement of plastic and elastic deformation due to third-order torque in self-ligated orthodontic brackets. *Am J Orthod Dentofac Orthop*. 2001;140:326–339.
46. Pandis N, Bourael C, Eliades T. Changes in the stiffness of the ligating mechanism in retrieved active self-ligating brackets. *Am J Orthod Dentofac Orthop*. 2007;132:834–837.
47. Eberting JJ, Straja SR, Tuncay OC. Treatment time, outcome and patient satisfaction comparisons of Damon and conventional brackets. *Clin Orthod and Res*. 2001;4:228–234.
48. Ollivere P. Treatment efficiency of self-ligating brackets. *Orthod Update*. 2012;5:15–19.
49. Miles PG. SmartClip versus conventional twin brackets for initial alignment: is there a difference? *Aust Orthod J*. 2005;21:123–127.
50. Miles PG, Weyant RJ, Rustveld L. A clinical trial of Damon 2 vs. conventional twin brackets during initial alignment. *Angle Orthod*. 2006;76:480–485.
51. Fleming PS, DiBiase AT, Sarri G, Lee RT. Efficiency of mandibular arch alignment with 2 preadjusted edgewise appliances. *Am J Orthod Dentofac Orthop*. 2009;135:597–602.
52. Pandis N, Polychronopoulou A, Eliades T. Self-ligation vs. conventional brackets in the treatment of mandibular crowding: a prospective clinical trial of treatment duration and dental effects. *Am J Orthod Dentofac Orthop*. 2007;132:208–215.
53. Scott P, DiBiase AT, Sherriff M, Cobourne MT. Alignment efficiency of Damon3 self-ligating and conventional orthodontic bracket systems: a randomized clinical trial. *Am J Orthod Dentofac Orthop*. 2008;134:470.el–470.e8.
54. Miles PG. Self-ligating versus conventional twin brackets during en-masse space closure with sliding mechanics. *Am J Orthod Dentofac Orthop*. 2007;132:223–225.
55. Fleming PS, DiBiase AT, Lee RT. Randomized clinical trial of orthodontic treatment efficiency with self-ligating and conventional fixed orthodontic appliances. *Am J Orthod Dentofac Orthop*. 2010;137:738–742.
56. Fleming PS, Johal A. Self-ligating brackets in orthodontics—a systematic review. *Angle Orthod*. 2010;80:575–584.
57. Songra G, Clover M, Atack NE, et al. Comparative assessment of alignment efficiency and space closure of active and passive self-ligating vs. conventional appliances in adolescents: a single-center randomized controlled trial. *Am J Orthod Dentofac Orthop*. 2014;145:569–578.
58. Harradine NWT. Point: self-ligating brackets increase treatment efficiency. *Am J Orthod Dentofac Orthop*. 2013;143:10.
59. Fleming PS, O'Brien K. Counterpoint: self-ligating brackets do not increase treatment efficiency. *Am J Orthod Dentofac Orthop*. 2013;143:11.
60. Scott P, Sherriff M, Di Biase AT, Cobourne MT. Perception of discomfort during initial orthodontic tooth alignment using a self-ligating or conventional bracket system: a randomized clinical trial. *Eur J Orthod*. 2009;30:227–232.
61. Pringle AM, Petrie A, Cunningham SJ, McKnight M. Prospective randomized clinical trial to compare pain levels associated with 2 orthodontic fixed bracket systems. *Am J Orthod Dentofac Orthop*. 2009;136:160–167.
62. Čelar A, Schedlberger M, Dörfler P, Bertl M. Systematic review on self-ligating vs. conventional brackets: initial pain, number of visits, treatment time. *J Orofac Orthop*. 2013;74:40–51.
63. Yamaguchi M. The Damon system and release of substance P in gingival crevicular fluid during orthodontic tooth movement in adults. *World J Orthod*. 2009;10:141–146.
64. Major TW, Carey JP, Nobes DS, et al. Measurement of plastic and elastic deformation due to third-order torque in self-ligated orthodontic brackets. *Am J Orthod Dentofac Orthop*. 2001;140:326–339.
65. Brauchli, Senn C, Wichelhaus A. Active and passive self-ligation—a myth? Part 1 torque control. *Angle Orthod*. 2012;82:663–669.
66. Sifakakis I, Pandis N, Makou M, et al. Torque efficiency of different archwires in 0.018- and 0.022-inch conventional brackets. *Angle Orthod*. 2014;84:149–154.
67. Al Fakir H, Carey JP, Melenka GW, et al. Investigation into the effects of stainless steel ligature ties on the mechanical characteristics of conventional and self-ligated brackets subjected to torque. *J Orthod*. 2014;41(3):188–200.
68. Damon DH. Treatment of the face with biocompatible orthodontics. In: Graber LW, Vanarsdall RL, Vig KWL, eds. *Orthodontics: Current Principles and Techniques*. 4th ed. St. Louis: Elsevier; 2005.

25

Lingual Appliance Treatment

Dirk Wiechmann and Dan Grauer

OUTLINE

Introduction, 761
Customized Treatment Goals and Lingual Appliances, 761
Use of a Target Setup Model System, 761
Accurate Realization of Individual Treatment Goals, 761
Reduced Risk of Decalcification, 762
Main Differences Between Labial and Lingual Techniques, 762
Anatomic Variations of the Lingual Tooth Surfaces, 762
First-Order Compensations, 763
Torque Control, 764
Exposure of Bonding Area, 764
Innovation in Completely Customized Lingual Appliances, 764
Efficient Leveling and Aligning with Lingual Appliances, 764
Aligning Type 1, 765
Aligning Type 2, 765
Aligning Type 3, 765
Summary, 765

INTRODUCTION

Appearance during orthodontic treatment with labial appliances has been related to social discomfort and lack of confidence in public.[1] Despite numerous efforts by experienced clinicians and continuous improvements, a lingual technique using conventional appliances has not been fully established.[2–9] Several completely customized and semicustomized lingual appliances and appliance systems are currently available; these include Harmony (American Orthodontics, Sheboygan, WI), Incognito (3M-Unitek, Monrovia, CA), Suresmile lingual (Orametrix, Richardson, TX), and WIN (DW Lingual Systems GmbH, Bad Essen, Germany). The Suresmile system differs in that instead of a custom lingual bracket, it uses a standard lingual bracket chosen by the clinician but custom-designed and computer-fabricated archwire(s).

In this chapter, the use of completely customized brackets and archwires is described and illustrated as used in the WIN System. Completely customized lingual appliances (custom-designed and manufactured brackets for each tooth and individually formulated archwires) have allowed clinicians to integrate the lingual technique successfully into their everyday practice.[10–14] These appliances have become the most widely used lingual appliances since their introduction in 2004 (Fig. 25-1). Although lingual appliances are the most aesthetic alternative for correcting malocclusions, other far more important reasons for using completely customized lingual appliances have emerged when compared with alternative treatments.

Customized Treatment Goals and Lingual Appliances

Use of a Target Setup Model System

The highly variable morphology of lingual surfaces of teeth and the need to reduce the profile of the lingual braces, increasing patients' comfort, require full customization of the lingual appliances. For each patient, a treatment goal is defined with the help of a target setup.[11,13] Establishing a target setup has both diagnostic and therapeutic advantages. Diagnostic advantages include the assessment of the desired treatment goals before initiation of treatment, which improves communication with the dental team and the patient;[15] the possibility of evaluating more than one treatment goal in complex cases; and a more accurate estimation of treatment time. The main treatment advantage is the fabrication of accurate appliances based on the target setup; these appliances are designed to move the teeth that need to be moved and remain passively on the teeth that should stay stationary,[16] as opposed to average preadjusted appliances that would produce unwanted tooth movements in some teeth because of anatomic variations. This can be explained by the fact that intraindividual variation in tooth morphology is greater than variation in preadjusted appliances' prescription.[17]

Accurate Realization of Individual Treatment Goals

The test of any appliance system is how accurately the clinician can achieve treatment goals. For each custom lingual appliance patient, a setup is fabricated according to the specific individual treatment plan goals. A customized prescription in six degrees of freedom is applied to each tooth, and the individual dental arch shape is formed. Attainment of treatment goals can be evaluated by comparing the orthodontic outcome with the target setup fabricated at the beginning of treatment. Several studies reported on the accuracy of completely customized lingual appliances. Pauls[18] compared setup and final models for 25 lingual appliance patients. They superimposed models from both time points and compared the position of the bracket in the setup model with the position of the bracket in the final model. The discrepancies in position between brackets were translated into three rotation and three translation parameters. The author concluded that the setup objectives were achieved in the finished cases.

Grauer and Proffit[19] compared setup and final models for 94 consecutive patients. Models were compared based on a surface-to-surface registration. The discrepancies between the

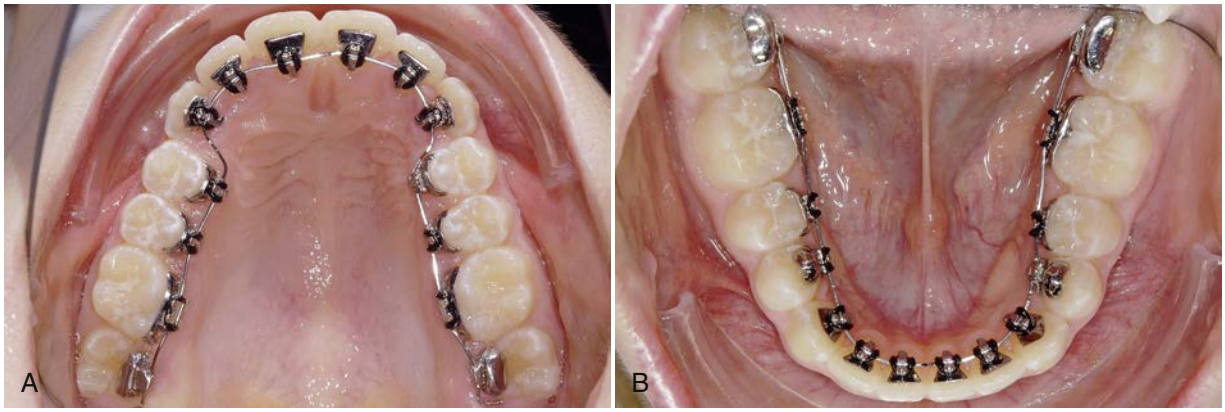


FIGURE 25-1 A, B, Completely customized lingual appliance. Note the occlusal coverage on the second molars and lower second premolars. Superelastic NiTi wires were inserted; these wires were designed and manufactured to achieve the desired individual arch form.

position of the teeth in the setup and outcome model were translated into rotation and translation parameters. Discrepancies in translation (mesiodistal, labiolingual, and vertical) and in rotational parameters (inclination, angulation, and axial rotation) between the setup and outcome were small for all teeth (generally <1 mm and 4 degrees) except for the second molars, where some larger discrepancies were observed. They concluded that fully customized lingual orthodontic appliances were accurate in achieving the goals planned at the initial setup.

A third study compared the desired intercanine distance at the target setup with the attained intercanine distance after treatment. The authors reported a mean deviation of less than 0.5 mm between the initial setup and finished treatment. This result was expected given that after teeth are aligned with no spaces among them, the change in arch form between canines has little influence on the arch perimeter.[20] Wiechmann et al.[21] assessed the inclination of the lower incisors after orthodontic treatment with completely customized lingual appliances combined with the Herbst appliance. There was no statistical difference ($p > 0.05$) between planned incisor inclinations of the target setup and achieved incisor inclinations on the day of debonding. The overall mean difference was 2.2 degrees ± 1.0 degree. It is important to note that when Herbst appliances are used, a wire that completely fills the bracket slot is used; hence, the full prescription of the appliance gets expressed.

Reduced Risk of Decalcification

White spot lesions (WSLs) associated with fixed orthodontic appliances are a common adverse effect of labial fixed orthodontic treatment; these are not distinct types of carious lesions but are the result of enamel demineralization as a stage of the carious process occurring around orthodontic fixed appliances.[22] The prevalence of WSLs occurring during orthodontic treatment is reported to range from 13% to 75%.[23–26] In a randomized clinical trial, it was shown that the incidence of WSLs is five times smaller when customized orthodontic appliances are used as opposed to conventional labial fixed appliances. Moreover, the severity of the lesions occurring during orthodontic treatment was 10 times smaller on the lingual side as opposed to the labial side.[27] In a recent study, the incidence of WSLs during orthodontic treatment with completely customized lingual appliances was determined and compared with the published incidence of WSLs when labial appliances are used. The authors concluded that the incidence of WSLs in maxillary front teeth was six times smaller when customized lingual appliances were used in terms of subjects and 12 times smaller in terms of teeth.[28] Based on this evidence, completely customized lingual appliances are classified as low risk for WSL (see Chapter 30).[22]

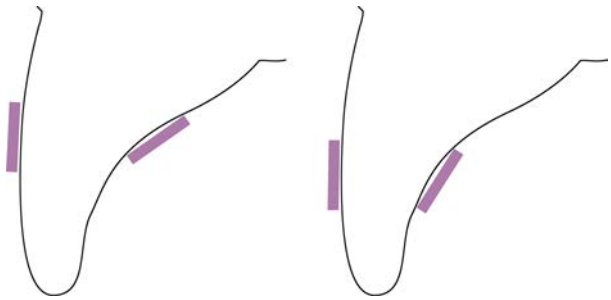


FIGURE 25-2 A slightly different positioning height of the bracket has only a small impact on torque (2 degrees) with a labial appliance. On the lingual aspect, the same height difference results in a major torque difference (22 degrees).

MAIN DIFFERENCES BETWEEN LABIAL AND LINGUAL TECHNIQUES

Anatomic Variations of the Lingual Tooth Surfaces

The labial and lingual differences in tooth morphology are substantial. Morphologic variability of the lingual surfaces of teeth results in a wide range of individual variation compared with the labial surface. For instance, the labial surface of an upper central incisor always follows a similar pattern, but its lingual surface shows marked morphologic variations among individuals. Therefore, designing the treatment appliance based on mean values, as with labial techniques, is impossible. Moreover, even a small height (vertical) deviation in the position of the brackets results in a marked effect on the third-order prescription (Fig. 25-2). Therefore, the application of the intended torque values can only be routinely achieved with an approach combining completely customized bracket bases with indirect bonding protocols. Without an understanding of the different requirements in bracket placement between lingual and labial

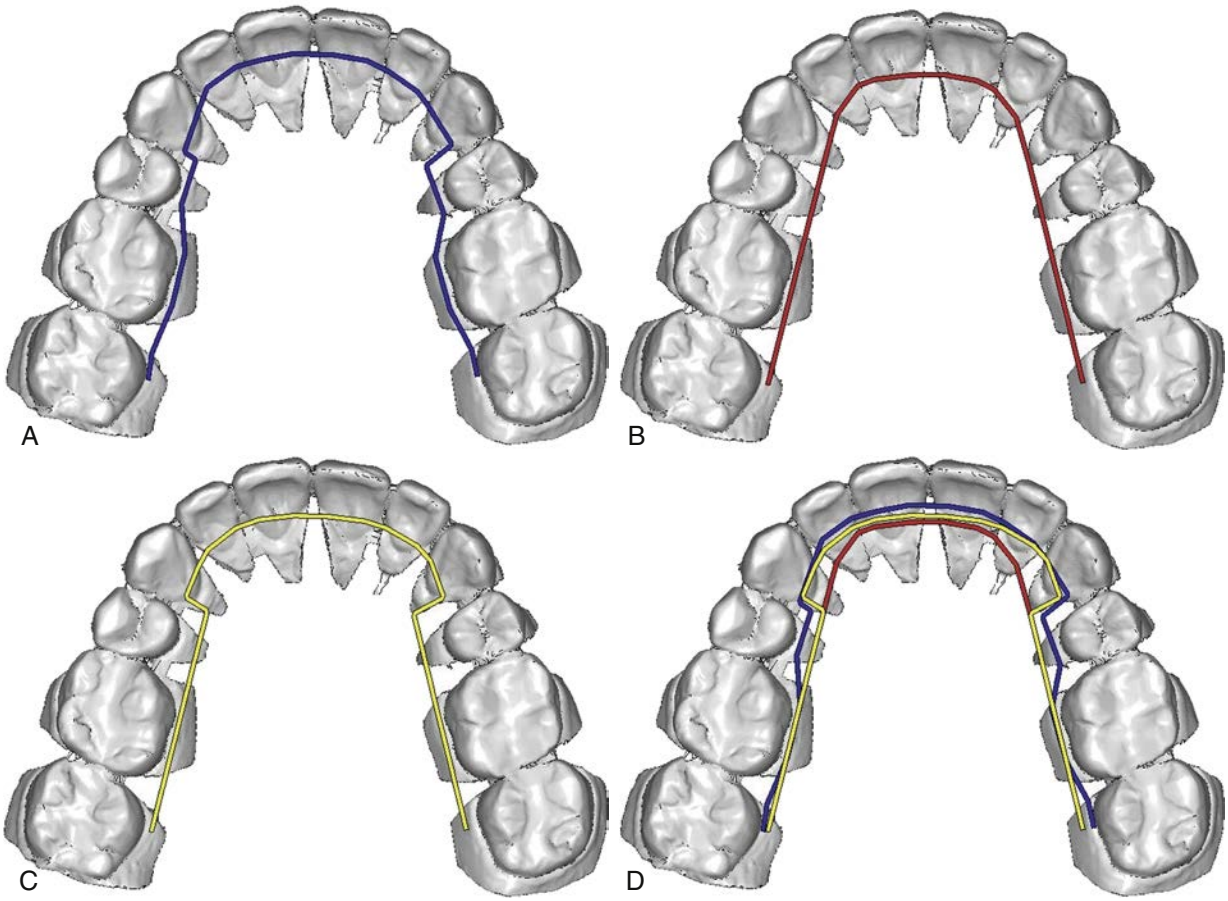


FIGURE 25-3 A, Different tooth thicknesses are compensated by first-order archwire bends. B, The archwire is straight. Compensation of the different tooth thicknesses must be accomplished by thicker brackets; therefore, additional composite is required under the brackets in the laboratory process, or the bracket bases themselves have to be thicker. C, Mushroom arch form. Two first-order bends are placed between the canines and first premolars. D, Superposition of the three types of archwires. In the anterior segment, major differences can be noticed. The archwire, which is completely individual in the first order (*blue*), allows for the flattest appliance. The straight archwire (*red*) produces by far the biggest restriction for the tongue.

appliances, just reproducing the approach of conventional labial techniques led to disillusionment with lingual appliances in the 1980s, especially in the United States.

First-Order Compensations

While looking at an ideal dental arch from the occlusal view, the labial surfaces of the teeth can be found along a regular arch line. On the lingual side, the teeth exhibit a pattern where surfaces are irregular. To develop an efficient lingual appliance, these differences in thickness occurring among teeth have to be compensated for with a laboratory process. There are three main strategies to compensate for these differences (Fig. 25-3):

1. **Compensation with first-order customized archwires that level out the tooth thickness differences** (see Fig. 25-3, *A*). The customization of the brackets only incorporates the customized programming of the second-order and third-order dimensions, which allows for a comparatively flat profile.[10,11,13]
2. **Compensation by adjusting the bracket base thickness.** In this strategy, brackets are set up not only with second-order and third-order programming but also with first-order programming.[8] This results in a considerable increase in appliance thickness, which may lead to patient discomfort. Because the brackets incorporate the three-dimensional programming in its entirety, "straight wires" may be used (see Fig. 25-3, *B*). Although the use of straight wire appliances has been established in labial techniques, it results in considerable disadvantages in the lingual technique for both the patient and the orthodontist because of the markedly thicker brackets. Hohoff et al.[29] and Stamm et al.[30] reported that thicker appliances cause more patient discomfort and more problems during speaking and eating. Tongue irritation is also observed more frequently when brackets encroach on tongue space. The interbracket distance becomes even shorter because of the increased bracket thickness. This not only makes inserting the archwires more challenging but also makes complete bonding of all brackets impossible at the start of treatment, even in moderate cases of crowding, because of the brackets' size. When thicker brackets are used, the distance between the center of resistance of the tooth and the point of force application increases, making it more challenging to correct

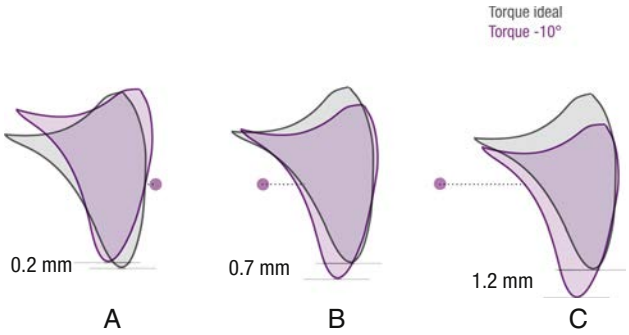


FIGURE 25-4 Effect of incorrect torque (−10 degrees) on the vertical position of the incisor edge of an upper central incisor. **A,** With a labial appliance, only 0.2 mm of vertical discrepancy can be noticed. **B,** The same incorrect torque creates an increased vertical discrepancy (0.7 mm) even if a very flat lingual appliance is used. **C,** Thicker lingual appliances (e.g., lingual straight wire appliances) are not only much more uncomfortable for the patient; they are also much more prone to third-order problems.

torque problems in the third dimension (Fig. 25-4). The larger and thicker the bracket, the higher the rate of bracket loss because there is more chance of biting and debonding a thicker bracket.

3. **Compensation by partial first-order bend of the archwires.** In this case, only the difference in thickness between the canine and the first premolar is compensated with a first-order bend of the archwire. The archwire is straight from canine to canine and from first premolar to second molar. The shape of this archwire setup reminds one of a mushroom, which is why the lingual technique was also called the "mushroom technique" by its pioneers[4] (see Fig. 25-3, *C*).

Torque Control

One major difference between labial and lingual appliances with regard to treatment mechanics is the importance of accurate torque control and its consequences on vertical tooth position. Incorrect torque control results in a completely different effect in a labial bracket versus a lingual bracket as illustrated in Figure 25-4. Two upper central incisors are depicted, one of which exhibits an ideal position; the other one displays a torque problem of −10 degrees. When a labial appliance is used, the effect of 10 degrees of torque discrepancy will be unnoticed by the patient, and only a very detail-oriented orthodontist could recognize the problem (see Fig. 25-4, *A*). When a lingual appliance is used, an incorrect torque of −10 degrees directly causes a visible malposition in the vertical plane, and the tooth appears extruded (see Fig. 25-4, *B*). This is even more severe as the distance between the tooth surface and the archwire increases, which is the standard situation when using a lingual straight wire approach with thicker brackets; vertical discrepancies are easily detected by the patient (see Fig. 25-4, *C*).

In state-of-the-art lingual orthodontic appliances, torque control is achieved by the exact fit of a rectangular archwire into the bracket slot. To ensure perfect third-order control, either full engagement of the slot by the archwires or smaller archwires with overcorrections are needed. Acceptable torque control in the course of the lingual treatment can be realized only if archwires and slots are precisely manufactured with only minimal tolerances.[31] The main cause for marked torque play in labial appliances, in particular in the "passive self-ligating systems," is their design with considerably oversized bracket slots.[32] Oversize of more than 15% on average of the nominal size may occur in these labial appliances. As an example, the slot in a 0.022-inch bracket system may have a real slot size of 0.025 inch. Nonetheless, reproducible, good outcomes can be obtained with these types of labial systems. The clinician must recognize that there are differences between the need for precise bracket–wire interfaces between the labial and lingual techniques, with lingual appliances demanding a higher focus on precision and potentially added torque to provide proper control. Consequently, the requirements for lingual appliances continue to be different even during routine orthodontic tooth movements such as anterior tooth retraction.

Exposure of Bonding Area

Most orthodontic patients are children and adolescents. Within this age group, the bonding area of the lingual surface can be reduced, especially in teeth that have just erupted. This is true, particularly for the upper and lower second molars and the lower premolars. Bonding conventional lingual appliances is therefore difficult or impossible in this age group. This is one of the main reasons why in the past, lingual treatment has been provided only to adults. The number of adults requesting orthodontic treatment has always been relatively low, so conventional lingual appliances were only appropriate for a limited group of patients. The use of the completely customized lingual appliances, in which bonding pads can be individually designed, and in cases of a reduced lingual bonding area, partially extended over the occlusal surface, now opens up a new target group that includes both children and adolescents (see Fig. 25-9, *H* and *I*).

Innovation in Completely Customized Lingual Appliances

In recent years, the lingual WIN System has increased its popularity among the customized lingual appliances around the world. Besides exact implementation of the planned treatment, the focus of this appliance is on simplification of clinical handling to the user's benefit. Users can rely exclusively on inserting traditional ligatures as are common in fixed labial appliances. Additionally, treatment duration is shorter on average compared with other customized lingual appliances because of very low rates of bracket loss[33] and a more efficient leveling and aligning stage. Because manufacturing expense is substantially lower than with other custom lingual bracket systems, lingual treatment has become affordable for a larger share of patients.

Efficient Leveling and Aligning with Lingual Appliances

The leveling and aligning stage presents important differences between common labial and lingual approaches. Labial appliances allow bonding of all brackets from the start of treatment in most cases. This is not often the case for lingual systems, where in the mandibular arch braces have to be bonded in a multistage process depending on the inserted system. Because of both the repeated performance of bracket bonding sessions and the more complex task this represents for the preparatory step of gaining space for the respective

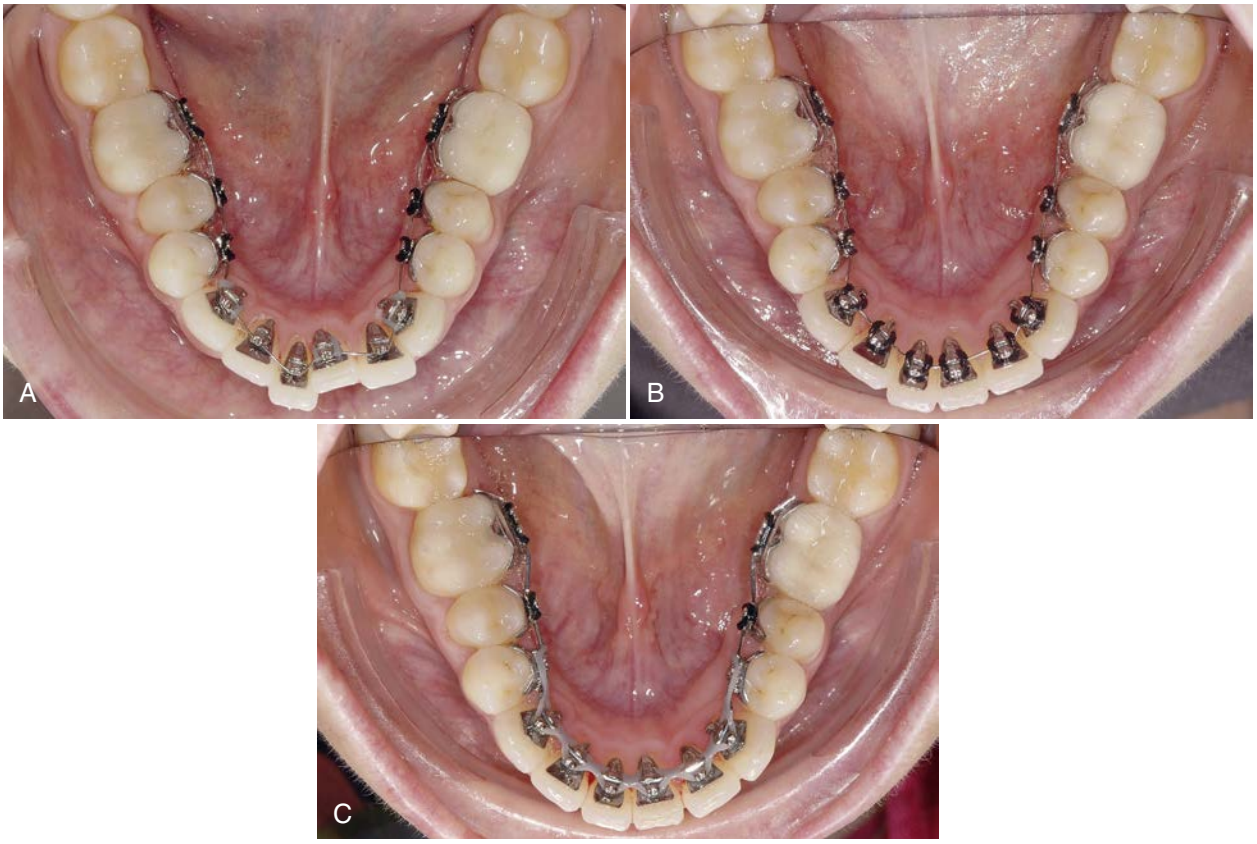


FIGURE 25-5 Aligning type 1. **A,** The first archwire, a 0.012-inch round SE-NiTi wire, is placed behind the incisal wings of the anterior teeth. **B,** Eight weeks later, the same archwire is placed into the normal slot with simple elastics. **C,** Nine weeks later, aligning is completed with a 0.016- × 0.022-inch SE-NiTi wire.

individual teeth and attachments, this multistage bonding process is associated with a considerable additional investment in treatment time. Three types of aligning are used in lingual orthodontics, depending on characteristics of the presenting malocclusion.

Aligning Type 1

Aligning type 1 is used when all brackets can be bonded from the start of treatment (Fig. 25-5). In cases of significant crowding, however, it is recommended not to insert the archwire into the bracket slot but rather one level higher, in the area of the wings. This enables initially longer archwire lengths in the interbracket area and resultant posterior tooth uprighting and anterior tooth proclination. At a later stage, the archwire can then be inserted into the slot proper using standard elastic ligatures.

Aligning Type 2

Aligning type 2 is indicated when there are brackets that cannot be bonded because of insufficient surface exposed (Fig. 25-6). In these cases, there is enough surface exposed to bond a smaller attachment constructed with a bonding pad and a flexible winglet. The thin NiTi archwire is set behind the winglet right at the start of treatment. As soon as space has been gained, the lingual bracket can be bonded in a subsequent bonding session. After all brackets are bonded, the clinical process described under Aligning Type 1 is carried out.

Aligning Type 3

Aligning type 3 is defined by a clinical situation when the dental surface is not exposed at all, typically when the tooth is impacted or deviated out of arch form (Fig. 25-7). In these cases, the insertion of thin NiTi archwires activated by stops is recommended. When the space has been opened and bonding can occur at this tooth, the respective treatment steps described under Aligning Type 1 or 2 are applied.

SUMMARY

Completely customized lingual appliances and techniques have revolutionized lingual orthodontics in recent years. In addition to the aesthetic advantages of these appliances, reduced risk of decalcification, superior third-order control, and accurate achievement of the treatment goals along with the possibility to treat children and adolescents have led to a better acceptance of lingual appliances in routine orthodontic practice. Indeed, the number of patients treated with lingual or combined lingual and labial appliances continues to rise worldwide. However, within the triad of orthodontist, patient, and appliance, which is of such great importance in any orthodontic therapy, even an ideal appliance alone will still not offer any guarantee of a successful treatment outcome. Rather, the know-how and continued attention to the details of diagnosis, treatment planning, and ongoing execution of biomechanics by the orthodontist finally and always determine the results.

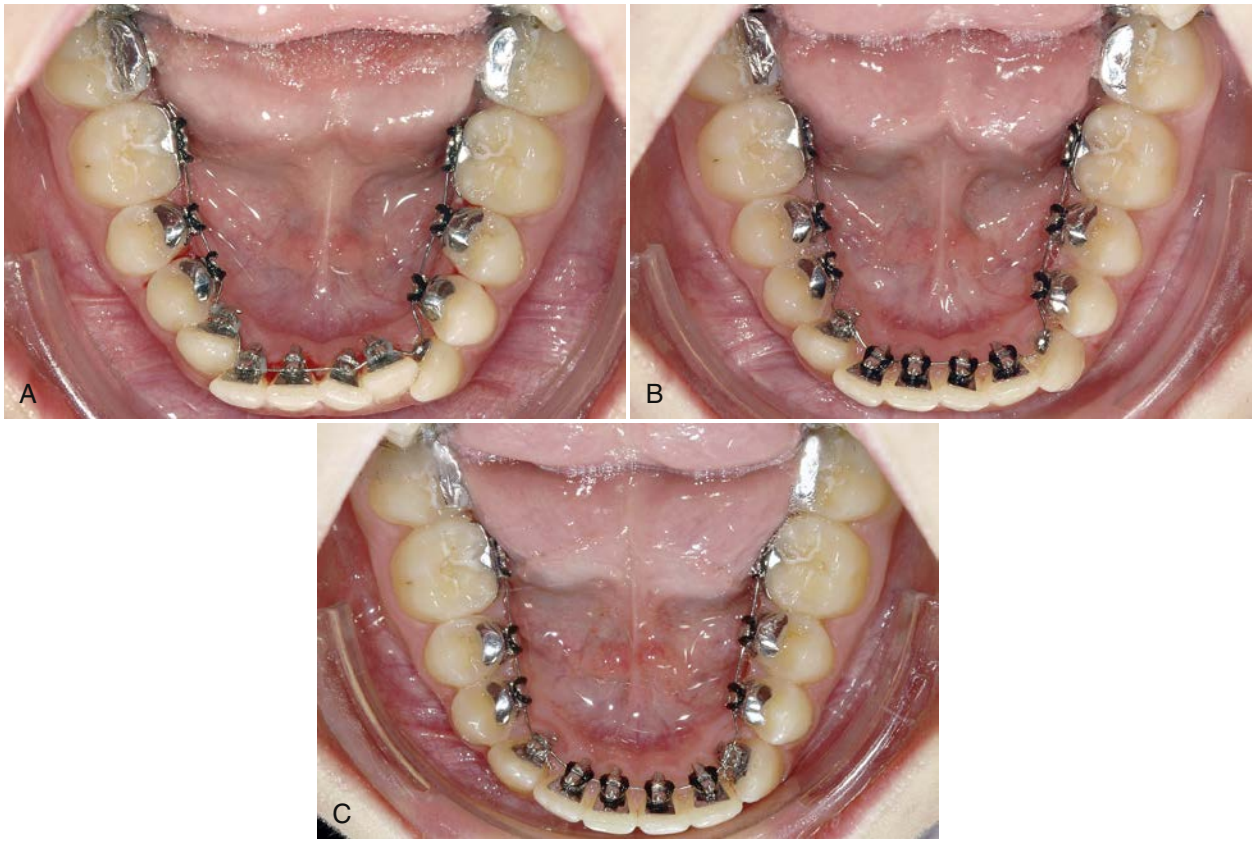


FIGURE 25-6 Aligning type 2. **A,** A bracket could not be bonded on the lower left canine at the initial stage. Therefore, a two dimensional control winglet is used for initial alignment of the tooth. Because of the amount of crowding, the archwire is placed behind the incisal wings. **B,** Twelve weeks later, the same archwire is placed into the normal slot. A lasso elastic is used to derotate the lower left canine. **C,** One month later, a bracket could be bonded. The archwire used is still the initial 0.012-inch superelastic NiTi wire.

FIGURE 25-7 Aligning type 3. **A, B,** A 0.012-inch round superelastic NiTi wire with two stops mesial of the first premolars is used for creating space. In the area of the blocked out lower right lateral incisor the archwire is compressed. **C,** Two months later, a winglet can be bonded to the lower lateral incisor. The two stops were removed, and now the archwire is placed behind the incisal wings. **D,** Three months later, a bracket can be bonded. Note: Only one wire was used.

CASE STUDY 25-1 LINGUAL TREATMENT OF ADULT PATIENTS

Completely customized lingual treatment of a man with previous asymmetric extraction. Treatment goals were incorporated into a diagnostic and therapeutic setup on which the appliances were fabricated. An indirect bonding procedure was used, and wires were designed and optimized to the desired dental arch form and needed mechanics. These included leveling and alignment, asymmetric space closure, Class II mechanics, correction of upper midline position, torque control, and finishing and detailing.

FIGURE 25-8 A–I, A 27-year-old patient with severe upper midline deviation to the left. The patient previously underwent orthodontic treatment with asymmetric extraction in the upper arch during adolescence. The right side presented a full Class II canine relationship. The lower midline was slightly shifted to the right. Relapse of the lower crowding occurred despite an original bonded mandibular retainer.

CASE STUDY 25-1 LINGUAL TREATMENT OF ADULT PATIENTS—cont'd

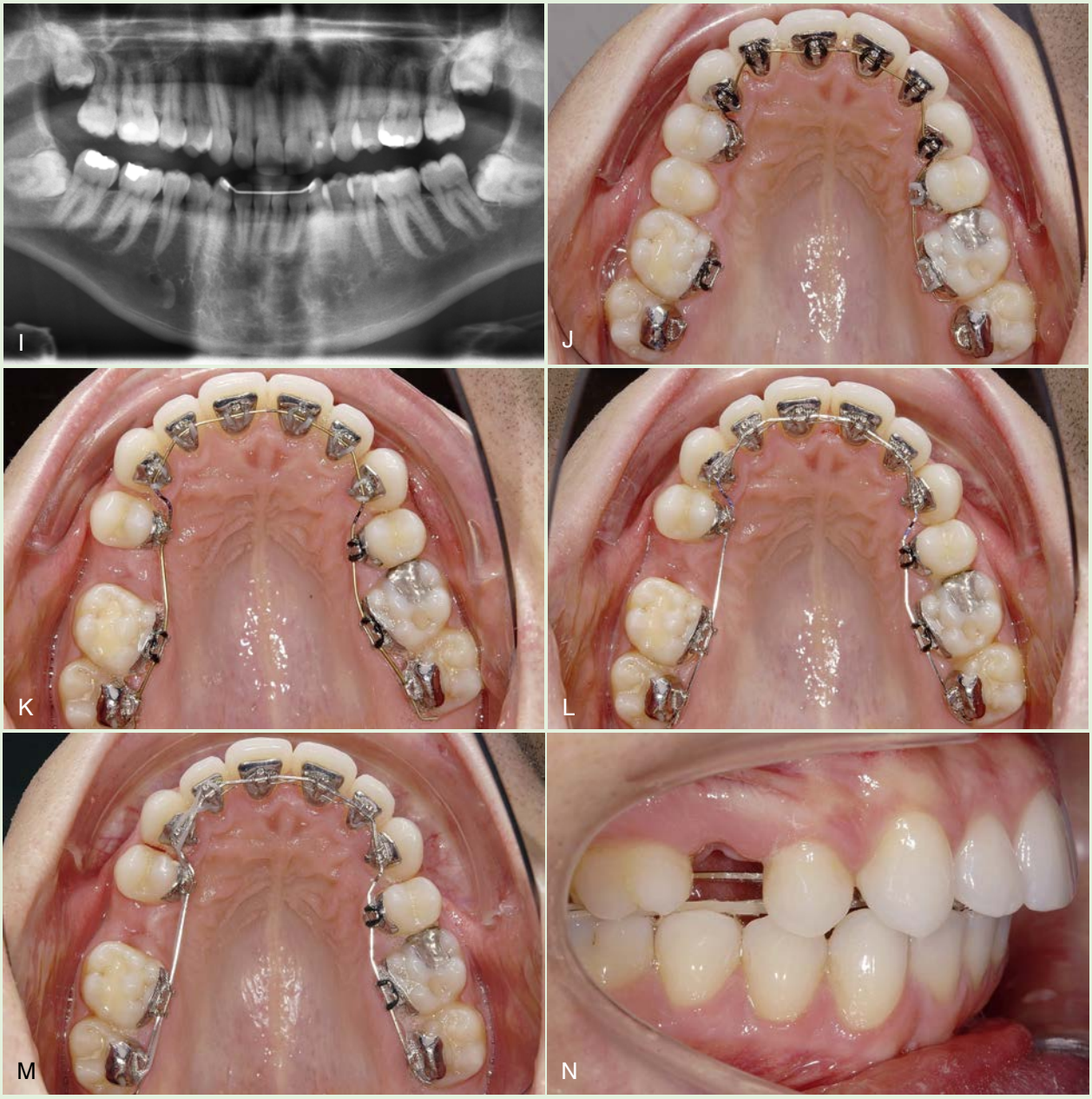


FIGURE 25-8, cont'd **J,** Immediately after upper arch bonding, a 0.012-inch round NiTi wire was placed for initial alignment. Indirect bonding protocol requires that the extractions are performed after bracket placement. **K,** A 0.016- × 0.022-inch NiTi wire was used for further alignment. The archwire was straight on its right side. **L,** A 0.016- × 0.024-inch stainless steel archwire was inserted for space closure and midline correction. Before the archwire insertion, a metal ligature was placed to attach the upper incisors and canines. The wire was ligated with metal ligatures. Note the different archwire shape on the right side (straight) and on the left side (individual). **M,** At the next appointment, the upper right canine was retied with a new steel ligature to improve tip control in the vertical slot during retraction. A power chain for space closure was attached to the lateral incisor for simultaneous wear of Class II elastics. **N,** The patient was able to attach Class II elastics on the hook of the canine.

Continued

CASE STUDY 25-1 LINGUAL TREATMENT OF ADULT PATIENTS—cont'd

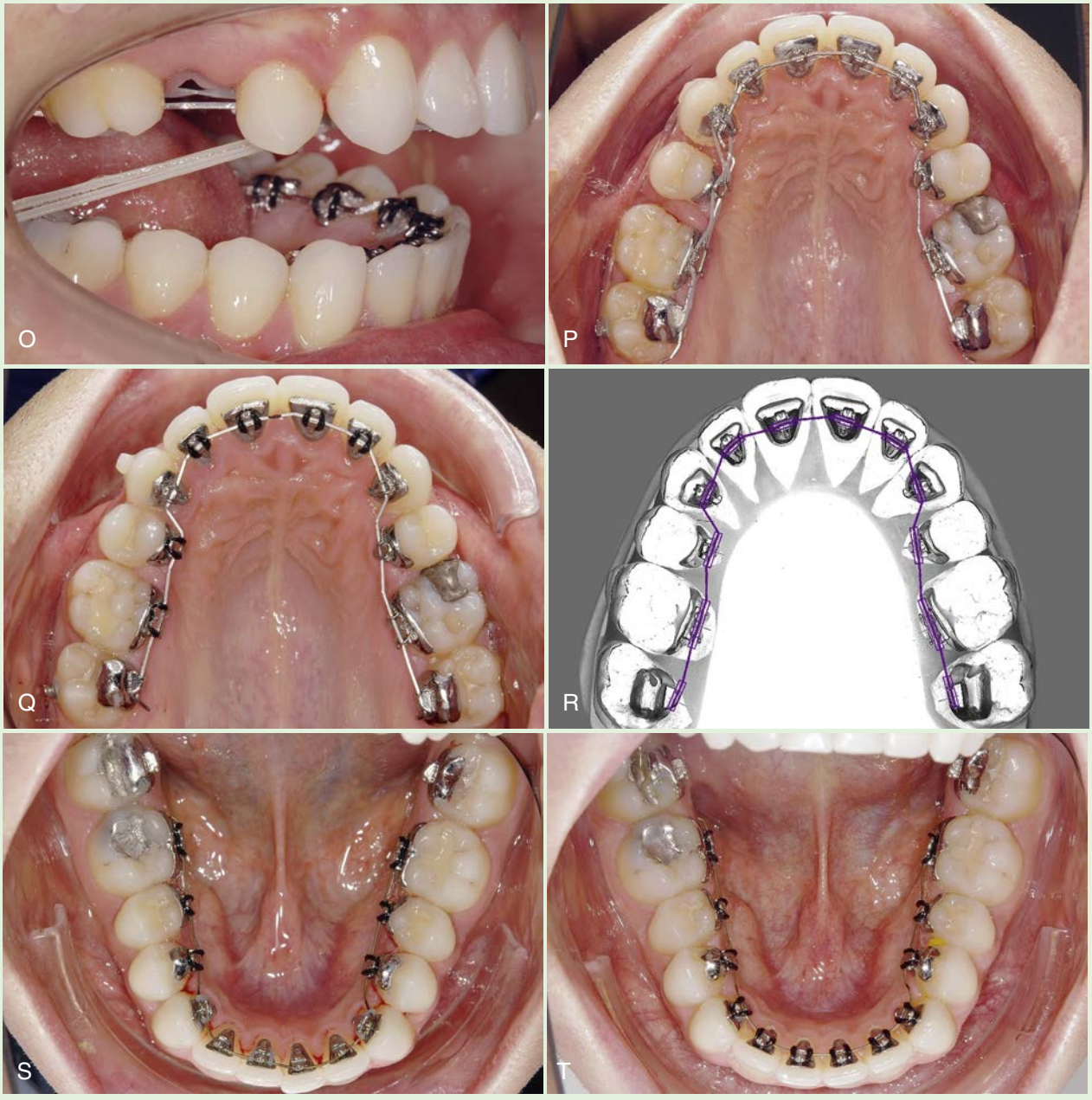


FIGURE 25-8, cont'd **N, O,** The remaining space had to be closed mainly by distalizing the anterior teeth to a Class I canine relationship. Class II elastics were worn full time to support space closure with power chains. **P,** The elastic extended from the upper canine hook to a labially bonded button on the lower second molar. At the final stage of space closure, double-cable mechanics with one power chain lingual and a companion clear chain buccal were used for residual space closure. The transparent labial power chain was attached to the archwire between the lateral incisor and the canine and connected to a labial button on the upper second molar. **Q,** After space closure, a 0.018- × 0.018-inch beta-titanium archwire was inserted for first-order detailing on the right side. To improve tip control, the upper right canine was ligated with a metal ligature. A vertical bend back prevented space from reopening. **R,** The final result and the treatment plan as represented by the virtual archwire are compared in the occlusal view. **S, T,** A 0.012-inch round NiTi wire was used for initial mandibular arch aligning. The wire was placed behind the wings of the brackets initially, and later the wire was ligated in to the slot.

CASE STUDY 25-1 LINGUAL TREATMENT OF ADULT PATIENTS—cont'd

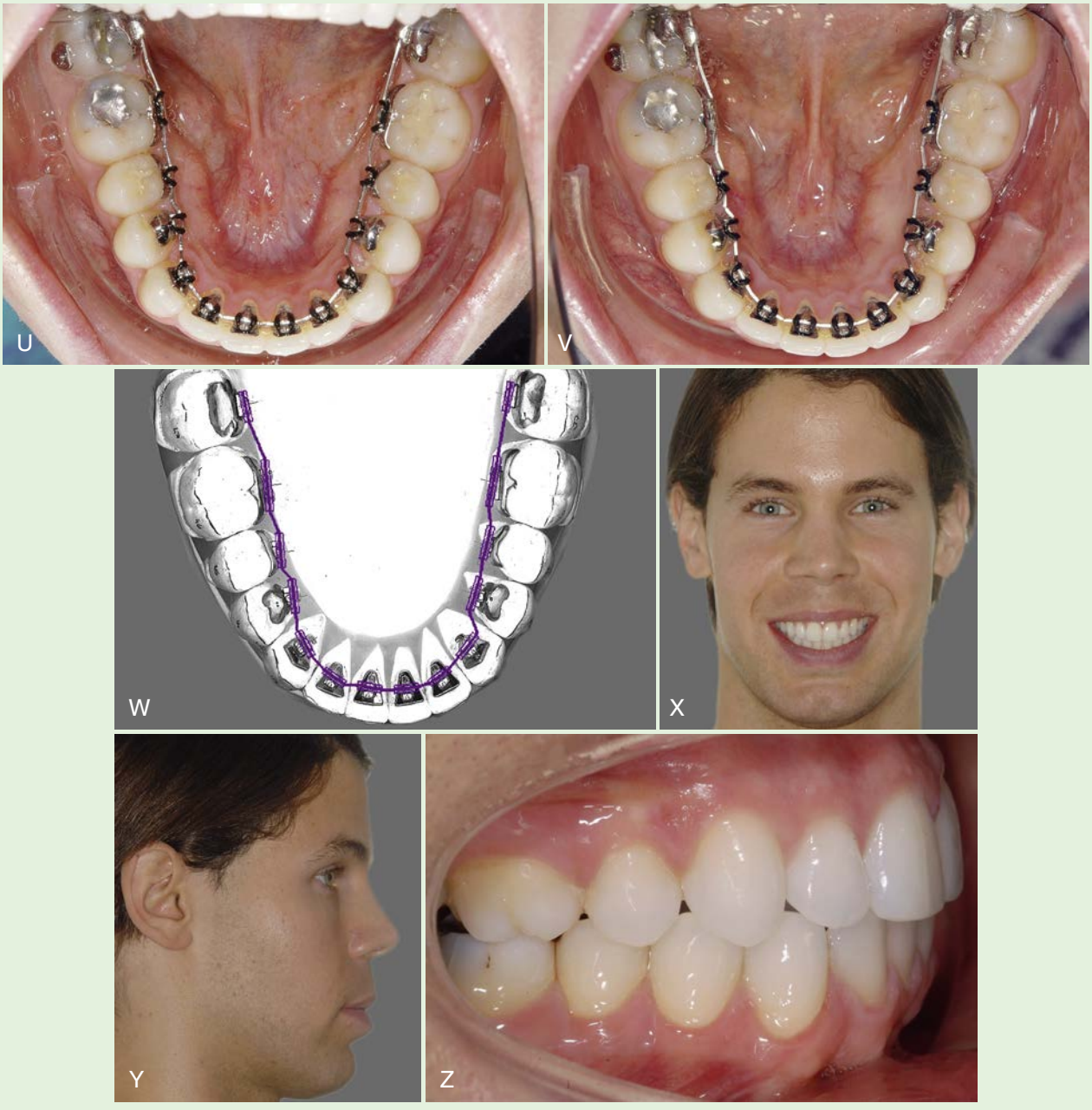


FIGURE 25-8, cont'd **U,** After the insertion of a 0.016-inch × 0.022-inch NiTi wire, a 0.016- × 0.024-inch stainless steel archwire was placed for intermaxillary elastic wear. **V,** Final torque control was achieved with a 0.018- × 0.018-inch beta-titanium archwire. Note that no over-ties were used. **W,** The final result and the treatment plan as represented by the virtual mandibular archwire are compared in the occlusal view.

Continued

CASE STUDY 25-1 LINGUAL TREATMENT OF ADULT PATIENTS—cont'd

FIGURE 25-8, cont'd AA–FF, The patient after 26 months of treatment. The upper midline deviation was corrected. Despite vertical insertion of the archwire in the frontal area, any unwanted tipping of the incisors was prevented. A Class I canine relationship was achieved on both sides. Upper and lower fixed retainers were bonded for retention.

CASE STUDY 25-2 LINGUAL TREATMENT OF CHILDREN AND ADOLESCENTS: NONEXTRACTION CASE WITH USE OF CLASS II ELASTICS

Completely customized lingual treatment of a female teenager with full dental Class II division 2. Treatment involved customization and fabrication of appliances through an individualized setup, use of half-occlusal coverage molar attachments for bite opening and debonding prevention, an extra-torque upper archwire for torque expression, Class II mechanics, and finishing and detailing.

FIGURE 25-9 A–G, A 14-year-old patient with a bilateral full Class II malocclusion and retroclined upper incisors. All first molars had an enamel defect that was restored with composite. Both the upper central incisors and upper canines showed negative torque.

Continued

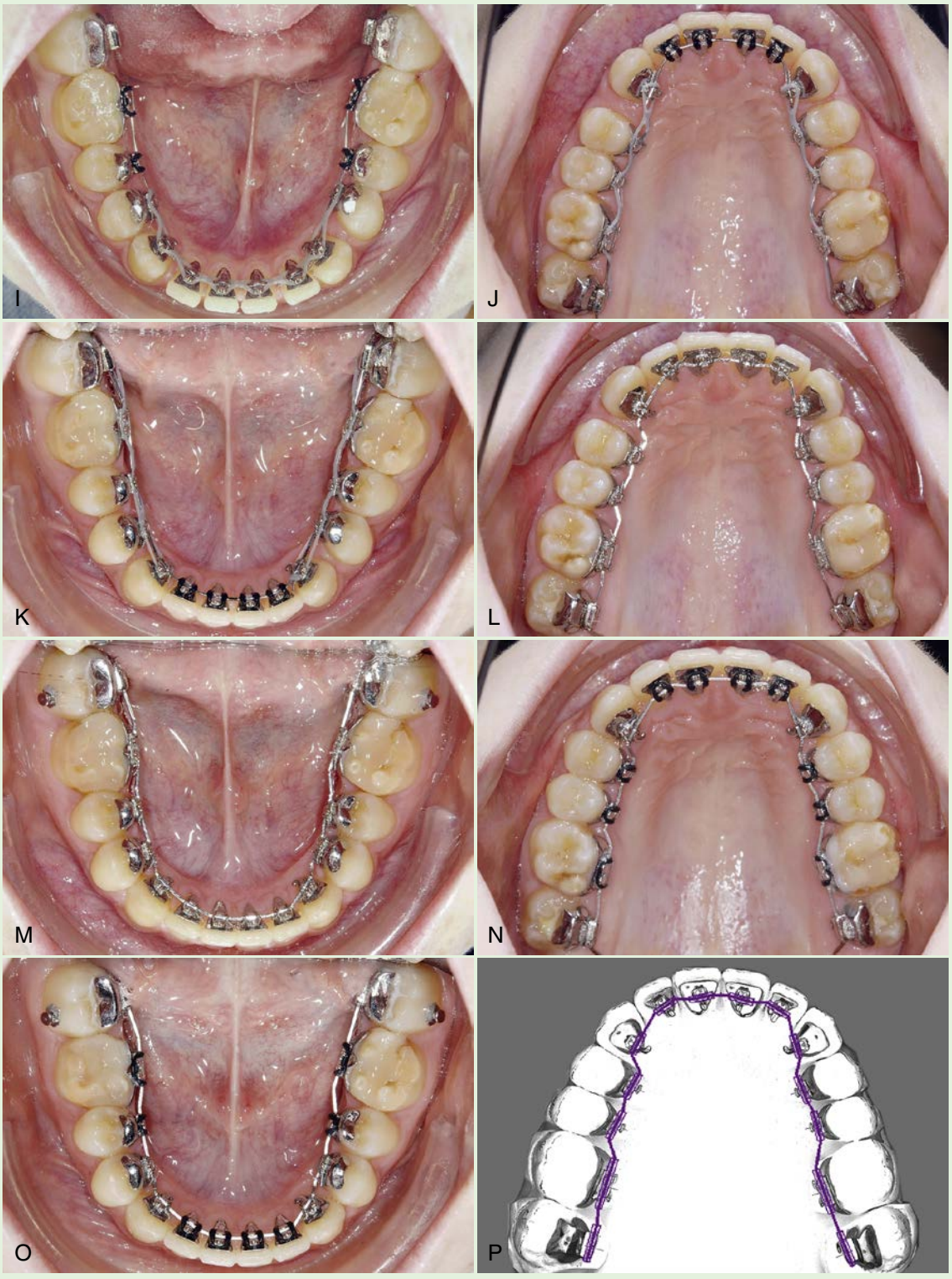


FIGURE 25-9, cont'd H, I, All brackets were bonded in the first appointment. 0.012-inch round NiTi archwires were placed in both arches. To prevent further space opening during leveling, a prestretched power chain was used in the lower arch. Partial extension of the bracket bases on the occlusal surfaces of all second molars helped to prevent bracket debonding and opened the bite. J, K, 0.016- × 0.022-inch NiTi archwires were placed in both arches for further leveling and aligning. No over-ties were used. L, M, 0.016- x 0.024-inch stainless steel archwires were placed in both arches. The upper archwire had extra torque of 13 degrees in the anterior region from canine to canine. Buttons were bonded on the labial surface of the lower second molars for Class II elastic wear. N, O, After achievement of slight overcorrection of the Class II relationship, 0.018- × 0.018-inch beta-titanium archwires were placed in both arches. To prevent space reopening, the wires were vertically bent back distal to the second molars in both arches.

FIGURE 25-9, cont'd P, Q, The final result and the treatment plan are compared by way of overlying the virtual wire on the treated model in the occlusal view. Total treatment time was 19 months. R, S, Upper and lower bonded retainers were placed for retention. The patient was asked to wear a night activator to stabilize the anteroposterior correction. T–V, At the end of leveling and aligning, the torque in the upper front teeth was too negative and prevented achieving a Class I relationship. The torque of the upper canines was also a major obstacle for the lower canines to move forward.

Continued

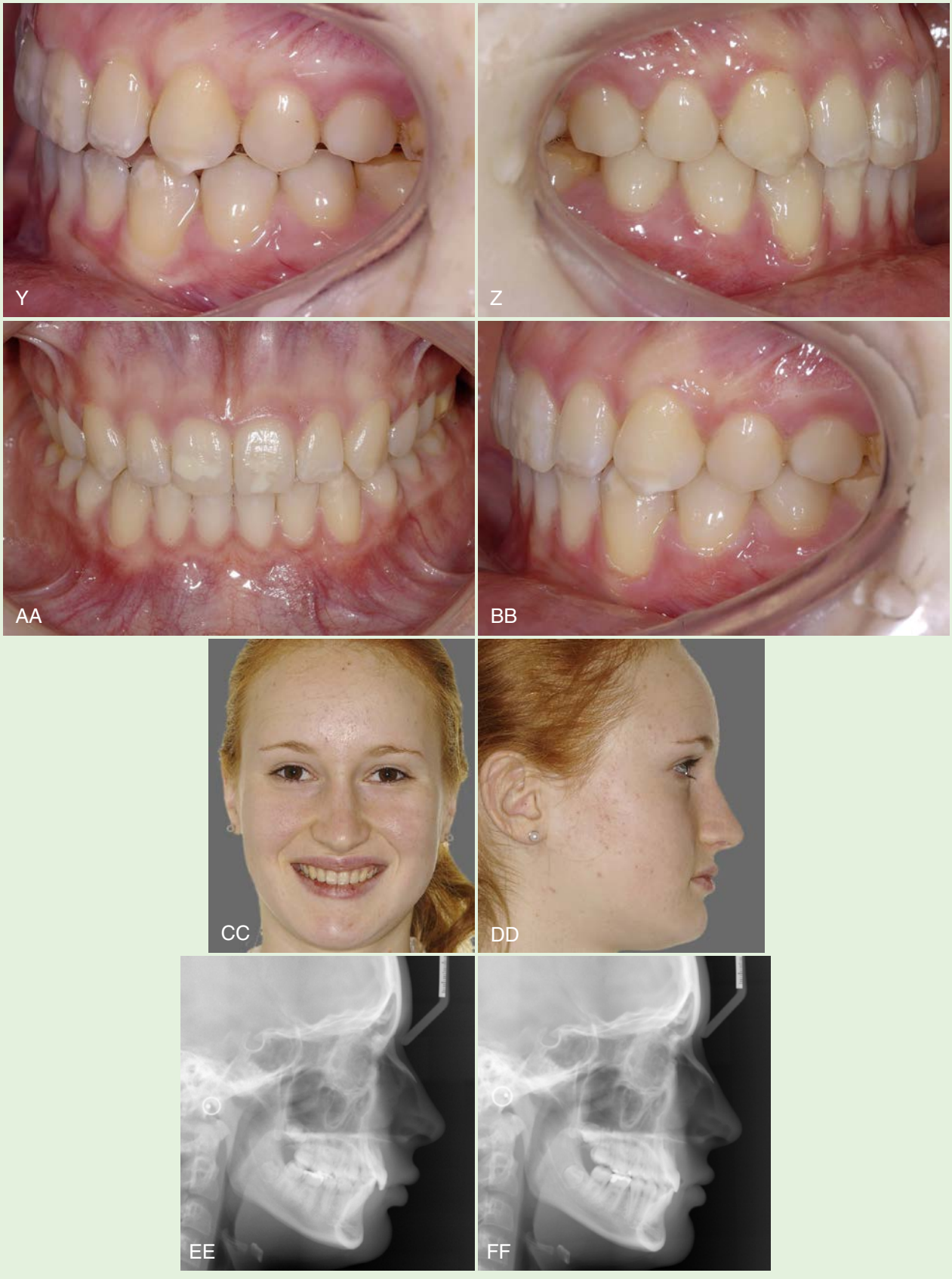


FIGURE 25-9, cont'd W—Y, Torque was corrected with the upper 0.016- × 0.024-inch stainless steel archwire mainly because of the extra torque built into the anterior area of the wire. Continued attention to torque management is critical to anterior retraction in lingual appliances because of the lingual bracket placement relative to the center of tooth rotation. The patient had to wear Class II elastics full time. Z–DD, Final results after 19 months of lingual treatment.

CASE STUDY 25-2 LINGUAL TREATMENT OF CHILDREN AND ADOLESCENTS: NONEXTRACTION CASE WITH USE OF CLASS II ELASTICS—cont'd

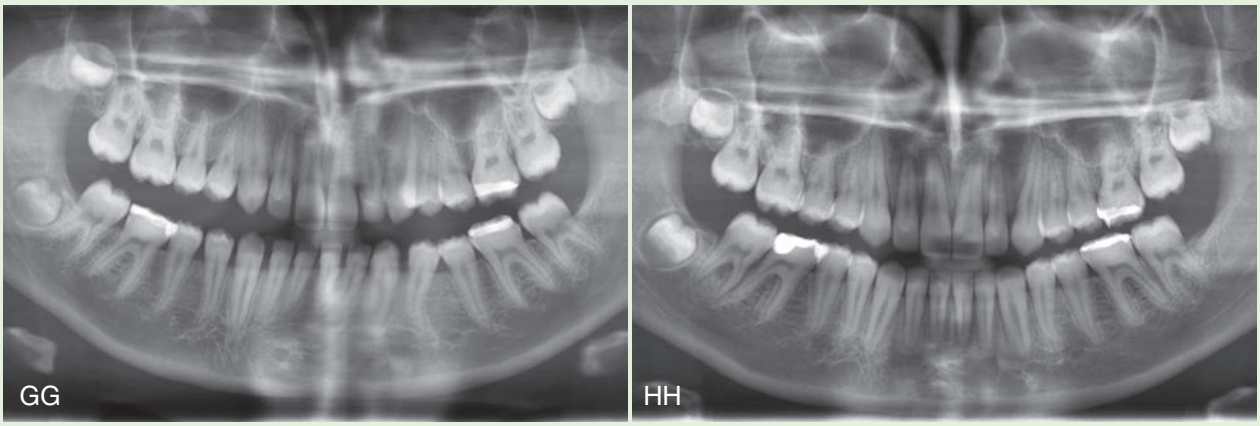


FIGURE 25-9, cont'd EE, FF, The lateral headfilm shows excellent torque control in both arches. Lower incisor proclination was prevented. GG, HH, The panoramic radiograph shows good tip control.

REFERENCES

1. Sergl HG, Klages U, Zentner A. Functional and social discomfort during orthodontic treatment—effects on compliance and prediction of patients' adaptation by personality variables. *Eur J Orthod*. 2000;22(3):307–315.
2. Alexander CM, Alexander RG, Gorman JC, et al. Lingual orthodontics. A status report. *J Clin Orthod*. 1982;16(4):255–262.
3. Fillion D. Lingual orthodontics: a system for positioning the appliances in the laboratory. *Orthod Fr*. 1989;60(2):695–704.
4. Fujita K. New orthodontic treatment with lingual bracket mushroom arch wire appliance. *Am J Orthod*. 1979;76(6):657–675.
5. Gorman JC. Treatment with lingual appliances: the alternative for adult patients. *Int J Adult Orthod Orthognath Surg*. 1987;2(3):131–149.
6. Kurz C. The use of lingual appliances for correction of bimaxillary protrusion (four premolars extraction). *Am J Orthod Dentofac Orthop*. 1997;112(4):357–363.
7. Kurz C, Swartz ML, Andreiko C. Lingual orthodontics: a status report. Part 2: research and development. *J Clin Orthod*. 1982;16(11):735–740.
8. Takemoto K, Scuzzo G. The straight-wire concept in lingual orthodontics. *J Clin Orthod*. 2001;35(1):46–52.
9. Wiechmann D. Lingual orthodontics (part 1): laboratory procedure. *J Orofac Orthop*. 1999;60(5):371–379.
10. Mujagic M, Fauquet C, Galletti C, et al. Digital design and manufacturing of the Lingualcare bracket system. *J Clin Orthod*. 2005;39(6):375–382.
11. Wiechmann D. A new bracket system for lingual orthodontic treatment. Part 1: theoretical background and development. *J Orofac Orthop*. 2002;63(3):234–245.
12. Wiechmann D. A new bracket system for lingual orthodontic treatment. Part 2: first clinical experiences and further development. *J Orofac Orthop*. 2003;64(5):372–388.
13. Wiechmann D, Rummel V, Thalheim A, et al. Customized brackets and archwires for lingual orthodontic treatment. *Am J Orthod Dentofac Orthop*. 2003;124(5):593–599.
14. Wiechmann D, Schwestka-Polly R, Hohoff A. Herbst appliance in lingual orthodontics. *Am J Orthod Dentofac Orthop*. 2008;134(3):439–446.
15. Kokich VG. Create the vision. *Am J Orthod Dentofacial Orthop*. 2011;140(6):751.
16. Proffit WR, Sarver DM. Contemporary orthodontic appliances. In: Proffit WR, ed. *Contemporary Orthodontics*. 5th ed. St. Louis: Mosby; 2013.
17. Miethke RR, Melsen B. Effect of variation in tooth morphology and bracket position on first and third order correction with preadjusted appliances. *Am J Orthod Dentofacial Orthop*. 1999;116(3):329–335.
18. Pauls AH. Therapeutic accuracy of individualized brackets in lingual orthodontics. *J Orofac Orthop*. 2010;71(5):348–361.
19. Grauer D, Proffit WR. Accuracy in tooth positioning with a fully customized lingual orthodontic appliance. *Am J Orthod Dentofacial Orthop*. 2011;140(3):433–443.
20. Thalheim A, Schwestka-Polly R. Clinical realisation of a setup in lingual orthodontics. *Inf Orthod Kiefer Orthop*. 2008;40:277–282.
21. Wiechmann D, Schwestka-Polly R, Pancherz H, Hohoff A. Control of mandibular incisors with the combined Herbst and completely customized lingual appliance—a pilot study. *Head Face Med*. 2010;6:3.
22. Heymann GC, Grauer D. A contemporary review of white spot lesions in orthodontics. *J Esthet Restor Dent*. 2013;25(2):85–95.
23. Fornell AC, Sköld-Larsson K, Hallgren A, et al. Effect of a hydrophobic tooth coating on gingival health, mutans streptococci, and enamel demineralization in adolescents with fixed orthodontic appliances. *Acta Odontol Scand*. 2002;60(1):37–41.
24. Gorelick L, Geiger AM, Gwinnett AJ. Incidence of white spot formation after bonding and banding. *Am J Orthod*. 1982;81(2):93–98.
25. Mitchell L. Decalcification during orthodontic treatment with fixed appliances: an overview. *Br J Orthod*. 1992;19(3):199–205.
26. Øgaard B, Larsson E, Henriksson T, et al. Effects of combined application of antimicrobial and fluoride varnishes in orthodontic patients. *Am J Orthod Dentofac Orthop*. 2001;120(1):28–35.
27. van der Veen MH, Attin R, Schwestka-Polly R, Wiechmann D. Caries outcomes after orthodontic treatment with fixed appliances: do lingual brackets make a difference? *Eur J Oral Sci*. 2010;118(3):298–303.
28. Wiechmann D, Klang E, Helms HJ, Knösel M. Lingual appliances reduce the incidence of white-spot lesions during orthodontic multibracket treatment. *Am J Orthod Dentofac Orthop*. 2015;148(3):414–422.
29. Hohoff A, Stamm T, Ehmer U. Comparison of the effect on oral discomfort of two positioning techniques with lingual brackets. *Angle Orthod*. 2004;74(2):226–233.
30. Stamm T, Hohoff A, Ehmer U. A subjective comparison of two lingual bracket systems. *Eur J Orthod*. 2005;27(4):420–426.
31. Meling TR, Odegaard J. The effect of cross-sectional dimensional variations of square and rectangular chrome-cobalt archwires on torsion. *Angle Orthod*. 1998;68(3):239–248.
32. Cash AC, Good SA, Curtis RV, McDonald F. An evaluation of slot size in orthodontic brackets—are standards as expected? *Angle Orthod*. 2004;74(4):450–453.
33. Knösel M, Klang E, Helms HJ, Wiechmann D. Lingual orthodontic treatment duration: performance of two different completely customized multi-bracket appliances (Incognito and WIN) in groups with different treatment complexities. *Head Face Med*. 2014;10:46.

26

Clear Aligner Treatment

David E. Paquette, Clark Colville, and Timothy Wheeler

OUTLINE

Brief History of Clear Aligner Treatment, 778
Scanning and Digitization, 780
Diagnosis and Treatment Planning for Clear Aligner Treatment, 782
ClinCheck—An Overview, 782
Biomechanics of Aligner Treatment, 783
Simple versus Difficult Movements, 785
Staging of Tooth Movement in ClinCheck, 786
Attachment Design in the Invisalign System, 788
Power Ridges and Pressure Areas in the Invisalign System, 791
Root Control in the Invisalign System, 792
Rotation Correction in the Invisalign System, 794
Extrusion Correction in the Invisalign System, 795
Auxiliaries and the Invisalign System, 796
Invisalign Teen, 797
Periodontal Considerations, 800
Summary, 800

BRIEF HISTORY OF CLEAR ALIGNER TREATMENT

Invisalign is an orthodontic technique that uses a series of computer-generated custom plastic aligners to guide the teeth gradually into proper alignment. Although the use of clear aligner treatment is not new, it is a growing part of the orthodontic market, and, as a result, many new products have become available. The concept on which treatment with clear aligners is based has been around since the 1940s. In 1945, H. D. Kesling[1] suggested that a series of tooth positioners be used to produce the types of movements that are needed for orthodontic treatment. Some years later, Nahoum[2] described a method to change tooth contours using thermoformed plastic. In 1971, Pontiz[3] introduced a thermoformed plastic appliance called the "invisible retainer" made on a master model that repositioned teeth with base-plate wax. He claimed that this appliance could produce limited tooth movement. McNamara and others[4] also described using invisible retainers to achieve minor tooth movement. Sheridan and colleagues[5] later developed a technique involving interproximal tooth reduction and progressive alignment using clear Essix appliances, and those techniques were further developed by Hilliard and Sheridan[6] with a series of special thermoforming pliers designed to enhance specific movements. Although these techniques based on Kesling's proposal of using removable appliances have been used to some degree in the past, the laboratory construction has always been tedious and has previously limited the widespread adoption of removable aligner techniques.

Clear aligners can be categorized into four basic categories as presented in Tables 26-1 to 26-4. Table 26-1 lists what are commonly known as positioners or guides along with their manufacturers, common use, and the material of which they are made. This category includes the original Allesee Orthodontic Appliances (AOA) positioner described by Kesling.[1] The second category (see Table 26-2) consists of those thermoformed appliances, sometimes known as Essix Retainers (Raintree Essix, DENTSPLY International, York, PA) and removable appliances referred to as spring aligners. The Essix appliance can be fabricated in the orthodontist's office or sent to a commercial laboratory, and it uses the techniques of Sheridan and Hilliard to move teeth. Table 26-3 lists those aligners that are fabricated from models that have had teeth cut out and manually moved to the correct position. If done in a series of models, an aligner can be fabricated from each model. These appliances can be used for minor movement of upper and lower anterior teeth and usually consist of three to five aligners. The last category, aligners fabricated from digitally manipulated models (see Table 26-4), is the largest growing area in aligner treatment. This growth is most likely attributable to the ability of these aligners to be used to treat malocclusions ranging from minor to complex, including both posterior and anterior teeth. Using digital technology to control tooth movement, intricate and precise tooth movements can be staged for each sequential aligner. However, tooth movement with aligners is as variable as it is with fixed appliances and can be dependent on individual variables (e.g., periodontal status, age, medication), as well as aligner variables (e.g., attachments, plastic). In this category, and the focus of this chapter, is Invisalign (Align Technology, Inc., Santa Clara, CA).

As a technique, Invisalign has now been commercially available to orthodontists since 1998. Invisalign treatment has a terminology that differs from traditional fixed appliances. A *midcourse correction* involves a temporary pause in treatment while a new scan is taken or impressions are made; treatment is then continued once new aligners are fabricated. The reasons for this midcourse correction can be many and include the following: lack of patient compliance; midtreatment restorative that rendered the current set of aligners unusable as a result of tooth morphologic structure; lack of one or more teeth tracking with aligners as planned, making the fit of the aligners unacceptable;

TABLE 26-1 Aligners Currently Available: Positioner and Guides

Product Name	Manufacturer	Description	Use	Material
Orthodontics Positioner	TP Orthodontics, Inc.	Custom stone setup done to finish	Remove braces 3–6 months earlier	Soft rubber or Crystal-Flex
Orthodontic Positioner	AOA	Custom setup on articulator	Minor tooth movement	Polyurethane
Nite-Guide; Occlus-o-Guide; Ortho-T; Preformed Positioner	Ortho-Tain	Premade guides for ages 5 years to adult	Arch formation and guided eruption; minor tooth movement	Polyurethane
Myobrace i-3, i-2, Myobrace	MRC	Premade guides for ages 2 years to adult; soft trainer and hard Myobrace Inner hard core and outer soft	Myofunctional development Use Frankel principles	Soft: silicone Hard: polyurethane

AOA, Allesee Orthodontic Appliances; *MRC,* Myofunctional Research Company.

TABLE 26-2 Aligners Currently Available: Activation Aligners and Appliances

Product Name	Manufacturer	Description	Use	Material
MTM Clear Aligner	DENSTPLY	Use of thermoforming pliers on clear retainer	Minor movement of U/L anterior teeth	Raintree Essix ACE plastic: Copolyester-proprietary
Straight 'N' Clear Cosmetic Correctors	GAC	Use of thermoforming pliers on clear retainer	Minor movement of U/L anterior teeth	Raintree Essix ACE plastic: Copolyester-proprietary
Inman Aligner	Inman Aligner	NiTi coil springs that activate labial and lingual bows	Minor movement of U/L anterior teeth	Acrylic
ClearAligner	ClearAligner	Spring aligner	Minor movement of U/L anterior teeth	No information

MTM, Minor Tooth Movement; *NiTi,* nickel titanium; *U/L,* upper and lower.

TABLE 26-3 Aligners Currently Available: Teeth Manually Reset

Product Name	Manufacturer	Description	Use	Material
Clear Image Aligners	Specialty Appliances	Up to 4 aligners/arch initially	Minor movement of U/L anterior teeth	No information
Red White Blue Simpli[5]	Ormoc AOA	Up to 3 aligners/arch Up to 5 aligners/arch; uses assist attachments	Minor movement of U/L anterior teeth; Move teeth up to 2.5 mm/tooth	No information
Triple Play	Ortho Organizers	Up to 3 aligners/arch	Minor movement of U/L anterior teeth	No information
MTM Clear Aligner	DENTSPLY International	No attachments; available as in-office and lab-created versions	Minor movement of U/L anterior teeth	Raintree Essix ACE: Copolyester-proprietary
Originator Clear Aligner System	TP Orthodontics	Available in 0.020-, 0.030-, 0.040-inch thickness	Minor to moderate U/L anterior crowding or spacing	
EZ-Align	Dynaflex	Up to 5 aligners	Minor movement of U/L anterior teeth	Dynaflex: Copolyester
Dual Laminate	TruTrain			Copolyester-PETG (hard/soft laminate)
Smart Moves	Great Lakes	Up to 3 setups/impression; 2 aligners/setup (1 hard aligner with soft center, 1 hard aligner) Individual tooth movement up to 1.0 mm/setup	Minor movement of U/L anterior teeth	Invisacryl: Copolyester proprietary
Graham Orthodontic Aligner	Graham Tool Co.		Anterior: 1–3-mm crowding; 1–2-mm spacing	Polyester-PETG (.040 inch)

AOA, Allesee Orthodontic Appliances; *MTM,* Minor Tooth Movement; *NiTi,* nickel titanium; *PETG,* polyethylene terephthalate glycol; *U/L,* upper and lower.

or a change in the treatment plan. Many orthodontists consider midcourse correction to be similar in concept to repositioning brackets partway through treatment with fixed appliances. Another term unique to aligner treatment is *refinement.* Refinement is similar to midcourse correction in that it generally involves pausing treatment and taking new impressions and ordering a new series of aligners. The only difference is that refinement takes place near the end of treatment when one or more teeth are apparently not positioned exactly as desired. This malposition could be attributable to small amounts of permanent distortion of the aligners, resulting in actual tooth positions that do not match the virtual treatment plan, or it could be because insufficient attention was paid to the details during the virtual setup phase. In any event, to achieve the desired results, another series of aligners is required. Refinement can be considered similar to artistic detail bends with fixed appliances. The final term unique to aligner treatment is *attachment.* Attachments are small composite additions to the tooth surface that

TABLE 26-4 Aligners Currently Available: Teeth Digitally Reset

Product Name	Manufacturer	Description	Use	Material
Invisalign Full, Invisalign Teen, Invisalign Assist, Invisalign Express 10, Invisalign Express 5	Align Technology, Inc.	Number of aligners dependent on treatment; simultaneous tooth movement of all teeth; SmartForce features (attachments, torque, bite opening)	Range from minor anterior tooth movement to complex treatment of all teeth	SmartTrack: Multilayer polyurethane and copolyester proprietary material (released in 2013)
Insignia: Clearguide Express	Ormco AOA	Up to 10 aligners/arch Move tooth up to 2.5 mm/tooth (0.25 mm/aligner) Monitor movement with "Heat N Bite"	Alignment of U/L anterior teeth	No information
Vivid Aligners	ODL	Sets of 3–5 aligners ; move tooth up to 0.5 mm/tooth		Zendura: rigid polyurethane
ClearCorrect	ClearCorrect	Aligners created in phases; models included to fabricate replacement aligners	Range from minor anterior tooth movement to treatment of all teeth	Zendura: rigid polyurethane
Minor Alignment Correction	Digi3DWorks	Return model with teeth moved for aligner to be made in office; will make aligner if desired	Alignment of U/L anterior teeth	Raintree Essix ACE: Copolyester proprietary
Clarus Clear Aligners	Clarus (Egypt)	Use attachments, buttons, and elastics Standard aligner: 4 aligners/mo, each worn for 1 wk, movement 0.5–0.7 mm/mo Smart aligner: 2 aligners/mo, each worn for 2 wks, movement 1.0 mm/mo	Range from minor anterior tooth movement to treatment of all teeth	Standard clear aligner material unknown; Smart clear aligner material unknown
AIRAligner	Nivol (Italy)	AIR One (one arch treatment); AIR Light (treatment of both arches <22 aligners); AIR Complete of (treatment both arches >22 aligners)	Move all teeth	No information
TwinAlignerSystem	Orthocaps (Germany)	Two aligners: hardCAPS for day wear, softCAPS for night wear	Move all teeth	hardCAPS, softCAPS: no information
In-LineClear Aligners	In-Line Orthodontic UK Ltd.	Twin layer laminate aligner; moves teeth up to 0.6 mm/aligner; aligner wear for 4–6 wks; IPR performed for anterior crowding	Minor to moderate U/L anterior crowding or spacing	No information
Clear aligners	NimroDENTAL Orthodontic Solutions (UK)	Few aligners	Minor U/L anterior crowding	No information
ClearPath	ClearPath Orthodontics (India)		Mild and moderate cases	No information
Clear aligners	3D Ortholine (UAE)		Move all teeth	Raintree Essix
iROK–CAT	iROK (China)	Aligners shipped in sets of four	Move all teeth	No information

AOA, Allesee Orthodontic Appliances; *IPR,* interproximal reduction; *U/L,* upper and lower; *3D,* three dimensional.

enhance areas of undercut either for retention or to facilitate specific movements. (Attachment design and placement are also discussed in greater detail later in this chapter.)

SCANNING AND DIGITIZATION

The unique combination of computerized virtual treatment planning and the first of its kind use of stereolithographic rapid prototyping technology for mass custom manufacturing gave Invisalign a commanding leadership role in aligner therapy. Over the course of the first decade of custom manufactured aligner treatment, the treating physician was required to obtain highly accurate impressions that were then sent to the manufacturing entity where the impressions were converted into digital data using industrial three-dimensional radiography.

The advent of intraoral scanners has made the process of digitization simpler and has eliminated the need for a third party to convert an impression into a virtual model. An instant virtual model from an intraoral scanner, in combination with multiple software platforms, now allows the orthodontist to manipulate teeth with or without the assistance of a technician. That, along with a three-dimensional printer, allows the orthodontist to make aligners easily in his or her office once the cost of the three components (scanner, software, and three-dimensional printer) becomes more affordable.

Let's look at intraoral scanners as the first step in the process. Every current scanner has a hand-held wand containing a camera that is connected to a computer for data collection and manipulation. The wand may project either laser or white light onto the tooth surface where it is reflected back to the camera, after which hundreds of thousands of measurements per inch are performed to recreate the three-dimensional representation of the teeth.

All intraoral scanning technologies using a wand have had to deal with cross-arch distortion as the information is stitched together to form a full arch image. Cross-arch distortion has not been as much of an issue in restorative dentistry because full-arch restorations are rare. In orthodontics, cross-arch distortion

will result in orthodontic appliances that simply do not properly fit. Fortunately, the scanner manufacturers have managed to correct such distortions. The technology engineered into the wand, which captures the surface data, determines the measurement speed, resolution, and accuracy of the scanner. Four types of imaging technologies are currently used: (1) triangulation, (2) parallel confocal imaging, (3) accordion fringe interferometry, and (4) three-dimensional in-motion video. In addition, intraoral ultrasound scanning, although not yet commercially available, is currently in development and should soon be released. Let's review each imaging technology in order.

CEREC (**C**hairside **E**conomical **R**estoration of **E**sthetic **C**eramics or **CE**ramic **REC**onstruction) (Sirona USA, Charlotte, NC) was the first in-office intraoral scanner introduced to dentistry in the early 1980s. It was originally developed to scan a crown preparation and then to send the data to a milling machine to create single crowns in the office without the need for impressions. CEREC works using triangulation. This technique measures the angles and distances from known points (laser source and sensor) with projected laser light. This technology requires a thin coating of opaque powder to be applied to the target tissue. The CEREC system determines the angle of reflection and the distance from the laser source to the object's surface as light reflects off the object.

Parallel confocal imaging (iTero, Align Technology, Inc., Santa Clara, CA) projects laser light through a pinhole to the target. The sensor is placed at the imaging plane where it is in focus (confocal). A small opening in front of the sensor blocks any light from above or below. Only the focused light reflecting off the target tissue will reach the sensor for processing. This type of system creates thousands of tomographic slices and stitches them together to form the three-dimensional picture.

Two sources of light are used with accordion fringe interferometry (AFI) to project three patterns of light, called "fringe patterns," onto the teeth and tissue (True Definition Scanner, 3M ESPE, St. Paul, MN). Based on the shape of the object, the fringe pattern distorts and takes on a new pattern. Surface data points of the fringe curvature are recorded by a high-definition video camera that is offset from the projector by approximately 30 degrees. Because the differences among the three precision optical measurements determine the distance measurement, the differential measurement is unaffected by changes in tooth colors and materials.

Three-dimensional, in-motion video (Lythos Digital Impression Systems, Ormco Corporation, Orange, CA) uses three tiny high-definition video cameras at the lens to capture three precise views of the target. A sensor behind the cameras converts the light energy into electrical signals, which allows the distances between two data points to be calculated simultaneously from two perspectives to create the three-dimensional data. The data points are captured in a video sequence and modeled in real time. Although powdering may be required to capture surface data points, only a light dusting is needed, compared with the thicker coating needed for triangulation. The ClearView SCAN (S-Ray Incorporated, Reno, NV), uses high-definition ultrasound imaging to capture both the tooth and the bone and soft tissue in three dimensions. Early prototypes suggest that these scans will use a disposable mouthpiece to scan the entire arch at once and will be significantly faster and more accurate than light-based scans.

The second part of the process is integrating the software that provides the ability for the technician or orthodontist to manipulate teeth in preparation for manufacturing aligners. The software must accomplish two things. First, it must fill any voids in the scan itself to produce a workable model; second, it must allow for tooth segregation and three-dimensional manipulation to produce the intended final setup. Furthermore, the amount of movement per aligner or stage must be determined by either the orthodontist with a three-dimensional digital orthodontic system (Orchestrate Orthodontic Technologies, Rialto, CA) or be programmed into the software in the form of a proprietary algorithm (Invisalign). If the aligners are manufactured by a vendor, then the orthodontist simply orders them once the setup is complete, and the rest is taken care of by the vendor of his or her choice.

If the orthodontist should decide that he or she wants to manufacture his or her own aligners, then a three-dimensional printer is required to produce the models on which the aligners will be formed. At the time of this writing, the three-dimensional printers with adequate resolution for fabricating aligners are still too expensive for the average orthodontist ($40,000 to $50,000). However, similar to most technologic advancements, the cost for these printers continues to decrease; it is not unreasonable that, in the very near future, in-office three-dimensional printing will be routine. Having said that, reviewing the four types of three-dimensional printers currently available is appropriate.

All three-dimensional printers build the object (for orthodontics a model of the teeth) in layers. The height of the model and the thickness of each layer determine how long it takes to print. Objects may be printed out of a variety of substances, depending on the printer and the intended use of the object. In **stereolithography**, liquid resin is held in a build tray and cured layer by layer with an ultraviolet laser light that "draws" a cross-section or outline of each layer in a bottom-up sequence until the model is submerged in the resin bath by the thickness of the build layer for each pass of the laser. Align Technology uses **stereolithography (SLA) models** to manufacture Invisalign. **Fused deposit modeling (FDM)** lays down layers of material heated just beyond its melting point, and the material immediately hardens as each layer is applied. The material in FDM printing is generally held on a replaceable spool. **Digital light processing (DLP)** is based on a chip technology developed by Texas Instruments and is commonly used in home theater projectors. With DLP printers, the process is similar to SLA modeling; however, an entire layer is cured at once, resulting in faster build times and a smoother surface finish. **PolyJet photopolymerization (PPP) printers** are similar to inkjet printers, but PPP printers work in three dimensions rather than two. In PPP printers, the material is sprayed out of nozzles and immediately cured with ultraviolet light. With some models, the build layers can be quite thin, resulting in minimal surface stratification. Similar to all technology, three-dimensional printers require routine maintenance for proper operation, as well as the selection of proper materials, the cost of which varies among both the manufacturer and the type of printer.

It would be remiss not to mention four-dimensional printing, the brainchild of Dr. Skylar Tibbits at Massachusetts Institute of Technology (MIT). Four-dimensional printing involves using three-dimensional printing and special geometric properties along with material characteristics to print objects that can change their configuration based on outside influences, such as motion or pressure. Imagine an aligner that creates its own dimple by increasing the pressure in a specific place on

the tooth if the tooth failed to respond as anticipated. This feature is only one aspect of four-dimensional printing that may assist orthodontists in improving efficiency and outcomes with aligner treatment.

DIAGNOSIS AND TREATMENT PLANNING FOR CLEAR ALIGNER TREATMENT

Diagnosis and treatment planning are the cornerstones for successful outcomes in orthodontic treatment. As described by Proffit in *Contemporary Orthodontics*,[7] the clinical examination, health history, and evaluation of quality diagnostic records lead to the development of a prioritized problem list. This problem list is further synthesized to produce realistic treatment objectives to correct the individual problems. The individual treatment objectives are then sequentially prioritized to form the optimal treatment plan. Specific mechanotherapy or force systems are then selected to achieve the treatment goals. The choice of one particular appliance or force system over another is a personal preference and infers that often more than one specific treatment plan is capable of producing the desired results.

As reported by Owens and colleagues[8] in 2006, the American Board of Orthodontics (ABO) developed the Case Management Form (CMF), which provides an objective tool to establish definitive treatment goals for the three major areas in the orthodontic treatment plan—skeletal analysis, dental analysis, and facial analysis. Based on the analysis of pretreatment records, the candidate records definitive therapeutic treatment goals for each of the three areas in three planes of space—vertical, sagittal, and transverse. The CMF defines the treatment goals for each of the tissues that can be altered during the orthodontic treatment. Treatment modalities (mechanotherapy or force systems) have the potential to affect each of the tissues. Some forms of mechanotherapy are specifically targeted to correct skeletal structures, such as a Hyrax appliance for the transverse correction of a narrow maxilla and a Herbst appliance designed to affect both the skeletal and the dental components in the patient with a deficient mandible. Clear aligner therapy provides a specifically controlled force system that primarily affects the maxillary and mandibular dentition in all three planes of space. Growing patients wearing aligners have the potential to have a small effect on the maxilla and the mandible, whereas in adults, the effect will be negligible.

Treatment planning, when considering clear aligner treatment using Invisalign-branded clear aligners, combines the process introduced by Proffit, Owens, and others. First, the determination should be made that clear aligner treatment is the appropriate force system, based on the treatment goals derived from the prioritized treatment objectives. More to the point, clear aligners are tooth-moving appliances that have very minimal impact on skeletal structures. Therefore clear aligners would not be the appropriate force system for transverse skeletal problems or for severe sagittal problems attributable to poorly aligned skeletal structures. Clear aligners are well suited and appropriate when the treatment goals can be achieved by tooth movement in all three planes of space. Examples are numerous and include all sagittal Angle molar classifications, with mild to moderate corrections in the transverse and vertical planes. Understanding the capabilities and limitations of moving teeth with clear aligners is essential to determining when their use is appropriate. The second part of treatment planning begins when the prescription form is submitted to Align Technology. The Invisalign prescription walks through a list of treatment objectives, and the orthodontist indicates how the treatment objectives should be achieved in the virtual setup. Having specific goals for the amount and direction of desired tooth movement in all three planes of space helps the orthodontist guide the technician to provide the initial setup that reflects the desired treatment result.

CLINCHECK—AN OVERVIEW

The virtual setup for Invisalign-branded clear aligner treatment is viewed by the orthodontist in a software program called ClinCheck. ClinCheck is not *the* treatment plan; rather, it is the three-dimensional interpretation of the treatment plan by the technician from the prescription form submitted online. The goal of the virtual setup, when modified and completed by the orthodontist, is to make the individual tooth movements shown in the ClinCheck depict the actual individual tooth movements required to correct the malocclusion. Strict attention to each crown and to the root movement in all three planes of space is required. A common misconception is that the technician providing the initial setup has diagnosed the patient's malocclusion and has sent back a ClinCheck treatment plan that is ready for approval. The technician is neither qualified nor responsible for delivering a treatment plan for the patient. The technician's responsibility is to provide a virtual set-up that (a) follows the instructions on the prescription form and (b) delivers tooth movements that fall within the predetermined defaults of Invisalign's Treat software. Although technicians receive training in basic occlusion principles and are well versed in orthodontic terminology, they do not have benefit of orthodontic training that incorporates biological principles of tooth movement, including concepts such as anchorage, reciprocal forces, and biomechanics. The technician uses a very robust proprietary software called Treat, and the orthodontist's interface with that program is a scaled-down version called ClinCheck. Treat software, although robust, is not a substitute for the didactic training received by an orthodontist in a postgraduate residency program.

Once the orthodontist has completed the virtual setup, the true power of the Treat program is exhibited by the application of SmartForce enhancements. SmartForce features are patented engineered solutions designed to create precise biomechanical forces on selected teeth or groups of teeth. Research and development of the SmartForce features, combined with the proprietary plastic, are unique to Invisalign. (SmartForce features are listed in Table 26-5.) These enhancements generally consist of customized computer-designed attachments added to the tooth surface or pressure areas applied to the tooth surface created by altering the digital model before fabricating the aligner. These features are discussed in further detail when discussing the biomechanics of tooth movement. Noting that SmartForce features are specific only to Invisalign-branded clear aligners is important. The treating orthodontist will enhance the predictability of treatment outcomes by gaining a thorough understanding of all the SmartForce features and the defaults that cause them to be applied to the virtual setup.

ClinCheck is also a proprietary software program unique to Invisalign-branded clear aligners. The treating orthodontist uses ClinCheck software to review the initial setup provided by the technician and to make modifications to the virtual setup until the desired tooth movements are displayed, at which time

TABLE 26-5 Invisalign SmartForce Features

		Name	Introduced
Optimized Attachments			
Extrusion	Maxillary centrals, laterals, cuspids		2008
	Mandibular centrals, laterals, cuspids		
	Mandibular bicuspids*	G5	2014
Rotation	Maxillary cuspids, bicuspids		2009
	Mandibular cuspids, bicuspids		
Extrusion/Root and Crown Tip**	Maxillary lateral incisors	Multi-plane	2011
Root Tip**	Maxillary centrals, cuspids, bicuspids	G3	2011
	Mandibular cuspids, bicuspids		
Pressure Points and Areas			
Lingual root torque	Maxillary centrals, laterals	Power Ridges	2007
	Mandibular centrals, laterals	Power Ridges	2008
Intrusion	Maxillary centrals, laterals	G5	2014
	Mandibular centrals, laterals, cuspids		
En Masse Features			
Anterior open bite closure	Maxillary incisors	G4	2012
Arch leveling: intrusion and extrusion	Maxillary incisors	G5	2014
	Mandibular incisors, cuspids		
	Maxillary bicuspids	G5	2014
	Mandibular bicuspids		
Extraction space closure	Maxillary molars, bicuspids, cuspids	G6	2015
	Mandibular molars, bicuspids, cuspids		

*Part of the intrusion en masse feature.
**Attachments may be combined with pressure points.

the treatment is approved and the aligners are manufactured. ClinCheck Pro (v4.1) is the most recent version of the software. ClinCheck Pro allows the orthodontist to control individual tooth movements in all three planes of space. In addition, arch shape can be changed by expanding or narrowing the posterior segments. All changes made by the orthodontist appear in real time with immediate adjustments automatically performed to adjacent teeth and arch form. All communications between the orthodontist and the technician are displayed on the right of the ClinCheck homepage and are listed in chronological order from the newest to the oldest, enabling all previous communications to be reviewed at any time (Fig. 26-1). ClinCheck Pro, with its ability to control tooth and arch movement, is a paradigm shift in the way orthodontists now achieve the desired virtual setup. Before the release of ClinCheck Pro (v3.1) in 2014, written communication with the technician was required to make modifications to the virtual treatment plan and often presented a barrier to getting the exact setup desired by the orthodontist. Another problem with previous versions was the inability to see the values for each tooth movement. The orthodontist had limited tools to assess the amount of tooth movement—a superimposition tool and a millimeter grid. Although these tools were helpful, they were inadequate to evaluate small changes in all three planes of space. The process of requesting changes and waiting to see the results was time consuming and often required multiple modifications before the treatment was accepted. With the introduction of ClinCheck Pro (v3.1) and subsequent updates in ClinCheck Pro (v4.1), the value of tooth movement measured to within hundredths of a millimeter are displayed on the bottom of the screen (Fig. 26-2). Orthodontists can now couple the proposed tooth movements in their treatment plans to the virtual setup and verify that all tooth movements are accurate in all three planes of space. The orthodontist's control of tooth movement not only increases the accuracy of the final positioning of the teeth, but it also saves considerable time by requiring fewer modifications.

BIOMECHANICS OF ALIGNER TREATMENT

Control of root position and anchorage is often the greatest challenge faced by any orthodontist. The question then becomes: Is treatment with Invisalign a practical alternative to fixed appliances? Several authors have examined the outcomes of Invisalign treatments. Patel and colleagues[9] found significant improvement in the peer assessment rating (PAR) index in patients treated with Invisalign. Vincent[10] found improvements in the ABO objective grading system (OGS) with tooth alignment but not in posterior occlusal contacts. Djeu and colleagues[11] compared Invisalign with fixed appliances and found the ABO OGS scores were improved more for the fixed appliance group than for the aligner group, whereas Brown and others[12] reported that, overall, Invisalign was found to be more effective than fixed appliances at producing the outcome defined by the OGS. In a systematic review in 2005, Lagravere and Flores-Mir[13] found the literature lacking and concluded that "Clinicians will have to rely on their Invisalign clinical experience, the opinions of experts and the limited published evidence when using Invisalign appliances." After the following section on biomechanics and Invisalign, the authors of this text hope the reader will be better equipped to make sound clinical decisions and have a greater understanding of the strengths and weaknesses of aligners.

One question to keep in mind during this entire discussion of biomechanics is the following: If teeth were capable of cognition, would they know what was applying the force? In 1999, Sims[14] predicted the future of orthodontics would include the abolition of bracket systems. In his 1986 textbook *Contemporary Orthodontics*,[15] Proffit stated that "adult patients traditionally have been somewhat reluctant to wear obvious fixed appliances and frequently indicate their preference for a removable appliance." He also described the characteristics required of an orthodontic appliance system. "No matter what the type of orthodontic appliance, it must meet certain basic design criteria: it (1) should not interfere with function; (2) should cause no harm to the oral tissues or interfere with the maintenance of good oral hygiene; (3) should be as light and inconspicuous as possible, yet sufficiently strong to withstand masticatory forces and a reasonable amount of abuse; (4) must be firmly retained in position; (5) must be capable of exerting an appropriately controlled force in the correct direction and delivering this force

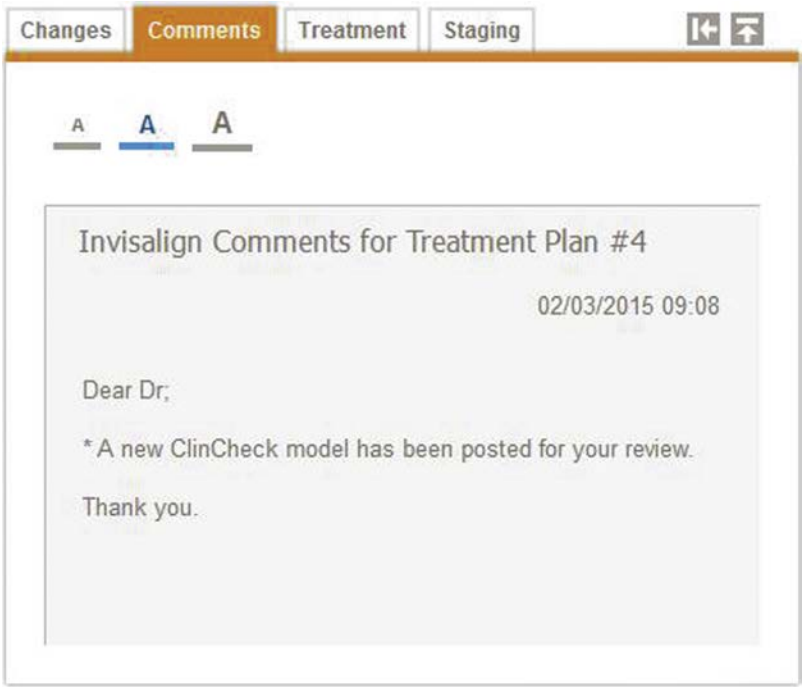


FIGURE 26-1 ClinCheck comments. (Courtesy of Align Technology, Inc.)

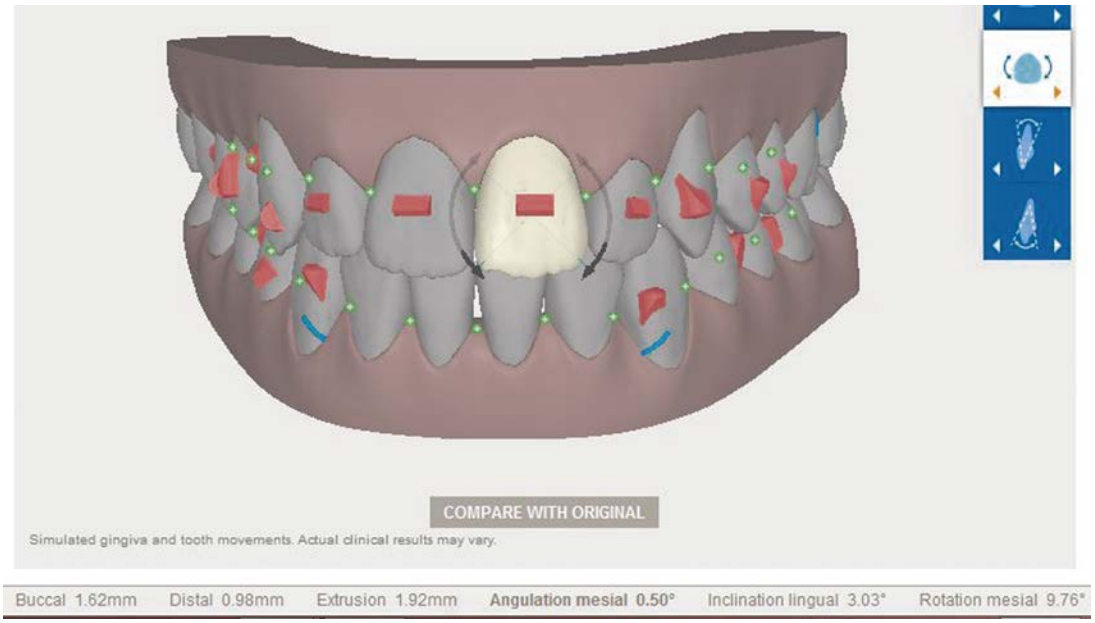


FIGURE 26-2 ClinCheck values of tooth movement. (Courtesy of Align Technology, Inc.)

for as long as possible between adjustment visits; and (6) should allow control of anchorage so that tooth movements other than those intended are minimized." On the surface, it would appear that Invisalign satisfies all of these criteria.

According to Proffit,[15] "removable appliances by their very nature produce simple tipping movements of teeth, making control of tooth position extremely difficult" and "as a practical matter, it can be difficult to maintain removable appliances in place against the displacing effects" of the forces required to produce controlled root movement. He concluded that the usual solution to this problem was to use fixed appliances. When describing the experiences with Invisalign at the University of the Pacific, Dugoni[16] wrote in 2002, "We then went on to patients in whom the mandibular incisors would have to be extracted to determine whether we could close those spaces in a parallel manner. This involved changes not only in the material but also in the technique. Ultimately, we were able to move the teeth bodily. Experimentation has continued with extraction cases to determine whether we could bodily move canines into first premolar positions after extractions." His conclusion was that "the use of the Aligners is far more complicated than most people believe. It takes a knowledgeable clinician with

considerable experience to use the appliance to its maximum. What that maximum is, I do not know."

To determine what the maximum might be with Invisalign in its current rendition of aligner materials, we must first examine the biomechanics of tooth movement with Invisalign.

Simple versus Difficult Movements

To examine the biomechanics of tooth movement with aligners, how aligners move teeth will first be described. With a typical fixed appliance, the wire is engaged in a bracket with the adhesive retaining the bracket on the tooth. The active archwire is elastically deformed and moves the tooth to a determined position as it returns to its original shape. With an aligner, the plastic encapsulates the tooth and, in doing so, must provide both retention and activation to move the teeth. In general, the natural undercuts of the teeth provide the retention and the active component to move teeth by the elastic deformation of the aligner, which is important for two reasons. First, the aligner elastic deformation cannot be as great as to overcome the retention forces; and second, certain directions allow the aligner a greater inherent ability to undergo elastic deformation. For instance, a faciolingual movement is fairly predictable because the entire body of the aligner can be elastically distorted; it then returns to its original shape, carrying the tooth with it. The total desired movement is then subdivided in such a way that the aligners remain within this range of elastic deformation, and a sequence of aligners is made to accomplish the entire desired movement. The number of aligners or stages, then, is based on the distance the tooth must be moved. In contrast, a vertical movement would require the aligner to stretch essentially within the matrix of the plastic and, at the same time, maintain retention of the tooth it is attempting to move. Because the ability for such elasticity within the plastic itself is very limited, these movements must be divided into very small increments and are considered difficult. Given this understanding of the basic nature of how aligners move teeth, it is not surprising that multiple movements are considered unpredictable with aligners. Some of these difficult movements include controlling torque, root parallelism, rotations, and extrusions. These issues are discussed within the context of traditional orthodontic biomechanics, specifically how aligners deal with forces and moments. The purpose of this chapter is not to provide a detailed review of orthodontic biomechanics; rather, its purpose is to discuss how aligners relate to these concepts.

The material composition of the aligner is important in delivering the desired properties to the aligner. Aligners are comprised of polyurethane, polyester, or some combination or modification of such (see Tables 26-1 through 26-4). Ideally, aligner plastic should be the appropriate stiffness to deliver a constant force. To accomplish this, the stress relaxation of the material needs to be low to ensure that the delivered force is constant. The material should be of high enough formability to enable it to conform precisely to teeth and to any attachments that may exist. Aligner plastic should be highly elastic; when it is stretched, it will return to the shape similar to an archwire should when activated. Last, the material should be highly aesthetic and comfortable for the patient to wear. Previous studies[17] have shown that aligners have less impact on the quality of life and less pain than orthodontic treatment with traditional braces. Research is currently being conducted in the area of aligner thermoplastics to examine the impact of aligner thickness[18] and the other force delivery properties of

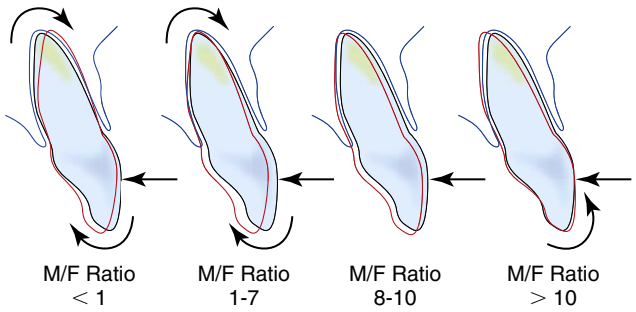


FIGURE 26-3 Effect of moment-to-force (M/F) ratios.

aligners.[19-22] It should be noted that the basis of current discussion using Invisalign is the proprietary plastic used to make the aligners known as SmartTrack (Align Technology, Inc.), a biocompatible multilayer thermoplastic polymer comprised of polyurethane and a copolyester, which was released in early 2013. A recent study[23] showed that the SmartTrack material achieved a significantly higher amount of prescribed tooth movement than the previous Align Technology's Exceed-30 aligner material that was used before 2013. Over the course of the past 20 years, the levels of force thought to be required to perform different movements has steadily decreased (see Table 26-1).

Continuing with that trend, recent studies have suggested that given time, even forces as low as 18*g* are sufficient to produce bodily movement.[24] Because the force delivered with an aligner made from Exceed 30 is initially 200*g* and decays to essentially a constant level of 40*g* within approximately 48 hours, delivering adequate forces to the teeth to create desired movements should pose no problem. Controlling those forces then becomes the issue.

How the force is delivered and the reaction of the tooth to that force are functions of multiple factors. These include the center of rotation, the center of resistance, and the point at which the force is applied.

The goal is to control root position during movement to achieve the desired end results with the minimum amount of complexity. Controlling the moment-to-force ratio can do this. Classic orthodontic biomechanics has described the effects of changing the moment-to-force ratio as illustrated in Figure 26-3.[25]

In traditional orthodontic biomechanics, the discussions are typically centered on fixed attachments in the form of brackets with forces applied by wires, with a small moment arm and relatively high forces required to meet moment-to-force ratios.

To better understand the dynamics of root control with aligners, the biomechanics of tooth movement with aligners are examined, and a comparison that, with an understanding of movement with fixed appliances, is made. Specifically, the design and placement of attachments and auxiliaries to accomplish controlled two-point force application are examined. In all of these discussions, it is important to understand that for effective tooth movement to take place, even in simple situations, aligners must be worn 22 hours a day, essentially the same as for fixed appliances.

One of the problems that is seen when attempting incisor root movements with aligners is that the intended movement and the actual movement are sometimes different. The reason for this difference is exactly what Proffit described as happening with removable appliances in general—not enough retention is

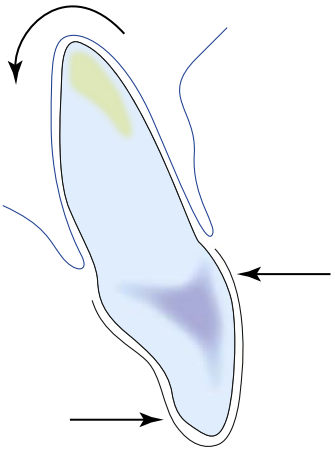


FIGURE 26-4 Theoretical moment-to-force ratio to achieve lingual root movement with an aligner.

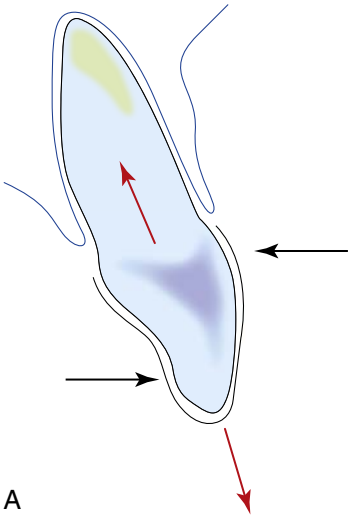


FIGURE 26-5 A, Diagram illustrating the clinical expression of moment-to-force ratio to achieve lingual root movement with an aligner. B, Photograph depicting the clinical expression of moment-to-force ratio to achieve lingual root movement with an aligner.

present to offset the force needed to generate the movement. The results are demonstrated in Figures 26-4 and 26-5, *A* and *B*.

STAGING OF TOOTH MOVEMENT IN CLINCHECK

An important aspect of controlling tooth movement with aligners is staging. *Staging* is the sequence in which and the speed at which teeth are moved with aligners. Figure 26-6 represents a classic staging diagram. As in the collision table,

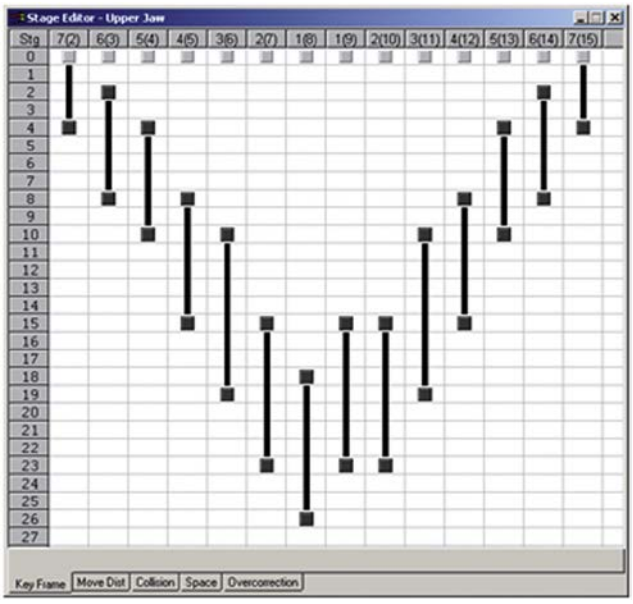


FIGURE 26-6 Segmented staging diagram.

the numbers across the top represent different teeth, and the vertical axis represents an aligner number. The difference is that the staging diagram is available to the orthodontist in ClinCheck. The vertical black bars in the diagram indicate the timing and rate of tooth movement. Each aligner number then represents one stage. The staging diagram has one shortcoming; that is, the rate of tooth movement is not illustrated. In other words, the staging diagram simply informs the orthodontist of whether the tooth is being moved. The orthodontist must then interpolate the rate of movement by estimating the total distance and dividing by the number of aligners, assuming equal rates of movement occur throughout the entire distance moved. In addition, the orthodontist has no way of knowing whether the movement represents linear movement or rotational movement. He or she only knows that the tooth is moving. The original default staging plans involved segmented tooth movements (see Fig. 26-6). The thought process was based on the classic notion of anchorage in which one group of teeth is held stationary while a smaller group of teeth is moved. Difficult movements were often left to the end of treatment and resulted in prolonging treatment time by adding multiple additional stages. To reduce excessive numbers of additional stages, some difficult movements were accelerated beyond a reasonable rate of movement. It sometimes became a self-fulfilling prophecy that difficult movements were unsuccessful with aligners. In addition, teeth that were supposedly held in place as an anchorage unit moved in the same manner that anchorage units move with fixed appliances.

An alternative to segmented staging that more closely mimics fixed appliance treatment is *simultaneous staging*. First suggested by Foy in 2004[26] and then refined by David Paquette (Staging Strategies, Effectiveness and Efficiency with Invisalign Treatment, 2005 Invisalign Summit, Las Vegas, NV) and then, after several years, the simultaneous movement concept was adopted by Align Technology in 2007 (Fig. 26-7). This standard simultaneous staging pattern is also referred to as *X staging pattern*. The basis for simultaneous movement is that all of the teeth within each arch are moved together from the initial stage through the final stage.

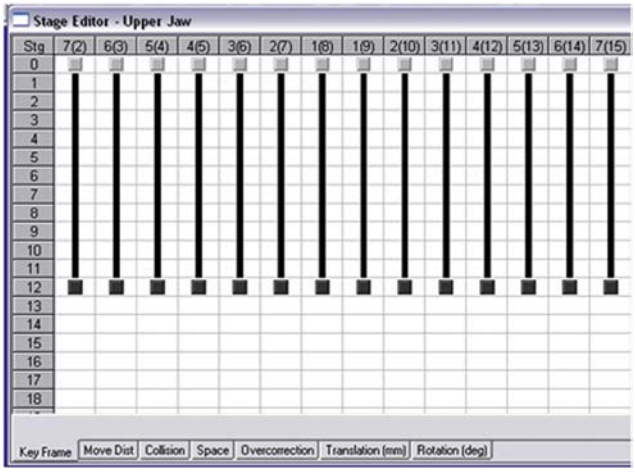


FIGURE 26-7 Simultaneous staging diagram.

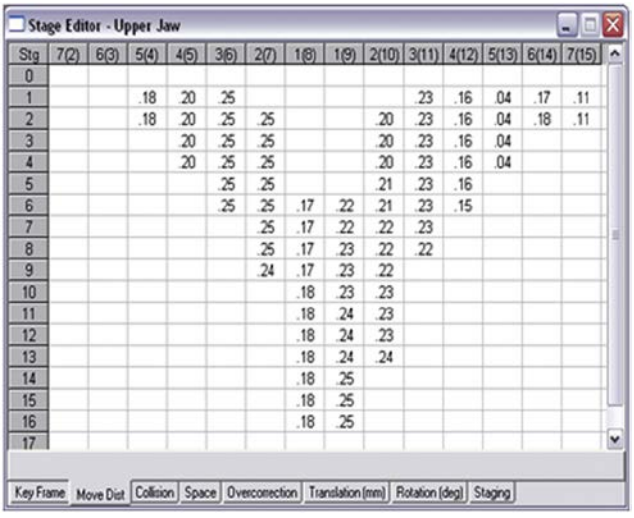


FIGURE 26-8 Segmented staging diagram linear movement.

The tooth that moves the most dictates the overall number of stages, based on the maximum allowable tooth velocity. Simultaneously moving the other teeth from the first to the last stage reduces the velocity for all the other movements and increases their predictability without increasing the overall number of aligners. A detailed examination of a series of screen captures from the Treat software provides the actual measures of intended velocities of tooth movement (Figs. 26-8 to 26-12). In Figure 26-8, all of the anterior teeth are moving at maximum velocity, 0.25 mm per stage. This velocity was established as the default for the maximum amount of tooth movement allowed per aligner in the first iteration of the Treat software and has not been changed up to the current version of the Treat software. This rate of linear velocity of movement per aligner has not been scientifically proven to be the correct amount of tooth movement in all three planes that can be achieved for each aligner change over time, but this rate of movement has, none- theless, persisted because empiric evidence suggests that it works on a large percentage of tooth movements.

In Figure 26-9, the upper left lateral was determined to be the rate-limiting tooth. When these movements were started from the beginning of treatment along with all of the other teeth, the result was a decrease from 16 stages to 12 stages, and all of the

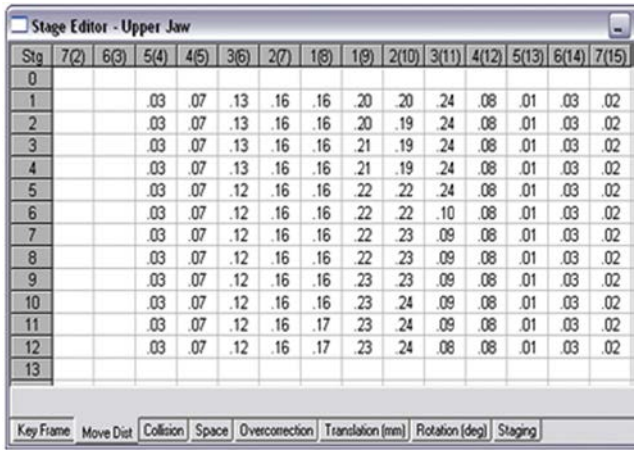


FIGURE 26-9 Simultaneous staging diagram linear movement.

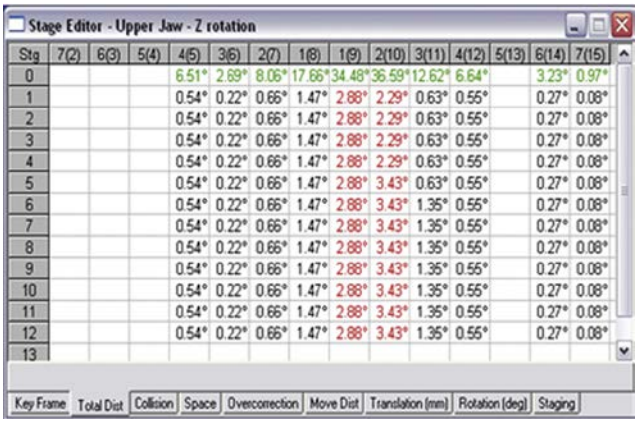


FIGURE 26-10 Simultaneous staging diagram rotational movement.

teeth except the rate-limiting tooth actually moved more slowly at each stage than previously.

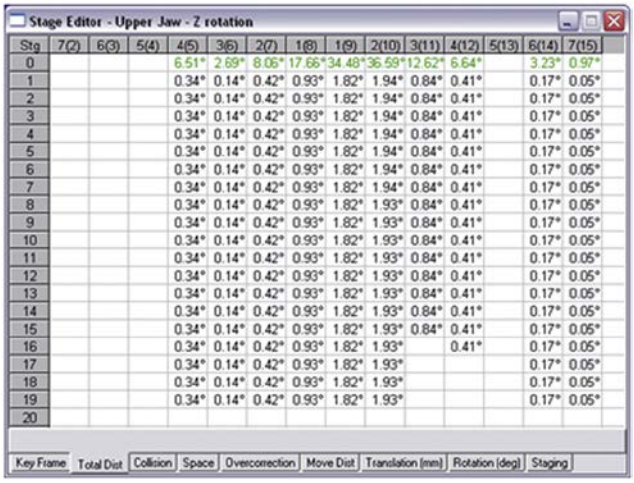
There are times when the linear velocity is not the rate limiting determinant; rather, the rotational velocity of tooth movement determines the minimum number of aligners for a given treatment sequence (see Figs. 26-9 and 26-10). In Figure 26-10, the linear movements were under the maximum velocity, resulting in the rotational velocity to be greater than desirable. To increase the predictability of treatment, rotational velocity will be limited to proceed at less than two degrees per stage (see Fig. 26-11). Two degrees is currently the standard default in the Treat software. When the rotational velocity is reduced to an acceptable limit, the number of stages once again increases to 19 stages.

The side effect of reducing the rotational velocity is to reduce further the linear velocity at the same time. The reduction of the velocity of linear movement of the upper left lateral from 0.24 mm per stage to 0.15 mm per stage is noted (see Figs. 26-8 to 26-12), and although the number of stages may increase slightly, the predictability of treatment outcomes is greatly improved.

Alternative staging patterns also exist that are associated with certain types of treatments and are briefly described. Distalization of the maxillary dentition, starting with the molars, followed by the bicuspids, and ending with the retraction of the anterior teeth, is a type of staging known as *V staging pattern* and typically applies only to the upper arch. Although this

pattern makes a great three-dimensional visualization of correcting a Class II Angle molar relationship to a Class I molar Angle relationship, the reality is that teeth that appear to be anchoring the arch are still subject to the forces applied by the aligner material. As previously mentioned, teeth that show no movement in a ClinCheck are still subject to forces that make any changes in the arch perimeter. Moving molars from a Class II relationship to a Class I relationship and then holding the teeth motionless, simulating maximum anchorage while the rest of the arch moves distally to a Class I relationship, is not something that will translate to the clinical setting. The other negative aspect of this staging pattern is the significant increase in overall treatment time.

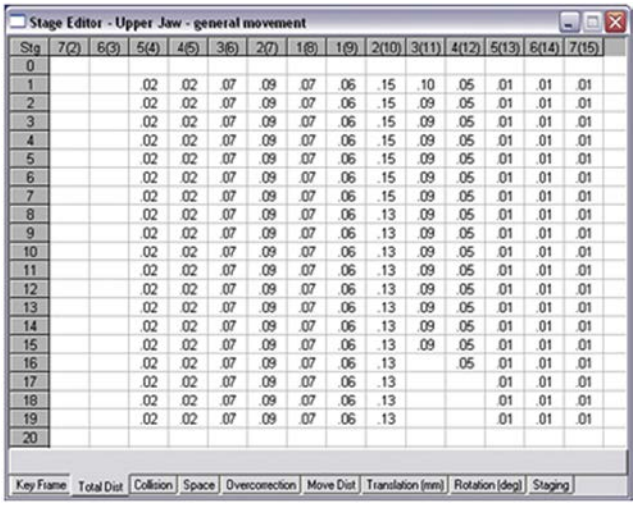
The opposite of V staging pattern, in which the anterior teeth move anteriorly, followed by posterior teeth moving anteriorly, is known as *A staging pattern*. This pattern could be used in either arch to open previously closed extraction spaces or to attempt to mesialize an entire arch in a segmental fashion. Again, this pattern will significantly lengthen the treatment time, as compared with X staging pattern staging, and has limited clinical applications.


Stage Editor - Upper Jaw - Z rotation

Stg	7(2)	6(3)	5(4)	4(5)	3(6)	2(7)	1(8)	1(9)	2(10)	3(11)	4(12)	5(13)	6(14)	7(15)
0				6.51°	2.69°	8.06°	17.66°	34.48°	36.59°	12.62°	6.64°		3.23°	0.97°
1				0.34°	0.14°	0.42°	0.93°	1.82°	1.94°	0.84°	0.41°		0.17°	0.05°
2				0.34°	0.14°	0.42°	0.93°	1.82°	1.94°	0.84°	0.41°		0.17°	0.05°
3				0.34°	0.14°	0.42°	0.93°	1.82°	1.94°	0.84°	0.41°		0.17°	0.05°
4				0.34°	0.14°	0.42°	0.93°	1.82°	1.94°	0.84°	0.41°		0.17°	0.05°
5				0.34°	0.14°	0.42°	0.93°	1.82°	1.94°	0.84°	0.41°		0.17°	0.05°
6				0.34°	0.14°	0.42°	0.93°	1.82°	1.94°	0.84°	0.41°		0.17°	0.05°
7				0.34°	0.14°	0.42°	0.93°	1.82°	1.94°	0.84°	0.41°		0.17°	0.05°
8				0.34°	0.14°	0.42°	0.93°	1.82°	1.93°	0.84°	0.41°		0.17°	0.05°
9				0.34°	0.14°	0.42°	0.93°	1.82°	1.93°	0.84°	0.41°		0.17°	0.05°
10				0.34°	0.14°	0.42°	0.93°	1.82°	1.93°	0.84°	0.41°		0.17°	0.05°
11				0.34°	0.14°	0.42°	0.93°	1.82°	1.93°	0.84°	0.41°		0.17°	0.05°
12				0.34°	0.14°	0.42°	0.93°	1.82°	1.93°	0.84°	0.41°		0.17°	0.05°
13				0.34°	0.14°	0.42°	0.93°	1.82°	1.93°	0.84°	0.41°		0.17°	0.05°
14				0.34°	0.14°	0.42°	0.93°	1.82°	1.93°	0.84°	0.41°		0.17°	0.05°
15				0.34°	0.14°	0.42°	0.93°	1.82°	1.93°	0.84°	0.41°		0.17°	0.05°
16				0.34°	0.14°	0.42°	0.93°	1.82°	1.93°		0.41°		0.17°	0.05°
17				0.34°	0.14°	0.42°	0.93°	1.82°	1.93°				0.17°	0.05°
18				0.34°	0.14°	0.42°	0.93°	1.82°	1.93°				0.17°	0.05°
19				0.34°	0.14°	0.42°	0.93°	1.82°	1.93°				0.17°	0.05°
20														

Key Frame | Total Dist | Collision | Space | Overcorrection | Move Dist | Translation (mm) | Rotation (deg) | Staging

FIGURE 26-11 Simultaneous staging diagram reduced rate of rotational movement.


Stage Editor - Upper Jaw - general movement

Stg	7(2)	6(3)	5(4)	4(5)	3(6)	2(7)	1(8)	1(9)	2(10)	3(11)	4(12)	5(13)	6(14)	7(15)
0														
1			.02	.02	.07	.09	.07	.06	.15	.10	.05	.01	.01	.01
2			.02	.02	.07	.09	.07	.06	.15	.09	.05	.01	.01	.01
3			.02	.02	.07	.09	.07	.06	.15	.09	.05	.01	.01	.01
4			.02	.02	.07	.09	.07	.06	.15	.09	.05	.01	.01	.01
5			.02	.02	.07	.09	.07	.06	.15	.09	.05	.01	.01	.01
6			.02	.02	.07	.09	.07	.06	.15	.09	.05	.01	.01	.01
7			.02	.02	.07	.09	.07	.06	.15	.09	.05	.01	.01	.01
8			.02	.02	.07	.09	.07	.06	.13	.09	.05	.01	.01	.01
9			.02	.02	.07	.09	.07	.06	.13	.09	.05	.01	.01	.01
10			.02	.02	.07	.09	.07	.06	.13	.09	.05	.01	.01	.01
11			.02	.02	.07	.09	.07	.06	.13	.09	.05	.01	.01	.01
12			.02	.02	.07	.09	.07	.06	.13	.09	.05	.01	.01	.01
13			.02	.02	.07	.09	.07	.06	.13	.09	.05	.01	.01	.01
14			.02	.02	.07	.09	.07	.06	.13	.09	.05	.01	.01	.01
15			.02	.02	.07	.09	.07	.06	.13	.09	.05	.01	.01	.01
16			.02	.02	.07	.09	.07	.06	.13		.05	.01	.01	.01
17			.02	.02	.07	.09	.07	.06	.13			.01	.01	.01
18			.02	.02	.07	.09	.07	.06	.13			.01	.01	.01
19			.02	.02	.07	.09	.07	.06	.13			.01	.01	.01
20														

Key Frame | Total Dist | Collision | Space | Overcorrection | Move Dist | Translation (mm) | Rotation (deg) | Staging

FIGURE 26-12 Segmented staging diagram reduced rate of linear movement.

M staging pattern is solely used for bicuspid extraction treatment. In this staging pattern, movement starts by first closing the extraction spaces, followed by the alignment of anterior teeth, and finishing with molar movement. This three-dimensional simulation attempts to mimic maximum anchorage of the molars by showing no movement as the remainder of the arch changes. Again, this simulation does not clinically and exactly translate as shown in the three-dimensional simulation because forces are being applied to the molars as the arch perimeter changes with each successive aligner. In response to the need to create a solution for the extraction treatment, which specifically addresses molar anchorage and root parallelism, G6, the latest SmartForce feature has been introduced. G6 combines SmartForce optimized attachments along with a specific staging pattern that is similar to the M staging pattern. To date, clinical results have not been evaluated to determine the clinical efficacy of G6 SmartForce enhancements.

Although significant clinical testing has gone into the development of staging patterns, understanding that the orthodontist can always request custom staging is important when he or she feels it is necessary to improve treatment outcomes. Ultimately, the orthodontist controls all aspects of the Invisalign treatment, including how much movement per aligner is desired. Although not recommended by Align Technology, some orthodontists greatly reduce the amount of linear movement per stage by increasing the number of active stage aligners using simultaneous tooth movement or X pattern staging for complex movements. Increasing the number of active stages with simultaneous tooth movement effectively reduces the amount of tooth movement per stage for all teeth in the arch. If the number of active stages were doubled, then the movement of the rate-determining tooth would decrease to nearly 0.12 mm per stage, and all of the other teeth in the arch would be significantly less than 0.12 mm linear movement per aligner. Reducing tooth movement per aligner then allows an accelerated changing schedule because of less elastic deformation of the aligner material. Many orthodontists who increase the number of active stage aligners deliver fresh aligners on a weekly basis to have the aligners provide a more consistent force, with less force diminution between aligner changes. Although twice as many aligners are the result, the total treatment time remains the same.

ATTACHMENT DESIGN IN THE INVISALIGN SYSTEM

One solution to aligner displacement is the proper design and placement of attachments. Attachments can be used for the retention of the aligner, as well as to enhance or facilitate specific tooth movements. Figure 26-13 illustrates the evolution of attachments used to help eliminate this problem. The goal is to provide a ledge for the aligner to grip that is perpendicular to the direction of displacement and of sufficient size to provide enough surface area to offset the force delivered. Another simple rule of thumb is to place the attachment far enough away from the gingival margin that the aligner will not spread or stretch and slip off the attachment. This concept is important because the aligners tend to relax over time; that is, they exert less force. The clinically observed side effect is that the gingival third tends to become less retentive, which is in contrast to the findings of Jones and colleagues,[27] based on laboratory results with in-office–fabricated aligners whose properties were not affected by the oral environment.

Movements that are termed difficult require a more sophisticated approach to attachment design than was used in the past. Recognizing the limitation of aligners and empirically designed attachments to accomplish certain tooth movements, engineers at Align Technology initiated efforts to design a better aligner and attachment system. Align Technology thus developed the Virtual Invisalign Laboratory, which is a sophisticated series of software tools that enable the evaluation of the expected clinical response to various attachment designs and placements. The approach, based on the principles of biomechanics, has three parts: virtual modeling, in vitro testing, and clinical evaluation of the resulting designs. Using this approach, the probability of achieving the movement displayed in the three-dimensional ClinCheck treatment is greatly enhanced.

Virtual modeling is first used to test a myriad of possible solutions and then to identify those that produce the desired force system. These models may include changes in attachment shape, as well as variations in the geometry of the aligner itself (Fig. 26-14, *A–C*). After considering possible designs, the models are then fabricated, and the force systems are measured using laboratory equipment specifically designed to measure force systems from aligner and attachment combinations. Successful designs are then moved into clinical testing.

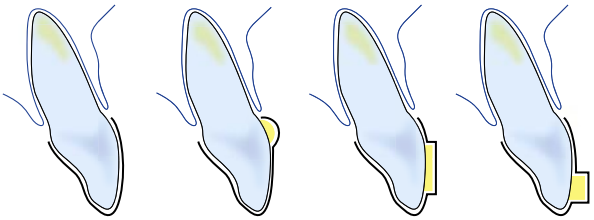


FIGURE 26-13 Evolution of attachments.

At the time of this printing, a significant number of *optimized* attachments have been developed and introduced to provide specific force systems for challenging tooth movements. These optimized attachments allow the proprietary aligner material (SmartTrack) to produce the required force, which creates the moment required to move the tooth as shown in the ClinCheck. The term *SmartForce* has been patented by Align Technology to describe the computer-generated attachment designs that are generated by Align Technology's Treat software. Each optimized attachment is now custom designed for a specific movement on a specific tooth for an individual patient. The optimized attachments are automatically generated by the Treat software. The orthodontist is responsible for moving each individual tooth to the desired position using ClinCheck Pro. The tooth movements prescribed by the orthodontist are measured in all three planes of space; when a default in any one direction is exceeded, the Treat software will place the SmartForce attachment on the tooth at the correct location to create the required force system to cause the tooth to move as depicted in the ClinCheck. Each SmartForce attachment feature has a default that must be exceeded to place the optimized attachment, and the technician cannot override the software to have these specific attachment designs placed on the teeth. As a result, it is incumbent for the orthodontist to understand each SmartForce feature and what movement is required to have the optimized attachment placed.

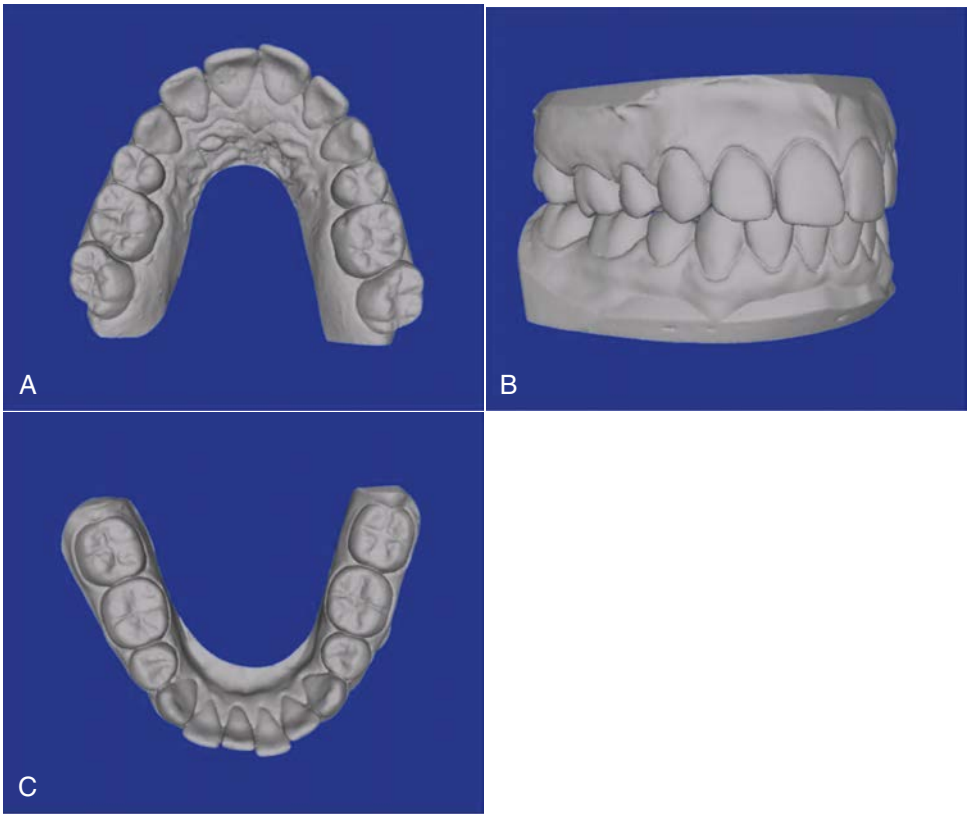


FIGURE 26-14 A–C, Virtual models generated from computer tomographic (CT) scans of polyvinyl siloxane (PVS) impressions.

One internal design feature from Align Technology allows computer-designed attachments to provide specific directions of force application upon inserting the first aligner. The initial force application applied to optimized attachments is generated by preactivating the aligner-attachment interface (see Fig. 26-14). Preactivation is achieved by using an attachment template for attachment placement, which will alter only the optimized attachments that are slightly offset from the position of the attachment in the subsequent aligner that will be placed. This offset positions the active surface of the attachment to engage fully the aligner material, thus eliminating a lag period of one to two aligners before achieving the full force system required to move the tooth as shown in the ClinCheck. In addition to preactivation, one additional feature has been designed to allow tooth movement using optimized attachments. Some optimized attachments will have excess space opposite the active surface to allow the tooth to move unimpeded in the correct direction. Although, clinically, this may give the appearance that the attachment is not fully seated in the aligner, this excess space is intentional by design.

As the number of optimized attachments has increased, it has become apparent that the ability to move teeth effectively with clear aligners is a function of the material, the velocity of tooth movement, and the ability to produce the correct force system by using computer-designed attachments and, last but not least, patient compliance.

Another group of attachments are available to be placed by the orthodontist when modifying a virtual treatment plan. These attachments, referred to as manually placed or physician-prescribed attachments, are those previously referred to in the section on ClinCheck. After turning the three-dimensional controls ON and selecting ATTACH & CUTS from the vertical dropdown list, the choice of attachments is visible. The design of these attachments was completely empiric and has evolved since the initial inception of the attachments. The first of the manually placed attachments was the ellipsoid attachment. This attachment is widely considered the least effective attachment because of its small size and lack of a defined active surface. All other attachments are variations of the initial rectangular attachment design. This design was the primary attachment for any tooth movement that was considered moderate or difficult, without deference to the actual forces and moments generated by its placement. The patient has difficulty inserting and removing the aligners, which is an inherent problem with rectangular attachments. If the attachment and the aligner are not completely coupled, then the result is an unwanted force system and unpredictable tooth movements (Figs. 26-15 and 26-16).

To facilitate greater ease of insertion and removal, as well as to eliminate the all-or-none situation, the beveled attachment was developed by rotating a portion of the rectangular attachment virtually into the tooth surface[28] (Figs. 26-17 and 26-18).

The beveled attachment can be used in multiple orientations by simply rotating the attachment in a different manner. The latest version of ClinCheck Pro (v4.1) allows the orthodontist to not only place and remove the manual attachments but also to orient the manually placed attachments by rotating, raising, or altering the bevel. Although the available attachments to be placed manually show a different orientation (e.g., vertical and horizontal), the ability for the orthodontist to alter the attachment location, orientation, and height makes all these choices duplicitous. Essentially, only two choices—ellipsoid or rectangular—are now available. The orthodontist can now make one rectangular attachment be any type of rectangular attachment he

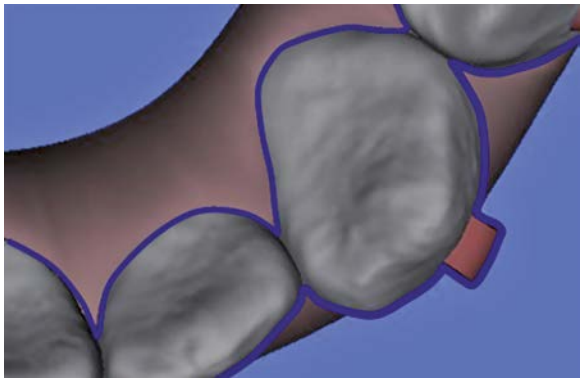


FIGURE 26-15 Aligner and rectangular attachment properly coupled.

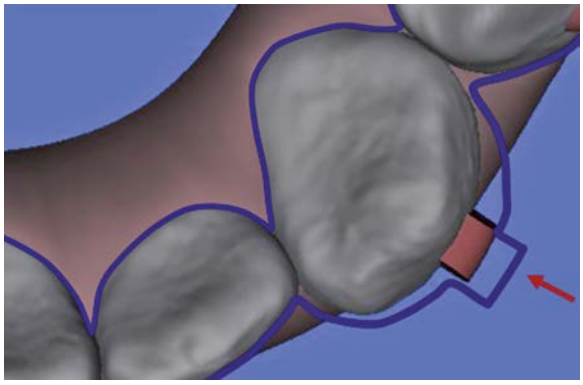


FIGURE 26-16 Aligner and rectangular attachment improperly coupled.

FIGURE 26-17 Beveled attachment.

FIGURE 26-18 Clinical appearance of the beveled attachment.

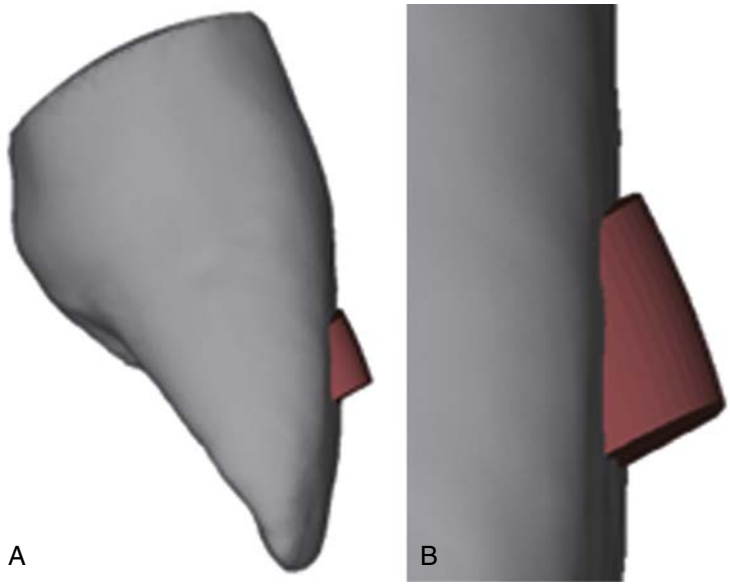


FIGURE 26-19 A, Gingivally beveled attachment. B, Gingivally beveled attachment close-up.

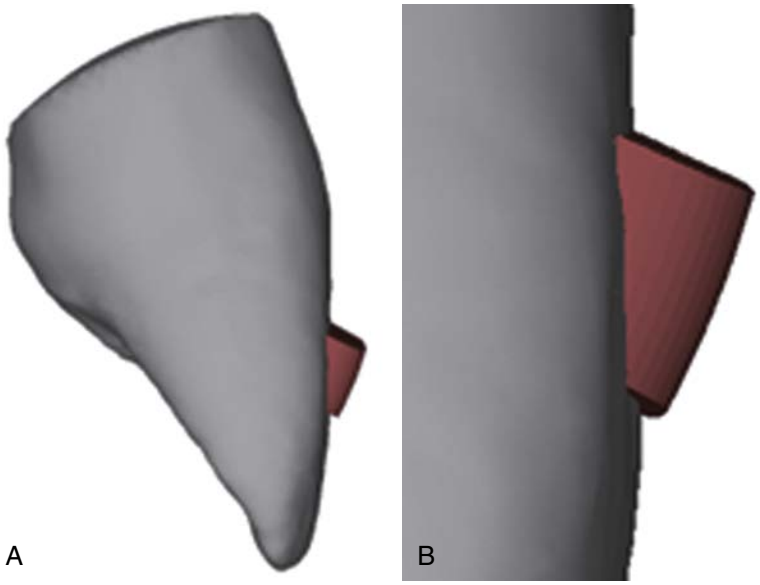


FIGURE 26-20 A, Occlusally beveled attachment. B, Occlusally beveled attachment close-up.

or she prefers—horizontal or vertical, bevel incisal or gingival. In addition, once the rectangular attachment is placed on the tooth, the length of the attachment can be selected by right clicking on the attachment, which will activate a pop-up list to select a 3-mm, 4-mm, or 5-mm attachment or to remove the attachment. In this way, attachments can be sized according to the orthodontist's preferences and can be used in any location that will enhance retention or movement. Theories suggest that rotating the bevel in specific directions will enhance specific movements.[29] Ongoing research will determine the effectiveness of such orientations. (Examples are given in Figs. 26-19 and 26-20.)

POWER RIDGES AND PRESSURE AREAS IN THE INVISALIGN SYSTEM

A net force of 40*g* (base level force of an aligner after 48 hours) intended to move the tooth lingually would require a moment of 320 to 400*g*/mm (moment-to-force [M/F] ratio 8 to 10) for bodily movement or greater than 400*g*/mm (force-to-moment [F/M] ratio less than 10) for lingual root movement (Figs. 26-21 and 26-22). Improper attachment design or placement allows the delivery of only 280*g*/mm moment in conjunction with 40*g* force, resulting in controlled lingual crown tipping (Fig. 26-23). Although the forces may be in opposite directions, the aligner provides the same level of force on both sides of the teeth. In the absence of spaces to close, just as with fixed appliances, some outside force system such as interarch elastics must be present to provide a net distalizing force on the maxillary anterior teeth to produce lingual root movement.

An alternative to attachments that help facilitate torque control is the SmartForce feature known as a *power ridge*. Similar to optimized attachments, power ridges are engineered corrugations placed at specific locations to enhance the undercut near the labial gingival margin of teeth undergoing torquing movements. The power ridges function in two ways: (1) to stiffen the gingival third of the aligner to make it more resilient; and (2) to

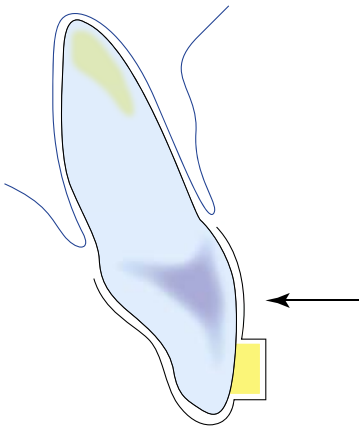


FIGURE 26-21 Force applied by aligner on facial surface.

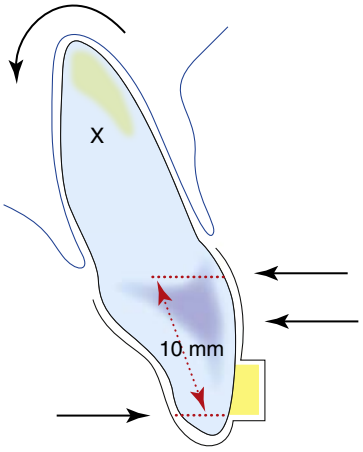


FIGURE 26-22 Force-to-moment diagram with attachment in incisal third of tooth.

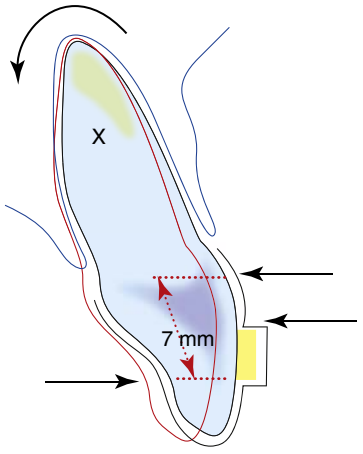


FIGURE 26-23 Force-to-moment diagram with attachment in middle third of tooth.

provide additional force as close to the gingival margin as possible to increase the effective moment arm of the aligner. The obvious advantage to power ridges is that attachments need not be placed or removed, and they are more aesthetically acceptable to the patient (Fig. 26-24). However, power ridges also have two disadvantages. First, they cannot be combined with any other attachment or SmartForce feature. For the force system to work

FIGURE 26-24 Power ridges.

as designed, the two points where the aligner places pressure on the tooth should be separated by as large a distance as possible, without interference from any other force system. In effect, the gingival protrusion in the aligner has a dual purpose. Not only does it create a force near the gingival margin, but it also lifts the aligner away from the facial surface of the tooth until the aligner re-engages the tooth on the lingual surface of the incisal edge of the tooth. The second problem with power ridges is that they can create irritation of the buccal tissues attributable to the protrusion of the margin of the aligner. Although generally acceptable to most patients, some will find this irritation very uncomfortable, more often on the lower arch than on the upper.

Power ridges were the first of a group of SmartForce features that are currently used to create force systems that clear aligners alone cannot achieve. These pressure areas may take the form of a power ridge, pressure point, or pressure area, and each may be a stand-alone feature or may work in conjunction with optimized attachments. Lingual root torque is achieved by the creation of power ridges as previously described. The multiplane attachment for the upper lateral incisors is an example of a pressure point on the lingual surface combined with an optimized attachment on the facial surface. Root control attachments on the upper and lower bicuspids will also use this combination of pressure points and optimized attachments to produce the required moment to correct root alignment on these teeth. The latest additions to this family of SmartForce features are pressure areas on the lingual surface of maxillary and mandibular centrals and laterals and the lower cuspids to direct intrusive forces more closely down the long axis of the tooth when any of these teeth are intruding more than 0.5 mm. Similar to optimized attachments, these pressure areas are all added by the Treat software when the amount and direction of movement extend beyond and surpass the default that triggers their placement. The orthodontist can control whether these features appear or disappear by moving the teeth above or below the threshold required by the software.

ROOT CONTROL IN THE INVISALIGN SYSTEM

Another aspect of biomechanics, especially pertinent to extraction treatment, is to control tipping to achieve root parallelism. When a force is applied in an attempt to move a canine distally, the tooth will rotate around the center of resistance. A sufficient moment is required to oppose the tipping movement. This area is more problematic because in a typical mesiodistal movement as in an extraction scenario, the aligner contacts the tooth on a surface that is parallel to the direction of force. The result is that little, if any, moment arm is created without the use of substantial attachments (Figs. 26-25 through 26-29).

An idea dating back to the late 1800s[30] was to place an attachment on the gingival aspect of a bracket extending toward the center of resistance in an attempt to decrease the amount of tipping when teeth are moved in a mesiodistal direction along

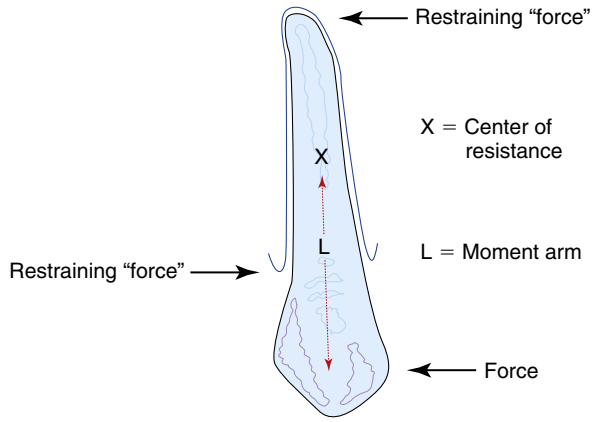


FIGURE 26-25 Force application against mesial of upper canine.

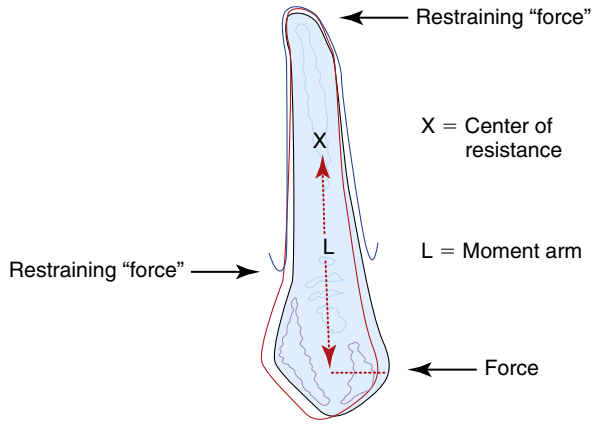


FIGURE 26-26 Effect of force application against mesial of upper canine.

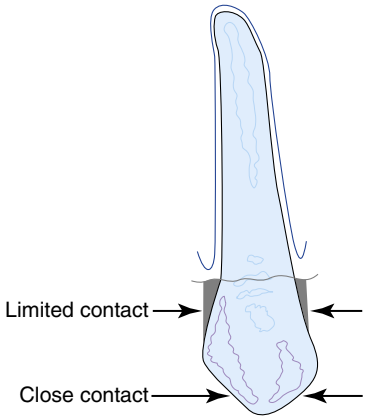


FIGURE 26-27 Diagram of aligner contact with upper canine.

the arch. These gingival extensions are often described as power arms. Power arms have been added to the force system with Invisalign in an attempt to alter the force-moment system (Figs. 26-30 and 26-31). In theory, the addition of a power arm auxiliary accomplishes two things. First, it moves the application of force closer to the center of resistance. Second, it creates a secondary moment attributable to pressure against the distal of the aligner. Unfortunately, the clinical application is not as beneficial as with fixed appliances because molar root control is more difficult than canine root control.

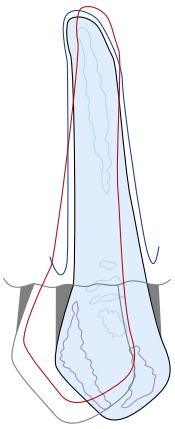


FIGURE 26-28 Displacement of aligner when force is applied against mesial of upper canine.

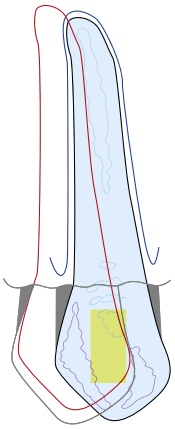


FIGURE 26-29 Controlled movement of upper canine with vertical attachment.

FIGURE 26-30 Handmade power arms in combination with aligners.

Align Technology has introduced engineered solutions in an attempt to mitigate the dumping in extraction cases and to help align the mesiodistal position of the roots of bicuspids, cuspids, and lateral and central incisors. The previous section notes that these root control enhancements are SmartForce features, consisting of pairs of optimized attachments or one optimized attachment and a corresponding pressure area. Short clinical crowns on the maxillary and mandibular bicuspids will dictate the use of the attachment and pressure area design. Maxillary central incisors, maxillary cuspids, and mandibular cuspids use only the paired attachments where two facial attachments will create the couple designed to tip the roots. The upper lateral

FIGURE 26-31 Manufactured power arms in combination with aligners.

FIGURE 26-32 Clinical photograph of lower incisor crowding.

FIGURE 26-33 Clinical photograph of root parallelism after single lower incisor extraction.

incisor multiplane feature uses an optimized attachment on the facial surface and a pressure area on the lingual surface.

The root positions of lower incisors have been successfully maintained during single incisor extraction treatment without the use of engineered root control features (Figs. 26-32 and 26-33), and cases of successful premolar extraction treatment using Invisalign have been reported.[31-33] Unfortunately, canines often remain upright during retraction into premolar spaces, whereas the clinical crowns of molars, especially maxillary molars, tend to tip mesially, which is frequently referred to as "dumping" (Figs. 26-34 to 26-36). Dumping occurs when the molars are being used as anchorage for anterior retraction. As the arch perimeter is reduced, closing extraction spaces, pressure is applied to the molar crowns in a mesial direction. This pressure is probably caused by the undesirable crown-to-root ratio combined with the large root surface area over which forces are distributed. G6, the latest release of SmartForce features, has antitipping attachments and pressure areas built into the molars to limit dumping of the clinical crowns in extraction cases. At this printing, no clinical cases have been reported that demonstrate the effectiveness of these latest root control

FIGURE 26-34 Mesial tipping of maxillary molar after premolar extraction.

FIGURE 26-35 Mesial tipping of maxillary molars after premolar extraction.

FIGURE 26-36 Mesial tipping of maxillary molars after premolar extraction.

features. To date, only the lower incisors are left without some form of engineered root control attachment design.

ROTATION CORRECTION IN THE INVISALIGN SYSTEM

Correcting rotations with aligners can be problematic for two primary reasons. First, aligners produce tooth movement by the plastic being slightly distorted and then elastically rebounding back to the predetermined shape, carrying the tooth with it. In the case of rotations, the aligner material is incapable of being distorted in a manner that can produce significant rotational movement. An analogous comparison would be attempting to rotate a tooth with a large steel archwire. Some suggest that beveled attachments with the bevel turned 90 degrees (i.e., mesiodistal; Fig. 26-37) would provide a surface that allows the aligner to rotate teeth. Even with a properly designed attachment, the second problem with rotations is that the tooth root is not a cylinder. Because of dilacerations and root surface variations, the computer software cannot adequately estimate

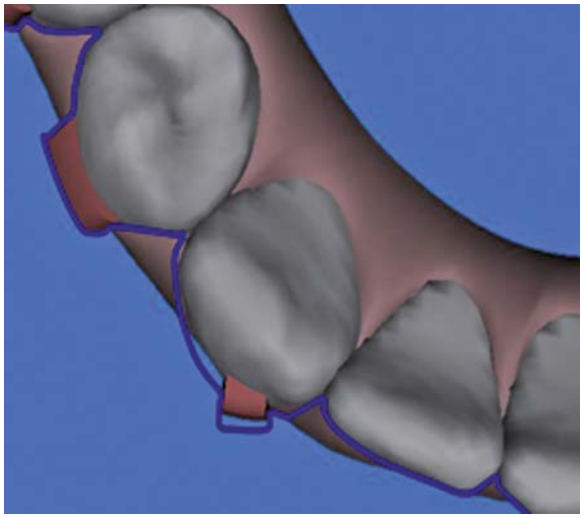


FIGURE 26-37 Rotation beveled attachment.

FIGURE 26-38 Rotational auxiliaries.

FIGURE 26-39 Rotational auxiliaries.

the true rotational long axis. In many cases, what is thought of as rotation of the tooth crown turns out to be bodily movement of the root surface; thus estimating the proper rate of tooth movement becomes impossible. When this happens in fixed appliances, it simply takes longer for the tooth to rotate; when it happens with aligners, the challenge is to keep the aligner fitting over the tooth. The result is the lack of desired movement; in addition, the aligner

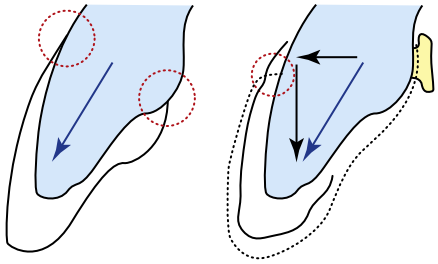


FIGURE 26-40 Extrusion auxiliary. Diagram of extrusion mechanics with button and trimmed aligner.

is now contacting different tooth surfaces than was intended. The result is either no movement or undesirable tooth movements. Before the addition of SmartForce attachments, many of which are specifically designed to correct rotated teeth, using auxiliaries before, during, or after aligner treatment was needed to accomplish the rotational correction (Figs. 26-38 and 26-39). With the advent of the newer optimized attachments, the predictability of rotational movements has significantly improved. Optimized attachments are now available for all bicuspids and cuspids. The default for the bicuspids and cuspids is rotation correction greater than 5 degrees in either direction. Molars and incisors do not have engineered rotation attachments to date. (Note that the upper lateral incisor attachment is not a rotation correction attachment; rather, it is designed for extrusion in combination with root or crown tip.)

EXTRUSION CORRECTION IN THE INVISALIGN SYSTEM

Extrusions were once thought to present problems with aligners. The reason for this assumption is similar to that of rotations. In the same way that the aligner itself is incapable of elastic deformation in the direction needed for effective rotational movement, the aligner cannot stretch within the plastic itself; consequently, an elastic deformation in the direction needed for extrusion is not possible. One method previously used to overcome this problem was to use the gingival beveled attachment to provide a longer surface area that can be elastically deformed and provide an extrusive force on the tooth. This model obviously had merit because Align Technology subsequently released an engineered solution to assist extrusion of maxillary and mandibular incisors and cuspids. The optimized attachments that assist extrusion are very similar to horizontal beveled attachments, beveled toward the gingival margin. The default that causes the attachment to be added to the virtual plan is extrusion of more than 0.50 mm down the long axis of the tooth. Relative extrusion, in which the crown of each incisor lingually reclines, may not trigger the placement of optimized extrusion attachments, even though the extrusion may be quite obvious. An additional SmartForce feature improves the tracking of anterior teeth extrusion when all four upper incisors are individually extruding more than 0.50 mm. This additional feature alters the digital model to create a pressure area at the bases of the optimized attachment on the upper lateral incisors. This pressure area produces a higher force on the optimized attachment to keep the teeth fully engaged in the aligner. The addition of optimized attachments for extrusion and pressure areas on the upper lateral incisors has made Invisalign the preferred appliance for treatment of mild to moderate anterior open bite treatment.

In particularly challenging situations, a button bonded to the tooth, together with an elastic, will assist with the extrusion (Figs. 26-40 to 26-42). This method is infrequently used

after the introduction of optimized attachments and can also be a very useful technique to finish treatment when only minor extrusion of a single tooth is required.

AUXILIARIES AND THE INVISALIGN SYSTEM

Other auxiliaries can be used to facilitate specific movements. Class II and Class III elastics are frequently needed, just as they are with fixed appliances. One can directly attach the elastics either to the aligner or to the buttons bonded to the teeth. If the elastics are directly attached to the aligner, then attachments are generally required to prevent displacement of the aligner. Engineers at Align Technology have developed an elastic hook with the proprietary name "precision hooks and button cutouts" that are manufactured into the aligner. The orthodontist can place either of these options when modifying the ClinCheck using the ATTACH & CUTS interface.

Miniscrews can also be effectively used with aligners in the same manner as with fixed appliances, either initially planned as part of the treatment or to help with movements that are not progressing as desired. Miniscrews can be used with aligners alone or in combination with other auxiliaries to simplify the movements the aligners are required to accomplish. The two most common uses of miniscrews with aligners are for vertical and sagittal movements. One such example is the extrusion of an upper canine. Figures 26-43 to 26-45 demonstrate placing a miniscrew in the lower arch and then running a rubber band from a clear button near the gingival on the upper canine to the miniscrew as the aligner guides the tooth into the correct position.

Another vertical movement that is easily enhanced with miniscrews is the intrusion of molars that have supererupted into an edentulous space. Figures 26-46 to 26-48 illustrate

FIGURE 26-41 Extrusion auxiliary. Clinical photograph of extrusion mechanics with lingual button and trimmed aligner.

FIGURE 26-42 Extrusion auxiliary. Clinical photograph of extrusion mechanics with facial button and trimmed aligner.

FIGURE 26-43 High canine.

FIGURE 26-44 Canine with miniscrew placed.

FIGURE 26-45 Final with canine extruded.

FIGURE 26-46 Supererupted molar.

placing a miniscrew on the buccal and lingual aspects of an upper molar. The patient then wears an elastic from one mini screw over the top of the aligner to the other miniscrew. Many of these patients need to undergo significant restorative treatment, and wearing aligners during the orthodontic setup is often preferred by the patient to wearing fixed appliances.

Miniscrews can expedite Class II correction. The first example involves placing a Carriere Distalizer appliance (Henry Schein Orthodontics, Carlsbad, CA) in the upper arch along with a miniscrew in the lower arch in the molar or retromolar area. A Class II elastic is then worn 24 hours a day; generally, a Class I molar and canine correction can be expected in approximately 12 to 16 weeks. Once the sagittal molar and cuspid correction has been accomplished, then arch alignment and finishing can be accomplished with Invisalign (Figs. 26-49 to 26-52).

FIGURE 26-47 Supererupted molar with miniscrews and aligners.

Another application of miniscrews with aligners is correcting an arch asymmetry by enhancing the distalization of one side. This correction can be accomplished by placing a miniscrew in the retromolar area, bonding buttons on the facial and lingual aspects of the upper first or second molar, and then connecting an elastic chain from the buttons to the miniscrew. If the intended movement is planned into the aligner treatment, then the mini screw provides the anchorage and allows simultaneous movement in the ClinCheck to reduce treatment time (Figs. 26-53 to 26-55).

The previous images demonstrate a few examples of using miniscrews along with aligners to accomplish otherwise difficult movements. There are obviously multiple other applications of miniscrews with aligners.

INVISALIGN TEEN

Invisalign Teen (Align Technology, Inc., Santa Clara, CA) is not so much a different appliance as it is a specific feature set. Originally, Invisalign was anticipated for use with adults and approved by the U.S. Food and Drug Administration (FDA) for those individuals with a fully erupted permanent dentition. It soon became apparent, however, that being able to treat the late mixed dentition with aligners provided certain benefits as well. The shortcomings to overcome were anticipating tooth eruption of one or more permanent teeth, being able to monitor patient compliance to discuss the progress (or lack thereof) with parents, proper control of torque without the need for attachments when crowns were not yet fully exposed, and, finally, avoiding practice management issues over lost aligners. Eruption tabs are used to prevent supereruption of unerupted second molars (Fig. 26-56). Tooth forms of approximate anticipated crown size are used to both create and hold room

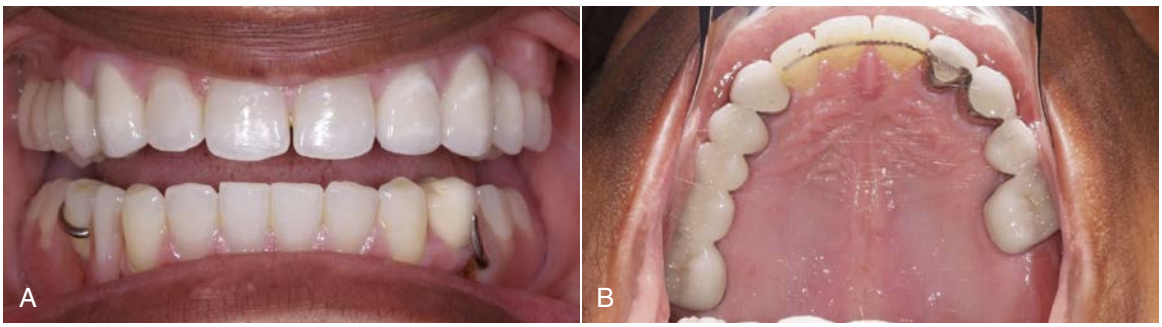


FIGURE 26-48 A, B, Final restorations of supererupted molar with miniscrews and aligners.

FIGURE 26-49 A, B, Initial class II malocclusions.

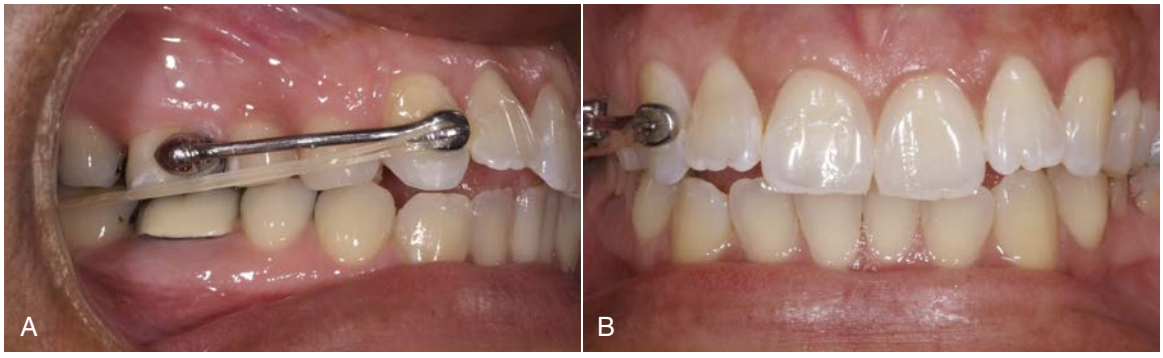


FIGURE 26-50 A, B, Carriere distalizer appliance and miniscrew placed.

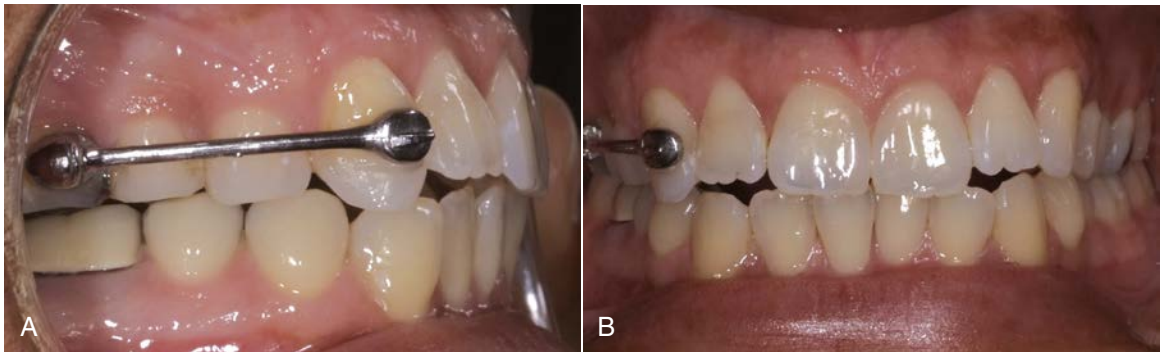


FIGURE 26-51 A, B, Class II malocclusions corrected to Class I.

FIGURE 26-52 A, B, Holding aligner placed with Class II elastic.

FIGURE 26-53 Pretreatment.

FIGURE 26-54 Elastic to miniscrew.

FIGURE 26-55 A, B, Asymmetry correction.

FIGURE 26-56 Eruption tabs.

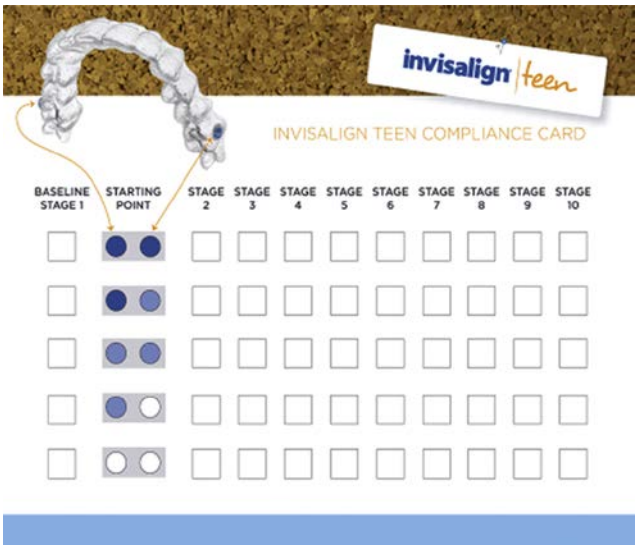


FIGURE 26-57 Compliance indicators.

and to guide eruption of actively erupting second premolars and canines, planning for refinement aligners with proper fit once the teeth are adequately erupted to capture properly the crowns in the impression. Wear indicators are placed on the facial surfaces of the first molars (Fig. 26-57). Two different types of chemical indicators are available that turn from dark blue to clear as the aligners are worn. These indicators are designed so that creative teenagers cannot realistically figure out a method to have both indicators change without actually wearing the aligners. All SmartForce enhancements, including optimized attachments, pressure points, and pressure areas that are previously discussed were developed for the Invisalign Teen product and are a routine part of the feature set. The practice management part is actually quite easy. Align Technology charges a premium and provides free replacements for lost aligners. In reality, the patient prepays for the privilege of having them replaced if lost. Many adults can benefit from the features in Invisalign Teen, and it is perfectly acceptable to order the Invisalign Teen product for an adult to access any of the unique product features.

Understanding when and where to anticipate the use of aligners in combination with other techniques or auxiliaries is critical to both getting satisfactory results and satisfying patients. Several variables combine to produce either acceptable desired results or unacceptable undesired tooth movements. A review of these results with reference to specific movements follows. The first and most important variable is duration of wear. Aligners are not retainers and must be consistently worn for approximately 22 hours of a 24-hour period, essentially acting similar to fixed appliances. If not consistently worn, then results are extremely unpredictable. Patients who do not comply with wearing aligners for the daily required duration generally have less than desirable results.

The next most important variables are clinical crown length and shape. The longer the clinical crown and the greater the natural undercuts to facilitate aligner retention, the more likely that the desired movement will take place, because there is a greater amount of surface area for the aligner to contact. Patients with very short clinical crowns are not good candidates to attempt some movements with Invisalign such as root alignment with premolar extraction treatment. Single lower central incisor extraction tends to be successful because of the length of the clinical crowns of the lower incisors relative to the forces applied.

Closing anterior spaces, especially with protrusive incisors that require some intrusion, is extremely predictable and requires no attachments. Closing minor anterior open bites is predictable because one secondary effect of aligner wear is the vertical control of dentoalveolar eruption of the posterior teeth. In growing patients this is particularly poignant. Because aligners tend to limit posterior tooth eruption, when opening a deep bite, special consideration should be given to

the virtual setup. If the curve of Spee is deep, then intruding lower second molars and lower incisors (using the aligner as if it were a reverse curve of Spee archwire) leaves only a small amount of extrusion of the premolars and first molars. Along with the reverse curve of Spee effect, heavy posterior occlusal contacts can be virtually created to compensate for the side effect of molars and bicuspids not being able to extrude easily. (Note that leveling the curve of Spee has now been made significantly easier with the addition of the virtual bite plane being added to the lingual aspect of the upper incisors. This addition was part of the G5 release in 2014 to address deep bite treatment). Opening the bite is accomplished by creating virtual collisions between the upper and lower molars when modifying the ClinCheck.

As previously discussed, some rotations are somewhat unpredictable as well. Often the decision is not whether to treat a patient with Invisalign but rather to predetermine whether adjunctive treatment may be required to gain the best end results for the patient and/or decrease overall treatment time. There are instances in which a short period of fixed appliances is of benefit to treat severe rotations, to align divergent roots, or to close significant spaces. This brief period of fixed appliances will be followed by comprehensive treatment with aligners. The benefit to this approach is the decrease in overall treatment time, as well as better results for difficult malocclusions. Success depends on patient demands and expectations, as well as the orthodontist's comfort with combining the two treatment modalities. No hard and fast rules have been written; each appliance may be used to do what it does best and tailor treatment to use whichever appliance meets the patient's personal needs and is the most efficient and effective in treating the orthodontic list of problems at hand.

PERIODONTAL CONSIDERATIONS

A growing body of evidence suggests that orthodontic treatment with aligners has less detrimental periodontal impact than that of fixed appliances. Miethke and Vogt[33] and Miethke and Brauner[34] compared the periodontal health of patients who underwent treatment with aligners with that of patients who underwent treatment with both labial fixed appliances and with lingual fixed appliances and found that the periodontal risk was no greater than with labial appliances and was lower than that of lingual fixed appliances. Boyd[35,36] found that periodontal health could actually improve during the course of treatment with Invisalign. He attributed this improvement to the patients' ability to remove the appliances and spend more time brushing and flossing their teeth and the aligner's ability to maintain an invisible appearance to the appliances.

SUMMARY

Successful treatment of many malocclusions with proper tip, torque, arch form, and aesthetic crown inclination is possible to achieve with clear aligners. Align Technology provides the most advanced clear aligner technology in the marketplace to date. Invisalign-branded clear aligners, using the proprietary plastic material and innovative SmartForce enhancements, provide predictable high-quality outcomes in the hands of a competently trained orthodontist. The appliance is neither intuitive to use nor easy to master. ClinCheck, although a great tool, requires considerable skill to manipulate the teeth to the desired positions. Besides the obvious aesthetic improvement over fixed appliances, periodontal health benefits may also be associated with Invisalign treatment. Understanding both the process of aligner manufacturing along with the biomechanics of tooth movement with aligners and applying that knowledge to treatment planning and clinical execution should enable the clinician to design a treatment plan with aligners alone or in combination with fixed appliances to make both simple and complex treatments predictable and routine.

CASE STUDY 26-1 CLASS I MODERATE CROWDING

A 39-year-old woman exhibited a typical orthodontic relapse, having been treated as a teenager and then failing to wear retainers. She exhibited significant lower crowding and dark triangles between upper incisors. Figure 26-58, *A–H*, shows her initial presentation. Figure 26-59, *A–H*, shows her final results. Figure 26-60, *A*, shows her final ClinCheck. Figure 26-60, *B*, shows her ClinCheck with staging, and Figure 26-61, *A–J*, shows the patient 2 years after treatment. To reduce dark triangles and control crown angulations, interproximal reduction (IPR) was prescribed and attachments were appropriately placed. Her treatment took 11 months, 9 upper aligners, 21 lower aligners, and 9 total appointments, including examination, impressions, and retainer delivery.

David E. Paquette

FIGURE 26-58 A–H, Initial photographs.

Continued

CASE STUDY 26-1 CLASS I MODERATE CROWDING—cont'd

FIGURE 26-59 A–H, Final photographs.

CASE STUDY 26-1 CLASS I MODERATE CROWDING—cont'd

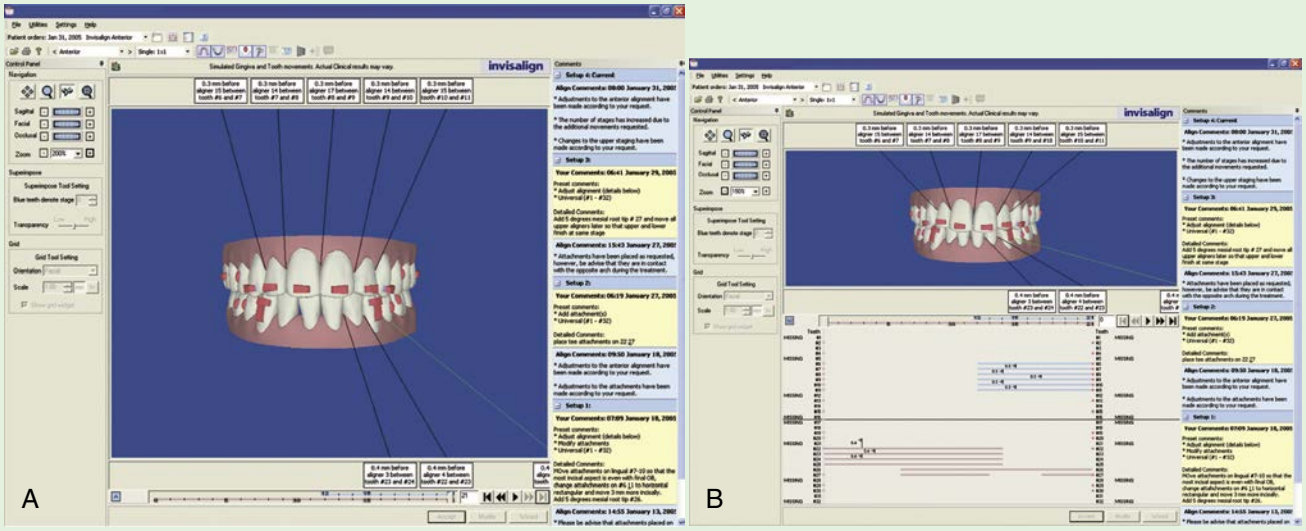


FIGURE 26-60 A, B, ClinCheck.

FIGURE 26-61 A–J, At 2 years after treatment.

Continued

CASE STUDY 26-1 CLASS I MODERATE CROWDING—cont'd

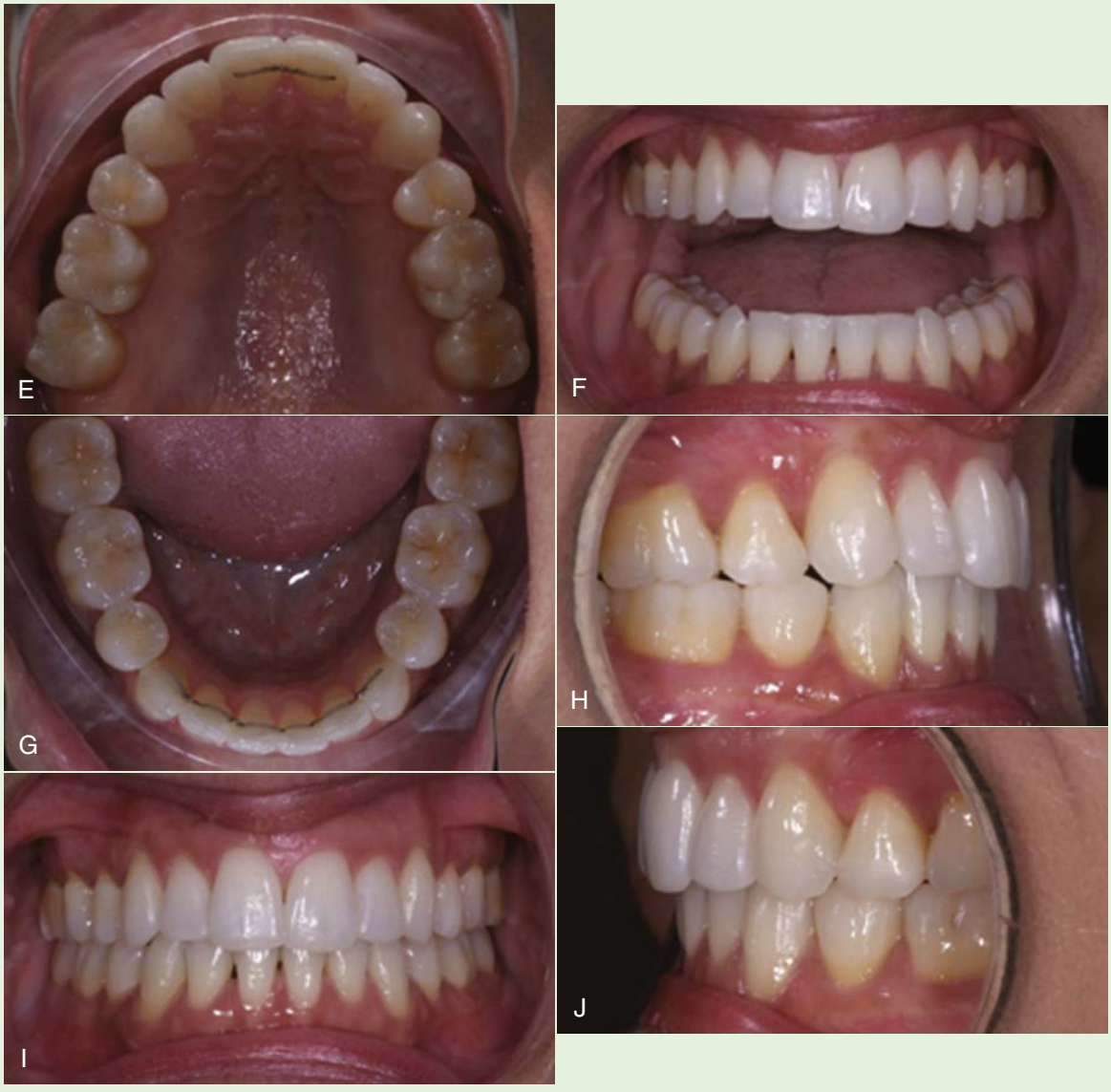


FIGURE 26-61 cont'd

CASE STUDY 26-2 CLASS I DEEP OVERBITE

JS, a 39-year-old man, has no history of orthodontic treatment. He had a deep bite with increased curve of Spee, narrow arches, and moderate crowding. Figure 26-62, *A–F*, show his initial presentation. Figure 26-63, *A–H*, shows his final results. Figure 26-64, *A*, shows his final ClinCheck, and Figure 26-64, *B*, shows his ClinCheck with staging. Figure 26-65, *A–C*, shows the superimpositions and method for leveling the curve of Spee. His treatment took 13 months, 26 aligners, and 10 total appointments including examination, impressions, and retainer delivery.

David E. Paquette

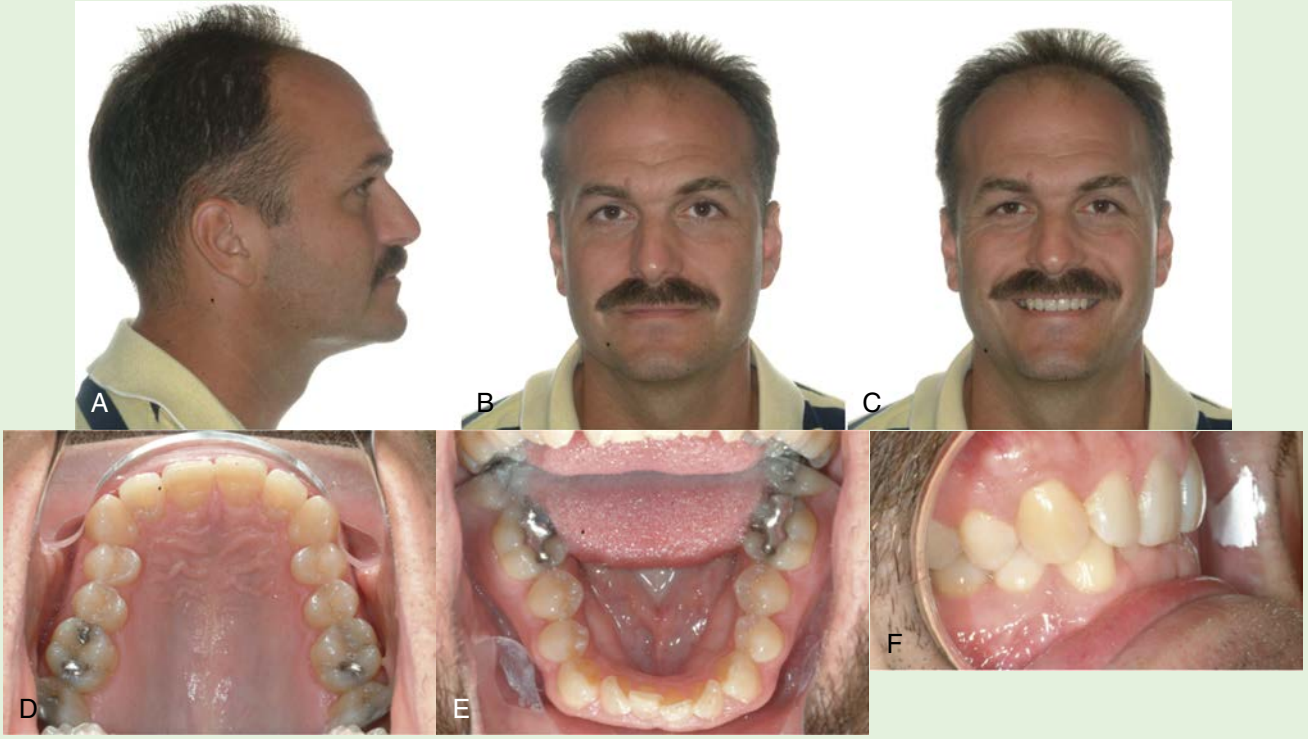


FIGURE 26-62 A–F, Initial photographs.

Continued

CASE STUDY 26-2 CLASS I DEEP OVERBITE—cont'd

FIGURE 26-63 A–H, Final photographs.

CASE STUDY 26-2 CLASS I DEEP OVERBITE—cont'd

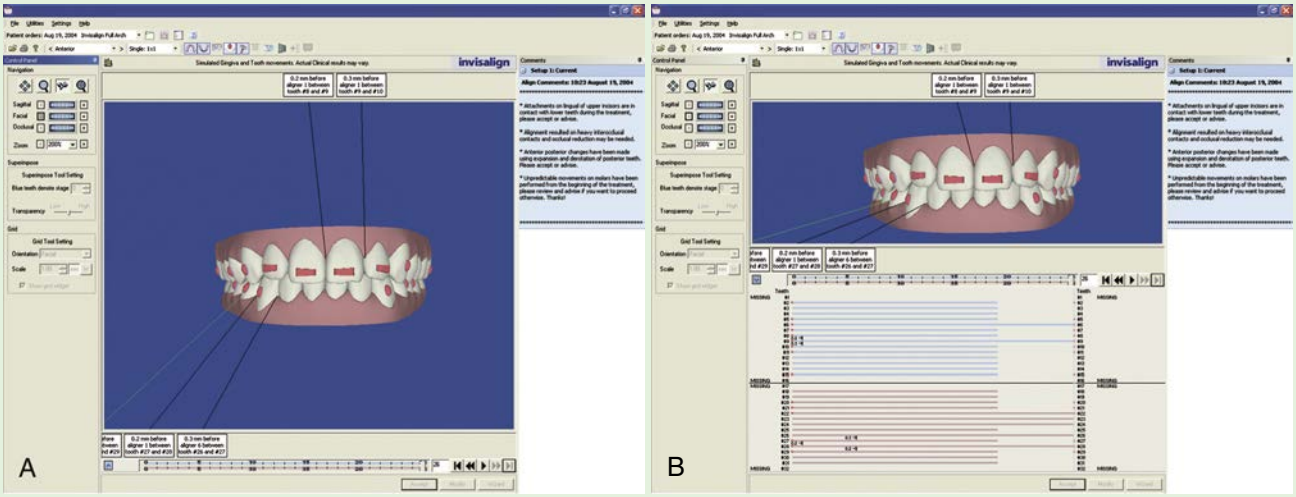


FIGURE 26-64 A, B, ClinCheck.

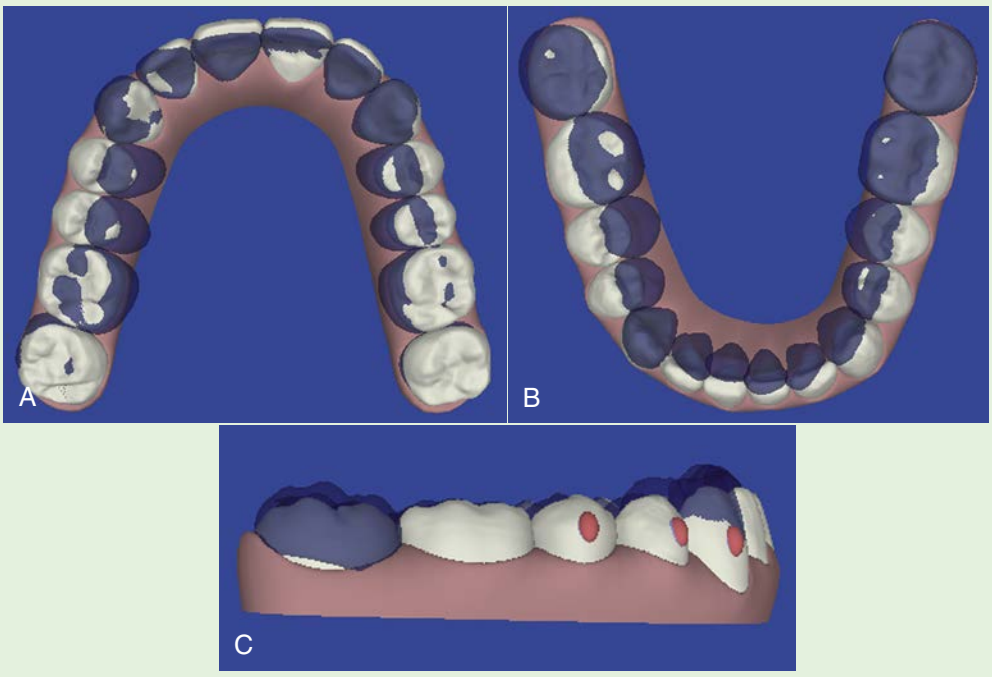


FIGURE 26-65 A–C, ClinCheck with superimposition.

CASE STUDY 26-3 CLASS II SUBDIVISION LEFT

CC, a 30-year-old woman, has no history of orthodontic treatment. She exhibited Class II subdivision left. Figure 26-66, *A–H*, shows her initial presentation. Figure 26-67, *A–H*, shows her final results. Figure 26-68, *A*, shows her final ClinCheck, Figure 26-68, *B*, shows her ClinCheck with staging, and Figure 26-69 shows how her Class II elastics were worn. Her treatment took 23 months, 44 aligners (26 initial and 18 refinement), and 14 total appointments including examination, impressions, and retainer delivery.

David E. Paquette

FIGURE 26-66 A–H, Initial photographs.

CASE STUDY 26-3 CLASS II SUBDIVISION LEFT—cont'd

FIGURE 26–67 A–H, Final photographs.

Continued

CASE STUDY 26-3 CLASS II SUBDIVISION LEFT—cont'd

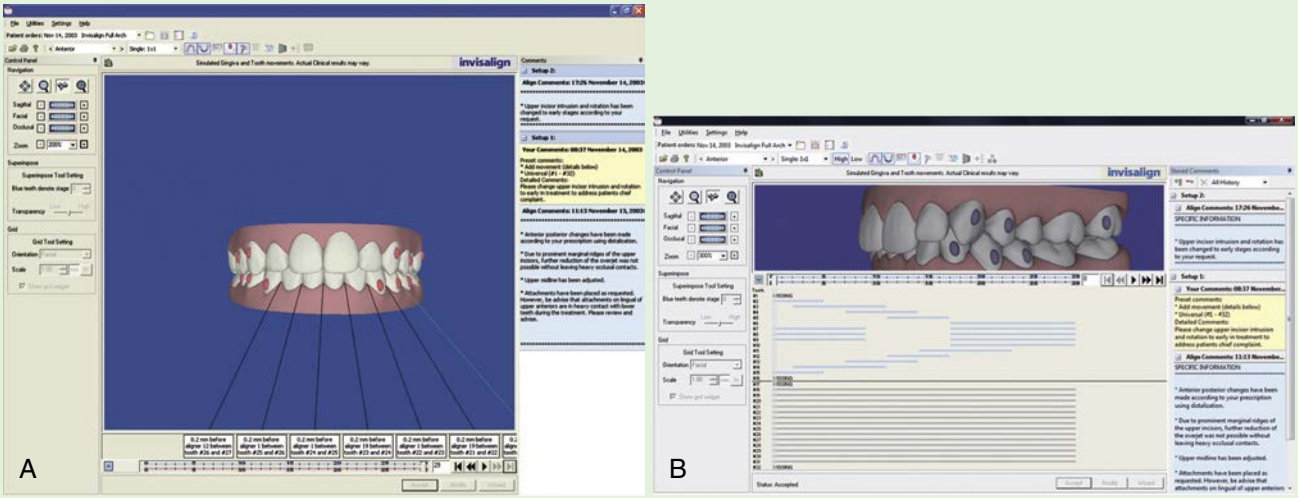


FIGURE 26-68 A, B, ClinCheck.

FIGURE 26-69 Class II elastics.

REFERENCES

1. Kesling HD. The philosophy of tooth positioning appliance. *Am J Ortho Oral Surg*. 1945;31:297–304.
2. Nahoum H. The vacuum-formed dental contour appliance. *N Y State Dent J*. 1964;30:385–390.
3. Ponitz RJ. Invisible retainers. *Am J Orthod*. 1971;59(3):266–272.
4. McNamara JA, Kramer KL, Juenker JP. Invisible retainers. *J Clin Orthod*. 1985;19:570–578.
5. Sheridan JJ, LeDoux W, McMinn R. Essix retainers: fabrication and supervision for permanent retention. *J Clin Orthod*. 1993;27(1):37–45.
6. Hilliard K, Sheridan JJ. Adjusting Essix appliance at chairside—these simple adjustments can be made in the operatory. *J Clin Orthod*. 2000;34(4):236–238.
7. Proffit WR. *Contemporary Orthodontics*. St. Louis: CV Mosby; 1986.
8. Owens Jr SE, Dykhouse VJ, Moffitt AH, et al. The case management form of the American Board of Orthodontics. *Am J Orthod Dentofacial Orthop*. 2006;129:325–329.
9. Patel M, Taylor M, McGorray S, et al. Outcome of Invisalign orthodontic treatment using PAR index. *J Dent Res*. 2004;83(Spec. Issue A):1282.
10. Vincent S. Evaluation of Invisalign® treatment utilizing the American Board of Orthodontics objective grading system for dental casts. *Am J Orthod Dentofacial Orthop*. 2005;127(2):268–269.
11. Djeu G, Shelton C, Maganzini A. Outcome assessment of Invisalign® and traditional orthodontic treatment compared with the American Board of Orthodontics objective grading system. *Am J Orthod Dentofacial Orthop*. 2005;128(3):292–298.
12. Brown P, Bayirli B, Gaynier B, Vazquez D. *Comparison of Invisalign® to fixed orthodontic treatment using ABO indices*; 2007. *J Dent Res*. 2007;86(Spec. Issue A):1916.
13. Lagravere MO, Flores-Mir C. The treatment effects of Invisalign® orthodontic aligners: a systematic review. *J Am Dent Assoc*. 2005;136(12):1724–1729.
14. Sims MR. Brackets, epitopes and flash memory cards: a futuristic view of clinical orthodontics. *Aust Orthod J*. 1999;15(5):260–268.
15. Proffit WR. Adjunctive treatment for adults. In: Proffit WR, Fields HJ, eds. *Contemporary Orthodontics*. St. Louis: CV Mosby; 1986.
16. Dugoni AA. What price progress? Reply. *Am J Orthod Dentofacial Orthop*. 2002;122(2):17A.
17. Miller KB, McGorray SP, Womack R, et al. A comparison of treatment impacts between Invisalign aligner and fixed appliance therapy during the first week of treatment. *Am J Orthod Dentofacial Orthop*. 2007;131(3):302.e1–9, PubMed PMID: 17346581.
18. Hahn W, Dathe H, Fialka-Fricke J, et al. Influence of thermoplastic appliance thickness on the magnitude of force delivered to a maxillary central incisor during tipping. *Am J Orthod Dentofacial Orthop*. 2009;136(1):12.e1–7, discussion 12-3: http://dx.doi.org/10.1016/j.ajodo.2008.12.015. PubMed PMID: 19577136.
19. Kwon JS, Lee YK, Lim BS, et al. Force delivery properties of thermoplastic orthodontic materials. *Am J Orthod Dentofacial Orthop*. 2008;133(2):228–234. http://dx.doi.org/10.1016/j.ajodo.2006.03.034. quiz 328.e1.
20. Hahn W, Engelke B, Jung K, et al. Initial forces and moments delivered by removable thermoplastic appliances during rotation of an

upper central incisor. *Angle Orthod*. 2010;80(2):239–246. http://dx.doi.org/10.2319/033009-181.1. PubMed PMID: 19905847.
21. Hahn W, Zapf A, Dathe H, et al. Torquing an upper central incisor with aligners–acting forces and biomechanical principles. *Eur J Orthod*. 2010;32(6):607–613. http://dx.doi.org/10.1093/ejo/cjq007. Epub 2010 May 12. PubMed PMID: 20462912.
22. Fang D, Zhang N, Chen H, et al. Dynamic stress relaxation of orthodontic thermoplastic materials in a simulated oral environment. *Dent Mater J*. 2013;32(6):946–951. Epub 2013 Nov 15. PubMed PMID: 24240895.
23. Patel ND. *Effect of aligner material, duration, and force level on tooth movement*; 2014. http://www.iadr.org/files/NSRG/LSRGNewsletter/UF_July2014.pdf.
24. Iwasaki LR, Haack JE, Nickel JC, et al. Human tooth movement in response to continuous stress of low magnitude. *Am J Orthod Dentofacial Orthop*. 2000;117(2):175–183.
25. Proffit WR. Mechanical principles in orthodontic force control. In: Proffit WR, ed. *Contemporary Orthodontics*. St. Louis: CV Mosby; 1986.
26. Foy M. *Personal communication, Invisalign® Alpha Group meeting, 2004*; 2004. Salt Lake City, UT.
27. Jones ML, Mah J, O'Toole BJ. Retention of thermoformed aligners with attachments of various shapes and positions. *J Clin Orthod*. 2009;43(2):113–117.
28. Nicozisis JL. Clinical report. *Clin Rep Techn*. 2006;2(1). http://www.westwindsororthodontics.com/docs/Summer_Clinical_Report.pdf.
29. Farrar JN. Regulation of teeth made easy. *Dental Cosmos*. 1878;20:18–22. http://quod.lib.umich.edu/d/dencos/acf8385.0020.001/30:7?rgn=main;view=image.
30. Hönn M, Göz G. A premolar extraction case using the Invisalign® system. *J Orofac Orthop*. 2006;67(5):385–394.
31. Womack WR. Case report: four-premolar extraction treatment with Invisalign. *J Clin Orthod*. 2006;40(8):493–500.
32. Paquette D. Extraction treatment with Invisalign. In: Tuncay O, ed. *The Invisalign® System*. London: Quintessence; 2006.
33. Miethke RR, Vogt S. A comparison of the periodontal health of patients during treatment with Invisalign® system and with fixed orthodontic appliances. *J Orofac Orthop*. 2005;66(3):219–229.
34. Miethke RR, Brauner K. A comparison of the periodontal health of patients during treatment with the Invisalign® system and with fixed lingual appliances. *J Orofac Orthop*. 2007;68:223–231.
35. Boyd R. Improving periodontal health through Invisalign® treatment. *Access*. 2005:Nov.;24–26. http://provider.invisalign.com/documents/publications/NovAlign102105_Deep_Bite_Crowding.pdf.
36. Boyd RL. Esthetic orthodontic treatment using the Invisalign® appliance for moderate to complex malocclusions. *J Dent Educ*. 2008;72(8):948–967.

27

Bonding in Orthodontics

Björn U. Zachrisson, Serdar Usumez, and Tamer Büyükyilmaz

OUTLINE

Introduction, 812
Materials and Devices Used in Orthodontic Bonding, 813
Brackets, 813
Ceramic Brackets, 813
Metal Brackets, 813
Adhesives, 813
Composite Resins, 813
Glass Ionomer Cements, 817
Light Sources, 817
Light-Emitting Diodes, 818
Bonding, 818
Bonding to Enamel, 819
Premedication, 819
Cleaning, 819
Enamel Conditioning, 819
Sealing and Priming, 821
Bonding, 821
Bonding to Artificial Tooth Surfaces, 825
Bonding to Porcelain, 825
Bonding to Amalgam, 827
Bonding to Gold, 829
Bonding to Composite Restoratives, 829
Indirect Bonding, 829
Clinical Procedure, 830
Indirect Bonding with Composite Custom Bracket Base, 830
Rebonding, 831
Conclusion, 832
Debonding, 833
Clinical Procedure, 833
Removal of Steel Brackets, 833
Removal of Ceramic Brackets, 834
Enamel Tearouts and Cracks (Fracture Lines), 834
Removal of Residual Adhesive, 834
Amount of Enamel Lost in Debonding, 837
Operator Safety during Debonding, 837
Prevention and Reversal of Decalcification, 838
Microabrasion, 838
Resin Infiltration, 840
Bonded Retainers, 840
Bonded Fixed Retainer Materials, 840
Bonded Fixed Retainer Adhesives, 840
Periodontal Health with Bonded Fixed Retainers, 841
Canine-to-Canine Lingual Retainer Bar, 842
Bonding the 3-3 Retainer Bar, 845
Failure Analysis and Long-Term Experience with the 3-3 Retainer Bar, 847
Multistranded Wire Retainers, 847
Bonding Multistranded Wire Retainer, 849
Failure Analysis and Repair, 852
Long-Term Experience with the Multistranded Wire Retainer, 852
Direct-Bonded Labial Retainers, 854
Technical Procedure, 855
Long-Term Results, 855
Other Applications of Bonding, 855
Bonding a Large Acrylic Appliance, 855
Technical Procedures, 859
Permanent Dentition, 859
Deciduous or Mixed Dentition, 859
Occlusal Buildup of Posterior Teeth, 860
Technical Procedure, 861
Conclusion, 862

INTRODUCTION

The introduction of bonding to orthodontics has had a dramatic effect on the profession. The many advantages of bonded brackets over cemented bands include the aesthetic improvement, the elimination of band seating, and the need for tooth separation, as well as the elimination of the thickness of the band material, which affected the arch length. Improved oral hygiene through easier access to the interproximal dental areas helps reduce the risk of

enamel decalcification. Accessibility to the interproximal area also allows for earlier detection of caries at these sites and their restoration, improved access to interproximal contacts for mesiodistal contouring, and the elimination of the need for posttreatment space closure. The ability to bond partially erupted and malaligned teeth enables earlier force application during treatment, which was not previously possible with banded attachments.[1–3] Direct bonding of orthodontic attachments also increased the acceptability of orthodontic appliances by the public and popularized orthodontic treatment, thus increasing the number of new enrollments each year. Advances in direct bonding also induced the emergence of some new techniques such as lingual orthodontics, which would have made no sense when used with circumferential bands.

Today, as the profession is approaching over 50 years of successful, reliable orthodontic bonding, a recent survey indicates that the decline of banding against bonding is noteworthy, and maxillary first molars are routinely banded by only less than one third of U.S. orthodontists. Presently, the median bond failure rate for practitioners in the United States is approximately 5% for labial and 4% for lingual appliances.[4,5] This finding is in accordance with the dramatic decrease of use of all types of headgears in the past 6 years.[5]

Achieving a low bond failure rate should be a high-priority goal, since replacing loose brackets is disturbing, time consuming, and costly. Consequently, an endless search is on for higher bond strengths, better adhesives, simpler procedures, and materials that will bond in the presence of saliva. However, most bond failures are a consequence of inconsistencies in the bonding technique and are not related to bonding resins, inadequate bond strengths, or the quality of the brackets being used.[6] Newer resin systems and alternative methods to bond to enamel may be giving the false impression that an orthodontist does not need to be as careful with the bonding procedures as before.

The basis for the adhesion of brackets to enamel has been enamel etching with phosphoric acid, as first proposed by Buonocore[7] in 1955. In the early 1970s, a considerable number of preliminary reports were published on different commercially available direct and indirect bonding systems. The first detailed posttreatment evaluation of direct bonding over a full period of orthodontic treatment in a large sample of patients was published in 1977.[1] Since then, product development in terms of adhesive resins, brackets, and technical details has occurred at a rapid rate. In fact, the progress has made it difficult for the practicing orthodontist to remain properly oriented.

The purpose of this chapter is to update current available information on bonding to natural and artificial teeth. A special emphasis is given to those techniques and materials that are demonstrated to be trending in recent studies.[4,5]

To help organize the contents, the chapter is divided into four parts:

1. Materials and devices used in orthodontic bonding
2. Bracket bonding
3. Debonding
4. Bonded retainers

MATERIALS AND DEVICES USED IN ORTHODONTIC BONDING

Brackets

The two most common types of orthodontic attachments are stainless steel and ceramic brackets, which constitute 91% (80% metal plus 11% ceramic) of all brackets used.[5]

FIGURE 27-1 Ceramic brackets provide an aesthetic way of attaching to tooth surfaces, making orthodontic treatment more acceptable, particularly for some patients.

Ceramic Brackets

Ceramic brackets have become an important although sometimes troublesome part of today's orthodontic practice. Ceramic orthodontic brackets are machined from monocrystalline or polycrystalline aluminum oxide. Theoretically, such brackets should combine the aesthetics of plastic and the reliability of metal brackets.

Ceramic brackets bond to enamel by two different mechanisms: (1) mechanical retention via indentations and undercuts in the base, and (2) chemical bonding by means of a silane-coupling agent. With mechanical retention, the stress of debonding is generally at the adhesive–bracket interface, whereas the chemical bonding may produce excessive bond strengths, with the stress at debonding shifted toward the enamel–adhesive interface (see Debonding). Chemically cured and light-cured adhesives are useful for ceramic brackets (Fig. 27-1).

Metal Brackets

Metal brackets depend on mechanical retention for bonding, and a mesh structure is the most common method of providing this[4] (Fig. 27-2, *A* and *D*). However, many bracket-based designs such as standard mesh base (Ultraminitrim, Dentaurum GmbH & Co., Ispringen, Germany); supermesh base (Microarch-GAC, Dentsply GAC, Islandia, NY), integral base (Dyna-Lock, 3M Unitek, Monrovia, CA); MicroLoc GAC (Dentsply GAC, Islandia, NY), microetched base (Miniature Twin, 3M Unitek, Monrovia, CA); and laser-structured base (Discovery brackets, Dentaurum GmbH & Co., Ispringen, Germany)[8–10] are available for clinical use.

Mesh pad is the system most commonly used for retention. The improvement of these variables has been the goal of many research projects.[11–16] A new type of laser-structured base retention was found to produce double the bond strength produced by foil mesh without compromising debonding characteristics.[17]

Adhesives

Composite Resins

Modern dental composite restorations can be considered to have originated in Bowen's classic work on the development of

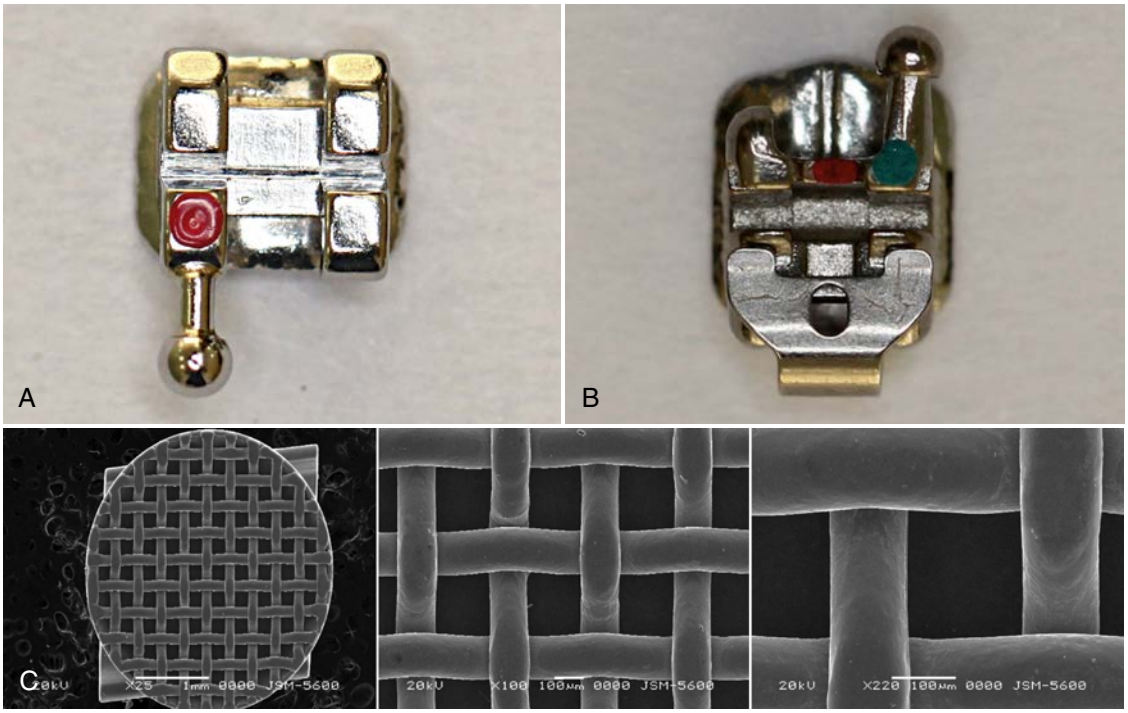


FIGURE 27-2 **A,** Metallic twin bracket sample (Mini Master Series, American Orthodontics, Sheboygan, WI). **B,** Metallic twin bracket sample with self-ligating clip (Empower Series, American Orthodontics, Sheboygan, WI). **C,** Bracket bases present a retentive base for holding the cured resin. Mesh pad is the system most commonly used for retention. Scanning electron microscopy (SEM) views of a standard mesh base bracket demonstrate undercut formations required for mechanical retention. (Ultraminitrim, Dentaurum GmbH & Co., Ispringen, Germany). (**A** and **B,** Courtesy Dr. Sabri Ilhan Ramoglu, Istanbul, Turkey. **C,** From Usumez S, Erverdi N. Adhesives and bonding in orthodontics. In: Nanda R, Kapila S, eds. *Current Therapy in Orthodontics*. 1st ed. St. Louis: Mosby; 2009:45–67.)

the monomer 2,2-bis-4(2-hydroxy-3-methacryloyloxypropoxy) phenylpropane.[18] The essential chemistry of the synthesis of this substance is the reaction of glycidyl methacrylate with bisphenol A to create a molecule informally known as bisphenol A–glycidyl methacrylate (bisGMA). The original aim of the synthetic studies of Bowen that led to the development of this monomer was to combine the advantages of the acrylic systems with those of epoxy systems based on bisphenol A but without the disadvantages. A fundamental difference is that cured acrylic resins form only linear polymers, whereas newer formulations may also be polymerized by cross-linking into a three-dimensional network. This cross-linking contributes to greater strength, lower water absorption, and less polymerization shrinkage.[19] The bisGMA is the basis of most of the contemporary composite resin systems clinically used today.[20,21]

Polymerization of composite resins can be initiated chemically or by light exposure.

No-mix adhesives. No-mix adhesives set when one paste under light pressure is brought together with a primer fluid on the etched enamel and bracket backing or when another paste on the tooth is to be bonded. No-mix adhesives are now routinely used by only 5% of the orthodontists in the United States, which indicates a dramatic decline, compared with a 2008 survey.[4,5]

Light-polymerized adhesives. The introduction of light-cure adhesives not only removed a step in the bonding procedure, but it also allowed practitioners the freedom to choose when to initiate the adhesive curing cycle after bracket placement. Light-curing resin composites were introduced to the market in the 1970s. In light-cure adhesives, the curing process begins when a photoinitiator is activated. Most dental photoinitiator systems use camphoroquinone as the diketone absorber, with the absorption maximum in the blue region of the visible light spectrum at a wavelength of 470 nanometers (nm).[22,23] The light-cured adhesives are routinely used today by more than 80% of orthodontists and are dominating the market[5] (Fig. 27-3).

These resins offer the advantage of extended, although not indefinite, working time. This advantage, in turn, provides the opportunity for assistants to place the brackets, with the orthodontist following up with any final positioning. Light-cured adhesives are particularly useful in situations during which a quick set is required, such as when rebonding one loose bracket or when placing an attachment on an impacted tooth after surgical uncovering, when the risk of blood contamination is present. Light-cured adhesives are also advantageous when extended working time is desirable, which is usually the case when difficult premolar bracket positions need to be checked and rechecked with a mouth mirror before the bracket positioning is considered optimal (Fig. 27-4).

Primers. Much confusion and uncertainty surround the use of sealants and primers in orthodontic bonding. Recent findings demonstrate that bonding with or without a primer (unfilled resin) before bracket placement is equally clinically successful as far as bracket failure rate is concerned.[24,25]

FIGURE 27-3 Current light polymerized adhesives are available in either syringe or capsule form and are best used with their respective primer (Transbond XT, 3M Unitek, Monrovia, CA).

FIGURE 27-4 Bracket position on difficult teeth may be checked with a mouth mirror.

Why, then, should a sealant be of any value in bracket bonding? If nothing else, a sealant permits a relaxation of moisture control because controlling moisture is no longer critical after resin coating. Sealants also provide cover for enamel in areas of adhesive voids, which is probably especially valuable with indirect bonding. The caries protection of sealant around the bracket base is more uncertain, and further studies are needed on the clinical merits of fluoride-containing sealants.

Moisture-Insensitive Primers. In an attempt to reduce the bond failure rates under moisture contamination, hydrophilic primers that can bond in wet fields (Transbond MIP, 3M Unitek, Monrovia, CA; Assure or Assure Plus, Reliance Orthodontic Products, Itasca, IL) have been introduced as a potential solution. Laboratory studies have demonstrated that water and saliva contamination of enamel during the bonding procedure lowers bond strength values of composite resins.[26,27] Although bond strengths were significantly lower under wet conditions than in dry conditions, the hydrophilic primers may be suitable in difficult moisture-control situations.[27,28] This may be the case in some instances of second molar bonding and when the risk for blood contamination is present on half-erupted teeth and on impacted canines. For optimal results, the moisture-insensitive primers should be used with their respective adhesive resins.

The hydrophilic resin sealants or primers polymerize in the presence of a slight amount of water, but they will not overcome routine saliva contamination. When bonding to enamel, the resin sealant or resin primer must be placed onto the prepared enamel before the pellicle (biofilm) formation from the saliva, which is not particularly difficult but is crucial to a successful enamel bond.[6]

Self-Etching Primers. Despite being demonstrated to provide only modest time saving (8 minutes for full mouth bonding),[29] the use of self-etch primers has steadily increased because of their great simplicity. A recent survey among U.S. orthodontists demonstrated that the routine use of a self-etching primer (SEP) is approximately 40%, which indicates a 10% increase in the past 6 years.[5] Combining conditioning and priming into one step may improve cost-effectiveness for clinicians and patients, provided the clinical bond failure rates are not significantly increased.

The unique characteristic of these bonding systems is that they combine the conditioning and priming agents into a single acidic primer solution for simultaneous use on both enamel and dentin; therefore separate acid etching of the enamel and subsequent rinsing with water and air spray is not required (Fig. 27-5). The active ingredient of the SEPs is a methacrylate phosphoric acid ester that dissolves calcium from hydroxyapatite. Rather than being rinsed away, the removed calcium forms a complex and is incorporated into the network when the primer polymerizes. Etching and monomer penetration to the exposed enamel rods are simultaneous, and the depth of etch and primer penetration is identical.

Three mechanisms act to stop the etching process. First, the acid groups attached to the monomer are neutralized by forming a complex with calcium from hydroxyapatite. Second, as the solvent is driven from the primer during the airburst step, the viscosity rises, slowing the transport of acid groups to the enamel interface. Finally, as the primer is light cured and the primer monomers are polymerized, transport of the acid groups to the interface is stopped.[30] Scanning electron microscopy (SEM) examination of the impression of SEP-treated enamel shows different surface characteristics from acid-etched enamel. Instead of the well-known distinct honeycombed structure with microtag and macrotag formation (Fig. 27-6, *A–F*), an irregular but smooth hybrid layer, 3 to 4 microns thick, and irregular tag formation with no apparent indentations of enamel prism or core material (Fig. 27-7, *A–C*) are found. The minimal etch obtained with the SEPs indicates that the majority of the bond may be more of a chemical bond with the calcium in the enamel than the mechanical bond achieved with a conventional phosphoric acid etch.[6]

The use of the new SEPs for orthodontic purposes has been extensively evaluated, and clinical bond strengths using SEPs may appear to be lower than those with conventional etching and priming.[31,32] Another recent systematic review and meta-analysis concludes that only weak insignificant evidence suggests higher odds of failure with an SEP than an acid etch over 12 months in orthodontic patients.[29] On the other hand, information on the effect of SEP application on enamel resistance against demineralization is controversial. Previous studies[33] show that the SEP provides no resistance to enamel demineralization and results in twice as many white spot lesions (WSLs), especially in patients with poor oral hygiene.[34] Recent studies, however, show that the enamel samples that are conditioned with the fluoride-releasing SEP (Transbond Plus, 3M Unitek, Monrovia, CA) display better remineralization.[35]

When deciding which SEP to use, each clinician must weigh bond failure rates and reduced resistance to demineralization against the time saved in bonding and debonding.

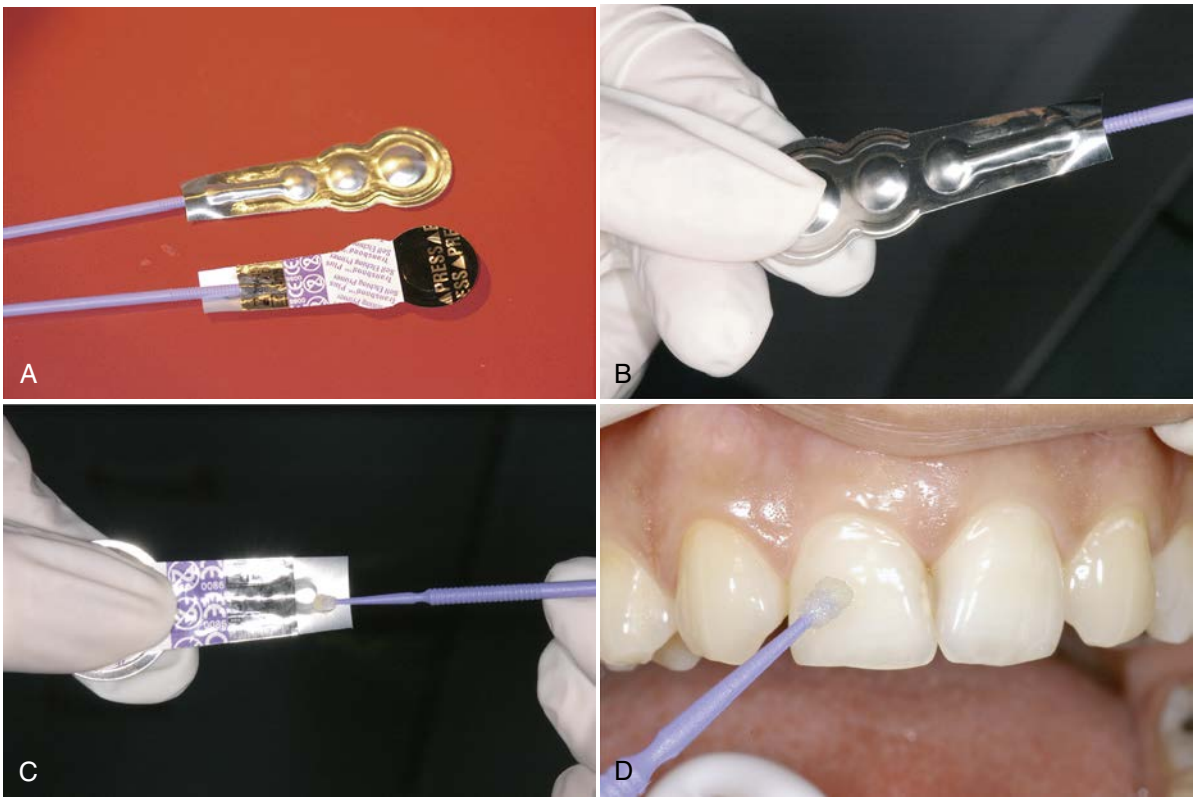


FIGURE 27-5 Application of a self-etching primer (Transbond Plus, 3M Unitek, Monrovia, CA) on enamel surface of maxillary incisor (see text for explanation).

Cytotoxicity of orthodontic resins. Regardless of the polymerization method, in vitro studies have shown that the polymerization reaction that produces the cross-linked polymer matrix from the dimethacrylate resin monomer is never complete, and approximately 15% to 50% of the methacrylic groups remain unreacted (32.4% and 44.5% for Transbond LR [3M Unitek, Monrovia, CA] and Lightcure LR [Reliance Orthodontic Products, Itasca, IL] orthodontic adhesives, respectively).[36–39] As a result of the efforts of the industry, the percentage of unbound monomers has decreased in the past 10 years, but the problem is still not eradicated. The quantity of residual monomers is less than one-tenth of the remaining methacrylic groups, which have been evaluated as no more than 1.5% to 5%, which is enough to contribute to major cytotoxic effects.[36,40]

Monomers identified in orthodontic composites (Transbond XT, Transbond LR [3M Unitek, Monrovia, CA]; Reliance Light Bond, Reliance FlowTain [Reliance Orthodontic Products, Itasca, IL] GC Fuji Ortho LC [GC America, Alsip, IL]) by liquid chromatography include bisGMA, triethylene glycol dimethacrylate (TEGDMA), urethane dimethacrylate (UDMA), and 2-hydroxylethyl methacrylate (HEMA) in the 0- to 99.8-μm range.[41] Resins and resin-modified glass ionomer cement (RM-GIC) also release ions such as fluoride, strontium, and aluminum.[36] These unbound free monomers seem to be directly responsible for the cytotoxicity of resin composites on pulp and gingival cells, and they are probably also implicated in the allergic potential of the material.[39] Recent in vivo research[42] shows morphologic signs of cytotoxicity of buccal cells after 6 months of fixed orthodontic treatment with different light-cured composites, with no genotoxic effects; and cell culture studies[43] suggest some adverse effects on cell viability with various current orthodontic adhesives. However, studies assessing orthodontic adhesives with a protocol that mimics the orthodontic bonding process are those of Eliades and colleagues,[44,45] which report no release of bisphenol-A or estrogenic effect. Another current systematic review[46] was unable to draw definitive conclusions as a result of a variety of setups and the different units allied to the diversity of reporting in different studies. For now, the conclusion can be reached that no controversy exists in the orthodontic community regarding the safety of the most commonly used materials. However, some simple and basic precautions may help fight the adverse effects of these materials for the patient population. First of all, the amount of composite resin used should be kept to a minimum, and any excess resin (flash) around the orthodontic attachments should be removed before the resin is polymerized. Minimizing the use of adhesive material may be of high importance when bonding fixed orthodontic retainers since these are left in the oral environment for a long time and are exposed to the cavity, unlike resin beneath the bracket base.[10] Degree of cure-conversion (DC) is another important factor, and current research emphasizes matching composites and light-curing units with one another to achieve satisfactory maximal biocompatibility and DC.[43] In addition, the speed of monomer release is at its maximum in the first 10 to 60 minutes.[41] Having a patient wash his or her mouth immediately after the bonding session and/or having him or her spit into a disposable cup for the first 30 minutes might be advisable as it is used after topical fluoride applications.[10]

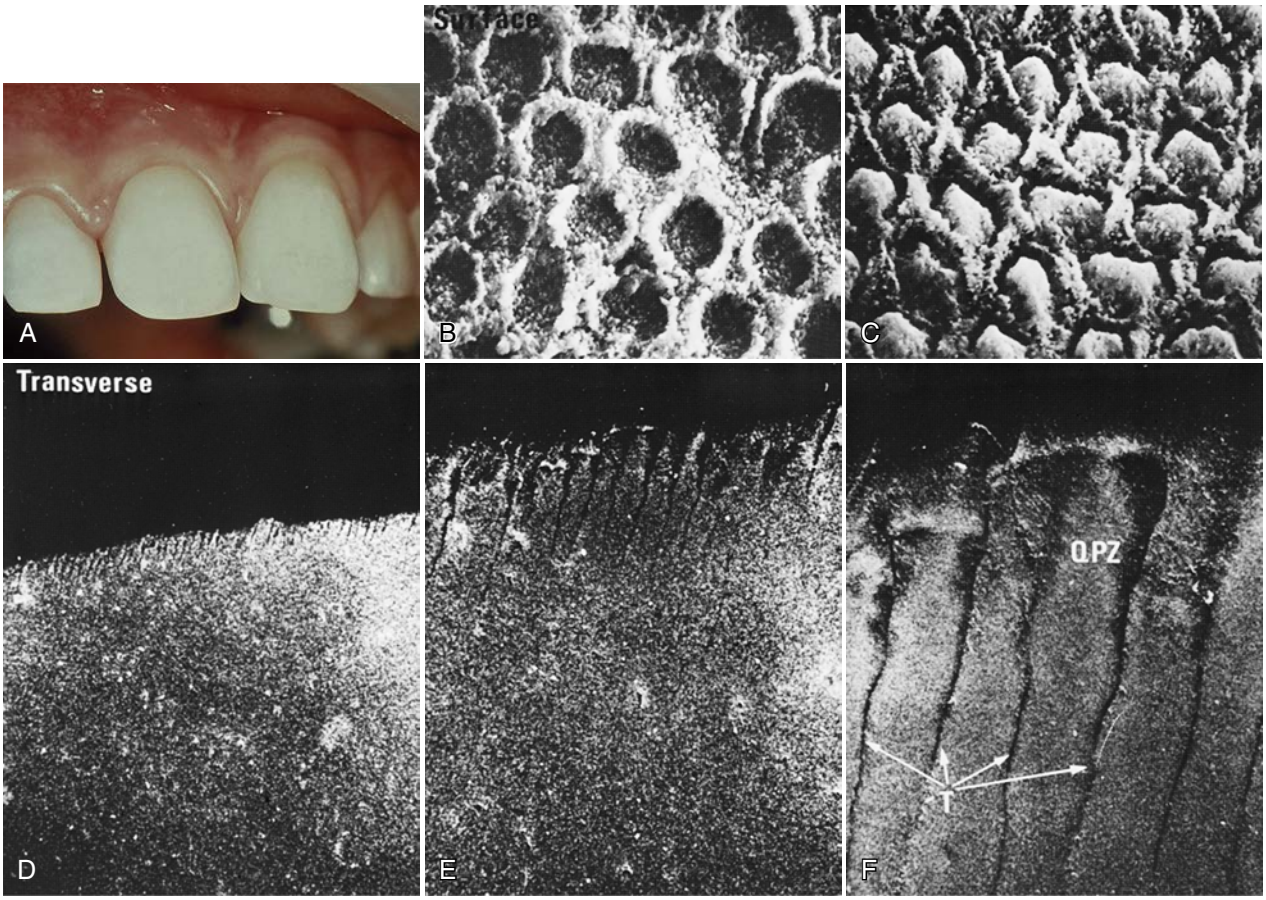


FIGURE 27-6 Acid-etch conditioning of enamel before bracket bonding. **A,** Frosty white appearance. **B, C,** Scanning electron micrograph of an enamel surface that has been etched with 37% phosphoric acid. (In **B** the prism centers have been preferentially removed, whereas in **C** the loss of prism peripheries demonstrates the head-and-tail arrangement of the prisms.) **D–F,** Transverse section of an etched porous enamel surface showing two distinct zones, the qualitative porous zone (QPZ) and the quantitative porous zone. In the latter, an even row of resin tags (*T*) may penetrate.

Glass Ionomer Cements

Glass ionomer cements were introduced in 1972, primarily as luting agents and as a direct restorative material, with unique properties for chemically bonding to enamel, dentin, and stainless steel and being able to release fluoride ions for caries protection. The second-generation water-hardening cements contain the same acids in freeze-dried form or in an alternative powdered copolymer of acrylic and maleic acids. Glass ionomer cements were modified to produce dual-cure or hybrid cements[19] ([Fig. 27-8], e.g., GC Fuji Ortho LC, GC America, Alsip, IL).

Glass ionomer and light-cured glass ionomer cements are the material of choice for cementing bands[5]; they are stronger than zinc phosphate and polycarboxylate cements, with improved adhesion to enamel and metal and less demineralization[47] at the end of treatment.

Glass ionomers are used by only 7% of the clinicians for direct bonding of brackets. For bonding of brackets and buccal tubes with resin-modified glass ionomer cements, few reports over a substantial time have been made with regard to clinical performance concerning the bond strength and incidence of demineralization. The pretreatment with polyacrylic acid facilitates a chemical bond between the glass ionomer and the enamel and thus should be performed before bracket bonding with the glass ionomer. When bond strength is the primary criterion for selecting an adhesive, composite resins are recommended.[48] Limiting the use of the glass ionomer with at-risk orthodontic patients is advisable to provide preventive actions and potentially remineralize early (subclinical) enamel demineralization.[47]

Light Sources

The variety of light sources available includes conventional and fast halogen lights, argon lasers, plasma arc lights, and light-emitting diodes (LEDs). A current systematic review and metaanalysis of randomized controlled trials and clinical controlled trials directly compare conventional halogen lights, LEDs, or plasma arc systems involving patients with full arch, fixed, or bonded orthodontic appliances (not banded) with follow-up periods of a minimum of 6 months. This systematic review suggests that no evidence supports the use of one light-cure type over another, based on the risk of attachment failure.[49] On the other hand, recent research demonstrates that LEDs today dominate not only the orthodontic but also the

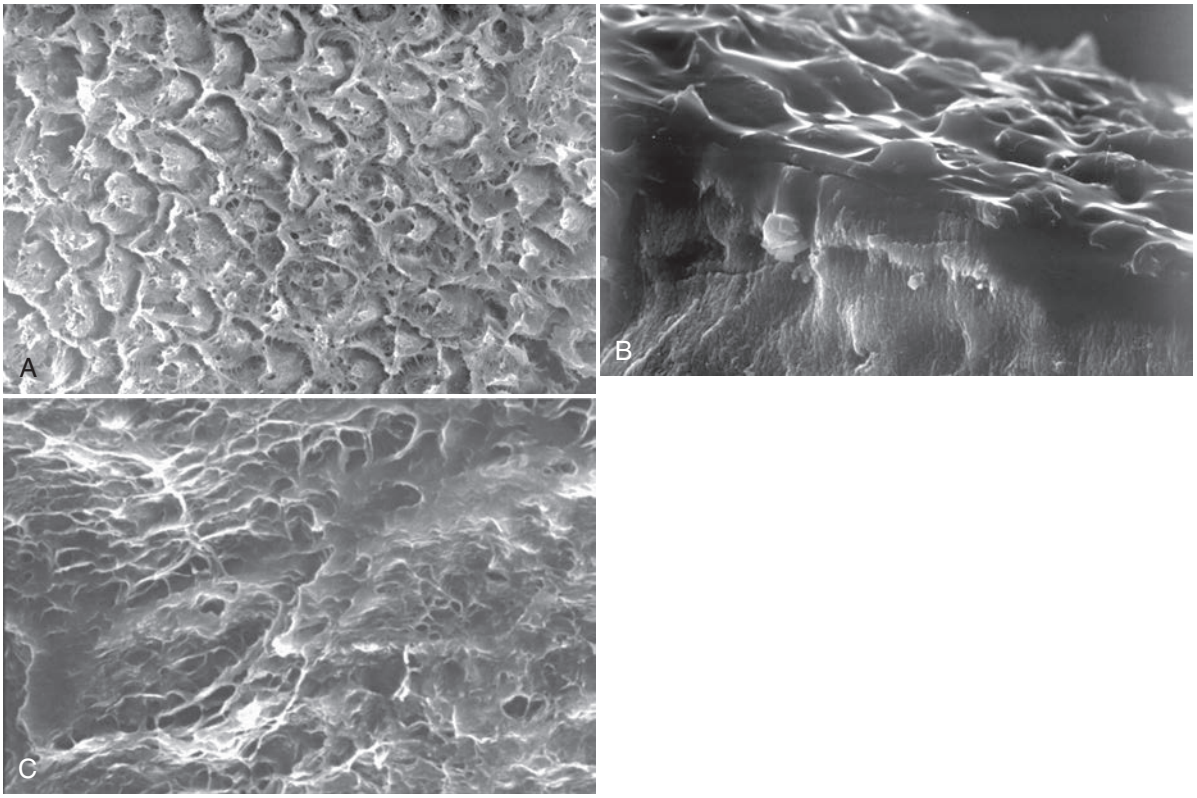


FIGURE 27-7 Comparison of scanning electron microscopy views of adhesive under the bracket base after phosphoric acid etching and the use of self-etching primer (Transbond Plus, 3M Unitek, Monrovia, CA). **A,** Adhesive is demonstrated under the bracket base after the removal of phosphoric acid–etched enamel. Note the exact replica of honeycomb appearance (×1500). **B,** Cross section shows Transbond Plus–treated enamel and outer surface of Transbond Plus layer on enamel (×2000). **C,** Adhesive is demonstrated under the bracket base after the complete removal of the Transbond Plus–treated enamel (×1500).

FIGURE 27-8 GC Fuji Ortho LC Automix (GC America, Alsip, IL) is a light-cured, resin-reinforced orthodontic glass ionomer adhesive that is used in combination with a 20% polyacrylic acid conditioner designed to etch the enamel mildly. (Courtesy of GC America, Alsip, IL.)

whole dental field.[5] Therefore only this group is discussed in detail in this chapter.

Light-Emitting Diodes

A solid-state LED technology for the polymerization of light-activated dental materials was proposed by Mills and colleagues.[50] LEDs use junctions of doped semiconductors to generate light instead of the hot filaments used in halogen bulbs.[51] They have a lifetime of over 20,000 hours and undergo little degradation of output over this time.[52] LEDs do not require filters to produce blue light, are resistant to shock and vibration, and take little power to operate.[50] Earlier LED designs provided unsatisfactory results with metal brackets, possibly attributable to their low power output.[53] Current LEDs, however, manage to combine high power output (from 1000 to 3200 mW/cm^2) with a very narrow wavelength range around 465 nm, which very nicely matches the absorption peak of camphoroquinone (Fig. 27-9).[10]

BONDING

Bonding of brackets and other orthodontic attachments is one of the most important stages of the whole treatment process. The simplicity of bonding can be misleading. The technique undoubtedly can be misused, not only by an inexperienced clinician but also by more experienced orthodontists who do not perform procedures with care.[19] Minor errors in this stage will be dramatically reflected in the active treatment phase in forms of improper alignment of teeth, premature failure of attachments, which will require time-consuming and costly replacements, and increased susceptibility to the formation of demineralization around the attachment. Success in bonding requires an understanding of and adherence to the accepted principles of orthodontic and preventive dentistry, which are cleaning the adhesive surfaces, providing good wetting, providing intimate

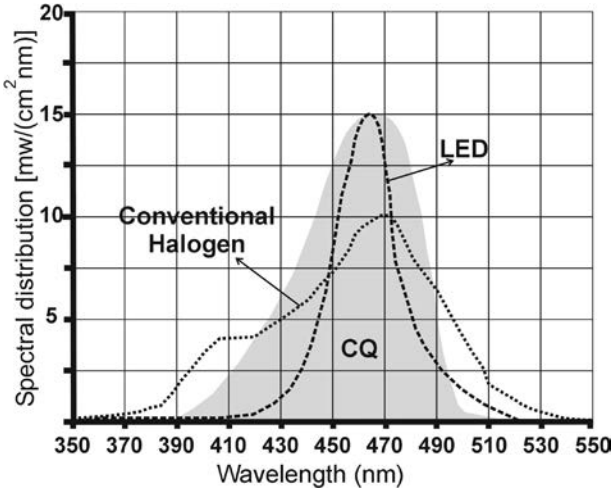


FIGURE 27-9 The spectral distribution and wavelength graphic demonstrates that the peak of light-emitting diode (LED) light sources better coincides with the absorption peak of camphoroquinone, which means that a photon emitted by an LED source is more likely to activate a camphoroquinone (CQ) molecule. (From Usumez S, Erverdi N. Adhesives and bonding in orthodontics. In: Nanda R, Kapila S, eds. *Current Therapy in Orthodontics*. 1st ed. St. Louis: Mosby; 2009:45–67.)

FIGURE 27-10 The Dri-Angle (Patterson Dental, St. Paul, MN) is an improvement on the cotton roll in the parotid area. The Dri-Angle covers the parotid or Stensen duct to restrict the flow of saliva. (Courtesy of Dental Health Products, Inc.)

adaptation, and making use of maximum bond strength and adequate curing (polymerization).[10]

Bonding to Enamel

Premedication

Direct or indirect bonding of orthodontic attachments is a pain-free procedure, whereas initial activation of the orthodontic attachments may cause significant discomfort to the patient, particularly after a few hours. Recent research reveals that patients premedicated with 550 mg naproxen sodium 1 hour before archwire placement have significantly lower levels of pain at 2 hours, 6 hours, and nighttime after adjustment than patients taking a placebo.[54] Analgesic premedication may be considered for selected patients before proceeding to the bonding stage.[10]

The steps of direct or indirect bracket bonding on facial or lingual surfaces of teeth are as follows:

- Cleaning
- Enamel conditioning
- Sealing and priming
- Bonding

Cleaning

An ideal bonding surface should be free of any debris, and cleaning of the teeth with pumice will remove plaque and the organic pellicle that normally covers all teeth.[55] Care must exercised to avoid traumatizing the gingival margin and initiating bleeding on teeth that are not fully erupted.[19]

The need for conventional pumice polishing before acid etching has been questioned[56,57] since neither bond strength nor enamel surface etch pattern is altered by pumicing clean enamel.[58] However, pumice prophylaxis does not appear to affect the bonding procedure adversely, and cleaning the tooth may be advisable to removing plaque and debris that otherwise might remain trapped at the enamel–resin interface, particularly when bonding posterior teeth that are sometimes out of reach of efficient brushing activity.[59] On the other hand, it should be noted that pumicing before the use of an SEP is crucial (see related section).[59,60]

Enamel Conditioning

Moisture control. Salivary control and maintenance of a dry working field are essential after rinsing and drying. Many devices on the market accomplish this:

- Lip expanders and cheek retractors
- Saliva ejectors
- Tongue guards with bite blocks
- Salivary duct obstructors
- Gadgets that combine several of these devices
- Cotton or gauze rolls

These products are being continually improved, and the clinician must decide which devices work best. For simultaneous molar-to-molar bonding in both arches, a technique using lip expanders (Dri-Angles, Patterson Dental, St. Paul, MN) to restrict the flow of saliva from the parotid duct (Fig. 27-10) and saliva ejectors both work well.[19]

Present research indicates that antisialagogue agents do not present a statistically significant effect on the observed bond failure rates and generally are not needed for most patients.[61]

Enamel pretreatment

Conventional Acid Etching. Isolation of the operative field is followed by etching of the enamel surface. An untouched enamel surface is hydrophobic, and wetting is limited, which makes bonding to an intimate enamel surface a challenging procedure. An enamel pretreatment or surface conditioning is necessary to make successful bonds. This pretreatment is usually accomplished by etching the surface using various acids. The most commonly used etchant is 37% orthophosphoric acid for 15 to 30 seconds.[4,5]

At the end of the etching phase, the etchant is rinsed off the teeth with abundant water spray. A high-speed evacuator is strongly recommended for increased efficiency in collecting the etchant-water rinse and to reduce moisture contamination

on the teeth and Dri-Angles (Patterson Dental, St. Paul, MN). Salivary contamination of the etched surface should best be avoided. (If contamination occurs, then rinsing with the water spray or re-etching for a few seconds is recommended; the patient must not rinse.) Since blood contamination has been shown to decrease the shear bond strength (SBS), teeth that are contaminated with blood should be rerinsed and dried.[62–64] Applying a protective liquid polish (BisCover, Bisco, Inc., Schaumburg, IL) to the etched surface before any contamination occurs has been shown to prevent the untoward effects of blood contamination.[62] This product may be beneficial in difficult bonding areas such as partially erupted or impacted teeth.

Next, the teeth are thoroughly dried with a moisture and oil-free air source to obtain the well-known dull, frosty appearance (see Fig. 27-6, *A*). Teeth that do not appear dull and frosty white should be re-etched.

This procedure probably reflects the general use of acid etching in orthodontics. Although considerable discussion of several aspects of enamel pretreatment remains, most of the debate concerning acid etching appears to be of limited clinical significance, because, apparently, good bond strength significantly depends more on both avoiding moisture contamination and achieving undisturbed setting of the bonding adhesive than on variations in the etching procedures.[19]

Some areas of debate and some short answers are provided:

Should the etching cover the entire facial enamel or only a small portion outside the bracket pad? Although laboratory research indicates increased susceptibility for WSLs by surplus orthodontic etching exceeding the bracket base area,[65] clinical experience over more than 25 years indicates that etching the entire facial enamel with solution is harmless—at least when a fluoride mouth rinse is regularly used.

Are gels preferable to solutions? Despite the fact that the liquid form is suggested for scientific studies, no apparent difference exists in the degree of surface irregularity after etching with an acid solution compared with etching with an acid gel.[66] Gels provide better control for restricting the etched area but may require a more thorough rinsing afterward.

What is the optimal etching time? Is it different for young and old teeth? Studies[66–70] and clinical experience indicate that 15 to 30 seconds is probably adequate for etching most young permanent teeth. However, important individual variation exists in enamel solubility among patients, between teeth, and within the same tooth. One benefit of conventional acid etching is that it tends to neutralize the differences among individuals and between teeth. Thus phosphoric acid etching of sufficient time can compensate for those individuals whose enamel is more resistant to acid.

Can recently bleached teeth be safely bonded? Today, bleaching is an increasing trend, and current information is conflicting, with some research[71] indicating no adverse effect, whereas other studies indicate that bleaching with 35% hydrogen peroxide significantly reduces bracket adhesion when bonded 24 hours after bleaching. However, no significant adverse effect of bleaching seems to occur after 7 days.[72] Therefore postponing the bonding procedure approximately 1 to 4 weeks for recently bleached teeth may be a good practice.

What is the preferred procedure for deciduous teeth? A recommended procedure for conditioning deciduous teeth is to sandblast with 50 microns of aluminum oxide for 3 seconds to remove some outermost aprismatic enamel and then etch for 30 seconds with phosphoric acid gel. The failure rate with this procedure for the authors of this text is less than 5%.[19]

Is prolonged etching necessary when teeth are pretreated with fluoride? Clinical and laboratory experience[66,67] indicates that extra etching time is not necessary when teeth have been pretreated with fluoride. When in doubt, the enamel should be checked for the uniformly dull and frosty white appearance after etching; if the desired appearance is confirmed, then surface retention is adequate for bonding. On the other hand, recent research shows that an adhesion promoter (Shofu, Inc., Kyoto, Japan) can recover the bond strength reduced by the long-term repeated topical applications of fluoride to the pre-fluoridation level.[73]

Is etching permissible on teeth with internal white spots, or is it more likely that the etchant will open up underlying demineralized areas? Caution should be exercised when etching over acquired and developmental demineralizations. The procedure is best avoided. If avoidance is impossible, then a short etching time, the application of a sealant or primer, and the use of direct bonding with extra attention to not having areas of adhesive deficiency are important. The presence of voids, together with poor hygiene, can lead to metal corrosion[74] and indelible staining of underlying developmental white spots.[75]

How much enamel is removed by etching, and how deep are the histologic alterations? Are they reversible? Is etching harmful? A routine etching removes from 3 to 10 microns of surface enamel.[75–78] Another 25 microns reveal subtle histologic alterations,[79–81] creating the necessary mechanical interlocks (see Fig. 27-1). Deeper localized dissolutions generally cause penetration to a depth of approximately 100 microns or more.[79,81,82] Although laboratory studies indicate that the enamel alterations are largely (although not completely) reversible,[81,83] the overall effect of applying an etchant to healthy enamel is not detrimental. This point is augmented by the fact that normal enamel is from 1000 to 2000 microns thick,[82,84] except where it tapers toward the cervical margin. Abrasive wear of facial enamel is normal and proceeds at a rate of up to 2 mm per year, and facial surfaces are self-cleaning and not prone to caries.[2]

What are other alternatives to etching with phosphoric acid (e.g., polyacrylic acid, maleic acid, SEPs)? The use of polyacrylic acid with residual sulfate is reported[74] to provide retention areas in enamel similar to those after phosphoric acid etching with less risk of enamel damage at debonding. However, other researchers have found much weaker bonds.[85–88] Research shows that 10% maleic acid, which is believed to decrease mineral loss alone, may produce similar bond strengths to 37% phosphoric acid.[89–93] However, the use of these milder acids has never been popularized.

Laser Etching. Laser treatment of dental enamel causes thermally induced changes within the enamel to a depth of 10 to 20 µm, depending on the type of laser and the energy applied to the enamel surface, in effect, etching takes place through a process of continuous vaporization and microexplosions due to vaporization of water trapped within the hydroxyapatite matrix (Fig. 27-11, *A–H*).[10] The degree of surface roughening is dependent on the system used and the wavelength of the laser.[94,95] The previous research demonstrated that achieved SBS diverge.[94,96,97] However, more recent findings with improved power and irradiation settings demonstrate more consistent enamel surface alterations and SBS values without any thermal damage.[98,99]

FIGURE 27-11 **A–F,** Lasers can be used to reshape the gingiva and to etch the enamel effectively with specific power settings (Gold Handpiece, Biolase Technology, Inc., Irvine, CA). **G,** With some new handpieces, the size and shape of the area to be etched can well be controlled (X-Runner handpiece, Fotona Technology, Ljubljana, Slovenia; San Clemente, CA). **H,** Laser irradiation causes thermal-induced changes within the enamel to a depth of 10 to 20 μm, depending on the type of laser and the energy applied to the enamel surface. The results regarding achieved shear bond strength diverge; in general, lasers are unable to produce a standard and reliable etching pattern at the moment. (**A–G,** Courtesy of Drs. Aslihan Usumez and Sertac Aksakalli, Istanbul, Turkey. **H,** From Usumez S, Erverdi N. Adhesives and bonding in orthodontics. In: Nanda R, Kapila S, eds. *Current Therapy in Orthodontics*. 1st ed. St. Louis: Mosby; 2009;45–67.)

The surface produced by laser etching is also claimed to be acid resistant as a result of the modified calcium-to-phosphorus (Ca/P) ratio, a reduced carbonate-to-phosphate ratio, and the formation of more stable and less acid-soluble compounds, thus reducing susceptibility to acid attack and caries.[100,101] Despite that it was shown[102] that subablative erbium:yttrium aluminium garnet (Er:YAG) laser irradiation before acid etching with a topical application of fluoride varnish increases the microhardness of enamel without reducing the SBS, current research[103] indicates that laser treatment of enamel does not significantly affect the mean percentage weights of elements. Therefore taking advantage of laser-induced caries resistance through an altered Ca/P ratio seems questionable in dental practice.

Sealing and Priming

After the teeth are completely dry and frosty white, a thin layer of bonding agent (sealant, primer) may be painted over the etched enamel surface. The coating may be thinned by a gentle air burst for 1 to 2 seconds (Fig. 27-12, *A* and *B*). A thick layer may cause drifting before curing is initiated and may interfere with the precise adaptation of the bracket base. Bracket placement should be immediately started after all of the etched surfaces are coated. Separate curing of the bonding agent is not necessary when light-cured products are used. The layer may be precured in hard-to-reach areas where moisture contamination is likely. Reapplication of the sealed layer is not required when saliva contamination occurs, but the area should be air dried before bracket placement.

Bonding

Immediately after all teeth to be bonded have been painted with a sealant or primer, the operator should proceed with the actual bonding of the attachments. At present, the majority of clinicians routinely bond brackets with the direct rather than the indirect technique. According to a 2014 survey in the United States, approximately 90% of orthodontists routinely use direct bonding. Indirect bracket bonding is routinely used labially by

approximately 18% of orthodontists, which indicates an increase in its use.[5] Only 15% of the specialists in the United States are routinely using the chemically cured one- or two-paste adhesives (this was 50% in 2008), whereas 80% of those who have adopted the light-initiated bonding resins prefer the no-mix versions.[4,5]

Many different adhesives exist for direct bonding, and new ones continually appear. However, the basic bonding technique is only slightly modified for varying materials, according to each manufacturer's instructions. The easiest method of bonding is to add a slight excess of adhesive to the backing of the attachment (Fig. 27-13) and then place the attachment on the tooth surface in its correct position.

When bonding attachments one at a time with a light-cured adhesive, the operator can work in a relaxed manner and obtain optimal bond strength for each bracket. Hurrying is not necessary; plenty of time is available for placing the bracket in its

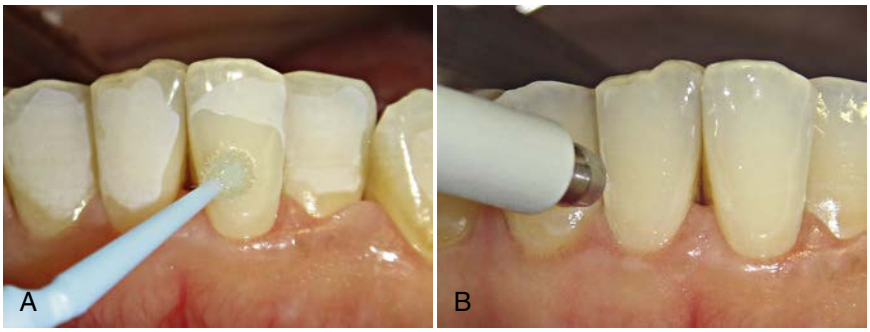


FIGURE 27-12 **A,** A thin layer of bonding agent (primer) is painted over the entire etched surface. **B,** The coating may be thinned by a light burst of air to avoid drifting of the attachment before curing and to improve its adaptation. (Courtesy of Dr. Sabri Ilhan Ramoglu, Istanbul, Turkey.)

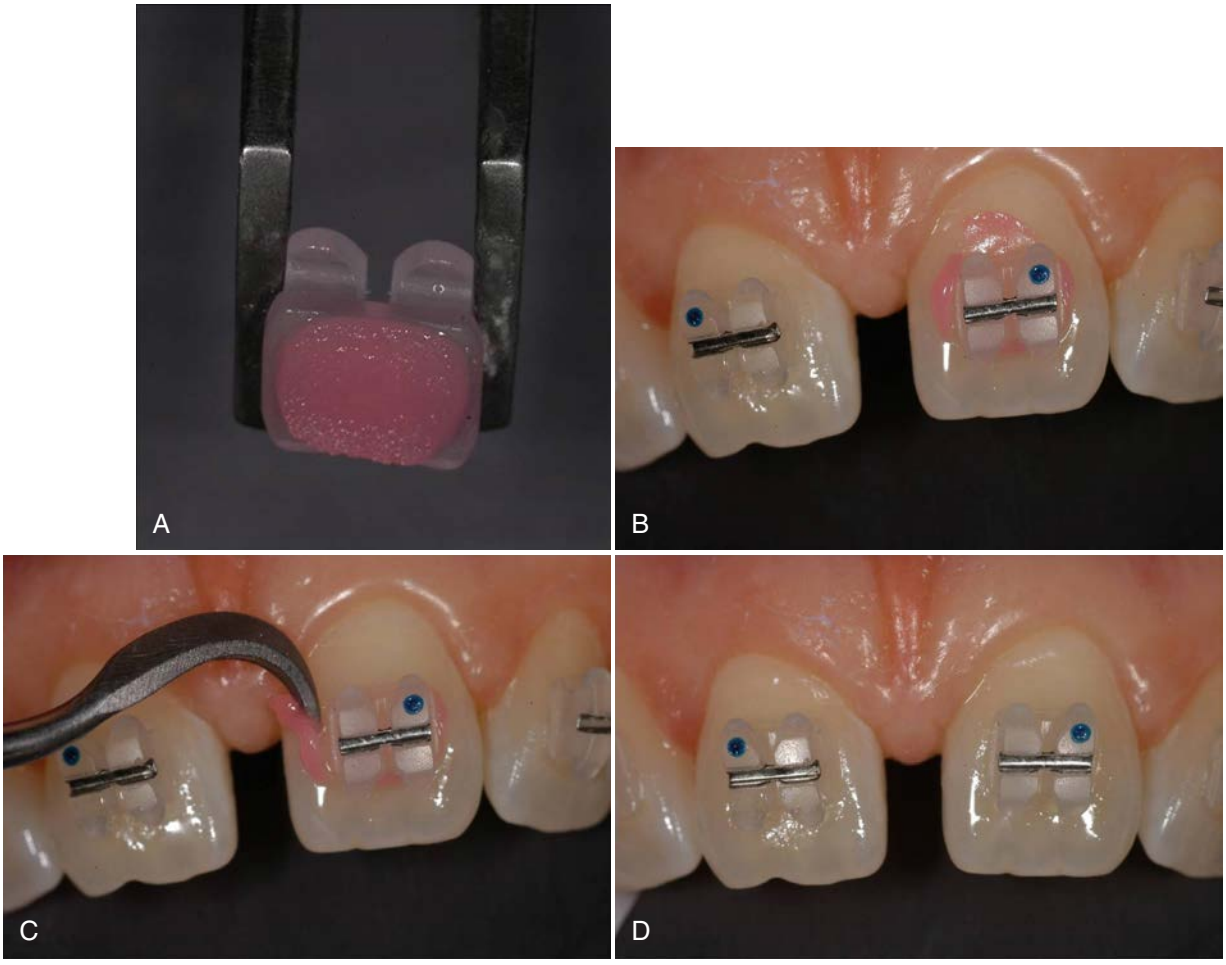


FIGURE 27-13 Direct bracket bonding with light-cured color-change adhesive resin. **A,** Adhesive resin on contact surface of bracket. **B,** Bracket transferred to tooth surface and oriented with placement scaler. **C,** Excess adhesive (*pink color*) is removed with the scaler before light activation. **D,** No color of adhesive resin after curing.

correct position, checking it, and, if necessary, repositioning it before light curing.

The recommended bracket bonding procedure[1,104] (with any adhesive) consists of the following steps[10]:

1. Transfer
2. Positioning
3. Fitting
4. Removal of excess
5. Curing

Transfer. The clinician grips the bracket with reverse action tweezers and then applies the mixed adhesive to the back of the bonding base. The clinician immediately places the bracket on the tooth, close to its correct position (see Fig. 27-13, *B*).

Positioning. The mandibular molar and premolar bracket wings must be kept out of occlusion, or the brackets may easily come loose. Therefore before positioning the brackets, the operator should do the following:

1. The patient is asked to bite with his or her teeth together; the operator should then evaluate the tooth area available for bonding.
2. The mandibular posterior brackets are bonded out-of-occlusion, which may necessitate adjusting bends in the archwires.

Later, the clinician uses a placement scaler to position the brackets mesiodistally and incisogingivally and to angulate them accurately, relative to the long axis of the teeth. Proper vertical positioning may be enhanced by different measuring devices or height guides.[105] A mouth mirror will aid in horizontal positioning, particularly on rotated premolars (see Fig. 27-4). Because of human limitations in the direct placement of brackets on both anterior and posterior teeth, using archwire bends or bracket repositioning to compensate for the inherent inaccuracies in bracket positions is still necessary.[105] Placing the brackets too far gingivally is important to avoid, unless dictated by the opposing teeth, which is sometimes the case in the lower arch. This leads to incomplete expression of the torque value built into the bracket and improper hygiene conditions. The brackets may come into contact with the gingival margin particularly after intrusive tooth movement as the gingival margin and the mucogingival junction moved in the same direction as the teeth by only 79% and 62%, respectively. A statistically significant decrease of the clinical crown length was also observed after intrusion.[106] The bite should be raised with occlusal stops from proper composite material when necessary (see Occlusal Buildup of Posterior Teeth).

Fitting. Next, the clinician turns the scaler and, with one-point contact with the bracket, firmly pushes toward the tooth surface.[107] The tight fit results in good bond strength, little material to remove on debonding, optimal adhesive penetration into bracket backing, and reduced slide when excess material peripherally extrudes. The clinician should remove the scaler after the bracket is in the correct position and should make no attempts to hold the bracket in place with the instrument. Even slight movement may disturb the setting of the adhesive. A totally undisturbed setting is essential for achieving adequate bond strength.[104]

Removal of excess. A slight bit of excess adhesive is essential to minimize the possibility of voids and to ensure that the adhesive will be buttered into the bracket backing when the bracket is being fitted. The excess is particularly helpful on teeth with abnormal morphologic structures. Excess adhesive will not be worn away by tooth brushing and other mechanical forces; it must be removed (especially along the gingival margin) with the scaler before the adhesive has set (see Fig. 27-13, *C*) or with burs after setting (Fig. 27-14).

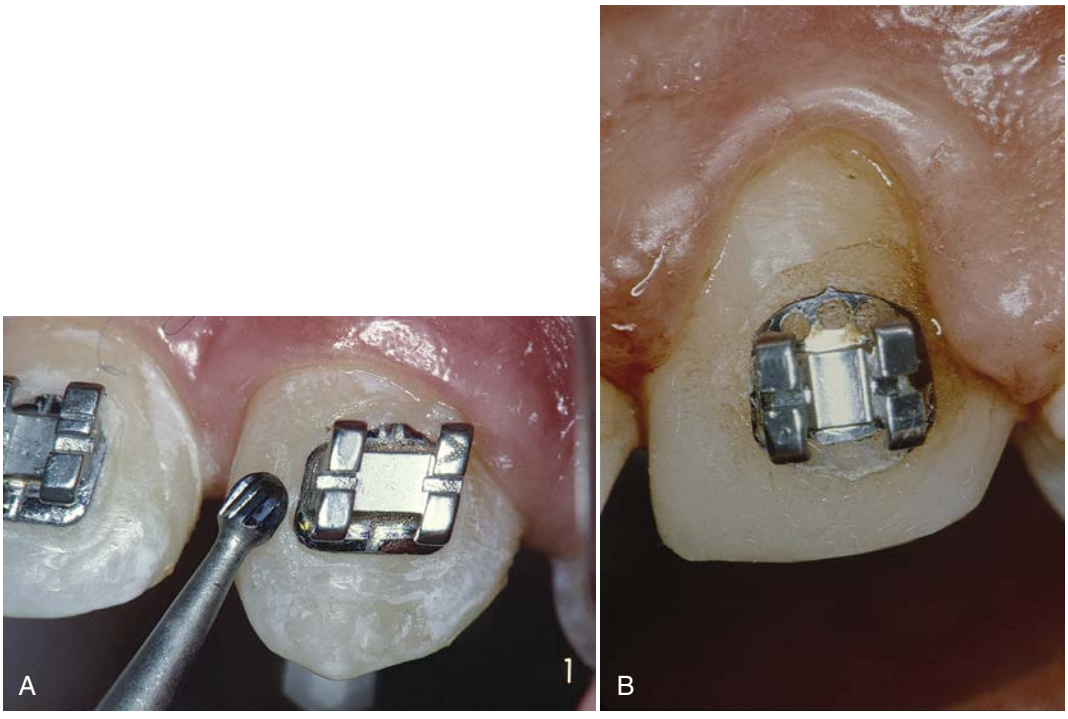


FIGURE 27-14 **A,** Use of a large (No. 7006) oval tungsten carbide bur for the removal of set adhesive around the bracket base. **B,** Relationship between excess adhesive and gingival inflammation. Note the hyperplastic gingival changes on the distal aspect, where excess adhesive is close to the gingival margin. Less reaction occurs on the mesial aspects, where adhesive is farther from the gingiva.

Removing the excess adhesive to prevent or minimize gingival irritation and plaque buildup around the periphery of the bonding base is most important (see Figs. 27-13 and 27-14). The removal of excess adhesive reduces periodontal damage and the possibility of decalcification. Clinically significant gingival hyperplasia and inflammation rapidly occur when excess adhesive comes close to the gingiva and is not properly removed.[1,104] In addition, the removal of excess adhesive can improve aesthetics, not only by providing a neat and clean appearance but also by eliminating exposed adhesive that might become discolored in the oral environment.

Some manufacturers add a coloring agent to assist in the visualization of the excess adhesive (APC II/Plus, 3M Unitek, Monrovia, CA) (see Fig. 27-14, *B* and *C*), although recent research revealed that this method does not reduce the amount of excessive adhesive around orthodontic brackets.[108] Another recent advance in this aspect is the introduction of a flash-free product (APC Flash-Free, 3M Unitek, Monrovia, CA) (Fig. 27-15). In this product, a new transparent APC adhesive formulation is contained within a form-fitting fiber mat on the base of the bracket. The manufacturer claims that when the flash-free adhesive coated bracket is placed on the tooth, the adhesive spreads out and conforms to the tooth surface, making uniform and consistent contact with no flash to clean. A recent study shows that the new flash-free adhesive performs just as well as the conventional adhesive with regard to bond quality and adhesive remnant cleanup; of the two products, clinicians preferred the flash-free adhesive over the conventional adhesive.[109] The clinical experience of the authors of this text, on the other hand, suggests that these appliances are best avoided when bonding to microdontic teeth or teeth with gross irregularities, as these brackets lack that small bit of excess adhesive that is essential to minimize the possibility of voids when a misfit between the bracket base and the enamel surface topography occurs.

When the procedure just described has been repeated for every bracket to be bonded, the clinician carefully checks the position of each bracket (see Fig. 27-4). Any attachment that is not in good position should be immediately removed with pliers and rebonded. After inserting a leveling archwire, the clinician instructs the patient how to brush properly around the brackets and archwires and gives a program of daily fluoride mouth rinses (0.05% sodium fluoride [NaF]) to follow.[110]

Curing. Once the bracket is secured in the desired position, the adhesive layer is cured with the light source. The correct setting should be preset before the curing. Today's light sources present different curing modes, including soft start modes to decrease polymerization contraction. On the other hand, polymerization contraction is irrelevant to the orthodontic setting where the bracket or the orthodontic attachment is actually free floating. Therefore presetting the light-curing unit to the maximum available setting or to the boost mode is advisable. Recent studies demonstrate no significant differences between the SBS values of brackets with curing distances of 0 to 4 mm.[111] The light is best initiated after being placed at the correct position and angulation as close to the bracket base as possible (Fig. 27-16). Divergent photon release will be avoided and curing efficiency increased if the light guide is brought into contact with the bracket after an initial cure of 1 to 2 seconds. Locating the guide tip before starting the light gun is crucial; with recent light sources, as little as even 2 or 3 seconds spent to position the guide tip correctly after shooting may well correspond to almost 40% of the total suggested curing time. Many manufacturers advise curing metal brackets from mesial and distal, direct bond molar tubes from mesial and distal or occlusal, and ceramic brackets through the bracket. Most current light sources can cure adhesives in approximately 10 or 5 seconds per metallic and ceramic brackets, respectively. A new plasma-emulating LED (VALO Ortho, Ultradent Products, Inc., South Jordon, UT) was demonstrated to cure resin under brackets in as few as 3 seconds,[43] which is also confirmed by the authors' clinical experience (Fig. 27-17). One clinical concern with this light source is the high heat reported by the patients at the gingiva, which is well tolerated when the patients are informed in advance. In the authors' recent laboratory studies, one of which was conducted under simulated blood circulation, the temperature increase in the pulp chamber was confirmed at 1.74° C, which is significantly lower than those resulting from longer exposures at lower power settings.[112,113] However, not every resin is compatible with every light source, and each combination should be individually assessed to achieve optimal results.[53,114,115]

FIGURE 27-15 The new flash-free product removes a step in bonding and allows the clinician to move directly from bracket placement to curing. (Courtesy of 3M, © 2016 3M. All rights reserved.)

FIGURE 27-16 The light guide is placed as close to the bracket base as possible and started to get the most benefit from the light energy. Physically touching the light guide to the bracket after 1 to 2 seconds is best to reduce divergent photon release.

Bonding to Artificial Tooth Surfaces

The number of adult patients being referred to orthodontic offices is steadily increasing,[5] and many adult patients have crown and bridge restorations fabricated from porcelain and precious metals in addition to amalgam restorations of molars. Banding becomes difficult, if not impossible, on the abutment teeth of fixed bridges. Recent advances in materials and techniques indicate, however, that effective bonding of orthodontic attachments to nonenamel surfaces now may be possible.[116,117] Particularly, the MicroEtcher (Danville Materials, Inc., San Ramon, CA) (Fig. 27-18), which uses 50-micron white or 90-micron tan aluminum oxide particles at approximately 7 kg/cm^2 pressure, has been advantageous for bonding to different artificial tooth surfaces. This tool is also useful for tasks such as rebonding loose brackets, increasing the retentive area inside molar bands,[13] creating micromechanical retention for bonded retainers, and bonding to deciduous teeth.

FIGURE 27-17 New high-intensity light-emitting diode (LED) curing lights are able to save the clinician chair time by reducing the curing time of orthodontics attachments to as few as seconds per tooth. (Courtesy of Ultradent Products, Inc., South Jordon, UT.)

Bonding to Porcelain

Most dental ceramic and metal ceramic crowns, bridges, and veneers are presently made from different feldspathic porcelains containing 10% to 20% aluminum oxide. However, such restorations can also be made from high-aluminous porcelains and glass ceramics.[118]

Several studies have reported that sandblasting porcelain alone is not suitable, but the addition of silane treatment may produce in vitro bond strengths that should be clinically successful[119]; however, these claims have not been verified by the authors' experiences, attributable to unacceptably high failure rates.

A more common and successful alternative to create an optimal retentive surface is the concept of etching the porcelain surface. The most commonly used porcelain etchant is 9.6% hydrofluoric acid in gel form. Although etching times vary anywhere from 1 to 2 minutes, this porcelain etchant has been reported to yield satisfactory results, and the gold standard seems to be 2 minutes, followed by silane application.[119–121] In the authors' hands, the addition of silane after hydrofluoric acid treatment did not significantly influence the bond strengths (failure rates of 8.2% versus 8.6%) (Figs. 27-19 and 27-20).

Hydrofluoric acid is strong and requires separately bonding to other teeth, careful isolation of the working area, cautious removal of the gel with a cotton roll, rinsing with high-volume suction, and immediate drying and bonding (see Fig. 27-19). The etchant creates microporosities on the porcelain surface that

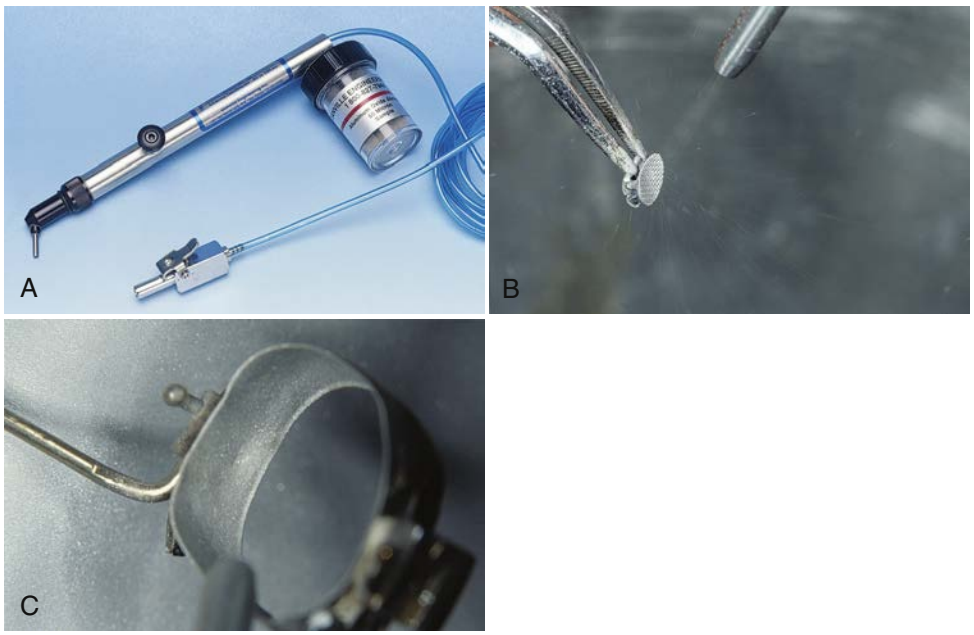


FIGURE 27-18 The MicroEtcher II (Danville Materials, Inc., San Ramon, CA) is an intraoral sandblaster approved by the U.S. Food and Drug Administration that is most useful for preparing microretentive surfaces in metals and other dental materials, whenever needed. **A,** The appliance consists of a container for the aluminum oxide powder, a pushbutton for fingertip control, and a movable nozzle where the abrasive particles are delivered. The MicroEtcher II is also useful for removing old composite resin and improving the retentive surface of loose brackets before rebonding (**B**) and the inside of the stainless steel molar bands (**C**).

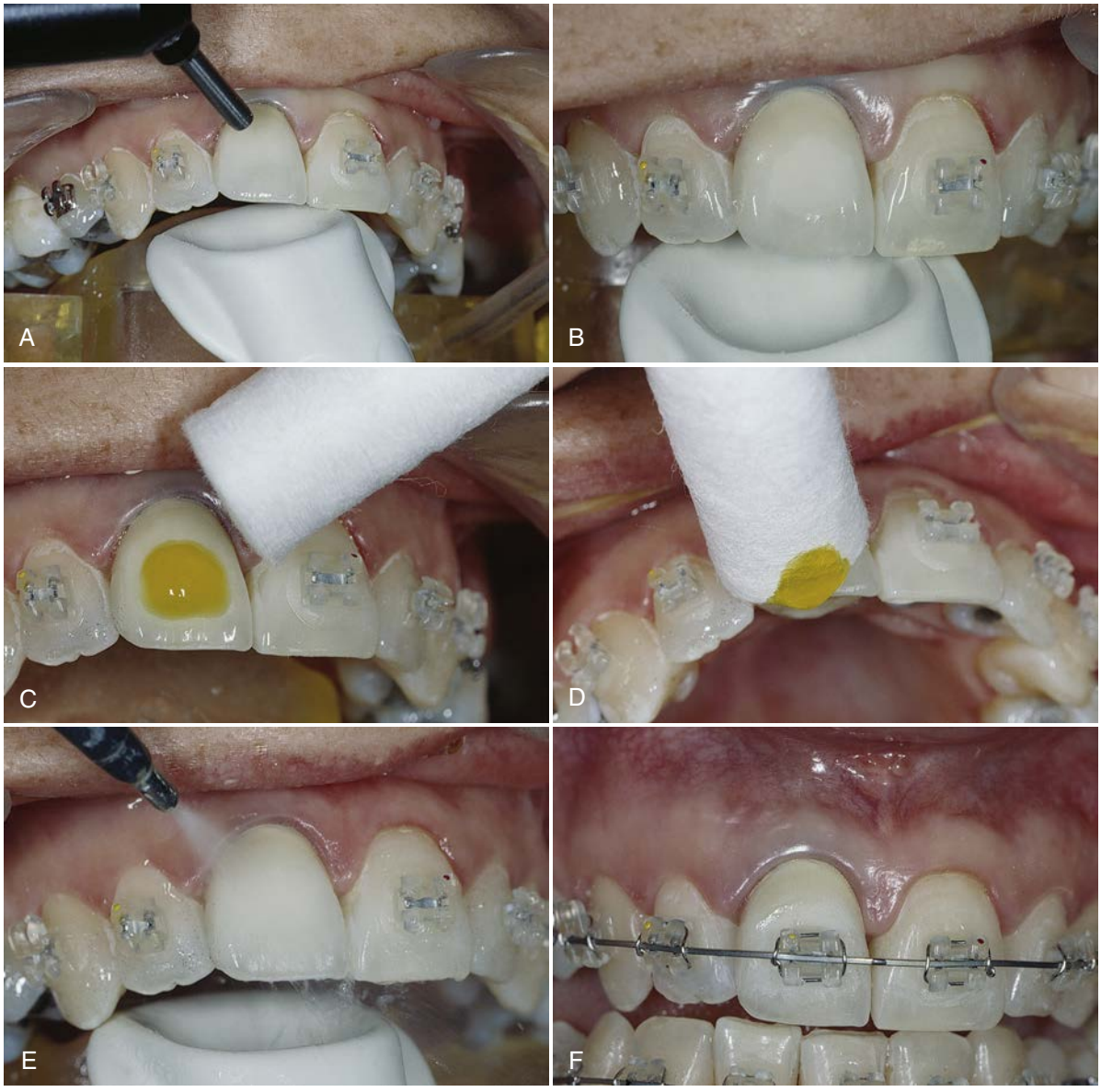


FIGURE 27-19 Technique for bracket bonding to porcelain surfaces includes reliable soft tissue retraction and separately bonding of the crown from other teeth. An area slightly larger than the bracket base is deglazed (**A, B**) before the hydrofluoric acid etching gel is applied for 2 minutes (**C**). The gel is removed with a cotton roll (**D**), and the teeth are rinsed with water and air spray under high-volume suction (**E**). **F,** Final result.

achieve a mechanical interlock with the composite resin.[122,123] The etched porcelain will have a frosted appearance similar to that of etched enamel.

Because of the caustic effects of hydrofluoric acid, several alternatives, including acidulated phosphate fluoride gel, or irradiation of ceramic surface with carbon dioxide or Er:YAG lasers were proposed all with similar or better results, compared with hydrofluoric acid.[124–127] However, none of these was popularized, attributable to either longer chair time or an unjustified amount of required investment.

For optimal bonding of orthodontic brackets and retainer wires to porcelain surfaces, the following technique is recommended (see Fig. 27-19):

1. Adequately isolate the working field, and bond the actual crown separately from the other teeth.
2. Use a barrier gel such as Kool-Dam (Pulpdent Corporation, Watertown, MA) (see Fig. 27-20) on mandibular teeth and whenever a risk exists that the hydrofluoric acid etching gel may flow into contact with the gingiva or soft tissues.
3. Deglaze an area slightly larger than the bracket base by sandblasting with 50 microns of aluminum oxide for 3 seconds.
4. Etch the porcelain with 9.6% hydrofluoric acid gel for 2 minutes.
5. Carefully remove the gel with a cotton roll, and then rinse using high-volume suction.
6. Immediately dry with air, and bond the bracket. Using a silane is optional.

Hydrofluoric acid will not be effective for bonding to high-alumina porcelains and glass ceramics, and new technique improvements are needed for successful orthodontic bonding

FIGURE 27-20 A, B, When hydrofluoric acid gel is used close to the gingival margin, particularly in the mandible, a light-cured blockout resin, such as Kool-Dam (Pulpdent Corporation, Watertown, MA) must be used to protect the soft tissues from the acid. C, A lower molar bracket must be positioned out of occlusion with the opposing teeth to avoid bracket loosening. If this is not possible, then the tie-wing in contact with the upper molar (usually the distal wing) should be ground with a green stone.

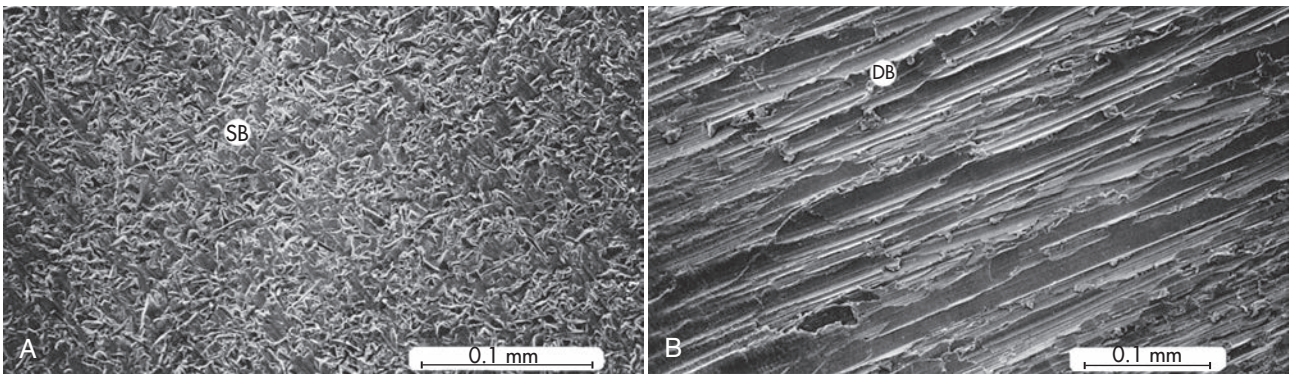


FIGURE 27-21 Scanning electron photomicrograph of a sandblasted (A) and diamond bur-roughened (B) metal surface. The use of the MicroEtcher (Danville Materials, Inc., San Ramon, CA) for approximately 3 seconds (*SB*) provides excellent micromechanical retention, whereas periodic ridges and grooves produced by medium-grit diamond bur (*DB*) have few undercuts for mechanical retention. *Bar*, 0.1 mm.

to such teeth. A newly introduced alternative technique to the use of hydrofluoric acid gel may be silica coating,[127–129] but further clinical trials are needed to obtain experience with the silica-coating technique.

Bonding to Amalgam

Improved techniques for bonding to amalgam restorations may involve (1) modification of the metal surface (sandblasting, diamond bur roughening) (Figs. 27-21 and 27-22), (2) the use of intermediate resins that improve bond strengths (e.g., ALL-BOND 2 [Bisco, Inc., Schaumburg, IL], Enhance and Metal Primer [Reliance Orthodontic Products, Itasca, IL]), and (3) new adhesive resins that chemically bond to nonprecious and precious metals (e.g., 4-methacryloxyethyl trimellitate anhydride [4-META] resins and 10-methacryloyloxi-decyl-dihydrogen-phosphate [10-MDP] bisGMA resins).[130,131]

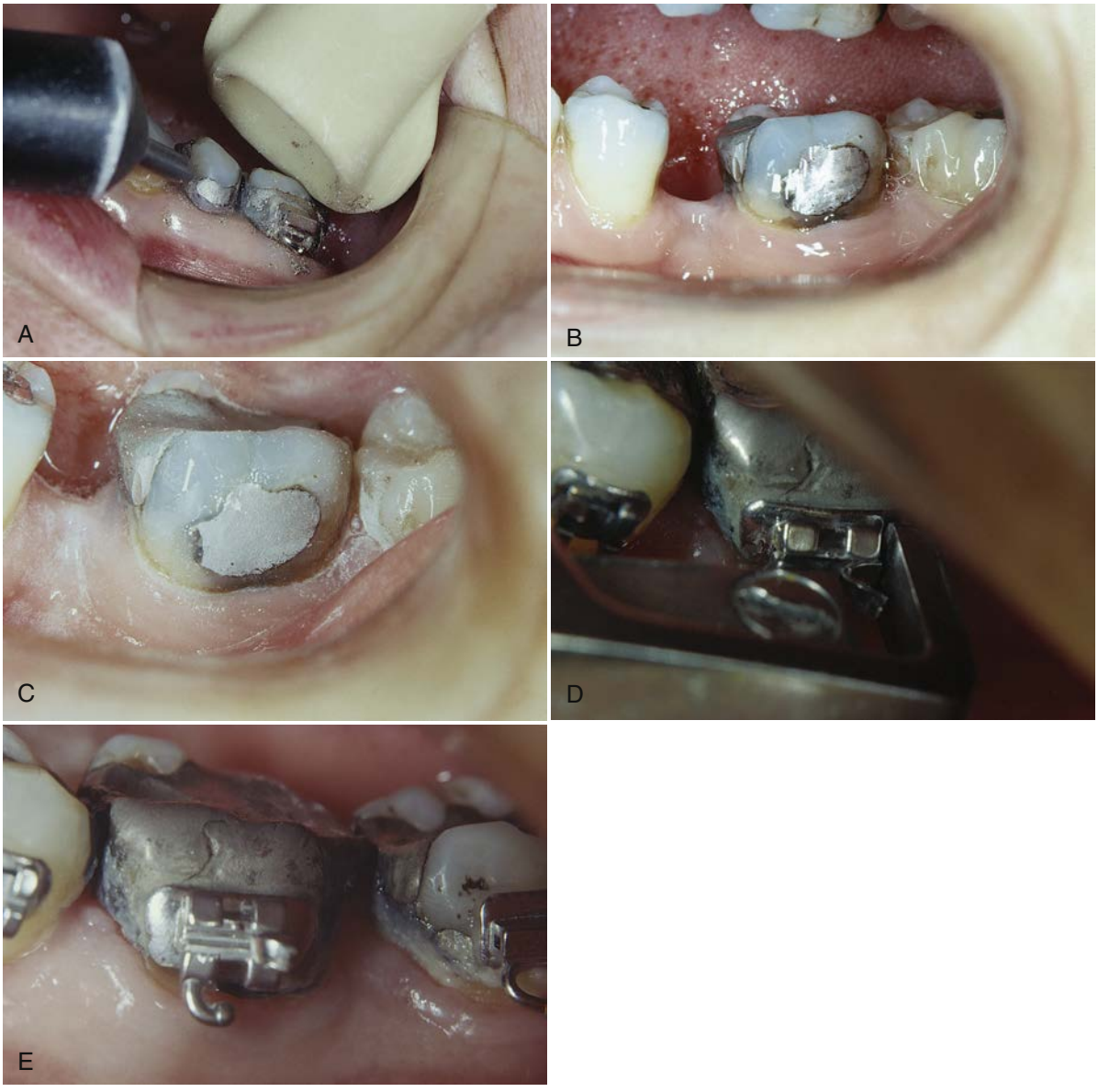


FIGURE 27-22 A, During air abrasion, high-velocity evacuation is necessary. B, C, Intraoral sandblasting of amalgam restorations produces frosted appearance, indicating increased micromechanical retention. D, E, Convertible cap removal on the attachment bonded to amalgam only on the mandibular first molar, indicating strength of bond.

Similar to bonding to porcelain, apparently a positive correlation does not exist between laboratory and clinical findings when it comes to orthodontic bonding to amalgam fillings. In vitro bonds to amalgam are significantly weaker than for similar brackets bonded to enamel of extracted human teeth.[130–132] However, the clinical performance with different techniques is satisfactory. In the first amalgam study in the authors' laboratory,[130] mean tensile bond strength to sandblasted amalgam tabs ranged from 3.4 to 6.4 megapascal (MPa), in contrast to control bonds to human enamel of 13.2 MPa. The strongest bonds to amalgam were obtained with a 4-META adhesive (Superbond C&B, Sun Medical Co., Ltd., Kyoto, Japan), but an intermediate resin (All-BOND 2, Bisco, Inc., Schaumburg, IL) and the Concise Enamel Bond (3M Dental Products, St. Paul, MN) were comparable with those of Superbond C&B.

A follow-up in vitro study with different intermediate primers on the three primary types of dental amalgams (spherical, lathe cut, admixed) showed better results for two 4-META primers (Metal Primer [Reliance Orthodontic Products, Itasca, IL], Amalgambond Plus [Parkell Co., Farmingdale, NY]) than for All-BOND 2.[132] Clinical observations have confirmed these results.

The following procedures are recommended for bonding to amalgam.

Small amalgam filling with surrounding sound enamel.

1. Sandblast the amalgam alloy with 50 microns of aluminum oxide for 3 seconds (see Fig. 27-22, *A-C*).
2. Condition the surrounding enamel with 37% phosphoric acid for 15 seconds.
3. Apply sealant, and bond with composite resin. Ensure the bonded attachment is not in occlusion with antagonists.

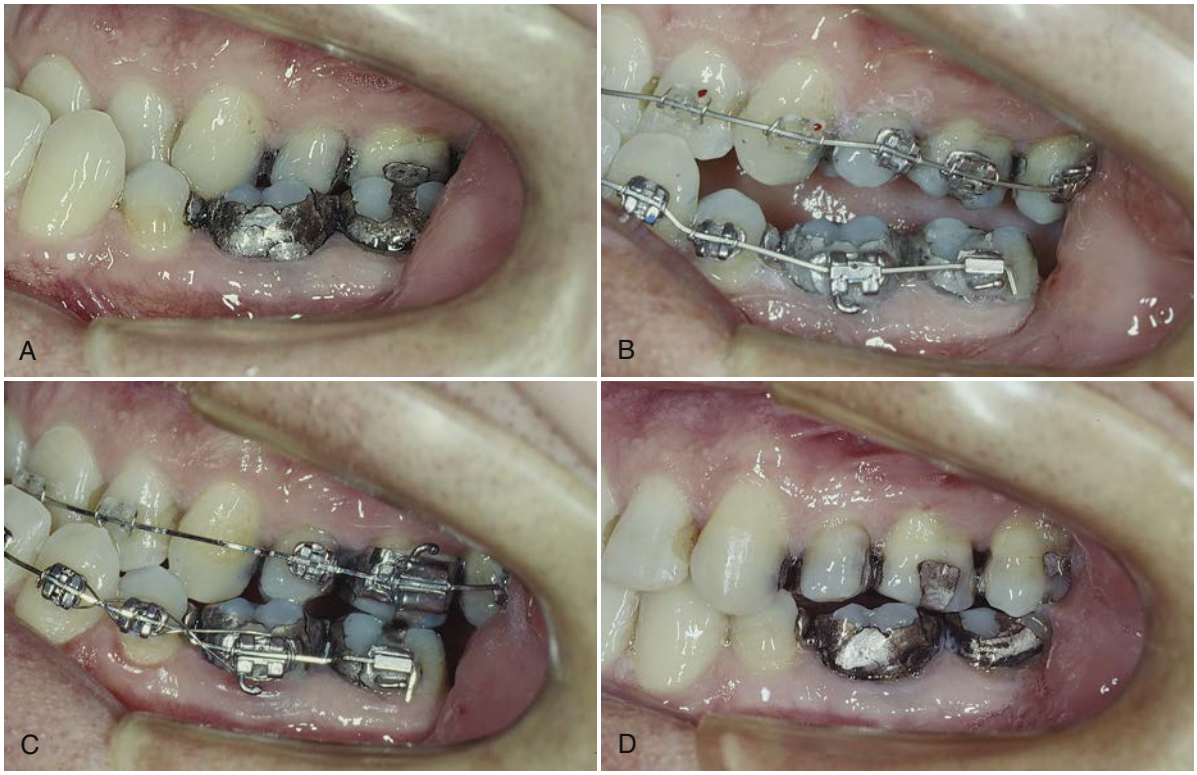


FIGURE 27-23 Orthodontic attachments bonded to large amalgam restorations on maxillary first and mandibular first and second molars in an adult Class III patient before (**A**), during (**B, C**), and after treatment (**D**). The superplastic (**B**) and rectangular stainless steel archwires (**C**) were bent over at the distal of the second molar during treatment without coming loose.

Large amalgam restoration or amalgam only.

1. Sandblast the amalgam filling with 50 microns of aluminum oxide for 3 seconds (Fig. 27-23; see also Fig. 27-22, *A–C*).
2. Apply a uniform coat of Reliance Metal Primer (Reliance Orthodontic Products, Itasca, IL), and wait for 30 seconds (or use another comparable primer according to the manufacturer's instruction).
3. Apply sealant, and bond with composite resin. Ensure the bonded attachment is not in occlusion with antagonists.

Of course, amalgam surfaces can be easily repolished with rubber cups and points after debonding.

Bonding to Gold

In contrast to bonding to porcelain and amalgam, excellent bonding to gold crowns is not yet available to orthodontic clinicians. This unavailability is surprising in light of the high bond strengths, which generally have been reported in different laboratory studies of gold alloys.[133] Different new technologies, including sandblasting, electrolytic tin-plating, or plating with gallium-tin solution (Adlloy), the use of several different types of intermediate primers, and new adhesives that chemically bond to precious metals (Superbond C&B [Sun Medical Co., Ltd., Kyoto, Japan] and Panavia Ex and Panavia 21 [Kuraray America, Inc., New York, NY])[117] have been reported to improve bonding to gold in laboratory settings. However, the high in vitro bond strengths to gold alloys have not been confirmed by satisfactory clinical results when bonding to gold crowns.

In the authors' experience, even a combination of intraoral sandblasting, coupled with the use of All-BOND 2 (Bisco, Inc., Schaumburg, IL) or 4-META primers and followed by bracket bonding with composite resin or special metal-bonding adhesives, may not optimally withstand the occlusal forces in clinical practice. Clinical studies are hampered by the fact that bracket bonding to gold restorations or retainer bonding to lingual metal-ceramic crowns (Fig. 27-24, *B*) is not frequently occurring in daily practice. A distinct difference between natural enamel or ceramic restorations and metal restorations is that curing light cannot travel through alloy or metal, and the amount of free radicals produced and the degree of conversion seem to be significantly less when bonding on the metal surface than on the enamel surface. Recently, it was demonstrated that primer precuring at the bracket base is required for secure bracket bonding on gold alloy surfaces using LED-curing units.[134] Further evaluation of this phenomenon is required for bonding to various gold alloys and large amalgam restorations.

Bonding to Composite Restoratives

The bond strength obtained with the addition of new composite to mature composite is substantially less than the cohesive strength of the material. However, brackets bonded to a fresh, roughened surface of old composite restorations after thorough air drying appear to be clinically successful in most instances.[135] The use of an intermediate primer is probably advantageous as well.

Indirect Bonding

Several techniques for indirect bonding are available. In some, the brackets are glued with a temporary material to the teeth on the patient's models, transferred to the mouth with a tray into which

FIGURE 27-24 Bonding to tooth surfaces of gold alloy includes bracket bonding to molar crowns (A) and retainer wire bonding to the lingual of metal-ceramic crowns (right and left lateral incisors and right central incisor in B). If unfilled, then 4-methacryloxyethyl trimellitate anhydride (4-META) resin is used for retainer bonding; it may be covered with more abrasion-resistant composite resin. (From Büyükyilmaz T, Zachrisson YØ, Zachrisson BU. Improving orthodontic bonding to gold alloy. *Am J Orthod Dentofacial Orthop* 1995;108:510–518. Used with permission from the American Association of Orthodontics.)

the brackets become incorporated, and then simultaneously bonded with a bisGMA resin. However, most current indirect bonding techniques are based on a modification introduced by Thomas,[136] which attaches the brackets with composite resin to form a custom base (Fig. 27-25). A transfer tray of silicone putty or thermoplastic material is used, and the custom bracket bases are then bonded to the teeth with a chemically cured sealant.

Many advocates believe that reduced chair time and the delegation of the procedure make indirect bonding cost effective.[137] The claimed primary advantages of indirect compared with direct bonding are that the brackets can be more accurately positioned in the laboratory, and the clinical chair time is decreased. In fact, Hodge and co-workers[138] investigated the cost effectiveness in a hospital dental clinic and found a significant cost savings when using indirect bonding versus direct bonding in that setting, but clinical and laboratory studies fail to support this advantage in labial cases with a small gain in the accuracy of the height of the bracket.[138–140] Recent clinical and laboratory research also indicates similar adhesive quality and bracket survival rate with those of direct bonding.[141–143]

However, the indirect bonding is technique sensitive, and the chairside procedure is more crucial, at least for inexperienced clinicians; removal of excess adhesive can be more difficult and more time consuming with some techniques; the risk for adhesive deficiencies under the brackets is greater; the risk for adhesive leakage to interproximal gingival areas can disturb oral hygiene procedures; and the failure rates with some methods seem to be slightly higher.[144,145] Yet, indirect bonding deserves attention, as the trend to use this method among orthodontists in the United States is increasing (18% in 2014 compared with 13% in 2008).[4,5] In addition, in lingual orthodontics, the indirect technique is also a prerequisite for good bracket alignment because direct visualization has evident difficulties.

Numerous products and methods that are specifically designed for indirect bonding procedures are available. Different types of custom base composites may be light cured, chemically cured, or thermally cured.[135,145,146] One system (from Reliance Orthodontic Products, Itasca, IL) recommends the use of a thermally cured base composite (Therma-Cure), Enhance adhesion booster, and a chemically cured sealant (Custom I.Q.). Another system (from 3M Unitek, Monrovia, CA) recommends the use of light-cured base composite (Transbond XT) and chemically cured sealant (Sondhi Rapid-Set) in the clinic (Fig. 27-26).

Clinical Procedure

As previously mentioned, several indirect bonding techniques have proved reliable in clinical practice (see Figs. 27-25 and 27-26). The techniques differ in the way the brackets are attached temporarily to the model, the type of transfer tray used (e.g., full-arch, sectioned full-arch, single tooth, double-tray system), the sealant or resin used, whether segmented or full bonding is used, and the way the transfer tray is removed so as not to exert excessive force on a still-maturing bond.

Indirect Bonding with Composite Custom Bracket Base

Most current techniques use composite resin custom bracket bases (light cured, thermally cured, or chemically cured) and a chemically cured sealant as the clinical bonding resin. The following procedure may be useful (see Fig. 27-26)[19]:

1. Take an impression, and pour a stone (not plaster) model.
2. Select brackets for each tooth.
3. Isolate the stone model with a separating medium.
4. Attach the brackets to the teeth on the model with light-cured or thermally cured composite resin, or use adhesive precoated brackets.
5. Check all measurements and alignments. Reposition if needed.
6. Make a transfer tray for the brackets. The material can be putty silicone, thermoplastics, or similar.
7. After removing the transfer trays, gently sandblast the adhesive bases with a microetching unit, taking care not to abrade the resin base.
8. Apply acetone to the bases to dissolve the remaining separating medium.
9. Prepare the patient's teeth for a direct application.
10. Apply Sondhi Rapid-Set (3M Unitek, Monrovia, CA) resin A to the tooth surfaces and resin B to the bracket bases. If Custom I.Q. (Reliance Orthodontic Products, Itasca, IL) is used, then apply resin B to the teeth and resin A to the bases. Alternatively, apply a thin coat of the mixture of part A and part B adhesive to

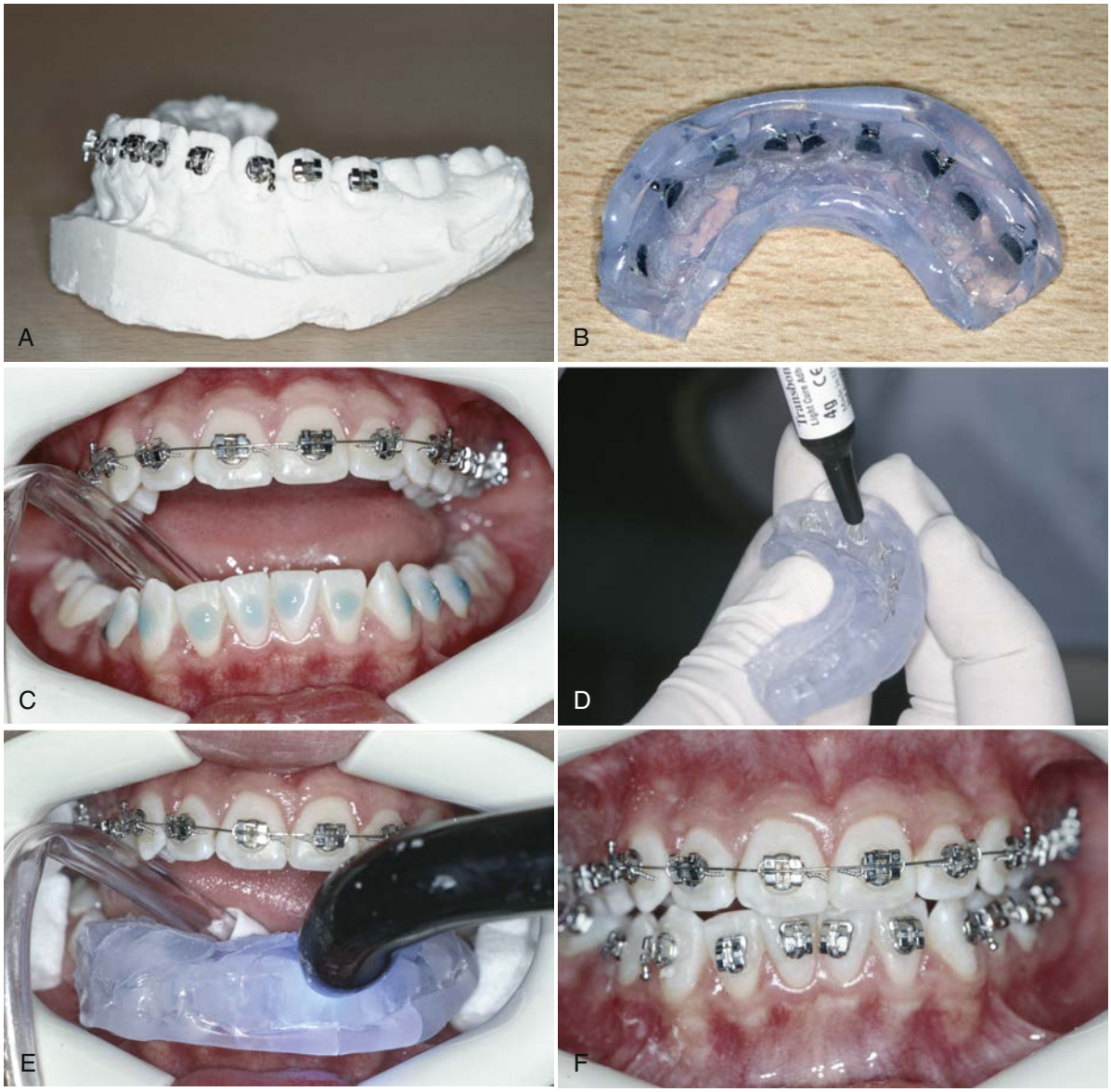


FIGURE 27-25 Indirect bonding using a Memosil 2 clear transfer tray (Heraeus Kulzer, Armonk, NY) and a light cure adhesive.

each custom resin base in the indirect bonding tray and to the tooth surface if using Transbond IDB Pre-Mix Chemical Cure Adhesive (3M Unitek, Monrovia, CA) (Fig. 27-27).

11. Seat the tray on the prepared arch, and apply equal pressure to the occlusal, labial, and buccal surfaces with the fingers. Hold for a minimum of 30 seconds, and allow for 2 minutes or more of curing time before removing the tray.
12. Remove excess flash of resin from the gingival and contact areas of the teeth with a scaler or contra-angle handpiece and tungsten carbide bur.

Rebonding

Bonded brackets that become loose during treatment, consuming significant chair time, are poor publicity for the office and are a nuisance to the orthodontist. The best way to avoid loose brackets is to adhere strictly to the rules for good bonding previously mentioned. Using a quick technique for rebonding loose brackets is also important.

The loose metal bracket removed from the archwire should first be inspected for any possible deformation of the slot that may have occurred during breakage. A bracket that seems to be deformed should be replaced with a new one. Any adhesive remaining on the tooth surface is removed with a tungsten carbide bur. The adhesive remaining on the loose bracket is best treated by sandblasting[147] (see Fig. 27-18) until all visible bonding material is removed from the base. The tooth is then etched with phosphoric acid gel for 15 seconds. On inspection, the enamel surface may not be uniformly frosty because some areas may still retain resin. The phosphoric acid will re-etch any exposed enamel and remove the pellicle on any exposed resin. After priming, the bracket is rebonded. The neighboring brackets are first religated, and then the rebonded bracket is ligated. The bond strength for sandblasted rebonded brackets is comparable to the success rate for new brackets,[147,148] but it should be noted that the brackets' SBS decreased as the size of the aluminum oxide particle used for sandblasting increased and as recycling was repeated.[149]

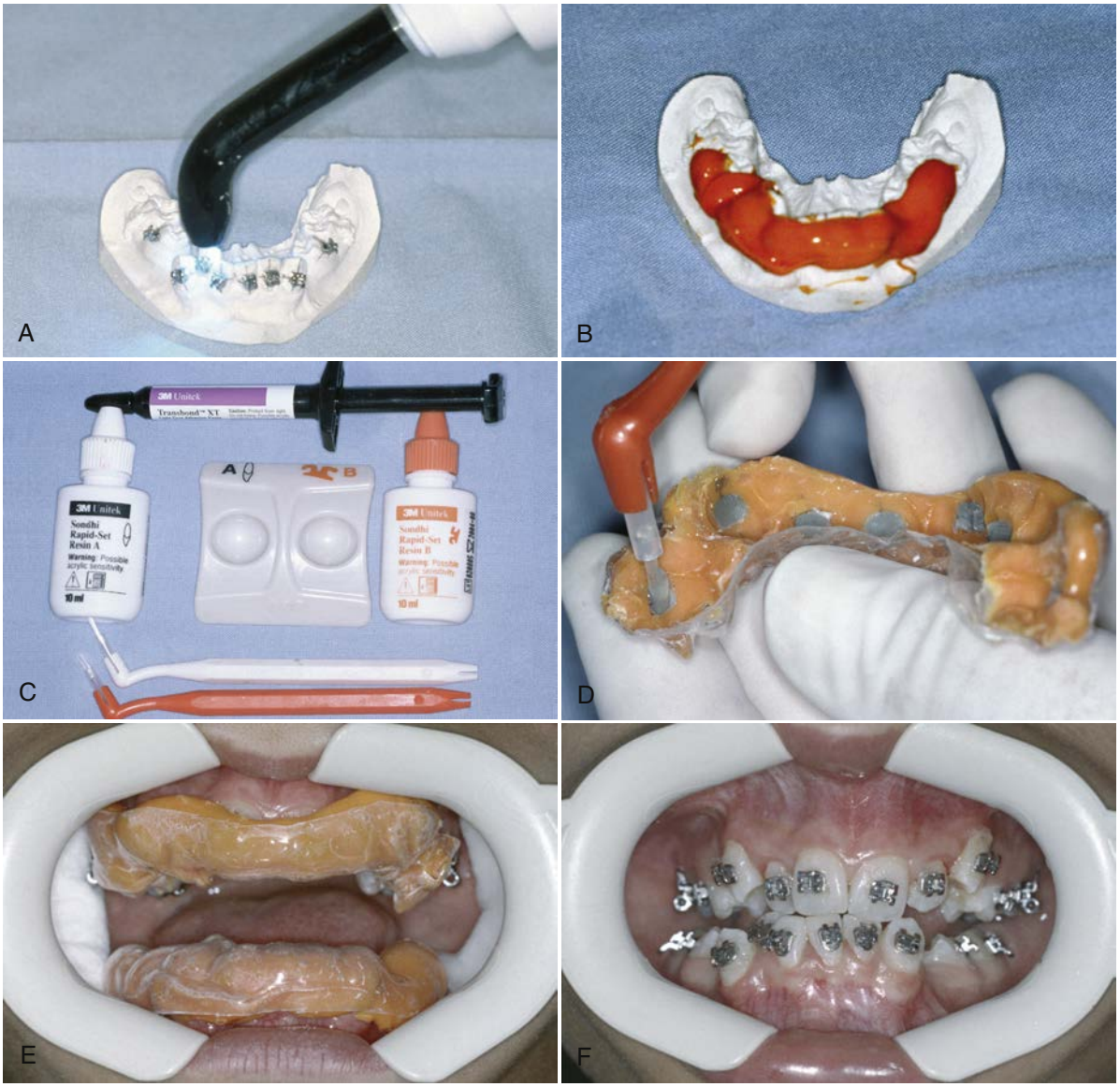


FIGURE 27-26 Indirect bonding using light-cured base composite (Transbond XT, 3M Unitek, Monrovia, CA) and chemically cured sealant (Sondhi Rapid Set, 3M Unitek, Monrovia, CA). (See text for details.)

FIGURE 27-27 Transbond IDB (3M Unitek, Monrovia, CA) is a premix chemical cure indirect bonding adhesive that can be used with custom-base adhesives. (Courtesy of 3M, © 2016 3M. All rights reserved.)

Reconditioning ceramic orthodontic brackets with an erbium, chromium:yttrium scandium gallium garnet (Er,Cr:YSGG) laser was found to be effective,[150] but a loose ceramic bracket should best be replaced with a new, intact bracket for optimal bond strength.

Conclusion

Bonding of brackets has changed the practice of orthodontics and has become a routine clinical procedure in a remarkably short time.[5]

In most routine situations, banding maxillary first molars provides a stronger attachment, and the availability of lingual sheaths (e.g., for transpalatal bars, elastics, and headgear) may give some interproximal caries protection. Finally, the procedure described for bonding mandibular second and third molars has proved to be successful in clinical use over many years. This success is particularly true in adolescents, whose teeth are erupting during the course of treatment. The

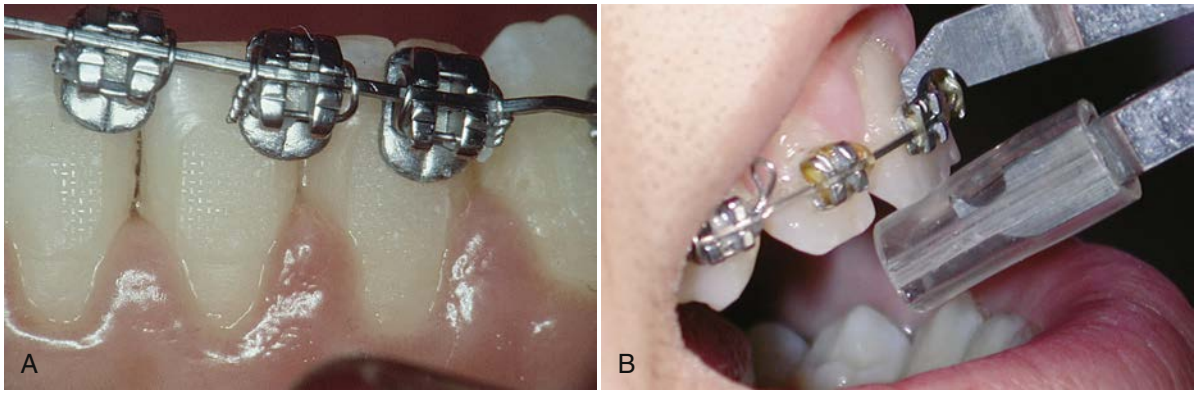


FIGURE 27-28 Bracket removal with pliers. Still ligated in place, the brackets are gripped, one by one, with 095 Orthopli Bracket-Removing Pliers (G&H Orthodontics, Inc., Franklin, IN) and lifted outward at a 45-degree angle. The indention in the pliers fits into the gingival tie-wings for a secure grip. This technique is quick and gentle and leaves the brackets intact and fit for recycling, if so desired. **A,** The bond breaks in the adhesive-bracket interface, and the pattern of the mesh backing is visible on the adhesive remaining on the teeth. **B,** The same technique is used for maxillary steel brackets.

mandibular second molar is better suited for bonding than for banding because gingival emergence of the buccal surface precedes emergence of the distal surface.

Modifications of technical devices, sealants and adhesives, attachments, and procedures are continuing. Careful study of the available information by the orthodontist will be mandatory in keeping up with the progress. However, cautious interpretation of in vitro studies is recommended because the in vivo results do not always reflect and verify the laboratory findings.[19] Long-term follow-up studies are needed in several areas such as bonding to amalgam, as well as for porcelain and gold, during a full period of routine orthodontic treatment in larger samples.[110]

Drawing valid clinical conclusions from laboratory bond strength studies is not possible for at least three different reasons: (1) a continually increasing tensile or shear load applied to bonded brackets in the laboratory is not representative for the force applications that occur clinically; (2) the type of debonding force in machines is not the same as the force applied in clinical debonding; and (3) the complex oral environment with variations in temperature, stresses, humidity, acidity, and plaque is not reproducible in the laboratory.[151]

The striking difference between the clinical and laboratory experiences with respect to bond strengths to porcelain, amalgam, and gold is challenging. The results from laboratory studies should be used only to indicate which products and materials seem most valuable to include in supplementary clinical evaluation. Clinical success is the final test.[116]

DEBONDING

Unlike other restorative practices in dentistry, the adhesive system set up in the beginning of the orthodontic treatment is removed and the brackets are removed after the completion of the therapy. The objectives of debonding are to remove the attachment and all the adhesive resin from the tooth and to restore the surface as closely as possible to its pretreatment condition without inducing iatrogenic damage. To obtain these objectives, a correct technique is fundamentally important. Debonding may be unnecessarily time consuming and damaging to the enamel if carelessly performed or performed with an improper technique.

Because several aspects of debonding are controversial, debonding is discussed in detail as follows:

- Clinical procedure
- Enamel tearouts and cracks (fracture lines)
- Removal of residual adhesive
- Amount of enamel lost in debonding
- Operator safety during debonding
- Prevention and reversal of decalcifications

Clinical Procedure

Although several methods have been recommended in the literature for bracket removal and adhesive cleanup, and some differences of opinion still exist, the techniques described have proved successful in the authors' experiences. Their rationales are mentioned throughout the ensuing discussion.

The clinical debonding procedure may be divided into two stages: (1) bracket removal and (2) removal of residual adhesive.

Removal of Steel Brackets

Whether metal or ceramic, the brackets should be individually removed to avoid force transfer from tooth to tooth, which may increase the risk of enamel crack formation.

Several different procedures are available for debracketing with pliers. An original method was to place the tips of a twin-beaked pliers against the mesial and distal edges of the bonding base and cut the brackets off between the tooth and the base. Several pliers are available for this purpose. A gentler technique is to squeeze the bracket wings mesiodistally and then lift the bracket off with a peel force. This technique is particularly useful on brittle, mobile, or endodontically treated teeth.

The recommended technique, in which brackets are not deformed, is illustrated in Figure 27-28. This technique uses a peeling-type force, which is most effective in breaking the adhesive bond. A peel force, as in peeling an orange, creates peripheral stress concentrations that cause bonded metal brackets to fail at low force values.[152] The break is likely to occur in the adhesive–bracket interface, thus leaving adhesive remnants on the enamel. Attempts to remove the bracket by shearing it off

(as is done in removing bands) can be traumatic to the patient and potentially damaging to the enamel.

Removal of Ceramic Brackets

With the introduction of ceramic brackets, a new concern over enamel fracture and loss from debonding has arisen because debonding ceramic brackets is more liable to enamel fracture formation since they more strongly adhere to the enamel surface and will not flex when squeezed with debonding pliers.[153,154] Because of differences in bracket chemistry and bonding mechanisms, various ceramic brackets behave differently on debonding. The risk is lower with ceramic brackets using mechanical retention than those using chemical retention.[153,155–157] More recent ceramic brackets have a mechanical lock base and a vertical slot that will collapse the bracket by squeezing. Debonding collapsible ceramic brackets with the archwire in place and ligated (Fig. 27-29) to hold together the debonded bracket parts is recommended.

Mesiodistally cutting off the brackets with gradual pressure from the tips of twin-beaked pliers oriented close to the bracket–adhesive interface is not recommended because doing so may introduce horizontal enamel cracks.[19] Low-speed grinding of ceramic brackets with no water coolant may cause permanent damage or necrosis of dental pulps. Therefore water cooling of the grinding sites is necessary.

Lasers also have the potential to be less traumatic and less risky for enamel damage. This procedure first appeared to facilitate the removal of porcelain laminate veneers, which are bonded using a very strong resin cement. Öztoprak and colleagues demonstrated that with a new scanning method, the Er:YAG laser is effective for reducing SBS of ceramic brackets from high values to levels for safe removal from the teeth in 9 seconds per bracket.[158] Additionally, in a recent study, Mundethu and co-workers[159] reported that clear brackets can be debonded with a single pulse when irradiated with the Er:YAG laser operating at 600 mJ, 800-μs pulse, 1.3-mm fiber tip. With this method, 19 out of 20 brackets successfully debonded with a single pulse.[159] In this method, laser energy is transferred through the ceramic and absorbed at the composite layer where microexplosions occur, resulting in the detachment of the bracket without any thermal damage to the tooth[159,160] (Fig. 27-30, *A* and *B*).

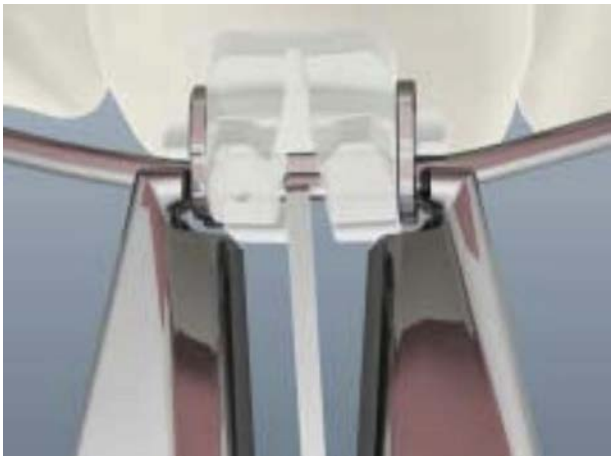


FIGURE 27-29 Debonding of collapsible ceramic brackets can be accomplished by using either Howe or Weingart pliers or respective debonding instrument of the company. Debonding collapsible ceramic brackets with the archwire in place and ligated is recommended. Whatever method is used, providing eye protection for the patient and operator to avoid scattering ceramic pieces is a good practice.

Enamel Tearouts and Cracks (Fracture Lines)

Localized enamel tearouts have been reported to occur associated with bonding and debonding metal and ceramic brackets. Ceramic brackets using chemical retention cause enamel damage more often than those using mechanical retention. A recent study demonstrated enamel tearouts in 26% of debonded polycrystalline brackets and in less than 1% of monocrystalline brackets with an average volume loss of 144 and 36 cubic micrometers (mm^3), respectively.[161] This damage probably occurs because the location of the bond breakage is at the enamel–adhesive interface rather than at the adhesive–bracket interface.

The prevalence and location of cracks among debonded, debanded, and orthodontically untreated teeth demonstrate no significant difference, and the most notable cracks (i.e., those invisible under normal office illumination) are on the maxillary central incisors and canines.[162] However, the sharp sound sometimes heard on the removal of bonded orthodontic brackets with pliers is possibly associated with the creation of enamel cracks. With ceramic brackets, the risk for creating enamel cracks is greater than for metal brackets. The lack of ductility may generate stress in the adhesive–enamel interface that may produce enamel cracks at debonding.

The clinical implications are (1) to use brackets that have mechanical retention and debonding instruments and techniques that primarily leave all or the majority of composite on the tooth (see Fig. 27-28, *A*) and (2) to avoid scraping away adhesive remnants with hand instruments.

Another important clinical implication may be the need for a pretreatment examination of cracks, notifying the patient and the parents if pronounced cracks are present (Fig. 27-31). The reason for this examination is that patients may be overly inspective after appliance removal and may detect cracks that were present before treatment of which they were unaware. They may question the orthodontist about the cause of the cracks. Without a pretreatment diagnosis and documentation (most cracks are not visible on routine intraoral slides), proving that such cracks are indeed unrelated to the orthodontic treatment is almost impossible.[19]

Removal of Residual Adhesive

Debonding of brackets usually leaves a residual adhesive volume of 0.6 to 2.48 mm^3 on the enamel surface.[163,164] Complete removal of all remaining adhesive is not easily achieved because of the color similarity between present adhesives and enamel. Spontaneous abrasive wear of present bonding resins is limited and should not be expected, and these remnants are also likely to become unaesthetically discolored with time.

The preferred method for the removal of excess adhesive is to use a suitable dome-tapered tungsten carbide bur (No. 1171 or No. 1172) in a contra-angle handpiece (Fig. 27-32). Clinical experience and laboratory studies[165] indicate that approximately 30,000 rpm is the optimum for rapid adhesive removal without enamel damage. Light painting movements of the bur should be used so as not to cause deep scratches on the enamel. Water cooling should not be used when the last remnants are removed because water lessens the contrast with enamel. Speeds higher than 30,000 rpm using fine fluted tungsten carbide burs may be

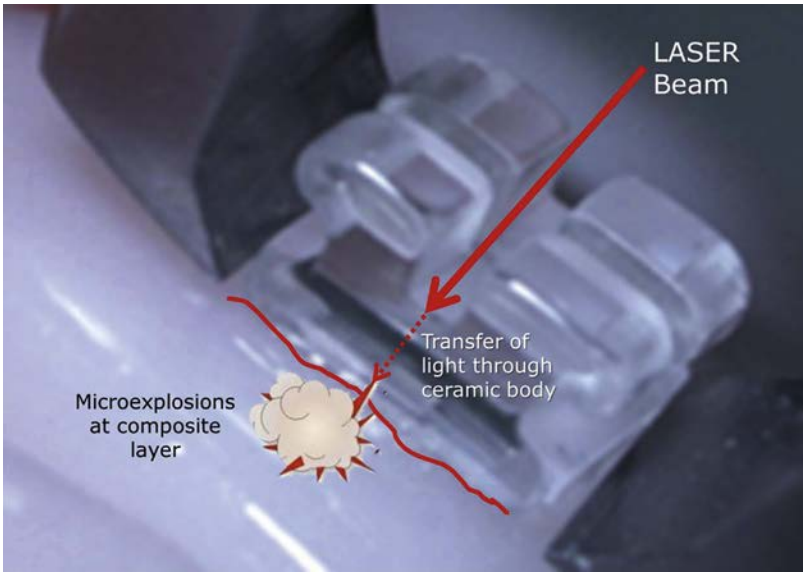


FIGURE 27-30 The laser tip is brought into contact with the ceramic bracket and initiated for laser-aided debonding. Laser energy is transferred through the ceramic and absorbed at the composite layer where microexplosions occur, resulting in the detachment of the bracket without any thermal damage to the tooth.

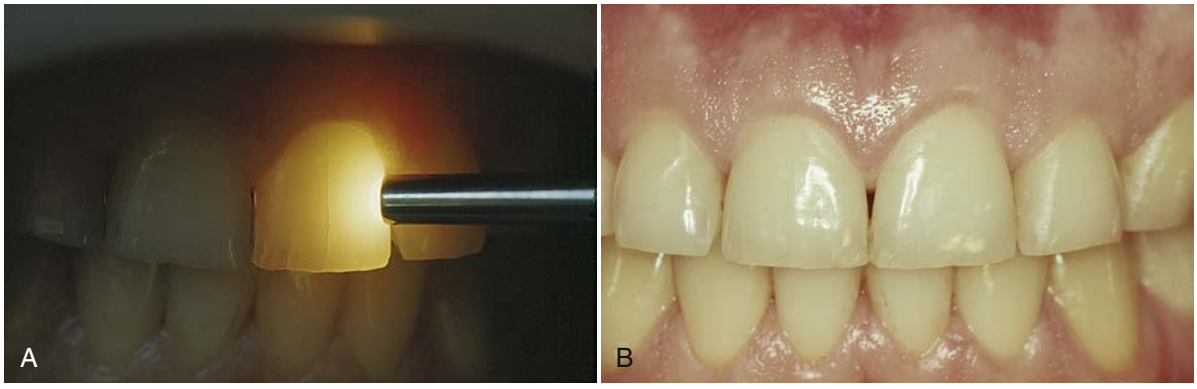


FIGURE 27-31 Enamel cracks generally are not visible on intraoral photographs. Several cracks are clearly seen on the left central incisor with fiberoptic transillumination (**A**) and are undetectable by routine photography (**B**). Note the vertical orientation of the cracks.

FIGURE 27-32 Adhesive remaining after debracketing may be removed with a tungsten carbide bur at approximately 30,000 rpm.

useful for bulk removal but are not indicated closer to the enamel because of the risk of marring the surface. Even ultrafine high-speed diamonds produce considerable surface scratches. Slower speeds (10,000 rpm and less) are ineffective, and the increased jiggling vibration of the bur may be uncomfortable to the patient.

Zachrisson and Årtun[165] compared different instruments commonly used in debonding procedures and ranked their degrees of surface marring on young permanent teeth. According to the results of this study, plain cut and spiral fluted tungsten carbide burs operated at approximately 25,000 rpm were the only instruments that provided the satisfactory surface appearance (Fig. 27-33); however, none of the instruments tested left the virgin tooth surface with its perikymata intact. The clinical implication of the study is that tungsten carbide burs produced the finest scratch pattern with the least enamel loss and are superior in their ability to reach difficult areas (Fig. 27-34). Recent research shows, however, that the use of carbide burs alone removes a substantial layer of enamel and roughens its surface and thus should be followed by multistep Sof-Lex disks and pumice slurry, which is

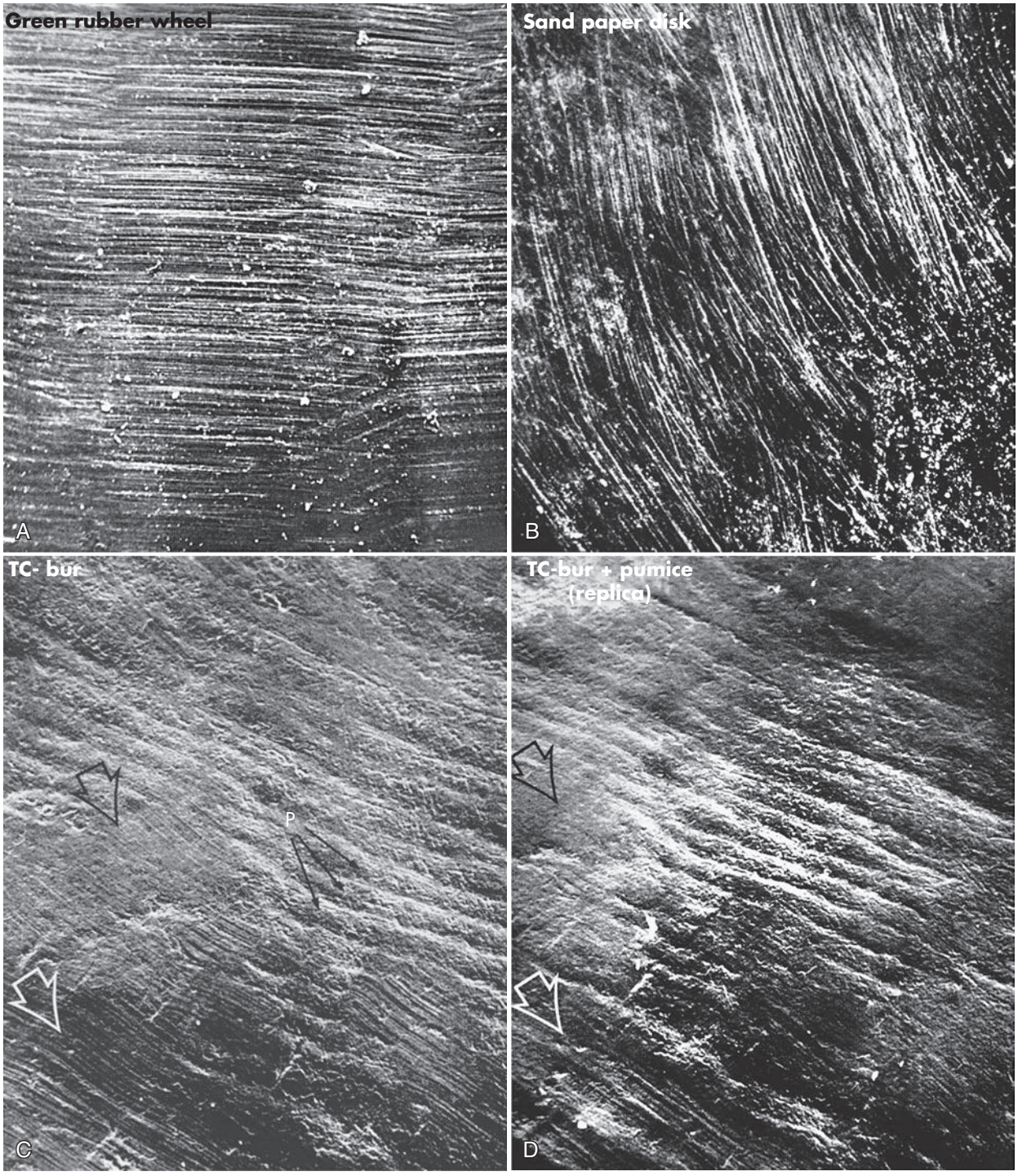


FIGURE 27-33 Comparison of the effect of three debonding techniques on the enamel surface. **A–C,** Scanning electron micrographs (×50) after adhesive removal without subsequent polishing. Note that the scratches are similar in appearance in **A** and **B**, but only sight faceting is visualized in **C** with fine scratches (*open arrows*) intermingled with the perikymata ridges (*P*). **D,** Same area as in **C** in replica after pumicing. The surface is smoother (*arrows*).

reported as a reliable method of polishing.[166] Using a carbide bur may also be followed by Astropol finish (F), polish (P), and high-gloss polisher (HP), as this combination is reported to provide a significantly lower enamel surface alteration with comparable composite remnants.[164]

Despite all efforts, a negligible amount of resin ranging from 0.11 to 0.22 mm^3 is left on the tooth surface, and the use of polishing systems with good composite polishing properties may leave a lustrous surface and therefore more composite remnants as they become invisible.[164]

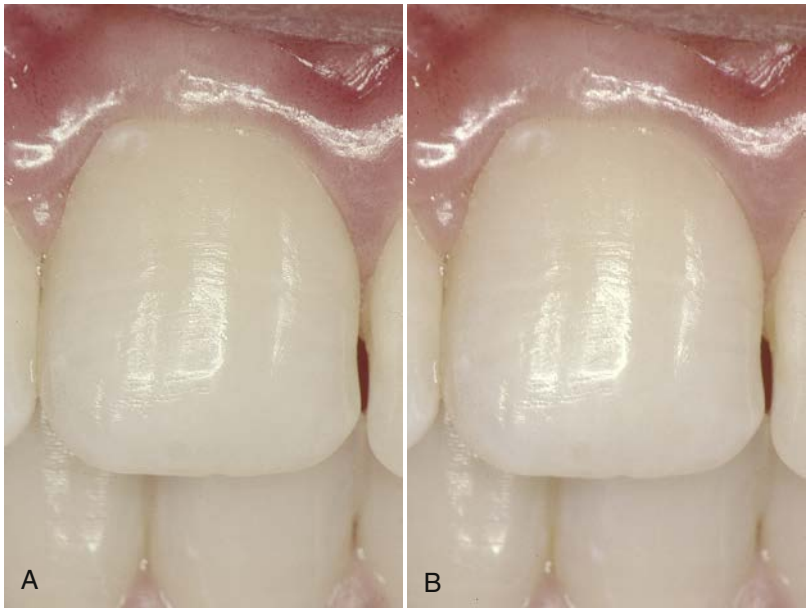


FIGURE 27-34 A, After debonding with a tungsten carbide bur at low speed. Gentleness of technique is reflected by the evident perikymata-like pattern on debonded teeth (B).

FIGURE 27-35 STAINBUSTER burs (Pearson Dental Supply Co., Sylmar, CA) gently remove cement, stains, and colored coatings from the surface of the enamel without abrading tooth enamel or ceramic. The burs are made up of the fiber sections with abrasive power, which cover the entire working surface and split up into small fragments as they act on a hard surface.

Some patients complain about color change of their teeth during and after orthodontic treatment. A recent study confirms this and states that both the orthodontic adhesive systems and the burs used to remove their residuals on tooth surfaces are responsible for this effect.[167] The authors suggest using STAINBUSTER burs (Pearson Dental Supply Co., Sylmar, CA) to remove the adhesive remnants close to the enamel surface[167] (Fig. 27-35).

Any recontouring considered necessary should be completed at this stage before proceeding to polishing. When all of the adhesive has been removed, the tooth surface is best polished with pumice (or a commercial prophylaxis paste) in a routine manner, which greatly improves the aesthetics of the teeth and the appreciation of the treatment results by the patient.

Amount of Enamel Lost in Debonding

Residual adhesive could be removed with minimal damage to the enamel by the careful use of a tungsten carbide bur, followed by finishing procedures,[168] but adequate clean-up without enamel loss is difficult to achieve.[164] Several factors, including the instruments used for prophylaxis and debonding and the type of adhesive resin used, dictates the amount of enamel actually removed, but the final surface topography is not influenced by different clean-up methods.[164]

Adequate removal of filled resin generally requires rotary instrumentation as described, and the reported amount of enamel loss is between 4.1 and 30 microns, which is approximately 0.05 mm^3 in volume.[161,164] In a clinical perspective, the enamel loss encountered with routine bonding and debonding procedures, exclusive of deep enamel fractures or gouges resulting from an injudicious use of hand instrument or burs, is not significant in terms of total thickness of enamel. The surfaces usually bonded have a thickness of 1500 to 2000 microns. The claim that removal of the outermost layer of enamel, which is particularly caries resistant and fluoride rich, may also be harmful is not in accordance with recent views on tooth surface dynamics and with clinical experience over many years. The facial tooth surfaces are left smooth and self-cleansing after debonding. Caries has been demonstrated not to develop in such sites even if the entire enamel layer is removed. Similarly, no histologic or clinical evidence of adverse effects was experienced after significant recontouring of canines that had been ground to resemble lateral incisors when approximately one half of the enamel thickness was removed as long as the surfaces were left smooth and sufficient water cooling was used.[84]

Operator Safety during Debonding

Another important but often ignored issue is the inhalation of aerosols produced during the removal of fixed orthodontic appliances. Recent research showed that aerosol particulates produced during enamel cleanup might be inhaled, irrespective of handpiece speed or the presence or absence of a water

FIGURE 27-36 Extreme degree of enamel demineralization after orthodontic treatment in a caries-prone patient **A,**. White spot lesions can occur on multiple teeth. **B,** The contour of the bonded brackets is visible on several teeth.

coolant. This aerosol may contain calcium, phosphorus, silica, aluminum, iron and lanthanum.[169] Blood, hepatitis B surface antigen (HBsAg), and hepatitis B virus–DNA were also detected in excess fluid samples of the two hepatitis B carriers.[170] Although the particles are most likely to be deposited in the conducting airways and terminal bronchi, some might be deposited in the terminal alveoli of the lungs and cleared only after weeks or months.[169]

Results of these reported studies indicate that orthodontists are exposed to high levels of aerosol generation and contamination during the debonding procedure, and preprocedural chlorhexidine gluconate mouth rinse appears to be ineffective in decreasing the exposure to infectious agents. Barrier equipment should be used to prevent aerosol contamination.[169,171]

Prevention and Reversal of Decalcification

White spots or areas of demineralization are carious lesions of varying extent. One prospective study[172] found that 50% of individuals undergoing fixed appliance treatment had nondevelopmental white spot lesions (WSLs), compared with 25% of a control group of patients. Another study[173] found that, even 5 years after treatment, orthodontic patients had a significantly higher incidence of WSLs than a control group of patients who had not had orthodontic treatment.

According to a recent survey, 37% of orthodontists reported that they had removed brackets because of patients' poor oral hygiene, and 69% of general dentists reported that they had treated WSLs during the previous year.[174] The presence of severe WSLs at the end of treatment decreases the value of efforts spent and disturbs the patient and the family. In addition, more than one-third of general dentists indicated that severe WSLs after orthodontic treatment could have a negative effect on a patient's perception of the treating orthodontist, thereby jeopardizing the referral base.[174] This obvious degree of iatrogenic damage suggests the need for preventive programs using fluoride associated with fixed appliance orthodontic treatment[19,175] (Fig. 27-36).

Daily rinsing with dilute (0.05%) sodium fluoride solution throughout the periods of treatment and retention, plus regular use of a fluoride dentifrice, is recommended as a routine procedure for all orthodontic patients.[110] Although effective, the weak fluoride mouth rinse has a few risks, and most patients can manage to use it easily for 1 to 2 years. Parents should also be adequately informed about the outcomes of failure to brush properly, and the definite responsibility also must be given to the patient to avoid decalcifications during treatment. In addition, painting a fluoride varnish[176] or other effective anticaries agents[177] over caries-susceptible sites at each visit may be useful in patients with hygiene problems.

Professional means of fluoride application have included fluoride-releasing bonding agents, fluoridated elastomeric ligature ties, fluoride varnish, and 10% casein phosphopeptide-amorphous calcium phosphate (GC Tooth Mousse, GC Corporation, Tokyo, Japan)[178–186] (Fig. 27-37). The professional application of 1% chlorhexidine collagen gel is also suggested to control *Streptococcus mutans* levels in an orthodontic patient with a high risk of caries.[187] The use of a polymeric tooth coating on the tooth surface around the brackets showed almost no demineralization-inhibiting effect.[188]

Visible white spots that develop during orthodontic therapy should not be treated with concentrated fluoride agents immediately after debonding because this procedure will arrest the lesions and prevent complete repair.[19] At present, a period of 2 to 3 months of good oral hygiene but without fluoride supplementation associated with the debonding session is recommended. This procedure should reduce the clinical visibility of the WSLs. High fluoride concentrations may tend to precipitate calcium phosphate onto the enamel surface and block the surface pores, which limit remineralization to the superficial part of the lesion, and the optical appearance of the WSL is not reduced.

Microabrasion

When the remineralizing capacity of the oral fluids is exhausted and WSLs are established (Fig. 27-38; see also Fig. 27-36), microabrasion is the optimal way to remove superficial enamel opacities. By the use of this technique, enamel stains can be eliminated with minimal enamel loss.[189]

Because microabrasion is comparably more invasive in nature, delayed application was thought to be beneficial, considering the spontaneous improvements of the lesion via saliva-based remineralization and spontaneous surface abrasion after debonding.[174,190]

Clinical procedure: A custom-made abrasive gel is prepared with 18% hydrochloric acid, fine powdered pumice, and glycerin. The active mixture is applied as follows[191]:

1. The gingiva is isolated using blockout resin or rubber dam. Dental floss may be useful to prevent soft tissue contact and injury from the acid.

FIGURE 27-37 MI Paste Plus (GC America Inc, Alsip, IL, USA) is a water-based, sugar-free crème containing Recaldent casein phosphopeptide–amorphous calcium phosphate (CPPACPF) and fluoride. When CPP-ACPF is applied to the tooth surfaces, it binds to biofilms, plaque, bacteria, hydroxyapatite, and surrounding soft tissue, localizing bioavailable calcium , phosphate and fluoride. (Courtesy of GC America Inc, Alsip, IL, USA)

FIGURE 27-38 White spot lesions before (**A**) and after (**B**) microabrasion. (See text for details.)

2. The abrasive gel is applied using an electric toothbrush for 3 to 5 minutes. The original toothbrush tip is modified by cutting the peripheral bristles to create a smaller brush tip to fit better on tooth surfaces.
3. Rinse for 1 minute.

To prevent enamel pitting, the acid should not be left on the tooth for an extended time. For best results and depending on the severity of the lesions, the procedure can be repeated monthly two to three times, which gradually makes the stains disappear. The microabrasion technique is effective in removing WSLs and streaks and brown-yellow enamel discolorations and considerably reduces the white opaque appearance of WSLs; however, this outcome is not resistant to discoloration.[191] In cases of more extensive mineral loss, however, grinding with diamond burs under water cooling or composite restorations is inevitable.

Resin Infiltration

More recently, a minimally invasive treatment approach was introduced, during which the WSL is infiltrated using a low-viscosity resin (ICON resin infiltrant, DMG, Hamburg, Germany) (Fig. 27-39). In this technique, the outer surface is transformed into a more permeable layer with the help of hydrochloric acid etching, and the porous structure beneath is infiltrated using a TEGDMA-based resin. It is noteworthy that this resin has a light refraction index similar to sound enamel, which improves the appearance of the lesion and reinforces the weakened enamel prism structure.[192–194]

The resin infiltration method was reported to produce satisfactory results, compared with remineralization using fluoride or amorphous calcium phosphate derivatives,[195–198] and is more resistant to discoloration, compared with microabrasion and in vitro fluoride treatments.[191]

FIGURE 27-39 Resin infiltration provides a minimally invasive treatment approach to transform the outer surface of affected teeth into a more permeable layer with the help of hydrochloric acid (HCl) etching, and infiltrating the porous structure beneath it using a triethylene glycol dimethacrylate (TEGMA)-based, low-viscosity resin. (Courtesy of DMG, Hamburg, Germany.)

Clinical procedure (Fig. 27-40):

1. Prophy teeth; rinse and dry.
2. Etch WSL with an Icon-Etch syringe (DMG America, Englewood, NJ), extending approximately 2 mm around the edges of the lesion for 2 minutes.
3. Rinse for 30 seconds, and completely dry with oil-free air.
4. Apply Icon-Dry (99% ethanol) (DMG America, Englewood, NJ) to the dried surface, and leave undisturbed for 30 seconds. Dry completely with air.
5. Apply ICON resin infiltrant (DMG, Hamburg, Germany) with a vestibular tip, and remove the direct overhead light source to avoid premature curing of the infiltrant. Leave undisturbed for 3 minutes. Maintain a moist surface by continuing to add infiltrant periodically during this time to ensure an adequate supply of resin to the lesion.
6. Remove any excess material, and light cure for 40 seconds.
7. Repeat the infiltration process with a new vestibular tip. Leave undisturbed for 1 minute, remove excess again, and light cure an additional 40 seconds.
8. Polish the teeth.

At this point, identifying lesions that are candidates for caries infiltration is critical; they contain the necessary porosities for infiltration. Lesions that occur from demineralization are the only suitable candidates for this procedure. Lesions from fluorosis, hypocalcification, hypoplasia, erosion, developmental anomalies, or trauma leading to enamel defects are not appropriate for caries infiltration.

BONDED RETAINERS

Permanent maintenance of the achieved result after successful treatment of malocclusion is undoubtedly a great, if not the greatest, problem for orthodontic clinicians, especially for adult patients.

A recent survey[5] indicates a dramatic increase in the tendency among U.S. orthodontists toward permanent retention and reports that fixed lingual retainers comprise approximately 85% of retention regimes followed. Bonded retainers have advantages such as:

1. Completely invisible from the front
2. Reduced need for long-term patient cooperation
3. Long-term (up to 10 years) and even permanent retention, whereas conventional retainers do not provide the same degree of stability

Bonded Fixed Retainer Materials

Early bonded fixed retainers were made with plain round or rectangular orthodontic wires,[199–203] but in 1977, Zachrisson proposed the potential advantage of the use of multistrand wire for their construction (see Multistranded Wire Retainers).[204] Novel materials proposed for lingual retainer fabrication include fiber-reinforced composite (everStick ORTHO, StickTech Ltd., Turku, Finland) and Ribbond polyethylene ribbon (Ribbond Inc., Seattle, WA). Prefabricated lengths of multistrand wire for bonded fixed retainers are also available on the market (Fig. 27-41, *A–D*).

Bonded Fixed Retainer Adhesives

Different composite resins have been advocated for bonding retainer wires.[205] Unlike the adhesive under a bracket, the lingual retainer resins remain exposed to the oral cavity and therefore require some specific physical properties. Several specific

lingual retainer adhesives may offer ease of application, optimal handling, improved patient comfort, and minimal abrasive wear (Fig. 27-42, *A* and *B*). Recent findings[114] indicate that light-activated composites may have these properties. The amount of total light energy delivered to the composite resin determines hardness, wear resistance, water absorption, residual monomer, and biocompatibility.[37,206] These highly filled, light-cured resin pastes are also said to be a better choice when longevity and durability are required.[19,37] A recent randomized trial indicates no evidence that the use of either chemical or light-cured composite is associated with a difference in failure rate or failure mode of bonded lingual retainers.[207]

Flowable resin composites have been made with a variety of formulas and viscosities for different uses.[208–213] Recently, using flowable composites, which were originally created for restorative dentistry by increasing the resin content of traditional microfilled composites, have been suggested for bonding lingual retainers.[214–216] These composites are claimed to be advantageous because no mixing is required, needle tips on the application syringes allow direct and precise composite placement, the composite is not sticky and flows toward the bulk of the material rather than away from it, no trimming and polishing are required, and chair time is reduced.[214] A recent study of the authors of this text demonstrated that flowable composites provide SBS and wire pull-out values comparable with a standard orthodontic resin and can be used as an alternative for direct bonding of lingual retainers.[217] However, higher microleakage scores at the wire-adhesive interface and unclear wear resistance make the use of flowable resin composites questionable.[218] These resins are best avoided in lingual retainer bonding until long-term clinical studies prove their dependability.

Periodontal Health with Bonded Fixed Retainers

Another issue with the use of bonded fixed retainers is the periodontal health. The gingival reaction depends on careful removal of excess adhesive at the time of retainer bonding by the clinician and on proper oral hygiene of the patients. Daily flossing in each interdental space is recommended with the use of a dental floss threader or Superfloss gingival to the wire.

Clinical data are conflicting; some research studies indicate no significant difference in plaque and calculous accumulation between round and spiral retainer wires,[219,220] whereas others report more plaque accumulation on the distal surfaces of the

FIGURE 27-40 **A,** Advanced enamel demineralization after fixed orthodontic therapy. **B,** Isolation of teeth as a preparation for infiltration treatment. The severity of decalcification is better appreciated after dehydration. **C,** Application of Icon-Etch (hydrochloric acid) (DMG America, Englewood, NJ) for 2 minutes. The enamel surfaces are then thoroughly rinsed. **D,** View of enamel after dehydration with Icon-Dry (ethanol) (DMG America, Englewood, NJ). The etch-and-dry cycles are repeated for a maximum of three times until an optimal visual effect is achieved. **E,** Application of the ICON resin infiltrant (DMG, Hamburg, Germany). The resin is light cured after two consecutive steps of infiltration. The cavity on tooth 12 is restored with resin composite. **F,** View of teeth after 1-week rehydration. **G,** Preoperative and postoperative cross-polarized images demonstrate the efficacy of infiltration therapy. (Courtesy of Dr. Zafer Çehreli, Ankara, Turkey.)

FIGURE 27-41 Materials proposed for lingual retainer fabrication include a fiber-reinforced composite (everStick ORTHO, StickTech Ltd., Turku, Finland) **(A)** and Ribbond polyethylene ribbon (Ribbond Inc., Seattle, WA) **(B)**. **C,** Prefabricated lengths of multistrand wire for bonded fixed retainers are also available today on the market but are prone to wear and break at interdental bridges in clinical service. **D,** Many clinicians choose to use Penta-One wire (Masel Orthodontics, Carlsbad, CA), a coaxial stainless steel wire in which five strands are wrapped around one, providing exceptional durability when used for lingual retainers. (**A,** Courtesy of GC Europe NV, Leuven, Belgium. **B,** Courtesy of Ribbond, Inc., Seattle, WA. **C,** Courtesy of Reliance Orthodontic Products.**D,** Courtesy of Masel Orthodontics.)

lower anterior teeth in patients with multistranded wire (MSW) retainers, compared with patients with round wire retainers.[221]

Many patients apparently have difficulties keeping the retainer area really clean, despite patient instruction in hygiene. Accumulations of supragingival calculus and stain are often noted along and beneath the retaining wire, whereas decalcification and caries are only exceptionally observed.[205,222] However, the presence of even large amounts of calculus around mandibular retainers is not alarming in young, healthy patients with no periodontal pockets according to a study, which compared the effect of toothbrushing after professional prophylaxis in patients with large amounts of calculus (removal requiring an average of 1 hour per patient) with the effect of toothbrushing as the sole hygiene method. The authors found no significant benefit of the calculus removal, which supports the hypothesis that it is not the calculus but the plaque that forms on it that has pathogenic potential.[223]

Recent research[221,224] demonstrates that the retainers are quite compatible with periodontal health even at long term, whereas other studies[225] question the appropriateness of lingual fixed retainers as a standard retention plan for all patients, regardless of their attitude to dental hygiene. Advanced periodontal cases probably need permanent retention (Fig. 27-43), but the effects of calculous accumulations on retainers in adults with existing periodontal problems are also unknown at present. Consequently, the selection of retention protocol and fixed retainer type may be based on case-specific parameters such as dental and gingival anatomy and oral hygiene status. In any case, the patient should be given detailed hygiene instruction for healthy preservation of the fixed lingual retainer.[10]

In the following section, two different retainer types are described.

1. Canine-to-canine lingual retainer bar (made of 0.030- to 0.032-inch plain round wire, bonded to the lower canines only)
2. Multistranded wire retainers (made of 0.0215-inch five-stranded wire, bonded to all teeth in a segment)

Canine-to-Canine Lingual Retainer Bar

In differential retention philosophy, the purpose of a bonded 3-3 retainer bar is to (1) prevent incisor recrowding, (2) hold the achieved lower incisor position in space, and (3) keep the rotation center in the incisor area when a mandibular anterior growth rotation tendency is present.

FIGURE 27-42 Specific lingual retainer adhesives may offer ease of application, optimal handling, improved patient comfort, and minimal abrasive wear over time. ((Top Image) Courtesy of 3M, © 2016 3M. All rights reserved; (Lower set of images) Courtesy Reliance Orthodontic Products, Inc.)

The standard appliance is bonded to the lingual surfaces of the canine teeth. The bar, which originally was constructed from plain blue Elgiloy wire with a loop at each terminal end for added retention[205] (Fig. 27-44, *A*), was replaced by a similar-diameter multistrand wire (Fig. 27-44, *B*). For some patients, this wire proved not solid enough and distorted, and the wire was difficult to bend to optimal fit. These drawbacks are eliminated in the third-generation design (Fig. 27-44, *C* and *D*), in which the bar is made from round 0.032-inch stainless steel or 0.030-inch gold-coated wire[204] and sandblasted on the ends for improved micromechanical retention. Bonding is performed with a chemically cured or light-cured composite resin because such adhesives provide the strongest bonds and show comparatively little abrasion over extended periods.[176]

In selected cases when the lower first premolars at the start of treatment are labially blocked out, severely rotated, or tipped, an extension of the 3-3 bar to include the first premolars (43-34 retainer) is useful. This extension is simply done by adding and bonding a small piece of thin wire between the premolar and canine (Figs. 27-45 and 27-46). The 43-34 design also may be used if, after orthodontic leveling of the six anterior mandibular teeth, the orthodontist desires to prevent their re-erupting above the functional occlusal plane.

Some companies supply preformed lingual 3-3 retainers with bonding pads, but these may be more difficult to fit and tightly bond. At the same time, obtaining maximal contact on the lingual surfaces of all four incisors may also be more difficult.

FIGURE 27-43 Adult female patient with advanced hard and soft periodontal tissue destruction and pathologic migration of the maxillary anterior teeth before (**A–E**), during (**F**), and after (**G–J**) orthodontic treatment. The improved dental result is retained by means of six-unit bonded lingual retainers in both dental arches (**H, I**). Some interdental gingival recession was unavoidable in the maxillary anterior region, but it does not clinically show much (**J**). The radiographs after treatment show no progression of periodontal tissue destruction, compared with the initial films (**C**).

FIGURE 27-43, cont'd

Bonding the 3-3 Retainer Bar

The following clinical recommendations (Fig. 27-47) represent basic principles that have been clinically tested over many years. Although taking shortcuts may seem possible, doing so is strongly discouraged; strict adherence to a meticulous technique has been found to be the key to long-term success.

1. While the orthodontic appliances remain in place, take a snap impression of the patient's teeth and pour a working model of hard stone.
2. Using the working model as a guide, bend a plain round stainless steel or gold-coated wire of 0.030- to 0.032-inch diameter with a fine, straight three-jaw or similar pliers

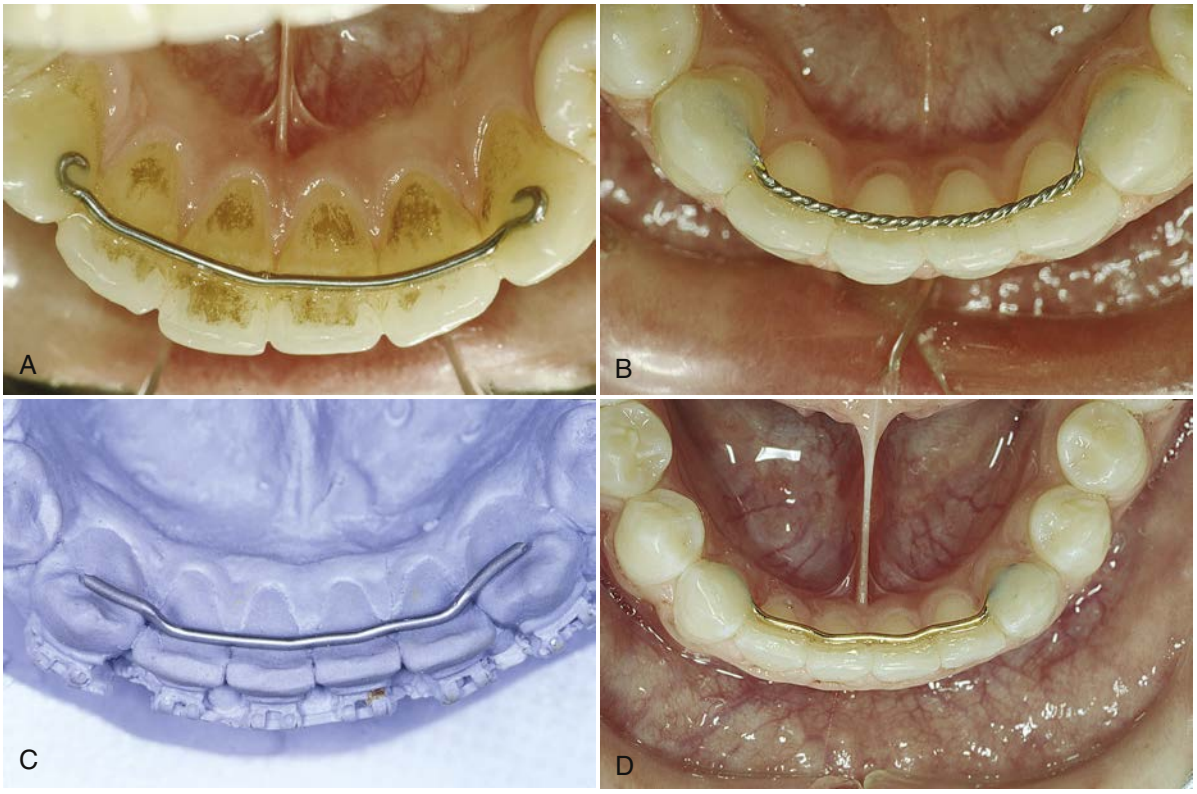


FIGURE 27-44 A, First-generation bonded mandibular lingual 3-3 retainer. B, Second-generation 3-3 retainer. C, D, Third-generation 3-3 retainer in stainless steel and gold-coated bar, respectively.

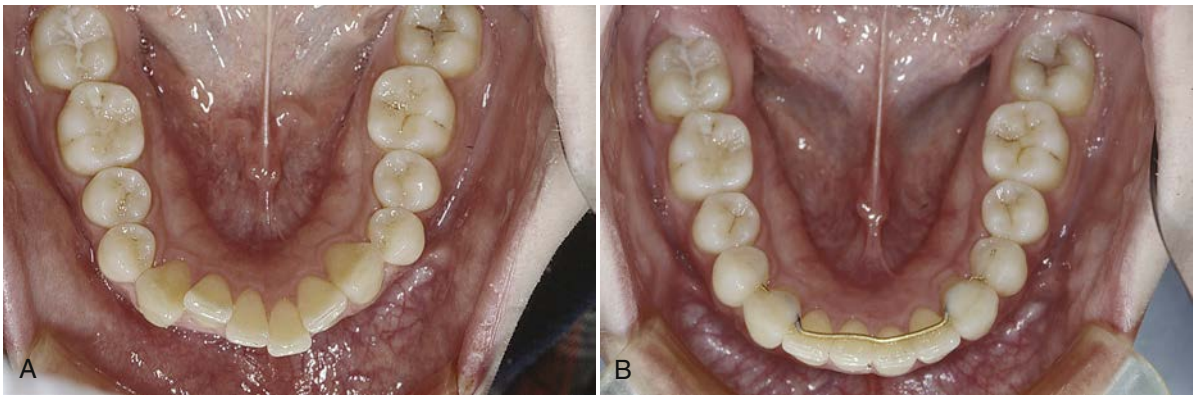


FIGURE 27-45 A, A 43-34 retainer can be used when the first premolars are labially blocked out or mesially tipped before treatment. B, The 0.030-inch 3-3 retainer bar is extended by means of a thin (0.0215-inch coaxial) gold-coated wire between the canine and the first premolar.

so that the wire precisely contacts the lingual surface of all mandibular incisors (see Fig. 27-47, *A*).

3. Sandblast the ends with 50 microns of aluminum oxide powder for approximately 5 seconds from different directions, using the MicroEtcher (Danville Materials, Inc., San Ramon, CA) (see Fig. 27-47, *B*) in a dust cabinet.
4. Clean the lingual surfaces of both canines with a tungsten carbide bur (No. 7006) or a large, round diamond bur.
5. Check the position of the wire in the mouth. When optimal, fix with three or four steel ligatures around the bracket wings of the incisors (see Fig. 27-47, *C* and *D*).
6. With the retainer wire in place, etch the lingual surfaces of the canines with colored phosphoric acid gel (see Fig. 27-47, *E*) for 30 seconds. Completely rinse and dry. Use a high-speed vacuum evacuator. Sealant is not needed on lingual surfaces, partly because of the reduced risk of decalcification. This fast and efficient procedure reduces the risk of moisture contamination.
7. Apply a thin coat of moisture-insensitive primer (Transbond MIP, 3M Unitek, Monrovia, CA) on the sandblasted ends of the retainer wire and on the etched enamel. This primer will reduce the risk of moisture contamination.
8. Apply the Transbond LR (3M Unitek, Monrovia, CA) adhesive to the right and left canines. Shape the resin bulk with fine brush strokes from the gingival margin to the incisal edge. A small amount of Transbond MIP on the

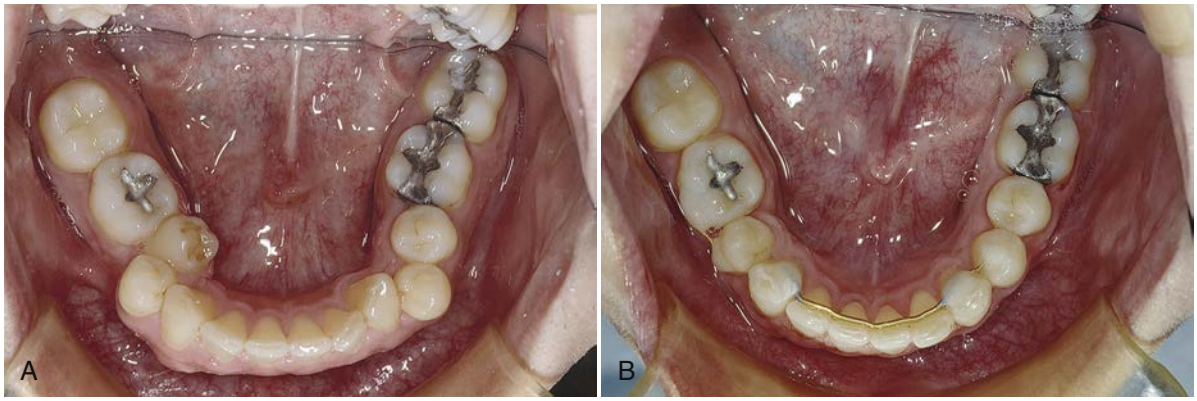


FIGURE 27-46 A, Adult patient with pretreatment blocked-out right second and left first premolar treated with the extraction of the second premolar on the right side. B, Final result is retained by means of a short labial gold-coated retainer in the closed extraction site and a 3-34 retainer.

brush tip will dilute the composite resin and make it flowable, creating a smooth, gentle contour in an incisogingival direction. It takes some experience to find the right consistency. If too much primer is added, then the adhesive will drift away from where it is placed and may interdentally flow and contact the gingiva. Optionally, the adhesive may be transferred from a mixing pad. The adhesive on the mixing pad should have a light-impermeable cover.

9. Light cure the composite resin according to instructions for light source used (e.g., 5 seconds for LED curing).
10. Cut the ligature wires. Trim (whenever necessary) along the gingival margin, and contour the bulk with an oval tungsten carbide bur (No. 7408; see Fig. 27-47, *I*) until it has a smooth contour in an incisogingival direction. Use a smaller bur (No. 2) interdentally.
11. Instruct the patient in proper oral hygiene and in the use of dental floss or Superfloss (Oral-B, South Boston, MA) beneath the retainer wire and along the mesial contact areas of both canines (Fig. 27-48). Instruct the patient to floss once daily to prevent the accumulation of calculus and plaque.

Failure Analysis and Long-Term Experience with the 3-3 Retainer Bar

Lingually bonded 3-3 retainers can provide excellent results[204,205,219,222,226] (see Fig. 27-44) if meticulous construction and bonding techniques are followed, along with some modifications of the original design.

Experience with bonded 3-3 retainer bars over 15 to 20 or more years is generally excellent, provided a careful bonding technique is used.[204,205,222] Particularly, the third-generation 3-3 retainer is a fine mandibular retainer.[226] Not only is the retainer solid, easy to place, and hygienic, but it also appears to be safer than mandibular retainers in which all six anterior teeth are bonded, which is equally important. A patient immediately notices whether a retainer comes loose when it is bonded only to the canines. The patient can then call for a rebonding appointment or remove the retainer, if necessary. For several years, a mandibular bar bonded only to the canines has been the authors' preferred retention method in adolescent and many adult patients.

Initial failures with first-generation bonded lingual 3-3 retainers were classified into two types. Type I failure related to the separation at the tooth-adhesive interface and occurred with the highest frequency. Type I failure most commonly resulted from moisture contamination or movement of the lingual bar during the initial polymerization of the composite. Type II failure occurred at the adhesive-retainer wire interface and resulted from inadequate bulk of adhesive for sufficient strength (or abrasive wear of the adhesive). An important note is that with adequate technique, one can avoid both types of failures. In other words, a clinician who experiences discouraging failure rates should reevaluate and improve the technique of making bonded lingual retainers.

The senior author's long-term (up to 12 years) experience with third-generation gold-coated 3-3 retainer bars[227] indicates the failure rates are considerably lower than those reported by others,[228] which is probably explained by the authors' careful bonding procedures (see Fig. 27-47).

Because the retainers are invisible, a problem may exist in deciding when to remove them. Extended retention periods (up to 10 years or longer) are now recommended by most clinicians. The long retention periods are favorable in many patients while waiting for the patient's third molars to erupt; long retention counters the effects of postpubertal growth activity and maxillomandibular adjustments, which may continue well into the second decade and longer. As an alternative, the bonded retainer may be replaced after several years with a removable one for long-term or permanent nighttime wear.[4, 5, 204, 224, 229, 230]

Multistranded Wire Retainers

Occasional cases of slight relapse anteriorly may occur when using retainers bonded only to the canines.[203] For this reason, a MSW retainer bonded to all anterior teeth may be indicated for adult patients with considerable pretreatment crowding.

Since the authors' early experiences in the mid-1970s with direct splinting of contacts between the incisors without wires were unsuccessful (they rapidly broke up into segments of one or two teeth because of their rigidity),[229] clinical experiments were begun with bonded flexible wires that allowed some physiologic movement of the individual teeth.[204] Such mobility was proven to be fundamental for the success of retainers bonded to several teeth in a segment.

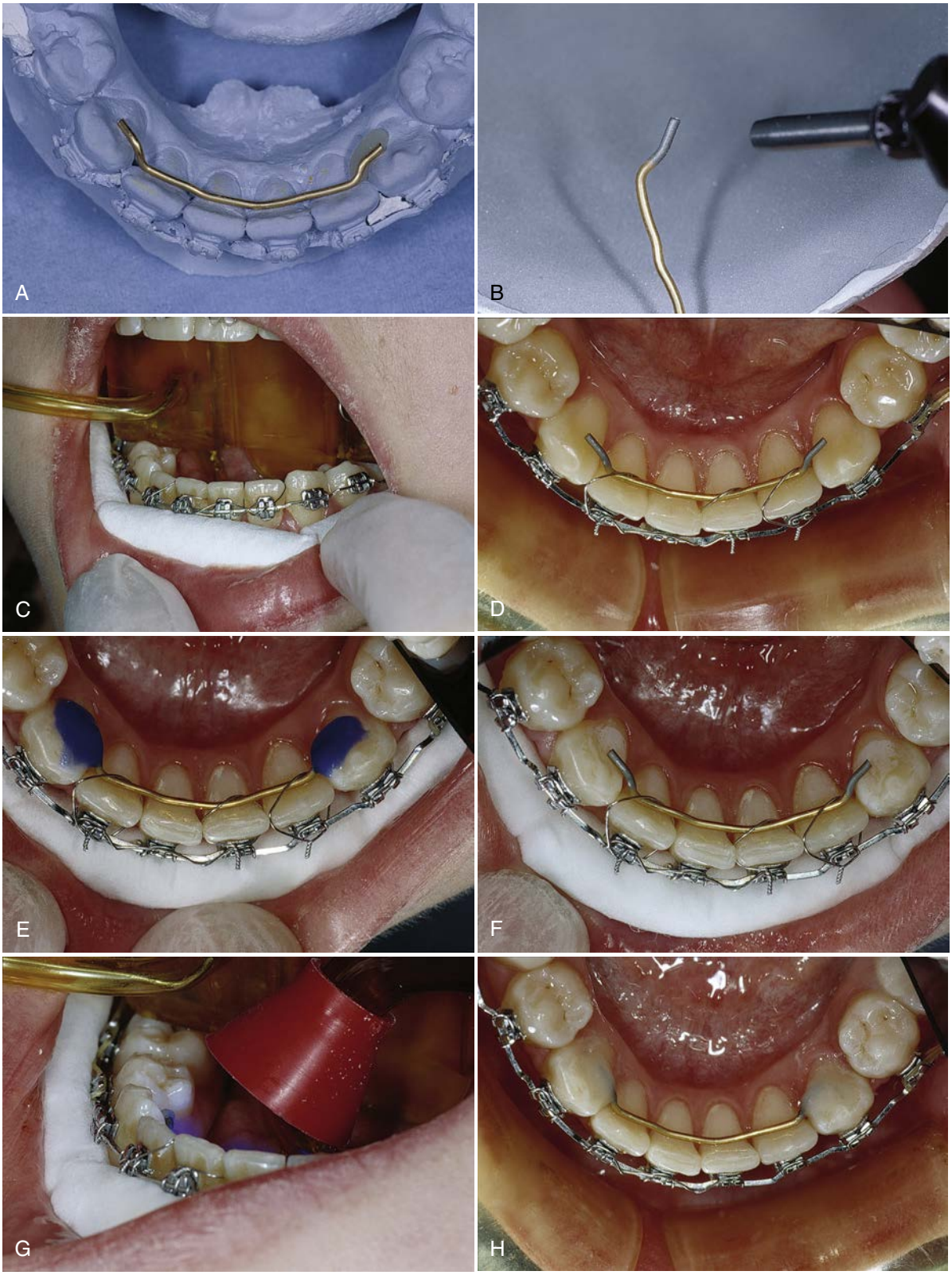


FIGURE 27-47 Making the bonded 3-3 retainer bar. **A,** Careful adaptation of retainer wire on stone model using fine three-jaw pliers. **B,** Sandblasting terminal ends of retainer bar. **C,** Lingual saliva ejector with high bite block (3M Unitek, Monrovia, CA) secures and optimally dries working field with no interfacing appliances. **D,** The 0.030-inch gold-coated wire is positioned by means of three steel ligatures. **E,** Ultratech 35% phosphoric acid gel for acid etching. **F,** Treated area clearly indicated. **G,** Initial tacking with small amount of flowable light-cured composite resin. **H,** Bulk of adhesive added to tacked retainer. **I,** Trimming adhesive with No. 7408 tungsten carbide bur. **J,** Final appearance.

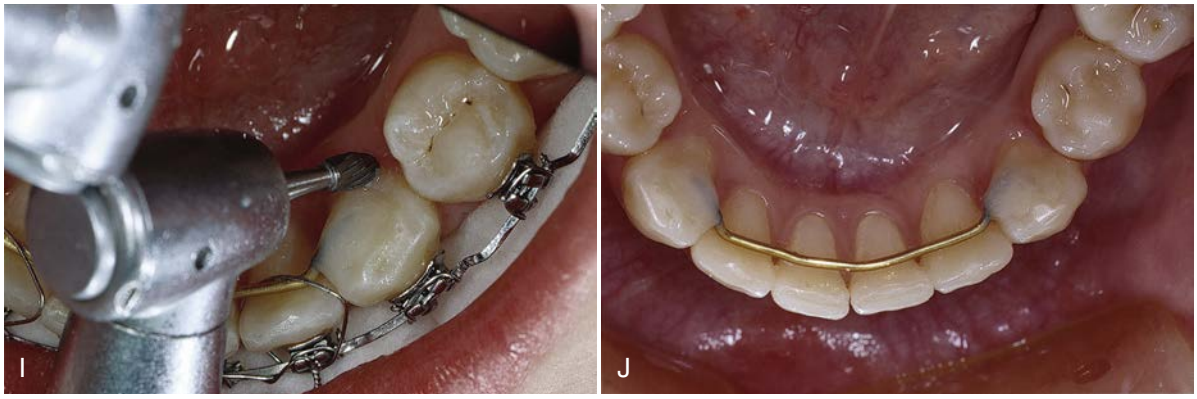


FIGURE 27-47, cont'd

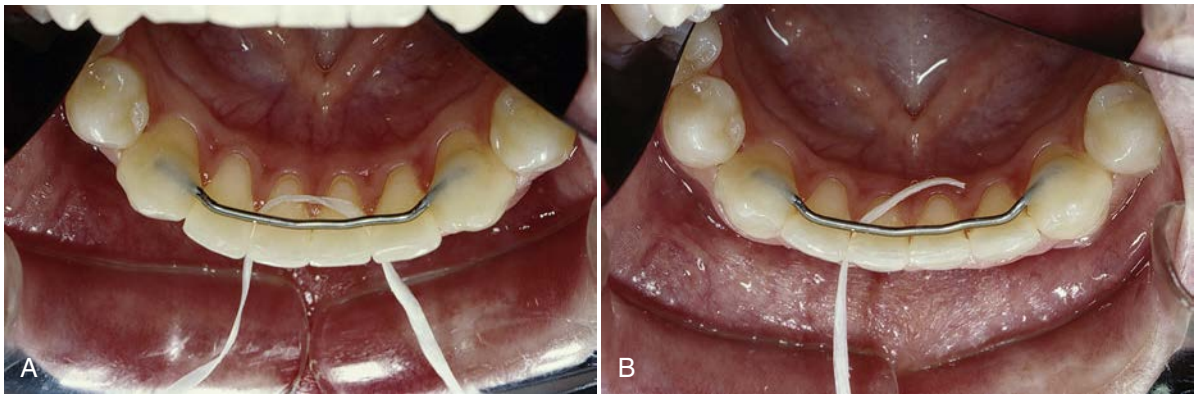


FIGURE 27-48 Interdental cleaning under a bonded 3-3 retainer. **A,** If a floss threader is not available, then a loop is formed over two incisors and moved under the retainer bar. **B,** When one end of the floss is pulled in, the other will snap free and can be grabbed with the fingers. Patients are instructed to move the floss over the interproximal surfaces once daily.

At least two indications or suggestions are helpful for using bonded MSW retainers (Fig. 27-49):

1. Prevention of space reopening
   a. Median diastemas
   b. Spaced anterior teeth
   c. Adult periodontal conditions with the potential for postorthodontic tooth migration
   d. Accidental loss of maxillary incisors requiring the closure and retention of large anterior spaces
   e. Mandibular incisor extractions
2. Holding of individual teeth
   a. Severely rotated maxillary incisors
   b. Palatally impacted canines

In these and other situations, the bonded spiral wire retainer can be used alone or with a removable retainer.

Bonding Multistranded Wire Retainer

Direct bonding of Multistranded wire retainer. The following clinical direct-bonding procedure is advocated for bonding with chemically cured or light-cured composite resin, respectively (Figs. 27-50 to 27-52):

1. Toward the end of orthodontic treatment, take a snap impression and pour a working model in stone.
2. Using fine, three-pronged wire-bending pliers and marking pen, closely and passively adapt the 0.0215-inch Penta-One steel wire (Masel Orthodontics, Carlsbad, CA) or gold-coated wire (Gold'n Braces, Inc., Palm Harbor, FL) to the crucial areas of the lingual surface of the teeth to be bonded. Cut the wire to the required length.
3. Check the retainer wire in the mouth for a good fit in an entirely passive state, and adjust, if necessary.
4. Clean the surfaces to be bonded with a tungsten carbide or diamond bur, and etch for 30 seconds with phosphoric acid gel (see Figs. 27-50 and 27-51).
5. Use a four-handed approach (or similar) for the initial tacking. Hold the wire by hand in the optimal position while tacking it to one incisor with a small amount of Transbond LR (3M Unitek, Monrovia, CA) (see Figs. 27-51, *E* and *F*, and 27-52, *B*). Check the wire for passive tension after tacking. If the wire is passive, then add more adhesive and light cure the remaining teeth; if it is not, then remove the wire and start over again.
6. Contour the bulk of adhesive with the brush dipped into the primer. Optionally, transfer the adhesive from a mixing pad, which should have a light-impermeable cover. Importantly, the adhesive must cover a large labiolingual area over the wire for strength and wear resistance. Trim with burs when necessary.
7. Instruct the patient in proper oral hygiene and in the use of dental floss and in each interdental area with a floss threader or Superfloss (Oral-B, South Boston, MA).

Indirect bonding of Multistranded wire retainer. The fixed lingual retainer also can be fabricated with an indirect technique

FIGURE 27-49 Four different clinical situations in which a lingual multistranded wire retainer is used for improved retention. The cases represent significant midline diastema of maxillary central incisors (**A, B**), bilaterally missing maxillary lateral incisors (**C, D**), one lower incisor extraction in Class III plus open-bite tendency case (**E, F**), and two palatally impacted maxillary canines (**G, H**). In **D,** the six-unit retainer is bonded in the occlusal fossa of the first premolars, whereas in **H,** a short labial retainer is bilaterally used to stabilize the mesially rotated and palatally displaced canines and the distally rotated first premolars.

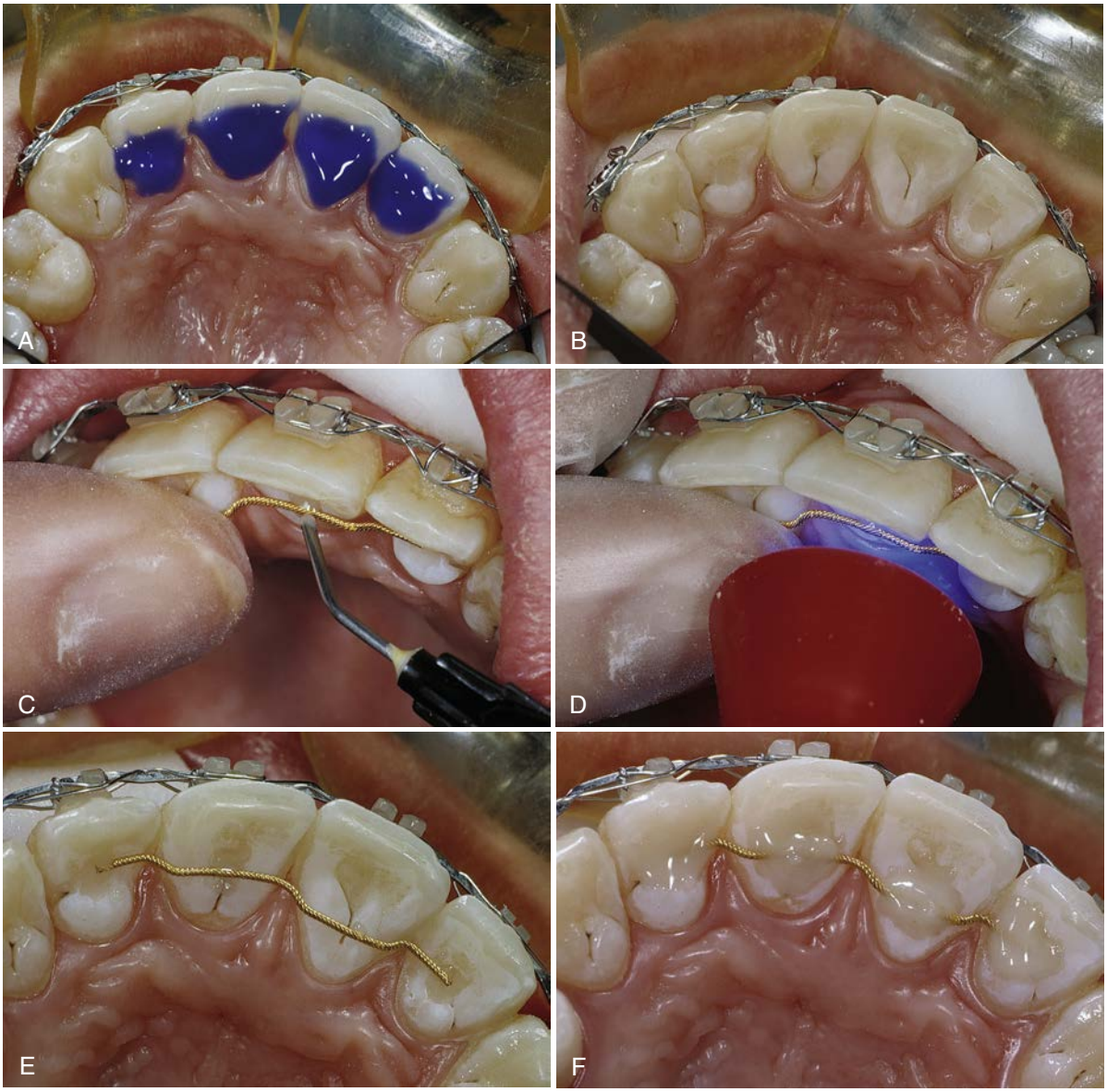


FIGURE 27-50 Fabrication of four-unit multistranded wire retainer with chemically cured composite resin. A gold-coated Penta-One wire is carefully adapted on a model with fine three-jaw pliers to fit the lingual contours of the incisors passively. **A, B,** Acid etching of the lingual surfaces of the upper incisors is demonstrated. **C, D,** The initial tacking to one incisor is made with flowable light-cured resin, with the wire held in the optimal position by a finger. This initial tacking to one tooth allows direct checking of position and fit of the retainer wire and is the key to avoid unwanted tooth movement as a side effect during the retention period. When correct and passive, the remaining teeth are tacked next with a small amount of light-cured flowable resin (**E**) before the bulk of adhesive is added in a gingival-occlusal movement (**F**). A thin mix of composite resin is then added with an explorer to fill in the bond mesially and distally on each tooth. Trimming is made with tungsten carbide burs (**G–I**). The No. 7006 is ideal incisal to the wire to avoid occlusal interference, whereas the contour gingival to the wire is made with the No. 7408 bur. **J,** Final result.

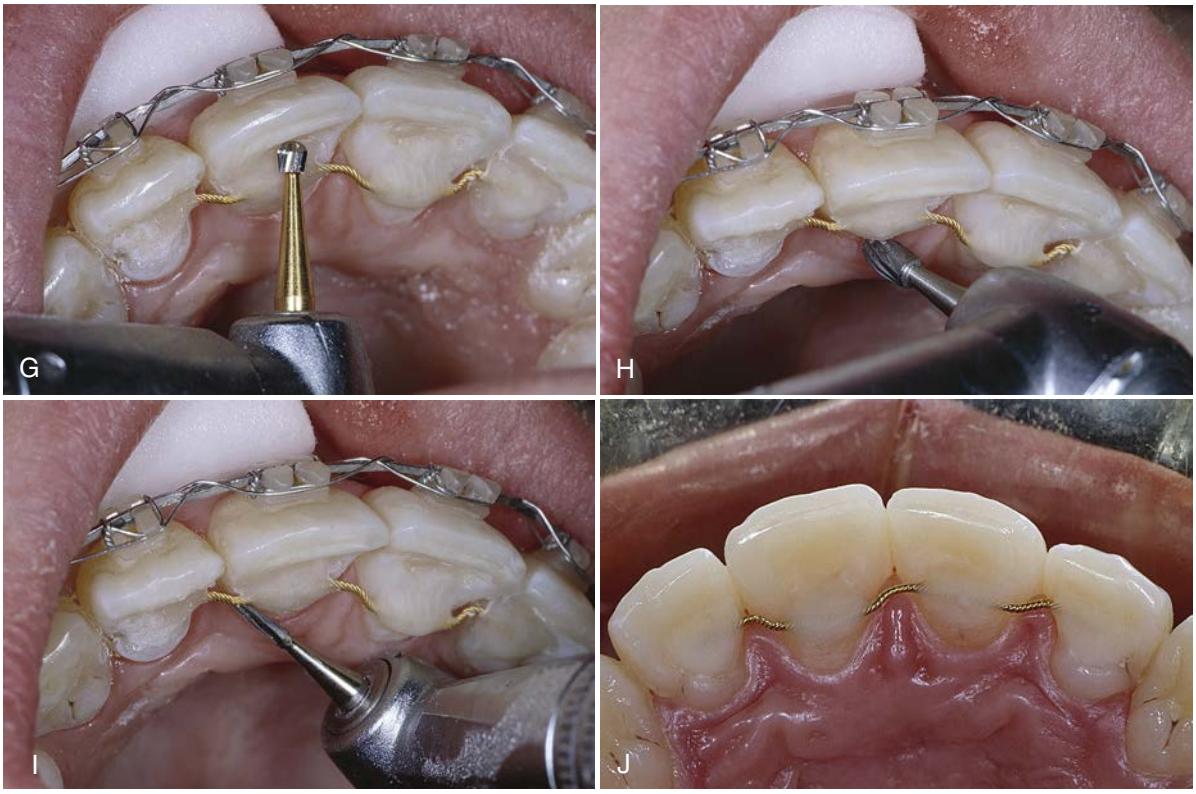


FIGURE 27-50, cont'd

as described elsewhere.[231] A practical approach for lingual retainers is to use indirect bonding with a 2-mm-thick polyethylene thermoplastic transfer tray and Transbond LR and Sondhi Rapid Set (3M Unitek, Monrovia, CA) as adhesive resins.[231] A recent randomized clinical trial indicates that indirect bonding is faster and shows a comparable failure rate.[232]

Failure Analysis and Repair

Experiments in the late 1970s and early 1980s used different sizes (0.015- to 0.020-inch diameter) and types of multistranded wires.[204] Early findings included the following:

1. The incidence of wire breakage appears to decrease with increasing wire diameter.
2. An unacceptable incidence of bond failures occurs when the wires are bonded to the lingual surfaces of premolars.[1,204]

The mandibular 3-3 bar and 321-123 retainer show similar success rates.[227] Dahl and Zachrisson reported in 1991 that for the five-stranded Penta-One wire (Masel Orthodontics, Bristol, PA), failure rates for loosening were 8% in the maxilla and 6% in the mandible; the failure rates for wire fracture were 3% in the maxilla and were nonexistent in the mandible.[233] Other authors, on the other hand, have reported failure rates of 37% to 46% over 2 to 3 years, which included any type of failure and may have exaggerated the overall failure rate.[207,234]

The discrepancies between the authors' experience and that of other studies probably can be explained by fewer occlusal interferences (with less contact with opposing teeth to allow for more wear) and by technical factors, such as adequate buccolingual width of composite over the wire, smooth contouring of the adhesive, completely undisturbed setting of the adhesive in every case, and careful adaptation of the wire to the lingual contours of the teeth. The reduction of wire breakage compared with earlier results is related to the increased flexibility of five smaller wires occupying the same diameter as the three larger wires in previous retainers. Because a common mode of failure with bonded MSW retainers is abrasion of composite and subsequent loosening of bonds between wire and composite,[220] one is advised to avoid occlusal contact or to add a thick layer of adhesive over the wire. Even in the absence of tooth contact, such as in the mandible, mechanical forces (e.g., tongue activity, tooth brushing) may cause notable abrasion over the years.

Because the failure rates significantly increase when the canines (and first premolars) are included in a maxillary MSW retainer, using a four-unit design, combined with a removable plate (Figs. 27-53 and 27-54) rather than a six-unit bonded retainer (Figs. 27-55 and 27-56), can be a safer alternative for routine retention in children and adults.[234]

The most common problem after wire fracture or the loosening of the bonding site(s) in MSW retainers is unwanted movement of one or more teeth. At this stage, the teeth are not firmly seated in their sockets and therefore can be generally forced back into position using techniques such as heavy pull with one or two steel ligatures (Fig. 27-57, *A* and *B*).

When the repair is made, a temporary contact splint using composite resin or a temporary bonded labial wire has proved to be of considerable value. The latter normally provides better stability and allows a good working area with undisturbed setting of the repair adhesive (Fig. 27-57, *C* and *D*). After the repair, the temporary labial wire (or contact splint) is removed with tungsten carbide burs.

Long-Term Experience with the Multistranded Wire Retainer

MSWs are invisible and can be placed out of occlusion in most instances. If not, then hiding the wire under a slight groove

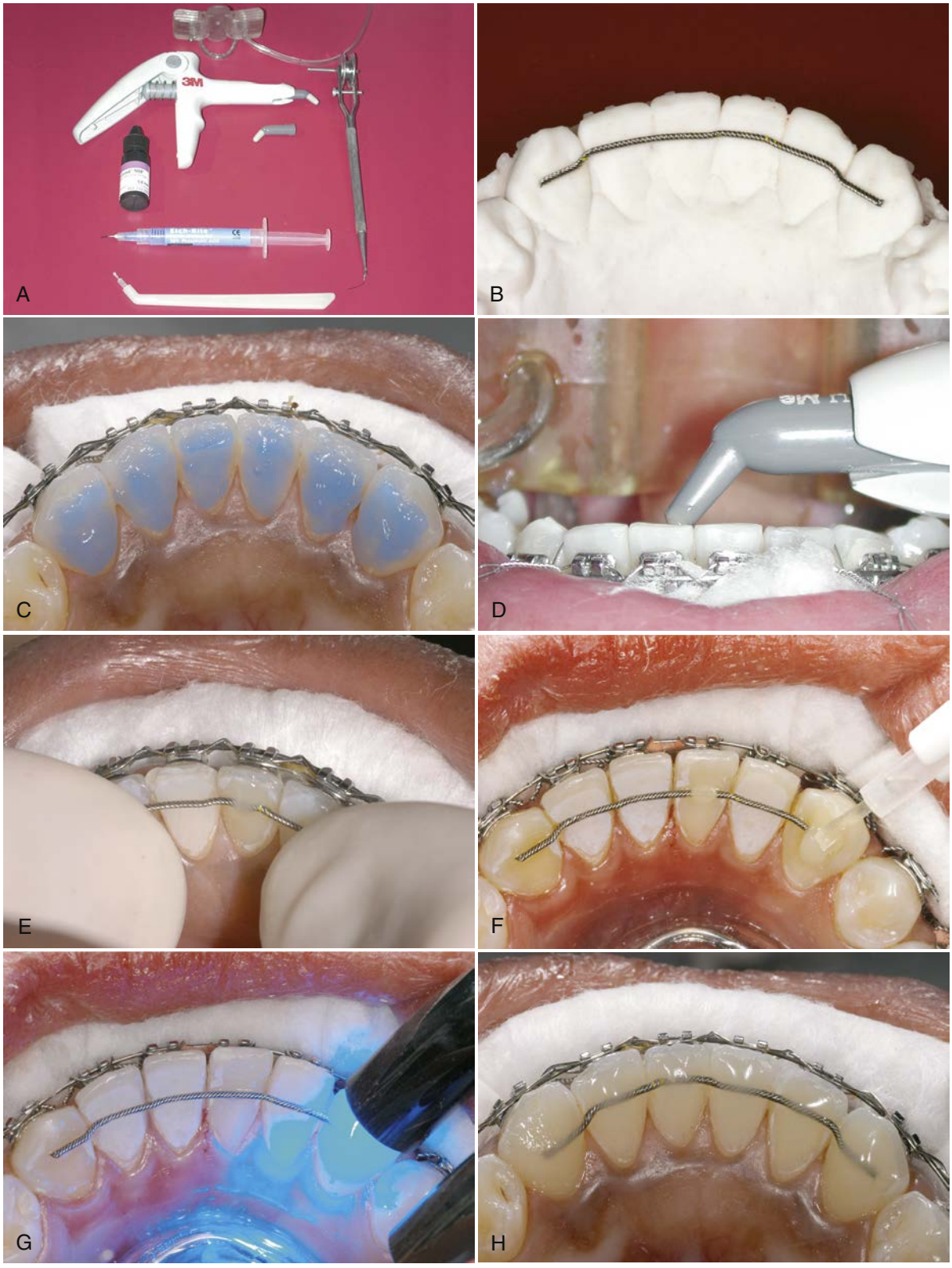


FIGURE 27-51 Instruments (A) and method (B–G) for fabrication of six-unit lingual multistranded wire retainer with light-cured composite resin. After a 0.0215-inch stainless steel or gold-coated Penta-One wire is adapted for optimal fit on the lingual surfaces of all teeth (B), the teeth are acid etched with phosphoric acid gel (C). Composite resin is added to one incisor (D, E) and light cured. After checking that the wire is passive and has a good fit to the remaining teeth, composite resin is added, shaped with the aid of liquid resin and fine brush (F), and light cured (G). H, Final result.

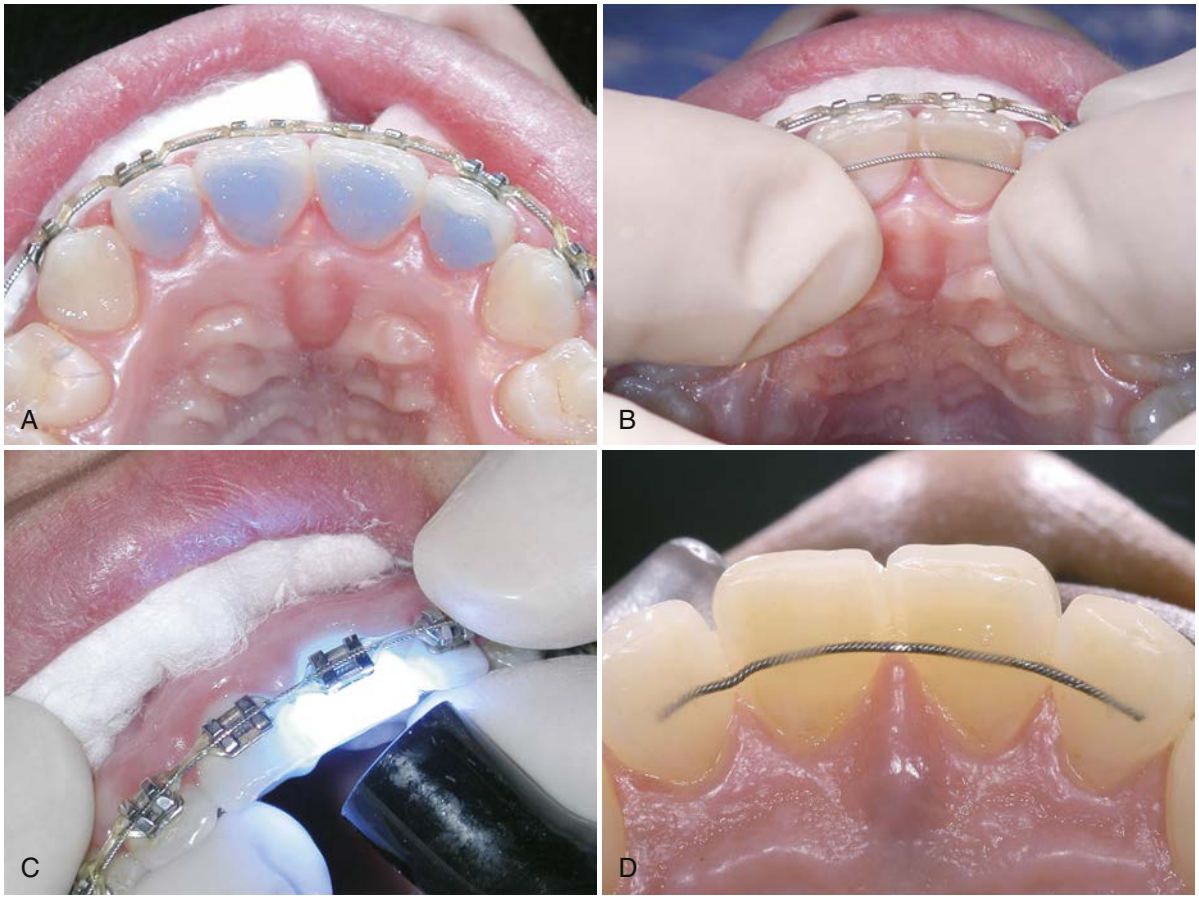


FIGURE 27-52 Fabrication of four-unit multistranded wire retainer with light-cured composite resin. **A,** Etching with phosphoric acid. **B,** Finger-holding of wire while tacking one incisor. **C,** Light curing the remaining teeth. **D,** Final result. (See text for details.)

in the enamel is a possibility and can be used alone or with removable retainers, and therefore the patient acceptance of the MSW retainer is excellent.[204,228,233] In addition, adults especially appreciate that the stability of the treatment result does not depend on their cooperation, which is the case when removable retainers are continuously worn or are worn at night.

MSW retainers allow safe retention of treatment results when proper retention is difficult or even impossible with traditional removable appliances. Recent research indicates the alignment of the mandibular anterior teeth is stable in 90.5% of patients at the 5-year follow-up, whereas in 9.5%, a mean increase of 0.81 mm in the irregularity index was observed.[235] MSW retainers are also better at maintaining incisor alignment than a round retainer bar.[221] The favorable long-term success can be attributed to the allowance of slight movement of all bonded teeth and segments of teeth with the MSW. Side effects in the form of undesirable movement of bonded teeth, on the other hand, may occur if the wire is too thin (three-stranded 0.015- to 0.0195-inch wires) or not entirely passive while bonding.[233,236]

When patients with previous multiple spacing of anterior teeth were in the retention phase of treatment, small spaces (1 to 2 mm) often opened distal to the terminal ends of the retainer wire after approximately 6 months. Because these spaces apparently did not open further, the conclusion was that they illustrated a settled occlusion with the MSW retainer in place in a new state of physiologic equilibrium.[205,228,233] Depending on the occlusion and the patient's dental awareness, such spaces could be filled with mesiodistally extended fillings or crowns or could be allowed to remain.

The 3-3 bar is a safe retainer, and this design may be recommended for most children. For adults and adolescent patients with pretreatment spacings and similar malocclusions, the bonding of all six anterior teeth may be preferable.

At present, little is known about the length of time that the bonded MSW retainer should be left in place. The type of original malocclusion and the patient's age and ability to keep the retainer clean may be decisive factors. As long as the retainer is intact, the treatment result is maintained; and as long as the patient performs adequate plaque control, no real reason exists to remove the retainer.

In selected cases, retainers may be used for permanent stabilization.[204,222] Further follow-up research is needed for semipermanent and permanent use of bonded retainers. As discussed for the 3-3 retainers, using the bonded lingual retainer for a prolonged retention period and then replacing it with a removable retainer for nighttime wear on a more permanent basis may be practical in some cases.

Direct-Bonded Labial Retainers

Clinical experimentation with short labial retainers was started in the late 1980s to try to improve the long-term results in

FIGURE 27-53 Recommended version of the removable plate to be used with a four-unit bonded lingual retainer. The rectangular (0.019- × 0.026-inch) labial wire of this plate distally extends to the lateral incisors and has a soldered extension wire to prevent flaring of the canines. A holding clasp of 0.8-mm round wire is distal to the second molars.

some specific retention situations.[228] The following were typical problems:

1. Inability to prevent some space reopening in closed extraction sites in adults
2. Tendency for some lingual relapse of previously palatal-impacted canines
3. Space reopening when molars and premolars had been mesially moved in cases with excess space

Common to these situations was that some support in the premolar area for 1 to 2 years appears advantageous to improve stability. The background for labially bonding retainer wires was based on unsatisfactory results when the orthodontist bonded wires to the lingual surface of premolars. The alternative—occlusally bonding the wire in the premolars—presents other problems. In most instances, antagonistic contact cannot be avoided unless a groove is prepared, which is probably not acceptable in routine situations. It was decided therefore to bond short retainer wires labially to examine success rates and patient reactions.

Technical Procedure

In principle, the fabrication of labial retainers is similar to the technique used for direct bonding of lingual retainers.

1. A straight piece of 0.0215-inch Penta-One wire (gold-coated or stainless steel) (Masel Orthodontics, Bristol, PA) is cut to the desired length.
2. After etching, the retainer wire is tacked on the teeth.
3. After the adhesive sets, a bulk of adhesive is added.
4. Contour trimming of excess is performed with tungsten carbide burs (No. 7408 and No. 7006), and interdental trimming is performed with small round burs (No. 1 or No. 2). Care is taken to avoid contact between composite and gingival margin at the bonding sites, as well as contact between the interdental papillae and the retainer wire.

Long-Term Results

The first follow-up study of direct-bonded labial retainers as reported by Axelsson and Zachrisson[228] demonstrated excellent results for short segments (two teeth) regarding bond success rate and, surprisingly, for patient acceptance. A gold-coated labial wire (Fig. 27-58) is understandably more acceptable than a steel wire, even if some of the plating may wear off over time. The failure rates for retainers of two teeth were approximately 4% over an average period of 2 years. The retainers were placed over closed extraction sites in adults (Fig. 27-59) or for added retention of previously palatally impacted canines (see Fig. 27-49, *H*).

When longer retainers (three to four teeth) were labially placed in the mandible, however, the bond failures increased significantly.[228]

OTHER APPLICATIONS OF BONDING

Bonding a Large Acrylic Appliance

Various acrylic appliances are used in clinical practice usually to expand the maxilla. Many of these appliances cover the upper buccal segment and require a certain amount of retention during their active phase. Usually the permanent dentition phase is uncomplicated with more than enough anatomic undercuts to hold the appliance in position. However, retaining such appliances in the deciduous or mixed dentition periods requires a dependable approach.

FIGURE 27-54 Recommended routine retention for adolescent patients. Young girl with unilateral crossbite (**A–C**) after orthodontic treatment involving four premolar extractions (**D, E**). **F,** After treatment. Retainers include an upper four unit multistranded wire retainer (**G**), a lower 3-3 bar (**H**), and a removable plate.

FIGURE 27-55 A–C, Combination of six-unit bonded lingual retainer and simplified Crozat appliance for retention in an adult female patient with an anteriorly constricted maxillary dental arch and rotated and blocked out lateral incisors and canines. E, The Crozat appliance is optimal for long-term retention of crossbites in adults. If the appliance is not worn for some time and slight transverse relapse occurs, then its flexibility allows for recovery (similar to a spring retainer), in contrast to what is possible with a conventional removable plate. Note improvement of smile fullness (F) compared with the start (A).

FIGURE 27-56 A, C, Recommended version of removable plate to be used with a six-unit bonded lingual retainer. The labial wire of this plate extends distal to the bonded retainer to avoid the risk of retainer wire fracture. The acrylic of the plate can be ground away from the teeth involved in the bonded retainer (B, D).

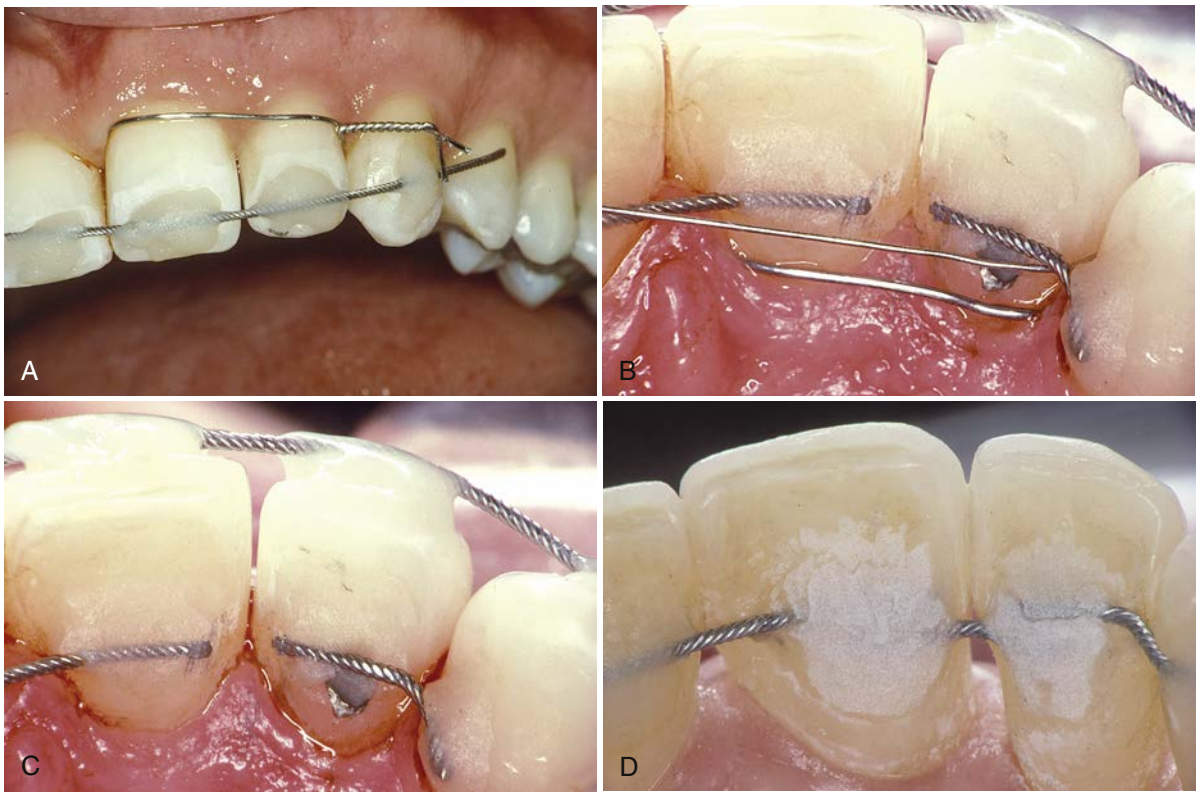


FIGURE 27-57 Repair of broken retainer (fatigue fracture of wire between left central and lateral incisor), using labial temporary wire for stabilization during rebonding. When the loose teeth have been pulled together with steel ligatures (A, B) to close a small space, the temporary wire is labially bonded with adhesive after a 5-second etch. After setting, the steel ligatures can be removed to provide a nice working field (C) where the repair wire can be bonded with no disturbed setting gingival to the main retainer wire (D).

FIGURE 27-58 Slight space reopening distally to short labial retainer in an adult woman requiring upper first premolar extraction. **A, B,** Gold-coated labial retainers. **C,** The reopening evidently reflects a tooth size discrepancy that can be addressed when remaking the amalgam fillings. **D,** The labial wire is inconspicuous on smiling.

Technical Procedures

Permanent Dentition

1. Clean the teeth to be bonded with pumice slurry. Wash thoroughly and air dry.
2. Cement the appliance with polycarboxylate cement. Have the patient bite down on a cotton roll or gauze. Remove the excess cement after approximately 30 to 60 seconds while still in the plastic phase.
3. Alternatively, if a certain amount of moisture cannot be avoided, use a hybrid glass ionomer cement (Multi-Cure Glass Ionomer Cement, 3M Unitek, Monrovia, CA). This cement works best on a moist surface; however, glass ionomers do not tolerate additional water or saliva introduced during the curing process. Cure for 10 seconds per tooth. Cement will also be chemically cured after 5 minutes.

Deciduous or Mixed Dentition

1. Clean the teeth to be bonded with pumice slurry. Thoroughly wash and air dry.
2. To improve the adhesion between the adhesive and the appliance, apply a uniform coat of plastic appliance conditioner (Reliance Plastic Appliance Conditioner, Reliance Orthodontic Products, Itasca, IL) or methacrylate monomer to the inner side of the acrylic appliance.
3. Acid etch the buccal and lingual tooth surfaces and the distal surface of the last molar. Occlusal surfaces should not be etched to facilitate clean-up at debonding. Thoroughly rinse and dry.
4. Apply a thin coat of adhesion booster (Assure, Reliance Orthodontic Products, Itasca, IL) to the etched deciduous enamel, and lightly air dry.
5. Load the appliance with light-cured band adhesive (Ultra Band-Lok, Reliance Orthodontic Products, Itasca, IL). When using colored band adhesives, turning off or dimming the overhead light is important, as these lights (particularly current LEDs) may cause premature curing of the resin.
6. Place the appliance into the mouth, and clean up the flash before light curing for 30 seconds per tooth with a conventional

FIGURE 27-59 **A–C,** Young adult female patient with typical Class II, Division 2 malocclusion before treatment. The maxillary first molar was extracted as part of treatment (**C**). The second molar and first premolar were held together with a short gold-coated labial retainer. The maxillary third molar is erupting. **D–F,** Note the improved maxillary canine occlusion and incisor torque.

halogen. Curing can be faster with new-generation light sources (Fig. 27-60).

Occlusal Buildup of Posterior Teeth

Occlusal buildup of posterior teeth is often necessary when bonding to the lower teeth in patients with deep bites or with severely malposed teeth or when a tooth or group of teeth in the crossbite is brought into the arch, which is easily achieved by composite occlusal buildups. Using colored light-cured band adhesives (Ultra Band-Lok, Reliance Orthodontic Products, Itasca, IL) is a good practice for easy detection and to remove the remnants completely.

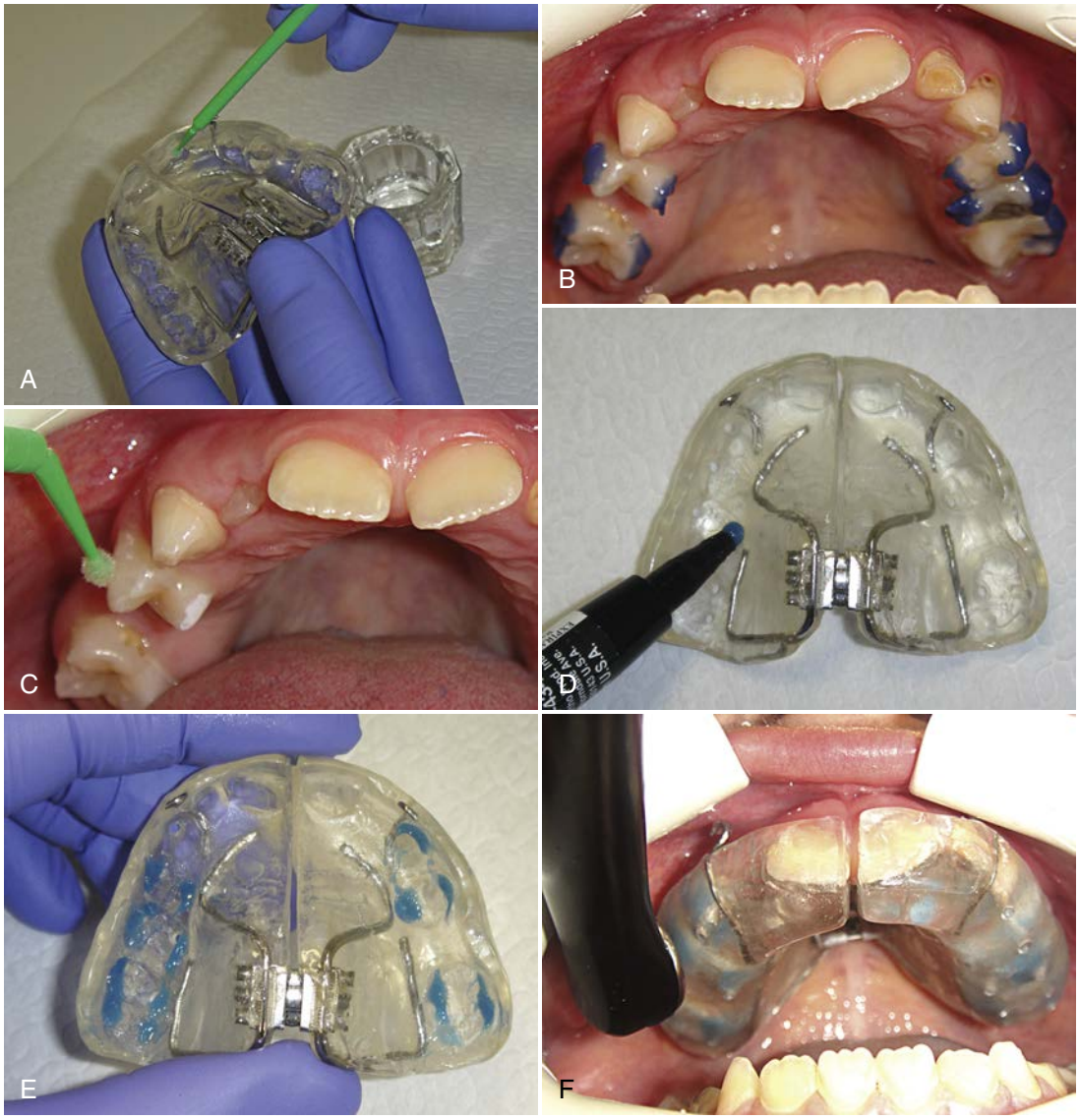


FIGURE 27-60 Various appliances may need to be bonded to teeth in the deciduous or mixed dentition period to provide better retention. All surfaces to be bonded should be cleaned with pumice slurry, thoroughly washed, and air dried. **A,** A uniform coat of Reliance Plastic Appliance Conditioner (Reliance Orthodontic Products, Itasca, IL) or methacrylate monomer is applied to the inner side of the acrylic appliance to improve adhesion between the adhesive and the appliance. **B,** The surfaces to be bonded are acid etched and thoroughly rinsed and dried. Etching the occlusal surfaces is avoided to facilitate clean-up during the debonding session. **C,** A thin coat of adhesion booster is applied to the etched enamel surfaces. **D,** The appliance is loaded with a light-cured band adhesive. **E,** An ungenerous amount of adhesive is loaded; the excess adhesive will move on to the occlusal surfaces and will complicate cleaning. **F,** The appliance is firmly placed into the mouth, and any flash is cleaned before light curing for 30 seconds per tooth with a conventional halogen. Curing can be faster with new-generation light sources. (Courtesy of Dr. Sabri Ilhan Ramoglu, Istanbul, Turkey)

Technical Procedure

1. Clean the occlusal surface to be bonded with pumice slurry. Thoroughly wash and air dry.
2. Acid etch the occlusal surface for 15 seconds, and avoid etching the deep occlusal groves for easy removal at the debonding session.
3. Apply colored band adhesive, and shape and adjust the resin bulk to the required height with fine brush strokes. As with appliance bonding, turning off or dimming the overhead light is important, as these lights (particularly current LEDs) may cause premature curing of the resin. Light cure for 30 seconds with a conventional halogen. Curing can be faster with new-generation light sources (Fig. 27-61).

Occasionally, some add-on may be required at the following appointments if the tooth with buildup gets intruded. The bulk can be easily removed by careful force application with a Weingart plier after treatment, and any remaining adhesive can be removed with a scaler.

FIGURE 27-61 **A,** The bite needs to be temporarily raised to bring the upper right lateral incisor into the arch. Temporary occlusal composite build-ups will be used for this purpose. **B,** The occlusal surface to be bonded is first cleaned with pumice slurry and thoroughly washed and air dried. **C,** The occlusal surface is acid etched for 15 seconds, avoiding etching the deep occlusal groves for easy removal during the debonding session. **D,** Note the frosty white appearance of the cusp tips and unconditioned occlusal grooves. **E,** Colored band adhesive is applied, and the resin bulk is shaped and adjusted to the required height with fine brush strokes and light cured. **F,** The raise of the bite can be seen. Note that an even occlusal contact should be provided on the contralateral side as well. (Courtesy of Dr. Sabri Ilhan Ramoglu, Istanbul, Turkey)

CONCLUSION

Today, adhesive resins, direct bonding, and light curing units are a very important part of the modern orthodontist's armamentarium. Our distinguished profession would not have been popularized without the application of direct bonding and advances in the material science.

Similar to any other material, composite resins and bonding have their particular benefits and drawbacks. Beyond a doubt, modern orthodontists need to have a thorough knowledge and comprehension of the materials available so that they can choose the best product available for their particular needs and to make the best use of them.

REFERENCES

1. Zachrisson BU. A posttreatment evaluation of direct bonding in orthodontics. *Am J Orthod*. 1977;71:173–189.
2. Mannerberg F. Appearance of tooth surface. *Odontol Rev*. 1960;11(Suppl 6):1–116.

3. Schaneveldt S, Foley TF. Bond strength comparison of moisture-insensitive primers. *Am J Orthod Dentofacial Orthop*. 2002;122:267–273.
4. Keim RG, Gottlieb EL, Nelson AH, et al. 2008 JCO study of orthodontic diagnosis and treatment procedures, part 1: results and trends. *J Clin Orthod*. 2008;42:625–640.
5. Keim RG, Gottlieb EL, Vogels DS, et al. 2014 JCO study of orthodontic diagnosis and treatment procedures, part 1: results and trends. *J Clin Orthod*. 2014;48:607–630.
6. Swartz ML. Orthodontic bonding. *Orthod Select*. 2004;16:1–4.
7. Buonocore MG. A simple method of increasing the adhesion of acrylic filling materials to enamel surface. *J Dent Res*. 1955;34:849–853.
8. Maijer R, Smith DC. Variables influencing the bond strength of metal orthodontic bracket bases. *Am J Orthod*. 1981;79:20–34.
9. Sharma-Sayal SK, Rossouw PE, Kulkarni GV, et al. The influence of orthodontic bracket base design on shear bond strength. *Am J Orthod Dentofacial Orthop*. 2003;124:74–82.
10. Usumez S, Erverdi N. Adhesives and bonding in orthodontics. In: Nanda R, Kapila S, eds. *Current Therapy in Orthodontics*. 1st ed. St. Louis: Mosby; 2009:45–67.
11. Bishara SE, Olsen M, VonWald L. Comparisons of shear bond strength of precoated and uncoated brackets. *Am J Orthod Dentofacial Orthop*. 1997;112:617–621.
12. Odegaard J, Segner D. Shear bond strength of metal brackets compared with a new ceramic bracket. *Am J Orthod Dentofacial Orthop*. 1988;94:201–206.
13. Millett D, McCabe JF, Gordon PH. The role of sandblasting on the retention of metallic brackets applied with glass ionomer cement. *Br J Orthod*. 1993;20:117–122.
14. Regan D, LeMasney B, Van Noort R. The tensile bond strength of new and rebonded stainless steel orthodontic brackets. *Eur J Orthod*. 1993;15:125–135.
15. Wang WN, Meng CL, Tarng TH. Bond strength: a comparison between chemical coated and mechanical interlock bases of ceramic and metal brackets. *Am J Orthod Dentofacial Orthop*. 1997;111:374–381.
16. MacColl GA, Rossouw PE, Titley KC, et al. The relationship between bond strength and orthodontic bracket base surface area with conventional and microetched foil-mesh bases. *Am J Orthod Dentofacial Orthop*. 1998;113:276–281.
17. Sernetz F, Binder F. Improvement of bond strength of orthodontic titanium brackets and tubes by laser structuring. *Proceedings of the 5th International Conference on Joining Ceramics, Glass and Metal; Jena, Germany. DVS Berichte Band*. 1997;184:82–85.
18. Bowen RL. Dental filling material comprising vinyl-silane treated fused silica and a binder consisting of the reaction product of bisphenol and glycidyl methacrylate. *US patent*. 1962. 3 066 122.
19. Zachrisson B, Büyükyılmaz T. Bonding in orthodontics. In: Graber TM, Vanarsdall R, Vig K, eds. *Orthodontics: Current Principles and Techniques*. 5th ed. St. Louis: Mosby; 2004:727–784.
20. Nicholson JW. *Composite resins. The Chemistry of Medical and Dental Materials*. Cambridge: Royal Society of Chemistry; 2001. 149–157.
21. Usumez A, Usumez S. Bonding and restorative composite materials in dentistry. In: Nicolais L, Borzacchiello A, Lee S, eds. *Wiley Encyclopedia of Composites*. 2nd ed. John Wiley & Sons; 2012:128–138.
22. Hotz P, McLean JW, Seed I, et al. The bonding of glass ionomer cements to metal and tooth substrates. *Br Dent J*. 1977;142:41–47.
23. Althoff O, Hartung M. Advances in light curing. *Am J Dent*. 2000;13: 77D–81D.
24. Rai AK. Evaluation of bracket failure rate in orthodontic patients bonded with and without primer. *Saudi J Dent Res*. 2015;6:48–53.
25. Nandhra SS. Is a primer needed for orthodontic bonding? A randomized controlled trial. D Clin Dent thesis, University of Leeds. *Biomed Res int*. 2013: 180137. 2012.
26. Sfondrini MF, Fraticelli D, Gandini P, et al. Shear bond strength of orthodontic brackets and disinclusion buttons: effect of water and saliva contamination. *Biomed Res Int*. 2013. http://dx.doi.org/10.1155/2013/180137. ID 180137, 6 pages.
27. Shukla C, Maurya R, Jain U, et al. Moisture insensitive primer: a myth or truth. *J Orthod Sci*. 2014;3:132–136.
28. Anand MK, Majumder K, Venkateswaran S, et al. Comparison of shear bond strength of orthodontic brackets bonded using two different hydrophilic primers: an in vitro study. *Indian J Dent Res*. 2014;25:191–196.
29. Fleming PS, Johal A, Pandis N. Self-etch primers and conventional acid-etch technique for orthodontic bonding: a systematic review and meta-analysis. *Am J Orthod Dentofacial Orthop*. 2012;142:83–94.
30. Cinader D. Chemical processes and performance comparisons of Transbond plus self-etching primer. *Orthod Perspect*. 2001;8:5–6.
31. Ireland AJ, Knight H, Sheriff M. An in vivo investigation into bond failure rates with a new self-etching primer system. *Am J Orthod Dentofacial Orthop*. 2003;124:323–326.
32. Chu CH, Ou KL, Dong de R, et al. Orthodontic bonding with self-etching primer and self-adhesive systems. *Eur J Orthod*. 2011;33:276–281.
33. Tanna N, Kao E, Gladwin M, et al. Effects of sealant and self-etching primer on enamel decalcification. Part I. An in-vitro study. *Am J Orthod Dentofacial Orthop*. 2009;135:199–205.
34. Ghiz MA, Ngan P, Kao E, et al. Effects of sealant and self-etching primer on enamel decalcification. Part II. An in-vivo study. *Am J Orthod Dentofacial Orthop*. 2009;135:206–213.
35. Visel D, Jäcker T, Jost-Brinkmann PG, et al. Demineralization adjacent to orthodontic brackets after application of conventional and self-etching primer systems. *J Orofac Orthop*. 2014;75:358–373.
36. Goldberg M. In vitro and in vivo studies on the toxicity of dental resin components: a review. *Clin Oral Investig*. 2008;12:1–8.
37. Üsümez S, Büyükyilmaz T, Karaman AI, et al. Degree of conversion of two lingual retainer adhesives cured with different light sources. *Eur J Orthod*. 2005;27:173–179.
38. Ozturk N, Üsümez A, Üsümez S, et al. Degree of conversion and surface hardness of resin cement cured with different curing units. *Quintessence Int*. 2005;36:771–777.
39. Tsitrou E, Kelogrigoris S, Koulaouzidou E, et al. Effect of extraction media and storage time on the elution of monomers from four contemporary resin composite materials. *Toxicol Int*. 2014;21:89–95.
40. Anand VS, Balasubramanian V. Effect of resin chemistry on depth of cure and cytotoxicity of dental resin composites. *Mater Sci Eng B*. 2014;181:33–38.
41. Agha MO, Üsümez S. *Evaluation of leaching of three different orthodontic bonding resins.* MS Dissertation submitted to Marmara University Health Sciences Institute; 2007.
42. Toy E, Yuksel S, Ozturk F, et al. Evaluation of the genotoxicity and cytotoxicity in the buccal epithelial cells of patients undergoing orthodontic treatment with three light-cured bonding composites by using micronucleus testing. *Korean J Orthod*. 2014;44:128–135.
43. Cörekçi B, Irgın C, Halıcıoğlu K, et al. Effects of plasma-emulating light-emitting diode (LED) versus conventional LED on cytotoxic effects and polymerization capacity of orthodontic composites. *Hum Exp Toxicol*. 2014;33:1000–1007.
44. Eliades T, Hiskia A, Eliades G, et al. Assessment of bisphenol-A release from orthodontic adhesives. *Am J Orthod Dentofacial Orthop*. 2007;131:72–75.
45. Eliades T, Gioni V, Kletsas D, et al. Oestrogenicity of orthodontic adhesive resins. *Eur J Orthod*. 2007;29:404–407.
46. Kloukos D, Pandis N, Eliades T. Bisphenol-A and residual monomer leaching from orthodontic adhesive resins and polycarbonate brackets: a systematic review. *Am J Orthod Dentofacial Orthop*. 2013;143:S104–S112. e1–e2.
47. Sudjalim TR, Woods MG, Manton DJ, et al. Prevention of demineralization around orthodontic brackets in vitro. *Am J Orthod Dentofacial Orthop*. 2007;131:705. e1– e9.
48. Bishara SE, Soliman M, Laffoon JF, et al. Shear bond strength of a new high fluoride release glass ionomer adhesive. *Angle Orthod*. 2008;78:125–128.
49. Fleming PS, Eliades T, Katsaros C, et al. Curing lights for orthodontic bonding: a systematic review and meta-analysis. *Am J Orthod Dentofacial Orthop*. 2013;143:S92–S103.
50. Mills RW, Jandt KD, Ashworth SH. Dental composite depth of cure with halogen and blue light emitting diode technology. *Br Dent J*. 1999;186:388–391.

51. Huh C, Lee KS, Kang EJ, et al. Improved light-output and electrical performance of InGaN-based light-emitting diode by microroughening of the *p*-GaN surface. *J Appl Phys*. 2013;93:9383–9385.
52. Parker ID, Cao Y, Yang CY. Lifetime and degradation effects in polymer light-emitting diodes. *J Appl Phys*. 1999;85:2441–2447.
53. Usümez S, Büyükyilmaz T, Karaman AI. Effect of light-emitting diode on bond strength of orthodontic brackets. *Angle Orthod*. 2004;74:259–263.
54. Polat O, Karaman AI, Durmus E. Effects of preoperative ibuprofen and naproxen sodium on orthodontic pain. *Angle Orthod*. 2005;75:791–796.
55. Aboush YE, Tareen A, Elderton RJ. Resin-to-enamel bonds: effect of cleaning the enamel surface with prophylaxis pastes containing fluoride or oil. *Br Dent J*. 1991;171:207–209.
56. Lew KK, Chew CL, Lee KW. A comparison of shear bond strengths between new and recycled ceramic brackets. *Eur J Orthod*. 1991;13:306–310.
57. Swartz ML. Why prophy prior to bracket bonding? *Clin Impressions*. 1994;3:11.
58. Ireland AJ, Sherriff M. The effect of pumicing on the in vivo use of a resin modified glass poly (alkenoate) cement and a conventional no-mix composite for bonding orthodontic brackets. *J Orthod*. 2002;29:217–220.
59. Burgess AM, Sherriff M, Ireland AJ. Self-etching primers: is prophylactic pumicing necessary? A randomized clinical trial. *Angle Orthod*. 2006;76:114–118.
60. Grover S, Sidhu MS, Prabhakar M. Evaluation of fluoride varnish and its comparison with pumice prophylaxis using self-etching primer in orthodontic bonding—an in vivo study. *Eur J Orthod*. 2012;34:198–201.
61. Ponduri S, Turnbull N, Birnie D, et al. Does atropine sulphate improve orthodontic bond survival? A randomized clinical trial. *Am J Orthod Dentofacial Orthop*. 2007;132:663–670.
62. Sayinsu K, Isik F, Sezen S, et al. Effect of blood and saliva contamination on bond strength of brackets bonded with a protective liquid polish and a light-cured adhesive. *Am J Orthod Dentofacial Orthop*. 2007;131:391–394.
63. Faltermeier A, Behr M, Rosentritt M, et al. An in vitro comparative assessment of different enamel contaminants during bracket bonding. *Eur J Orthod*. 2007;29:559–563.
64. Prasad M, Mohamed S, Nayak K, et al. Effect of moisture, saliva, and blood contamination on the shear bond strength of brackets bonded with a conventional bonding system and self-etched bonding system. *J Nat Sci Biol Med*. 2014;5:123–129.
65. Knösel M, Bojes M, Jung K, et al. Increased susceptibility for white spot lesions by surplus orthodontic etching exceeding bracket base area. *Am J Orthod Dentofacial Orthop*. 2012;141:574–582.
66. Brännström M, Malmgren O, Nordenvall KJ. Etching of young permanent teeth with an acid gel. *Am J Orthod*. 1982;82:379–383.
67. Brännström M, Nordenvall KJ, Malmgren O. The effect of various pretreatment methods of the enamel in bonding procedures. *Am J Orthod*. 1978;74:522–530.
68. Newman GV. A posttreatment survey of direct bonding of metal brackets. *Am J Orthod*. 1978;74:197–206.
69. Nordenvall KJ, Brännström M, Malmgren O. Etching of deciduous teeth and young and old permanent teeth. A comparison between 15 and 60 seconds of etching. *Am J Orthod*. 1980;78:99–108.
70. Wang WN, Lu TC. Bond strength with various etching times on young permanent teeth. *Am J Orthod Dentofacial Orthop*. 1991;100:72–79.
71. Uysal T, Basciftci FA, Usümez S, et al. Can previously bleached teeth be bonded safely? *Am J Orthod Dentofacial Orthop*. 2003;123:628–632.
72. do Rego MV, dos Santos RM, Leal LM, et al. Evaluation of the influence of dental bleaching with 35% hydrogen peroxide in orthodontic bracket shear bond strength. *Dental Press J Orthod*. 2013;18:95–100.
73. Endo T, Ishida R, Komatsuzaki A, et al. Effects of long-term repeated topical fluoride applications and adhesion promoter on shear bond strengths of orthodontic brackets. *Eur J Dent*. 2014;8:431–436.
74. Maijer R, Smith DC. Corrosion of orthodontic bracket bases. *Am J Orthod*. 1982;81:43–48.
75. Ceen RF, Gwinnett AJ. Indelible iatrogenic staining of enamel following debonding. *J Clin Orthod*. 1980;14:713–715.
76. Pus MD, Way DC. Enamel loss due to orthodontic bonding with filled and unfilled resins using various clean-up techniques. *Am J Orthod*. 1980;77:269–283.
77. Silverstone LM. The effect of fluoride in the remineralization of enamel caries and caries-like lesions in vitro. *J Public Health Dent*. 1982;42:42–53.
78. Thompson RE, Way DC. Enamel loss due to prophylaxis and multiple bonding/debonding of orthodontic attachments. *Am J Orthod*. 1981;79:282–295.
79. Burapavong V, Marshall GW, Apfel DA. Enamel surface characteristics on removal of bonded orthodontic brackets. *Am J Orthod*. 1978;74:176–187.
80. Gwinnett AJ. The bonding of sealants to enamel. *J Am Soc Prev Dent*. 1973;3:21–29.
81. Silverstone LM. Remineralization phenomena. *Caries Res*. 1977;11:59–84.
82. Diedrich P. Enamel alterations from bracket bonding and debonding: a study with the scanning electron microscopy. *Am J Orthod*. 1981;79:500–522.
83. Silverman E, Cohen M, Demke RS, et al. A new light-cured glass ionomer cement that bonds brackets to teeth without etching in the presence of saliva. *Am J Orthod Dentofacial Orthop*. 1995;108:231–236.
84. Zachrisson BU, Mjör IA. Remodeling of teeth by grinding. *Am J Orthod*. 1975;68:545–553.
85. Artun J, Bergland S. Clinical trials with crystal growth conditioning as an alternative to acid-etch pretreatment. *Am J Orthod*. 1984;85:333–340.
86. Bishara SE, Fehr DE. Ceramic brackets: something old, something new, a review. *Semin Orthod*. 1997;3:178–188.
87. Farquhar RB. Direct bonding comparing a polyacrylic acid and a phosphoric acid technique. *Am J Orthod*. 1986;90:187–194.
88. Read MJ, Ferguson JW, Watts DC. Direct bonding: crystal growth as an alternative to acid-etching? *Eur J Orthod*. 1986;8:118–122.
89. Bas-Kalkan A, Orhan M, Üsümez S. The effect of enamel etching with different acids on the bond strength of metallic brackets. *Turkish J Orthod*. 2007;20:35–42.
90. Olsen ME, Bishara SE, Damon P, et al. Evaluation of Scotchbond multipurpose and maleic acid as alternative methods of bonding orthodontic brackets. *Am J Orthod Dentofacial Orthop*. 1997;111:498–501.
91. Hermsen RJ, Vrijhoef MM. Loss of enamel due to etching with phosphoric or maleic acid. *Dent Mater*. 1993;9:332–336.
92. Baratieri LN, Monteiro Jr S. Influence of acid type (phosphoric or maleic) on the retention of pit and fissure sealant: an in vivo study. *Quintessence Int*. 1994;25:749–755.
93. Goes MF, Sinhoreti MA, Consani S, et al. Morphological effect of the type, concentration and etching time of acid solution on enamel and dentin surface. *Braz Dent J*. 1998;9:3–10.
94. Usümez S, Orhan M, Usümez A. Laser etching of enamel for direct bonding with an Er,Cr:YSGG hydrokinetic laser system. *Am J Orthod Dentofacial Orthop*. 2002;122:649–656.
95. von Fraunhofer JA, Allen DJ, Orbell GM. Laser etching of enamel for direct bonding. *Angle Orthod*. 1993;63:73–76.
96. Basaran G, Ozer T, Berk N, et al. Etching enamel for orthodontics with an erbium, chromium:yttrium-scandium-gallium-garnet laser system. *Angle Orthod*. 2007;7:117–123.
97. Berk N, Basaran G, Ozer T. Comparison of sandblasting, laser irradiation, and conventional acid etching for orthodontic bonding of molar tubes. *Eur J Orthod*. 2008;30:183–189.
98. Sağır S, Usumez A, Ademci E, et al. Effect of enamel laser irradiation at different pulse settings on shear bond strength of orthodontic brackets. *Angle Orthod*. 2013;83:973–980.
99. Topcuoglu T, Oksayan R, Ademci KE, et al. Effects of water flow rate on shear bond strength of orthodontic bracket bonded to enamel surface after Er:YAG laser ablation. *Photomed Laser Surg*. 2013;31:486–491.
100. Fowler BO, Kuroda S. Changes in heated and in laser irradiated human tooth enamel and their probable effects on solubility. *Calcif Tissue Int*. 1986;38:197–208.
101. Keller U, Hibst R. Ultrastructural changes of enamel and dentin following Er:YAG laser radiation on teeth. *Proc SPIE*. 1990;1200:408–415.
102. Fornaini C, Brulat N, Milia G, et al. The use of sub-ablative Er:YAG laser irradiation in prevention of dental caries during orthodontic treatment. *Laser Ther*. 2004;23:173–181.
103. Secilmis A, Usumez A, Usumez S, et al. Evaluation of mineral content of enamel prepared by erbium, chromium:yttrium-scandium-gallium-garnet laser. *Lasers Med Sci*. 2010;25:467–472.

104. Zachrisson BU, Brobakken BO. Clinical comparison of direct versus indirect bonding with different bracket types and adhesives. *Am J Orthod*. 1978;74:62–78.
105. Armstrong D, Shen G, Petocz P, et al. A comparison of accuracy in bracket positioning between two techniques—localizing the centre of the clinical crown and measuring the distance from the incisal edge. *Eur J Orthod*. 2007;29:430–436.
106. Erkan M, Pikdoken L, Usumez S. Gingival response to mandibular incisor intrusion. *Am J Orthod Dentofacial Orthop*. 2007;132. 143.e9– e13.
107. Jost-Brinkmann PG, Schiffer A, Miethke RR. The effect of adhesive layer thickness on bond strength. *J Clin Orthod*. 1992;26:718–720.
108. Armstrong D, Shen G, Petocz P, et al. Excess adhesive flash upon bracket placement. A typodont study comparing APC PLUS and Transbond XT. *Angle Orthod*. 2007;77:1101–1108.
109. Grünheid T, Sudit GN, Larson BE. Debonding and adhesive remnant cleanup: an in vitro comparison of bond quality, adhesive remnant cleanup, and orthodontic acceptance of a flash-free product. *Eur J Orthod*. 2015;37:497–502. 10. 1093/ejo/cju080. Epub 2014.
110. Zachrisson BU. Fluoride application procedures in orthodontic practice: current concepts. *Angle Orthod*. 1975;45:72–81.
111. Jain A, Ray S, Mitra R, et al. Light cure tip distance and shear bond strength: does it have any clinical significance? *J Ind Orthod Soc*. 2013;47:135–142.
112. Malkoç S, Uysal T, Usümez S, et al. In-vitro assessment of temperature rise in the pulp during orthodontic bonding. *Am J Orthod Dentofacial Orthop*. 2010;137:379–383.
113. Ramoglu SI, Karamehmetoglu H, Sari T, et al. Temperature rise caused in the pulp chamber under simulated intrapulpal microcirculation with different light-curing modes. *Angle Orthod*. 2015;85:381–385. http://dx.doi.org/10.2319/030814-164.1. Epub 2014 Oct 15.
114. Usümez S, Büyükyilmaz T, Karaman AI. Effects of fast halogen and plasma arc curing lights on the surface hardness of orthodontic adhesives for lingual retainers. *Am J Orthod Dentofacial Orthop*. 2003;123:641–648.
115. Price RB. Light curing guidelines for practitioners: a consensus statement from the 2014 symposium on light curing in dentistry, Dalhousie University, Halifax, Canada. *J Adhes Dent*. 2014;16:303–304.
116. Zachrisson BU. Orthodontic bonding to artificial tooth surfaces: clinical versus laboratory findings. *Am J Orthod Dentofacial Orthop*. 2000;117:592–594.
117. Zachrisson BU, Büyükyilmaz T. Recent advances in bonding to gold, amalgam, and porcelain. *J Clin Orthod*. 1993;27:661–675.
118. Zachrisson YO, Zachrisson BU, Büyükyilmaz T. Surface preparation for orthodontic bonding to porcelain. *Am J Orthod Dentofacial Orthop*. 1996;109:420–430.
119. Kocadereli I, Canay S, Akça K. Tensile bond strength of ceramic orthodontic brackets bonded to porcelain surfaces. *Am J Orthod Dentofacial Orthop*. 2001;119:617–620.
120. Grewal Bach GK, Torrealba Y, Lagravère M. Orthodontic bonding to porcelain: a systematic review. *Angle Orthod*. 2014;84:555–560.
121. Güler AU, Yilmaz F, Yenisey M, et al. Effect of acid etching time and a self-etching adhesive on the shear bond strength of composite resin to porcelain. *J Adhes Dent*. 2006;8:21–25.
122. al Edris A, al Jabr A, Cooley RL, et al. SEM evaluation of etch pattern by three etchants on three porcelains. *J Prosthet Dent*. 1990;64:734–739.
123. Simonsen RJ, Calamia JP. Tensile bond strength of etched porcelain. *J Dent Res*. 1983;61:279.
124. Kukiattrakoon B, Thammasitboon K. Optimal acidulated phosphate fluoride gel etching time for surface treatment of feldspathic porcelain: on shear bond strength to resin composite. *Eur J Dent*. 2012;6:63–69.
125. Chen JR, Oka K, Kawano T, et al. Carbon dioxide laser application enhances the effect of silane primer on the shear bond strength between porcelain and composite resin. *Dent Mater J*. 2010;29:731–737.
126. Yassaei S, Moradi F, Aghili H, et al. Shear bond strength of orthodontic brackets bonded to porcelain following etching with Er:YAG laser versus hydrofluoric acid. *Orthodontics (Chic.)*. 2013;14:e82–e87.
127. Karan S, Büyükyilmaz T, Toroğlu MS. Orthodontic bonding to several ceramic surfaces: are there acceptable alternatives to conventional methods? *Am J Orthod Dentofacial Orthop*. 2007;132. 144.e7–e14.
128. Schmage P, Nergiz I, Herrmann W, et al. Influence of various surface-conditioning methods on the bond strength of metal brackets to ceramic surfaces. *Am J Orthod Dentofacial Orthop*. 2003;123:540–546.
129. Saraç YŞ, Külünk T, Elekdağ-Türk S, et al. Effects of surface-conditioning methods on shear bond strength of brackets bonded to different all-ceramic materials. *Eur J Orthod*. 2011;33:667–672.
130. Zachrisson BU, Büyükyilmaz T, Zachrisson YO. Improving orthodontic bonding to silver amalgam. *Angle Orthod*. 1995;65:35–42.
131. Germec D, Cakan U, Ozdemir FI, et al. Shear bond strength of brackets bonded to amalgam with different intermediate resins and adhesives. *Eur J Orthod*. 2009;31:207–212.
132. Büyükyilmaz T, Zachrisson BU. Improving orthodontic bonding to silver amalgam. Part 2. Lathe-cut, admixed and spherical amalgams with different intermediate resins. *Angle Orthod*. 1998;68:337–344.
133. Büyükyilmaz T, Zachrisson YO, Zachrisson BU. Improving orthodontic bonding to gold alloy. *Am J Orthod Dentofacial Orthop*. 1995;108:510–518.
134. Shon WJ, Kim TW, Chung SH, et al. The effects of primer precuring on the shear bond strength between gold alloy surfaces and metal brackets. *Eur J Orthod*. 2012;34:72–76.
135. Klocke A, Shi J, Kahl-Nieke B, et al. Bond strength with custom base indirect bonding techniques. *Angle Orthod*. 2003;73:176–180.
136. Thomas RG. Indirect bonding: simplicity in action. *J Clin Orthod*. 1979;13:93–106.
137. Sheridan JJ. The Readers' Corner. 2. What percentage of your patients are being treated with Invisalign appliances? *J Clin Orthod* 200;38:544–545.
138. Hodge TM, Dhopatkar AA, Rock WP, et al. The Burton approach to indirect bonding. *J Orthod*. 2001;28:267–270.
139. Koo BC, Chung CH, Vanarsdall RL. Comparison of the accuracy of bracket placement between direct and indirect bonding techniques. *Am J Orthod Dentofacial Orthop*. 1999;116:346–351.
140. Kalange JT, Thomas RG. Indirect bonding: a comprehensive review of the literature. *Semin Orthod*. 2007;13:3–10.
141. Menini A, Cozzani M, Sfondrini MF, et al. A 15-month evaluation of bond failures of orthodontic brackets bonded with direct versus indirect bonding technique: a clinical trial. *Prog Orthod*. 2014;15:70. http://dx.doi.org/10.1186/s40510-014-0070-9.
142. Bozelli JV, Bigliazzi R, Barbosa HA, et al. Comparative study on direct and indirect bracket bonding techniques regarding time length and bracket detachment. *Dental Press J Orthod*. 2013;18:51–57.
143. Swetha M, Pai VS, Sanjay N, et al. Indirect versus direct bonding—a shear bond strength comparison: an in vitro study. *J Contemp Dent Pract*. 2011;12:232–238.
144. Klocke A, Shi J, Kahl-Nieke B, et al. In vitro investigation of indirect bonding with a hydrophilic primer. *Angle Orthod*. 2003;73:445–450.
145. Polat O, Karaman AI, Buyukyilmaz T. In vitro evaluation of shear bond strengths and in vivo analysis of bond survival of indirect-bonding resins. *Angle Orthod*. 2004;74:405–409.
146. Cozzani M, Menini A, Bertelli A. Etching masks for precise indirect bonding. *J Clin Orthod*. 2010;44:326–330.
147. Aksu M, Kocadereli I. Influence of two different bracket base cleaning procedures on shear bond strength reliability. *J Contemp Dent Pract*. 2013;14:250–254.
148. Sonis AL. Air abrasion of failed bonded metal brackets: a study of shear bond strength and surface characteristics as determined by scanning electron microscopy. *Am J Orthod Dentofacial Orthop*. 1996;110:96–98.
149. Montero MM, Vicente A, Alfonso-Hernández N, et al. Comparison of shear bond strength of brackets recycled using micro sandblasting and industrial methods. *Angle Orthod*. 2015;85:461–467. http://dx.doi.org/10.2319/032414-221.1.
150. Ahrari F, Fekrazad R, Kalhori KA, et al. Reconditioning of ceramic orthodontic brackets with an Er,Cr:YSGG laser. *Lasers Med Sci*. 2013;28:223–228.
151. Zachrisson BU. Bonding in orthodontics. In: Graber TM, Vanarsdall RG, eds. *Orthodontics: Current Principles and Techniques*. 3rd ed. St. Louis: Mosby; 2000:557–646.
152. Oilo G. Bond strength testing—what does it mean? *Int Dent J*. 1993;43:492–498.

153. Redd TB, Shivapuja PK. Debonding ceramic brackets: effects on enamel. *J Clin Orthod*. 1991;25:475–481.
154. Artun JA. Post-treatment evaluation of multibonded ceramic brackets in orthodontics. *Eur J Orthod*. 1997;19:219–228.
155. Deleted in proof.
156. Viazis AD, Cavanaugh G, Bevis RR. Bond strength of ceramic brackets under shear stress: an in vitro report. *Am J Orthod Dentofacial Orthop*. 1990;98:214–221.
157. Winchester LJ. Bond strengths of five different ceramic brackets: an in vitro study. *Eur J Orthod*. 1991;13:293–305.
158. Oztoprak MO, Nalbantgil D, Erdem AS, et al. Debonding of ceramic brackets by a new scanning laser method. *Am J Orthod Dentofacial Orthop*. 2010;138:195–200.
159. Mundethu AR, Gutknecht N, Franzen R. Rapid debonding of polycrystalline ceramic orthodontic brackets with an Er:YAG laser: an in vitro study. *Lasers Med Sci*. 2014;29:1551–1556.
160. Nalbantgil D, Tozlu M, Oztoprak MO. Pulpal thermal changes following Er-YAG laser debonding of ceramic brackets. *Scientific World Journal*. 2014;2014:912429.
161. Suliman SN, Trojan TM, Tantbirojn D, et al. Enamel loss following ceramic bracket debonding: a quantitative analysis in vitro. *Angle Orthod*. 2015;85:651–656. http://dx.doi.org/10.2319/032414-224.1.
162. Zachrisson BU, Skogan O, Höymyhr S. Enamel cracks in debonded, debanded, and orthodontically untreated teeth. *Am J Orthod*. 1980;77:307–319.
163. Lee YK, Lim YK. Three-dimensional quantification of adhesive remnants on teeth after debonding. *Am J Orthod Dentofacial Orthop*. 2008;134:556–562.
164. Ryf S, Flury S, Palaniappan S, et al. Enamel loss and adhesive remnants following bracket removal and various clean-up procedures in vitro. *Eur J Orthod*. 2012;34:25–32.
165. Zachrisson BU, Artun J. Enamel surface appearance after various debonding techniques. *Am J Orthod*. 1979;75:121–137.
166. Janiszewska-Olszowska J, Szatkiewicz T, Tomkowski R, et al. Effect of orthodontic debonding and adhesive removal on the enamel—current knowledge and future perspectives—a systematic review. *Med Sci Monit*. 2014;20:1991–2001.
167. Boncuk Y, Çehreli ZC, Polat-Özsoy Ö. Effects of different orthodontic adhesives and resin removal techniques on enamel color alteration. *Angle Orthod*. 2014;84:634–641.
168. Ferreira FG, Nouer DF, Silva NP, et al. Qualitative and quantitative evaluation of human dental enamel after bracket debonding: a noncontact three-dimensional optical profilometry analysis. *Clin Oral Investig*. 2014;18:1853–1864.
169. Day CJ, Price R, Sandy JR, et al. Inhalation of aerosols produced during the removal of fixed orthodontic appliances: a comparison of 4 enamel cleanup methods. *Am J Orthod Dentofacial Orthop*. 2008;133:11–17.
170. Toroglu MS, Bayramoglu O, Yarkin F, et al. Possibility of blood and hepatitis B contamination through aerosols generated during debonding procedures. *Angle Orthod*. 2003;73:571–578.
171. Toroğlu MS, Haytaç MC, Köksal F. Evaluation of aerosol contamination during debonding procedures. *Angle Orthod*. 2001;71:299–306.
172. Hadler-Olsen S, Sandvik K, El-Agroudi MA, et al. The incidence of caries and white spot lesions in orthodontically treated adolescents with a comprehensive caries prophylactic regimen—a prospective study. *Eur J Orthod*. 2012;34:633–639.
173. Karabekiroglu S, İleri Z, Kahraman FB, et al. The effects of fixed orthodontic treatment period on white spot lesion prevalence and DMFT index. *J Istanbul Univ Faculty Dent*. 2014;48:27–35.
174. Hamdan AM, Maxfield BJ, Tüfekçi E, et al. Preventing and treating white-spot lesions associated with orthodontic treatment: a survey of general dentists and orthodontists. *J Am Dent Assoc*. 2012;143:777–783.
175. Benson PE, Shah AA, Millett DT, et al. Fluorides, orthodontics and demineralization: a systematic review. *J Orthod*. 2005;32:102–114.
176. Brobakken BO, Zachrisson BU. Abrasive wear of bonding adhesives: studies during treatment and after bracket removal. *Am J Orthod*. 1981;79:134–147.
177. Büyükyilmaz T, Tangugsorn V, Ogaard B, et al. The effect of titanium tetrafluoride (TiF4) application around orthodontic brackets. *Am J Orthod Dentofacial Orthop*. 1994;105:293–296.
178. Pascotto RC, Navarro MF, Capelozza Filho L, et al. In vivo effect of a resin-modified glass ionomer cement on enamel demineralization around orthodontic brackets. *Am J Orthod Dentofacial Orthop*. 2004;125:36–41.
179. Caldeira ÉM Osório A, Oberosler EL, et al. Antimicrobial and fluoride release capacity of orthodontic bonding materials. *J Appl Oral Sci*. 2013;21:327–334.
180. Chung CK, Millett DT, Creanor SL, et al. Fluoride release and cariostatic ability of a compomer and a resin-modified glass ionomer cement used for orthodontic bonding. *J Dent*. 1998;26:533–538.
181. Frazier MC, Southard TE, Doster PM. Prevention of enamel demineralization during orthodontic treatment: an in vitro study using pit and fissure sealants. *Am J Orthod Dentofacial Orthop*. 1996;110:459–465.
182. Todd MA, Staley RN, Kanellis MJ, et al. Effect of a fluoride varnish on demineralization adjacent to orthodontic brackets. *Am J Orthod Dentofacial Orthop*. 1999;116:159–167.
183. Wiltshire WA. In vitro and in vivo fluoride release from orthodontic elastomeric ligature ties. *Am J Orthod Dentofacial Orthop*. 1999;115:288–292.
184. Gillgrass TJ, Creanor SL, Foye RH, et al. Varnish or polymeric coating for the prevention of demineralization? An ex vivo study. *J Orthod*. 2001;28:291–295.
185. Mattick CR, Mitchell L, Chadwick SM, et al. Fluoride-releasing elastomeric modules reduce decalcification: a randomized controlled trial. *J Orthod*. 2001;28:217–219.
186. Tüfekçi E, Pennella DR, Mitchell JC, et al. Efficacy of a fluoride-releasing orthodontic primer in reducing demineralization around brackets: an in-vivo study. *Am J Orthod Dentofacial Orthop*. 2014;146:207–214.
187. Alves PV, Alviano WS, Bolognese AM, et al. Treatment protocol to control *Streptococcus mutans* level in an orthodontic patient with high caries risk. *Am J Orthod Dentofacial Orthop*. 2008;133:91–94.
188. Derks A, Katsaros C, Frencken JE, et al. Caries-inhibiting effect of preventive measures during orthodontic treatment with fixed appliances. A systematic review. *Caries Res*. 2004;38:413–420.
189. Gelgör IE, Büyükyilmaz T. A practical approach to white spot lesion removal. *World J Orthod*. 2003;4:152–156.
190. Bergstrand F, Twetman S. A review on prevention and treatment of post-orthodontic white spot lesions—evidence-based methods and emerging technologies. *Open Dent J*. 2011;5:158–162.
191. Yetkiner E, Wegehaupt F, Wiegand A, et al. Colour improvement and stability of white spot lesions following infiltration, micro-abrasion, or fluoride treatments in vitro. *Eur J Orthod*. 2014;36:595–602.
192. Paris S, Meyer-Lueckel H, Cölfen H, et al. Penetration coefficients of commercially available and experimental composites intended to infiltrate enamel carious lesions. *Dent Mater*. 2007;23:742–748.
193. Paris S, Meyer-Lueckel H, Cölfen H, et al. Resin infiltration of artificial enamel caries lesions with experimental light curing resins. *Dent Mater J*. 2007;26:582–588.
194. Kielbassa AM, Muller J, Gernhardt CR. Closing the gap between oral hygiene and minimally invasive dentistry: a review on the resin infiltration technique of incipient (proximal) enamel lesions. *Quintessence Int*. 2009;40:663–681.
195. Knösel M, Eckstein A, Helms HJ. Durability of esthetic improvement following Icon resin infiltration of multibracket-induced white spot lesions compared with no therapy over 6 months: a single-center, split-mouth, randomized clinical trial. *Am J Orthod Dentofacial Orthop*. 2013;144:86–96.
196. Kim Y, Son HH, Yi K, et al. The color change in artificial white spot lesions measured using a spectroradiometer. *Clin Oral Investig*. 2013;17:139–146.
197. Paris S, Schwendicke F, Keltsch J, et al. Masking of white spot lesions by resin infiltration in vitro. *J Dent*. 2013;41(Suppl 5):e28–e34.
198. Soviero V, Paris S, Leal SC, et al. Ex vivo evaluation of caries infiltration after different application times in primary molars. *Caries Res*. 2013;47:110–116.
199. Knierim RW. Invisible lower cuspid to cuspid retainer. *Angle Orthod*. 1973;43:218–220.

200. Rubenstein BM. A direct bond maxillary retainer. *J Clin Orthod*. 1976;10:43.
201. Carter RN. Simplified direct-bonded retainer. *J Clin Orthod*. 1978;12:221.
202. Lubit EC. The bonded lingual retainer. *J Clin Orthod*. 1979;13:311–313.
203. Artun J, Spadafora AT, Shapiro PA. A 3-year follow-up study of various types of orthodontic canine-to-canine retainers. *Eur J Orthod*. 1997;19:501–509.
204. Zachrisson BU. Clinical experience with direct-bonded orthodontic retainers. *Am J Orthod*. 1977;71:440–448.
205. Bearn DR. Bonded orthodontic retainers: a review. *Am J Orthod Dentofacial Orthop*. 1995;108:207–213.
206. Uzel A, Buyukyilmaz T, Kayalioglu M, et al. Temperature rise during orthodontic bonding with various light-curing units—an in vitro study. *Angle Orthod*. 2006;76:330–334.
207. Pandis N, Fleming PS, Kloukos D, et al. Survival of bonded lingual retainers with chemical or photo polymerization over a 2-year period: a single-center, randomized controlled clinical trial. *Am J Orthod Dentofacial Orthop*. 2013;144:169–175.
208. Attar N, Tam LE, McComb D. Flow, strength, stiffness and radiopacity of flowable resin composites. *J Can Dent Assoc*. 2003;69:516–521.
209. Lee IB, Son HH, Um CM. Rheologic properties of flowable, conventional hybrid, and condensable composite resins. *Dent Mater*. 2003;19:298–307.
210. Arslan S, Demirbuga S, Ustun Y, et al. The effect of a new-generation flowable composite resin on microleakage in Class V composite restorations as an intermediate layer. *J Conserv Dent*. 2013;16:189–193.
211. Bonilla ED, Yashar M, Caputo AA. Fracture toughness nine flowable resin composites. *J Prosthet Dent*. 2003;89:261–267.
212. Labella R, Lambrechts P, Van Meerbeek B, et al. Polymerization shrinkage and elasticity of flowable composites and filled adhesives. *Dent Mater*. 1999;15:128–137.
213. Tavassoli Hojati S, Alaghemand H, Hamze F, et al. Antibacterial, physical and mechanical properties of flowable resin composites containing zinc oxide nanoparticles. *Dent Mater*. 2013;29:495–505.
214. Elaut J, Asscherickx K, Vande Vannet B, et al. Flowable composites for bonding lingual retainers. *J Clin Orthod*. 2002;36:597–598.
215. Geserick M, Wichelhaus A. A color-reactivated flowable composite for bonding lingual retainers. *J Clin Orthod*. 2004;38:165–166.
216. Geserick M, Ball J, Wichelhaus A. Bonding fiber-reinforced lingual retainers with color-reactivating flowable composite. *J Clin Orthod*. 2004;38:560–562.
217. Tabrizi S, Salemis E, Usumez S. Flowable composites for bonding orthodontic retainers. *Angle Orthod*. 2010;80:195–200.
218. Uysal T, Ulker M, Baysal A, et al. Different lingual retainer composites and the microleakage between enamel-composite and wire-composite interfaces. *Angle Orthod*. 2008;78:941–946.
219. Artun J. Caries and periodontal reactions associated with long-term use of different types of bonded lingual retainers. *Am J Orthod*. 1984;86:112–118.
220. Artun J, Urbye KS. The effect of orthodontic treatment on periodontal bone support in patients with advanced loss of marginal periodontium. *Am J Orthod Dentofacial Orthop*. 1988;93:143–148.
221. Al-Nimri K, Al Habashneh R, Obeidat M. Gingival health and relapse tendency: a prospective study of two types of lower fixed retainers. *Aust Orthod J*. 2009;25:142–146.
222. Artun J, Spadafora AT, Shapiro PA. A 3-year follow-up study of various types of orthodontic canine-to-canine retainers. *Eur J Orthod*. 1997;19:501–509.
223. Gaare D, Rølla G, Aryadi FJ, et al. Improvement of gingival health by toothbrushing in individuals with large amounts of calculus. *J Clin Periodontol*. 1990;17:38–41.
224. Booth FA, Edelman JM, Proffit WR. Twenty-year follow-up of patients with permanently bonded mandibular canine-to-canine retainers. *Am J Orthod Dentofacial Orthop*. 2008;133:70–76.
225. Pandis N, Vlahopoulos K, Madianos P, et al. Long-term periodontal status of patients with mandibular lingual fixed retention. *Eur J Orthod*. 2007;29:471–476.
226. Zachrisson BU. Third-generation mandibular bonded lingual 3-3 retainer. *J Clin Orthod*. 1995;29:39–48.
227. Zachrisson BU. Long-term experience with direct-bonded retainers: update and clinical advice. *J Clin Orthod*. 2007;41:728–737.
228. Axelsson S, Zachrisson BU. Clinical experience with direct-bonded labial retainers. *J Clin Orthod*. 1992;26:480–490.
229. Zachrisson BU. The acid etch technique in orthodontics. Clinical studies. In: Silverstone LM, Dogon L, eds. *Proceedings of an International Symposium on the Acid Etch Technique*. St. Paul: North Central Publishing Company; 1975:265–275.
230. Zachrisson BU. Tooth movements in the periodontally compromised patient. In: Lindhe J, Karring T, Lang NP, eds. *Clinical Periodontology and Implant Dentistry*. 5th ed. Oxford, UK: Blackwell Munksgaard; 2008:1241–1279.
231. Karaman AI, Polat O, Büyükyilmaz T. A practical method of fabricating a lingual retainer. *Am J Orthod Dentofacial Orthop*. 2003;124:327–330.
232. Bovali E, Kiliaridis S, Cornelis MA. Indirect vs direct bonding of mandibular fixed retainers in orthodontic patients: a single-center randomized controlled trial comparing placement time and failure over a 6-month period. *Am J Orthod Dentofacial Orthop*. 2014;146:701–708.
233. Dahl EH, Zachrisson BU. Long-term experience with direct-bonded lingual retainers. *J Clin Orthod*. 1991;25:619–630.
234. Zachrisson BU. Multistranded wire retainers. From start to success. *Am J Orthod Dentofacial Orthop*. 2015;148:724–727.
235. Salehi P, Zarif Najafi H, Roeinpeikar SM. Comparison of survival time between two types of orthodontic fixed retainer: a prospective randomized clinical trial. *Prog Orthod*. 2013;14:25.
236. Renkema AM, Renkema A, Bronkhorst E, et al. Long-term effectiveness of canine-to-canine bonded flexible spiral wire lingual retainers. *Am J Orthod Dentofacial Orthop*. 2011;139:614–621.
237. Katsaros C, Livas C, Renkema AM. Unexpected complications of bonded mandibular lingual retainers. *Am J Orthod Dentofacial Orthop*. 2007;132:838–841.

28

Management of Impactions

Adrian Becker and Stella Chaushu

OUTLINE

Teeth Normally Erupt! (Causes), 868
Is There an Impaction? (Diagnosis), 869
Which Teeth Are the Most Likely to Be Affected? (Prevalence), 870
Where Is the Tooth? (Positional Diagnosis), 870
Assessment of the Overall Malocclusion (Treatment Planning), 871
Resolving the Impaction, 873
Surgical Exposure, 873
Surgical Options, 873
Attachments, 875
Traction Mechanisms, Their Range and Directional Potential, 876
Failure—Patient-Dependent Factors, 876
Failure—Orthodontist-Dependent Factors, 876
Failure—Surgeon-Dependent Factors, 877

TEETH NORMALLY ERUPT! (CAUSES)

The most important and basic attribute of each tooth in the dentition is that it erupts into the oral environment. In the absence of eruption of a tooth in its due time, it must be assumed that something is wrong. Therefore the key to success in resolving noneruption or tooth impaction is to find the cause and eliminate it. Possible reasons may be listed under the following headings:

1. **Primary displacement of the tooth bud:** Displacement occasionally occurs with canines, but other teeth are not immune. The tooth develops in an ectopic location (Fig. 28-1); although it may have normal eruptive potential, its eruptive movements may not bring it in an appropriate direction, and thus the tooth may not emerge into the oral cavity. Aside from impaction, primary displacement is sometimes associated with tooth transposition. Treatment: The tooth is surgically exposed, followed by biomechanical redirection or extraction.
2. Space inadequacy: Inadequate space includes crowding and space loss in the dental arch, such as the drifting that occurs after the early extraction of deciduous teeth. Teeth possess the intrinsic potential for eruption but, under these conditions, are prevented from erupting. Treatment: Space is re-created by moving neighboring teeth or by remedial extraction of other teeth.
3. Local obstruction anomaly: Examples of obstruction-related anomalies are supernumerary teeth,[1] odontomes (Fig. 28-2), dentigerous cysts, and gingival fibrosis. Teeth possess the intrinsic potential for eruption but, under these conditions, are obstructed. Treatment: The causative entity is eliminated, with or without the provision of extrusive orthodontic mechanics.[2]
4. Trauma: Early trauma to the deciduous anterior dentition may sometimes be transmitted to the developing permanent teeth, particularly the central incisors. Trauma may result in damage to the unerupted tooth, usually by causing attenuation or arrest of the development of the root. The result can be a short or dilacerate root and failure of the tooth to erupt (Fig. 28-3, *A*, *B*). Treatment: If long-term prognosis is poor, the tooth is surgically exposed, followed by orthodontic redirection with the intention of bringing the tooth into the arch, with its accompanying alveolar bone to provide a healthy base for a subsequent implant. In an extraction case, consideration should be given to extracting this tooth rather than extracting a normal healthy premolar.

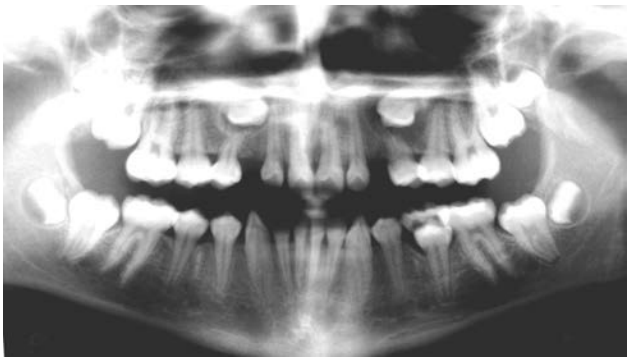


FIGURE 28-1 Maxillary bilateral impacted canines are transposed with the first premolars on this panoramic view. Shortening of the tooth length is due to the projection of the x-ray beams almost along their long axes. The apices of both canines are palatal and their crowns labial, in this case of primary displacement of the tooth buds.

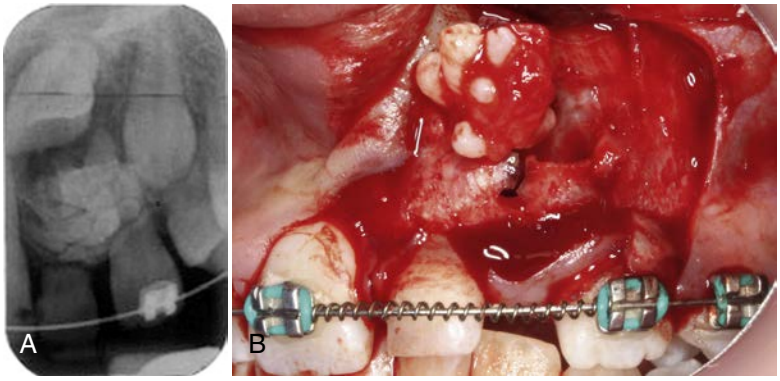


FIGURE 28-2 A, B, A composite odontoma is revealed in a periapical radiograph and at the time of surgical exposure before extraction of the odontoma.

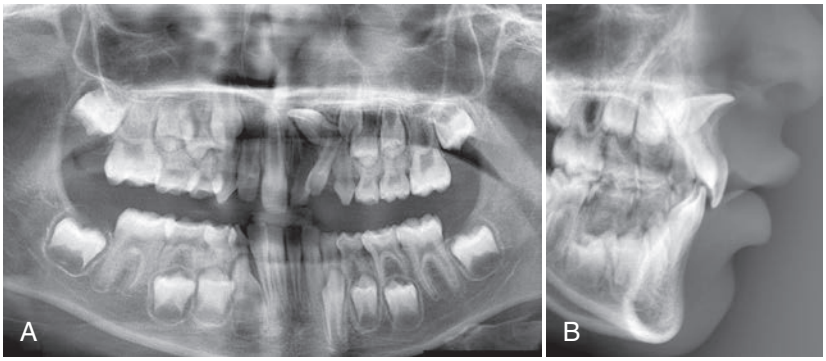


FIGURE 28-3 A, B, A severely dilacerated maxillary central incisor is revealed in the panoramic and lateral cephalometric views. The incisal edge is located within the anterior nasal spine.

5. Local pathologic cause of periodontic origin: For normal eruption, a tooth must have a periodontal ligament that is intact around the entire root surface. When a break of its integrity occurs, as in ankylosis, the tooth is in direct contact with the alveolar bone and will not erupt further. Invasive cervical root resorption (ICRR) (Fig. 28-4), on the other hand, is caused by infiltration of clastic cells through gaps in the cementum layer. There is loss of periodontal ligament (PDL) integrity and bony deposition in the depth of the ICRR lesion, adjacent to the resorption front.[3] Treatment: Therapeutic luxation followed by immediate traction may overcome an ankylosis. ICRR can be treated by surgically exposing the lesion and obliterating the resorption crater with filling material, provided it is not too extensive.
6. General conditions: With a primary failure of eruption (PFE),[4–6] cleidocranial dysplasia (CCD)[7] (Fig. 28-5) and hypoplastic amelogenesis imperfecta,[8] the teeth have little or no intrinsic potential for eruption. Treatment: Attempts to erupt the teeth in PFE and, in some cases with amelogenesis imperfecta, will usually fail, due to defective intrinsic factors that are not well understood. After the surgical elimination of over-retained deciduous teeth and multiple supernumerary teeth in CCD, the application of efficient extrusive mechanics will readily bring about a positive eruptive response on the part of the teeth.[9]

IS THERE AN IMPACTION? (DIAGNOSIS)

At the age of 7 years, one expects to see erupted maxillary central incisors in the mouth. Similarly, at the ages of 11 and 12 years, the maxillary canines should have appeared. These determinations are made using a correlation of the chronologic age with the eruption age of the patient. However, this method is not reliable because great variability exists in the ages at which teeth erupt, most often attributable to local factors. A more reliable assessment method is to use a key that is linked to dental development—the dental age of the patient.[10,11] Teeth normally erupt when two-thirds to three-quarters of their estimated final root length has developed. In many cases, several teeth may erupt before their time because of pathologic conditions and early extraction of their deciduous

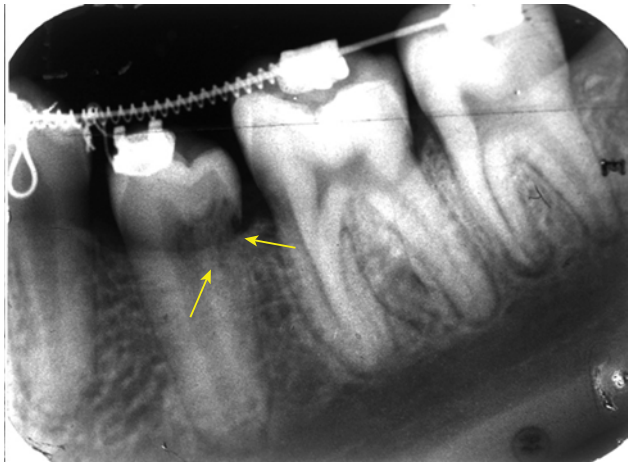


FIGURE 28-4 An invasive cervical root resorption (ICRR) lesion (*arrows*) is affecting the second premolar, which resisted active eruptive traction.

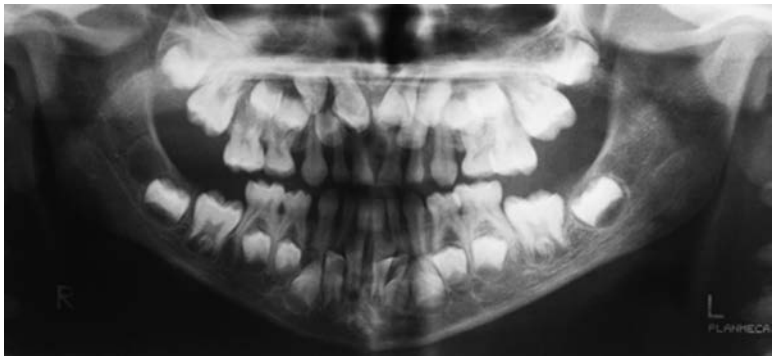


FIGURE 28-5 The dentition of an 11-year-old boy with cleidocranial dysplasia is depicted in this panoramic view. Note the very late dental development and the presence of several supernumerary teeth.

predecessors. Similarly, many instances of delayed eruption occur because of late shedding of the deciduous teeth. Diagnosis is made using radiographs, which may show unerupted teeth with advanced root development in excess of three-quarters of the final root length. For the most part and after the extraction of over-retained deciduous teeth, these teeth may still be expected to erupt normally, even if a little late. In contrast, an impacted tooth has been defined as "a permanent tooth whose root is developed in excess of this length and whose spontaneous eruption is not expected in a reasonable time"[11] (Fig. 28-6).

FIGURE 28-6 Horizontally impacted second permanent molars are revealed with almost completed root development.

WHICH TEETH ARE THE MOST LIKELY TO BE AFFECTED? (PREVALENCE)

Third molars are by far the most common teeth to be impacted, usually attributable to their location in relation to the length of the dental arch. Because of this tooth size–arch length discrepancy, conservative alignment of these teeth is not frequently advised, since no arch length into which they may be uprighted exists. On the other hand, when second premolars are absent, consideration of treatment aimed at mesially drawing first and second molars to provide space for the resolution of an otherwise impacted third molar often may be justified (Fig. 28-7, *A–C*).

Although less common, the maxillary canine is a far more important tooth to treat than its 1% to 2% impaction prevalence in most populations would suggest.[12–15] This is attributable to its position at the front of the mouth, its relative importance vis-à-vis the occlusion, and the patient's appearance.

Impacted maxillary central incisors[1,16–18] are the most disfiguring among all impacted teeth; fortunately, the frequency of their occurrence in the general population is very low. Prevalence figures from population studies are often inflated by the fact that affected individuals are far more likely to seek treatment and thus be included in an investigative sample of patients in dental hospitals, which unjustly purports to be a random sample.

WHERE IS THE TOOTH? (POSITIONAL DIAGNOSIS)

If the tooth has more than three-quarters of its expected root length and its eruption is not expected in a reasonable period, then the tooth needs assistance in resolving the impaction and, from the previous definition, the initiation of treatment is already late.

Before orthodontic treatment of the impacted tooth can be planned, accurately diagnosing its three-dimensional location in the jaw and its relation to the adjacent teeth and other anatomic structures is essential. The gathering of important relevant positional information is best achieved in four distinct diagnostic stages in the present context.

a. *Clinical examination.* When the patient attends a first visit, he or she generally comes without any diagnostic records. The intraoral examination should be conducted as with any orthodontic case, with an emphasis on searching for clues to the existence and location of unerupted teeth, based on certain features seen in the crown form and orientation of other teeth and other anatomic clues.[19] Thus the presence of a distally or labial flared lateral incisor, or a palpable bulge in the tissues overlying the palate, may indicate the location of an aberrant canine. Mobility or lack of mobility of deciduous molars at a certain age or early shedding of a deciduous canine may be very significant in this context.

b. *Plain-film radiography.* Perhaps the most popular film used by orthodontists in the earlier stages of diagnosis is the panoramic radiograph. It provides a good overall depiction of the dentition, much of the surrounding anatomy, and an occasional pathologic finding (Fig. 28-8). It offers a good two-dimensional indication of the presence and orientation of unerupted teeth but, by itself, lacks the facility to provide adequate buccolingual information[20,21] without the addition of supplemental films from other radiographic views, such as periapical, occlusal, lateral skull, and posteroanterior skull.

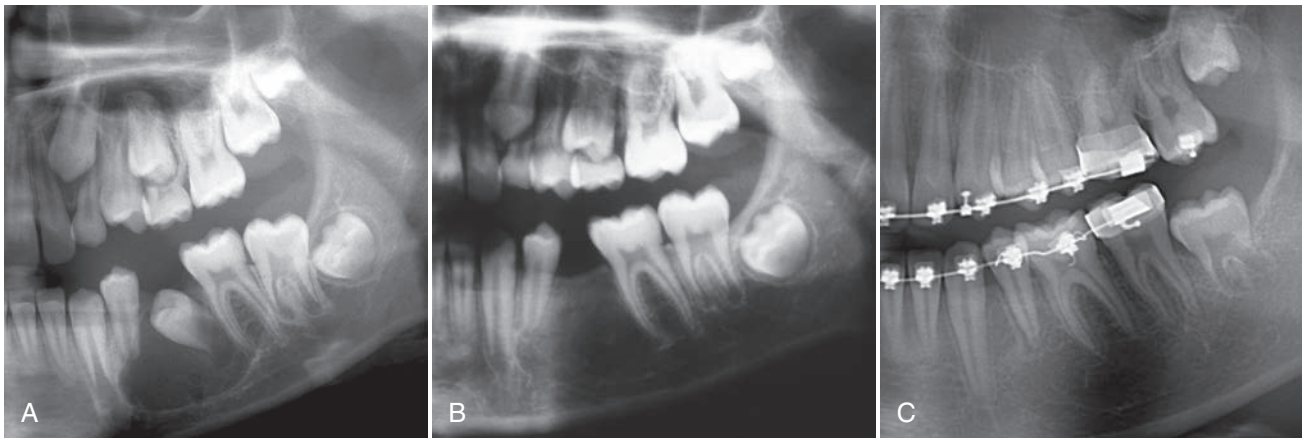


FIGURE 28-7 A, The right side of a panoramic radiograph shows a radicular cyst, which was enucleated with the second premolar tooth. B, The same view is shown after full healing and alveolar bone regeneration had occurred. C, The same view is shown during the final stages of orthodontic treatment, which is directed at mesially moving first and second permanent molars into the space to free the former impacted third molar to erupt normally.

FIGURE 28-8 A panoramic view prescribed to locate the position of the impacted maxillary canine reveals the chance finding of a radicular cyst associated with a nonvital second deciduous molar and an unerupted second premolar.

c. *Three-dimensional imaging.* Cone-beam computed tomography (CBCT) was introduced to dentistry, in general, and to orthodontics, in particular, at the turn of the 21st century; with CBCT came a quantum leap in the ability of the practitioner to see and easily understand the location and orientation of the tooth within the maxillofacial complex, because of its three-dimensional imaging.[22–24] When the reconstruction of the raw data has been properly prepared, it is difficult to understand how an orthodontist or a surgeon can possibly arrive at a mistaken positional diagnosis (Fig. 28-9, *A–E*).

Unerupted teeth cannot be evaluated for color, surface mottling, and enamel blemishes until they are surgically exposed. Assessment of tooth shape and size, crown, and root anomaly before exposure can be revealed only with the use of radiographs and/or CBCT imaging.[25] With the advent of these advanced radiographic imaging aids, the practitioner is responsible for rigorously scouring these diagnostic records for information regarding the existence of pathologic lesions, such as root resorption in teeth adjacent to an impacted tooth, which will undermine the long-term prognosis. Diagnosing conditions such as ankylosis and invasive cervical root resorption[26] (see Fig. 28-4) is significantly more difficult, because these conditions are insidious, asymptomatic, and not easily identified until they are quite extensive. Their presence will cause total resistance to any attempt to move the affected tooth.

By carefully appraising the wealth of information that will have been gathered, the orthodontist will be in a position to assess whether helping this tooth become an integral part of the envisaged orthodontic occlusal scheme for the specific patient is viable and whether the therapeutic cost of achieving this is justified.

ASSESSMENT OF THE OVERALL MALOCCLUSION (TREATMENT PLANNING)

In regard to the impacted tooth, four possible lines of treatment are available:

1. **Prevention and interception.** The literature is replete with studies investigating the efficacy of various measures intended to prevent, intercept, and/or mitigate the severity of impacted maxillary canine teeth, although little has been written in relation to other teeth. The measures have included the prophylactic extraction of the deciduous canines,[27–29] the deciduous canines and deciduous first molars,[30,31] space maintenance with a transpalatal arch,[32,33] molar distalization,[27,34] and maxillary expansion[32–34]; all of these measures have been successful to a limited degree, provided the diagnosis has been made early.
2. **Substitution.** Whether it is because of its surgical inaccessibility, its extreme displacement, its unacceptable shape or size, the presence of a pathologic finding, an unacceptable management problem in the child, or another legitimate reason for not attempting resolution, the simplest line of treatment is extraction. In this instance, substitution of the tooth by moving an adjacent tooth into its place, prosthodontic substitution with the use of an implant, or autotransplantation of another tooth will be considered. This treatment option is outside the scope of this chapter(see Chapter 20).
3. **Remedial extraction as part of an overall treatment plan.** If the overall treatment plan calls for the extraction of teeth because of crowding of the dentition or for another reason, then

consideration needs to be given to whether the impacted tooth should be one of the teeth of choice, together with other teeth in the remaining three quadrants of the arch. Again, the decision depends on factors such as the ability to disguise another tooth to simulate the missing tooth, particularly if a canine or incisor is the tooth of concern, and whether the appearance and longevity of the overall outcome would thus be improved. This treatment option is beyond the scope of this chapter.

4. **Resolution.** Resolving the impaction of the affected tooth and bringing it into its place in the dental arch—the orthodontic-surgical modality—is the most desirable result in the majority of cases that are seen. Resolving the impaction is predicated on the condition that the outcome conforms to the rigorous standards of health, function, and appearance of the tooth itself and of the dentition as a whole.

The remainder of the discussion in this chapter will be devoted to "resolution"— the orthodontic-surgical modality and how it may best be honed to produce the finest clinical outcomes in accordance with these standards.

With the decision to include the impacted tooth as an integral unit in the dentition, the overall malocclusion must be assessed in the manner described in earlier chapters of this text, regarding the orthodontic evaluation of every other case. In the fully erupted dentition, the initial task of the active orthodontic appliance therapy is to align and level the teeth in a single stage.

In the context of this chapter, leveling and aligning all erupted teeth will be completed and space will be prepared for the later alignment of the impacted tooth. A heavy passive archwire is then placed in the brackets of the aligned teeth with the express purposes of holding this alignment and of amalgamating the teeth into a compound anchor unit. The biomechanic appliance therapy for the resolution of the displacement of an impacted tooth will derive anchorage from this solid base.[35] Supplemental anchorage, in the form of intermaxillary elastics, soldered palatal arches, extraoral headgears, and temporary anchorage devices (TADs), should be considered in the initial treatment planning, if and when needed.

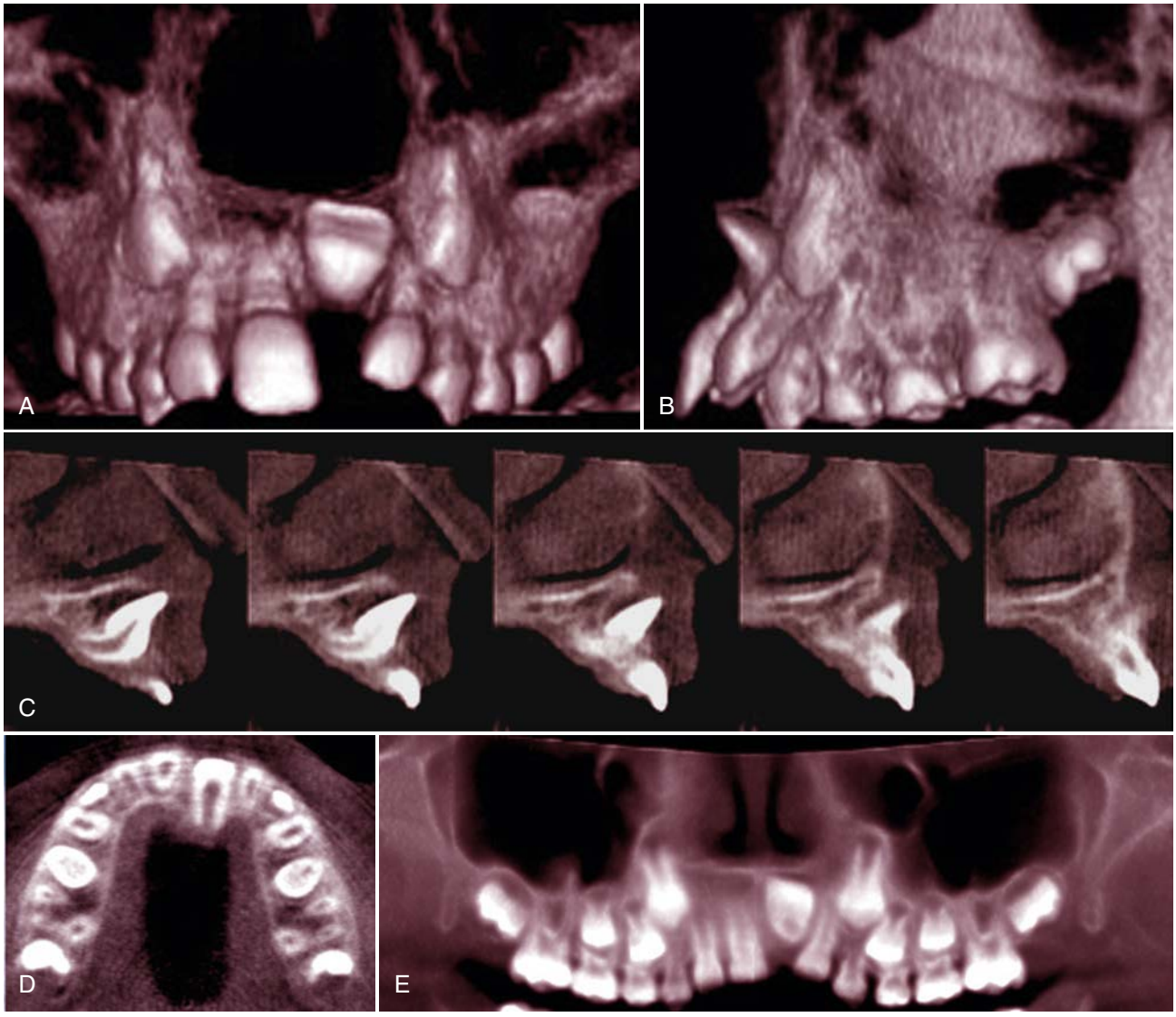


FIGURE 28-9 A–E, Cone-beam computed tomography (CBCT) images show classic dilaceration of the maxillary left central incisor on anterior and lateral three-dimensional views, cross-sectional cuts, an axial cut, and a panoramic view.

Occasional important exceptions to the treatment protocol are outlined here, specifically relating to the timing of the surgical exposure. Thus when the impacted canine is associated with resorption of the root of an adjacent (usually incisor) tooth, this resorption may be very aggressive and, in the absence of timely interceptive measures, can result in loss of the affected tooth. In these cases, the first step should be distancing the canine from the incisor, an intervention that has been proved to arrest the resorptive process.[36]

RESOLVING THE IMPACTION

In the absence of a pathologic condition, having diagnosed the location and orientation of the tooth in the three planes of space and having related its proximity to the locations of the adjacent teeth and other structures, the orthodontist should be able to plan an orthodontic strategy that will provide a path through potential obstructions and bring about the eruption of the tooth in the dental arch.

Many impacted teeth may be moved from their ectopic developmental position directly into their designated place in the line of the arch, in a simple tipping movement, without interference from the roots of adjacent teeth. For others, traction may need to be initiated in one direction to avoid an obstacle, and, once clear, the force may be redirected to bring the teeth into their place. Perhaps the most common example of when an indirect approach is needed is in relation to many palatally impacted maxillary canines. A typical scenario has the root apex of the canine high and in the line of the arch, with its apex-to-crown long axis deflected in a downward, forward, and palatally inclined vector. The crown is impacted on the palatal side, between the roots of the central and lateral incisors. The incisors may be in good positions, but the root of the lateral incisor, with its crown-to-apex orientation, lies in the canine's direct path to its intended location in the dental arch.

The panoramic radiographic presentation of this scenario is two-dimensional; because of this, defining the buccolingual orientation of the lateral incisor is impossible. The root of this incisor gives the misleading impression of being upright and parallel to that of the central incisors in the buccolingual plane, which will often encourage the orthodontist to plan a simple swing of the canine crown in an attempt to tip the tooth directly toward the labial archwire, not recognizing the obstacle in its path. Movement of the canine will be arrested by the clash with the lateral incisor root and the attempt will fail.[37-41] To avoid failure from this source, the canine should be vertically drawn downward toward the tongue, thereby erupting into the palatal area, inferior to the incisor root. From there, it may be tipped in a beeline to the archwire, in the normal manner.[37,38]

SURGICAL EXPOSURE

Surgical intervention in association with the presence of impacted teeth is needed for two primary purposes:

1. To eliminate a physical obstacle that restricts the normal eruption path of the tooth
   a. Resistant overlying soft tissue
   b. Supernumerary tooth
   c. Odontoma
2. To expose the tooth to provide access for the orthodontist to exercise control over its eruption
   a. To maintain the patency of the exposure and to supervise the tooth's natural autonomous eruption before an attachment is bonded to it for mechanical alignment[40]
   b. To permit attachment bonding and extrusive traction[37,38]
   c. To re-expose a tooth that has become reburied in the healing tissues

SURGICAL OPTIONS

Two basic approaches are used to expose impacted teeth. In the *open eruption exposure technique,* bone and soft tissues are removed around the crown, and it remains open to the oral environment at the end of the procedure (Fig. 28-10, *A*, *B*). The cut tissues are prevented from rehealing over the tooth by a broad elimination of the tissues down to the cement-enamel junction (CEJ) with or without the placement of a surgical pack,[39,41] which is left in place for 2 to 3 weeks or longer to permit healing to occur around it, while preventing the tissues from recovering the tooth. An attachment may be placed on the tooth and traction applied at the time of surgery or at a subsequent visit to the orthodontist after healing has occurred and the pack is removed (see Fig. 28-10, *B*).

Exposure must be immediately made in the mucosa overlying the unerupted tooth, and traction will bring the tooth into the oral environment through the tissues at this point. If the

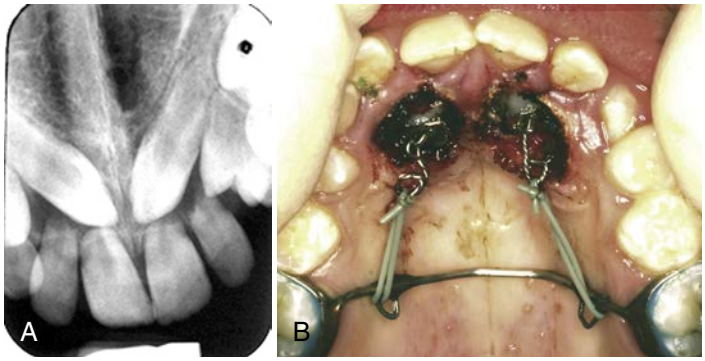


FIGURE 28-10 A, A periapical view reveals two impacted maxillary canines close to the midline that have caused the virtual disappearance of the roots of the four incisors. B, The same patient as shown in A is depicted after individual bilateral open surgical procedures with attachments are bonded to the palatal surfaces of the two canines and traction is immediately applied.

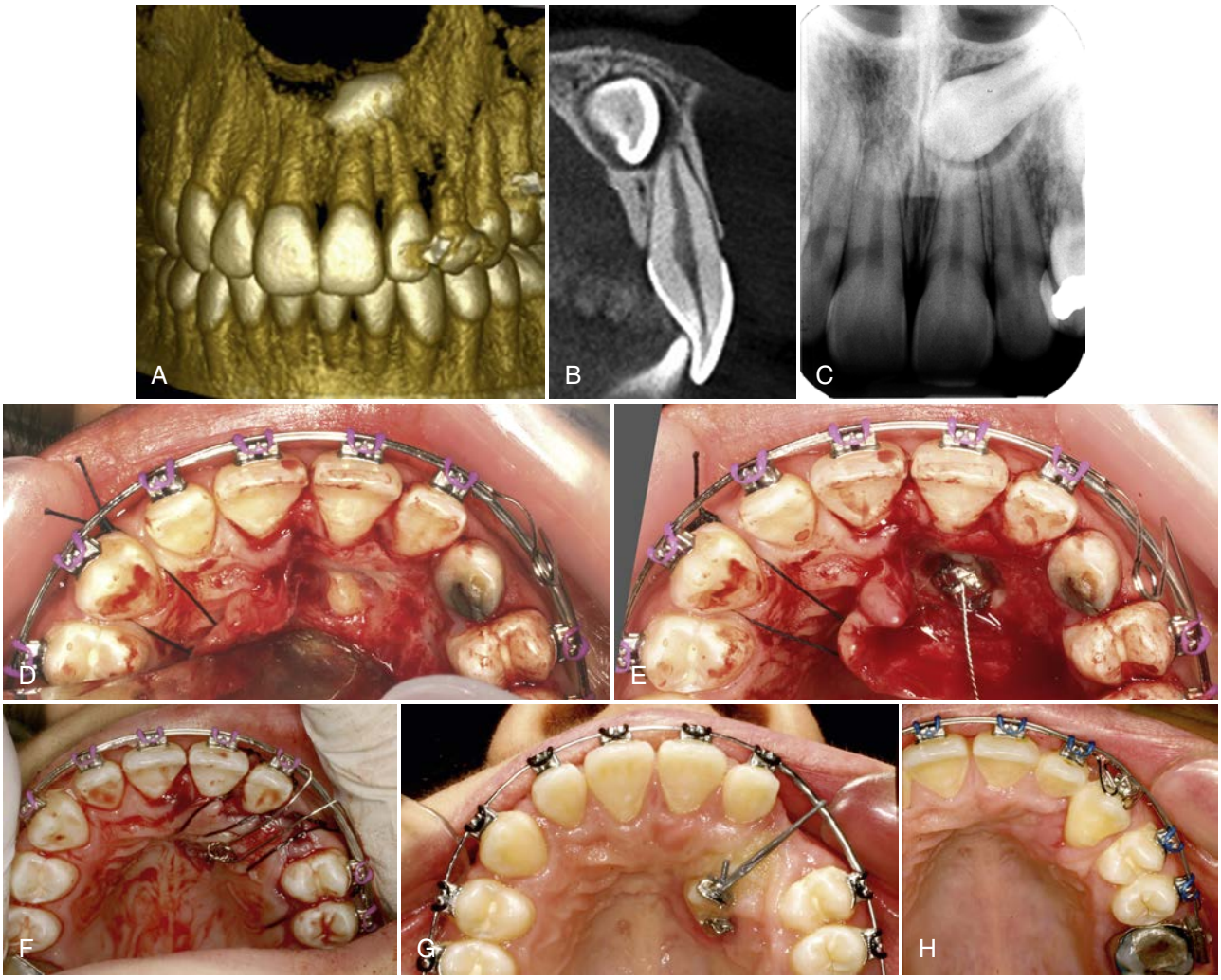


FIGURE 28-11 **A–C,** Diagnostic three-dimensional, cross-sectional, and periapical views of a very difficult palatal canine. **D–F,** With a very high canine, a closed surgical exposure has many advantages. Note the minimal exposure, attachment bonding, and fully replaced flap, with an auxiliary ballista-type spring already providing traction before the patient leaves the surgical unit. **G, H,** The canine is first erupted away from the incisor roots and into the midpalate before laterally moving into its place in the arch.

overlying tissues are comprised of nonkeratinized oral mucosa, as seen in impacted teeth that are exposed on the labial side, then the finally erupted tooth will be invested with oral mucosa, which is friable and easily damaged.

To avoid this undesirable sequel, a modification of the open exposure technique is recommended,[42] during which the tooth is exposed using a partial thickness apically repositioned flap of keratinized attached gingiva from the crest of the ridge or from the gingival crevice of the simultaneously extracted deciduous predecessor. In this way, the repositioned flap will come down together with the erupting tooth to provide it with a labial attachment of keratinized epithelium.[42]

In the *closed eruption exposure technique,* a wide surgical flap including attached gingiva is reflected, and the thin bone overlying the tooth is peeled away to reveal the follicle beneath. The follicle is minimally opened to permit a small area of the crown to be exposed. A more radical excision of bony or soft tissues is not needed, and surgery in the area of the CEJ is both superfluous and potentially damaging. The bonding area should be as small as possible and governed by the ability of the surgeon to maintain hemostasis while the orthodontist bonds the attachment with a twisted steel ligature or gold chain leading to the exterior.[43] The entire surgical flap is then resutured to its former place, leaving the steel ligature or gold chain as the means by which orthodontic traction may be applied to the impacted tooth[44,45] (Fig. 28-11, *A–H,* and 28-12, *A–E*), with the intention of erupting it through the attached gingiva, thereby simulating normal eruption.

Most studies have shown the superiority of the closed procedure with regard to the periodontal outcomes, especially for labially positioned canines and incisors,[44–53] although recent investigations do not support this view.[46,54] An open surgical procedure increases the risk for causing the loss of attached gingiva and long unaesthetic clinical crowns. Moreover, a closed procedure is also related to a shorter recovery time with less postoperative pain, especially for palatally displaced canines.[55–57]

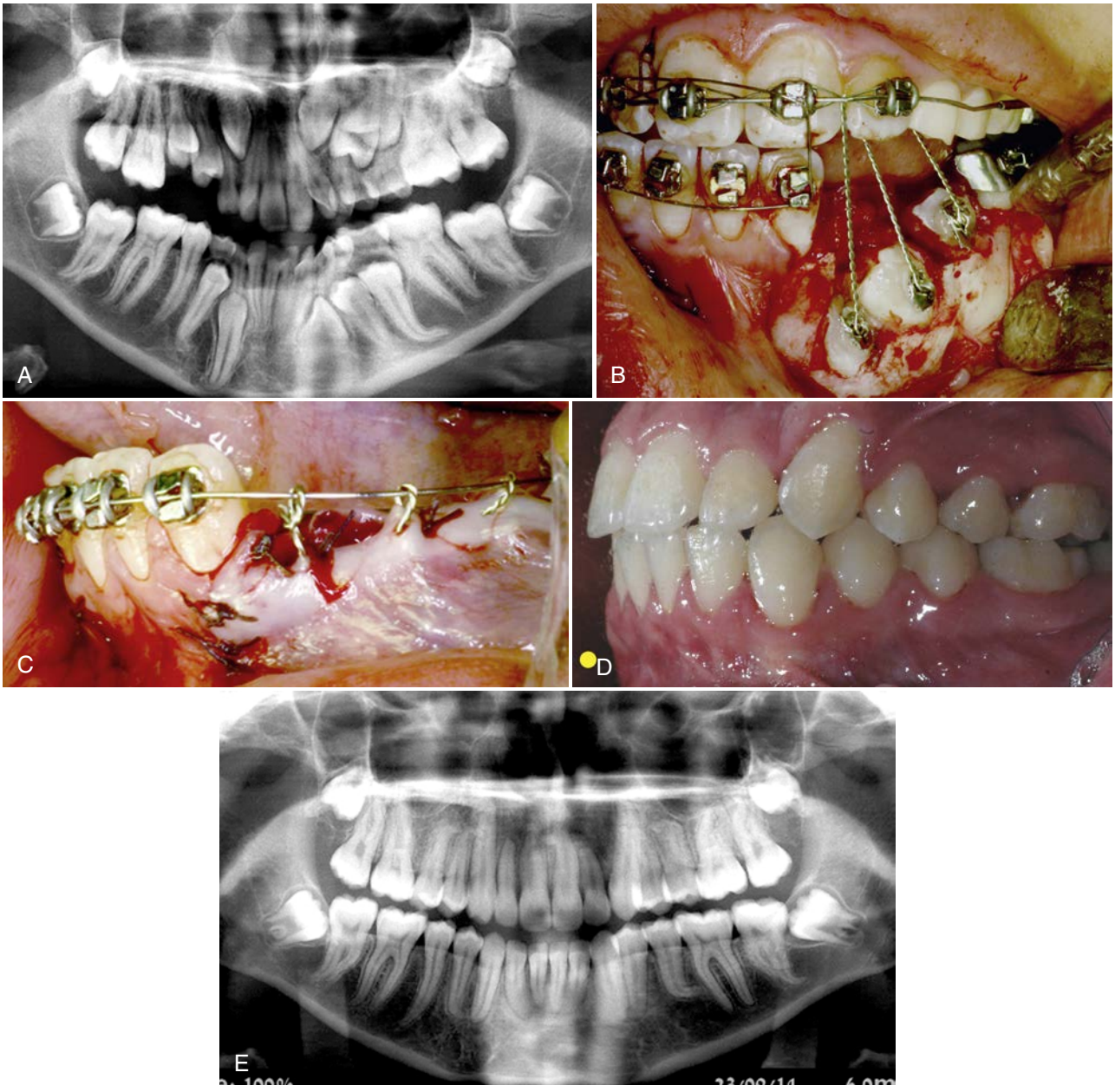


FIGURE 28-12 A–E, A young adult is suffering from nonsyndromic multiple impacted teeth. All four canine and premolar areas were treated with closed eruption exposures in a single surgical session under general anesthesia. Only the left mandibular quadrant is shown here.

ATTACHMENTS

By placing a sophisticated prescription bracket on a tooth, the operator acquires the capability to move a tooth in all directions, right and left, up and down, intruding and extruding, and it can also perform tipping, rotation, and torquing movements. However, these attributes presuppose its placement at a predetermined height on the midbuccal aspect of the crown of the tooth—the sole location for which the exquisite engineering specifications of the prescription bracket were designed. When placed elsewhere, it offers no advantage over a simple eyelet (see Fig. 28-10, *B*) and has many drawbacks.[11] At surgery, achieving the ideal placement of an attachment is not often possible because access to the midbuccal site is restricted by the adjacent incisor root or because considerably more bone and soft tissue would need to be removed to get there, as opposed to exposing the most superficially located bonding site.

The physical dimensions, shape, and protrusion of the angular corners of a precision orthodontic bracket will traumatize the gingival tissues through which it passes from its initial subgingival location, even following an open exposure procedure, during which a degree of rehealing of the tissues over the tooth and the consequent soft tissue impingement always occur.

Even assuming the presence of ideal conditions, to efficiently do more than extrude and tip a tooth that is not engaged in an archwire is not possible (see Fig. 28-11, *E–G*). Extrusion and tipping movements can be more readily achieved with a simple eyelet. This attachment is of more modest and rounded dimensions, more easily placeable in tight circumstances, much

kinder to the tissues, and easier to ligate. For all these reasons, an eyelet should be used during or shortly after surgical exposure (see Fig. 28-12) and substituted only by a regular bracket when the tooth reaches the archwire.[46–48]

Ideally, the attachment is bonded in the surgical unit when the exposure has been performed. In preparation for its bonding, a soft stainless steel ligature of 0.014-inch gauge is threaded though the eyelet and tightly twisted to be fashioned into a hook or loop for traction. A pre-made chain/attachment may also be used.

TRACTION MECHANISMS, THEIR RANGE AND DIRECTIONAL POTENTIAL

Direct traction to the intended location of the tooth in the dental arch is the simplest and most frequently used mechanism and is most suitable when the tooth is displaced but has a direct and unobstructed path to that site. Elastic thread is directly tied between the attachment on the affected tooth and the heavy archwire that will have been placed at the end of the alignment and leveling stage. In this way, the tooth is moved in the desired direction and all the other teeth that are ligated into the archwire act as a composite anchor, while the rigidity of the archwire resists bowing adjacent to the ligated area. A common alternative in these cases is to use a nickel-titanium archwire, fully tied in to the brackets of all the teeth including the impacted tooth. However, this should only be used when the impacted tooth is very mildly displaced. Because of the flexibility of the wire, the brunt of the reactive force is borne by the two teeth that are adjacent to the impacted tooth, and these will move toward the impaction. Thus in the case of a palatal canine, these adjacent teeth would quickly move into a crossbite relation with their antagonists, and a flattening of the arch form would result.

Displaced canines that are more mesially or more palatally displaced are also considerably higher than the occlusal plane and thus must be brought occlusally before or at the same time as they are moved to their places; otherwise, they will become buried in the vertical alveolar process.

In the scenario discussed earlier, in which the root of a lateral incisor blocks the direct path for a palatal canine, evasive action must be taken. This is best performed by first drawing the tooth down from its initial location and erupting it in the palatal area to circumvent the incisor root. From there, a direct and unimpeded path to the archwire will be acquired.

To achieve this, a ballista-type auxiliary spring may be used, which has two versions. In addition to the heavy stabilizing arch, a second full arch of 0.016-inch steel wire incorporating a vertical loop with a terminal helix is tied into all the brackets piggyback style[38] (see Fig. 28-11, *F*). Alternatively, a unilateral sectional rectangular cross-section wire may be used, which is slotted into an auxiliary molar tube. The sectional wire mesially extends with a 90-degree vertically downward bend that is adjacent to the prepared space in the arch.[58] A small helix is incorporated into the end of this wire. In both cases, the auxiliary wire is tied into its place, with the vertical portion passively hanging down (see Fig. 28-11, *D*, *E*). This step is best performed while the surgeon is waiting for the local anesthetic injections to take effect and before the surgical procedure has begun.

After bonding of the eyelet attachment in a closed eruption procedure, the surgical flap is fully replaced and resutured with the twisted 0.014-inch steel ligature puncturing it at a point immediately overlying the recovered tooth. The vertical loop of the auxiliary ballista-type auxiliary spring is palatally turned upward and ensnared into the steel ligature that will be bent into a hook around it. The vertical loop is drawn up under finger pressure to lie against the palatal mucosa (see Fig. 28-11, *F*). The round wire loop or mesial portion of the rectangular auxiliary thus applies a measurable extrusive force on the tooth with a long range of action. In an open procedure, the attachment may be bonded several days or weeks later, but preferably, bonding is done at the time of surgery by following the same protocol.

FAILURE—PATIENT-DEPENDENT FACTORS

Intrinsic patient factors can contribute to the failure in the resolution of an impacted tooth. These include an abnormal morphologic structure of the crown of a tooth or an inappropriate configuration of its roots. Abnormalities of the tooth itself, such as ankylosis or ICRR,[26] will cause the tooth to resist all attempts at its movement. With advancing age, the dental follicle surrounding the crown of an impacted tooth may break down, bringing about direct contact with the alveolar bone. This, in the long term, may lead to a replacement resorption of the crown enamel and no response to orthodontic traction (Fig. 28-13).

Resorption of the root of an adjacent tooth has been found to occur in two-thirds of the patients with impacted canines,[23] and female patients with normally sized lateral incisors are more susceptible.[59] Although resorption of the root is very minor in its extent in most cases, it is progressive and may threaten the longevity of the affected tooth (see Fig. 28-10, *A*). As a general rule, distancing the canine from the immediate area will bring about a cessation of the resorption.[36] In these cases, the sequence of treatment should be changed to moving the canine away from the resorbing area in question, even before space has been made.

Needless to add, patient compliance in relation to the attendance for treatment, oral hygiene, and a preparedness to cooperate in the placement of prescribed supplementary aids, such as headgears and rubber bands, are important factors, and the absence of patient compliance will undermine the chances of success.

FAILURE—ORTHODONTIST-DEPENDENT FACTORS

Regardless of a potentially favorable location of the impacted tooth, insufficient space in the arch will cause the treatment to flounder; an appreciation of the fact that the space created must extend all the way up to the apical area of the neighboring teeth

FIGURE 28-13 Periapical view of the anterior maxilla of a 65-year-old female patient shows the indistinct presence of two impacted canines. Most of the follicles have disappeared, and the margins are blurry, attributable to replacement resorption of the enamel and dentin of the crown.

is essential. Occasionally, the palatal root of a premolar may be inadequately uprighted after space opening or it may possess a mesial curvature, causing it to obstruct the path of eruption of a palatal canine (Fig. 28-14).

It has been reported elsewhere that "Diagnosis of the location of the tooth and its immediate relationship with the roots of the adjacent teeth is generally treated with cavalier and often negligent simplicity, even though modern technology has provided the tools to achieve this with great accuracy in all 3 dimensions."[35] With an inaccurate positional diagnosis, it follows that traction will be applied in the wrong direction. It follows, too, that without the benefits of CBCT imaging and its proven superior diagnostic capabilities, the diagnosis of root resorption of adjacent teeth will be frequently missed.

Inefficient traction mechanisms can lead to failure, such as frequently tying elastic thread over a short distance. Elasticity of stretched elastic thread fatigues very rapidly, the knot does not hold well, and the aberrant tooth fails to move. The rebound effect of a deflected overlay nickel-titanium wire depends, for its efficiency, on being able to slide freely in adjacent brackets, which is not often easy to guarantee. Simple custom-made deimpaction springs need to be designed to deliver low-force values over a broad range of movement. These springs can be made to fit the particular circumstance at hand, and this surely is the *forte* of the experienced orthodontist.

In the more extreme cases of ectopy and in situations where significant root movement is needed, a lack of appreciation of the considerable anchorage requirements of the case and the need to exploit all available means of enhancing them will inevitably lead to inefficient mechanotherapy and unnecessarily longer treatment, if not abject failure.[35]

FAILURE—SURGEON-DEPENDENT FACTORS

For the surgeon, the orientation of the long axis of an impacted tooth is immaterial. The surgeon needs only to know where the crown of the tooth lies, and this positional information can usually be provided by plain periapical and panoramic views alone. Nevertheless, the CBCT records should be made available to the surgeon to prevent a *rummaging* exposure, in which insufficient positional information is available and the surgical field is opened up too widely with excessive amounts of bone removed in the quest for the buried tooth. This excessive removal may cause injury to the impacted tooth itself, to an adjacent tooth, and to the soft tissues. Mistaken positional diagnosis also has serious consequences in the medicolegal context. In the more extreme cases, law reports are replete with cases in which exposure of a labially impacted tooth has been attempted from the palatal side. Other frequent mistakes include radical elimination of tissue beyond the CEJ, exposing the root surface and damaging the cementum layer, which potentially raises the likelihood that ankylosis or invasive root resorption will occur.

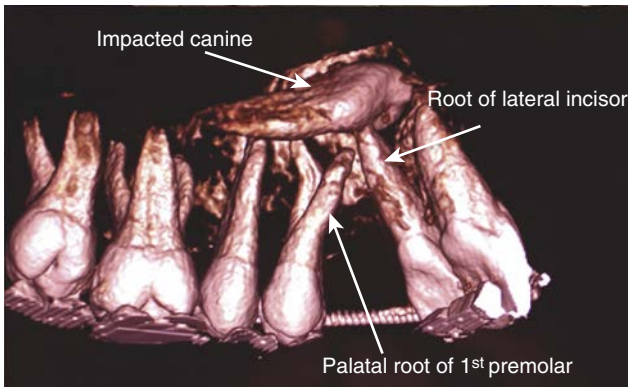


FIGURE 28-14 The three-dimensional cone-beam computed tomography (CBCT) lingual view reveals the left side of the maxilla in a patient with a horizontally impacted canine high above the roots of the adjacent teeth. Although space has been created at the occlusal level, note the mesial curvature of the palatal root of the premolar. The distance between the premolar and lateral incisor roots does not permit the passage of the canine.

In many cases and knowing that surgery will be necessary, the patient is incorrectly referred first to the surgeon, who is tempted to expose the impacted tooth without further discussion. Surgery without prior strategic orthodontic planning in these cases might occasionally be helpful; for the most part, however, it will have been a futile exercise if directional traction is needed in the resolution of the impaction and eventual alignment of the tooth.

Many orthodontists are happy for the surgeon to bond an attachment to the tooth at the time of surgery, but it should be remembered that, for the surgeon, attachment bonding is not a routine procedure; the chances of failure—and therefore a repeated surgical procedure—are higher. The surgeon is not familiar with the alternative directions of traction and will not know where to place the attachment or where to trail the gold chain or steel ligature from the attachment to the exterior. Many surgical procedures are sadly repeated, during which the attachment is already in place but simply redirecting these connectors is necessary.

The interrelations between the orthodontist and surgeon regarding the treatment of impacted teeth are particularly prone to mistakes and misunderstandings in surgical decisions. Each of these could sound the death knell to a treatment plan that may have appeared *a priori* to have been straightforward, with a potentially good prognosis.

Accordingly, the conclusion to be drawn from this is that the orthodontist has a major interest in being present at the surgeon's side during the exposure procedure. In addition to bonding the attachment while the surgeon maintains ideal hemostasis and dry conditions for acid-etch bonding, the orthodontist often needs a specific type of exposure—open or closed, a specific direction of traction, and the enormous benefit of immediate application of the traction mechanism. Short of the orthodontist attending the surgery, a mutual understanding of the orthodontic requirements and excellent communication are needed before and after the surgical procedures are completed. The surgeon may see the patient once more on a follow-up visit to remove sutures. However, the orthodontist will regularly see the patient over a long period to align the affected tooth as an integral part of the overall orthodontic treatment and in what is generally a demanding biomechanical venture. The fact that the most critical part of the procedure is well accomplished will simplify and streamline the treatment and offer an optimal prognosis.

REFERENCES

1. Tay F, Pang A, Yuen S. Unerupted maxillary anterior supernumerary teeth: report of 204 cases. *ASDC J Dent Child*. 1984;51(4):289–294.
2. Schmidt AD, Kokich VG. Periodontal response to early uncovering, autonomous eruption, and orthodontic alignment of palatally impacted maxillary canines. *Am J Orthod Dentofacial Orthop*. 2007;131(4):449–455.

3. Brosjo M, Anderssen K, Berg JO, Lindskog S. An experimental model for cervical resorption in monkeys. *Endod Dent Traumatol*. 1990;6(3):118–120.
4. Proffit WR, Vig KW. Primary failure of eruption: a possible cause of posterior open-bite. *Am J Orthod*. 1981;80(2):173–190.
5. Frazier-Bowers SA, Koehler KE, Ackerman JL, Proffit WR. Primary failure of eruption: further characterization of a rare eruption disorder. *Am J Orthod Dentofacial Orthop*. 2007;131(5): 578; e1–e11.
6. Frazier-Bowers SA, Puranik CP, Mahaney MC. The etiology of eruption disorders—further evidence of a "genetic paradigm." *Semin Orthod*. 2010;16(3):180–185.
7. Becker A, Lustmann J, Shteyer A. Cleidocranial dysplasia: Part 1—general principles of the orthodontic and surgical treatment modality. *Am J Orthod Dentofacial Orthop*. 1997;111(1):28–33.
8. Seow WK. Dental development in amelogenesis imperfecta: a controlled study. *Pediatr Dent*. 1995;17(1):26–30.
9. Becker A, Shteyer A, Bimstein E, Lustmann J. Cleidocranial dysplasia: Part 2—treatment protocol for the orthodontic and surgical modality. *Am J Orthod Dentofacial Orthop*. 1997;111(2):173–183.
10. Demirjian A, Goldstein H, Tanner JM. A new system of dental age assessment. *Hum Biol*. 1973;45(2):211–227.
11. Becker A. *The Orthodontic Treatment of Impacted Teeth*. 3rd ed. Oxford: Wiley-Blackwell; 2012.
12. Dachi SF, Howell FV. A survey of 3, 874 routine full-month radiographs. II. A study of impacted teeth. *Oral Surg Oral Med Oral Pathol*. 1961;14:1165–1169.
13. Brin I, Becker A, Shalhav M. Position of the maxillary permanent canine in relation to anomalous or missing lateral incisors: a population study. *Eur J Orthod*. 1986;8(1):12–16.
14. Thilander B, Jakobsson SO. Local factors in impaction of maxillary canines. *Acta Odontol Scand*. 1968;26(2):145–168.
15. Sacerdoti R, Baccetti T. Dentoskeletal features associated with unilateral or bilateral palatal displacement of maxillary canines. *Angle Orthod*. 2004;74(6):725–732.
16. Zilberman Y, Malron M, Shteyer A. Assessment of 100 children in Jerusalem with supernumerary teeth in the premaxillary region. *ASDC J Dent Child*. 1992;59(1):44–47.
17. Brook AH. Dental anomalies of number, form and size: their prevalence in British schoolchildren. *J Int Assoc Dent Child*. 1974;5(2):37–53.
18. Di Biase DD. The effects of variations in tooth morphology and position on eruption. *Dent Pract Dent Rec*. 1971;22(3):95–108.
19. Baccetti T. A controlled study of associated dental anomalies. *Angle Orthod*. 1998;68(3):267–274.
20. Chaushu S, Chaushu G, Becker A. The use of panoramic radiographs to localize displaced maxillary canines. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 1999;88(4):511–516.
21. Chaushu S, Chaushu G, Becker A. Reliability of a method for the localization of displaced maxillary canines using a single panoramic radiograph. *Clin Orthod Res*. 1999;2(4):194–199.
22. Chaushu S, Chaushu G, Becker A. The role of digital volume tomography in the imaging of impacted teeth. *World J Orthod*. 2004;5(2):120–132.
23. Walker L, Enciso R, Mah J. Three-dimensional localization of maxillary canines with cone-beam computed tomography. *Am J Orthod Dentofacial Orthop*. 2005;128(4):418–423.
24. Becker A, Chaushu S, Casap-Caspi N. Cone-beam computed tomography and the orthosurgical management of impacted teeth. *J Am Dent Assoc*. 2010;141(Suppl 3):14S–18S.
25. Bodner L, Bar-Ziv J, Becker A. Image accuracy of plain film radiography and computerized tomography in assessing morphological abnormality of impacted teeth. *Am J Orthod Dentofacial Orthop*. 2001;120(6):623–628.
26. Becker A, Abramovitz I, Chaushu S. Failure of treatment of impacted canines associated with invasive cervical root resorption. *Angle Orthod*. 2013;83(5):870–876.
27. Baccetti T, Leonardi M, Armi P. A randomized clinical study of two interceptive approaches to palatally displaced canines. *Eur J Orthod*. 2008;30(4):381–385.
28. Bazargani F, Magnuson A, Lennartsson B. Effect of interceptive extraction of deciduous canine on palatally displaced maxillary canine: a prospective randomized controlled study. *Angle Orthod*. 2014;84(1):3–10.
29. Naoumova J, Kurol J, Kjellberg H. Extraction of the deciduous canine as an interceptive treatment in children with palatal displaced canines—part I: shall we extract the deciduous canine or not? *Eur J Orthod*. 2015;37(2):209–218.
30. Alessandri Bonetti G, Incerti Parenti S, Zanarini M, Marini I. Double vs single primary teeth extraction approach as prevention of permanent maxillary canines ectopic eruption. *Pediatr Dent*. 2010;32(5):407–412.
31. Alessandri Bonetti G, Zanarini M, Incerti Parenti S, et al. Preventive treatment of ectopically erupting maxillary permanent canines by extraction of deciduous canines and first molars: a randomized clinical trial. *Am J Orthod Dentofacial Orthop*. 2011;139(3):316–323.
32. Baccetti T, Sigler LM, McNamara Jr JA. An RCT on treatment of palatally displaced canines with RME and/or a transpalatal arch. *Eur J Orthod*. 2011;33(6):601–607.
33. Sigler LM, Baccetti T, McNamara Jr JA. Effect of rapid maxillary expansion and transpalatal arch treatment associated with deciduous canine extraction on the eruption of palatally displaced canines: a 2-center prospective study. *Am J Orthod Dentofacial Orthop*. 2011;139(3):e235–e244.
34. Armi P, Cozza P, Baccetti T. Effect of RME and headgear treatment on the eruption of palatally displaced canines: a randomized clinical study. *Angle Orthod*. 2011;81(3):370–374.
35. Becker A, Chaushu G, Chaushu S. Analysis of failure in the treatment of impacted maxillary canines. *Am J Orthod Dentofacial Orthop*. 2010;137(6):743–754.
36. Becker A, Chaushu S. Long-term follow-up of severely resorbed maxillary incisors after resolution of an etiologically associated impacted canine. *Am J Orthod Dentofacial Orthop*. 2005;127(6):650–654; quiz 754.
37. Becker A, Zilberman Y. The palatally impacted canine: a new approach to treatment. *Am J Orthod*. 1978;74(4):422–429.
38. Kornhauser S, Abed Y, Harari D, Becker A. The resolution of palatally impacted canines using palatal-occlusal force from a buccal auxiliary. *Am J Orthod Dentofacial Orthop*. 1996;110(5):528–534.
39. Kokich VG, Mathews DP. Surgical and orthodontic management of impacted teeth. *Dent Clin North Am*. 1993;37(2):181–204.
40. Kokich VG. Preorthodontic uncovering and autonomous eruption of palatally impacted maxillary canines. *Semin Orthod*. 2010;16:205–211.
41. Mathews DP, Kokich VG. Palatally impacted canines: the case for preorthodontic uncovering and autonomous eruption. *Am J Orthod Dentofacial Orthop*. 2013;143(4):450–458.
42. Vanarsdall RL, Corn H. Soft-tissue management of labially positioned unerupted teeth. *Am J Orthod*. 1977;72(1):53–64.
43. Becker A, Shpack N, Shteyer A. Attachment bonding to impacted teeth at the time of surgical exposure. *Eur J Orthod*. 1996;18(5):457–463.
44. Becker A, Chaushu S. Palatally impacted canines: the case for closed surgical exposure and immediate orthodontic traction. *Am J Orthod Dentofacial Orthop*. 2013;143(4):451–459.
45. Chaushu S, Dykstein N, Ben-Bassat Y, Becker A. Periodontal status of impacted maxillary incisors uncovered by 2 different surgical techniques. *J Oral Maxillofac Surg*. 2009;67(1):120–124.
46. Becker A, Kohavi D, Zilberman Y. Periodontal status following the alignment of palatally impacted canine teeth. *Am J Orthod*. 1983;84(4): 332–336.
47. Kohavi D, Zilberman Y, Becker A. Periodontal status following the alignment of buccally ectopic maxillary canine teeth. *Am J Orthod*. 1984;85(1):78–82.
48. Kohavi D, Becker A, Zilberman Y. Surgical exposure, orthodontic movement, and final tooth position as factors in periodontal breakdown of treated palatally impacted canines. *Am J Orthod*. 1984;85(1):72–77.
49. Crescini A, Clauser C, Giorgetti R, et al. Tunnel traction of infraosseous impacted maxillary canines. A three-year periodontal follow-up. *Am J Orthod Dentofacial Orthop*. 1994;105(1):61–72.
50. Pearson MH, Robinson SN, Reed R, et al. Management of palatally impacted canines: the findings of a collaborative study. *Eur J Orthod*. 1997;19(5):511–515.
51. Burden DJ, Mullally BH, Robinson SN. Palatally ectopic canines: closed eruption versus open eruption. *Am J Orthod Dentofacial Orthop*. 1999;115(6):640–644.

52. Becker A, Brin I, Ben-Bassat Y, et al. Closed-eruption surgical technique for impacted maxillary incisors: a postorthodontic periodontal evaluation. *Am J Orthod Dentofacial Orthop*. 2002;122(1):9–14.
53. Chaushu S, Brin I, Ben-Bassat Y, et al. Periodontal status following surgical-orthodontic alignment of impacted central incisors with an open-eruption technique. *Eur J Orthod*. 2003;25(6):579–584.
54. Parkin N, Benson PE, Thind B, Shah A. Open versus closed surgical exposure of canine teeth that are displaced in the roof of the mouth. *Cochrane Database Syst Rev*. 2008;(4):CD006966.
55. Chaushu G, Becker A, Zeltser R, et al. Patients' perceptions of recovery after exposure of impacted teeth with a closed-eruption technique. *Am J Orthod Dentofacial Orthop*. 2004;125(6):690–696.
56. Chaushu S, Becker A, Zeltser R, et al. Patients' perceptions of recovery after surgical exposure of impacted maxillary teeth treated with an open-eruption surgical-orthodontic technique. *Eur J Orthod*. 2004;26(6):591–596.
57. Chaushu S, Becker A, Zeltser R, et al. Patients, perception of recovery after exposure of impacted teeth: a comparison of closed- versus open-eruption techniques. *J Oral Maxillofac Surg*. 2005;63(3):323–329.
58. Jacoby H. The "ballista spring" system for impacted teeth. *Am J Orthod*. 1979;75(2):143–151.
59. Chaushu S, Kaczor-Urbanowicz K, Zadurska M, Becker A. Predisposing factors for severe incisor root resorption associated with impacted maxillary canines. *Am J Orthod Dentofacial Orthop*. 2015;147(1):52–60.

29

Management of Dental Luxation and Avulsion Injuries in the Permanent Dentition

Patrick Turley

OUTLINE

Tissue Response to Trauma, 880
Root Resorption, 880
Pulpal Response, 880
Pulp Necrosis, 880
Pulp Canal Obliteration, 881
Alveolar Fracture, 881
Management of Trauma and Immediate Sequelae, 881
Concussion and Subluxation, 881
Lateral Displacement, 881
Extrusive Luxation, 881
Intrusive Luxation, 883
Avulsion, 883
Treatment of Avulsed Permanent Teeth with Closed Apex, 883
Treatment of Avulsed Permanent Teeth with Open Apex, 883
Prevention, 884

Trauma to the dentoalveolar region is a common occurrence in children, and estimates suggest that 25% of all children will suffer dental trauma during their developmental years, with more than 60% of these reported to be luxation-type injuries.[1–3] Because of this high frequency, dentists and dental specialists who treat children should be familiar with the principles of managing dental trauma. The management of traumatic injuries has not traditionally been an area of practice for the orthodontist; consequently, he or she is often not well equipped to handle the injury. The frequency of orthodontic appointments, however, results in the orthodontist developing a close relationship with the patient and family, and since the injury often damages the orthodontic appliance, the orthodontist is often contacted first. The purpose of this chapter is to familiarize the orthodontist with the management of dental luxation and avulsion injuries, which by their nature (malposition) and treatment requirements (orthodontic splinting or alignment) can fall within the purview of the practicing orthodontist.

TISSUE RESPONSE TO TRAUMA

The proper management of luxation and avulsion injuries requires an understanding of the immediate and short-term response to the involved tissues: the periodontal ligament (PDL), pulp, and alveolar bone.

ROOT RESORPTION

Trauma to the tooth often results in damage to the PDL. Trauma to the PDL, not necessarily the pulp, usually results in teeth unable to survive a traumatic injury. The amount of damage depends on the severity and type of injury and may include tearing, severing, or compression of the ligament. Root resorption is the common response to PDL injury; generally, the three types are surface, inflammatory, and replacement resorption.[4] *Surface resorption* is the least invasive, although it may involve both cementum and dentin; it is self-limiting and shows spontaneous repair. Surface resorption radiographically appears as small excavations on the root surface adjacent to a PDL space of normal width. *Inflammatory resorption* results from infected necrotic pulp tissue, which then affects the traumatized PDL.[5] This type can quickly destroy root structure, and without endodontic treatment, the loss of the tooth usually occurs within 2 to 10 months. Characterized by bowl-shaped excavations in the root surface with an adjacent radiolucency in the bone, inflammatory resorption can be radiographically seen as early as 3 to 4 weeks after an avulsion injury. *Replacement resorption* usually follows severe injuries in which the PDL has been removed or severely damaged.[6] Ankylosis accompanies replacement resorption, and the slow process of continuous replacement of root structure by bone results in the eventual loss of the tooth over a period of years. Radiographically, the root surface is highly irregular, with the normal lamina dura absent and the tooth structure obviously diminishing. Progressive root resorption (inflammatory and replacement resorption) occurs most frequently after intrusion or avulsion injuries.[1] Removing the pulp tissue and placing calcium hydroxide in the canal is the treatment for inflammatory resorption.[1,7,8] The same treatment regimen is also used for replacement resorption, although this type of resorption is more difficult to control.[7,9]

PULPAL RESPONSE

Pulp Necrosis

Pulp response and survival depend on the type (severity) of the injury and the stage of root end development.[10] The diameter of the apical foramen is the most important factor in pulp survival.[11,12] Teeth with open apices show a higher survival rate, especially with the less severe injuries such as concussion, luxation, lateral displacement, and extrusion. Pulps of intruded and avulsed teeth with closed root apices rarely survive. To prevent

the necrotic pulp from potentiating the process of progressive root resorption, endodontic treatment with calcium hydroxide is recommended within a few weeks after the injury.[1,9,13] Later, when no signs of root resorption are evident, calcium hydroxide is replaced with traditional endodontic therapy. In the case of a nonvital pulp with incomplete root formation, root-end induction procedures must be accomplished first.[14]

Pulp Canal Obliteration

In a significant number of cases (15% to 22%), the pulp may undergo a slow process of obliteration. Calcification usually starts coronally and may completely obliterate the pulp chamber.[11,15,16] Pulp canal obliteration more frequently occurs in teeth with open apices that have experienced a severe luxation injury and seems to be a sequel to revascularization and/or innervation of a damaged pulp. Since only a minority of these teeth will develop future pupal necrosis, endodontic treatment is not usually recommended.[16,17] However, because of the difficulty of performing endodontic treatment after pupal obliteration has occurred, some endodontists recommend prophylactic treatment, once root formation is complete and pupal obliteration is beginning to be apparent.

Internal resorption is an infrequent finding (2% to 4%) with luxated permanent teeth.[18]

ALVEOLAR FRACTURE

Luxation injuries are often associated with a fracture of alveolar bone. Fracture lines may be located at any level from the marginal bone to the root apex. In addition to regular periapical views, radiographs with varying horizontal angles, an occlusal view and/or a panoramic radiograph, or focused cone-beam computed tomography (CBCT) can be helpful in determining the course and position of the fracture lines.[19] In these cases, both the tooth and the attached alveolus need to be reduced and stabilized. The type of stabilization required depends on the stability of the segment after repositioning. Large, mobile segments may require more stable fixation for a period of 4 weeks, and smaller segments that reposition well may be treated with lighter stabilization for a shorter period. Clinical examination may reveal granulation tissue in the gingival crevice or the secretion of pus from the pocket.[1] Loss of marginal bone frequently occurs with intrusion injuries and other injuries associated with alveolar fracture.[16]

MANAGEMENT OF TRAUMA AND IMMEDIATE SEQUELAE

With an understanding of the common tissue response that follows a traumatic injury, the practitioner can better manage the different injuries that may occur. The management of traumatic injuries to be discussed is based on Guidelines published by the International Association of Dental Traumatology (IADT).[19] Luxation injuries are classified as concussion, subluxation, extrusive luxation, lateral luxation, and intrusive luxation. Avulsion is classified separately. The most noted change over the past 30 years in the treatment of these injuries has been the change to short-term and flexible, rather than rigid, splints to stabilize luxated, avulsed, and root-fractured teeth.[20–22] An extension of these methods has been the use of techniques to orthodontically rather than manually reposition teeth.

The PDL is anatomically classified as a fibrous joint. Similar to other joints in the body, rigid and long-term stabilization can result in hard tissue healing or ankylosis.[23] The routine use of physical therapy after an orthopedic joint surgery is because functional stimuli depress osteogenesis but enhance fibrous tissue healing.[24] Hence luxation and avulsion injuries are best stabilized by flexible means for only 7 to 10 days.[9,25,26] If the displaced teeth can be readily repositioned by hand, then the wire may be directly bonded to the teeth. If some further repositioning is required, then orthodontic brackets can be placed to include stable teeth mesial and distal to the injured teeth. In the early mixed dentition with only central incisiors erupted, primary canines and/or primary molars may be bonded. A light braided or nickel-titanium alloy wire is then ligated in place.

Concussion and Subluxation

Concussion is an injury to the tooth and supporting structures without abnormal loosening or displacement, but it is tender to the touch or to tapping. The frequency of pulp necrosis and canal obliteration is low (2%), with necrosis occurring in teeth with completed root formation.[10] Subluxation is a similar injury except for the presence of increased mobility. Slight bleeding from the gingival crevice may be seen, which indicates damage to the periodontal tissues. Usually no immediate treatment is required unless severe mobility and/or multiple tooth injuries exist. In this situation, a flexible splint for patient comfort can be placed for up to 2 weeks. To relieve occlusal interferences, glass ionomer cement can be applied to the occlusal surface of the molars to open the bite temporarily. A soft diet for 2 weeks is recommended. The frequency of pulp necrosis and pulp canal obliteration ranges from 26% to 47%, always occurring in teeth with completed root formation.[10] Progressive root resorption is very infrequent (4%).[16]

Lateral Displacement

Lateral displacement occurs when the tooth is displaced in a direction other than axially. Because of the lateral support of adjacent teeth, this direction is often labial or palatal and is accompanied by comminution or fracture of the alveolar socket.[1] If the tooth is labially displaced, then it should be repositioned along with any attached alveolar bone. Lacerated gingiva should be adapted to the neck of the tooth and sutured. Light wire orthodontic stabilization is recommended for 4 weeks. When the crown is palatally displaced, the root apex is often displaced through the vestibular labial plate, locking it in this position.[19] Repositioning requires labial digital pressure over the apical area and on the lingual side of the crown to unlock the apex. Forceps may be needed to disengage the tooth from its bony lock. The tooth is gently repositioned into its original location. This procedure may require infraorbital regional block anesthesia on the appropriate side of the maxilla. When treatment for lateral displacement is delayed (>24 hours), the tooth is often consolidated in the new position, making reduction difficult or even contraindicated. In these situations, orthodontic appliances can be used to reposition the tooth gradually (Fig. 29-1). Periodontal and pulpal status should be radiographically monitored. Completed root formation is the major factor associated with the development of pulp necrosis.[10,11] Pulp survival is 93% with incomplete root formation, as opposed to 23% with complete root formation.[10] Progressive root resorption is uncommon with this injury.

Extrusive Luxation

An extrusion injury is characterized by a partial displacement of the tooth out of the alveolar socket. The tooth appears elongated and is excessively mobile. Treatment involves gently repositioning the tooth and stabilizing it with a light orthodontic wire for 2 weeks.

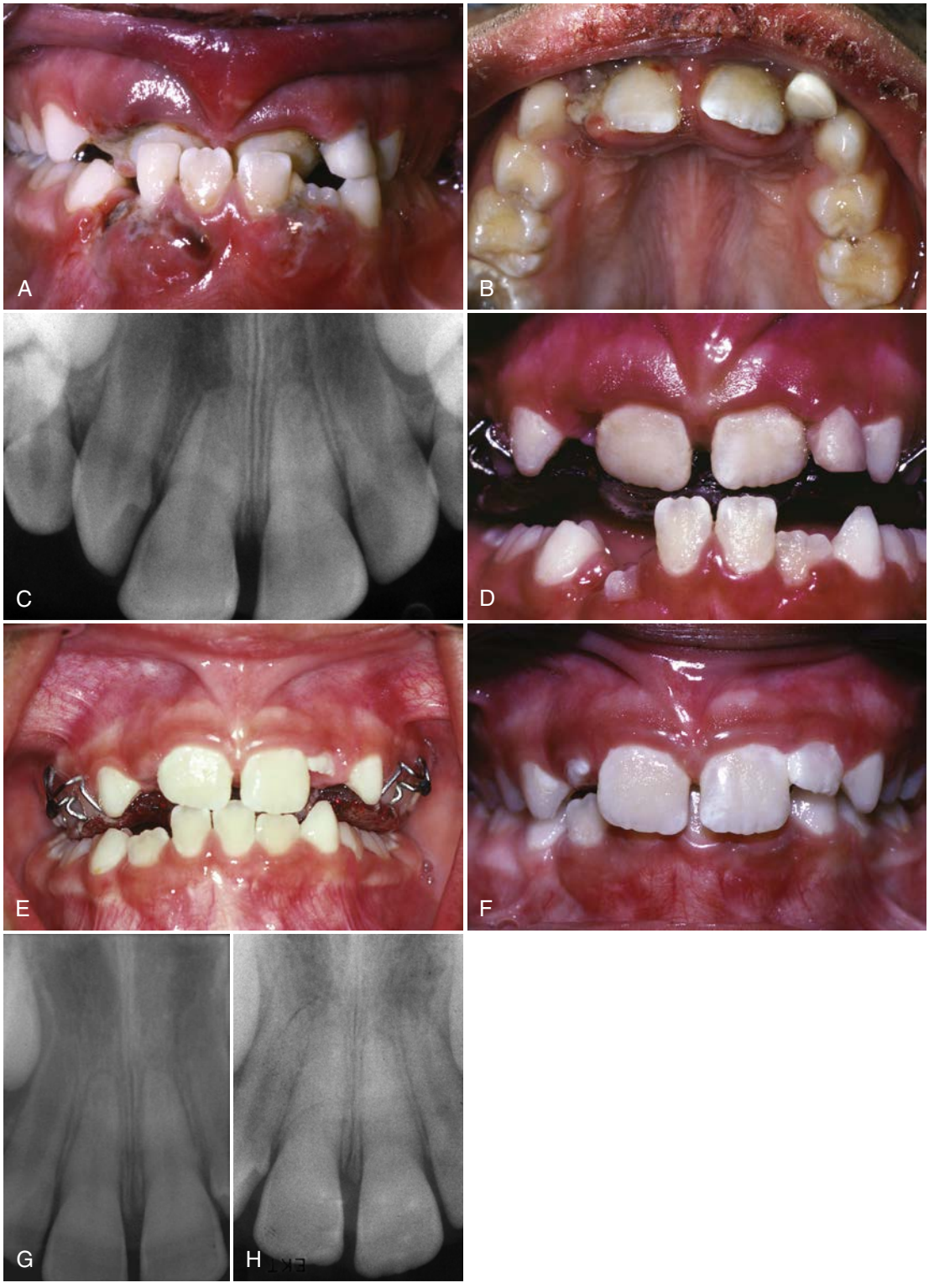


FIGURE 29-1 A, B, Lateral luxation in an 8-year-old girl is seen the day after the accident. C, Radiograph shows incomplete root formation of traumatized central incisors. D, A removable orthodontic appliance was used to open the bite and procline the incisors. E, Tooth position is improving 4 months after the trauma. F, Tooth position is visualized 7 months after the trauma. G, Root formation is continuing and pulp chamber shows narrowing 8 months after the injury. H, Radiograph 2 years after the trauma shows pulp canal obliteration.

Similar to lateral displacement, if the injury is treated many hours after the injury, then orthodontic brackets and a light archwire can be used to intrude the tooth. Pulp survival rates are 90% if the tooth has a wide open apex; in cases with completed root formation, pulp survival is less than 50%.[10] Progressive root resorption is significantly lower (7%) than with avulsion or intrusion injuries.[6,16] Pulp canal obliteration occurs in 24% of cases and is more frequent in teeth with incomplete root formation. The patient should be endodontically evaluated. If several signs and symptoms suggest a necrotic pulp, then endodontic treatment is indicated.

Intrusive Luxation

The least common displacement injury in the permanent dentition is intrusive luxation, a displacement of the tooth into the alveolar bone. These injuries are associated with a comminution or fracture of the alveolar bone. Pupal necrosis, progressive root resorption, and marginal bone loss are common sequelae to severe intrusion.[10,15,16] IADT guidelines recommend allowing *mild* intrusions to erupt without intervention.[19] If no movement occurs after 2 to 4 weeks, reposition orthodontically (or surgically) before ankylosis develops. If the tooth is intruded more than 7 mm, surgical or orthodontic repositioning is recommended. Manual/surgical repositioning has been shown to increase the frequency of complications; hence we prefer orthodontic extrusion.[10,16] In addition, our research has shown ankylosis may develop as early as 7 to 10 days following the injury; hence we prefer to initiate orthodontics within the first week following the injury for anything but a very mild intrusive injury.[27] A tooth that is tightly wedged into the bone with no mobility will not move under normal orthodontic forces and may predispose to ankylosis. These teeth should be luxated to produce some mobility before orthodontic activation (Fig. 29-2).[28] Severely displaced teeth embedded deep into bone may also need to be partially repositioned to allow the placement of orthodontic appliances and access for endodontic treatment. Because pulp survival with closed root apices is uncommon, pulp extirpation and calcium hydroxide fill are indicated within 14 days of the injury.[13,19] Teeth with immature root formation also should be observed for pathologic abnormalities since most (60%) of these pulps will not survive.[10]

Avulsion

The avulsion injury is seen in 0.5% to 3% of all dental injuries and is characterized by a complete displacement of the tooth out of the alveolar socket.[1,2] This injury is accompanied by comminution or fracture of the alveolar socket. Critical factors to the long-term survival of these teeth are the physiologic status of the PDL cells,[19] extraoral period,[6,8,29] stage of root development,[4,30] storage medium,[31,32] and method of stabilization.[23–26,33] Approximately 90% of the teeth reimplanted within 30 minutes showed no root resorption.[4] The best storage medium is in the patient's mouth; hence, parents or guardians should be instructed over the telephone how to reposition the tooth back into the socket. If the tooth cannot be repositioned, then it can be placed in the vestibule or under the child's tongue. If this is not possible, the tooth should be transported in a proper storage medium. Hanks balanced salt solution (HBSS), a tissue culture medium, is commercially available to dentists (www.Save-A-Tooth.com) and has been shown to improve the viability of remaining PDL cells.[34] Milk may be as good a storage medium as any of the commercially prepared solutions for up to 6 hours.[31] The tooth should not be stored in water.

The type of treatment for an avulsed tooth ultimately depends on two factors: (1) the maturity of the root (open or closed apex) and (2) the viability of the PDL cells.[19] The condition of the cells depends on the storage medium and the time out of the mouth. The clinician should classify the tooth into one of three groups before instituting treatment.

1. PDL cells are most likely viable as a result of immediate or short-term reposition.
2. PDL cells may be viable but compromised as a result of dry time approximately 60 minutes or less and kept in a storage medium.
3. PDL cells are nonviable as a result of oral dry time longer than 60 minutes.

TREATMENT OF AVULSED PERMANENT TEETH WITH CLOSED APEX

A tooth with a closed apex that arrives at the office having been replanted after a short time has an excellent chance of survival. The orthodontist should clinically and radiographically verify the position of the tooth. After cleaning the area with water spray, saline, or chlorhexidine, a flexible splint is applied and worn for up to 2 weeks. Endodontic treatment should begin 7 to 10 days after replantation but before removing the splint.

The tooth that arrives at the office in a physiologic storage medium or after a dry time of less than 60 minutes should be replanted by the orthodontist. The root surface and apical foramen are cleaned with saline, and the tooth is soaked in saline while preparing to replant. A local anesthesia is administered, and the socket is irrigated with saline. The alveolar socket is examined, and any fractured or displaced socket wall is repositioned. The tooth is slowly replanted with slight digital pressure, and the normal position of the tooth is clinically and radiographically verified. A flexible splint is applied, and endodontic treatment should begin 7 to 10 days after the replantation but before removing the splint.

If the closed apex tooth has dry time longer than 60 minutes, then the PDL cells are necrotic; hence, the eventual outcome is ankylosis and root resorption and the eventual loss of the tooth. Before replantation, the orthodontist should carefully remove attached nonviable soft tissue with gauze. Endodontic treatment can be performed before or after replantation. Replantation is performed as previously described, and a flexible splint is applied and worn for 4 weeks. To slow down osseous replacement of the tooth, some have suggested treating the root surface with 2% sodium fluoride for 20 minutes before replantation.

TREATMENT OF AVULSED PERMANENT TEETH WITH OPEN APEX

Teeth immediately replanted with open apices are treated as previously described but are not endodontically treated since the pulp may revascularize. If that does not occur, then endodontic treatment will be indicated.

Teeth with open apices in a proper storage medium or with a dry time less than 60 minutes should be replanted as previously described. Before replantation, the topical application of antibiotics (minocycline or doxycycline) has been shown to enhance the chances for revascularization, 1 mg per 20 mL of saline for a 5-minute soak.[35,36] The pulp space may revascularize; however, infection-related root resorption is very rapid in immature teeth. Therefore close endodontic monitoring is recommended.

Teeth with open apices and a dry time longer than 60 minutes have a poor long-term prognosis. They should be managed similarly to teeth with closed apices.

FIGURE 29-2 A, Severe intrusive luxation is revealed in a 23-year-old man. The tooth has no mobility. B, Radiograph of intruded tooth shows that no lamina dura is visible, which is attributable to tearing and crushing of periodontal ligament. C, Tooth is luxated and slightly extruded to provide a periodontal ligament space, some mobility, and access for orthodontic attachments and endodontic intervention with calcium hydroxide. D, Orthodontic brackets and light arch wire are placed the same day. E, Alignment of tooth is visualized 10 weeks after the trauma. F, Radiograph reveals alignment of the tooth 10 weeks after the trauma. Calcium hydroxide root canal was accomplished 2 weeks after the injury. No progressive root resorption or periapical pathologic condition is noted. G, Image reveals alignment after treatment. H, Radiograph 9 months after the trauma shows definitive root canal treatment. Tooth #9 subsequently became nonvital and was treated.

Tetanus prophylaxis should be administered if the child's immunization status is compromised or if the tooth or wound is dirty. Antibiotic coverage also should be prescribed in the first week—penicillin V or amoxicillin in an appropriate dose for age and weight. Avulsion injuries with closed apices rarely show pupal revascularization. As a result, with an extended extraoral period or a closed apex, the pulp should be extirpated after waiting 7 to 10 days and before splint removal. Calcium hydroxide should be placed in the canal to help prevent root resorption.[37] After 1 month, calcium hydroxide is replaced by a definitive root canal filling. Placing calcium hydroxide too soon, such as immediately after reimplantation, will promote inflammation that can lead to PDL damage.[38] Lengheden and colleagues[39,40] have suggested that long-term calcium hydroxide root canal fill may also have deleterious effects.

PREVENTION

Numerous studies have documented the increased incidence of traumatic dental injuries with Class II malocclusions.[41–43] Preventive measures should therefore include early correction of cases with significant overjet and lip incompetence. Evidence suggests that providing early orthodontic treatment for children with prominent upper front teeth is more effective in reducing the incidence of incisal trauma than providing one course of treatment when the child is in early adolescence.[44] Athletic mouth guards should be used for contact sports and can be fabricated to accommodate fixed orthodontic appliances, tooth movement, and exfoliation-eruption processes in the mixed dentition.[45–47] Stock mouth guards, available at sporting good stores, must be held in place with constant occlusal pressure,

because of their poor fit. Boil-and-bite guards may provide a better fit, but some evidence suggests that neither type reduces the incidence of trauma and may actually give the athlete a false sense of security. Custom-fitted vacuum-formed mouth guards, or pressure laminating, which provides the best fit and shape stability, is the mouth guard of choice and can be fabricated in the office with proper equipment or by numerous commercial laboratories.

Traumatic injuries should always be managed with the cause of the injury in mind. Estimates suggest that 1 out of 10 children are physically abused, of whom 75% are reported to have injuries of the head and neck area.[48] Numerous studies note the low reporting frequency of dentists,[49–53] and preliminary data suggest orthodontists rarely report abuse.[54] Trauma prevention requires orthodontists to be knowledgeable of the signs of abuse and the obligation and methods for reporting. Avulsed or discolored teeth, bruises on the cheek or around the neck, burns in the shape of hot objects, and bite marks are often associated with child abuse. Children displaying psychosomatic complaints and seductive behavior or those with unusual knowledge of sexual behavior may have suffered sexual abuse. A strong correlation exists between dental neglect and physical neglect. Mandates in all 50 states require that dental professionals be aware of and report instances of child abuse and neglect to the proper state child protection authorities. State laws also protect the dental professional from civil retribution.[55,56]

REFERENCES

1. Andreasen JO, Andreasen FM, Andersson L. *Textbook and Color Atlas of Traumatic Injuries to the Teeth*. 4th ed. Oxford, UK: Wiley-Blackwell; 2007.
2. Glendor U. Epidemiology of traumatic dental injuries—a 12 year review of the literature. *Dent Traumatol*. 2008;24(6):603–611.
3. Perez R, Berkowitz R, Mcllveen L, Forrester D. Dental trauma in children: a survey. *Endod Dent Traumatol*. 1991;7:212–213.
4. Andreasen JO, Hjorting-Hansen E. Replantation of teeth. II. Histological study of 22 replanted anterior teeth in humans. *Acta Odontol Scand*. 1966;24: 287–306.
5. Andreasen JO. Relationship between surface and inflammatory resorption and changes in the pulp after replantation of permanent incisors in monkeys. *J Endod*. 1981;7:294–301.
6. Andreasen JO, Hjorting-Hansen E. Replantation of teeth. I. Radiographic and clinical study of 110 human teeth replanted after accidental loss. *Acta Odontol Scand*. 1966;24:263–286.
7. Andreasen JO. Treatment of fractured and avulsed teeth. *ASDC J Dent Child*. 1971;38:29–31.
8. Cvek M. Treatment of non-vital permanent incisors with calcium hydroxide. II. Effect on external root resorption in luxated teeth compared with effect of root filling with guttapucha. A follow-up. *Odontol Revy*. 1973;24:343–354.
9. Andreasen JO. Challenges in clinical dental traumatology. *Endod Dent Traumatol*. 1985;1:45–55.
10. Andreasen FM, Pederson BV. Prognosis of luxated permanent teeth—the development of pulp necrosis. *Endod Dent Traumatol*. 1985;1:207–220.
11. Andreasen FM, Thomsen BL, Andersen PK. Occurrence of pulp canal obliteration after luxation injuries in the permanent dentition. *Endod Dent Traumatol*. 1987;3:103–115.
12. Andreasen FM, Zhijie Y, Thomsen BL. Relationship between pulp dimensions and development of pulp necrosis after luxation injuries in the permanent dentition. *Endod Dent Traumatol*. 1986;2:90–98.
13. Spalding PM, Fields Jr HW, Torney D, et al. The changing role of endodontics and orthodontics in the management of traumatically intruded permanent incisors. *Pediatr Dent*. 1985;7:104–110.
14. Frank AL. Therapy for the divergent pulpless tooth by continued apical formation. *J Am Dent Assoc*. 1966;72:87–93.
15. Turley PK, Joiner MW, Hellstrom S. The effect of orthodontic extrusion on traumatically intruded teeth. *Am J Orthod*. 1984;85:47–56.
16. Andreasen JO. Luxation of permanent teeth due to trauma. A clinical and radiographic follow-up study of 189 injured teeth. *Scand J Dent Res*. 1970;78:273–286.
17. Jacobsen I, Kerekes K. Long-term prognosis of traumatized permanent anterior teeth showing calcifying processes in the pulp cavity. *Scand J Dent Res*. 1977;85:588–598.
18. Crona-Larsson G, Bjarnason S, Norén J. Effect of luxation injuries on permanent teeth. *Endod Dent Traumatol*. 1991;7(5):199–206.
19. DiAngelis AJ, Andreasen JO, Ebeleseder KA, et al. International Association of Dental Traumatology guidelines for the management of traumatic dental injuries: 1. Fractures and luxations of permanent teeth. *Dent Traumatol*. 2012;28:2–12.
20. Kahler B, Heithersay GS. An evidence-based appraisal of splinting luxated, avulsed and root-fractures teeth. *Dent Traumatol*. 2008;24(1):2–10.
21. Andreasen JO, Andreasen FM, Mejàre I, Cverk M. Healing of 400 intra-alveolar root fractures. 2. Effect of treatment factors such as treatment delay, repositioning, splinting type and period and antibiotics. *Dent Traumatol*. 2004;20(4):203–211.
22. Hinckfuss SE, Messer LB. Splinting duration and periodontal outcomes for replanted avulsed teeth: a systematic review. *Dent Traumatol*. 2009;25: 150–157.
23. Andreasen JO. The effect of splinting upon periodontal healing after replantation of permanent incisors in monkeys. *Acta Odontol Scand*. 1975;33(6):313–323.
24. Clark Jr HB, Hayes PA. A study of the comparative effects of "rigid" and "semirigid" fixation on the healing of fractures of the mandible in dogs. *J Bone Joint Surg Am*. 1963;45–A:731–741.
25. Nasjleti CE, Castelli WA, Caffesse RG. The effects of different splinting times on replantation of teeth in monkeys. *Oral Surg Oral Med Oral Pathol*. 1982;53(6):557–566.
26. Andersson LM, Lindskog S, Blomlöf L, et al. Effect of masticatory stimulation of dentoalveolar ankylosis after experimental tooth replantation. *Endod Dent Traumatol*. 1985;1:13–16.
27. Chung KR, Turley PK. The onset of ankylosis following intrusive luxation injuries. *Korean J Orthod*. 1991;21:259–272.
28. Turley PK, Crawford LB, Carrington KW. Traumatically intruded teeth. *Angle Orthod*. 1987;57:234–244.
29. Grossman LI, Ship II . Survival rate of replanted teeth. *Oral Surg Oral Med Oral Pathol*. 1970;29:899–906.
30. Skieller V. The prognosis for young teeth loosened after mechanical injuries. *Acta Odontol Scand*. 1960;18(2):171–181.
31. Hiltz J, Trope M. Vitality of human lip fibroblasts in milk, Hanks balanced salt solution and Viaspan storage media. *Endod Dent Traumatol*. 1991;7:69–72.
32. Krasner P, Person P. Preserving avulsed teeth for replantation. *J Am Dent Assoc*. 1992;123:80–88.
33. Hurst RV. Regeneration of periodontal and transseptal fibers after autografts in Rhesus monkeys: a qualitative approach. *J Dent Res*. 1972;51:1183–1192.
34. Krasner P, Rankow HJ. *New Philosophy Treatment of the Avulsed Permanent Tooth: Recommended Guidelines of the American Association of Endodontics*; 1995; Chicago.
35. Cvek M, Cleaton-Jones P, Austin J, et al. Effect of topical application of doxycycline on pulp revascularization and periodontal healing in reimplanted monkey incisors. *Endod Dent Traumatol*. 1990;6:170–176.
36. DiAngelis AJ, Leonard MS. Management of tooth avulsions. *Dent Today*. 1998;17:72–75.
37. Andreasen JO, Kristerson L. The effect of extra aveolar root filling with calcium hydroxide on periodontal healing after replantation of permanent incisors in monkeys. *J Endod*. 1981;7:349–354.
38. Lindskog S, Blomlöf L, Hammarström L. Cellular colonization of denuded root surfaces in vivo: cell morphology in dentin resorption and cementum repair. *J Clin Periodontal*. 1987;14:390–395.
39. Lengheden A, Blomlöf L, Lindskog S. Effect of delayed calcium hydroxide treatment on periodontal healing in contaminated replanted teeth. *Scand J Dent Res*. 1991;99:147–153.

40. Lengheden A, Blomlöf L, Lindskog S. Effect of immediate calcium hydroxide treatment and permanent root-filling on periodontal healing in contaminated replanted teeth. *Scand J Dent Res*. 1991;99:139–146.
41. Burden DJ. An investigation of the association between overjet size, lip coverage, and traumatic injury to maxillary incisors. *Eur J Orthod*. 1995;17(6):513–517.
42. Bauss O, Röhling J, Schwestka-Polly R. Prevalence of traumatic injuries to the permanent incisors in candidates for orthodontic treatment. *Dent Traumatol*. 2004;20:61–66.
43. Ben Bassat Y, Brin I, Brezniak N. Can maxillary incisor trauma be predicted from cephalometric measurements? *Am J Orthod Dentofacial Orthop*. 2001;120(2):186–189.
44. Thiruvenkatachari B, Harrison JE, Worthington HV, O'Brien KD. Orthodontic treatment for prominent upper front teeth (class II malocclusion) in children. *Cochrane Database Syst Rev*. 2013;11. http://dx.doi.org/10.1002/14651858.CD003452.pub3. CD003452.
45. Croll TP, Castaldi CR. The custom-fitted athletic mouthguard for the orthodontic patient and for the child with a mixed dentition. *Quintessence Int*. 1989;20(8):571–575.
46. Padilla R, Balikov S. Sports dentistry: coming of age in the '90s. *J Calif Dent Assoc*. 1993;21:27–37.
47. ADA Council on Access. Prevention and Interprofessional Relations; ADA Council on Scientific Affairs. Using mouthguards to reduce the incidence and severity of sports-related oral injuries. *J Am Dent Assoc*. 2006;137(12):1712–1720.
48. da Fonseca MA, Feigal RJ, ten Bensel RW. Dental aspects of 1248 cases of child maltreatment on file at a major county hospital. *Pediatr Dent*. 1992;14:152–157.
49. Hazelwood Al. Child abuse: the dentist's role. *NY State Dent J*. 1970;36:289–291.
50. Becker DB, Needleman HL, Kotelchuck M. Child abuse and dentistry: orofacial trauma and its recognition by dentists. *J Am Dent Assoc*. 1978;97:24–28.
51. Kittle PE, Richardson DS, Parker JW. Two child abuse/child neglect examinations for the dentist. *ASDC J Dent Child*. 1981;48:175–180.
52. Stanley RT. Child abuse—what's a dentist to do? *Ohio Dent J*. 1981;55:16–27.
53. Malecz RE. Child abuse, its relationship to pedodontics: a survey. *ASDC J Dent Child*. 1979;46:193–194.
54. Ramos-Gomez F, Rothman D, Blain S. Knowledge and attitudes among California dental care providers regarding child abuse and neglect. *J Am Dent Assoc*. 1998;129:340–348.
55. Katner DR, Brown CE. Mandatory reporting of oral injuries indicating possible child abuse. *J Am Dent Assoc*. 2012;143(10):1087–1092.
56. Guideline on Oral and Dental Aspects of Child Abuse and Neglect, American Academy of Pediatrics Committee on Child Abuse and Neglect and the American Academy of Pediatric Dentistry, *AAPD Clinical Practice Guidelines*. 2010;37(6): 172–175. Available at: http://www.aapd.org/media/policies_guidelines/g_childabuse.pdf. Accessed June 26, 2016.

30

Iatrogenic Effects of Orthodontic Treatment

Philip Edward Benson, Norah Lisa Flannigan, Glenn Sameshima, and M. Ali Darendeliler

OUTLINE

PREVENTION AND MANAGEMENT OF DEMINERALIZED WHITE LESIONS, by Philip Edward Benson and Norah Lisa Flannigan, 887
Prevalence of Demineralized White Lesions, 887
Detection and Measurement of Demineralized White Lesions, 888
Transverse Microradiography, 888
Quantitative Light-Induced Fluorescence, 888
Quantitative Light-Induced Fluorescence–Digital, 888
Prevention of Demineralized White Lesions, 888
Preventing the Loss of Mineral from Enamel (Demineralization), 889
Promoting the Uptake of Mineral into Enamel (Remineralization), 892
Treatment of Demineralized White Lesions, 895
Fluoride, 896
Casein Phosphopeptide–Amorphous Calcium Phosphate, 896
Resin Infiltration, 896
ROOT RESORPTION, by Glenn Sameshima and M. Ali Darendeliler, 896
Etiologic Factors, 896
Risk Factors, 897
Diagnostic Factors, 897
Tooth Anatomy, 897
Demographic Factors, 897
Malocclusion Factors, 898
Patient Medical History and Habits, 899
Treatment Factors, 900
Mechanical Factors, 900
Magnitude of Applied Force, 900
Intermittent versus Continuous Force, 900
Early Treatment, 900
Expansion, 900
Extractions, 900
Prolonged Treatment Time, 900
Apical Root Displacement, 900
Management, 901
Imaging, 901
History, 901
Progress Review, 902
What to Do If Root Resorption Is Detected at Progress, 902
When Does External Apical Root Resorption Start? 902
When Does External Apical Root Resorption Stop? 903
What Happens to Teeth with Short Roots Long Term? 903
Are There Any Methods to Detect Root Resorption before It is Visible on Radiographs? 904

PREVENTION AND MANAGEMENT OF DEMINERALIZED WHITE LESIONS*

Despite many advances in orthodontic techniques, the occurrence of demineralized white lesions (DWLs) during treatment remains a serious side effect, particularly when using fixed appliances (Fig. 30-1). Early enamel demineralization clinically exhibits as *white spot lesions*, defined by Fejerskov and colleagues as "the first sign of a caries lesion on enamel that can be detected with the naked eye."[1] The *milky white* appearance is caused by an increased scattering of light that is attributable to the loss of crystal structure and is exaggerated when the enamel is dried.[2]

The risk of demineralization is greatest during fixed orthodontic treatment as a result of plaque collection and retention around the irregular surfaces of the attachments.[3] These stagnation areas also limit the self-cleansing mechanism of salivary flow,[4] resulting in the accumulation of cariogenic bacterial species.[5] After 14 days of completely undisturbed plaque, changes in the enamel are visible with air drying. After 3 to 4 weeks, the outermost surface exhibits further porosity, and clinical changes can be seen without air drying.[1] The rate of progression of mineral loss around fixed orthodontic appliances can be faster than the traditional caries process, with demineralization being clinically apparent within 6 months of starting treatment.[6]

Prevalence of Demineralized White Lesions

The reported proportion of patients with DWLs after fixed orthodontic treatment widely varies in the literature between 2% and 96%.[7–9] This variation can be attributed to differences in the standardization of clinical examinations, as well as the use of various detection tools, with different sensitivities and specificities in diagnosing demineralization.[10] Al Maaitah and colleagues[11] described

* Philip Edward Benson and Norah Lisa Flannigan.

FIGURE 30-1 The typical appearance of demineralized white lesions is revealed on the day a fixed appliance is removed.

a prevalence of 71.7% DWLs in 230 participants after orthodontic treatment. This finding was determined using a technique called *quantitative light-induced fluorescence* (QLF) (Inspektor Research Systems, Bussum, Netherlands), which has a high sensitivity for detecting DWLs. Julien and colleagues reported an incidence of 23.4% DWLs in 885 participants using digital photography.[12]

Some differences can be found in the literature about which teeth are most commonly affected with DWLs during orthodontic treatment. Several studies conclude that maxillary incisors are particularly susceptible,[7,13] whereas other studies found that canines and molars are more frequently affected.[3] Evidence suggests that patients with DWLs, particularly on the anterior teeth, and their parents, perceive the aesthetics to be poorer after treatment.[14] Ogaard has shown that a difference in prevalence of DWLs between those who have and those who have not had orthodontic treatment is still present 5 years after the appliances have been removed.[9]

Detection and Measurement of Demineralized White Lesions

Transverse Microradiography

The generally accepted gold standard method of measuring demineralization and remineralization is transverse microradiography (TMR). This technique is destructive, requiring cuts to be made to the enamel or dentine and microsamples to be removed for analysis. The basis of TMR is the measurement of x-ray absorption by a tooth section, compared with absorption by a simultaneously exposed standard.[15] In this technique, planoparallel sections (approximating 80 μm for enamel) are cut from the sample to be investigated. The sections, which are cut perpendicular to the anatomic tooth surface, are placed on high-resolution photographic film, along with an aluminum calibration stepwedge and irradiated with monochromatic x-ray beams.[16] Absorption of the x-ray beams by the tooth sample and stepwedge is directly reflected in the optical density of the developed film (microradiograph).

Analysis of the mineral content and distribution is calculated by means of Angmar's formula[17] from the optical density of the tooth sample and stepwedge images. Densitometry-based systems and, more recently, image analysis systems comprising a charge-coupled device (CCD) video camera and dedicated software are used for the evaluation of microradiographic optical densities and for the generation of mineral content profiles.[18] From the analysis, three primary parameters are obtained: mineral loss (ΔZ units Vol%.μm), lesion depth (Ld) (μm units), and lesion width (Lw) (μm units). ΔZ is the integrated difference between the microradiograph of the sample with mineral loss and that of the sound sample, whereas Ld and Lw values are determined from mineral distribution (Fig. 30-2).[16]

A major disadvantage of the TMR process is that either the tooth must be removed from the mouth and destroyed or a small enamel sample attached to the appliance (in situ method, Fig. 30-3) must be removed. If the tooth to be examined has to be extracted as part of an orthodontic treatment plan, then only the first few weeks of orthodontic treatment can be studied. Alternatively, an in situ enamel sample might not be truly representative of the environment around a bracket.[19]

This limitation of TMR, as well as the need for early detection of DWLs to prevent irreversible damage to the enamel, has made optical methods of demineralization analysis increasingly popular. Available optical methods include QLF, which allows the detection of demineralization before the lesions are clinically visible to a trained examiner (subclinical).[20]

Quantitative Light-Induced Fluorescence

QLF is a nondestructive diagnostic technique that uses visible light for the early detection of demineralization in enamel (Fig. 30-4). The principle behind the technique is that enamel will undergo autofluorescence under certain conditions. Demineralized enamel will fluoresce less, and this loss of fluorescence can be detected, quantified, and longitudinally monitored using QLF (Fig. 30-5). The teeth under investigation are illuminated from a lamp with a peak intensity of 370 nanometers (nm). A yellow high-pass filter (520 nm) is placed in front of a CCD microcamera, which captures the tooth image.[21] A live image of the tooth is displayed on a personal computer (PC) screen and can be stored for analysis.[22,23] The analysis software detects the darker area of the image and fluorescence radiance of sound enamel at the lesion site via a reconstruction algorithm.[21] QLF has been validated against TMR in permanent teeth and has shown excellent agreement.[24]

Quantitative Light-Induced Fluorescence–Digital

The quantitative light-induced fluorescence–digital (QLF-D) Biluminator (Inspektor Research Systems, Bussum, Netherlands) is a novel device based on QLF technology (Fig. 30-6). It takes two successive images, a white light (WL) image, which is a conventional digital photograph, and a QLF image. As the two images are almost simultaneously taken, it ensures consistency with regard to magnification and allows for comparisons between the images. Using this system, demineralization can be readily identified and assessed (Fig. 30-7). Plaque is barely visible in a WL image; however, with the change in the filter system, the emitted red fluorescence makes plaque visible (Fig. 30-8). The current cost of the complete QLF-D system is approximately 9000 British pounds (14,000 U.S. dollars). Clinicians have implemented the use of QLF-D onto the orthodontic clinic for routine plaque screening and also for monitoring initial subclinical lesions. Early detection allows the clinician to apply preventive measures before DWLs are clinically visible and avoid the development of unsightly lesions during the course of the orthodontic treatment.

Prevention of Demineralized White Lesions

In 1999, Featherstone proposed a model to explain the caries process called The Caries Balance concept (Fig. 30-9).[25] The model has been amended several times, but, essentially, it consists of three factors that lead to the loss of enamel, or demineralization: (1) fermentable carbohydrate, (2) acidogenic

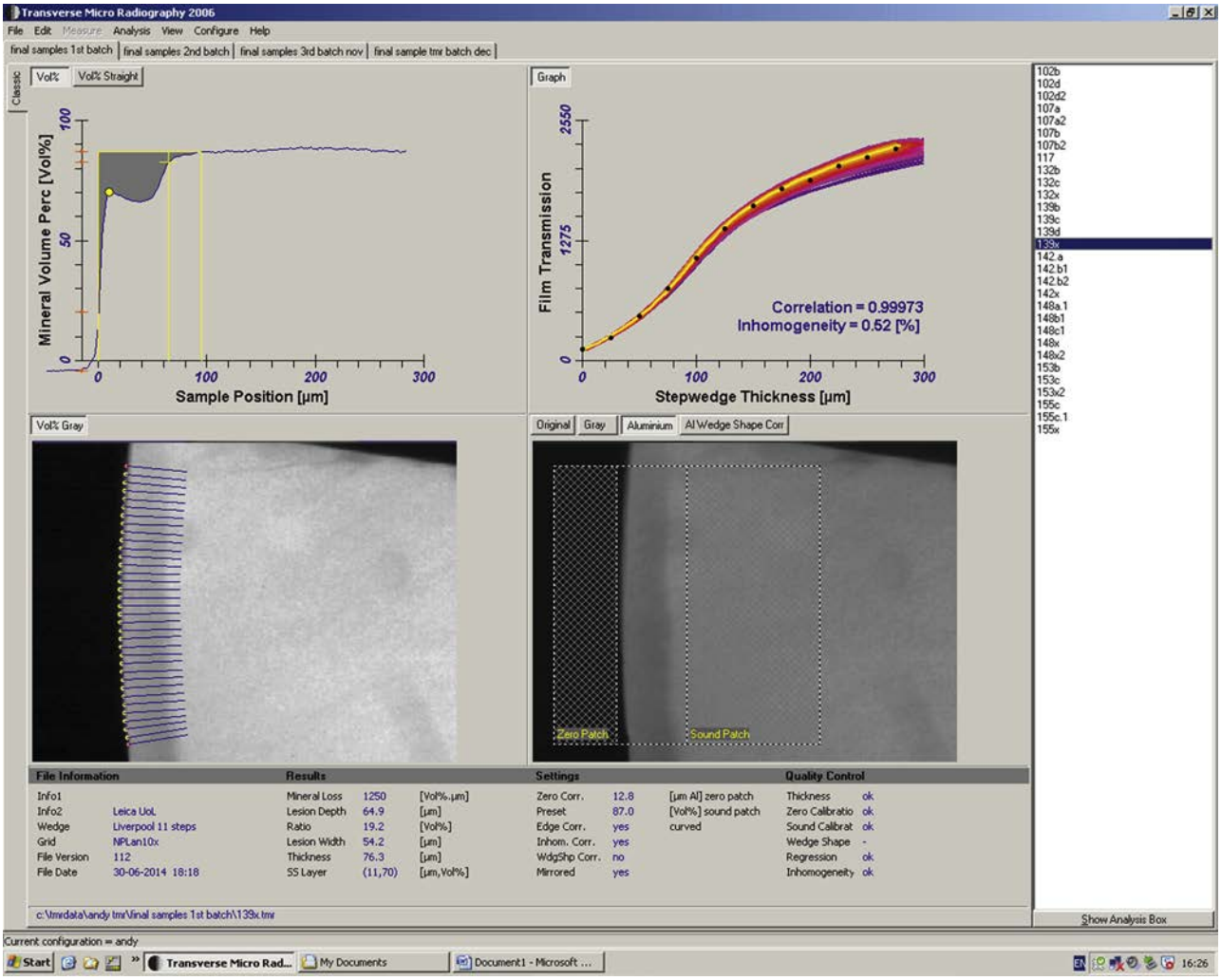


FIGURE 30-2 Output from dedicated transverse microradiography (TMR) software (Inspektor Research Systems, Bussum, Netherlands) illustrates subsurface mineral loss. (Courtesy of Inspektor Research Systems, Bussum, Netherlands.)

FIGURE 30-3 This example of an in situ enamel sample is used with a fixed appliance. (Reproduced with permission from Doherty UB, Benson PE, Higham SM. Fluoride-releasing elastomeric ligatures assessed with the in situ caries model. *Eur J Orthod* 2002;24:371–378.)

bacteria, and (3) reduced salivary flow; and three factors that lead to the uptake of mineral into enamel, or remineralization: (1) fluoride, (2) antimicrobial agents, and (3) salivary flow.[26] The Caries Balance model can be used to develop ways of preventing DWLs in orthodontic patients through:

- Preventing the loss of mineral from enamel (demineralization)
- Promoting the uptake of mineral into enamel (remineralization)

Preventing the Loss of Mineral from Enamel (Demineralization)

There are three ways of preventing demineralization:

1. Reducing the amount of acidogenic plaque through mechanical and/or chemical methods
2. Reducing the frequent consumption of sugar (diet advice)
3. Sealing the enamel

Reducing the amount of acidogenic plaque. Studies have suggested that inadequate oral hygiene before the placement of appliances is one of the most predictable risk factors for the development of DWLs during orthodontic treatment[11,27,28]; therefore clinicians must be satisfied that patients exhibit an acceptable level of oral health before placing any appliances.

FIGURE 30-4 Quantitative light-induced fluorescence (QLF) (Inspektor Research Systems, Bussum, Netherlands) hardware and camera handpiece (*inset*). (Courtesy of Inspektor Research Systems, Bussum, Netherlands.)

Reducing the levels of acidogenic plaque can be achieved through mechanical and/or chemical methods.

Mechanical. Plaque removal may be improved either through the use of more effective devices or by helping patients more efficiently use their devices (toothbrushing advice). Evidence suggests that electric toothbrushes are better than manual toothbrushes for removing plaque in people with[29,30] and without orthodontic appliances.[31] Little evidence supports the effectiveness of interdental cleaning, such as flossing, in orthodontic[32] or nonorthodontic populations.[33]

Some evidence suggests that intensive motivation techniques[34,35] or the use of reminders, such as weekly text messages to parents,[36] improve gingival health in the short term; however, more long-term studies over the full length of orthodontic treatment are required.[37] In addition, it has yet to be shown that all the effort put into encouraging patients to clean their appliances leads to a lower incidence of DWLs, but new lesions might be less severe.[38]

Chemical. Regular use of a chlorhexidine mouth rinse[39,40] or toothpaste[41] can reduce plaque and gingivitis in young people undergoing orthodontic treatment without the adverse effect of increased tooth staining. Concentrated chlorhexidine varnish can also reduce the numbers of *Streptococcus mutans* for up to 4 weeks in the mouths of young people with high initial levels wearing a fixed appliance.[42] Little evidence suggests that this reduction in the number or change in the type of bacteria in plaque leads to fewer new DWLs in orthodontic patients. One study found that the addition of chlorhexidine to a fluoride varnish regularly applied through orthodontic treatment did not provide additional protection from DWLs[43]; another study showed that the use of a triclosan/copolymer toothpaste in a nonorthodontic population leads to a small reduction in caries.[44] No reliable evidence suggests that adding an antimicrobial agent to a bonding material will prevent demineralization.[45]

Reducing the frequent consumption of sugar (diet advice). In addition to lowering the number of acidogenic bacteria,

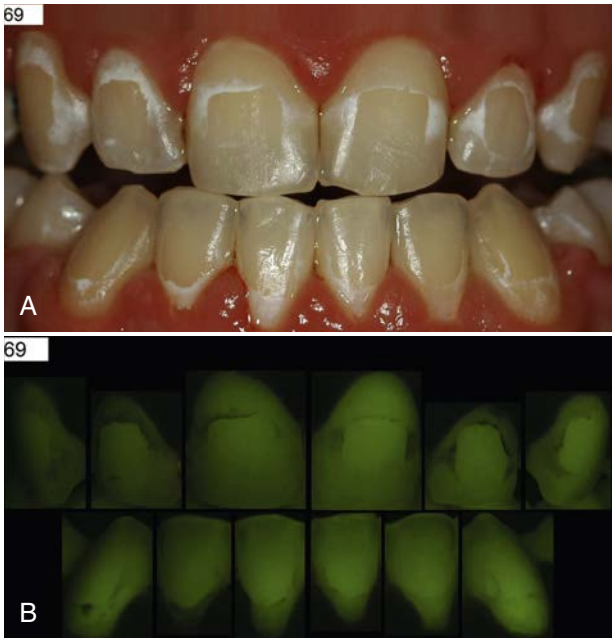


FIGURE 30-5 Two images illustrate the clinical photograph with demineralized white lesions on the day of debonding **(A)** and the corresponding quantitative light-induced fluorescence (QLF) image **(B)**.

FIGURE 30-6 The quantitative light-induced fluorescence–digital (QLF-D) Biluminator (Inspektor Research Systems, Bussum, Netherlands) equipment **(A)** and in use **(B)**.

FIGURE 30-7 White light (WL) **(A)** and quantitative light-induced fluorescence (QLF) **(B)** images are simultaneously taken with the quantitative light-induced fluorescence–digital (QLF-D) Biluminator, demonstrating demineralization.

FIGURE 30-8 White light (WL) **(A)** and quantitative light-induced fluorescence (QLF) **(B)** are images simultaneously taken with the quantitative light-induced fluorescence–digital (QLF-D) Biluminator, demonstrating plaque accumulation.

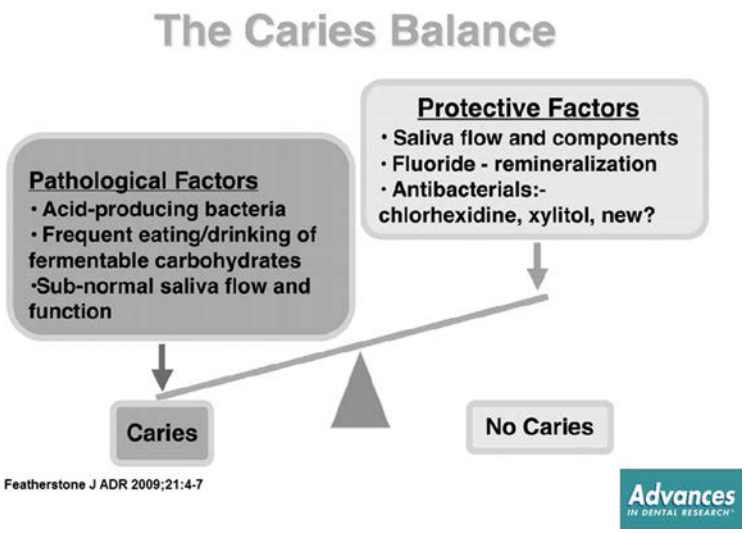


FIGURE 30-9 The Caries Balance model proposed by Featherstone. (Reproduced with permission from Featherstone JD. Remineralization, the natural caries repair process—the need for new approaches. *Adv Dent Res* 2009;21:4–7.)

another important way of reducing demineralization is to decrease the intake of fermentable carbohydrates used by the bacteria to produce acid, which dissolves the mineral. A few studies examined the effect of diet advice on orthodontic patients. One qualitative study found that a significant proportion of participants in the early stages of fixed appliance treatment said that they had heeded the advice of their orthodontist by reducing the frequency and amount of sugar they consumed[46]; however, little evidence in the wider literature suggests that professional advice changes the diet over the long term and is effective at reducing the level of caries.[47]

Patient and parental education and regular review of oral hygiene are important; however, additional measures to prevent demineralization are also necessary.

Sealing the enamel. Another proposed method of preventing mineral loss is to place a resin sealant over most of the labial enamel surface, when brackets are bonded to the tooth. The initial results with this technique were disappointing, possibly attributable to a loss of the sealant.[48,49] More recent studies have found a reduction in DWLs when the enamel surrounding the brackets is sealed with resin.[50–52] One study found a small reduction in the incidence but not in the severity of DWLs.[53] There does not seem to be any advantage in using a sealant containing fluoride, compared with one without fluoride.[54]

Promoting the Uptake of Mineral into Enamel (Remineralization)

Preventing the loss of mineral from the teeth of patients undergoing orthodontic treatment is desirable, but another approach is to change the balance within the mouth toward remineralization of the enamel. Remineralization is the natural repair of a carious lesion through the processes of inorganic chemistry,[26] and it has been extensively studied in the laboratory, as well as in the human mouth, and can be promoted in a number of ways.

Increasing the availability of fluoride in the mouth. Frequent exposure of dental enamel to fluoride has been known to reduce the incidence and severity of caries for years; however, only relatively recently has it been recognized that the primary mechanism of achieving this reduction, once the teeth have erupted, is through the promotion of remineralization. Laboratory studies have shown that remineralization is enhanced in the presence of fluoride, and the process actually makes enamel more resistant to further demineralization.[26] Ensuring that fluoride is available in the mouth can be achieved in a number of ways.

Toothpaste. Toothpaste is probably the most common regular source of fluoride for most orthodontic patients. Although little evidence suggests that toothpaste, containing a standard concentration of fluoride, prevents enamel caries in orthodontic patients, there is sufficient evidence in a general population[55]; therefore every orthodontic patient should be encouraged to use toothpaste with a fluoride concentration of at least 1000 parts per million (ppm), twice a day.[56] Some evidence suggests that toothpaste containing a higher concentration of fluoride (5000 ppm) is more effective at preventing DWLs in orthodontic patients than toothpaste with the conventional level of fluoride (1450 ppm).[57]

Mouth Rinses. Many orthodontists recommend the regular use of a fluoride mouth rinse to their patients to reduce the incidence of DWLs, even though very little reliable evidence suggests that fluoride mouth rinses are effective.[58] One study found that an amine fluoride–stannous fluoride toothpaste and mouth rinse combination was marginally more effective than a sodium fluoride toothpaste and mouth rinse combination at preventing DWLs in orthodontic patients.[59] Although little evidence supports the theory that the regular use of a fluoride mouth rinse is effective in orthodontic patients, a reasonable amount of reliable evidence in the nonorthodontic population suggests that it provides additional protection from caries to children over and above other sources of fluoride.[60] Orthodontic patients should therefore be encouraged to use a regular, daily fluoride mouth rinse (230 to 250 ppm) throughout the fixed appliance treatment. Unfortunately, only a minority of orthodontic patients will follow these instructions.[61]

Varnish. One good quality clinical trial has shown that regular professional applications of a varnish containing a high

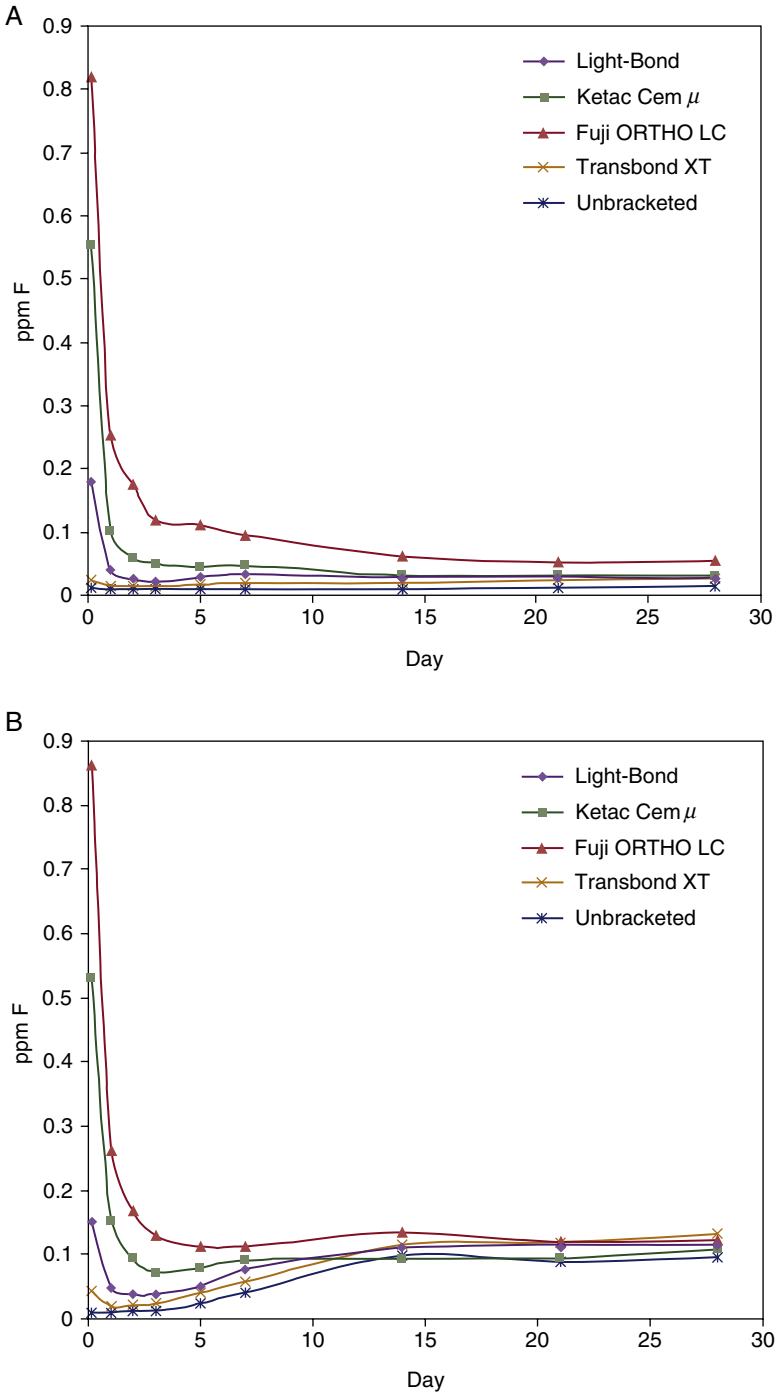


FIGURE 30-10 A, Graph demonstrates typical fluoride release profiles from materials in the laboratory with rapid loss within days, followed by very low but sustained fluoride release thereafter. B, Graph demonstrates that immersion in a fluoride mouth rinse leads to some increased release of fluoride. (Reproduced with permission from Chin MY, Sandham A, Rumachik EN, et al. Fluoride release and cariostatic potential of orthodontic adhesives with and without daily fluoride rinsing, *Am J Orthod Dentofacial Orthop* 2009;136:547–553.)

level of fluoride (7700 ppm wet; approximately 30,000 ppm dry) at each orthodontic adjustment visit reduces the incidence of DWLs, compared with a placebo varnish (absolute risk reduction 18%, number needed to treat 5.5).[62] Although this study is only one of its kind, regular applications of a fluoride-containing varnish may be considered a potentially effective approach, particularly in patients whose oral hygiene worsens during treatment.

Bonding Materials. Adding fluoride to the material that bonds the attachment to the tooth would seem to be an ideal means of delivering fluoride where it is needed. Unfortunately, fluoride released from these materials might not be sustained; they tend to show high levels of fluoride release initially, before the concentration quite dramatically drops (Fig. 30-10).[63] Some fluoride materials, such as glass ionomer cement, have the capacity to recharge, that is, to absorb fluoride from their

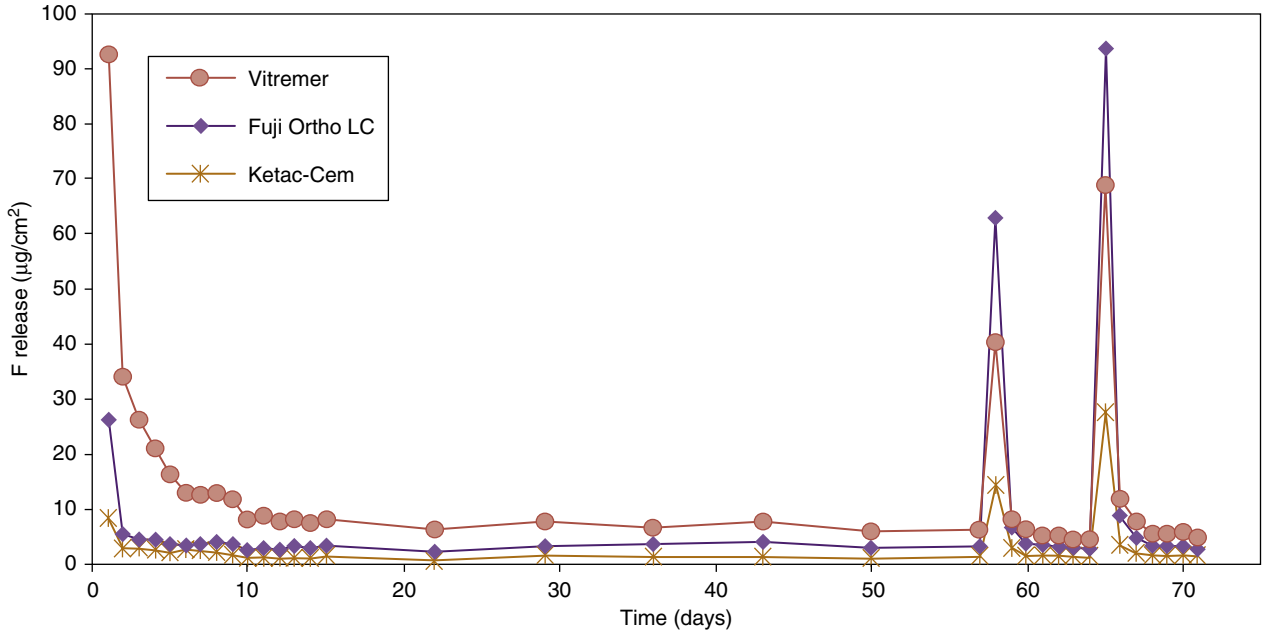


FIGURE 30-11 Graph shows the recharge of fluoride-containing materials when placed in 1.23% sodium fluoride gel for 4 minutes to simulate topical fluoride application. (Reproduced with permission from Lin YC, Lai YL, Chen WT, et al. Kinetics of fluoride release from and reuptake by orthodontic cements. *Am J Orthod Dentofacial Orthop* 2008;133:427–434.)

surroundings, as well as release it (Fig. 30-11)[64]; however, it is not entirely clear whether the fluoride released will be at a sufficient level over an adequate time (full length of orthodontic treatment) to prevent DWLs. The results of adding fluoride to composite materials have generally been disappointing.[65–68] Conventional glass ionomer cements showed promise in preventing DWLs,[69,70] but they are significantly weaker than composite materials, and the proportion of bond failures is high.[71] Newer resin-modified glass ionomer cements are stronger, but little reliable clinical evidence suggests that their use for bonding brackets prevents DWLs over the full course of treatment. Compomer, which is a hybrid between conventional resin-based and glass ionomer cement showed some initial promise,[72] but little research has been undertaken with the material since.

Elastics. In addition to placing fluoride in the orthodontic bonding material, adding fluoride to the elastomeric ligatures or elastic chain placed over the brackets would also seem logical. Fluoride would then be delivered to where it is most needed, and elastics are regularly changed, leading to a fresh boost of fluoride levels. Initial results with elastomeric ligatures were promising[73,74]; however, incorporating fluoride into the elastic changed the physical properties of the elastics, which rapidly deteriorated in the mouth (Fig. 30-12); consequently, finding any fluoridated elastomeric ligatures or elastic chain on the market today is very difficult.

Slow-Release Devices. Two primary types of devices are designed to provide slow and sustained release of fluoride in the mouth—copolymer membrane and glass beads.[75] One study reported the use of a copolymer membrane device,[76] and one reported the use of glass beads,[77] both in orthodontic patients (Fig. 30-13). Both reports may be considered at high risk of bias. Limited evidence proposes that slow-release devices are effective in the general population[78]; however, these devices could potentially provide sustained release of fluoride, and further research into their use for orthodontic patients might prove fruitful.

FIGURE 30-12 The fluoridated elastomeric ligature on the upper right lateral incisor has considerably deteriorated, compared with the conventional ligatures after 6 weeks in the mouth. The fluoridated ligature on the upper right central incisor has been lost. (Reproduced with permission from Benson PE, Douglas CW, Martin MV. Fluoridated elastomers: effect on the microbiology of plaque. *Am J Orthod Dentofacial Orthop* 2004;126:325–330.)

Diet. Limited evidence submits that fluoride in the diet or added to foods, such as milk or salt, is effective at preventing DWLs in orthodontic patients or in the general population.[79,80]

Unfortunately, at the present time, insufficient reliable evidence exists that recommends the most effective method of delivering fluoride to the orthodontic patient to prevent demineralization.[58] Hopefully, this lack of reliable evidence will be remedied in the future through the results of properly

FIGURE 30-13 Fluoride-containing glass bead on an orthodontic archwire. (Photograph supplied by Professor Jack Toumba and reproduced with permission. From Benson PE. Prevention of demineralization during orthodontic treatment with fluoride-containing materials or casein phosphopeptide–amorphous calcium phosphate. In: Huang GJ, Richmond S, Vig KWL, eds. *Evidence-Based Orthodontics*. Oxford: Wiley-Blackwell; 2011:149–165.)

designed clinical trials carried out over the full course of orthodontic treatment.[81] The current view of some cariologists is that supplemental topical fluoride alone might not be sufficient to prevent dental caries completely and that other ways need to be explored.[26]

Increasing the availability of calcium and phosphate. Ample evidence states that fluoride promotes the remineralization of enamel; however, this process also requires the presence of calcium and phosphate ions. Fluorapatite (chemical formula $Ca_{10}[PO_4]_6F_2$) contains three times more phosphate and five times more calcium than fluoride ions; therefore a relative deficiency of calcium and phosphate may delay or stop remineralization altogether.[82] To remedy this, a number of compounds containing calcium and phosphate have been proposed, the most thoroughly investigated being casein phosphopeptide–amorphous calcium phosphate (CPP-ACP).

Casein Phosphopeptide–Amorphous Calcium Phosphate. CPP-ACP has been considered an anticariogenic agent for over 20 years.[82,83] Previous clinical use of calcium and phosphate ions to enhance remineralization had not been a success because of the low solubility of calcium and phosphates, especially in the presence of fluoride ions.[83] CPP-ACP is a substance based on a group of peptides, casein phosphopeptides (CPPs), which have been shown to stabilize calcium and phosphate as nanoclusters of ions, in a metastable solution, known as amorphous calcium phosphate (ACP).[84] The CPP-ACP nanocomplexes can be used to intraorally deliver high concentrations of bioavailable calcium and phosphate ions to prevent demineralization and to allow a greater potential for remineralization.[85] CPP-ACP has been shown to aid more rapid remineralization of enamel subsurface lesions and can stabilize 100 times more calcium phosphate than neutral pH solutions.[86] It inhibits caries activity in a dose-response manner.[84,87] In addition, the incorporation of CPP into salivary pellicles reduces the adherence of cariogenic streptococcal species.[88] Anticariogenic activity has also been attributed to the ability of CCP to localize ACP at the tooth surface and, as such, leads to increased calcium phosphate levels in plaque.[86]

Reynolds and colleagues[84] showed that the use of a 3% CPP-ACP mouthwash increased the calcium content of plaque. This increase led to the idea that CPP-ACP would act as a reservoir in plaque for buffering free calcium and phosphate ion activities, maintaining a supersaturated state with respect to enamel. CPP-ACP has been shown to reduce caries activity in human in situ studies,[83,84,89–91] although the results of some trials suggest that the addition of CPP-ACP does not confer any additional benefits to that of fluoride toothpaste.[92,93]

A few studies have examined the effectiveness of CPP-ACP for the prevention of DWLs in orthodontic patients. Robertson and colleagues[94] compared nightly tray applications of a cream containing CPP-ACP and 900 ppm fluoride-containing cream with a placebo cream applied after brushing with toothpaste. They found that participants using the CPP-ACP and fluoride-containing cream after toothbrushing had fewer lesions forming and more lesions regressing (54% reduction in demineralization), compared with those using the placebo product; however, because of limitations in the study methodology, including this study in the latest Cochrane systematic review[58] was not possible.

A laboratory study examined the effect of incorporating CPP-ACP into a glass ionomer cement[95]; however, no clinical trials of this product have been undertaken.

Increase salivary flow or increase the pH of saliva

Chewing Gum. The use of sugar-free chewing gum has been suggested as a way of controlling caries in the general population.[96] Chewing gum stimulates the flow of saliva, increases its bicarbonate content and, consequently, increases the alkalinity of saliva, and has been shown to reduce both the amount of plaque in orthodontic patients.[97] Chewing mastic gum reduces the number of cariogenic bacteria in patients wearing fixed appliances[98]; however, adding chlorhexidine to gum did not significantly decrease plaque levels further, compared with gum without chlorhexidine.[97]

The addition of CCP-ACP into chewing gum has been shown to increase the amount of remineralization in non-orthodontic patients,[99] and a large randomized, controlled caries clinical trial found that CPP-ACP containing sugar-free chewing gum significantly slowed the progression of caries and enhanced the regression of caries, compared with the control subjects with sugar-free gum.[100] However, this finding has yet to be tested in patients wearing orthodontic appliances.

Treatment of Demineralized White Lesions

The aim of all orthodontists, with the help of their patients and parents, should be to attempt to prevent DWLs from appearing during orthodontic treatment; however, unfortunately, DWLs are still a common sight when the appliance is removed. It has been shown that regression of early DWLs can occur in the presence of saliva, without the additional use of fluoride or other remineralizing agents, once appliances have been removed,[101] but overall regression can be slow and limited.[3] Arends and Christoffersen have shown that even if remineralization of lesions does occur, the white marks may permanently remain.[102] As with the prevention of DWLs, a paucity of evidence suggests the best method of managing DWLs once the appliance is removed.[103]

Fluoride

As previously stated, ample evidence from numerous laboratory studies proposes that fluoride promotes remineralization; however, limited evidence is available regarding the concentration and frequency of fluoride that is most effective at reducing the visibility and the need for restorative treatment in patients with DWLs after their orthodontic appliance has been removed.

Du and colleagues[104] showed a greater reduction in readings, using a handheld laser caries detection aid (DIAGNOdent), when fluoride varnish was applied to DWLs post orthodontic treatment, as compared with a saline placebo; however, how the DIAGNOdent readings relate to the actual appearance of DWLs is not clear. Concern has been expressed about applying a high concentration of fluoride to a DWL, which might lead to hypermineralization of the surface and the persistence of a visible subsurface white lesion.[9,105]

Willmot undertook a double-blind, parallel groups, randomized controlled trial comparing a low fluoride (50 ppm) mouth rinse and toothpaste combination and a nonfluoride mouth rinse and toothpaste combination.[106] He found that, over 6 months, an average reduction of 57% in the size of the DWLs was reported, but no difference between the two groups was recorded. Baeshen and colleagues[107] found a significant reduction in both DIAGNOdent readings and the International Caries Detection and Assessment System scores when patients, with a minimum of four DWLs after orthodontic treatment, were asked to chew sticks (Miswaks) containing fluoride up to five times daily. There was also a reduction in the DWLs in patients who were given nonfluoridated sticks, but the reduction was not as large.

Casein Phosphopeptide–Amorphous Calcium Phosphate

In theory, the use of CPP-ACP in addition to fluoride should enhance the remineralization process, as CPP-ACP interacts with fluoride ions to produce nanoclusters of calcium, phosphate, and fluoride.[83] This CPP-ACP and fluoride complex thereby delivers fluoride, not only to the surface of the lesion but also to the subsurface, promoting remineralization. This complex also provides a source of soluble calcium, phosphate, and fluoride that is more resistant to pH changes. Reynolds and colleagues found that a mouth rinse with 2% CPP-ACP and 450 ppm fluoride significantly increased supragingival plaque fluoride content.[83] They also found that toothpaste containing 2% CPP-ACP and 1100 ppm fluoride was 2.6 times more effective than toothpaste with only 1100 ppm fluoride in remineralization of enamel subsurface lesions in situ.

The evidence for the effectiveness of CPP-ACP applications on the reduction of DWLs once orthodontic appliances have been removed is equivocal. Two studies found a positive effect of using a CPP-ACP containing cream, in addition to fluoride,[108,109] and three studies found a limited effect[110–112]; therefore further work in this area is required before a definitive answer is found.

Resin Infiltration

A new method of caries infiltration for early DWLs that has been suggested[113] involves etching the area with a 15% hydrochloric (HCl) acid gel and drying it with ethanol before placing a low-viscosity light-cured resin. Although the short-term results in patients with postorthodontic DWLs are promising,[114,115] no long-term data concerning staining or discoloration of the resin are available. Remineralization with calcium and phosphate must remain the ideal method of managing postorthodontic DWLs.

The development of unsightly white and/or brown demineralized lesions on the teeth during fixed orthodontic treatment remains a significant problem. Various approaches of preventing and treating DWLs during and after orthodontic treatment are available. Identifying the most effective and efficient of these approaches is the current challenge.

ROOT RESORPTION†

ETIOLOGIC FACTORS

The etiologic causes and origins of root resorption as a result of orthodontic forces are multifactorial and presently not clearly understood. Paradoxically, root resorption is common, but clinically significant root resorption is rare. Resorption of the root can occur any time an injury occurs, causing inflammation to the periodontal ligament (PDL) and/or to the pulp. Classification of root resorption is generally divided into internal resorption and external resorption. There are two types of internal resorption: (1) internal inflammatory resorption and (2) internal replacement resorption. These occur secondary to an insult to the dental pulp and are not related to orthodontic tooth movement. External resorption is classified into four categories: (1) surface resorption, (2) external inflammatory root resorption, (3) replacement resorption, and (4) ankylosis. Surface resorption is the physiologic process of resorption and repair that the root sustains during normal physiologic activity (e.g., mastication). External inflammatory root resorption includes any resorption mediated by the inflammatory process and includes resorption caused by orthodontic tooth movement and trauma, among others. This type of resorption can occur anywhere on the root resurface where there is periodontal attachment. Of particular interest to the orthodontist is the occurrence of external apical root resorption.

In normal orthodontic tooth movement, external root resorption is a naturally occurring side effect of the physiologic process of resorption and deposition as the bone remodels to accommodate the moving tooth caused by a cascading series of events initiated by pressure and tension in the PDL. On the pressure side of the root, as clastic cells are recruited during the initial inflammatory process, cementoclasts remove cementum, which is normal and has been shown to occur without exception. Along the side of the root, the cementum is repaired as soon as the force expression diminishes and cementoblasts replace the cementoclasts. This ongoing resorption-repair process along the sides of the root generally occurs without consequence to the health and longevity of the tooth. Lighter forces have been shown to produce fewer craters on the root surface than have heavier forces.[116] For reasons that are not completely understood, the resorption–repair process is different at the apex. Once resorption starts at or near the apex, it does not always repair. The role of exposed dentin or the possible involvement of the pulp through the apices may be a factor. The apex, itself, is complex with multiple foramina and complicated surface anatomy. The composition of cementum is variable near the apex. Stresses on the neurovascular bundle exiting the pulp may be involved. Distinguishing between the two forms (location) of external root resorption is important; what may be true in the former may not necessarily be so for the latter. To differentiate the unique type of resorption that takes place at the root apex, the term *external apical root resorption* (EARR) is the most appropriate appellation.

† Glenn Sameshima and M. Ali Darendeliler.

The complex nature of EARR probably precludes the identification of all patients at high risk as there will inevitably be cases of unknown causes, likely attributable to random events in the physiologic functioning of the individual. However, an individual's genetic predisposition is generally accepted as the primary cause of EARR. Figure 30-14 illustrates the percentage of EARR by cause, based on current research. The percentage attributed to biomechanical factors has become smaller as more studies have emerged. Studies of families with severe root resorption have established a firm genetic component implicating the interleukin gene family (see Chapter 2 for details).[117–119] In a twin study evaluating root resorption after full orthodontic treatment, an overall hereditary estimate of 0.34 was obtained.[120] Several candidate genes have been proposed, with the most likely scenario being that more than one gene will be implicated, and secondary or tertiary control of gene expression is undoubtedly involved as well. What genetic predisposition means to the clinician is that if a patient had significant EARR at the end of orthodontic treatment, then the risk for EARR is greater than usual when treating siblings. In addition, if a parent had severe EARR, then the orthodontist must more carefully look at the children and perhaps even the grandchildren of the patient as higher risk as well.

In summary, the physiologic cause is understood, but why it occurs more readily in some patients and not others is not known. Similar to most disease states with complex symptomatologic characteristics but little morbidity and mortality, EARR is epigenetic in origin with layered causes that will remain elusive for a considerable time to come.

RISK FACTORS

How does the clinician prevent EARR from occurring? Radiographically detectable root resorption is common. In the literature, the vast majority of teeth undergoing orthodontic tooth movement have been shown to have measurable EARR.[121,122] The crucial question, then, is how can *severe* root resorption be prevented? The orthodontic literature is replete with EARR investigations. The following paragraphs summarize the findings to give the clinician some insight into the risk factors.

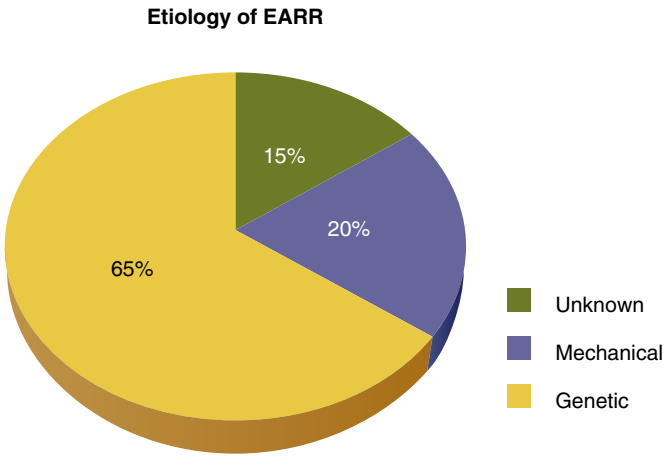


FIGURE 30-14 Etiology of external apical root resorption (EARR). The percent explained by genetics has significantly increased in the past decade, whereas the percent explained by treatment factors has proportionally decreased. (Adapted from Dr. James Hartsfield.)

Diagnostic Factors

Retrospective studies with large sample sizes have shown that the maxillary incisors have the greatest amount of root resorption of all teeth,[122–129] and lateral incisors are more resorbed than central incisors. The reported mean (average) amounts for all cases were approximately 1.2 to 1.6 mm.[122] Maxillary and mandibular canines follow in mean amounts of EARR with the rest of the dentition having less, but the clinician will see isolated teeth, particularly mandibular first bicuspids, with significant EARR.[122] Molars rarely resorb, but if they do, it is usually the mesial root(s). No studies explain why maxillary lateral incisors have more EARR, but several factors have been given serious consideration. Brin and others have shown that lateral incisors often have undetected EARR caused by erupting canines.[130,131] (The canine guidance theory of eruption proposed by Ericson and Kurol in 1988 supports these findings.[132]) Maxillary lateral incisors have the highest frequency of dilacerations of any tooth and also have a high frequency of microdont, peg shape, barrel shape, and being congenitally missing. Because of the variation in crown shape and size, the orthodontist must often adjust the position and torque of a maxillary lateral incisor more than any other tooth. Finally, maxillary incisors may have a composition of cementum that is different from the other teeth, and they are developmentally distinct early in origin as a unit. Interestingly, no difference in EARR among small or peg or barrel laterals has been found, compared with a normal antimere.[133]

Tooth Anatomy

Short teeth are at no higher risk than normal length teeth. Teeth with an odd root shape are probably at higher risk as shown by clinical and finite element models[122,134] (Figs. 30-15 and 30-16). Studies have shown that dilacerated roots, pointed roots, and pipette-shaped roots may have a higher risk for EARR.[122] The dilacerated portion of the root is often resorbed during active tooth movement. Using the two-dimensional representations that are routinely used (conventional radiographs) makes assessing the shape difficult. Although shape as a nominal variable in two dimensions is reproducible, recent three-dimensional studies have shown that dilacerations can occur perpendicular to the plane of the periapical, rendering the shape invisible in two-dimensional studies.[135] Figure 30-17 clearly demonstrates this with a root tip dilacerated completely in the palatal direction. Decalcified teeth and teeth with a high crown-to-root ratio are not at higher risk. Teeth with a history of trauma may be at risk, but teeth with prior resorption are not.[136,137] One study found that teeth with longer roots had greater EARR.[122]

Demographic Factors

For years, it was thought that women and teenagers were at greater risk, but the majority of studies show no difference in sex or age; most of the studies that did find a difference showed those of increasing age and men may be at higher risk.[122–129,138–142] Clinical experience and the known role genetics plays in the occurrence of EARR have led some to suspect ethnicity; one study found Hispanic patients to have more EARR than Caucasians or Asians.[122] In addition, some

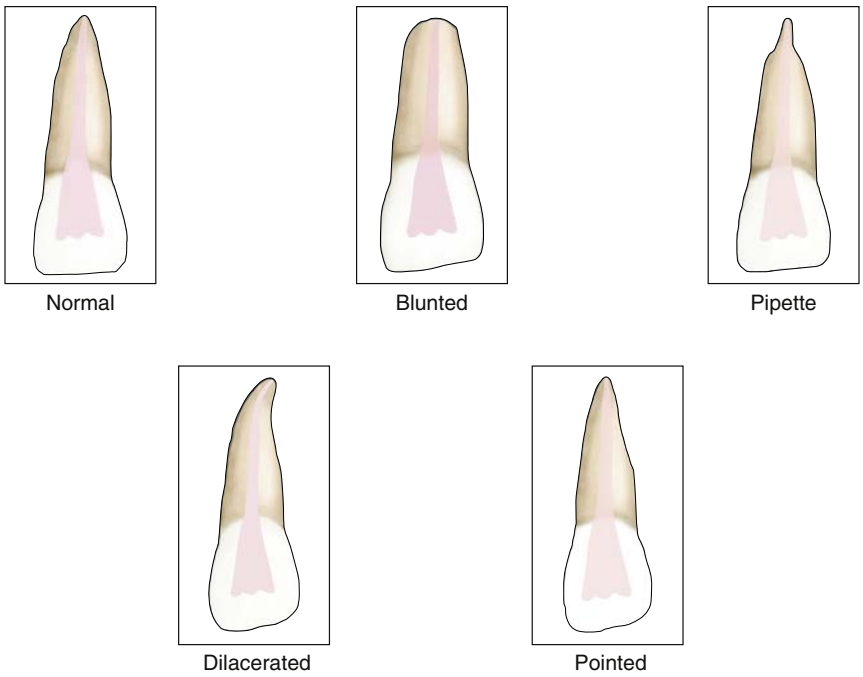


FIGURE 30-15 Classification of root shape. The five most commonly seen root shapes are visualized on periapical x-ray images. The pipette shape is almost unique to maxillary central incisors. Maxillary lateral incisors have, by far, the highest frequency of dilacerations. It is acknowledged that these classifications are fairly general and that all roots have an irregular surface and contour.

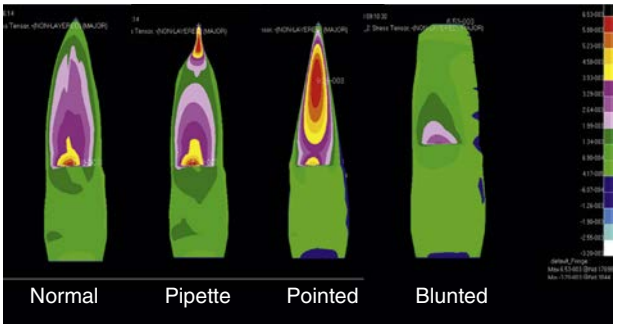


FIGURE 30-16 Finite element analysis of three root shapes. This figure shows the principal von Mises stresses from a pure linear tipping movement (areas of maximum stress on the root surface) of a static model of a maxillary central incisor. In the normal-shaped root, the stresses are concentrated at the cement–enamel junction at the alveolar crest. Blunted root shapes show a similar pattern. However, in the pipette-shaped and pointed-shaped roots, the stresses occur at the apical end and apical third, respectively, mirroring the increased external apical root resorption (EARR) clinically observed in these shapes.

evidence suggests that a significant variation exists among offices, even when many confounding variables are considered in the study.[122]

Malocclusion Factors

No evidence associates Angle classifications of malocclusion, cephalometric measurements, or the amount of crowding that may warrant extraction treatment with EARR.[122–129,138–142] The amount of apical displacement has been shown to be a primary factor causing EARR.[143–145] This, as well as a hypofunctioning PDL, makes anterior open bite cases more prone to EARR.[145]

Root Canal Treatment. Teeth that have been successfully treated endodontically were thought to be resistant to EARR; Sameshima and Sinclair found no EARR in any root-filled teeth in their study of 1000 patients,[122] as did two other studies.[147,148] Castro found no difference in EARR in posterior teeth between root-filled and vital teeth, but the amount of EARR in both groups was less than measurement error.[149] A systematic review[150] found no difference between root-filled and vital teeth but was very limited in validity and scope and included one study with a sample size of 16 and another using panoramic films for analysis. The latter is the only paper that found EARR in root-filled teeth.[139] Teeth with immature apices do not resorb, although the roots may not achieve full length if orthodontically moved[151,152] (Fig. 30-18).

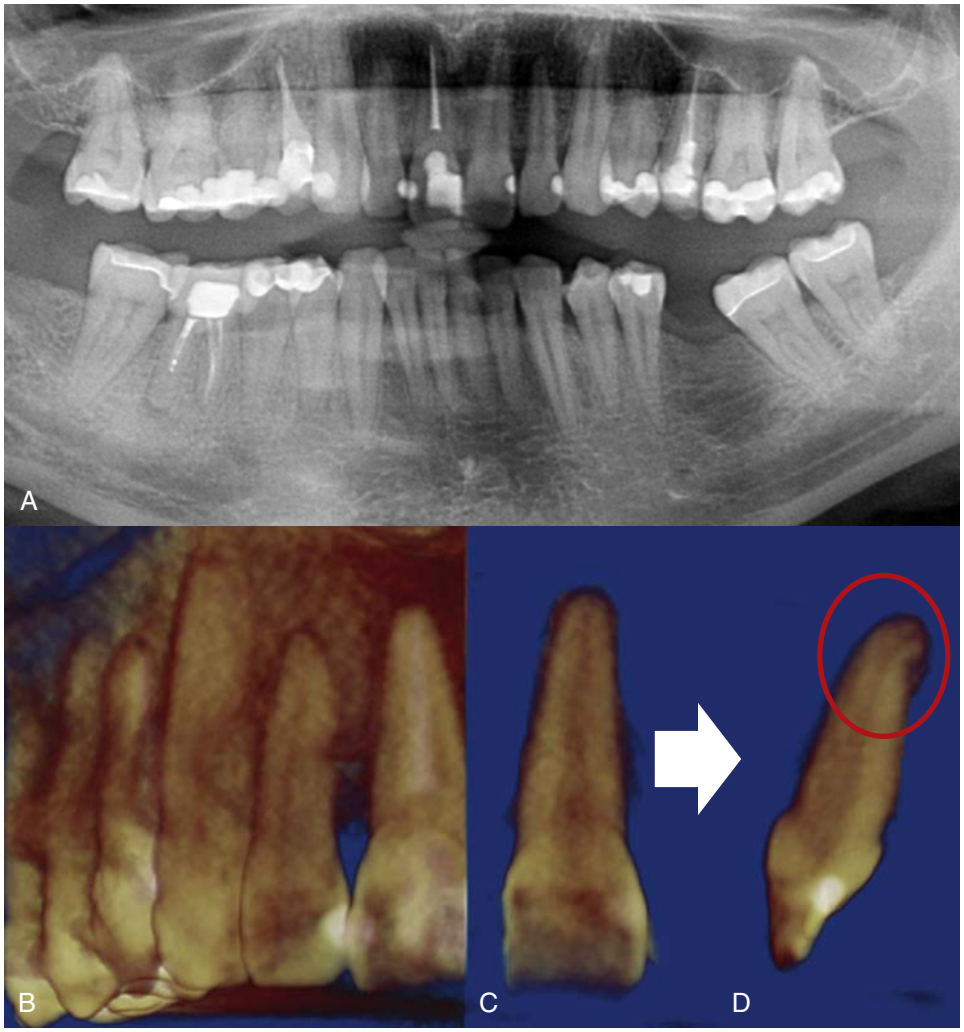


FIGURE 30-17 External apical root resorption (EARR) in 3D images. **A.** Initial PAN shows abnormal root shape for all four maxillary incisors. **B.** CBCT generated image processed with Dolphin 3D **C.** Segmented left central incisor (see arrow on PAN) from frontal and mesial views shows a palatal dilaceration that would not be visible on any conventional radiograph.

Patient Medical History and Habits

Primarily based on case reports, a few medical conditions, such as Turner syndrome, familial dysostosis (also known as familial expansile osteolysis),[153] and possibly any patient with severe and/or uncontrolled endocrine problems, are known to place patients at higher risk for EARR. Asthma has long been suspected of being a risk factor for EARR. The link with inflammation provides a sound theoretical basis, and three studies have found increased risk in patients with asthma.[154–156] Patients with allergies were found not to have higher prevalence in one study,[157] but they did have significantly higher risk factors for EARR in another study[154] with a proposed mechanism reported in an animal study by Murata.[158] Habits such as tongue thrusting, nail biting, or bruxing as independent risk factors for EARR have received much speculation but are supported by no clear evidence. Additionally, the clinician must be aware that documented cases of idiopathic root resorption (with no history of orthodontia) exist, sometimes involving multiple teeth and severe enough that teeth are eventually lost.[159–162]

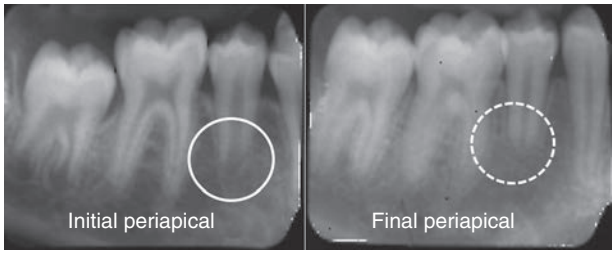


FIGURE 30-18 Movement of teeth with immature apices: initial periapical radiograph (*left*) and final periapical radiograph of mandibular bicuspids (*right*). Note no shortening of the root occurred, but, based on other teeth in the same state of development, the tooth did not achieve its predicted final length. Generally, however, immature root apices do not resorb.

Treatment Factors

Mechanical Factors

Treatment philosophy, bracket type and slot size, archwire composition or sequence, and use of headgear, among others, have not been found to be significant risk factors.[142,163,164] In particular, the claim that moving teeth with self-ligating brackets cause less EARR has been proven to be false.[165] Among clinicians, a nagging suspicion remains that inconsistent use of elastics by the patient can be a contributing factor, especially if carried out over a long period, although evidence is fleeting.

Recent case reports show that clear aligner treatment that applies enough force to move the roots the same amount as fixed appliances do will cause similar EARR.[166–168] Limited evidence supports the fact that orthodontic patients treated with periodontally accelerated osteogenic orthodontics do not have an increased risk for EARR.[169,170]

Magnitude of Applied Force

Both animal and human studies have provided strong evidence that force magnitude is directly proportional to the severity of root resorption.[116,171–174] Given the choice between heavy and light forces applied at the bracket–archwire interface, the clinical choice is light forces, although the distinction between light versus heavy forces in clinical practice is not a simple one to make. Indeed, although considerable evidence suggests that light forces cause less resorption along the root surface, there is no evidence that light forces directly translate to resorption, which occurs at the root apex.

Intermittent versus Continuous Force

A pause in tooth movement allows the resorbed cementum to heal. It follows that most of the studies comparing intermittent with continuous forces found that the latter is associated with greater resorption.[175–178] However, the amount of movement using intermittent forces is less than the amount produced by continuous forces.

Early Treatment

Brin and Bollen found that the spontaneous unraveling of incisor crowding often observed with serial extraction during early treatment does not prevent EARR in patients treated with late extractions, when the patients are treated in phase II.[179] If an advantage to early treatment exists, relative to EARR, it is that immature apices are not as susceptible (see above).

Expansion

An observational study using CBCT shows that rapid maxillary expansion (RME) or other similar fixed devices may increase the risk of EARR. Significant volume lost, linear surface area changes, and thinning and/or shortening of maxillary first molar and premolar roots were common findings with the use of tooth-borne RME therapy.[180,181] When slow and rapid expansion therapies were compared, no difference in the amount of EARR was found.

Extractions

The extraction of teeth has been found to increase risk.[182,183] The risk is the same whether the first bicuspid, second bicuspids, or upper bicuspids only are extracted.[122] For upper bicuspids only, it is a covariant with horizontal apical displacement and overjet correction.[122]

Prolonged Treatment Time

In theory, treatment that takes longer than usual has the potential for wearing down the system, for lack of a better description. There are likely limits to the amount of remodeling cycles the root apex can withstand. The model of EARR proposed by Al Qasami supports this theory.[118] The majority of studies that have measured an association between treatment duration and EARR have found a positive one.[122–129] The longer the active treatment time, the greater the amount of EARR,[182,183] which may also be related to root (apical) displacement (see the next paragraph). This theory would also partially explain why round-tripping of teeth produces more EARR, and jiggling in inconsistent wear of finishing elastics is thought to increase risk. Recently, the effects of 4 weeks of jiggling movement were studied; the conclusion was that short-term buccopalatal light and heavy jiggling movements do not increase the amount of EARR.[184] However, vertical jiggling movements, in comparison with buccopalatal jiggling movements, were shown to increase the instances of EARR.[185]

Apical Root Displacement

Most studies have found the distance the apex is displaced is a significant risk factor, but not all studies agree. Until recently, accurately measuring the actual displacement of the tooth has been difficult; serial cephalometric tracings superimposed on the maxilla were used to measure horizontal and vertical displacement of the apex quantitatively (Fig. 30-19). One of the many limitations of this method of measurement is the uncertainty in knowing which central incisor is traced on each film. Imminent studies with three-dimensional superimposition may provide more accurate answers and will show true displacement in all three planes of space. Moving the apex of maxillary central incisors against the cortical bone was shown in one study using cephalometric films to increase EARR, but it has been difficult to replicate the study,[127] and ongoing studies with stereolithography (STL) files may provide more solid evidence. Anecdotally, when practitioners used to rotate and torque the maxillary first molars (mesial buccal root) against the cortical bone for anchorage, EARR of the mesiobuccal root was often observed.

The fact that apical displacement is a risk factor to consider is also evident in the heroic distances that a tooth can now be moved using skeletal temporary anchorage devices (TADs). The orthodontist must take frequent progress films whenever moving teeth a long distance to correct overjet or camouflage malocclusion by using TADs to avoid extractions[186] (Fig. 30-20). Pure or absolute intrusion of teeth is now possible using TADs. Figure 30-21, *A*, *B*, demonstrate cases where absolute intrusion of molars with TADs resulted in EARR. The clinician will find that the risk of EARR significantly increases when absolute intrusion is performed.

Although no supporting evidence exists, a strong suspicion suggests that maxillary teeth in LeFort I osteotomies have a higher incidence of severe root resorption. Patients with controlled periodontal disease have no higher risk for EARR and periodontal bone loss, but attachment loss of 1 mm is considered as serious as 3 mm of EARR.[187] EARR results in a large crown-to-root ratio; but unless the tooth will support a load-bearing prosthesis, the longevity of the tooth is not affected. Figure 30-22, *A*, *B*, illustrates two typical cases of EARR of all four maxillary incisors found in a study of consecutively treated cases from a private practice.

A list of risk factors for EARR is shown in Box 30-1.

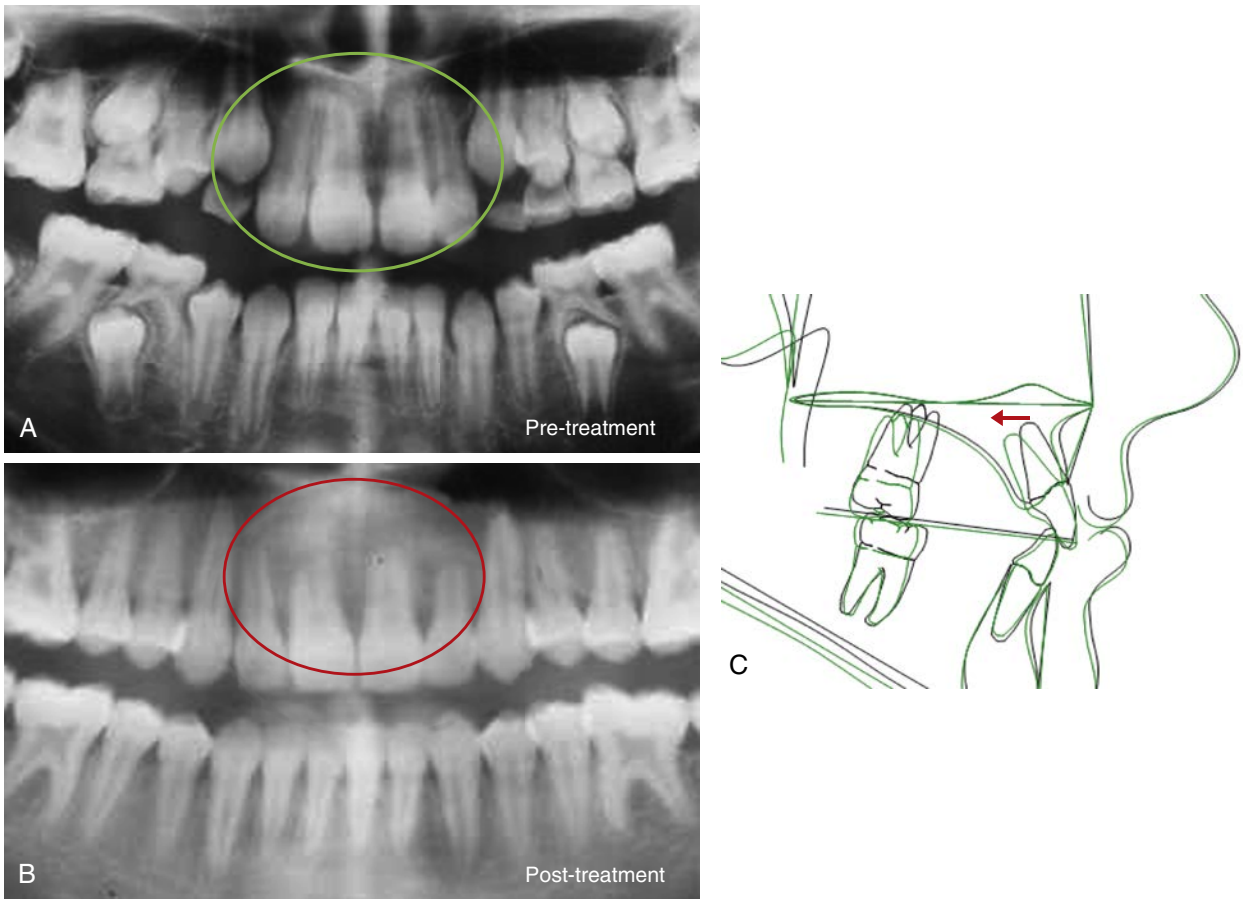


FIGURE 30-19 External apical root resorption (EARR) and horizontal root displacement. **A,** Pre-treatment. **B,** Post treatment showing significant EARR in all maxillary incisors (circled). **C,** Cephalometric superimposition shows amount of apical displacement (arrow).

BOX 30-1 Summary of Risk Factors

1. Siblings with external apical root resorption (EARR) from orthodontic treatment
2. Positive medical history for known conditions
3. Dilacerated, pipette, or pointed root shape
4. Long apical displacement
5. Long treatment time
6. Extractions
7. Open bite and deep bite
8. Excess overjet
9. Hispanic ethnicity

MANAGEMENT

Clinical management of EARR is summarized in Box 30-2.

Imaging

An initial periapical radiograph or limited-field CBCT is essential in adult patients to examine properly and with clarity the root morphologic structure and the location of the roots. Periapical radiographs have been shown to be superior to panoramic images for detecting EARR.[188] Apical displacement in all three planes of spaces often exceeds what can be observed in two-dimensional radiographs of the same area of the head. Limited-field CBCT with accurate software can now make details of the root apex and apical resorption quite visible.[189–191]

BOX 30-2 Clinical Management

1. Produce good pretreatment images.
2. If risk factors present, then document a special entry in the informed consent.
3. If risk factors present, take periapical radiographs at 6 and 12 months or when apical displacement has started.
4. During treatment:
   a. If external apical root resorption (EARR) is greater than 2 mm, then stop treatment for 4 months.
   b. If EARR is greater than 4 mm or more than one-third of the root, then stop active tooth movement and consider terminating treatment.
5. If severe EARR occurs on more than two adjacent teeth, the treatment must be terminated.
6. EARR stops when appliances are removed.
7. Patient and referring dentist must be kept informed at all time points.
8. If short roots are present at the beginning of treatment:
   a. Delay applying appliances on the affected tooth as long as possible.
   b. Avoid torque and apical displacement.
   c. Take more frequent periapical radiographs.

History

A careful history is essential. If a patient is at high risk, especially for families, then specific documentation of this level of risk must be included in the informed consent for treatment.

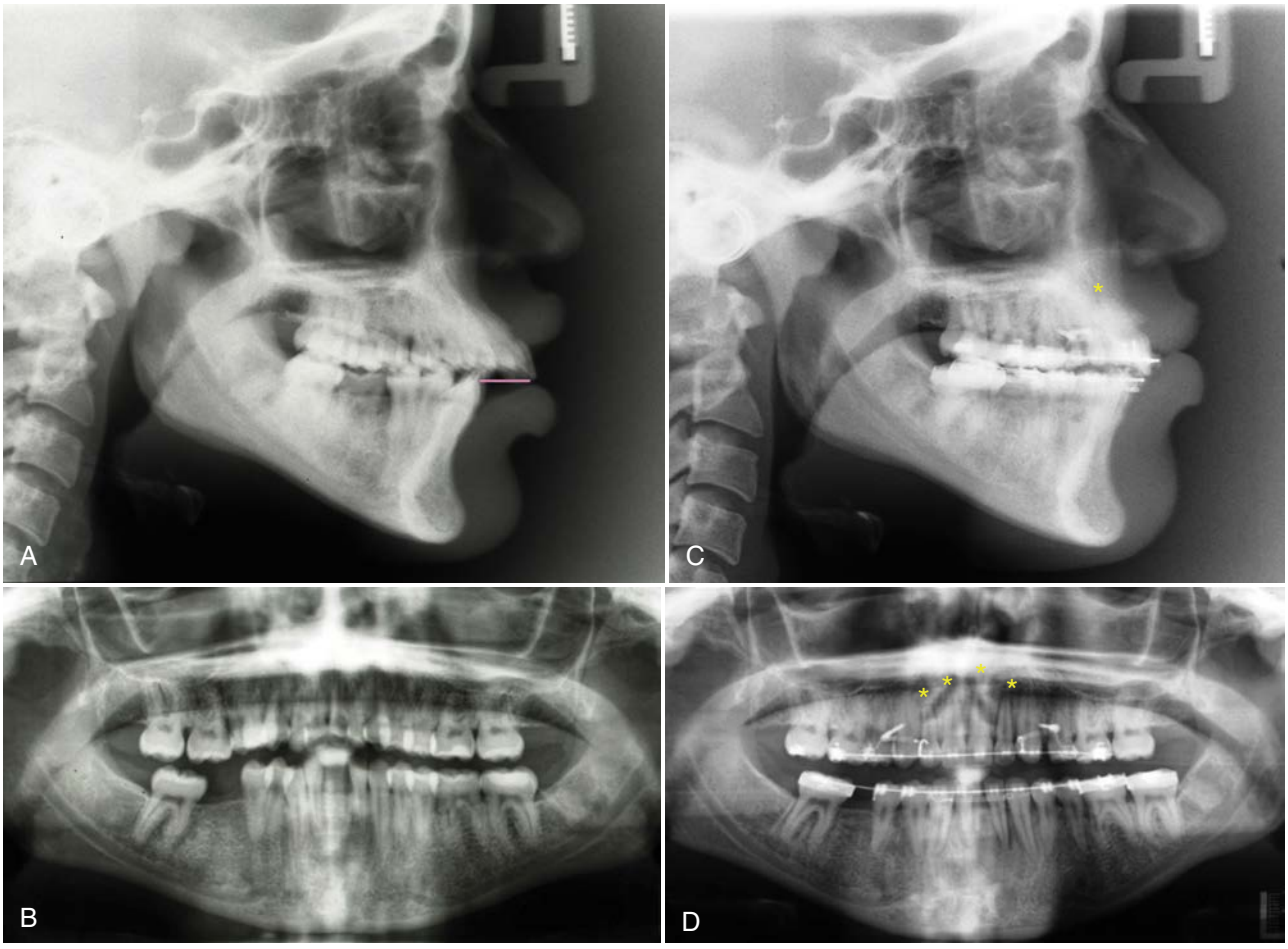


FIGURE 30-20 Extreme horizontal retraction of maxillary arch with temporary anchorage devices (TADs). Pre- (**A,B**) and progress (**C,D**) PAN and cephalometric radiographs. External apical root resorption (EARR) of approximately 25% was found on all four maxillary incisors after the apices were retracted over 5 mm over a 30-month period. Patient had no other risk factors. Case illustrates the distances possible and the caution needed with absolute skeletal anchorage.

Progress Review

Documenting the progress for all cases is a good practice, but documentation is particularly important if risk factors for EARR (and other potential issues such as periodontal problems) are present at the beginning of treatment. A commonly practiced time to take progress records seems to be 1 year into treatment. However, if a patient starts with short roots (approaching a crown-to-root ratio of one), then periapical radiographs should be taken sooner and more frequently. This need must be explained to the patient during the informed consent process before appliances are placed.

What to Do If Root Resorption Is Detected at Progress

The decision to continue active tooth movement is dependent on planned further movement of the tooth in question and the amount of resorption visible on the radiograph. It must be noted that there are no consensus standards on what constitutes severe EARR. A survey found that general dentists were of the opinion that 35% root shortening should terminate orthodontic treatment; orthodontists did not agree.[192] Generally for a normal length and nonperiodontally involved tooth, if the amount of resorption is greater than 2 mm, then the best course of action is to stop active treatment immediately and wait for 4 months. Ideally, the tooth should not be in hyperfunction and no force applied, which usually means placing a passive archwire to hold the teeth exactly where they are. After this resting period, treatment can continue (Fig. 30-23). No increased risk for the EARR has shown to resume, but overtorquing the tooth is unwise, and the orthodontist may have to compromise the amount of detailing as well. If the amount of EARR is 4 mm or more in a patient who has been in treatment for a long time and the apex has already been moved a significant distance (1 mm or more), then the orthodontist will have to decide whether to terminate treatment (deband the case) or modify the plan to finish without moving the affected teeth (Fig. 30-24). In both situations, the patient and his or her dentist must be informed of the change in plan.

When Does External Apical Root Resorption Start?

It has been hypothesized that EARR will start to occur when the root apex is displaced—in any direction. Artun found that teeth

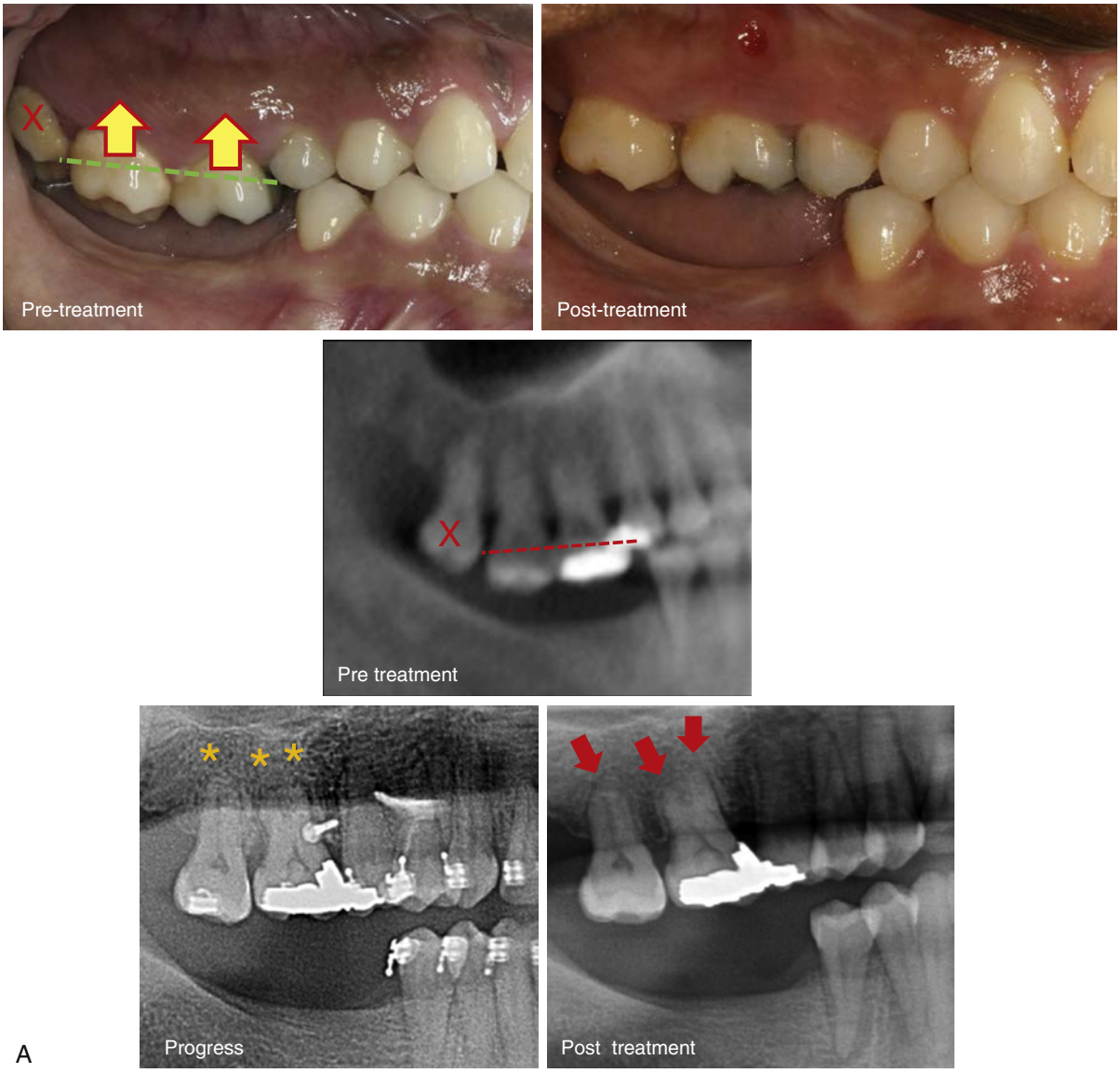


FIGURE 30-21 Absolute intrusion with temporary anchorage devices (TADs) for overerupted, unopposed maxillary molars is demonstrated. TADs were placed buccal and lingual to intrude the maxillary right second and first molars approximately 4 mm (*yellows arrows*). External apical root resorption (EARR) in the intruded molars was 1-2 mm at progress (asterisks) and 2-3 mm (*red arrows*) at completion of treatment.

with EARR at 6 months after fixed appliance placement were the most likely to have severe EARR by the end of treatment.[193]

When Does External Apical Root Resorption Stop?

It has been clinically observed that as soon as active forces are removed from the tooth, EARR stops. Studies have shown the reparative process (cementoblast activity) is completed within a few weeks. Generally, removable appliances do not cause EARR, and retainers with springs will not cause further resorption; however, tooth positioners may produce enough force to continue EARR (no evidence in the literature confirms either way).

What Happens to Teeth with Short Roots Long Term?

Teeth with congenitally extremely short roots do not spontaneously fall out unless periodontal disease remains untreated. The current faith in the permanency and longevity (forever) of implants in the dental world has become a call for the extraction of any compromised teeth (including teeth with EARR) and replacement with implants (Fig. 30-25). Case reports and long-term cohort studies have shown that no relationship exists between teeth with short roots and morbidity (loss of the tooth). Remington in 1989 recalled 100 patients with EARR after 14 years—no teeth had been compromised.[194] In 2002, Savage and Kokich presented long-term follow-up studies of three patients with severe EARR; the authors emphasized the need for a comprehensive, interdisciplinary approach to maintain the health of the afflicted teeth.[195] Lateral incisors with severe root resorption (not apical) from erupting canines can still be orthodontically moved; and even with a 20% increase in the crown-to-root

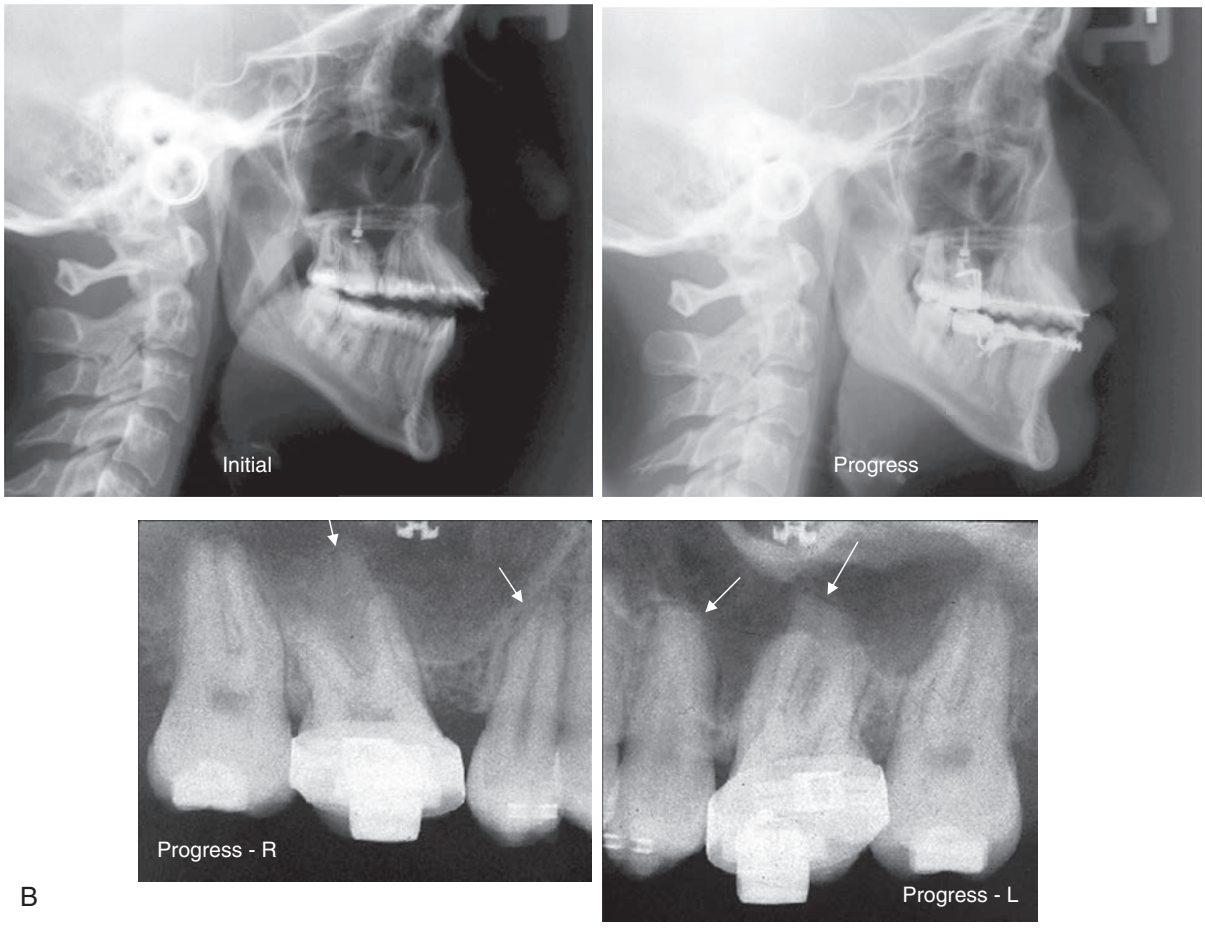


FIGURE 30-21, cont'd B, Molar intrusion to close anterior bite is demonstrated. EARR was detected on the bicuspids and first molars with possible impaction of apices with floor of maxillary sinus. Buccal segments intruded absolutely 2 to 4 mm. No EARR was found on anterior teeth. Case debanded soon after these in-progress periapical images were taken.

ratio, they are stable long term.[196] Jonsson and colleagues evaluated mobility in patients recalled 25 years later and found increased tooth mobility if the root length was less than 9 mm.[197] A single case of severe EARR of all four maxillary incisors treated with fixed appliances was stable 25 years later.[198] However, a prospective study following patients with and without short roots long term has not yet been published.

Finally, the orthodontist must also be aware of an uncommon form of root resorption called *invasive cervical root resorption* (ICRR). Heathersay[199] and others[200] describe ICRR as starting with an inflammatory process that penetrates the cementum from the PDL (thus it is external in origin) apical to the epithelial attachment. The lesion then resorbs the dentin and enamel, generally leaving the dentin surrounding the pulp intact but causing a hollowing of the tooth. The lesion is not visible early unless a radiograph happens to have the correct angulation of the lesion in cross section. Usually the first sign of ICRR is when the clinician notices a pink-appearing crown near the cement-enamel junction (CEJ). This pink hue is due to the granulation tissue filling in the resorbed dentin and enamel under the resorbed part of the crown. The tooth remains vital and asymptomatic. Unfortunately, by the time the patient or dentist notices the problem, enough destruction has occurred that heroic measures must be taken, with extraction often the outcome. The orthodontist must be aware of this; although no direct cause has been proven, orthodontic treatment has been identified as one of the most commonly associated factors with ICRR. Heathersay's classic paper identified eleven potential predisposing factors. Orthodontics was the most common sole factor (47 patients with 62 affected teeth).[199] Trauma was the second most frequent sole factor (31 patients with 39 affected teeth).[199] Other associated factors were bleaching, restorations, and combinations of factors; 33 cases were not associated with any predisposing factors.[199]

Are There Any Methods to Detect Root Resorption before It is Visible on Radiographs?

Many possibilities exists, but, practically speaking, the choices are limited. Methods to detect root resorption are available, but the ability to find markers has been promising and no practical methods have been developed for clinical use. Gingival crevicular fluid (GCF) is an intriguing possibility. Mah and Prasad compared levels of dentin phosphoproteins in the GCF among three groups.[201] They found significantly higher levels in resorbing primary teeth and teeth undergoing active

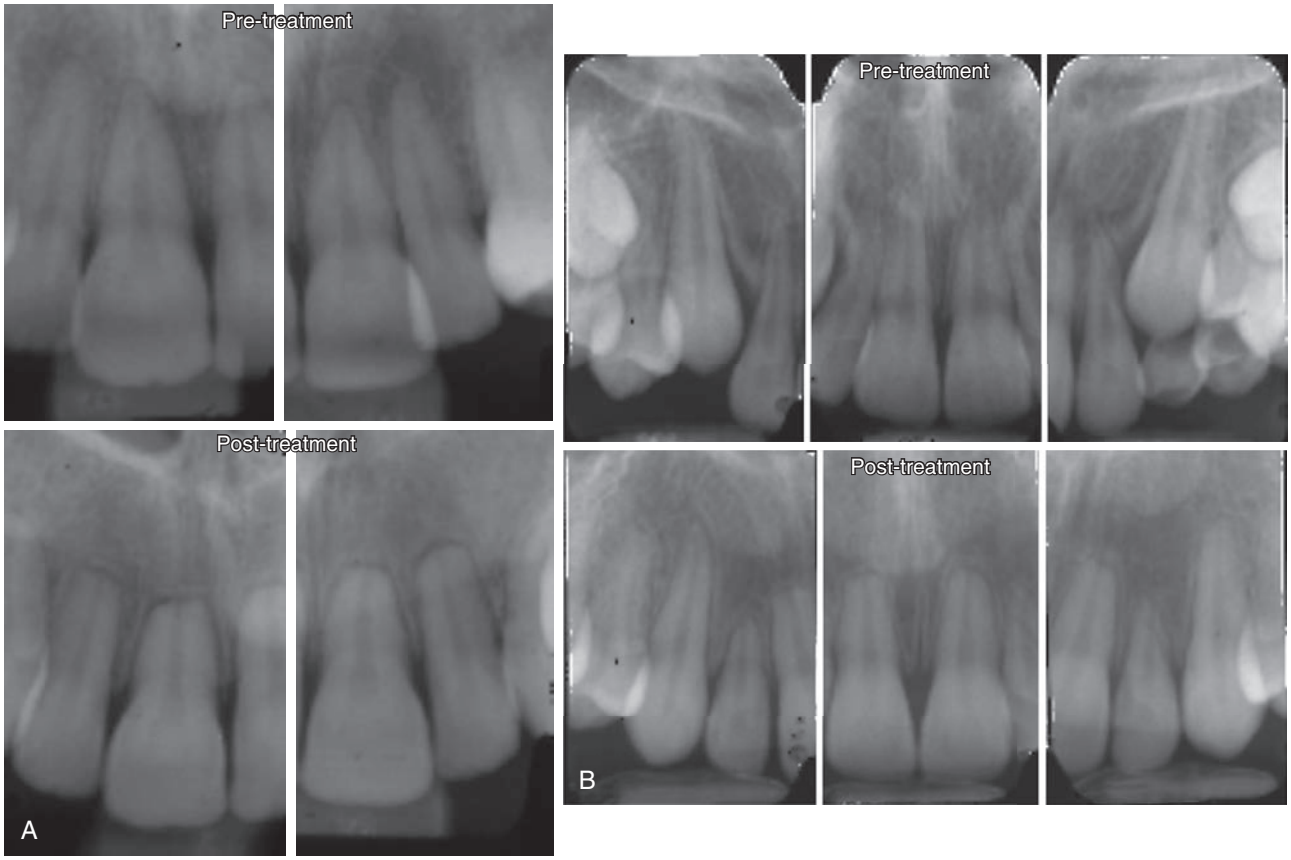


FIGURE 30-22 **A,** An extraction case shows severe root resorption of all four maxillary incisors. Pretreatment risk factors include an older sibling with moderate external apical root resorption (EARR). Female Caucasian, 12 years of age. 4-mm overjet. Normal overbite. Class II, Division 1. Extraction of four bicuspids. Pointed and dilacerated root shapes. Duration of treatment was 33 months. Root apex horizontally displaced 3.5 mm. **B,** A nonextraction case shows severe root resorption of all four maxillary incisors. Female Caucasian, 12 years of age. Negative health history for risk factors. Maxillary left lateral may have preexisting root damage from erupting canine. Nonextraction, deep bite with mild crowding. Extended treatment time. Root apex horizontally displaced 1.5 mm and vertically displaced 2.0 mm.

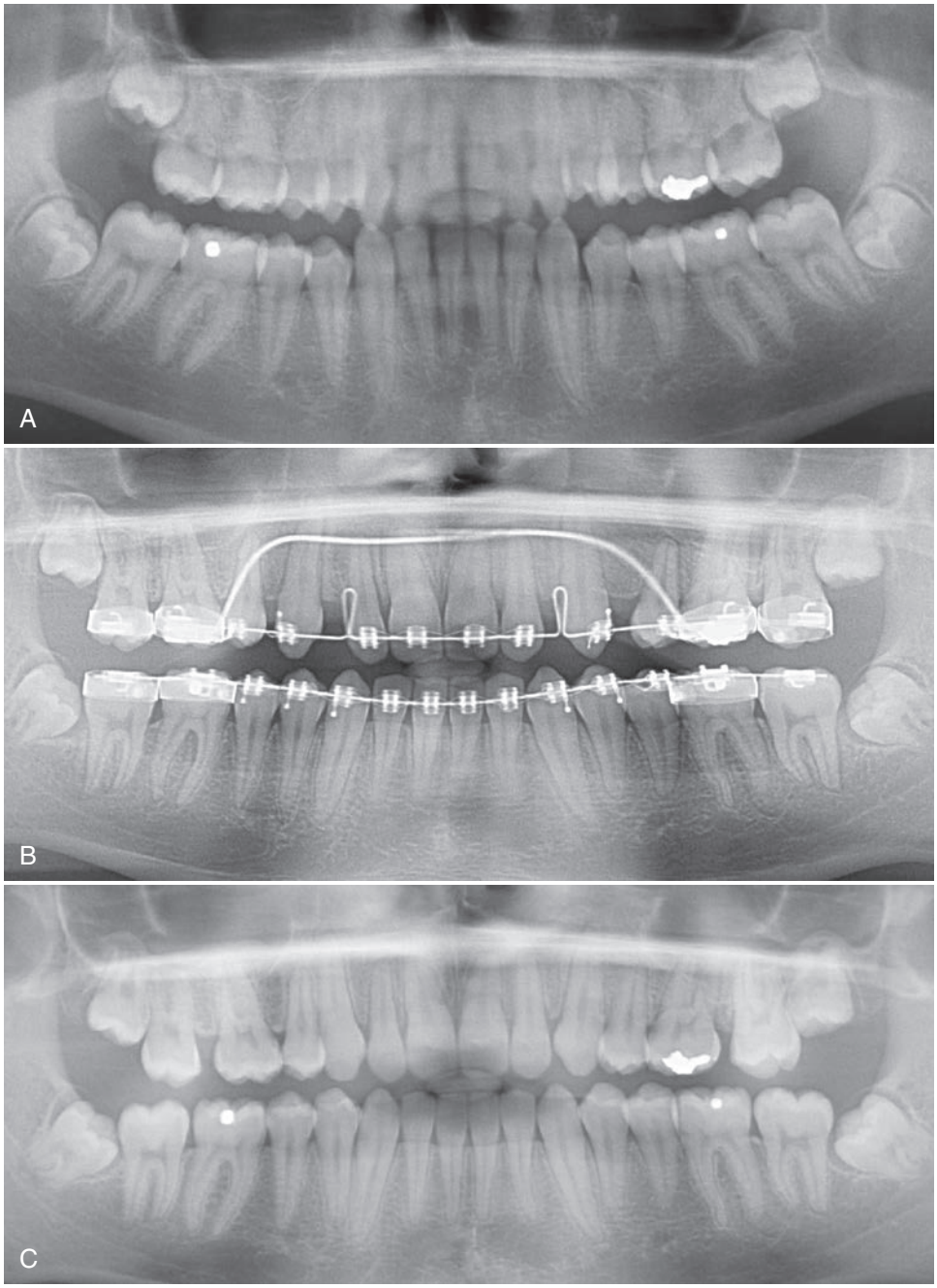


FIGURE 30-23 Management of external apical root resorption (EARR) at progress. When root resorption is found during treatment, the recommended course of action is to stop treatment temporarily. **A**, Initial panoramic x-ray image. **B,** Progress during retraction of incisors; note EARR on all four teeth. **C**, Final panoramic x-ray image. No further EARR is observed. Treatment was stopped for 6 months after EARR was found at progress.

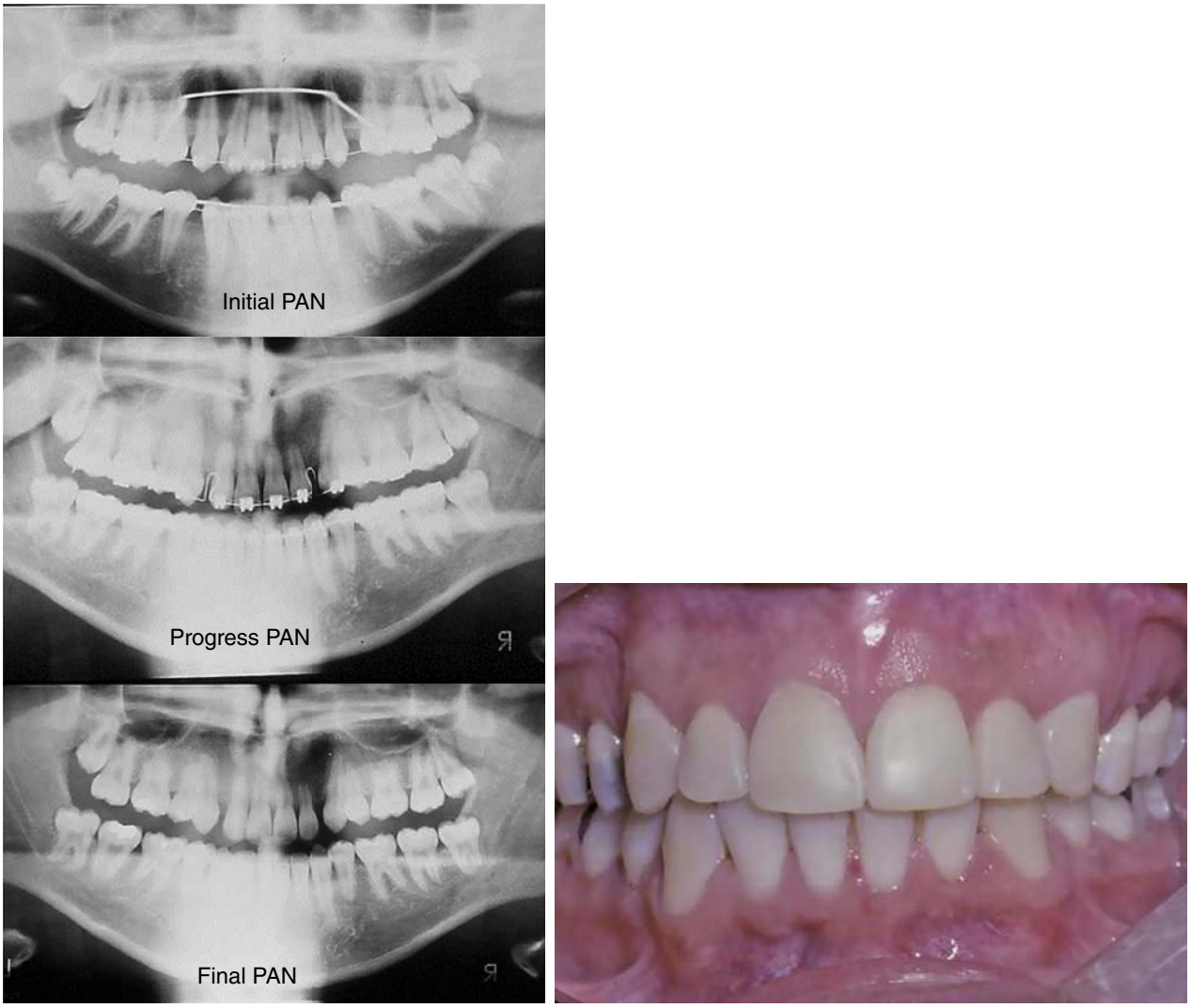


FIGURE 30-24 Management of severe external apical root resorption (EARR) after long treatment time. Initial (*left top*). After 3 years of treatment (*left center*) Final PAN (*left bottom*) and final photos (*right*)–(note restorations). By mutual agreement between the orthodontist and the patient, treatment was terminated; the patient did not want to stop treatment for 4 months then continue.

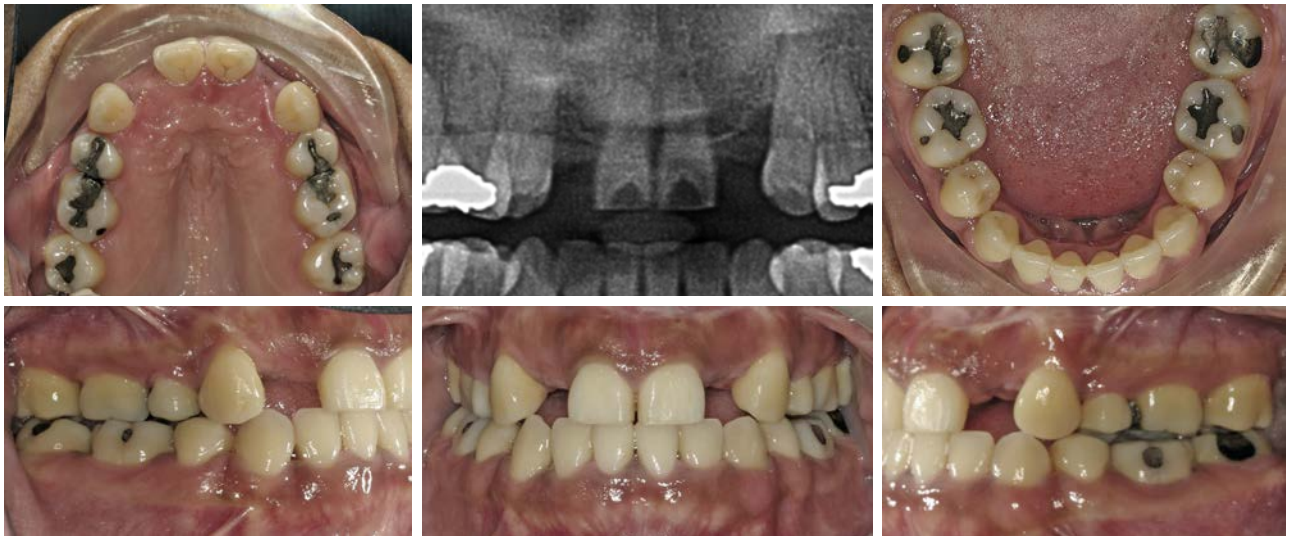


FIGURE 30-25 External apical root resorption (EARR) and implants visualized on posttreatment panoramic x-ray and intraoral images. Patient was seen by a prosthodontist who recommended extraction of the central incisors because "their prognosis was poor." Approximately one third of the root had been lost in 2 years of orthodontic treatment with fixed appliances. No mobility or periodontal problems were present. The patient eventually sought second and third opinions, and a restorative dentist replaced the missing maxillary lateral incisors without incident.

orthodontic tooth movement. Similar findings were reported by Balducci and colleagues.[202] George and Evans examined GCF levels of dentin phosphoproteins and other markers and found differences between teeth in patients with root resorption and a control group with no forces.[203]

There are genes (see previous discussion in this chapter and in Chapter 2) that identify patients at greater risk for EARR, but genetic material can only be obtained from blood or buccal swabs. However, a new method for detecting markers in saliva has been developed at the University of California, Los Angeles (UCLA). If reliable markers for either risk factors or active EARR can be established, then the technology exists to detect them in saliva, which is easier to collect.[204] An interesting study by Ramos showed that levels of a specific immunoglobulin are elevated during pretreatment in patients who subsequently had severe root resorption; someday, this could be another way to test for risk if elevated levels of a specific immunoglobulin can be detected in saliva.[205]

EARR is paradoxical; it occurs in nearly every tooth but is a benign side effect. Severe EARR is rare but can be destructive and affect more than one tooth. The primary risk factors supported by the literature and clinical observation are presented (see Box 30-1). Proper management of EARR should include an assessment of risk factors, taking quality images, and following established procedures if severe EARR is detected during treatment (see Box 30-2). Finally, it must be emphasized that the mere fact of a short root is not cause for the extraction of the tooth and replacement with an implant.

REFERENCES

References for Prevention and Management of Demineralized White Lesions

1. Fejerskov O, Nyvad B, Kidd EAM. *Dental Caries: The Disease and its Clinical Management*. 3rd ed. Chichester: John Wiley & Sons; 2015.
2. Angmar-Månsson B, ten Bosch JJ. Optical methods for the detection and quantification of caries. *Adv Dent Res*. 1987;1:14–20.
3. van der Veen MH, Mattousch T, Boersma JG. Longitudinal development of caries lesions after orthodontic treatment evaluated by quantitative light-induced fluorescence. *Am J Orthod Dentofacial Orthop*. 2007;131:223–228.
4. Rosenbloom RG, Tinanoff N. Salivary *Streptococcus mutans* levels in patients before, during, and after orthodontic treatment. *Am J Orthod Dentofacial Orthop*. 1991;100:35–37.
5. Naranjo AA, Triviño ML, Jaramillo A, et al. Changes in the subgingival microbiota and periodontal parameters before and 3 months after bracket placement. *Am J Orthod Dentofacial Orthop*. 2006;130:275. e17–e22.
6. Tufekci E, Dixon JS, Gunsolley JC, et al. Prevalence of white spot lesions during orthodontic treatment with fixed appliances. *Angle Orthod*. 2011;81:206–210.
7. Gorelick L, Geiger AM, Gwinnett AJ. Incidence of white spot formation after bonding and banding. *Am J Orthod*. 1982;81:93–98.
8. Mitchell L. Decalcification during orthodontic treatment with fixed appliances—an overview. *Brit J Orthod*. 1992;19:199–205.
9. Ogaard B. Prevalence of white spot lesions in 19-year-olds: a study on untreated and orthodontically treated persons 5 years after treatment. *Am J Orthod Dentofacial Orthop*. 1989;96:423–427.
10. Boersma JG, van der Veen MH, Lagerweij MD, et al. Caries prevalence measured with QLF after treatment with fixed orthodontic appliances: influencing factors. *Caries Res*. 2005;39:41–47.
11. Al Maaitah EF, Adeyemi AA, Higham SM, et al. Factors affecting demineralization during orthodontic treatment: a post-hoc analysis of RCT recruits. *Am J Orthod Dentofacial Orthop*. 2011;139:181–191.
12. Julien KC, Buschang PH, Campbell PM. Prevalence of white spot lesion formation during orthodontic treatment. *Angle Orthod*. 2013;83:641–647.
13. Geiger AM, Gorelick L, Gwinnett AJ, Griswold PG. The effect of a fluoride program on white spot formation during orthodontic treatment. *Am J Orthod Dentofacial Orthop*. 1988;93:29–37.
14. Maxfield BJ, Hamdan AM, Tufekçi E, et al. Development of white spot lesions during orthodontic treatment: perceptions of patients, parents, orthodontists, and general dentists. *Am J Orthod Dentofacial Orthop*. 2012;141:337–344.
15. Ten Bosch JJ, Angmar-Månsson B. A review of quantitative methods for studies of mineral content of intra-oral caries lesions. *J Dent Res*. 1991;70:2–14.
16. Arends J, ten Bosch JJ. Demineralization and remineralization evaluation techniques. *J Dent Res*. 1992;71. Spec No:924–928.
17. Angmar B, Carlstrom D, Glas JE. Studies on the ultrastructure of dental enamel. IV. The mineralization of normal human enamel. *J Ultrastruct Res*. 1963;8:12–23.
18. Lagerweij MD, de Josselin de Jong E, ten Cate JM. The video camera compared with the densitometer as a scanning device for microradiography. *Caries Res*. 1994;28:353–362.
19. Benson PE, Pender N, Higham SM. An in situ caries model to study demineralisation during fixed orthodontics. *Clin Orthod Res*. 1999;2:143–153.
20. Pretty IA, Pender N, Edgar WM, Higham SM. The in vitro detection of early enamel de- and re-mineralization adjacent to bonded orthodontic cleats using quantitative light-induced fluorescence. *Eur J Orthod*. 2003;25:217–223.
21. de Josselin de Jong E, Sundström F, Westerling H, et al. A new method for in vivo quantification of changes in initial enamel caries with laser fluorescence. *Caries Res*. 1995;29:2–7.
22. Benson PE, Pender N, Higham SM. Quantifying enamel demineralization from teeth with orthodontic brackets—a comparison of two methods. Part 1: repeatability and agreement. *Eur J Orthod*. 2003;25:149–158.
23. van der Veen MH, Thomas RZ, Huysmans MC, de Soet JJ. Red autofluorescence of dental plaque bacteria. *Caries Res*. 2006;40:542–545.
24. Pretty IA, Edgar WM, Higham SM. The effect of dehydration on quantitative light-induced fluorescence analysis of early enamel demineralization. *J Oral Rehabil*. 2004;31:179–184.
25. Featherstone JD. Prevention and reversal of dental caries: role of low level fluoride. *Community Dent Oral Epidemiol*. 1999;27:31–40.
26. Featherstone JD. Remineralization, the natural caries repair process—the need for new approaches. *Adv Dent Res*. 2009;21:4–7.
27. Chapman JA, Roberts WE, Eckert GJ, et al. Risk factors for incidence and severity of white spot lesions during treatment with fixed orthodontic appliances. *Am J Orthod Dentofacial Orthop*. 2010;138:188–194.
28. Benkaddour A, Bahije L, Bahoum A, Zaoui F. Orthodontics and enamel demineralization: clinical study of risk factors. *Int Orthod*. 2014;12:458–466.
29. Erbe C, Klukowska M, Tsaknaki I, et al. Efficacy of 3 toothbrush treatments on plaque removal in orthodontic patients assessed with digital plaque imaging: a randomized controlled trial. *Am J Orthod Dentofacial Orthop*. 2013;143:760–766.
30. Mazzoleni S, Bonaldo G, Pontarolo E, et al. Experimental assessment of oral hygiene achieved by children wearing rapid palatal expanders, comparing manual and electric toothbrushes. *Int J Dent Hyg*. 2014;12:187–192.
31. Yaacob M, Worthington HV, Deacon SA, et al. Powered versus manual toothbrushing for oral health. *Cochrane Database Syst Rev*. 2014;6. http://dx.doi.org/10.1002/14651858.CD002281.pub3. CD002281.
32. Goh HH. Interspace/interdental brushes for oral hygiene in orthodontic patients with fixed appliances. *Cochrane Database Syst Rev*. 2007. http://dx.doi.org/10.1002/14651858.CD005410.pub3. CD005410.
33. Sambunjak D, Nickerson JW, Poklepovic T, et al. Flossing for the management of periodontal diseases and dental caries in adults. *Cochrane Database Syst Rev*. 2011. http://dx.doi.org/10.1002/14651858.CD008829.pub2. CD008829.
34. Acharya S, Goyal A, Utreja AK, Mohanty U. Effect of three different motivational techniques on oral hygiene and gingival health of patients undergoing multibracketed orthodontics. *Angle Orthod*. 2011;81:884–888.

35. Lalic M, Aleksic E, Gajic M, et al. Does oral health counseling effectively improve oral hygiene of orthodontic patients? *Eur J Paediatr Dent*. 2012;13:181–186.
36. Eppright M, Shroff B, Best AM, et al. Influence of active reminders on oral hygiene compliance in orthodontic patients. *Angle Orthod*. 2014;84:208–213.
37. Gray D, McIntyre G. Does oral health promotion influence the oral hygiene and gingival health of patients undergoing fixed appliance orthodontic treatment? A systematic literature review. *J Orthod*. 2008;35:262–269.
38. Hadler-Olsen S, Sandvik K, El-Agroudi MA, Øgaard B. The incidence of caries and white spot lesions in orthodontically treated adolescents with a comprehensive caries prophylactic regimen—a prospective study. *Eur J Orthod*. 2012;34:633–639.
39. Brightman LJ, Terezhalmy GT, Greenwell H, et al. The effects of a 0.12% chlorhexidine gluconate mouthrinse on orthodontic patients aged 11 through 17 with established gingivitis. *Am J Orthod Dentofacial Orthop*. 1991;100:324–329.
40. Anderson GB, Bowden J, Morrison EC, Caffesse RG. Clinical effects of chlorhexidine mouthwashes on patients undergoing orthodontic treatment. *Am J Orthod Dentofacial Orthop*. 1997;111:606–612.
41. Oltramari-Navarro PV, Titarelli JM, Marsicano JA, et al. Effectiveness of 0.50% and 0.75% chlorhexidine dentifrices in orthodontic patients: a double-blind and randomized controlled trial. *Am J Orthod Dentofacial Orthop*. 2009;136:651–656.
42. Attin R, Yetkiner E, Aykut-Yetkiner A, et al. Effect of chlorhexidine varnish application on *Streptococcus mutans* colonisation in adolescents with fixed orthodontic appliances. *Aust Orthod J*. 2013;29:52–57.
43. Øgaard B, Larsson E, Henriksson T, et al. Effects of combined application of antimicrobial and fluoride varnishes in orthodontic patients. *Am J Orthod Dentofacial Orthop*. 2001;120:28–35.
44. Riley P, Lamont T. Triclosan/copolymer containing toothpastes for oral health. *Cochrane Database Syst Rev*. 2013;12. CD010514.
45. Pereira-Cenci T, Cenci MS, Fedorowicz Z, Azevedo M. Antibacterial agents in composite restorations for the prevention of dental caries. *Cochrane Database Syst Rev*. 2013;12. CD007819.
46. Abed Al Jawad F, Cunningham SJ, Croft N, Johal A. A qualitative study of the early effects of fixed orthodontic treatment on dietary intake and behaviour in adolescent patients. *Eur J Orthod*. 2012;34:432–436.
47. Harris R, Gamboa A, Dailey Y, Ashcroft A. One-to-one dietary interventions undertaken in a dental setting to change dietary behaviour. *Cochrane Database Syst Rev*. 2012;3. CD006540.
48. Banks PA, Richmond S. Enamel sealants: a clinical evaluation of their value during fixed appliance therapy. *Eur J Orthod*. 1994;16:19–25.
49. Wenderoth CJ, Weinstein M, Borislow AJ. Effectiveness of a fluoride-releasing sealant in reducing decalcification during orthodontic treatment. *Am J Orthod Dentofacial Orthop*. 1999;116:629–634.
50. Heinig N, Hartmann A. Efficacy of a sealant: study on the efficacy of a sealant (Light Bond) in preventing decalcification during multibracket therapy. *J Orofac Orthop*. 2008;69:154–167.
51. Benham AW, Campbell PM, Buschang PH. Effectiveness of pit and fissure sealants in reducing white spot lesions during orthodontic treatment. A pilot study. *Angle Orthod*. 2009;79:338–345.
52. Ghiz MA, Ngan P, Kao E, et al. Effects of sealant and self-etching primer on enamel decalcification. Part II: an in-vivo study. *Am J Orthod Dentofacial Orthop*. 2009;135:206–213.
53. O'Reilly MT, De Jesus Viñas J, Hatch JP. Effectiveness of a sealant compared with no sealant in preventing enamel demineralization in patients with fixed orthodontic appliances: a prospective clinical trial. *Am J Orthod Dentofacial Orthop*. 2013;143:837–844.
54. Leizer C, Weinstein M, Borislow AJ, Braitman LE. Efficacy of a filled-resin sealant in preventing decalcification during orthodontic treatment. *Am J Orthod Dentofacial Orthop*. 2010;137:796–800.
55. Marinho VC, Higgins JP, Sheiham A, Logan S. Fluoride toothpastes for preventing dental caries in children and adolescents. *Cochrane Database Syst Rev*. 2003. CD002278.
56. Walsh T, Worthington HV, Glenny AM, et al. Fluoride toothpastes of different concentrations for preventing dental caries in children and adolescents. *Cochrane Database Syst Rev*. 2010. CD007868.
57. Sonesson M, Twetman S, Bondemark L. Effectiveness of high-fluoride toothpaste on enamel demineralization during orthodontic treatment—a multicenter randomized controlled trial. *Eur J Orthod*. 2014;36:678–682.
58. Benson PE, Parkin N, Dyer F, et al. Fluorides for the prevention of early tooth decay (demineralised white lesions) during fixed brace treatment. *Cochrane Database Syst Rev*. 2013;12. CD003809.
59. Øgaard B, Alm AA, Larsson E, Adofsson U. A prospective, randomized clinical study on the effects of an amine fluoride/stannous fluoride toothpaste/mouthrinse on plaque, gingivitis and initial caries lesion development in orthodontic patients. *Eur J Orthod*. 2006;28:8–12.
60. Marinho VC, Higgins JP, Logan S, Sheiham A. Fluoride mouthrinses for preventing dental caries in children and adolescents. *Cochrane Database Syst Rev*. 2003;(3). CD002284.
61. Geiger AM, Gorelick L, Gwinnett AJ, Benson BJ. Reducing white spot lesions in orthodontic populations with fluoride rinsing. *Am J Orthod Dentofacial Orthop*. 1992;101:403–407.
62. Stecksen-Blicks C, Renfors G, Oscarson ND, et al. Caries-preventive effectiveness of a fluoride varnish: a randomized controlled trial in adolescents with fixed orthodontic appliances. *Caries Res*. 2007;41:455–459.
63. Chin MY, Sandham A, Rumachik EN, et al. Fluoride release and cariostatic potential of orthodontic adhesives with and without daily fluoride rinsing. *Am J Orthod Dentofacial Orthop*. 2009;136:547–553.
64. Lin YC, Lai YL, Chen WT, et al. Kinetics of fluoride release from and reuptake by orthodontic cements. *Am J Orthod Dentofacial Orthop*. 2008;133:427–434.
65. Mitchell L. An investigation into the effect of a fluoride releasing adhesive on the prevalence of enamel surface changes associated with directly bonded orthodontic attachments. *Brit J Orthod*. 1992;19:207–214.
66. Turner PJ. The clinical evaluation of a fluoride-containing orthodontic bonding material. *Brit J Orthod*. 1993;20:307–313.
67. Trimpeneers LM, Dermaut LR. A clinical evaluation of the effectiveness of a fluoride-releasing visible light-activated bonding system to reduce demineralization around orthodontic brackets. *Am J Orthod Dentofacial Orthop*. 1996;110:218–222.
68. Banks PA, Burn A, O'Brien K. A clinical evaluation of the effectiveness of including fluoride into an orthodontic bonding adhesive. *Eur J Orthod*. 1997;19:391–395.
69. Marcusson A, Norevall LI, Persson M. White spot reduction when using glass ionomer cement for bonding in orthodontics: a longitudinal and comparative study. *Eur J Orthod*. 1997;19:233–242.
70. Shungin D, Olsson AI, Persson M. Orthodontic treatment-related white spot lesions: a 14-year prospective quantitative follow-up, including bonding material assessment. *Am J Orthod Dentofacial Orthop*. 2010;138:136.e1–e8; discussion 136–137.
71. Norevall LI, Marcusson A, Persson M. A clinical evaluation of a glass ionomer cement as an orthodontic bonding adhesive compared with an acrylic resin. *Eur J Orthod*. 1996;18:373–384.
72. Chung CK, Millett DT, Creanor SL, et al. Fluoride release and cariostatic ability of a compomer and a resin-modified glass ionomer cement used for orthodontic bonding. *J Dent*. 1998;26:533–538.
73. Banks PA, Chadwick SM, Asher-McDade C, Wright JL. Fluoride-releasing elastomerics—a prospective controlled clinical trial. *Eur J Orthod*. 2000;22:401–407.
74. Mattick CR, Mitchell L, Chadwick SM, Wright J. Fluoride-releasing elastomeric modules reduce decalcification: a randomized controlled trial. *J Orthod*. 2001;28:217–219.
75. Pessan JP, Al-Ibrahim NS, Buzalaf MA, Toumba KJ. Slow-release fluoride devices: a literature review. *J Appl Oral Sci*. 2008;16:238–246.
76. Marini I, Pelliccioni GA, Vecchiet F, et al. A retentive system for intra-oral fluoride release during orthodontic treatment. *Eur J Orthod*. 1999;21:695–701.
77. Luther F, Tobin M, Robertson AJ, Toumba KJ. Fluoride-releasing glass beads in orthodontic treatment to reduce decay: a randomized, controlled clinical trial. *World J Orthod*. 2005;6:166–167.
78. Chong LY, Clarkson JE, Dobbyn-Ross L, Bhakta S. Slow-release fluoride devices for the control of dental decay. *Cochrane Database Syst Rev*. 2014;11. CD005101.

79. Sköld-Larsson K, Sollenius O, Karlsson L, et al. Effect of fluoridated milk on enamel demineralization adjacent to fixed orthodontic appliances. *Acta Odontol Scand*. 2013;71:464–468.
80. Cagetti MG, Campus G, Milia E, Lingström P. A systematic review on fluoridated food in caries prevention. *Acta Odontol Scand*. 2013;71:381–387.
81. Benson PE. Fluoride-containing materials and the prevention of demineralization during orthodontic treatment—which research method should we now use? *Semin Orthod*. 2010;16:293–301.
82. Reynolds EC. Casein phosphopeptide–amorphous calcium phosphate: the scientific evidence. *Adv Dent Res*. 2009;21:25–29.
83. Reynolds EC, Cai F, Cochrane NJ, et al. Fluoride and casein phosphopeptide–amorphous calcium phosphate. *J Dent Res*. 2008;87:344–348.
84. Reynolds EC. Anticariogenic complexes of amorphous calcium phosphate stabilized by casein phosphopeptides: a review. *Spec Care Dentist*. 1998;18:8–16.
85. Cochrane NJ, Reynolds EC. Casein phosphates in oral health. In: Wlison M, ed. *Food Constituents and Oral Health: Current Status and Future Prospects*. Cambridge, UK: Woodhead Publishing Limited; 2009:185–224.
86. Reynolds EC. Remineralization of enamel subsurface lesions by casein phosphopeptide–stabilized calcium phosphate solutions. *J Dent Res*. 1997;76:1587–1595.
87. Reynolds EC, Cain CJ, Webber FL, et al. Anticariogenicity of calcium phosphate complexes of tryptic casein phosphopeptides in the rat. *J Dent Res*. 1995;74:1272–1279.
88. Schupbach P, Neeser JR, Golliard M, et al. Incorporation of caseinoglycomacropeptide and caseinophosphopeptide into the salivary pellicle inhibits adherence of *mutans* streptococci. *J Dent Res*. 1996;75:1779–1788.
89. Reynolds EC, Cai F, Shen P, Walker GD. Retention in plaque and remineralization of enamel lesions by various forms of calcium in a mouthrinse or sugar-free chewing gum. *J Dent Res*. 2003;82:206–211.
90. Cai F, Shen P, Morgan MV, Reynolds EC. Remineralization of enamel subsurface lesions in situ by sugar-free lozenges containing casein phosphopeptide–amorphous calcium phosphate. *Aust Dent J*. 2003;48:240–243.
91. Walker G, Cai F, Shen P, et al. Increased remineralization of tooth enamel by milk containing added casein phosphopeptide–amorphous calcium phosphate. *J Dairy Res*. 2006;73:74–78.
92. Rao SK, Bhat GS, Aradhya S, et al. Study of the efficacy of toothpaste containing casein phosphopeptide in the prevention of dental caries: a randomized controlled trial in 12- to 15-year-old high caries risk children in Bangalore, India. *Caries Res*. 2009;43:430–435.
93. Sitthisettapong T, Phantumvanit P, Huebner C, Derouen T. Effect of CPP-ACP paste on dental caries in primary teeth: a randomized trial. *J Dent Res*. 2012;91:847–852.
94. Robertson MA, Kau CH, English JD, et al. MI Paste Plus to prevent demineralization in orthodontic patients: a prospective randomized controlled trial. *Am J Orthod Dentofacial Orthop*. 2011;140:660–668.
95. Al Zraikat H, Palamara JE, Messer HH, et al. The incorporation of casein phosphopeptide–amorphous calcium phosphate into a glass ionomer cement. *Dent Mater*. 2011;27:235–243.
96. Burt BA. The use of sorbitol- and xylitol-sweetened chewing gum in caries control. *J Am Dent Assoc*. 2006;137:190–196.
97. Cosyn J, Verelst K. An efficacy and safety analysis of a chlorhexidine chewing gum in young orthodontic patients. *J Clin Periodontol*. 2006;33:894–899.
98. Aksoy A, Duran N, Toroglu S, Koksal F. Short-term effect of mastic gum on salivary concentrations of cariogenic bacteria in orthodontic patients. *Angle Orthod*. 2007;77:124–128.
99. Shen P, Cai F, Nowicki A, et al. Remineralization of enamel subsurface lesions by sugar-free chewing gum containing casein phosphopeptide–amorphous calcium phosphate. *J Dent Res*. 2001;80:2066–2070.
100. Morgan MV, Adams GG, Bailey DL, et al. The anticariogenic effect of sugar-free gum containing CPP-ACP nanocomplexes on approximal caries determined using digital bitewing radiograph. *Caries Res*. 2008;42:171–184.
101. Ogaard B, Ten Bosch JJ. Regression of white spot enamel lesions. A new optical method for quantitative longitudinal evaluation in vivo. *Am J Orthod Dentofacial Orthop*. 1994;106:238–242.
102. Arends J, Christoffersen J. The nature of early caries lesions in enamel. *J Dent Res*. 1986;65:2–11.
103. Chen H, Liu X, Dai J, et al. Effect of remineralizing agents on white spot lesions after orthodontic treatment: a systematic review. *Am J Orthod Dentofacial Orthop*. 2013;143:376–382.e3.
104. Du M, Cheng N, Tai B, et al. Randomized controlled trial on fluoride varnish application for treatment of white spot lesion after fixed orthodontic treatment. *Clin Oral Investig*. 2012;16:463–468.
105. Ogaard B, Rølla G, Arends J, ten Cate JM. Orthodontic appliances and enamel demineralization. Part 2. Prevention and treatment of lesions. *Am J Orthod Dentofacial Orthop*. 1988;94:123–128.
106. Willmot DR. White lesions after orthodontic treatment: does low fluoride make a difference? *J Orthod*. 2004;31:235–242; discussion 202.
107. Baeshen HA, Lingström P, Birkhed D. Effect of fluoridated chewing sticks (Miswaks) on white spot lesions in postorthodontic patients. *Am J Orthod Dentofacial Orthop*. 2011;140:291–297.
108. Andersson A, Sköld-Larsson K, Hallgren A, et al. Effect of a dental cream containing amorphous cream phosphate complexes on white spot lesion regression assessed by laser fluorescence. *Oral Health Prev Dent*. 2007;5:229–233.
109. Bailey DL, Adams GG, Tsao CE, et al. Regression of post-orthodontic lesions by a remineralizing cream. *J Dent Res*. 2009;88:1148–1153.
110. Beerens MW, van der Veen MH, van Beek H, ten Cate JM. Effects of casein phosphopeptide amorphous calcium fluoride phosphate paste on white spot lesions and dental plaque after orthodontic treatment: a 3-month follow-up. *Eur J Oral Sci*. 2010;118:610–617.
111. Bröchner A, Christensen C, Kristensen B, et al. Treatment of post-orthodontic white spot lesions with casein phosphopeptide–stabilised amorphous calcium phosphate. *Clin Oral Investig*. 2011;15:369–373.
112. Huang GH, Roloff-Chiang B, Mills BE, et al. Effectiveness of MI Paste Plus and PreviDent fluoride varnish for treatment of white spot lesions: a randomized controlled trial. *Am J Orthod Dentofacial Orthop*. 2013;143:31–41.
113. Paris S, Hopfenmuller W, Meyer-Lueckel H. Resin infiltration of caries lesions: an efficacy randomized trial. *J Dent Res*. 2010;89:823–826.
114. Eckstein A, Helms HJ, Knosel M. Camouflage effects following resin infiltration of postorthodontic white-spot lesions in vivo: one-year follow-up. *Angle Orthod*. 2015;85:374–380.
115. Knosel M, Eckstein A, Helms HJ. Durability of esthetic improvement following Icon resin infiltration of multibracket-induced white spot lesions compared with no therapy over 6 months: a single-center, split-mouth, randomized clinical trial. *Am J Orthod Dentofacial Orthop*. 2013;144:86–96.

References for Root Resorption

116. Darendeliler MA, Kharbanda OP, Chan EK, et al. Root resorption and its association with alterations in physical properties, mineral contents and resorption craters in human premolars following application of light and heavy controlled orthodontic forces. *Orthod Craniofac Res*. 2004;7(2):79–97.
117. Al-Qawasmi RA, Hartsfield Jr JK, Everett ET, et al. Genetic predisposition to external apical root resorption. *Am J Orthod Dentofacial Orthop*. 2003;123(3):242–252.
118. Artun J, Van't Hullenaar R, Doppel D, Kuijpers-Jagtman AM. Identification of orthodontic patients at risk of severe apical root resorption. *Am J Orthod Dentofacial Orthop*. 2009;135(4):448–455.
119. Sharab LY, Morford LA, Dempsey J, et al. Genetic and treatment-related risk factors associated with external apical root resorption (EARR) concurrent with orthodontia. *Orthod Craniofac Res*. 2015;18(Suppl 1):71–82.
120. Ngan DC, Kharbanda OP, Byloff FK, Darendeliler MA. The genetic contribution to orthodontic root resorption: a retrospective twin study. *Aust Orthod J*. 2004;20(1):1–9.
121. Kokich VG. Orthodontic and nonorthodontic root resorption: their impact on clinical dental practice. *J Dent Educ*. 2008;72(8):895–902.
122. Sameshima GT, Sinclair PM. Predicting and preventing root resorption: Part I. Diagnostic factors. *Am J Orthod Dentofacial Orthop*. 2001;119(5):505–510.
123. Sameshima GT, Sinclair PM. Characteristics of patients with severe root resorption. *Orthod Craniofac Res*. 2004;7(2):108–114.

124. Vlaskalic V, Boyd RL, Baumrind S. Etiology and sequelae of root resorption. *Semin Orthod*. 1998;4:124–131.
125. Brezniak N, Wasserstein A. Orthodontically induced inflammatory root resorption. Part II: the clinical aspects. *Angle Orthod*. 2002;72(2):180–184.
126. Levander E, Bajka R, Malmgren O. Early radiographic diagnosis of apical root resorption during orthodontic treatment: a study of maxillary incisors. *Eur J Orthod*. 1998;20(1):57–63.
127. Kaley J, Phillips C. Factors related to root resorption in edgewise practice. *Angle Orthod*. 1991;61:125–132.
128. Linge L, Linge BO. Patient characteristics and treatment variables associated with apical root resorption during orthodontic treatment. *Am J Orthod Dentofacial Orthop*. 1991;99:35–43.
129. Sehr K, Bock NC, Serbesis C, et al. Severe external apical root resorption—local cause or genetic predisposition? *J Orofac Orthop*. 2011;72(4):321–331.
130. Brin I, Becker A, Zilberman Y. Resorbed lateral incisors adjacent to impacted canines have normal crown size. *Am J Orthod Dentofacial Orthop*. 1993;104(1):60–66.
131. Ericson S, Kurol PJ. Resorption of incisors after ectopic eruption of maxillary canines: a CT study. *Angle Orthod*. 2000;70(6):415–423.
132. Ericson S, Kurol J. Early treatment of palatally erupting maxillary canines by extraction of the primary canines. *Eur J Orthod*. 1988;10(4):283–295.
133. Kook YA, Park S, Sameshima GT. Peg-shaped and small lateral incisors not at higher risk for root resorption. *Am J Orthod Dentofacial Orthop*. 2003;123(3):253–258.
134. Shaw AM, Sameshima GT, Vu HV. Mechanical stress generated by orthodontic forces on apical root cementum: a finite element model. *Orthod Craniofac Res*. 2004;7(2):98–107.
135. Hecht R. Root shape frequency and direction of dilaceration: a CBCT study. Los Angeles: MS thesis. University of Southern California; 2013.
136. Brin I, Ben-Bassat Y, Heling I, Engelberg A. The influence of orthodontic treatment on previously traumatized permanent incisors. *Eur J Orthod*. 1991;13(5):372–377.
137. Kindelan SA, Day PF, Kindelan JD, et al. Dental trauma: an overview of its influence on the management of orthodontic treatment. Part 1. *J Orthod*. 2008;35(2):68–78.
138. Tieu LD, Saltaji H, Normando D, Flores-Mir C. Radiologically determined orthodontically induced external apical root resorption in incisors after non-surgical orthodontic treatment of class II division 1 malocclusion: a systematic review. *Prog Orthod*. 2014;15:48.
139. Ioannidou-Marathiotou I, Zafeiriadis AA, Papadopoulos MA. Root resorption of endodontically treated teeth following orthodontic treatment: a meta-analysis. *Clin Oral Investig*. 2013;17(7):1733–1744.
140. Rakhshan V, Nateghian N, Ordoubazari M. Risk factors associated with external apical root resorption of the maxillary incisors: a 15-year retrospective study. *Aust Orthod J*. 2012;28(1):51–56.
141. Weltman B, Vig KW, Fields HW, et al. Root resorption associated with orthodontic tooth movement: a systematic review. *Am J Orthod Dentofacial Orthop*. 2010;137(4):462–476; discussion 12A.
142. Segal GR, Schiffman P, Tuncay O. Meta analysis of the treatment-related factors of external apical root resorption. *Orthod Craniofac Res*. 2004;7:71–78.
143. Zhou Y. Open bite as a risk factor for orthodontic root resorption. *Eur J Orthod*. 2015;37(1):118–119.
144. Motokawa M, Terao A, Kaku M, et al. Open bite as a risk factor for orthodontic root resorption. *Eur J Orthod*. 2013;35(6):790–795.
145. Harris EF, Butler ML. Patterns of incisor root resorption before and after orthodontic correction in cases with anterior open bites. *Am J Orthod Dentofacial Orthop*. 1992;101(2):112–119.
146. Motokawa M, Terao A, Karadeniz EI, et al. Effects of long-term occlusal hypofunction and its recovery on the morphogenesis of molar roots and the periodontium in rats. *Angle Orthod*. 2013;83(4):597–604.
147. Mirabella AD, Artun J. Risk factors for apical root resorption of maxillary anterior teeth in adult orthodontic patients. *Am J Orthod Dentofacial Orthop*. 1995;108(1):48–55.
148. Spurrier SW, Hall SH, Joondeph DR, et al. A comparison of apical root resorption during orthodontic treatment in endodontically treated and vital teeth. *Am J Orthod Dentofacial Orthop*. 1990;97(2):130–134.
149. Castro I, Valladares-Neto J, Estrela C. Contribution of cone beam computed tomography to the detection of apical root resorption after orthodontic treatment in root-filled and vital teeth. *Angle Orthod*. 2015;85(5):771–776.
150. Walker SL, Tieu LD, Flores-Mir C. Radiographic comparison of the extent of orthodontically induced external apical root resorption in vital and root-filled teeth: a systematic review. *Eur J Orthod*. 2013;35(6):796–802.
151. Do PT. The effect of orthodontic treatment on the developing roots of premolars. Los Angeles: MS thesis. University of Southern California: 2002.
152. Mavragani M, Bøe OE, Wisth PJ, Selvig KA. Changes in root length during orthodontic treatment: advantages for immature teeth. *Eur J Orthod*. 2002;24(1):91–97.
153. Wallace RG, Barr RJ, Osterberg PH, Mollan RA. Familial expansile osteolysis. *Clin Orthop Relat Res*. 1989;248:265–277.
154. Nishioka M, Ioi H, Nakata S, et al. Root resorption and immune system factors in the Japanese. *Angle Orthod*. 2006;76(1):103–108.
155. Davidovitch Z, Lee YJ, Counts AL, et al. The immune system possibly modulates orthodontic root resorption. In: Davidovitch Z, Mah J, eds. *Biological Mechanisms of Tooth Movement and Craniofacial Adaptation*. Boston, MA: Harvard Society for the Advancement of Orthodontics; 2000:207–217.
156. McNab S, Battistutta D, Taverne A, Symons AL. External apical root resorption of posterior teeth in asthmatics after orthodontic treatment. *Am J Orthod Dentofacial Orthop*. 1999;116(5):545–551.
157. Owman-Moll P, Kurol J. Root resorption after orthodontic treatment in high- and low-risk patients: analysis of allergy as a possible predisposing factor. *Eur J Orthod*. 2000;22(6):657–663.
158. Murata N, Ioi H, Ouchi M, et al. Effect of allergen sensitization on external root resorption. *J Dent Res*. 2013;92(7):641–647.
159. Hegde S, Shubha AB, Kakti A, Rao BD. Extensive idiopathic external apical root resorption on a 13 year old child. *J Clin Pediatr Dent*. 2012;36(3):289–292.
160. Moazami F, Karami B. Multiple idiopathic apical root resorption: a case report. *Int Endod J*. 2007;40(7):573–578.
161. Kanungo M, Khandelwal V, Nayak UA, Nayak PA. Multiple idiopathic apical root resorption. *BMJ Case Rep*. 2013;23:2013. http://dx.doi.org/10.1136/bcr-2013-009696. pii: bcr2013009696.
162. Soğur E, Soğur HD, Baksi Akdeniz BG, Sen BH. Idiopathic root resorption of the entire permanent dentition: systematic review and report of a case. *Dent Traumatol*. 2008;24(4):490–495.
163. Sameshima GT, Sinclair PM. Predicting and preventing root resorption: Part II. Treatment factors. *Am J Orthod Dentofacial Orthop*. 2001;119(5):511–515.
164. Alzahawi K, Færøvig E, Brudvik P, et al. Root resorption after leveling with super-elastic and conventional steel arch wires: a prospective study. *Prog Orthod*. 2014;15(1):35.
165. Jacobs C, Gebhardt PF, Jacobs V, et al. Root resorption, treatment time and extraction rate during orthodontic treatment with self-ligating and conventional brackets. *Head Face Med*. 2014;10:2.
166. Brezniak N, Wasserstein A. Root resorption following treatment with aligners. *Angle Orthod*. 2008;78(6):1119–1124.
167. Barbagallo LJ, Jones AS, Petocz P, Darendeliler MA. Physical properties of root cementum: Part 10. Comparison of the effects of invisible removable thermoplastic appliances with light and heavy orthodontic forces on premolar cementum. A microcomputed-tomography study. *Am J Orthod Dentofacial Orthop*. 2008;133(2):218–227.
168. Krieger E, Drechsler T, Schmidtmann I, et al. Apical root resorption during orthodontic treatment with aligners? A retrospective radiometric study. *Head Face Med*. 2013;9:21.
169. Machado IM, Ferguson DJ, Wilcko WM, Wilcko MT. Reaborcion radicular despues del tratamiento orodoncico con o sin corticotomia alveolar. *Reb Venezuela Ortho*. 2002;19:647–653.
170. Ren A, Lv T, Kang N, et al. Rapid orthodontic tooth movement aided by alveolar surgery in beagles. *Am J Orthod Dentofacial Orthop*. 2007;131(2):160.e1–e10.
171. King GJ, Fischlschweiger W. The effect of force magnitude on extractable bone resorptive activity and cemental cratering in orthodontic tooth movement. *J Dent Res*. 1982;61:775–779.

172. Vardimon AD, Graber TM, Voss LR, Lenke J. Determinants controlling iatrogenic external root resorption and repair during and after palatal expansion. *Angle Orthod*. 1991;61:113–122.
173. Harris DA, Jones AS, Darendeliler MA. Physical properties of root cementum: part 8. Volumetric analysis of root resorption craters after application of controlled intrusive light and heavy orthodontic forces: a microcomputed tomography scan study. *Am J Orthod Dentofacial Orthop*. 2006;130:639–647.
174. Paetyangkul A, Türk T, Elekdağ-Türk S, et al. Physical properties of root cementum: Part 16. Comparisons of root resorption and resorption craters after the application of light and heavy continuous and controlled orthodontic forces for 4, 8, and 12 weeks. *Am J Orthod Dentofacial Orthop*. 2011;139(3):e279–e284.
175. Acar A, Canyürek U, Kocaaga M, Erverdi N. Continuous vs. discontinuous force application and root resorption. *Angle Orthod*. 1999;69:159–163.
176. Levander E, Malmgren O, Eliasson S. Evaluation of root resorption in relation to two orthodontic treatment regimes. A clinical experimental study. *Eur J Orthod*. 1994;16:223–228.
177. Maltha JC, Dijkman GEHM. Discontinuous forces cause less extensive root resorption than continuous forces [abstract]. *Eur J Orthod*. 1996;18:420.
178. Weiland F. Constant versus dissipating forces in orthodontics: the effect on initial tooth movement and root resorption. *Eur J Orthod*. 2003;25:335–342.
179. Brin I, Bollen AM. External apical root resorption in patients treated by serial extractions followed by mechanotherapy. *Am J Orthod Dentofacial Orthop*. 2011;139(2):e129–e134.
180. Akyalcin S, Alexander SP, Silva RM, English JD. Evaluation of three-dimensional root surface changes and resorption following rapid maxillary expansion: a cone beam computed tomography investigation. *Orthod Craniofac Res*. 2015;18(Suppl 1):117–126.
181. Forst D, Nijjar S, Khaled Y, et al. Radiographic assessment of external root resorption associated with jackscrew-based maxillary expansion therapies: a systematic review. *Eur J Orthod*. 2014;36(5):576–585.
182. Sameshima GT, Sinclair PM. Predicting and preventing root resorption: Part II. Treatment factors. *Am J Orthod Dentofacial Orthop*. 2001 May;119(5):511–515.
183. de Freitas JC, Lyra OC, de Alencar AH, Estrela C. Long-term evaluation of apical root resorption after orthodontic treatment using periapical radiography and cone beam computed tomography. *Dental Press J Orthod*. 2013;18(4):104–112.
184. Eross E, Turk T, Elekdag-Turk S, et al. Physical properties of root cementum: Part 25. Extent of root resorption after the application of light and heavy buccopalatal jiggling forces for 12 weeks: a microcomputed tomography study. *Am J Orthod Dentofacial Orthop*. 2015;147(6):738–746.
185. Ng The extent of orthodontically induced inflammatory root resorption following transverse and vertical jiggling movement with heavy forces for 12 weeks: a micro-CT study. Sydney, Australia: MS thesis. University of Sydney; 2014.
186. Liou EJ, Chang PM. Apical root resorption in orthodontic patients with en-masse maxillary anterior retraction and intrusion with miniscrews. *Am J Orthod Dentofacial Orthop*. 2010;137(2):207–212.
187. Kalkwarf KL, Krejci RF, Pao YC. Effect of apical root resorption on periodontal support. *J Prosthet Dent*. 1986;56(3):317–319.
188. Sameshima GT, Asgarifar KO. Assessment of root resorption and root shape: periapical vs panoramic films. *Angle Orthod*. 2001;71(3):185–189.
189. Lund H, Gröndahl K, Hansen K, Gröndahl HG. Apical root resorption during orthodontic treatment. A prospective study using cone beam CT. *Angle Orthod*. 2012;82(3):480–487.
190. Kapila SD, Nervina JM. CBCT in orthodontics: assessment of treatment outcomes and indications for its use. *Dentomaxillofac Radiol*. 2015;44(1). http://dx.doi.org/10.1259/dmfr.20140282. 20140282.
191. Castro IO, Alencar AH, Valladares-Neto J, Estrela C. Apical root resorption due to orthodontic treatment detected by cone beam computed tomography. *Angle Orthod*. 2013;83(2):196–203.
192. Lee KS, Straja SR, Tuncay OC. Perceived long-term prognosis of teeth with orthodontically resorbed roots. *Orthod Craniofac Res*. 2003;6(3):177–191.
193. Artun J, Smale I, Behbehani F, et al. Apical root resorption six and 12 months after initiation of fixed orthodontic appliance therapy. *Angle Orthod*. 2005;75(6):919–926.
194. Remington DN, Joondeph DR, Artun J, et al. Long-term evaluation of root resorption occurring during orthodontic treatment. *Am J Orthod Dentofacial Orthop*. 1989;96(1):43–46.
195. Savage RR, Kokich Sr VG. Restoration and retention of maxillary anteriors with severe root resorption. *J Am Dent Assoc*. 2002;133(1):67–71.
196. Becker A, Chaushu S. Long-term follow-up of severely resorbed maxillary incisors after resolution of an etiologically associated impacted canine. *Am J Orthod Dentofacial Orthop*. 2005;127(6):650–654.
197. Jönsson A, Malmgren O, Levander E. Long-term follow-up of tooth mobility in maxillary incisors with orthodontically induced apical root resorption. *Eur J Orthod*. 2007;29(5):482–487.
198. Marques LS, Chaves KC, Rey AC, et al. Severe root resorption and orthodontic treatment: clinical implications after 25 years of follow-up. *Am J Orthod Dentofacial Orthop*. 2011;139(4 Suppl):S166–S169.
199. Heithersay GS. Invasive cervical resorption: an analysis of potential predisposing factors. *Quintessence Int*. 1999;30(2):83–95.
200. Patel S, Kanagasingam S, Pitt Ford T. External cervical resorption: a review. *J Endod*. 2009;35(5):616–625.
201. Mah J, Prasad N. Dentine phosphoproteins in gingival crevicular fluid during root resorption. *Eur J Orthod*. 2004;26:25–30.
202. Balducci L, Ramachandran A, Hao J, et al. Biological markers for evaluation of root resorption. *Arch Oral Biol*. 2007;52(3):203–208.
203. George A, Evans C. Detection of root resorption using dentin and bone markers. *Orthod Craniofac Res*. 2009;12:229–235.
204. Yoshizawa JM, Schafer CA, Schafer JJ, et al. Salivary biomarkers: toward future clinical and diagnostic utilities. *Clin Microbiol Rev*. 2013;26(4):781–791.
205. Ramos Sde P, Ortolan GO, Dos Santos LM, et al. Anti-dentine antibodies with root resorption during orthodontic treatment. *Eur J Orthod*. 2011;33(5):584–591.

Minimally and Noninvasive Approaches to Accelerate Tooth Movement

Ignacio Blasi, Jr., and Dubravko Pavlin

OUTLINE

MICRO-OSTEOPERFORATIONS, by Ignacio Blasi, Jr., 913
Introduction, 913
Biological Mechanism, 913
Techniques and Indications, 914
Techniques, 914
Indications, 916

LOW-LEVEL MECHANICAL VIBRATIONS, by Dubravko Pavlin, 918
Introduction, 918
Clinical Studies, 918
Vibration and Treatment with Clear Aligners, 921
Biological Mechanism of Bone Response to Vibration, 921

MICRO-OSTEOPERFORATIONS*

Introduction

Prolonged treatment time in orthodontics is an undesirable side effect for both the patient and the clinician. Usually, between 2 and 3 years of treatment are required for a case to be properly completed,[1,2] which depends on a variety of factors including the biological response of each individual to orthodontic forces, the complexity of the case, skeletal discrepancies, the amount of dental camouflage of skeletal problems, treatment mechanics, and patient compliance. Regardless of the length of required treatment, it is important to emphasize that a clinician should provide to the patient the best treatment outcome possible. For example, in a case with an excessive overjet and lack of anterior guidance, coupling of the anterior segments should not be compromised because of a prolonged treatment time.

The first of the two fundamental principles in minimizing the length of orthodontic treatment is a proper diagnosis and an individualized treatment plan that includes clear treatment objectives to correct a malocclusion and to provide optimal occlusion without trespassing on the anatomic boundaries and compromising aesthetics, while avoiding any harm to the adjacent tissues. The second principle is an understanding of orthodontic biomechanics, which allows the clinician to develop a sound mechanical plan and to select appropriate appliances to achieve the treatment goals specific for each patient.

In addition to these two basic principles, several approaches have been proposed to accelerate orthodontic tooth movement. These include self-ligating brackets,[3–5] robotic prefabricated wires (e.g., SureSmile, OraMetrix, Inc., Richardson, TX),[6,7] indirect bonding technique,[8–10] low-level laser therapy (e.g., OrthoPulse, Biolux Research Ltd., Vancouver, BC, Canada),[11–15] electrical currents stimulation,[16–19] pulsed electromagnetic fields,[20] piezoelectricity,[21] injections of pharmacologic agents such as prostaglandins,[22–25] Relaxin[26–29] and platelet-rich plasma (PRP),[30] decortication and related techniques (see Chapter 22),[31,32] as well as distraction osteogenesis,[33–35] corticision,[36,37] osteoperforations (e.g., Propel, Propel Orthodontics, Ossining, NY),[38] and low-level mechanical vibration (e.g., AcceleDent, OrthoAccel Technologies, Inc., Bellaire, TX).[39–41] However, limited clinical and scientific evidence can be found for the effectiveness of most of these techniques.[42,43]

Performing micro-osteoperforations is a minimally invasive technique used to accelerate the rate of tooth movement by stimulating the patient's own biologic response as an attempt to shorten treatment time. It can be also used to facilitate and accomplish certain difficult and challenging tooth movements in a more predictable manner. The concept is similar to other surgical techniques, such as alveolar decortication and variations, damaging or traumatizing the cortical alveolar bone to cause a localized inflammatory response and thereby increase the regular bone turnover (the regional accelerated phenomenon[44,45] [RAP]) and increase the rate of tooth movement.

Biological Mechanism

Orthodontic tooth movement occurs in the presence of a mechanical stimulus sequenced by remodeling of the alveolar bone and the periodontal ligament (PDL). Orthodontic tooth movement consists of three phases: (1) initial phase, (2) lag phase, and (3) postlag phase.[46]

The initial phase consists of an immediate and rapid movement and occurs within 24 to 48 hours after the initial application of force to the tooth. The rate of movement is largely attributed to the displacement of the tooth in the PDL space, causing its compression and undermining bone resorption on the pressure side. Bone resorption occurs through osteoclastic activity by creating bone lacunae that will later be filled in by osteoblast cells.

The lag phase lasts 20 to 30 days and displays relatively little to no tooth movement. This phase is marked by PDL hyalinization in the region of compression where the blood supply is cut off. No subsequent tooth movement occurs until the cells complete the removal of all of the necrotic tissues. Once the PDL regenerates,

*Ignacio Blasi Jr.

tooth movement continues. The postlag phase follows the lag phase, during which the rate of movement increases.[47,48]

When tooth movement occurs, the fibers on the tension side are stretched and resist further movement. Light continuous tension causes elongation of fiber bundles and subsequent bone formation mediated by osteoblasts. Osteoblasts are differentiated from the local precursor cells, called *mesenchymal stem cells*. Mature osteoblasts form the osteoids, and the mineralization processes follow.[49] In addition, endothelial nitric oxide synthase (eNOS) and enzyme profiles indicate bone formation in the tension area.[50]

Different cytokines and hormones are involved in the biological mechanism of tooth movement. Tumor necrosis factor–alpha (TNF-α), interleukin-1 beta (IL-1β), interleukin-6 (IL-6), prostaglandin E2 (PGE2), and other inflammatory cytokines can facilitate osteoclastic bone resorption processes[51,52] through activation of the nuclear factor kappa B (RANK) and the nuclear factor kappa B ligand (RANKL). In addition, osteoblastic cells regulate osteoclastic differentiation by synthesizing RANKL.[52,53]

Different cytokiness such as TNF-α levels are increased at the gingival sulcus with orthodontic tooth movement.[54,55] To assess the impact of tooth movement, studies blocking these factors have been conducted and have demonstrated less tooth movement when compared with the control group.[56–59] In an animal study, mechanical forces were applied to tumor necrosis factor receptor (TNFR)-deficient mice to investigate the role of TNF signaling in orthodontic tooth movement. The experiment demonstrated that tooth movement was delayed 6 days after the application of the appliance in TNFR-deficient mice compared with wild-type mice. Therefore these results revealed certain roles of TNF in orthodontic movement, and the assumption that increasing the levels of these factors can accelerate tooth movement is a reasonable conclusion.[60]

In a study in which micro-osteoperforations were performed in combination with orthodontic tooth movement in a rat model, the expression of different inflammatory markers was observed to increase. It was hypothesized that these markers, caused by the micro-osteoperforations, led to increased osteoclast activity and to increased speed of tooth movement (Fig. 31-1, *A*).[61]

Cytokines such as IL-1, IL-6, IL-8, and TNF-α have been proven to be linked with bone remodeling during orthodontic movement.[52] Alikhani and colleagues[38] demonstrated that some chemokines (CCL-2, CCL-3, CCL-5, and IL-8) and cytokines (IL-1, TNF-α, and IL-6) had increased levels during orthodontic tooth movement and that osteoperforations significantly increased the expression of these factors. Their study suggested that the higher presence of these cytokines is related with more osteoclast activity and therefore increased tooth movement (Fig. 31-1, *B*).

Techniques and Indications

The key to success when performing micro-osteoperforations depends first of a proper diagnosis and case selection and second of an appropriate application of the technique.

Micro-osteoperforations and decortication with bone grafting (see Chapter 22) employ the same biological and physiologic principles to accelerate tooth movement. Although both techniques accelerate the rate of tooth movement, they can be used for different treatment objectives. The objective for the first technique is the velocity of the tooth movement or achieving a complex movement; for the second technique, a periodontal benefit could be the principal objective and the acceleration of tooth movement a beneficial side effect. Therefore a careful periodontal evaluation should first be completed in each case.

Micro-osteoperforations involve a less invasive approach than corticotomy. The perforations are performed through the gingival tissues, penetrating the cortical plate (Fig. 31-2). There is no need to raise a mucogingival flap, create any incisions or perform a tissue punch to access the cortical bone. The osteoperforations are performed with a bone screw, which is self-drilling and self-tapping, and no pilot hole is necessary. The Propel device (Propel Orthodontics, Ossining, NY) is a good example. The first-generation Propel device was a single-use, sterile, disposable manual perforator similar in size to a small handheld screwdriver. It had a light-emitting diode (LED) depth stop indicator that illuminated once the desired perforation depth was achieved. The second-generation Propel device has a heavier, balanced metal handle and disposable screw tips with marks to indicate the depth of the perforation. The third-generation consists of a disposable screw tip, an automatic electric torque driver and a contra-angle.

The technique is performed by the orthodontist, requires no surgical instrumentation, and can be performed in a standard dental setting using a traditional aseptic protocol.

Techniques

Knowing and evaluating the anatomy surrounding the tissues of the site to be treated are important; the mandibular nerve and the maxillary sinuses should be located. Palpating and exploring the area for root proximity to ensure safety while performing the perforations are essential. A current panoramic or a cone-beam computed tomography (CBCT) x-ray image might be helpful tool a to verify the patient's anatomy and root location. The oral cavity should be aseptic to diminish the bacterial count on the site to be treated.

Either local infiltrative or topical anesthetic could be used to anesthetize the area. If topical anesthetic is chosen, then the compounded formulas are the best to be used.[62] Profound gel (Steven's Pharmacy, Costa Mesa, CA) is a mixture of 10% prilocaine, 10% lidocaine, and 4% tetracaine. The ingredients in the Baddest Topical in Town (BTT 12.5) (Woodland Hills Pharmacy, Woodland Hills, CA) are 12.5% lidocaine, 12.5% tetacaine, 3% prilocaine, and 3% phenylephrine gel. Before applying the topical anesthesia, the gingiva should be thoroughly dried with gauze to remove the saliva and salivary proteins that can act as a barrier to the medications within the compounded formula. The topical anesthetic should be in contact with the gingiva for at least 4 minutes or until the tissue gets a corrugated look. Local infiltration may be used to numb the patient in a more predictable manner and is definitely the choice for palatal perforations. The area should be tested to ensure numbness.

The osteoperforations should penetrate through the cortical plate into the cancellous bone to ensure trauma to the alveolar bone and to achieve an inflammatory response greater than orthodontic forces alone. The depths of the perforations are dependent upon the soft tissue and bone thickness. A transgingival perforation with a periodontal probe is recommended to determine the soft tissue thickness. Katranji and colleagues[63] reported that the buccal plate of the dentate maxilla and mandible ranged from 1.6 to 2.2 mm in thickness. The average cortical thickness in the maxilla is 2.23 mm in the molar, 1.62 mm in the premolar, and 1.59 mm in the anterior regions. In the mandible, the cortical thickness is 1.98 mm in the molar, 1.20 mm in the premolar, and 0.99 mm in the anterior regions. They found that the thinnest area is in the lower anterior region and the thickest area in the upper posterior region.[63] Although the cortical thicknesses may vary among individuals, the soft tissue

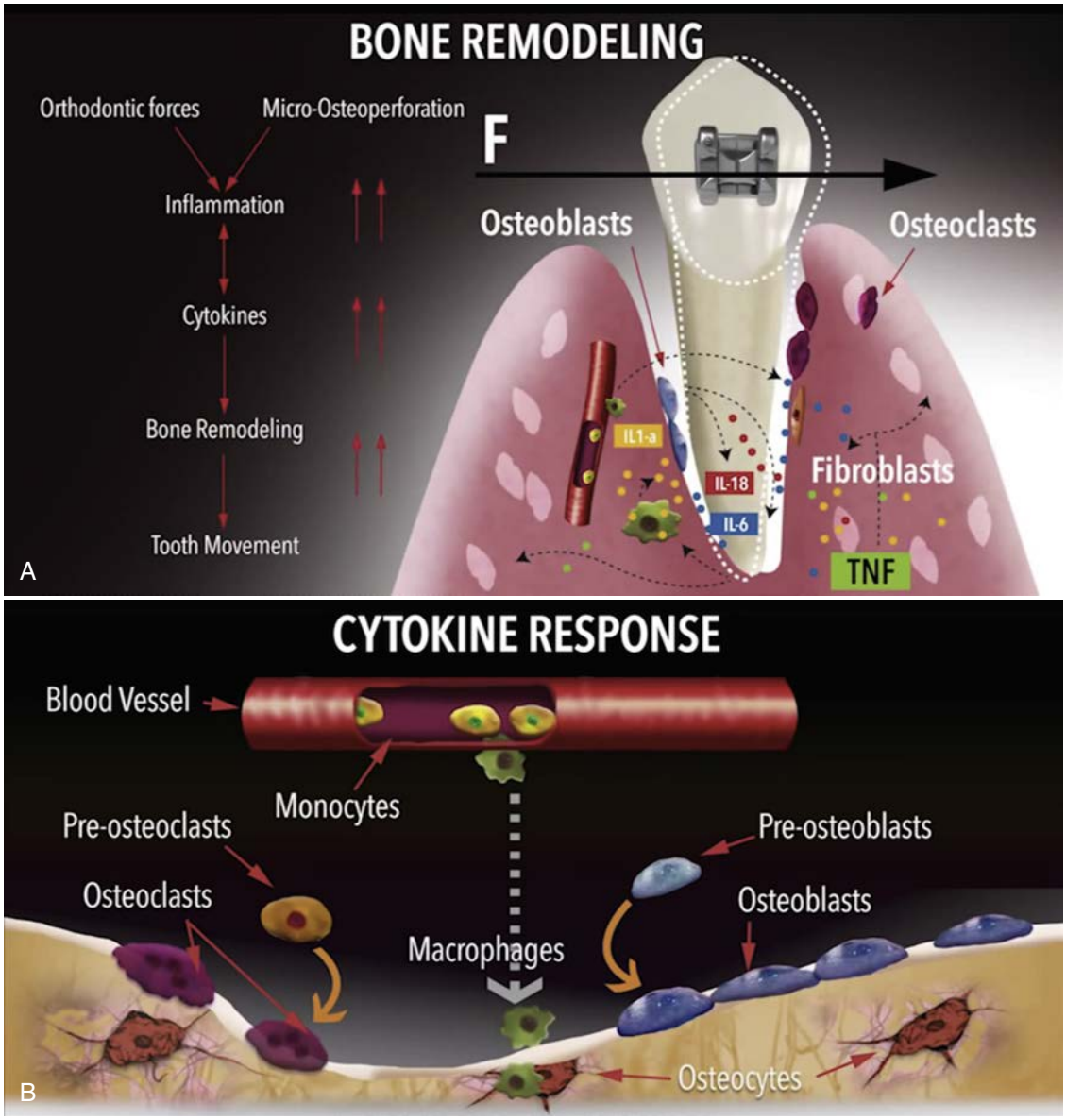


FIGURE 31-1 A, Cascade of events occurs with the initiation of tooth movement. Cytokine chemical messengers mediate osteoblast and osteoclast communication to remodel bone. B, Schematic representation of bone remodeling. *F,* force *g*; *IL1-α,* interleukin-1 alpha; *TNF,* tumor necrosis factor. (From Propel Orthodontics, Ossining, NY.)

thickness should be assessed and accounted for when calculating the depth of the perforations. For example, if osteoperforations are to be performed in the premolar area with a soft tissue thickness of 3 mm, then the selected length of the screw should be no less than 5 mm. In addition, the clinician may feel less resistance once the cortical plate is penetrated.

Once the depth is calculated, the perforations can be performed through the gingiva in the selected area. The perforations can be made either on the attached keratinized tissue or on the free gingival mucosa.

Ideally, three micro-osteoperforations in each interdental space of the area selected should be buccally and/or lingually performed. However, the practitioner may perform as many osteoperforations as desired. The perforations can be made in a linear or triangular distribution (Fig. 31-3). In the areas where performing three or more perforations is anatomically not possible as a result of root proximity, one or two may be sufficient. A patient could receive either a single micro-osteoperforation application or repeated multiple applications at different times to maximize the benefits of the biological stimulation. Studies have reported that an aggressive technique triggers higher osteoclastic activity and/or lower alveolar bone density, which in turn accelerates orthodontic tooth movement.[30] Therefore one can assume that a series of osteoperforations performed at periodic intervals will maintain a high level of inflammation. Further research is still needed to determine the exact number of perforations and frequency that is adequate to achieve the desired biological response.

Minor bleeding may occur after performing the procedure, especially in the alveolar mucosa. In general, minor discomfort is reported. However, some patients may experience some level of pain around the treatment site. If any discomfort is present, then it should be treated with acetaminophen (Tylenol). Nonsteroidal

anti-inflammatory drugs (NSAIDs), such as ibuprofen (Advil), should be completely avoided. NSAIDs may diminish the effect created by the inflammation to accelerate the tooth movement.

Alikhani and colleagues[38] evaluated the effect of micro-osteoperforations on the rate of tooth movement. In this randomized, single-blinded clinical trial, the rate of canine retraction with and without perforations was studied. This trial also evaluated the stimulation of inflammatory markers and the discomfort of the patients during treatment. The sample of 20 patients (ages ranging between 19.5 and 33.1 years) was divided in two groups. The control group consisted of three men and seven women, and the experimental group consisted of five men and five women. Both groups had similar malocclusions (Class II, Division II) and were treated with maxillary first premolar extractions. The retraction mechanics of the maxillary canines consisted of nickel-titanium closing-coil springs activated from a temporary anchorage device (TAD) to a power arm on the canine bracket close to the center of resistance of the tooth. Its activation was delayed 6 months to minimize any inflammatory effect from the extraction procedure. The canines were checked for any occlusal interference that could affect the movement. The experimental group received three micro-osteoperforations distal to the canine on one side only, whereas the control group did not receive any perforations. The rate of canine retraction, measured at 28 days after activation, was a statistically significant (2.3-fold) increase in the experimental side when compared with the control side. Gingival crevicular fluid samples were collected from the canines to evaluate for inflammatory markers. The protein analysis was performed in different time points—before retraction and 24 hours, 7 days, and 28 days after activation. Increases in the levels of cytokines and chemokines analyzed 24 hours in both groups were significant, compared with their baseline values (before retraction). The differences between the two groups were also significantly different, with the experimental group higher than the control group. At day 28, only interluekin-1 was still significantly elevated in the control group and in the experimental group. The difference between the two groups was not statistically significant. Although the patients in this study reported local tolerable discomfort at the experimental site, it did not require additional medication.

FIGURE 31-2 This diagram illustrates the micro-osteoperforation into the alveolar bone. The insertion is performed flapless through the gingiva to reach the alveolar bone. (From Propel Orthodontic, Ossining, NY.)

Indications

Orthodontic tooth movement depends on a multitude of factors, including skeletal pattern of the patient, musculature, occlusal forces, anatomic characteristics of the jaws, and mechanical orthodontic forces.

One important factor is the alveolar bone shape and its density. Lekholm described a classification system of bone and divided it in four categories: type 1 to type 4 (from dense cortical bone to low-density trabecular bone).[64] The mandible generally has denser corticated bone than the maxillae, and in both arches, the thickness of the cortical plate decreases and trabecular space porosity increases moving posteriorly. As the bone density is reduced, the rate of tooth movement increases. For this reason, maxillary molars present a lower degree of

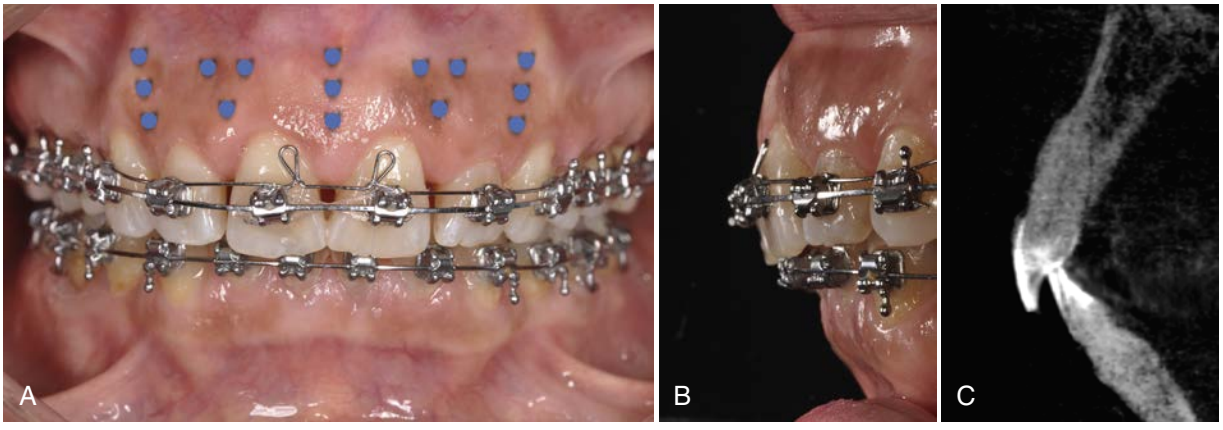


FIGURE 31-3 A 64-year-old woman with minimal tooth movement. **A,** Micro-osteoperforations can be performed in a linear or triangular shape distribution, depending on anatomic limitations. **B,** Note the vertically angulated upper incisors and the torquing auxiliary to upright the root over the alveolar bone. **C,** Cone-beam computed tomography (CBCT) sagittal cut at the level of the upper central incisors shows minimal buccal plate present. Note the root position of the incisor against the cortical plate that may have had an impact on decreasing the rate of tooth movement. In this case, micro-osteoperforations can be buccally performed between the interdental spaces in combination with palatal perforations to accelerate the bone turnover.

anchorage when compared with mandibular molars. A remark supporting this concept is the observation of higher tooth movement in children when compared with adults.[65] Therefore the micro-osteoperforation technique may help surpass these physiologic and anatomic barriers.

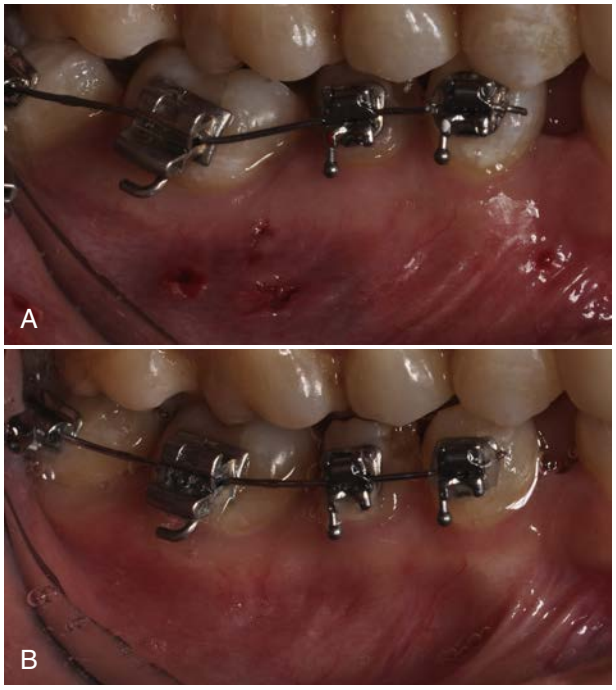


FIGURE 31-4 A, Three micro-osteoperforations were performed between the second premolar and the mesially inclined first molar in the leveling initial phase of treatment. B, The molar was uprighted after one visit. Note healing of the soft tissues.

Orthodontic tooth movement may be problematic when the alveolar width between the buccal and lingual cortical plates is not appropriate to accommodate the complete anatomic dimension of the roots. Consequently, tooth movement through the cortical plate may be reduced and buccal and/or lingual bone dehiscences and/or fenestrations might develop.[66] Therefore a proper periodontal evaluation is crucial (see Chapter 22). Bone augmentation procedures designed to increase the alveolar width in combination with orthodontic treatment are suggested to prevent these undesirable consequences.[67]

Micro-osteoperforations do not change the basal bone and/or alveolar bone. They do not expand the limits of the envelope of discrepancy for the maxillary and mandibular arches; tooth movement is still limited by anatomic alveolar boundaries. Primary indications are to (1) accelerate the rate of tooth movement to shorten the treatment time, (2) facilitate the tooth movement for challenging movements, and (3) modify anchorage.

The technique is contraindicated in systemically compromised patients (American Society of Anesthesiology (ASA) Class III), patients requiring chronic NSAID or steroid prescriptions, and patients in treatment with bisphosphonates. The technique is minimally invasive; however, potential disadvantages include damage to surrounding tissues, root perforation, and potential patient discomfort.

Micro-osteoperforations can be used in many different situations, depending on what is needed for each individual case. The clinician might decide to use this approach in simple cases where crowding and rotations are difficult movements to correct or simply to shorten the treatment time. It could be used during the initial stages of treatment for faster leveling and alignment (Fig. 31-4), during the working stages of space closure (Fig. 31-5), for single or multiple intrusions or extrusions (e.g., to correct a deep bite by intrusion of the lower incisors or to correct an open bite by intrusion of the posterior segments), for protraction (Fig. 31-6) of the dentition to be achieved in

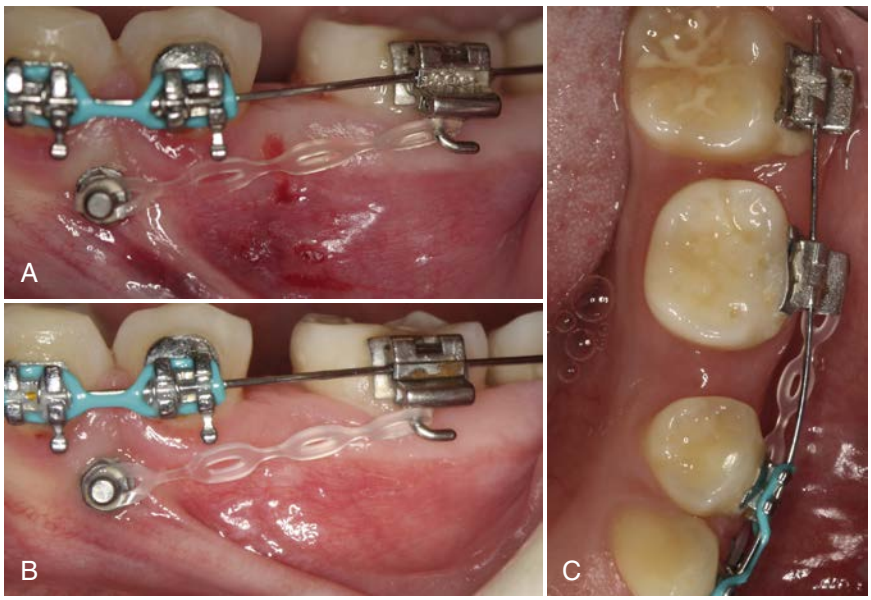


FIGURE 31-5 A, Micro-osteoperforations were performed in a lineal distribution on the open space to accelerate the rate of tooth movement. B, C, Two weeks after the procedure, the tissue is healing within normal limits, and forward movement is being achieved. The beneficial effects of the osteoperforations are accelerating the tooth movement and minimal root resorption of this temporary tooth.

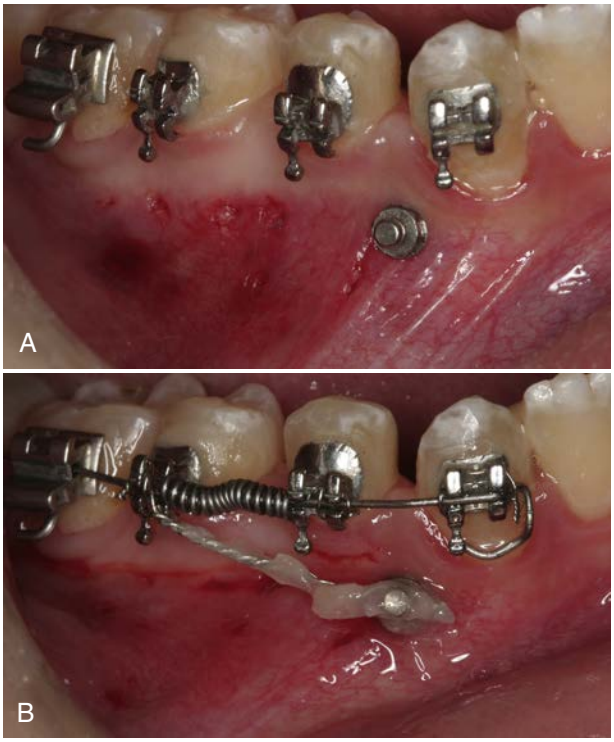


FIGURE 31-6 A, B, Six micro-osteoperforations are made in a triangular shape for protraction on the mandibular segment for faster tooth movement.

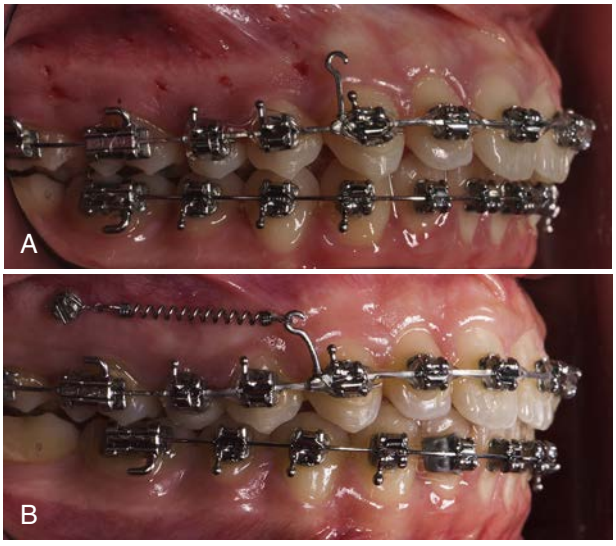


FIGURE 31-7 A 16-year-old female adolescent with unilateral Class II malocclusion and upper midline discrepancy. **A,** Micro-osteoperforations were performed in each interdental space on the posterior segment to maximize the inflammation effects for the challenging movement. **B,** Progress 4.5 months later, with two sessions of osteoperforations. The use of a temporary anchorage device and extraction of the 3rd molar also facilitated the distalization in a more predictable manner.

a more predictable way, and/or for distalization (Fig. 31-7) or mesialization of a single tooth or a whole segment (e.g., a Class II [see Fig. 31-7] or Class III correction). Micro-osteoperforations could be used for any limited treatment or adjunct orthodontics such as molar uprighting. It can be hypothesized that it could diminish the amount of external tooth resorption as a result of the decrease in orthodontic treatment time.[68,69]

In cases where this procedure may facilitate a specific challenging movement, the practitioner should prepare the teeth to get the maximum benefit from the biologic response of the osteoperforations.

In summary, the micro-osteoperforation technique is a clinical approach that can be performed to generally accelerate tooth movement or to achieve a particular movement that is needed in a particular area. Frequent repeated applications (approximately every 4 to 6 weeks until the desired movement is completed) should decrease the treatment time by maintaining a high level of the inflammatory markers that stimulates the osteoblast and osteoclast activity.

Although finishing a case is subjective, there are standards that must be met in every case. Treatment time is an important consideration in orthodontics, but quality should not be compromised for speed.

LOW-LEVEL MECHANICAL VIBRATIONS†

Introduction

A plethora of evidence from orthopedic research suggests that low-level mechanical oscillatory signals (vibrations) have a positive effect on bone metabolism, increasing the rate of remodeling in long bones during adaptation to mechanical loading.[70] This type of mechanical stimulation is currently used as a nonpharmacologic intervention in the prevention of osteoporosis.[71] Results from multiple clinical trials demonstrate that the application of low-magnitude, high-frequency mechanical stimulation causes in an increase in bone density and a decrease in bone loss in postmenopausal women.[72] In addition to clinical trials, animal studies have provided evidence from the cranial suture model[73,74] and long bone periosteum,[75] suggesting that dynamic loading can substantially improve bone formation. Results from a study using a rodent model showed that dynamic vibrational loading increased the rate of orthodontic tooth movement, with no negative side effects on the periodontium and surrounding tissues of the alveolar bone.[76] Loading with a pulsating force for 1.5 hours per day over 3 weeks resulted in approximately 1.3 to 1.4 times greater tooth movement than loading with a static force. However, until recently, clinical evidence concerning the effect of vibratory loading on orthodontic tooth movement was lacking, which prompted several clinical studies discussed in this section.

Clinical Studies

Based on an increasing body of evidence that low-level mechanical cyclic loading results in an anabolic effect on bone metabolism and stimulates remodeling in long bones, vertebrae, and cranial sutures, and that orthodontic tooth movement in rodents is also stimulated by this type of loading, a clinical trial was conducted at the Department of Orthodontics in San Antonio, Texas, to test the effect of vibrations on tooth movement in patients undergoing orthodontic treatment.[77] The objective of this study was to test the hypothesis that low-level mechanical vibration, as an adjunct to standard orthodontic

†Dubravko Pavlin

FIGURE 31-8 A, AcceleDent Aura. B, Patient wears the AcceleDent for 20 minutes per day by lightly biting on the mouthpiece. (Courtesy of OrthoAccel Technologies, Inc., Bellaire, TX.)

treatment, has a stimulatory effect on the rate of tooth movement in orthodontic patients treated for 20 minutes per day by the AcceleDent device (OrthoAccel Technologies, Inc., Bellaire, TX), which delivers a vibrational force of 0.25 newton (N) (25 grams) with a frequency of 30 Hertz (Hz) (Fig. 31-8). This study was preceded by a pilot study[78] and followed by two other clinical studies addressing similar topics, which are discussed later in this chapter.

The San Antonio study conducted at the Department of Orthodontics was a prospective, randomized, controlled, double-blind, parallel group clinical trial (described in detail elsewhere)[77]; a brief summary of its protocol and results is provided. The study fully adheres to the CONSORT guidelines and CONSORT 2010 checklist[79] for conducting and reporting randomized clinical trials (RCTs). The power analysis required a minimum sample size of 32, but the final total sample was increased to 45 subjects. Subjects were randomly assigned to a vibration group (n=23, vibration applied using the AcceleDent device) or to a control group (n=22, using a device in which vibration was internally disabled). A third-party vendor provided a computer-generated randomization schedule. One subject inclusion criterion was that the treatment plan required the extraction of the upper bicuspids. All subjects were treated by orthodontic residents under the supervision of one of the principal investigators. In both groups, a routine set of initial orthodontic records, including study models, photographs, and radiographs, were obtained. All subjects were bonded with a 0.022- × 0.028-inch prescription edgewise MBT Appliance System with twin brackets (3M Unitek, St. Paul, MN). Compliance with the device was assessed using a logbook with a daily record form, requiring the subjects to enter the times of initiation and cessation of device use. In both groups, the use of AcceleDent was prescribed for 20 minutes per day from the start of the treatment.

After the initial alignment, a mini-implant TAD (Tomas [temporary orthodontic micro anchorage system] anchorage pin, Dentaurum GmbH & Co., Ispringen, Germany), 1.6 mm in diameter and 9 mm in length, was inserted into the interdental bone between the roots of the maxillary second premolar and the first molar and immediately loaded using a nickel-titanium closing-coil spring stretched between the mini-implant and the hook on the canine bracket to provide a force of approximately 180 grams (Fig. 31-9). To determine the amount of canine movement, the distance between the canine and the TAD was measured before each closing-coil spring activation using a digital caliper placed parallel to the occlusal plane.

The analysis of the intent-to-treat (ITT) sample (n=45) reported that the rate of tooth movement in the treatment group (1.16 mm/month) was higher than in the control group (0.79 mm/month).[77] The rate of movement of the retracting cuspids in the per protocol (PP) sample (the patients in which treatment was finished according to the protocol n=39) was also faster when vibration was applied. Interpretation of the results is more meaningful by focusing on the ITT sample, since after enrollment and randomization, some subjects (6) were withdrawn from the group for various reasons (pregnancy, small extraction space after alignment, loose TADs), which could result in a bias in the remaining PP group.

The secondary outcome of this RCT was to determine the effect of AcceleDent-induced vibration on the rate of initial alignment of lower anterior teeth. This analysis was conducted by measuring the change in the arch perimeter before and after alignment on the plaster models of the lower arch. Because the arch perimeter measurement is not suitable in nonextraction cases, only the patients with extractions were analyzed, which reduced the sample size. Despite that limitation, initial findings indicate that the rate of alignment was increased by approximately 51% in the subjects exposed to vibration.

Results from other outcomes of this RCT were related to patients' safety, comfort, and ease of use of the AcceleDent device. The most significant safety outcome was to determine whether the low-level vibration increases the risk of external apical root resorption (EARR) during orthodontic treatment. Both cementum and bone are the mineralized tissues of the periodontium that can undergo resorption when exposed to a compressive stress during mechanical loading by orthodontic forces.[80,81] The anabolic effect on bone produced by vibratory (cyclic) loading reflects the increased rates of bone resorption and bone formation, thus raising the concern that tooth cementum could be subject to a higher level of resorption when exposed to concomitant stresses from orthodontic forces and vibrations. The root

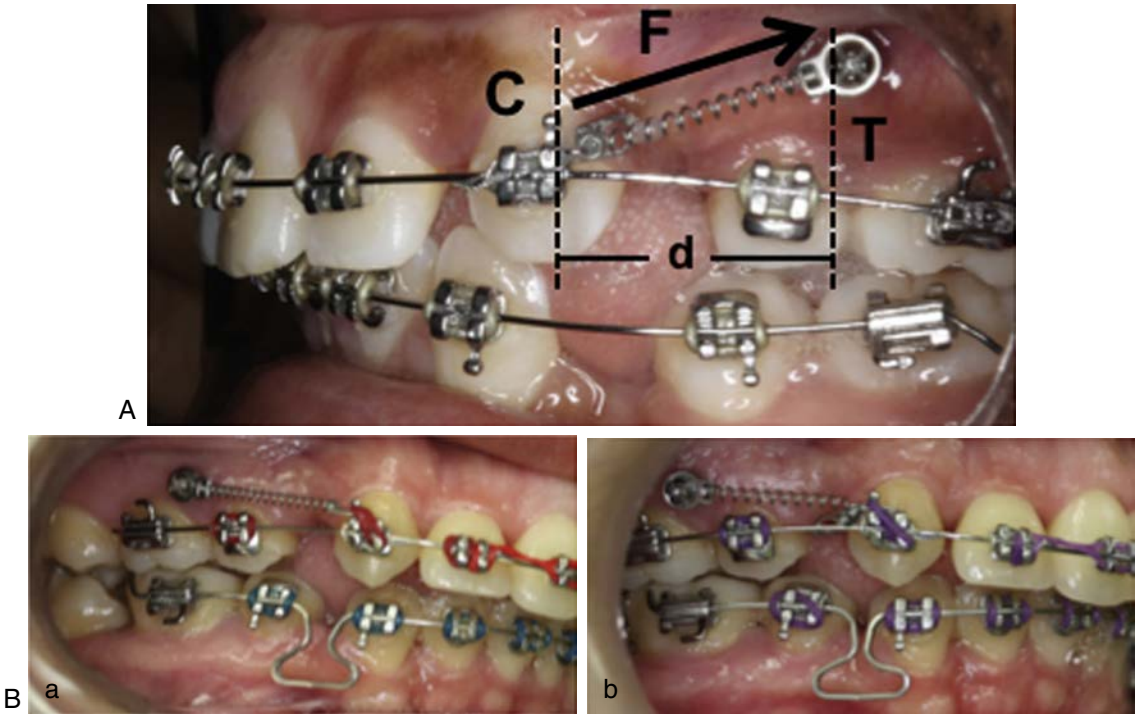


FIGURE 31-9 **A,** Orthodontic appliance for separate canine retraction. *C,* Canine being retracted; *T,* temporary anchorage device (TAD) (Tomas [temporary orthodontic micro-anchorage system] anchorage pin, Dentaurum, GmbH & Co., Ispringen, Germany), 1.6 mm in diameter and 9 mm in length was inserted between the maxillary second premolar and the first molar, under local anesthetic, and immediately loaded; *F,* retraction force of 180 *g* of force, measured by Dontrix gauge (American Orthodontics, Sheboygan, WI) was applied by a nickel-titanium closing-coil spring activated between the TAD and the canine bracket; *d,* distance measured parallel to the occlusal plane using a digital caliper before each closing-coil spring activation or reactivation. An average value from two measurements was entered at each visit; visits were approximately 4 weeks apart. The spring was truncated as needed and re-tied to deliver 180 *g* of force. **B,** Representative example of retraction mechanics for space closure. **a,** The activated closing-coil spring is in place at the beginning of space closure. **b,** One month later, the space opened mesial to the canine.

length analysis was conducted using panoramic radiographs from the subjects participating in the RCT, which were taken in the same laboratory before the start of treatment, at the end of space closure, and at the end of treatment in both the vibration and control groups. Total tooth length was measured using the digital ruler in the Medicor Imaging Picture Archive Communication System (MiPACS, Medicor Imaging, Charlotte, NC). Included were all the teeth from first molar to first molar, except for the first bicuspids because of the variability of root shape. The canines appeared to move predominantly by translation, based on the direction of the reversal lines in alveolar bone adjacent to the root, which represent the areas of the onset of the new bone formation on the tension side of the periodontium. The initial analysis of total tooth length showed no significant difference in this parameter between the AcceleDent device and the sham group at either the end of space closure or the end of treatment, indicating that mechanical vibration as an adjunct to orthodontic loading did not increase the risk of EARR in orthodontic patients.

Several outcomes of this RCT related to the patients' comfort and ease of using the AcceleDent device. The subjects were assessed at each study visit using a visual analog scale (VAS) to evaluate the following parameters: discomfort, hygiene, drooling, schedule disruption, reliability, ease of use, noise, cleanliness and maintenance, and overall satisfaction with the device. The VAS was based on a 100-point scale, and the scores from the control and AcceleDent groups were compared. The initial assessments are showing very similar VAS scores for both groups, indicating that the patients are very satisfied with treatment and the device is easy to use without disruption of their daily activities.

In addition to the RCT focusing on cuspid retraction discussed previously, the increased interest in accelerated tooth movement has resulted in several recent studies that focused on the effect of supplemental vibratory loading during the initial alignment/leveling stage of treatment. Miles and colleagues conducted a randomized trial with 66 patients, using the Tooth Masseuse device[82]. They found no effect on the rate of reduction of irregularity during anterior alignment. They also did not find any difference in alleviation of pain.

Three other studies have been published on the AcceleDent device. The most recent, by Woodhouse and colleagues, was a three-armed randomized trial, comparing AcceleDent with fixed appliances, sham AcceleDent with fixed appliances, and

FIGURE 31-10 Invisalign case, courtesy of Dr. Sam Daher. Number of aligners: 52; refinement aligners: 7; total: 59 aligners. Normal treatment time: 59 × 2 weeks = 118 weeks (2 years and 3 months). Treated with AcceleDent: aligners changed as follows: 7 days for initial 52 aligners and every 5 days for the 7 refinement aligners. Total treatment time (actual) was 1 year, 1 month. A: Initial, B: Progress, C: Final.

fixed appliances only[83]. The authors reported no difference in the time to reach initial or final tooth alignment among the three groups. Bowman in a retrospective study, reported a faster rate of alignment in an AcceleDent group, but also stated that the difference was not statistically different[84]. Bowman did report a significantly faster rate of leveling in the AcceleDent group. In addition, Kau and colleagues reported on a group of 14 patients (case series) all treated with fixed appliances and the AcceleDent device. Although there was no comparison group, the authors reported that the rate of tooth movement seen in their study was 2-3 times faster than conventional tooth movement (estimated to be about 1 mm per month)[78] .

Vibration and Treatment with Clear Aligners

The use of the AcceleDent device as an adjunct to treatment with Invisalign is becoming increasingly popular among the clinicians in the United States and in other parts of the world. Although no clinical studies have been published on this subject, numerous personal reports of practitioners indicate that the treatment time can be cut by 50% or more when the AcceleDent device is used during treatment with Invisalign. The proponents of this approach report that the time for wearing each aligner can be cut down from the prescribed 2 weeks to 1 week or less when vibration is applied. Studies are needed to determine the mechanics underlying such a sharp decrease in treatment time. The fact that the tight contact of the aligner with the entire tooth surface allows a more efficient transmission of vibration to the root and surrounding bone is a logical speculation. Figure 31-10 shows a representative patient with Invisalign aligner trays whose treatment time was reduced by more than 50% with the adjunctive use of the AcceleDent device.

Biological Mechanism of Bone Response to Vibration

The biological mechanism underlying the anabolic effect of cyclic loading on bone metabolism is not fully understood. Several signaling pathways have been implied in the response of bone cells to mechanical loading,[85,86] and further studies are needed to determine which of these pathways is activated by low-level vibrations. Since oscillatory forces, even at the extremely low levels used in this study, are readily transmitted beyond the bone surface and into the deep compartments of bone, the speculation that the initial response occurs in the cells embedded in bone matrix, such as osteocytes, is reasonable.

B

C

FIGURE 31-10, cont'd

Our group and others have previously identified osteocytes as the early mechanoresponsive cells in the alveolar bone during orthodontic tooth movement.[87] As early as 6 hours after the onset of mechanical loading, a surge in the expression of genes for osteocalcin and dentin matrix protein was detected in the alveolar osteocytes. These mechanically induced signaling pathways could be triggered by fluid shear stress in osteocyte lacunae and canaliculi or by piezoelectric potentials induced by bone bending, all of which can occur during vibrations. In addition, bone microfractures, at the level similar to or lower than those exerted by physical activity, may be a contributing factor in the early response to oscillatory loading. Further studies showed that these early mechanoresponsive events in osteocytes are followed by increased differentiation of osteoblasts[88] and stimulation of bone characteristic genes (alkaline phosphatase, type I collagen) in these cells after 24 hours and up to 5 days.[89–90] Because similar signaling and gene regulatory events are most likely involved in response to orthodontic loading of bone with and without vibrations, one of the key questions to be addressed in future studies is whether cyclic loading superimposed on the force systems from an orthodontic appliance produces a faster and quantitatively higher levels of the same types of early anabolic signals or if completely different pathways of signaling and genetic responses are initiated that result in stimulation of bone remodeling and faster tooth movement.

REFERENCES

References for Micro-Osteoperforations

1. Fink DF, Smith RJ. The duration of orthodontic treatment. *Am J Orthod Dentofacial Orthop*. 1992;102(1):45–51.
2. Fisher MA, Wenger RM, Hans MG. Pretreatment characteristics associated with orthodontic treatment duration. *Am J Orthod Dentofacial Orthop*. 2010;137(2):178–186.
3. Budd S, Daskalogiannakis J, Tompson BD. A study of the frictional characteristics of four commercially available self-ligating bracket systems. *Eur J Orthod*. 2008;30(6):645–653.
4. Chen SS, Greenlee GM, Kim JE, et al. Systematic review of self-ligating brackets. *Am J Orthod Dentofacial Orthop*. 2010;137(6): 726.e721–726, e718; discussion 726–727.
5. Henao SP, Kusy RP. Frictional evaluations of dental typodont models using four self-ligating designs and a conventional design. *Angle Orthod*. 2005;75(1):75–85.
6. Alford TJ, Roberts WE, Hartsfield Jr JK, et al. Clinical outcomes for patients finished with the SureSmile™ method compared with conventional fixed orthodontic therapy. *Angle Orthod*. 2011;81(3):383–388.
7. Saxe AK, Louie LJ, Mah J. Efficiency and effectiveness of SureSmile. *World J Orthod*. 2010;11(1):16–22.
8. Grauer D, Wiechmann D, Heymann GC, et al. Computer-aided design/computer-aided manufacturing technology in customized orthodontic appliances. *J Esthet Restor Dent*. 2012;24(1):3–9.
9. Koo BC, Chung CH, Vanarsdall RL. Comparison of the accuracy of bracket placement between direct and indirect bonding techniques. *Am J Orthod Dentofacial Orthop*. 1999;116(3):346–351.
10. Silverman E, Cohen M, Gianelly AA, et al. A universal direct bonding system for both metal and plastic brackets. *Am J Orthod*. 1972;62(3):236–244.
11. Cruz DR, Kohara EK, Ribeiro MS, Wetter Nu. Effects of low-intensity laser therapy on the orthodontic movement velocity of human teeth: a preliminary study. *Lasers Surg Med*. 2004;35(2):117–120.
12. Yamaguchi M, Hayashi M, Fujita S, et al. Low-energy laser irradiation facilitates the velocity of tooth movement and the expressions of matrix metalloproteinase-9, cathepsin k, and alpha(v) beta(3) integrin in rats. *Eur J Orthod*. 2010;32(2):131–139.
13. Campanha BP, Gallina C, Geremia T, et al. Low-level laser therapy for implants without initial stability. *Photomed Laser Surg*. 2010;28(3): 365–369.
14. Altan BA, Sokucu O, Ozkut MM, Inan S. Metrical and histological investigation of the effects of low-level laser therapy on orthodontic tooth movement. *Lasers Med Sci*. 2012;27(1):131–140.
15. Fujita S, Yamaguchi M, Utsunomiya T, et al. Low-energy laser stimulates tooth movement velocity via expression of RANK and RANKL. *Orthod Craniofac Res*. 2008;11(3):143–155.
16. Beeson DC, Johnston LE, Wisotzky J. Effect of constant currents on orthodontic tooth movement in the cat. *J Dent Res*. 1975;54(2):251–254.
17. Davidovitch Z, Finkelson MD, Steigman S, Shanfeld JL. Electric currents, bone remodeling, and orthodontic tooth movement. I. The effect of electric currents on periodontal cyclic nucleotides. *Am J Orthod*. 1980;77(1):14–32.
18. Davidovitch Z, Finkelson MD, Steigman S, Shanfeld JL. Electric currents, bone remodeling, and orthodontic tooth movement. II. Increase in rate of tooth movement and periodontal cyclic nucleotide levels by combined force and electric current. *Am J Orthod*. 1980;77(1):33–47.
19. Kim DH, Park YG, Kang SG. The effects of electrical current from a micro-electrical device on tooth movement. *Korean J Orthod*. 2008;38(5):337–346.
20. Showkatbakhsh R, Jamilian A, Showkatbakhsh M. The effect of pulsed electromagnetic fields on the acceleration of tooth movement. *World J Orthod*. 2010;11(4):e52–e56.
21. Shapiro E, Roeber FW, Klempner LS. Orthodontic movement using pulsating force-induced piezoelectricity. *Am J Orthod*. 1979;76(1): 59–66.
22. Garlet TP, Coelho U, Silva JS, Garlet GP. Cytokine expression pattern in compression and tension sides of the periodontal ligament during orthodontic tooth movement in humans. *Eur J Oral Sci*. 2007;115(5): 355–362.
23. Lee KJ, Park YC, Yu HS, et al. Effects of continuous and interrupted orthodontic force on interleukin-1beta and prostaglandin E2 production in gingival crevicular fluid. *Am J Orthod Dentofacial Orthop*. 2004;125(2):168–177.
24. Leiker BJ, Nanda RS, Currier GF, et al. The effects of exogenous prostaglandins on orthodontic tooth movement in rats. *Am J Orthod Dentofacial Orthop*. 1995;108(4):380–388.
25. Yamasaki K, Miura F, Suda T. Prostaglandin as a mediator of bone resorption induced by experimental tooth movement in rats. *J Dent Res*. 1980;59(10):1635–1642.
26. Liu ZJ, King GJ, Gu GM, et al. Does human relaxin accelerate orthodontic tooth movement in rats? *Ann N Y Acad Sci*. 2005;1041:388–394.
27. Madan MS, Liu ZJ, Gu GM, King GJ. Effects of human relaxin on orthodontic tooth movement and periodontal ligaments in rats. *Am J Orthod Dentofacial Orthop*. 2007;131(1):8.e1–8.e10.
28. McGorray SP, Dolce C, Kramer S, et al. A randomized, placebo-controlled clinical trial on the effects of recombinant human relaxin on tooth movement and short-term stability. *Am J Orthod Dentofacial Orthop*. 2012;141(2): 196–203.
29. Stewart DR, Sherick P, Kramer S, Breining P. Use of relaxin in orthodontics. *Ann N Y Acad Sci*. 2005;1041:379–387.
30. Liou EJ. Accelerated orthodontic tooth movement. In: Miles PG, Rinchuse DJ, Rinchuse DJ, eds. *Evidence-Based Clinical Orthodontics*. Chicago: Quintessence Publishing Company; 2012:179–199.
31. Wilcko WM, Wilcko T, Bouquot JE, Ferguson DJ. Rapid orthodontics with alveolar reshaping: two case reports of decrowding. *Int J Periodontics Restorative Dent*. 2001;21(1):9–19.
32. Hoogeveen EJ, Jansma J, Ren Y. Surgically facilitated orthodontic treatment: a systematic review. *Am J Orthod Dentofacial Orthop*. 2014;145(4 Suppl):S51–S64.
33. Iseri H, Kisnisci R, Bzizi N, Tüz H. Rapid canine retraction and orthodontic treatment with dentoalveolar distraction osteogenesis. *Am J Orthod Dentofacial Orthop*. 2005;127(5):533–541; quiz 625.
34. Liou EJ, Figueroa AA, Polley JW. Rapid orthodontic tooth movement into newly distracted bone after mandibular distraction osteogenesis in a canine model. *Am J Orthod Dentofacial Orthop*. 2000;117(4):391–398.

35. Liou EJ, Huang CS. Rapid canine retraction through distraction of the periodontal ligament. *Am J Orthod Dentofacial Orthop*. 1998;114(4):372–382.
36. Kim SJ, Park YG, Kang SG. Effects of corticision on paradental remodeling in orthodontic tooth movement. *Angle Orthod*. 2009;79(2):284–291.
37. Murphy CA, Chandhoke T, Kalajzic Z, et al. Effect of corticision and different force magnitudes on orthodontic tooth movement in a rat model. *Am J Orthod Dentofacial Orthop*. 2014;146(1):55–66.
38. Alikhani M, Raptis M, Zoldan B, et al. Effect of micro-osteoperforations on the rate of tooth movement. *Am J Orthod Dentofacial Orthop*. 2013;144(5):639–648.
39. Alikhani M, Khoo E, Alyami B, et al. Osteogenic effect of high-frequency acceleration on alveolar bone. *J Dent Res*. 2012;91(4):413–419.
40. Nishimura M, Chiba M, Ohashi T, et al. Periodontal tissue activation by vibration: intermittent stimulation by resonance vibration accelerates experimental tooth movement in rats. *Am J Orthod Dentofacial Orthop*. 2008;133(4):572–583.
41. Pavlin D, Anthony R, Raj V, Gakunga PT. Cyclic loading (vibration) accelerates tooth movement in orthodontic patients: a double-blind, randomized controlled trial. *Semin Orthod*. 2015;21(3):187–194.
42. Gkantidis N, Mistakidis I, Kouskoura T, Pandis N. Effectiveness of non-conventional methods for accelerated orthodontic tooth movement: a systematic review and meta-analysis. *J Dent*. 2014;42(10):1300–1319.
43. Long H, Pyakurel U, Wang Y, et al. Interventions for accelerating orthodontic tooth movement: a systematic review. *Angle Orthod*. 2013;83(1):164–171.
44. Frost HM. The biology of fracture healing. an overview for clinicians. Part I. *Clin Orthop Relat Res*. 1989;248:283–293.
45. Frost HM. The biology of fracture healing. an overview for clinicians. Part II. *Clin Orthop Relat Res*. 1989;248:294–309.
46. Burstone CJ. The biomechanics of tooth movement. In: Kraus BS, Riedel RA, eds. *Vistas in Orthodontics*. Philadelphia: Lea & Febiger; 1962:197–213.
47. Krishnan V, Davidovitch Z. Cellular, molecular, and tissue-level reactions to orthodontic force. *Am J Orthod Dentofacial Orthop*. 2006;129(4):469.e1–469.e32.
48. Reitan K. Tissue behavior during orthodontic tooth movement. *Am J Orthod*. 1960;46:881–900.
49. Sprogar S, Vaupotic T, Cör A, et al. The endothelin system mediates bone modeling in the late stage of orthodontic tooth movement in rats. *Bone*. 2008;43(4):740–747.
50. Tan SD, Xie R, Klein-Nulend J, et al. Orthodontic force stimulates eNOS and iNOS in rat osteocytes. *J Dent Res*. 2009;88(3):255–260.
51. Pacios S, Kang J, Galicia J, et al. Diabetes aggravates periodontitis by limiting repair through enhanced inflammation. *FASEB J*. 2012;26(4):1423–1430.
52. Masella RS, Meister M. Current concepts in the biology of orthodontic tooth movement. *Am J Orthod Dentofacial Orthop*. 2006;129(4):458–468.
53. Karsenty G. The complexities of skeletal biology. *Nature*. 2003;423(6937):316–318.
54. Uematsu S, Mogi M, Deguchi T. Interleukin (IL)-1 beta, IL-6, tumor necrosis factor-alpha, epidermal growth factor, and beta 2-microglobulin levels are elevated in gingival crevicular fluid during human orthodontic tooth movement. *J Dent Res*. 1996;75(1):562–567.
55. Lowney JJ, Norton LA, Shafer DM, Rossomando EF. Orthodontic forces increase tumor necrosis factor alpha in the human gingival sulcus. *Am J Orthod Dentofacial Orthop*. 1995;108(5):519–524.
56. Jäger A, Zhang D, Kawarizadeh A, et al. Soluble cytokine receptor treatment in experimental orthodontic tooth movement in the rat. *Eur J Orthod*. 2005;27(1):1–11.
57. Chae HS, Park HJ, Hwang HR, et al. The effect of antioxidants on the production of pro-inflammatory cytokines and orthodontic tooth movement. *Mol Cells*. 2011;32(2):189–196.
58. Knop LA, Shintcovsk RL, Retamoso LB, et al. Non-steroidal and steroidal anti-inflammatory use in the context of orthodontic movement. *Eur J Orthod*. 2012;34(5):531–535.
59. Iwasaki LR, Haack JE, Nickel JC, et al. Human interleukin-1 beta and interleukin-1 receptor antagonist secretion and velocity of tooth movement. *Arch Oral Biol*. 2001;46(2):185–189.
60. Yoshimatsu M, Shibata Y, Kitaura H, et al. Experimental model of tooth movement by orthodontic force in mice and its application to tumor necrosis factor receptor-deficient mice. *J Bone Miner Metab*. 2006;24(1):20–27.
61. Teixeira CC, Khoo E, Tran J, et al. Cytokine expression and accelerated tooth movement. *J Dent Res*. 2010;89(10):1135–1141.
62. Kravitz ND. The use of compound topical anesthetics: a review. *J Am Dent Assoc*. 2007;138(10):1333–1339; quiz 1382.
63. Katranji A, Misch K, Wang HL. Cortical bone thickness in dentate and edentulous human cadavers. *J Periodontol*. 2007;78(5):874–878.
64. Lekholm U, Zarb GA. Patient selection and preparation. In: Branemark PI, Zarb GA, Albrektsson T, eds. *Tissue Integrated Prostheses: Osseointegration in Clinical Dentistry*. Chicago: Quintessence Publishing Company; 1985:199–209.
65. Roberts WE. Bone physiology, metabolism, and biomechanics in orthodontic practice. In: Graber TM, Vanarsdall RL, Vig KW, eds. *Orthodontics: Current Principles and Techniques*. 4th ed. St. Louis: Mosby; 2005:221–292.
66. Köle H. Corticalisschwächung zur Unterstützung bei der kieferorthopädischen Behand-lung. *Fortschr KieferGesichtschir*. 1958;4:161–166.
67. Diedrich PR. Guided tissue regeneration associated with orthodontic therapy. *Semin Orthod*. 1996;2(1):39–45.
68. Fox N. Longer orthodontic treatment may result in greater external apical root resorption. *Evid Based Dent*. 2005;6(1):21.
69. Segal GR, Schiffman PH, Tuncay OC. Meta analysis of the treatment-related factors of external apical root resorption. *Orthod Craniofac Res*. 2004;7(2):71–78.

References for Low-Level Mechanical Vibrations

70. Rubin C, Turner AS, Müller R, et al. Quantity and quality of trabecular bone in the femur are enhanced by a strongly anabolic, noninvasive mechanical intervention. *J Bone Miner Res*. 2002;17(2):349–357.
71. Rubin C, Judex S, Qin YX. Low-level mechanical signals and their potential as a non-pharmacological intervention for osteoporosis. *Age Ageing*. 2006;35(Suppl 2):ii32–ii36.
72. Rubin C, Recker R, Cullen D, et al. Prevention of postmenopausal bone loss by a low-magnitude, high-frequency mechanical stimuli: a clinical trial assessing compliance, efficacy, and safety. *J Bone Miner Res*. 2004;19(3):343–351.
73. Kopher RA, Mao JJ. Suture growth modulated by the oscillatory component of micromechanical strain. *J Bone Miner Res*. 2003;18(3):521–528.
74. Kopher RA, Nudera JA, Wang X, et al. Expression of in vivo mechanical strain upon different wave forms of exogenous forces in rabbit craniofacial sutures. *Ann Biomed Eng*. 2003;31(9):1125–1131.
75. Peptan AI, Lopez A, Kopher RA, Mao JJ. Responses of intramembranous bone and sutures upon in vivo cyclic tensile and compressive loading. *Bone*. 2008;42(2):432–438.
76. Nishimura M, Chiba M, Ohashi T, et al. Periodontal tissue activation by vibration: intermittent stimulation by resonance vibration accelerates experimental tooth movement in rats. *Am J Orthod Dentofacial Orthop*. 2008;133(4):572–583.
77. Pavlin D, Anthony R, Raj V, Gakunga PT. Cyclic loading (vibration) accelerates tooth movement in orthodontic patients: a double-blind, randomized controlled trial. *Semin Orthod*. 2015;21(3):187–194.
78. Kau CH, Nguyen JT, English JD. The clinical evaluation of a novel cyclical force generating device in orthodontics. *Orthod Prac*. 2010;1:1–4.
79. Schulz KF, Altman DG, Moher D. CONSORT Group. CONSORT 2010 statement: updated guidelines for reporting parallel group randomised trials. *BMJ*. 2010;340:c332.
80. Segal GR, Schiffman PH, Tuncay OC. Meta analysis of the treatment-related factors of external apical root resorption. *Orthod Craniofac Res*. 2004;7(2):71–78.
81. Weltman B, Vig KW, Fields HW, et al. Root resorption associated with orthodontic tooth movement: a systematic review. *Am J Orthod Dentofacial Orthop*. 2010;137(4):462–476; discussion 412A.
82. Miles P, Smith H, Weyant R, Rinchuse DJ. The effects of a vibrational appliance on tooth movement and patient discomfort: a prospective randomised clinical trial. *Aust Orthod J*. 2012;28(2):213–218.

83. Woodhouse NR, DiBiase AT, et al. Supplemental vibrational force during orthodontic alignment: a randomized trial. *J Dent Res*. 2015;94(5):682–689.
84. Bowman SJ. The effect of vibration on the rate of leveling and alignment. *J Clin Orthod*. 2014;48(11):678–688.
85. Rubin J, Rubin C, Jacobs CR. Molecular pathways mediating mechanical signaling in bone. *Gene*. 2006;367:1–16.
86. Pavlin D, Gluhak-Heinrich J. Effect of mechanical loading on periodontal cells. *Crit Rev Oral Biol Med*. 2001;12(5):414–424.
87. Gluhak-Heinrich J, Ye L, Bonewald LF, et al. Mechanical loading stimulates dentin matrix protein 1 (DMP1) expression in osteocytes in vivo. *J Bone Miner Res*. 2003;18(5):807–817.
88. Pavlin D, Goldman ES, Gluhak-Heinrich J, et al. Orthodontically stressed periodontium of transgenic mouse as a model for studying mechanical response in bone: the effect on the number of osteoblasts. *Clin Orthod Res*. 2000;3(2):55–66.
89. Pavlin D, Dove SB, Zadro R, et al. Mechanical loading stimulates differentiation of periodontal osteoblasts in a mouse osteoinduction model: effect on type I collagen and alkaline phosphatase genes. *Calcif Tissue Int*. 2000;67(2):163–172.
90. Pavlin D, Zadro R, Gluhak-Heinrich J. Temporal pattern of stimulation of osteoblast-associated genes during mechanically-induced osteogenesis in vivo: early responses of osteocalcin and type I collagen. *Connect Tissue Res*. 2001;42(2):135–148.

32

Biodigital Orthodontics: Integrating Technology with Diagnosis, Treatment Planning, and Targeted Therapeutics

Rohit ChamanLal Sachdeva

OUTLINE

Introduction, 926
Current Craft-Based Care Model, 926
Patient-Centered and Professional-Based Model of Care, 927
Integrated Digital Technology Platform: SureSmile and the OraMetrix Digital Laboratory, 927
Technology, 927
OraMetrix Digital Laboratory and Manufacturing, 930
SureSmile in Clinical Practice, 930
Pre-SureSmile Therapeutic Phase: The Digital Diagnostic Model, 930
SureSmile Therapeutic Phase, 934
Outcome Evaluation Phase, 942
Proactive Decision Making, 942
Principle of Smart Orthodontics, 942
Principle of Risk Analysis, 944
Principle of Constraint Recognition and Management, 944
Principle of Anticipatory Orthodontics, 944
Principles of Targeted Therapeutics, 946
Adaptive Prescription, 946
Principle of Image-Guided Smart Bracket Placement and Archform Selection, 947
Principle of Minimal Archwire Replacement, 947
Principle of Constraint Management, 950
Principle of Concurrent Mechanics, 950
Principle of Additive or Subtractive Bending, Preferential Staging, and Shaping, 951
Principles of Consistent Force Systems and Reactive Force Management, 954
Principle of Complementary Force System, 954
Principle of Optimization of Archwire and Material Selection, 954
Efficacy and Effectiveness, 957
Summary, 958

INTRODUCTION

The integration of digital technologies in diagnosis, treatment planning, appliance design, construction, and manipulation is changing the manner in which orthodontics is provided to patients. New opportunities for improved treatment, coupled with shorter and more comfortable patient experiences, are now available with these new paradigms. This chapter describes the practice of diagnostically driven, robotic-assisted (DDRA) orthodontics using as the technology example SureSmile technology: a total patient management system (OraMetrix, Inc., Richardson, TX). The foundation of DDRA orthodontics lies in a patient-centered, professional-based care model. The goal of DDRA orthodontics is to improve treatment outcomes continuously by providing targeted personalized care and minimizing variation in all aspects of care delivery and error proofing. Patient care is managed by applying sound principles grounded in conventional orthodontic biomechanics, using SureSmile's integrated digital technology platform and applying unique diagnostic, care planning, and therapeutic strategies developed, in large degree, by the author of this chapter. These strategies are enabled by capabilities that include three-dimensional (3D) imaging, a decision support system, robotic-assisted archwire bending,[1–7] and 3D printing with stereolithography (STL).

CURRENT CRAFT-BASED CARE MODEL

The current practice of orthodontic care may be characterized as *reactive*.[1] It has historically been appliance driven and error prone. Error is seeded early in the treatment as a result of a relatively cursory diagnosis and a limited treatment plan. The therapeutic strategy with the straight archwire approach to treatment relies on the intelligence built into the bracket prescription and its placement. In reality, great variation exists in the prescription of the brackets because of inaccurate manufacturing practices. Furthermore, both direct and indirect bonding bracket placement procedures are fraught with errors driven by perception, lack of reliable global and local reference coordinates (because of differences in tooth morphologic structure), and technique sensitivity. These sources of variations add to the error trap.[2]

Initially, treatment begins with the objective of removing "noise" in the malocclusion through alignment and leveling

through a replacement approach—for example, the use of a series of archwires that are progressively changed until a working wire is established. Transition to this archwire generally marks the beginning of the finishing stage of treatment. The choice of archwires and archform by a clinician is commonly based on feel, clinical judgment, philosophic bent, or historical holdover. This approach to treatment adds another layer of complexity to patient care that consists of variation and error.

Generally, finishing begins approximately 1 year into the care cycle. In reality, this is often the error correction phase of treatment, which is managed very inefficiently through reactive processes. The doctor is challenged in managing issues arising from inadequate diagnosis and care planning, vagaries in appliance prescription, and misapplication and mismanagement of therapeutic appliances, all at a time when patient compliance to treatment is at its lowest ebb (Fig. 32-1, *A*, *B*).

This means that for all extant purposes, the patient is essentially placed into a doctor-devised orthodontic "intensive care unit" style of treatment in which the doctor applies every conceivable therapeutic strategy at his or her disposal to *rescue* the patient. This treatment can include bracket repositioning and/or archwire modification through manual bending,* reinforcement of greater patient compliance to wear elastics, or even reconsideration of treatment objectives.

This cycle of reactive therapeutics is driven by the fact that the clinician lacks the tools to validate the robustness of his or her decisions and has little assurance that the bracket repositions or the archwire bends are correct. As a result, the clinician awaits the patient's next visit to assess treatment response to verify the soundness of his or her decisions and the interventions made during the previous visit.

Unfortunately, the time dependency of this response adds another layer of complexity. A judgment has to be made as to when the next patient visit should be scheduled. Typically, at this point, the clinician resorts to following the patient at shorter intervals. The irony of this approach to managing care is that it triggers overuse of therapeutic interventions and invariably disrupts practice scheduling, jaundices the patient's care experience, and negatively affects the patient's lifestyle.

In summary, the current care model in managing orthodontic patients is *craft-based* and *doctor-centric* and is therapeutically driven by personal knowledge that is immune to clinical benchmarking. Customization is handcrafted, which increases the risk of vulnerability to both variation and error in care. Such treatment practices add to high internal costs, increase the care cycle, and place the patient at risk.

PATIENT-CENTERED AND PROFESSIONAL-BASED MODEL OF CARE

Professional-based care is diagnostically driven, proactively managed, and evidence based. The doctor is systems minded and manages care through streamlined processes and the use of appropriate technologies to minimize variation; its goal is to deliver error-proof care. The doctor promotes a generative practice environment with continuous quality improvement initiatives. Finally, the professional caregiver is mindful of waste and seeks to lower internal costs and shorten the care cycle (Fig. 32-2, *A–D*).

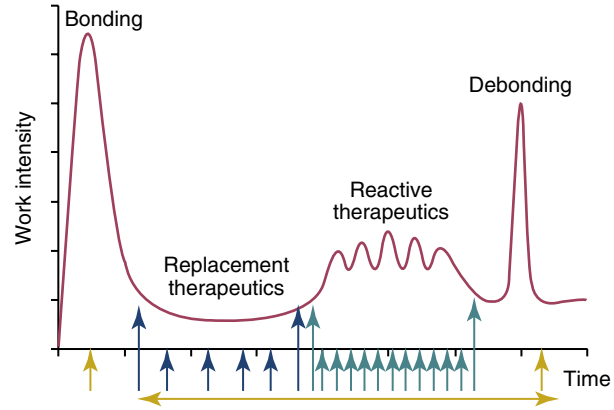
INTEGRATED DIGITAL TECHNOLOGY PLATFORM: SURESMILE AND THE ORAMETRIX DIGITAL LABORATORY

Technology

To demonstrate a global integration of technology to assist in patient care, the SureSmile system (OraMetrix, Inc., Richardson, TX) has been selected and will remain the focus of this chapter. There are other systems and some to come that also use some of the concepts articulated herein, but at the date of this publication, SureSmile provides the most comprehensive approach for clinicians, using a wide variety of labial, lingual, and aligner appliances. SureSmile is an integrated series of digital technologies, services, and product solutions that enables the orthodontist to practice a patient-centered, professional-based care model. The hardware technology includes a 3D image acquisition device (Fig. 32-3, *A*) and/or the use of 3D cone-beam computed tomography (CBCT) scans. SureSmile's software solution provides a comprehensive set of utilities that help the practice manage the patient in both the physical and virtual world. The software provides such tools as (1) image management; (2) patient tracking; (3) workflow automation; (4) decision support; (5) prescriptive archwire design and fabrication, indirect bonding trays, and aligner design; (6) outcome evaluation; and (7) patient reports. These tools allow the clinician to diagnose, communicate, plan care, and design prescriptive customized orthodontic archwires, custom removable tooth aligners, and indirect bonding trays (Fig. 32-3, *B*).

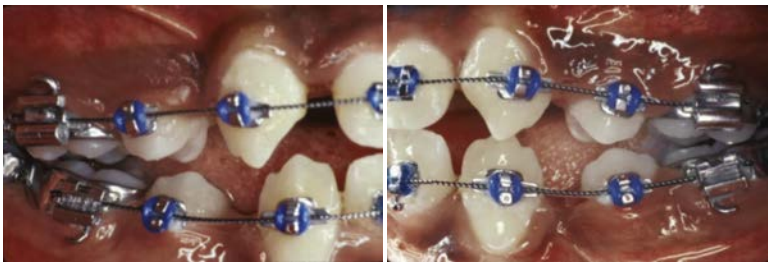
Care in the digital environment with SureSmile is entirely managed in a workflow process–driven environment. The orthodontist's care plan is used to create a target setup that, in turn, provides input parameters to drive a six-axis, archwire-bending robot remotely situated from the practice (Fig. 32-3, *E*). Materials such as copper-nickel-titanium (CuNiTi), beta titanium (titanium molybdenum alloy [TMA]), Elgiloy (Rocky Mountain Orthodontics, Denver, CO), and Azurloy (Ormco, Glendora, CA), comprising a range of cross sections (0.16- to 0.19-inch × 0.25-inch) and shapes (round, square, and rectangular), are bent by the robot to accomplish tooth movement prescribed by the orthodontist, with an angular and linear accuracy of 1 degree and 0.1 mm, respectively. Archwires can be for both labial and lingual techniques and for passive fixed retainers. Such versatility offers unlimited control to the doctor to achieve his or her treatment objectives. Furthermore, archwire design is not limited by bracket choice, and brackets can be placed on the labial or lingual aspects, depending on the therapeutic choice. The clinician is free to use any bracket system or prescription. In addition, the clinician can design aligners with SureSmile software and obtain staged models from OraMetrix to fabricate aligners in his or her office. If the doctor prefers, he or she can obtain STL files of their SureSmile patients and have the staged models printed by their preferred laboratory. Last, customized indirect bonding trays can be designed with SureSmile software with the indirect trays printed by OraMetrix.

* OraMetrix has introduced indirect bonding trays to reduce the error of the method. The results of clinical testing suggest that 3D printed trays enable bracket placement with the precision of 3 degrees and .2mm for horizontal and vertical position on labial tooth surfaces.



Error latency

Error recognition (micro aggregation)



Error recognition (macro aggregation)
- Finishing starts here
- Reactive correction

FIGURE 32-1 A, Craft-based care model in orthodontics. This stage begins with Replacement therapeutics followed by Reactive therapeutics, which is characterized by high work intensity and appointment density to correct for error. B, Finishing stage in orthodontics generally represents the error correction phase of treatment and commonly follows a blunt end path of error (i.e., errors are accumulated over time during the course of treatment).

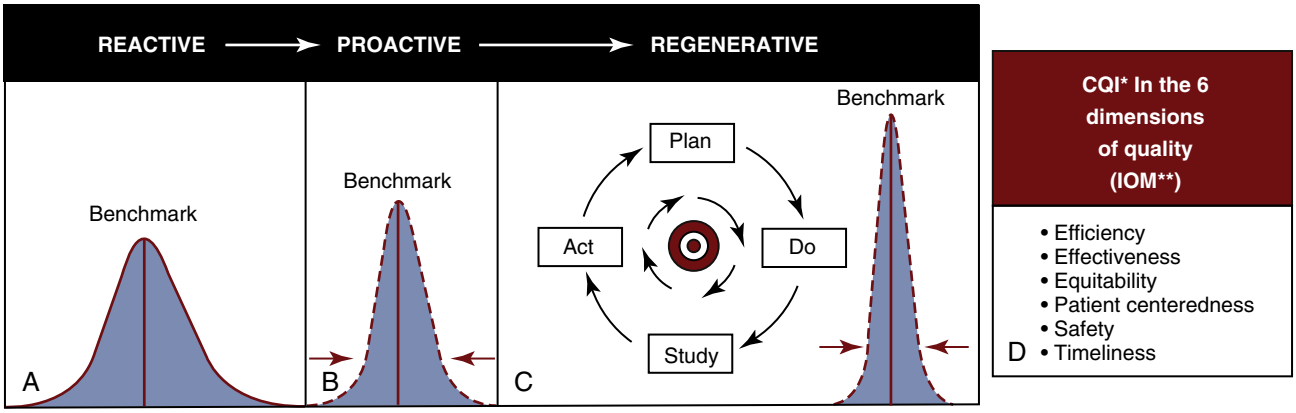


FIGURE 32-2 Practice models. **A,** Characterized by extreme variation. **B,** Characterized by active processes to reduce variation and error. **C, D,** Characterized by proactive management with continuous quality improvement (CQI) initiatives with the goal of continuously improving the six dimensions of quality recommended by the Institute of Medicine (IOM).

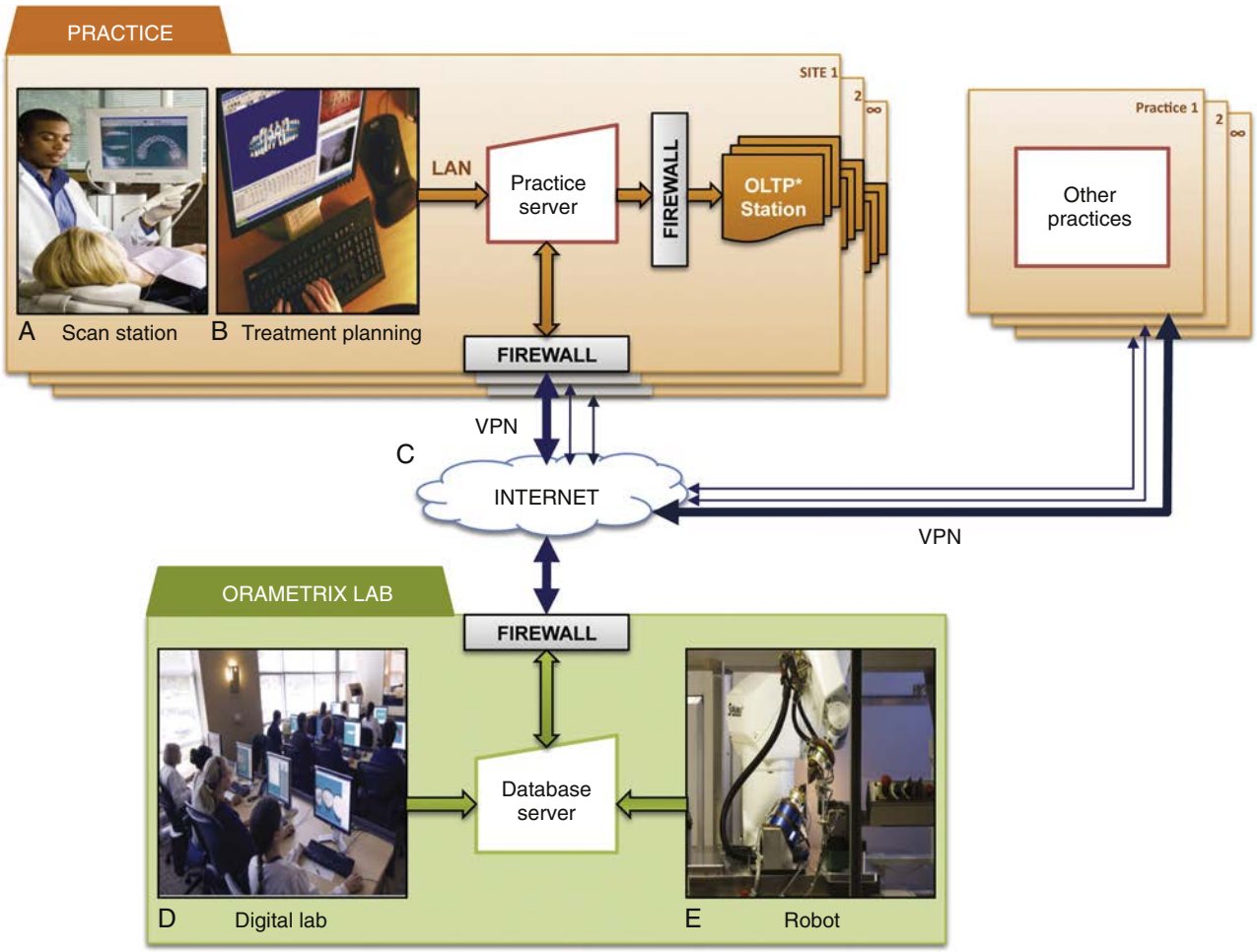


FIGURE 32-3 SureSmile Integrated Technology and Service Environment. **A,** Digital scanner and work station. **B,** Treatment planning work node. **C,** Practice laboratory connectivity. **D,** Remote digital laboratory. **E,** Six-axis wire bending robot. *LAN,* Local area network; *OLTP,* online transaction processing; *VPN,* virtual private network.

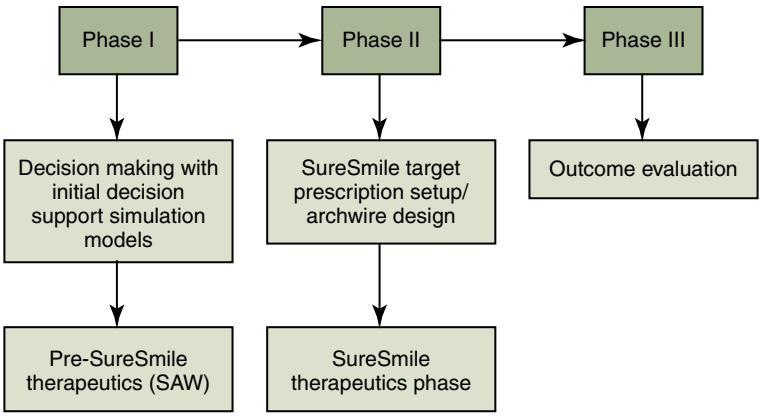


FIGURE 32-4 SureSmile workflow schema. Phase I is characterized by diagnosis, decision making, and managing the patient with straight archwire (SAW) therapy. This is followed by Phase II during which the custom setup and archwire are designed. The patient is managed with SureSmile-targeted personalized archwires. Finally, in Phase III the treatment results are evaluated.

OraMetrix Digital Laboratory and Manufacturing

The SureSmile laboratory is remote to the practice and provides the practice with digital office services. These include (1) modeling all of the 3D images (e.g., the initial pretreatment *diagnostic* models, therapeutic models, and posttreatment final models) and (2) creating the targeted setup based on the orthodontist's prescription. Bidirectional transfer of data between the practice and OraMetrix occurs using a high-speed Internet connection. The customized archwires, indirect bonding trays, and staged models for aligners are manufactured at the SureSmile production facilities and shipped to the practices on demand (Fig. 32-3, *C*, *D*).

SURESMILE IN CLINICAL PRACTICE

This section provides the reader with an overview of the digital integration and process in a technologically advanced practice, described herein using SureSmile. (The discussion in this chapter is limited to the use of SureSmile software tools in the management of patients with the added use of robotically constructed, individually prescribed archwires. Other technologies are commercially available that may also integrate computer-assisted treatment planning and appliance fabrication using some of the principles discussed herein.) SureSmile protocol can be conveniently divided into three distinct phases as shown in Figure 32-4.

Pre-SureSmile Therapeutic Phase: The Digital Diagnostic Model

The integration with the digital diagnosis and treatment planning starts with the consultation appointment and collation of a patient's diagnostic records. These include the patient's chief complaint and medical, dental, and orthodontic history. Records such as extraoral and intraoral photographs and cephalometric and panoramic or CBCT radiographs and models are taken to supplement the clinical findings (Fig. 32-5, *A–C*).

The orthodontist can create a digital diagnostic simulation (DDS) model to use for decision support during care planning. The DDS model can be created by scanning the rough pour of a patient's plaster model with the OraScanner. In addition, a direct in vivo CBCT scan of the patient can be processed to create the DDS model or a physical model scanned with CBCT. Digital models from other vendors in the STL format can also be used and converted into SureSmile DDS models.

The scan of the models is electronically sent to the digital laboratory for modeling. The laboratory sets the global orientation (position of the maxilla in space) and bite registration of the model and separates each tooth into an individual object to allow simulations of individual teeth (Fig. 32-6; see also Fig. 32-5).

The DDS model represents a virtual model of the patient's malocclusion with movable teeth (Fig. 32-7, *B*). The models can be used for 3D visualization and measurement; for example, the physician can perform a Bolton analysis and a dynamic arch length discrepancy by affecting the boundary conditions and also measure the American Board of Orthodontics (ABO) Discrepancy Index. All of these functions are automatically performed with the software (Fig. 32-7, *A–D*).

Decision support with 3D simulation allows the doctor to validate his or her mental model of the patient's treatment plan. The clinician can rapidly simulate multiple treatment scenarios to establish proactive treatment strategies and achieve efficient and effective orthodontic treatment (Fig. 32-7, *E–H*). The DDS model serves as an excellent interactive visual communication tool to discuss the patient's needs and treatment plan. Furthermore, consults can be given in a virtual environment with all stakeholders remotely located by using third-party Web-based collaborative tools such as GoToMeeting (www.gotomeeting.com).

The DDS model may also be used for planning strategic bracket placement (especially important in lingual treatment). Once the initial plan is complete, therapy for labial appliances begins with a conventional straight archwire. There are a number of reasons for this. It fits well within the practice workflow, and some initial alignment of the teeth allows for easier insertion of the customized SureSmile archwires later, minimizing the risk of tooth-wire and/or tooth-bracket collisions and in regions of severe crowding between the bends in the archwire and brackets. The use of additional therapeutic strategies such as constraint management and concurrent mechanics to help achieve more effective and efficient alignment and leveling with

FIGURE 32-5 A, B, Initial SureSmile patient electronic record consists of a standard collage of the patient's two-dimensional images. C, Three-dimensional digital diagnostic simulation (DDS) model.

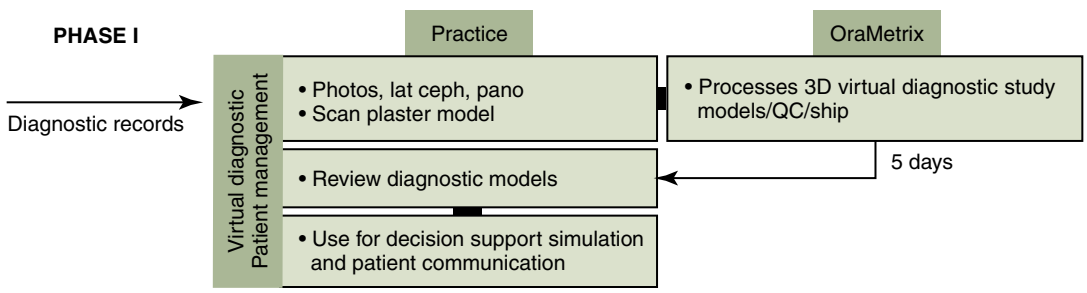
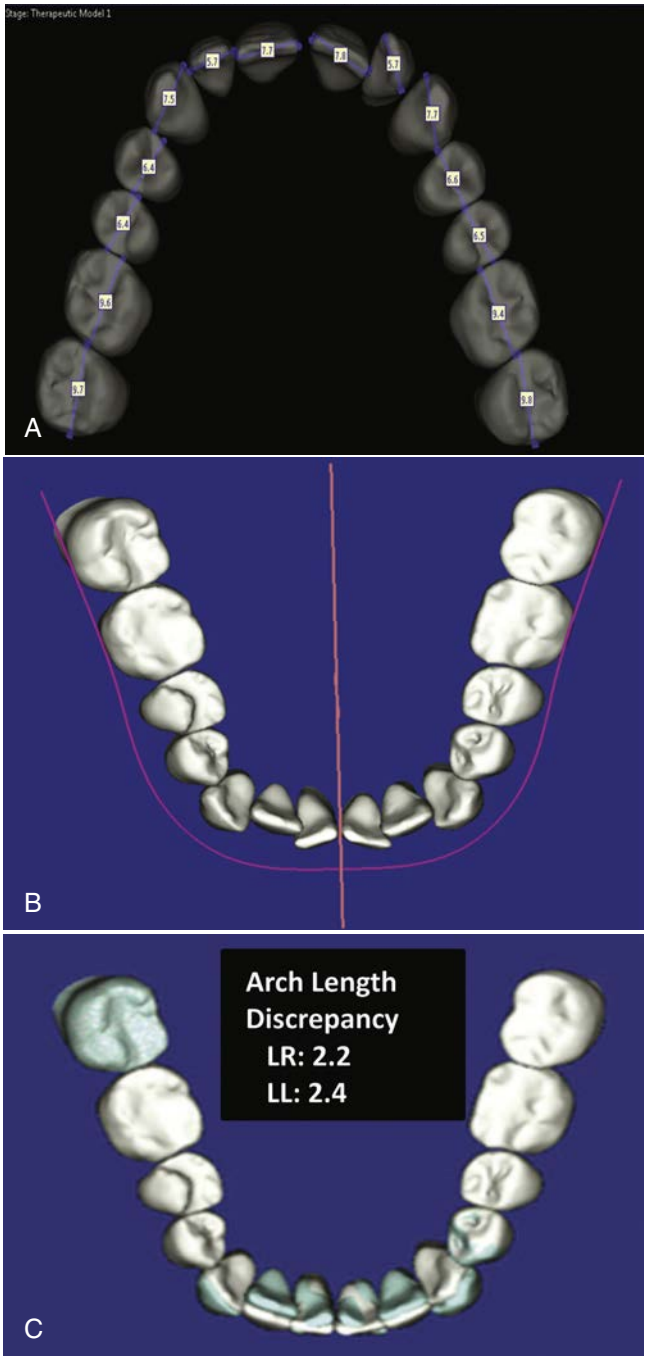


FIGURE 32-6 Practice and laboratory work tasks and production time schedule in Phase I.



Bolton ratio (6-6): 95.3 (3-3): 81.6
Maxilla sum (6-6): 87.1 mm (3-3): 42.1 mm
Mandible sum (6-6): 83.0 mm (3-3): 34.4 mm
Surplus (6-6): Lower 3.4 mm
Surplus (3-3): Lower 1.9 mm

FIGURE 32-7 A, Bolton analysis. B, Estimation of mandibular crowding. C, Boundary constraints for tooth movement are used to evaluate dynamic arch length discrepancy. Blue-green colored teeth represent current status, with white teeth the simulated goal.

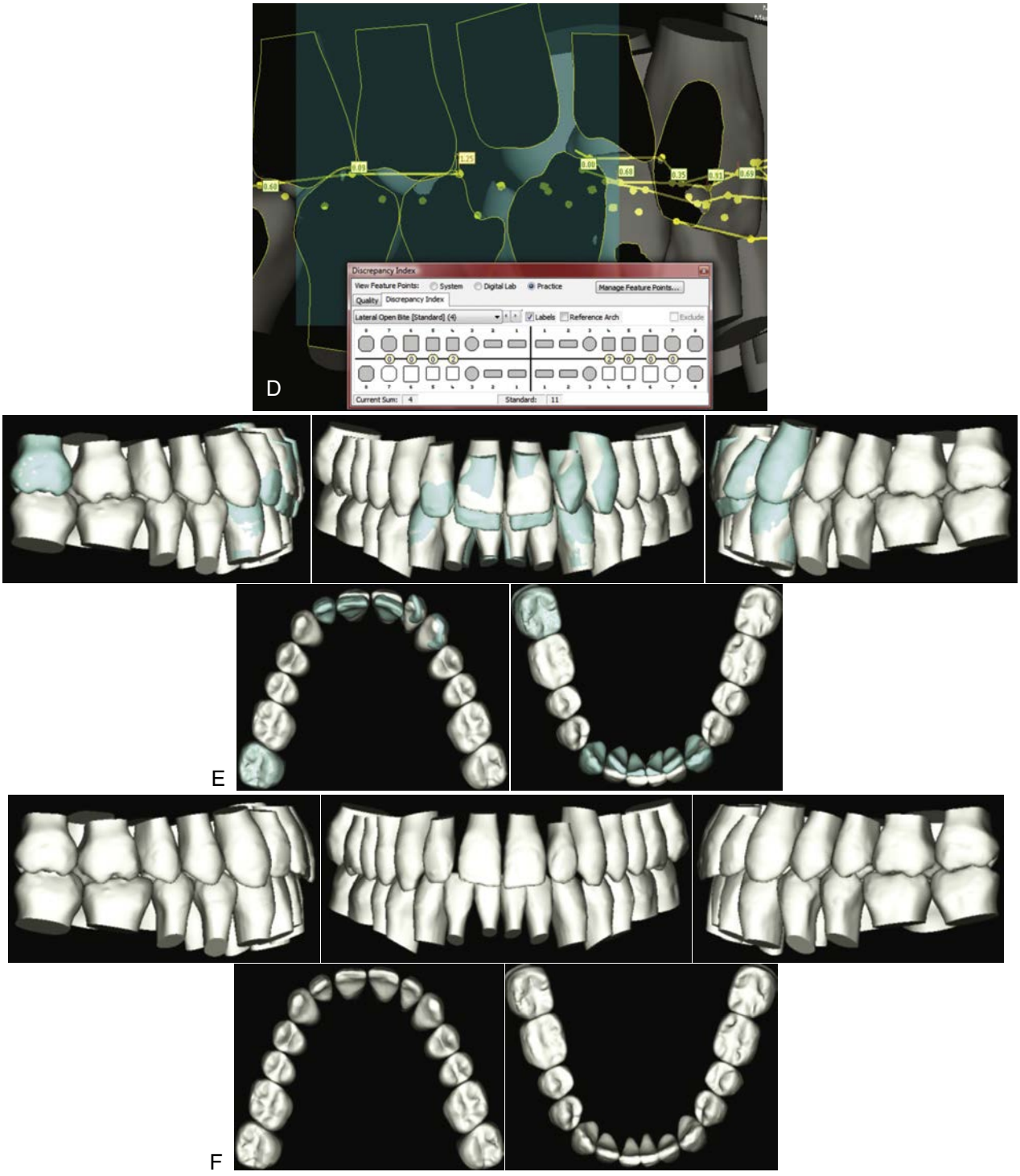


FIGURE 32-7, cont'd D, Discrepancy index is automatically measured.

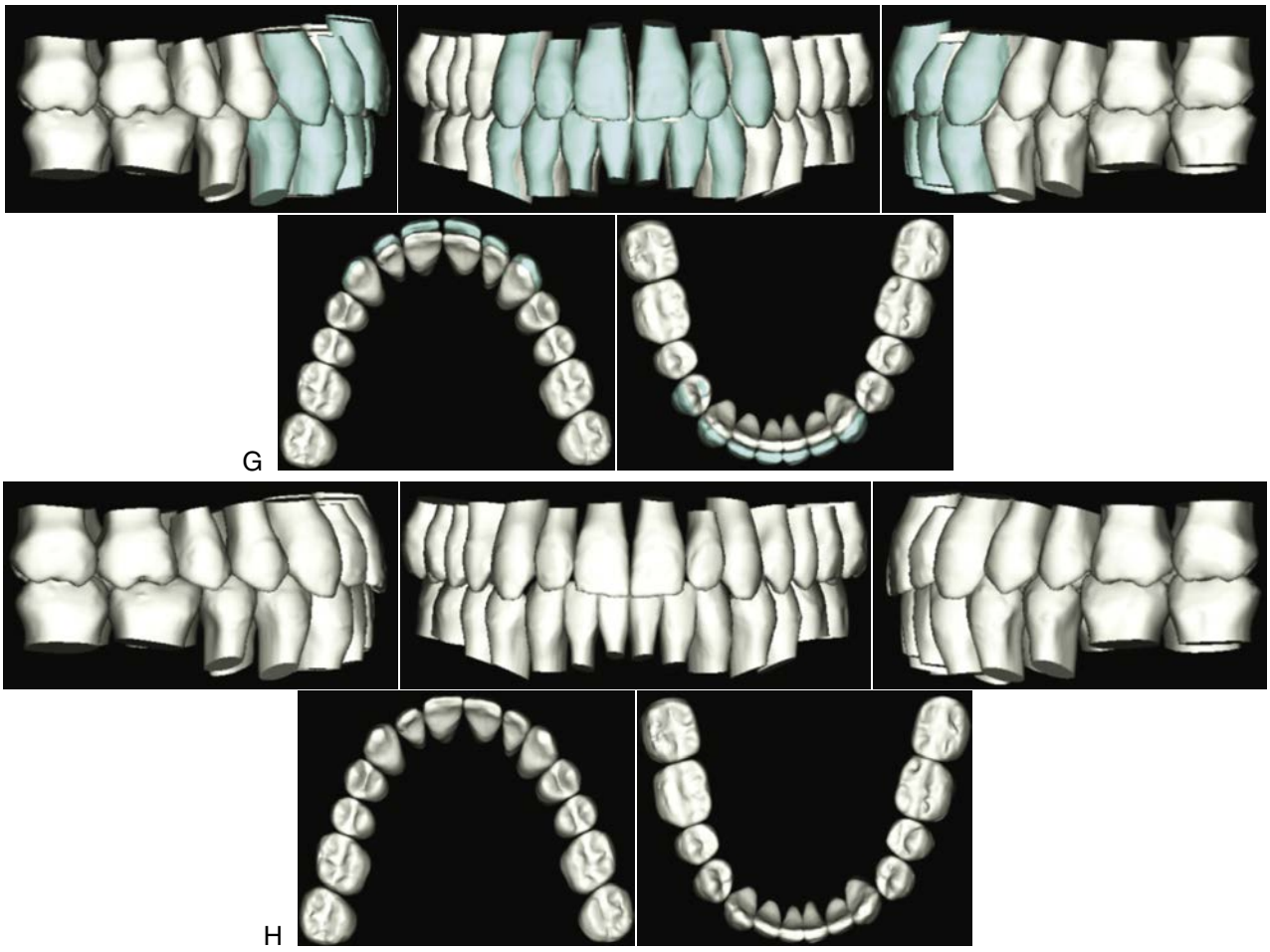


FIGURE 32-7, cont'd E–H, Decision support with simulation. The blue-green model represents the initial condition, and the white model represents the simulation.

earlier torque control is also recommended to optimize treatment efficiency (Fig. 32-8, *A-E*). These approaches are discussed in detail later in this chapter. Generally speaking, for most types of malocclusions, the pre-SureSmile therapeutics phase does not exceed 6 months. However, with appropriate patient selection, an experienced clinician can start treatment with SureSmile-customized archwire from the start of treatment or very soon thereafter, which requires following the fast-track clinical pathway (Fig. 32-9).

SureSmile Therapeutic Phase

The SureSmile therapeutic phase is immediately triggered after initial alignment and leveling have been achieved, and the intra-arch form has been partially established. In a normal premolar extraction case, approximately 4 mm of extraction space should be closed before using SureSmile. Sagittal corrections can be performed either before or concomitantly to using SureSmile.

An in vivo scan is taken of the patient to capture the current 3D positions of the dentition and the brackets. This scan is called the *therapeutic scan* (see Fig. 32-8, *B*). The SureSmile intraoral scanning device (OraScanner or other OraMetrix-accepted scanner) or CBCT may be used to capture the therapeutic scan. The choice of using either of the technologies is determined by the doctor's preferences and abilities and the patient's needs.

The OraScanner uses structured white light to capture the image of the dentition and therefore poses no radiation risk to the patient. Image capture with an OraScanner is not sensitive to patient motion. An OraScanner does not capture root position but displays gingival tissue and has a higher resolution range of 30 to 50 microns (see Fig. 32-8, *C*). At this publication date, the iTero Element Scanner (Align Technologies, Santa Clara, CA) and TRIOS (3Shape) may also be used in lieu of the OraScanner.

Image acquisition with CBCT takes less than 10 seconds at a voxel size of 0.2 mm and is extremely sensitive to patient motion. Furthermore, although the root's position can be captured, the gingival tissue cannot be visualized at this point.

Once captured, the therapeutic scan is sent to the laboratory for postacquisition processing. The laboratory technician sets the global position and bite registration of the model, based on the submitted photographs and radiographs. The teeth are separated to create individual objects. The captured bracket image on the therapeutic scan is matched against 3D-bracket templates of the doctor-prescribed bracket, and the brackets are precisely registered on individual teeth in space with an accuracy of 10 microns (see Fig. 32-8, *D*). The therapeutic model is then electronically shipped to the practice (see Fig. 32-8, *E*). The orthodontist then orders an individualized setup prescription.

The clinician can use the therapeutic scan to evaluate treatment progress by best-fit registration on the diagnostic model

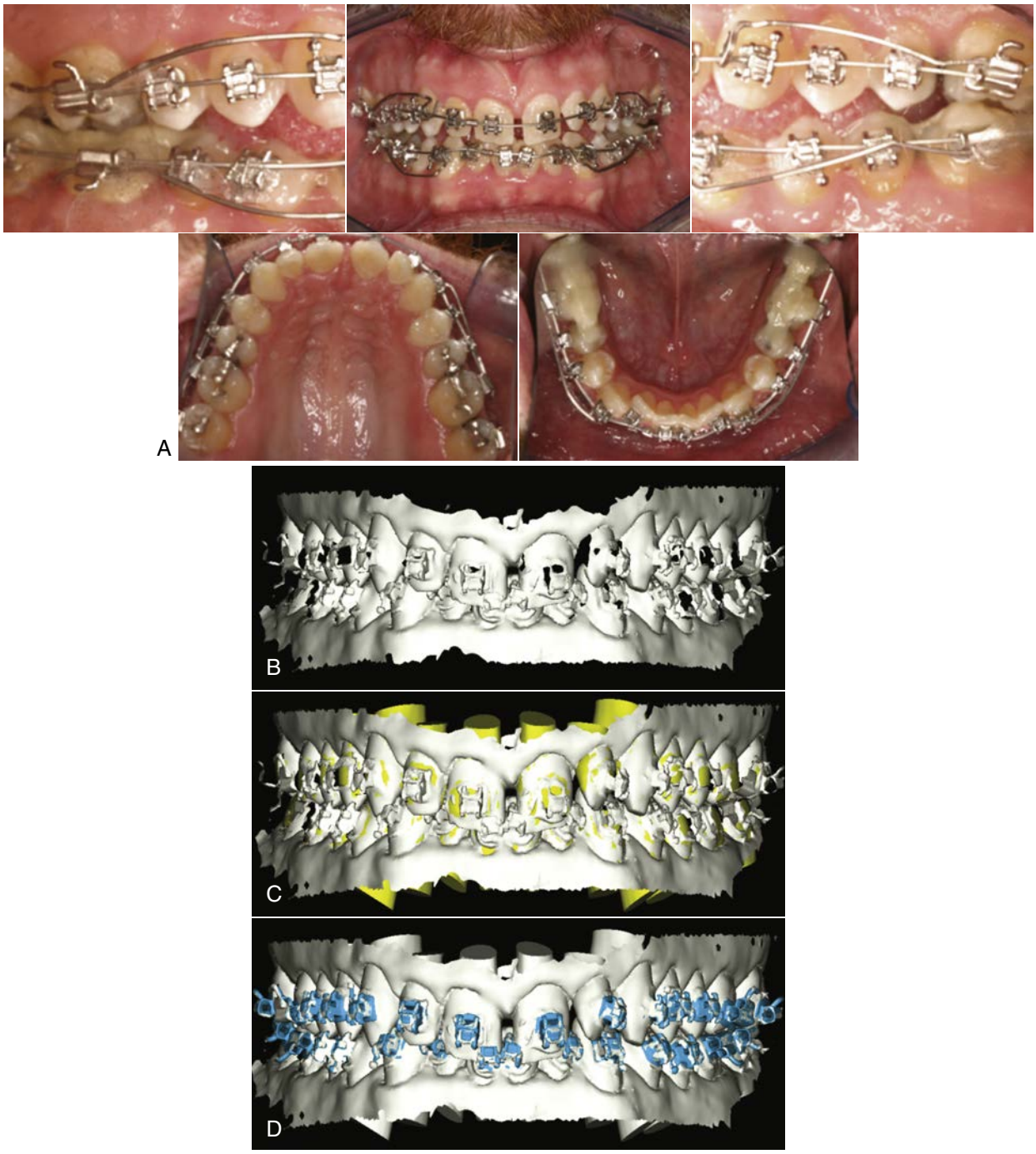
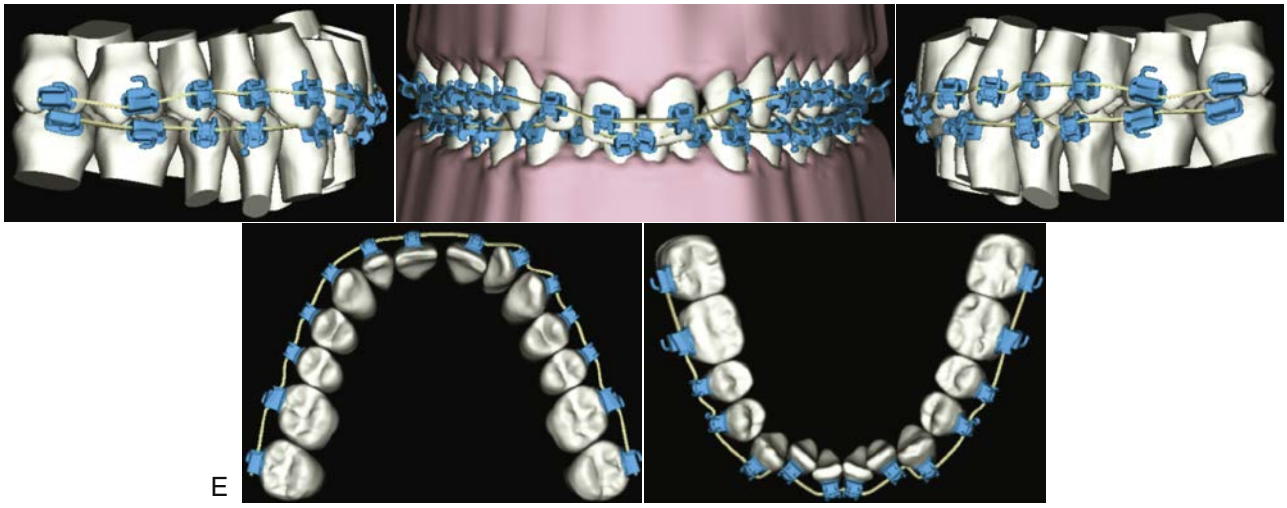
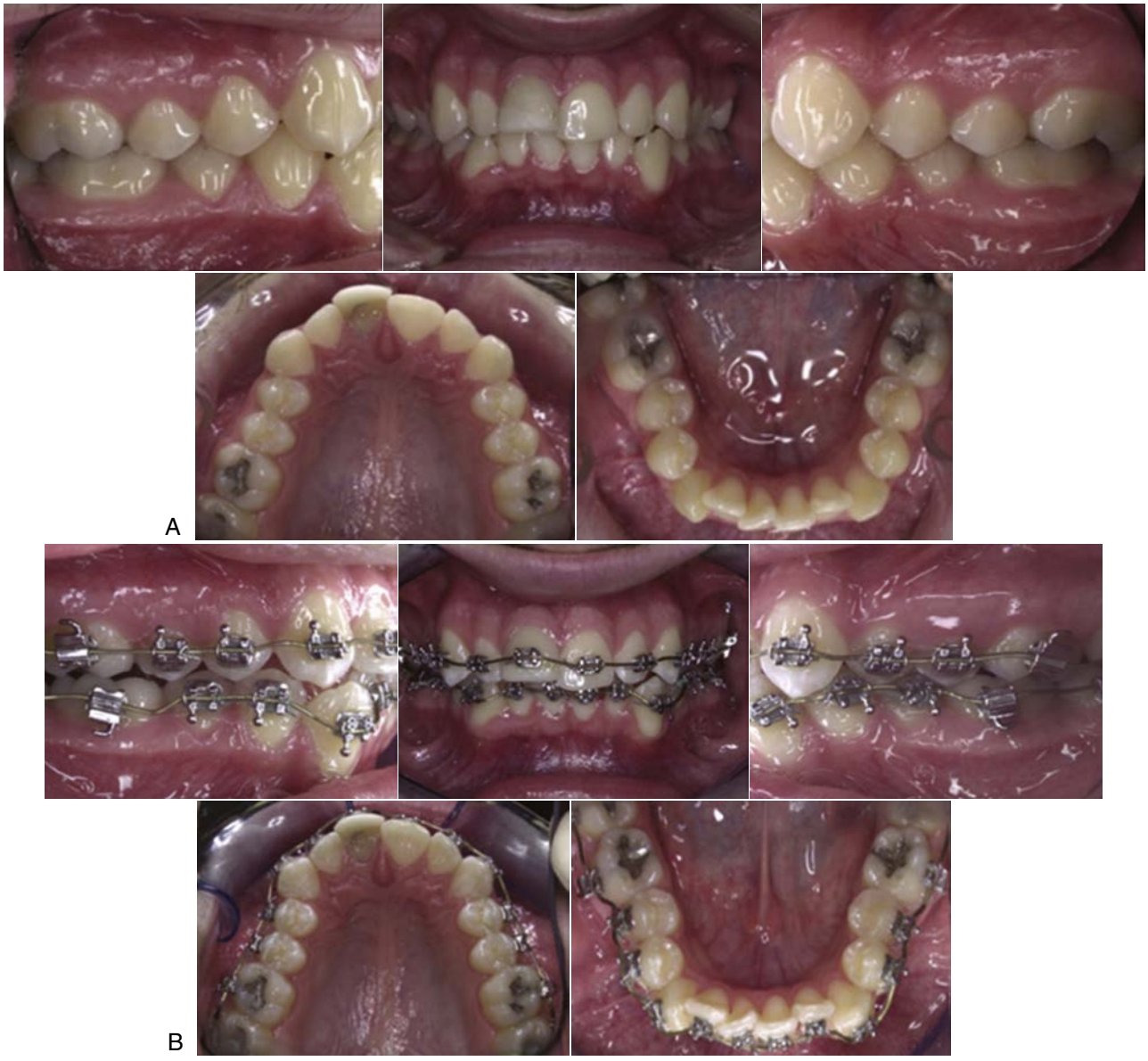


FIGURE 32-8 Intraoral images and corresponding therapeutic model. **A,** Initial pre-SureSmile therapeutics 17- × 25-inch copper-nickel-titanium (CuNiTi), 17- × 25-inch titanium molybdenum alloy (TMA) tip back springs helps arch leveling and posterior bite turbos to prevent interarch tooth collisions. **B,** Therapeutic raw scan data are demonstrated. **C,** Raw scan with tooth template is used for void filling. **D,** Bracket registration is visualized on a raw scan.



E

FIGURE 32-8, cont'd E, The therapeutic model shows the tooth and bracket position at the time of scan; in addition, the state of archwire deactivation can be displayed.



A

B

FIGURE 32-9 A–D, Full expression archwires (100%). In this patient, a full expression 17- × 25-inch copper-nickel-titanium (CuNiTi) archwire was used from the beginning (9/24/02) to the end of treatment (8/15/03). This is an example of a fast-track clinical pathway.

FIGURE 32-9, cont'd

and assess how the straight archwire has worked. The therapeutic model is used to establish the setup prescription (Fig. 32-10). A number of approaches are used to provide this prescription; these include clinician notes, a 3D simulation, and/or filling a prescription form. The choice is driven by the doctor's preference and consideration of the most effective way for the orthodontist to communicate with the laboratory technologists regarding the particular needs of the patient and the goals for treatment. A combination of approaches is usually used.

The prescription for the setup model is driven by considering six boundary conditions. The first letters of each of these boundary conditions form a memory mnemonic MACROS: (1) location of the **M**idline, (2) **A**rchform type, (3) **C**lass of occlusion, (4) choice of **R**eference teeth (i.e., the tooth positions that the clinician wishes to maintain), (5) **O**cclusal plane (commonly the aesthetic), and (6) **S**pecial instructions, which may include instructions such as the need for preferential spacing for restorative purposes and amount of interproximal reduction (IPR) permitted, among others. Additionally, the doctor can set elastic boundaries to allow a range of tooth movements based upon a professional assessment of the biologic constraints (see Fig. 32-10).

The setup prescription is electronically submitted to the laboratory, and the completed digital prescriptive setup is then returned to the practice. The clinician uses an editable checklist to evaluate the setup, complemented with a grading and measuring system (the doctor may choose to use the ABO objective grading system [OBG] and/or other systems). This detailed review of the setup model is critical to the success step in the management of the patient since the setup design directly affects the archwire prescription and represents the treatment goal (visualizing the end to develop the means!). The clinician can make or request the laboratory technologist to make changes in the setup (Fig. 32-11, *A-D*), but the overall treatment plan and plan of treatment are determined as a result of the orthodontist's specific patient prescription.

Tooth displacement with respect to the treatment setup and the working design of the prescriptive archwire(s) is also evaluated (Fig. 32-12, *A-B*).

Once the prescriptive setup is accepted, the orthodontist focuses on the archwire design. This phase includes design modifications such as setting limits of archwire expression and staging archwires, adding expansion or curve of Spee, overcorrection or limiting archwire bracket play by adding additional

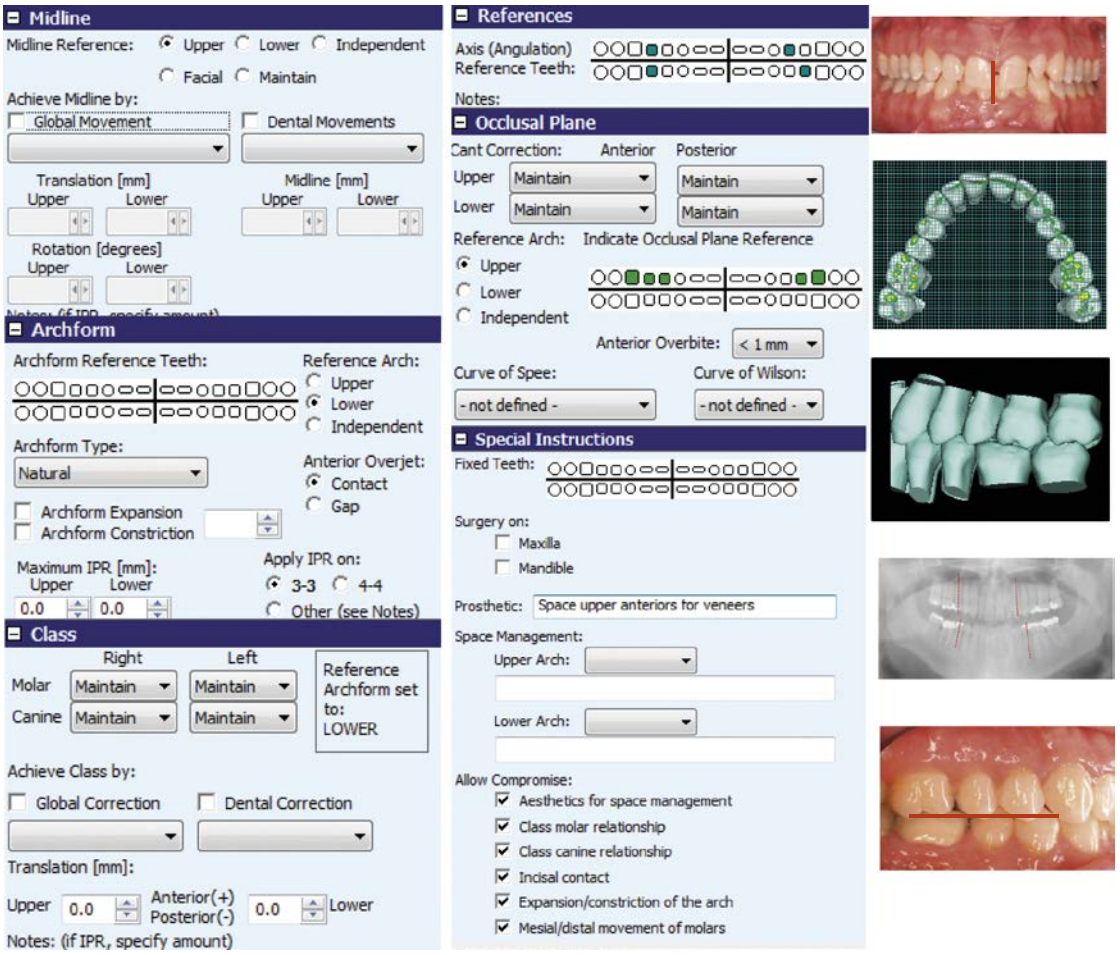


FIGURE 32-10 Electronic prescription form. The doctor fills in the form using a workflow-driven checklist using MACROS (Midline, Archform, Class, Reference teeth, Occlusal Plane, and Special Instructions) and can also set elastic boundary preferences (i.e., range of acceptable treatment limits).

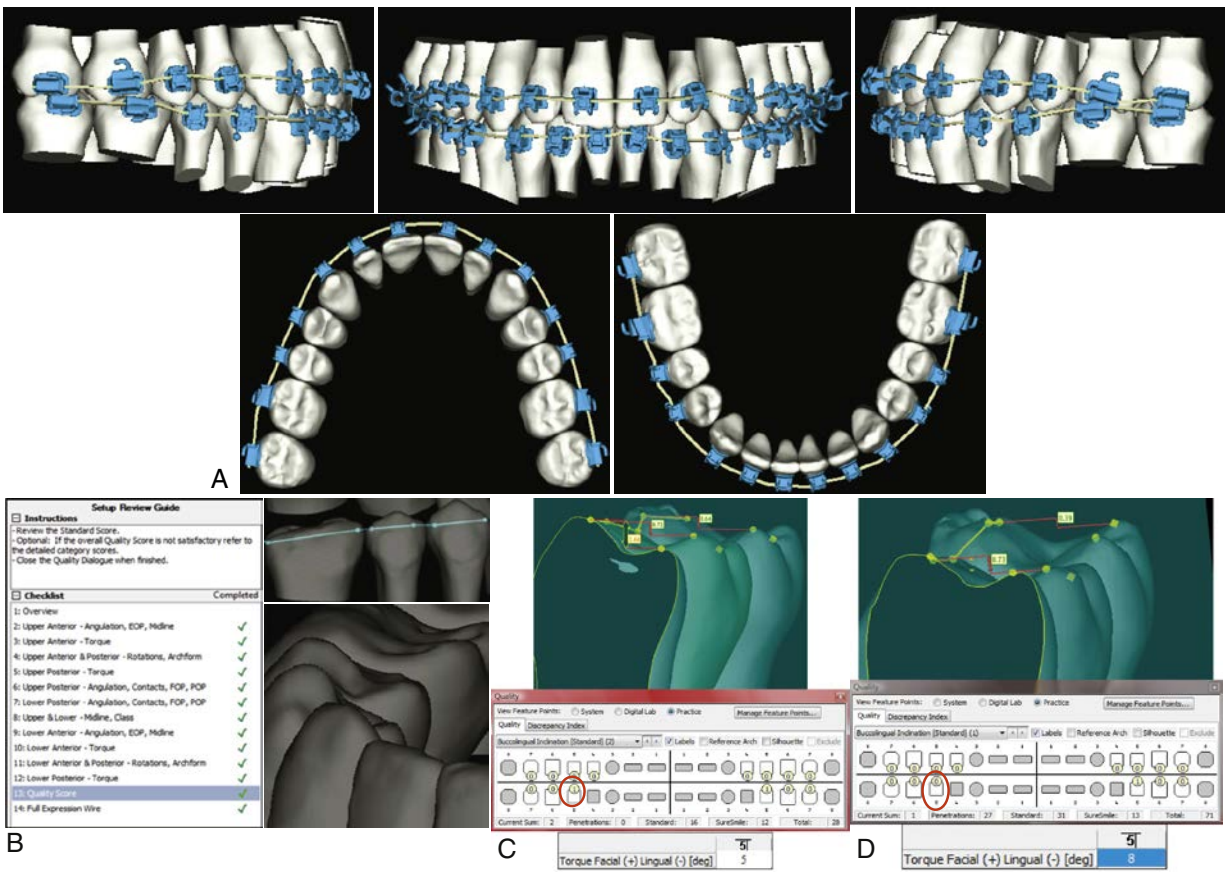


FIGURE 32-11 A, Target setup, based upon the doctor's prescription. B, Practice-based checklist. C, D, Measuring and grading tools to evaluate the setup. Modifications can be made at practice site.

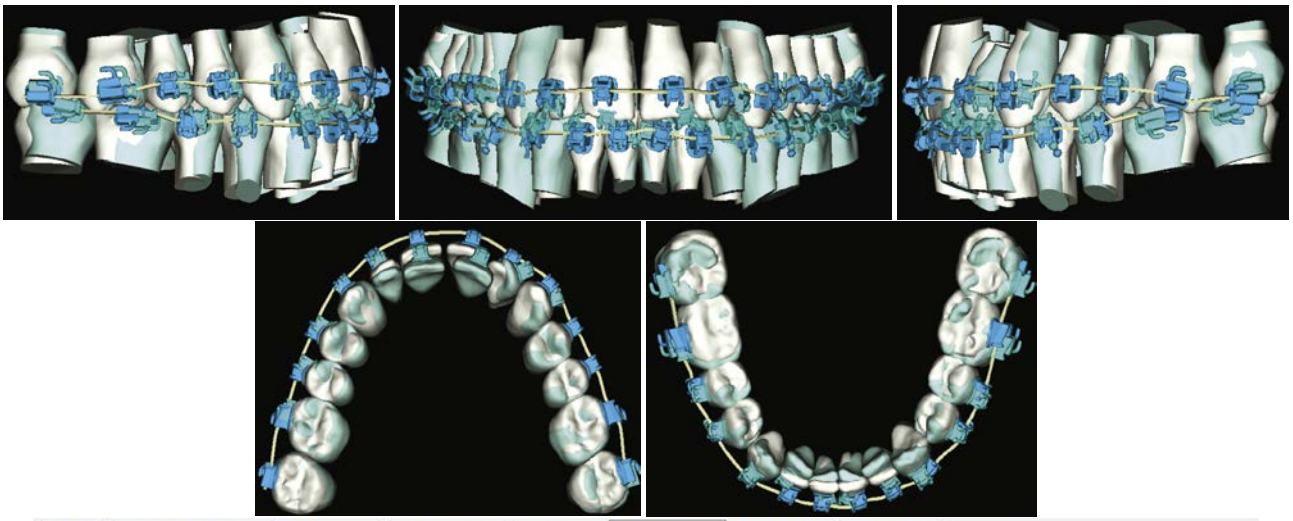
torque in the archwire, and choosing archwire material and cross section.

This capability offers the clinician a wealth of therapeutic strategies to control tooth movement. (How these are used is discussed later in this chapter in greater detail.) Optimal archwire installation strategies can be simulated to minimize any archwire bend-bracket collisions on insertion or during tooth movement. Modifications of archwire design in terms of location of wire bends can be also made to minimize this occurrence (Fig. 32-13).

After the submission of the archwire prescription, archwires are fabricated in the OraMetrix laboratory, and the practice is sent the prescribed customized archwires. Before shipping, all archwires are scanned with a special laser scanner to measure the accuracy and precision of the archwire bends. Archwires are measured to an accuracy of 20 microns. This procedure is one of many steps instituted by the manufacturing facility to minimize errors in production through quality assurance and control.

Figure 32-14 shows a summary of Phase II practice—laboratory activity and customary timelines for product delivery. The customized wires are generally inserted within 6 weeks of taking the therapeutic scan. Instructions for inserting the archwires are provided, and the archwires are laser marked to minimize placement error. Patients are commonly seen 8 weeks after custom-designed wire insertion (Fig. 32-15).

At this visit, the doctor and the staff use a guided checklist to evaluate the progress of care. Using a checklist helps prevent any oversights during the evaluation process. By the 16th week, if the target occlusion has not yet been reached, the doctor may use a second level of support checklist and a guided therapeutic archwire modification schema to manage corrections. Archwire modifications may be accomplished at the chairside with the *virtual plier*.



A

Order | Global Registration | References | Occlusal Plane | Arch Form | **Upper Disp.** | Lower Disp. | Fixed Teeth

Active Stage: Plan 2 [0.3 DL] Compare with: Reference Stage: Therapeutic Model 1

	8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
Mesial (+) Distal (-) [mm]		-0.2	-0.2	-0.4	-0.3	-0.5	-0.1	-0.6	0.8	1.2	0.8	0.6	0.6	0.6	0.4	
Buccal (+) Lingual (-) [mm]		0.4	0.5	0.5	0.5	0.4	0.7	1.8	2.1	0.3	0.3	0.3	0.3	0.3		
Occlusal (+) Gingival (-) [mm]		0.5		0.2		0.2	-0.8	-2.1	-2.0	-0.4	0.5		0.2		0.5	
Torque Facial (+) Lingual (-) [deg]		-6		3	2	1	6	10	10	-3			-3	-3	-9	
Angulation Mesial (+) Distal (-) [deg]							-1	1	2						8	
Rotation Mesial (+) Distal (-) [deg]			-9	-14	-11	18	-12	-4	9	38	29	-6	-6	-5		
Mesial Gap (+) IPR (-) [mm]						0.8		0.3	0.3		0.8					

☑ Malocclusion Includes Max. / Mand. Alignment Adjustments ◉ Tooth Displacements ○ Bracket Displacements

Order | Global Registration | References | Occlusal Plane | Arch Form | Upper Disp. | **Lower Disp.** | Fixed Teeth

Active Stage: Plan 2 [0.3 DL] Compare with: Reference Stage: Therapeutic Model 1

	8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
Mesial (+) Distal (-) [mm]		-0.9	-1.0	-1.1	-1.0	-0.4	-0.1		0.3	-0.6	-0.6	-0.9	-0.9	-0.9	-0.6	
Buccal (+) Lingual (-) [mm]		-1.2	-0.1	-0.1	-0.7	-0.8	1.5	0.5	0.3	1.1	-1.0	-0.2	-0.5	-0.7	-1.3	
Occlusal (+) Gingival (-) [mm]		-0.3	0.4		0.2	-0.5	-1.1	0.5	0.3	-0.9	-0.6	0.3		0.3	-0.4	
Torque Facial (+) Lingual (-) [deg]		4		5	7	11	11	7	8	14	13	11		-5	3	
Angulation Mesial (+) Distal (-) [deg]		-9					-3	5		-3		-1		-3	-12	
Rotation Mesial (+) Distal (-) [deg]			-5	-1	20	18	20	-14	-28	13	-9	7	-4	-6	-4	
Mesial Gap (+) IPR (-) [mm]						-0.2	-0.2	-0.2	-0.2	-0.2	-0.2					

☑ Malocclusion Includes Max. / Mand. Alignment Adjustments ◉ Tooth Displacements ○ Bracket Displacements

FIGURE 32-12 A, Therapeutic model (*blue*) versus setup model (*white*) helps plan realistic tooth movement.

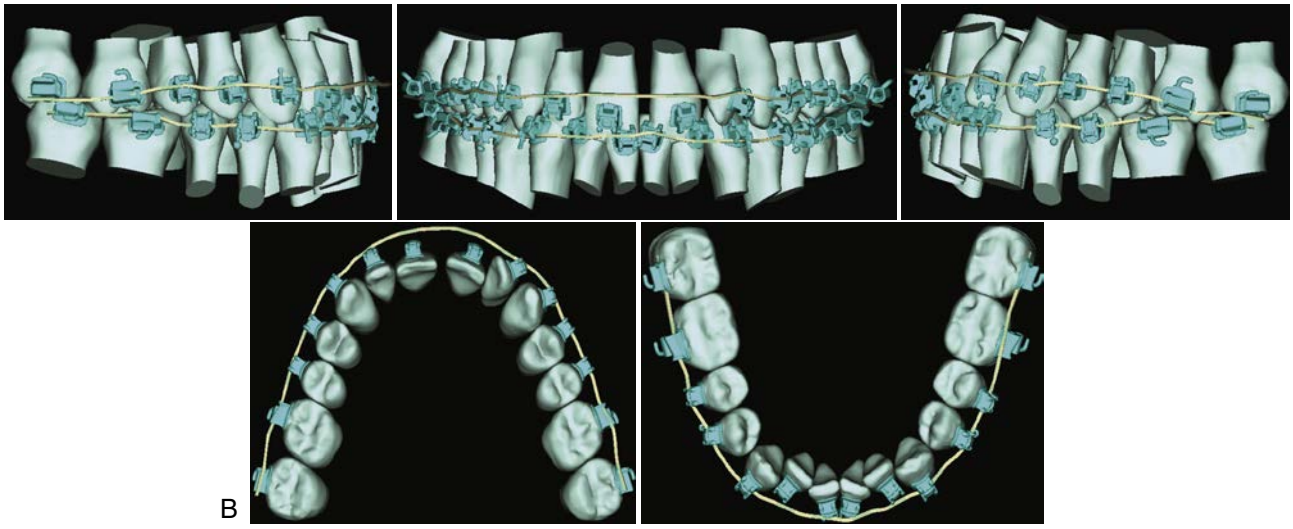
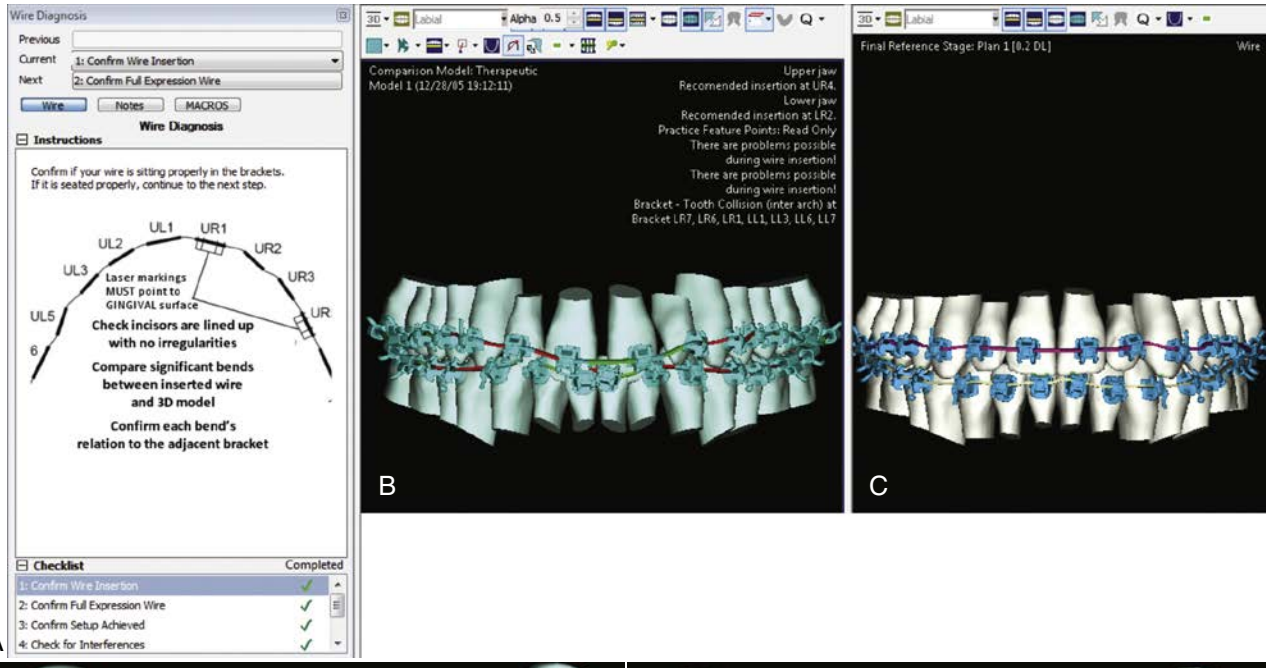


FIGURE 32-12, cont'd B, Therapeutic model versus archwire helps read the archwire design.



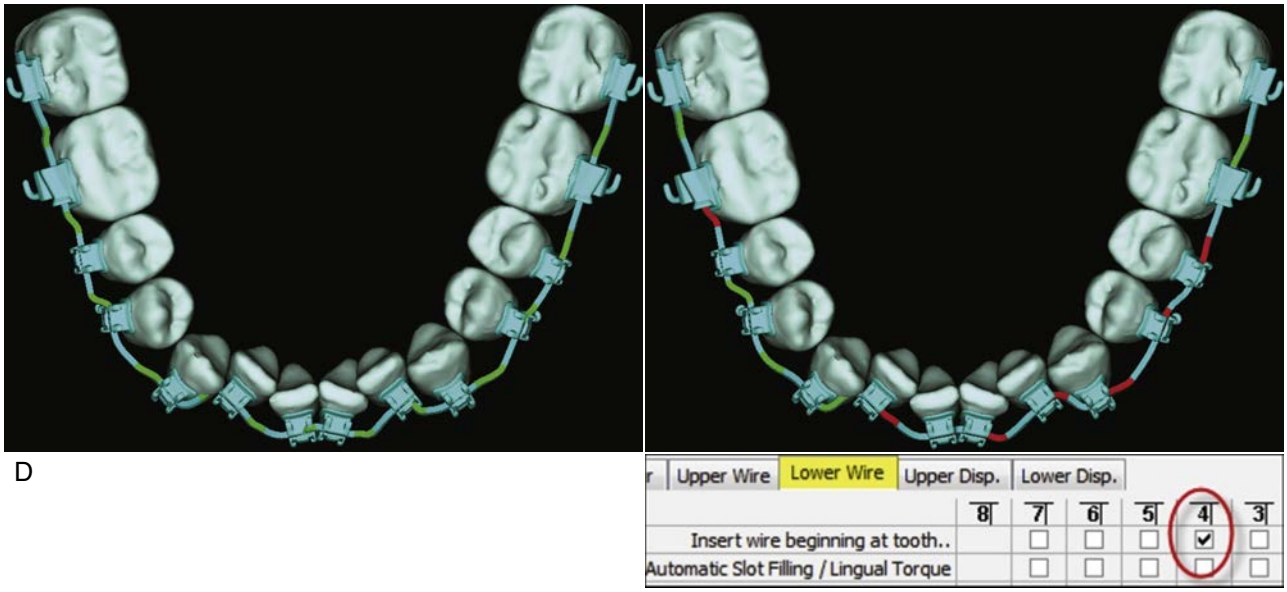


FIGURE 32-13 Archwire collision identification aids to see where interferences among teeth, brackets, and wires may come in to play during treatment. **A,** Template generated for practice demonstrating optimal location for tying archwire. **B, C,** Archwire installation can be simulated on the therapeutic model to detect collisions. **D,** Archwire is in its final passive state.

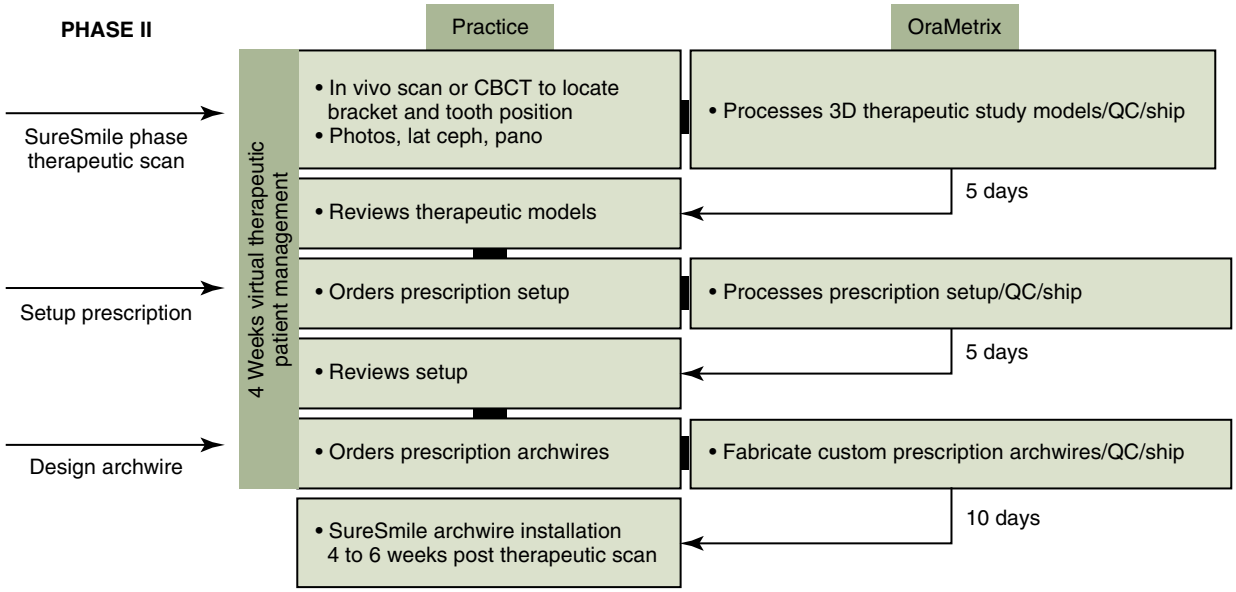


FIGURE 32-14 Phase II SureSmile work tasks at the practice and laboratory with work delivery times.

FIGURE 32-15 A, Insertion of 19- × 25-inch SureSmile archwire. B, Eight weeks after insertion.

There is great assurance in using the robot-assisted modified archwires since bends in areas requiring no modifications are maintained. Keeping tooth position unchanged is extremely difficult, if not impossible, to do with conventional archwires since their repeated removal, modification, and insertion deform the archwire and may lead to overworking a hand-bent archwire and to breakage. Replicating the geometry of the broken archwire is frustrating and usually cannot be accomplished with accuracy. In addition, if needed, the clinician may order a backup pair of bendable prescriptive archwires made from TMA or Azurloy (Ormco, Glendora, CA) in advance and make minor modifications at the chairside.

Generally speaking, most treatment with SureSmile prescription archwires is completed within 6 months after the placement of the SureSmile wires. Clinical judgment and the image of the target setup with the final position of the brackets on are used to gauge whether the prescriptive archwire has worked out and the treatment goals are achieved.

The final bracket position on the target setup is used to evaluate whether the archwire has worked out to completion (Fig. 32-16). The value of this verification step cannot be underestimated; in many busy practices, the treatment goals are generally forgotten and patient care continues, often with little progress achieved over time—a phenomenon the author of this chapter terms "orthodontic flagellation," which is costly to both the patient and the practice. A visual reference definitively provides an effective "stopping rule" for care. Furthermore, patients commonly forget the goals of treatment. When a visual treatment plan is designed with the patient's participation in advance and shared during treatment, the patient is aware of the end goal and is more accepting of the outcome. Furthermore, the plan is always visually accessible on a computer to show a patient if the need arises in response to special patient and/or parent questions. This approach is unlike the conventional practice where two different versions exist—one in the doctor's mind and the other in the patient's mind. A visual treatment objective provides a set point for motivating patients and parents.

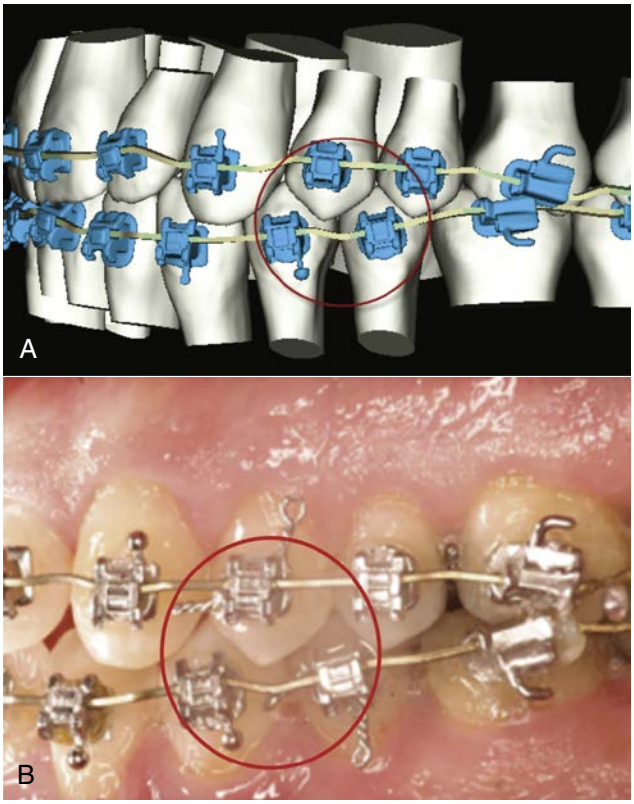


FIGURE 32-16 Final bracket position (*red circles*) on the target setup (A) is matched against the clinical situation (B) to assess whether the archwire has worked out.

Outcome Evaluation Phase

This final model can be evaluated by 3D inspection or by using the automatic grading system and by a comparative analysis using the best fit registration method against the diagnostic, therapeutic, or target setup (Figs. 32-17 and 32-18). Furthermore, to better analyze the patient's response to treatment and the physician's ability to achieve his or her treatment objectives, the models can be selectively superimposed by best fit registration to the diagnostic, therapeutic, or setup models.

PROACTIVE DECISION MAKING

A number of approaches may be used in planning the care for a patient using the DDS model and/or the Dental Technology Services (DTS) model. For an accomplished clinician, these simulations do not take longer than 10 minutes to accomplish. Effectively using these proactive simulation tools is a skill and can be learned only through deliberate practice. Practitioners new to digital tool sets should schedule extra time for simulation and other tasks as they build these skills. When the knowledge gained from these simulations is appropriately incorporated and sequenced in the management of a patient, not only do they add to the efficiencies in care and better outcomes, but they also increase patient safety by alerting the orthodontist to potential hazards.

Principle of Smart Orthodontics

The objective of simulations is to plan care with three objectives. The treatment plan should (1) be achievable, (2) require minimal tooth displacement, and (3) realistically reflect the ability of the therapeutic appliance under consideration. An example of using this approach in the correction of a functional shift accompanied with a posterior crossbite in the second molar area is shown in Figures 32-19 through 32-22.

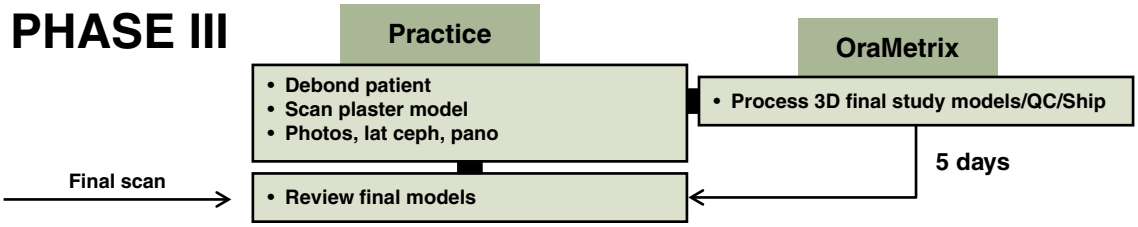
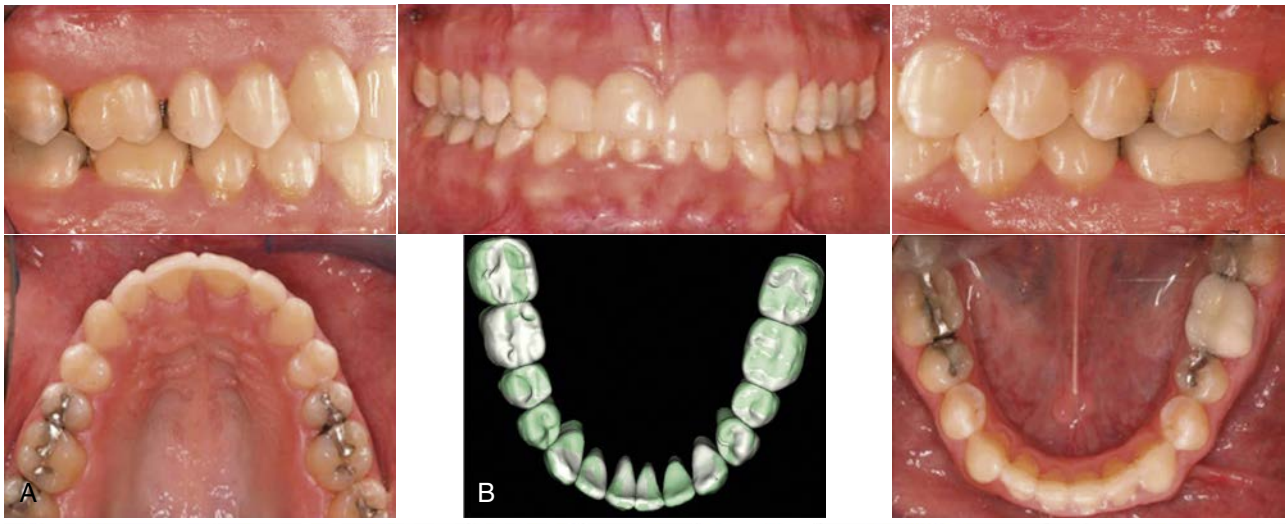


FIGURE 32-17 SureSmile process for patient debonding and final assessment.



er | Global Registration | References | Occlusal Plane | Arch Form | Upper Disp. | Lower Disp. | Fixed Teeth | Upper Brackets | Lower Brackets

Stage: Simulation 5 Compare with: Reference Stage: Diagnostic Model 1

	8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
Mesial (+) Distal (-) [mm]		0.2	0.2	0.2	0.1									0.1	0.1	
Buccal (+) Lingual (-) [mm]					0.5	-0.1									-1.2	
Occlusal (+) Gingival (-) [mm]							-0.1					-0.3		0.3		
Torque Facial (+) Lingual (-) [deg]					-7						-4					
Angulation Mesial (+) Distal (-) [deg]					1									-5		
Rotation Mesial (+) Distal (-) [deg]					-11	3		3	3		4					

FIGURE 32-18 A, Final photographs with four anterior teeth restored with veneers. B, Superimposition of final models versus setup.

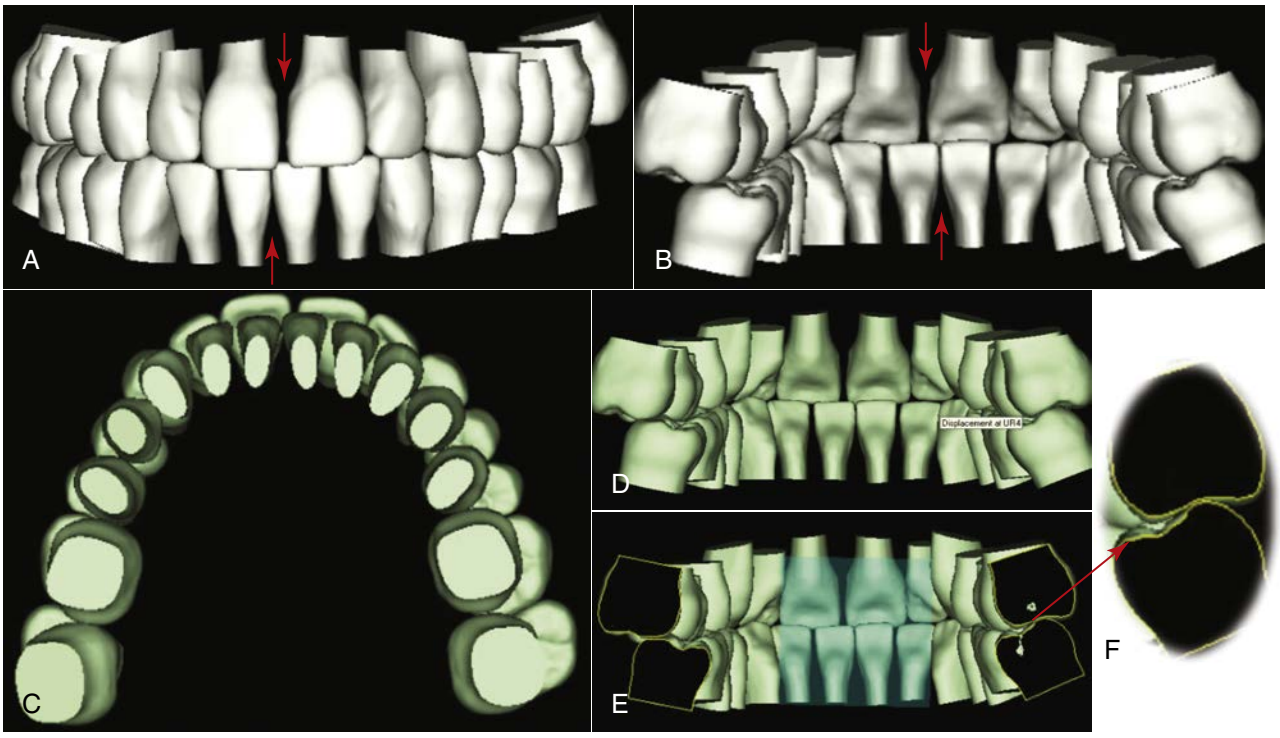


FIGURE 32-19 Smart tooth movement. A–E, Initial models show functional shift and buccal crossbite in left second molar region (*arrows*). F, Note the contact in the upper right molar area (*arrow*).

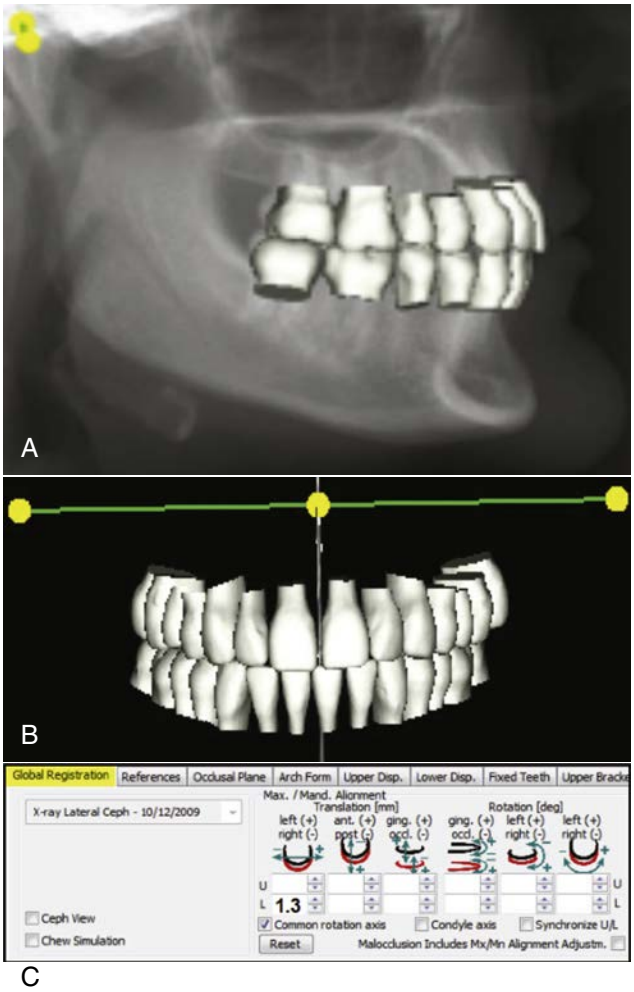


FIGURE 32-20 Smart tooth movement. **A, B,** Center of rotation simulates a mandibular shift. **C,** Input for simulation of mandibular shift.

Principle of Risk Analysis

The author of this chapter uses this simulation to visualize whether the plan may result in the violation of anatomic constraints (Fig. 32-23). In addition, since the gingival tissue is designed to move in a one-to-one ratio with tooth movement, the effect that tooth movement has on gingival architecture can be seen and plans can be made in advance for it (Fig. 32-24). The patient can also be proactively informed about the potential risks associated with treatment.

Principle of Constraint Recognition and Management

This simulation enables the orthodontist to better understand where the teeth will collide during alignment and plan for strategic and proactive IPR (Fig. 32-25). He or she can also see whether bracket interferences will occur and how these interferences could affect tooth movement.

Principle of Anticipatory Orthodontics

The orthodontist can develop chronologic milestone-driven, best-estimate prediction simulations that may be shared with both patients and staff. Patients find them very useful in tracking their care progress. They become active collaborators in care, and their personal motivation to adhere to the treatment regimen substantially increases. The simulations also allow the staff members to better understand treatment objectives, and the images provide them a template to assess orthodontic vital signs at each visit. The simulations also provide an excellent visual communication tool for both the clinician and staff to discuss the patient's progress in treatment at the chairside (Fig. 32-26). This collaborative care is another hallmark of a patient-centered practice. After years of performing such predictive simulations and comparing them with the actual tooth movement, patterns in tooth movement can be recognized that may be incorporated in the simulations to achieve more realistic expectations.

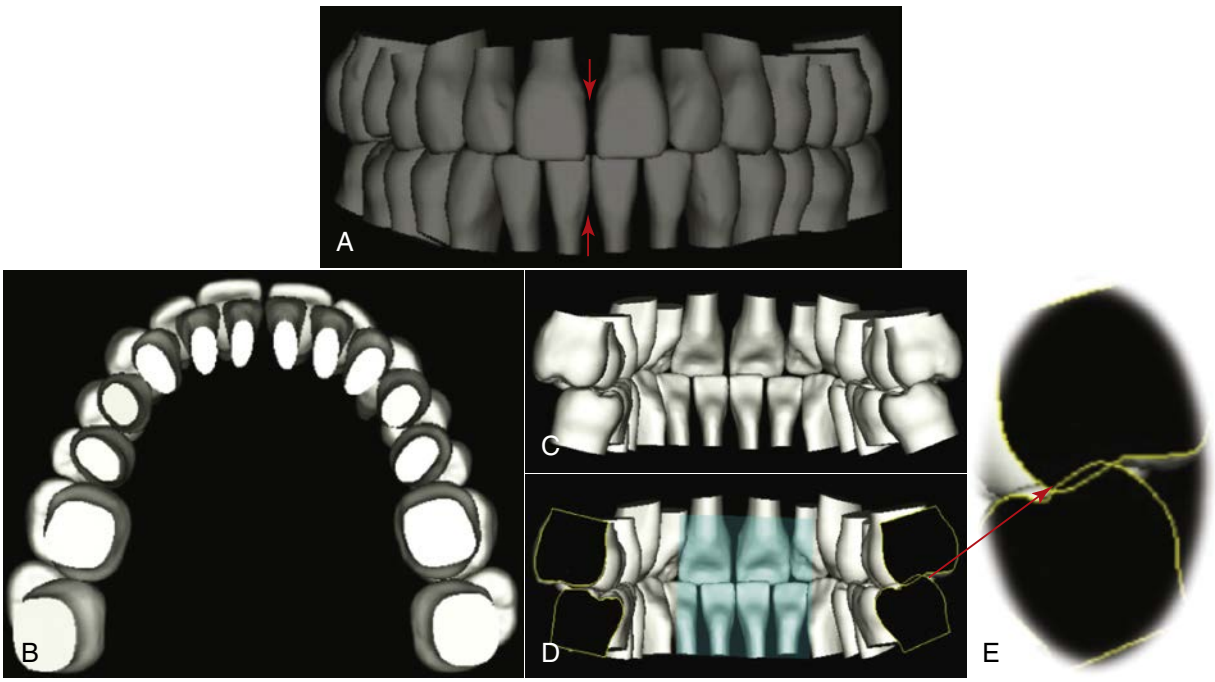
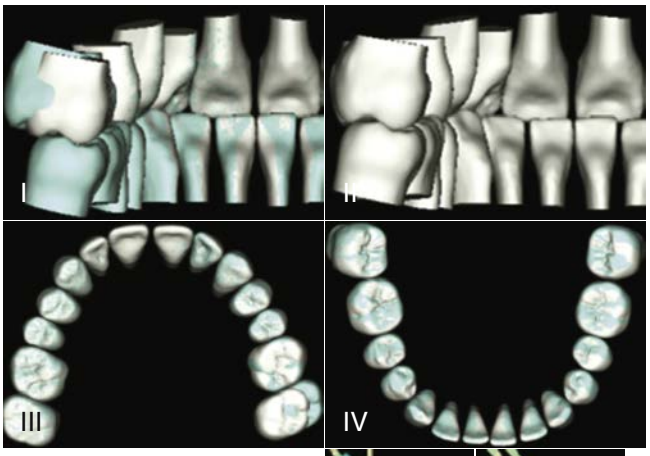
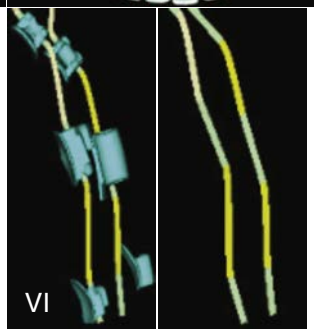


FIGURE 32-21 **A–D,** Mandible is shifted to the patient's left. The simulation of a mandibular shift corrects the midline but does not completely resolve the crossbite in the left molar region; crossbite also appears in the right molar region. **E,** Interference (*arrow*) appears on the inclined planes as in the upper right molar area as the mandibular shift is corrected.

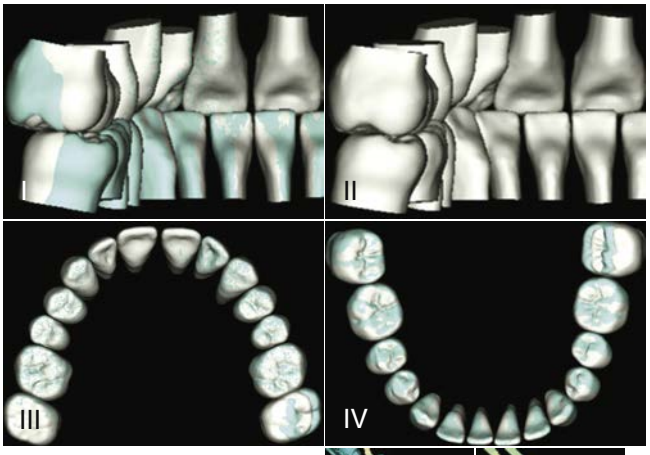


	6	7
Mesial (+) Distal (−) [mm]		−0.5
Buccal (+) Lingual (−) [mm]		−2.8
Occlusal (+) Gingival (−) [mm]	0.1	0.9
Torque facial (+) Lingual (−) [deg]		−2
Angulation mesial (+) Distal (−) [deg]		5
Rotation mesial (+) Distal (−) [deg]	−8	−8
Mesial gap (+) IPR (−) [mm]	0.1	0.1

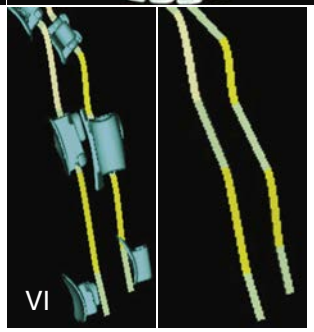


V

A



	7	7
Mesial (+) Distal (−) [mm]		
Buccal (+) Lingual (−) [mm]	−1.3	1.3
Occlusal (+) Gingival (−) [mm]	0.2	0.2
Torque facial (+) Lingual (−) [deg]	−5	3
Angulation mesial (+) Distal (−) [deg]		



V

B

FIGURE 32-22 Smart tooth movement. The residual crossbite is corrected by two different kinds of movements of the molars. **A,** *I–IV,* Upper left 7 is moved lingually. *V,* Tooth displacement values. *VI,* Targeted archwire design. **B,** *I–IV,* Residual crossbite, corrected by the movement of the upper and lower 7s, is consistent with the use of crossbite elastics. *V,* The displacement values of the upper and lower 7s are reciprocal. *VI,* Targeted archwire is designed for reciprocal movement. This movement may be easier to accomplish and faster than the correction planned in movement A.

FIGURE 32-23 A–D, Cephalometric superimposition of the simulation and original models, registered on the lower second molars. Simulation model is in white, and initial model is in blue-green.

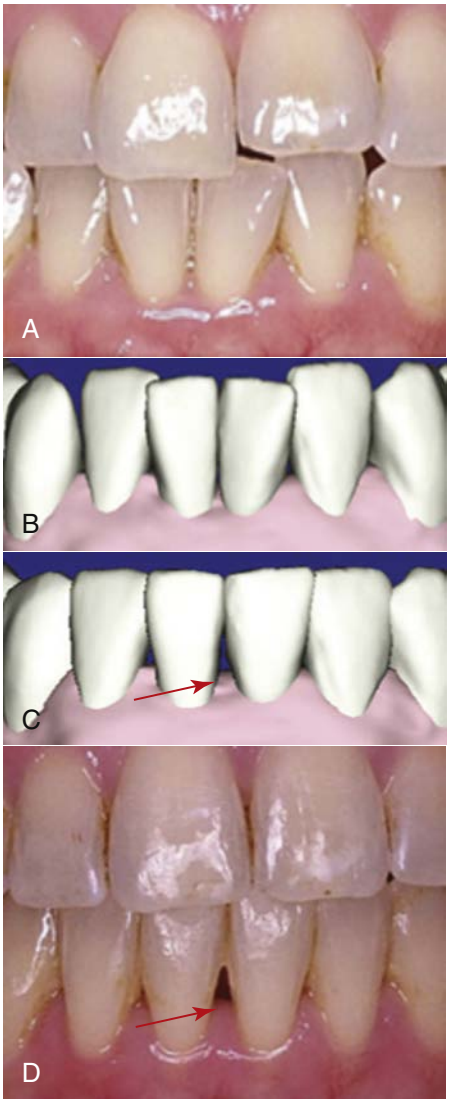


FIGURE 32-24 A, Virtual diagnostic model shows crowding of anterior teeth. B, Intraoral diagnostic image shows crowding of anterior teeth. C, Virtual simulation model (interproximal reduction [IPR] scenario) shows the largest black triangle (*arrow*) between the central incisors. D, In vivo condition at the end of treatment is demonstrated.

PRINCIPLES OF TARGETED THERAPEUTICS

Achieving effective treatment outcomes in an efficient manner requires a proactive therapeutic strategy driven by a diagnosis in all of the therapeutic phases of treatment. A full-expression archwire based on the targeted setup is only one of the many therapeutic solutions that can be designed with digital analytic and treatment planning techniques and fabricated with the robot to achieve predictable and controlled tooth movement. The versatility of designing diagnostic-driven and condition-based appliances for managing a dynamic biologic system is where the true strength of robotic-assisted orthodontics resides. The following text describes the concept of the adaptive prescription, which provides the foundation of the design principles in managing patient care with robot-assisted orthodontics.

Adaptive Prescription

The straight archwire concept of treatment is designed on the principle of a deterministic system. The operating paradigm is that a bracket with an ideal prescription ideally placed on a tooth will lead to an ideal response. This strategy, at best, is naive and needs to be challenged. A bracket cannot be ideally placed most of the time. Anatomic constraints attributable to severely rotated teeth, partially erupted teeth, and variation in tooth morphologic structure are some clinical examples that limit their ideal placement.

Furthermore, only a slight alteration in bracket position, such as to avoid interferences, can substantially affect the expression of a bracket prescription, forcing the clinician to practice reactive orthodontics. The altered expression of the bracket as a result of repositioning can only be corrected by archwire bending (Fig. 32-27).

Even with fully erupted teeth and the use of indirect bonding techniques, ideal bracket placement remains difficult for some because of the lack of reliability in seating the transfer trays.[8,9]

In addition, indirect bonding does not overcome the impact of inconsistencies in tooth morphologic structure; it simply provides an efficient way to improve precision and place brackets en masse at the chairside. Another problem with the straight arch appliance brackets resides with the bracket prescription. Straight wire prescriptions are standard and do not reflect the individual patient's unique needs. Furthermore, the bracket slot dimensions often demonstrate poor manufacturing tolerances and diminish precise control.[10,11] It might be argued that customized brackets may solve this issue, but this is not necessarily true.[12,13] The slots in customized brackets are milled just as

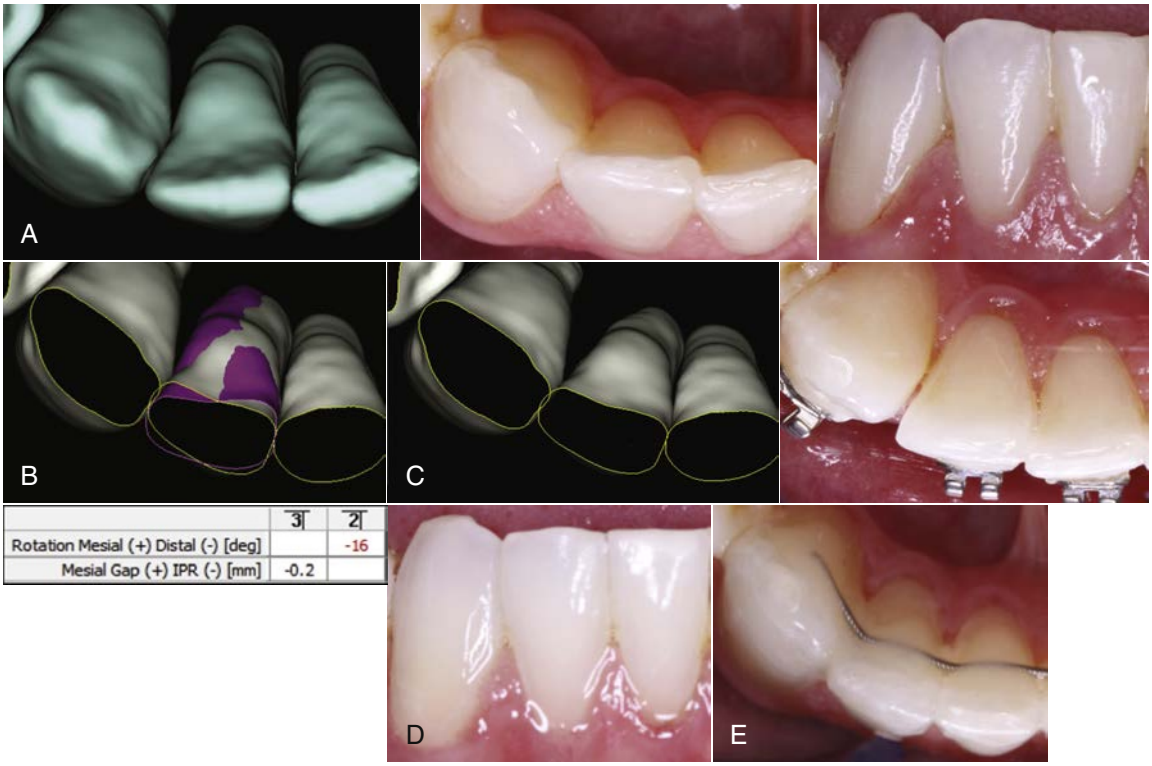


FIGURE 32-25 Constraint detection and management. **A,** Mesial outrotation of lower left lateral incisor is simulated. **B, C,** When the tooth is rotated 16 degrees (assuming no movement of the canine), then collision will occur on the distal surface of the lateral and mesial surfaces of the canine. The overlap automatically demonstrates the magnitude of the collision. The author of this text uses this as a guide to show how much interproximal reduction (IPR) is performed. **D, E,** Final photographs.

for the off-the-shelf brackets and therefore are subject to the same issues of poor manufacturing tolerances. Again, the conflict between vertical bracket placement and tooth morphologic structure cannot be reconciled by customized brackets. Any change in torque values exhibits itself not only in relative vertical discrepancies but also in buccolingual dimensions, setting up a vicious error-generating cycle.

Most important, however, is that orthodontic care occurs in an environment that reflects a complex adaptive system. John Holland has defined the complex adaptive system as, "A dynamic network of many agents acting in parallel, constantly acting/reacting to other agents." The overall behavior of the system is a result of a huge number of decisions made every moment with multiple variables affecting treatment response (Fig. 32-28). Many clinical instances, such as poor patient compliance, unplanned anchorage loss, and spurious tooth movement, force the doctor to reassess his or her treatment objectives in midtreatment. In such situations, deterministic-designed appliance systems fail to account for the altered therapeutic requirements to manage the new care objectives. They do not provide the clinician the flexibility to override the prescription built into the appliance, short of rebooting or resorting to craft-based reactive therapy. Robotic-assisted archwire bending driven by therapeutic diagnosis provides the doctor unprecedented control to modify the targeted prescription at will and to adaptively manage the dynamic clinical situation. Building constriction or expansion or selectively staging the expression in a static prescription offered by a bracket remains almost impossible without resorting to archwire shaping or bending.

Conventional straight archwire therapy and indirect bonding with or without customized prescriptive brackets are limited in their capacity to provide efficient control in the management of orthodontic patients. An adaptive prescription built into the archwire with robotic-assisted orthodontics provides a more realistic approach to manage orthodontic care, which is a manifestation of a complex adaptive system.

Principle of Image-Guided Smart Bracket Placement and Archform Selection

By using 3D-modeling simulation tools, the author of this chapter tries to understand the impact of tooth morphologic structure on bracket-archwire interaction and also uses the tool to determine where the brackets should be placed to achieve the treatment goals, minimize collisions, and avoid the risk of bracket failure. Additionally, the simulations are used to develop a visual guide to better position the brackets at chairside (Figs. 32-29 and 32-30) or in an indirect bonded setup. With the simulation tools, plans for the selection or modification of the straight archwire form, which will be initially used in the pre-SureSmile phase of treatment, are made.

Principle of Minimal Archwire Replacement

In the pre-SureSmile therapeutic phase, superelastic nitinol archwires are commonly used, and the goal is to minimize archwire changes through the appropriate selection of material, archwire cross section, and transformation temperature (Fig. 32-31). If tooth geometry leads to inconsistent force systems, segmental

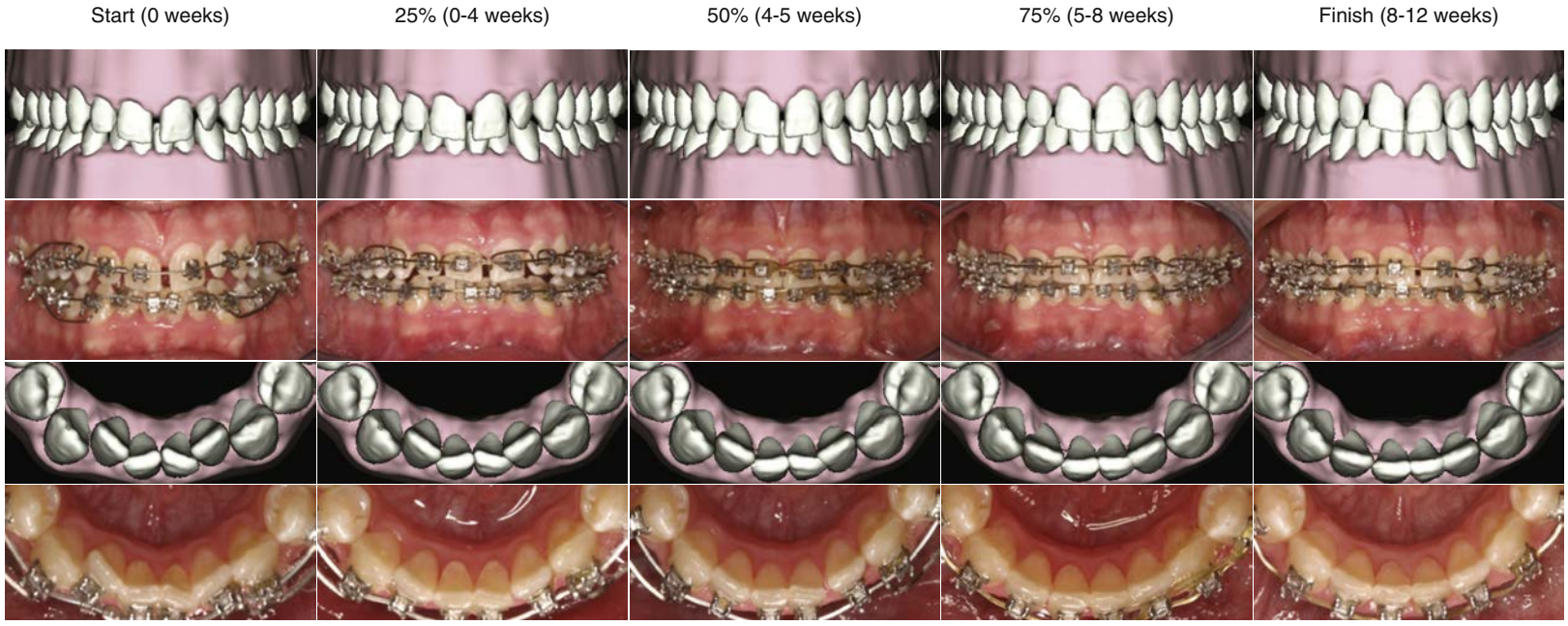


FIGURE 32-26 Anticipatory orthodontics. Progress milestones have been established with simulations.

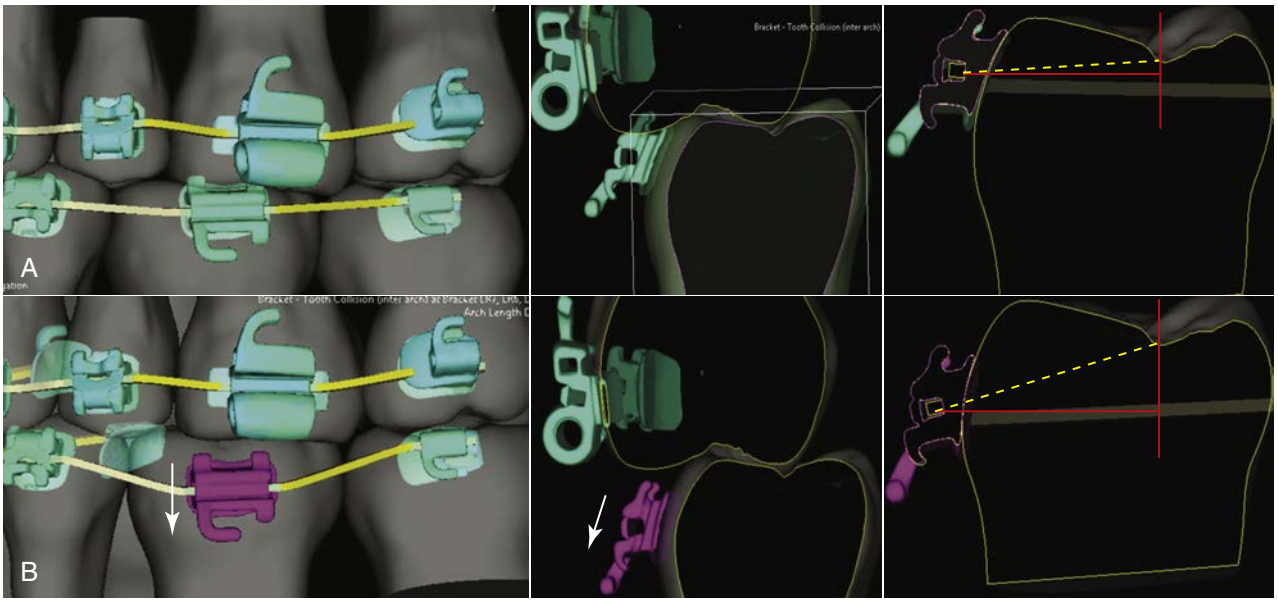


FIGURE 32-27 Reactive orthodontics are driven by bracket position. This example demonstrates the effect of vertical bracket repositioning on torque. **A,** Clipping plane view shows that the occlusal wings of the lower first molar are colliding with the upper first molar. Note the torque of the bracket. **B,** The bracket is gingivally repositioned to avoid collision. Note the effect this has on torque expression. Many times, adjustments are made in bracket placement without compensating for these effects, which then leads to reactive therapeutics with the archwire bending to manage the resulting spurious tooth movement.

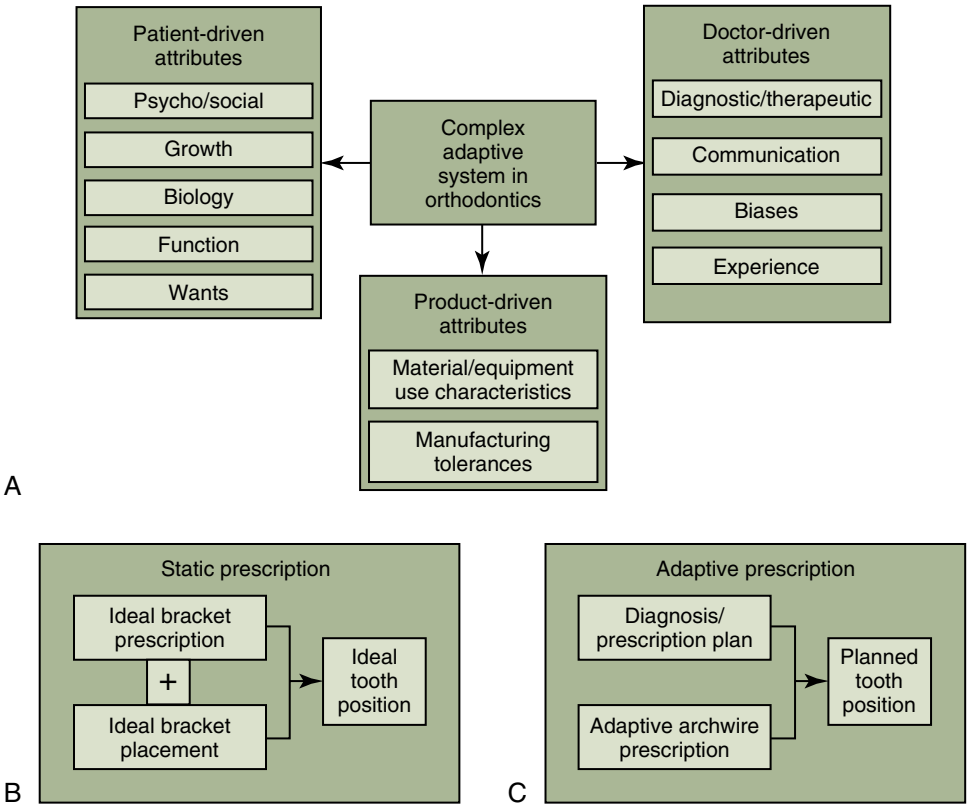


FIGURE 32-28 **A,** Factors affecting treatment response. **B,** Assumptions in appliance-driven mechanotherapy. **C,** Adaptive prescription therapy best serves in the management of the complex adaptive system such as in orthodontic care.

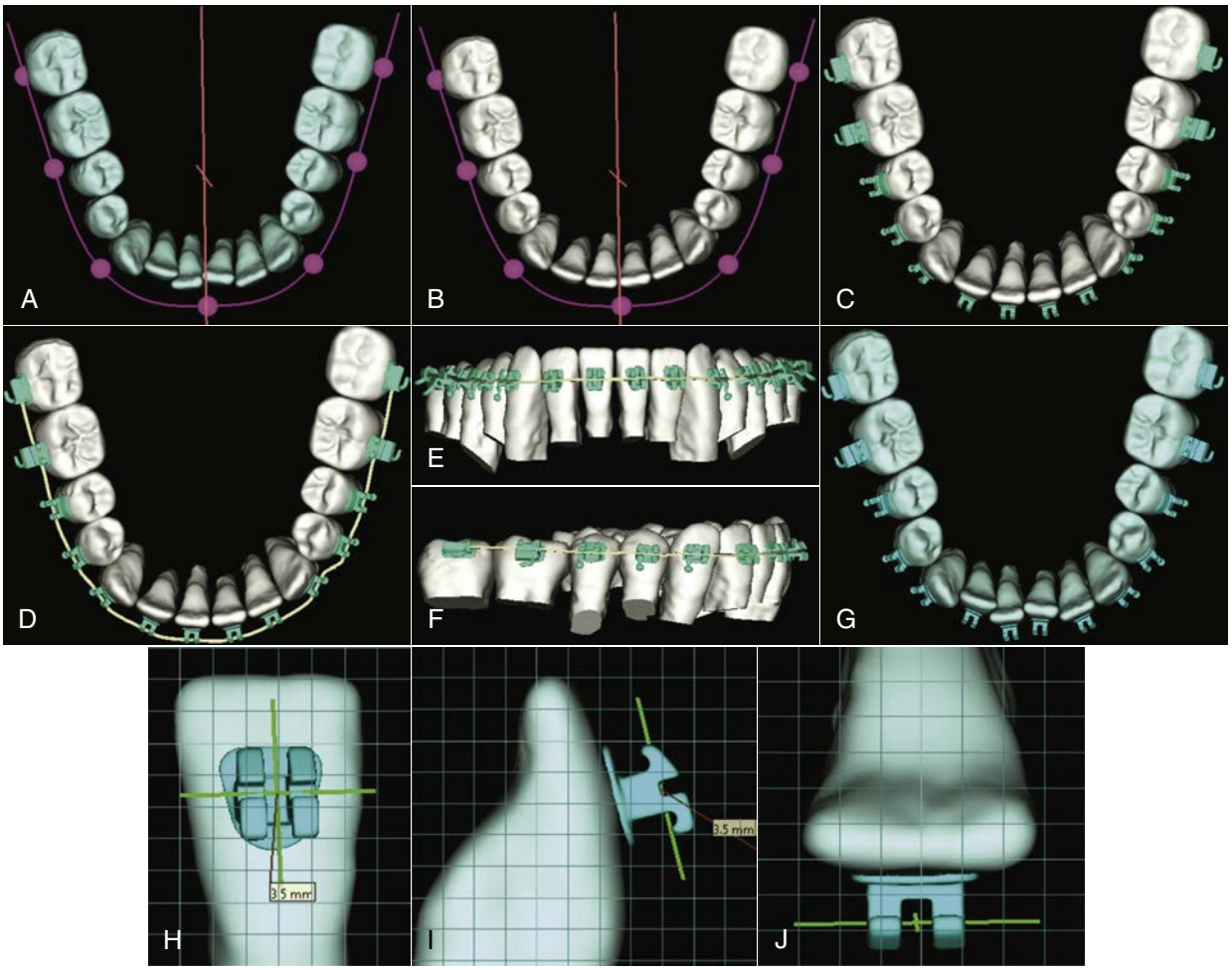


FIGURE 32-29 Smart bracket placement. **A,** Initial model with selected treatment arch form. **B,** Simulation of tooth alignment and leveling. **C,** Automatic bracket placement achieves the desired result. **D–F,** Despite optimization of bracket placement, archwire bends will be required to achieve desired result. **G–J,** Coordinate transfer used to place brackets on original model. The bracket position on each tooth can be visualized and measured to guide placement.

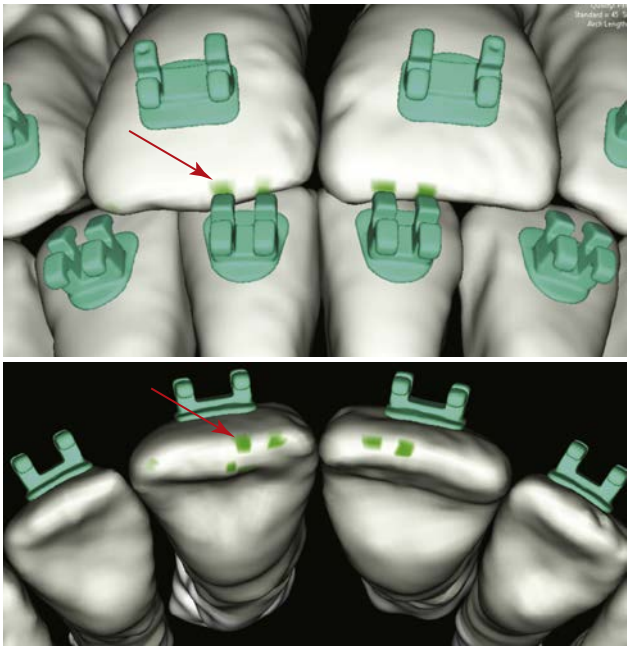


FIGURE 32-30 Bracket collision (*arrows*) can be detected in advance of placement and avoided.

mechanics may be used to achieve the desired control. The principle of concurrent mechanics (discussed later in this chapter) also minimizes replacement therapy.

Principle of Constraint Management

As mentioned earlier, if warranted by treatment objectives, IPR may be performed to minimize any potential collisions between the teeth, which allows for quicker alignment and leveling. Interferences between opposing teeth can prevent their movement through collision. The clinician may also bond bite blocks early in the treatment to disclude the arches and to prevent collisions from occurring (Fig. 32-32).

Principle of Concurrent Mechanics

Auxiliary appliances, such as torquing springs, tip-back springs, and elastics, may be used from the start of treatment when required. Complementary force systems may be generated to gain better control of tooth movement and to achieve multiple treatment goals, such as alignment leveling and space closure in parallel (Figs. 32-33 and 32-34).

FIGURE 32-31 Minimal replacement alignment achieved in 4 months with initial 0.19- × 0.25-inch copper-nickel-titanium (CuNiTi) A_F 40° C archwire that was fully engaged.

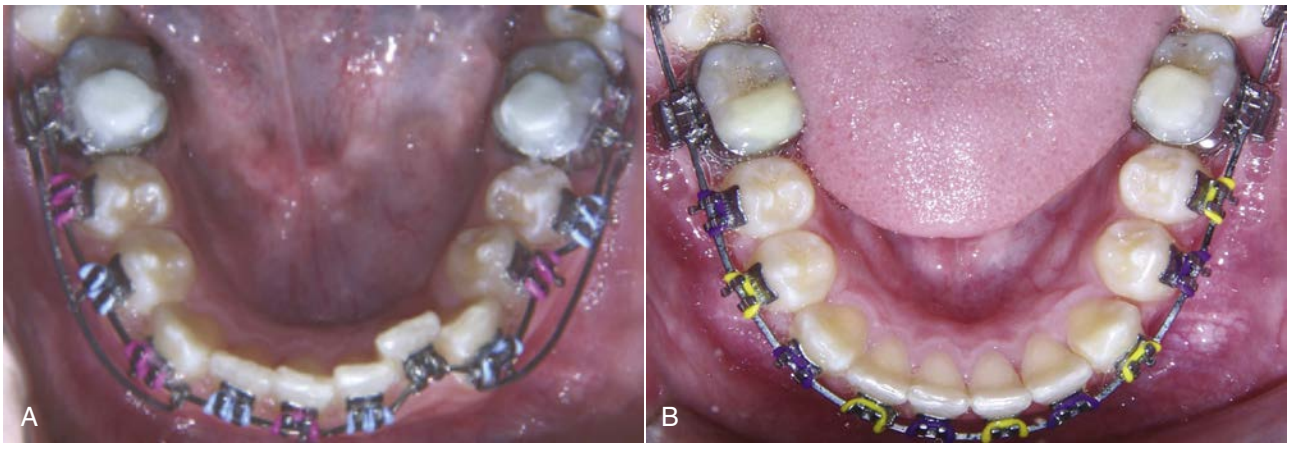


FIGURE 32-32 A, B, Posterior bite blocks are bonded to prevent interarch collisions.

Principle of Additive or Subtractive Bending, Preferential Staging, and Shaping

The expression of the digitally designed and robotically constructed archwire can be very effectively influenced to provide unprecedented control of tooth movement by either adding or subtracting bends at any point in the archwire or as a whole to achieve different levels of archwire expression.

A full-expression, 100%-active archwire can be designed for use from the start of treatment (see Fig. 32-9). The expression may also be incrementally staged to control the amount of tooth displacement, especially in situations during which the patient has a low threshold to pain, is not compliant, or is on an extended vacation (Fig. 32-35).

An added dimension of control can be achieved by limiting bends to any one plane of space. For example, control can be achieved by removing all bends except for the first order or creating a straight segment to achieve sliding mechanics during space closure (Fig. 32-36). A curve of Spee, reverse curve, expansion, and constriction can all be designed into the archwire to build additive forces or subtract the reactive forces observed when using elastics.

FIGURE 32-33 A–D, Concurrent mechanics; ART (Art Root Torquing) (Atlanta Orthodontics, Atlanta, GA). Torquing springs are being used at the onset of treatment with 17- × 25-inch copper-nickel-titanium (CuNiTi) 35° C archwire to correct edge-to-edge bite. The bite is anteriorly opened with posterior bite blocks to minimize anterior interference during correction.

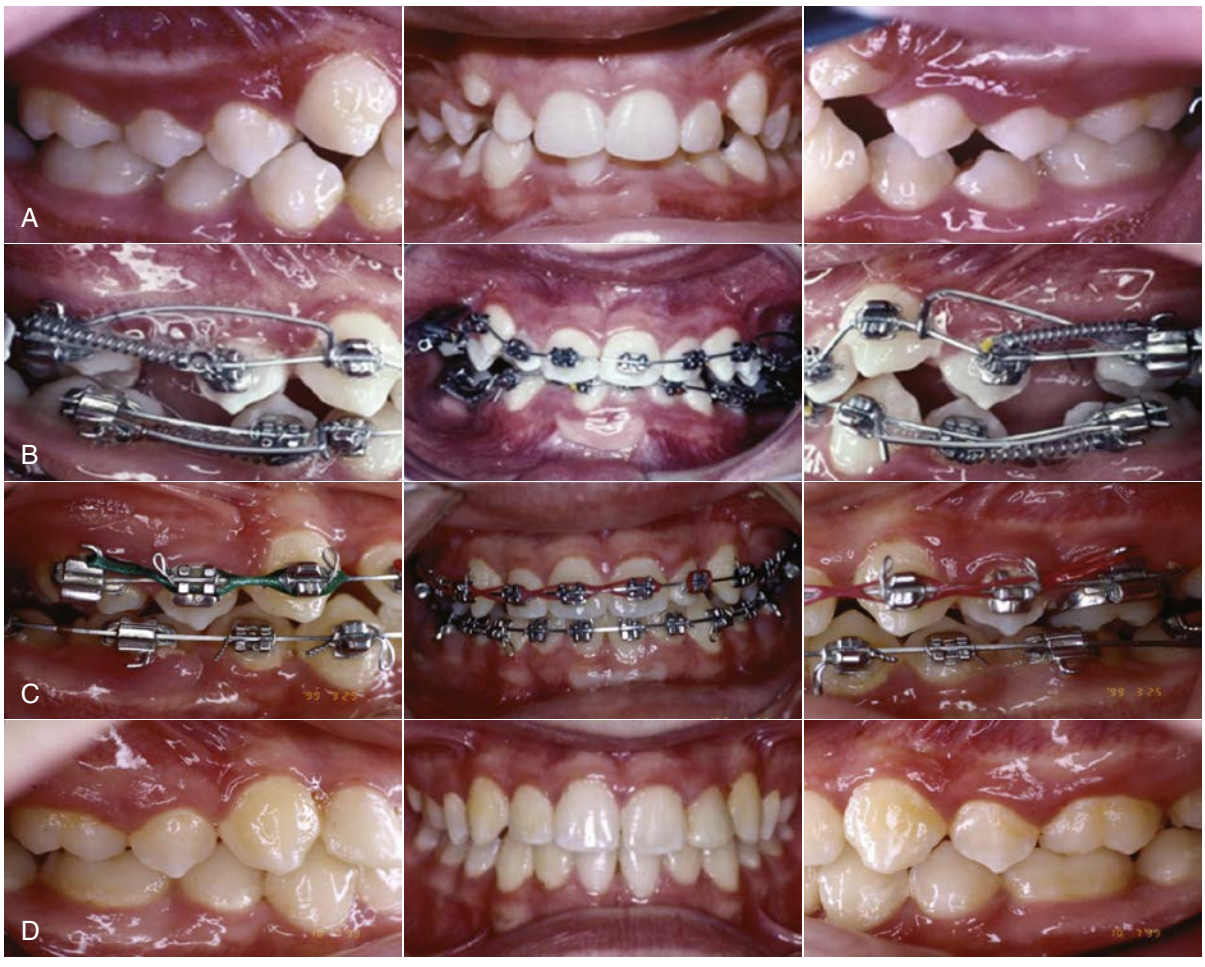


FIGURE 32-34 A–D, Concurrent mechanics. Sliding mechanics are being used to close space. The initial wire is a 0.17- × 0.25-inch copper-nickel-titanium (CuNiTi) A_F 35° C. Tip-back springs, fabricated from 0.17- × 0.25-inch titanium molybdenum alloy (TMA), were used to aid in leveling and minimizing the tip forward of the buccal segments and distal crown tipping of the canines during space closure.

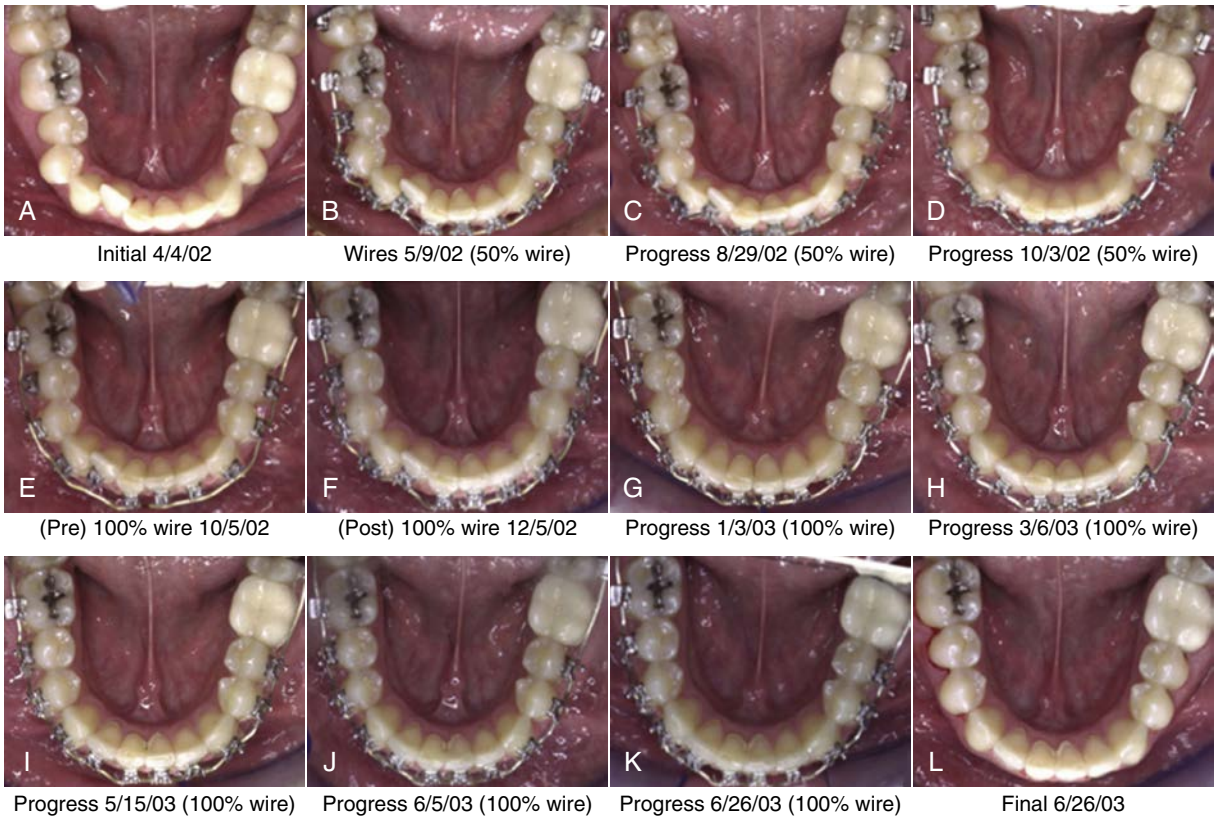


FIGURE 32-35 Progressive SureSmile expression archwire. **A,** Initial. **B,** SureSmile archwire with 50% expression inserted. **C, D,** Progress with 50% SureSmile expression archwire. **E,** SureSmile archwire with 100% expression inserted. **F–K,** Progress with SureSmile 100% expression archwire. **L,** Finish.

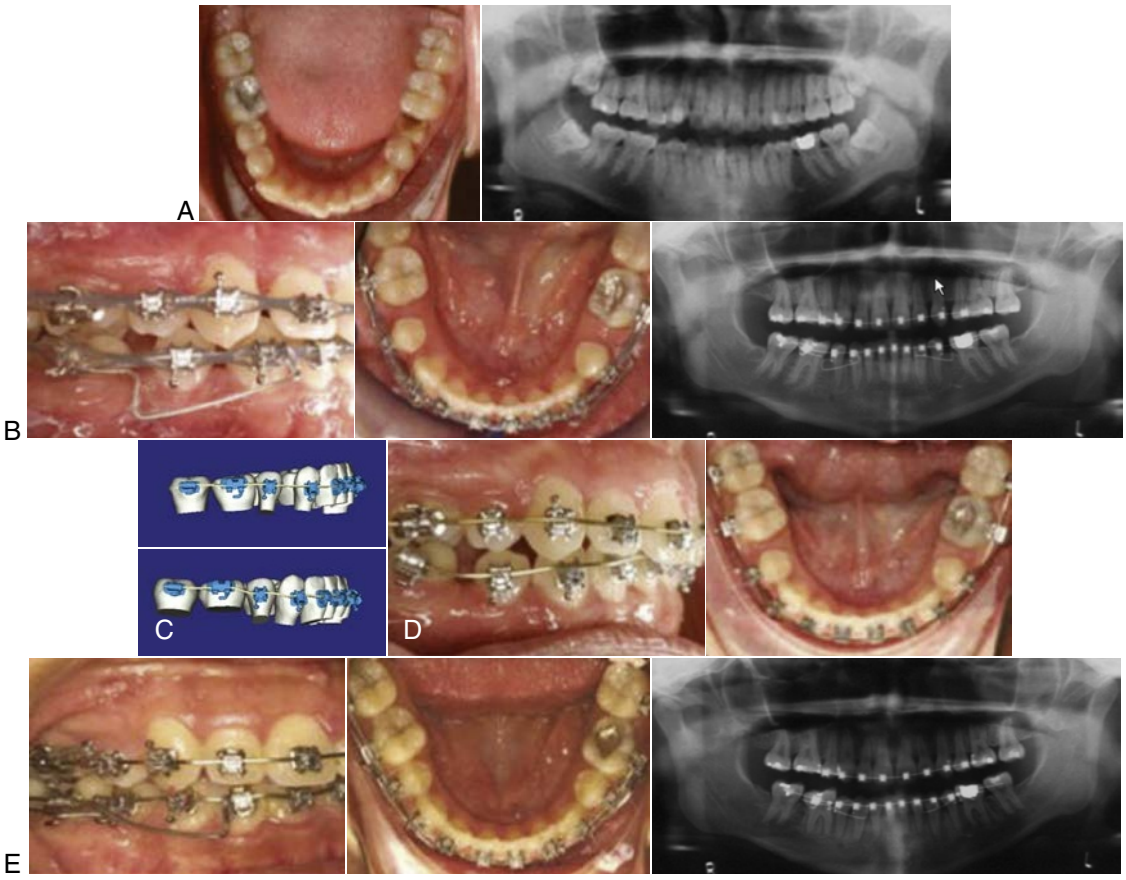


FIGURE 32-36 SureSmile archwire designed to enable sliding mechanics. **A,** Initial photograph and panoramic radiograph. **B,** Initial half of the space has been closed on straight archwire. **C,** Two types of SureSmile archwires are available. The first archwire is designed for space closure. It is straight distal to the canines and has full anterior expression. The second archwire will be used after the space is closed. It is the final archwire. **D,** Progress with SureSmile wires. **E,** Space is closed on the SureSmile wire.

Principles of Consistent Force Systems and Reactive Force Management

The unmanaged use of straight archwires will create inconsistent force systems in many situations, leading to spurious tooth movement (Fig. 32-37). Designing passive and reactive elements can prevent this movement. Furthermore, hybrid archwires can be designed with both active and passive elements to better distribute the reactive forces and target the active forces (Fig. 32-38). Passive archwires can be designed to minimize the side effects of Class II mechanics (Fig. 32-39).

Principle of Complementary Force System

Many times, toward the end of treatment, mild to moderate Class II or Class III correction remains unachievable, despite long-term elastic wear. The most common reason for this is that bracket archwire coupling may impose reactive force constraints and fight the Class II or Class III elastic forces. This eventuality may be designed for in bracket placement but becomes a daunting task; it requires precision bracket placement in all three planes of space. This kind of complex placement is difficult to accomplish clinically. Instead, when using a digital tool set, such situations can be anticipated on the target setup, and the virtually designed archwire can be made to complement the force system generated by the Class II or Class III elastics, springs, or functional appliances (Fig. 32-40).

Principle of Optimization of Archwire and Material Selection

The availability of different archwire materials, sizes, and shapes increases the doctor's need to be judicious when selecting archwires and to guide his or her decisions based on material characteristics, the patient's condition, the desired treatment objectives, and whether elastics are being used. High load–deflection rate materials such as Elgiloy (Rocky Mountain Orthodontics, Denver, CO) offer greater control of reactive segments, whereas superelastic archwires provide long-acting sustained forces that are beneficial for active movements over the long term.

The capability of being able to bend nitinol wire and retain its superelastic characteristics with robotic-assisted archwire bending offers the orthodontist a number of distinct advantages: (1) the ability to establish torque control very early in treatment extends its use and minimizes the number of wire replacements needed; (2) biologically friendly forces can be applied through the entire range of care; and (3) torque control

FIGURE 32-37 Passive SureSmile bypass wire controls inconsistent force systems. **A,** A common side effect of extruding canines with a continuous archwire is the intrusion of the anteriors, which creates an anterior open bite (*arrows*). **B,** The therapeutic strategy to minimize this side effect is to design a passive bypass archwire and piggyback an active archwire to extrude the cuspids. The bypass archwire helps stabilize the reactive segment and minimize side effects.

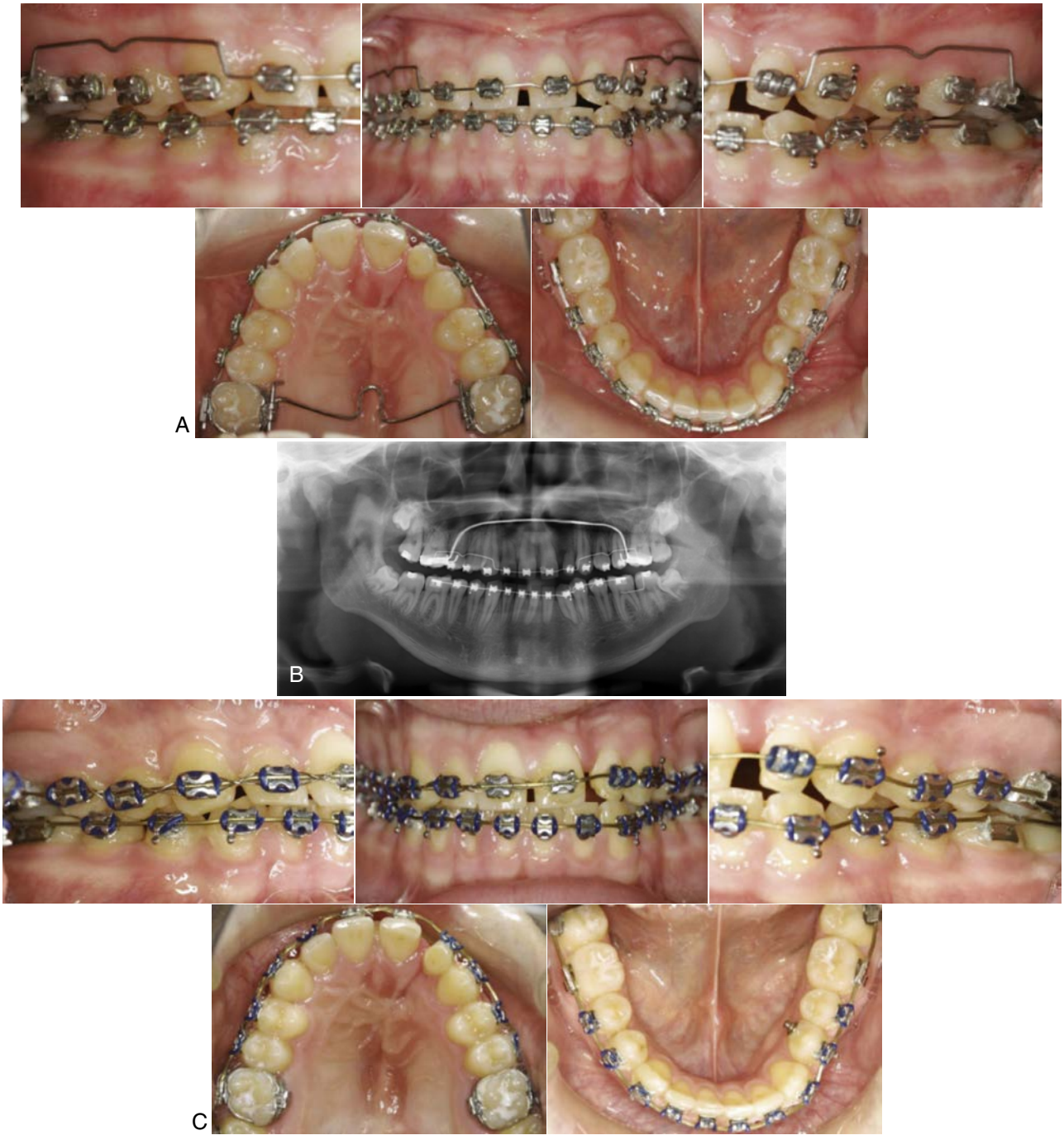


FIGURE 32-38 A–F, Hybrid SureSmile archwire design. A combination of passive and partially progressive bends is used to expand the lower left buccal segment. A continuous archwire would have had the tendency to expand both sides by creating a passive archwire from the lower left canine to the second molar on the right. A relatively rigid reactive segment is established that better distributes the reactive forces. In addition, by designing progressive expansion active bends 50%, followed by 100% on the left buccal segment, the displacement of the teeth is better titrated, minimizing the potential of spurious movement.

D

E

F

FIGURE 32-38, cont'd

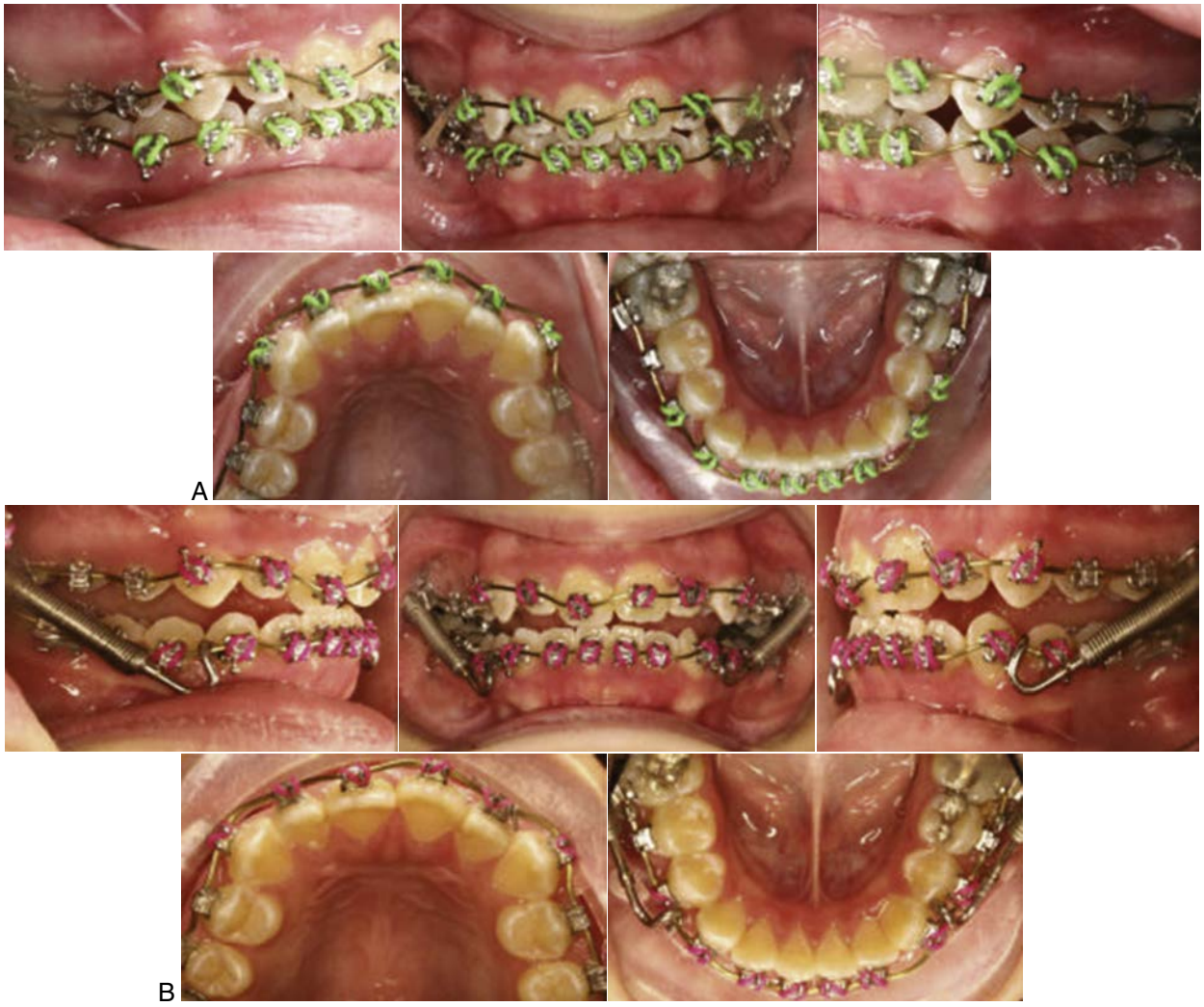


FIGURE 32-39 A, B, Class II correction with Forsus Fatigue Resistant Device (3M Unitek, St. Paul, MN). SureSmile spring/passive 19- × 25-inch copper-nickel-titanium (CuNiTi) A_F 35° C archwires are used for anchorage in the upper and lower dentition.

does not require the use of a near full-size archwire but can be accomplished by accommodating for bracket play in the region of interest. Such a situation can occur when substantial anterior crowding demands the anterior use of a smaller cross-section archwire, whereas posteriorly, a single tooth such as the second molar requires torque.

EFFICACY AND EFFECTIVENESS

A number of studies have been designed to understand the efficiency and effectiveness of using robotic-assisted orthodontics. Saxe and colleagues[14] studied 62 patients treated with advanced digital technology with a similar control sample and showed that the mean total months for treatment using the SureSmile process was 14.7 months versus 20 months for conventional methods ($p < 0.001$). The ABO OGS grades were 14% lower (better) than conventional treatment. A recent study by Alford and colleagues[15] also reported that treatment time with SureSmile was reduced by approximately 30%, compared with conventional treatment, and that the cast/radiographic evaluation (CRE) scores were 11% better.

To this point, over 12,000 patient records have been analyzed to understand the effectiveness of SureSmile. The results clearly demonstrate that technology-driven diagnosis and treatment favorably influence treatment time across the spectrum of practices and types of malocclusions. The median treatment time for SureSmile patients with Class I is 15 months (conventional method, 24 months); Class II, 13 months (conventional method, 23 months); and Class III, 16 months (conventional method, 25 months).[16] Recent research conducted by Patel and colleagues[17] at the University of Oklahoma demonstrated no statistical differences in root resorption between robotic-assisted technology and conventional treatment. Future studies need to be conducted to understand further both the efficiency and the effectiveness of this technology in the management of orthodontic patient care.

FIGURE 32-40 Complementary force system. **A,** The patient has a Class III occlusion on the right buccal segment. **B,** Before the SureSmile therapeutic phase, the Class III correction remains unresolved, although the patient has been cooperative and has been wearing Class III elastics for 6 months. **C, D,** In the target setup, the dentition in the buccal segment is subtly set to correct the Class III in the lower bicuspid canine area. The archwire bracket geometry reflects this. **E,** The patient continued the elastic wear with the SureSmile archwire. Correction in occlusion is achieved as planned in the setup by building dentoalveolar compensations.

SUMMARY

Our profession is at the threshold of reinventing itself. We have the ability to transform our orthodontic care environment in which quality thrives by using enabling technology and applying the science of process management to evolve from a craft-based to a patient-centered and professional-based care model.

We have the unique opportunity to adapt and customize care to specific patient needs, to streamline our clinical protocols by embracing evidence-based care and evidence farming, to establish new benchmarks in care, and to embrace system-minded thinking.

Some may perceive these advances as a step toward the loss of personal autonomy and craft. Some may also fear that the new integrated digital tools and processes widen the distance between orthodontists and their patients, causing a loss of the human connection that most of us prize. Both of these fears are misplaced. Technology and processes, when viewed as enabling, provide unprecedented control for the doctor and staff members to deliver better patient-centered care.

We must view each practice as a *practice of one* and hold ourselves accountable to giving the best in care for each patient. We must subscribe to the practice of providing the right treatment for the right patient at the right time and to avoid, at every cost, the lure of market-driven orthodontics.

Acknowledgments

I would like to express my sincere thanks to Drs. Takao Kubota, Jun Uechi, Richard Ingraham, Edward Lin, Ralph Mueller, and Mark Feinberg for kindly providing some of their patient records for this publication. I cannot thank my mentors enough, Drs. Charles Burstone, Michael Marcotte, Larry White, and Carl Gugino, for unselfishly sharing their knowledge of orthodontics with me. I am eternally indebted to my former residents and patients for giving me the privilege to learn from them. To my friends and colleagues, Drs. Mark Knoefel, John Lohse, Takao Kubota, Doug Haberstock, Blair Adams, Geoff Hall, Michael Stosich, Sam Wong, Antonella Maselli, Raffaele Schiavoni, Con Vanco, Todd Hickman, Michael Mahafee, and in addition, Karen Moawad: not only has your presence in my life professionally enriched me, but, more important, you have allowed me to see beyond the "to-do list" and enjoy the present.

CASE STUDY 32-1

Patient Histories

Diagnostically driven, robotic-assisted orthodontics provides a very effective tool to achieve targeted therapeutics with predictable and controlled tooth movement at any point during the care cycle. Furthermore, any type of malocclusion can be treated with this approach, independent of the bracket type used. The following patient histories demonstrate the wide applicability of this approach. The shorter care cycles are a result of proactive planning, process control, and targeted therapeutics. Teeth are not moved faster, but they are moved smarter (Figs. 32-41 to 32-46).

A

Initial

B

Bonded

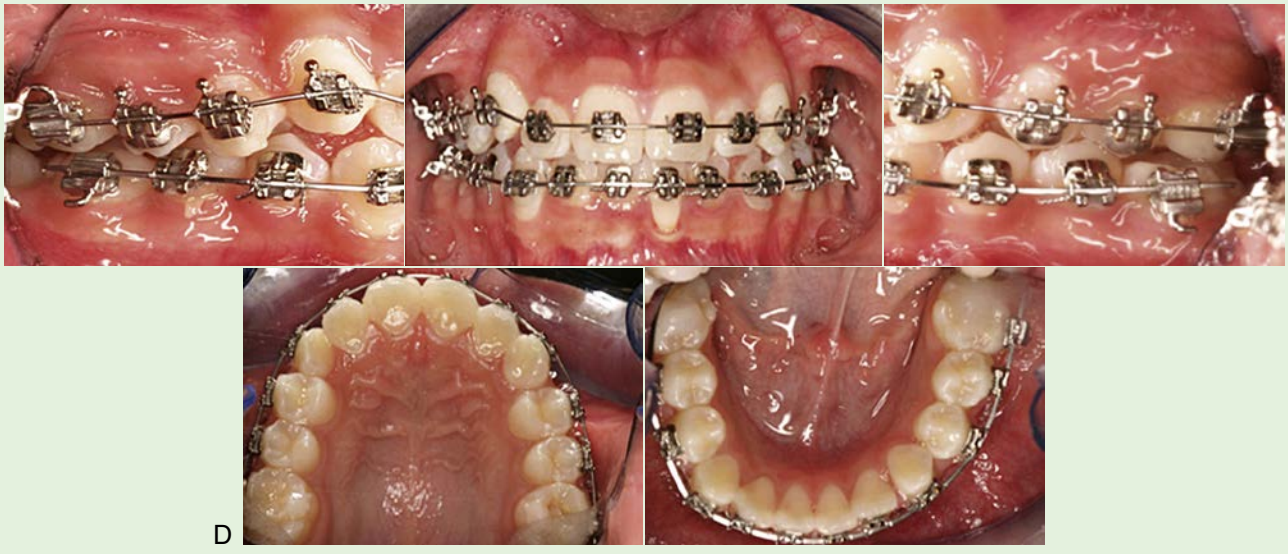
FIGURE 32-41 **A–I,** Class I severe crowding. Ormco (Orange, CA) 0.018 slot mini diamond brackets. Initial alignment wire 0.018 A_F 17- × 25-inch copper-nickel-titanium (CuNiTi) A_F 35° C, followed by 17- × 25-inch CuNiTi A_F 35° C in the lower arch; 17- × 25-inch CuNiTi A_F 35° C Sure-Smile prescriptive archwire.

Continued

CASE STUDY 32-1—cont'd

C

Progress

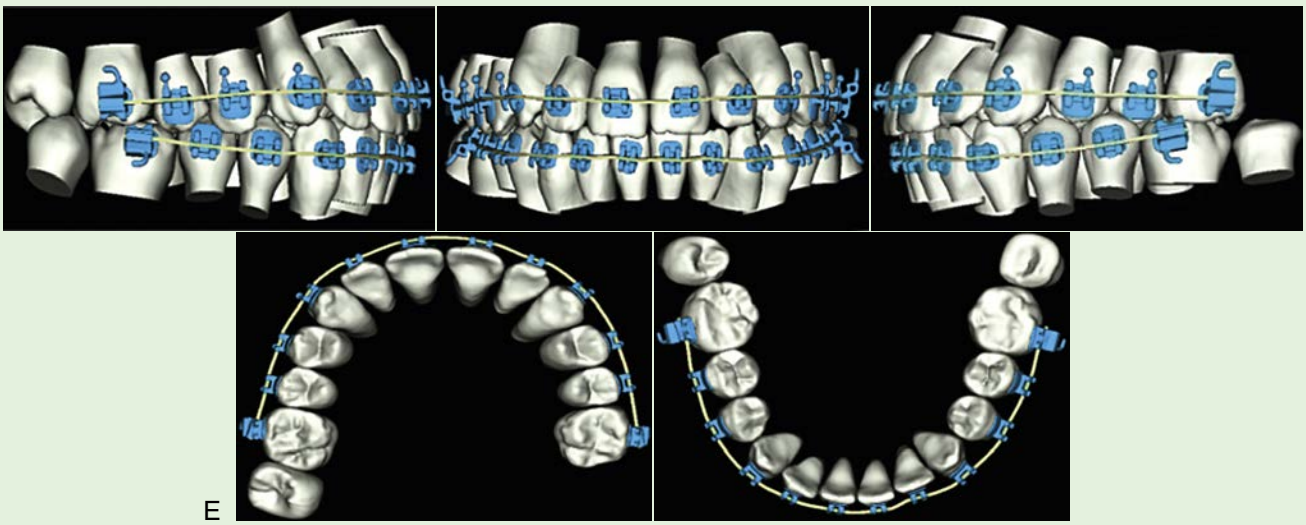


D

Progress

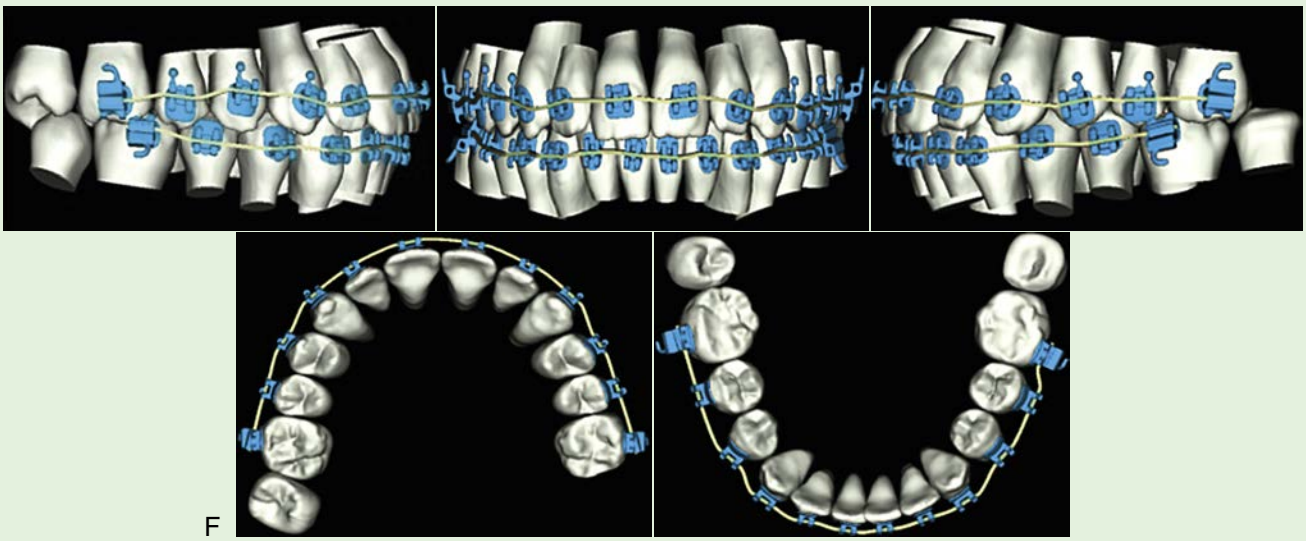
FIGURE 32-41, cont'd

CASE STUDY 32-1—cont'd



E

Therapeutic scan



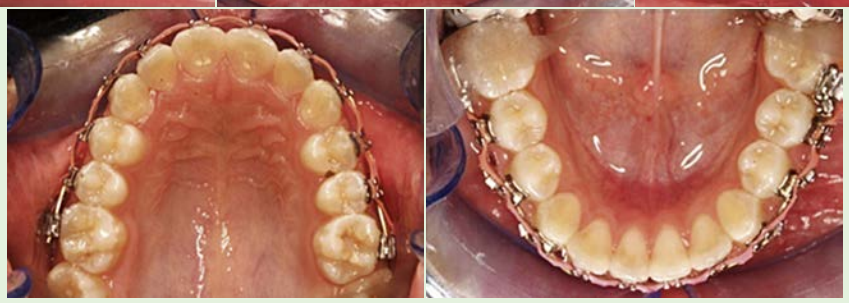
F

Setup

FIGURE 32-41, cont'd

Continued

CASE STUDY 32-1—cont'd

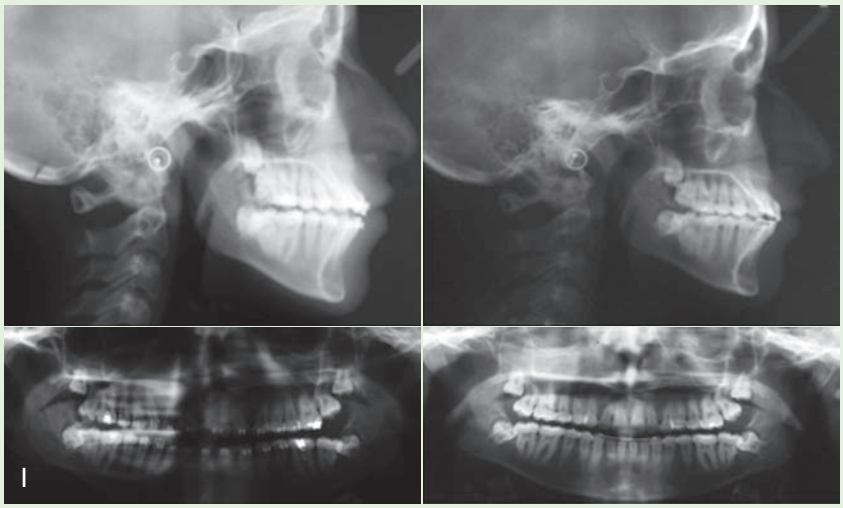


G

Prescriptive archwire

H

Final



I

Initial x-rays Final x-rays

FIGURE 32-41, cont'd

CASE STUDY 32-1—cont'd

A

Post
Invisalign

B

Post Invisalign

FIGURE 32-42 A–I, Class I with extraction. Patient was initially treated with Invisalign (Align Technology, Santa Clara, CA). Upper Inspire ICE and lower Damon System 2 (Ormco, Glendora, CA), 0.22 slot size bracket. Initial alignment wire 17- × 25-inch copper-nickel-titanium (CuNiTi) A_F 35° C followed by 19- × 25-inch CuNiTi A_F 35° C SureSmile prescriptive archwire. A revision wire 19- × 25-inch CuNiTi A_F 35° C was used. Additionally, ART (Art Root Torquing) spring torque auxiliaries were used to augment anterior root correction.

Continued

CASE STUDY 32-1—cont'd

C

Progress

D

Prescriptive archwire

FIGURE 32-42, cont'd

CASE STUDY 32-1—cont'd

E Progress

F Progress

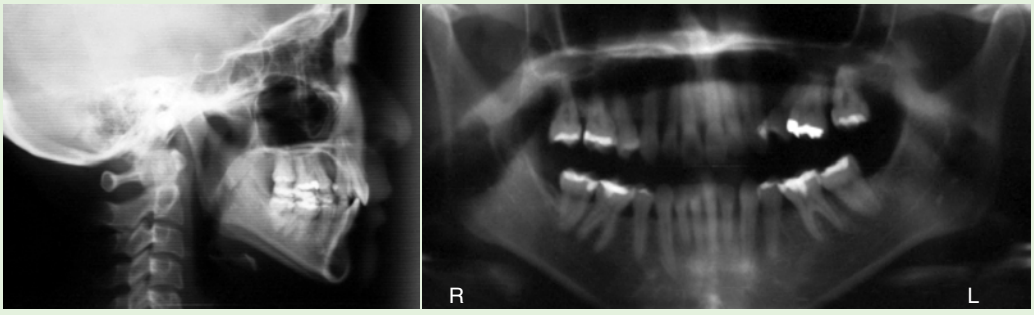
FIGURE 32-42, cont'd

Continued

CASE STUDY 32-1—cont'd

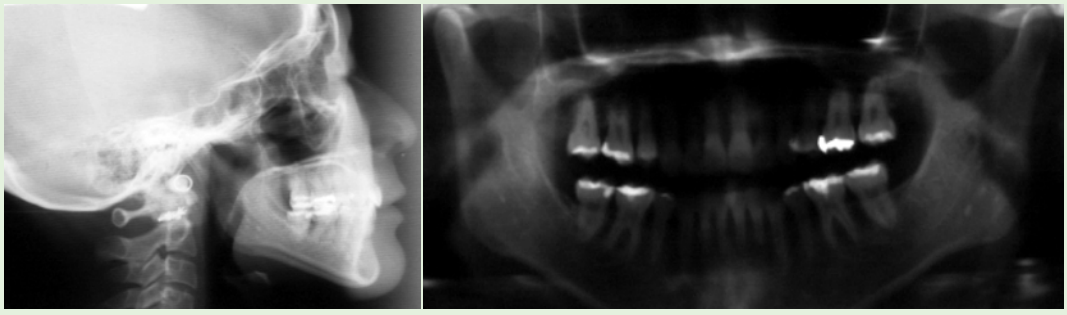
G

Final



H

Initial x-rays



I

Final x-rays

FIGURE 32-42, cont'd

CASE STUDY 32-1—cont'd

FIGURE 32-43 A–H, Patient with a Class II malocclusion. The upper and lower arches are bonded with 0.18-inch Dentsply GAC (Islandia, NY) In-Ovation 7-7 bracket systems. Class II correction achieved before therapeutic scan with 17- × 25-inch copper-nickel-titanium (CuNiTi) A_F 35° C. SureSmile prescriptive archwires were used in the therapeutic phase of treatment.

Continued

CASE STUDY 32-1—cont'd

C

Final

D

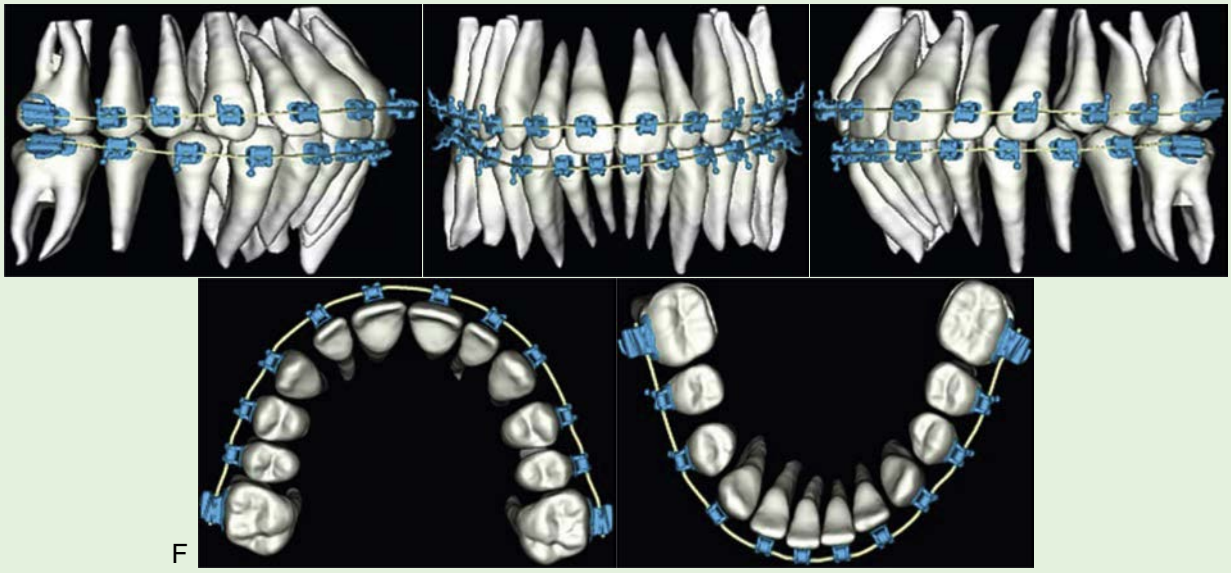
Initial x-rays

FIGURE 32-43, cont'd

CASE STUDY 32-1—cont'd

E

Initial model



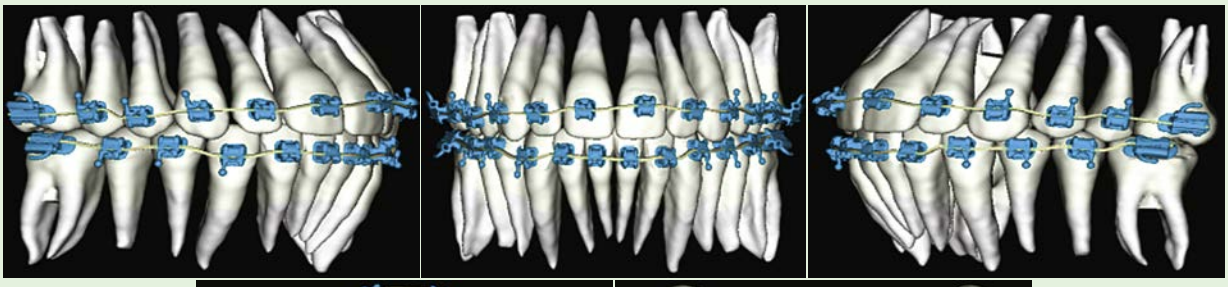
F

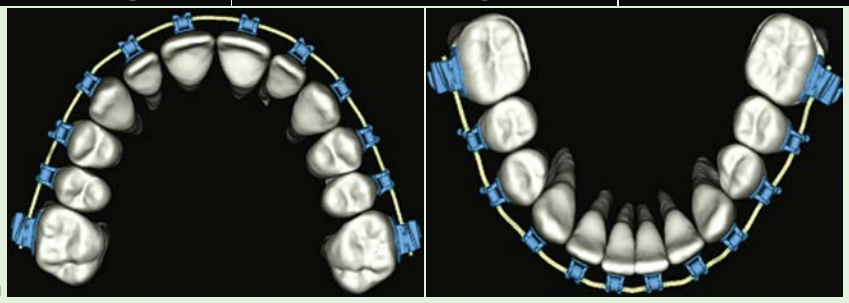
Therapeutic model

FIGURE 32-43, cont'd

Continued

CASE STUDY 32-1—cont'd





G

Setup

H

Final x-rays

FIGURE 32-43, cont'd

CASE STUDY 32-1—cont'd

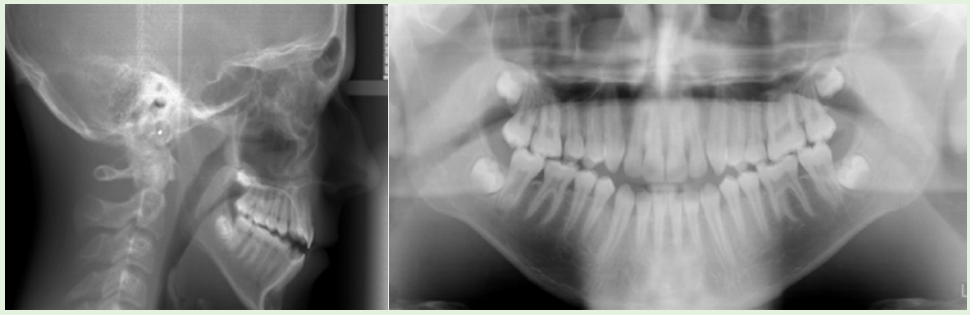
FIGURE 32-44 A–H, Open bite with mandibular shift. Patient is bonded with Mini Twin bracket system (3M Unitek, St. Paul, MN) with a slot size 0.018 inch. Initial archwire 17- × 25-inch copper-nickel-titanium (CuNiTi) A_F 35° C. SureSmile prescriptive archwire 17- × 25-inch CuNiTi A_F 35° C with Class III. Elastic wear on the right.

Continued

CASE STUDY 32-1—cont'd

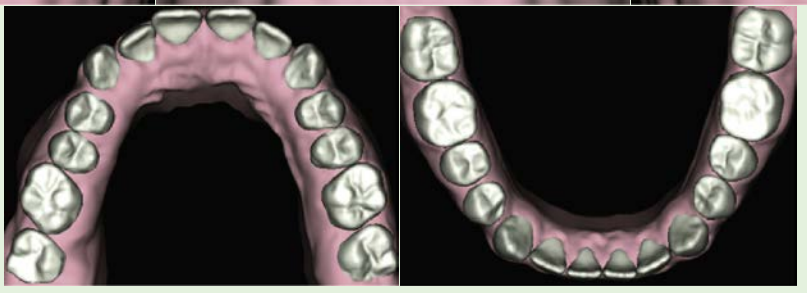
C

Final



D

Initial x-rays

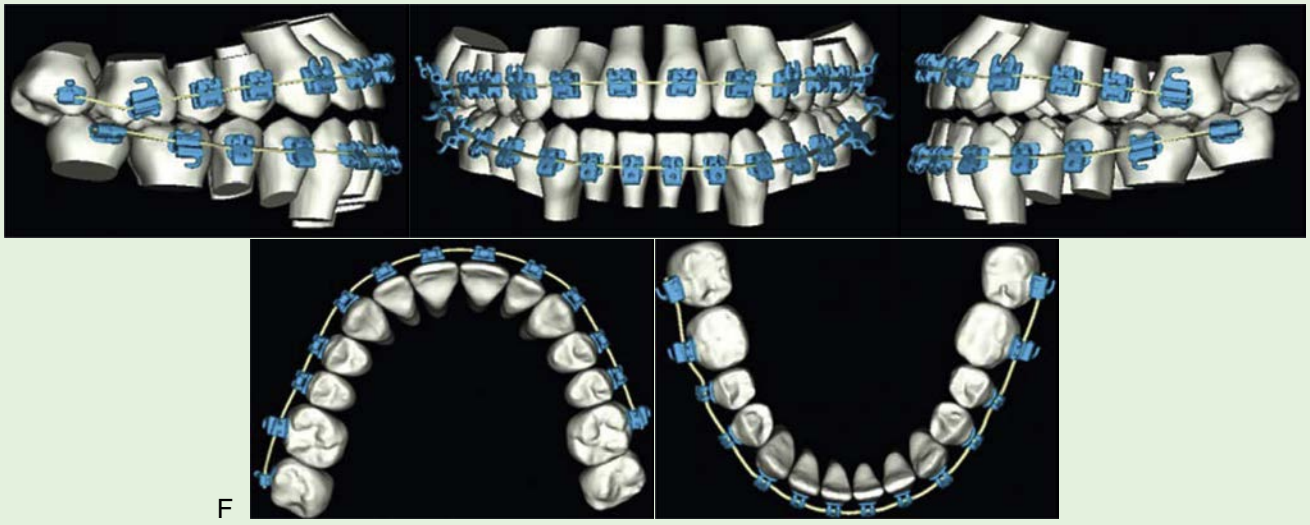


E

Initial model

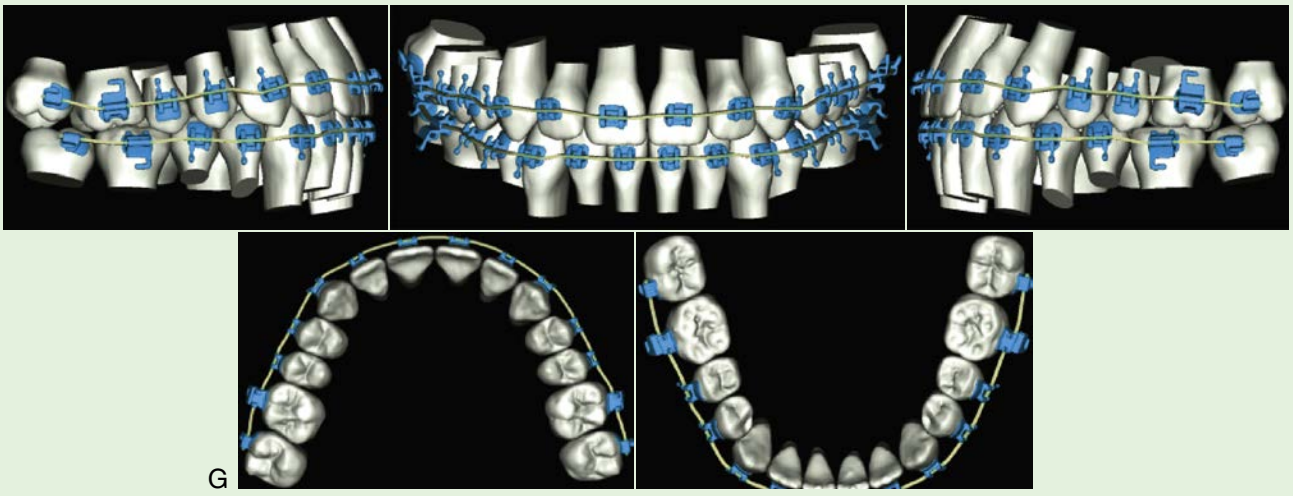
FIGURE 32-44, cont'd

CASE STUDY 32-1—cont'd



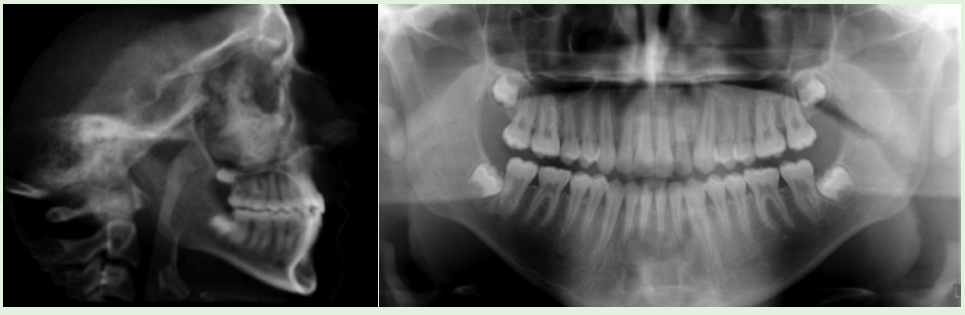
F

Therapeutic model



G

Setup



H

Final x-rays

FIGURE 32-44, cont'd

Continued

CASE STUDY 32-1—cont'd

A

Initial

B

Therapeutic

FIGURE 32-45 A–H, Class III open bite. Bonded upper with GAC In-Ovation bracket system (Dentsply GAC, Islandia, NY) and lower Time-2 brackets (American Orthodontics, Sheboygan, WI) with 0.018 slot size. Initial archwire 17- × 25-inch copper-nickel-titanium (CuNiTi) A_F 35° C; SureSmile prescription archwire 17- × 25-inch CuNiTi A_F 35° C.

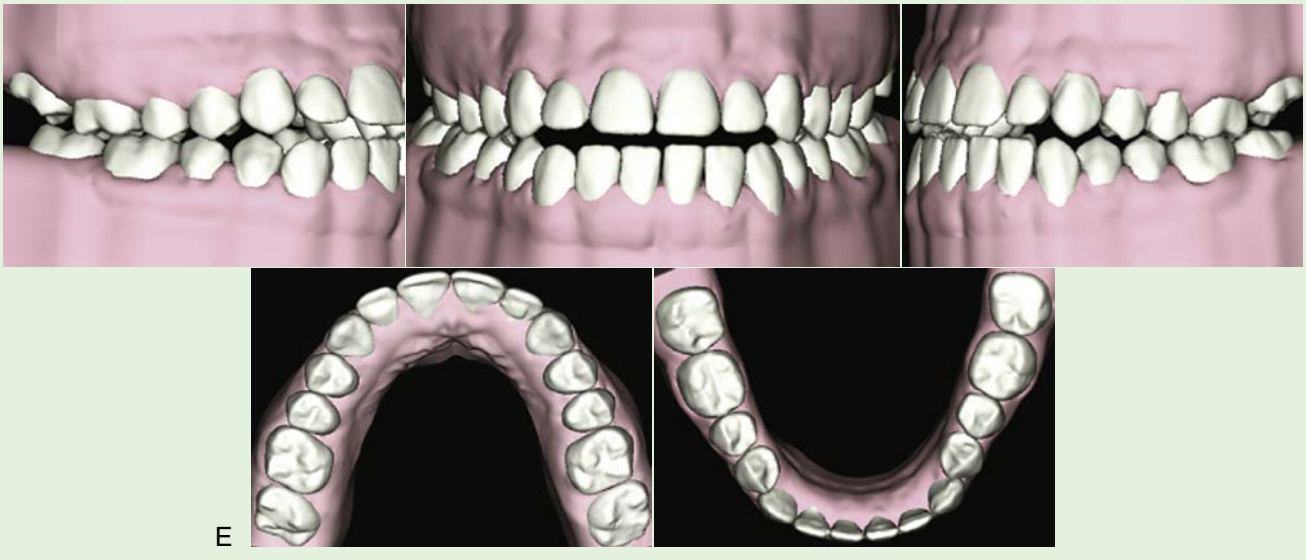
CASE STUDY 32-1—cont'd

C

Final

D

Initial x-rays



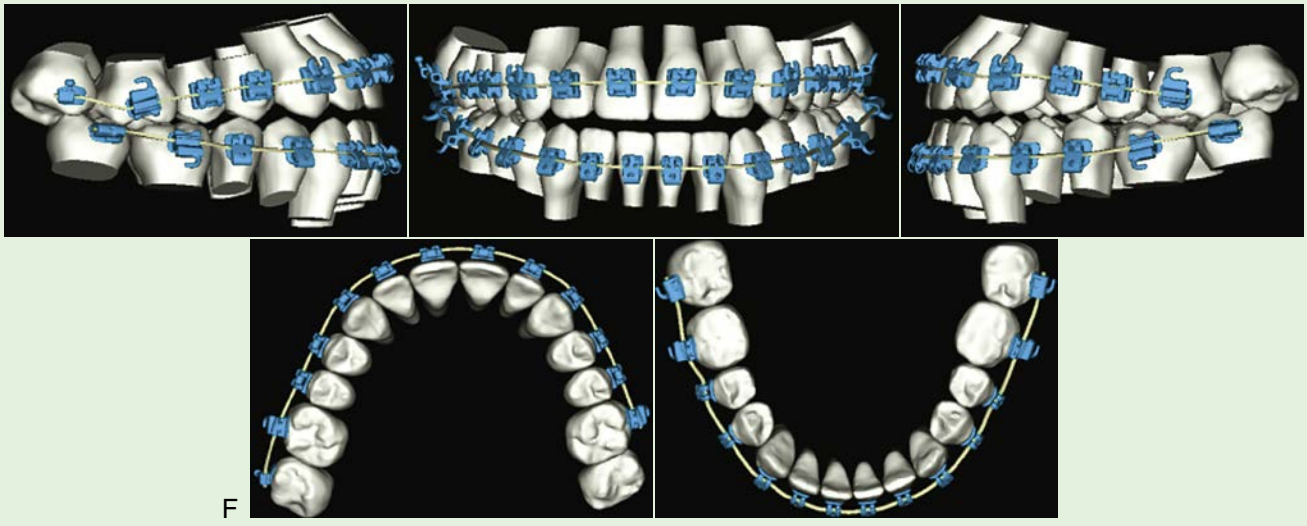
E

Initial model

FIGURE 32-45, cont'd

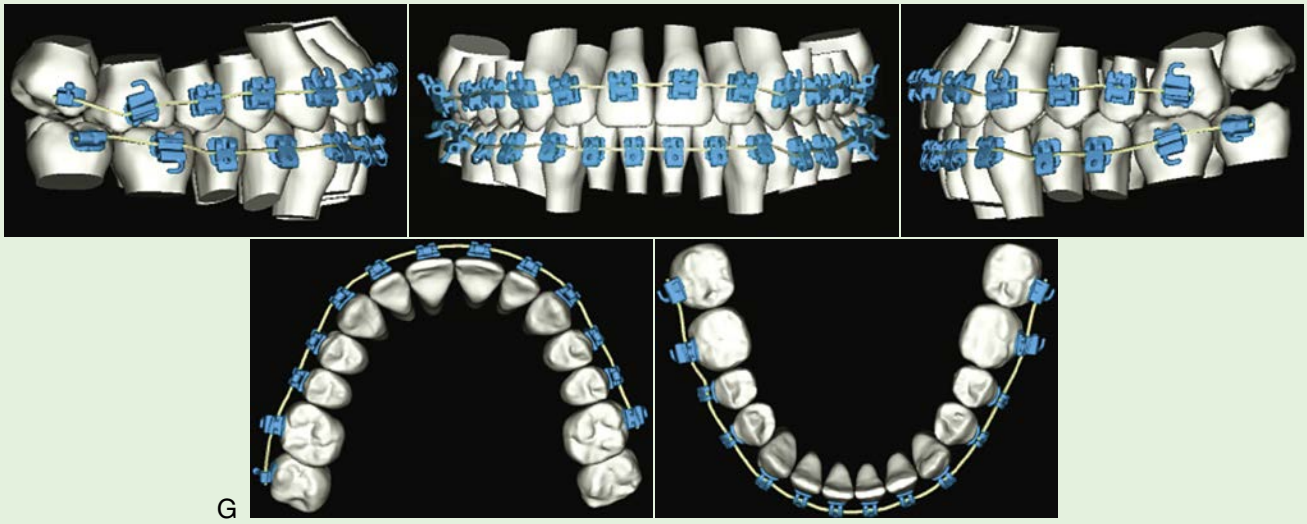
Continued

CASE STUDY 32-1—cont'd



F

Therapeutic model



G

Setup

H

Final x-rays

FIGURE 32-45, cont'd

CASE STUDY 32-1—cont'd

A

Initial

B

Therapeutic

FIGURE 32-46 A–F, Mutilated dentition, Damon bracket system, 0.014-inch Damon copper-nickel-titanium (CuNiTi), followed by 0.16- × 0.22-inch CuNiTi A_F 35°C lower wire and upper Sure-Smile prescriptive archwire 17- × 25-inch CuNiTi A_F 35° C followed by 19- × 25-inch CuNiTi A_F 35° C.

Continued

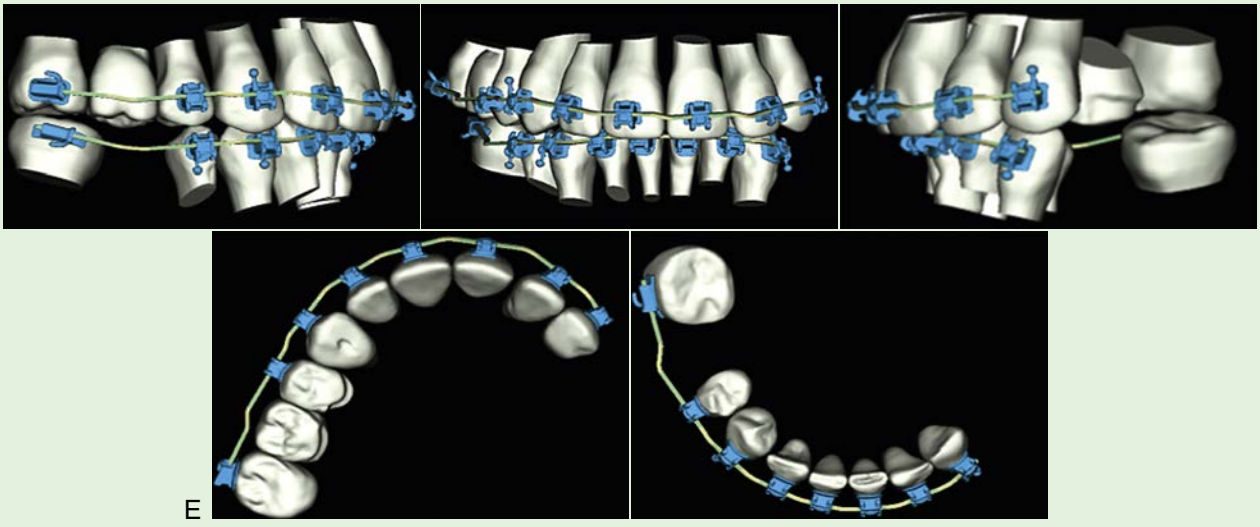
CASE STUDY 32-1—cont'd

C Final

D Initial model

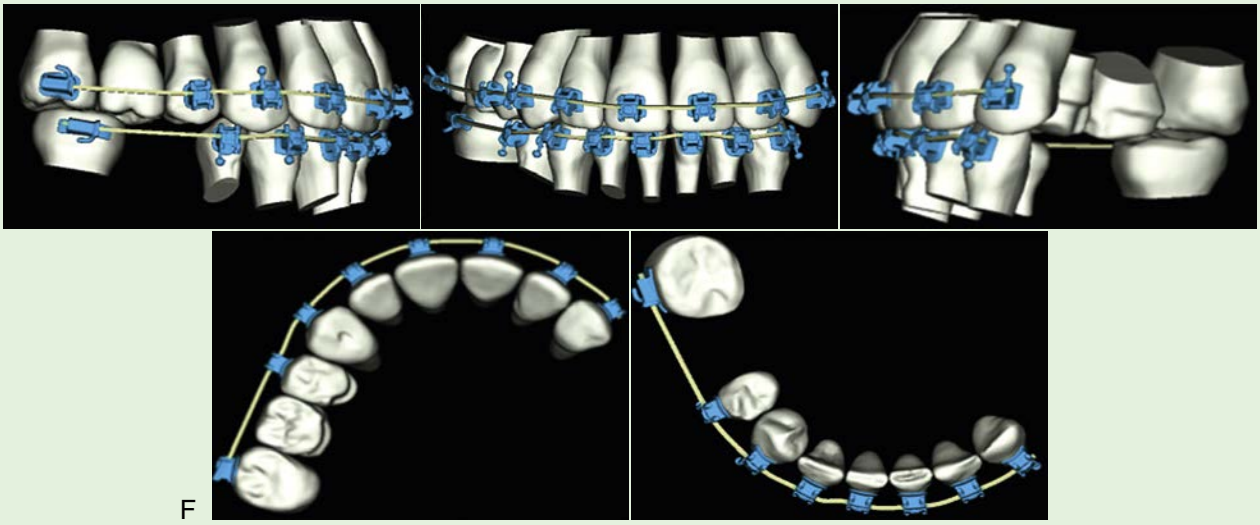
FIGURE 32-46, cont'd

CASE STUDY 32-1—cont'd



E

Therapeutic model



F

Setup

FIGURE 32-46, cont'd

REFERENCES

1. Mah J, Sachdeva R. Computer-assisted orthodontic treatment: the SureSmile process. *Am J Orthod Dentofacial Orthop*. 2001;120(1): 85–87.
2. Sachdeva R. Sure-Smile: technology-driven solution for orthodontics. *Tex Dent J*. 2002;119(7):608–615.
3. Sachdeva R, Frugé JF, Frugé AM, et al. SureSmile: a report of clinical findings. *J Clin Orthod*. 2005;39(5):297–314.
4. Sachdeva RC. SureSmile's promise: digital care solutions for the orthodontic industry. *Orthod CYBER Journal*. 2001.
5. JCO INTERVIEWS Dr. Rohit CL. Sachdeva on a total orthodontic care solution enabled by breakthrough technology. *J Clin Orthod*. 2000;34(4):223–232.
6. Scholz R, Sachdeva RC. Interview with an innovator: SureSmile Chief Clinical Officer Rohit C. L. Sachdeva. *Am J Orthod Dentofacial Orthop*. 2010;138:231–238.
7. Sachdeva R, Feinberg MP. Reframing clinical patient management with SureSmile technology. *PSCO NewsWire*. 2009;2(1). Available at http://www.pcsortho.org/newswire/March-09.cfm.
8. Hodge T, Dhopatkar A, Rock W, Spary D. A randomized clinical trial comparing the accuracy of direct versus indirect bracket placement. *J Orthod*. 2004;31(2):132–137.
9. Balut N, Klapper L, Sandrik J, Bowman D. Variations in bracket placement in the preadjusted orthodontic appliance. *Am J Orthod Dentofacial Orthop*. 1992;102(1):62–67.
10. Cash A, Good S, Curtis R, McDonald F. An evaluation of slot size in orthodontic brackets—are standards as expected? *Angle Orthod*. 2004;74(4):450–453.
11. Matasa CG. Preadjusted appliances: one shoe fits all? (II) Can brackets alone make them … straight? *Orthod Materials Insider*. 1994;7(3).
12. Scholz R, Sarver D. Interview with an Insignia doctor: David M. Sarver. *Am J Orthod Dentofacial Orthop*. 2009;136(6):853–856.
13. Kanavakis G, Spinos P, Polychronopoulou A, et al. Orthodontic journals with impact factors in perspective: trends in the types of articles and authorship characteristics. *Am J Orthod Dentofacial Orthop*. 2006;130(4):516–522.
14. Saxe A, Louie L, Mah J. Efficiency and effectiveness of SureSmile. *World J Orthod*. 2010;11(1):16–22.
15. Alford TJ, Roberts WE, Hartsfield JK, et al. Clinical outcomes for patients finished with SureSmile™ method compared with conventional fixed orthodontic therapy. *Angle Orthod*. 2011;81(3):383–388.
16. Sachdeva R, Aranha SL, Egan ME, et al. Treatment time: SureSmile vs conventional. *Orthodontics (Chic.)*. 2012;13:72–85.
17. Patel N. *Linear Analysis of Anterior Root Resorption Utilizing Cone-Beam CT: SureSmile Versus Conventional Edgewise*. M.S. Thesis, University of Oklahoma; 2010.

33

Stability, Retention, and Relapse

Donald R. Joondeph, Greg Huang, and Robert Little

OUTLINE

History of Retention, 981
Occlusal School, 981
Apical Base School, 981
Mandibular Incisor School, 982
Musculature School, 982
Retention Theories Proposed in the Literature, 982
Theory 1: Teeth That Have Been Moved Tend to Return to Their Former Position, 982
Theory 2: Elimination of the Cause of the Malocclusion Will Prevent Recurrence, 982
Theory 3: Malocclusion Should Be Overcorrected, 982
Theory 4: Proper Occlusion Is an Important Factor in Holding Teeth in Their Corrected Positions, 982
Theory 5: Bone and Adjacent Tissues Must Be Allowed to Reorganize around Newly Positioned Teeth, 982
Theory 6: If the Lower Incisors Are Placed over Basal Bone, They Are More Likely to Remain in Good Alignment, 983
Theory 7: Corrections Carried out during Periods of Growth Are Less Likely to Relapse, 983
Theory 8: Arch Form, Particularly the Mandibular Arch, Cannot Be Permanently Altered with Appliance Therapy, 983
Other Factors Related to Retention, 983
Tooth-Size Discrepancies, 983
Interproximal Reduction, 983
Growth Factors and Posttreatment Change, 984
Third Molars, 984
Duration of Retention, 984
Occlusal Adjustment, 984
UW Postretention Registry: Lessons Learned, 984
Clinical Applications of Retention, 989
Retention Appliances, 991
Positioner in Retention Planning, 994
Duration of Retention, 994
Recovery after Relapse, 994
Summary, 995

A working definition of retention in relation to orthodontics might be stated as follows: the holding of teeth in optimal aesthetic and functional positions.

The requirements for retention are often decided at the time of diagnosis and treatment planning. The correct problem list or diagnosis, a logical treatment plan, and the timing of treatment must be directed toward achieving favorable aesthetics, ideal function, and, as much as possible, the permanent maintenance of these goals. Incorrect diagnosis or treatment, on the other hand, can complicate the requirements for retention. For instance, gross expansion of the dental arches, severe changes in arch form, incomplete resolution of anteroposterior malrelationships, and incomplete correction of dental rotations may require additional retentive measures.

HISTORY OF RETENTION

For many years, clinicians did not agree about the need for retention. Hellman[1] said, "We are in almost complete ignorance of the specific factors causing relapses." Different schools of thought have existed over time, and present-day concepts generally combine several of the following historic concepts regarding retention.

Occlusal School

Kingsley[2] stated, "The occlusion of the teeth is the most potent factor in determining the stability in a new position." Many early investigators[3–18] agreed that proper occlusion was of primary importance in retention.

Apical Base School

In the middle 1920s, a second school of thought formed secondary to the writings of Axel Lundstrom,[19] who suggested that the apical base was one of the most important factors in the correction of malocclusion and the maintenance of a corrected occlusion. McCauley[20] also suggested that intercanine width and intermolar widths should be maintained as originally presented to minimize retention problems. Strang and

Thompson[21] further confirmed and substantiated this concept. Finally, Nance[22] also noted that, "arch length may be permanently increased only to a limited extent."

Mandibular Incisor School

Grieve[23] and Tweed[24,25] suggested that the mandibular incisors must be placed and kept upright over basal bone to maximize their stability.

Musculature School

Rogers[26] introduced a consideration of the necessity of establishing proper muscular balance. Others corroborated his thoughts. Over time, orthodontists have come to realize that retention is not separate from active orthodontic treatment; rather, retention is part of treatment itself and must be included in treatment planning. Stability has become a primary objective in orthodontic treatment; without it, ideal function, optimal aesthetics, or both may be lost.[27] Care must exercised to establish a proper occlusion within the bounds of normal muscle balance and with careful regard given to the apical base, as well as to the relationship of the maxillary and mandibular bases to one another.

RETENTION THEORIES PROPOSED IN THE LITERATURE

The orthodontic literature describes many theories regarding retention. Some are presented here, along with supporting literature.

Theory 1: Teeth That Have Been Moved Tend to Return to Their Former Position[27-29]

Investigators do not agree on the reason for this tendency. Suggested influences include morphogenetic pattern of musculature,[30] apical base,[31–33] transseptal fibers,[34] and bone morphology. Regardless of the reason, most clinicians agree that teeth should be held in their corrected positions for some time after changes are made in their position. Only a limited number of individuals have suggested that retention is routinely not necessary.[12,35]

Theory 2: Elimination of the Cause of the Malocclusion Will Prevent Recurrence[22]

Until more is known about the causative factors that are related to particular types of malocclusions, little can be done regarding their elimination. When obvious habits such as thumb or finger sucking or lip biting contribute to a malocclusion, recognizing them as etiologic factors is significantly easier. To improve retention prognosis, however, identifying and preventing, as much as possible, the causative factors that contribute to the malocclusion are important.

One of the more insidious habits that operate against successful retention is tongue posture and function, which results in anterior and, occasionally, lateral open bites. The mere fact that the patient has been directed along a course of tongue or speech therapy and is able to meet all the exercise requirements of the therapist on conscious command does not guarantee correction of this subconscious behavior pattern.[36] In fact, several investigators have suggested that open bite malocclusion may be attributable to mouth breathing, resulting from nasopharynx obstruction that could result from anatomic blockage, allergic disease,[28] or adenoid hyperplasia.[37] Mouth breathing may also be habitual, which then necessitates a compensatory low anterior tongue posture and rotated mandibular position for the individual to be able to breathe. Dentofacial changes associated with this functional alteration appear to become more severe with age[28] and are a challenge to diagnose accurately and to treat and retain successfully. This retention issue has been discussed by Lopez-Gavito and colleagues,[31] who conducted a long-term evaluation of patients with initial open bite malocclusions. Despite attempts to control posterior maxillary vertical changes during active treatment and retention, the authors of this study found that 35% of the patients with an initial open bite had an open bite of 3 mm or more 10 years out of retention.

Theory 3: Malocclusion Should Be Overcorrected

A common practice of many orthodontists is to overcorrect a Class II malocclusion into an edge-to-edge incisor relationship. One must be aware, however, that an overcorrection such as this could be the result of muscle *memory*, rather than the achievement of a true skeletal or dental correction. The use of prolonged Class II elastics, for instance, may produce a forward postural displacement of the mandible, which is difficult to detect until elastics have been discontinued long enough to allow normal mandibular posture to return, emphasizing the need to check patients in the centric relation position.

Overcorrection of a deep overbite is an accepted procedure by many clinicians. Satisfactory maintenance of overbite correction depends on the establishment of satisfactory correction, or overcorrection, during active treatment, as well as retention appliances, such as an anterior bite plate, designed to prevent vertical relapse.

One of the more common types of relapses is the tendency for a previously rotated tooth to attempt movement toward its former position. Overrotation is not often practical, and, furthermore, little evidence exists that indicates its effectiveness in preventing a return to the initial position. Preventing the anterior teeth from erupting rotated in the first place is often possible by providing space for unimpeded eruption through the use of orthodontic expansion appliances or by early removal of primary teeth. As Reitan has stated, "The principle is that if the tooth had never been rotated initially, it would have less tendency to re-rotate at a later time."[38] Additionally, increased stability of corrected rotations through minor surgical procedures has also been suggested by Allen,[39] Boese,[40] Brain,[41] and finally, Edwards,[42] who recommended transseptal fiberotomy.

Theory 4: Proper Occlusion Is an Important Factor in Holding Teeth in Their Corrected Positions

Fidler and colleages[43] studied the long-term stability of well-finished Class II cases a mean of 14 years after the discontinuation of all retention appliances. The authors concluded that successful achievement of a good occlusion after active orthodontic treatment was very stable in the long term. In contrast, Norwick[44] evaluated the long-term stability of cases in which the final orthodontic result was judged to be unsatisfactory. Norwick found a statistically significant relapse in overjet and posterior dental relationships a mean of 14 years 8 months out of retention. When compared with successfully treated patients, the unsuccessful sample exhibited changes in overjet and posterior dental relationships that appear to be the result of inadequate occlusal correction at the end of active treatment.

Theory 5: Bone and Adjacent Tissues Must Be Allowed to Reorganize around Newly Positioned Teeth

In general, the orthodontist should use some type of fixed or removable retention appliance to maintain the achieved correction. Historic evidence shows that the alveolar bone and

periodontal ligament around the teeth that have been moved are altered, and it takes considerable time before complete reorganization can occur. Some authors advocate fixed wire retainers (1) as *permanent* retention, whereas others suggest that retainers should have no *positive fixation* to allow for the natural functioning of teeth.[37,45] Oppenhein[46] argued that repair of tissues around the teeth more rapidly occurs if no fixed retention appliance is used.

Whether stability increases with prolonged retention is also difficult to determine as documentation and a control of variables, such as cooperation, length of retention time, growth, and appliance design, make reaching valid conclusions difficult.

Present-day orthodontic concepts consider tooth position to result from a state of equilibrium between the muscular environment and the influence of the transseptal and periodontal fibers surrounding the teeth.[47]

Theory 6: If the Lower Incisors Are Placed over Basal Bone, They Are More Likely to Remain in Good Alignment

According to this theory, direct attention should be paid to the proper angulation and placement of the mandibular incisors.[23,24,48–51] The difficulty in evaluating this theory is determining whether incisors have been placed upright over basal bone. The term *upright* may be defined as perpendicular to the mandibular plane, plus or minus five degrees, or some other specified angulation, to the occlusal or Frankfort horizontal planes. Specifying exactly where basal bone begins or ends with a satisfactory method of quantification is also difficult.[33,52–54]

In growing patients, the mandibular anterior segment may exhibit a physiologic migration or eruption pattern in relation to the mandibular body in a posterior direction that is apart from the intended orthodontic tooth movement.[55–58] If the mandibular anterior teeth are lingually moved during orthodontic treatment, then one can hypothesize that this movement may be in harmony with the normal eruption of these teeth and that retentive care may be minimized.[59]

Theory 7: Corrections Carried out during Periods of Growth Are Less Likely to Relapse

Orthodontic treatment therefore should be instituted, if possible, when the patient is young and actively growing. Little direct evidence exists to substantiate this theory, but it is plausible. If orthodontists are in any way able to influence the growth and development of the maxilla or mandible, then certainly one may logically presume that this growth can be influenced only while the patient is growing.[60–63] In 1924, Matthew Federspiel[64] said, "I am free to confess that we find it almost impossible to treat a true type of disto-occlusion without extraction after the developmental period and keep it in position." When treatment depends on inhibition or an alteration of the growth direction, as anticipated with headgear or functional appliances, both fixed and removable treatment must be sufficiently initiated early to utilize active growth.

Early diagnosis and treatment planning appear to afford several advantages with regard to long-term stability. The institution of early intervention can prevent progressive, irreversible tissue or bony changes,[65] maximize the use of growth and development with concomitant tooth eruption, allow interception of the malocclusion before excessive dental and morphologic compensations occur (which may become more difficult to resolve) and allow correction of skeletal malrelationships while sutures are morphologically immature and more amenable to alteration.

Theory 8: Arch Form, Particularly the Mandibular Arch, Cannot Be Permanently Altered with Appliance Therapy

Treatment should be directed toward maintaining the arch form presented by the malocclusion as much as possible.[21] Nance[22] reported that attempts to alter the mandibular arch form were met with failure and recommended maintenance of the mandibular arch form as a treatment objective. In 1944, McCauley[20] stated, "Since these two mandibular dimensions, molar width and canine width, are of such an uncompromising nature, one might establish them as fixed quantities and build the arches around them." Strang[21] reiterated in 1946, "I am firmly convinced that the axiom of the mandibular canine width may be stated as follows: The width as measured across from one canine to the other in the mandibular denture is an accurate index to the muscular balance inherent to the individual and dictates the limits of the denture expansion in this area of treatment."

OTHER FACTORS RELATED TO RETENTION

Tooth-Size Discrepancies

A discrepancy in tooth size is an often-overlooked problem in retention. Ballard[66] reported that 90% of the casts of the 500 patients he examined had tooth-size discrepancies. When maxillary teeth are too large in relation to mandibular anterior teeth, the maxillary teeth must be in one of the following positions to compensate: (1) deeper overbite, (2) greater overjet, (3) combination of deeper overbite and excess overjet, (4) crowded maxillary anterior segment, or (5) tendency toward Class III posterior occlusion.

If the mandibular anterior teeth are too large in relation to the maxillary anterior teeth, then compensations may include (1) end-to-end incisor relationship, (2) space in the maxillary anterior segment, which may be closed through restoration, (3) incomplete resolution of mandibular anterior crowding, or (4) treatment of the maxillary posterior teeth to a mesial position, relative to the mandibular buccal segments.

Estimating and evaluating these compensations is possible by fabricating a diagnostic setup or by using a mathematical formula to determine the degree and location of a tooth-size discrepancy between the maxillary and mandibular arches.[67] Three-dimensional imaging allows a very precise means of direct tooth-size measurement and resultant planning for inherent discrepancies.

Interproximal Reduction

Measurements from dental study models or digital images can determine tooth-size ratios and the degree and location of a tooth-size discrepancy. When anterior tooth-size discrepancies exceed 2.5 mm, a trial setup is usually indicated to evaluate the anticipated final occlusion. If the teeth can only be satisfactorily occluded with interproximal reduction (IPR) in the setup, then the clinician should include IPR of these same teeth in the mouth.

IPR can usually be accomplished with more control and ease during the finishing stages of therapy. Approximately 2.5 mm of enamel can be removed from the maxillary or mandibular six anterior teeth (0.25 mm from each surface). To determine the surface best reduced, the size and shape of the teeth are considered in an attempt to correct any intraarch asymmetries and to minimize gingival interproximal *black triangles*. Enamel thickness should also be considered through the use of accurate periapical radiographs. Removing excessive enamel from the contact point to the extent that the crown width becomes narrower than the cervical dimension should be avoided, since doing so will prevent closing of the spaces in a normal contact relationship, and incisor root proximity could occur.

Growth Factors and Posttreatment Change

Growth can be an aid in the correction of many types of orthodontic problems, but it may also contribute to changes in treated orthodontic cases. For instance, although orthodontists take advantage of growth when treating patients through growth modification appliances such as headgear[68] or fixed or removable functional appliances, alterations of the postorthodontic occlusion can occur, not through tooth change but through disparate maxillary and mandibular growth. Progressive asymmetric growth after treatment may also be demonstrated with concomitant occlusal changes, despite successfully maintaining the teeth in their posttreatment positions.

With the possible exception of those who have an open-bite malocclusion, patients who have a Frankfort mandibular angle (FMA) within the normal range can generally be expected to show a reduction in this angle with continued growth. If, during orthodontic treatment, the FMA increases, then it may be expected to return to its former relationship, or less, if the patient continues to grow. If no further growth takes place, then the return to the original FMA will likely not occur. If, however, further growth occurs and the maxillary and mandibular teeth are retained in a position of minimal overbite, then the subsequent increase in posterior facial height and a leveling of the FMA may occur without an accompanying increase or relapse in the deep overbite.

Increases in the angulation of the occlusal plane to facial planes during orthodontic treatment are undesirable; the general tendency is for these angles to reverse or decrease after therapy. Class II mechanics, for instance, can rotate the occlusal plane in a clockwise direction by extruding mandibular buccal segments, proclining and intruding lower incisors and extruding and uprighting the maxillary incisors. If no further growth is forthcoming after the occlusal plane has been rotated in this manner, then the subsequent recovery change of the occlusal plane angle may mean the return of the original Class II buccal relationship also with a return of excess overjet as the dental changes reverse.[69]

The implications of mandibular growth and latent mandibular growth in the true Class III malocclusion are very well known, and, accordingly, orthodontists do not usually attempt to perform definitive treatment until all growth has ceased.

Removable functional appliances may play an important role during retention to assist in maintaining the correction of skeletal components. These types of removable appliances can be worn during sleep to minimize changes in the maxillomandibular relationship that may take place, secondary to unharmonious growth. Functional appliances may also be an effective way to encourage differential eruption and tooth movement in compensation for skeletal changes resulting from posttreatment growth.

Third Molars

Third molars are often implicated as causing crowding of the mandibular anterior teeth in the late teens and early 20s. Some authors have attributed the presence of third molars to long-term mandibular dental instability,[70–73] but others have published data suggesting that third molars play little, if any, role in long-term mandibular arch changes.[74–77]

Additionally, two systematic reviews address this issue. In the earlier review by Mettes,[78] one randomized trial[79] was identified for inclusion in the review that indicated third molars did not have an impact on lower incisor crowding over a 5-year period. In the more recent review by Zawawi,[80] 12 studies were identified for inclusion, although only 3 were prospective trials. The authors of this review indicated that most of the included studies had a high risk of bias, and they reported that most of the studies did not indicate a relationship between third molars and lower incisor crowding. However, they also stressed that the quality of the evidence on this topic prevented them from drawing any definitive conclusions.

Implicating third molars as a cause of crowding is tempting. Some postulate that mesial drift plus third-molar eruption may be cofactors that cause the onset of incisor crowding. However, many patients who have had third molars removed experience incisor crowding that progressively worsens.[74] Similarly, many individuals who are congenitally missing third molars will develop incisor crowding over time.[74]

Duration of Retention

Does prolonged retention provide greater stability? A prior study indicates no consensus has been reached on how long to use retention appliances.[81] Also, to date, no satisfactory comparisons have been made of patients with comparable malocclusions similarly treated but having different retention protocols and durations. Most clinicians would consider "longer is better" but recognize individual differences among patients. Long-term maturational changes are often confused with *relapse* and, no matter what the label, may be attenuated with semipermanent retainer wear. As a clinician, determining exactly how long (hours, days, months, years) to use retainers after active treatment and with what frequency is difficult. To complicate the issue, accurately assessing the actual time of retainer wear by the patient is often difficult. In the absence of conclusive evidence on the appropriate duration of orthodontic retention, the concept of semipermanent retention may be the safest bet for a patient, as long as retainers continue to be monitored on a regular basis by the orthodontist and/or general practitioner.

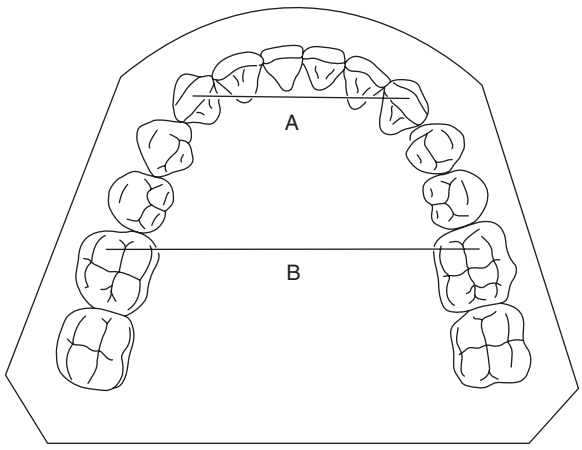
Occlusal Adjustment

Treatment objectives should include the basic principles of gnathology such as canine disclusion and incisal guidance whenever possible. Stability may be improved with occlusal adjustment, concomitant with or after orthodontic treatment, that includes the removal of interferences in centric relation, as well as cross-tooth and cross-arch deflective interferences not eliminated during orthodontic finishing. Attempts to achieve final functional balance immediately after the appliances are removed may not be desirable. The dental changes or settling that naturally occurs after appliance removal should be taken into consideration before undertaking extensive equilibration procedures. Indeed, any occlusal adjustment in the first 6 months after appliance removal should be solely performed to eliminate gross interferences. Restorations can be recontoured, cusps and incisal edges adjusted, and teeth aesthetically recontoured where excessive wear or lack of function has produced undesirable dental morphologic outcomes. Long-term considerations include periodic observation of posttreatment changes with possible detailed occlusal adjustment, as needed, to achieve desirable and functionally beneficial results.

UW POSTRETENTION REGISTRY: LESSONS LEARNED

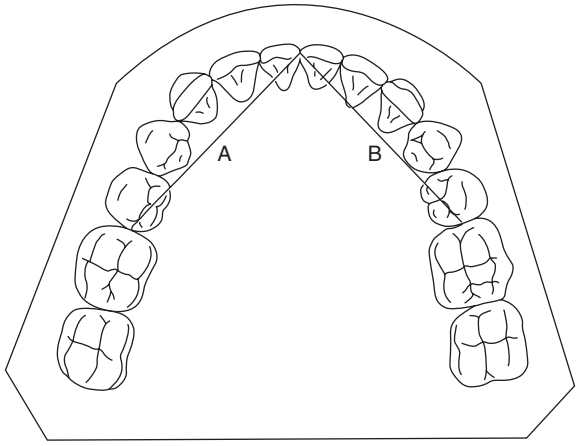
The idea for the postretention registry at the University of Washington (UW) should be credited to Dr. Richard Riedel (Fig. 33-1), former chairman of the Department of Orthodontics. As the story goes, Dr. Riedel wished to determine how his

FIGURE 33-1 Dr. Richard Riedel, founder of the postretention registry at the University of Washington, Seattle.



A = intercanine width
B = intermolar width

FIGURE 33-2 Arch width dimensions.



A + B = arch length

FIGURE 33-3 Arch length measurements.

American Board of Orthodontics cases held up many years after treatment. Upon collecting these records, he learned so much that he decided to increase the target population to all of his former patients. Later, Dr. Little joined Dr. Riedel in this effort, and they expanded the search to include cases from the graduate clinic, faculty practice, alumni, and other interested orthodontists. The result of this collaboration is a collection of approximately 900 cases that have follow-up records at least 10 years after the end of retention, that is, 10 years after all retainers have been removed or discontinued. This sample was not randomly collected nor was it consecutively chosen. Thus the potential for some selection bias is present. If these data could be collected again, then more emphasis would be given to the parameters for sample selection. However, Dr. Riedel tried to include all subjects who were willing to undergo the collection of long-term records, regardless of their long-term outcome. If there is bias, then it might be toward over-representation of patients who exhibited relapse; those who were stable may have had less reason to return to their orthodontists long-term. Nevertheless, the size of the sample is impressive, and given the known limitations of the observational data, this registry has certainly assisted orthodontists in understanding the changes that occur in alignment and arch dimensions many years after treatment.

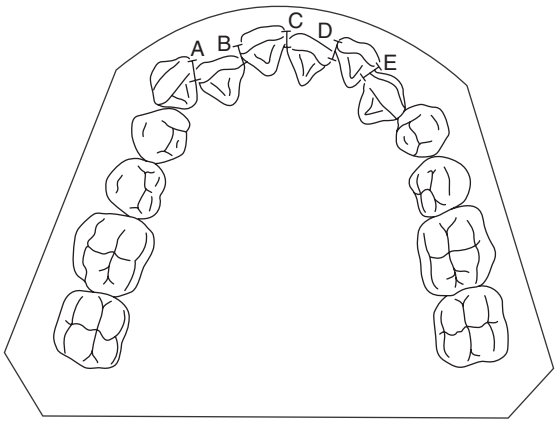
As stated by Edward Angle[82] himself, "The period of retention of teeth, after they have been moved into normal occlusion, is one of the most important in treatment, and so complicated and persistent are the delicate forces that tend to derangement of the established occlusion as to necessitate the most thoughtful consideration of the problems involved, and a degree of skill in overcoming them which much experience alone can develop, even among those with talent for the work." That statement was written over 100 years ago, and the orthodontic professionals are still challenged by these same forces. As mentioned earlier, many theories have been proposed regarding retention and stability, such as occlusion, muscle balance, incisor proportions, the apical base width, and the placement of incisors over basal bone. Although all of these theories are interesting, the reality is that, today, orthodontists are still debating how to prevent teeth from moving after treatment. What lessons can be learned about relapse emerge from the orthodontic postretention UW registry?

1. **Postretention changes are unpredictable.** Upon meeting a new patient, how often have we wished for a "crystal ball" to predict influences such as growth, treatment response, and cooperation? Having this knowledge in advance would certainly allow us to customize our treatment. The same could be said for retention—if only we knew in advance whose teeth would have a high-relapse potential and whose teeth would stay in their corrected positions. This knowledge would certainly allow us to tailor the types of retainers we prescribe, as well as the wear regimen. However, as helpful as these predictors could be, the hundreds of cases in the registry seem to indicate that postretention changes are unpredictable. Based upon intercanine and intermolar distances (Fig. 33-2), as well as arch length (Fig. 33-3) and the Irregularity Index scores (Fig. 33-4), predicting the long-term outcome based on the initial or end-of-treatment condition was difficult[83] (Fig. 33-5). Therefore long-term retention seems to be the only predictable way to ensure a stable result. Even extraction cases, in which incisors are placed over basal bone, displayed only acceptable alignment in approximately one third of the treated cases after 10 years,[83] and by 20 years, this rate was down to 10%.[84] Although many

cases relapsed toward the original pattern, this movement was not always the case; we were sometimes surprised by teeth moving opposite of the original positions.

Of course, problems with long-term retention can occur using fixed retainers. Oral hygiene can be difficult, and the possibility for caries under or around the lingual retainers certainly exists. Most orthodontists believe periodically checking these retainers is their obligation to ensure that they do not become loose. When they do loosen, relapse can sometimes occur alarmingly quickly, perhaps faster than the patient can make an appointment to have the retainer repaired. On rare occasions, twisted wires can remain bonded to the teeth but, through the relaxation or distortion of the wire, express torque can cause roots or crowns to move considerable distances.

Given these limitations, bonded lower retainers are still a more reliable choice for long-term retention than removable retainers, which are notorious for being lost, thrown away, broken, and inadequately used. A study published in 2010 reported that 40% of orthodontists are routinely prescribing lower fixed retention, whereas 28% prescribe Hawley retainers (DDS Lab, Tampa, FL) and 18% recommend clear thermoplastic retainers.[85] In the maxillary arch, Hawley retainers were prescribed by 58% of the practitioners, and clear thermoplastic retainers were recommended by 30%.



A + B + C + D + E = irregularity index

FIGURE 33-4 Calculation of the Irregular Index score.

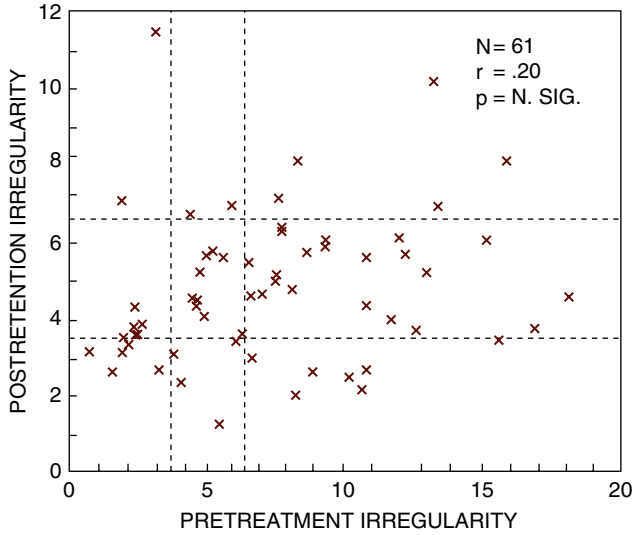


FIGURE 33-5 Relationship between pretreatment irregularity and postretention irregularity.

2. Arch length and width constrict over time, especially intercanine width. Trends were noted in the postretention cases, and a particularly strong one was constriction in arch dimensions, especially intercanine width (Fig. 33-6). This trend may be related to the popular notion in dentistry known as the *mesial drift*, in which posterior teeth have a tendency to mesially migrate over time. Since the basal bone houses the roots and the lips and tongue provide a corridor for the crowns to exist in a state of equilibrium, mesial drift would eventually result in the crowding of the front teeth, especially the lower incisors. Thus to the extent possible, attempting to maintain or decrease intercanine width seems prudent. In patients with mandibular incisor crowding, aligning the incisors will always result in an increase in intercanine width and/or protrusion unless IPR or incisor extraction is performed. Even extraction of premolars cannot prevent an increase in intercanine width unless the above conditions are met, as the canines are moving into a wider area of the arch to allow alignment of incisors (see Fig. 33-6). Thus, in reality, alignment without an increase in intercanine distance is a difficult task to accomplish, and we must be prepared to deal with the arch constriction that almost inevitably follows.[83] Further complicating our understanding, not all cases show intercanine constriction after retention, a few actually show expansion.

Using the UW sample, investigators assessed the stability of cases undergoing lower incisor extraction. Interestingly, these cases seemed to exhibit better incisor irregularity scores long-term, compared with other types of extraction and nonextraction samples.[86] One might argue that incisor irregularity scores should be lower with incisor extraction cases, since they have fewer contacts to count. However, the investigators controlled for this factor by calculating an average of the incisor irregularity scores; the lower scores were still significant. As previously mentioned, incisor extraction is one of the few treatment modalities we have that can actually allow a reduction in intercanine width, and perhaps this reduction plays a role in the improved stability.

In recent decades, arch expansion using various techniques has become a popular method to address crowding. In particular, claims have been made that treatment with self-ligating brackets allows bone development and, subsequently, better stability. However, little scientific evidence indicates that these claims are true. Although the long-term cases in the UW retention registry were not treated with self-ligating brackets, their results indicate that patients undergoing arch development may be among the most unstable cases encountered.[87] Whether the arch development was in the transverse and/or in the anterior dimension, these patients' arches tended to relapse significantly when retention was discontinued.

3. Rotations usually relapse toward initial positions. If teeth relapse, then predicting that they will relapse toward their original positions seems logical. One philosophy of treatment to counteract this relapse potential is called 11/10 orthodontics,[88] in which teeth are overcorrected by approximately 10% in rotations or labial and/or lingual movement. The idea is that when the tooth relapses, the 1/10 overcorrection will be lost, leaving the tooth in the correct position. Unfortunately, predicting the amount and direction of relapse has proven to be difficult. Although some teeth, perhaps the majority, relapse toward their original position, approximately 20% of teeth unexpectedly relapse away from their original position,[89] making it very difficult to decide whether overcorrection will

be harmful or detrimental (Fig. 33-7). Some have advocated a supracrestal fiberotomy to assist with preventing relapse, and some research suggests that this surgical technique can be effective.[37,42] For teeth that have undergone a fiberotomy, there is no guarantee that rotational relapse will not occur. The primary point is that the degree of rotational relapse is lessened but not fully eliminated after fiberotomy. Additionally, the cost and thought of undergoing surgery can be unpopular, especially if stability is not guaranteed.

To prevent relapse of lower incisors, lingual bonded retainers are very common. The two styles that are commonly used are either bonded only to the canines or bonded to each anterior tooth. Although not bonding to incisors that were normally positioned at the pretreatment phase has advantages for oral

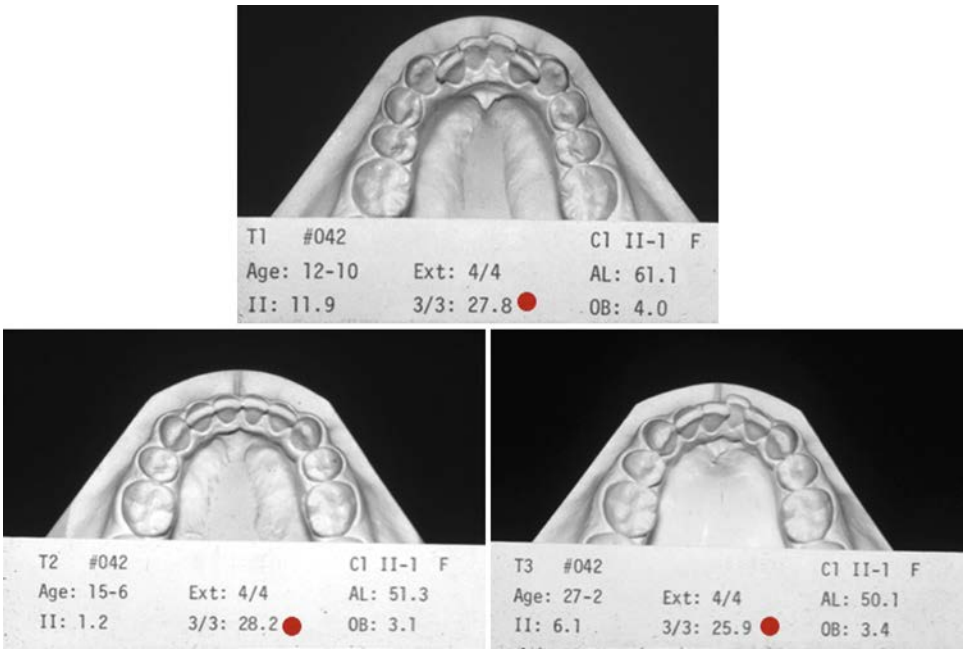


FIGURE 33-6 Photographs of constriction of mandibular intercanine dimension, from ages 12 years 10 months, 15 years 6 months, and 27 years 2 months.

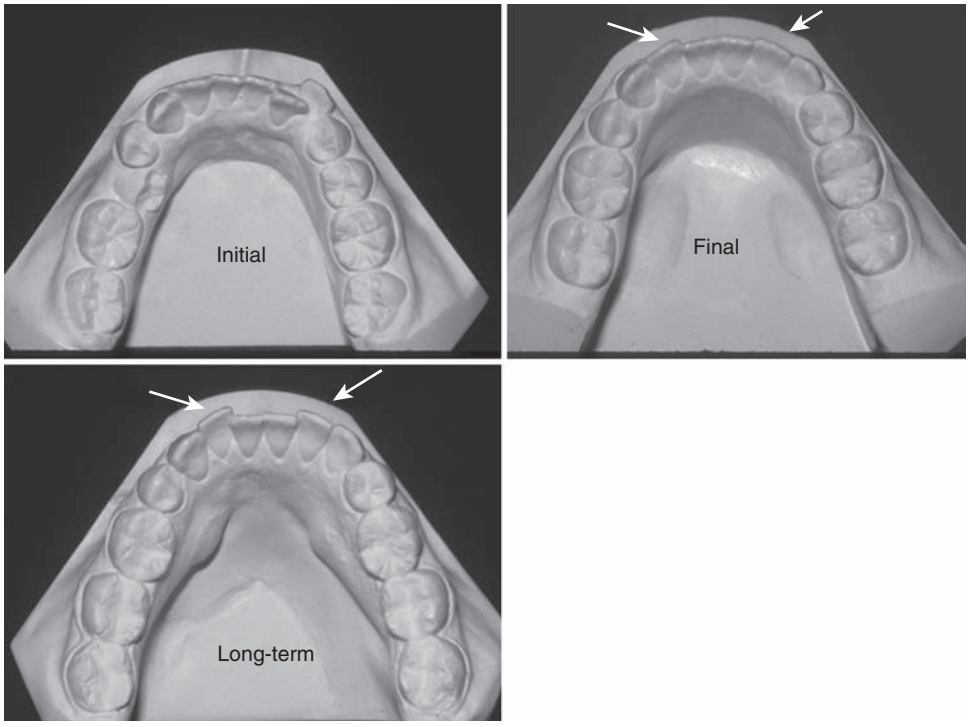


FIGURE 33-7 Photographs of 11/10 orthodontics, as the potential for rotations to move away from the pretreatment condition.

hygiene, any incisors that are not bonded can sometimes move labially or lingually or rotate, even though lip pressure should generally prevent this movement from happening (Fig. 33-8). Retainers bonded to each anterior tooth will prevent posttreatment movement, but hygiene can be more difficult, and more bonds are there to possibly fail.

4. **Some cases will display more favorable results.** A group of patients that seem to fare better than most are the ones who have adequate or excess arch length at pretreatment (Fig. 33-9). It seems intuitive that those with more than enough space are less likely to crowd in the long term. These patients probably have large jaws, small teeth, or a combination of the two, and either factor provides a considerable advantage for the maintenance of long-term alignment. Additionally, patients with adequate or excess arch length have the benefit of maintaining or decreasing their intercanine width during orthodontic treatment, similar to the incisor extraction cohort. One long-term study demonstrated that these patients rarely showed more than mild crowding.[90]

5. **Early treatment may not improve long-term stability.** One may ask whether the management of crowding at a younger age is associated with better long-term stability. Three approaches might be taken. The first strategy is early expansion or arch enlargement. There are some who advocate early expansion to address arch length issues.[91] However, early expansion is not immune from relapse. In fact, patients undergoing mixed dentition enlargement of the lower arch showed the poorest postretention results in our sample, with only 10% having acceptable long-term results.[87]

A second strategy is serial extraction. Unfortunately, this technique does not appear to have a better long-term outcome than premolar extraction in the permanent dentition.[92] Perhaps the reason for this is that patients are candidates for serial extraction because they have insufficient arch length at an early age and are destined for recrowding after treatment and retention, just as are most patients with crowded full dentition.

Orthodontists have hypothesized that those serial extraction cases who demonstrate self-improvement of alignment during the observation period and before full treatment will likely be more stable after retention. Unfortunately, this theory has not proven to be true. Those who favorably react during the observation period may or may not significantly relapse after retention, just as those who do not fare well during the observation period may or may not do well after retention. Serial extraction outcomes seem to be unpredictable in influencing alignment in the long term.

A third approach is utilizing the leeway space. Hayes Nance advocated this strategy, and, in fact, some evidence suggests that utilizing the leeway space may result in superior long-term outcomes.[93] Probably, however, patients who are candidates for preserving leeway space are not as crowded as those who undergo serial extraction; thus the initial crowding should be considered when assessing the long-term outcomes of patients undergoing serial extraction versus the utilization of the leeway space. If the leeway space is equal or greater than anterior crowding, then this strategy appears to give good results. If the leeway space is insufficient to permit anterior alignment without expansion, then this strategy will likely fail to improve retention.

FIGURE 33-8 Photograph of an incisor moving away from a retainer only bonded to the canines. (Brackets were placed immediately before taking this photograph.)

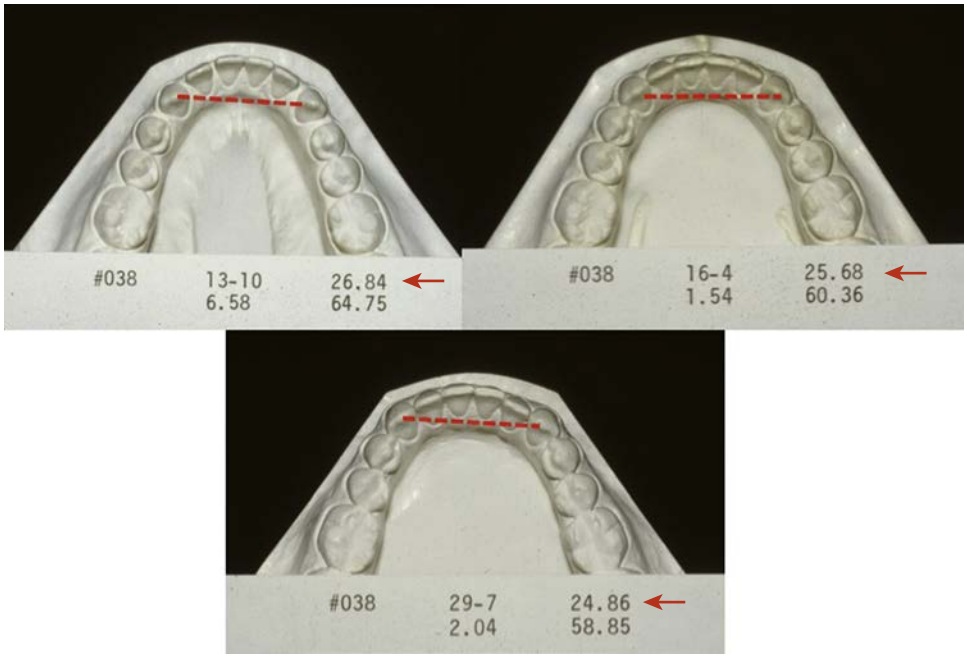


FIGURE 33-9 Photographs of good alignment over a 13-year period in a patient with pretreatment spacing (*red dotted lines*).

6. Early postretention stability is usually a mirage. Many of our patients with fixed retainers displayed a period of relative stability after the removal of retainers. However, this stability proved to be short lived; after a few years, more and more crowding occurred. One might wonder when the relapse ceases. Following cases from 10 and 20 years after retention, apparently, there is no end point. The rate of relapse seems to slow in these cases, but slow, undesirable alignment changes continued to happen.[84] Some patients seemed to have a period of accelerated tooth movement immediately after the fixed retainers were removed. For patients with a strong relapse tendency, many years of fixed retainers may not be enough. Unfortunately, once a bonded retainer is placed, the only way to test stability is by removing the retainer. A graduated lessening of retainer use, which can be done with removable retainers, is not an option.

7. Life-long retention is the best insurance policy. If there is a common theme to our long-term findings, then it is that life-long retention is the best insurance policy that can be provided to a patient. Of course, it is probably not realistic to expect patients to wear their retainers forever. Removable retainers can be lost, worn out, and forgotten, and fixed retainers eventually become loose. Perhaps one strategy is to enlist patients to become partners in the retention process. After all, they will quickly learn if their teeth have a tendency to move or shift, especially if they are using removable retainers. Retention visits should provide an opportunity to discuss retainer use and any observed relapse in a candid and cooperative fashion and to transfer ownership of posttreatment changes to the patient. Fixed retainers usually limit changes in the lower arch; however, when removable retainers are used, patients must be told that they should use their retainers as much as needed to maintain tooth position. Letting patients know that they are in charge and that you will provide assistance can be helpful. Then, when posttreatment movements do occur (and they will for most patients), offering retreatment options and letting patients decide how they would like to proceed are both significantly easier.

8. Extraction of third molars is not necessarily helpful to prevent crowding. One of the most common retention questions that orthodontists hear is whether third molars will cause crowding when they erupt, ruining the results of orthodontic treatment. Third molars often appear during the time that lower incisor relapse occurs, and ascribing the crowding to the eruption of the third molars is only natural. However, the best evidence to date, which comes from two systematic reviews,[78,80] indicates that third molars do not seem to play a large role in lower anterior crowding. In fact, even those with third molars removed or congenitally absent develop lower anterior crowding in the late teenage years and in the early 20s.[74] Coincidently, the typical age of third molar eruption seems to happen when active relapse begins to occur, even for those who have congenitally absent third molars. Nevertheless, this urban myth has proven extremely difficult to dispel, even among general dentists, oral surgeons, and orthodontists.

9. Here is some good news—maxillary teeth fare better. There are some positive findings from the UW retention registry. It appears that relapse occurs for the upper incisors to a lesser degree, and, in addition, the upper incisors do not necessarily mirror the alignment changes observed in the lower arch.[94]

10. Treat to excellence. Quality matters! There were also some interesting findings concerning finishing cases to the highest standards. A study investigating changes in patients with varying degrees of excellent to poor final results showed that an overall regression to the mean long term occurred; that is, cases with excellent finishes tended to deteriorate, whereas cases with poor finishes tended to show some long-term improvement in the scores from the American Board of Orthodontics objective grading system (ABO-OGS).[95] Although this finding might seem counterintuitive, the fact that patients with the best ABO-OGS scores have little room to improve should be given consideration. On the other hand, patients with poor ABO-OGS scores tended to improve in several areas, including marginal ridges, buccolingual inclination, and occlusal contacts. However, one additional important finding was that the cases with the best finishes had the best long-term ABO-OGS scores, indicating that high-quality treatment does, in fact, provide long-term benefits, compared with that of lesser quality treatment.

Feeling a little discouraged after reading the findings of the UW postretention registry is natural. Many of our patients did not hold up well in lower anterior alignment 10 or 20 years after discontinuing retainer wear. Certainly, after all the effort of straightening teeth and correcting occlusions, it would be ideal if teeth would remain in stable positions. However, we are reminded that our bodies undergo changes during our entire lifetimes; perhaps the concept that a perfect occlusion will be stable is unrealistic. In fact, some have proposed using the term *physiologic maturation* or *normal aging* rather than relapse to describe the changes that occur after treatment.

Of course, we should strive for excellence, as the study by Nett and Huang[95] indicates that better occlusions will, on average, exhibit better alignment in the long term. However, above all else, perhaps we need to have candid and honest discussions with our patients before initiating treatment to ensure that they will understand the importance of retainers. Patients should also understand that, despite the best efforts of orthodontists, teeth will and do move, change, and shift over time in somewhat unpredictable ways. Additionally, we also should acknowledge that our understanding of retention is less than perfect. In fact, a Cochrane systematic review published in 2006 concludes that, "There are insufficient research data on which to base our clinical practice on retention at present."[96]

CLINICAL APPLICATIONS OF RETENTION

Retention planning will divided into three categories, depending on the type and extent of retention treatment instituted: (1) limited retention, (2) moderate retention in terms of time and appliance wearing, and (3) permanent or semipermanent retention.

I. Limited retention required.
   A. Corrected crossbites
      1. Anterior: when adequate overbite has been established
      2. Posterior: when axial inclinations of posterior teeth remain reasonable after corrective procedures have been completed. An exception is posterior crossbites treated with either orthopedic or surgical expansion of the mid- palatal suture. In these cases, overcorrection and long-term retention are desirable.
   B. Dentitions that have been treated by serial extraction. The percentage of complete satisfaction secondary to extraction and autonomous eruption alone is typically rather low and depends on the degree of perfection desired by the patient and orthodontist. When extraction of the mandibular second premolars is possible or when they are congenitally absent, the resultant occlusions often are more satisfactory than those in first premolar serial extraction cases.[97]

C. High canine extraction cases.
D. Cases calling for extraction of one or more teeth such as subdivision types of malocclusions, or when the posterior teeth are in Class II relationship and the anterior teeth are treated to a Class I canine position. Occasionally, lingual bonded retainers can be used to prevent extraction spaces from opening.
E. Corrections that have been achieved by retardation of maxillary growth after the patient is through growing.
F. Dentitions in which the maxillary and mandibular teeth have been separated to allow for eruption of teeth previously blocked out. Typical examples are partially impacted mandibular second premolars and maxillary canines.

II. Moderate retention required.
A. Class I nonextraction cases, characterized by protrusion and spacing of maxillary incisors. These cases require retention until normal lip and tongue function has been achieved.
B. Class I or Class II extraction cases probably require that the teeth be held in contact, particularly in the maxillary arch, until lip and tongue function can achieve a satisfactory balance, as in the nonextraction group. Especially true in extraction cases is that the maxillary incisors can be retracted so far as to be unrestrained by lip pressure and to impinge on the space occupied by the tongue before treatment. Use of a maxillary removable retainer until normal functional adaptation has occurred generally is desirable. The time of application of this type of retention can be reduced as the patient adapts to new tooth positions, proceeding from full-time wearing of appliances to nighttime wear only, then perhaps every other night, once or twice a week, and finally wearing retainers at night as needed when they feel tight on insertion. Corrections of adult dentitions probably require longer retentive procedures. A predetermined time schedule for removing retentive appliances probably is undesirable in these cases, and the time of retention depends directly on the patient 's compliance and reactions during retention. Tongue and myofunc- tional therapy may be advantageous in this group of corrected cases.
C. Corrected deep overbites in Class I or Class II malocclusions usually require retention in a vertical plane.[98–100]
1. If anterior teeth were depressed to achieve overbite correction, a bite plate on a maxillary retainer is desirable. To be effective in retaining overbite correction, the bite plate should be worn continuously for perhaps the first several months, including while the patient is eating. In deep overbite cases, overcorrection is usually desirable and occlusal adjustment to an ideal functional relationship is a consideration.
2. If overbite correction was achieved as a result of clockwise mandibular rotation, vertical dimension should be held at least until growth of mandibular ramal height catches up and neuromuscular balance can adapt.

A measure of bite opening can be assessed in the degree of change between the mandibular plane and a cranial base plane, such as the Frankfort or sella-nasion. Positive changes of the mandibular plane angle suggest continued retention until growth compensates or until the orthodontist determines that growth is complete. This seems to suggest that correction of deep overbites is desirable before the completion of facial growth. The work of Matthews in this field concludes that beneficial results may be achieved by beginning overbite correction as early as the primary or mixed dentition.

Severe occlusal plane tipping also may require extended retention protocols and possibly additional maxillary restraint as well. Examination of cephalometric films taken at about 6-month intervals will reveal whether adaptive growth changes have taken place.[101]

D. Early correction of rotated teeth[33,38] to their normal positions.
1. Perhaps before root formation has been completed
2. In the mandibular incisor area, a fixed lingual or removable type of appliance with a labial bow is probably best. In this area the occlusal splint-type or Hawley spring retainer may be useful (Fig. 33-10). More recently, gingi- vectomy procedures have offered hope for increased stability of corrected rotations. Early correction of rotations, transeptal fiberotomy and long-term fixed retention may prove to be the most satisfactory.[39–42,46]

E. Cases involving ectopic eruption of teeth or the presence of supernumerary teeth require varying retention times, usually prolonged, and occasionally a fixed or permanent retentive device, such as bonded lingual retainers.
1. Supernumerary teeth may be encountered in the maxillary anterior area and, on removal of these teeth, the maxillary incisors often erupt slowly and incompletely. When the latter have been brought to a normal level through orthodontic therapy, bonded lingual wires are desirable because these teeth seemingly have a tendency to reintrude when released. Palatally impacted canines, once brought into arch alignment, also may benefit from fixed lingual retention wires bonded to adjacent teeth to prevent reintrusion and palatal relapse (Fig. 33-11).
2. Excessive spacing between maxillary incisors requires prolonged retention after space closure.

F. The corrected Class II, Division 2 malocclusion generally requires extended retention to allow for the adaptation of musculature. These cases, because they are

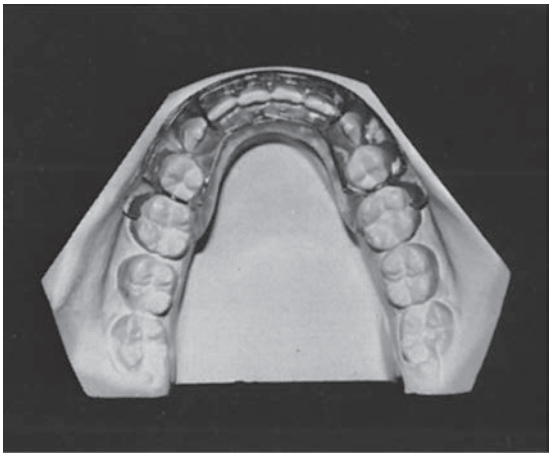


FIGURE 33-10 Mandibular splint retainer. Wire position can be altered to avoid passing through an extraction site or coming in contact with the maxillary dentition.

FIGURE 33-11 Lingual bonded retainers designed to prevent relapse of canines that were initially palatally impacted. A removable retainer is made to fit over and/or around the sectional wires to retain tooth position in other planes of space.

FIGURE 33-12 Lingual bonded 0.014-inch stainless steel wire is applied to hold midline diastema from relapsing. Care should be taken to keep retainer out of occlusion with mandibular incisors.

typically treated without extractions, generally undergo some increase in mandibular intercanine width and present with malalignment of maxillary incisors, which needs to be maintained during retention.

III. Permanent or semipermanent retention required.
   A. Cases in which expansion has been the choice of treatment, particularly in the mandibular arch, may require permanent or semipermanent retention to maintain normal contact alignment. In many instances, patients have aesthetically desirable dentofacial relationships, and extraction of permanent teeth would create aesthetically undesirable results. The orthodontist may be faced with a choice between stability and aesthetics, and often the patient's aesthetic requirements take precedence.
   B. Cases of considerable or generalized spacing may require permanent retention after space closure has been completed.
   C. Instances of severe rotation, particularly in adults, or severe labiolingual malposition may require permanent retention, as provided by lingual bonded retainers.
   D. Spacing between maxillary central incisors in otherwise normal occlusions sometimes requires permanent retention, particularly in adult dentitions (Fig. 33-12).

RETENTION APPLIANCES

The Hawley retainer[9] (DDS Lab, Tampa, FL) is one of the most frequently used retentive appliances available to the clinical orthodontist. The palatal or lingual portion is usually constructed of acrylic and may completely cover the palatal mucosa or can be constructed in a horseshoe shape contacting the lingual surfaces of the teeth and only a limited amount of palatal mucosa. A labial bow of round stainless steel 0.020- to 0.036-inch wire is usually constructed to contact the labial surfaces of

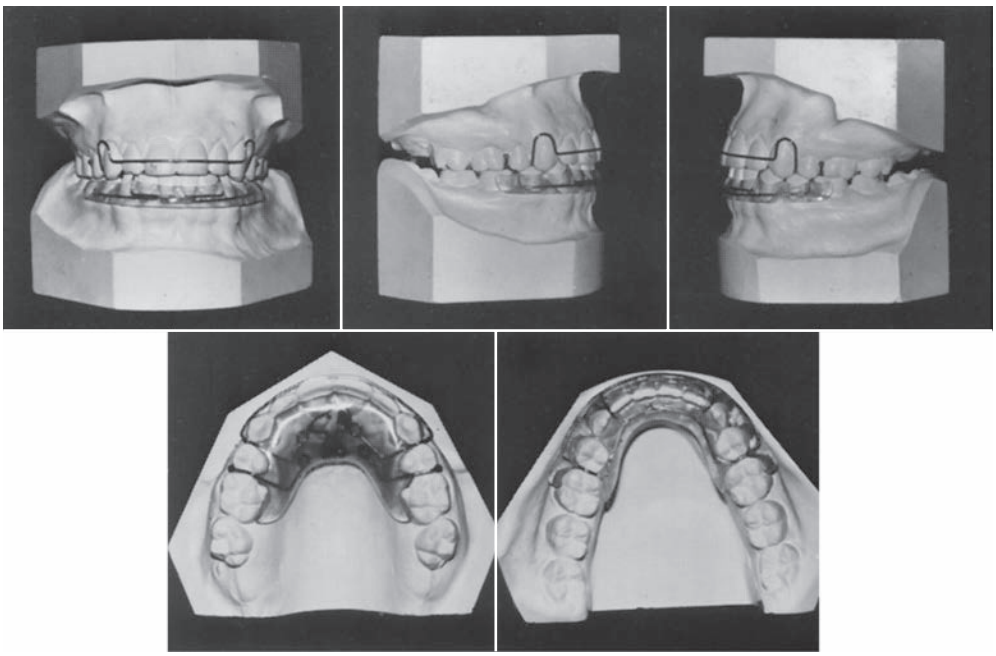


FIGURE 33-13 Typical Hawley and splint retainers. The models should be articulated in occlusion to ensure that the patient is not prematurely contacting on retainer wires or acrylic unless an anterior bite plate is desired. The maxillary labial bow may also cross the occlusion at the canine-lateral incisor embrasure with distal clasping of the canine to avoid a wire in the extraction site that might inadvertently aid reopening of the contact.

four or six maxillary anterior teeth (Fig. 33-13). Constructing the wire to cross incisally through the contact area between the canine and the lateral incisor may minimize occlusal interference in the area of the mandibular canine. If such construction is used, then inclusion of a restraint on the labial aspect of the canines is desirable to prevent their extrusion or labial drift.

The clinician may wish to alter the type and position of retention clasps to avoid traumatic occlusal interferences and to assist in eruption guidance or minor tooth movements, such as erupting second molars (Fig. 33-14). Additionally, the labial bow and retention clasps should not pass through extraction sites, if possible, because that may encourage the tendency for the space to reopen, especially in adult patients.

Circumferential maxillary retainers provide excellent retention with the added benefits of eliminating potential occlusal interferences and aiding in holding the space closed (Fig. 33-15). The clinician may place *keeper* wires between the lateral incisors and canines or flow acrylic on the wire facial to the maxillary anterior teeth to enhance the stability of the labial wire. The clinician also can efficiently adjust labial bow tension if recurved loops are included. Palatal acrylic should be distally extended to retain second molar position, which is particularly important in patients who have undergone changes in transverse dimension during active treatment, such as maxillary surgical or orthopedic sutural expansion.

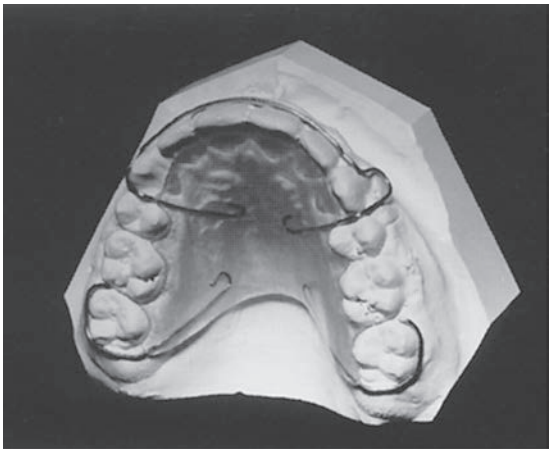


FIGURE 33-14 Maxillary Hawley retainer with C clasps is applied to guide second molar eruption.

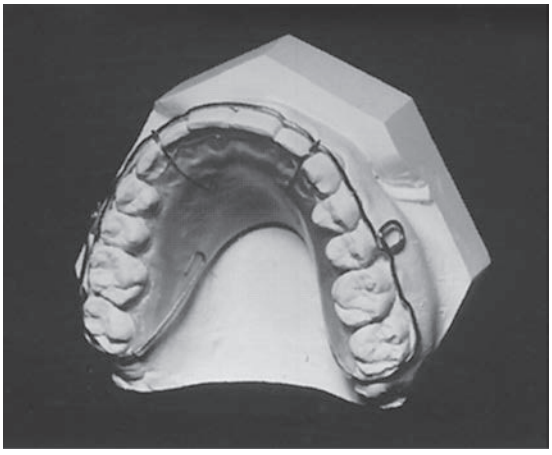


FIGURE 33-15 Maxillary circumferential retainer. Palatal acrylic can be reduced to minimize effects on speech and swallowing patterns. Closing loops allow adjustment of tension.

Pontics may be included in the retainer design to enhance aesthetics and to retain the edentulous area(s) during the transition from fixed appliances to prosthetic replacement of missing teeth (Fig. 33-16). Lingual bonded segmental wires are used to prevent the teeth from drifting into the edentulous areas, and a *flipper* with pontic(s), which is made to fit over the bonded wires, is worn during the day for aesthetics (Fig. 33-17). After the placement of the implants, the retainer can be relieved in the gingival area to allow maintenance of dental aesthetics while avoiding tissue impingement during the osseointegration stage. The clinician may adjust the retainer after completing the prosthetic phase simply by removing the pontic and recontouring the palatal acrylic to fit the final restoration.

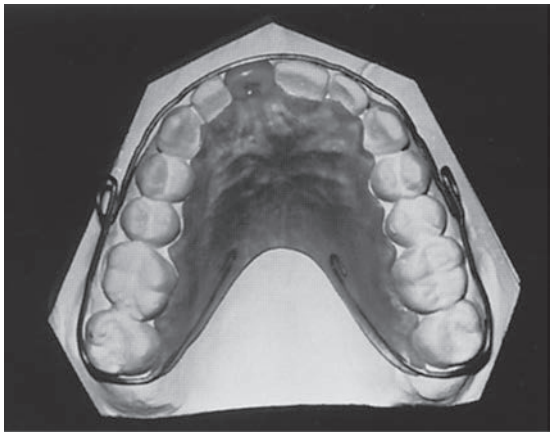


FIGURE 33-16 Maxillary circumferential retainer with pontic is applied to replace missing right central incisor.

FIGURE 33-17 A, Maxillary flipper with pontic to replace right lateral incisor is worn during the day for aesthetics. The flipper is made to fit over and/or around sectional wires bonded on the lingual side to prevent teeth from drifting into edentulous areas. B, Flipper is in place for aesthetic replacement of the maxillary right lateral incisor. Note the use of pink acrylic gingivally to blend into oral mucosa.

A clear removable retainer with pontic(s) held in place within the plastic is also typically made to be worn at night or for use in emergency situations if the regular retainer is lost or broken. The clear retainer is stronger and acts as a night guard to prevent breakage of the pontic from grinding or clenching teeth during sleep (Fig. 33-18).

Clear retainers are commonly used and have the benefits of excellent aesthetics, broad tooth contact to hold alignment, and function as a night guard for those patients who exhibit mild to moderate parafunction such as clenching or grinding teeth while sleeping (Fig. 33-19). These clear retainers are best adjusted or ground into occlusion to ensure even contact throughout the arches in centric relation occlusion.

The clinician also may alter the size of a retainer to accommodate special needs. A mandibular spring retainer, for instance, may include only six or eight anterior teeth to eliminate the difficulty of seating such an appliance over buccal segment undercuts, which are fairly common in adult patients with long clinical crown heights or undercuts created by excess lingual crown torque or restorations (Fig. 33-20).

The clinician may also use fixed retention appliances to retain potential areas of relapse more securely and to reduce the dependency on patient compliance (Fig. 33-21). The development of bonding materials has facilitated the fabrication of segmental retainers that splint two or more teeth together to maintain intraarch tooth position. The wires used for such retainers should be flexible (0.0175-inch twisted or 0.014- to 0.016-inch stainless steel) to allow physiologic tooth movement. Bonded retainers may also be effectively used to hold extraction spaces closed if relapse is anticipated (Fig. 33-22). Canine-to-canine bonded retainers can also be distally extended to include premolars if the premolars were initially ectopic or if holding the extraction spaces closed is the intent, as in adult extraction cases (Fig. 33-23). The clinician can also fabricate removable retainers

FIGURE 33-18 Clear retainer with the maxillary right lateral incisor pontic used during appliance therapy transferred and included in the retainer.

FIGURE 33-19 Example of clear maxillary overlay retainer.

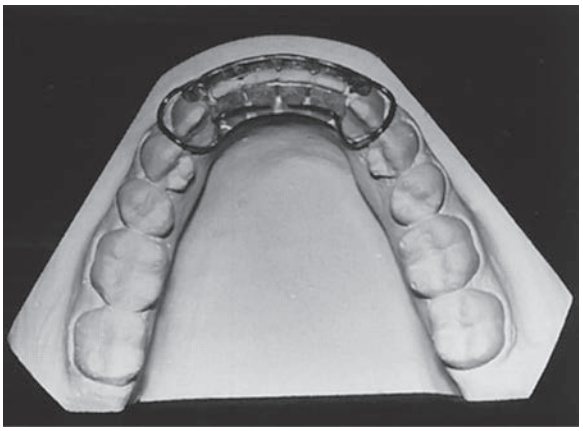


FIGURE 33-20 Mandibular spring retainer modified to maintain only anterior tooth position.

FIGURE 33-21 **A,** Mandibular bonded retainer. A braided wire is bonded to each tooth but allows physiologic tooth movement. **B,** A maxillary bonded retainer may also be placed to prevent relapse of initial spacing or irregularity. This retainer must be positioned and contoured to prevent premature mandibular incisor or canine contact.

FIGURE 33-22 Sectional wire bonded on the lingual to hold an extraction site closed.

FIGURE 33-23 Mandibular bonded retainers can be designed to extend distally to include premolars if premolars were initially ectopic or to hold extraction sites closed.

to fit over bonded retainers to minimize potential relapse in the other planes of space and to maintain interarch relationships.

Studies[102,103] conclude that fixed bonded retainers are as dentally and periodontally healthy as wearing removable retainers if they are maintained with proper oral hygiene techniques. The clinician can consider, however, eventually removing fixed retention and fabricating removable retainers to be used as needed to minimize the impact of long-term relapse and maturation changes.

POSITIONER IN RETENTION PLANNING

The positioner appears to be particularly beneficial in reestablishing normal tissue tone and firmness where gingival hyperplasia has occurred during treatment. The advantages of a positioner are that it is clean and unlikely to be broken, tends to stimulate tissue tone, and constantly works toward maintaining or improving tooth position. The disadvantages are its limited time of wear, because the patient can neither eat nor talk with the positioner in place, and the possibility that it may keep teeth loose by producing intermittent forces contrary to natural muscle balance. The positioner is probably contraindicated in patients who have a tendency for nasal airway obstruction and are mouth breathers because of the difficulty of breathing with a positioner in place.

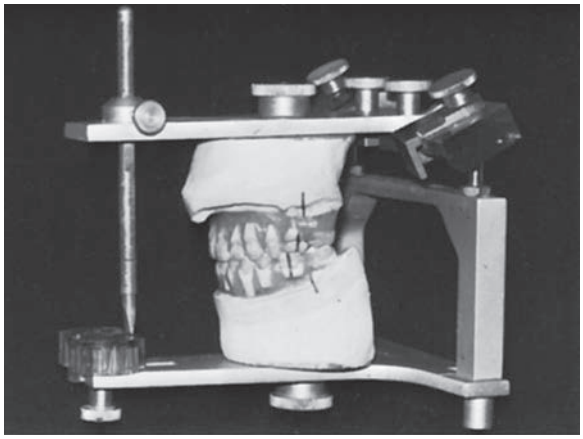


FIGURE 33-24 Diagnostic setup completed on an adjustable articulator after a facebow transfer. Fabrication of the positioner on this type of instrument allows the patient to bite into the positioner without altering condylar position.

If a positioner is to be used on what has been a deep overbite malocclusion, then construction of a maxillary retainer with a bite plane to be used when the patient is unable to wear the positioner is a beneficial adjunct. Construction and placement of a positioner that fits over a fixed appliance, such as a lingual bonded retainer, are also considerations.

Positioner setups and fabrication should be done on an adjustable articulator after a facebow transfer (Fig. 33-24) to allow the patient to function into the positioner without altering condyle-fossa relationships.

DURATION OF RETENTION

As noted from the literature, various authors suggest retention protocols that range from no retention whatsoever[35,83] to permanent retention. Most clinicians will institute some form of mandibular retention until completion of growth is evident. The indefinite use of retainers should also be strongly considered, as needed, to ensure the maintenance of tooth relationships in the long term. The clinician should discuss retention procedures with patients before starting orthodontic treatment to impress upon them the importance of the retentive phase that follows active treatment. Retention may be described as a 12-month *healing phase*, during which the newly made movements of teeth are stabilized, and a *maintenance phase*, to guard against the maturational changes in tooth position that occur over time. The latter phase is, of course, long term.

RECOVERY AFTER RELAPSE

If, despite the utmost care in treatment and retention, relapse or further postorthodontic maturational change occurs, then the following suggestions may be useful:

1. Retreatment may take the form of rebanding or rebonding some or all teeth. Admittedly, this measure is extreme, but it may be necessary to accomplish the desired correction.

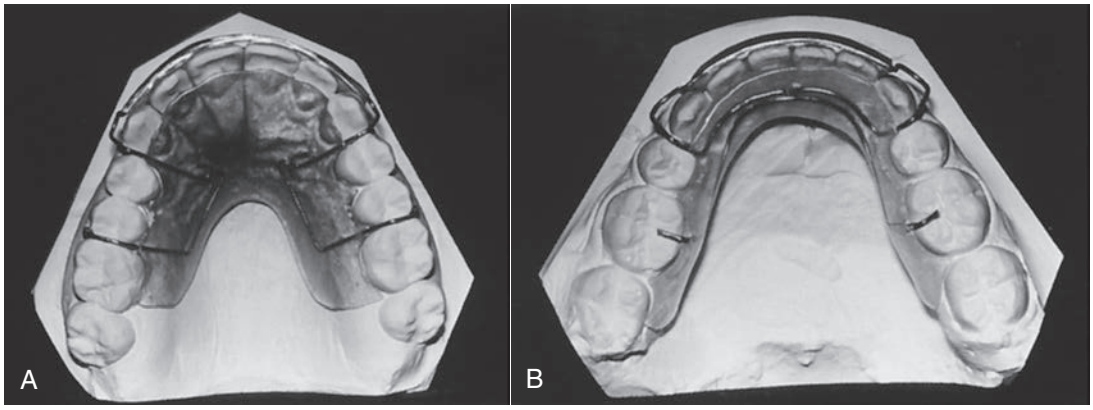


FIGURE 33-25 Maxillary (A) and mandibular (B) spring retainers. Incisors have been realigned in the working model.

Permanent and long-term retention is likely preferable after retreatment. In any case, discovering and eliminating the factors that appear contributory to the relapse should be attempted.

2. The mandibular lingual arch admirably serves to realign teeth in instances of mandibular collapse or crowding. Light pressure against the mandibular anterior teeth may be used to realign them. In this situation, a removable lingual arch that facilitates adjustment and recontouring is an advantage.
3. Springs and clasps can be added to removable retainers to assist in repositioning and controlling labiolingual deviations.
4. Spring retainers using facial and lingual acrylic for added leverage or clear aligners may be used for realignment. Teeth are sectioned and aligned as in a diagnostic setup or aligned on a computer simulation and the active retainer or aligner(s) is(are) fabricated to the realigned relationship (Fig. 33-25). The same spring retainer(s) used for alignment can be used to maintain the achieved correction. Interproximal stripping is sometimes beneficial as determined by the diagnostic wax-up or computer simulation.
5. A headgear or functional appliance may be used against the maxillary arch during growth to provide maintenance or recorrection in instances of relapse toward a Class II relationship.
6. Habit training in the form of myofunctional therapy may be beneficial when abnormal habit patterns have contributed to orthodontic relapse. Removable appliances are also helpful as tongue restraints and trainers.
7. Occlusal adjustment and interproximal tooth size reduction may be a consideration to achieve an optimal aesthetic and functional result. Interproximal reduction (IPR) or stripping in the crown width may reduce the tendency for further relapse.
8. In certain cases, the clinician may even suggest that the patient accept minimal relapse rather than continue with prolonged retreatment and retention.

SUMMARY

Retention is not a separate problem but is and will continue to be a problem to be considered during the initial and ongoing diagnosis and treatment planning for patients. As Oppenheim[46] so aptly stated, "Retention is one of the most difficult problems in orthodontia; in fact, it is the problem."

REFERENCES

1. Hellman M. Fundamental principles and expedient compromises in orthodontic procedures. In: *Transactions of the American Association of Orthodontists*. St. Louis: Mosby; 1945.
2. Kingsley N. *A Treatise on Oral Deformities as a Branch of Mechanical Surgery*. New York: Appleton & Co.; 1880.
3. Case CS. Retention of dental orthopedia. In: *A Practical Treatise on the Principles of Dental Orthopedia and Prosthetic Correction of Cleft Palate*. Chicago: CS Case; 1921.
4. Dewey M. *Practical Orthodontia*. St. Louis: Mosby; 1920.
5. Dewey M. Some principles of retention. *Am Dent J*. 1909;8:254.
6. Lischer BE. *Orthodontics*. Philadelphia: Lea & Febiger; 1912.
7. Guilford SH. *Orthodontia*. Philadelphia: Spangler & Davis; 1893.
8. Baker HA. Discussion of Dr. Pullen's paper. *Dent Items Interest*. 1907;29:389.
9. Hawley CA. A removable retainer. *Dent Cosmos*. 1919;61:449.
10. Jackson VH. *Orthodontia and Orthopedia of the Face*. Philadelphia: JB Lippincott; 1904.
11. Markus MB. A review and consideration of the problem of retention. *Am J Orthod Oral Surg*. 1938;24:203.
12. Mershon JV. The removable lingual arch appliance. In: *The First International Orthodontic Congress*. St. Louis: Mosby; 1972.
13. Northcroft G. The problem of retention with a view to permanence of result and minimum of danger. *Br Dent J*. 1915;36:898.
14. Ottolengui R. Discussion of Dr. Pullen's paper. *Dent Items Interest*. 1907;29:388.
15. Pullen HH. Some considerations in retention. *Dent Items Interest*. 1907;29:287.
16. Salzmann JA. *Principles of Orthodontics*. Philadelphia: JB Lippincott; 1943.
17. Simons ME, Joondeph DR. Change in overbite: a ten-year postretention study. *Am J Orthod*. 1973;64:349–367.
18. Thorne NAH. Experiences on widening the median maxillary suture. *Eur Orthod Soc Trans*. 1956;32:279.
19. Lundström A. Malocclusions of the teeth regarded as a problem in connection with the apical base. *Int J Orthod Oral Surg*. 1925;11:591.
20. McCauley DR. The cuspid and its function in retention. *Am J Orthod*. 1944;30:196–205.
21. Strang RHW, Thompson WM. *Textbook of Orthodontia*. 5th ed. Philadelphia: Lea & Febiger; 1958.
22. Nance H. The limitations of orthodontic treatment; diagnosis and treatment in the permanent dentition. *Am J Orthod*. 1947;33:253–301.
23. Grieve GW. The stability of the treated denture. *Am J Ortho Oral Surg*. 1944;30:171–195.
24. Tweed CH. Why I extract teeth in the treatment of certain types of malocclusion. *Alpha Omegan*. 1952;46:93–104.
25. Tweed CH. Indications for extraction of teeth in orthodontic procedure. *Am J Orthod Oral Surg*. 1944;30:405–428.

26. Rogers AP. Making facial muscles our allies in treatment and retention. *Dent Cosmos*. 1922;64(7):711–730.
27. Graber TM. Postmortems in posttreatment adjustment. *Am J Orthod*. 1966;52:331–352.
28. Bresolin D, Shapiro PA, Shapiro GG, et al. Mouth breathing in allergic children: its relationship to dentofacial development. *Am J Orthod*. 1983;83:334–340.
29. Mills JRE. A long-term assessment of the mechanical retroclination of lower incisors. *Angle Orthod*. 1967;37:165–174.
30. Welch KN. *A study of treatment and postretention dimensional changes in mandibular dental arches,* Master's thesis. Seattle: University of Washington; 1965.
31. Lopez-Gavito G, Wallen TR, Little RM, Joondeph DR. Anterior open-bite malocclusion: a longitudinal 10-year postretention evaluation of orthodontically treated patients. *Am J Orthod*. 1985;87:175–186.
32. Moorrees CF, Chadha JM. Available space for the incisors during dental development. *Angle Orthod*. 1965;35:12–22.
33. Richardson ER, Brodie AG. Longitudinal study of maxillary width. *Angle Orthod*. 1964;34:1–15.
34. Reitan K. Clinical and histologic observations on tooth movement during and after orthodontic treatment. *Am J Orthod*. 1967;53:721–745.
35. Englert G. Further determination and implementation of a clinical idealogy. *Angle Orthod*. 1960;30:14–25.
36. Walters DC. Changes in the form and dimensions of dental arches resulting from orthodontic treatment. *Angle Orthod*. 1953;23:3–18.
37. Linder-Aronson S. Adenoids: Their effect on mode of breathing and nasal airflow and their relationship to characteristics of the facial skeleton and the dentition. A biometric, rhino-manometric and cephalometro-radiographic study on children with and without adenoids. *Acta Otolaryngol Suppl*. 1970;265:1–132.
38. Reitan K. Tissue rearrangement during retention of orthodontically rotated teeth. *Angle Orthod*. 1959;29:105–113.
39. Allen RL. *A study of the regression phenomenon of orthodontically rotated teeth in the human patient with and without surgical removal of the supra-alveolar fiber apparatus,* Master's thesis. Seattle: University of Washington; 1969.
40. Boese LR. *Increased stability of orthodontically rotated teeth following gingivectomy,* Master's thesis. Seattle: University of Washington; 1968.
41. Brain WE. The effect of surgical transsection of free gingival fibers on the regression of orthodontically rotated teeth in the dog. *Am J Orthod*. 1969;55:50–70.
42. Edwards JG. A study of the periodontium during orthodontic rotation of teeth. *Am J Orthod*. 1968;54:441–461.
43. Fidler B, Artun J, Joondeph D, Little R. Long-term stability of Angle Class II, division 1 malocclusions with successful occlusal results at the end of active treatment. *Am J Orthod*. 1995;107:276–285.
44. Norwick K. *Long-term stability of Angle Class II, Division 1 malocclusion with unsuccessful versus successful occlusal results at the end of active treatment.* Master's thesis. Seattle: University of Washington; 1995.
45. Weinstein S. Minimal forces in tooth movement. *Am J Orthod*. 1967;53:881–903.
46. Oppenheim A. The crisis in orthodontia. Part I. 2. Tissue changes during retention: Skogsborg's septotomy. *Int J Orthod Dent Child*. 1934;20:639–644.
47. Horowitz SL, Hixon EH. Physiologic recovery following orthodontic treatment. *Am J Orthod*. 1969;55:1–4.
48. Hernandez JL. Mandibular bicanine width relative to overbite. *Am J Orthod Dentofacial Orthop*. 1969;56:445–467.
49. Lindquist JT. The lower incisor—its influence on treatment and esthetics. *Am J Orthod Oral Surg*. 1958;44:112–140.
50. Storey E. Tissue response to the movement of bones. *Am J Orthod*. 1973;64:229–247.
51. Waldron R. Reviewing the problem of retention. *Am J Orthod Dentofacial Orthop*. 1942;28:770–791.
52. Howes AE. Case analysis and treatment planning based upon the relationship of tooth material to its supporting bone. *Am J Orthod*. 1947;33:499–533.
53. Howes AE. Model analysis for treatment planning: a portion of symposium on case analysis and treatment planning. *Am J Orthod Dentofacial Orthop*. 1952;38:183–207.
54. Lude JC. Technique for the determination of the size of the mandibular apical base; its application to growth studies. *Angle Orthod*. 1967;37:272–284.
55. Baum AT. Orthodontic treatment and the maturing face. *Angle Orthod*. 1966;36:121–135.
56. Bjork A. Facial growth in man, studied with the aid of metallic implants. *Acta Odontol Scand*. 1955;13:9–34.
57. Bjork A. Variations in the growth pattern of the human mandible: longitudinal radiographic study by the implant method. *J Dent Res*. 1963;42:400–411.
58. Bjork A. A sutural growth of the upper face studied by the implant method. *Rep Congr Eur Orthod Soc*. 1964:49–65.
59. Schaeffer A. Behavior of the axes on human incisor teeth during growth. *Angle Orthod*. 1949;19:254–275.
60. Matthews JR. Clinical management and supportive rationale in early orthodontic therapy. *Angle Orthod*. 1961;31:35–52.
61. Kloehn SJ. At what age should treatment be started? *Am J Orthod Dentofacial Orthop*. 1955;41:262–278.
62. Moore AW. Orthodontic treatment factors in Class II malocclusion. *Am J Orthod Oral Surg*. 1959;45:323–352.
63. Witzel D. *Long-term stability of the mandibular arch following differential management of arch length deficiencies,* Master's thesis. Seattle: University of Washington; 1978.
64. Federspiel M. On gnathostatic diagnosis in orthodontics. *Int J Orthod*. 1924;10:783.
65. Ochsenbein C, Maynard JG. The problem of attached gingiva in children. *ASDC J Dent Child*. 1974;41:263–272.
66. Ballard ML. Asymmetry in tooth size: a factor in the etiology, diagnosis and treatment of malocclusion. *Angle Orthod*. 1944;14:67–70.
67. Stoner MM, Lindquist JT, Jack M, et al. A cephalometric evaluation of fifty-seven consecutive cases treated by Dr. Charles H. *Tweed. Angle Orthod*. 1956;26:68–98.
68. Litowitz R. A study of the movements of certain teeth during and following orthodontic treatment. *Angle Orthod*. 1948;18:113–132.
69. Mershon JV. The removable lingual arch appliance. In: *The First International Orthodontic Congress*. St. Louis: Mosby; 1972.
70. Bergström K, Jensen R. Responsibility of the third molar for secondary crowding. *Dent Abstr*. 1961;6:544.
71. Lindquist B, Thilander B. Extraction of third molars in cases of anticipated crowding in the lower jaw. *Am J Orthod Dentofacial Orthop*. 1982;81:130–139.
72. Schwarze CW. The influence of third molar germectomy: a comparative long-term study. In: *Transactions of the Third International Orthodontic Congress*. St. Louis: Mosby; 1975.
73. Simon P. *Fundamental Principles of a Systemic Diagnosis of Dental Anomalies*. Boston: Stratford Press; 1926.
74. Ades AG, Joondeph DR, Little RM, Chapko MK. A long-term study of the relationship of third molars to changes in the mandibular dental arch. *Am J Orthod Dentofacial Orthop*. 1990;97:323–335.
75. Kaplan R. Mandibular third molars and postretention crowding. *Am J Orthod*. 1974;66:411–430.
76. Richardson M. Late third molar genesis: its significance in orthodontic treatment. *Angle Orthod*. 1980;50:121–128.
77. Shanley LS. The influence of mandibular third molars on mandibular anterior teeth. *Am J Orthod Oral Surg*. 1962;48:786–787 (abstract).
78. Mettes TD, Ghaeminia H, Nienhuijs ME, et al. Surgical removal versus retention for the management of asymptomatic impacted wisdom teeth. *Cochrane Database Syst Rev*. 2012;13:6. CD003879.
79. Harradine NW, Pearson MH, Toth B. The effect of extraction of third molars on late lower incisor crowding: a randomized controlled trial. *Br J Orthod*. 1998;25:117–122.
80. Zawawi KH, Melis M. The role of mandibular third molars on lower anterior teeth crowding and relapse after orthodontic treatment: a systematic review. *Scientific World Journal*. 2014;2014:615429.
81. Gallerano R. *Mandibular anterior crowding, a postretention study,* Master's thesis. Seattle: University of Washington; 1976.
82. Angle EH. *Treatment of Malocclusion of the Teeth Angle's System*. 7th ed. Philadelphia: SS White Dental Manufacturing Co.; 1907.
83. Little RM, Wallen TR, Riedel RA. Stability and relapse of mandibular anterior alignment—first premolar extraction cases treated by traditional edgewise orthodontics. *Am J Orthod*. 1981;80:349–365.

84. Little RM, Riedel RA, Artun J. An evaluation of changes in mandibular anterior alignment from 10 to 20 years postretention. *Am J Orthod Dentofacial Orthop*. 1988;93:423–428.
85. Manish V, Hughes E. Results of a survey-based study to identify common retention practices in the United States. *Am J Orthod Dentofacial Orthop*. 2010;137:170–177.
86. Riedel RA, Little RM, Bui T. Mandibular incisor extraction–postretention evaluation of stability and relapse. *Angle Orthod*. 1992;62:103–116.
87. Little RM, Riedel RA, Stein A. Mandibular arch length increase during the mixed dentition: postretention evaluation of stability and relapse. *Am J Orthod Dentofacial Orthop*. 1990;97:393–404.
88. Zachrisson B, JCO/interviews Dr. Bjorn U. Zachrisson on excellence in finishing. Part 2. *J Clin Orthod*. 1986;20:536–556.
89. Johnson Egger N. *Long-term stability of maxillary anterior alignment,* Master's thesis. Seattle: University of Washington; 1992.
90. Little RM, Riedel RA. Postretention evaluation of stability and relapse—mandibular arches with generalized spacing. *Am J Orthod Dentofacial Orthop*. 1989;95:37–41.
91. McNamara Jr JA, Brudon WL. *Orthodontic and Orthopedic Treatment in the Mixed Dentition*. Ann Arbor, MI: Needham Press; 1993.
92. Little RM, Riedel RA, Engst ED. Serial extraction of first premolars—postretention evaluation of stability and relapse. *Angle Orthod*. 1990;60:255–262.
93. Dugoni SA, Lee JS, Varela J, Dugoni AA. Early mixed dentition treatment: postretention evaluation of stability and relapse. *Angle Orthod*. 1995;65(5):311–320.
94. Huang L, Artun J. Is the postretention relapse of maxillary and mandibular incisor alignment related? *Am J Orthod Dentofacial Orthop*. 2001;120(1):9–19.
95. Nett BC, Huang GJ. Long-term posttreatment changes measured by the American Board of Orthodontics objective grading system. *Am J Orthod Dentofacial Orthop*. 2005;127(4):444–450.
96. Littlewood SJ, Millett DT, Doubleday B, et al. Retention procedures for stabilising tooth position after treatment with orthodontic braces. *Cochrane Database Syst Rev*. 2006;1. CD002283.
97. McNeill RW, Joondeph DR. Congenitally absent maxillary lateral incisors: treatment planning consideration. *Angle Orthod*. 1973;43:24–29.
98. Sproule W. *Dentofacial changes produced by extraoral cervical traction to the maxilla of the* Macaca mulatta: *a histologic and serial cephalometric study,* Master's thesis. Seattle: University of Washington; 1968.
99. Magill JM. Changes in the anterior overbite relationship following orthodontic treatment in extraction cases. *Am J Orthod Dentofacial Orthop*. 1960;46:755–788.
100. Schudy FF. The control of vertical overbite in clinical orthodontics. *Angle Orthod*. 1968;38:19–39.
101. Douglas J. *A serial cephalometric evaluation of the dentofacial changes in patients treated with Class II intermaxillary forces,* Master's thesis. Seattle: University of Washington; 1973.
102. Artun J. Caries and periodontal reactions associated with long-term use of different types of bonded lingual retainers. *Am J Orthod*. 1984;86:112–118.
103. Booth FA, Edelman JM, Proffit WR. Twenty-year follow-up of patients with permanently bonded mandibular canine-to-canine retainers. *Am J Orthod Dentofacial Orthop*. 2008;133:70–76.

34

Interceptive Guidance of Occlusion with Emphasis on Diagnosis

Jack G. Dale* and Hali C. Dale

OUTLINE

Diagnostic Records, 997.e3
Intraoral, Panoral, or Cone-Beam Computed Tomography Images, 997.e3
Cephalometric Radiographs, 997.e3
Facial Photographs, 997.e3
Study Models, 997.e4
Intraoral Photographs, 997.e4
Diagnosis, 997.e5
The Face, 997.e5
Proportional Facial Analysis, 997.e5
The Standard, 997.e7
Alveolodental Protrusion, 997.e7
Alveolodental Retrusion, 997.e7
Prognathism, 997.e7
Retrognathism, 997.e8
The Teeth, 997.e13
Clinical Analysis, 997.e13
Dental Development in the Mixed Dentition, 997.e15
Total Space Analysis, 997.e22
Conventional Method, 997.e22
Tweed Method, 997.e23
Total Space Analysis, 997.e23
Timing of Guided Primary Tooth Removal, 997.e27
Treatment, 997.e28
Class I Treatment, 997.e30
Treatment Procedures: Interceptive Guidance, Active Treatment, 997.e30
Serial Extraction in Class I Treatment, 997.e30
Premolars, 997.e39
Typical Patient for Class I Serial Extraction, 997.e40
Key Measurements for Diagnosis and Case Evaluation, 997.e45

"This "Classic Chapter in Orthodontics" is taken from our fifth edition of Orthodontics: Current Principles and Techniques published by Elsevier in 2012. It is presented as then written. The chapter contains perspectives and information that remain valuable to orthodontic residents and clinicians."

As we learn more about growth and its potentials, more about the influences of function on the developing denture, and more about the normal mediodistal position of the denture in its relation to basal jawbones and head structures, we will acquire a better understanding of when and how to intervene in the guidance of growth processes so that Nature may better approximate her growth plan for the individual patient. In other words, knowledge will gradually replace harsh mechanics, and in the not-too-distant future the vast majority of orthodontic treatment will be carried out during the mixed-dentition period of growth and development and before the difficult age of adolescence.

—Charles H. Tweed

The term *serial extraction* was first introduced by Kjellgren[1,2] in 1929. Special knowledge is required to carry out this procedure successfully. Unfortunately, Kjellgren's phrase resulted in the indiscriminate removal of teeth by individuals who have not appreciated the requisite knowledge. A common misconception is that the procedure is easy because it implies simply the removal of teeth serially.

Hotz,[3] however, referred to the procedure as *guidance of eruption*. This is a better title than Kjellgren's because it implies that knowledge of growth and development is necessary to direct the teeth as they erupt into occlusion. The term *guidance of occlusion* is even more appropriate because clinicians are interested in the final destination of eruption: the occlusion. One must have a thorough understanding of growth and development with all its ramifications and must be aware of occlusion of the dentition, with its relationship to the craniofacial structures and function. This chapter discusses the serial extraction of the primary teeth to guide the eruption of the permanent teeth into

*An article in memoriam of author Jack G. Dale can be found at the following link: http://www.sciencedirect.com/science/article/pii/S0889540616001827

a favorable occlusion to intercept, in part, the occurrence of a major malocclusion.

When the practitioner is contemplating the correction of an orthodontic problem, the most crucial decision to make is whether teeth should be extracted. Even more demanding is adding the morphogenetic pattern and the dimensions of time, complicating that with the growth and development of the dentition and the craniofacial complex and carrying out the procedure serially.[2]

Serial extraction is not easy, as so many mistakenly believe. Serial extraction should never be initiated without a comprehensive diagnosis. Teeth may be extracted with the greatest of ease during a so-called serial extraction procedure. However, if the basic principles of diagnosis are ignored, the result will be failure and disappointment. Serial extraction will be injurious not only to the patient but also to the reputation of the practitioner and ultimately to the profession. Legal concerns regarding risk management include promiscuous extraction.[4]

Unfortunately, since Bunon[5] introduced serial extraction to the profession 268 years ago, it has been grossly misunderstood. Lack of understanding and knowledge has produced disastrous results, including a deterioration of the dentition and facial balance. Serial extraction has been criticized and maligned unfairly by individuals who have never used the procedure in practice or acquired the necessary knowledge to perform it well.

If serial extraction is based on a thorough diagnosis and carried out carefully and properly on a select group of patients, the procedure can be an excellent and valuable treatment. Serial extraction can reduce appliance treatment time, the cost of treatment, discomfort to the patient, potential iatrogenic sequelae, and time lost by the patient and the parents.

To intercept a malocclusion as early as possible and to reduce or, in rare instances, avoid fixed appliance mechanotherapy at the sensitive teenage period is logical. Why allow an unfavorable dental, skeletal, or soft tissue relationship to exist for a number of years if it can be corrected, or significantly improved, early with a minimum of appliance treatment time?

Before attempting the treatment of an orthodontic patient using guidance of occlusion, the practitioner must be prepared to meet the challenge of diagnosis. Without question, the secret of success in orthodontic treatment is a thorough understanding of mixed dentition diagnosis. The orthodontist can have the most comprehensive and sophisticated treatment plan at work in the patient's mouth; however, if the plan is implemented in the wrong patient, treatment will fail. This chapter discusses guidance of occlusion with a special emphasis on diagnosis based treatment planning.

Charles Tweed, whose words are quoted at the beginning of this chapter, was a man who devoted more than 40 years of his life to the treatment of the permanent dentition and the development of precision edgewise mechanotherapy, sometimes referred to as "harsh mechanics." However, during the last 13 years of his life, he was vitally interested in treatment during the mixed dentition period, including preorthodontic guidance, guidance of occlusion, and serial extraction.

Tweed and many others found that serial extraction, especially in Class I tooth size–jaw size discrepancy malocclusions, improves the alignment of the teeth when they emerge into the oral cavity. As a result, it creates a better environment "so that Nature may better approximate her growth plan." Tweed also discovered that the interception of dentofacial deformities using "growth and its potentials" and biologic principles rather than "harsh mechanics" was an exhilarating and rewarding experience.

FIGURE 34-1 Class II, Division 2, malocclusion with an impacted permanent maxillary right canine. During a misguided serial extraction program, one mandibular incisor and the maxillary right first premolar were extracted.

FIGURE 34-2 A 13-year-old girl who did not have the benefit of serial extraction and early treatment.

Early consideration of the corrective measures necessary to remedy any type of malocclusion should be a prime concern of the practitioner. Whatever method is chosen, treatment time should be at a minimum.[6]

Patient requests for more technique sensitive lingual, esthetic labial and/or aligner appliances can make serial extraction even more important. There are increasing concerns of tissue damage and the iatrogenic potential of our armamentaria and materia technica (see Chapter 30).[4] The best way to hide appliances is to not use them or at least to diminish their use. By using the benefits of serial extraction in Class I malocclusions, orthodontists can concentrate more fully on the time-consuming and technically demanding Class II and III malocclusions; thus, these orthodontists increase their usefulness to their profession and to society.

Serial extraction often has been criticized as being a bad procedure, and it certainly can be more harmful than beneficial if not done properly (Fig. 34-1). The two girls in Figures 34-2 and 34-3 are 13 years of age, just at the beginning of adolescence. The girl in Figure 34-2 has a severe Class I tooth size–jaw size discrepancy. Her treatment required the extraction of four premolar teeth and 30 months of mechanotherapy beginning at 13 years. The retention period was prolonged because the teeth had been allowed to remain in a crowded irregular relationship for several years. The girl in Figure 34-3 had a similar malocclusion, but

FIGURE 34-3 A 13-year-old girl who had the benefit of serial extraction and early treatment.

her treatment was completed by 13 years. Serial extraction was begun at 8 years, and the multibonded appliance was inserted at 12 years. The retention period was minimal. Thus, two girls with similar malocclusions were treated differently. Whereas one was just beginning 30 months of treatment at 13 years, the other already was finished and had a more stable result.

The recommended routine office procedure performed before serial extraction requires a complete orthodontic evaluation, including a comprehensive patient history and a thorough clinical and diagnostic record evaluation followed by an appropriate parent or guardian consultation (see Chapters 8-11 and 16).

DIAGNOSTIC RECORDS

A complete set of diagnostic records is acquired to develop the diagnosis, including panoral radiographs and cephalometric radiographs (or cone-beam computed tomography [CBCT]), oriented facial photographs, study models, and intraoral photographs of the dentition. The objective of taking quality records is to secure clinical accuracy for the purpose of establishing a sound diagnosis and being able to communicate the therapeutic needs to the parent and patient as well as general or pediatric dentist.[7]

Intraoral, Panoral, or Cone-Beam Computed Tomography Images

Dental radiographs must be taken for the following reasons:

1. Protection of the patient and documentation for the orthodontist
2. Detection of congenital absences of teeth
3. Detection of supernumerary teeth
4. Evaluation of the dental health of the permanent teeth (detection of pathologic conditions in the early stages)
5. Assessment of trauma to the teeth after an injury
6. Detection of evidence of a true hereditary tooth size–jaw size discrepancy such as the resorptive pattern on the mesial of the roots of the primary canines
7. Determination of dental age of the patient by assessing the length of the roots of permanent unerupted teeth and the amount of resorption of primary teeth, as in dental age analysis
8. Calculation of the total space analysis (see Figs. 34-46 to 34-56)
9. Detection of root resorption before, and by means of comparison, during and after treatment (Fig. 34-4)
10. Evaluation of third molars before, during, and after treatment

FIGURE 34-4 Root resorption of the maxillary central incisors by impacted canines. (From Dale JG. Trauma: its influence on orthodontic treatment planning. *Dent Clin North Am* 1982;26:565.)

11. Posttreatment radiographic evaluation allows for appraisal and documentation of the dental health after orthodontic treatment.

Cephalometric Radiographs

Cephalometric radiographs are used for the following:

1. Evaluation of craniofaciodental relationships before treatment
2. Assessment of the soft tissue matrix
3. Classification of facial patterns (as in the proportional facial analysis) (see Figs. 34-8 to 34-17)
4. Calculation of tooth size–jaw size discrepancies (as in the total space analysis)
5. Determination of mandibular position
6. Prediction of growth and development
7. As a basis for monitoring of skeletodental relationships during treatment
8. Detection of pathologic conditions before and after treatment
9. Assessment of trauma after facial injuries
10. These radiographs and their pretreatment and posttreatment evaluation allow for long-term improvement in treatment planning.

Facial Photographs

Figures 34-5 and 34-6 illustrate the requirements of the American Board of Orthodontics for oriented facial and intraoral photographs.[8] Facial patterns play an important role in diagnosis and treatment planning, especially in serial extraction. From a practical standpoint, the photographs allow the orthodontist to better identify the patient. The prime objective of diagnosis and treatment in relation to the face should be the creation of harmony and balance: a favorable, proportionate relationship between the teeth, skeletal pattern, and soft tissue matrix, including the profile. Similar

Requirements:
Lateral view:
- Ears displayed
- Full face displayed in natural head position
- Left eyelash slightly visible

Frontal views:
- Teeth in occlusion
- Eyes are open and looking into camera
- Ears exposed
- Lips relaxed and in contact
- No distracting eyewear or jewelry
- Cropped to reveal facial symmetry and balance
- Inter-pupillary line bisecting the frame revealing top of shoulders and neck
- Use the same format for the smiling and nonsmiling photographs

FIGURE 34-5 American Board of Orthodontics requirements for facial photographs. (From American Board of Orthodontics. *Specific Instructions for Candidates*. St. Louis: 2016. www.americanboardortho.com/orthodontic-professionals/about-board-certification/clinical-examination/case-record-preparation/photograph-requirements.)

to cephalometric radiographs, facial patterns are invaluable for the following:

1. Evaluation and documentation of craniofacial (and dental) relationships and proportions before treatment
2. Assessment of soft tissue profile and muscle balance
3. Proportional facial analysis and evaluation of symmetry
4. Total space analysis in tandem with occlusal curve analysis
5. Monitoring of treatment progress
6. These photographs and their pretreatment and posttreatment evaluation allow for long-term improvement in treatment planning.

Study Models

Study models provide a three-dimensional record of the dentition and are essential for many reasons.[8] Although CBCT radiographic imaging and occlusal scanning and other imaging technologies may eventually allow us to replace this record, models are used for the following:

1. To calculate total space analysis
2. To assess and document the dental anatomy
3. To assess and document the intercuspation
4. To assess and document arch form
5. To assess and document the curve of occlusion (occlusal curve analysis)
6. To evaluate functional occlusion, potentially with the aid of articulators
7. As a basis from which to measure progress during treatment
8. To detect abnormalities (e.g., localized enlargements and distortion of arch form)
9. These records and their pretreatment and posttreatment evaluation allow for long-term improvement in treatment planning.

Intraoral Photographs

Figure 34-6 illustrates the requirements of the American Board of Orthodontics for intraoral photographs.[8]

Intraoral photographs are valuable to evaluate and document the immediate soft and hard tissue relationships surrounding the teeth. They present a time-stamped record of the visual characteristics of the gingiva as well as the enamel before placement of any appliances. Intraoral photographs add the dimension of color to the records, which aids in assessing and recording the health or disease of the teeth and soft tissue structures.[4]

Regarding serial extraction, photographs allow the clinician to record the steps in the technique, frequently without making study models. This is important for self-evaluation and discussion with patients and parents as well as our general and pediatric dental colleagues. A picture is truly worth a thousand words,

- Mid-palatal raphe centered
- Frame the entire arch with minimal amount of soft tissue displayed

- Fill the frame with the entire mandibular arch at least through the first molars
- Labial surface of the central incisors parallel to the bottom of the frame
- Midline centered in the frame

- Anteriorly include the contralateral central incisor
- Posteriorly include at least the first molars
- Occlusal plane should be parallel to the frame
- Occlusal plane should be horizontal and should bisect the photograph
- There should be equal display of the posterior dentition
- Use the same format as for the right lateral intra-oral photograph

FIGURE 34-6 American Board of Orthodontics requirements for intraoral photographs. (From American Board of Orthodontics. *Specific Instructions for Candidates*. St. Louis: 2016. www.americanboardortho.com/orthodontic-professionals/about-board-certification/clinical-examination/case-record-preparation/photograph-requirements.)

and with the ability to share digital images, we are better able to communicate and consult on important treatment decisions. The ability of a lay parent or guardian to see problems that have been identified, visualize changes, and share with family throughout treatment is a great benefit as well.

DIAGNOSIS

To differentiate, categorize, and treat specifically, successfully, and routinely requires an understanding of the fundamental principles of diagnosis. Several analyses related to the face and teeth are discussed in this section. These examples of the procedures should be followed if treatment is to be successful and serial extraction effective.

The Face

The infant face is not simply a miniature of the adult face (Fig. 34-7). The linkage of the terms *growth* and *development* indicates that the enlargement of the face involves more than progressive increases in size. Growth is a differential process in which some parts enlarge more or less than others and in a multitude of directions. Growth is a gradual maturational process taking many years and requiring a succession of changes in regional proportions and relationships of various parts. Many localized alterations occur that are associated with a continuous process of soft and hard tissue remodeling. An understanding of the mechanisms of growth and development of the face is essential to the practice of dentistry (see Chapter 1). To achieve this understanding, one must have some knowledge of faces.

Normal growth is unbalanced and therefore progressive, and, noticeably, alterations in facial form and pattern take place. Differential growth processes not only produce a wide range of topographic facial variations but also constitute the developmental basis for malocclusions and congenital facial abnormalities. To control and use the complex processes of growth in clinical procedures, an understanding of various concepts is essential for dentists.

Proportional Facial Analysis

The proportional facial analysis is basically a classification of facial patterns based on the Steiner analysis,[9] the Merrifield and Tweed cephalometric analyses,[10,11] and especially the counterpart analysis of Enlow and Hans.[12] Proportional facial analysis includes an evaluation of the following relationships (Fig. 34-8, *A*):

- Anterior cranial base (1, 2)
- Posterior cranial base (2, 3)
- Cranial base angle (1, 2, 3)
- Ramus of the mandible (3, 4)
- Corpus of the mandible (4, 5)
- Gonial angle (3, 4, 5)
- Nasomaxillary complex (6, 7, 8, 9)
- Maxillary dentition (10, 11)
- Mandibular dentition (12, 13)

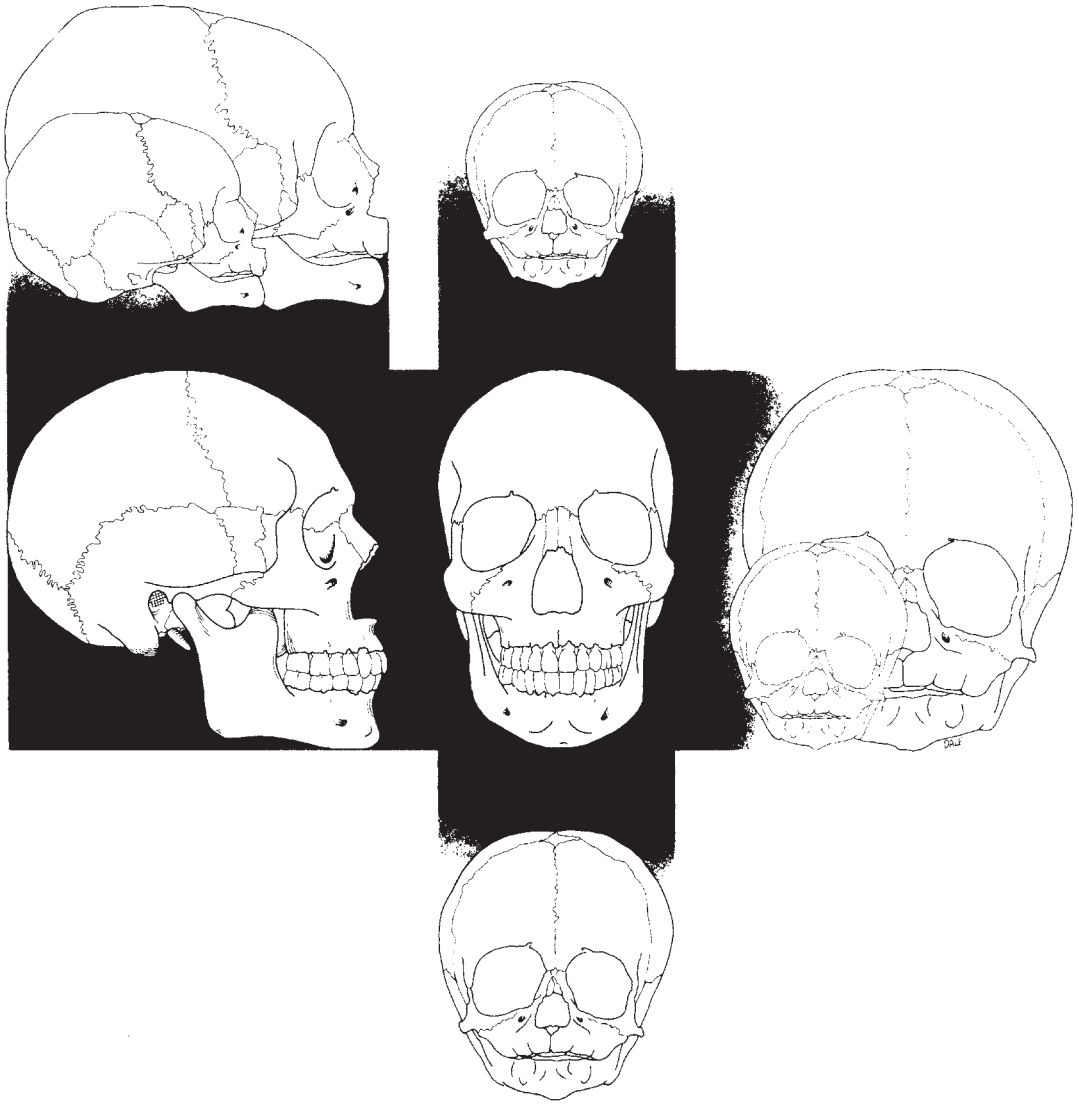


FIGURE 34-7 Changes in facial proportions throughout growth and development from infancy to maturity. To show the regional differences in height, depth, and breadth, the newborn skull has been enlarged to the same size as the adult skull. (From Enlow DH, Dale JG. Childhood facial growth and development. In: Ten Cate AR, ed. *Oral Histology: Development, Structure, and Function*. 5th ed. St. Louis: Mosby; 1998.)

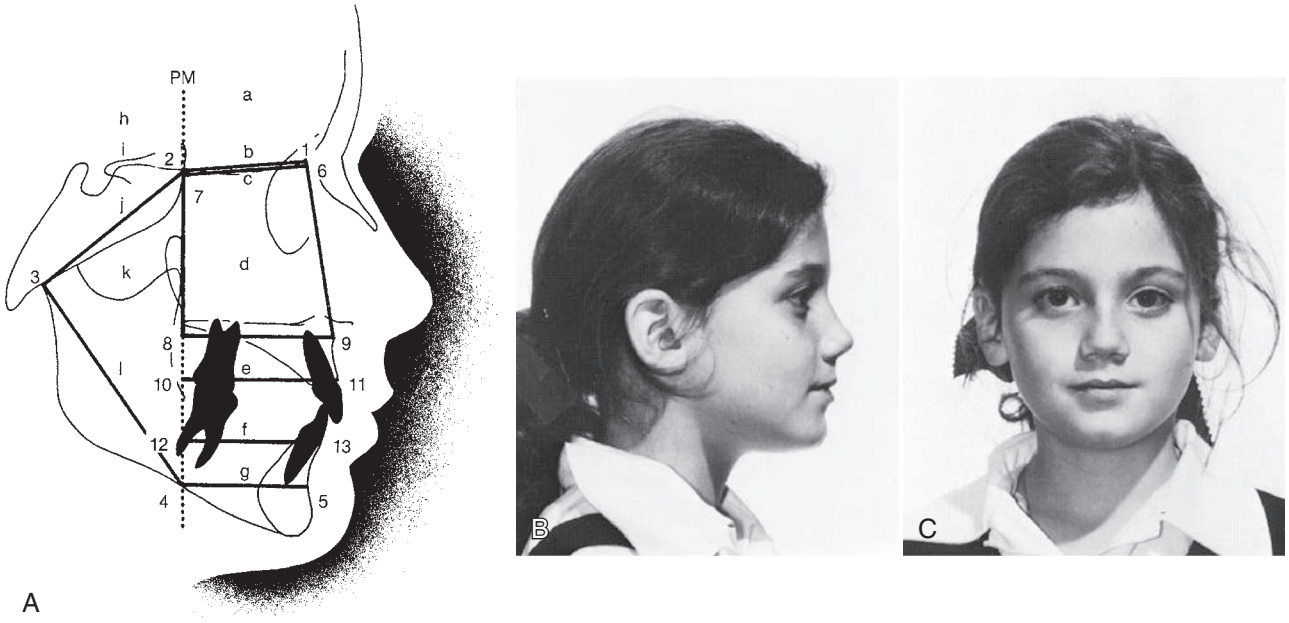


FIGURE 34-8 Standard or orthognathic facial pattern. **A,** The basic units associated with the proportional facial analysis. **B** and **C,** The patient.

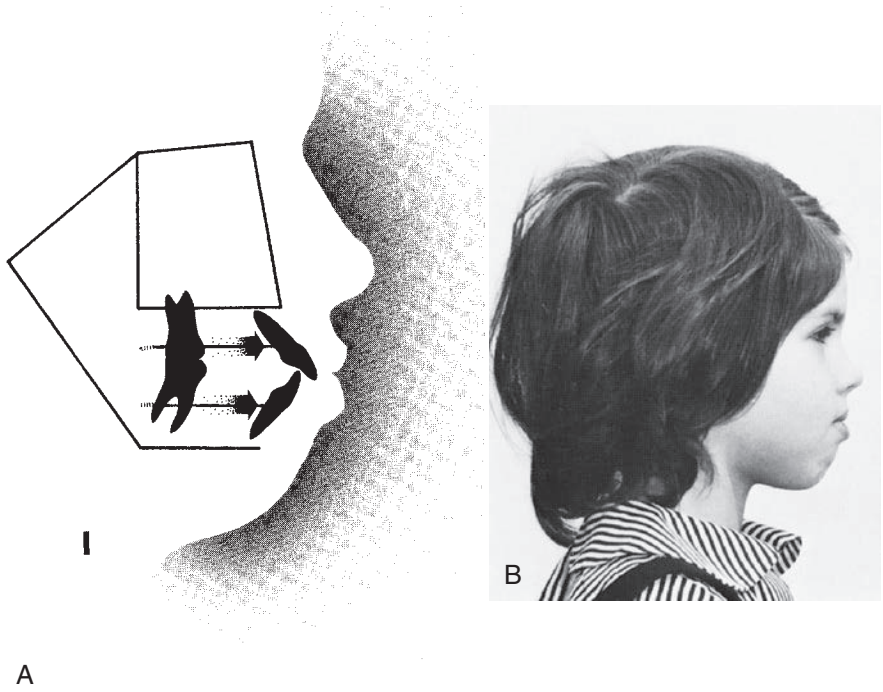


FIGURE 34-9 A, Class I maxillary and mandibular alveolodental protrusion. B, Typical patient profile.

The posterior maxillary (PM) plane is possibly the most significant plane in the craniofacial complex. The plane delineates, naturally, the various anatomic counterparts and is a developmental interface between the series of counterparts in front of and behind it. The PM plane thus retains a number of basic relationships throughout the growth process.

The Standard

The standard or orthognathic face, as in Figure 34-8, *B* and *C*, exhibits a harmonious relationship between the facial structures and the cranium, between the maxilla and the mandible, between the maxilla and the maxillary dentition, between the mandible and the mandibular dentition, between the maxillary dentition and the mandibular dentition, and between the soft tissue profile and the underlying hard tissue structures ("the mesiodistal position of the denture in its relation to the basal jawbones and head structures").[13]

Alveolodental Protrusion

Class I: Maxillary & Mandibular Alveolodental Protrusion. The maxillary and the mandibular dentitions are forward, and the teeth are in a Class I relationship (Fig. 34-9). This facial pattern in general responds well to extraction and, when caution is exercised, to serial extraction.[14,15]

Class II: Maxillary Alveolodental Protrusion. The maxillary dentition is forward, and the teeth are in a Class II relationship, but everything else is favorable (Fig. 34-10). Routinely, this malocclusion can be treated by the extraction of two maxillary first premolars and by serial extraction in the maxilla only.

Class III. In the interest of brevity and relevance, the Class III category of this analysis is not included in the discussion. Class III malocclusions are not suitable for serial extraction procedures. Mixed dentition Class III treatment is a complicated undertaking, is often extensive and prolonged, and may have to be repeated (see Chapter 16).

Alveolodental Retrusion

Class I: Maxillary & Mandibular Alveolodental Retrusion. The maxillary and mandibular dentitions are back, and the teeth are in a Class I relationship (Fig. 34-11). The orthodontist must be particularly careful in treating this type of facial pattern. If possible, treatment should be done without the extraction of teeth. This appearance can be seen in individuals who have had no orthodontic treatment and in patients who have had treatment with extractions and without. A dished-in face cannot always be avoided, regardless of the treatment.

Class II: Mandibular Alveolodental Retrusion. The mandibular dentition is back, and the teeth are in a Class II relationship (Fig. 34-12). Again, the practitioner must be careful about extracting teeth in this type of facial pattern.

Prognathism

Class I: Maxillary & Mandibular Prognathism. In maxillary and mandibular prognathism, both jaws are forward, and the teeth are in a Class I relationship (Fig. 34-13). Often this is a most aesthetic facial pattern. Many motion picture stars exhibit this type of profile. If the teeth are severely crowded, serial extraction should be performed. However, because of the increase in the size of the jaws, extractions are not always indicated.

Class II: Maxillary Prognathism. In maxillary prognathism, the maxilla is forward, and the teeth are in a Class II relationship (Fig. 34-14). This relationship may be because the maxilla itself is forward or may result from a long anterior cranial base. Also, the cranial base angle may be flat, creating a downward and forward position of the nasomaxillary complex. This in turn may rotate the mandible down and back. These are difficult Class II malocclusions to treat and certainly demand more

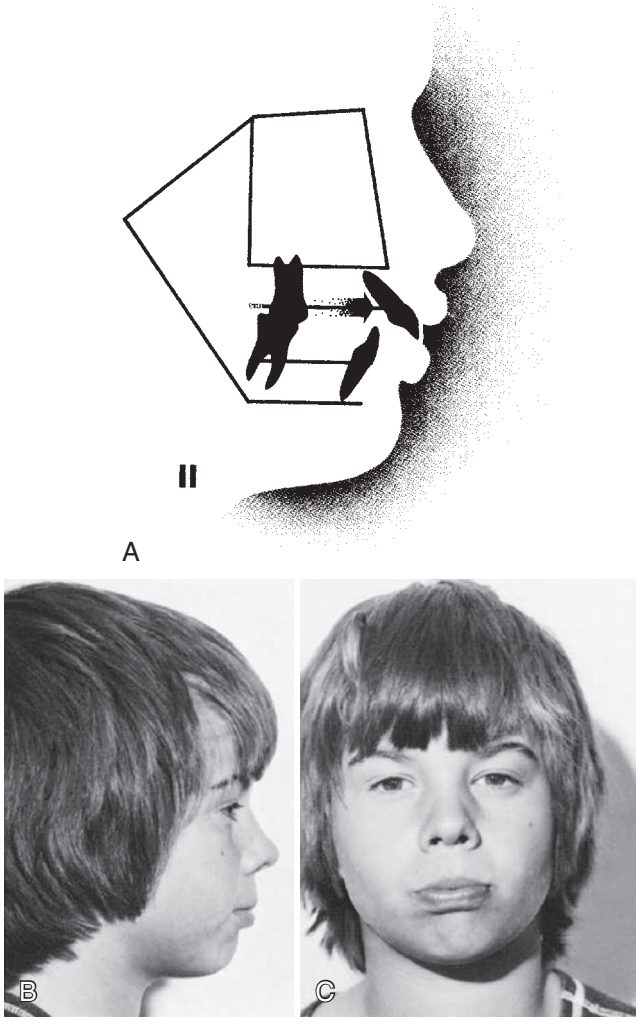


FIGURE 34-10 A, Class II maxillary alveolodental protrusion. B and C, Typical patient profile and frontal views demonstrating perioral muscle strain.

than serial extraction. The malocclusions are complicated skeletal discrepancies that may require not only comprehensive mechanotherapy but also surgical intervention. Midface protrusions, as they are called sometimes, are difficult profiles to correct satisfactorily by orthodontic treatment alone. The nose, at the end of treatment, often is prominent.

Retrognathism

Class I: Maxillary & Mandibular Retrognathism. In Class I malocclusions exhibiting maxillary and mandibular retrognathism, the maxilla and mandible are back in relation to the other craniofacial structures (Fig. 34-15). It is difficult to produce a favorable profile in these patients because of the lack of horizontal growth and the recessive nature of their profiles. Every effort should be made to encourage a forward development of the jaws. However, this is difficult because the dentition is in a Class I relationship. Over a long time, such a profile can become more recessive and, if serial extraction is being done, the orthodontist could be saddled with the blame. It is important to explain to the parents during the case presentation that the patient appears to have an unfavorable growth pattern that may get worse regardless of treatment.

Class II: Mandibular Retrognathism. In Class II malocclusions exhibiting mandibular retrognathism, the mandible is back. The corpus of the mandible may be small (Fig. 34-16), or the ramus may be narrow. The gonial angle may be proportionately acute. Excessive vertical development may be evident in the nasomaxillary complex (Figs. 34-16 and 34-17), which in turn rotates the mandible down and back, producing a retrognathic mandible and a tendency to open bite. If this is the cause of the retrognathic mandible, the treatment of preference may be surgery or, if less extreme, use of temporary anchorage devices (TADs) to intrude posterior dental segments to allow the forward rotation of the mandible. Regardless, a patient with this facial pattern is not a good candidate for serial extraction. McNamara and Brudon[16] found the most common characteristic of Class II malocclusions to be a retrognathic mandible, with excessive vertical development.

FIGURE 34-11 A, Class I maxillary mandibular alveolodental retrusion. B, Typical patient profile.

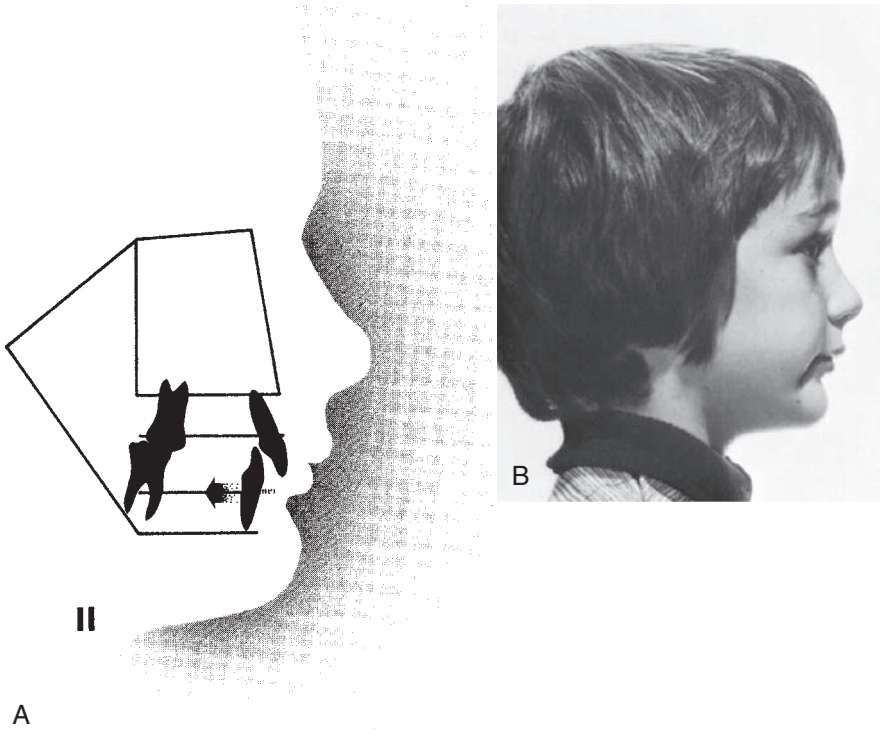


FIGURE 34-12 A, Class II mandibular alveolodental retrusion. B, Typical patient profile with adverse lip posture and function supporting the malocclusion during function.

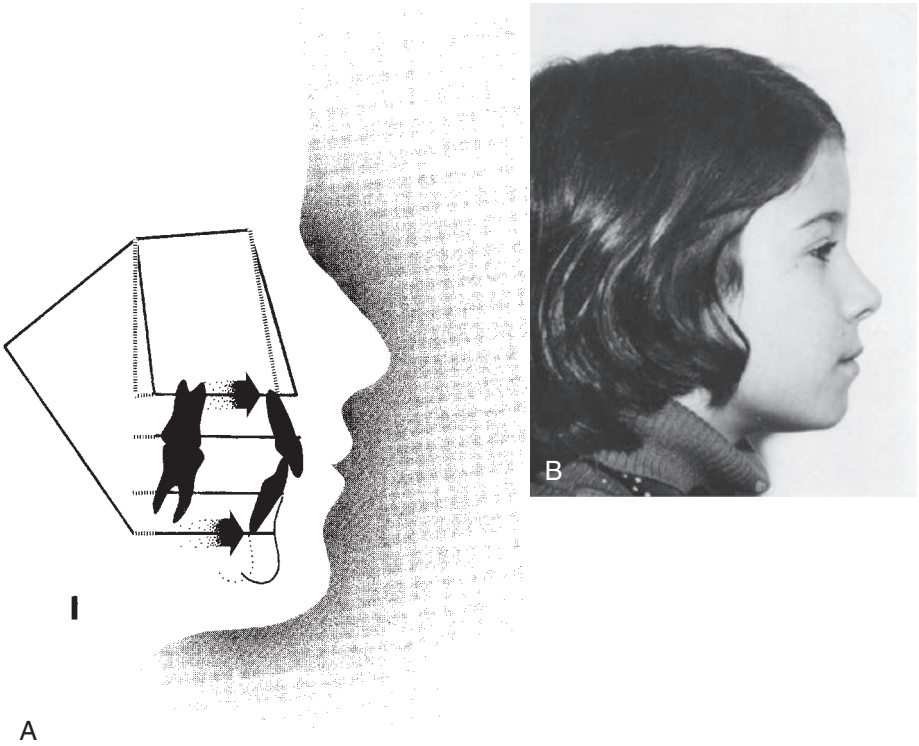


FIGURE 34-13 A, Class I maxillary and mandibular prognathism. B, Typical patient profile.

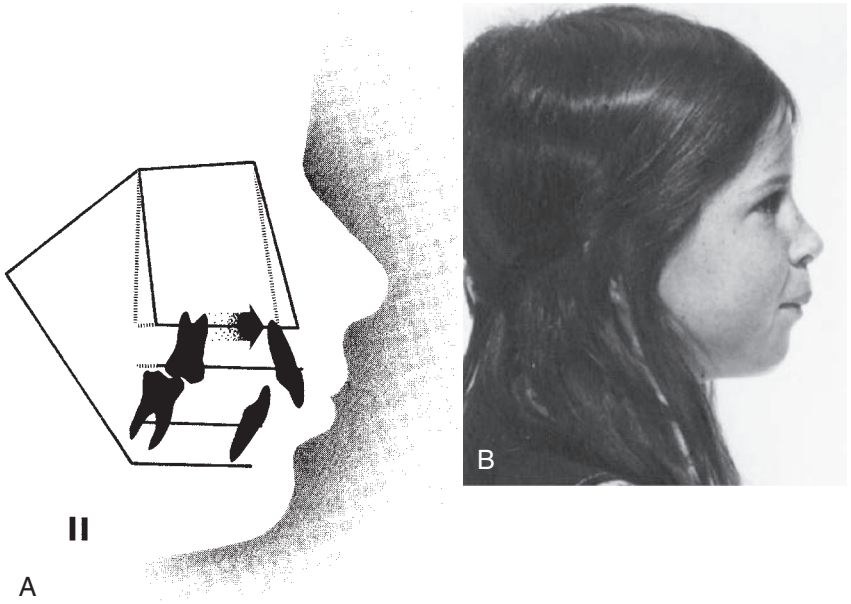


FIGURE 34-14 A, Class II maxillary prognathism. B, Typical patient profile.

According to the proportional facial analysis, a Class I malocclusion could be associated with a facial pattern characterized by a maxillary and mandibular alveolodental protrusion, maxillary and mandibular alveolodental retrusion, maxillary and mandibular prognathism, maxillary and mandibular retrognathism (even orthognathism), or a combination of these features.

What is evident from this analysis is that to treat all Class I malocclusions in the same manner would not be using sound clinical judgment. Class I malocclusions with excessive vertical development of the nasomaxillary area might be treated better by posterior segment impaction with TADS or combined orthodontics and orthognathic surgery or alternate tooth extraction protocols rather than by serial extraction followed by removal of the four first premolars.

Similarly, a Class II malocclusion could be a result of maxillary alveolodental protrusion; mandibular alveolodental retrusion; a prognathic maxilla caused by a forward nasomaxillary complex, long anterior cranial base, or flat cranial base angle; a retrognathic mandible caused by a short corpus, narrow ramus, acute gonial angle, and excessive vertical development of the nasomaxillary complex; or a combination of these features.

What is even more evident is that one should give serious thought before using serial extraction in Class II malocclusions. Serial extraction aids in the correction of tooth size–jaw size discrepancies but not necessarily in the correction of a Class II relationship. Therefore, the orthodontist must be prepared to place appliances for an extended time. The diagnosis is particularly important because growth guidance appliances and/or permanent teeth other than the four first premolars may be extracted in the treatment of these malocclusions, which is referred to later in the discussion of total space analysis.

The proportional facial analysis describes clearly the multifactorial basis for malocclusion and also explains the way compensation may occur to prevent or minimize a discrepancy. A patient's facial pattern may include a Class I profile with harmony and balance that results from a small cranial base angle (which is a Class III characteristic) and a short corpus (which is a Class II characteristic). Because the maxilla and the mandible tend toward retrognathism, the profile that results is in balance.

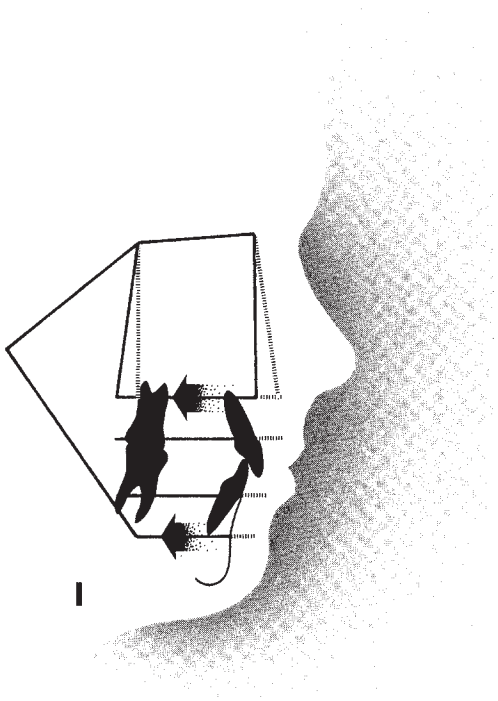


FIGURE 34-15 Class I maxillary and mandibular retrognathism.

Similarly, a combination of factors could result in a more severe malocclusion. A patient might have a Class I malocclusion as a result of excessive vertical development (a Class II characteristic) and a large gonial angle (a Class III characteristic). However, in this instance, the two factors do not balance one another but combine to make the malocclusion more severe. This Class I dentition would not be a good candidate for premolar extraction or serial extraction.

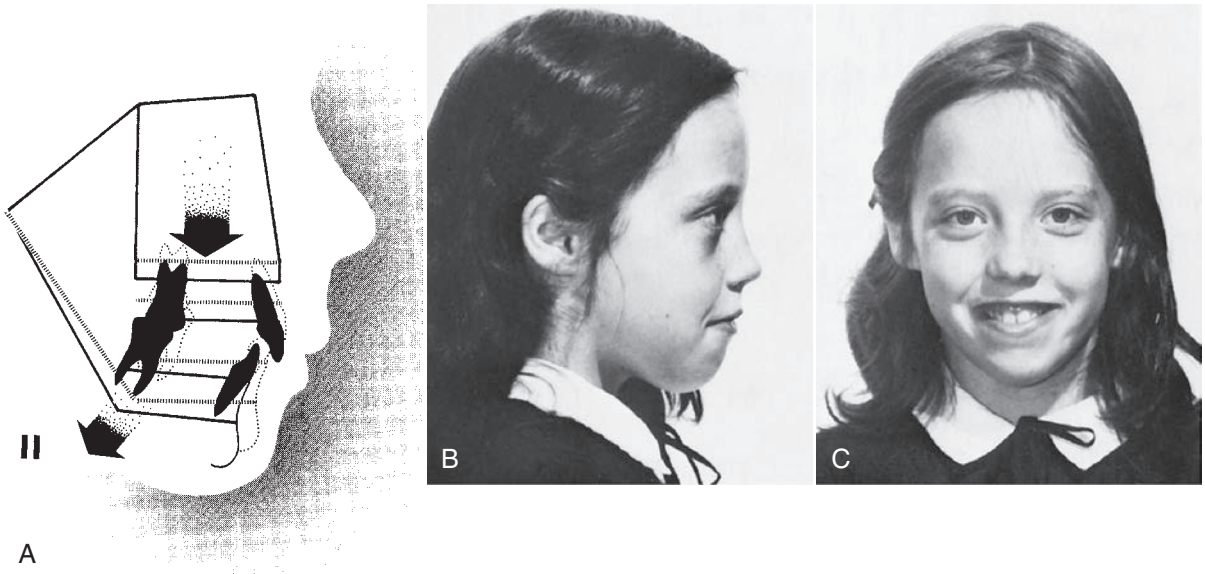


FIGURE 34-16 A, Class II mandibular retrognathism with increased vertical facial height. B and C, Typical patient profile and frontal view. Patients with excessive vertical development reveal substantial gingival tissue when smiling albeit philtrum height is within norm.

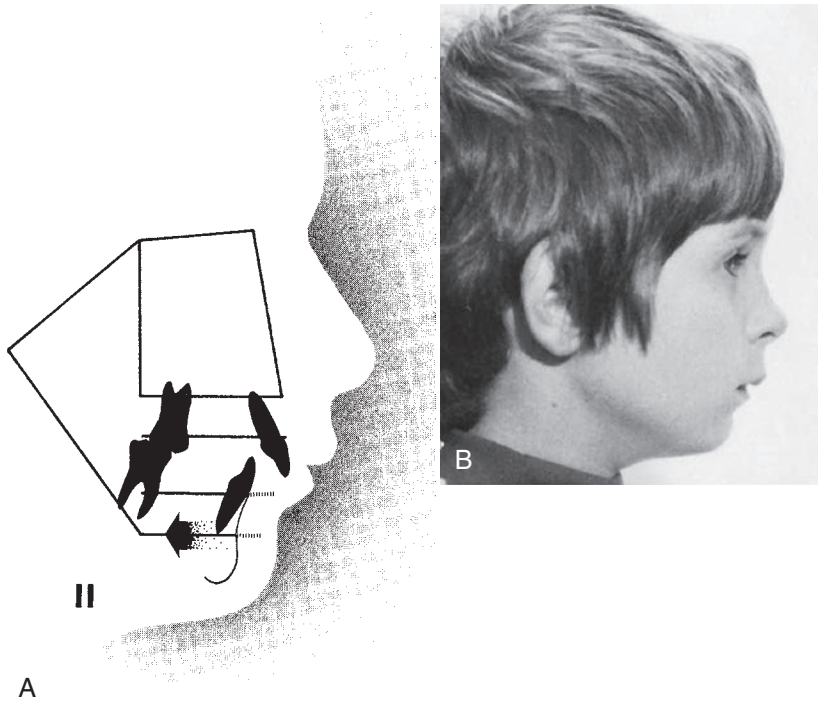


FIGURE 34-17 A, Class II mandibular retrognathism. B, Typical patient profile.

High Angle (Hyperdivergent). The hyperdivergent facial pattern includes a steep mandibular plane and usually is associated with a prognathic maxilla; a retrognathic mandible; a maxillary & mandibular alveolodental protrusion; an open bite relationship of the incisor teeth; an incompetent lip relationship; a long, sloping forehead with a heavy glabella and supraorbital rims; a nose that is long and thin; and a flattened recessive chin exhibiting muscle tension (Fig. 34-18).

Cephalometrically, the mandibular plane to S-N angle is greater than 32 degrees in the Steiner analysis. The Frankfurt mandibular plane angle (FMA) is greater than 25 degrees in the Tweed analysis. Additional features include dolichocephalic head form, leptoprosopic facial form, large gonial angle, short ramus, small coronoid process, antegonial notching, long anterior face height, short posterior face height, long lower face height relative to upper face height, large cranial base angle (which is responsible for a downward and forward position of the nasomaxillary complex), and a downward and backward position of the mandible. This pattern in turn contributes to a steep occlusal plane (OP) and frequently to an exaggerated curve of occlusion. The following are characteristic: microgenia, narrow and long symphysis, high and narrow palate, tooth size–jaw size discrepancy caused by large teeth, impacted third molars, small interincisal angle, overerupted incisors despite an open bite tendency, convex soft tissue profile, weak temporal muscles, restricted pharyngeal space with the tongue forward, mouth breathing, narrow nasal apertures, vertical mandibular growth, and ectomorphic body type with slow skeletal development and poor posture. The extraction of teeth is routinely part of the orthodontic treatment plan in these facial patterns. Extraction is necessary to relieve crowding, assist in the correction of the open bite tendency, and position the mandibular incisors upright. If not too severe, serial extraction may be permissible.

Low Angle (Hypodivergent). The low-angle facial pattern routinely exhibits a low MPA accompanied by a favorable horizontal skeletal relationship or an orthognathic facial pattern, maxillary and mandibular alveolodental retrusion, deep overbite relationship of the incisor teeth, prominent chin, straight or dished-in soft tissue profile, and a shorter nose (Fig. 34-19).

Cephalometrically the mandibular plane to S-N is less than 32 degrees in the Steiner analysis. The FMA is less than 25 degrees in the Tweed analysis. Additional features include a broad brachycephalic head form, a euryprosopic facial form, wide-set eyes, prominent cheekbones, a bulbous forehead, less prominent glabella and supraorbital ridges, a small gonial angle, a broad and long ramus, large coronoid processes, and no antegonial notching on the lower border of the mandible. The anterior face height equals the posterior face height. Compared with the upper face height, the lower face height is small. A small cranial base angle is responsible for an upward and backward position of the nasomaxillary complex and an upward and forward position of the mandible. Macrogenia

FIGURE 34-18 High-angle facial pattern. Facial planes are divergent. Chin profile projection is weak.

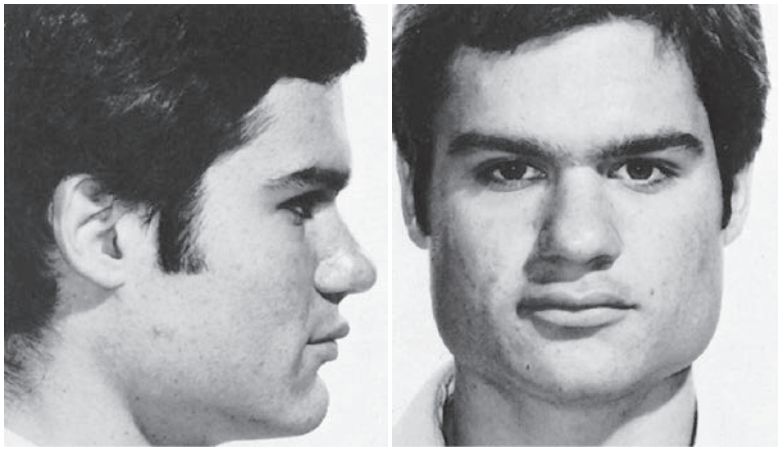


FIGURE 34-19 Low-angle facial pattern. Facial planes are convergent. Chin profile projection is strong.

is common. The palatal vault is flat with a wide dental arch and potentially small teeth. The mandibular incisors may be crowded as a result of a deep overbite, or spaced dentition may result. More abrasion of teeth occurs along with early formation and eruption of teeth; thin lips; deep mentalis sulcus; heavy masseter muscles, with vertical pull; strong temporal muscles; and large pharyngeal space, with a posterior tongue position. Less tongue thrusting occurs when swallowing with low-angle facial patterns. The nasal apertures are large. The mandibular growth is horizontal. The body is endomorphic with advanced skeletal age and upright posture. These patients have less need for extraction; indeed, every effort is made to avoid the extraction of teeth. Arch length can be gained by correcting the deep overbite and positioning the mandibular teeth upright. Serial extraction, in most instances, is contraindicated.

The Teeth

According to the Burlington Orthodontic Research Project,[17] 34% of 3-year-old children enjoy a normal occlusion. By the time they reach 12 years of age, only 11% have a normal intercuspation—a reduction of 23%. Local environmental factors are largely responsible. For example, crowded dentition results from a loss of arch length caused by the premature loss of primary teeth.

Serial extraction is an interceptive procedure designed to assist in the correction of hereditary tooth size–jaw size discrepancies. Because the malocclusions of 66% of 3-year-old children are hereditary (with a significant number of tooth size–jaw size discrepancies), serial extraction is an invaluable adjunct to interceptive treatment. This is especially true in the case of Class I malocclusions and to a lesser extent in the Class II malocclusions.

Class I malocclusions are ideal for serial extraction because the dentition is basically in a favorable relationship and successful treatment is possible with a minimum of mechanotherapy.[18,19] The ideal conditions for serial extraction are (1) a true, relatively severe hereditary tooth size–jaw size discrepancy; (2) a mesial step mixed dentition developing into a Class I permanent relationship; (3) a minimal overjet relationship of the incisor teeth; (4) minimal overbite; and (5) a facial pattern that is orthognathic or with a slight alveolodental protrusion.

Clinical Analysis

Hereditary Crowding. The signs of a true hereditary tooth size–jaw size discrepancy may be outlined as follows:

1. Maxillary and mandibular alveolodental protrusion without interproximal spacing
2. Crowded mandibular incisor teeth
3. A midline displacement of the permanent mandibular incisors, resulting in the premature exfoliation of the primary canine on the crowded side (Fig. 34-20)
4. A midline displacement of the permanent mandibular incisors with the lateral incisors on the crowded side blocked out, usually lingually (Fig. 34-21) but occasionally labially
5. A crescent area of external resorption on the mesial aspect of the roots of the primary canines caused by crowded permanent lateral incisors
6. Bilateral primary mandibular canine exfoliation, resulting in an upright positioning of the permanent mandibular incisors; this in turn increases overjet (Fig. 34-22) and/or the overbite as lower incisors retract
7. A splaying out of the permanent maxillary or mandibular incisor teeth caused by the crowded position of the unerupted canines (Fig. 34-23)
8. Gingival recession on the labial surface of the prominent mandibular incisor
9. A prominent bulging in the maxilla or mandible caused by the crowding of the canines in the unerupted position
10. A discrepancy in the size of the primary and permanent teeth, reducing the leeway space
11. Ectopic eruption of the permanent maxillary first molars, resulting in the premature exfoliation of the primary second molars, which indicates a lack of development in the tuberosity area (see Fig. 34-36)

FIGURE 34-20 Premature exfoliation of one primary mandibular canine with a resulting midline discrepancy.

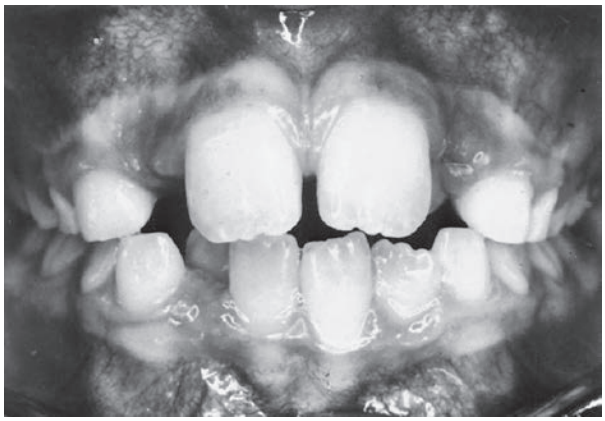


FIGURE 34-21 One permanent mandibular lateral incisor blocked out lingually with a midline discrepancy.

FIGURE 34-22 Bilateral exfoliation of primary mandibular canines resulting in an increase in overjet, with uprighting of lower incisors.

FIGURE 34-23 Splayed maxillary lateral incisors. This is often coupled with mesial eruption of the permanent maxillary cuspids with potential for damage to the lateral incisor roots if appliances are placed.

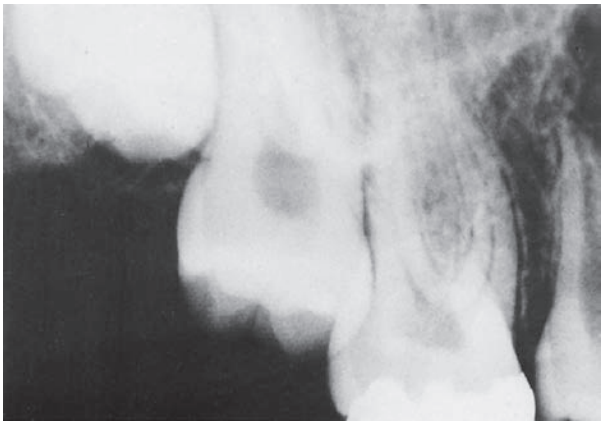


FIGURE 34-24 Palisading of the maxillary molars. The clinician should monitor for impaction of the second molars with potential need for early third molar removal.

12. A vertical palisading of the permanent maxillary first, second, and third molars in the tuberosity area, again indicating a lack of jaw development (Fig. 34-24)
13. Impaction of the permanent mandibular second molars in the absence of treatment

True hereditary tooth size–jaw size discrepancies must be differentiated from crowded dentitions resulting from factors that are more environmental in nature. True hereditary crowding likely will be treated with the aid of extractions and, if discovered early, with serial extraction. However, crowding resulting from environmental factors may be treated without extractions.

Environmental Crowding. Environmental crowding may result under the following conditions:

1. Trauma that affects surrounding hard and soft tissues
2. Iatrogenic malocclusion caused by ill-conceived space management
3. Discrepancy in the size of individual teeth (Fig. 34-25)
4. Discrepancy between mandibular tooth size and maxillary tooth size
5. Aberration in the shape of teeth (i.e., extra cusps)
6. Aberration in the eruptive pattern of the permanent teeth (Fig. 34-26)

FIGURE 34-25 Discrepancy in the size of individual teeth.

FIGURE 34-26 Maxillary canine erupted in the premolar position. This may be related to a transposed canine and ectopic premolar or an absent premolar.

FIGURE 34-27 Rotated maxillary premolar consuming more space than it would in its normal position.

7. Transposition of teeth (see Fig. 34-38)
8. Uneven resorption of primary teeth
9. Rotation of premolar teeth causing loss of space for erupting canines (Fig. 34-27)
10. Ankylosis of primary teeth, most often in first and second primary molars, with adjacent tooth tipping
11. Reduction of arch length caused by interproximal caries in the primary teeth (see Fig. 34-33)

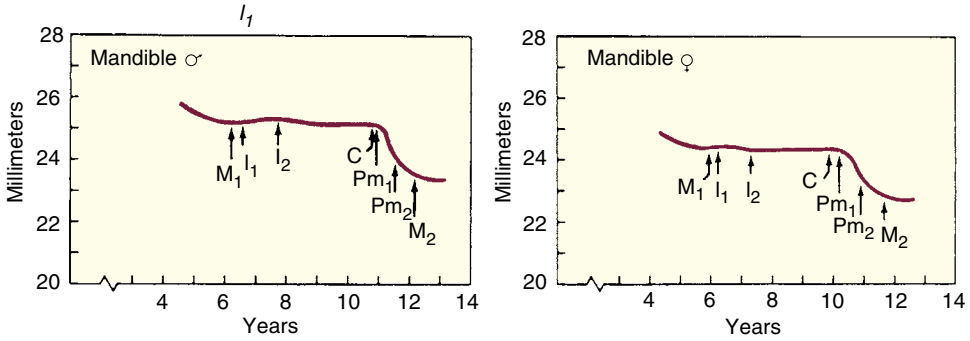


FIGURE 34-28 Reduction in arch length as a result of the moving forward of posterior teeth. M_1, first molar; I_1, central incisor: I_2, lateral incisor; C, canine; Pm_1, first premolar; Pm_2, second premolar; M_2, second molar. (From Moorrees CF, Reed RB. Changes in dental arch dimension expressed on the basis of tooth eruption as a measure of biologic age. *J Dent Res* 1965;44:129.)

12. Premature loss of primary teeth resulting in a reduction of arch length from subsequent drifting of permanent teeth (see Figs. 34-34 to 34-37, *A*)
13. Altered emergence sequence (see Fig. 34-43)
14. Exfoliation sequence of primary teeth (see Fig. 34-37, *A*)
15. Prolonged retention of primary teeth

Because serial extraction is performed during the mixed dentition period, careful examination of the transformation from the primary to the permanent dentition is of utmost importance. A thorough understanding of the eruption sequence and range of timing in tooth eruption is critical. One of the most important daily aspects in mixed dentition patient evaluation is the observation of the tooth eruption pattern and follow-up on alterations in the patient's sequence of eruption. Although there are many permutations on "normal," intercepting individual, specific developing problems results in reduced needs for mechanotherapy.

Dental Development in the Mixed Dentition

At present, overwhelming scientific evidence indicates that the posterior teeth move forward throughout life. This tends to reduce arch length. Moorrees[20] has established that arch length decreases 2 to 3 mm between 10 and 14 years, when the primary molars are being replaced by the permanent premolars (Fig. 34-28). He also has demonstrated that the arch circumference is reduced about 3.5 mm in the mandible of boys and 4.5 mm in girls during the mixed dentition period.

DeKock[21] measured a 10% decrease in arch length for males and 9% for females over a period of 10 years beyond the period of the mixed dentition, from 12 years to 26 years (Fig. 34-29). Brodie[22] has observed that in newborn infants, the tongue tends to fill the oral cavity and often encroaches on the alveolar ridge area. As a consequence of the more rapid anterior growth of the jaws in the postnatal period, the tongue lags behind and comes to occupy a more posterior position in the oral cavity. This is consistent with the upright positioning of the incisors that occurs in many adolescents as noted by Enlow,[23] Bjork[24] (Fig. 34-30), Tweed,[11] and others.

These findings suggest that as facial growth continues in an anterior direction into adulthood, it thrusts the mandible into the facial musculature, which produces a posterior force vector on the crowns of the incisors. Thus, arch length decreases from the anterior and posterior sides. To repeat, serial extraction is

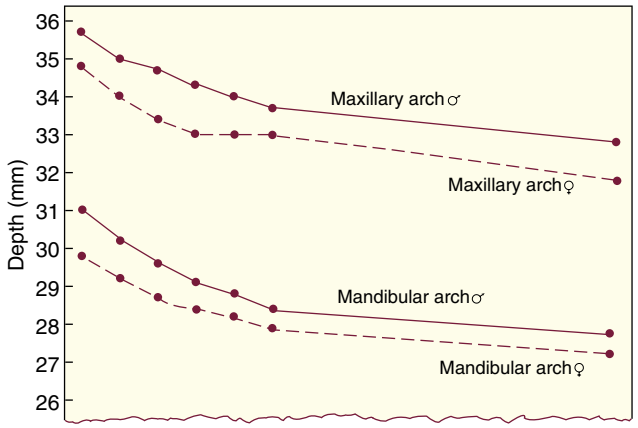


FIGURE 34-29 Reduction in arch depth. (From DeKock WH. Dental arch depth and width studies longitudinally from 12 years of age to adulthood. *Am J Orthod* 1972;62:56.)

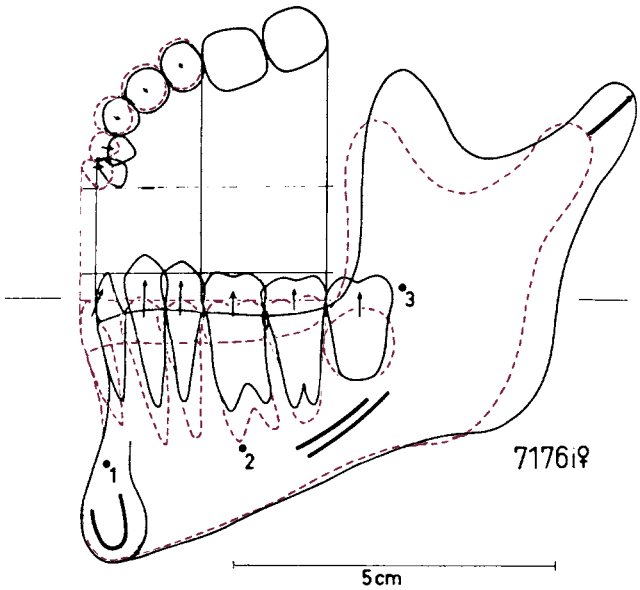


FIGURE 34-30 Upright positioning of incisors with forward growth. (From Bjork A. Variations in the growth pattern of the human mandible: longitudinal radiographic study by the implant method. *J Dent Res* 1963;42:400.)

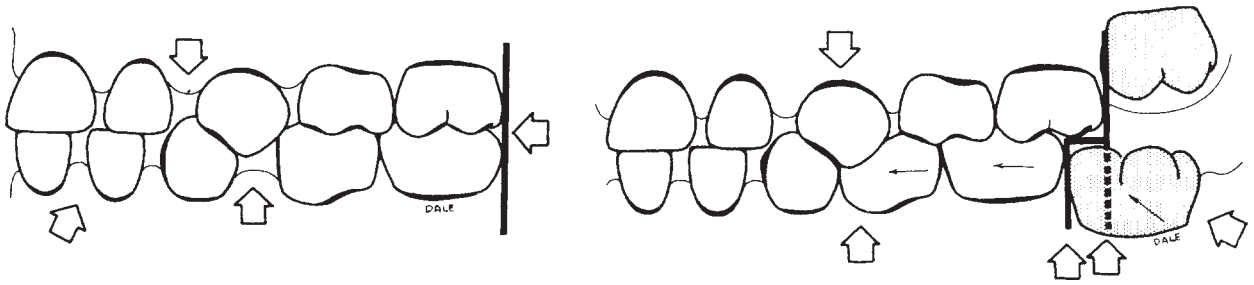


FIGURE 34-31 Early mesial shift. Note the use of normal developmental spaces in the mixed dentition to bias the occlusal development for a mesial shift of the mandibular teeth versus the maxillary teeth. (Adapted from Baume LJ. Physiological tooth migration and its significance for the development of occlusion. II. The biogenesis of the accessional dentition. *J Dent Res* 1950;29:331.)

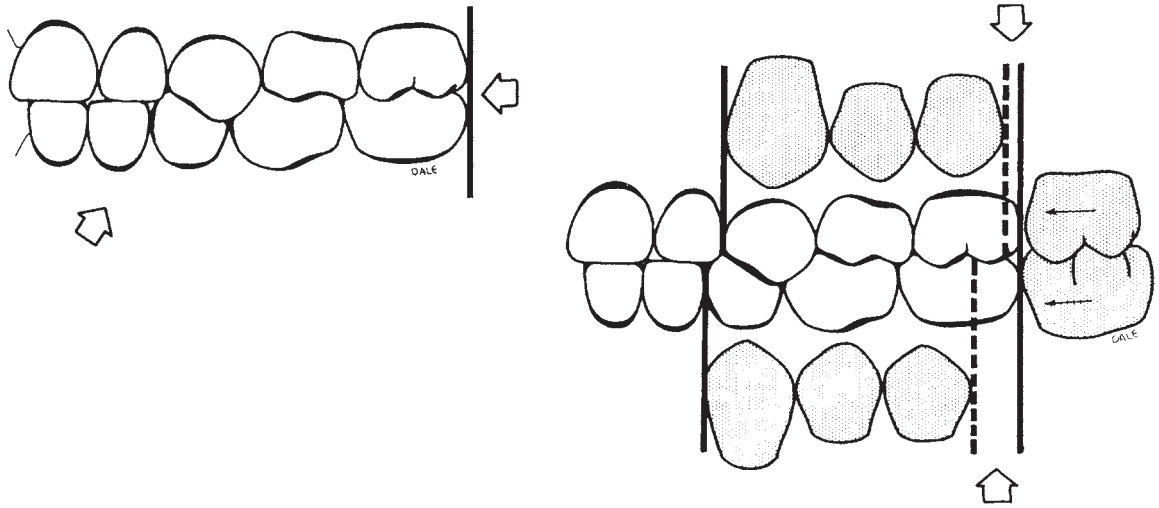


FIGURE 34-32 Late mesial shift. This pattern is seen when there is minimal developmental spacing in the primary and early mixed dentition. (Adapted from Baume LJ. Physiological tooth migration and its significance for the development of occlusion. II. The biogenesis of the accessional dentition. *J Dent Res* 1950;29:331.)

based on the fact that arch length does not increase. If crowding is evident at 8 years, it will not improve with further growth and development.

First Molars. Several situations can exist for the permanent first molars. In patients with a spaced primary dentition and a straight terminal plane relationship of the primary molars, the permanent mandibular first molars emerge at about 6 years, move the primary molars mesially, close the space distal to the primary canines, convert the straight terminal plane to a mesial step relationship, reduce arch length in the mandibular dentition, and allow the permanent maxillary molars to emerge into a Class I relationship. This has been referred to as the *early mesial shift* (Fig. 34-31).

In patients with a closed primary dentition and a straight terminal plane, the permanent maxillary and mandibular first molars emerge into a cusp-to-cusp relationship simply because no spaces exist. At about 11 years, the primary mandibular second molars are exfoliated, and the permanent mandibular first molars migrate mesially into the excess leeway space provided by the differences in mesiodistal dimensions of the primary second molars and the permanent second premolar teeth. Again, this reduces arch length, converts the straight terminal plane to a mesial step, and provides for a Class I relationship of the permanent first molars. This shift has been referred to as the *late mesial shift* (Figs. 34-28 and 34-32). If the permanent maxillary first molars emerge before the mandibular molars, just the reverse of the early mesial shift—an abnormal Class II relationship—will occur, and a reduction in the maxillary arch length will result.

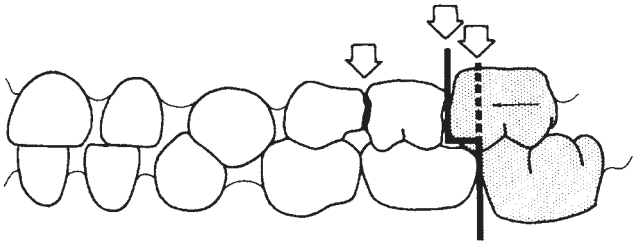


FIGURE 34-33 Reduction in arch length as a result of caries.

If extensive interproximal caries is allowed to develop in the maxilla, a similar situation will occur: a reduction of arch length causing crowding (Fig. 34-33). If the caries is so extensive that extraction of the primary maxillary second

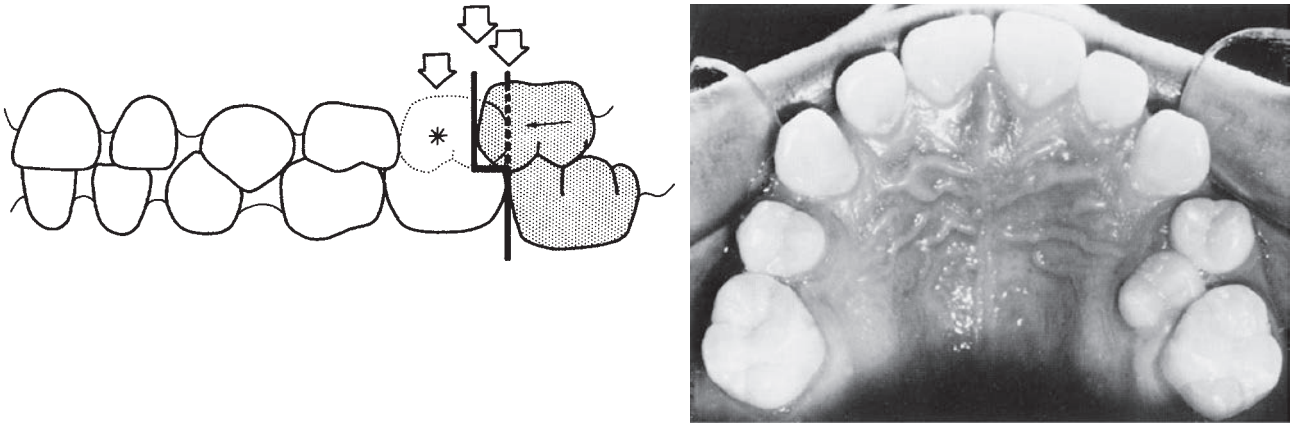


FIGURE 34-34 Reduction in arch length as a result of premature loss of primary maxillary second molars.

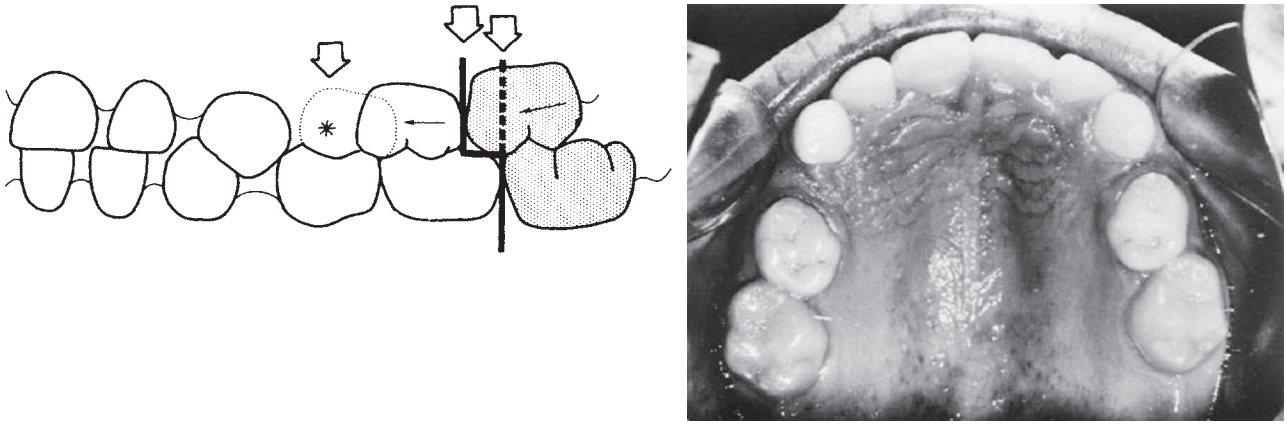


FIGURE 34-35 Reduction in arch length as a result of premature loss of the primary maxillary first molars.

molars is necessary, again, crowding will result (Fig. 34-34). Similarly, and this is contrary to popular belief, premature loss of the primary maxillary first molars will cause crowding (Fig. 34-35).

Ectopic eruption of the permanent maxillary first molars, resulting in premature exfoliation of the primary second molars and loss of arch length, indicates a lack of development of the tuberosity. This results in not only crowding but also a Class II molar relationship (Fig. 34-36).

If the exfoliation sequence of the primary second molars is reversed and the maxillary molar is lost before the mandibular, a Class II relationship of the permanent first molars will result. Again, arch length will be reduced, and crowding will occur in the maxilla (Fig. 34-37, *A*).

However, if the primary mandibular second molar is lost far too early, the mandibular arch length will be reduced to such an extent that the normal leeway will be exceeded and crowding will occur (Fig. 34-37, *B*).

The normal leeway, according to Lo and Moyers,[25] is 2.6 mm (1.3 mm on each side) in the maxilla and 6.2 mm (3.1 mm on each side) in the mandible. However, this leeway varies considerably and should be measured on each patient. It is possible for a discrepancy to exist between the size of the primary teeth and the size of the permanent teeth to such an extent that no positive leeway is available. A negative leeway may exist. According

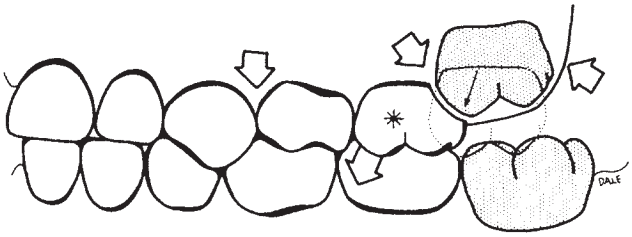


FIGURE 34-36 Reduction in arch length as a result of ectopic eruption of the permanent maxillary first molars.

to Horowitz and Hixon,[26] correlations in size between a single primary tooth and its successor range from $r = 0.2$ to $r = 0.6$. This means that anywhere from 4% to 36% of the successors are favorably correlated in size to the primary teeth. In other words, if the primary tooth is small, its successor will be small. According to Arita and Iwagaki,[27] Hixon and Oldfather,[28] Lewis and Lehman,[29] Moorrees and Chadha,[30] and Jensen et al.,[31] the size correlation between all the primary teeth and their successors is approximately $r = 0.5$. Therefore, 25% of the time, a positive relationship exists. That is not too favorable for optimal occlusion.

Incisors. Several situations can exist also for the incisors. Ideally, the primary spacing of the spaced primary dentition will be sufficient, together with other factors, to allow for the

accommodation and favorable alignment of the succedaneous permanent incisors (Fig. 34-38).

In primary dentitions in which there is no interdental spacing, the permanent mandibular lateral incisors emerge, and the primary mandibular canines are moved laterally. Thus, a space is created that enables the permanent maxillary lateral incisors to emerge into a favorable alignment. This is referred to as *secondary spacing* and was first described by Baume (Fig. 34-39).[32] Secondary spacing also occurs when the permanent mandibular central incisors are emerging.

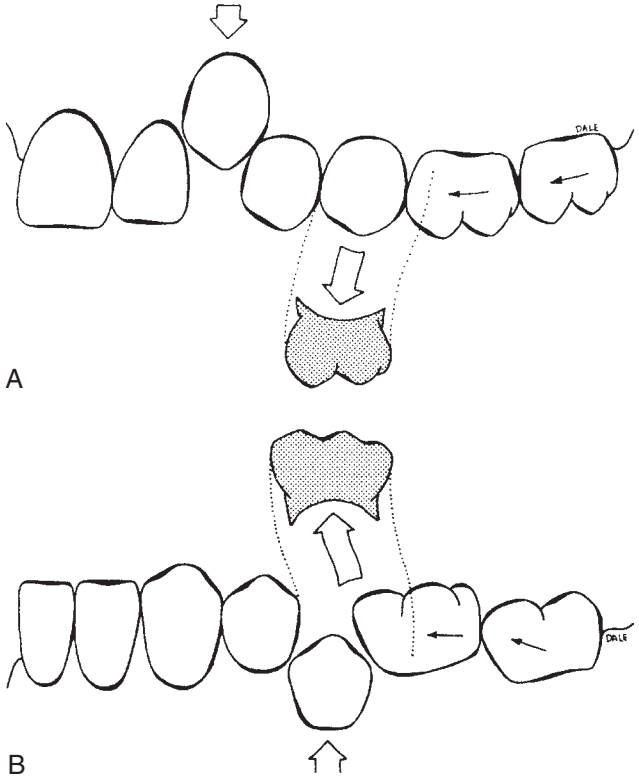


FIGURE 34-37 A, Reduction in arch length as a result of premature exfoliation of the primary maxillary second molar. B, Reduction in arch length as a result of premature loss of the primary maxillary or mandibular second molar.

Both of these observations have been substantiated by the findings of Moorrees[20] that show an increase in intercanine width during the period of incisor emergence (Fig. 34-40). Moorrees also has demonstrated an increase in arch circumference in the maxilla of boys of 1.5 mm and of girls of 0.5 mm during this period of development.[33]

If the primary canines are reduced in size or extracted when this natural phenomenon is occurring, an increase in intercanine distance and secondary spacing may not occur. Thus, borderline discrepancies may be converted from a nonextraction treatment to extraction.

Research conducted by Moorrees and Chadha[34] has revealed an increase in the crowding of maxillary and mandibular incisors when they are emerging into the oral cavity. However, 2 mm of crowding in the incisor segment in the mandible of boys will recover to no crowding by 8 years on the average. Girls recover to about 1 mm of crowding. The maxillary dentitions of boys and girls do not exhibit the same tendency to crowding. However, during the eruption of the incisors, 2 to 3 mm of spacing is reduced to 0 (Fig. 34-41). This is a significant finding because it tells the clinician not to be alarmed with a slight amount of crowding in the early stages of emergence of the permanent incisors. The reduction in size, or extraction, of the primary canines should be deferred. In fact, the patient may not require extraction at all.

Intercanine distance increases more in the maxilla and in closed dentitions, which lack primary tooth interdental spaces. A true hereditary tooth size–jaw size discrepancy is characterized by permanent mandibular lateral incisors and a premature exfoliation of the primary canines (Fig. 34-42).

Mayne[35] described a concept he termed *incisor liability*. In his discussion, he outlined the way in which incisor liability could be used clinically to determine anterior crowding. He described the following principal variables:

1. The four permanent maxillary incisor teeth are, on the average, 7.6 mm larger than the primary incisors.[36] The four permanent mandibular incisors are 6.0 mm larger. This size differential is the incisor liability. The liability varies greatly from individual to individual, and for this reason, the patient's own tooth measurements should be used in the analysis. A favorable incisor liability exists when the spacing of the

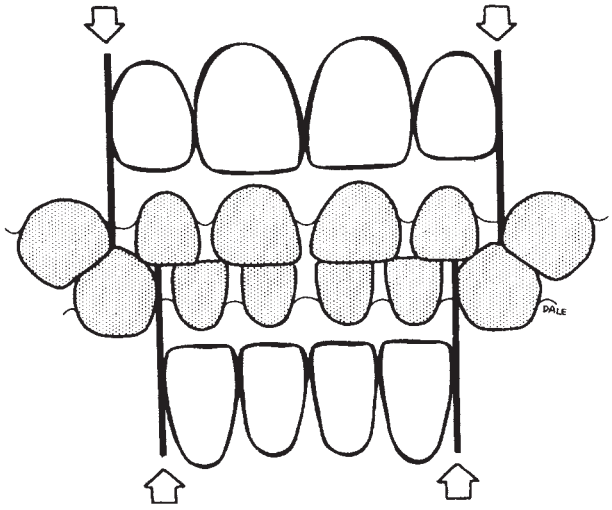


FIGURE 34-38 Primary spacing. Allows larger incisors to erupt with reduced chance of crowding.

primary anterior teeth is sufficient to allow for the eruption of the permanent incisors without any crowding (see Fig. 34-38).
2. A more precarious incisor liability situation exists when no primary spacing is present in a closed primary dentition. Then the individual must rely on the development of secondary spacing to create sufficient space for the permanent incisors to emerge without crowding (see Fig. 34-39).
3. An impossible situation exists when the incisor liability is of such magnitude that growth and development will never be able to meet the space demands required by the permanent incisors. Such patients are doomed to severe crowding and irregularity from the outset (see Fig. 34-42).
4. In the primary dentition, the interdental spacing may range between 0 and 10 mm in the maxilla, with an average of 4 mm, and between 0 and 6 mm in the mandible, with a range of 3 mm.
5. During the period of permanent incisor eruption, notable amounts of intercanine arch width development occur in the maxillary and mandibular dentition. In the mandible, the increase occurs between 6 and 9 years for boys and between 6 and 8 years for girls. In the maxilla, it increases longer, to 16 years in boys and 12 years in girls (see Fig. 34-40).
6. After 10 years of age, little intercanine arch width change is expected in the mandible of boys or girls. The average increase in the mandibular dentition of boys and girls is about 3 mm; in the maxilla, it is about 4.5 mm (see Fig. 34-40).
7. The permanent incisor teeth erupt slightly labial to the arch position of the primary incisors and for a time at least are more procumbent.[35]

Incisor crowding may be assessed by using the variables just described. First, a favorable situation, described previously,

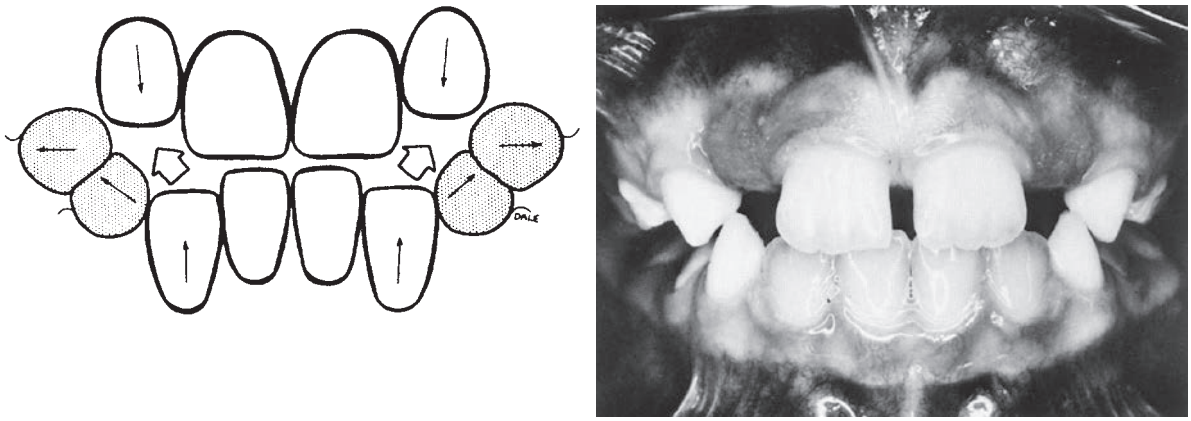


FIGURE 34-39 Secondary spacing occurring when the permanent mandibular lateral incisors are emerging.

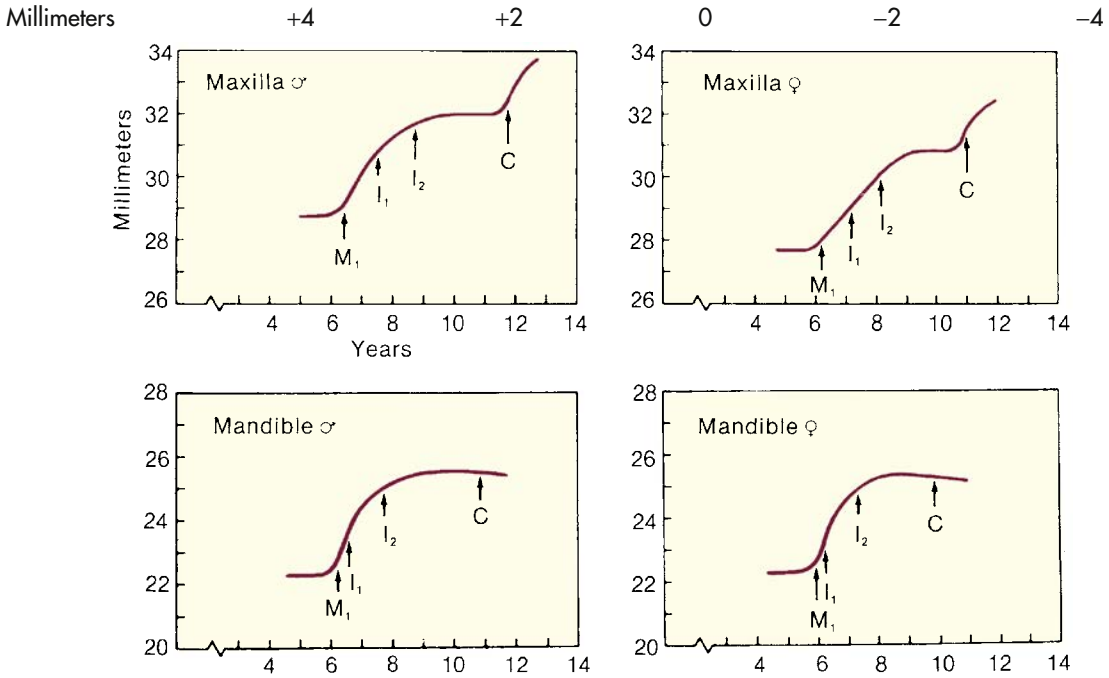


FIGURE 34-40 Average intercanine distance. *M1*, First molar; *I1*, central incisor; *I2*, lateral incisor; *C*, canine. (From Moorrees CF, Reed RB. Changes in dental arch dimension expressed on the basis of tooth eruption as a measure of biologic age. *J Dent Res* 1965;44:129.)

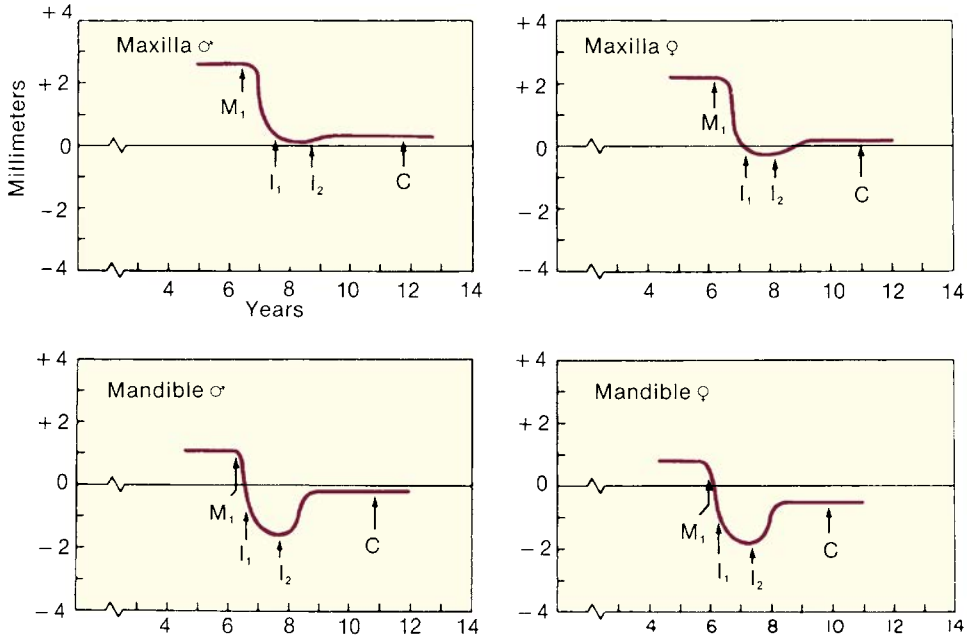


FIGURE 34-41 Available space in the incisor segment. (From Moorrees CF, Chadha IM. Available space for the incisors during dental development: a growth study based on physiological age. *Angle Orthod* 1963;33:44.)

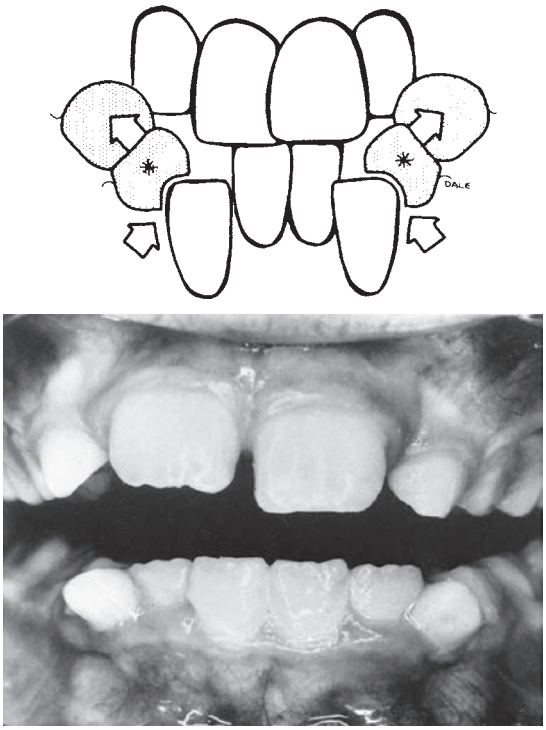


FIGURE 34-42 Lingual eruption of the permanent mandibular lateral incisors as a symptom of crowding and its effect on secondary spacing. Asterisks in drawing demonstrate inadequate arch length for erupting canines.

TABLE 34-1 Incisor Liability

Situation	Maxilla (mm)	Mandible (mm)
Favorable: Primary Spacing		
Incisor liability	−6.2	−4.3
Interdental spacing	+4.0	+3.0
Incisor position	+2.2	+1.3
—	6.2	4.3
Amount of crowding	—	—
Precarious: Secondary Spacing		
Incisor liability	−9.2	−7.3
Interdental spacing	+4.0	+3.0
Intercanine arch width	+3.0	+3.0
Incisor position	+2.2	+1.3
—	9.2	7.3
Amount of crowding	—	—
Impossible: Crowding Exceeds Compensations		
Incisor	−14.2	−12.3
Interdental spacing	+4.0	+3.0
Intercanine arch width	+3.0	+3.0
Incisor position	+2.2	+1.3
—	9.2	7.3
Amount of crowding	5.0	5.0

exists when sufficient primary spacing is present in the spaced primary dentition to allow for the eruption of the permanent incisors without crowding (Table 34-1; see also Fig. 34-38).

Second, a precarious situation exists with a primary dentition with no anterior developmental spaces. A substantial increase in intercanine width is necessary to provide secondary spacing so that the permanent incisors erupt without appreciable crowding (see Fig. 34-39 and Table 34-1). In these patients, the practitioner avoids interproximal reduction or extraction of primary canines.

Finally, an impossible situation exists with a true hereditary tooth size–jaw size discrepancy and an incisor liability that cannot be compensated by interdental spacing, an increase in intercanine arch width, or labial positioning of the permanent incisors (see Fig. 34-42 and Table 34-1). Serial extraction can be beneficial for these individuals.

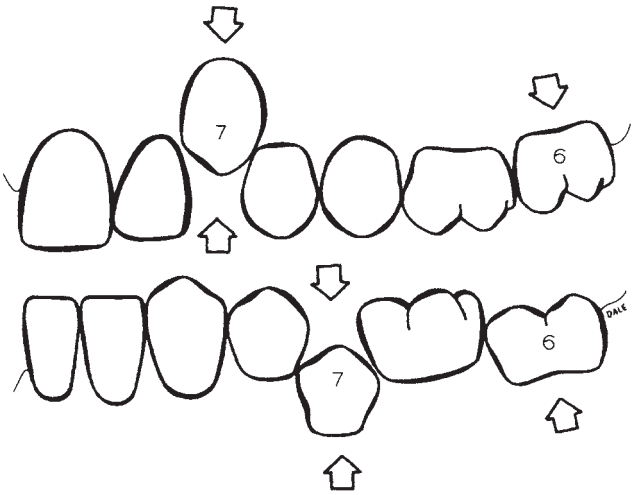


FIGURE 34-43 Unfavorable emergence sequence. The permanent second molars are emerging early, blocking the space for maxillary canines and mandibular second premolars.

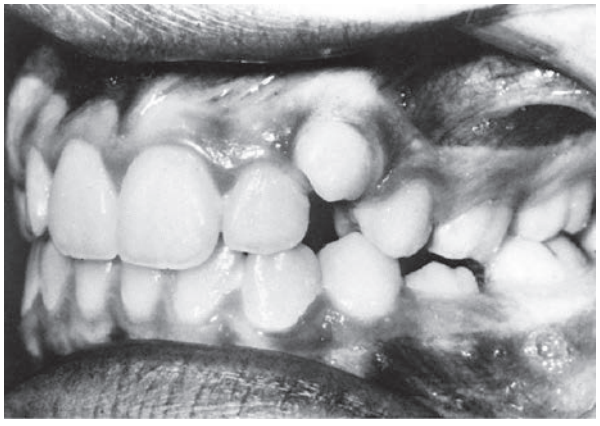


FIGURE 34-44 Prolonged retention of the primary maxillary second molar causing crowding of the permanent canine.

Canines, Premolars, and Second Molars. The most frequently occurring sequence of eruption in the maxilla is as follows: permanent first molar, central incisor, lateral incisor, first premolar, second premolar, canine, second molar. The most frequently occurring sequence in the mandible is permanent first molar, central incisor, lateral incisor, canine, first premolar, second premolar, second molar. According to Lo and Moyers,[25] this combination produces the highest incidence of favorable occlusion. More girls than boys have a favorable combination.

FIGURE 34-45 Prolonged retention of the mandibular second deciduous molar, causing crowding of the first premolar.

An unfavorable sequence can produce crowding. For instance, if the second molars erupt early, they may affect the canines in the maxilla and the second premolars in the mandible (Fig. 34-43). Maxillary second molars erupt ahead of mandibular molars in 89.11% of Class II patients, but in only 56.5% do maxillary first molars erupt ahead of their mandibular counterparts.[25] Therefore, second molars are more important than first molars in the development of a Class II relationship.

Early exfoliation of primary teeth can disrupt the alignment of the dentition, reducing arch length in the maxilla (see Figs. 34-34, 34-35, and 34-37) and mandible (see Fig. 34-37, *B*).

Prolonged retention of primary teeth can cause crowding of the permanent teeth in the maxilla (Fig. 34-44) or mandible (Fig. 34-45).

Clinically, the primary second molars appear to resist mesial migration to some extent. Rarely does the orthodontist observe primary mandibular second molars tipped mesially after the premature loss of primary first molars. However, the permanent mandibular first molars are observed to have an exaggerated mesial inclination after the premature loss of the primary second molars. The relative position of the unerupted premolars is crucial in this situation. When the primary mandibular second molars are lost prematurely, the permanent second premolars are often deep in the alveolar bone. This may encourage the permanent first molar to tip forward. When the primary mandibular first molar is lost prematurely, the permanent first premolar (which is scheduled to emerge before the second premolar) is not so deeply embedded in bone. Therefore, the tendency for tipping of the primary second molar is not as great. The unerupted first premolar tends to support the primary second molar.

Contrary to popular belief, arch length can be reduced, and space can be lost after premature exfoliation or extraction of the primary first molar (see Fig. 34-35). This is especially true in the mandible, where the formation of a knife-edge ridge of alveolar bone obstructs and retards the emergence of the underlying first premolar.

The premature loss of primary molars immediately leads one to consider space maintenance. However, the use of space maintainers depends on a thorough diagnosis and may be modified with subsequent treatment planning. If the patient has a normal dentition or the treatment plan does not include the extraction of permanent teeth, a space maintainer may be placed. Indeed, in the borderline situation, maintenance of the "E" space may be all that is indicated to maintain a nonextraction treatment approach (See also Chapter 16).

In the maxilla, about 90% of the time, the first premolar emerges ahead of the canine. This is favorable for serial extraction and maintenance of the overbite because the maxillary incisors are not held forward. About 10% of the time, the sequence is unfavorable for serial extraction and the overbite.

In the mandible, about 80% of the time, the canine emerges ahead of the first premolar.[25] This eruption sequence decreases overbite because it maintains the mandibular incisors in a forward position.[37] However, the sequence is not favorable for serial extraction. About 20% of the time, the sequence is favorable for serial extraction but not for maintenance of the overbite; that is, the first premolars are ahead of the canines.

Serial extraction is beneficial to prevent resorption of the roots of the maxillary lateral incisors by impacted canines. When the primary canine is extracted in serial extraction, the space between the lateral incisor and the primary first molar is reduced substantially. However, when the primary first molar is extracted at the proper time, the first premolar erupts, allowing the unerupted permanent canine to move away from the root of the lateral incisor. On extraction of the first premolar, the canine then erupts into place without difficulty and without danger to the lateral incisor. Certain conditions exist, which will be discussed later, under which primary first molars are extracted before the primary canines to allow the premolars to erupt. This extraction may even be done, in the presence of concern, in nonextraction malocclusions. To their knowledge, the authors have not had a lateral incisor root resorbed by an impacted canine in 33 years of practice using serial extraction procedures.

Total Space Analysis

The mixed dentition analyses historically were dentally oriented. Unfortunately, even today, tooth size–jaw size discrepancies diagnosed in the mixed dentition period still are evaluated by dental-oriented analyses. These analyses will be referred to as conventional methods. Their aim is to evaluate, as accurately as possible, future crowding in the permanent dentition using a prediction of the mesiodistal width of the permanent mandibular canines and premolars. The value obtained is added to the already known measurement of the permanent mandibular incisors. This represents space required. The space required is subtracted from the arch circumference of space available. If the result is significantly negative, future crowding can be predicted.

Tweed,[38] studying the relationship between the mandibular incisors and the mandibular plane, found that if the teeth are not in a stable relationship with the basal bone after treatment, the result may relapse. In view of this, the dental-oriented mixed dentition analysis alone is not adequate. A facial-oriented analysis, incorporating the relations of the incisor teeth to the basal bone, is preferable. This analysis will be described as the Tweed method.

Furthermore, the various mixed dentition analyses demonstrate the discrepancy only and do not indicate the exact area where the discrepancy occurs. In many instances, the problem is confined to one specific area. Because one can direct treatment specifically to one area, knowledge of the area affected is desirable. The total space analysis provides this precise information.[39]

Garcia-Hernandez and Dale[40] completed a study of 60 Class I mixed dentition patients in the authors' practice. One objective was to compare the conventional method, the Tweed method, and the total space analysis method in the diagnosis of tooth size–jaw size discrepancies before serial extraction. Sixty individuals (30 boys and 30 girls) with a mean chronologic age of 8 years, 4 months (range, 7 years 7, months to 11 years, 3 months) were selected from the records of the practice.

Conventional Method

The "conventional" calculations were done as follows. For space required, the four mandibular incisors were measured at their greatest mesiodistal crown diameter by means of a sliding Boley gauge with pointed beaks. All measurements were made with the gauge parallel to the incisal edges of the teeth, and all readings were to the nearest 0.1 mm (Fig. 34-46). The values for unerupted canines and premolars were obtained by measuring their greatest mesiodistal crown diameter or their images on the periapical radiograph (Fig. 34-47).

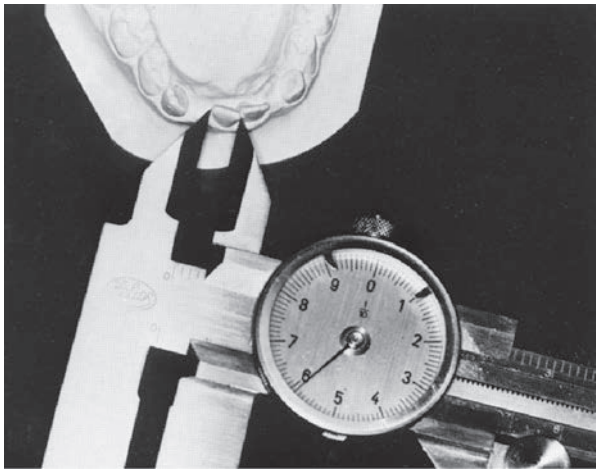


FIGURE 34-46 Measurement of space required on study models with a Boley gauge. Digital models allow this to be done efficiently with a variety of space analyses and size discrepancy evaluations.

FIGURE 34-47 Measurement of space required on the periapical radiograph with a Boley gauge.

To reduce the radiographic enlargement, the formula recommended by Huckaba[41] was used in all radiographic measurements:

$$X = (y)\left(x^1\right)/\left(y^1\right)$$

where X is the estimated size of the permanent tooth, x^1 is the radiographic size of the permanent teeth, y is the size of the primary mandibular second molar on the cast (Fig. 34-48), and y^1 is the radiographic size of the primary molar.

The values obtained for the mandibular incisors on the cast and those for the canines and premolars on the radiograph were added to provide the space required.

The space available was obtained by extending a brass wire from the mesiobuccal of the first permanent molar on one side to the mesiobuccal of the molar on the opposite side, passing through the buccal cusps and incisal edges of the remaining teeth (Fig. 34-49). The wire was straightened carefully and measured with a pointed Boley gauge to the nearest 0.1 mm.

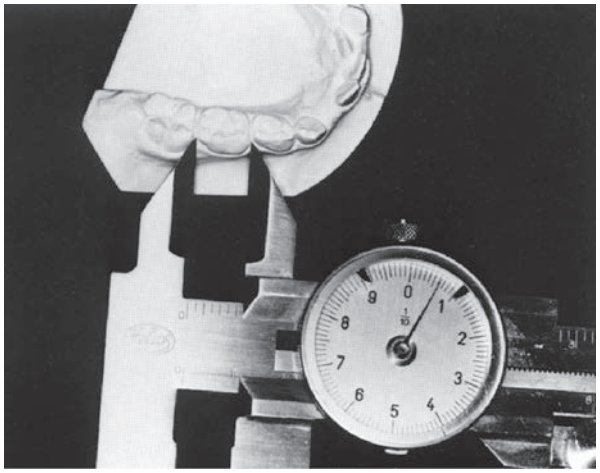


FIGURE 34-48 Measurement of the primary second molar on the study model with a Boley gauge to aid in assessing a radiographic enlargement.

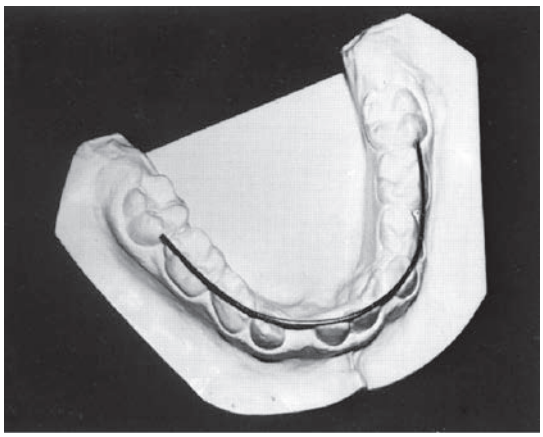


FIGURE 34-49 Measurement of space available on the study model with a brass ligature wire. Digital model analysis makes this manual measurement step unnecessary.

The difference in the values obtained for space required and space available was the amount of the discrepancy.

Tweed Method

The values for space required and space available were obtained as required for the conventional method (see Figs. 34-46 to 34-49). An assessment of the relations between the axial inclination of the mandibular incisors and the basal bone was made on a tracing of the lateral cephalogram. The amount of alveolodental protrusion or retrusion was assessed and incorporated into the mixed dentition analysis (Fig. 34-50).

Tweed Foundation research has established the following relationships:

- When the FMA is between 21 and 29 degrees, the Frankfort mandibular incisor axis angle (FMIA) should be 68 degrees.
- When the FMA is 30 degrees or greater, the FMIA should be 65 degrees.
- When the FMA is 20 degrees or less, the incisor mandibular plane angle (IMPA) should not exceed 92 degrees.

If for a specific FMA (30 degrees) the FMIA (49 degrees) did not correspond, an objective line was traced to form the required FMIA (65 degrees). Then the distance between this objective line and the line that passed through the actual axial inclination of the mandibular incisors was measured on the OP with pointed calipers to the nearest 0.1 mm (6 mm). This figure was multiplied by 2 to include right and left sides (12 mm). The total was the cephalometric correction, which then was added to the difference between space required in space available to yield the total discrepancy (see Fig. 34-50).

Total Space Analysis

This method was divided into three areas—anterior, middle, and posterior—and the resulting values for each area were added together to yield the final deficit.

Anterior Area. For the anterior area, the calculation of the difference between space required and space available was done as before. However, the space required included, in addition to tooth measurement and cephalometric correction, soft tissue modification.

Tooth Measurement. Measurements of mandibular incisor widths on the cast were added to the values obtained from the radiographic measurements of the canines. Both measurements were made according to a previously described technique (see Figs. 34-46 to 34-48).

Cephalometric Correction. The cephalometric correction was calculated as for the Tweed method. However, instead of measurements being made of the distance (in millimeters) on the OP between the objective line and the line indicating the true axial inclination of the mandibular incisors, the actual FMIA (in degrees) was subtracted from the proposed angle, and the difference (in degrees) was multiplied by a constant (0.8) to give the difference in millimeters (Fig. 34-51).

Soft Tissue Modification. Whereas the Tweed method added the anterior skeletal dental (hard tissue cephalometrics) relationships to the dental-oriented mixed dentition analysis, this method also adds a consideration of the soft tissue profile. Thus, the teeth, jaws, and soft tissue are involved in the assessment.

The soft tissue modification was derived by measuring the Z angle of Merrifield[10,39] and adding the cephalometric correction (in degrees) to it. If the corrected Z angle was greater than 80 degrees, the mandibular incisor inclination was modified

		Objective
Frankfort–mandibular angle (FMA)	30°	30°
Incisor–mandibular plane angle (IMPA)	101°	85°
Frankfort–mandibular incisor angle (FMIA)	49°	65°
A point–nasion–B point angle (A-N-B)	6°	3°
Occlusal plane (OP)	13°	9.3°
Z angle	68°	80 ± 5°
Chronologic age, 7 yr 6 mo		
Dental age, 6 yr 6 mo		

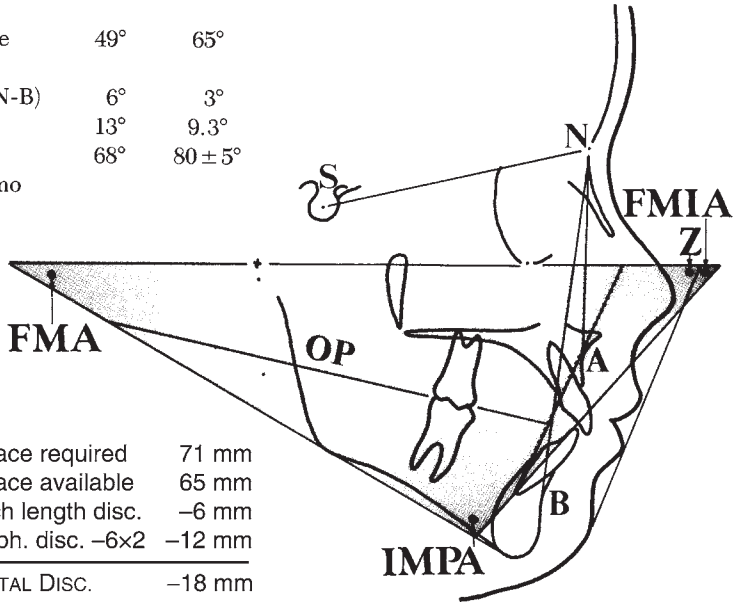


FIGURE 34-50 Tweed method. Cephalometric (ceph.) correction to factor dental protrusion into space analysis. In this patient, according to Tweed goals, the lower incisal edge is 6 mm ahead of where it should be. If the incisor is to be uprighted to the Tweed goal, 6 mm per side of space (12 mm total) are required.

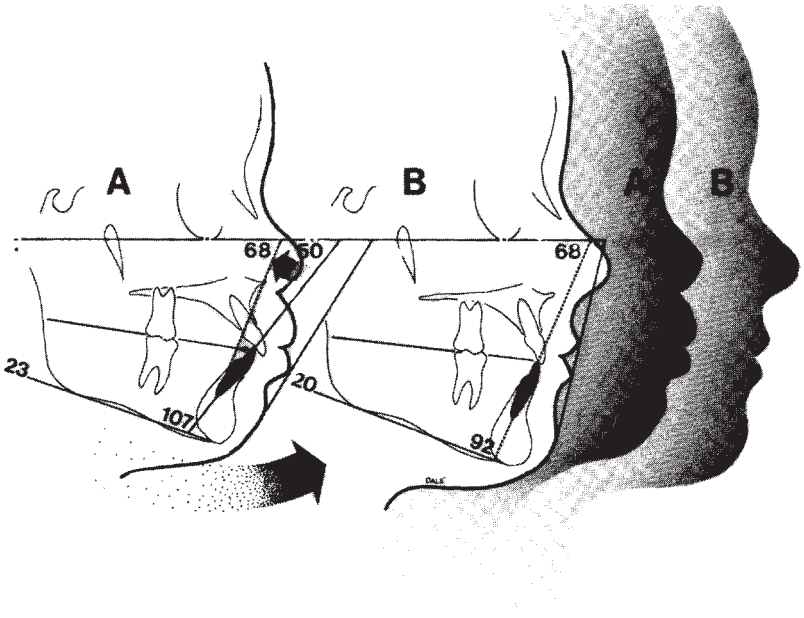


FIGURE 34-51 Total space analysis. Anterior arch cephalometric correction. For patient ML, using FMIA actual minus goal provides an 18 degree difference. Multiplied by .8 would indicate 14.4 mm of needed arch length to achieve the Tweed goal for lower incisor angulation.

as necessary (up to an IMPA of about 92 degrees). If the corrected angle was less than 75 degrees, additional upright positioning of the mandibular incisors was necessary. Upper lip thickness was measured from the vermilion border of the lip to the greatest curvature of the labial surface of the central incisor. Total chin thickness was measured from the soft tissue chin to the N-B line.

If lip thickness was greater than chin thickness, the difference (in millimeters) was determined, multiplied by 2, and added to the space required. If it was less than or equal to chin thickness, no soft tissue modification was necessary.

Figure 34-52 portrays patient M.L., described in Figure 34-51, who had a typical Class I malocclusion with an alveolodental protrusion and an incompetent lip relationship (see

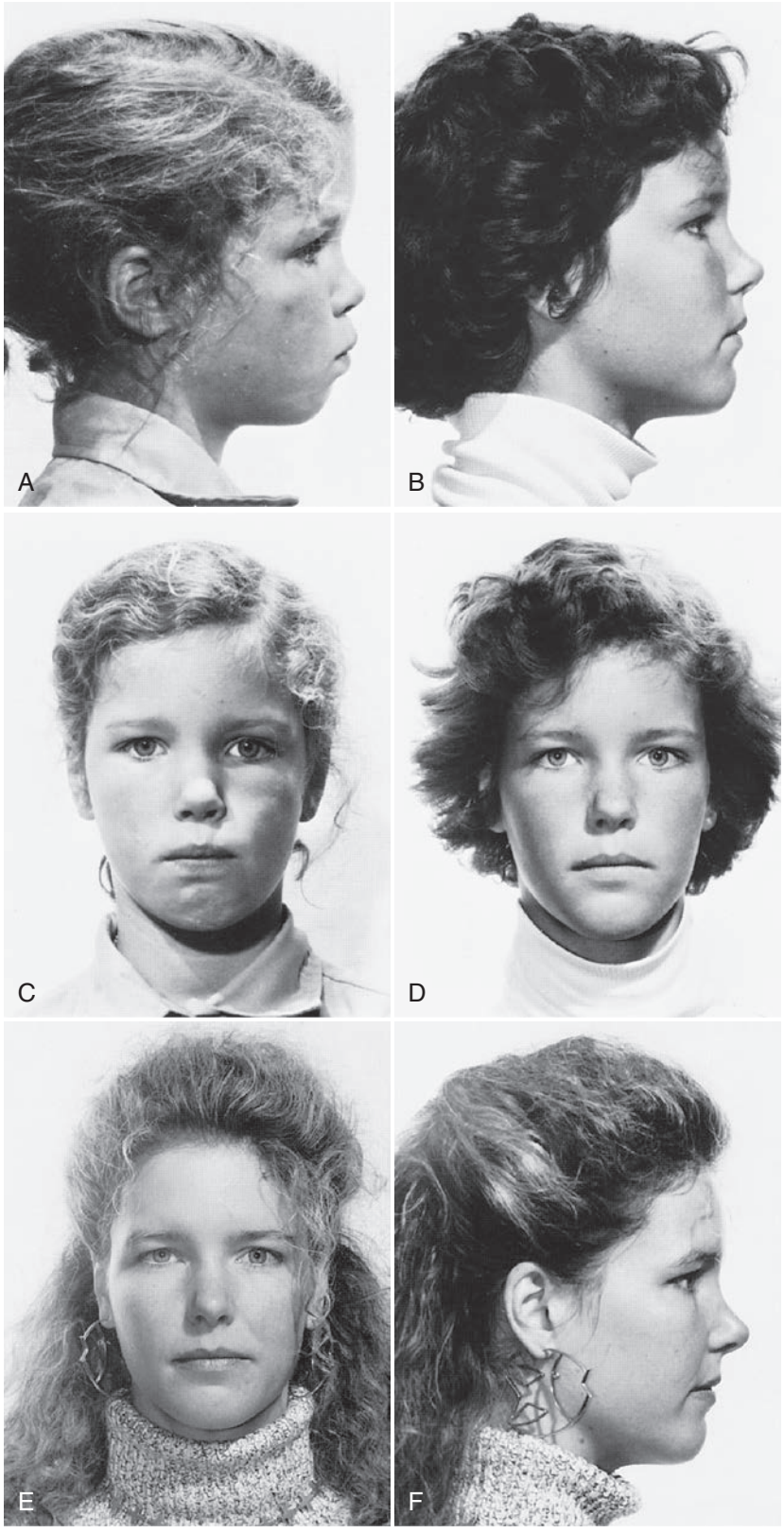


FIGURE 34-52 Patient M.L. **A** and **C,** Before treatment. **B** and **D,** After treatment. **E** and **F,** Balance and harmony persist as an adult 15 years after orthodontic treatment.

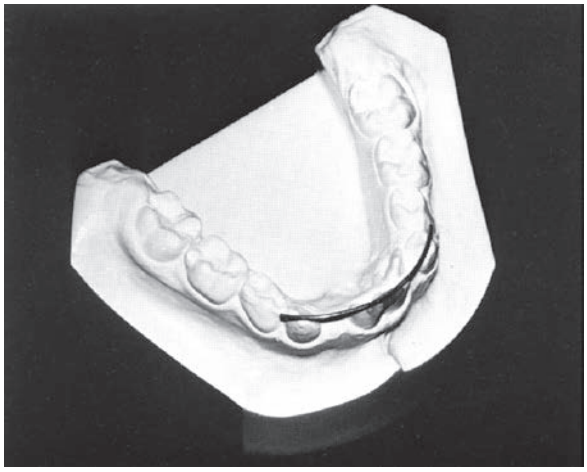


FIGURE 34-53 Total space analysis. Measurement of space available in the anterior area.

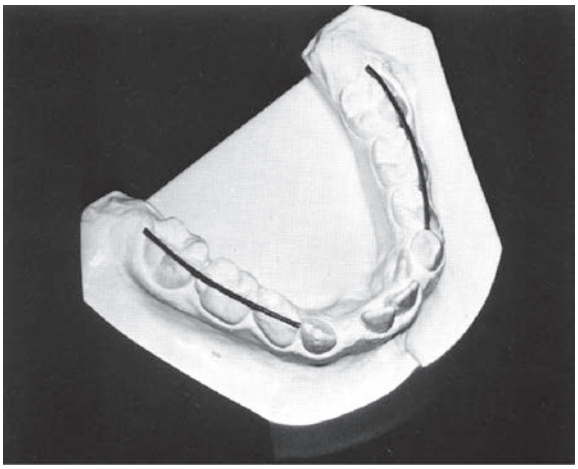


FIGURE 34-55 Total space analysis. Measurement of space available in the middle area.

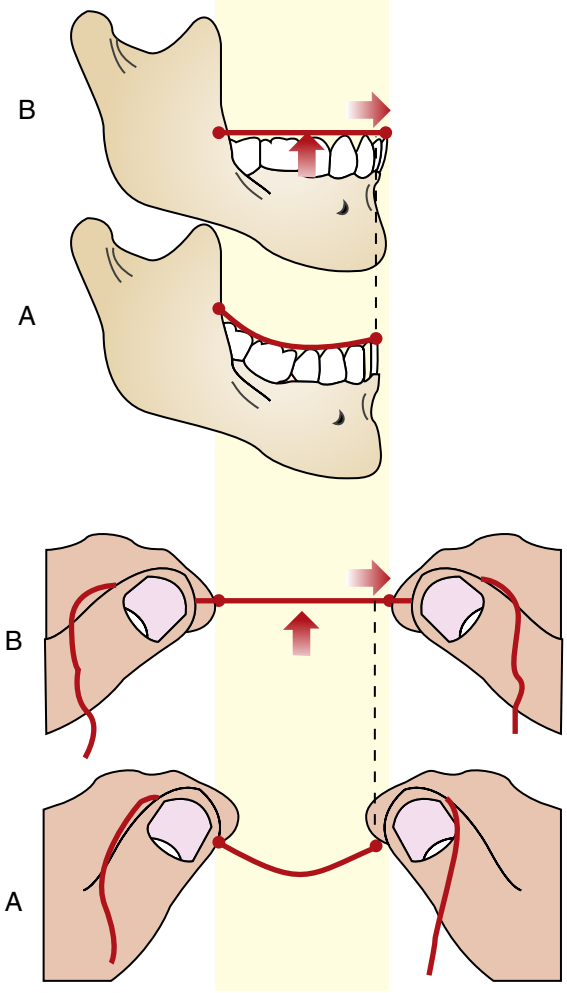


FIGURE 34-54 Total space analysis. A, curve of occlusion; B, leveled curve of occlusion increasing length.

Fig. 34-52, *A*). Note the improvement in muscle balance and facial harmony after serial extraction and active treatment (see Fig. 34-52, *B*).

The space available was obtained by placing a brass wire from the mesiobuccal of the primary first mandibular molar to the mesiobuccal of the opposite molar (Fig. 34-53). The wire then was straightened and measured with a pointed caliper to the nearest 0.1 mm. This value was subtracted from the total space required to yield the deficit.

Middle Area. For the middle area, calculations were made of the difference between space required and space available. In this instance, however, the mandibular curve of occlusion was considered.

Tooth Measurement. The crown widths of the permanent mandibular first molars were measured at their greatest mesiodistal diameter on the cast. These values were added to the measurements of the premolars obtained from the radiographs (see Figs. 34-46 to 34-48).

Curve of Occlusion. The following describes the procedures for the calculations for the curve of occlusion.

The space required to level the mandibular curve of occlusion was determined (Fig. 34-54). A flat object was placed on the occlusal surfaces of the mandibular teeth, contacting the permanent first molars and the incisors. The deepest point between this flat surface and the occlusal surfaces of the primary molars was measured on both sides. The curve of occlusion formula was applied, and the space required for leveling was determined. This was added to the tooth measurements to complete the space required. The curve of occlusion formula used the greatest depth of each side:

$$(\text{Right side depth} + \text{Left side depth})/2 + 0.5 \text{ mm}$$

The space available was determined by placing two brass wires from the mesiobuccal of the primary first molars to the distobuccal of the permanent first molars (Fig. 34-55). These were measured as before, added together, and subtracted from the space required.

Posterior Area. For the posterior area, the space required and space available were determined (including that presently available and that predicted). The space required consisted of the sum of the mesiodistal widths of the two second and third molars, which were unerupted in these patients (Fig. 34-56). Because they were unerupted, radiographic enlargements had to be calculated. However, the formula used for the conventional method was modified. In this instance, the permanent mandibular first molars were substituted for the

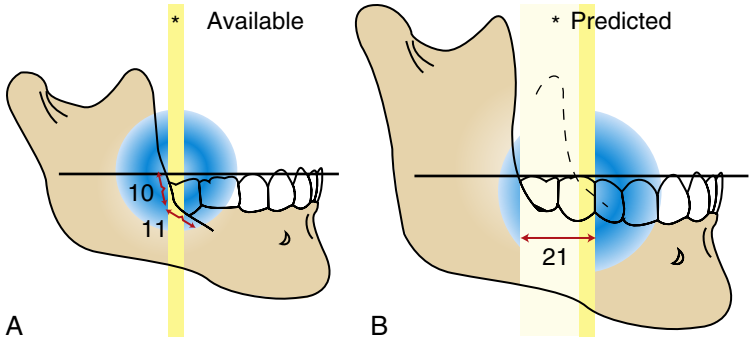


FIGURE 34-56 Total space analysis. Measurement of the (A) available and (B) predicted space in the posterior area.

primary second molars. A second complication was that often the third molars were not visible on the radiographs. In this case, Wheeler's measurements were used, and the calculation was as follows:

$$X = y - x^1/y^1$$

where X is the estimated value of the permanent mandibular third molar in the individual patient, x^1 is Wheeler's values for third molars, y is the actual size of permanent mandibular first molar on the cast, and y^1 is Wheeler's value for first molars.

The space available consisted of the space presently available plus the estimated increase or prediction. The estimated increase was 3 mm per year (1.5 mm for each side) until 14 years in girls and 16 years in boys. Therefore, the age of the patient was subtracted by 14 or 16. The result was multiplied by 3 to obtain the estimated increase for the individual patient. The space presently available was obtained by measuring the distance on the OP between a perpendicular line drawn from the OP tangent to the distal surface of the permanent first molar to the anterior border of the ramus on the lateral cephalometric tracing (see Fig. 34-56). After the presently available space and the predicted space were totaled to give the space available, the space required was subtracted.

Discussion. Because the total space analysis involves the permanent molars, it was not possible to compare its results with those of the other two procedures. However, when the results of the conventional method are compared with those of the Tweed method, the differences become highly significant.

The assessment of axial inclination of the mandibular anterior teeth with respect to the basal bone must be included in the analysis of mixed dentition malocclusion so that there will be harmony in the facial profile and stability in the dentition. The cephalometric correction in the Tweed method accounts for 9.8% of the space required, a considerable amount.

Table 34-2 illustrates the differences between the conventional method and the Tweed method in one of the subjects of the investigation. Comparison of the techniques makes it evident that completely different treatment plans will result.

Patient M.L. (see Figs. 34-51 and 34-52) exemplifies an ideal serial extraction situation: a Class I mixed dentition malocclusion with an anterior discrepancy in the form of an alveolodental protrusion and a posterior discrepancy with a medium angle facial pattern. The first premolars were extracted to correct the anterior discrepancy, and the third molars were extracted to correct the posterior discrepancy.

TABLE 34-2 Mixed-Dentition Analysis: Comparison of the Conventional and Tweed Methods in a Sample Patient

Sample Patient	Conventional Method (mm)	Tweed Method (mm)
Space required	−69.3	−69.3
Space available	+69.1	+69.1
Discrepancy	−0.2	−0.2
Cephalometric Correction	—	−8.0
Total discrepancy	−0.2	−8.2
Difference between two methods	—	−8.0

Patient M.R. (see Figs. 34-85 and 34-87) also is a good example of a favorable serial extraction situation: a Class I mixed dentition malocclusion with an anterior discrepancy in the form of a moderate alveolodental protrusion and a posterior discrepancy with a high-angle facial pattern. The same teeth were extracted as in patient M.L. for different reasons. The anterior discrepancy is more related to the alveolodental protrusion in patient M.L. than in patient M.R. The posterior discrepancy is more related to the high-angle facial pattern in patient M.R. than in patient M.L. Thus, each individual is treated differently depending on the results of the various diagnostic procedures, including total space analysis. Total analysis may thus result in extraction of varied teeth or nonextraction, with mechanotherapy varying to match the correction of individual discrepancies from a balanced dentofacial form.

Timing of Guided Primary Tooth Removal

Three factors may be applied by the clinician in deciding the optimal time for the removal of teeth in the guidance of occlusion:

1. The effect of extraction of the primary tooth on the eruption of its permanent successor
2. The amount of root formation at the time of emergence
3. The length of time for the attainment of various stages of root development

Serial extraction, if carried out too early in the primary dentition, can delay the eruption of permanent successors. In the case of early extraction of the primary molar, Fanning[42] reported an initial spurt in eruption of the premolar. This leveled off and the tooth then remained stationary, erupting later than its antimere with a normally shedding primary precursor.

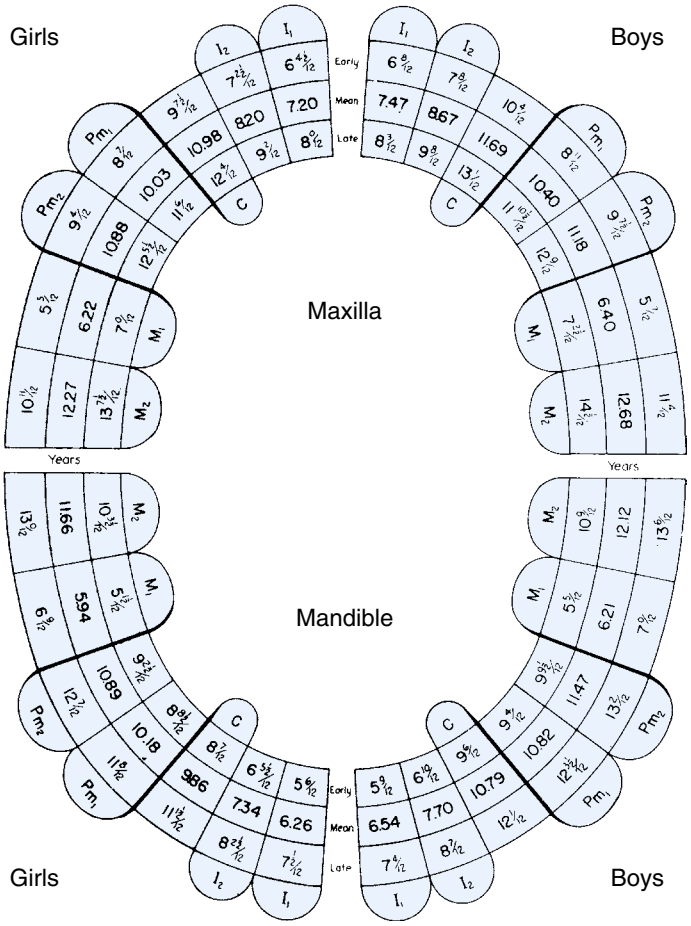


FIGURE 34-57 Assessment of dental age by means of tooth emergence (±1 standard deviation). This is a classic study taken from records of 93,000 children and demonstrates individual tooth variability and sex-linked timing of eruption. (From Hurme VO. Ranges in normalcy in eruption of permanent teeth. *J Dent Child* 1949;16:11.)

If serial extraction is initiated with the extraction of the primary canines, the length of the roots of the premolars is not an important consideration. If, however, the orthodontist is contemplating initiating serial extraction by removing the primary first molars, the length of the root of the premolar is an important consideration and guide for the commencement of the procedure.[43]

The relative eruptive rates of the permanent canines and first premolars influence the decision about which primary teeth should be extracted. If, for example, during an examination of the radiographs, the orthodontist observes the permanent mandibular first premolar crown ahead of the permanent canine crown, the premolar with less than one half its root formed, and the mandibular incisors crowded, the primary canine should be extracted to relieve the crowding. The primary first molar should be left until the first premolar has attained one half its root length. If, on examining the radiographs, the orthodontist observes the premolar crown even with the canine crown, the premolar with one half its root formed, and an alveolodental protrusion, the primary first molar should be extracted to encourage the emergence of its successor.

Dental age, assessed particularly by root length, is an essential requirement in the decision of a serial extraction program and in the initiation of interceptive and definitive fixed appliance treatment. A knowledge of root development, relative eruptive rates, and the emergence of permanent teeth (Fig. 34-57), together with root resorption of the primary teeth and the factors that influence these processes, is mandatory in the timing of serial extraction.

TREATMENT

The authors adhere to the principles and objectives of the Tweed philosophy. Clinically, Tweed advocated maximal facial harmony and balance. To achieve this goal, he recognized that mandibular incisor teeth must be placed upright over basal bone. The primary objective in treatment is to obtain maximal health, aesthetics, function, and stability. Specifically, it is excellence in tooth alignment, arch form, and axial inclination of the teeth, with optimal occlusal intercuspation of the maxillary and mandibular dentitions.

Objectives of treatment include the following:

1. Decrease in the FMA, indicating a favorable rotation of the mandible.
2. Decrease in the IMPA, indicating the reduction of the alveolodental protrusion.
3. Increase in the FMIA, also indicating an upright positioning of the mandibular incisor teeth.
4. Decrease in the OP angle throughout treatment, indicating that one is not extruding the posterior teeth or dumping the anterior teeth forward.

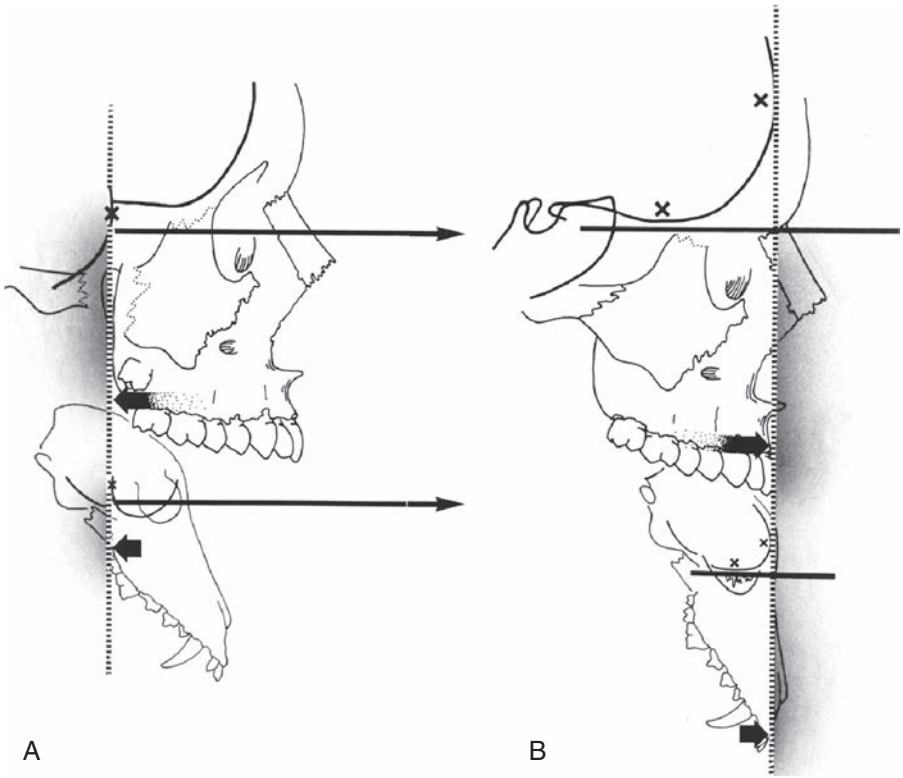


FIGURE 34-58 A, Posterior limits of the nasomaxillary area: a line perpendicular to the line of sight passing through the junction of the anterior and middle cranial fossae. B, Anterior limit of the nasomaxillary complex: a line extending from the internal surface of the frontal bone and running perpendicular to the ethmoid. (Adapted from Enlow DH, Hans MG. *Essentials of Facial Growth*. 2nd ed, Ann Arbor, MI: Needham Press; 2008.)

5. Decrease in the ANB (A point–nasion–B point) angle, indicating a correction of the skeletal discrepancy.
6. Increase in Merrifield's Z angle, indicating improvement in facial harmony and balance.
7. No lateral, anterior, or posterior expansion of the dentition. This is substantiated by the scientific investigations of Enlow,[23] who describes specifically the boundaries of the nasomaxillary complex (Fig. 34-58).

Many orthodontists have found from bitter experience that they cannot extend arch length posteriorly, anteriorly, or laterally unless the position of the teeth results from environmental factors such as premature loss of the primary teeth. Most of the problems during treatment and retention have been associated with creating space and maintaining alignment where teeth have not been extracted.[44] Teeth that have not been placed in the correct position relative to the skeletal pattern and soft tissue matrix are more likely to change after fixed appliances have been removed. The ideal conditions for stability are achieved when teeth are placed in a harmonious relationship early. Long-term use of mandibular fixed retainers may be needed to reduce age-related maturational changes in the lower anterior segment. These should be maintained in patients for a prolonged time to preserve the incisor alignment when the mandible grows horizontally (see Chapters 1 and 33).

Serial extraction allows teeth to become aligned when they emerge into the oral cavity rather than to remain in a crowded unfavorable condition for several years. In the case of alveolodental protrusion, the procedure allows the mandibular incisors to be positioned upright lingually into a position of balance. This alone will reduce mechanotherapy by 6 months and contribute to the stability of the treatment result. In many of these patients, the need for retention is minimal. However, the orthodontist must remember that under ideal serial extraction conditions, the orthodontist is not dealing with severe skeletal discrepancies or severe overjet and overbite problems. The need for long-term retention is greater when skeletal discrepancies have complicated the orthodontic treatment.

One may ask, "If the roots of the permanent incisor teeth complete their formation in a more favorable position, is their stability enhanced?" It seems logical that if a tooth completes its formation in a site where it will remain when treatment is completed, it will be more stable. Conversely, if a tooth is left in a crowded, tipped, and rotated position for several years and then moved to a new position relatively rapidly, it will be less stable for a time and will require a longer retention period.

One also may ask, "Does the initiation of serial extraction with the removal of primary teeth always mean that the permanent teeth will be removed?" After it has been initiated, serial extraction more often than not will culminate in the extraction of four premolar teeth because arch length, which is deficient to begin with, is reduced even more. Despite a thorough diagnosis, the treatment plan occasionally needs to be changed to nonextraction treatment. *The practitioner should always be prepared to treat without extraction if it appears that this can be done successfully with stability.* At the beginning of treatment, the parents should be informed that extractions may be necessary to produce a successful and stable treatment result. Later, they will be relieved and happy if extractions are not necessary.

An important objective in using the serial extraction technique is to make treatment easier and mechanotherapy less complicated, less expensive, and shorter (especially during the teenage years). Treatment can be divided into four categories:

1. A period of interceptive guidance, extending about 5 years from age 7.5 to 12.5 years. This consists entirely of the guidance of occlusion, including serial extraction or space maintenance (or both) and is the most ideal service that can be provided. Unfortunately, production of excellent occlusion with serial extraction, or space maintenance alone (or both) is possible in only a few patients. When guidance without further appliances is accomplished, it is most rewarding and satisfying because the results are achieved without mechanotherapy.
2. An initial period of interceptive guidance, extending about 4 years from age 7.5 to 11.5 years, plus a second period of fixed appliance treatment extending about 1 year from 11.5 to 12.5 years). Class I and specific types of dental Class II malocclusions fall into this category.
3. An initial period of interceptive treatment, extending about 1 year from 8.5 to 9.5 years, plus a period of interceptive guidance extending about 2 years from 9.5 to 11.5 years and a second period of mechanotherapy extending about 1.5 years from 11.5 to 13 years. Class II malocclusions fall primarily into this category.
4. A period of fixed appliance treatment, extending for 1.5 to 3 years from age 11.5 to 14.5 years. Serial extraction is not involved in this treatment. Wherever possible, the orthodontist should try to avoid extensive treatment in the teenage period because of potential adverse sociopsychological concerns that influence patient cooperation.

Of course, these are general classifications. They may vary considerably depending on the individual patient, the malocclusion, and the dental age.

CLASS I TREATMENT

Serial extraction has been classically characterized by the removal of the primary canines, primary first molars, and permanent first premolars. With scientific investigation and clinical experience, serial extraction has become increasingly sophisticated and precise. Results will be more rewarding if the orthodontist does not cling to a particular sequence but varies it according to the diagnosis and therapeutic goals. The sequence that the orthodontist believes is indicated for each patient should be selected.[45]

Treatment Procedures: Interceptive Guidance, Active Treatment

Serial Extraction in Class I Treatment

Group A: Anterior Discrepancy—Crowding. This is a typical serial extraction problem: severe crowding, a developing Class I malocclusion, a favorable overjet–overbite relation of the incisor teeth, and an ideal orthognathic facial pattern (Fig. 34-59). Examination of the radiographs often reveals a crescent pattern of resorption on the mesial of the primary canine roots (see Fig. 34-6). This is an indication of a true hereditary tooth size–jaw size discrepancy. Radiographic evaluation indicates that the first premolars are emerging favorably, ahead of the permanent canines. None of the unerupted permanent teeth have reached one half root length. Because of this, the primary first molars would not be extracted. The primary canines should be extracted to relieve the incisor crowding.

Step 1: Extraction of the primary canines

Step 2: Extraction of the primary first molars. The incisor crowding has improved, the overbite has increased, and the extraction site is reduced in size (Fig. 34-60). The radiographs reveal that the first premolars have reached one half root length. Now is the time to extract the pri-

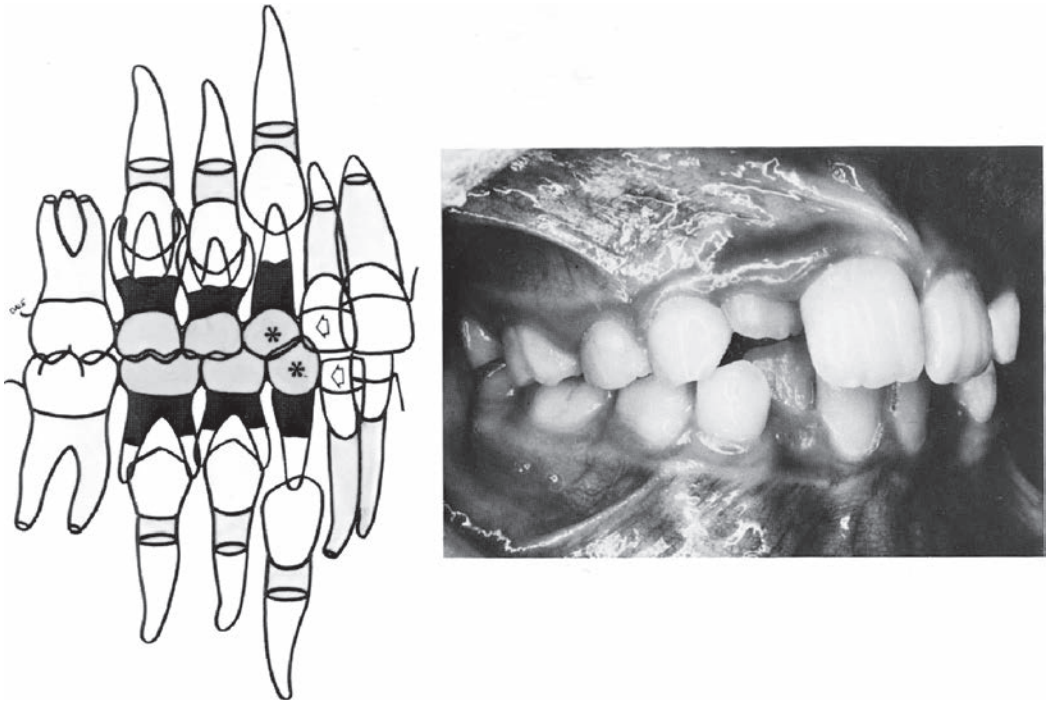


FIGURE 34-59

mary first molars to encourage the eruption of the first premolar teeth.

Step 3: Extraction of the first premolars. These teeth are emerging into the oral cavity (Fig. 34-61). Because the permanent canines have developed beyond one half root length, indicating that they are prepared to accelerate their eruption, the orthodontist extracts the premolars.

Step 4: Fixed appliance treatment. This is the typical result of serial extraction, a relatively deep overbite with a distoaxial inclination of the canines, a mesioaxial inclination of the second premolars, a Class I molar relationship, an improved alignment of the incisors, and residual spaces at the extraction sites (Fig. 34-62).

Step 5: Retention. When mechanotherapy is completed, an ideal occlusion should be observed, with minimal overjet–overbite relationship of the anterior teeth, parallel canine and premolar roots, ideal arch form, and no spaces (Fig. 34-63). In addition, the dentition should be aligned in harmony with the craniofacial skeleton and soft tissue matrix.

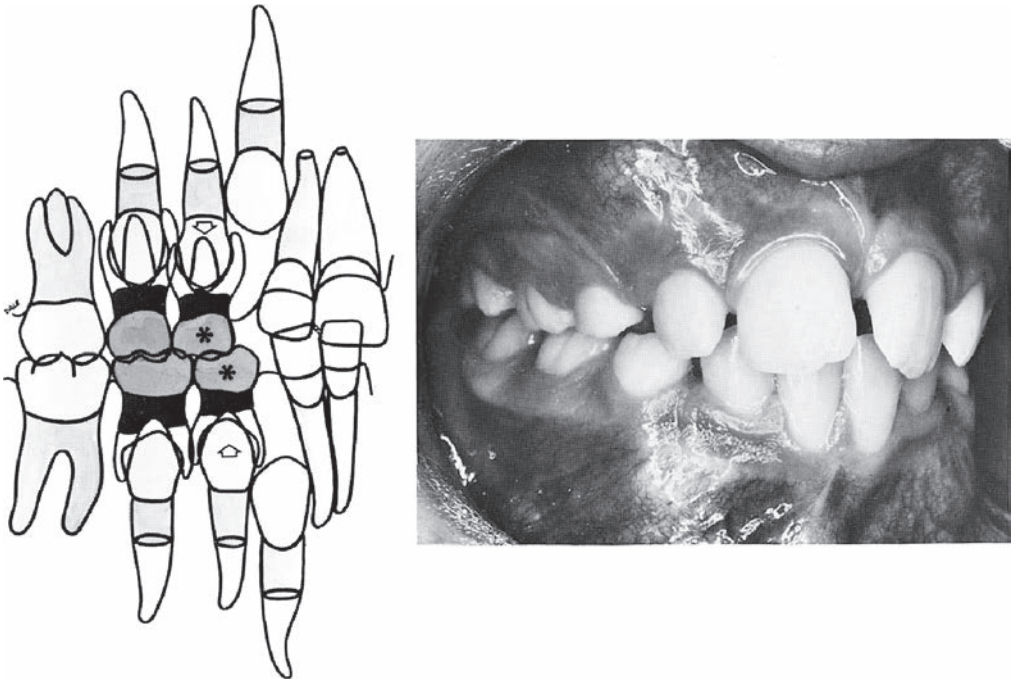


FIGURE 34-60

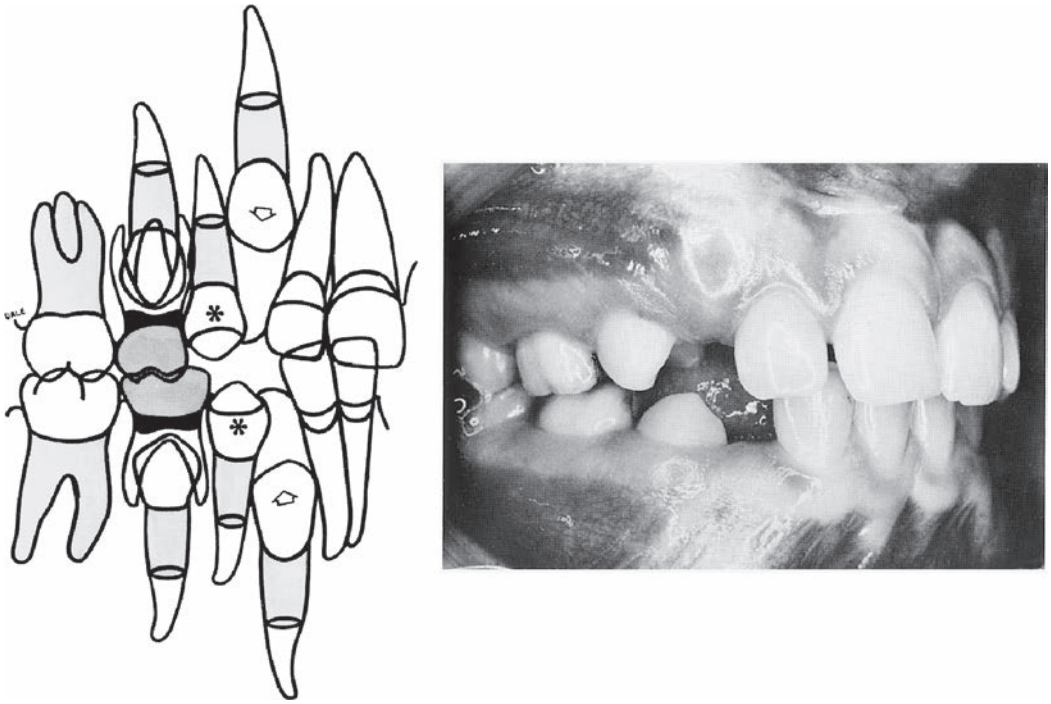


FIGURE 34-61

Step 6: Post retention. Again, an ideal occlusion with stability should be evident (Fig. 34-64). Initiating the serial extraction procedure with elimination of the primary mandibular canines tends to deepen the overbite. The primary first molars should be extracted when the underlying first premolars have reached one half their root length. If this is done, risk of collapse will be minimal. If the mandibular incisors are crowded, the primary canines should be extracted first, in preference to the primary first molars. The orthodontist rarely will be satisfied with the improved alignment of the incisors when the primary molars are extracted first. Again, the decision is based on the relative position and length of the roots of the first premolars and canines. Figure 34-64 illustrates the occlusion 18 years after treatment.

Group B: Anterior Discrepancy—Alveolodental Protrusion. A minor irregularity of the incisor teeth exists. Instead of crowding, the patient has an alveolodental protrusion (Fig. 34-65). The crowns of the first premolars and canines are at the same level. However, the canines are beyond one

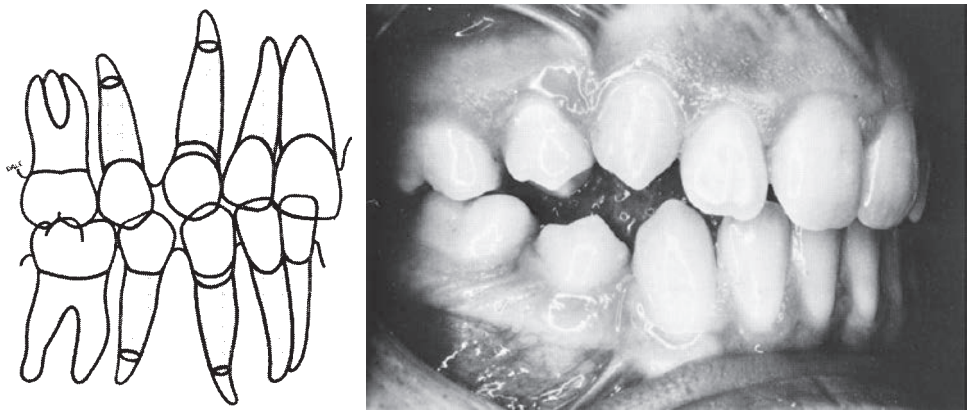


FIGURE 34-62

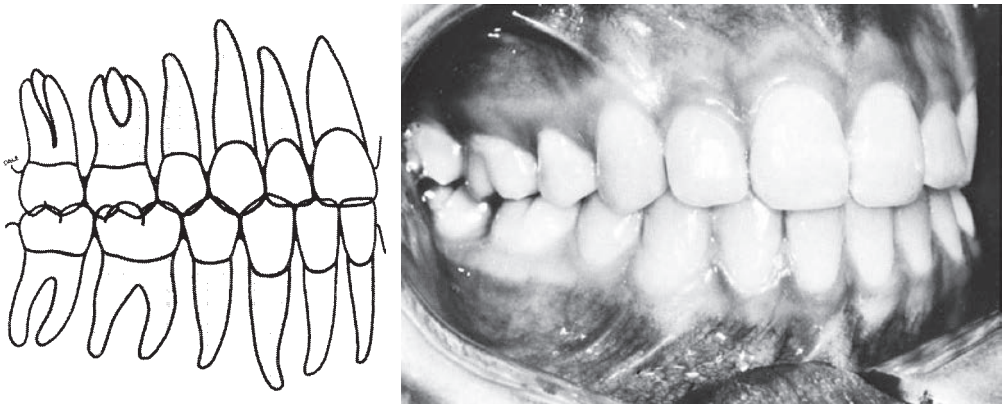


FIGURE 34-63

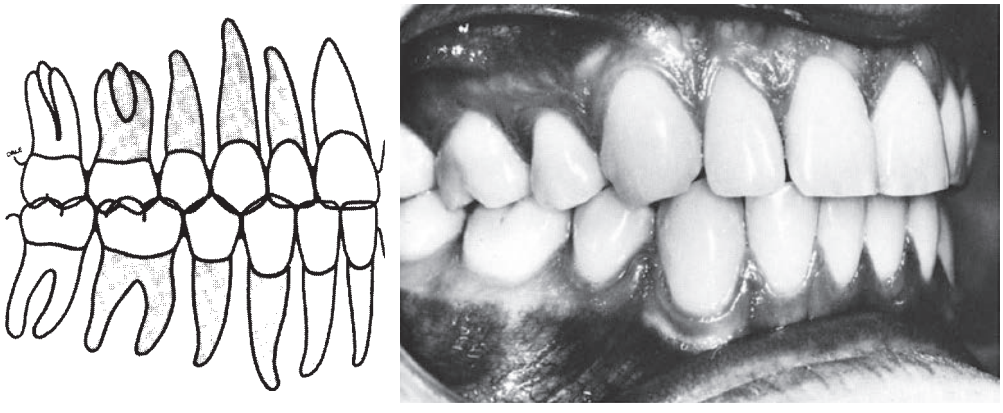


FIGURE 34-64

half root length and are erupting faster than the premolars. Because the first premolars have one half their root length developed, the primary first molars should be extracted to accelerate eruption of the first premolars. This will ensure that the premolars emerge into the oral cavity ahead of the canines. Timing is most important to prevent the formation of a knife-edge ridge.

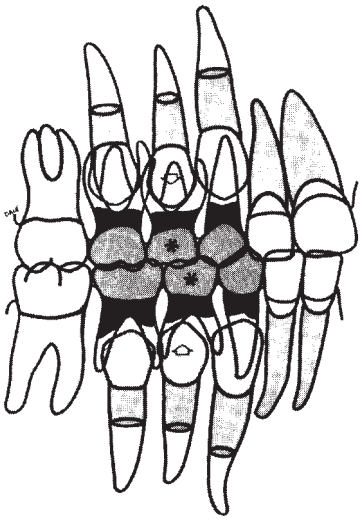


FIGURE 34-65

Step 1: Extraction of the primary first molars
Step 2: Extraction of the primary canines and first premolars. When the first premolars have emerged sufficiently, they are extracted along with whatever primary canines remain (Fig. 34-66). No effort is made to prevent lingual tipping of the incisor teeth because the objective is to reduce the alveolodental protrusion.
Step 3: Fixed appliance treatment. Note how beautifully the dentition is aligning itself (Fig. 34-67). Little mechanical treatment will be required.
Step 4: Retention. Retention in the mandible is less crucial because minimal irregularity was present before treatment (Fig. 34-68).

Group C: Middle Discrepancy—Impacted Canines. The tooth size–jaw size discrepancy is severe, causing premature exfoliation of the primary canines (Fig. 34-69). Note the splaying of the incisors because of crowding in the apical area. Often parents interpret this spacing as evidence for nonextraction treatment. The orthodontist must explain that this is a sign of severe crowding. The radiograph will reveal that the first premolars are ahead of the canines in eruption and have attained one half their

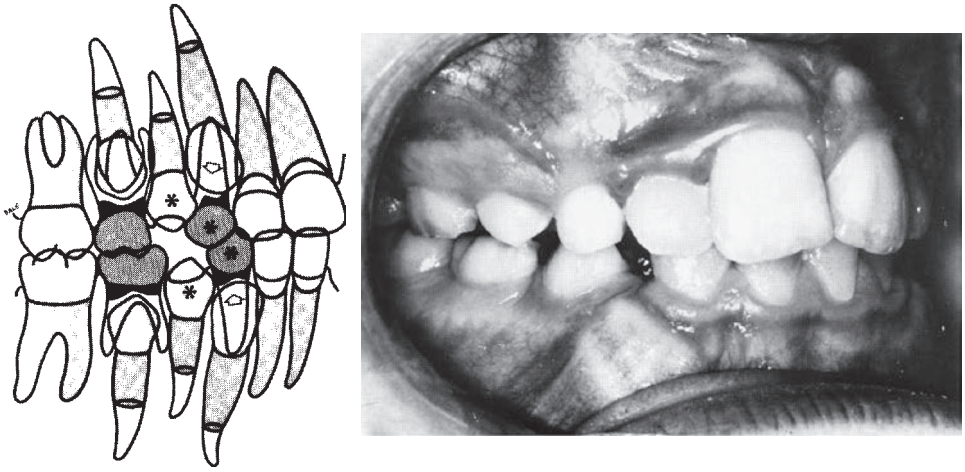


FIGURE 34-66

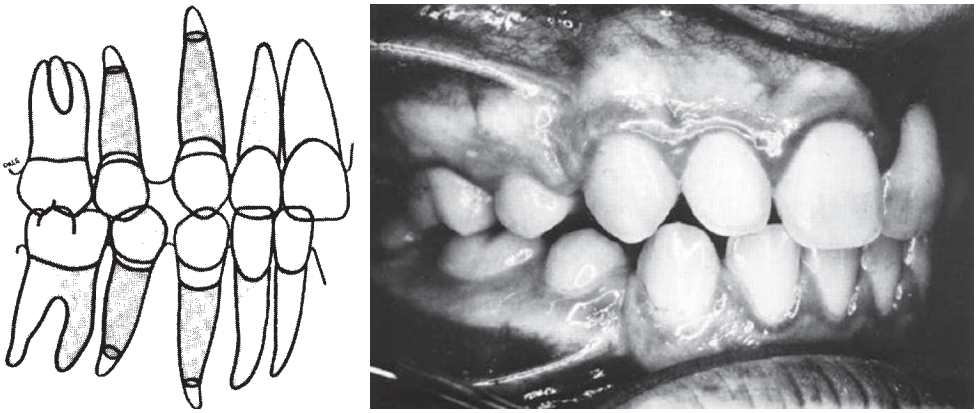


FIGURE 34-67

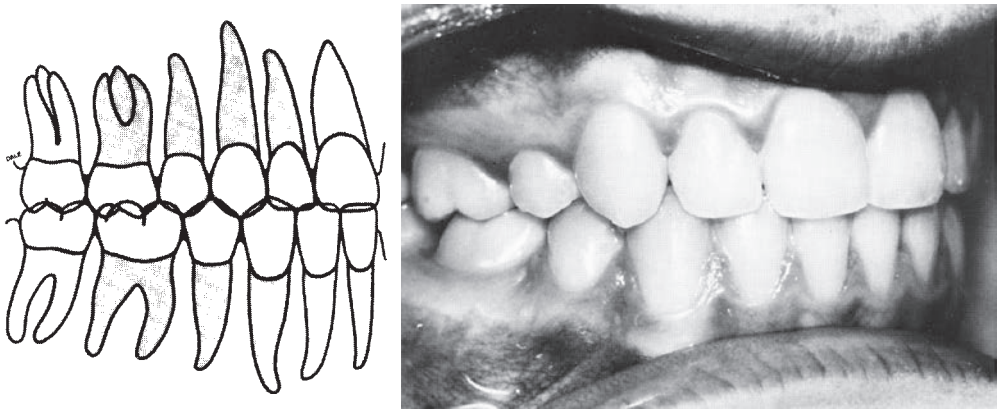


FIGURE 34-68

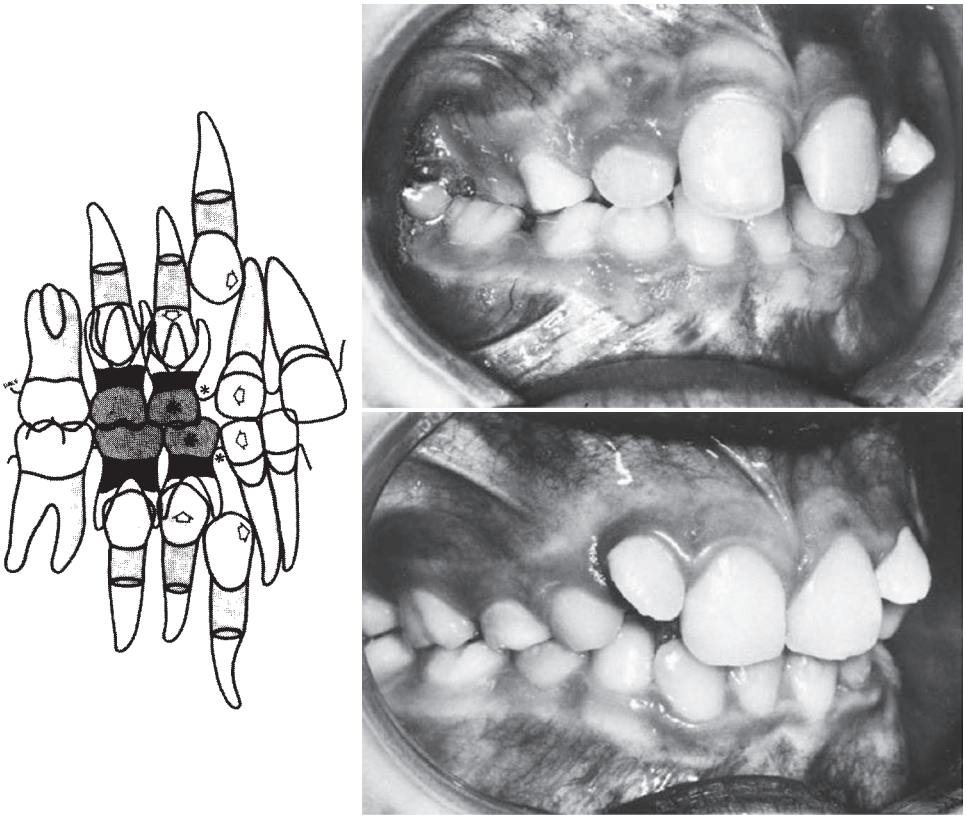


FIGURE 34-69

root length. Treatment begins with the extraction of the primary first molars (remember, here primary canines have been lost).

The impacted permanent maxillary canines may cause severe splaying of the maxillary incisors to such an extent that the lateral incisors do not contact the primary canines (see Fig. 34-69, *bottom right*). In this situation, extracting the primary canines first does little good. Better advice is to extract the primary first molars to encourage the first premolars to emerge as early as possible. The canines then will have space to migrate away from the apices of the incisors and begin their eruption into the oral cavity. In this instance, the practitioner should be concerned more with correcting canine crowding than with incisor irregularity. Every effort should be made to avoid correcting the incisors with fixed appliances for fear of causing the incisor roots to resorb as lateral incisor roots move distally.

Step 1: Extraction of the primary first molars
Step 2: Extraction of the first premolars. For reasons explained in group A, step 3, the first premolars are now due for extraction (Fig. 34-70).
Step 3: Fixed appliance treatment. Note the typical result of serial extraction (Fig. 34-71).
Step 4: Retention. Again, note the desired result of mechanotherapy (Fig. 34-72).

Group D: Enucleation in the Mandible. If it is evident that the canines will emerge into the oral cavity ahead of the first premolars, the primary first molars can be extracted and the first

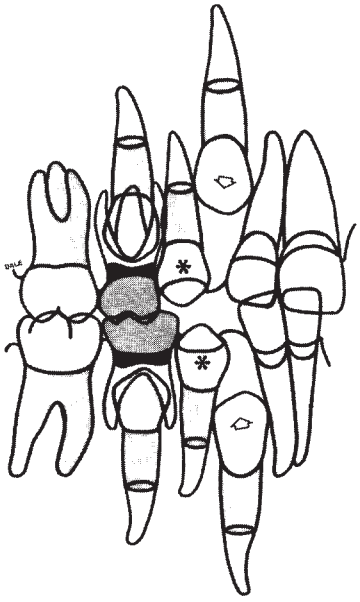


FIGURE 34-70

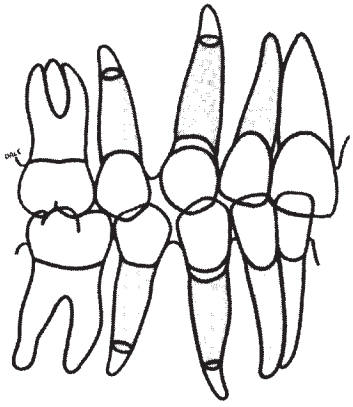


FIGURE 34-71

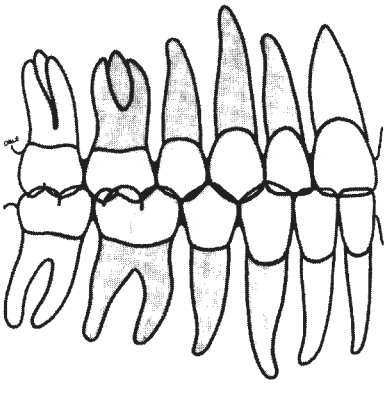


FIGURE 34-72

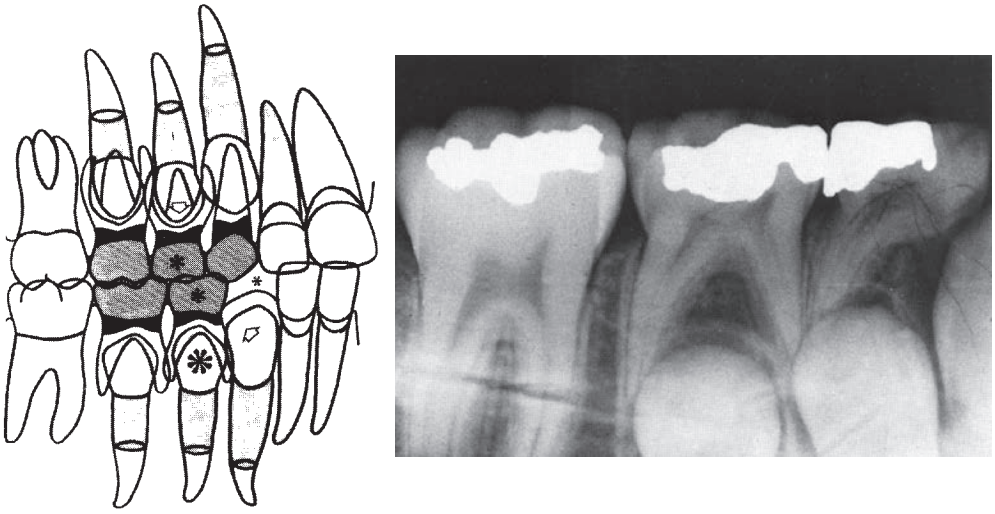


FIGURE 34-73

premolars may be enucleated (Fig. 34-73). This will encourage distal migration of the canines as they erupt.

Step 1: Extraction of the primary first molars and enucleation of the mandibular first premolars
Step 2: Extraction of the primary maxillary canines and maxillary first premolars. In the maxilla, the first premolars usually emerge before the canines (Fig. 34-74). Therefore, enucleation is less likely to be indicated. At this point, the mandibular canines can be observed emerging favorably into the oral cavity.
Step 3: Mechanotherapy (Fig. 34-75). The patient is now ready for the fixed appliance multibracket appliance treatment.
Step 4: Retention

Group E: Enucleation in the Maxilla and Mandible. On occasion, the canines in the maxilla and mandible erupt before the first premolars (Fig. 34-76). If this is the case, the orthodontist might elect to extract the primary canines and first molars and enucleate the first premolars. This is acceptable if absolutely no opportunity exists to place fixed appliances at the completion of serial extraction. Otherwise, an alternative to enucleation is almost always preferable (see group F).

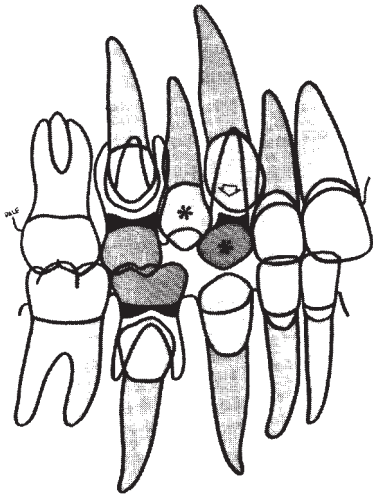


FIGURE 34-74

Step 1: Extraction of primary canines and primary first molars and enucleation of the first premolars
Step 2: Mechanotherapy (Fig. 34-77). The patient is now ready for the appliance treatment.
Step 3: Retention

Group F: Alternative to Enucleation. When the permanent canines are erupting ahead of the first premolars, enucleation of the premolars should be avoided if an opportunity exists to place fixed appliances at the completion of serial extraction (Fig. 34-78). When the first premolars have attained one half their root length, the primary first molars should be extracted.

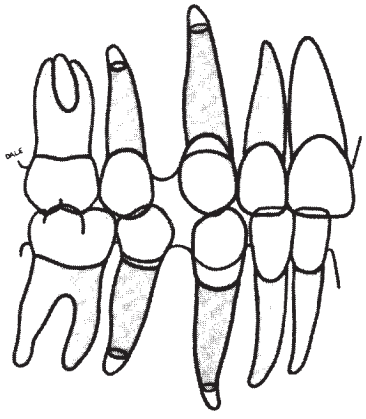


FIGURE 34-75

Step 1: Extraction of the primary first molars
Step 2: Extraction of the primary maxillary canines, maxillary first premolars, and primary mandibular second molars. About 6 to 9 months later, when the emerging mandibular first premolar appears to be obstructed by the mesial contour of the primary second molar, the orthodontist should extract the offending tooth (Fig. 34-79). However, this sequence is usually not necessary in the maxillary dentition.
Step 3: Extraction of the mandibular first premolars. When these teeth emerge sufficiently, they are extracted (Fig. 34-80).

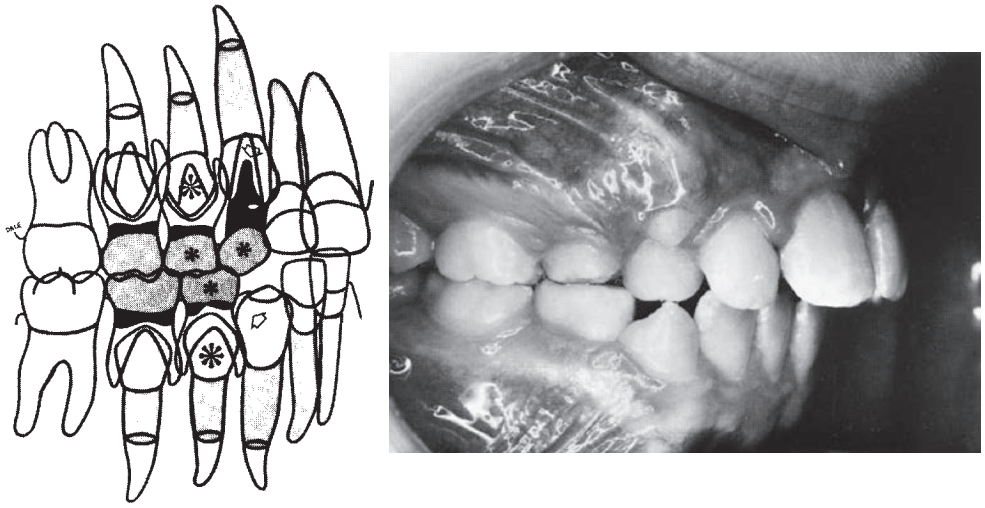


FIGURE 34-76

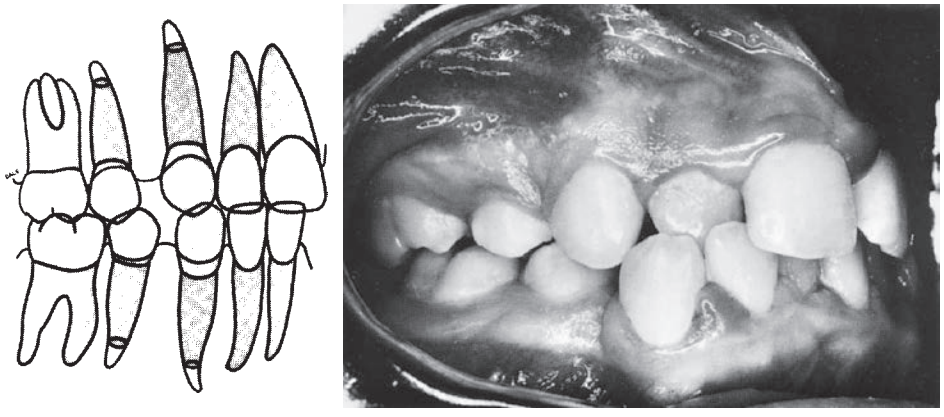


FIGURE 34-77

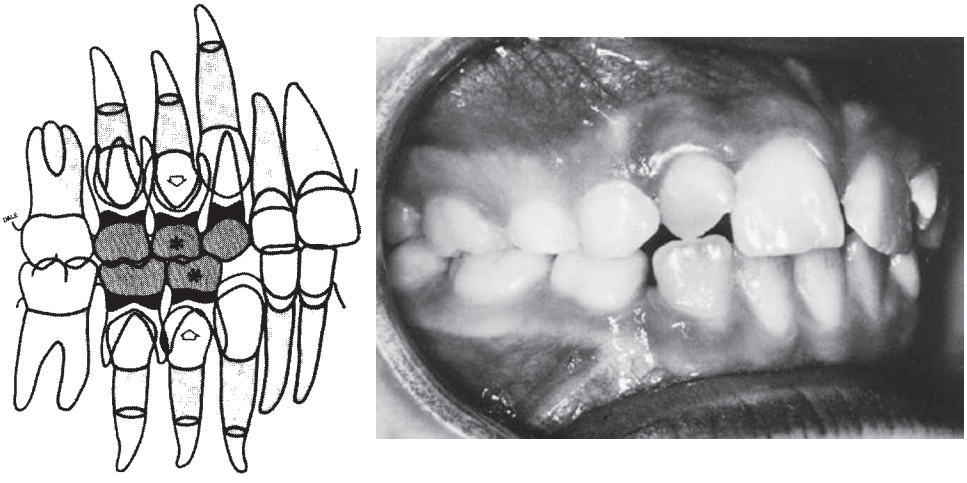


FIGURE 34-78

Step 4: Mechanotherapy. With this particular sequence, the least desirable extraction result is achieved (Fig. 34-81). However, extraction does not prolong the fixed appliance treatment significantly.

Step 5: Retention

Group G: Interproximal Reduction. Rarely should the mesial surfaces of the primary canines be reduced. These rare occurrences include when the practitioner does not intend to extract the permanent teeth and where a localized interference results in rotation of the lateral incisor.

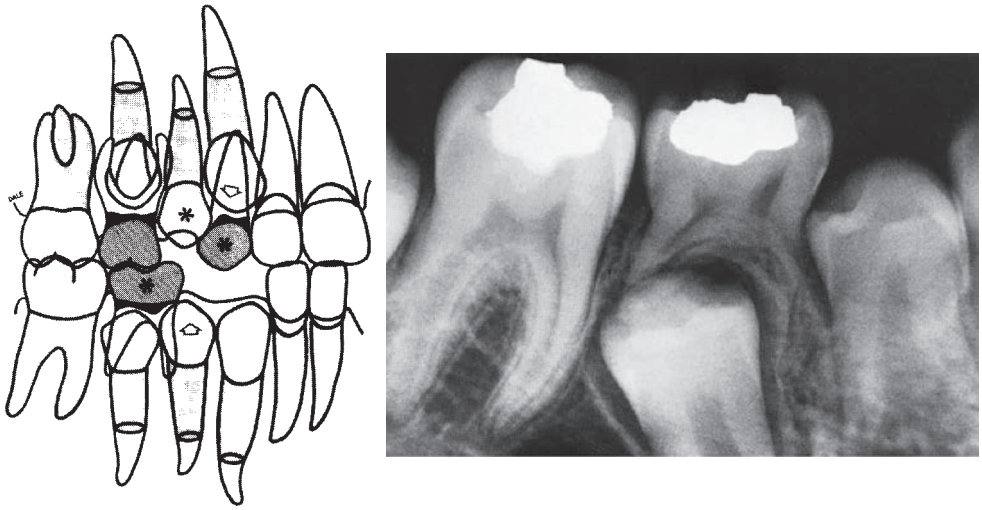


FIGURE 34-79

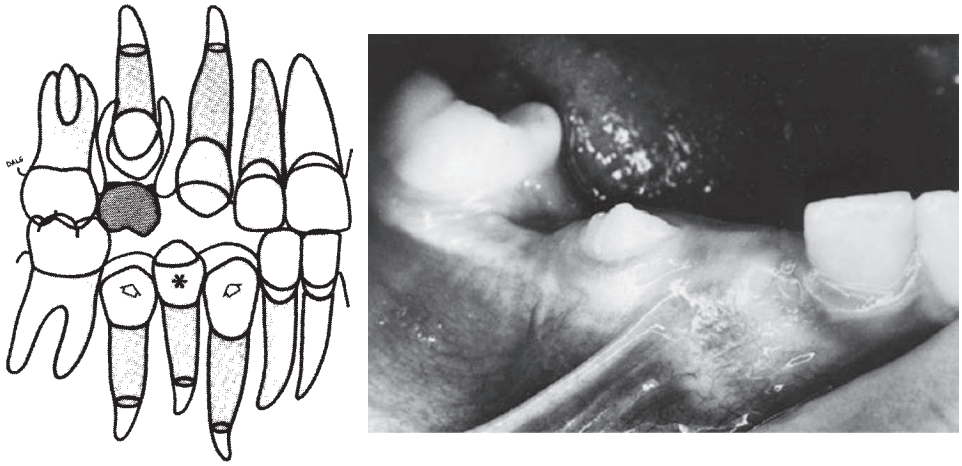


FIGURE 34-80

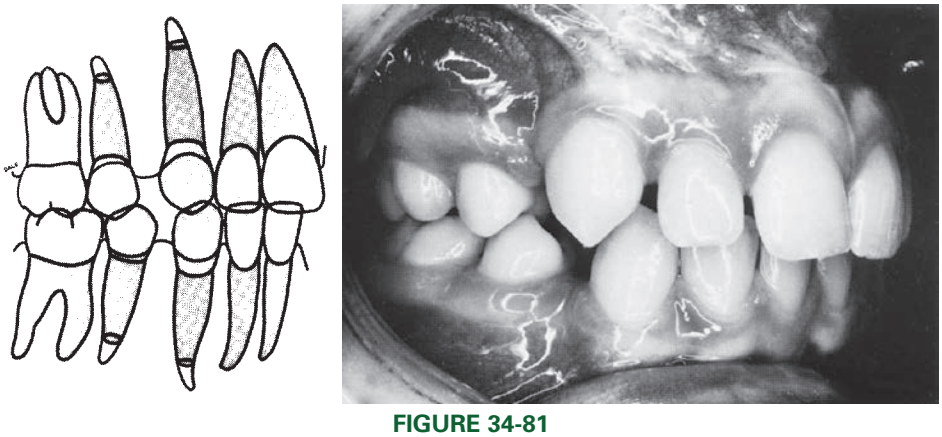


FIGURE 34-81

Occasionally, in nonextraction malocclusions, the primary second molars will be retained for an unusually long time. Because these teeth are wider mesiodistally than the underlying second premolars, they force the first premolars into a forward position in the dental arch and thus influence the permanent canines (Fig. 34-82, *A*). The long retention of these primary second molars also may interfere with the eruption of the first premolars after the permanent canines have emerged (Fig. 34-82, *B*). In each instance, the mesial surface of the primary second molars should be reduced by the amount of the leeway space. This usually results in a favorable alignment of the permanent teeth (Fig. 34-83).

The orthodontist may find it necessary at times to reduce the distal surfaces of the primary second molars (Fig. 34-84)

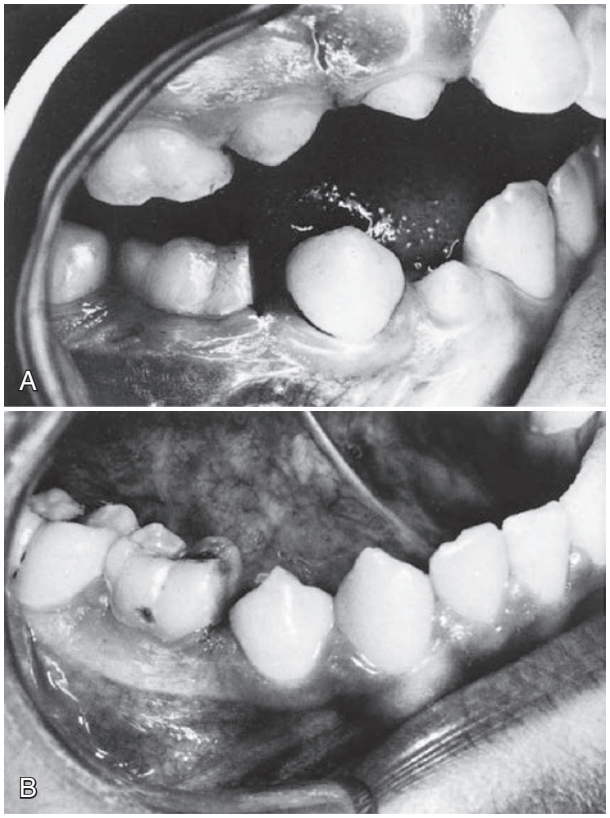


FIGURE 34-82 Interproximal reduction of the primary second molar to allow for emergence of the permanent canine (A) and the first premolar (B).

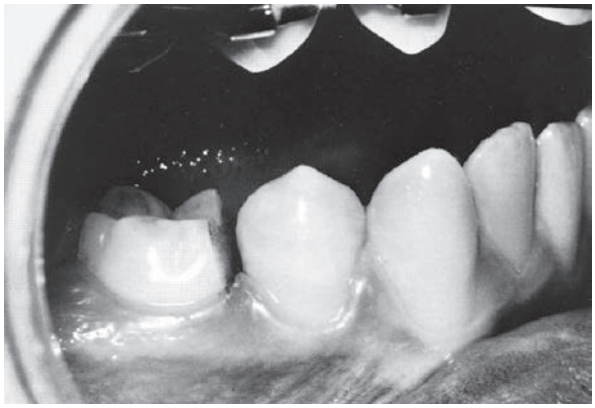


FIGURE 34-83 Favorable alignment after reduction of the primary second molar.

to ensure conversion of a straight terminal plane into a mesial step in preparation for a Class I relationship of the permanent first molars. This is accomplished when the primary maxillary second molars are exfoliated before those in the mandible and when space is present to allow the molars to move mesially. Ideally, the mandibular molars should be lost first. (For more detailed discussion of interproximal reduction of primary teeth, read the articles by Hotz,[3] Northway,[46,47] and Valencia et al.[48]).

Group H: Congenital Absence. Of utmost importance in patients with congenital absence of some teeth is to proceed with the conventional orthodontic diagnosis and treatment planning as if the teeth were present. Then treatment may proceed to cope

FIGURE 34-84 Reduction of the distal surface of the primary second molar to convert a straight terminal plane into a mesial step.

with the missing tooth or teeth. Whether the teeth are missing as a result of injury, disease, or developmental aberration matters little. Which teeth are missing and what is left to work with are the important factors in total correction of the malocclusion.

Maxillary Incisors. The maxillary lateral incisors are frequently missing or malformed. The percentage varies depending on the study consulted but in most practices is about 5% of the patients treated.

If a maxillary lateral incisor is congenitally absent, the dentition should be allowed to emerge completely before fixed appliance treatment begins. The orthodontist and primary care dentist should examine the crown of the canine carefully after emergence before deciding to place it in the lateral incisor position or its usual position.[49] The orthodontist should not rely on radiographs to evaluate the shape of unerupted teeth. Placing implant or fixed bridge restorations rather than using canines as lateral incisors may be needed if the size discrepancy exceeds the ability to restore the cuspid to appear as a lateral incisor. When canines are used as laterals, the orthodontist needs to be aware of arch size discrepancies between the arches and the potential to develop interproximal spaces during the retention period; in many instances, aesthetic considerations are the final deciding factor on whether or not to proceed with cuspid substitution.[50–52]

Mandibular Incisors. The patient in Figure 34-85 was treated by extraction of the mandibular central incisors. Such extraction disturbs arch length balance. Patients who have severe recession and malposition of the central incisors can be treated by routine serial extraction. The extraction of mandibular incisors, especially during development, definitely is not recommended; it complicates the total treatment plan and necessitates a compromise result. Every effort should be made to maintain the mandibular incisors, especially in unilateral situations.

Sometimes the orthodontist is faced with a single missing lower incisor. To repeat, every effort should be made to maintain the mandibular incisors, especially in the unilateral situation and during development. If one of the mandibular incisors is missing, it may be advisable to avoid extracting a premolar in that quadrant and use the canine as an incisor. If it is an extraction case, the molars on the affected side will be in a Class I relationship; if it is not, they will be in a Class III relationship.

PREMOLARS

Figure 34-26 illustrates a patient who had two maxillary first premolars missing. The clinical appearance that resulted was

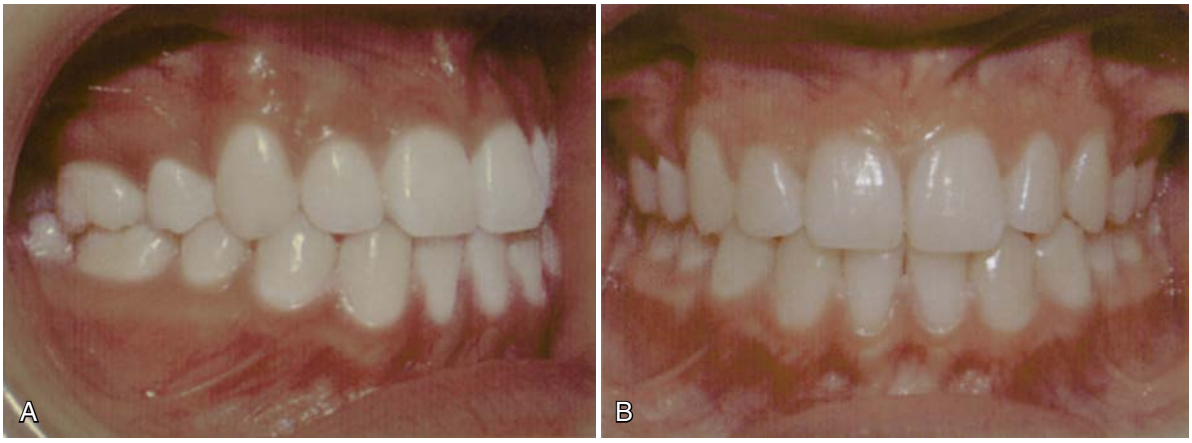


FIGURE 34-85 A and B, Alignment of the mandibular dentition after treatment in a patient with congenital absence of two incisors. The first premolars were extracted in the maxilla.

unusual. The canines erupted into the premolar locations, and the primary canines were retained. The treatment plan was altered to include extraction of the primary canines and movement of the permanent canines forward into their proper position with a resultant.

Class II Molar Relationship. Mandibular second premolars frequently are missing. Depending on the basic orthodontic diagnosis, treatment may involve moving the molars forward or replacing the missing teeth with implants or bridges. Treatment for Class II malocclusions that involved extractions may include moving the maxillary protruding incisors into the space provided by the extraction of the maxillary first premolars. Correction for a Class II molar relationship may involve moving the mandibular molars forward into the second premolar area, where the teeth are congenitally absent.

Mandibular second premolars constantly present difficulties in orthodontic treatment. They often are missing or erupting in the wrong direction and often are badly shaped with the potential for a size difference from the left to right side. The use of TADs to bring lower molars mesially without taxing anterior anchorage allows for space closure without adverse lingual tipping of the lower incisor teeth.

When one mandibular second premolar is congenitally missing, the clinician should proceed with serial extraction as normally done, extracting all four primary canines or primary first molars, depending on the development. When the four first premolars have emerged, three of them are extracted. In a quadrant where the second premolar is missing, the primary second molar is extracted. The first premolar then is moved into the second premolar site. This allows space for the canine to emerge. Occasionally, the primary second molar must be extracted earlier to allow the first premolar to emerge into a more distal position.

Garn and Lewis[53] have shown that in a congenital absence patient, more often than not, the treatment will not require extraction. In other words, congenital absence of teeth is related to small teeth; thus, serial extraction is not indicated.

If the diagnosis indicates the extraction of four second premolars or if the four teeth are congenitally missing, serial extraction is not indicated. Second premolar extractions are prescribed for a borderline tooth size–jaw size discrepancy in which the incisors are upright over basal bone. With extraction of the second premolars, the minimal crowding in the anterior area is corrected by using a fraction of the extraction space and without producing an alveolodental retrusion. It is difficult to encourage the emergence of second premolars ahead of the first premolars by extraction of the primary second molars early. Second premolars are notoriously slow in their eruption. Furthermore, if the primary second molars are extracted early, the permanent first molars may move mesially too rapidly. Second premolars are extracted when they have emerged partially into the oral cavity.

Often the question is asked about creating a dished-in face as the ultimate result of serial extraction when the four first premolar teeth are extracted. All orthodontists should be concerned about creating dished-in faces. *That is why serial extraction should be done only on a specific group of patients and only after a comprehensive diagnosis.* Because of this concern, the clinician should monitor the position of the mandibular incisors constantly. These should be allowed to tip lingually to the desired position and then held.

Some patients have a dished-in profile whether treated by extraction or nonextraction or left untreated. They exhibit a maxillary and mandibular alveolodental retrusion, a low MPA, a deep overbite of the incisor teeth, a short anterior facial height, a prominent chin, a prominent nose, and a tense perioral musculature. If a severe tooth size–jaw size discrepancy is superimposed, extraction may be called for to produce a stable result. Deferring consideration of extraction until all the permanent teeth have emerged is usually advisable with this facial pattern rather than performing serial extraction. If the orthodontist performs serial extraction, the orthodontist must use extreme caution and use holding appliances.[42,54,55]

A word of caution is necessary at this point regarding holding appliances, the labial upright positioning of permanent mandibular incisors, and the distal movement of permanent mandibular first molars by the use of fixed appliances including lip bumpers. Even the use of a lower fixed lingual arch can result in the impaction of the erupting mandibular second molars, another reason for continued monitoring during tooth eruption.

Typical Patient for Class I Serial Extraction

A typical Class I malocclusion with an anterior discrepancy manifested by an alveolodental protrusion and a posterior discrepancy manifested by a high-angle configuration is illustrated by patient M.R. (Figs. 34-86 and 34-87). The dentition when

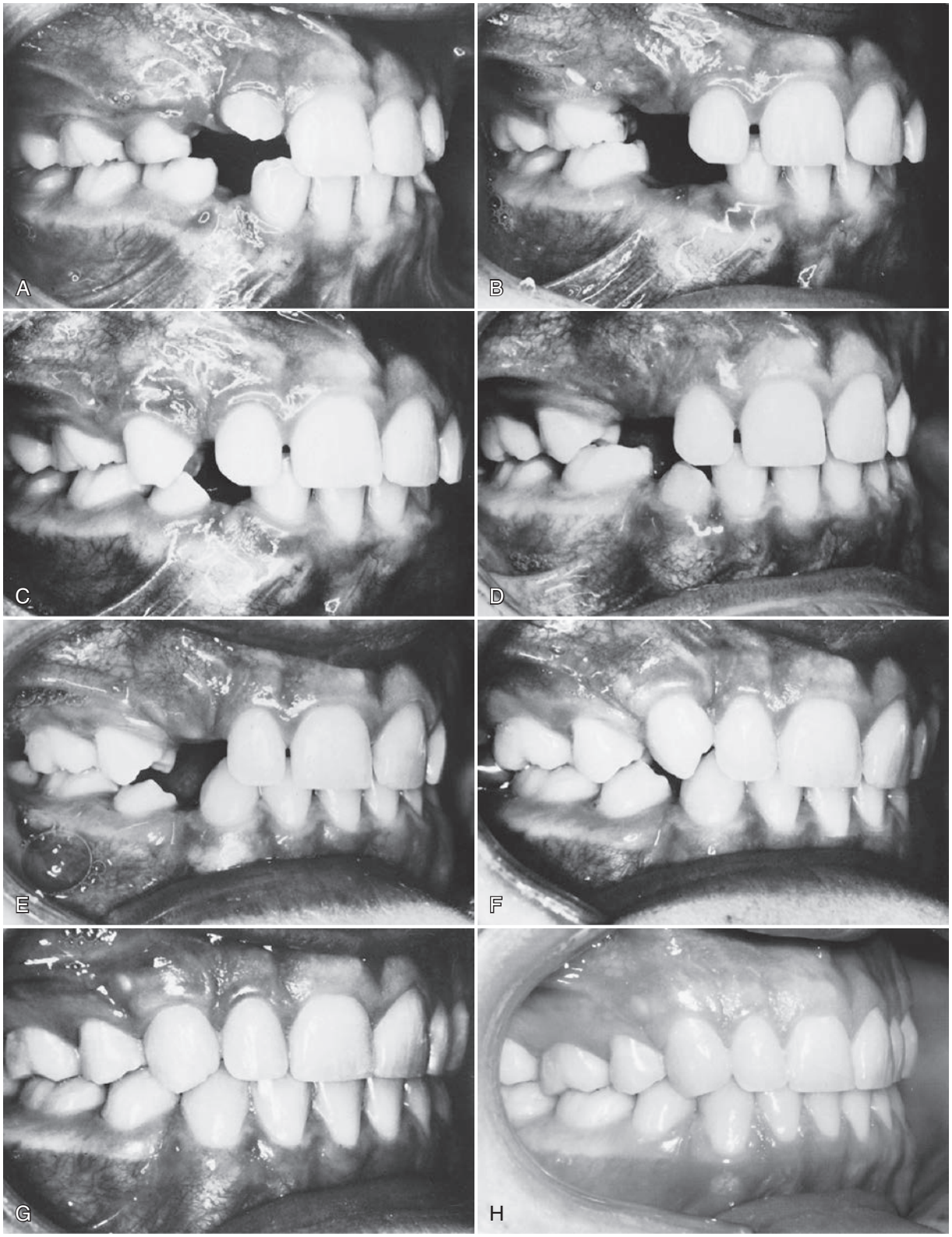


FIGURE 34-86 Patient M.R. **A,** At initial examination. The primary canines have been extracted. **B,** After extraction of primary first molars. **C,** Before extraction of the first premolars. **D,** Emergence of the permanent mandibular canines. **E,** Emergence of the second premolars. **F,** Before fixed appliance treatment. **G,** After fixed appliance treatment. **H,** Fifteen years after treatment. **I,** Response during interceptive guidance. **J,** Considerably less response during edgewise treatment. **K,** Response as indicated by the five cephalometric tracings (*1–5*) that were taken in each step in the treatment: *1,* before treatment; *2,* after extraction of the primary canines; *3,* after extraction of the primary first molars; *4,* after extraction of the first premolars; and *5,* after fixed appliance treatment.

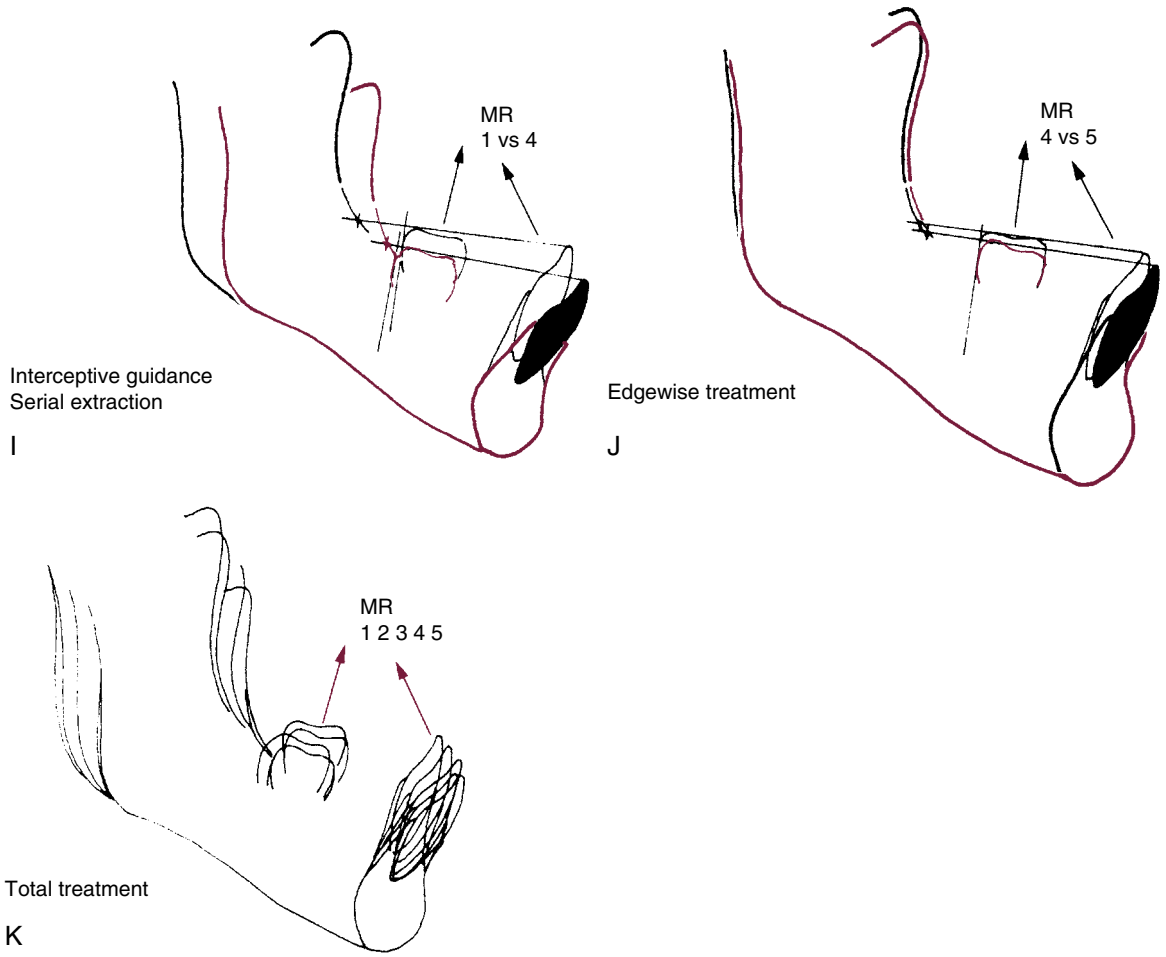


FIGURE 34-86—CONT'D

the patient came for examination and consultation is presented in Figure 34-86, *A*. The primary canines already had been extracted. In such patients, the clinician extracts the primary first molars to encourage the emergence of the first premolars so that they can be extracted as early as possible. This corrects the alveolodental protrusion by upright positioning of the incisor teeth.

With a relatively straight alignment of the incisor teeth, one does not need to extract the primary canines early. In Figure 34-86, *B*, the primary first molars have been extracted. Note the early eruption of the maxillary first premolar. A large restoration and a periapical infection of the primary molars possibly were factors in this situation. The orthodontist should wait until the other premolars have emerged into the oral cavity before advising extraction of these teeth (see Fig. 34-86, *B*). Note that in Figure 34-86, *C*, the four premolars have been extracted, and the incisor teeth are beginning to become upright over basal bone. In Figure 34-86, *D*, the permanent mandibular canines are beginning to emerge into the oral cavity. Note that the maxillary right primary second molar has been lost early. The premolar has emerged, and the first molar has drifted somewhat mesially. In Figure 34-86, *E*, all the primary second molars have now been lost, and the second premolars are emerging. Note the further upright positioning of the incisor teeth. With the emergence of the permanent maxillary canines and second molars, this patient is now ready for fixed appliance treatment (see Fig. 34-86, *F*). Figure 34-86, *G*, illustrates the end result, and *H*, the occlusion 15 years after retention. Cephalometric radiographs were taken of patient M.R. at the following times:

1. Before treatment
2. After extraction of the primary canines
3. After extraction of the primary first molars
4. After extraction of the permanent first premolars
5. After edgewise treatment

The response to serial extraction during interceptive guidance—superimpositions of cephalometric tracings numbers 1 and 4 (see Fig. 34-86, *I*)—is significantly greater than the response during the edgewise treatment—superimposition of cephalometric tracings 4 and 5 (see Fig. 34-86, *J*).

Figure 34-86, *K*, illustrates the response during the total treatment: interceptive guidance and edgewise appliance. The mandibular incisors gradually move upright, reducing the alveolar dental protrusion; the mandibular molars move upward and forward, accounting for an upward and forward rotation of the mandible.

Figure 34-87 presents the facial appearance before and after serial extraction and fixed appliance treatment. Note that this patient before treatment has a moderate high-angle facial pattern and alveolodental protrusion. After treatment the facial configuration exhibits harmony and balance. The smile has improved after serial extraction and again after multibanded treatment. Balance and harmony persist 15 years after treatment.

FIGURE 34-87 Facial appearance of patient M.R. Before (**A** and **B**) and after (**C** and **D**) serial extraction. **E** and **F,** After the fixed appliance treatment. **G** and **H,** Fifteen years after treatment. Cephalometric analysis of M.R. **I,** Before serial extraction. **J,** After serial extraction. **K,** After fixed appliance treatment. *ANB,* A point–nasion–B point; *FMIA*, Frankfort mandibular incisor axis angle; *IMPA*, incisor mandibular plane angle; *OP*, occlusal plane; *SNA*, sella-nasion-subspinale; *SNB*, sella-nasion-supramentale; *Z*, Z angle. The angle between the Frankfort Plane and Z Line (a line tangent to the chin and the vermilion border of both lips).

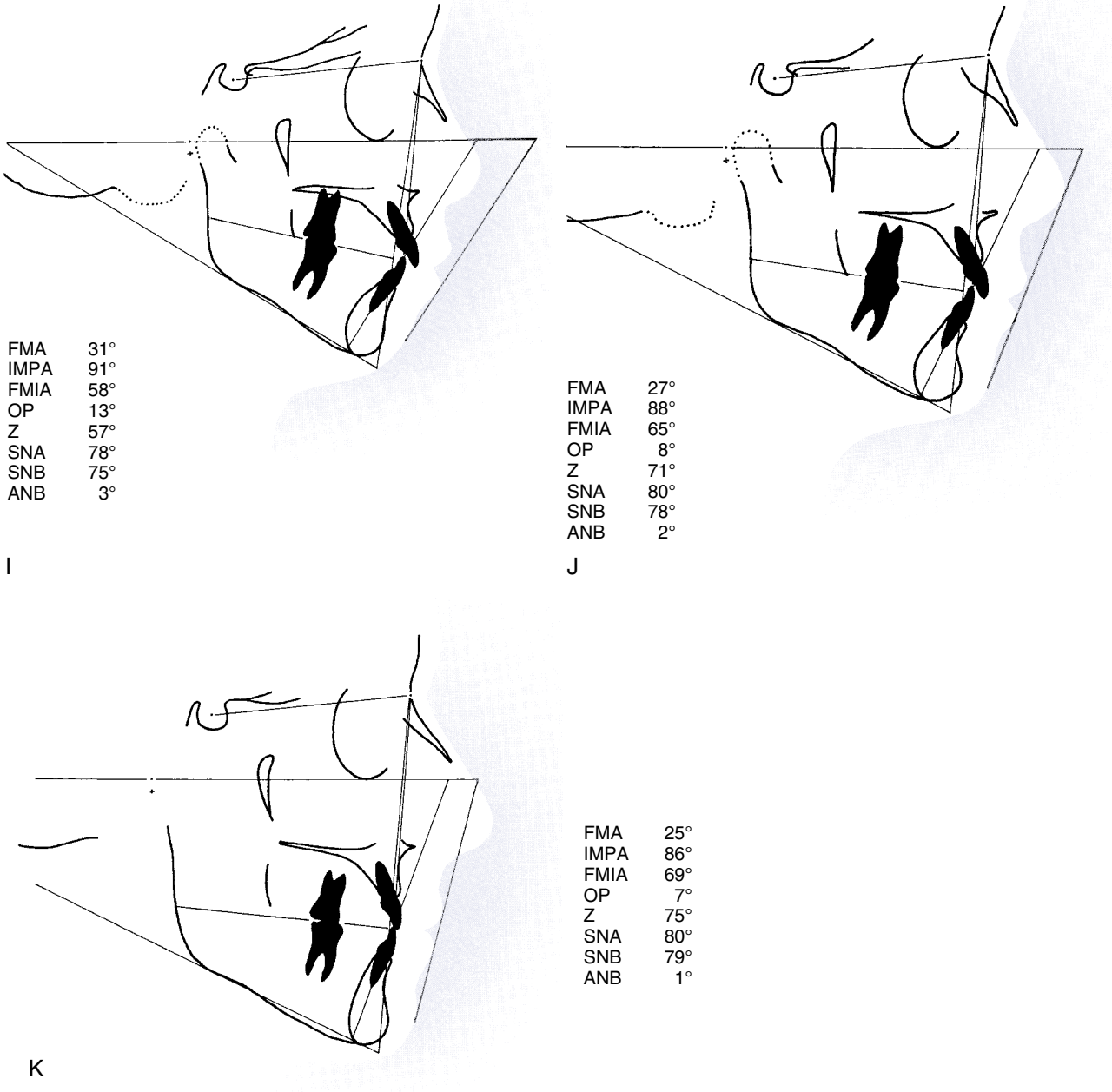


FIGURE 34-87—CONT'D

Before treatment, the cephalometric analysis of patient M.R. showed an FMA of 31 degrees. This represents a high-angle face. Throughout the course of serial extraction, the FMA was reduced to 27 degrees and throughout the course of fixed appliance treatment to 25 degrees. The 6-degree reduction indicated a favorable upward and forward rotation of the mandible. The patient's IMPA before treatment was 91 degrees, suggesting a moderate alveolodental protrusion for a high-angle facial pattern. During serial extraction, the mandibular incisors were positioned upright to 88 degrees and during active treatment to 86 degrees. This reduction of 5 degrees in the IMPA resulted in a slightly more balanced and harmonious soft tissue profile. The overall increase in the FMIA of 11 degrees (from 58 to 69 degrees) reflected a reduction in the FMA and the IMPA and a favorable rotation of the mandible and correction of the alveolodental protrusion. The reduction of the OP angle from 13 to 8 degrees during serial extraction indicated a mesial migration of the posterior teeth and an upright positioning of the mandibular incisor teeth, both favorable responses in the treatment of this particular facial pattern. The reduction of the OP angle from 8 to 7 degrees during active treatment indicated good control. The overall increase in the Z angle of 18 degrees (from 57 to 75 degrees) reflected a favorable rotation of the mandible and correction of the alveolodental protrusion. The maxilla grew slightly during the serial extraction period from 78 to 80 degrees and remained relatively constant during active treatment. Mandibular growth and rotation accounted for

3 degrees during the serial extraction period and for 1 more degree during the active period of treatment. Throughout total treatment, the ANB was reduced from 3 degrees to 1 degree, indicating a modest improvement in the jaw relationship (see Fig. 34-87, *I–K*).

Important in patient M.R.'s case was that the tooth size–jaw size discrepancy not be corrected by extension of the teeth posteriorly, anteriorly, laterally, or vertically. If this had been done in such a high-angle facial pattern, the molar extrusion would have resulted in an unfavorable downward and backward rotation of the mandible. With extrusion of the molars and the dumping forward of the incisors, the OP would have been tipped unfavorably and the alveolodental protrusion worsened. With the mandibular rotation and the tipped OP, the maxillary incisor would have been rabbited downward and backward, revealing more gingival tissue. Concomitant effects include impaction of the posterior molars and expanded dental arches. Furthermore, the mandibular incisors would have been tipped off basal bone, creating an unstable situation that required prolonged retention. The harmony and balance of the facial profile could have been worsened considerably.[56]

The total space analysis indicated an anterior and posterior space discrepancy. The anterior discrepancy was corrected by serial extraction and extraction of the permanent first premolars. The posterior discrepancy was corrected by extraction of the permanent third molars.

KEY MEASUREMENTS FOR DIAGNOSIS AND CASE EVALUATION

The Tweed Foundation has conducted several studies on the difference between successful and unsuccessful treatment.[57–63] They have indicated six measurements that are crucial in the evaluation of this difference: FMA, ANB, Z angle, OP, sella-nasion-supramentale (SNB), and the ratio of the posterior face height–to–anterior face height ratio (PFH/AFH) (Box 34-1). We have designed a "wiggle" to illustrate successful treatment (Fig. 34-88). Patient M.R.'s six measurements before serial extraction (see Fig. 34-88, *A*) were to the left of the wiggle, in the retrognathic, high-angle vertical growth area: FMA = 31, ANB = 3, Z angle = 57, OP = 13, SNB = 75, and PFH/AFH = 0.65.

After serial extraction (see Fig. 34-88, *B*), all six measurements were within the normal range: FMA = 27, ANB = 2, Z

BOX 34-1 Six Crucial Measurements for Treatment Success

FMA (Frankfurt mandibular plane angle) Indicates mandibular response. In most patients, successful treatment is associated with a favorable counterclockwise rotation indicated by a reduction of FMA.

ANB (A point–nasion–B point) angle Indicates the correction of a skeletal discrepancy. Successful treatment is associated with a reduction in ANB in most instances.

Z angle Indicates the alveolar dental and soft tissue profile response. With successful treatment, improvement in the soft tissue balance and harmony and reduction in the alveolar dental protrusion occur. This is associated with an increase in the Z angle.

OP (occlusal plane) Indicates the control of treatment. If the mandibular molars are prevented from extruding and the mandibular incisors are not "dumped" forward, the occlusal plane does not tip forward. If anchorage is prepared and the incisors are positioned upright, the OP decreases. Successful treatment is associated with a flattening of the OP.

SNB (sella-nasion-supramentale) angle Indicates the mandibular response. In most patients, if the treatment is successful, the SNB angle will increase.

PFH/AFH (posterior facial height/anterior facial height) Indicates the mandibular response. In successful treatment the PFH increases relatively more than the AFH; therefore, the ratio increases.

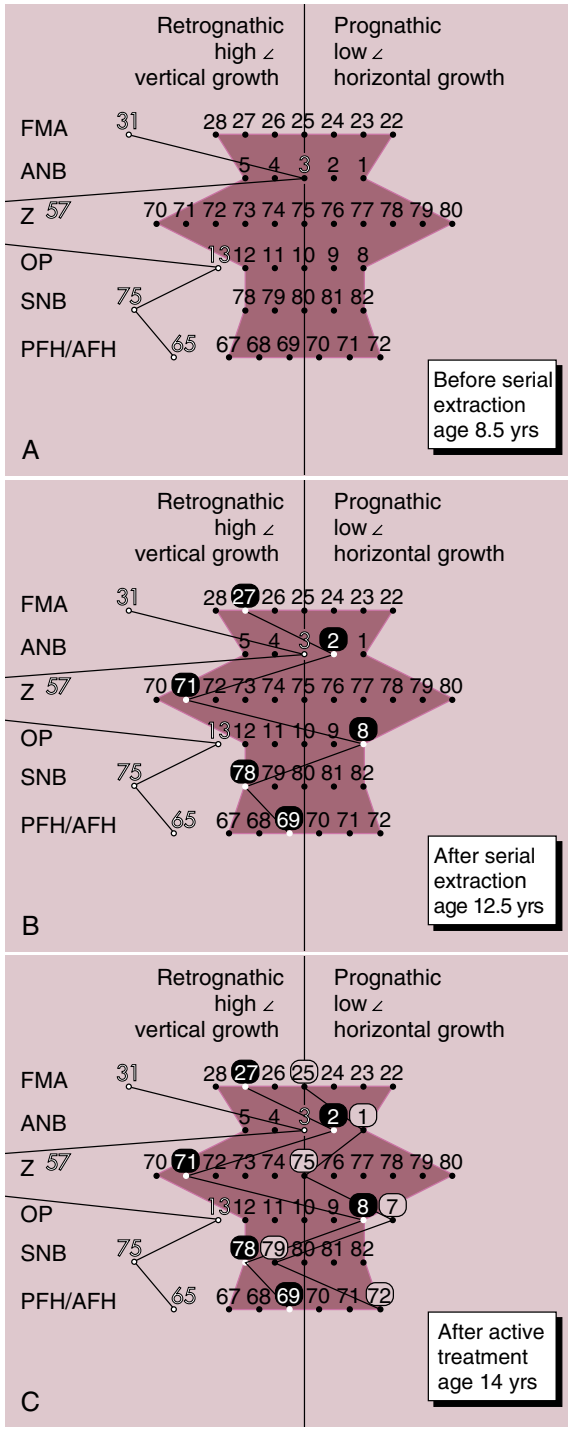


FIGURE 34-88 **A,** The graphic "wiggle" for patient M.R. before serial extraction. **B,** The wiggle for patient M.R. after serial extraction. **C,** The combined graphic wiggle for patient M.R. Note the improved measurements after both serial extraction and fixed appliances. *ANB,* A point–nasion–B point; *FMA,* Frankfurt mandibular plane angle; *OP,* occlusal plane; *PFH/ANH,* posterior face height–to–anterior face height ratio; *SNB,* sella-nasion-supramentale; *Z,* Z angle.

angle = 71, OP = 8, SNB = 78, and PFH/AFH = 0.69. Clinically, this is a highly successful response, especially without mechanotherapy.

After edgewise treatment (see Fig. 34-88, *C*), the measurements increased slightly but remained within the normal range, except for one reading, the OP, which was overcorrected slightly.

> *When facial deformity has been corrected and mental anguish eliminated, a dull and unhappy facial expression becomes bright and happy. What greater reward could any orthodontist want or expect?*
>
> ***—Charles H. Tweed***

The earlier this beneficial result occurs, the better.

ACKNOWLEDGMENT

The concepts behind guided extraction and orthodontic treatment have been developed over a period of time and are reflected herein to be most current. Over the years, there have been a number of "champions" of the technique who worked hard to teach the concepts involved. Dr. Warren R. Mayne, one of the original champions, worked with Dr. Tom Graber and contributed the guided extraction chapter in the first and second editions of this text. Both the authors and the editors recognize his initial contributions. We are all happy to see the concepts underlying guided extraction still relevant for today's clinician.
https://coursewareobjects.elsevier.com/objects/elr/ExpertConsult/Graber/orthodontics6e/extras/

REFERENCES

1. Kjellgren B. Serial extraction as a corrective procedure in dental orthopedic therapy. *Eur Orthod Soc Trans*. 1947–1948:134.
2. Kjellgren B. Serial extraction as a corrective procedure in dental orthopaedic therapy. *Eur J Orthod*. 2007;29(Suppl 1):37–50.
3. Hotz R. Guidance of eruption versus serial extraction. *Am J Orthod*. 1970;58:1.
4. Graber TM, Eliades T, Athanasiou A. *Risk Management in Orthodontics*. Chicago: Quintessence; 2004.
5. Bunon R. *Essay sur les maladies des dents; ou I'on propose les moyens de leur procurer une bonne confirmation des la plus tendre enfance, et d'en assurer la conservation pendant tout le cours de la vie*. Paris: 1743.
6. Wagers LE. Preorthodontic guidance and the corrective mixed-dentition treatment concept. *Am J Orthod*. 1976;69:1.
7. Moskowitz E. Consultations in the "real world". *Am J Orthod Dentofac Orthop*. 2005;127(3):358–359.
8. American Board of Orthodontics. *Specific instructions for candidates*; 2016. St. Louis www.americanboardortho.com/orthodontic-professionals/about-board-certification/clinical-examination/case-record-preparation/photograph-requirements.
9. Steiner C. Cephalometrics in clinical practice. *Angle Orthod*. 1959;29:8.
10. Merrifield LL. The profile line as an aid in critically evaluating facial esthetics. *Am J Orthod*. 1966;52:804.
11. Tweed CH. Treatment planning and therapy in the mixed-dentition. *Am J Orthod*. 1963;49:900.
12. Enlow DH, Hans MG. *Essentials of Facial Growth*. 2nd ed. Ann Arbor, MI: Needham Press; 2008.
13. Tweed CH. The diagnostic facial triangle in the control of treatment objectives. *Am J Orthod*. 1969;55:651.
14. Bills DA, Handelman CS, BeGole EA. Bimaxillary dentoalveolar protrusion: traits and orthodontic correction. *Angle Orthod*. 2005;75(3):333–339.
15. Hodges A, Rossouw PE, Campbell PM, et al. Prediction of lip response to four first premolar extractions in white female adolescents and adults. *Angle Orthod*. 2009;79(3):413–421.
16. McNamara JA, Brudon WL. *Orthodontics and Dentofacial Orthopedics*. Ann Arbor, MI: Needham Press; 2004:54.
17. *Burlington Orthodontic Research Project*. University of Toronto: Faculty of Dentistry, report no. 3; 1957.
18. Yoshihara T, Matsumoto Y, Suzuki J, et al. Effect of serial extraction alone on crowding: relationships between tooth width, arch length, and crowding. *Am J Orthod Dentofac Orthop*. 1999;116:691–696.
19. Yoshihara T, Matsumoto Y, Suzuki J, et al. Effect of serial extraction alone on crowding: spontaneous changes in dentition after serial extraction. *Am J Orthod Dentofac Orthop*. 2000;118:611–616.
20. Moorrees CF. Changes in dental arch dimension expressed on the basis of tooth eruption as a measure of biologic age. *J Dent Res*. 1965;44:129.
21. DeKock WJ. Dental arch depth and width the studies longitudinally from 12 years of age to adulthood. *Am J Orthod*. 1972;62:56.
22. Brodie A. Late growth changes in the human face. *Angle Orthod*. 1953;23:146.
23. Enlow DH. *The Human Face: An Account of the Postnatal Growth and Development of the Craniofacial Skeleton*. New York: Paul B. Hoeber; 1968.
24. Bjork A. Variations in the growth pattern of the human mandible: longitudinal radiographic study by the implant method. *J Dent Res*. 1963;42:400.
25. Lo RT, Moyers RE. Studies on the etiology and prevention of malocclusion. I. The sequence of eruption of the permanent dentition. *Am J Orthod*. 1953;39:460.
26. Horowitz SL, Hixon EH. *The Nature of Orthodontic Diagnosis*. St. Louis: Mosby; 1966.
27. Arita M, Iwagaki H. *Studies on the Serial Observations of Dental-Facial Region in the Japanese Children*. Tokyo: Nihon University School of Dentistry; 1963.
28. Hixon EH, Oldfather RE. Estimation of the sizes of unerupted cuspid and bicuspid teeth. *Angle Orthod*. 1958;28:236.
29. Lewis SM, Lehman IA. A quantitative study of the relation between certain factors in the development of the dental arch and the occlusion of the teeth. *Int J Orthod*. 1932;18:1015.
30. Moorrees CF, Chadha JM. Crown diameters of corresponding tooth groups in deciduous and permanent dentition. *J Dent Res*. 1962;41:466.
31. Jensen E, Kai-Jen Yen P, Moorrees CF. Mesiodistal crown diameters of the deciduous and permanent teeth in individuals. *J Dent Res*. 1957;36(1):39.
32. Baume LJ. Physiological tooth migration and its significance for the development of occlusion. III. The biogenesis of the successional dentition. *J Dent Res*. 1950;29:338.
33. Moorrees CF. Growth changes of the dental arches: a longitudinal study. *J Can Dent Assoc*. 1958;24:449.
34. Moorrees CF, Chadha JM. Available space for the incisors during dental development: a growth study based on physiological age. *Angle Orthod*. 1965;35:12.
35. Mayne WR. Serial extraction. In: Graber TM, ed. *Current Orthodontic Concepts and Techniques*. Philadelphia: WB Saunders; 1969.
36. Black GV. *Descriptive Anatomy of the Human Teeth*. 5th ed. Philadelphia: SS White Dental; 1902.
37. Baker C. Development of the occlusion of the teeth. *J Am Dent Assoc*. 1944;31:1470.
38. Tweed CH. *Clinical Orthodontics*. St. Louis: Mosby; 1966.
39. Merrifield LL. Differential diagnosis with total space analysis. *J Charles H Tweed Int Found*. 1978;6:10.
40. Garcia-Hernandez J, Dale JG. *Facial Considerations in Mixed-Dentition Analysis in Preparation for Guidance of Occlusion*. Postgraduate thesis, University of Toronto; 1979.
41. Huckaba GW. Arch size analysis and tooth size prediction. *Dent Clin North Am*. 1964:431.
42. Fanning EA. Effect of extraction of deciduous molars on the formation and eruption of their successors. *Angle Orthod*. 1962;32:44.
43. Rowland P, Poling R, Slater D, Hobsion R, Steen N. Can crown-root ratio predict premolar eruption? *Am J Orthod*. 2006;129(3):331–336.
44. Merrifield LL. The systems of directional force. *J Charles H Tweed Int Found*. 1982;10:15.
45. Jacobson A, Miller MA. A cephalometric assessment of children treated with two serial extraction patterns followed by fixed orthodontic treatment. *Am J Orthod Dentofac Orthop*. 2006;129(4):590.
46. Northway W. Hemisection. One large step toward management of congenitally missing lower second premolars. *Angle Orthod*. 2004;74(6):792–799.

47. Northway W. The nuts and bolts of hemisection treatment: managing congenitally missing mandibular second premolars. *Am J Orthod Dentofacial Orthop*. 2005;127(5):606–610.
48. Valencia R, Saadia M, Grinberg G. Controlled slicing in the management of congenitally missing second premolars. *Am J Orthod Dentofac Orthop*. 2004;125:537–543.
49. Beyer A, Tausche E, Boening K, Harzer W. Orthodontic space opening in patients with congenitally missing lateral incisors. *Angle Orthod*. 2007;77(3):404–409.
50. Sayinsu K, Nalbantgil D. Orthodontic treatment of a patient with traumatic loss of maxillary incisors. *World J Orthod*. 2008;9(1):43–47.
51. Tausche E, Harze W. Treatment of a patient with Class II malocclusion, impacted maxillary canine with a dilacerated root, and peg-shaped lateral incisor. *Am J Orthod*. 2008;133(5):762–770.
52. Kokich VG, Crabill KE. Managing the patient with missing or malformed maxillary central incisors. *Am J Orthod*. 2006;129(4 Suppl):S55–S63.
53. Garn SM, Lewis AB. The gradient and the pattern of crown-size reduction in simple hypodontia. *Angle Orthod*. 1970;40:51.
54. Geron S, Shpack N, Kandos S, et al. Anchorage loss—a multifactorial response. *Angle Orthod*. 2003;73(6):730–773.
55. Kau CH, Durning P, Richmond S, et al. Extractions as a form of interception in the developing dentition: a randomized controlled trial. *J Orthod*. 2004;31:107–114.
56. Basciftci FA, Usumez S. Effects of extraction and nonextraction treatment on Class I and Class II subjects. *Angle Orthod*. 2003;73(1):36–42.
57. Gebek TR, Merrifield LL. Orthodontic diagnosis and treatment analysis: concepts and values, part I. *Am J Orthod Dentofac Orthop*. 1995;107:434.
58. Gramling JF. A cephalometric appraisal of the results of orthodontic treatment on 50 unsuccessfully corrected difficult Class II malocclusions. *J Charles H Tweed Int Am J Orthod Dentofacial OrthopFound*. 1987;15:102.
59. Gramling JF. A cephalometric appraisal of the results of orthodontic treatment on 50 unsuccessfully corrected difficult Class II malocclusions. *J Charles H Tweed Int Am J Orthod Dentofacial Orthop Found*. 1987;15:112.
60. Gramling JF. The probability index. *Am J Orthod Dentofac Orthop*. 1995;107:165.
61. Horn AJ. Facial height index. *Am J Orthod Dentofac Orthop*. 1992;102:180.
62. Merrifield LL. Dimensions of the denture: back to basics. *Am J Orthod Dentofac Orthop*. 1994;106:535.
63. Merrifield LL, Klontz HK, Vaden JL. Differential diagnostic analysis system. *Am J Orthod Dentofac Orthop*. 1994;106:641.

ADDITIONAL READINGS

Al-Jasser NM, Bello LL. Time of eruption dentition in Saudi children. *J Contemp Dent Pract*. 2003;4(3):65–75.
Baume LJ. Physiological tooth migration and its significant for the development of occlusion. I. The biogenetic course of the deciduous teeth. *J Dent Res*. 1950;29:123.
Baume LJ. Physiological tooth migration and its significance for the development of occlusion. II. The biogenesis of accessional dentition. *J Dent Res*. 1950;29:331.
Baume LJ. Physiological tooth migration and its significance for the development of occlusion. IV. The biogenesis of overbite. *J Dent Res*. 1950;29:440.
Baume LJ. Developmental and diagnostic aspects of the primary dentition. *Int Dent J*. 1959;9:349.
Baumrind S, Korn EL, Boyd RL, Maxwell R. The decision to extract. I. Interclinician agreement. *Am J Orthod Dentofac Orthop*. 1996;109:297–309.
Baumrind S, Korn EL, Boyd RL, Maxwell R. The decision to extract. II. Analysis of clinicians' stated reasons for extraction. *Am J Orthod Dentofac Orthop*. 1996;109:393–402.
Becker A, Sharabi S, Chaushu S. Maxillary tooth size variation in dentition with palatal canine displacement. *Eur J Orthod*. 2002;24(3):313–318.
Bishara SE, Chadha JM, Potter RB. Stability of intercanine width, overbite, and overjet correction. *Am J Orthod*. 1973;62:588–594.
Bishara SE, Cummings DM, Jakobsen JR, et al. Dentofacial and soft tissue changes in Class II Division I cases treated with and without extractions. *Am J Orthod Dentofac Orthop*. 1995;107:28–37.
Bishara SE, Khadivi P, Jakobsen JR. Changes in tooth size-arch length relationships from the deciduous to the permanent dentition: a longitudinal study. *Am J Orthod Dentofac Orthop*. 1995;108:607–613.
Bishara SE, Jakobsen JR, Treder J, et al. Arch width changes from 6 weeks to 45 years of age. *Am J Orthod Dentofac Orthop*. 1997;111:401–409.
Bogaerts K, Leroy R, Lesaffre E, et al. Modelling tooth emergence data based on multivariate interval-censored data. *Stat Med*. 2002;21(24):37775–37787.
Boley JC. Serial extraction revisited: 30 years in retrospect. *Am J Orthod Dentofac Orthop*. 2002;121(6):575–577.
Bolton WA. The clinical application of a tooth size analysis. *Am J Orthod*. 1962;48:504.
Bowman SJ, Johnston LE. The esthetic impact of extraction and nonextraction treatments on Caucasian patients. *Angle Orthod*. 2000;70(1):3–10.
Cassidy KM, Harris EF, Tottey EA, et al. Genetic influence on dental arch form in orthodontic patients. *Angle Orthod*. 1998;68:445–454.
Chaushu S, Sharabi S, Becker A. Dental morphologic characteristics of normal versus delayed developing dentitions with palatally displaced canines. *Am J Orthod Dentofac Orthop*. 2002;12(4):339–346.
Chaushu S, Sharabi S, Becker A. Tooth size in dentitions with buccal canine ectopia. *Eur J Orthod*. 2003;25(5):485–491.
Chaushu S, Zilberman Y, Becker A. Maxillary incisor impaction and its relationship to canine displacement. *Am J Orthod Dentofac Orthop*. 2003;124(2):144–150.
Choi NK, Yang KH. A study on the eruption timing of primary teeth in Korean children. *J Dent Child*. 2001;68(4):244–249.
Clifton OW. Lower first premolar extraction: a viable alternative? *Am J Orthod Dentofac Orthop*. 1986;90(2):158–163.
Dale H. *A cephalometric evaluation of the curve of Spee in relation to facial types assessed in the vertical direction*. Master's thesis, Los Angeles, University of Southern California; 1990.
Dale H. *A severe Class II malocclusion treated by serial extraction and Tweed-Merrifield edgewise mechanotherapy analysed by the "wiggle" and "difficulty" procedures*. Paris: Presented at the twenty-first biennial meeting of the Charles H Tweed International Foundation; 1996.
Dale J, JCO interviews. Dr. Jack G. Dale on serial extraction. *J Clin Orthod*. 1976;10:44.
Dale J, VIP interview. Jack G. Dale. *World J Orthod*. 2006;7(1):95–99.
Dewel BF. Serial extractions in orthodontics: indications, objections, and treatment procedures. *Int J Orthod*. 1954;40:906.
Dewel BF. A critical analysis of serial extraction in orthodontic treatment. *Am J Orthod*. 1959;45:424.
Dewel BF. Serial extraction: its limitations and contraindications in orthodontic treatment. *Am J Orthod*. 1967;53(12):904–921.
Dewel BF. Extraction in orthodontic: premises and prerequisites. *Angle Orthod*. 1973;43(1):65–87.
Diamanti J, Townsend GC. New standards for permanent tooth emergence in Australian children. *Aust Dent J*. 2003;48(1):39–42.
Dibbets JM, van der Weele LT. Long-term effects on orthodontic treatment, including extraction, on signs and symptoms attributed to CMD. *Eur J Orthod*. 1992;14(1):16–20.
Elstramd KR, Christiansen J, Christiansen ME. Time and duration of eruption of first and second permanent molars: a longitudinal investigation. *Commun Dent Oral Epidemiol*. 2003;31(5):344–350.
Enlow DH, Dale JG. Childhood facial growth and development. In: Ten Cate AR, ed. *Oral Histology: Development, Structure, and Function*. 5th ed. St. Louis: Mosby; 1998.
Garfinkle RL, Artese A, Kaplan RG, et al. Effect of extraction in the late mixed-dentition on the eruption of the first premolar in *Macaca nemestrina*. *Angle Orthod*. 1980;50:23–27.
Gianelly AA, Hughes HM, Wohlgemuth P, Gildea G. Condylar position and extraction treatment. *Am J Orthod Dentofac Orthop*. 1988;93(3):201–205.
Gianelly AA, Anderson CK, Boffa J. Longitudinal evaluation of condylar position in extraction and nonextraction treatment. *Am J Orthod Dentofac Orthop*. 1991;100(5):416–420.
Gilmore CA, Little RM. Mandibular incisor dimensions and crowding. *Am J Orthod*. 1984;86(6):493–502.

Graber TM. Serial extraction: a continuous diagnostic and decisional process. *Am J Orthod*. 1971;60(6):541–575.
Greer GW, Artese A, Gutierrez A, et al. Effect of extraction in the early mixed-dentition on the eruption of the first premolar in *Macaca nemestrina*. *Angle Orthod*. 1980;50:34.
Gron AM. Prediction of tooth the emergence. *J Dent Res*. 1962;41:573.
Hägg U, Taranger J. Maturation indicators and the pubertal growth spurt. *Am J Orthod*. 1982;82:299.
Harila-Kaera V, Heikkien T, Alvesalo L. The eruption of permanent incisors and first molars in prematurely born children. *Eur J Orthod*. 2003;25(3):293–299.
Heath J. The interception of malocclusion by planned serial extraction. *N Z Dent J*. 1953;49:77.
Heath J. Dangers and pitfalls of serial extraction. *Eur Orthod Soc Trans*. 1961;37:60.
Hotz R. Active supervision of the eruption of teeth by extraction. *Eur Orthod Soc Trans*. 1947–1948:34.
Jacobs J. Cephalometric and clinical evaluation of Class I discrepancy cases treated by serial extraction. *Am J Orthod*. 1965;51(6):401–411.
Jacobs SG. A re-assessment of serial extraction. *Aust Orthod J*. 1987;10(2): 90–97.
James RD. A comparative study of facial profiles in extraction and non extraction treatment. *Am J Orthod Dentofac Orthop*. 1998;114:265–276.
Jegou I. Indication and limits on the choice of extraction of 14–24 and 35–45 in treating Angle Class-II malocclusion by a Tweed-Merrifield analysis of the total space. *Orthod Fr*. 1984;55(2):485–494.
Jones O, Ortiz C, Schlein R. Orthodontic management of a patient with Class I malocclusion and severe crowding. *Am J Orthod Dentofac Orthop*. 1990;98(3):189–196.
Katona TR, Qian H. A mechanism of noncontinuous supraosseous tooth eruption. *Am J Orthod Dentofac Orthop*. 2001;120(3):263–271.
Kennedy DB, Joodeph DR, Osterberg SK, et al. The effect of extraction and orthodontic treatment on dentoalveolar support. *Am J Orthod*. 1983;84(3):183–190.
Kjellberg H, Beiring M, Albertsson Wikland K. Craniofacial morphology, dental occlusion, tooth eruption, and dental maturity in boys of short stature with or without growth hormone deficiency. *Eur J Oral Sci*. 2000;108(5):359–367.
Klapper L, Navarro SF, Bowman D, Pawlowski B. The influence of extraction and nonextraction orthodontic treatment on brachyfacial and dolichofacial growth patterns. *Am J Orthod Dentofac Orthop*. 1992;101(5):425–430.
Krogman WM. Biological timing and dento-facial complex. *J Dent Child*. 1968;35:175.
Kurol J. Early treatment of tooth-eruption disturbances. *Am J Orthod Dentofac Orthop*. 2002;121(6):588–591.
Leighton BC, Hunter WS. Relationship between lower arch spacing/crowding and facial height and depth. *Angle Orthod*. 1982;82:418.
Leonardi R, Annunziata A, Licciardello V, Barbato E. Soft tissue changes following the extraction of premolars in nongrowing patients with bimaxillary protrusion. *Angle Orthod*. 2010;80(1):211–216.
Little RM. The effects of eruption guidance and serial extraction on developing dentition. *Pediatr Dent*. 1987;9(1):65–70.
Little RM, Riedel RA, Artun J. An evaluation of changes in mandibular anterior alignment from 10-20 years post retention. *Am J Orthod Dentofac Orthop*. 1988;93:423–428.
Little RM, Wallen TR, Riedel RA. Stability and relapse of mandibular anterior alignment: first premolar extraction cases treated by traditional edgewise orthodontics. *Am J Orthod*. 1981;80:349–365.
Little RM, Riedel RA, Engst ED. Serial extraction of first premolars: postretention evaluation of stability and relapse. *Angle Orthod*. 1990;60(4):255–262.
Little RM. *Stability and Relapse: Early Treatment of Arch Length Deficiency*. Phoenix, AZ: International Symposium on Early Orthodontic Treatment; February 8–10, 2002.
Lloyd ZB. Serial extraction as a treatment procedure. *Am J Orthod*. 1956;42:728.
Luecke PE, Johnston LE. The effect of maxillary first premolar extraction and incisor retraction on mandibular position: testing the central dogma of "functional orthodontics". *Am J Orthod Dentofac Orthop*. 1992;101(1):4–12.
Luppanapornlarp S, Johnston LE. The effects of premolar extraction: a long-term comparison of outcomes in "clear-cut" extraction and non extraction Class II patients. *Angle Orthod*. 1993;63(4):257–272.
Marshall S, Dawson D, Southard KA, et al. Transverse molar movements during growth. *Am J Orthod Dentofac Orthop*. 2003;124(6):615–624.
McLaughlin RP, Bennett JC. The extraction-nonextraction dilemma as it relates to TMD. *Angle Orthod*. 1995;65(3):175–186.
McNamara JA, Brudon WL. *Orthodontic and Orthopedic Treatment in the Mixed Dentition*. 1st ed. Ann Arbor, MI: Needham Press; 1993.
McReynolds DC, Little SR. Mandibular second premolar extraction: postretention evaluation of stability and relapse. *Angle Orthod*. 1991;6(2):133–144.
Merrifield LL, Gebek TR. Orthodontic diagnosis and treatment analyses: concepts and values, part II. *Am J Orthod Dentofac Orthop*. 1995;107:541.
Moorrees CF. *The Dentition of the Growing Child: A Longitudinal Study of Dental Development Between 3 and 18 Years of Age*. Cambridge, MA: Harvard University Press; 1959.
Moorrees CF. Variability of dental and facial development. *Ann N Y Acad Sci*. 1966;134:846.
Moorrees CF, Fanning EA, Grön AM. Consideration of dental development in serial extraction. *Angle Orthod*. 1963;33:44.
Moorrees CF, Fanning EA, Grön AM, et al. Timing of orthodontic treatment in relation to tooth formation. *Eur Orthod Soc Trans*. 1962;38:87.
Moorrees CF, Fanning EA, Hunt Jr EE. Age variation of formation stages for ten permanent teeth. *J Dent Res*. 1963;42:1490.
Moorrees CF, Fanning EA, Hunt Jr EE. Formation and resorption of three deciduous teeth in children. *Am J Phys Anthropol*. 1963;21:99.
Moorrees CF, Kent Jr RL. Patterns of dental maturation. In: McNamara JA, ed. *The Biology of Occlusal Development. Monograph 6, Craniofacial Growth Series*. Ann Arbor, MI: University of Michigan; 1978.
Moorrees CF, Reed RB. Biometrics of crowding and spacing of the teeth in the mandible. *Am J Phys Anthropol*. 1954;12:77.
Moorrees CF, Reed RB. Correlations among crown diameters of human teeth. *Arch Oral Biol*. 1964;115:685–697.
Moorrees CF, Reed RB, Chadha JM. Growth changes of the dentition defined in terms of chronologic and biologic age. *Am J Orthod*. 1964;50:789.
Morrow LA, Robbins JW, Jones DL, et al. Clinical crown length changes from age 12-19 years: a longitudinal study. *J Dent*. 2000;28(7):469–473.
Mugonzibwa EA, Kuijpers-Jagtman AM, Laine-Alava MT, et al. Emergence of permanent teeth in Tanzanian children. *Commun Dent Oral Epidemiol*. 2002;30(6):455–462.
Mulvany DA. Space gain following the removal of first or second molars without orthodontic treatment—case reports. *Funct Orthod*. 2002;19(2):18–25.
Ngan PW, Kao EC, Wei SH. Guidance of eruption for general practitioners. *Int Dent J*. 2003;53(2):100–113.
Norman F. Serial extraction. *Angle Orthod*. 1965;35:149.
Odenrick L, Trocmé M. Facial, dentoalveolar and dental morphology in serial or early extraction. *Angle Orthod*. 1985;55(3):206–214.
Ortial JP. The Tweed technic: choice of extraction and treatment strategy I. *Attual Dent*. 1987;3(16):10–11.
Papandreas SG, Buschang PH, Alexander RG, et al. Physiologic drift of the mandibular dentition following first premolar extractions. *Angle Orthod*. 1993;63(2):127–134.
Paquette DE, Beattie JR, Johnston L. A long-term comparison of nonextraction and premolar extraction edgewise therapy in "borderline" Class II patients. *Am J Orthod Dentofac Orthop*. 1992;102:1–14.
Parner ET, Heidmann JM, Kjaer I, et al. Biological interpretation of the correlation of permanent teeth in Danish children. *Arch Oral Biol*. 2001;46(5):425–431.
Persson M, Persson E, Skagius S. Long-term spontaneous changes following removal of all first premolars in Class I cases with crowding. *Eur J Orthod*. 1989;11:271–282.
Psoter WJ, Morse DE, Pendrys DG, et al. Median ages or eruption of the primary teeth in white and Hispanic children in Arizona. *Pediatr Dent*. 2003;25(3):257–261.
Renfoe EW. The philosophy of extraction in orthodontics: 1966 (classified article). *Int J Orthod*. 1989;27(1-2):3–8.
Richardson ME. Late lower arch crowding in relation to primary crowding. *Angle Orthod*. 1982;52:300.

Richardson ME. The effect of mandibular first premolar extraction on third molar space. *Angle Orthod*. 1989;59(4):291–294.
Richardson ME. Late lower arch crowding: the role of the transverse dimension. *Am J Orthod Dentofac Orthop*. 1995;107:613–617.
Richardson ME. Late lower arch crowding: the aetiology reviewed. *Dent Update*. 2002;29(5):234–238.
Ringenberg QM. Influence of serial extraction on growth and development of maxilla and mandible. *Am J Orthod*. 1967;53(1):19–26.
Robertson NAE, Cavina R, Moss JP. The effect of the extraction of lower first premolars on the migration of teeth in the human dental arch. *J Dent Res*. 1979;58:1252–1256.
Rousset MM, Boualam N, Delfosse C. Occlusion and rhythm of eruption. *Bull Group Int Rech Sci Stomatol Odontol*. 2001;43(2):53–61.
Saatçi P, Yukey F. The effect of premolar extractions on tooth-size discrepancy. *Am J Orthod Dentofac Orthop*. 1997;111:428–434.
Sanin C, Savara B, Sekiguchi T. Longitudinal dentofacial changes in untreated persons. *Am J Orthod*. 1969;55:135.
Schirmer UR, Wiltshire WA. Manual and computer-aided space analysis: a comparative study. *Am J Orthod Dentofac Orthop*. 1997;112:676–680.
Schudy FF. The rotation of the mandible resulting from growth: its implications in orthodontic treatment. *Angle Orthod*. 1965;35:36.
Sharpe W, Reed B, Subtelny JD, et al. Orthodontic relapse, apical root resorption, and crestal alveolar bone levels. *Am J Orthod*. 1987;91:252–258.
Shearer DH, Woodside DG. Postretention mandibular incisor stability in serial premolar extraction cases. *Am J Orthod*. 1995;107(4):459.
Slaj M, Jezina MA, Lauc T, et al. Longitudinal dental arch changes in the mix dentition. *Angle Orthod*. 2003;73(5):509–514.
Staggers JA. A comparison of results of second molar and first premolar extraction treatment. *Am J Orthod Dentofac Orthop*. 1990;98(5):430–436.
Staggers JA. Vertical changes following first premolar extractions. *Am J Orthod Dentofac Orthop*. 1994;105(1):19–24.
Stahl F, Grabowski R. Maxillary cine displacement and genetically determined predisposition to disturbed development of the dentition. *J Orofac Orthop*. 2003;64(3):167–177.
Steyn CL, Harris AM, du Preez RJ. Anterior arch circumference: how much? *Angle Orthod*. 1996;66:457–461.
Surbeck BT, Artun J, Hawkins NR, et al. Association between initial post treatment and postretention alignment of maxillary anterior teeth. *Am J Orthod Dentofac Orthop*. 1998;113:186–195.
Taguchi Y, Yano Y, Kobayashi H, et al. Retarded eruption of maxillary second premolars associated with late development of the germs. *J Clin Pediatr Dent*. 2001;25(3):175–179.
Takanobu H, Little RM. Early versus late treatment of crowded first premolar extraction cases: post retention evaluation of stability and relapse. *Angle Orthod*. 1998;68:61–68.
Tanner JM. *Growth at Adolescence*. 2nd ed. Cambridge, MA: Blackwell Scientific; 1962.
Toshniwal NG. A review of serial extraction. *J Indian Dent Assoc*. 1990;61(12):291.
Tsai HH. Eruption process of the second molar. *J Clin Pediatr Dent*. 2000;67(4):275–281.
Tsai HH. Development changes of mandibular permanent canines and premolars in early mixed dentition. *J Clin Pediatr Dent*. 2001;26(1):61–64.
Tsai HH. Eruption process of upper permanent canine. *J Clin Pediatr Dent*. 2001;25(3):175–179.
Tung AW, Kiyak HA. Psychological influences on the timing of orthodontic treatment. *Am J Orthod Dentofac Orthop*. 1998;113:29–39.
Tweed CH. The Frankfort-mandibular plane angle in orthodontic diagnosis classification, treatment planning, and prognosis. *Am J Orthod Oral Surg*. 1946;32:175.
Tweed CH. The Frankfort-mandibular incisor angle in orthodontic diagnosis, treatment planning, and prognosis. *Angle Orthod*. 1954;24:121.
Tweed CH. Pre-orthodontic guidance procedure, classification of facial growth trends, treatment timing. In: Kraus BS, Riedel RA, eds. *Vistas in Orthodontics*. Philadelphia: Lea & Febiger; 1962.
Vaden JL, Kiser HE. Straight talk about extraction and non extraction: a differential diagnostic decision. *Am J Orthod Dentofac Orthop*. 1996;109:445–452.
Wagers LE. Preorthodontic guidance and the corrective mixed-dentition treatment concept. *Am J Orthod*. 1976;69(1):1–28.
Wagner M, Berg R. Serial extraction or premolar extraction in the permanent dentition? Comparison of duration and outcome of orthodontic treatment. *J Orofac Orthop*. 2000;61(3):207–216.
Wasserstein A, Shalish M. Adequacy of mandibular premolar position despite early loss of its deciduous molar. *J Dent Child*. 2002;69(3):233–234, 254–258.
Watanabe CT, Watanabe JI, Taguchi T, et al. Eruption disturbances of mandibular permanent incisors. *J Clin Pediatr Dent*. 2001;25(3):181–185.
Weinberg MA, Eskow RN. An overview of delayed passive eruption. *Compend Contin Educ Dent*. 2000;21(6):511–514.
Wilson JR, Little RM, Joondeph DR, Doppel DM. *(1999)* Comparison of soft tissue profile changes in serial extraction and late premolar extraction. *Angle Orthod*. 1999;69(2):165–172.
Wise GE, Frazier-Bowers S, D'Souza RN. Cellular, molecular, and genetic determinants of tooth eruption. *Crit Rev Oral Biol Med*. 2002;13(4):323–334.
Yamaguchi K, Nanda RS. The effects of extraction and nonextraction treatment on the mandibular position. *Am J Orthod Dentofac Orthop*. 1991;100(5):443–452.
Yang EY, Kiyak HA. Orthodontic treatment timing: a survey of orthodontists. *Am J Orthod Dentofac Orthop*. 1998;113:96–103.
Yoshihara T, Matsumoto Y, Suzukui J, et al. Effect of serial extraction alone on crowding: spontaneous changes in dentition after serial extraction. *Am J Orthod Dentofac Orthop*. 2000;118(6):611–616.
Yoshihara T, Matsumoto Y, Suzukui J, et al. Effect of serial extraction alone on crowding relationship between closure of residual extraction space and changes in dentition. *J Clin Pediatr Dent*. 2002;26(2):147–152.

35

Functional Appliances

*Thomas M. Graber**

OUTLINE

Origin, 997.e50
Andresen Activator, 997.e51
Bite-Opening Controversy, 997.e53
Head Posture during Sleep, 997.e53
Working Hypotheses, 997.e54
Sagittal Change, 997.e54
Vertical Opening Variations, 997.e56
Mechanisms of Class II Correction with Functional Orthopedics, 997.e57
Class II Correction: A Likely Scenario, 997.e59
Bionator, 997.e59
Construction Bite, 997.e62
Anterior Mandibular Posturing, 997.e63
Hamilton Expansion Activator, 997.e64
Fränkel Appliance, 997.e68
Double Plates, 997.e69
Clark Twin Block Appliance, 997.e70
Magnetic Appliances, 997.e71
Stöckli-Teuscher Combined Activator-Headgear Orthopedics, 997.e75
Fixed Functional Appliances, 997.e81
Herbst Appliance, 997.e81
Sagittal Changes, 997.e85
Vertical Changes, 997.e85
Long-Term Posttreatment Changes, 997.e88
Jasper Jumper, 997.e90
Summary, 997.e92

This "Classic Chapter in Orthodontics" is taken from our fourth edition of Orthodontics: Current Principles and Techniques published by Elsevier in 2005. It is presented as then written. The chapter contains perspectives and information that remain valuable to orthodontic residents and clinicians.

The stomatognathic system consists of the teeth, the periodontal membranes, alveolar and basal bone, the temporomandibular joints (TMJ), and the motivating and draping neuromusculature. This is a living, viable, and remarkably adaptive system, particularly during the period of growth and development of the craniofacial complex. Bone may be one of the hardest tissues in the human body, but it is also one of the most responsive to environmental stimuli. For example, persons have known for many centuries that the membranous bone cranial structures can be deformed by binding an infant's skull, as was done by the Inca Indians.[1] Pathologic manifestations can result in bizarre craniofacial asymmetries and deformities that involve cranial and mandibular structures. Practitioners can learn much about the normal condition from pathologic conditions.

Orthopedic surgeons have corrected skeletal endochondral deformities for many years, primarily in growing individuals. However, influencing endochondral bone is known to be a greater challenge. The chapters in this book by Thilander, Hatch, and Sun (Chapter 3) and Roberts and Huja (Chapter 4) describe the *raison d'être* for this tissue reaction, and the reader should become familiar with the minutiae to better understand important factors such as the way appliances work, the best time to use them, and their limitations. An orthodontic practitioner cannot help but be excited by the prospect of using the patient's own functional forces to achieve orthopedic and orthodontic correction of dentofacial abnormalities. Indeed, previous overconcentration on the mechanical aspects of orthodontics has been a barrier to the full realization of the magnitude of influence that is possible. Orthodontics is not only the appliance, but it is about which appliances, why, when, and for how long.

ORIGIN

Theories on bone plasticity may be traced to Wolff[2] and Roux,[3] who believed that form and function were intimately related. Changes in functional stress produced changes in internal bone architecture and external shape. Recent research has supported Roux's concept of functional "shaking of the bone," and the anabolic stimulus applied to achieve the optimal morphogenetic pattern.[4–6]

Early in the twentieth century, Pierre Robin of France introduced the plastic monobloc as a passive positioning device. This device was used in neonates with micromandibular development to prevent glossoptosis, which is literally a blocking of the airway by the tongue.[7] This congenital abnormality of development has been called Pierre Robin syndrome and is usually associated with cleft palate. The Robin appliance was modified from bite-jumping vulcanite maxillary anterior guide planes designed by Norman Kingsley.[8] However, the Kingsley guide plane was attached to teeth, whereas Robin had to use his monobloc as a removable device because newborns have no teeth.

*An article in memoriam of author Thomas M. Graber can be found at the following link: http://www.sciencedirect.com/science/article/pii/S0889540615008835

ANDRESEN ACTIVATOR

Viggo Andresen of Norway also was familiar with the writings of American authors Norman Kingsley[8] and Calvin Case.[9] Their use of bite-jumping appliances was common among other orthodontists at the end of the nineteenth century. Even Angle originally resorted to and recommended such appliances for patients with mandibular retrusion.[10] Also on Andresen's bookshelf was a favorite of his, the orthodontic textbook of Benno Lischer,[11] published in 1912. Lischer presented graphic illustrations of the effect of abnormal perioral muscle function on the teeth and investing tissues. One conclusion to be drawn from Lischer's theory is that if compensatory and adaptive lip and tongue function could exacerbate excessive overjet in types of Class II malocclusions and if abnormal swallowing and prolonged finger-sucking habits could create anterior open bite and narrow maxillary arches, then could not the same muscles be used to correct these and other problems? This theory was particularly true of patients with Class II, Division 1 conditions who had protruding maxillary teeth and retrusive lower dentition, a combination that created excessive overjet and evoked compensatory, adaptive function. Why not use these same deforming muscles to reverse the procedure? Holding the mandible forward long enough could reverse the deleterious effects of a trapped lower lip, lack of normal lip contact, and abnormal respiration, characteristics associated with malocclusion and illustrated as early as 1861 by artist George Catlin (cited in Angle[12].)

Actually, Andresen was not thinking of "guiding growth" at that time but only of eliminating the adverse effects of abnormal function. This working hypothesis was tested on his own daughter, who was wearing fixed orthodontic appliances and who was going away to camp over the summer. Andresen removed the fixed appliances and placed a modified Hawley-type retainer on the maxillary arch. However, he added a lingual horseshoe flange that guided the mandible 3 to 4 mm forward when the teeth were brought into maximal closure allowed by the interposed acrylic guide plane[13–16] (Fig. 35-1), which was done to prevent any relapse over the 3-month vacation period. On his daughter's return, Andresen was surprised to see that nighttime wearing of the appliance not only eliminated the abnormal neuromuscular compensations, but it also produced a complete sagittal correction and significantly improved the facial profile. The result was stable. When trying this system on numerous other growing patients who were going away for the summer and then on patients undergoing routine care throughout the year, Andresen observed significant sagittal basal bone and neuromuscular improvement that he had not been able to achieve with devices such as conventional fixed appliances and intermaxillary elastics.[14]

The sagittal changes that Andresen observed occurred because the appliance increased the activity of the protractor and elevator muscles, with concomitant relaxation and stretching of the retractors. The elimination of the abnormal perioral muscle function by mandibular protraction prevented the deforming and restrictive action of the trapped lower lip and the hyperactivity of the mentalis and submandibular muscle groups (Figs. 35-2 and 35-3). Compensatory mandibular opening caused by concomitant mouth breathing also could be eliminated. The change in muscle pattern would thus produce not only a new and more favorable pattern but also a change in bony structures as the muscles adapted to the new functional stresses (Fig. 35-4).[6,17–63]

The intermittent forces produced by the loose appliance were thought to create favorable changes in tooth position, even though the appliance was worn only at night. Later, it was postulated that the appliance had an added benefit; it was thought to serve as a night guard, preventing the potentially deleterious effects of nocturnal parafunctional activity, actually stimulating normal muscle activity and enhancing the metabolic pump activity of the retrodiskal pad. This repetitive pump activity increased blood flow into the highly vascular posterior attachment on guided mandibular protraction and forced out the catabolic by-products on mandibular retraction.[64,65] The obvious benefits of a protracted, unloaded condyle were hypothesized to be enhancement of condylar growth increments and a more favorable upward and backward condylar growth direction.[4] Fundamental tissue research by Bierbaek and colleagues,[66] Braun and associates,[67] Buschang and Santos-Pinto,[24] Chen and others,[68] Decrue and Wieslander,[69] Droel and Isaacson,[70] Du and colleagues,[27] El-Bialy and associates,[71] Garattini and others,[72] Graber,[65] Ikai and colleagues,[32] Kantomaa and Pirttiniemi,[34] McNamara and associates,[43] Pancherz,[47] Paulsen,[49,50,73] Pirttiniemi and others,[51] Popowich and colleagues,[74] Rabie and associates,[75] Ruf and Pancherz,[53] Vardimon and others,[76] Voudouris and colleagues,[77–79] Ward and associates,[80] Watted and others,[61] Woodside and colleagues,[81] and Xiong and associates[82] stresses the changing position of the glenoid fossa during growth and the potential for influencing it, even as the Incas changed growth eons ago.

As with Robin's monobloc, Andresen's first appliances were passive, having no intrinsic force systems. Only when the patient bit into the appliance would the extrinsic force be in

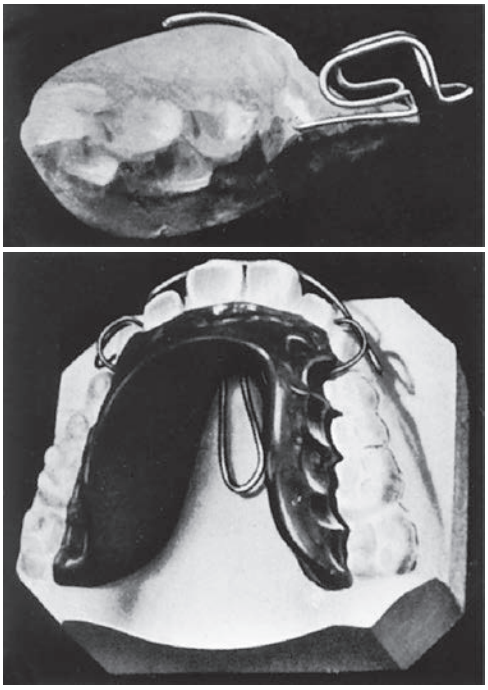


FIGURE 35-1 Andresen-Häupl activator. This device has no tooth-moving parts. The appliance was made to treat Class II, Division 1 malocclusions. Instead of palatal coverage, a heavy Coffin spring has been used to ensure stability and yet allow tongue contact with the mucosa. The loops in the canine region stand away from the teeth, allowing maxillary intercanine development. These loops are the forerunners of the Balters Bionator screening loops, which extend to the distal aspect of the deciduous second molar, and also of the Fränkel buccal shields, which are also meant to hold off cheek pressure. (From Andresen V, Häupl K. *Funktionskieferorthopädie: die Grundlagen des "Norwegischen Systems."* 2nd ed. Leipzig, Germany: H Meusser; 1936.

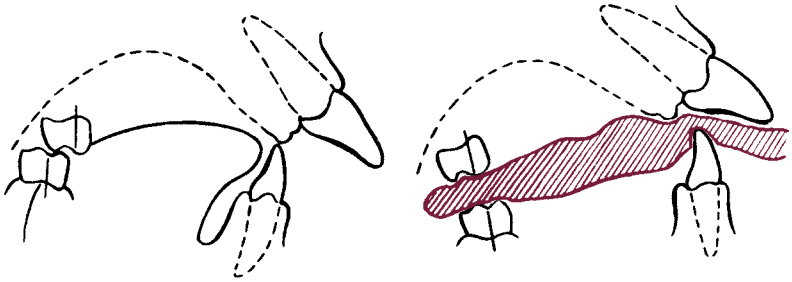


FIGURE 35-2 Construction bite. Correcting the buccal segment relationships from Class II to Class I is accomplished through mandibular advancement of approximately 6 mm. Note that the overjet is still excessive because of labially malposed maxillary incisors, which can be corrected after the buccal segment relationship has been normalized. An interocclusal clearance of 3 to 4 mm allows the mandible to come forward sufficiently to correct the buccal segment intercuspation. (Redrawn from Andresen V, Häupl K. *Funktionskieferorthopädie: die Grundlagen des "Norwegischen Systems."* 2nd ed. Leipzig, Germany: H Meusser; 1936.)

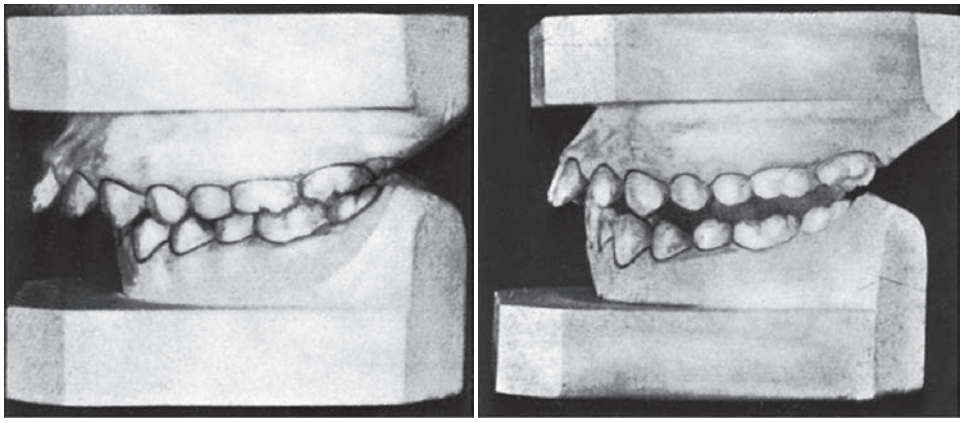


FIGURE 35-3 Lateral view of study models of the patient in Figure 35-2, a severe Class II, Division 1 malocclusion. The lingual inclination of the lower incisors is a favorable condition for functional therapy, as is the deeper-than-normal overbite. (From Andresen V, Häupl K. *Funktionskieferorthopädie: die Grundlagen des "Norwegischen Systems,"* 3rd ed. Leipzig, Germany: JA Barth; 1942.)

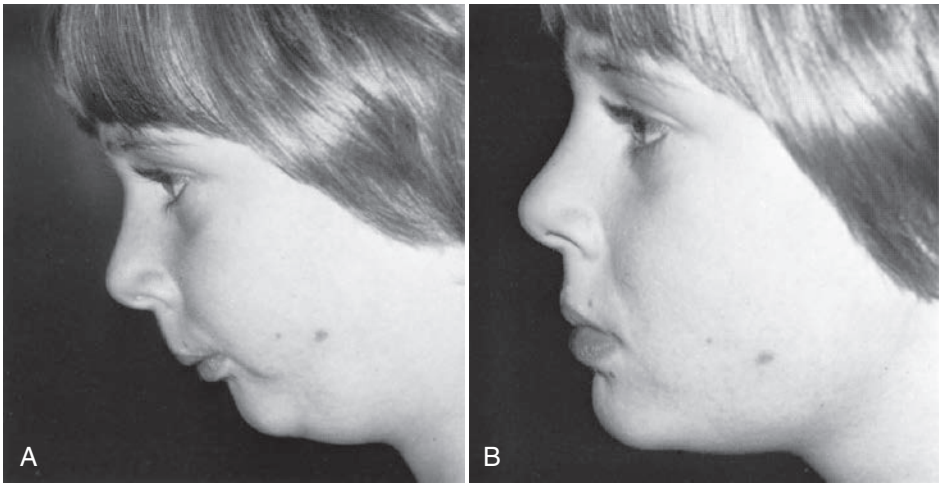


FIGURE 35-4 Profile with jaws in habitual Class II occlusion. **A,** Note the improvement when the mandible is protracted into a Class I relationship. **B,** The patient has a tendency to elevate the head when protruding the mandible, a maneuver that frees the airway.

effect. The growth increments were hypothesized to be greater at night, a belief that later was validated by Hotz,[83] Igarishi and colleagues,[84] Korkhaus,[85] and Petrovic and associates.[86]

The original name Andresen used for this type of treatment was *biomechanical orthodontics*. Only later, after teaming up with Karl Häupl and doing further work on the concepts and technique refinements, was the name changed to *functional jaw orthopedics*, which was more descriptive.[10,15] The concepts were broadened to include the potential for altering skeletal relationships, depending on the amount and

direction of jaw growth. The forward posturing of the mandible, as determined by the carefully manipulated construction bite, was reasoned to produce beneficial sagittal skeletal change by harnessing the growth potential. This was a contradiction of early bite-jumping procedures, which were deemed strictly positional changes of the mandibular condyle in the fossa; hence, the functional orthopedic connotation. The treatment became known as the Norwegian system to much of the orthodontic world, although Andresen was a Dane and Häupl was a German (both taught at the dental school in Oslo, Norway).[87]

BITE-OPENING CONTROVERSY

Although the concept of sagittal construction bite advancement was generally accepted by clinicians in Europe (it varied from 3 to 6 mm, depending on the severity of the anteroposterior dysplasia and the resultant abnormal buccal segment interdigitation), the theory pertaining to the amount of vertical opening and its effect on the muscles produced considerable controversy. Selmer-Olsen,[8] who became professor of orthodontics at the University of Oslo after World War II, believed that the muscles could not actually be stimulated during sleep; nature had designed them to rest at night, and swallowing occurred only four to eight times an hour. He interpreted the activator action as stretching of the muscles, fascial sheets, and ligaments when the mandible was opened beyond the postural resting position (i.e., viscoelastic response). In truth, the activator was a foreign body, and the tooth-moving force produced did not result from the kinetic energy of muscle function but from the potential energy of stretched tissues. Woodside and colleagues[81,88] called this the viscoelastic properties of the tissues. Current debate on the precise nature of the activator effect still reflects the divergence of opinion.[89] According to Lysle Johnston,[90] condylar unloading is a factor. Research by Graber[4,64] indicates that enhanced metabolic activity, accomplished through the stretched and enlarged TMJ retrodiskal pad during protraction, is a too-long-overlooked consideration.

Andresen and Häupl's interpretation presupposed freedom for the mandible to assume the physiologic rest position. Slagsvold,[91] later professor of orthodontics at Oslo, reported that his own observations did not completely substantiate this premise. Nevertheless, he concurred that forward posturing should not exceed the rest position vertical opening of 2 to 4 mm. Too wide an opening made compliance more difficult and could produce a depressing force on the posterior teeth—hardly desirable in deep-bite, Class II malocclusions. Grude[92] and Fränkel[93] strongly support this construction bite limit. Fränkel recommended incremental small advancements of 2 to 3 mm for his appliances rather than "the great leap forward" of 5 to 7 mm. Reactivation of optimal tissue response, as well as enhanced patient compliance, are factors. Petrovic and colleagues,[86] Komposch and Hockenjos,[35] and McNamara and associates[42] provide laboratory verification of this fact.

The philosophy of Harvold[94] and Woodside[88] has been to exceed the freeway space limits, if for no other reason than to keep the appliance in place at night during sleep so as to maintain a corrective stimulus. As for the cognitive interpretations of Selmer-Olsen, subsequent research by Ahlgren,[17] Herren,[95] Komposch and Hockenjos,[35] Sander,[54] Schmuth,[96] and Watted and colleagues[61] corroborated the finding that the activator does not *activate* muscles during sleep (Fig. 35-5).

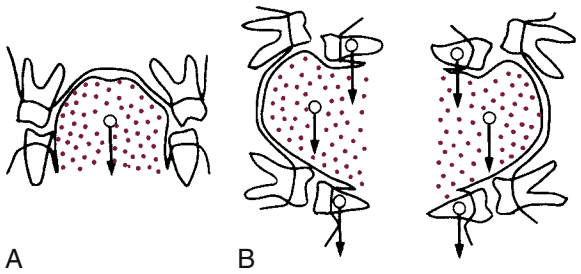


FIGURE 35-5 Various forces act on the upper and lower arches. In this case, the influence of gravity is different when the head is erect (A) than during sleep when the head is turned to one side or another (B). In the latter case, the activator tends to prevent the normal change or adaptation to varying postural and gravitational forces that elicits a positive reaction from the orofacial muscles, particularly the protracting and retracting muscles. Because the patient swallows only four to eight times an hour during sleep, this positive reaction is more likely responsible for any changes effected by the appliance. (From Herren P. The activator's mode of action. *Am J Orthod* 1959;45:512–527. With permission from the American Association of Orthodontists.)

Head Posture during Sleep

Another variable is the many changes of head posture that occur during sleep, altering the magnitude and direction of force.[91] At any particular moment, the mandibular rest position depends on the head and body posture; therefore the restriction of muscle movement required to create the desired mandibular position change without the activator in place constantly varies, involving different muscle groups and creating different force vectors on the activator (see Fig. 35-5). The plane of sleep (light or deep), the intraoral air pressure, the dream cycle, and the state of mind are additional conditioning factors, all uncontrolled by the clinician. Only the mandibular position (and the potential effect on the glenoid fossa), as held by the appliance, is controllable. However, the splinting or guiding effect on the mandible is such that it can help influence the net effect of the variable forces. If the activator is properly designed and worn as prescribed, then the result of all the controlled and uncontrolled forces is usually designed to enhance adaptation to the position created by the appliance (Figs. 35-6 and 35-7).[6,56]

Herren[95] has tried to assess the magnitude of the resultant forces acting on the mandible and the interaction between them. The weight of the mandible, tongue, and appliance is constant, averaging approximately 250 g. Muscle tonus and tensions vary, depending on factors such as the degree of stretching, the control from the central nervous system, the relation with the bed or pillow, and whether the head is prone or supine. If the patient is dorsally recumbent while sleeping and the head is upright, then the muscle forces must balance the weight of the lower jaw, associated soft tissue structures, and appliance. The multiple forces are different in prone, supine, and rotated combinations. The forces created are basically intermittent, however, even though some of the teeth or groups of teeth may be subject to pressure for a protracted period. The loose appliance is responsible for some of the intermittent action. Removal of the appliance during the day also produces a net intermittent action, calling up a new muscle engram to establish the neuromuscular engram consistent with diurnal postural changes and activities.*

* References 17, 19, 40, 45, 81, 92, 97–99.

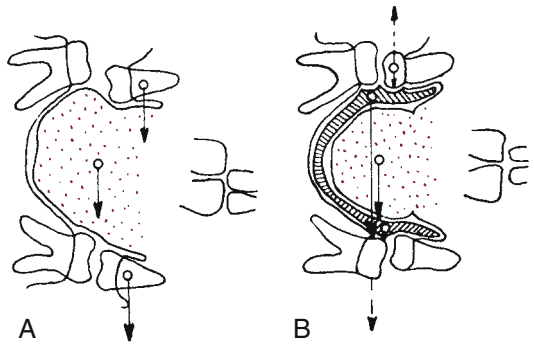


FIGURE 35-6 Instead of evoking postural change in body position during sleep (A), as in Figure 35-5, the activator splints the jaws in a more normal transverse relationship while exerting pressure on specific teeth in the buccal segments (B). (Redrawn from Herren P. The functional mechanism of the activator. *SSO Schweiz Monatsschr Zahnheilkd* 1953;63:829–879.)

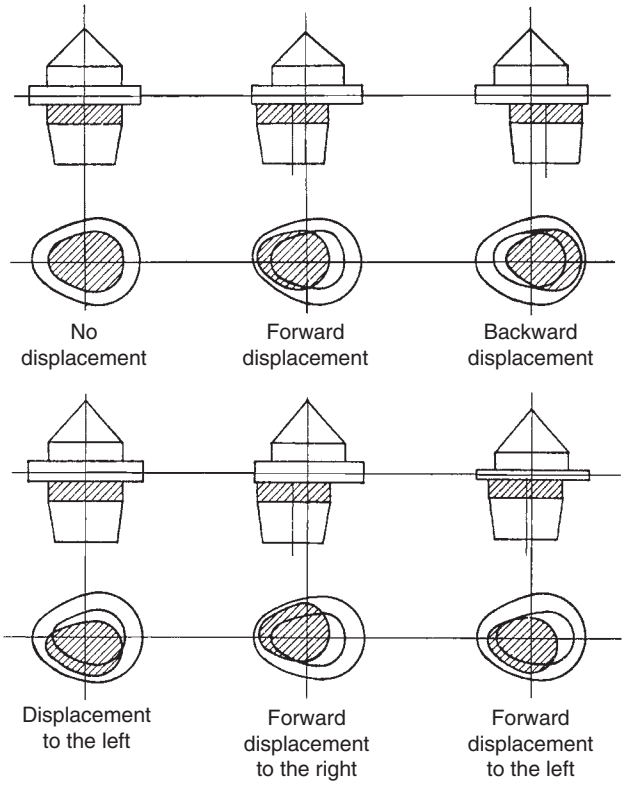


FIGURE 35-7 The direction of forces acting on the activator at any particular time depends on the spatial relationship induced by postural changes. The view is from the top of the head, with the face to the left of the ovoid outline. (Redrawn from Herren P. The functional mechanism of the activator. *SSO Schweiz Monatsschr Zahnheilkd* 1953;63:829–879.)

Working Hypotheses

For conventional nighttime wear of the classic activator, two working hypotheses have been proposed for the effect on the teeth themselves:

1. The original Andresen-Häupl rationale attributed tooth movement to a loose-fitting, functionally generated mobility that provided intermittent forces created by the elevator and protractor muscles and by the elements of the buccinator mechanism. These forces jolted or shook the teeth, pushing the appliance back into position on the maxillary teeth when it dropped away. This hypothesis was supported by the Roux concepts. A prerequisite was that the bite opening should not exceed the normal interocclusal clearance or freeway space. This approach also enhanced the metabolic pumplike action of the TMJ retrodiskal pad, stimulating anabolic and catabolic exchange.[4,64,72]
2. The second hypothesis denies that enhanced functional activity is induced at night by the bite-opening activator and implies that the activator at rest moves the teeth and creates the desired changes. Current long-range case analysis of treated individuals shows some support for this thesis.[20,21,100] The interocclusal mass of acrylic exceeds the physiologic resting position. The appliance thus is squeezed between the jaws most of the time, splinting them in the desired sagittal relationship and preventing postural changes that would normally occur without the activator in place. The resulting intermittent forces vary from time to time in frequency and magnitude, but the net result is the distribution of functional forces to the activator, which transmits them to the teeth and alveolar bone, enhancing the adaptive process. The forces produce a strain in the tissues (i.e., viscoelastic stretch) and essentially act as mechanical phenomena[20,22,81,88,100] (Fig. 35-8).

Normal respiration is optimized with forward mandibular posturing, particularly in young patients with excessive epipharyngeal tissue. This appliance thus prevents the development of open bite problems, overeruption of posterior teeth, steeper mandibular plane inclination, and compensatory tongue posture changes that might be deleterious. This hypothesis has been validated by subsequent studies by Woodside and colleagues.[†]

Sagittal Change

The forward posturing of the mandible, produced by the construction bite, creates the desired sagittal change (Fig. 35-9). This aspect depends on the desired growth guidance and adaptive processes for the skeletal and dental modifications. Occasionally, a functional retrusion is eliminated, particularly in deep bite malocclusions, which has long gone unrecognized because only the apparent condylar position in the glenoid fossa was used as a criterion and the more retruded fossa in Class II problems was not.[66,70] Controversy still surrounds the interpretations, despite extensive research by Cevidanes and colleagues,[106] Graber and associates,[10] McDougall and McNamara,[39] McNamara and colleagues,[40,42] Pancherz,[46–48,107,108] Petrovic and associates,[86] Ruf and Pancherz,[54] Voudouris and colleagues,[78,79] and others.[*] These studies clearly indicate an enhancement of growth while the functional appliance is worn—that is, more condylar growth increments and a change in the direction of growth to a more upward and backward vector. Stutzmann's research demonstrates an actual change in the internal condylar structure, with a more posterior directional orientation of the trabeculae.[86] Some evidence also exists of minor retardation of maxillary sagittal growth.[10] Vertical growth, of course, can be controlled by the interocclusal acrylic mass, preventing or allowing eruption of posterior teeth. As previously mentioned (and a factor that has been too long neglected), the change in growth direction in the fossa, as well as modification of the fossa, have both been demonstrated by Decrue and Wieslander,[69] Droel and Isaacson,[70] Ikai and colleagues,[32] McNamara and associates,[43]

[†] References 19, 81, 88, 97–99 ,101–105.
[*] References 18, 20, 22, 25, 31, 36, 38, 45, 49 ,50, 58, 72, 83, 109–117.

FIGURE 35-8 A, When the patient is upright, muscle tension, muscle tonus, and atmospheric pressure balance the weight of the mandible, the associated tissues, and the activator. B, If the patient reclines during sleep while wearing the activator, then gravity, muscle tension, and tonus act in the same direction. However, during sleep, the lips drop open, mouth breathing ensues, and function is minimal.

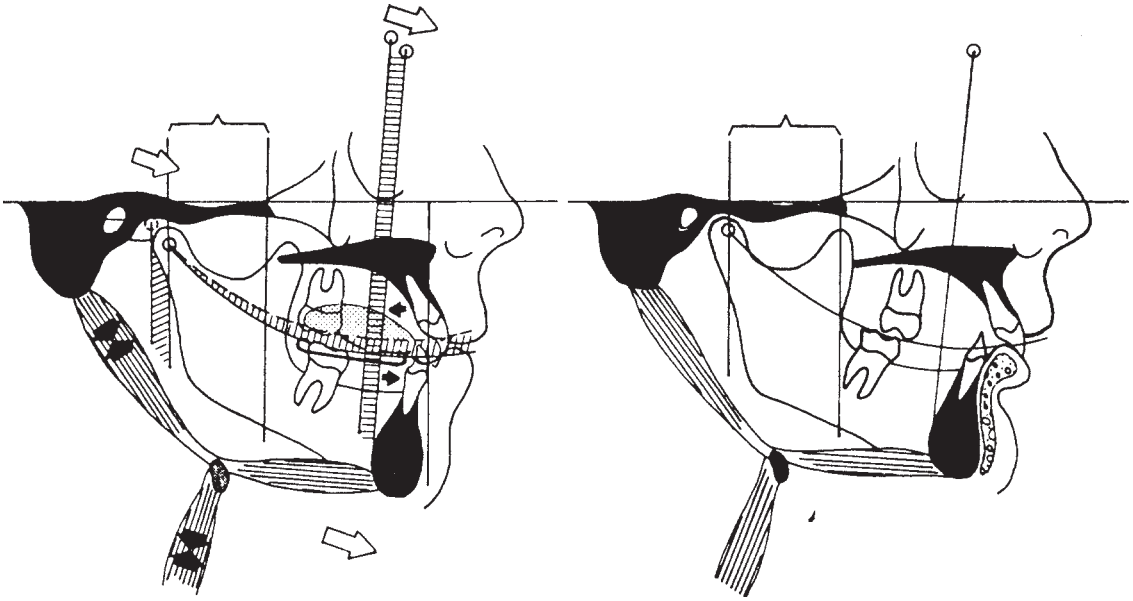


FIGURE 35-9 Modified cephalometric tracings illustrate the immediate effect of placing a functional appliance that postures the mandible forward (*arrows*). The condyle is brought forward and downward on the eminence, which removes the usual functional stresses. The retractor muscles are activated, exerting some force against the maxillary arch. Reduced masseter activity is evident. (Redrawn from Fanghänel J, et al. Changing muscle activity with functional appliances. *Eur J Orthod* 1998;20:468.)

Pancherz,[47] Paulsen,[49] Ruf and Pancherz,[53] Woodside and colleagues,[81] and others.[†]

Unfortunately, definitive incremental changes are hard to measure because of the limitations of two-dimensional radiographic superimpositions. However, Buschang and Santos-Pinto[24] have shown significant change in the position of the temporomandibular fossa with respect to the anterior cranial base. This component of sagittal change might be the most significant. Such change must occur during the growth period. Therefore sagittal change is a combination of incremental increases in condylar growth, a more favorable direction of condylar growth, repositioning and adaptation of the fossa and articular eminence, and some degree of withholding of the maxillary dentoalveolar area by the appliance, depending on factors such as the structural design, duration of wear, and possible use of an extraoral appliance.[119]

[†] References 9, 27, 34, 61, 64, 66, 72, 80, 100, 118.

Melanson and Van Dyken,[117] as well as Johnston,[90] believe that mere unloading of the condyle by forward posturing can be a significant factor. This belief is based on the seminal study conducted some years ago by Melanson and Van Dyken at the University of Michigan, in which unloading the condyles in primates actually stimulated condylar growth. The results can vary, as shown by Graber[120] in an analysis of 58 patients treated with the Fränkel functional regulator. When the enhanced metabolic action is coupled with neuromuscular and appliance stimulation, significant improvement can be attained during a period of active dentofacial growth that is not possible with conventional fixed mechanotherapy alone.[65]

The same analysis can be applied to the Herbst fixed functional appliance, as reintroduced and modified by Pancherz* (see also Graber[64,121]). (For an excellent discussion of this appliance and its modified versions, see Chapter 16 of *Dentofacial Orthopedics with Functional Appliances*, 2nd ed., by Graber and associates,[122] as well as a discussion later in this chapter.) Conversely, in early clinical reports based on minimal material and experience in the appliances, Creekmore and Elkund,[123] Gianelly and colleagues,[112] and Ricketts[124] noted little enhancement of mandibular length. However, long-term studies by Ruf and Pancherz[53,54] showed an additional 1 to 2 mm of growth over the normal mandibular growth pattern for Herbst cases. If left alone, the mandibular length for most Class II, Division 1 malocclusions is deficient. Furthermore, in addition to some enhanced condylar proliferation, the direction of growth changes to a more upward and backward vector for the condyle. Finally, significant glenoid fossa adaptation and positional change occurs, as previously noted.† Elimination of neuromuscular perversions that accentuate the overjet could be a significant factor in the ultimate status. This is too often ignored by "one phase, get in–get out" philosophies. The net effect is that these patients attain the achievable optimum of pattern, assuming that treatment is timed to coincide with the period that has the greatest growth and developmental potential for sagittal correction. Although such appliances can be used in a patient who is past the maximal growth period, more dentoalveolar response and more likelihood of proclination exist in the lower incisors.[22,57,95,126–129] The correct comparisons of treated functional cases are made not with a normal sample but with comparable untreated patients of the same age, pattern, gender, category of malocclusion, and degree of mandibular underdevelopment at the time of initiation of treatment.

Vertical Opening Variations

A number of modifications of the original Andresen appliance has been introduced. Many of the modifications are based on constructions of different vertical opening requirements and different amounts of forward posturing. The question of whether to posture the mandible in "one great leap forward" or in sequential steps has also given rise to different designs. Petrovic and colleagues[86] show clear evidence of greater response with periodic, sequential advancement procedures. Whether the appliance is to be worn only at night or full time is another factor in the design, as is the decision regarding whether the appliances are to be anchored to the maxilla (as with the Fränkel functional regulator and the Bionator) or kept loose with minimal wire appurtenances.[93]

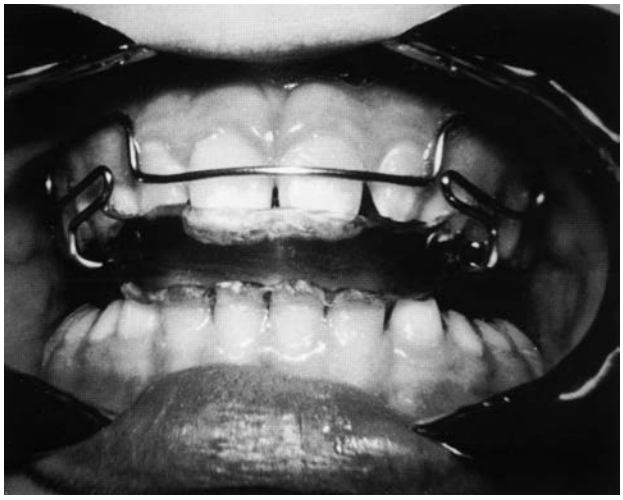


FIGURE 35-10 A number of activator models (e.g., Woodside, Herren, Harvold) require a larger vertical opening, which exceeds the freeway space at postural rest position and elicits the viscoelastic or stretch properties of the associated tissues. This illustration shows an 8-mm opening between the first molars. The lower incisors are capped to provide more stability and to offset the tendency of these teeth to procline.

The current use of skeletal design functional appliances mostly follows the original small opening, incremental sagittal advancement concept, and encourages daytime wear. The frequency of deglutition is increased, and phasic muscle activity is enhanced. Thilander and Filipsson[59] conducted electromyographic studies on daytime wear and found an insignificant increase in muscle forces when no active function occurred. During mandibular activity, however, increased force was exerted, especially by the retracting muscles. Increased deglutitional activity also was associated with the enhanced salivary flow. Sander[55] and Schmuth[96] have also studied the role of functional appliances. Their research indicates that patients with large protrusive construction bites tend to disclude the appliances during the day and at night, reducing the desired effect and jiggling selective teeth.[55] As with normal subjects, however, a broad range of individual activity exists from patient to patient.

Although openings as large as 12 to 14 mm have been successfully treated by clinicians such as Eschler,[130] Schwarz,[131] Woodside and colleagues,[81] and Herren[95] (Fig. 35-10), two factors favor the reduced opening and incremental sagittal correction.

Patient acceptance and compliance are significant factors for all removable activators. Daytime use mitigates against bulky functional appliances that interfere with speech, swallowing, eating, and other activities. The wider opening appliances have a smaller sagittal correction. They have the advantage of depressing the posterior segments if an occlusal cover is used, which allows the mandible to move upward and forward if the patient has an open bite tendency and reduced interocclusal clearance. This result is desirable in open bite problems but not in deep bite malocclusions. Histologic evidence also supports periodic incremental advancement because of the periodically enhanced condylar and fossa response with each adjustment.*

* References 46–48, 53, 54, 107, 108.

† References 24, 32, 34, 43, 47, 49, 51, 54, 61, 64, 66, 70, 72, 80, 81, 120, 125.

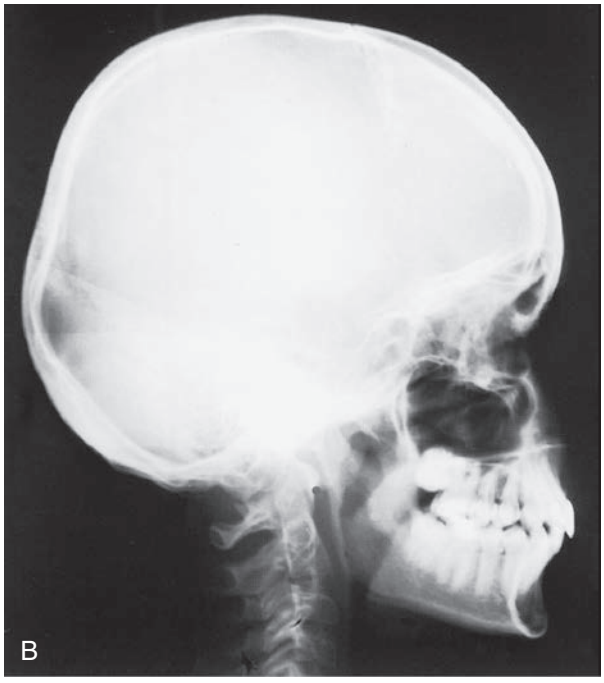
* References 32, 35, 42, 43, 84, 118, 132–134.

FIGURE 35-11 **A, B,** Class II, Division 1 malocclusion in an 11-year-old boy with a retained infantile swallowing habit. Initial success with the modified Bionator appliance was limited because of the lack of significant growth and patient cooperation. **C, D,** The patient became motivated after 2 years of intermittent wear, and the results seen were achieved. Pubertal growth acceleration obviously helped. (**A** and **B,** Courtesy Erich Fleischer.)

With a single 6- to 7-mm advancement, the condylar and fossa growth stimulus is shorter in duration, daytime wear becomes more difficult, and adverse labial proclination of mandibular incisors may be greater.[10,29,93] Again, for a fuller discussion of these differences and for design prescriptions for specific malocclusion characteristics, see *Dentofacial Orthopedics with Functional Appliances*, 2nd ed., by Graber and associates[10] or *Removable Orthodontic Appliances* by Graber and Neumann.[135]

MECHANISMS OF CLASS II CORRECTION WITH FUNCTIONAL ORTHOPEDICS

The efficacy of functional appliances no longer needs to be proved, unless one is ignoring many thousands of case reports. Dramatic facial and skeletal changes seldom seen with conventional fixed appliances are not only possible but fairly routine (Figs. 35-11 to 35-14). However, the



	10 yr 3 mo	12 yr 9 mo
S-N-A	84°	83°
S-N-B	76°	78°
A-N-B	8°	5°
A to N (perpendicular)	1 mm	–1 mm
$\underline{1}$ to A	+5 mm	+2 mm
$\overline{1}$ to pogonion	+5 mm	+6 mm
Pogonion to N (perp)	–10 mm	–8 mm
Maxillary length	90 mm	91 mm
Mandibular length	107 mm	114 mm
Skeletal differential	17 mm	23 mm
Saddle angle	125°	127°
Ar to Ptm	31 mm	31 mm
Mandibular plane angle	19°	20°
Anterior face height	68 mm	69 mm
Posterior face height	51 mm	56 mm
Anterior cranial base length	70 mm	71 mm
$\underline{1}$ to S-N	110°	102°
$\overline{1}$ to Mandibular plane	104°	108°

C

FIGURE 35-12 Lateral cephalograms of the patient in Figure 35-11 before treatment **(A)** and 2½ years later **(B)**. **C,** Cephalometric measurements.

precise reasons that these changes occur are more obscure and vary from patient to patient, as orthopedic surgeons have observed with their growth guidance procedures in long bones. Unfortunately, the overly simplistic attribution of the change to condylar growth has clouded the picture and is similar to looking at the road as a horse with blinders does. Orthodontic practitioners are dealing with a cybernetic process.[10] The morphogenetic pattern, growth timing and direction of component parts, neuromuscular patterns, functional displacement, dentoalveolar compensation, fossa displacement and remodeling, type of appliance, and patient compliance are the most important variables but not the only ones.[136] Factors such as tissue response and rate of fibroblast turnover are discussed in the superb chapters by Thilander, Hatch, and Sun (see Chapter 3) and Roberts and Huja (see Chapter 4), all recognized as outstanding authorities. No longer can orthodontists simply estimate the amount of condylar change needed to provide the correction. The change is a multifactorial reaction that is too diverse to determine by measuring only one component.

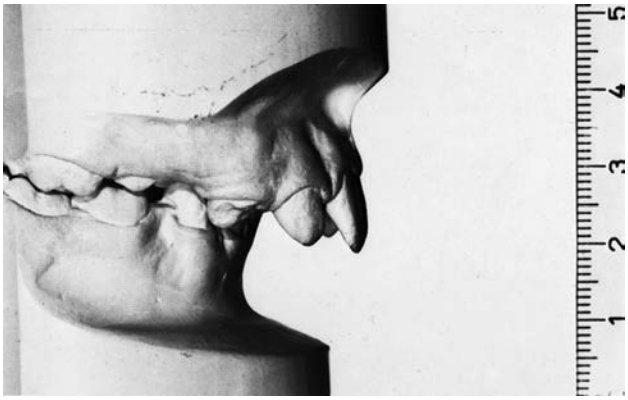


FIGURE 35-13 Initial study model depicts a boy, 9 years and 7 months old, who has a severe Class II, Division 1 malocclusion. (Courtesy Erich Fleischer.)

As research continues, the fact that nighttime wearing of appliances largely serves as a transient posturing splint is becoming increasingly clear. Telemetric studies, notably those of Sander,[55] show that function is not enhanced by wearing appliances only during sleep. Nevertheless, the forward posturing of the condyle does enhance metabolic action in the TMJ, as shown by Ward and colleagues.[80] Increased anabolic and catabolic exchange, not merely forward positioning, may account for enhanced growth of the condyle and posterior fossa wall proliferation.[4,32,65]

Class II Correction: A Likely Scenario

The amount of sagittal correction is 6 to 7 mm. The following anticipated growth and adaptational increments (assuming a favorable direction) are typical:

1. Condylar growth amount during treatment: 1 to 3 mm—The mandible outgrows the maxilla by 1 to 5 mm, with higher increments predominantly occurring in males.
2. Fossa displacement, growth, and adaptation—Recent research shows this growth to be 3 to 5 mm, with a dominant vertical vector[24] and a positive correlation with the pubertal growth spurt.*
3. Functional retrusion (see Fig. 35-52): This retrusion does not always occur in Class II malocclusions and can vary from 0.5 to 1.5 mm. Isberg and Isaacson,[139] as well as Kantomaa and Pirttiniemi,[34] show strikingly reduced TMJ metabolism in primates with functionally retruded condyles. Research conducted by Graber,[65] as well as that of McNamara[41] and Boman and Blume (see Graber[118]), confirms these observations. The controversy results from not knowing the technique for a correct determination (see Fig. 35-52).[118]
4. More favorable growth direction shown by trabecular orientation: Trabecular orientation changes and can account for between 0.5 and 1.5 mm.
5. Withholding of downward and forward maxillary growth[117]: Maxillary growth accounts for between 1 and 1.5 mm of the change.
6. Differential upward and forward eruption of lower buccal segments: Accounts for between 1.5 and 2.5 mm of Class II correction, depending on the depth of overbite.
7. Headgear effect[61,137]: Is between 0.0 and 0.5 mm

* References 24, 53, 54, 81, 88, 137, 138.

Obviously, no reliable method exists for predicting the contribution of each component for the individual patient. Not all functional assessments and physiologic phenomena are reducible to two-dimensional cephalometric tracings with angles and figures or to articulators. Orthodontic practitioners are as precise in their efforts as the best orthopedic surgeons, who would not think of using a cephalometric analysis and an articulator for any joint they treat. The seven components previously listed provide a potential for the needed sagittal change. However, skeletal malocclusions are three dimensional. Weinstein once said, "We are a generation of profiles!" This statement was based on the orthodontic preoccupation with only the lateral cephalogram and an unwillingness to use anteroposterior views to determine equally important transverse characteristics.[26,140] Today's three-dimensional diagnostic procedures provide more accurate information. Early functional appliances incorporated expansion screws or jackscrews to cope with the narrow maxillary arch of most Class II, Division 1 malocclusions (the third dimension), particularly when the narrowed maxillary arch is caused by abnormal perioral muscle function. Whether by slow or rapid palatal expansion or by fixed or removable appliances, modern orthodontics recognizes the need to produce a three-dimensional correction. In no way is this an excuse to expand teeth off basal bone, however. Axel Lundström's epic paper[116] on the apical base in 1923 is still valid. Some recognition of the need for a three-dimensional therapeutic attack on the problem must be incorporated in the functional appliance, or transverse correction can be done before the placement of functional appliances. Nature provides the answer to questions about the amount of expansion that can be achieved when perverted perioral muscle activity is eliminated. In many cases, wearing a functional appliance is likely to eliminate abnormal and deforming neuromuscular activity without active expansion adjustments. Too much rapid palatal expansion is being done without checking the inclination of the buccal segment teeth in their relation to basal structures before and after treatment. Herein lies a major precipitator of posttreatment relapse.

Bionator

The bulkiness of the activator and its limitation to nighttime wear have deterred clinicians interested in achieving a mixed dentition correction of sagittal discrepancies and eliminating their attendant neuromuscular perversions. The Bionator, as developed by Balters,[141] is the prototype of a less bulky appliance (Fig. 35-15). Its lower portion is narrow, and its upper portion has only the labial wire and the buccal screening wire extension, plus a stabilizing cross-palatal bar that actually can be adjusted for bilateral expansion, if needed. The palate is free for proprioceptive contact by the tongue. The appliance may be worn all the time except for meals, which is critical for maximum response.

According to Balters,[141] the equilibrium between the tongue and the circumoral buccinator mechanism is responsible for the shape of the arches and intercuspation. This hypothesis supports the early form and function concepts of van der Klaauw as cited by Graber[118] and the later functional matrix hypothesis of Moss.[142] The purpose of the Bionator is to establish good muscle coordination and to eliminate potentially deforming growth restrictions, while unloading the condyle through a protrusive mandibular position. The upper and lower incisors are usually in contact during wear. Balters attributed a major role to normal or abnormal tongue function and posture in deglutitional

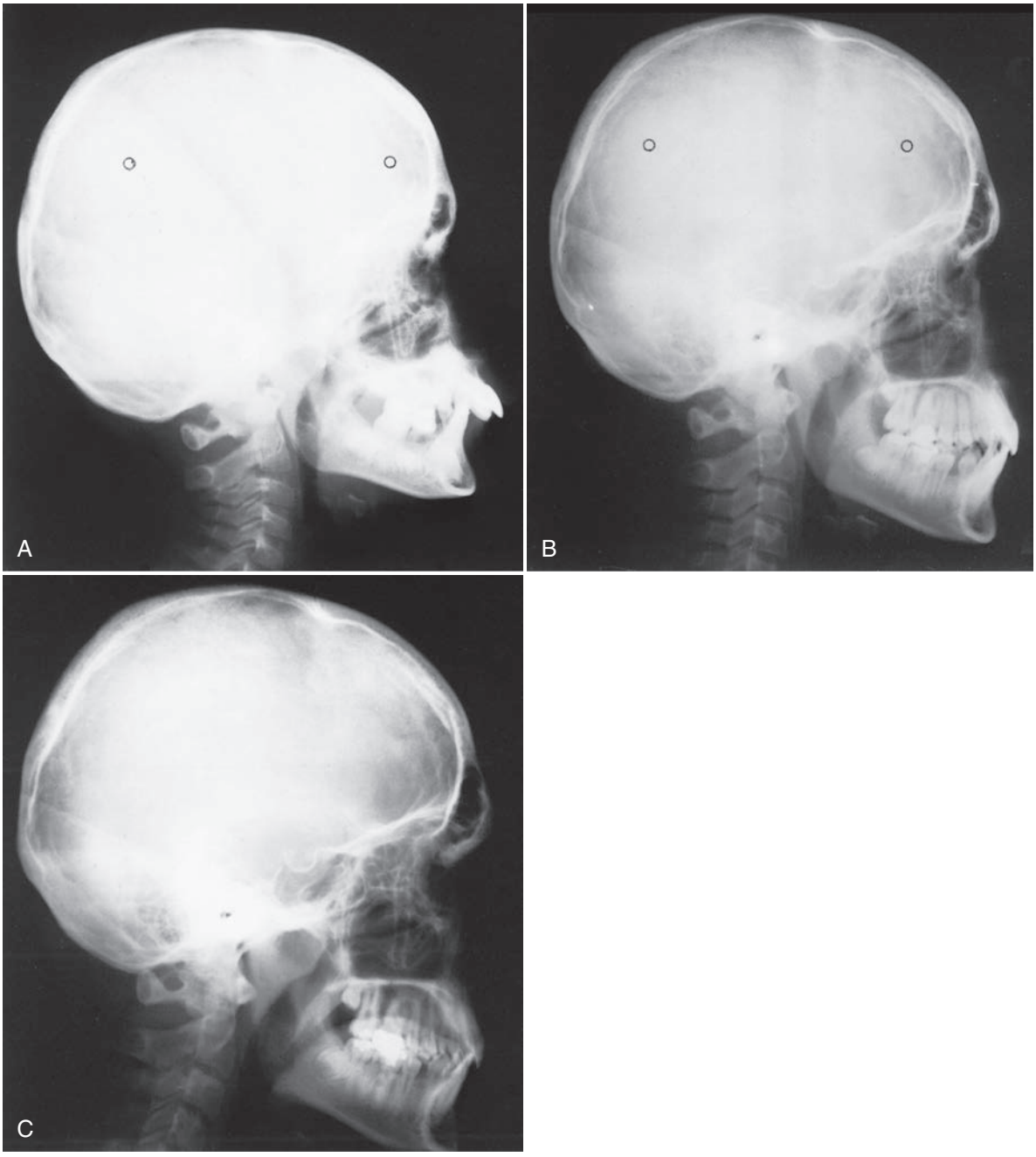


FIGURE 35-14 Lateral cephalograms and measurements of patient in Figure 35-13. **A,** Severe overjet is exacerbated by a lower lip trap habit. **B,** At the end of treatment (patient's age 13 years and 7 months). **C,** Patient at age 16 years and 2 months. **D,** Cephalometric analysis summary. (**A–C,** Courtesy Erich Fleischer.)

D

	9 yr 7 mo	13 yr 7 mo	16 yr 2 mo
S-N-A	82°	83°	80°
S-N-B	75°	78°	79°
A-N-B	7°	5°	1°
A to N (perpendicular)	+3 mm	+4 mm	+1 mm
$\underline{1}$ to A	+7 mm	+2 mm	+6 mm
$\overline{1}$ to pogonion	0 mm	+5 mm	+1 mm
Pogonion to N (perp)	–3 mm	–1 mm	+4 mm
Maxillary length	89 mm	95 mm	92 mm
Mandibular length	107 mm	102 mm	126 mm
Skeletal differential	18 mm	25 mm	34 mm
Saddle angle	130°		128°
Ar to Ptm	34 mm	35 mm	35 mm
Mandibular plane angle	17°	20°	15°
$\underline{1}$ to S-N	115°	95°	98°
$\overline{1}$ to mandibular plane	99°	103°	98°
Anterior facial height	55 mm	67 mm	70 mm
Posterior facial height	43 mm	49 mm	55 mm
Anterior cranial base length	66 mm	69 mm	72 mm

FIGURE 35-14, cont'd

maturation; Ballard[19] later elaborated on this. Research has shown that the tongue exerts three to four times as much force on the dentition as the circumoral muscles. If the morphogenetic pattern, resting force and viscoelastic properties, tissue rigidity, atmospheric pressure, and intercuspation are included, then obviously the dentition and its surrounding structures are in a multisystem balance.[64,88]

The standard Bionator (Fig. 35-16) consists of a lower horseshoe-shaped acrylic lingual flange that extends from the distal of the last erupted molar around to the corresponding point on the other side. For the upper arch, the appliance has only posterior acrylic lingual extensions that cover the molar and premolar regions. The upper and lower parts are interocclusally joined in the correct construction bite (protrusive) relationship. The anterior portion is kept free so as not to interfere with tongue posture and function. No acrylic capping of the lower incisors is done, although some clinicians choose this variance to attempt to control proclination of the lower incisors.

The function and posture of the cheeks and lips are guided by two wire configurations: the palatal bar and the labial bow with buccal extensions (Fig. 35-17). The palatal bar, which is made of 1.2-mm hard stainless steel, lies 1 mm away from the palatal mucosa and runs distally to a line drawn between the distal surfaces of the maxillary first molars. The palatal bar has a Coffin spring–like configuration. The bar stabilizes and also anteriorly orients the tongue to contact the palate. The labial and buccal bow is comprised of 0.9-mm stainless steel (Fig. 35-18) and does not actually contact the incisors or gingival tissue. The position of the wire produces a negative pressure, with the wire supporting lip closure. If necessary, the wire can be adjusted during the course of treatment, after the arch has been widened, to close the spaces and to retract the maxillary incisors. Selective eruption can be achieved during treatment by trimming the acrylic.

Of all the functional appliances, the Bionator and the Clark twin block (see Chapter 16) are the most popular. They are less subject to distortion than the efficient Bimler appliance,[21]

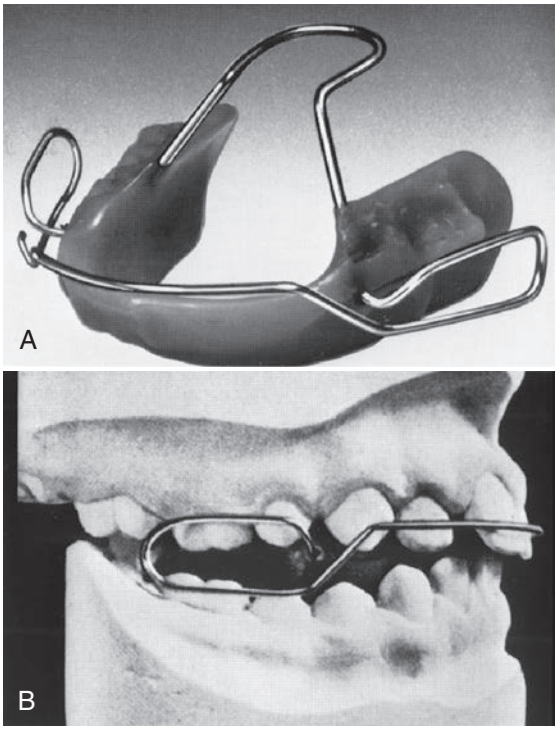


FIGURE 35-15 Bionator modified from Balters' original design. A, Note the use of a heavy transpalatal Coffin spring instead of acrylic construction. The large gauge labial and buccal wire is not a tooth-moving appurtenance but a screening wire. To enhance eruption, the acrylic can be removed in the interdental area. B, The construction bite is usually taken in an end-to-end incisal relationship, allowing enough room for passage of interocclusal wires. The appliance is an effective adjunct in many temporomandibular dysfunction problems.

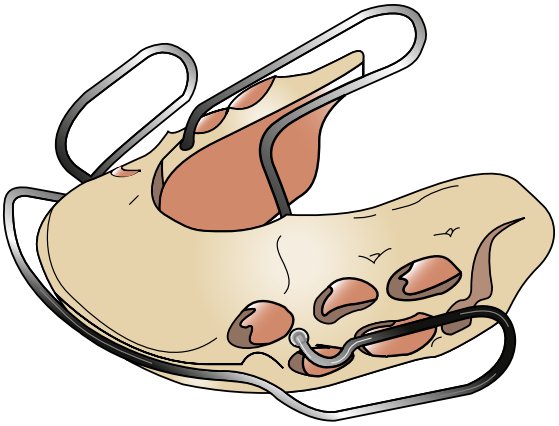


FIGURE 35-16 Basic Bionator appliance. Open palate enhances tongue proprioception and discourages abnormal growth. Heavy buccal and labial wires serve to screen off muscle forces.

which has more wire framework that can be distorted. The successful use of the Bionator in TMJ disturbances has been well documented[10] (see Figs. 35-11 to 35-14). For a fuller discussion of the Bionator philosophy and appliance variations for Class II, Class III, and open bite malocclusions, see Chapter 11 in *Dentofacial Orthopedics with Functional Appliances*, 2nd ed., by Graber and associates.[10]

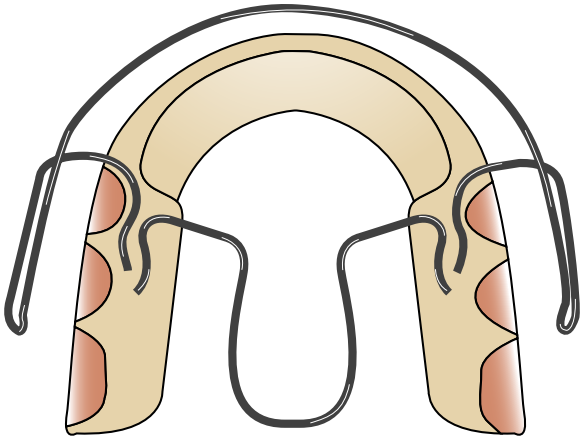


FIGURE 35-17 Palatal Coffin wire of 1.2-mm hard stainless steel is placed slightly away from the palatal tissue but did not interfere with proprioceptive tongue contact.

CONSTRUCTION BITE

Probably the most important aspect of fabrication is the assessment of optimal horizontal and vertical displacement before construction of the appliance. As previously noted, no cookbook exists for determining the relationships. Much depends on the specific appliance being used, the case selection, the dentofacial morphologic form and structure, the probable growth pattern and projected increments and direction, and the need for other therapeutic assists (e.g., fixed appliances, extraoral force, extractions).

The Bionator, originally developed by Balters,[10,141] is one of the most commonly used removable functional appliances today because of its simplicity, sturdiness, ease of construction, ability to be modified, patient acceptance, and use in temporomandibular disorders (TMDs); therefore this section is devoted to a discussion of the construction bite for that type of appliance[†] (Fig. 35-19). The technique can be adapted to other designs. A complete diagnostic discipline is essential before selecting a functional appliance for a patient.[134] As Rakosi and colleagues[52,148] emphasize, completing a three-dimensional cephalometric radiographic study and functional analysis for the mixed dentition, use of a functional appliance is no less important than it is for a full, fixed mechanotherapy problem. Facial pattern is important; growth amounts and growth direction are equally important (i.e., growth forecasting, such as developed by the late Dr. Ricketts,[124] and computerized digital projection to ensure that the patient is aware of the problem and the projected result [see Chapters 8,9 and 11]). Only if the patient satisfies the demanding functional appliance criteria and only if patient compliance is likely to be good should such an appliance be used. The operator should always keep in mind that a functional appliance is primarily an interceptive device, seldom capable of achieving full correction and complete dental detailing. The appliance is a first-phase attack on the total problem. The argument of one-phase versus two-phase treatment will not be settled here. Distinguished proponents ascribe to both approaches. However, with proper case selection, proper wear, and proper patient compliance, the author's firm conviction, based on thousands of treated cases, good and bad, is that

[†] References 26, 38, 57, 141, 143–147.

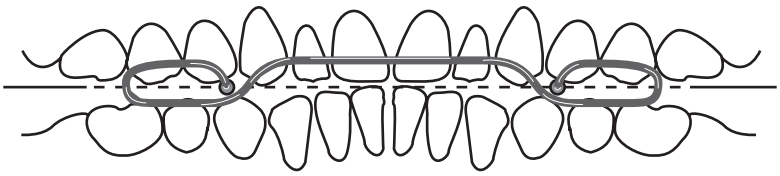


FIGURE 35-18 End-to-end construction bite and configuration of 0.9-mm stainless steel labial and buccal screening wire extends to the distal of the second deciduous molars. The wire has no contact with teeth. Fränkel added acrylic to the posterior aspect of the screening wire to make more effective buccal shields.

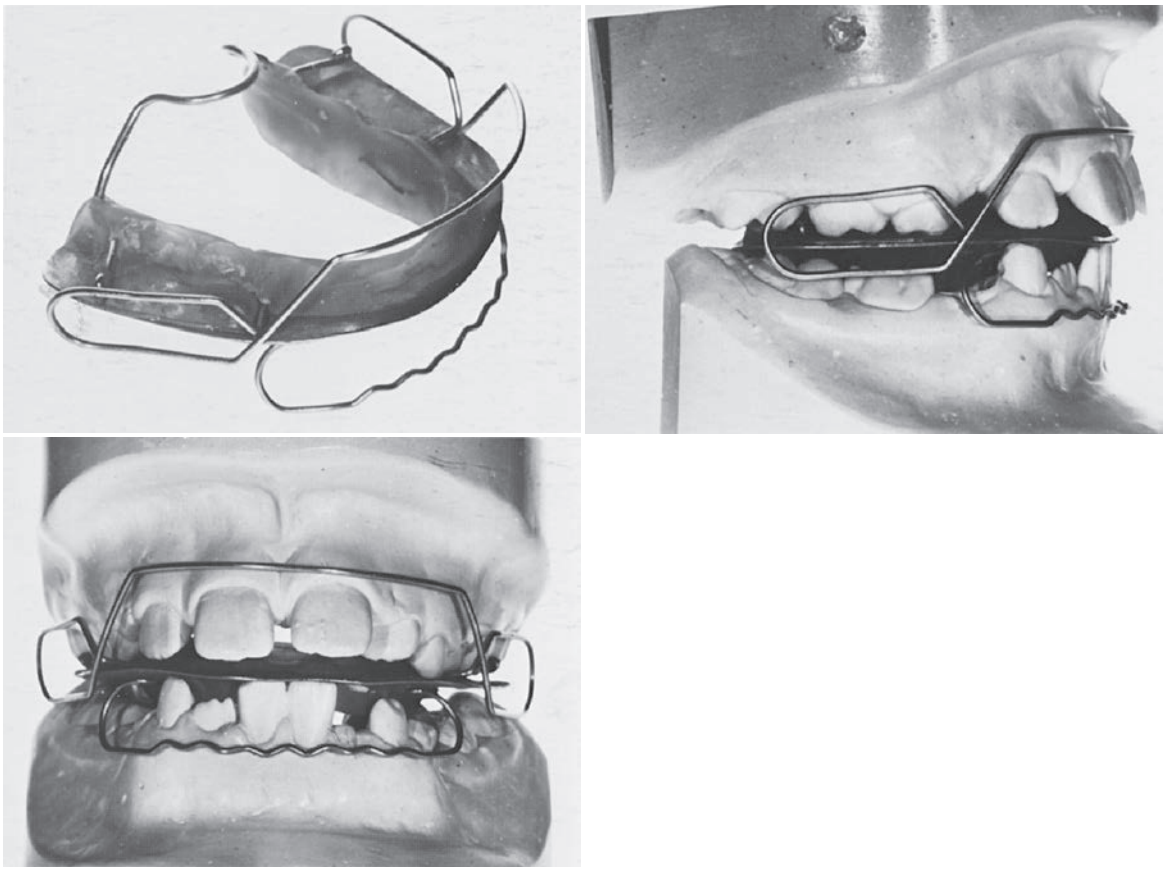


FIGURE 35-19 Fleischer Bio-Bionator is placed on working models in construction bite relationship. A lower screening wire has been added to prevent a lower lip trap and to allow the tongue to procline lingually malposed lower incisors. This arrangement is similar to the lip shields of the Fränkel function regulator.

skeletal and neuromuscular changes can be attained with functional appliances in first-phase therapy that cannot be duplicated with single-phase treatment. No patents or commercial ties exist to influence this observation.

In this short survey chapter, the concern is for Class II malocclusion, the most frequent challenge and the one with the greatest potential for therapeutic success. For a fuller discussion of techniques and appliances and for Class III and open bite problems, see Chapter 13 in *Dentofacial Orthopedics with Functional Appliances*, 2nd ed., by Graber and associates.[10]

Anterior Mandibular Posturing

In the past, if the sagittal discrepancy was not too great, the forward posturing of the mandible was such that the upper and lower incisors contacted in an end-to-end relationship (see Fig. 35-19). However, the patient's adjustment to the mandibular protrusion is easier if the advancement is made in 3-mm increments. Evidence exists that this method is more successful, despite the added work. Usually adjustment can be done with a 12- to 16-week interval in the sequential advancement. Procumbency of the lower incisors is less likely with sequential advancement, and patient compliance is better. As Petrovic and colleagues[10,86,133] have shown, tissue reaction is also more favorable, and Rabie and colleagues[75,149,150] conclusively demonstrated this on treated cases. Case selection will help determine the decision. Lingually inclined lower incisors, as with a lower lip trap problem, may respond better with a 5- to 6-mm advancement. As a general rule, the anterior advancement should be 3 mm or more from the most posterior condylar position in the fossa. Paulsen concurs.[73]

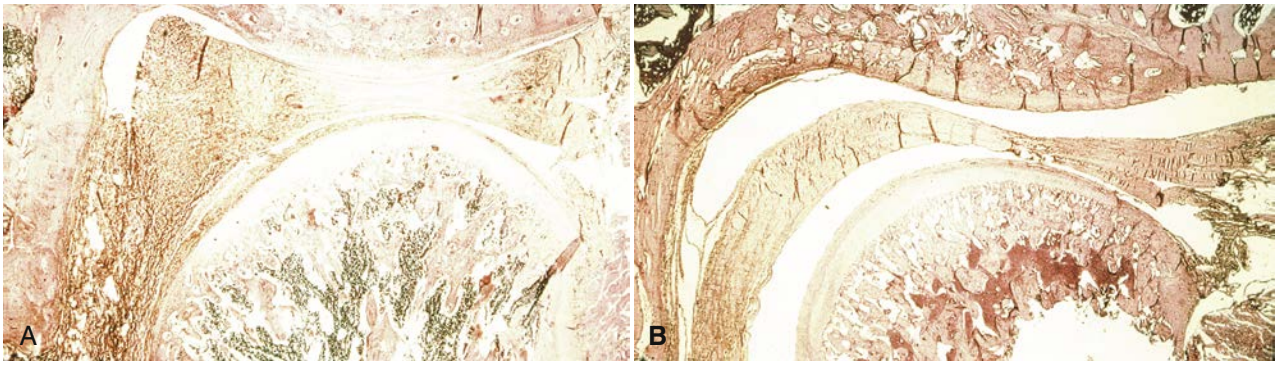


FIGURE 35-20 Cryogenic primate specimens. **A,** Control shows normal morphologic and positional relationships of the condyle, disk, eminence, and retrodiskal attachment. **B,** Significant change is visible in only 6 weeks of wear of a retrusive splint. Dramatic diminution of disk and retrodiskal tissue has occurred. Clearly, forced retrusion has the potential to damage retrodiskal tissue and reduce metabolism, that is, growth and development. (**A,** Courtesy Isberg; from Graber TM. The unique nature of the temporomandibular joint metabolism; the clinical implications. In: Rabie AM, Urist MR, eds. *Bone Formation and Repair*. Amsterdam: Elsevier; 1997:143–157.)

A good technique is to have the patient move the mandible forward as far as possible and then drop back 3 to 4 mm. If a functional shift to one side or the other occurs because of a narrow maxillary arch and convenience swing to one side, then lining up the maxillary and mandibular midlines with the construction bite can eliminate this shift. Rakosi, with his tremendous experience with functional and fixed appliances through the years, has carefully described the construction bite technique, with detailed illustrations and descriptions, to ensure the best possible results, in his chapter in *Dentofacial Orthopedics with Functional Appliances*, 2nd ed., by Graber and associates.[10]

Because the maxillary incisors are initially and frequently tipped labially and are often spaced, using an active plate for a short period or limited fixed appliances to retract and rotate maxillary incisors before placing the functional appliance may be preferable to reduce the magnitude of initial mandibular protraction and to enhance patient compliance. What should be made clear is that modern orthodontics is not an either/or therapy. Mixing fixed and functional appliances is easy and often desirable with modern bonding techniques, and functional appliances are not the "easy and cheap" alternative to classic full-mouth fixed hardware. Bonding anterior brackets and placing molar bands on the maxillary arch is a ½-hour time investment with major returns in better functional appliance retention and control of maxillary incisors, rather than resorting merely to tipping them lingually with the labial bow of the removable appliance.

Despite the controversy that still exists over the idea of the existence of posterior condylar displacement in some Class II deep bite malocclusions, seminal research by Dayton Blume and Vernon Boman demonstrates this phenomenon in selected cases. Some patients with TMJ sequelae are relieved only by forward posturing of the mandible (see Fig. 35-52, which shows research findings of Blume and Boman). This possibility should be checked with the diagnostic functional analysis. Tomographic radiographs and scintigraphy, such as that used by Paulsen,[108] Ruf and Pancherz,[53,54] and Pancherz,[47] show this positional status, taking a postural resting position and maximal occlusion lateral views. However, the astute clinician can see the same by observation and palpation. Recent research by Isberg and Isaacson,[139] Kantomaa and Pirttiniemi,[34] and Pirttiniemi and colleagues,[51] as well as the author of this text,[65] shows that retrusive forces on the condyle in the TMJ can impinge on the retrodiskal pad, reducing the metabolic exchange and the potential nutrient elements for TMJ and condylar growth (Fig. 35-20). Definite reduction in the collagen and proteoglycan content of the condyle shows the effect of reduced anabolic and greater catabolic activity.[51] Condylar advancement enhances metabolic activity.[65] As previously noted, the vertical opening is within the limits of the freeway space. Elevator and protractor muscles are activated, and a greater hyoid group compensation or retracting force occurs, which is distributed against the maxillary arch. This action only slightly inhibits maxillary downward and forward relocation, as studies by McNamara and colleagues demonstrate[39–42,134,151] (see also Chapter 16). Biting into the appliance and swallowing enhance the neuromuscular and osseous adaptation (both fossa and condyle) to the new posture.

Hamilton Expansion Activator

The Hamilton expansion activator is a variant of the traditional monobloc. David Hamilton was often called "the thinking man's orthodontist." The first generation of such appliances was removable and yet achieved stable correction. Current bonded appliances reduce the need for patient compliance and have a high level of success (Figs. 35-21 to 35-24). The simultaneous use of two palatal jackscrews permits differential expansion control, which is highly desirable. Too often, a single palatal expansion screw does not provide the varying need of expansion in anterior and posterior maxillary buccal segments. The Hamilton philosophy and appliances are described in detail in *Dentofacial Orthopedics with Functional* Appliances, 2nd ed., by Graber and associates.[10] Hamilton[113] points out, as do Graber,[122] Gianelly and associates,[112] and others, that most patients with these functional appliances need concurrent partial fixed appliances, transverse correction, or a second period of therapy to complete the required detailing. This fact is vital to orthodontists' understanding of the multiple challenges of correcting a large variety of malocclusions, depending on a multiplicity of causative factors, sexual dimorphism, and when the orthodontist first sees the patient.

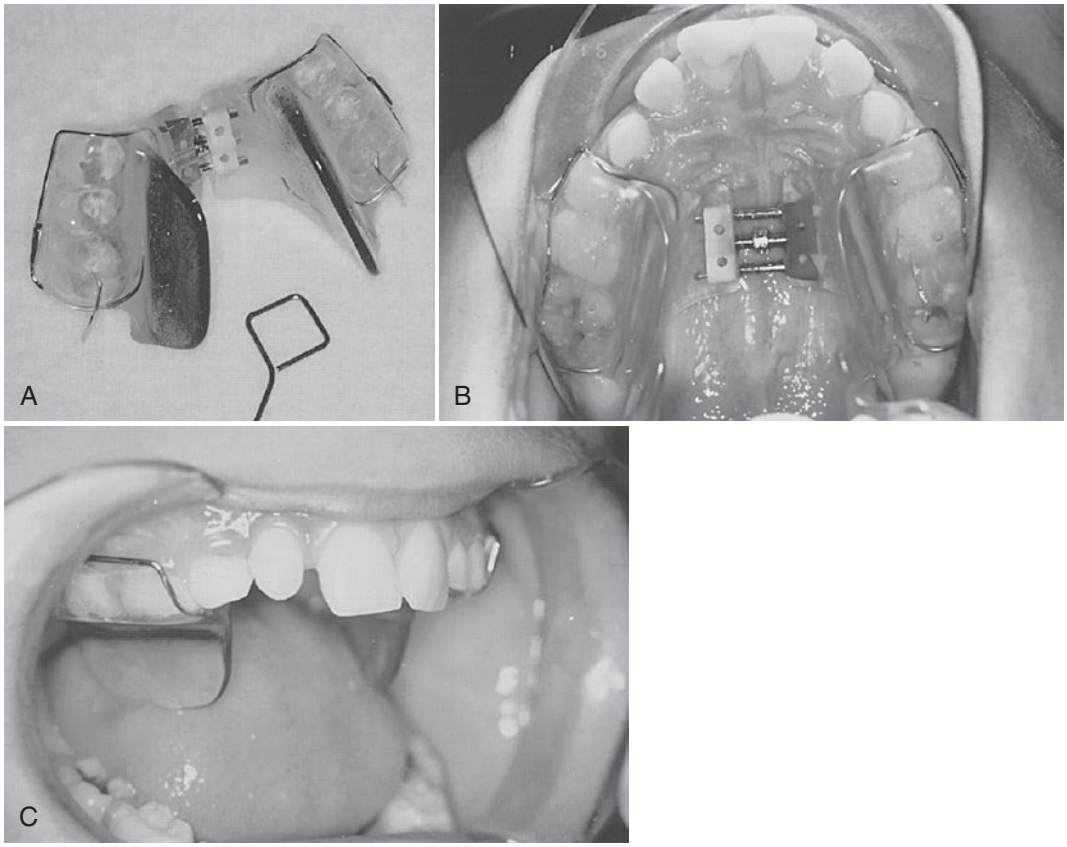


FIGURE 35-21 Early version of Hamilton bonded activator (from mandibular aspect). **A,** The palatal jackscrew is partly open. (Later versions have two palatal expansion screws for more effective localized expansion.) The dark lingual flanges extend toward the viewer. Impressions of the lower posterior teeth are embedded in the occlusal acrylic in the forward postural relationship. **B,** The Hamilton appliance is bonded to the maxillary arch; again, the lingual flanges vertically extend toward the viewer. **C,** Lateral view shows the lingual flanges of the bonded appliance. These lingual flanges guide the mandible into its correct anterior construction bite via proprioception.

FIGURE 35-22 A, B, Initial facial and profile views show a female patient with a Class II, Division 1 malocclusion with anterior open bite, no interocclusal clearance, excessive overjet, a constricted maxilla, and narrow and retruded mandibular arch. **C, D,** All of these characteristics cause compensatory and deforming perioral neuromuscular activity.

FIGURE 35-22, cont'd E–H, Posttreatment frontal and lateral facial and intraoral views show significant facial sagittal jaw improvement. **I,** A twin-screw palatal expansion appliance provides selective buccal segment expansion. This appliance was worn for 8 weeks and was followed by a Hamilton bonded activator (see Figs. 35-21 and 35-23). **J–M,** Intraoral views of the patient at age 11 years and 6 months indicate that minimum fixed appliance treatment will be required to finish tooth minor alignment and to establish a normal overbite. This is a classic example of a proper two-stage orthopedic appliance management for the twenty-first century.

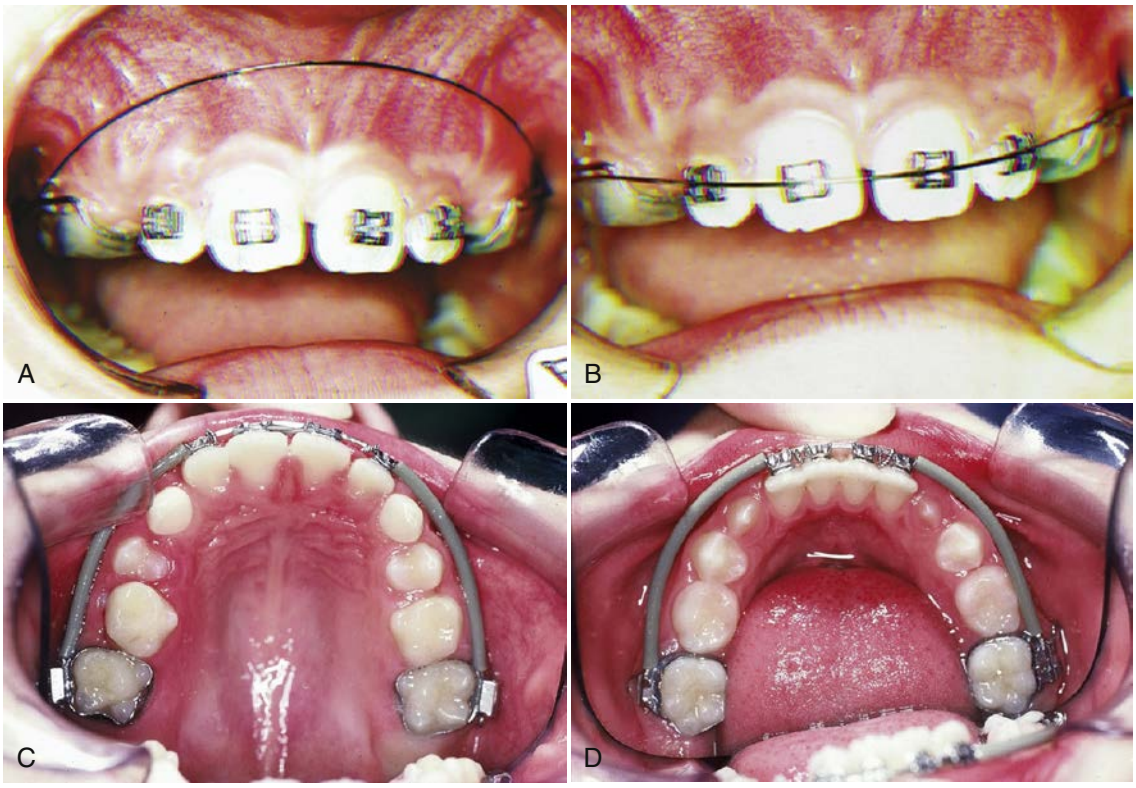


FIGURE 35-23 A, B, Molars and incisors are usually bonded, and light arch wire force quickly aligns malposed teeth and corrects any vertical discrepancies. C, D, Buccal tubes have been added on first molars to align the incisors with the bonded brackets.

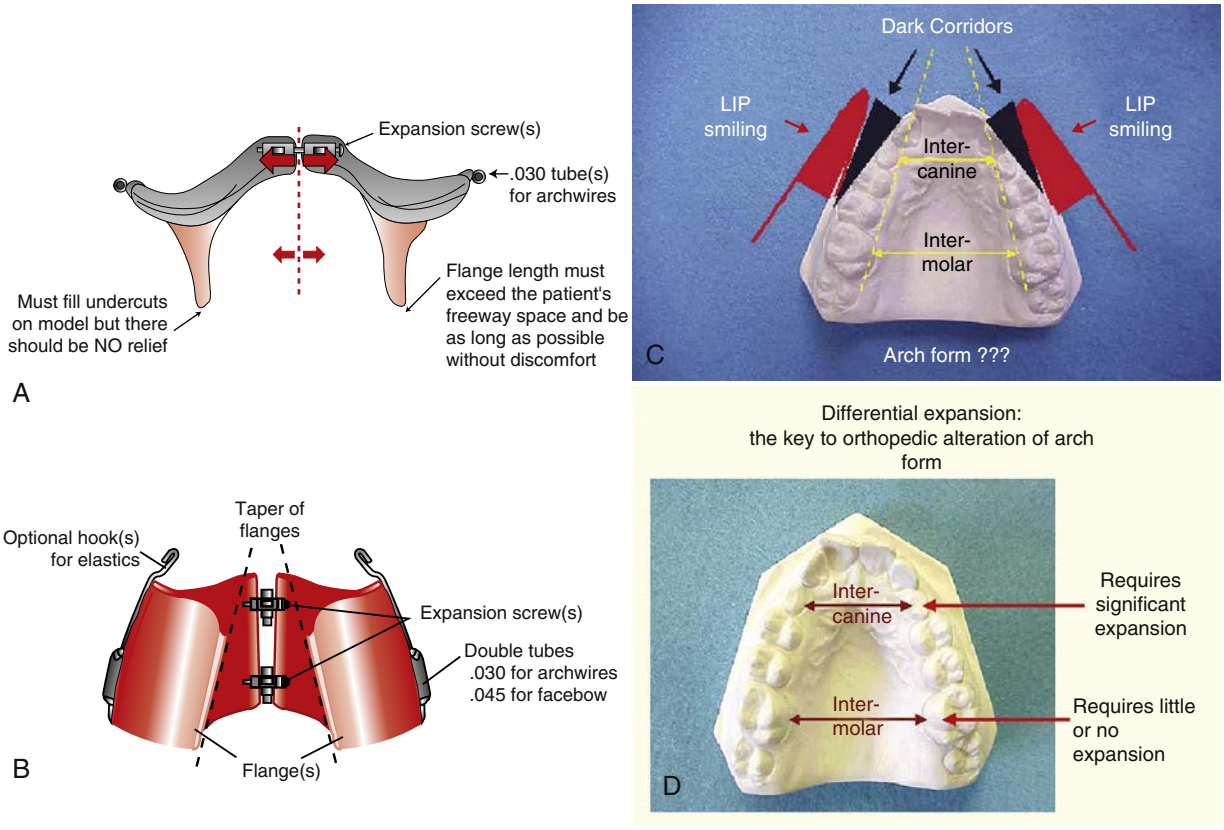


FIGURE 35-24 Hamilton dual arch expansion appliance; it can be removable or bonded to the maxillary arch. A, Note the length of the lingual mandibular flanges. One or two expansion screws can be used. Buccal tubes may be added for headgear or arch wire use. B, Drawing shows palatal view with lingual flanges extending toward the viewer. Note the double buccal tubes and twin expansion screws. Lingual extensions guide the mandible forward by proprioceptive contact with mandibular lingual mucosa. A maxillary labial arch may be added, as well as an acrylic cap for the lower incisors. This expansion/functional appliance is similar to the Balters' Bionator. The maxillary expansion screw void can be filled with acrylic after the desired expansion is achieved. C, Original maxillary narrow arch form is demonstrated with the canine dark corridors highlighted. D, Final arch form is natural.

Fränkel Appliance

Rolf Fränkel[93] probably did more to interest American orthodontists in functional appliances than any single clinician. His outstanding long-term three-dimensional results, the spectacular improvements that have stood the test of time and were done with the highest integrity, showed what can be done with carefully selected patients, properly designed appliances, an exercise routine (oral gymnastics), and maximal patient compliance. His impeccably researched clinical results are still the gold standard for all functional appliances, fixed and removable[106] (Figs. 35-25 and 35-26). Fränkel's insistence on full-time wear and on making the appliance (the functional regulator) an exercise device with oral gymnastics during the day demonstrates the significant role of function, per se. His requirement for anchoring the appliance at the maxillary first molar–second deciduous molar embrasure emphasizes the need for this or a similar design to maximize withholding the horizontal growth of the maxillary arch. The use of buccal shields to screen off potentially narrowing muscle forces and of lip pads in the lower labial vestibule to prevent abnormal perioral muscle function and lingualizing forces makes eminently good sense (see Fig. 35-25). Enlow and colleagues,[138] Moffett,[152] Graber,[30,121,153] and others[88,134,141] validate the fact that periosteal pull (i.e., viscoelastic stretch), as created by the Fränkel buccal shields, has the potential to stimulate bone growth (i.e., expansion of the transverse dimension) and is so well illustrated in cases treated with the functional regulator (see Fig. 35-30). Fränkel's step-by-step advancement, so easily achieved by the unique appliance design, has proved to provide the best and most stable results, and these results are applicable to other functional appliances.

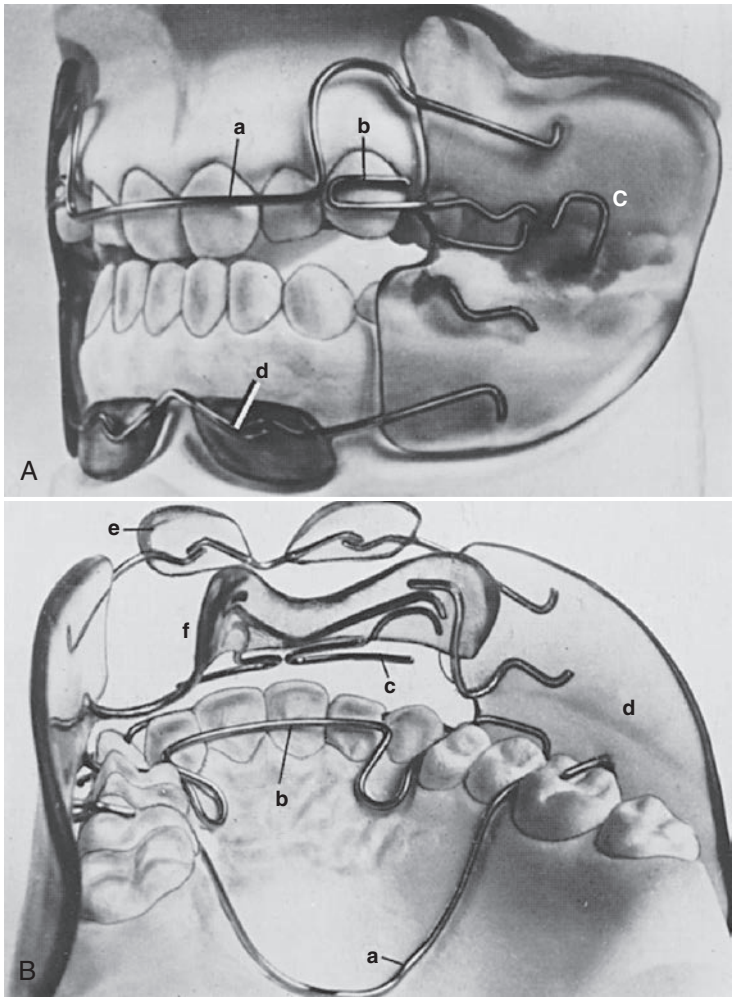


FIGURE 35-25 Fränkel function regulator makes maximum use of the vestibule as an area of operation, screening away abnormal lip and cheek pressures. **A,** Labial and buccal views. The wire assembly anchors the appliance on the maxillary arch at the mesial first molar embrasure. (*a*) Labial bow; (*b*) canine loop; (*c*) buccal shields; (*d*) lip pads (pelots). **B,** No interocclusal acrylic is used, and no interference with eruption of mandibular teeth occurs. A lingual acrylic pad or plate is the only contact with the lower arch, maintaining it in a protrusive relationship in the trough provided by the lingual acrylic pad and the lip pads. Sagittal advancement is usually accomplished in two steps of 3 mm each, with a simple advancement of the posturing trough. Less than full-time wear (except at mealtimes) diminishes the effectiveness of the appliance. (*a*) Cross-palatal stabilizing wire on the maxillary arch; (*b*) maxillary looped lingual arch or protrusion bow; (*c*) lower lingual wires; (*d*) buccal shield; (*e*) lip pads; (*f*) lower lingual pad or plate.

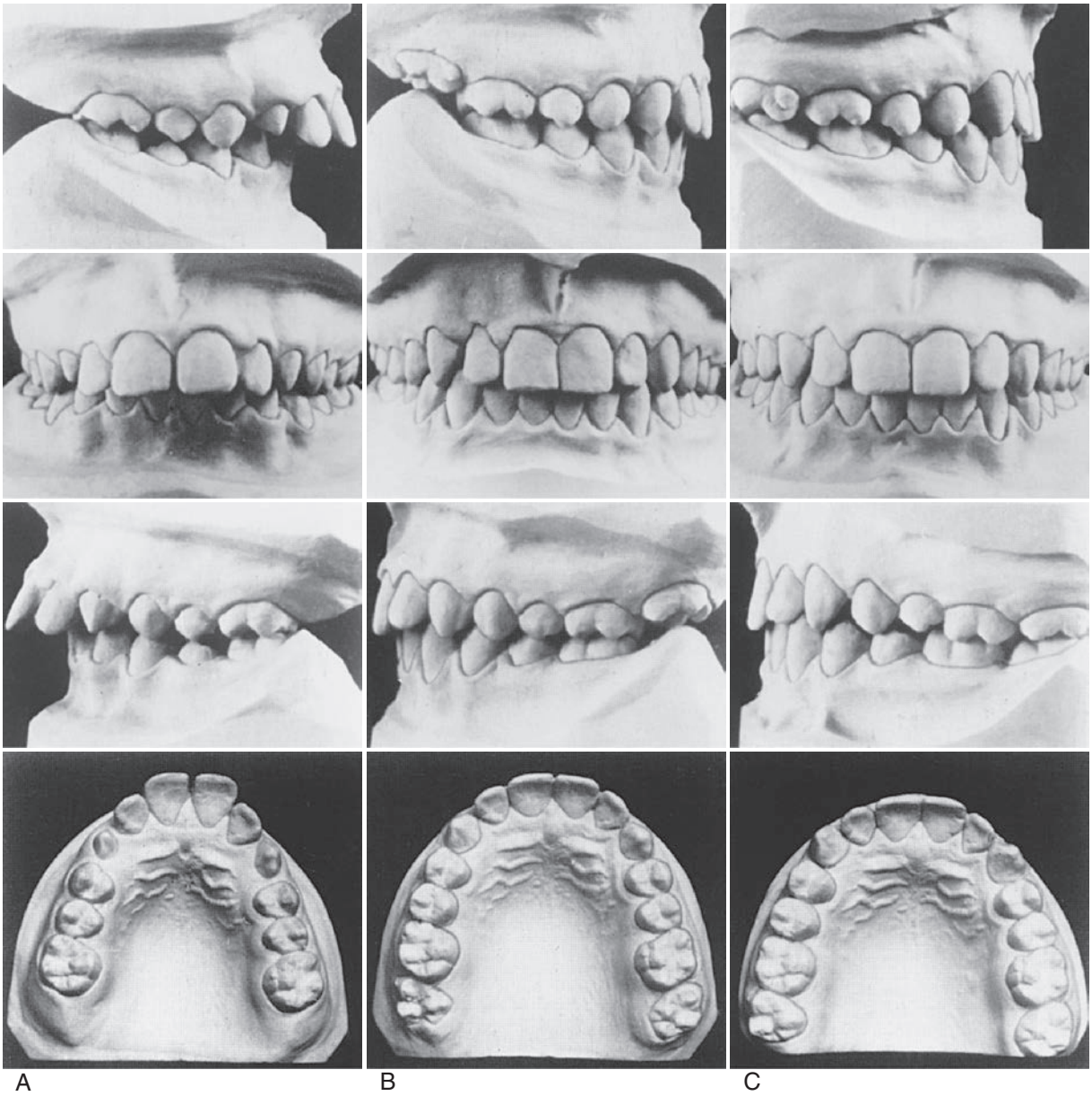


FIGURE 35-26 Fränkel therapy is responsible for this treatment result. **A,** Beginning study models. **B,** Autonomous expansion achieved by screening off restrictive muscle activity. **C,** After 3½ years of treatment, with no retainer control. Note the autonomous arch expansion. (Courtesy Rolf Fränkel.)

Unlike the activator, however, which can be modified in many ways and still produce an acceptable result, the Fränkel appliance demands a rigid discipline of design, fit, and use, as well as proper treatment timing and case selection. Patient daytime wear is a motivational challenge for the orthodontist and has been an Achilles' heel in that too many clinicians have not been able to replicate Fränkel's spectacular results.[154,155] This failure is partly because of deficiencies in appliance construction and a lack of experience on the part of the clinician; primarily, however, failure is the result of the lack of sufficient compliance on the part of too many patients (i.e., full-time wear). Of all the appliances, the functional regulator is the one that mostly depends on function, proper fabrication, sufficient length of wear, and cooperation. Some of its features have been incorporated into modifications of fixed functional appliances and activators with beneficial results. However, the popularity of the pure functional regulator has waned, not because of questions about the validity of the philosophy, but because of pragmatic use, more demanding laboratory procedures, and compliance demands. (For a full discussion of this approach, see *Dentofacial Orthopedics with Functional Appliances*, 2nd ed., by Graber and associates.[10])

Double Plates

Martin Schwarz[131,156] recognized early that a monobloc was bulky and difficult to wear. Therefore he introduced the double plate functional approach. Upper and lower removable appliances were held in place by wire appurtenances. Opposing occlusal

guiding ramps postured the mandible forward as the jaws were closed. Daytime wear and patient compliance were significantly improved. The Schwarz plate was the historical precursor of William Clark's twin block appliance, which has now become the most widely used functional appliance in the world.[126]

Clark Twin Block Appliance

The Clark twin block appliance is a modern, highly successful functional appliance approach modeled after the Schwarz double plate.[37,44,126,157] William Clark has developed this combination of upper and lower appliances to a high degree of efficiency. His book describes all the technical features in detail and shows many examples of successfully treated cases of all categories.[126] Occlusal guide planes, selective grinding, guided eruption, and a midpalatal jackscrew allow for three-dimensional control.

Using wire appurtenances and a jackscrew with the upper and lower inclined planes, the clinician has three-dimensional control (Figs. 35-27 to 35-35). Ease of full-time wear and selective guidance of eruption are attractive features. Similar to Hamilton, McNamara, and others, Clark recognizes the need to restore normal maxillary width and accomplishes this in his active arch development stage of treatment. Some clinicians modify the Clark maxillary component with two expansion screws for differential expansion, similar to Hamilton (see Chapter 16). As with other functional appliances, this modification may also be made ahead of time for transverse deficiencies. The twin block appliances can also be bonded, as with the Hamilton expansion activator. If desired, rare earth magnets in an attraction mode can be incorporated into the guide planes, as Moss[142] has done with his functional appliance design. The magnets stimulate repetitive functional activity and TMJ metabolism and optimize response, similar to the Vardimon magnetic appliance discussed later in this chapter.[60,158,159]

The orthopedic phase with the twin blocks recognizes the need for eruption of lower molars to eliminate excessive overbite and deficient vertical dimension. Because the mandibular molars are not covered, they are free to erupt in an upward and forward vector, aiding in the Class II sagittal correction (see Fig. 35-29). Of course, in open bite cases, the molars are covered to prevent eruption and to close down the bite.

Functional appliances resort to reciprocal Class II dental correction in a noninvasive manner, avoiding the potential iatrogenic sequelae of periodontal involvement, gingival inflammation, crestal bone loss, and root resorption or dilaceration. This reciprocal upper and lower dental adjustment must be

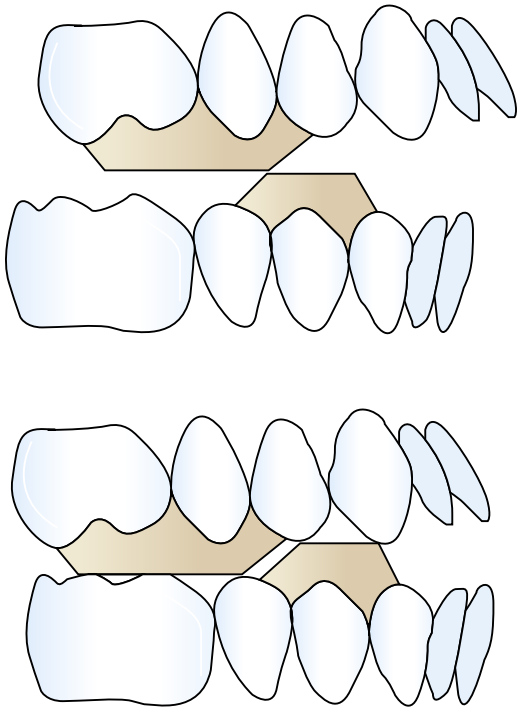


FIGURE 35-27 Clark twin blocks are modified double plates with occlusal inclined planes. They were introduced by Martin Schwarz to reduce the bulkiness of the traditional monobloc. The twin blocks meet at approximately 70 degrees, with the lower inclined plane mesial in a Class II correction.

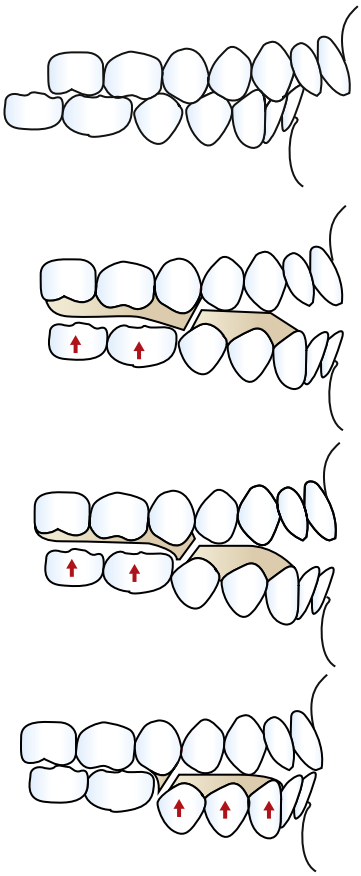


FIGURE 35-28 Twin block occlusal relief used to expedite eruption of lower molars and reduce overbite.

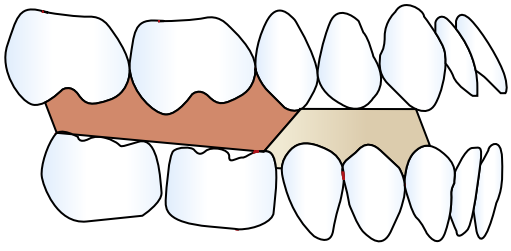


FIGURE 35-29 In patients with open bites, occlusal cover is maintained over posterior teeth to achieve all possible depression of the upper and lower molars.

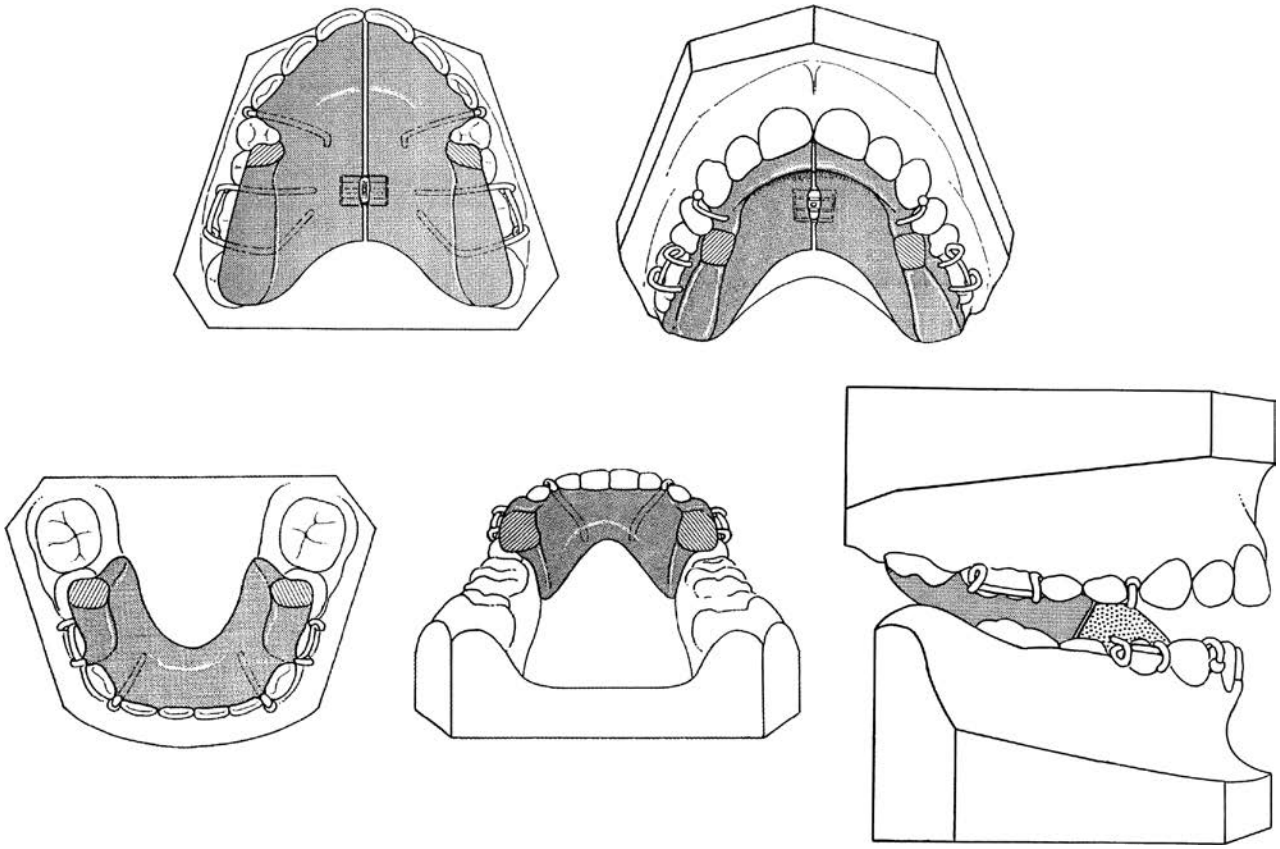


FIGURE 35-30 Standard Clark twin blocks. Hash marks denote the occlusal inclined guiding planes. (Courtesy of W. Brudon and J.A. McNamara Jr, University of Michigan. IN Clark WJ: Twin block functional therapy, ed. 2, London, 2002, Elsevier, Ltd)

controlled, of course. Too often, conventional functional appliances do not control mandibular incisors, which become excessively proclined during treatment. To obtain dental correction with biocompatible measures is not maltreatment, however. For example, lower incisors that are initially lingually inclined as the result of a perverted lip trap can be greatly improved. Acrylic capping of lower incisors also reduces the tendency to procumbency. In any event, Hansen and Pancherz[31] note considerable recovery from lower incisor proclination after treatment in their long-term studies.

If rare earth magnets are used for phase I of twin block therapy, then they are embedded in attraction mode on the 70-degree inclined planes of upper and lower appliances. Functional activity and interarch contact are thus enhanced. If the appliance is not bonded, then the patient is instructed to wear it all the time, removing it only for cleaning. Moss[160] has reported using repelling rare earth magnets in a different configuration, again to assist in forward posturing of the mandible. A thin layer of acrylic covers opposing magnetic surfaces to prevent breaking the parylene seal and thus fostering corrosion. After the rapid and desired sagittal correction has been achieved, the twin blocks are worn for an additional 2 to 3 months to stabilize the occlusion and to allow for needed dental compensation (e.g., posterior eruption, which occurs more slowly). Compensation is a common problem for all functional appliances because sagittal correction is achieved before vertical treatment objectives are reached. The acrylic is trimmed away from the contiguous maxillary occlusal surfaces to allow for unimpeded molar and premolar eruption. As Buschang and Santos-Pinto[24] note, nine times as much vertical growth as sagittal growth occurs in the human face. The lower appliance is usually discontinued unless arch length has been gained, and an inclined plane is added to the maxillary appliance, which serves as an active or short-term retainer for phase II during the continued eruption of posterior teeth as they lock into correct interdigitation. Many studies emphasize the importance of a *locked occlusion* to prevent sagittal relapse.*

Magnetic Appliances

Blechman,[163,164] Bondemark and Kurol,[165] and Vardimon and colleagues,[60,159] as well as Darendelilier and associates[166] and Joho and Darendelilier,[167] have been aware of the potential for using rare earth magnets in orthodontics and dentofacial orthopedics for some time. Blechman,[168] the true pioneer, has been intimately involved in the medical and dental use of rare earth magnets. Recent medical research corroborates his observations that static magnet fields may have an electric field effect that potentiates tissue response.[65,168,169] Despite widespread and increasing use of magnetic adjuncts in general orthopedic problems, in vitro orthodontic research has produced mixed

* References 20, 31, 37, 44, 47, 53, 54, 57, 60, 159, 161, 162.

FIGURE 35-31 Deforming perioral muscle function is noted in an 8-year-old boy before twin block treatment.

results thus far. Is it a question of not knowing what to look for? Heightened blood flow is clearly evident, and the alignment of blood cells is influenced by rare earth magnets, but the magnitude or character of force, its duration, and whether continuous or intermittent force is applied are questions currently being addressed, particularly in medical orthopedics, in which considerable improvement has been noted in specific types of problems, such as back and joint disorders, fracture nonunion, and pain threshold management. The clinical use of magnets in such cases is widespread.

In orthodontics, Vardimon and colleagues[170] have demonstrated the clinical use of magnets for the deimpaction of maxillary canines, which can achieve the desired result in as little as one third of the time required by conventional traction technique. The article received the B.F. Dewel Award in 1992 as the best article in the *American Journal of Orthodontics and Dentofacial Orthopedics* the previous year. With the magnetic deimpaction, the tissue attachments and alveolar bone height are normal rather than the compromised gingival margins observed in conventional *expose and pull* techniques. No evidence of root resorption was seen.

Despite the increased exercise effect of magnets incorporated into functional appliances, which enhances the TMJ–condylar growth metabolism, no compelling evidence currently exists of a significantly enhanced localized tissue effect. This lack of evidence may be because the magnets are not contiguous to the tissues but are separated from them by acrylic barriers. At this time, magnetic functional appliances do not appear to be much more effective than nonmagnetic designs in a nongrowing child. Improper case selection, improper design, and injudicious use can cause unwanted lower incisor proclination, as with any other functional appliance. Incorporating Fränkel-type buccal

FIGURE 35-32 The patient in Fig. 35-31 is seen after 14 months of twin block treatment. Note the use of a biteplate during this support phase to stimulate posterior eruption because vertical dimension improvement often takes longer than sagittal correction.

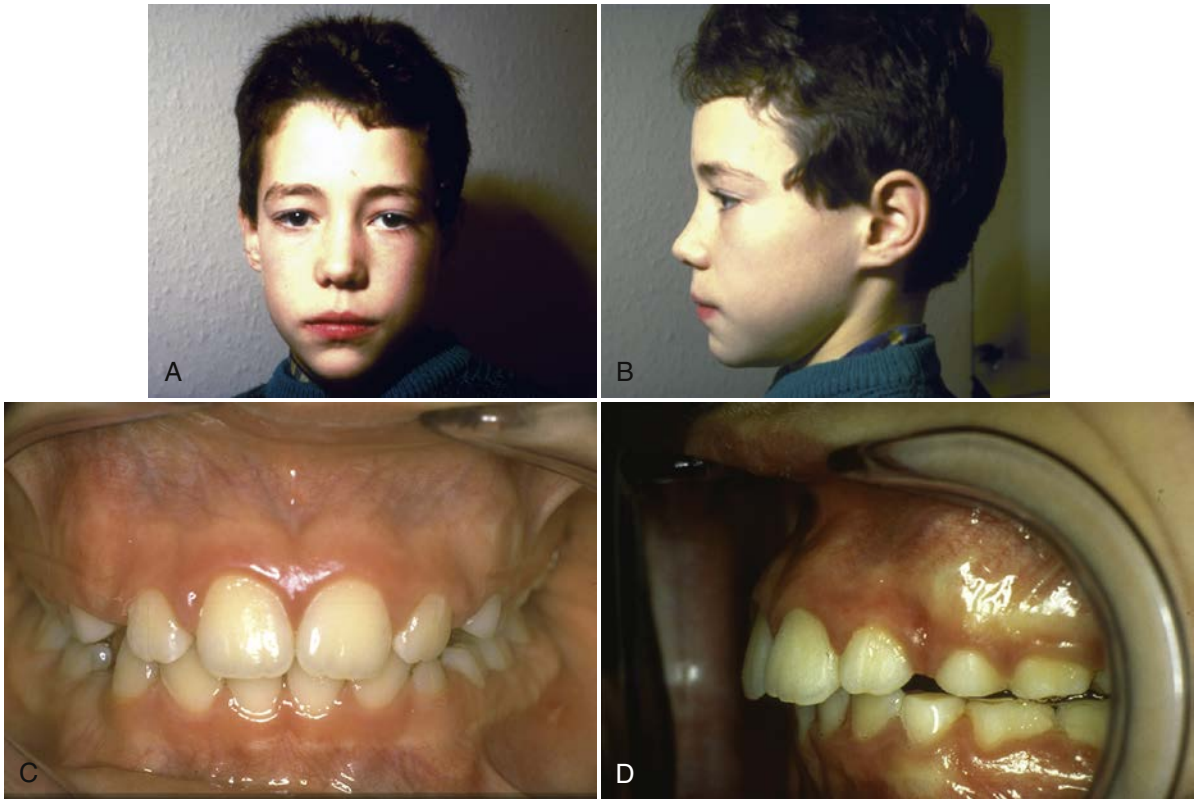


FIGURE 35-33 Facial and intraoral views depicts the patient in Figs. 35-31 and 35-32.

FIGURE 35-34 Facial (**A, B**) and intraoral (**C, D**) views of the patient in Figs. 35-31 to 35-33 are depicted after a short period of fixed mechanotherapy for detailing.

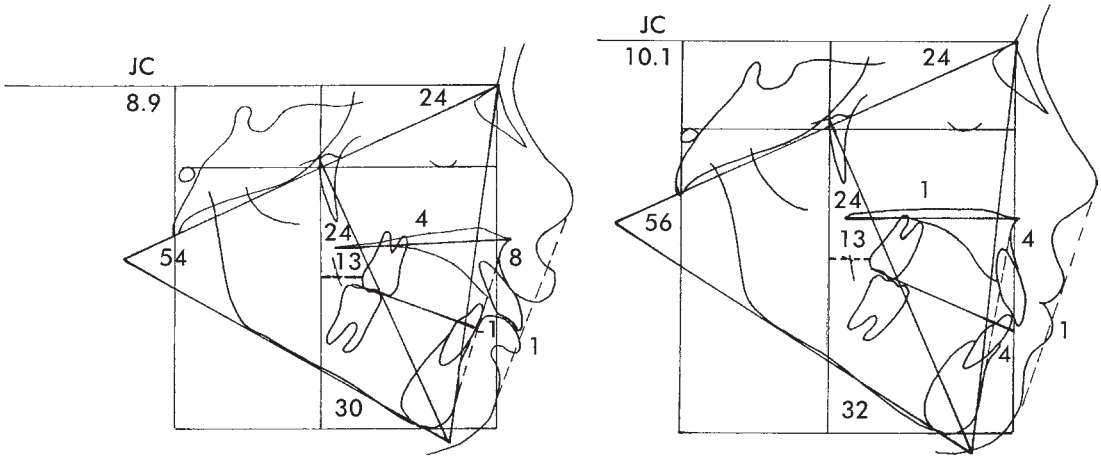


FIGURE 35-35 Cephalometric tracings of the patient from Figs. 35-31 and 35-32 were created after a 14-month interval. (Courtesy WJ Clark.)

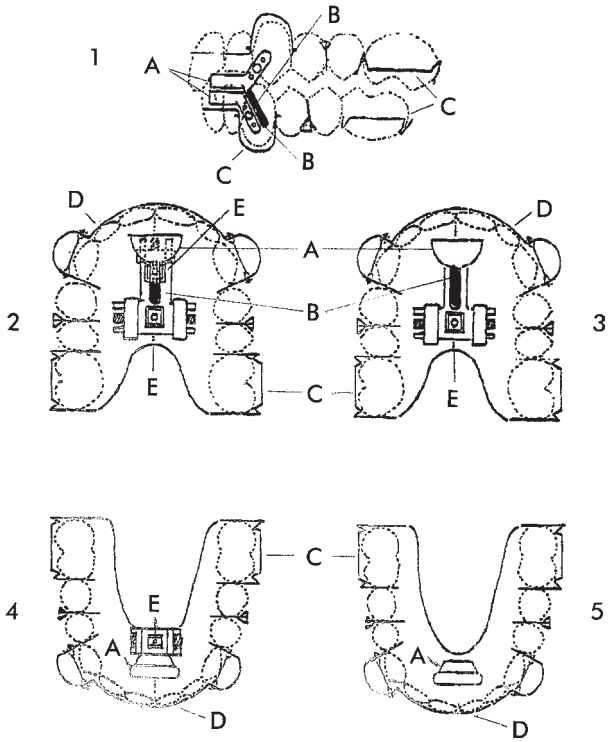


FIGURE 35-36 Functional magnetic appliance. **1,** Lateral view with magnets together in a protrusive relationship. **2,** Maxillary appliance with jackscrews for expansion and incisor segment protraction. **3,** Maxillary appliance with expansion jackscrew only. Note the guidance prongs in the maxillary appliance. **4,** Mandibular appliance with expansion jackscrew. **5,** Mandibular appliance with magnet only (*A*) embedded in lingual acrylic (see Fig. 35-38). *A,* Miniaturized rare earth magnets coated with parylene to prevent corrosion; *B,* maxillary forward guidance prong; *C,* retention clasps; *D,* labial wire; *E,* jackscrew. (Courtesy Andy Vardimon.)

shields or the buccal loops of the Bionator appliance in patients with narrow maxillary arches or perverted perioral muscle function may be beneficial in magnetic and other types of functional appliances, replacing the jackscrew. The functional appliance, as with any other mechanism, should be *the thinking orthodontist's approach* and not a procrustean bed or mechanical straitjacket for all patients.

Vardimon and associates have had significant success with magnetic functional appliances (Figs. 35-36 and 35-37). A report in the *American Journal of Orthodontics and Dentofacial Orthopedics* highlights the uniform success achieved.[76] Two case reports are shown in Figures 35-38 and 35-39 to demonstrate Vardimon's success with magnetic appliances.

Stöckli-Teuscher Combined Activator-Headgear Orthopedics

Long-term research with even the most efficient removable functional appliances demonstrates a minimal distalizing effect on the maxillary complex.[23,42,100,158,171] A withholding of the anterior component of maxillary change is probably the most that can be expected. Fixed functional appliances such as the Pancherz modification of the Herbst appliance, the Jasper Jumper, and the De Vincenzo Eureka Spring, all described in this book, can definitely distalize the maxillary molars and may also have some effect on the maxillary downward and forward growth pattern with possible enhancement of horizontal mandibular growth.

Traditionally, despite the mandibular underdevelopment in most Class II malocclusions, American orthodontists have attacked the maxilla with extraoral force, producing a compromise result to fit the maxilla to the deficient mandible. Calling it *camouflage treatment* does not make it any more legitimate. Because the classic activator is primarily directed against the mandible, what is needed in the best of all worlds is a device that affects a correction using significant changes in both the maxillary and the mandibular basal structures (i.e., growth patterns), plus dentoalveolar compensatory changes. Thus the mechanotherapy must adapt treatment to dentofacial growth in an optimal manner, eliminating three-dimensional malocclusion problems.

Normally, the sagittal and vertical growth of the condyles and change in the TMJ glenoid fossa position and morphologic structure are balanced by the downward and forward growth of the maxilla. The great anatomist Harry Sicher attributed the bulk of maxillary basal change to sutural growth.[10] As Coben[172] emphasizes, however, growth of the sphenooccipital synchondrosis is a significant factor in both a lengthening of the cranial base and the sagittal midface development, as well as influencing the position of the glenoid fossa (Figs. 35-40 to 35-43). This critical area has been largely ignored in orthodontic research and diagnosis.

If the magnitude and nature of the growth processes are such that the vertical component is dominant, then the mandibular symphysis will move more vertically. If the horizontal vector is greater, then the chin point will move more sagittally. Functional appliances, alone, actually attack only part of the problem. Korkhaus noted, referring to functional orthopedics, "We effect the correction of distoclusion exclusively through the mesial development of the entire lower jaw."[173] However, is this enough, particularly when definite limitations exist on the therapeutic change of the mandibular position and morphologic structure?

Stöckli and Teuscher persuasively argue in the second and third editions of this text that a combined assault against the maxilla using a functional appliance and extraoral force produces a more dramatic and stable correction skeletally, cosmetically, and functionally. Figures 35-41 to 35-44 illustrate the challenges. Figure 35-41 shows average natural growth and displacement of the maxilla and mandible. The result is harmonious downward and forward growth. If mandibular growth is deficient, then a more horizontal lower jaw vector must be stimulated to compensate for the sagittal discrepancy, essentially producing the entire correction with mandibular basal change and stable dental compensation (see Fig. 35-42). Research shows that solely skeletal correction is not possible in many cases with functional or conventional fixed appliances.[174] The orthognathic surgeon recognizes the problem and can effect such a skeletal change through surgical repositioning or distraction osteogenesis. However, this procedure is beyond the domain of conventional orthodontics and this chapter. Figure 35-43 shows the potential of combined assault on the maxillary and mandibular sagittal discrepancy. A more realistic orthopedic possibility is combined maxillary withholding, plus mandibular and fossa forward displacement, plus vertical control of dental and skeletal spatial relationships. Neither functional appliances nor extraoral force alone can routinely

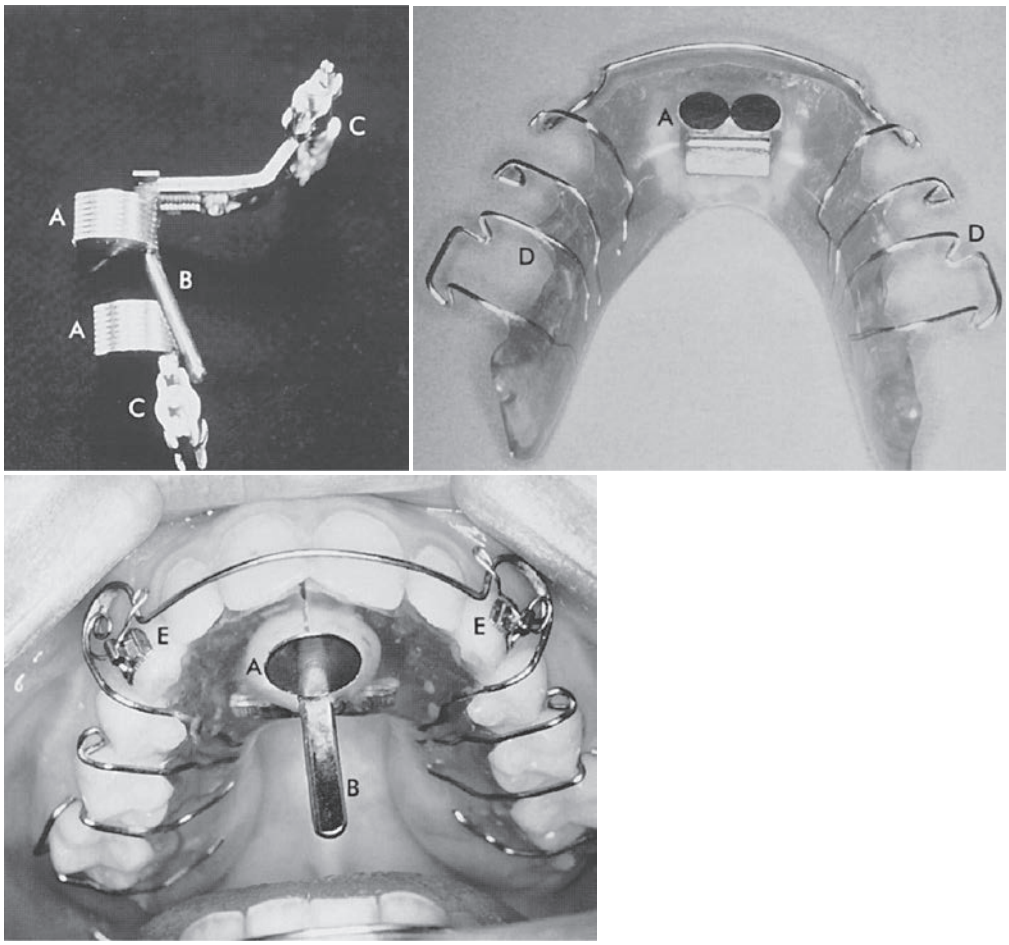


FIGURE 35-37 Vardimon functional magnetic appliance. Upper and lower modified Schwarz plates, similar to the Clark twin block appliances, are used. Instead of bilateral-angled magnetic inclines, lingual rare earth magnets are buried in the acrylic at the midline. A maxillary guiding prong that slides on the lingual acrylic of the lower appliance during closure brings the mandible forward into the desired protrusive relationship as the attractive magnets contact. This unloads the condyle in much the same way as other functional appliances, except that the functional stimulus occurs more often with the *make-and-break* magnetic contact. Temporomandibular joint metabolism is enhanced in this manner. Patient compliance is apparently improved. Transverse control can be achieved by incorporating palatal jackscrews if needed. The upper and lower magnets have not been incorporated into the acrylic; rather, they are positioned to show the glide path as they approach each other. Magnetic attraction exists as far as 2 cm (*top views*). Small elastics hooked above bonded canine brackets to enhance retention of the maxillary functional magnetic appliance (*middle view*). *A,* Magnets; *B,* guidance prong; *C,* metal tags for incorporation of upper and lower appliances into the acrylic; *D,* retention clasps; *E,* elastics hooked over bonded brackets.

FIGURE 35-38 Patient FN was 11 years, 3 months old when functional magnetic system (FMS) treatment was initiated. His Class II malocclusion was due to prognathic maxilla and mild retrognathic mandible (McNamara analysis: Point A to Nasion Perpendicular line = 4mm, Pogonion to Nasion Perpendicular line = −8mm. Retrognathic mandible according to Steiner analysis: SNA angle = 80 degrees, SNB angle = 72 degrees.) Pretreatment records: **A,** Profile. **B,** 3/4 Profile,. **C,** Overjet. **D,** Frontal view with excessive overjet; **E,** Left occlusion. First FMS treatment stage lasted 9 months. A full Class I molar relationship was established, with the canine in almost a Class I relationship, which resulted in overjet remnant. **F,** Upper FMS appliance. **G,** Lower FMS appliance. **H, I,** Increased bite clearance with the FMS. **J,** End of FMS treatment. **K, L,** Second edgewise treatment stage: during treatment. **M, N,** Posttreatment profile and smile. **O,** Schematic illustration provides the percentage correction of each component. Without taking growth into account, major treatment changes occurred in the mandible (75%) and the upper molar (75%). However, after subtracting growth, the mandibular skeletal contribution dropped to 12.3% and the maxillary dental contribution (molar distalization) increased to 92%. *OLp,* Occlusal line perpendicular. (**A–J,** Courtesy Abraham Kyriakides. **K–N,** Courtesy Eleni Dre.)

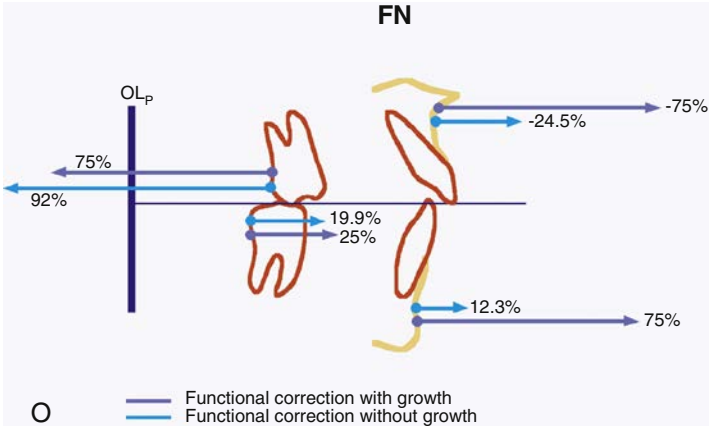


FIGURE 35-38, cont'd

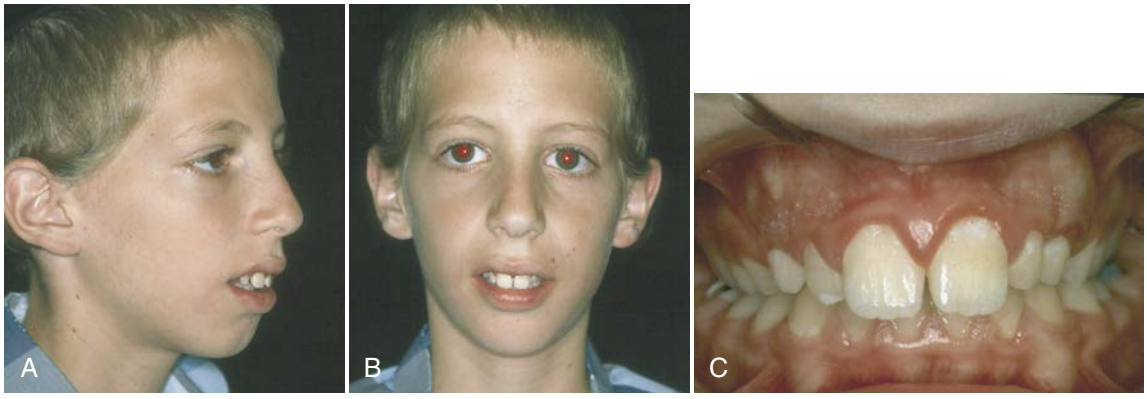


FIGURE 35-39 JM, a 12½-year-old male patient, had a Class II malocclusion by virtue of retrognathic mandible (SNB = 71 degrees, Pogonion to Nasion Perpendicular line = −17 mm), overjet of 9 mm associated with deep bite (6 mm), and hypoplasia of upper lateral incisors. Pretreatment records: **A,** Profile. **B,** Smile. **C,** Frontal view.

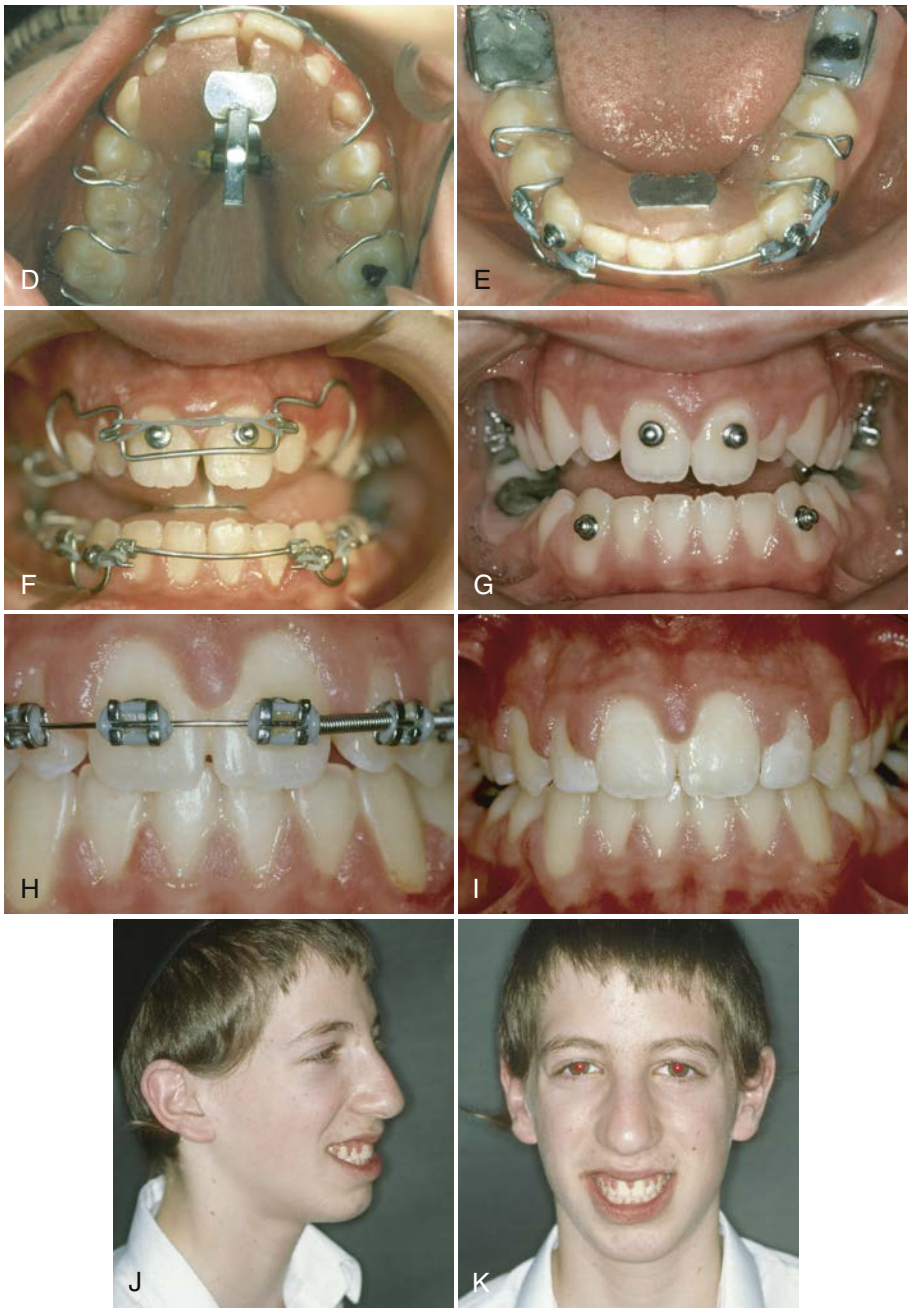


FIGURE 35-39, cont'd D, E, Treated with the functional magnetic system (approximately 24 months) because of an initial poor compliance that required bonding the appliance for 3 weeks. F, G, The upper central incisors served as abutments for the functional magnetic system appliance by reason of the delayed eruption of the upper canines. H, The bite clearance was increased to facilitate the guidance of eruption of the posterior segment. I, Posttreatment occlusion included restorative recontouring of the lateral incisors with composite. The pubertal gingival hypertrophy was left untreated because of expected self-improvement after bracket debonding. J, K, Post-treatment profile and smile. L, Schematic illustration of functional correction, mandibular skeletal contribution was reduced by one half (20.2%) when growth was considered. This reduction was followed by an increase in upper distalization contribution (from 27.3% to 40%). *OLp,* Occlusal line perpendicular. (A–H courtesy Costas Ergatoudes; I–K courtesy Vasilis Kalamatas.)

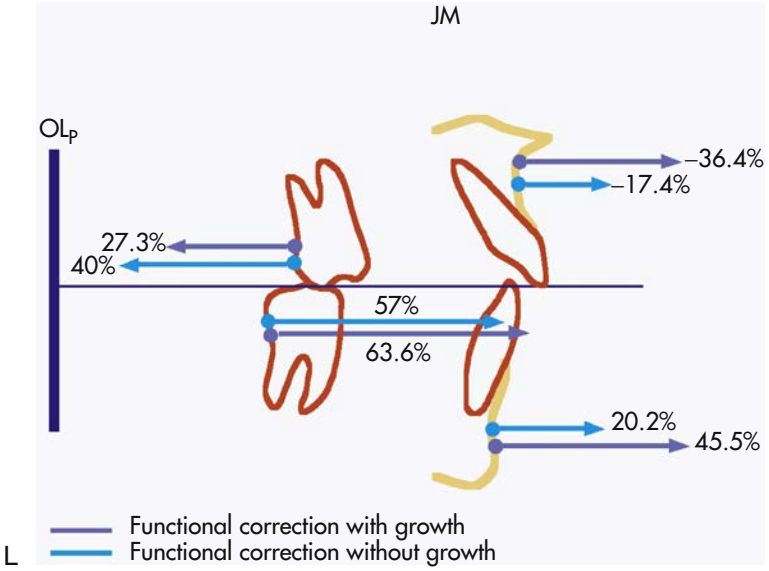


FIGURE 35-39, cont'd

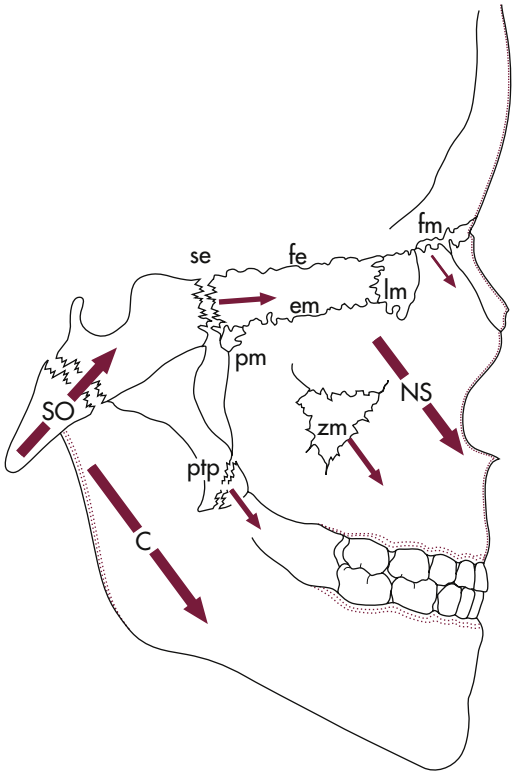


FIGURE 35-40 Growth directions of the cranial base and facial sutures, with a resultant expanding V, is accomplished as the cranial portion moves upward and forward from the sphenooccipital synchondrosis (*SO*) and the facial portion moves downward and forward. *C,* Reflection of condylar and ramal growth; *NS,* nasal septum growth vector; *se,* sphenoethmoidal suture; *pm,* pterygomaxillary fissure; *ptp,* pterygopalatine suture; *fe,* frontoethmoidal suture; *em,* ethmoidal-maxillary suture; *lm,* lacrimal-maxillary suture; *fm,* frontomaxillary suture; *zm,* zygomaticomaxillary suture. (Adapted from Coben SE: Growth and Class II Treatment, Am J Orthod Dentofacial Orthop 52:5-26, 1966. With permission from the American Association of Orthodontists.)

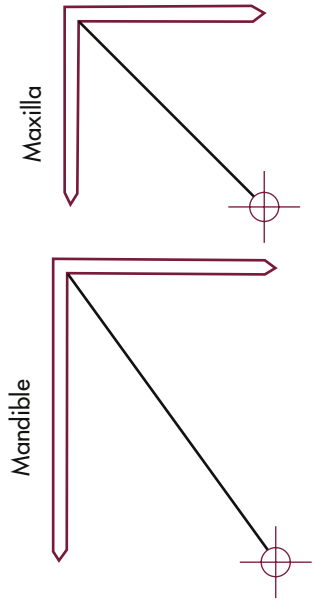


FIGURE 35-41 Average natural displacement pattern of the maxilla (Point A) and the mandible (pogonion), relative to the anterior cranial base. The horizontal and vertical vectors (*white*) and the resultant vector of displacement are shown. In an average facial growth pattern, the forward component of the mandibular symphysis displacement is slightly greater than that of the anterior apical base of the maxilla. The difference is marked at the bottom. Of course, as Coben points out, if superimposition occurred on the entire cranial base, including the sphenooccipital synchondrosis, then the result would be different.

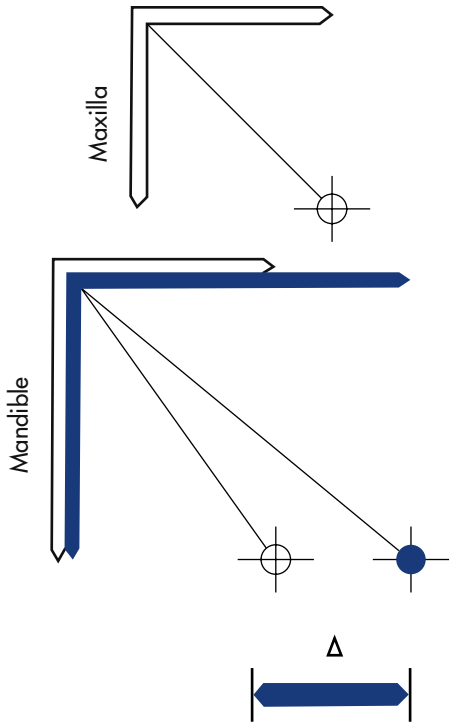


FIGURE 35-42 The concept of stimulating specific forward growth, with mandibular displacement (*black*) to bring about the so-called jumping of the bite in Class II treatment with functional orthopedics, can be an unfulfilled orthodontic "dream" because the actual change that occurs without appliance interference cannot be measured at the same time. The morphogenetic pattern, treatment timing, appliance used, and patient compliance are important determining factors. For these reasons, forecasting is a difficult, if not impossible, task.

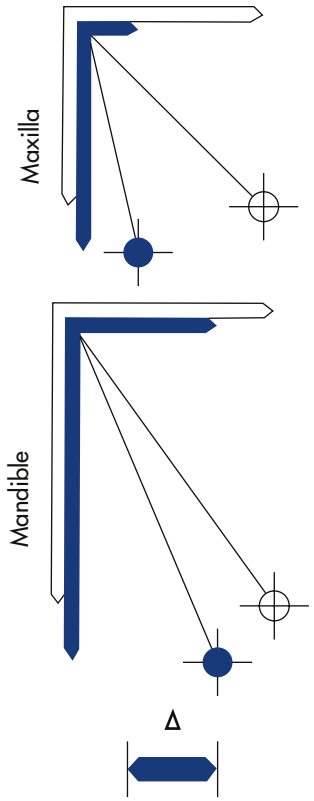


FIGURE 35-43 Reduction of the sagittal component of maxillary skeletal and dentoalveolar structures (*black*) is compared with the average values. Stöckli and Teuscher believe that this possibility is more realistic with functional appliances, despite the research of McNamara and others, which shows little effect of functional appliances on the maxilla. The use of an extraoral appliance (headgear) does enhance sagittal withholding or a distalizing effect on the maxilla, hence the justification for combined functional appliances and headgear. In many cases, however, rotational and elongating reactions by the maxilla and dentoalveolar structures create additional vertical demands and reduce the potential sagittal correction, causing an opening mandibular rotation that exacerbates the apical base discrepancy (*lower diagram*). Because of dominant vertical growth, as shown by Buschang, appliance control of this vector is essential (i.e., vertical growth is much greater than sagittal growth).

provide the desired correction.* A study by Altenburger and Ingervall,[100] comparing combination headgear-activator results with the results achieved using an activator alone, still does not show a significant distalizing effect with combination therapy. However, in a more recent study, Du, Hagg, and Rabie[27] show significant molar distalization when incorporating a headgear with the Herbst appliance.

Again, the reader is referred to the excellent chapter by Stöckli and Teuscher in the second edition of this book for techniques and treatment.[173] Figures 35-44 and 35-45 illustrate the activator-headgear appliance. Proper construction implies that the extraoral force component considers any possible rotational effect from extraoral force, which must pass as close as possible through the maxillary center of resistance.

Perhaps the best compromise, ensuring optimal stability of the functional appliance when the headgear is worn, is to place first molar bands and buccal tubes, insert the occipital pull extraoral force arms into the buccal tubes, and have the wire clasps of the functional appliance snap above the buccal tubes to give maximal retention for day and night wear of the functional appliance. Three-dimensional control of molar anchor teeth is a top priority, preventing unwanted opening of the bite. This two-jaw approach enhances anchorage and dentoalveolar compensation and provides optimal growth guidance. Various functional appliances can be used—a Bionator, twin block, or monobloc appliance—but with reduced bulk to permit full-time wearing.

Fixed Functional Appliances

Herbst Appliance

At the International Dental Congress of 1909 in Berlin, Emil Herbst presented a fixed bite-jumping device called Scharnier, or joint[179] (Figs. 35-46 to 35-48). The idea of continually keeping the mandible forward and eliminating the need for patient compliance, as is required with removable functional appliances, appealed to clinicians. In 1934, Herbst and Martin Schwarz wrote a series of articles describing their case selection,

* References 61, 113, 129, 158, 171, 175–178.

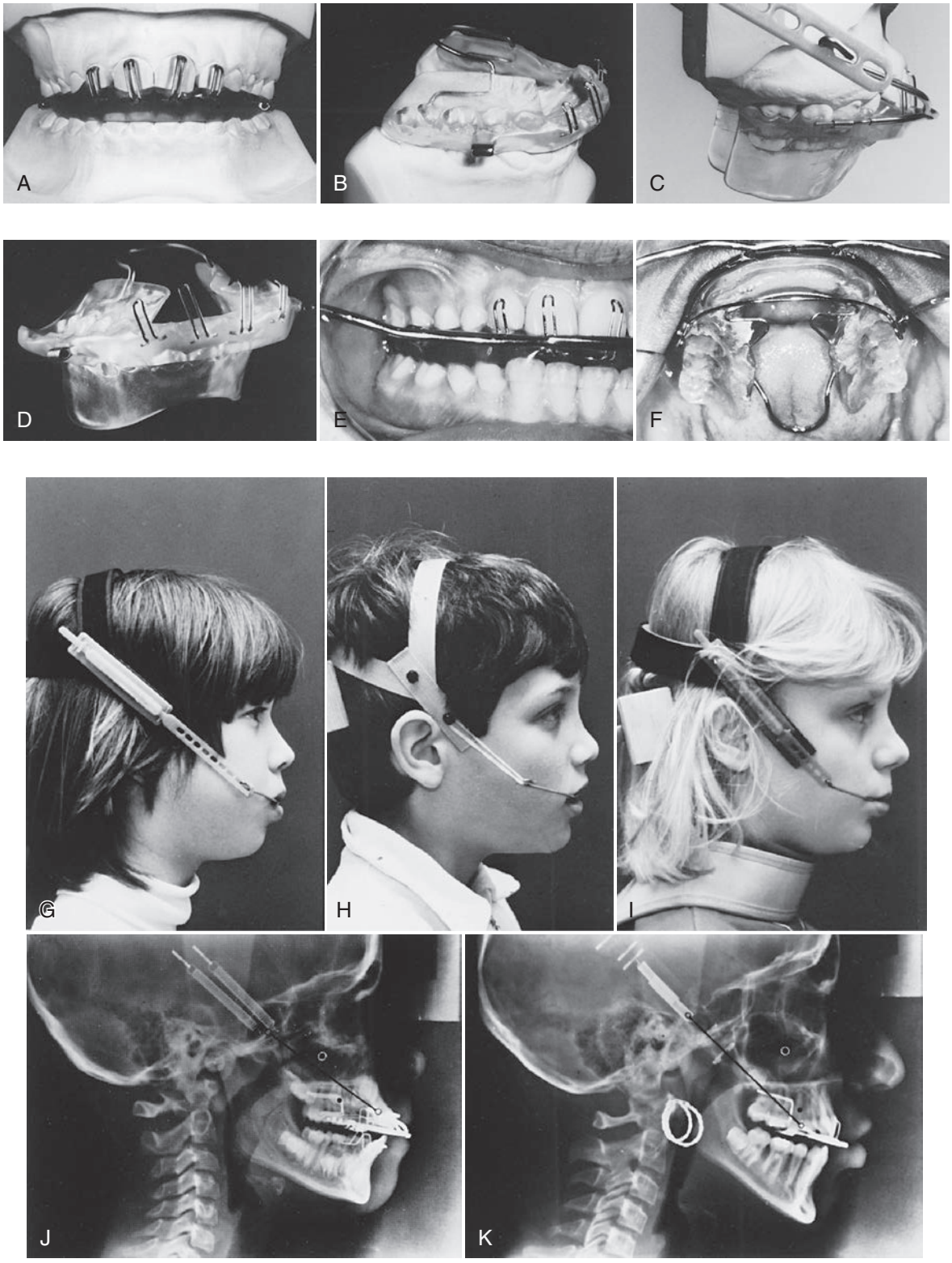


FIGURE 35-44 Activator-headgear appliance and extraoral attachments. **A,** Vestibular extensions and torquing springs are demonstrated. **B,** The appliance is placed on the lower cast. Note the anterior area with torquing springs, palatal extension of acrylic, and transpalatal wire. **C,** The appliance is placed on the upper cast with the facebow inserted. Note the lower lingual flange extension for engaging the mandibular arch, similar to the Hamilton appliance. **D,** Appliance view again shows the transpalatal bar, torquing springs, and lingual extensions. **E,** The activator-facebow is in place. **F,** The activator is in place on the lower arch, showing the insertion of the facebow. Note the tongue against the transpalatal Coffin spring. In this construction, labial and lingual wires are used instead of torquing springs. **G,** An anteriorly placed force vector is estimated in this case to pass just anterior to the center of resistance of the maxilla. Some anterior rotation of the dentition must be expected, and ample condylar growth is required. **H,** Average steepness of the force vector is estimated to pass slightly superior to the center of resistance of the upper dentition. The posterior rotational effect on the maxilla and the anterior rotational effect on the dentition should neutralize each other to ensure that no change is expected in the inclination of the occlusal plane. **I,** The posteriorly placed force vector is estimated in this case to pass just inferior to the center of resistance of the dentition. Posterior rotation of the maxilla and dentition must be anticipated. This setup is used for patients with an open bite tendency or when the prognosis of condylar growth is poor. **J, K,** Lateral cephalograms are provided to check force vectors (*line connecting the circle at the end of the outer arms of the facebow and the circle at the springs of the headcap*) to the centers of resistance of the maxilla (*white circle*) and upper dentition (*black dot*).

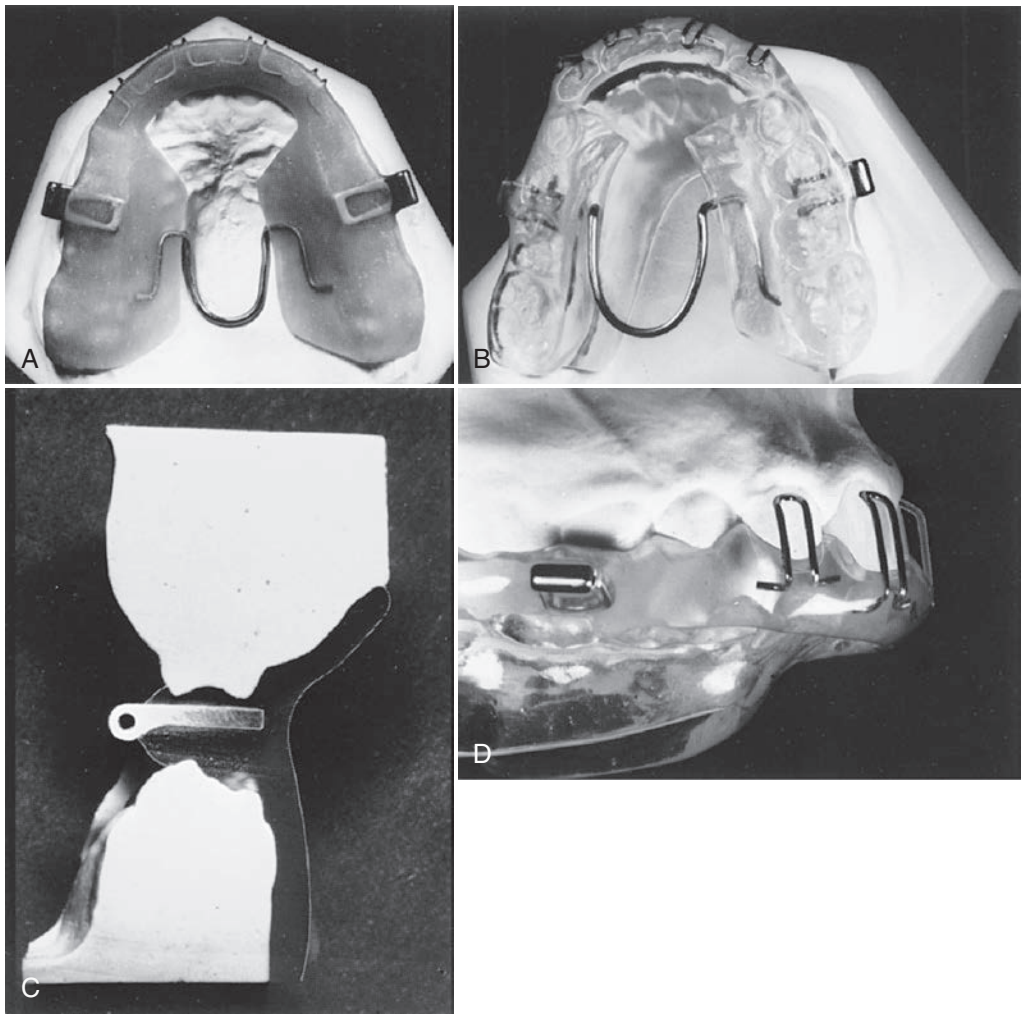


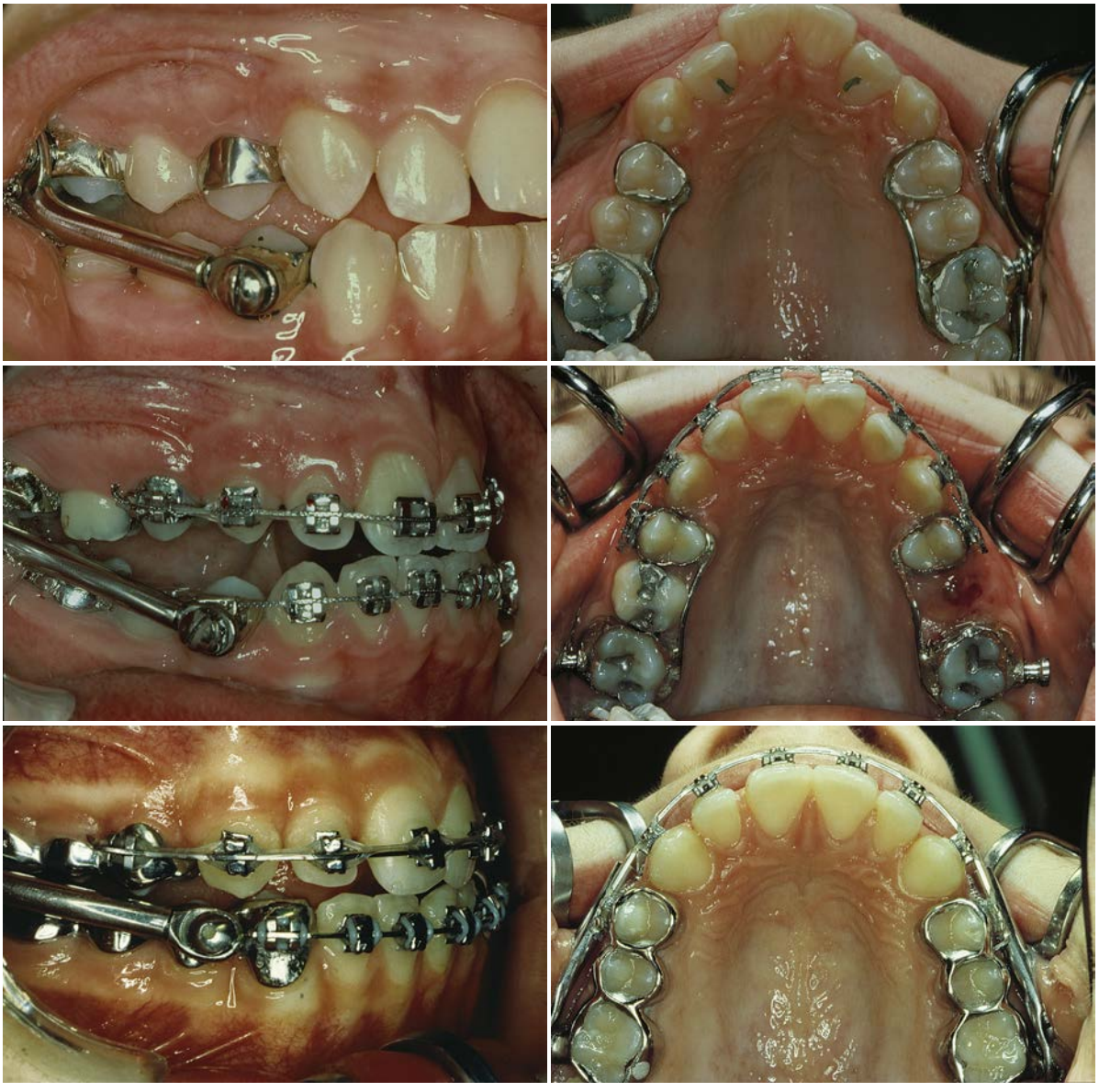
FIGURE 35-45 Maxillary component of the Stöckli-Teuscher appliance. **A,** Extension on the palatal side. Placement of headgear tubes and palatal wire; retention area of torquing springs. **B,** Occlusal and incisal replica relief of the upper teeth. **C,** Interocclusal placement of the headgear tube. **D,** Design and position of the torquing springs. Only the palatally curved tip should touch the crown, contiguous to the gingival margin. Note buccal tubes.

FIGURE 35-46 Working hypothesis of the Herbst appliance. (Courtesy Dentaurum, Berlin, Germany.)

FIGURE 35-47 The disassembled telescopic mechanism (plunger and tube) of the Herbst appliance is available in pairs. (Courtesy Hans Pancherz.)

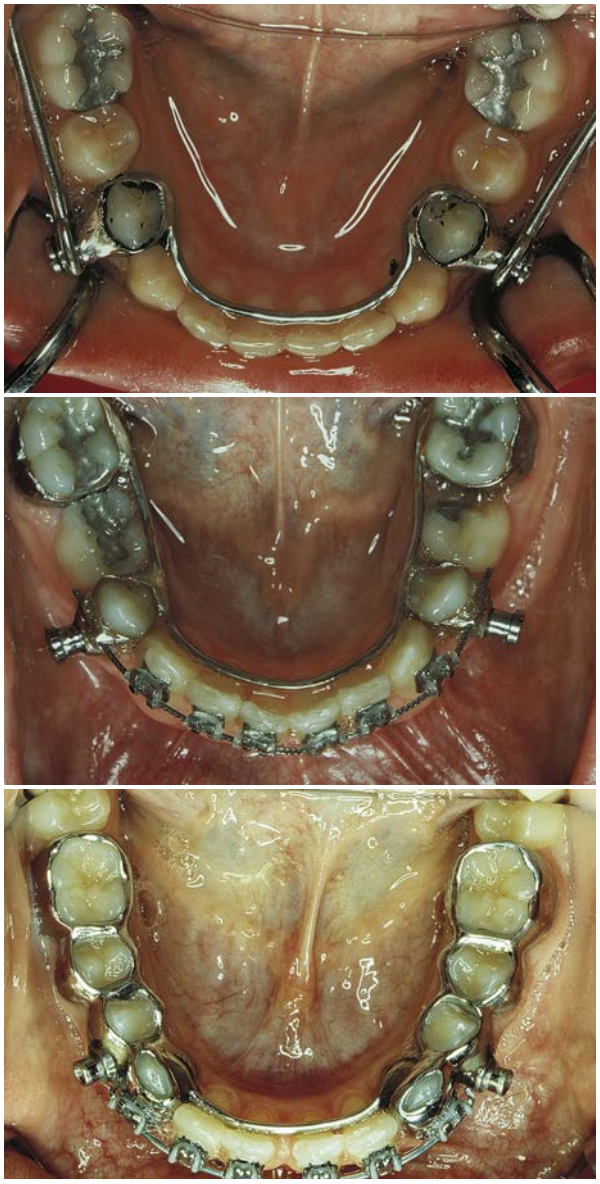
experiences, problems, and solutions.[180] Patients with retrognathic mandibles and TMJ problems responded best. Breakage had been reduced by some design changes of the tube and plunger assembly. However, after this, little appeared in the literature until the concept was resurrected by Hans Pancherz.[107] In 1979, Pancherz's article in the *American Journal of Orthodontics* called attention to the possible stimulation of mandibular growth. Subsequent articles have elaborated on modifications and the short- and long-term effects on jaw relationship, occlusion, and masticatory efficiency.[31,50,114,181] Ruf and Pancherz's long-term research[53,54] and clinical use have prompted others to use this approach or modify the appliance (e.g., Jasper Jumper, Eureka Spring).[182]

The Herbst appliance can be compared with an artificial joint between the maxilla and mandible. The bilateral telescopic mechanism maintains the protracted position of the mandible,



A B

FIGURE 35-48 Anchorage system of Pancherz's version of the Herbst appliance. **A,** Partial anchorage (banded appliance). **B,** Total anchorage (banded appliance). **C,** Total anchorage (cast splint appliance).



C

FIGURE 35-48, cont'd

even during function (see Fig. 35-48). This appliance and its use are fully described by Hans Pancherz in an extensive chapter in *Dentofacial Orthopedics with Functional Appliances*, 2nd ed., by Graber and associates.[10] Each device consists of a tube, a plunger, two pivots, and two locking screws that prevent the telescoping elements from slipping past the pivots (see Figs. 35-46 and 35-47). The pivot for the tube is usually soldered to the maxillary first molar band, and the pivot for the plunger is attached to the mandibular first premolar band. Pancherz found that waiting until the premolars erupted before starting therapy still allows enough time for harnessing adequate remaining growth, particularly in boys.

Generally, the protraction of the mandible for the Herbst appliance is similar to that for the Bionator, with the incisors in an end-to-end relationship, although the incremental advancement approach can be used in large sagittal discrepancies (Fig. 35-49). Du, Hagg, and Rabie[27] prefer incremental advancement and show significant benefit with this method, duplicating the research of Petrovic and Stutzmann on rats.

The length of the tube-plunger assembly determines the amount of advancement. Although conventional orthodontic bands can be used, Pancherz prefers his current approach of casting splints of chromium-cobalt alloy that incorporate molars and premolars and are cemented as units with glass ionomer cement (see Fig. 35-48, *C*). This method saves chair time and causes few clinical problems. For patients with narrow arches, a first-phase expansion can be done with a palatal expansion appliance (one or two expansion screws), or a quad helix appliance can be incorporated into the maxillary appliance (Fig. 35-50). Construction details, per-visit tasks, and the effects of Herbst therapy are thoroughly described in *Dentofacial Orthopedics with Functional Appliances*, 2nd ed., by Graber and associates[10] and in the periodical literature.

The Herbst appliance is a powerful and effective functional system in the treatment of Class II malocclusions.[53] As with other functional appliances, viscoelastic stretch is important. Normalization of occlusion is generally accomplished in 6 to 8 months. Overcorrection of sagittal arch relationships and incomplete cuspal interdigitation, resulting from slower eruption of posterior teeth, are to be expected as settling occurs. Skeletal and dental changes contribute to the treatment result.[54,127,183] Fränkel[93] was critical of the short duration of Herbst appliance wear and recommended prolonged active removable appliance retention and myofunctional exercises during the growth period, as do orthopedic surgeons for growth guidance problems. This practice takes advantage of continued jaw growth. The author of this chapter supports this concept. The downside is that prolonged patient compliance is more difficult.

Sagittal Changes

Figure 35-51 illustrates the restraint of the maxillary arch and the stimulation of mandibular growth.[10] Bone remodeling processes are clearly evident on the lower border of the mandible. Histologic studies confirm these observations.[1] The ultimate condylar position in the fossa is unaffected by Herbst appliance therapy, unless the original problem was a Class II, Division 2 malocclusion. However, and this is again emphasized, modification of the TMJ fossa itself occurs during the growth period. The lower incisors are proclined, and the maxillary molars are posteriorly moved, as with a high-pull headgear.[31] During the first year after treatment, the occlusion settles and the sagittal relationships recover approximately 30% of their previous dimensions. Approximately 90% of the posttreatment occlusal changes occur during the first 6 months and are primarily of dental origin. Lower incisor procumbency rebounds to a significant degree. Lower molars and upper molars tend to move posteriorly and anteriorly, respectively, after treatment.[53]

An unfavorable maxillomandibular growth relationship contributes to early posttreatment changes only to a minor degree. A catch-up in maxillary growth and a minor reduction in mandibular growth increments are apparent in patients treated with the Herbst appliance.[31,54] Again, continued functional retention may enhance stability.

Vertical Changes

In Class II malocclusions with deep bites, overbite may be significantly reduced with Herbst therapy.[31,53] The change primarily results from eruption of lower molars and the intrusion of

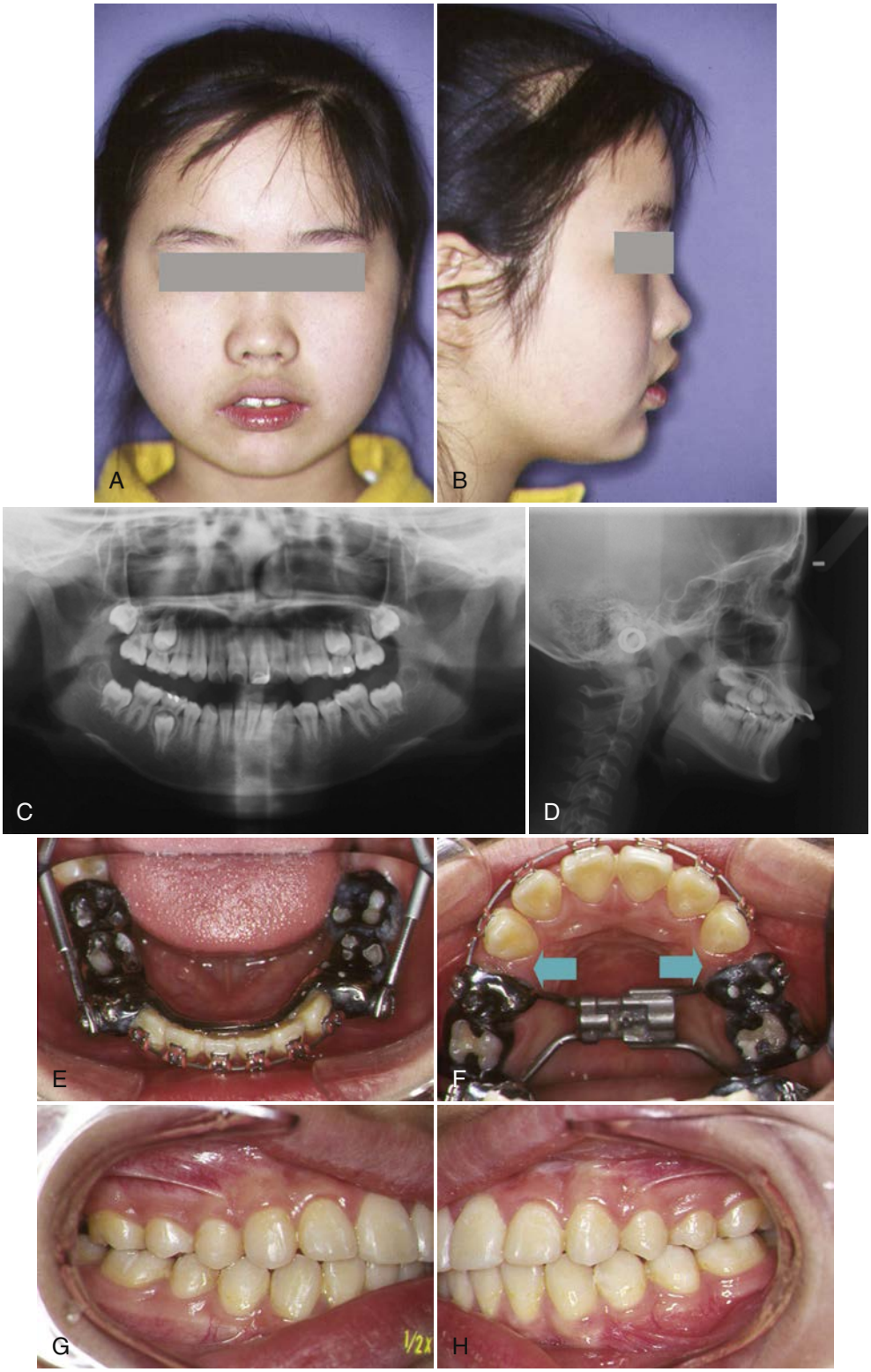
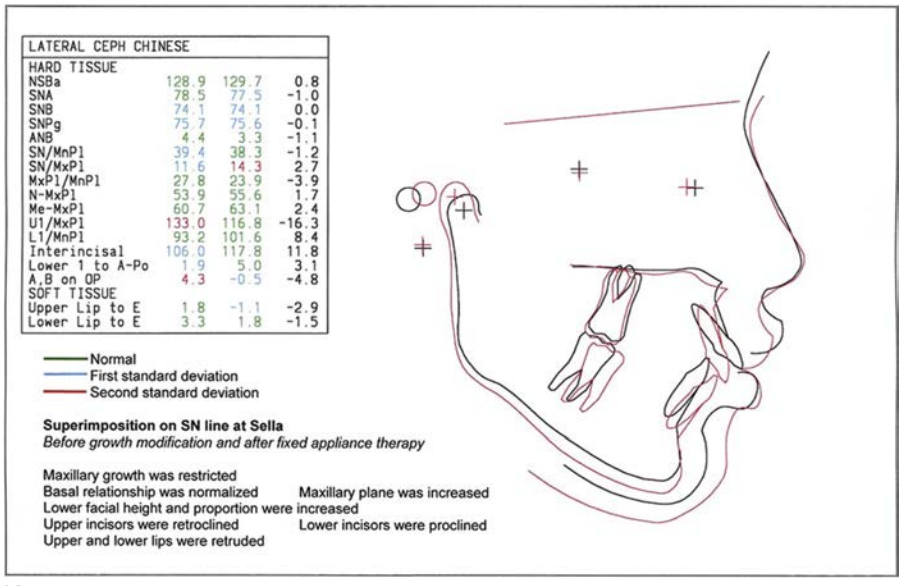


FIGURE 35-49 Case report by Rabie shows the protraction of the mandible for the Herbst appliance. **A,** Pretreatment frontal view. **B,** Pretreatment lateral profile. **C,** Pretreatment panoramic radiograph. **D,** Pretreatment cephalometric radiograph. **E,** Lower occlusal view with the appliance in place. **F,** Upper occlusal view, noting the space gained by the distalization effect of the Herbst appliance (*arrows*). **G, H,** Posttreatment occlusal views. **I,** Posttreatment frontal view. **J,** Posttreatment lateral profile. **K,** Superimposition of cephalometric tracing: before treatment, after Herbst appliance, and after full treatment. (Courtesy ABM Rabie, Hong Kong University, Hong Kong.)



K

FIGURE 35-49, cont'd

FIGURE 35-50 A, Upper cast splint Herbst appliance is used with quad helix lingual arch for expansion. B, A rapid palatal expansion jackscrew may also be used with cast Herbst appliance.

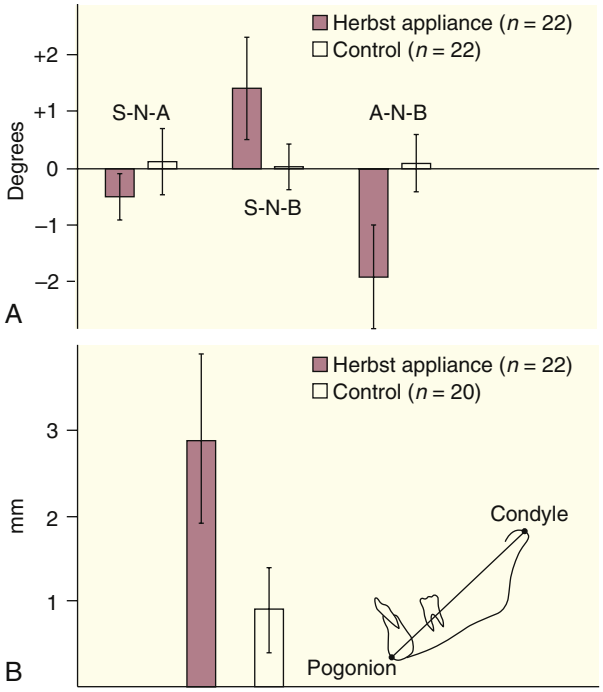


FIGURE 35-51 A, Mean and standard deviation changes in SNA angle, SNB angle, and ANB angle over 6 months. B, Increase in mandibular length over a 6-month period is demonstrated.

the lower incisors. As previously noted, proclination of lower incisors contributes to the seeming intrusion of these teeth. Nevertheless, the appliance has a limited effect on the maxillary and mandibular jaw positions, as expressed by the palatal plane angle and the mandibular plane angle. One must stress again that part of the change is an improvement in the glenoid fossa position and morphologic structure, a change long overlooked until the seminal research of Peter Buschang[24] and Hans Ullrich Paulsen.[49,50]

Long-Term Posttreatment Changes

Several effects have been observed 5 to 10 years after treatment in patients treated with the Herbst appliance. A Class I dental arch relationship is maintained by a stable cuspal interdigitation of the upper and lower arches, whereas relapse tends to occur in cases with unstable occlusal conditions.[54] Teeth locked in a proper relationship are more likely to transfer continuing maxillary growth forces to the mandible. Pancherz emphasized a functionally stable occlusion as possibly more important than posttreatment growth increments. The most common combination of factors leading to varying degrees of relapse are too early treatment, mixed dentition treatment, persistent lip or tongue dysfunction habits, unstable posttreatment occlusion, and insufficient length of appliance wear and retention measures. However, in Pancherz's view, unfavorable posttreatment growth is not a significant factor for occlusal relapse.

Although too early treatment is often associated with relapse, it actually may not be responsible for relapse. More likely, in the mixed dentition, a solid Class I interdigitation is not possible with the deciduous molars. Pancherz believes therefore that the unstable occlusion, not the timing, is responsible.[53] Pancherz notes, however, that the most favorable time to initiate treatment is during the peak pubertal growth period.

Pancherz is totally objective in his intensive and impressive long-term studies. In addition, Pancherz notes from his own and a number of other studies that the basal skeletal relationship is improved but not totally normalized, whereas the occlusal relationship is essentially normalized.[31,46] The implication is that the Herbst appliance is capable of producing sagittal changes that can partially compensate for aberrant skeletal relationships.

A recent study by Ruf and Pancherz[54] assesses the TMJ adaptation through prospective magnetic resonance imaging and roentgenographic techniques. A similar paper was accepted for publication in the *American Journal of Orthodontics and Dentofacial Orthopedics* in 1999. Both papers clearly show significant remodeling with Herbst fixed functional treatment. Strangely enough, comprehensive, long-term studies of the modification of temporomandibular morphologic structure and position in the growing face have yet to be done for fixed appliances, although Paulsen and colleagues[49,50] show convincing evidence of fossa remodeling with sophisticated tomography for short-term Herbst treatment. This long-overlooked phenomenon has been emphasized in this chapter, and references to the seminal work done by Bierbaek and colleagues,[66] Buschang and Santos-Pinto,[24] Decrue and Weislander,[69] Droel and Isaacson,[70] Hotz,[83] Ikai and associates,[32] Kantomaa and Pirttiniemi,[34] Pancherz,[47] and Paulsen[49] clearly indicate the tissue changes, as demonstrated by the Buschang study of changes in the position of the glenoid fossa during growth. It is only logical, then, that the same implications should apply for conventional removable functional appliances. Buschang and Santos-Pinto[24] show the significant potential to affect the changing position of the membranous bone TMJ elements, both fossa and articular eminence. Nature already has pointed the way to this possibility by showing adaptations to deep bite (deep fossa, steep eminence) and Class III malocclusions (shallow fossa, eminently shallow curve).[29]

In another provocative unpublished study that the author directed some years ago at Northwestern University by Dayton Blume and Vernon Boman, postural resting and habitual occlusion condylar and fossa relations were measured in samples of Class I and Class II malocclusions. A significantly higher percentage of condylar retrusion was noted in the Class II sample (Fig. 35-52). Knowing what orthodontists now know about the potentially deleterious effects of condylar retrusion, the logical question may be, "How much of the underdeveloped mandible resulted from interference with metabolism, as dramatically illustrated by Graber,[65] Isberg,[139] Jasper and McNamara,[140] Ward and associates,[80] and others?" (See Fig. 35-20.)

The effects on the facial profile have also been studied by Pancherz and associates.[45,48,114] In patients who were treated for 7 to 8 months and reexamined 5 to 10 years after treatment, a general reduction of hard and soft tissue profile convexity was noted (Fig. 35-53). The upper lip becomes less protrusive, whereas the lower lip remains almost unchanged. However, considerable individual variations were noted (Fig. 35-54).

Overall, the following long-term posttreatment changes can be expected:

- Reduction of the soft tissue profile convexity (excluding the nose), with an increase in mandibular prognathism (see Fig. 35-53)

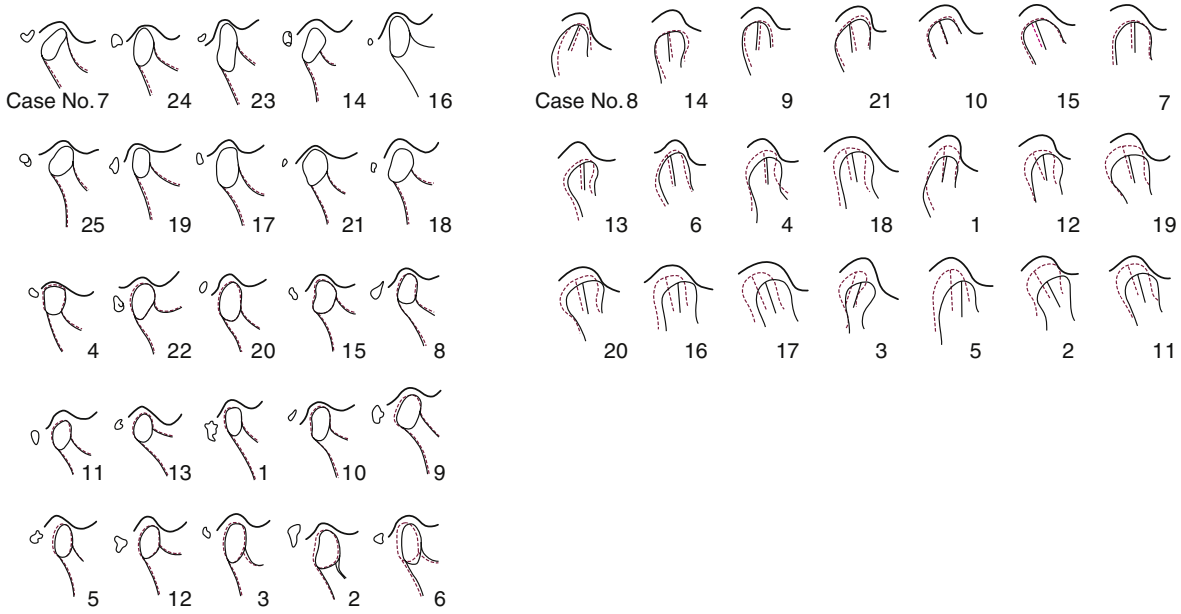


FIGURE 35-52 Unpublished radiographic research by Dayton Blume and Vernon Boman show the path of closure from the postural resting position (*black solid line*) to habitual occlusion (*red dotted line*) in normal patients (*left*) and those with a Class II malocclusion (*right*). This evidence indicates significantly more translatory action (functional retrusion) with an upward and backward path of closure for Class II malocclusions. Normally, condylar movement is essentially rotary from postural rest to maximal intercuspation (*left*). (From Graber TM. Anatomische und physiologische aspekte bei der behandlung von kiefergelenksstörungen [The anatomical and physiological aspects in the treatment of temporomandibular joint disorders]. *Fortschr Kieferorthop* 1991;52:126–132.)

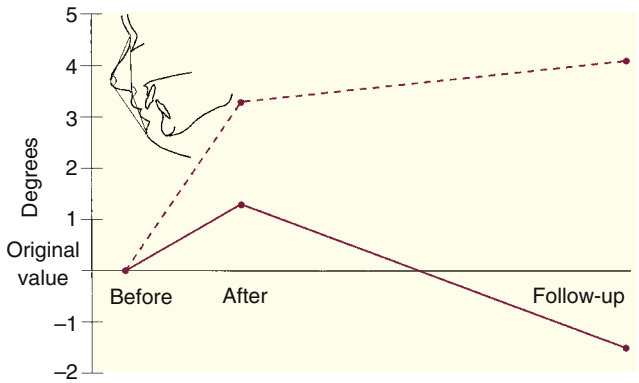


FIGURE 35-53 Mean changes in the soft tissue facial profile angles, excluding the nose (*dotted line*) and including the nose (*solid line*), in 49 subjects treated with the Herbst appliance. Positive values imply profile convexity reduction. Negative values imply profile convexity increase. Measurements were taken before treatment (original value of 0), after 7 months of appliance wear, when the appliance was removed, and at follow-up 5 to 10 years after treatment. (Data from Pancherz H, Fischer S. Amount and direction of temporomandibular joint growth changes in Herbst treatment: a cephalometric long-term investigation. *Angle Orthod* 2003;73:493–501.)

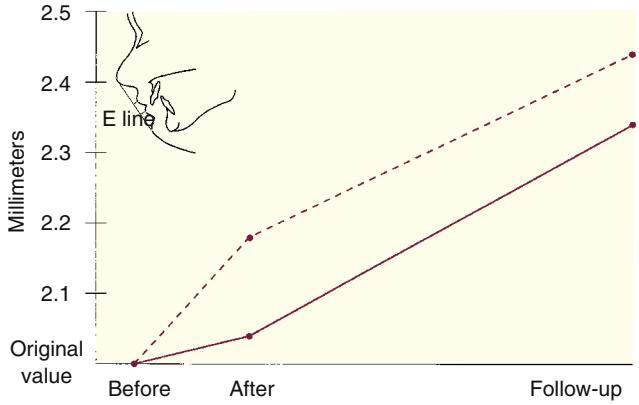


FIGURE 35-54 Mean changes in the position of the upper lip (*dotted line*) and the lower lip (*solid line*) in relation to the aesthetic line in 49 subjects treated with the Herbst appliance. Negative values imply lip retrusion. Measurements were taken before treatment, when the appliance was removed, after 7 months of appliance wear, and 5 to 10 years after treatment. (Data from Pancherz H, Fischer S. Amount and direction of temporomandibular joint growth changes in Herbst treatment: a cephalometric long-term investigation. *Angle Orthod* 2003;73:493–501.)

- Increase in the soft tissue profile convexity (including the nose), largely because of normal nasal growth (see Fig. 35-53)
- Retrusion of the upper and lower lips in relation to the aesthetic line because of normal nose and chin growth (see Fig. 35-54)

Jasper Jumper

The popular Jasper Jumper is a modification of the Herbst appliance just described. The interarch flexible force module of the Jasper Jumper allows the patient greater freedom of movement (Fig. 35-55). As with the Pancherz version of the Herbst appliance, the Jasper Jumper resorts to pushing forces, in contrast to conventional intermaxillary elastics (Figs. 35-56 to 35-59). Repetitive forward posturing is the key element. Metabolism, again, is stimulated to provide the achievable optimum result.

McNamara[41] notes that the treatment response of the Jasper Jumper is almost equally divided between basal and dental effects.[10] On average, a 2-mm increase in mandibular length occurs. Little maxillary skeletal change has been noted. As with all functional appliances, the potential for modification of the fossa in position and morphologic structure exists in the growing individual. One must analyze this using reliable three-dimensional landmarks rather than conventional two-dimensional radiographic, cephalometrically constructed measure points and line and angular reconstructions (cephalometric tracings).[140,184–186]

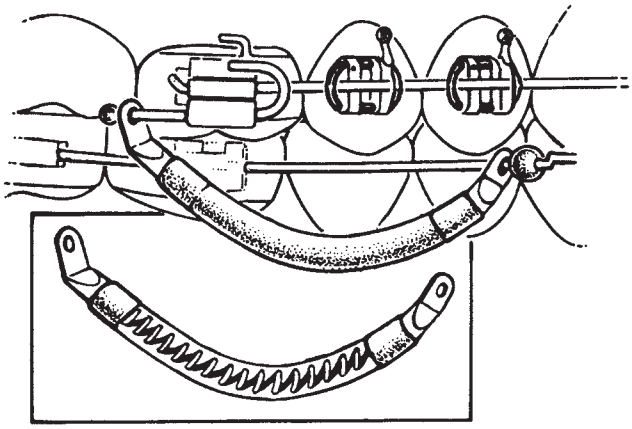


FIGURE 35-55 Jasper Jumper essentials. The distal end of the force module is attached to the maxillary dental arch by means of a ball pin and double molar tubes. The appliance can be activated by anteriorly moving the ball pin. The alignment of the spring within the jumper mechanism is shown in the inset.

According to McNamara,[116] the most pronounced dentoalveolar change with the Jasper Jumper and similar appliances, is a relative posterior movement of the maxillary buccal segments of approximately 2.5 mm. Proclination of the lower incisors is also reported, as with the conventional Herbst appliance.*

As with the Pancherz version of the Herbst appliance, the Jasper Jumper has the advantage of a shorter active treatment time, which is a double-edged sword because less time is available to harness growth increments. Patient compliance needs are minimal. The appliance is less rigid than the classic Herbst device and uses a flexible pushing device, also enlisting the buccinator mechanism (buccal muscle forces) (see Figs. 35-55 to 35-59). In addition to sagittal forces, the Jasper Jumper has a transverse expansion vector acting on the maxillary molars, which must be watched. A transpalatal arch counteracts unwanted buccal malposition of the upper first molars. The use of a lower lingual arch enhances mandibular anchorage (see Fig. 35-59).[10,152,184]

Most of the Pancherz team's observations concerning the conventional Herbst appliance apply to the Jasper Jumper, although some clinicians believe that the magnitude of change is less with the Jasper Jumper. John DeVincenzo has designed several fixed functional appliance modifications that could be used, including his own effective Eureka Spring. DeVincenzo[69,143] attributes the bulk of change to dentoalveolar compensation, with minimal skeletal reaction.

Detailed treatment adjustments are presented in Chapter 17 of *Dentofacial Orthopedics with Functional Appliances*, 2nd ed., by Graber and associates.[10] The reader is strongly advised to read Part 2 of that chapter on asymmetric dentofacial orthopedics. A significant number of Class II malocclusions are not bilateral mirror images. Barry Mollenhauer's discussion applies not only to the Jasper Jumper but also to all fixed functional appliance modifications.[10,63,184,188]

* References 25, 140, 154, 184, 187–189.

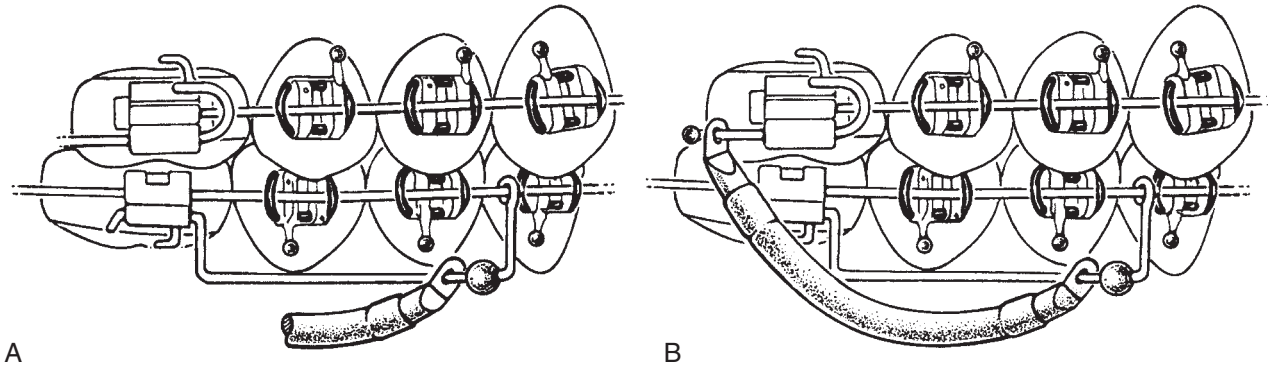


FIGURE 35-56 Outriggers (auxiliary arch wires) are used to anchor the force module. **A,** The rectangular auxiliary wire is anteriorly looped over the main archwire and is posteriorly cinched back through the auxiliary tube. **B,** A ball pin is inserted through the distal hole of the jumper module, anteriorly placed through the face-bow tube on the upper first molar band, and cinched forward to activate the module.

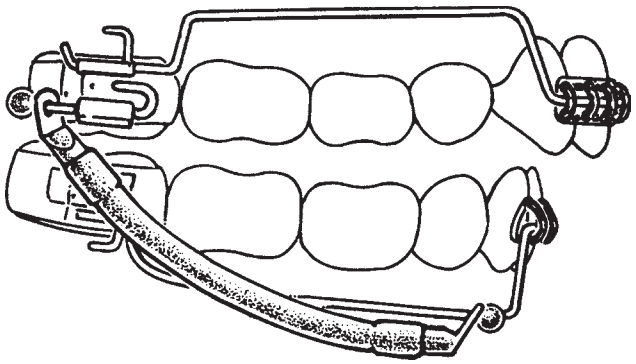


FIGURE 35-57 The force module is used in a patient with mixed dentition. In this instance, a bayonet bend is distally placed to the canine, and a Lexan ball anteriorly acts as a stop for the force module. In this example, the upper and lower rectangular utility arches connect the anterior and posterior teeth.

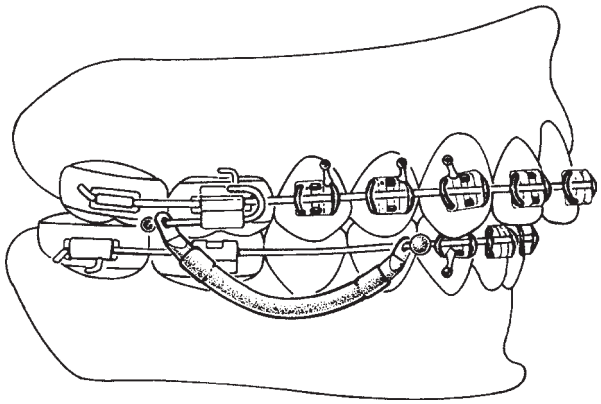
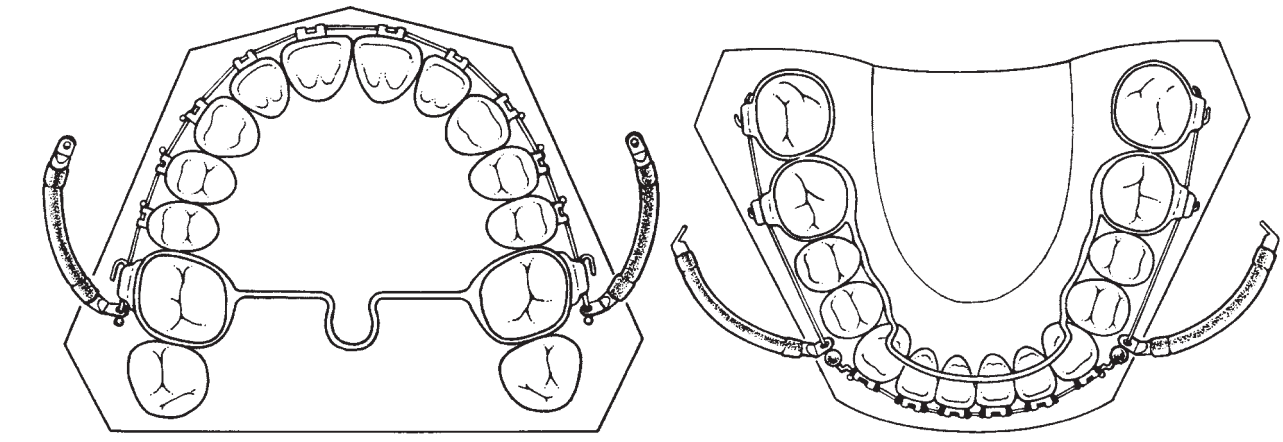


FIGURE 35-58 Maximal anchorage is set up for the force module. The maxillary and mandibular archwires extend to the second molars and are posteriorly cinched back. Tiebacks can also be used. The offset bend in the main archwire (see Fig. 35-59) is obscured by the Lexan ball.



A B

FIGURE 35-59 A, The transpalatal arch, combined with fixed appliances, is used to enhance maxillary anchorage. B, A lower lingual arch, combined with fixed appliances, enhances mandibular anchorage.

SUMMARY

The author of this chapter hopes that this overview of the biologic approach—enlisting the patient's own muscles, function, growth pattern, optimal metabolic stimulation, and compliance—will excite the reader enough to stimulate, the reading of the accompanying list of references. Functional appliances do not replace fixed attachments. Indeed, the combined use of brackets, bands, and extraoral force has the potential for the best possible and most stable long-term results. Treatment timing and length of treatment are important considerations for growth guidance as orthopedic surgeons show. The ultimate goal of all orthodontists is to be applied biologists.

https://coursewareobjects.elsevier.com/objects/elr/ExpertConsult/Graber/orthodontics6e/extras/

REFERENCES

1. Graber TM, Chung DB, Aoba TJ. Dentofacial orthopedics versus orthodontics. *J Am Dent Assoc*. 1967;75:1145–1166.
2. Wolff J. *Gesetz der Transformation der Knochen*. Berlin: Hirschwold; 1892.
3. Roux W. *Gessamelte Abhandlungen über Entwicklungsmechanik der Organismen*. Leipzig: W. Engelmann; 1895.
4. Graber TM. The unique nature of temporomandibular joint metabolism: the clinical implications. In: Rabie AM, Urist MR, eds. *Bone Formation and Repair*. Amsterdam: Elsevier; 1997:143–157.
5. Pilon JJGM. *Orthodontic Forces and Tooth Movement,* Doctoral dissertation. Nijmegen, Netherlands: University of Nijmegen; 1996.
6. Sfondrini G, Reggiani C, Gandini P, et al. Adaptations of masticatory muscles to a hyperpropulsive appliance in the rat. *Am J Orthod Dentofacial Orthop*. 1996;110:612–617.
7. Robin P. Observation sur un nouvel appareil de redressement. *Rev Stomatol*. 1902;9:423–432.
8. Kingsley NW. *Oral Deformities*. New York: Appleton & Son; 1880.
9. Case C. Open bite malocclusion. *Dent Rev*. 1984;6:124.
10. Graber TM, Rakosi T, Petrovic AG, eds. *Dentofacial Orthopedics with Functional Appliances*. 2nd ed. St. Louis: Mosby; 1997.
11. Lischer BE. *Principles and Methods of Orthodontics*. Philadelphia: Lea & Febiger; 1912.
12. Angle EH. *The Treatment of Malocclusion of the Teeth*. 7th ed. Philadelphia: S. S. White Manufacturing; 1907.
13. Andresen V. Beitrag zur retention. *Z Zahnartzl Orthop*. 1910;3:121.
14. Andresen V. Über das sogenannte "Norwegische system der funktions-kieferorthopädie." *Dtsch Zahnartzl Wochenschr*. 1936;39:235.
15. Andresen V, Häupl K. *Funktionskieferorthopädie: die Grundlagen des "Norwegischen Systems."* 2nd ed. Leipzig, Germany: Hermann Meusser; 1936.
16. Andresen V, Häupl K, Petrik L. *Funktionskieferorthopädie*. 6th ed. Munich, Germany: Johann Ambrosium Barth Publishing; 1957.
17. Ahlgren J, Bendéus M. Changes in length and torque of masticatory muscles produced by the activator appliance. A cephalometric study. *Swed Dent J Suppl*. 1982;15:27–35.
18. Baccetti T, Franchi L, McNamara Jr JA, Tollaro I. Early dentofacial features of Class II malocclusion: a longitudinal study from the deciduous through the mixed dentition. *Am J Orthod Dentofacial Orthop*. 1997;111:502–509.
19. Ballard CF. A consideration of the physiological background of mandibular posture and movement. *Dent Pract*. 1955;6:80–89.
20. Barton S, Cook PA. Predicting functional appliance treatment outcome in Class II malocclusions—a review. *Am J Orthod Dentofacial Orthop*. 1997;112:282–286.
21. Bimler HP. The Bimler appliance. In: Graber TM, Neuman B, eds. *Removable Orthodontic Appliances*. 2nd ed. Philadelphia: WB Saunders; 1984:410–519.
22. Björk A. The principle of the Andresen method of orthodontic treatment: a discussion based on cephalometric x-ray analysis of treated cases. *Am J Orthod*. 1951;37:437–458.
23. Brieden CM, Pangrazio-Kulbersh V, Kulbersh R. Maxillary skeletal and dental changes with Fränkel appliance therapy—an implant study. *Angle Orthod*. 1984;54:226–232.
24. Buschang PH, Santos-Pinto A. Condylar growth and glenoid fossa displacement during childhood and adolescence. *Am J Orthod Dentofacial Orthop*. 1998;113:437–442.
25. Cash RG. Adult nonextraction treatment with a Jasper Jumper. *J Clin Orthod*. 1991;25:43–47.
26. DeVincenzo JP, Winn MW. Orthopedic and orthodontic effects from the use of a functional appliance with different amounts of protrusive activation. *Am J Orthod Dentofacial Orthop*. 1989;96:181–190.
27. Du X, Hägg U, Rabie B. Effects of headgear Herbst and mandibular step-by-step advancement versus conventional Herbst appliance and maximum jumping of the mandible. *Eur J Orthod*. 2002;24:167–174.
28. Fricker JP, ed. *Orthodontics and Dentofacial Orthopaedics*. Canberra, Australia: Tidbinbilla Pty. Ltd.; 1998.
29. Graber TM, ed. *Physiological Principles of Functional Appliances*. St. Louis: Mosby; 1985.
30. Graber TM. The neuromuscular system and its significance for clinical orthodontics. *Eur J Orthod*. 1998;20:470.
31. Hansen K, Pancherz H. Long-term effects of Herbst treatment in relation to normal growth and development: a cephalometric study. *Eur J Orthod*. 1992;14:285–295.
32. Ikai A, Sugisaki M, Young-Sung K, Tanabe H. Morphologic study of the mandibular fossa and the eminence of the temporomandibular joint in relation to facial structures. *Am J Orthod Dentofacial Orthop*. 1997;112:634–638.
33. Kalra V, Burstone CJ, Nanda R. Effects of a fixed magnetic appliance on the dentofacial complex. *Am J Orthod Dentofacial Orthop*. 1989;95:467–478.
34. Kantomaa T, Pirttiniemi P. Changes in proteoglycan and collagen content in the mandibular condylar cartilage of the rabbit caused by an altered relationship between the condyle and glenoid fossa. *Eur J Orthod*. 1998;20:435–441.
35. Komposch G, Hockenjos C. Die Reaktionsfahrigkeit des temporomandibularen Knörpels. *Fortschr Kieferorthop*. 1977;28:121–132.
36. Lange DW, Kalra V, Broadbent Jr BH, et al. Changes in soft tissue profile following treatment with the Bionator. *Angle Orthod*. 1995;65:423–430.
37. Lund DI, Sandler PJ. The effects of Twin Blocks: a prospective controlled study. *Am J Orthod Dentofacial Orthop*. 1998;113:104–110.
38. Mamandras AH, Allen LP. Mandibular response to orthodontic treatment with the Bionator appliance. *Am J Orthod Dentofacial Orthop*. 1990;97:113–120.
39. McDougall PD, McNamara JA. Arch width development in Class II patients with the Fränkel appliance. *Am J Orthod*. 1982;82:10–22.
40. McNamara Jr JA. Functional adaptations in the temporomandibular joint. *Dent Clin North Am*. 1975;19:457–471.
41. McNamara Jr JA. *Temporomandibular Joint Adaptation to Functional Protrusion.* Paper presented at the one hundred third annual session of the American Association of Orthodontists. Honolulu: Hawaii; May 6, 2003.
42. McNamara Jr JA, Bookstein FL, Shaughnessy TG. Skeletal and dental changes following functional regulator therapy on Class II patients. *Am J Orthod*. 1985;88:91–110.
43. McNamara Jr JA, Hinton RJ, Hoffman DL. Histologic analysis of temporomandibular joint adaptations to protrusive function in young adult rhesus monkeys. *Am J Orthod*. 1982;82:288–298.
44. Mills CM, McCulloch KJ. Treatment effects of the twin block appliance. *Am J Orthod Dentofacial Orthop*. 1998;114:15–24.
45. Obijou C, Pancherz H. Herbst appliance treatment of Class II, Division 2 malocclusions. *Am J Orthod Dentofacial Orthop*. 1997;112:287–291.
46. Pancherz H. The effect of continuous bite jumping on the dentofacial complex: a follow-up study after Herbst appliance treatment of Class II malocclusion. *Eur J Orthod*. 1981;3:49–60.
47. Pancherz H. The effects, limitations, and long-term dentofacial adaptations to treatment with the Herbst appliance. *Semin Orthod*. 1997;3:232–243.

48. Pancherz H, Fischer S. Amount and direction of temporomandibular joint growth changes in Herbst treatment: a cephalometric long-term investigation. *Angle Orthod*. 2003;73:493–501.
49. Paulsen HU. Morphological changes of the TMJ condyles of 100 patients treated with the Herbst appliance in the period of puberty to adulthood: a long-term radiographic study. *Eur J Orthod*. 1997;19:657–668.
50. Paulsen HU, Rabøl A, Sørensen SS. Bone scintigraphy of the human temporomandibular joints during Herbst treatment. *Eur J Orthod*. 1998;20:369–374.
51. Pirttiniemi P, Kantornaa T, Poikela A, Pietila K. Electrical masseter muscle stimulation alters condylar shape. *Eur J Orthod*. 1998;20:478–479.
52. Rakosi T. The activator. In: Graber TM, Rakosi T, Petrovic A, eds. *Dentofacial Orthopedics with Functional Appliances*. 2nd ed. St. Louis: Mosby; 1997:19–67.
53. Ruf S, Pancherz H. The mechanism of Class II correction during Herbst therapy in relation to the vertical jaw base relationship: a cephalometric roentgenographic study. *Angle Orthod*. 1997;67:271–276.
54. Ruf S, Pancherz H. Temporomandibular joint growth adaptation in Herbst treatment: a prospective magnetic resonance imaging and cephalometric roentgenographic study. *Eur J Orthod*. 1998;20:375–388.
55. Sander FG. The effects of appliances and Class II elastics on masticatory patterns. In: McNamara Jr JA, Ribbens KA, Howe RP, eds. *Clinical Alteration of the Growing Face. Monograph 14, Craniofacial Growth Series. Center for Human Growth and Development*. Ann Arbor, MI: University of Michigan; 1983:155–178.
56. Sectakof PA. *The Effects of Functional Appliances on Functional Activities of Jaw Muscles in Macaca Fascicularis.* Master's thesis. Toronto: University of Toronto; 1992.
57. Stromeyer EL, Caruso JM, DeVincenzo JP. A cephalometric study of the Class II correction effects of the Eureka Spring. *Angle Orthod*. 2002;72(3):203–210.
58. Tallgren A, Christiansen RL, Ash Jr M, Miller RL. Effects of a myofunctional appliance on orofacial muscle activity and structures. *Angle Orthod*. 1998;68:249–258.
59. Thilander B, Filipsson R. Muscle activity related to activator and intermaxillary traction in angle Class II Division 1 malocclusions. An electromyographic study of the temporal, masseter, and suprahyoid muscles. *Acta Odontol Scand*. 1966;24:241–257.
60. Vardimon AD, Fricke J, Spack N, et al. Principles and cephalometric assessment of functional magnetic correction. *Eur J Orthod*. 1998;20:483–484.
61. Watted N, Witt E, Koch R, Kenn W. A nuclear magnetic resonance study of the temporomandibular joint changes following functional orthopaedic treatment using the Würzburg approach. *Eur J Orthod*. 1998;20:484–485.
62. Wieslander L. Long-term effect of treatment with the headgear-Herbst appliance in the early mixed dentition. Stability or relapse? *Am J Orthod Dentofacial Orthop*. 1993;104:319–329.
63. Yamin-Lacouture C, Woodside DG, Sectakof PA, Sessie BJ. The action of three types of functional appliances on the activity of the masticatory muscles. *Am J Orthod Dentofacial Orthop*. 1997;112:560–572.
64. Graber TM. The clinical implications of the unique metabolic processes in the human temporomandibular joint. In: Sachdeva RC, Hans-Peter Bantleon, Larry White, Jeff Johnson, et al., ed. *Orthodontics for the Next Millennium*. Ormco; 1997.
65. Graber TM. *Nobel Prize Research and its Impact on Growth Guidance and TMJ Metabolism.* Paper presented at the one hundred third annual session of the American Association of Orthodontists; May 6, 2003. Honolulu, Hawaii.
66. Birkebaek L, Melsen B, Terp S. A laminagraphic study of the alterations in the temporo-mandibular joint following activator treatment. *Eur J Orthod*. 1984;6:257–266.
67. Braun S, Kittelson R, Kim K. The G-axis: a growth vector for the mandible. *Angle Orthod*. 2004;74:328–331.
68. Chen JY, Will LA, Neiderman R. Analysis of efficacy of functional appliances on mandibular growth. *Am J Orthod Dentofacial Orthop*. 2002;122:470–476.
69. Decrue A, Wieslander L. Fossa articularis changes using Herbst appliance after mandibular advancement. [Article in German]. *Zahnarztl Prax*. 1990;41:360–362, 364–365.
70. Droel R, Isaacson RJ. Some relationships between glenoid fossa position and various skeletal discrepancies. *Am J Orthod*. 1972;61:64–78.
71. el-Bialy T, el-Moneim Zaki A, Evans CA. Effects of ultrasound on rabbit mandibular incisor formation and eruption after mandibular osteodistraction. *Am J Orthod Dentofacial Orthop*. 2003;124:427–434.
72. Garattini G, Levrini L, Crozzoli P, Levrini A. Skeletal and dental modifications produced by the Bionator III appliance. *Am J Orthod Dentofacial Orthop*. 1998;114:40–44.
73. Paulsen HU. *Personal communication*; Feb 2004.
74. Popowich K, Nebbe B, Major PW. Effect of Herbst treatment on temporomandibular joint morphology: a systematic literature review. *Am J Orthod Dentofacial Orthop*. 2003;123:388–394.
75. Rabie AB, She TT, Hägg U. Functional appliance therapy accelerates and enhances condylar growth. *Am J Orthod Dentofacial Orthop*. 2003;123:40–48.
76. Vardimon AD, Köklü S, Iseri H, et al. An assessment of skeletal and dental responses to the functional magnetic system (FMS). *Am J Orthod Dentofacial Orthop*. 2001;120:416–426.
77. Katsavrias EG, Voudouris JC. The treatment effect of mandibular protrusive appliances on the glenoid fossa for Class II correction. *Angle Orthod*. 2004;74:79–85.
78. Voudouris JC, Woodside DG, Altuna G, et al. Condyle-fossa modifications and muscle interactions during Herbst treatment, part 1. New technological methods. *Am J Orthod Dentofacial Orthop*. 2003;123:604–613.
79. Voudouris JC, Woodside DG, Altuna G, et al. Condyle-fossa modification and muscle interactions during Herbst treatment, part 2. Results and conclusions. *Am J Orthod Dentofacial Orthop*. 2003;124:13–29.
80. Ward DM, Behrents RG, Goldberg JS. Temporomandibular synovial fluid pressure response to altered mandibular positions. *Am J Orthod Dentofacial Orthop*. 1990;98:22–28.
81. Woodside DG, Metaxas A, Altuna G. The influence of functional appliance therapy on glenoid fossa remodeling. *Am J Orthod Dentofacial Orthop*. 1987;92:181–198.
82. Xiong H, Hägg U, Tang GH, et al. The effect of continuous bite jumping in adult rats: a morphological study. *Angle Orthod*. 2004;74:86–92.
83. Hotz R. *Orthodontics in Daily Practice*. Bern, Switzerland: Hans Huber Publishers; 1974.
84. Igarashi K, Miyoshi K, Shinoda H, et al. Diurnal variation in tooth movement in response to orthodontic force in rats. *Am J Orthod Dentofacial Orthop*. 1998;114:8–14.
85. Korkhaus G. Die Auswertung des Fernröntgenbildes in der Kieferorthopädie. *Dtsch Zahn Mund Kieferheilkd*. 1936;3:715.
86. Petrovic AG, Oudet CL, Stutzmann JJ. Temporal organization of rat and human skeletal cells: circadian frequency and organizement of cell generation time. In: Edmunds L, ed. *Cell Cycle Clocks*. New York: Marcel Dekker, Inc.; 1984:325–349.
87. Selmer-Olsen R. En kritisk bertrakning over "Der Norsk System." *Nor Tannlaegeforen Tid*. 1937;47:85–91.
88. Woodside DG. Do functional appliances have an orthopedic effect? *Am J Orthod Dentofacial Orthop*. 1998;113:11–14.
89. Ryan MJ, Schneider BJ, BeGole EA, Muhi ZF. Opening rotations of the mandible during and after treatment. *Am J Orthod Dentofacial Orthop*. 1998;114:142–149.
90. Johnston LE. Personal communication on unloading the condyle. *Sept*. 1998.
91. Slagsvold O, Kolstad I. Class II Division 1 Malocclusion Treated with Activators: A Study of Posttreatment Stability. Unpublished. 1978.
92. Grude R. Myo-functional therapy: a review of various cases some years after their treatment by the Norwegian system had been completed. *Nor Tanlaegeforen Tid*. 1952;62:1–7.
93. Fränkel R. *Technik und Handhabung der Funktionsregler*. Berlin: VEB Verlag Volk & Gesundheit; 1973.
94. Harvold EP. *The Activator in Interceptive Orthodontics*. St. Louis: Mosby; 1974.
95. Herren P. The activator's mode of action. *Am J Orthod*. 1959;45(7):512–527.
96. Schmuth GPF. Consideration of Functional Aspects in Dentofacial Orthopedics and Orthodontics. Sheldon Friel Memorial Lecture. Presented at the 94th European Orthodontic Congress in Mainz, Germany, June 1998. *Am J Orthod Dentofacial Orthop*. 1999;116(4):373–381.
97. Huggare JA, Laine-Alava MT. Nasorespiratory function and head posture. *Am J Orthod Dentofacial Orthop*. 1997;112:507–511.

98. Ono T, Ishiwata Y, Kuroda T. Inhibition of masseteric electromyographic activity during oral respiration. *Am J Orthod Dentofacial Orthop*. 1998;113:518–525.
99. Ozbek MM, Memikoglu TU, Gögen H, et al. Oropharyngeal airway dimensions and functional-orthopedic treatment in skeletal Class II cases. *Angle Orthod*. 1998;68:327–336.
100. Altenburger E, Ingervall B. The initial effects of treatment of Class II, Division 1 malocclusions with the van Beek activator compared with the effects of the Herren activator and an activator-headgear combination. *Eur J Orthod*. 1998;20:389–397.
101. Clark GT, Arand D, Chung E, Tong D. Effect of anterior mandibular positioning on obstructive sleep apnea. *Am Rev Respir Dis*. 1993;147: 624–629.
102. Scholle HC, Schurmann NP, Kopp S. Selective masseter and temporal muscle activation depending on the morphofunctional situation in the orofacial region. *Eur J Orthod*. 1998;20:482.
103. Tourne L, Schweiger J. Immediate postural response to total nasal obstruction. *Am J Orthod Dentofacial Orthop*. 1996;110:606–611.
104. Vig KD. Nasal obstruction and facial growth: the strength of evidence for clinical assumptions. *Am J Orthod Dentofacial Orthop*. 1998;113:603–611.
105. Yamada T, Tanne K, Miyamoto K, Yamauchi K. Influence of nasal respiratory obstruction on craniofacial growth in young *Macaca fuscata* monkeys. *Am J Orthod Dentofacial Orthop*. 1997;111:38–43.
106. Cevidanes L, Franco AA, Scanavini A, et al. Clinical outcomes of Fränkel appliance therapy assessed with a counterpart analysis. *Am J Orthod Dentofacial Orthop*. 2003;123:379–387.
107. Pancherz H. Treatment of class II malocclusions by jumping the bite with the Herbst appliance. A cephalometric investigation. *Am J Orthod*. 1979;76:423–442.
108. Pancherz H. The modern Herbst appliance. In: Graber TM, Rakosi T, Petrovic A, eds. *Dentofacial Orthopedics with Functional Appliances*. 2nd ed. St. Louis: Mosby; 1997:336–366.
109. Bishara S. Mandibular changes in persons with untreated and treated Class II, Division 1 malocclusion. *Am J Orthod Dentofacial Orthop*. 1998;113:661–673.
110. Bishara S, Zaher AR, Cummins DM, Jakobsen JR. Effects of orthodontic treatment on the growth of individuals with Class II, Division 1 malocclusion. *Angle Orthod*. 1994;64:221–230.
111. Björk A, Palling M. Adolescent age changes in sagittal jaw relations, alveolar prognathy, and incisal inclination. *Acta Odontol Scand*. 1955;12:201–232.
112. Gianelly A, Brosnan P, Martignoni M, Bernstein L. Mandibular growth, condyle position, and Fränkel appliance therapy. *Angle Orthod*. 1983;53:131–142.
113. Hamilton D. Early treatment: the emancipation of dentofacial orthopedics. In: Graber TM, Rakosi T, Petrovic A, eds. *Dentofacial Orthopedics with Functional Appliances*. 2nd ed. St. Louis: Mosby; 1997.
114. Konik M, Pancherz H, Hansen K. The mechanism of Class II correction in late Herbst treatment. *Am J Orthod Dentofacial Orthop*. 1997;112:87–91.
115. Kusumoto K, Sato A, Mitani H. The evaluation of the orthopedic appliances by using standard growth curves of maxilla and mandible. *J Jpn Orthod Soc*. 1996;55(4):311–321.
116. Lundström A. *Malocclusion of the Teeth as Regarded as a Problem with the Apical Base*. Doctoral dissertation. Stockholm: Karolinska University; 1923.
117. Melanson E, Van Dyken C. *Studies in Condylar Growth*. Master's thesis. Ann Arbor, MI: University of Michigan; 1972.
118. Graber TM. The anatomical and physiological aspects in the treatment of temporomandibular joint disorders. *Fortschr Kieferorthop*. 1991;52: 126–132.
119. Haralabakis NB, Halazonetics DJ, Sifakakis IB. Activator versus cervical headgear: superimposed cephalometric comparison. *Am J Orthod Dentofacial Orthop*. 2003;123:296–305.
120. Graber TM. *The Effect of Buccal Shields on the Maxillary Dental Arch Width in the Squirrel Monkey (Saimiri Sciureus)*. Paper presented at the eighty-third annual session of the American Association of Orthodontists. Boston: May 4–10, 1983.
121. Graber TM. *Experimental and Clinical Studies of the Effect of the Fränkel FR Appliance in Primates and Humans*. Annual meeting of the American Association of Orthodontists. Boston: May 4–10, 1983.
122. Graber TM. Combined extraoral and functional appliances. In: Graber TM, Rakosi T, Petrovic A, eds. *Dentofacial Orthopedics with Functional Appliances*. 2nd ed. St. Louis: Mosby; 1997.
123. Creekmore TD, Eklund MK. The possibility of skeletal anchorage. *J Clin Orthod*. 1983;17:266–269.
124. Ricketts RM. *Orthopedics in the Eyes of the Clinician. Symposium on Clinical and Bioengineering Aspects of Dentofacial Orthopedics*. Farmington, CT: University of Connecticut; 1981.
125. Enlow DH. Growth and the problem of local control mechanism. *Am J Anat*. 1973;136(4):403–405.
126. Clark WJ. *Twin Block Functional Therapy: Applications in Dentofacial Orthopaedics*. 2nd ed. London: Mosby; 2002.
127. Coelho Filho CM. The mandibular protraction appliance no 3. *J Clin Orthod*. 1998;32:379–384.
128. Dischinger T. Edgewise Herbst appliance. *J Clin Orthod*. 1995;29:738–742.
129. Ghafari J, Shofer FS, Jacobsson-Hunt U, et al. Headgear versus function regulator in early treatment of Class II Division 1 malocclusion: a randomized clinical trial. *Am J Orthod Dentofacial Orthop*. 1998;113:51–61.
130. Eschler J. Die Kieferdehnung mit funktionskieferorthopädischen Apparaten: der Funktionator. *Zahnarztl Welt*. 1962;63:203.
131. Schwarz AM. The effects of the activator. *Fortschr Kieferorthop*. 1952;13:117–138.
132. Baume LJ, Derichsweiler H. Is the condylar growth center responsive to orthodontic therapy? An experimental study in *Macaca mulatta*. *Oral Surg Oral Med Oral Pathol*. 1961;14:347–362.
133. Charlier JP, Petrovic A, Hermann-Stutzmann J. Effects of mandibular hyperpropulsion on the prechondroblastic zone of young rat condyle. *Am J Orthod*. 1969;55:71–74.
134. McNamara Jr JA, Huge SA. The functional regulator (FR-3) of Fränkel. *Am J Orthod*. 1985;88:409–424.
135. Graber TM, Neumann B, eds. *Removable Orthodontic Appliances*. 2nd ed. Philadelphia: W.B. Saunders; 1984.
136. Tulloch JF, Phillips C, Koch G, Proffit WR. The effect of early intervention on skeletal pattern in Class II malocclusion: a randomized clinical trial. *Am J Orthod Dentofacial Orthop*. 1997;111:391–400.
137. Burkhardt DR, McNamara Jr JA, Baccetti T. Maxillary molar distalization or mandibular advancement: a cephalometric comparison of comprehensive orthodontic treatment including the pendulum and the Herbst appliances. *Am J Orthod Dentofacial Orthop*. 2003;123:108–116.
138. Enlow DH, DiGangi D, McNamara Jr JA, Mina M. An evaluation of the morphogenic and anatomic effects of the functional regulator utilizing the counterpart analysis. *Eur J Orthod*. 1988;10:192–202.
139. Isberg A, Isacsson G. Tissue reactions associated with internal derangement of the temporomandibular joint. *Acta Odontol Scand*. 1986;44:160–164.
140. Jasper JJ, McNamara Jr JA. The correction of interarch malocclusions using a fixed force module. *Am J Orthod Dentofacial Orthop*. 1995;108: 641–650.
141. Balters W. *Eine Einführung in die Bionatorheilmethode: ausgewählte Schriften und Vorträge*. Heidelberg, Germany: C Hermann; 1972.
142. Moss ML. The functional matrix hypothesis revisited.1. The role of mechanotransduction. *Am J Orthod Dentofacial Orthop*. 1997;112:8–11.
143. DeVincenzo J. The Eureka Spring: a new interarch force delivery system. *J Clin Orthod*. 1997;31:454–467.
144. Eirew HL, McDowell F, Phillips JG. The Fräenkel appliance—avoidance of lower incisor proclination. *Br J Orthod*. 1981;8:189–191.
145. Fleischer E, Fleischer A. Bionator modification: the Bio-M-S therapy. In: Graber TM, Neumann B, eds. *Removable Orthodontic Appliances*. 2nd ed. Philadelphia: W.B. Saunders; 1984:387–409.
146. Miyamoto K, Yamada K, Ishizuka Y, et al. Masseter muscle activity during the whole day in young adults. *Am J Orthod Dentofacial Orthop*. 1996;110:394–398.
147. Tulloch JF, Phillips C, Proffit WR. Benefit of early Class II treatment: progress report of a two-phase randomized clinical trial. *Am J Orthod Dentofacial Orthop*. 1998;113:62–72.
148. Rakosi T, Jonas I, Graber TM. *Orthodontic Diagnosis (Color Atlas of Dental Medicine*. Berlin: Thieme; 1993.
149. Rabie AB, Wong L, Hägg U. Correlation of replicating cells and osteogenesis in the glenoid fossa during stepwise advancement. *Am J Orthod Dentofacial Orthop*. 2003;123:521–526.

150. Rabie AB, Wong L, Tsai M. Replicating mesenchymal cells in the condyle and the glenoid fossa during mandibular forward positioning. *Am J Orthod Dentofacial Orthop*. 2003;123:49–57.
151. McNamara JA. Fabrication of the acrylic splint Herbst appliance. *Am J Orthod Dentofacial Orthop*. 1988;94:10–18.
152. Moffett BC. A research perspective on craniofacial morphogenesis. *Acta Morphol Neerl Scand*. 1972;10:91–101.
153. Graber TM. The Fränkel function regulator. In: Graber TM, Rakosi T, Petrovic A, eds. *Dentofacial Orthopedics with Functional Appliances*. 2nd ed. St. Louis: Mosby; 1997:219–274.
154. Cope JB, Buschang Ph, Cope DD, et al. Quantitative evaluation of craniofacial changes with Jasper Jumper therapy. *Angle Orthod*. 1994;64:113–122.
155. Creekmore TD, Radney LJ. Fränkel appliance therapy: orthopedic or orthodontic? *Am J Orthod*. 1983;83:89–108.
156. Schwarz AM, Gratzinger M. *Removable Orthodontic Appliances*. Philadelphia: W.B. Saunders; 1966.
157. Clark WJ. The twin-block technique. In: Graber TM, Rakosi T, Petrovic AG, eds. *Dentofacial Orthopedics with Functional Appliances*. 2nd ed. St. Louis: Mosby; 1997:268–298.
158. Bondevik O. Treatment needs following activator-headgear therapy. *Angle Orthod*. 1995;65:417–422.
159. Vardimon K, Köklü A, Iseri H, Shpack N, Fricke J, Mete L. An assessment of skeletal and dental responses to the functional magnetic system (FMS). *Am J Orthod Dentofacial Orthop*. 2001;120(4):416–426.
160. Moss JP. *Personal communication*; Oct. 21, 1999.
161. O'Brien K, Wright J, Conboy F, et al. Effectiveness of early orthodontic treatment with the twin-block appliance: a multicenter, randomized, controlled trial. Part I. Dental and skeletal effects. *Am J Orthod Dentofacial Orthop*. 2003;124:234–243.
162. O'Brien K, Wright J, Conboy F, et al. Effectiveness of treatment of Class II malocclusion with the Herbst or twin-block appliances: a randomized, controlled trial. *Am J Orthod Dentofacial Orthop*. 2003;124:128–137.
163. Blechman AM. Magnetic force systems in orthodontics. Clinical results of a pilot study. *Am J Orthod*. 1985;87:201–210.
164. Blechman AM, Steger ER. A possible mechanism of action of repelling, molar distalizing magnets. Part I. *Am J Orthod Dentofacial Orthop*. 1995;108:428–431.
165. Bondemark L, Kurol J. Distalization of maxillary first and second molars simultaneously with repelling magnets. *Eur J Orthod*. 1992;14:264–272.
166. Darendeliler MA, Sinclair PM, Kusy RP. The effects of samarium-cobalt magnets and pulsed electromagnetic fields on tooth movement. *Am J Orthod Dentofacial Orthop*. 1995;107:578–588.
167. Darendeliler MA, Joho JP. Magnetic activator device II (MAD II) for correction of Class II, Division 1 malocclusions. *Am J Orthod Dentofacial Orthop*. 1993;103:223–229.
168. Blechman AM. Pain-free and mobility-free orthodontics? *Am J Orthod Dentofacial Orthop*. 1998;113:379–383.
169. Golyakhovsky V. *Local and Continuous Exposure to a Permanent Magnetic Field as a Means of Boosting Reparative Osteogenesis*. Proceedings of the Kubyshev Regular Conference on Magnetic Fields. Kubyshev, USSR: June 1976 (as translated from Russian by AM Blechman in Blechman AM. Pain-free and mobility-free orthodontics? *Am J Orthod Dentofacial Orthop*. 1998;113:379–383).
170. Vardimon AD, Graber TM, Drescher D, Bourauel C. Rare earth magnets and impaction. *Am J Orthod Dentofacial Orthop*. 1991;100(6):494–512.
171. Bondevik O. How effective is the combined activator-headgear treatment? *Eur J Orthod*. 1991;13:482–485.
172. Coben SE. The spheno-occipital synchondrosis: the missing link between the profession's concept of craniofacial growth and orthodontic treatment. *Am J Orthod Dentofacial Orthop*. 1998;114:709–712.
173. Stöckli PW, Teuscher UM. Combined activator headgear orthopedics. In: Graber TM, Vanarsdall RL, eds. *Orthodontics: Current Principles and Technique*. 2nd ed. St. Louis: Mosby; 1994:437–506.
174. Tanne K, Matsubara S, Sakuda M. Stress distribution in the maxillary complex from orthopedic-headgear forces. *Angle Orthod*. 1993;63:111–118.
175. Battagel JM. The relationship between hard and soft tissue changes following treatment of Class II, Division 1 malocclusion using Edgewise and Fränkel appliance techniques. *Eur J Orthod*. 1990;12:154–165.
176. Braun S, Johnson B, Hnat WP, Gomez JA. Evaluation of the vertical forces generated by cervical biteplate facebow. *Angle Orthod*. 1993;63:119–126.
177. Dugoni SA, Lee JS, Varela J, Dugoni AA. Early mixed dentition treatment: postretention evaluation of stability and relapse. *Angle Orthod*. 1995;65:311–320.
178. Keeling SD, Wheeler TT, King GJ, et al. Anteroposterior skeletal and dental changes after early Class II treatment with Bionators and headgear. *Am J Orthod Dentofacial Orthop*. 1998;113:40–50.
179. Herbst E. *Atlas und Grundriss der Zahnärtzlichen Orthopädie*. Munich, Germany: JF Lehmann Verlag; 1910.
180. Herbst E. Dreissigjahrige erfahrungen mit demretentionscharnier. *Zahnartzl Rundsch*. 1934;43:1515–1524.
181. West RP. The adjustable bite corrector. *J Clin Orthod*. 1995;29:650–657.
182. Greenfield RL. Fixed piston appliance for rapid Class II correction. *J Clin Orthod*. 1995;29:174–183.
183. Coelho Filho CM. Clinical applications of the mandibular protraction appliance. *J Clin Orthod*. 1997;31:92–102.
184. Jasper JJ, McNamara JA, Mollenhauer B. The modified Herbst appliance (Jasper Jumper). In: Graber TM, Rakosi T, Petrovic A, eds. *Dentofacial Orthopedics with Functional Appliances*. 2nd ed. St. Louis: Mosby; 1997:367.
185. Weiland FJ, Bantleon HP. Treatment of Class II malocclusions with the Jasper Jumper appliance—a preliminary report. *Am J Orthod Dentofacial Orthop*. 1995;108:341–350.
186. Weiland FJ, Ingervall B, Bantleon HP, Droacht H. Initial effects of treatment of Class II malocclusion with the Herren activator, activator-headgear combination, and Jasper Jumper. *Am J Orthod Dentofacial Orthop*. 1997;112:19–27.
187. Blackwood 3rd HO. Clinical management of the Jasper Jumper. *J Clin Orthod*. 1991;25:755–760.
188. Erdogan E, Erdogan E. Asymmetric application of the Jasper Jumper in the correction of midline discrepancies. *J Clin Orthod*. 1998;32:170–180.
189. Schwindling FP. *Jasper Jumper Color Atlas*. Merzig, Germany: Schwindling; 1997.

36

Treatment of the Face with Biocompatible Orthodontics

Dwight Damon

OUTLINE

The Damon System Concept, 997.e96
Early Observations of Damon System Treatment, 997.e97
Contemporary Orthodontic Philosophies, 997.e98
Achieve Facial Harmony via Facially Driven Treatment Planning, 997.e98
Use of Nonextraction Therapy Where Possible and Light-Force Mechanics, 997.e113
Force Management, 997.e113
Achieving Extremely Light-Force Mechanics: A Passive Tube, 997.e113
A Look at Sliding Mechanics, 997.e114
Case for Using Extremely Light Forces in Passive Tubes, 997.e115
Computed Tomographic Scans Demonstrate Healthy Bone Structure after Treatment with the Low-Force Damon Tube System, 997.e118
Case Presentations and Clinical Analyses, 997.e118
Treatment with the Herbst Appliance of Growing Patients with Skeletal Class II Dentition, 997.e144
Clinical Principles for Using the Herbst Appliance, 997.e144
Clinical Application of the Herbst Appliance, 997.e145
Damon System Essentials, 997.e160
Damon System Appliance, 997.e160
Damon Standard Prescription, 997.e160
High-Torque and Low-Torque Alternatives to the Damon Standard Prescription, 997.e161
Damon System Archwires and Archwire Sequencing, 997.e161
Light Round Wire Phase, 997.e162
High-Tech Edgewise Phase, 997.e162
Major Mechanics Phase, 997.e162
Finishing Phase, 997.e163
Tieback Usage with the Damon System, 997.e163
Using Elastics with the Damon System, 997.e163
Lingual Retainer Wire and Splint Retainer, 997.e163
Summary, 997.e165

THE DAMON SYSTEM CONCEPT

The philosophy underlying the intended use of the Damon System (Ormco Corporation, Orange, CA) is to approximate biologically induced, tooth-moving forces in each phase of orthodontic treatment. The Damon System achieves this goal by means of a passive, virtually friction-free, self-locking, fixed-appliance conduit that maximizes the full potential of today's high-tech archwires. By doing so, the Damon System provides a reliable and simple means of achieving the best possible facial balance for each patient through the use of light forces that foster corrective functional adaptation of the archform while maximizing patient comfort during treatment. This functional adaptation is similar to the *Fränkel effect* in its posterior arch-widening results. Traditional treatment planning has long been based on maintaining the original archform for stability. In patients with muscle imbalance and collapsed archforms, tooth mass often had to be eliminated. Extensive clinical results indicate that clinicians can now maintain most complete dentitions, even in severely crowded arches, by using very light–force, high-tech archwires in the passive Damon appliance that alter the balance of forces among the lips, tongue, and muscles of the face. This alteration creates a new force equilibrium that allows the archform to reshape itself to accommodate the teeth; the body, not the clinician, determines where the teeth should be positioned. The author refers to this phenomenon as "physiologically determined" tooth positioning. Computed tomographic (CT) scans taken of patients just debonded and those in retention for longer than 5 years demonstrate that using light forces in a passive tube with a small wire-to-lumen ratio enables teeth to be bodily moved, without excessive tipping, in all planes of space and that alveolar bone will follow. This compelling research calls for a significant shift in thinking and treatment planning, reducing and even eliminating the need for molar distalization, extractions (excluding those deemed appropriate for bimaxillary protrusive cases), and rapid palatal expansion.

Practicing orthodontist Alan Pollard describes the Damon appliance system as unique in offering "rapid alignment with gentle forces, functional adaptation and accurate, predictable tooth positioning with micro precision." He continues, "It provides a well-documented means, a virtually friction-free tube, by which the most advanced wire technologies can work to their maximum advantage, an aim most of us have aspired to but have not been able to achieve in conventionally ligated edgewise systems."[1]

Pollard refers to the Damon appliance as a *tube*. Tube is a useful and accurate geometric description of this passive appliance. The appliance is a tube with tie-wings rather than a bracket and, as such, has a static facial wall when its locking mechanism is closed. To emphasize the important difference between this passive appliance and active self-locking brackets, the Damon bracket appliance is referred to as a tube throughout this discussion.

This chapter presents documentation that substantiates existing clinical findings on patients treated with extremely light forces via the Damon System. Before doing so, the author reviewed the early observations he made in using the system and the two important governing principles (achieving facial harmony and using nonextraction therapy where possible, about which most orthodontists agree today) and their close alignment with Damon System principles. Review of these principles leads to a systematic discussion of the vascularity of the periodontal ligament and alveolar complex. CT scans of various types of cases demonstrate stable and even improved alveolar bone formation after bodily tooth movement with light forces via the Damon System. Treatment results of a wide range of malocclusions demonstrate the application and versatility of the system. After these presentations, the section, "Damon System Essentials," touches on torque selection, the Damon archform and archwire sequencing, interarch elastic configurations, and the use of the retention splint.

EARLY OBSERVATIONS OF DAMON SYSTEM TREATMENT

When the author first started using this new passive tube technology in the mid-1990s, it became obvious that alveolar bone, tissue, and teeth responded differently from those treated with conventional high-force mechanics. The following observations were made from some of the earliest cases treated with the first Damon SL tubes. These observations totally changed the author's treatment planning and have led to improvements in bracket-tube geometries and materials. Most of the photographs in this section exhibit much earlier and larger iterations of the Damon tube appliance.

1. *The system offers minimal negative impact on the archform when aligning severely malpositioned teeth.* When using conventionally tied appliances and engaging a high upper canine, the expected impact would be for the adjacent lateral incisor and first premolar to intrude or superiorly move with the incisors anteriorly flaring. With this tube technology and low-force, low-friction mechanics, these adverse responses are minimized or eliminated (Fig. 36-1).
2. *The appropriate force and wire-to-lumen ratio produces a Fränkel-type, arch-widening effect in the posterior, whereas lower canine width stays approximately the same.* The action of the small round archwire (0.014 inch) in the large lumen produces posterior transverse arch-widening that accommodates most complete dentitions, even in severely crowded arches and without the use of high-force palatal expansion (Fig. 36-2, *A* and *B*). Usually minimal change occurs in lower incisor position on the composite head film tracing (Fig. 36-2, *C*). (The only exception is when the canines are severely positioned anteriorly or lingually to the lateral incisors, which is consistent with other types of clinical mechanics with or without extractions.) Establishing the appropriate force and wire-to-lumen ratio in the initial phase of treatment is critical for the orthodontist. The initial archwire (Ni-Ti Align SE or Damon Copper Ni-Ti (Ormco Corporation, Orange, CA) must not exceed 0.014 inch in diameter and must remain an adequate time to allow its full expression (Fig. 36-3). Moving too quickly or forcing archwire changes through the Ni-Ti Align SE or the Damon Copper Ni-Ti phases may disrupt the adaptation process, totally altering the impact on the face, bone, muscles, and soft tissues.
3. *The orbicularis oris and mentalis muscles create a lip bumper effect, which minimizes anterior movement of the incisors.* Based on experience with conventional mechanics, orthodontists expect to see labial movement of anterior teeth when trying to accommodate teeth in a crowded arch without making room for them via extractions. In

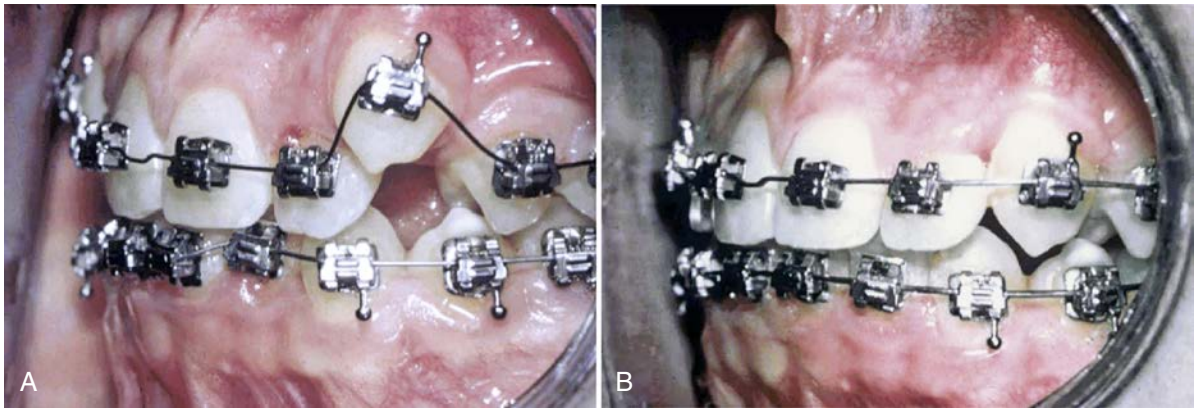


FIGURE 36-1 A, B, When bringing a high canine into position, light-force tube technology produces minimal impact on the adjacent teeth and little flaring of the incisors.

using the tube system, the author noticed that the anterior movement of the incisors was minimal, and the lips and muscles of the face become powerful allies with this low-force system. The patient in Figure 36-4, *A* and *B*, is an early example of this phenomenon. Because of her flat midface, treating this patient without extractions was essential. With a minimal incisor inclination change, treatment brought a pleasing dental result (Fig. 36-4, *C-H*). The composite head film shows minimal anterior movement of the incisors (see Fig. 36-4, *D*). The patient also had excellent tissue and periodontal response (see Fig. 36-4, *E*) 3½ years in retention.

4. *In cases treated with nonextraction, as the arch widens in the posterior, the tongue usually lifts and moves forward, creating a new force equilibrium between it and the lips and muscles of the face.* This phenomenon provides the muscles of the face and tongue a second chance to balance themselves and for the archform to continue to reshape itself to accommodate the teeth (Fig. 36-5).
5. *In bimaxillary protrusive cases treated through extraction therapy, treatment mechanics are greatly simplified with the lip bumper or headgear effect of the facial muscles, thus minimizing the demand on posterior anchorage.* The crowded anterior teeth, engaged with an archwire no stronger than 0.014-inch Ni-Ti or Copper Ni-Ti, take the path of least resistance into the extraction site (Fig. 36-6).

CONTEMPORARY ORTHODONTIC PHILOSOPHIES

Achieve Facial Harmony via Facially Driven Treatment Planning

Most contemporary clinicians have expanded their primary focus toward creating beautiful and handsome faces, with "putting plaster on the table" filling a vital but contributory role. Treatment planning is now concentrated on developing a result that is conducive, rather than detrimental, to long-term maturation of the face; that is, treating the 13-year-old patient with the future 50-year-old adult in mind.

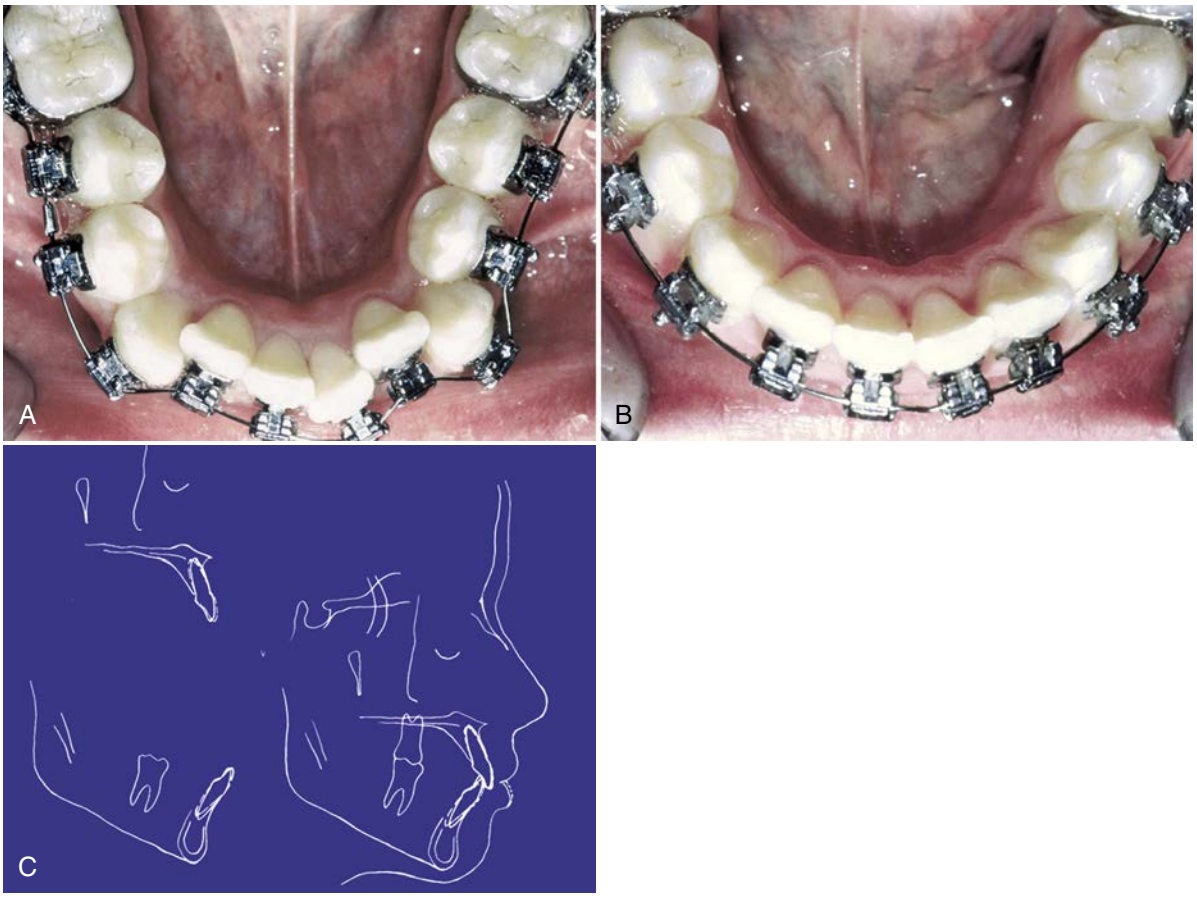


FIGURE 36-2 A, B, The action of the small round archwire (0.014 inch) in the large lumen of the Damon System tube appliance produces a Fränkel-type arch-widening effect in the posterior, usually with minimal change in the lower incisor position on the composite head film tracing **(C)**.

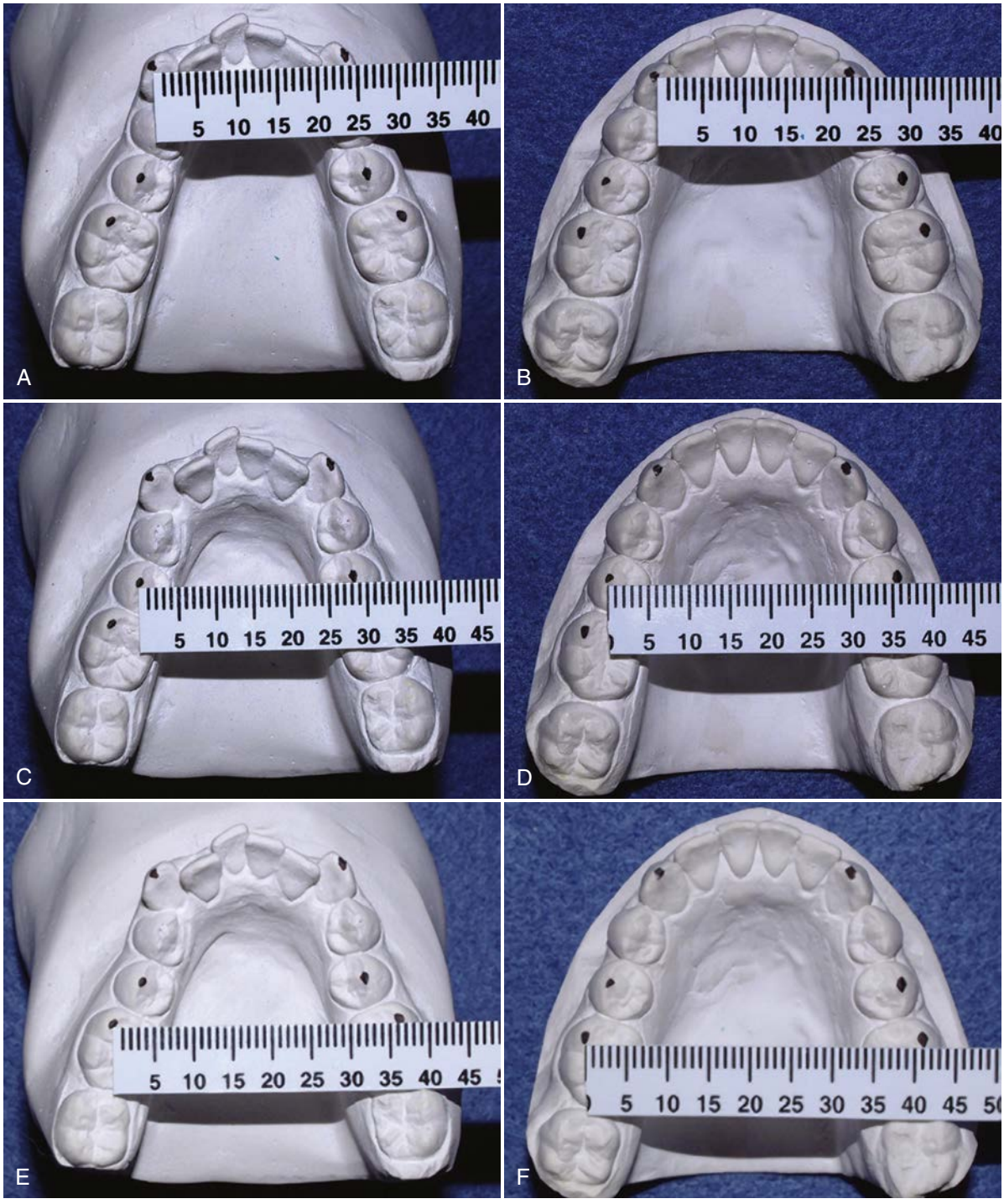


FIGURE 36-3 Lower canine width stays approximately the same if the initial nickel-titanium (Ni-Ti) archwire does not exceed 0.014 inch in size. This early case demonstrates a −1-mm change in canine width, an 8-mm change in first premolar width, and a 5-mm change in first molar width. **A,** Pretreatment. Canines: 23.5 mm. **B,** Posttreatment. Canines: 22.5 mm; change: −1 mm. **C,** Pretreatment. First premolars: 28 mm. **D,** Posttreatment. First premolars: 36 mm; change: 8 mm. **E,** Pretreatment. First molars: 36 mm. **F,** Posttreatment. First molars: 41 mm; change: 5 mm.

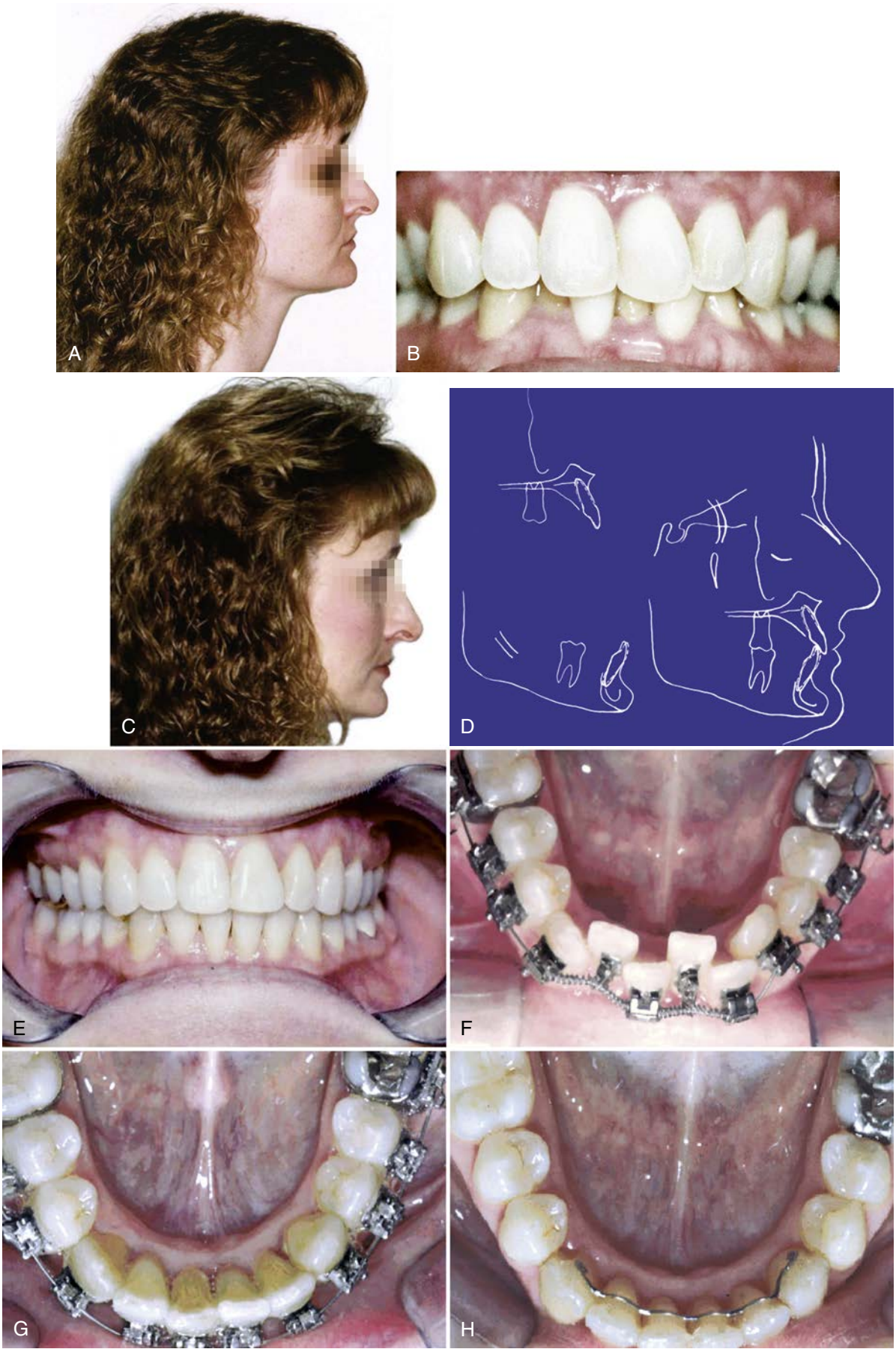


FIGURE 36-4 The lip bumper effect of the orbicularis oris and mentalis muscles precludes dumping of the incisors. **A, B,** Pretreatment records. **C-E,** Posttreatment records. **F,** Pretreatment lower arch. **G,** Third appointment after 3½ months of treatment. **H,** Retention record at 3½ years.

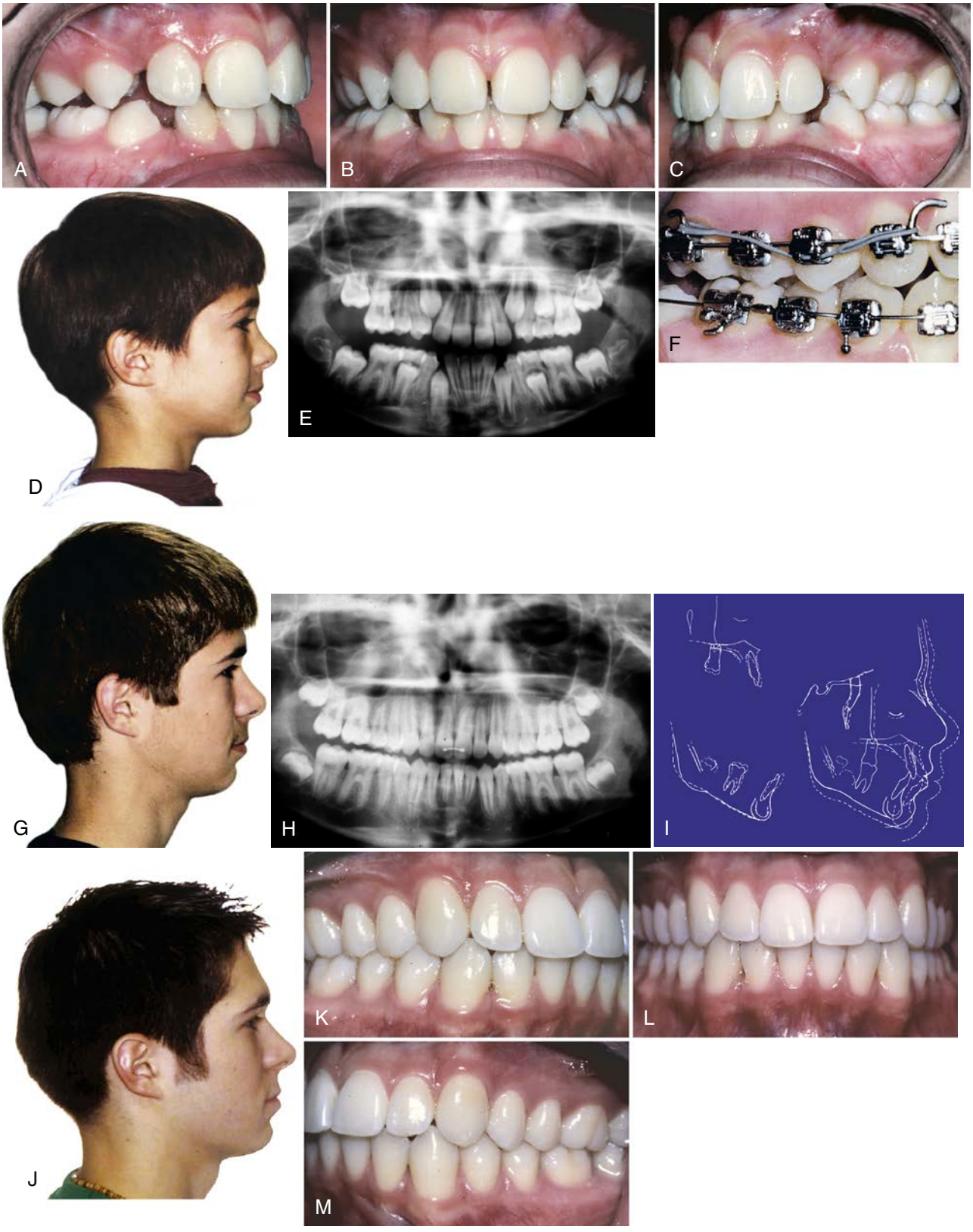


FIGURE 36-5 In cases treated with nonextraction, as the arch widens in the posterior, the tongue lifts and moves forward, creating a new force equilibrium between it and the lips and muscles of the face. **A-E,** Pretreatment. **F,** Tongue responds to the change in the posterior arch width and lifts into new position, often creating a posterior open bite. **G-I,** Posttreatment. **J-M,** Results after 4 years and 3 months in retention with no retainer wear for the last 2 years.

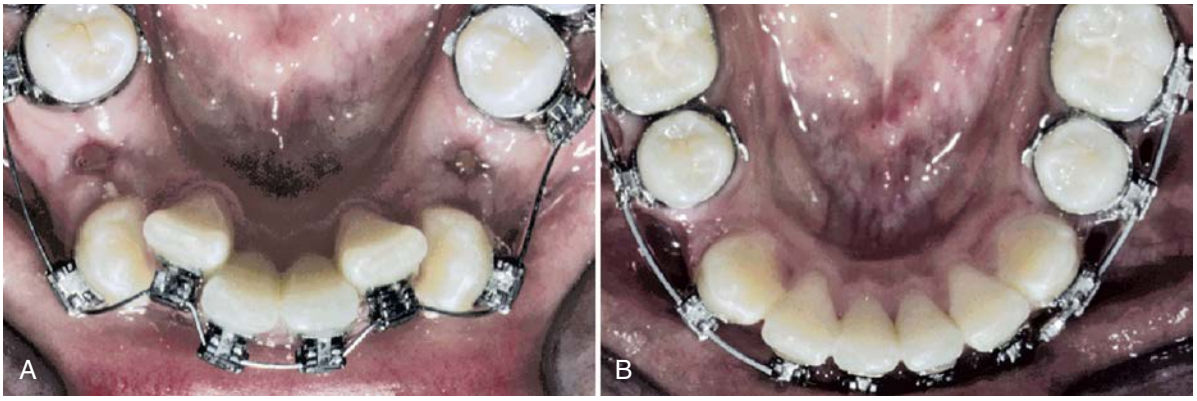


FIGURE 36-6 Bimaxillary protrusive extraction case. Treatment mechanics were greatly simplified with the lip bumper or headgear effect of the facial muscles minimizing the demand on posterior anchorage. As the crowded teeth aligned, they took the path of least resistance, which was into the extraction space. **A,** Pretreatment. **B,** At 6 months, 2 weeks.

CASE STUDY 36-1 Achieving Facial Harmony with Facially Driven Treatment Planning

Case 1 (CB) demonstrates a good prospect for this method of treatment planning, which provides expanded options for extremely low-force mechanics. CB exhibits a Class I face (Class II dentally on the right) with severe crowding in both arches and a unilateral posterior crossbite. His canines were totally blocked and had erupted labially to the lateral incisors in both arches with minimal bone and thin labial tissue (Fig. 36-7). The upper second premolars were erupting toward the palate. CB resembles his father, who is tall with a strong nose and chin, so one can assume that his well-proportioned nose and chin buttons will dramatically change as his face matures. He lacks facial support laterally; his midface is somewhat concave with an obtuse nasolabial angle. Along with a thinning of the lips, such common facial traits are prevalent in patients with collapsed and crowded dental arches.

The treatment objectives were as follows:

1. Gain maxillary and mandibular arch length to achieve facial balance with a positive impact on patient profile.
2. Establish upper and lower incisor position to provide lip support.
3. Establish maxillary and mandibular posterior arch width to support midface.
4. Establish ideal maxillary lip-to-tooth relationship.
5. Design treatment mechanics to eliminate the need for high-force rapid palatal expansion.
6. With low-force mechanics working with the orofacial muscle complex, bone, and tissue, establish a physiologically determined tooth position.

Conventional treatment planning would suggest that four first premolars should be extracted, and early in the author's career, the extractions would have been done without thinking about the long-term impact on this young man's profile. The long-term result of many such treatment plans produced flat or dished-in faces. In this case, evaluating where this profile could be at 30 years of age is absolutely critical. Even with extractions, a lower bonded retainer would still have been necessary to maintain lower incisor position over the long term. If lower bonded retainers are required for stability, then why not treat the face using light-force archwires in a passive tube system to gain arch length, especially when one can do so with far less trauma than higher force extraction therapy?

Box 36-1 outlines the treatment sequence of the case. Special torques were selected for this case: +7 degrees for the upper central incisors and +3 degrees for the upper lateral incisors. The torque used on the lower central incisors and lateral incisors was −6 degrees. Before beginning orthodontic treatment, the author prescribed that the patient have his primary second molars extracted. Because of the severe labial position of the canines, extraction was necessary to gain space immediately; consequently, the author began treatment in the maxilla with a rectangular wire (0.014- × 0.025-inch Ni-Ti Align SE) with medium-light Ni-Ti springs activated 1.5 to 2.0 times the width of a bracket (Fig. 36-8). Beginning treatment with a rectangular Ni-Ti wire is not a recommended treatment protocol for Damon System mechanics; but, in this case, the upper anterior teeth were well aligned and the canines were rapidly erupting through the cortical plate. The author needed to apply the gentle space-opening mechanism of Ni-Ti coil springs from the four anterior teeth to the molars to gain arch length and transverse arch width, making room for the canines to descend into their normal position. The protocol mandates a 0.014-inch Ni-Ti archwire as the initial archwire; however, because of the interbracket distance and the need to engage a Ni-Ti coil spring, the case required the stability of a rectangular wire. As the space became available, the canines came down naturally and were not bracketed until they were well-positioned in the archform. A mandibular 0.014-inch Ni-Ti Align SE sectional archwire was placed in the more crowded lower arch only lateral to lateral incisor because, with a span so great, the archwire would have disengaged had the wire engaged the first molars (Fig. 36-9 to Fig. 36-11; see also Fig. 36-8).

Given the opportunity, this case should have been started long before the canines erupted through the tissue; however, it does provide an excellent example of why orthodontists should design treatment mechanics that do not overpower the biologic system. At the beginning of treatment, the canines are labial to the lateral incisors in both arches. With conventional mechanics, the challenge is gaining room for them to be included in the arch without negatively affecting the lateral roots. With light-force tube mechanics, the orbicularis oris and mentalis muscles minimize the anterior flaring of the incisors but also encourage the distal positioning of the canines as space is gained in the arch with posterior adaptation. Using this treatment protocol for 8 years, the author has not observed a negative impact on the roots of the lateral incisors.

The Damon System treated this patient to a dentally and facially pleasing result without extractions or other high-force mechanics (Fig. 36-12). One should think how differently this 18-year-old's profile, and indeed his entire face, would have looked had four premolars been removed. Figure 36-13 tracks the maturation of the profile of this patient from 13 years, 4 months to 20 years, 8 months of age, clearly demonstrating how well this system served him in precluding extraction therapy.

Years after this case was completed, the author decided that taking CT scans of patients after treatment would be helpful in demonstrating the health of the bony architecture when treated with low-force tube mechanics. Figures 36-14 and 36-15 compare plaster models of the upper and lower arches taken initially and on the day of debonding with CT scans taken 5 or more years after treatment. Without high-force rapid palatal

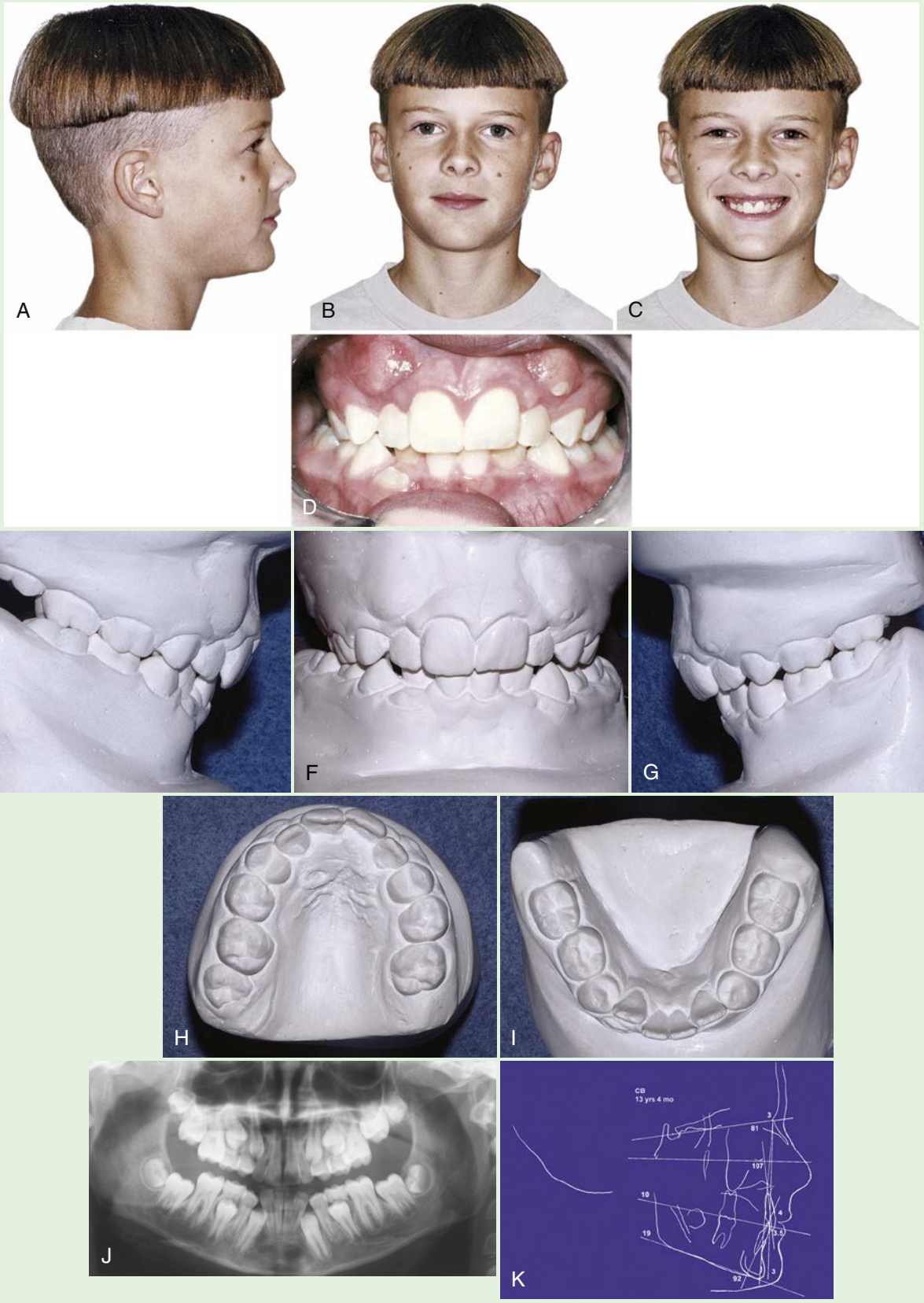


FIGURE 36-7 A-K, Pretreatment records for case 1 (CB, age 13 years, 6 months). Diagnosis: Class I face (Class II dentally on the right) with severe crowding in both arches and a unilateral posterior crossbite. The canines are totally blocked to the labial of the lateral incisors in both arches with thin bone and tissue surrounding them. The upper second premolars are erupting toward the palate. CB laterally lacks facial support; his midface is somewhat concave with an obtuse nasolabial angle.

Continued

CASE STUDY 36-1 Achieving Facial Harmony with Facially Driven Treatment Planning—cont'd

BOX 36-1 Case 1 (CB) Treatment Sequence

First appointment: Banded maxillary and mandibular first molars. Bonded central and lateral incisors (see Fig. 36-8). Because of the severe labial position of the canines, it was necessary to gain space, therefore began treatment with a maxillary rectangular wire (0.014- × 0.025-inch Ni-Ti Align SE) with medium-light Ni-Ti springs activated 1.5 to 2.0 times the width of a bracket. Placed mandibular 0.014-inch Ni-Ti Align SE sectional archwire.

Second appointment at 2 months, 1 week: Placed maxillary and mandibular 0.016- × 0.025-inch Ni-Ti Align SE. Activated Ni-Ti springs 1.5 width of the bracket.

Third appointment at 4 months, 1 week: Checked only the status (see Fig. 36-9).

Fourth appointment at 6 months, 1 week: Activated the springs.

Fifth appointment at 8 months: Placed maxillary and mandibular 0.019- × 0.025-inch stainless steel arch wire (see Fig. 36-10). Took a panoramic radiograph to evaluate the position of erupting permanent teeth.

Sixth appointment at 10 months, 2 weeks: Adjusted the maxillary archwire.

Seventh appointment at 12 months, 2 weeks: Bonded maxillary canines and first and second premolars. Placed continuous 0.014-inch Ni-Ti Align SE in the maxillary arch.

Eighth appointment at 14 months, 3 weeks: Repositioned maxillary left lateral. Waiting on the eruption of the permanent teeth. Patient did not keep appointments for the next 5 months.

Ninth appointment at 19 months, 3 weeks: Bonded mandibular canines and first and second premolars. Placed continuous mandibular 0.014-inch Ni-Ti Align SE. Placed maxillary 0.016- × 0.025-inch Ni-Ti Align SE.

Tenth appointment at 22 months, 1 week: Placed maxillary 0.019- × 0.025-inch stainless steel posted archwire. Placed mandibular 0.016- × 0.025-inch Ni-Ti Align SE. Took head film.

Eleventh appointment at 24 months, 3 weeks: Took panoramic radiograph to evaluate root position (see Fig. 36-11). Adjusted maxillary archwire.

Twelfth appointment (at 26 months, 2 weeks): Bonded mandibular second molars. Placed 0.016-inch Ni-Ti Align SE overlay to engage mandibular second molars.

Thirteenth appointment at 29 months: Bonded upper second molars. Placed maxillary 0.016-inch Ni-Ti SE overlay. Placed crimpable hooks on 0.016- × 0.025-inch Ni-Ti Align SE. Started full-time bilateral V-elastics and anterior trapezoid elastics.

Fourteenth appointment at 30 months, 3 weeks: Adjusted maxillary archwire. Placed mandibular 0.016- × 0.025-inch posted stainless steel archwire in lower arch because additional play between archwire and bracket slot was needed for settling. Continued bilateral V-elastics full time.

Fifteenth appointment at 32 months, 1 week: Adjusted maxillary and mandibular archwires. Continued V-elastics full time.

Sixteenth appointment at 33 months, 2 weeks: Debonded both arches, and placed retention wires. Bonded 0.016- × 0.022-inch Bond-a-Braid braided wire to the maxillary arch lateral to lateral incisor. Placed 0.026-inch round stainless steel wire on the mandibular arch canine to canine, bonding only the canines (see Fig. 36-12). Prescribed upper and lower slipcover retainers for night wear only.

expansion, the maxillary first premolar width increased by 11 mm and the maxillary first molars increased by 14 mm with desired tipping. An estimation of the second premolar change is 12 to 13 mm. The lower first premolar width increased by 11 mm, and the lower first molar width increased by 5 mm. Canines had erupted high through the cortical plate in both arches and could not be measured.

The plaster models and the horizontal CT scans of the upper arch (Fig. 36-16) demonstrate that the majority of tooth movement occurred with minimal tipping, resulting in a well-shaped archform. Figure 36-17 demonstrates that the arch length gain was in the transverse and was achieved through bodily movement of teeth and desirable tipping of posterior teeth. In addition, the palatal anatomic improvement is also evident. Figure 36-18 shows vertical CT scans of the upper second premolars and upper second molars. All the vertical CT scans illustrate excellent tooth position and health of the surrounding bone structure that resulted from bodily tooth movement without high-force rapid palatal expansion. Moreover, the scans illustrate the delicacy of the bony architecture in this area and should challenge all clinicians to consider lowering forces and using an alternative to rapid palatal expansion.

Orthodontists can make a number of interesting observations from the CT scans. The vertical CT scan of the lower second molar (Fig. 36-19) was eye opening for the author. Before using low-force tube mechanics, the author had always assumed that in trying to gain lower arch length, conventional mechanics caused the second molars to erupt lingually; however, in looking at the CT scans, the author came to another conclusion. In crowded cases, the tongue is more passive than normal and typically does not lift or function into the palate. In swallowing at least 1000 times a day and articulating sounds, a normally functioning tongue continually moves up and down in the palate, thus shaping the architecture of the lingual cortical plate, especially in the lower second molar area. In a normal functioning case, the lingual cortical plate is more upright than in crowded cases where the tongue is passive, sitting low in the mouth; thus in such cases, the cortical plate is lingually inclined in the second molar area. The net result is that as the second molar erupts, its crown follows the path of least resistance between the buccal and lingual cortical plate, which usually results in a lingually tipped clinical crown. A second observation is that on nearly all CT scans taken, the apex of the lower second molar root is usually positioned within the lingual cortical plate. This observation supports the recommended treatment protocol of the system to cut the stainless steel working archwire between the first and second molars when applying Class II elastic mechanics. Because the apex of the second molar is sitting in cortical bone, which has low vascularity, orthodontists only need to engage it in cases of maximum anchorage. Ricketts spoke of the anchorage value present when tipping the lower roots against or into the buccal cortical plate.

The horizontal CT scan of the lower arch (Fig. 36-20) offers an important vantage point when considering orthodontic force. The bony contours on the labial, buccal, and lingual sides are demonstrated. Much of the tooth structure normally sits within the thin-layer, low-vascular cortical bone, which supports the concept of using extremely low forces. The scan lends support to the idea that in using low-force mechanics, cortical bone will follow tooth movement in all planes of space. The author believes that when orthodontists can simulate the forces of natural tooth movement—as orthodontists have done over the years with the Fränkel appliance—the body has an incredible ability to adapt. An interesting analogy is how the cortical plate adapts (Fig. 36-21) around a horizontally impacted tooth as it slowly develops. Figure 36-22 shows records taken after 5 years, 3 months in retention. Despite the fact that CB had not worn his retainers for more than 4 years, the dentition remained stable.

CASE STUDY 36-1 **Achieving Facial Harmony with Facially Driven Treatment Planning—cont'd**

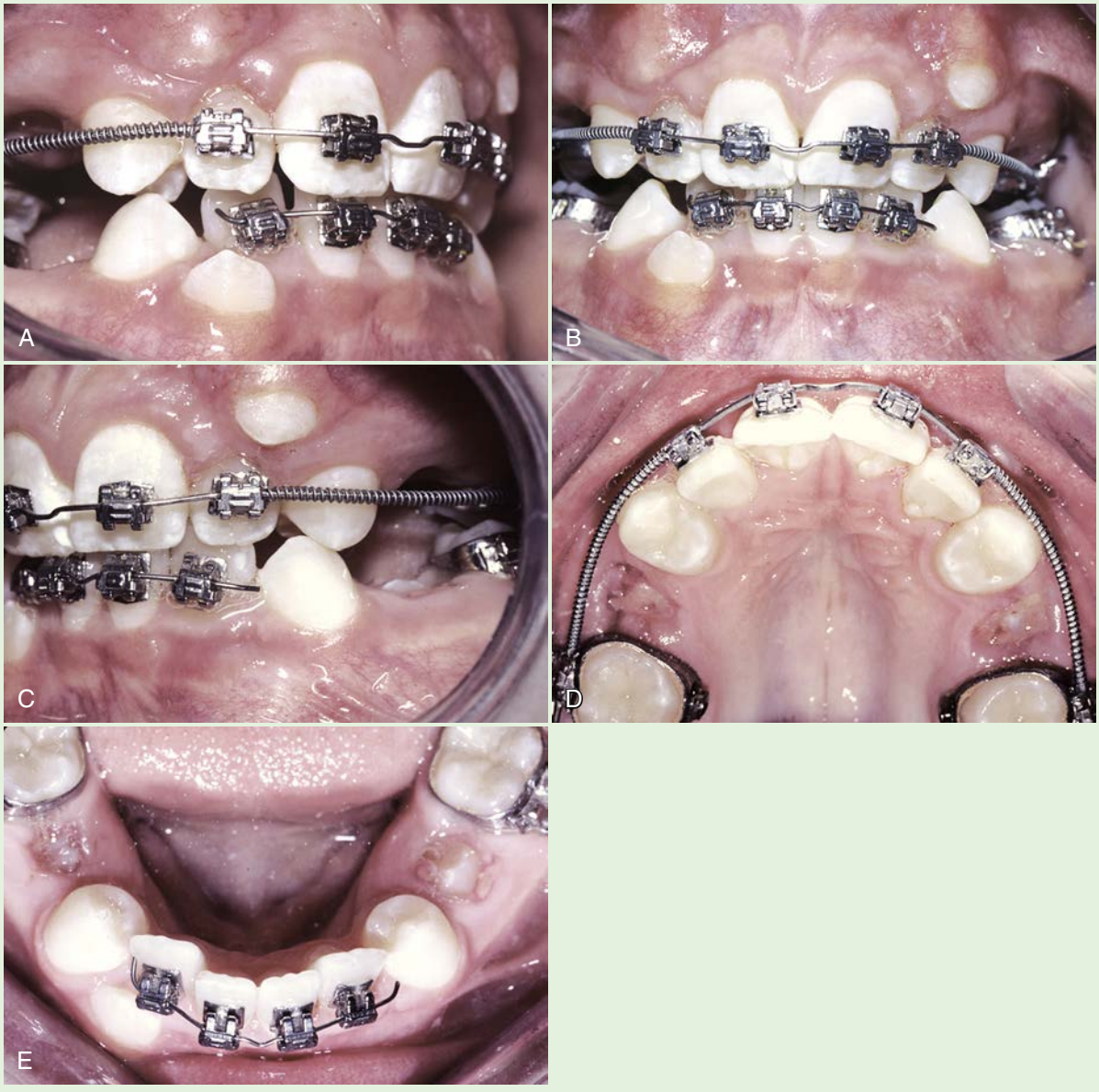


FIGURE 36-8 A-E, Bonding appointment for CB.

Continued

CASE STUDY 36-1 **Achieving Facial Harmony with Facially Driven Treatment Planning—cont'd**

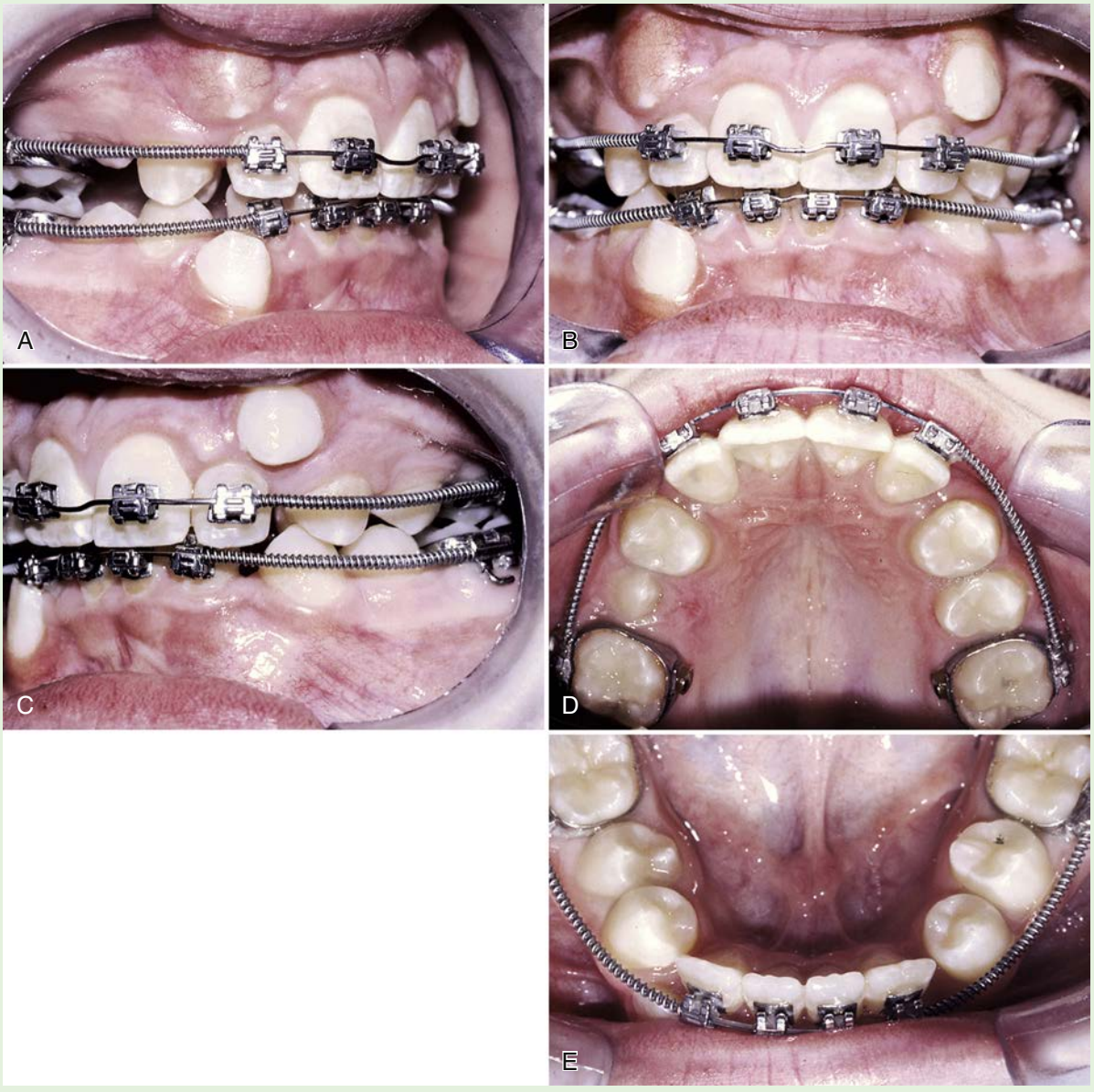


FIGURE 36-9 A-E, Third appointment for CB at 4 months, 1 week.

CASE STUDY 36-1 **Achieving Facial Harmony with Facially Driven Treatment Planning—cont'd**

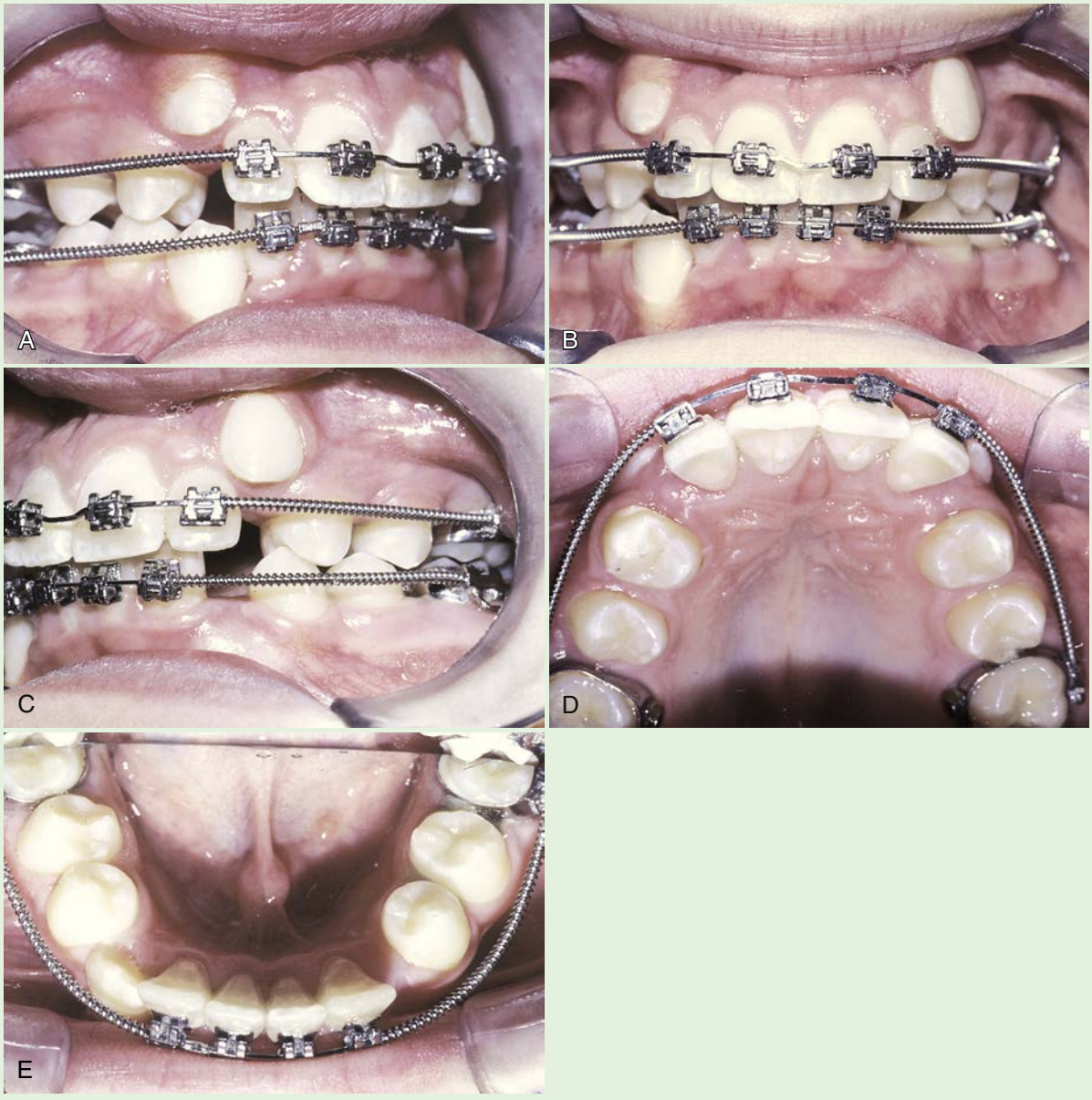


FIGURE 36-10 A-E, Fifth appointment for CB at 8 months.

FIGURE 36-11 Panoramic radiograph taken during the eleventh appointment for CB at 24 months, 3 weeks.

Continued

CASE STUDY 36-1 **Achieving Facial Harmony with Facially Driven Treatment Planning—cont'd**

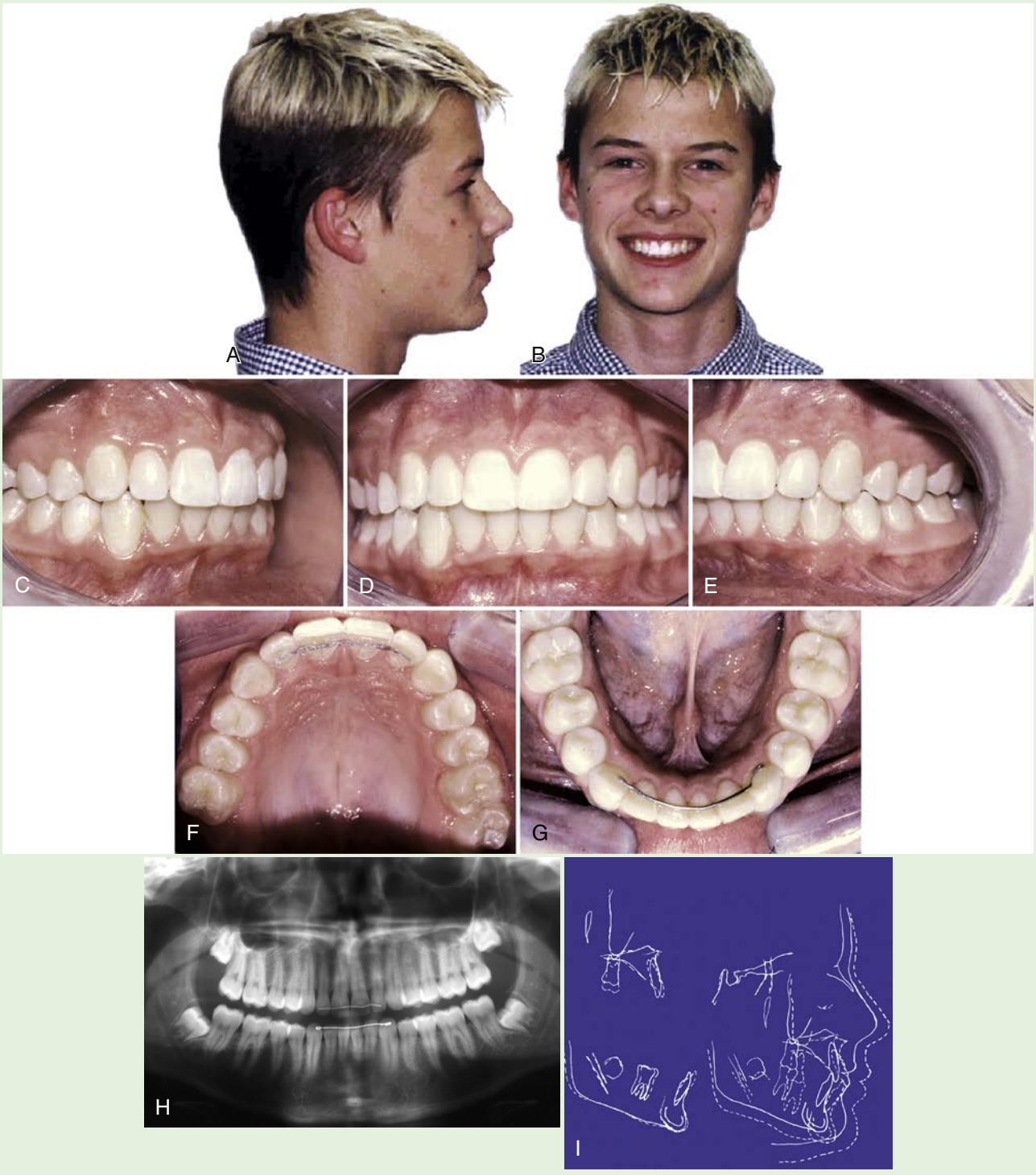


FIGURE 36-12 A-I, Posttreatment records of CB. Total treatment time: 33 months, 2 weeks with 16 appointments. The length of treatment was dictated, in part, by the time required for the canines to erupt into the arch and the fact that the patient did not keep his appointments for 6 months during midtreatment.

CASE STUDY 36-1 Achieving Facial Harmony with Facially Driven Treatment Planning—cont'd

FIGURE 36-13 These photographs track the maturation of CB's profile from 13 years, 4 months to 20 years, 8 months, clearly demonstrating how well the Damon System served CB in precluding extraction therapy.

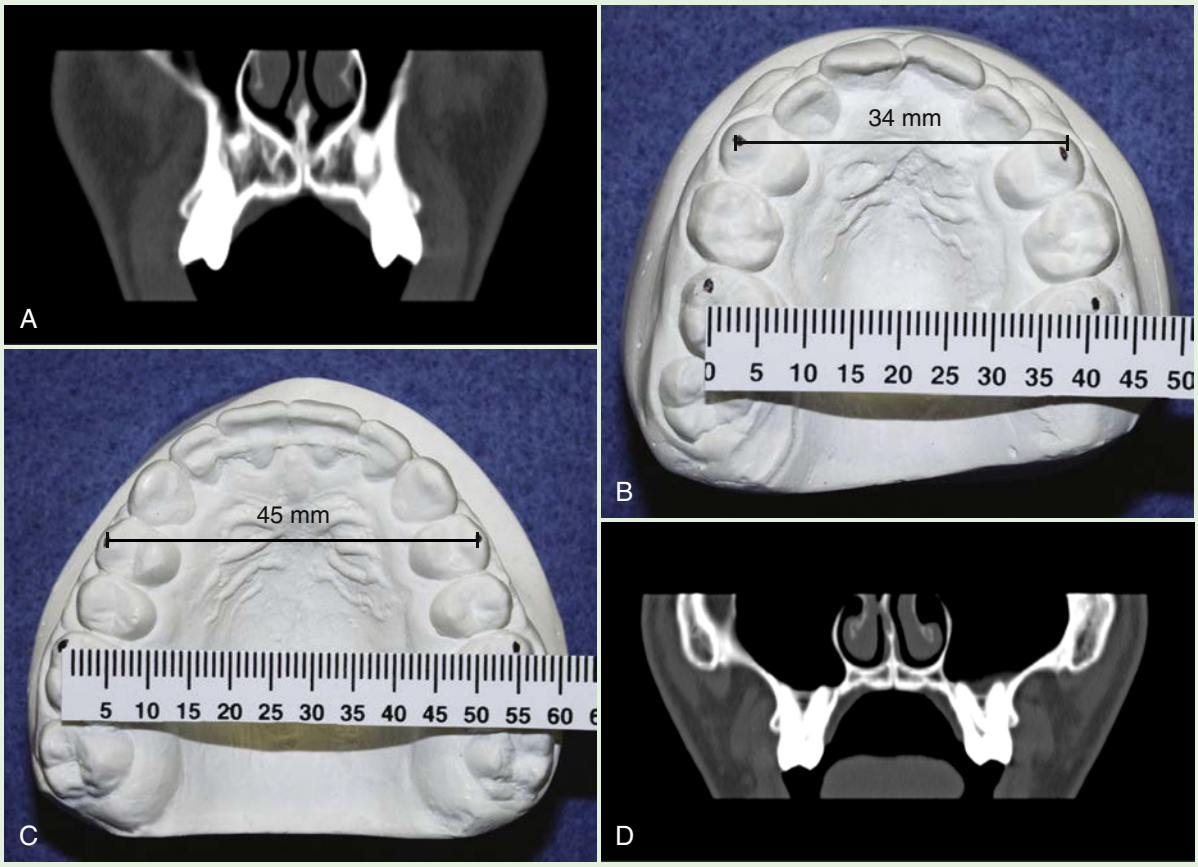


FIGURE 36-14 Comparisons of pretreatment and posttreatment upper arch plaster models for CB with computed tomographic scans. Pretreatment and posttreatment plaster models of the upper arch show 11-mm change in first premolar width (and an estimated 12- to 13-mm change in second premolar width) and 14-mm change in first molar width. The bony contours on the buccal of the first premolars and first molars are evident. Vertical scans taken 5 years, 3 months into retention show healthy alveolar bone. **A,** Scan of upper first premolars at 5 years, 3 months in retention. **B,** Pretreatment. First premolars: 34 mm; first molars: 41 mm. **C,** Posttreatment. First premolars: 45 mm; first molars: 55 mm. **D,** Scan of upper first molars 5 years, 3 months in retention.

Continued

CASE STUDY 36-1 **Achieving Facial Harmony with Facially Driven Treatment Planning—cont'd**

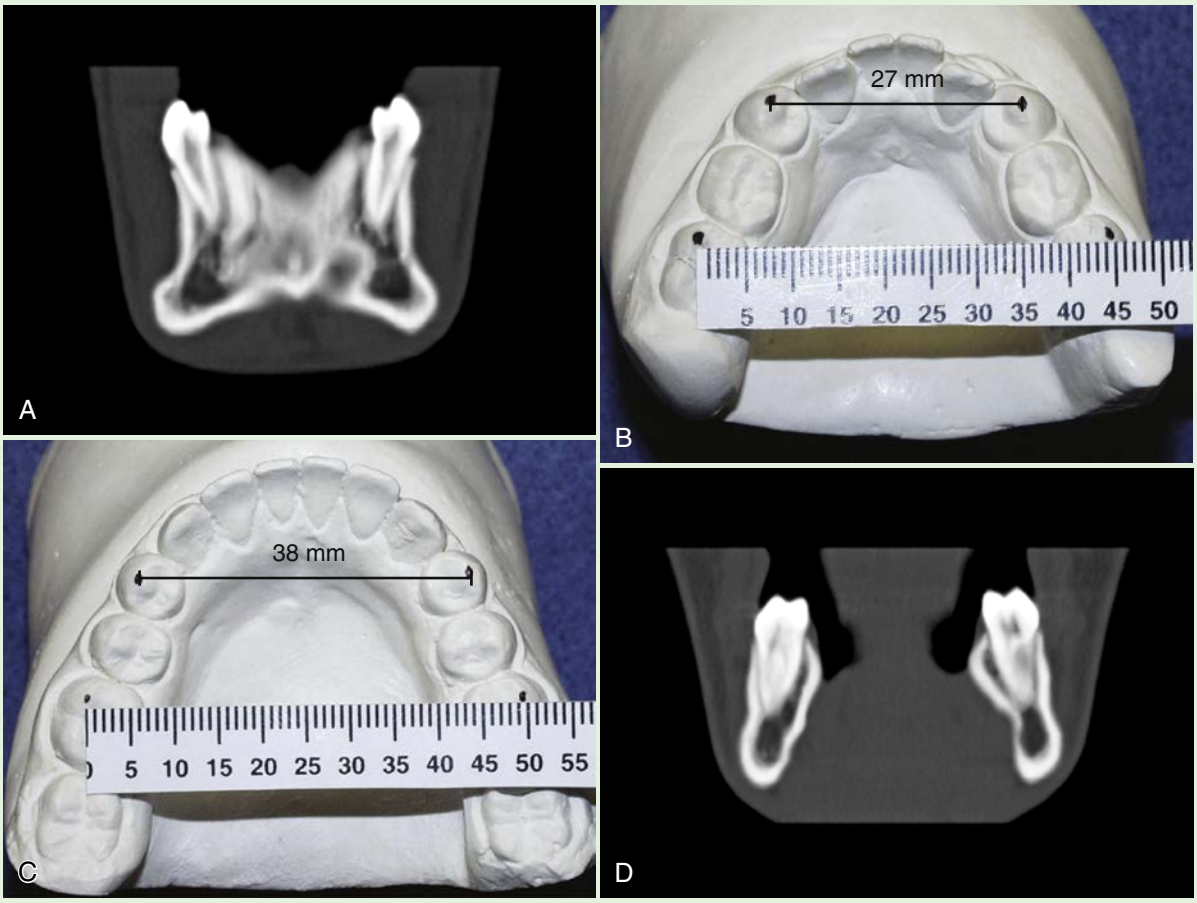


FIGURE 36-15 Comparisons of pretreatment and posttreatment lower arch plaster models for CB with computed tomographic scans. Pretreatment and posttreatment models of lower arch show 11-mm change in first premolar width and 5-mm change in first molar width. Vertical scans, taken 5 years, 3 months into retention, show healthy alveolar bone. **A,** Scan of lower first premolars 5 years, 3 months in retention. **B,** Pretreatment. First premolars: 27 mm; first molars: 44.5 mm. **C,** Posttreatment. First premolars: 38 mm; first molars: 49.5 mm. **D,** Scan of lower first molars 5 years and 3 months in retention.

FIGURE 36-16 The horizontal computed tomographic scan of CB's upper arch illustrates the well-shaped archform.

CASE STUDY 36-1 **Achieving Facial Harmony with Facially Driven Treatment Planning—cont'd**

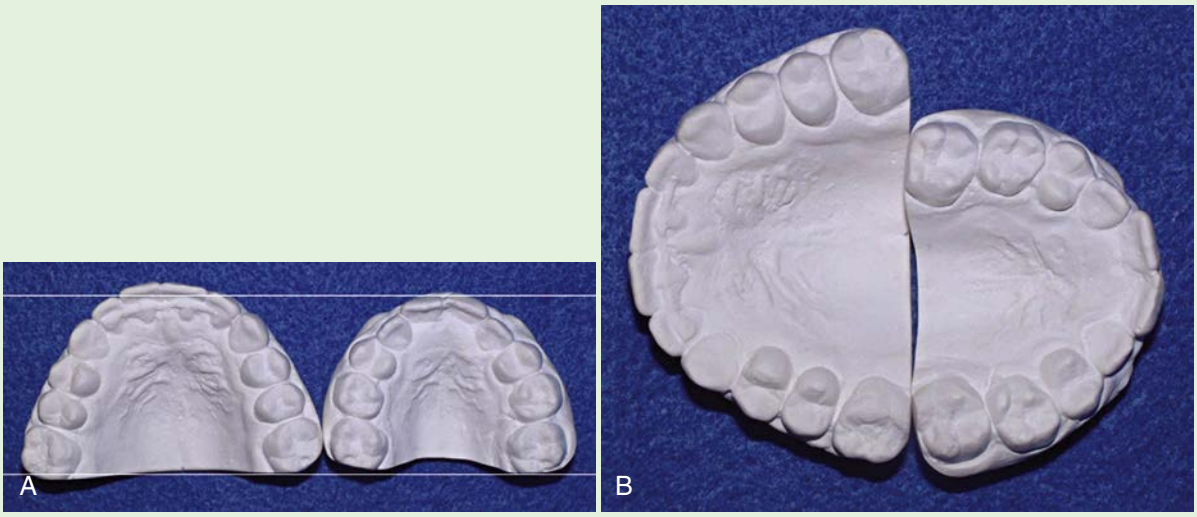


FIGURE 36-17 A, B, Plaster models demonstrate that the arch length gain was in the transverse achieved through bodily movement of the teeth and desirable tipping of the posterior teeth. The improvement in palatal contours is also evident.

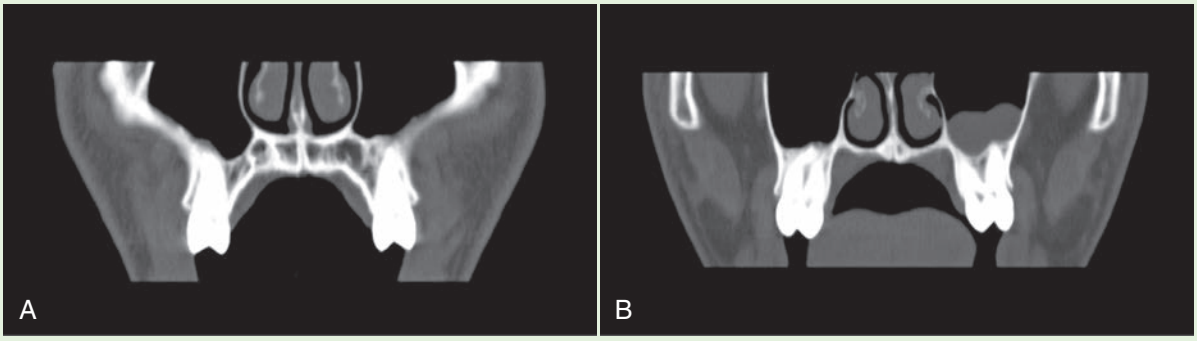


FIGURE 36-18 Computed tomographic scans of CB taken 5 years, 3 months into retention. A, Scan of upper second premolars illustrates the excellent tooth position and surrounding bone structure that resulted from bodily movement without a high-force rapid palatal expander. B, Scan of upper second molars illustrates the delicate bony architecture of this area and challenges clinicians to consider lowering forces and using an alternative to rapid palatal expansion.

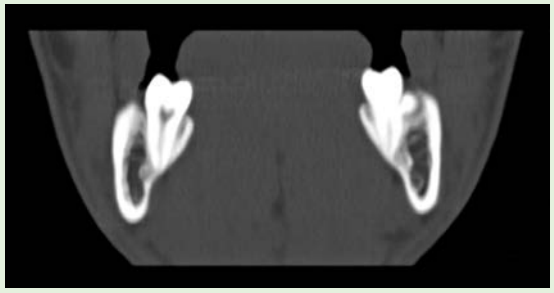


FIGURE 36-19 The bony architecture in the second molar area of a collapsed arch encourages second molars to erupt lingually inclined. By helping the tongue assume a normalized position via posterior arch adaptation, second molars have a greater chance of erupting in a more upright position.

Continued

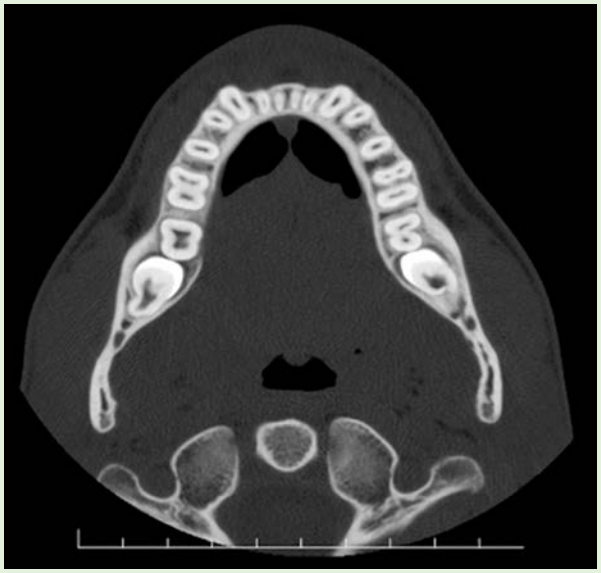


FIGURE 36-20 The fact that much of the tooth structure normally sits in a thin layer of cortical bone of low vascularity supports the concept of using extremely low forces.

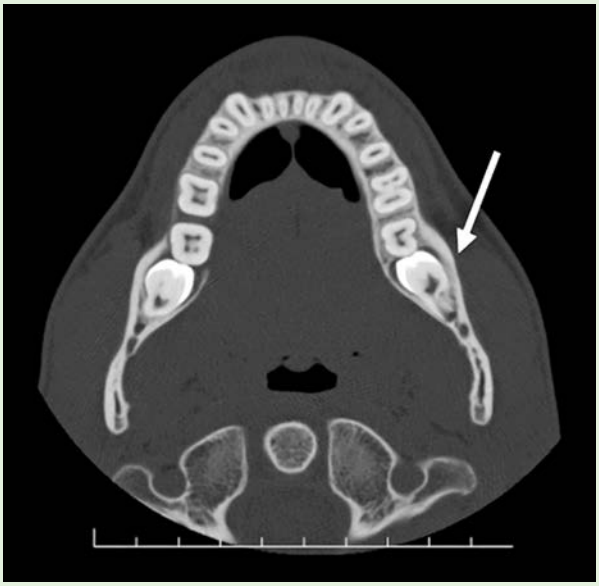


FIGURE 36-21 Computed tomographic scan depicts how the cortical bone has adapted to the third molar *(arrow)* and supports the idea that cortical bone will follow tooth movement in all planes of space.

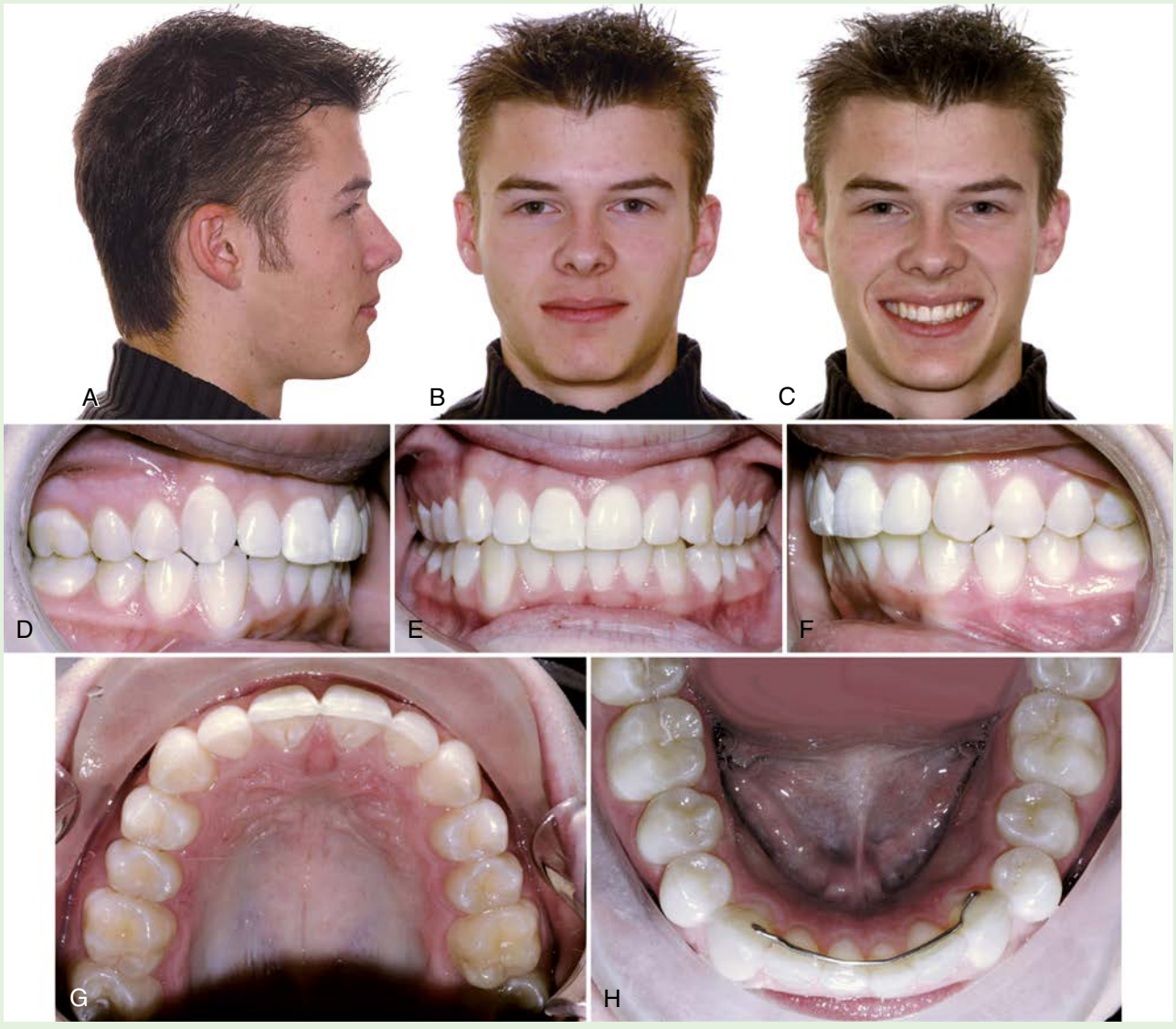


FIGURE 36-22 A-H, Retention records of CB taken at 5 years, 3 months into retention. Despite the fact that the patient did not wear night retainers for more than 4 years, the dentition remains stable.

Use of Nonextraction Therapy Where Possible and Light-Force Mechanics

A second trend—toward nonextraction therapy—is an admirable orthodontic goal. The principle means of achieving nonextraction, however, negates another important biologically sound principle: that of using low-force orthodontic mechanics. Heretofore, other than the Fränkel and similar appliances that have proved difficult to apply and to clinically manage, the principal means of orthodontically gaining arch length were distalizing molars or using high-force rapid palatal expansion devices. Each of these methods has its drawbacks because the forces are high when one considers the fragile vascular architecture (illustrated later in Fig. 36-28). CBCT scans of the midface after rapid palatal expansion show wide ranging effects on skeletal and dental structures. Distalizing molars is also time consuming. Although Damon System practitioners are sometimes reluctant to relinquish their rapid palatal expanders, the author believes that their use has an unnecessarily detrimental effect on the biologic structure and function of the entire dentofacial system. Rather than causing patients to suffer the trauma of opening the palatal sutures, distalizing the molars, or extractions (and the subsequent closure of the extraction sites and adding treatment length), clinicians can now maintain many complete dentitions by using very light-force, high-tech archwires in passive tubes, thus gaining arch length.

FORCE MANAGEMENT

Achieving Extremely Light-Force Mechanics: A Passive Tube

The author has spent nearly 20 years carefully evaluating the rationale for his clinical mechanics. Early on, it became apparent that using a force system was not "biologically sensible." To use a bracket system to move teeth along an archwire that was tightly tied and nearly locked the wire to the base of the bracket slot made little sense. To reach orthodontic goals, orthodontists are challenged to use forces that are consistent with biologically sound principles of tooth movement.

The author believed that an appliance capable of accommodating a biologically attuned system might best be developed by looking into the field of self-locking brackets. Orthodontic history indicates that, from its early years, clinicians perceived the advantages of engaging archwires in tubes rather than ligating them into brackets. One of the earliest tube systems, the twin-wire cap-and-channel appliance, possessed some of the attributes of current self-locking systems but was limited by hard-to-place caps and the absence of space-age wires, preadjusted slots, and modern mechanics. In 1966, the Snap Ring appliance was introduced (3M Unitek, Monrovia, CA). This appliance was not self-locking in today's sense of the term, but it simulated the concept.[2] A staple applicator attached a metal C-ring that locked over the outer portion of a round bracket. The system was limited by the large bracket size and the same shortcomings of the earlier cap-and-channel system.

A later example was the Edgelok bracket (Ormco Corporation, Orange, CA), which was introduced in the late 1960s (Fig. 36-23). Craig Andreiko, director of research and development at Ormco, described the bracket as the "first true self-ligating tube-type appliance." He went on to explain that, "It was comfortable, had a positive-seating mechanism that formed the facial wall that was easy to open and close. It had the misfortune of being introduced around the same time as the StraightWire appliance from 'A' Company. It also suffered from the prevailing edgewise treatment sentiment of the time that dictated an archwire selection and timing that stifled the effectiveness of even the more resilient and efficient archwires that were becoming available; nevertheless, it enjoyed widespread usage for a number of years, waning as preadjusted appliances took hold in the marketplace."[3]

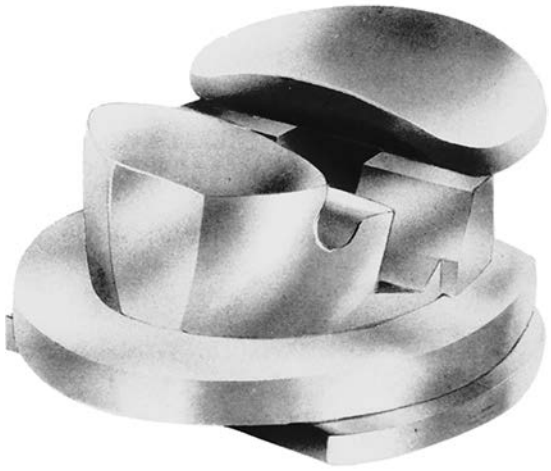


FIGURE 36-23 The Ormco Edgelok self-ligating bracket was introduced in the late 1960s.

Another major advancement of that era was the SPEED appliance (Strite Industries, Cambridge, Canada), an active self-ligating appliance. The use of the SPEED appliance has been shown to reduce archwire changing chair time to as little as 25% of that of conventional twin brackets. The evolution of self-locking appliances greatly accelerated after 1995 with the introduction of active and passive appliances, including the Damon System.

Coinciding with the advances in self-locking appliances has been the evolution of the space-age nickel-titanium wires introduced by George Andreasen and the University of Iowa in cooperation with 3M Unitek. Although the profession widely recognized this development as a landmark advance in providing appropriate, light, continuous orthodontic force, it has been slow to realize its full potential. Ray Morrow, former 3M Unitek research and development project manager for Nitinol wire, related the early problems with usage of the archwires that are analogous with today's failures to take full advantage of their remarkable potential: "Initially, just as they were accustomed to using stainless steel archwires, many clinicians tried to place bends in the wires, so many complaints were received about breakage from what actually was the result of improper usage of the early, somewhat brittle wires. In response, Unitek altered its manufacturing process in order to produce less elastic wires that permitted more bending without breaking. It was some time before proper usage of the wires ended the complaints, even as wires with greater elasticity and resistance to bending were introduced by Unitek and, later, by other companies."[4]

The author has included this bit of history because, even today, the ultimate clinical benefits attainable with the remarkable space-age wires are being severely limited. Steel and elastomeric ligations are primary causes of this problem because they create binding and friction; active self-locking appliances provide a significant improvement but still exert facial-side pressure in many phases of treatment that limit the freedom of

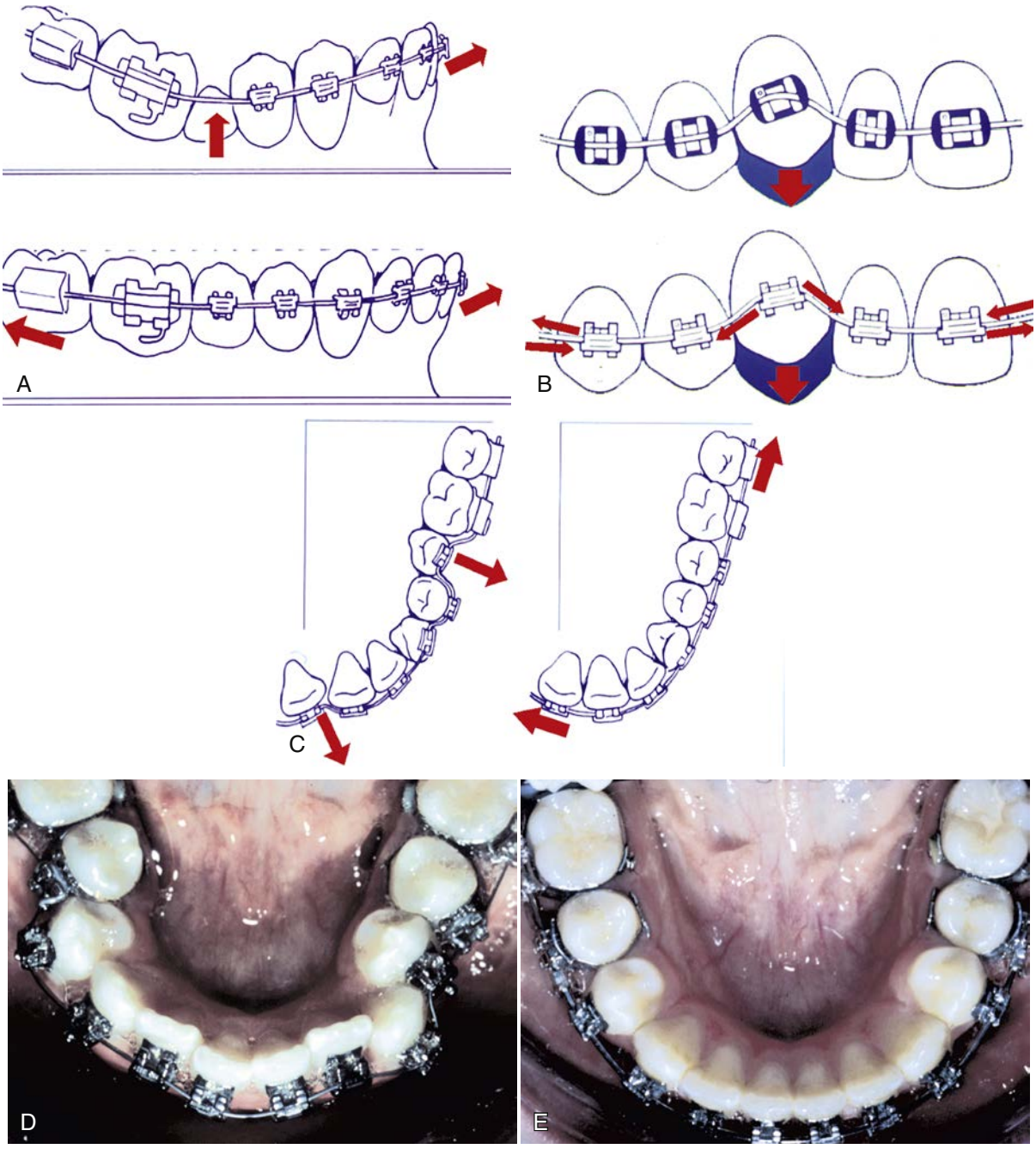


FIGURE 36-24 Sliding mechanics is involved with **(A)** leveling the arch, **(B)** aligning high canines, **(C)** correcting tooth rotations, and **(D, E)** changing archform.

the archwire to perform. Maximizing treatment results requires a passive self-locking appliance (tube), combined with careful selection of archwires (allowing plenty of play in early treatment and in midtreatment) and timing (allowing the wires to work and fully express their potential). The Damon System meets these demands to reach the optimal force range that stimulates cellular activity without occluding blood flow in the periodontium.

A Look at Sliding Mechanics

Many types of space-closure mechanics that require sliding the brackets along the wire as they start to level and align have been used for years, so achieving space closure with the Damon tube is easy for clinicians to visualize and accept. Over the past 30 years, clinicians have moved away from starting cases with multiloop stainless steel archwires in favor of using high-tech continuous wires free of any loops or bends. For teeth to align in the early phases of treatment, sliding mechanics must take place. Leveling the arch, aligning high canines, correcting tooth rotations, and changing archform (Fig. 36-24) are good examples of the use of sliding mechanics. Orthodontists have accepted and used tubes on molars for many years with great success. Passive self-ligation gives the clinician the opportunity to have a tube on every tooth, enhancing the performance of low-force high-tech archwires with low-friction sliding mechanics.

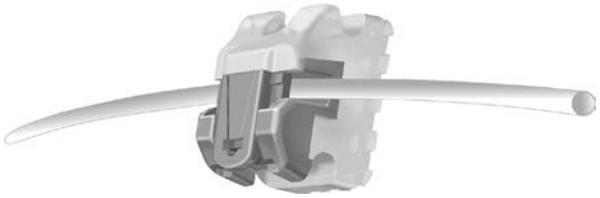


FIGURE 36-25 The passive Damon System 3 tube and bracket from Ormco.

The Damon System (Fig. 36-25) has been developed to align each phase of treatment with these natural force systems of normal growth and development. It became apparent that the application of biologically appropriate forces at each stage of treatment is essential for a long-term favorable outcome. As previously mentioned, the Damon appliance is commonly referred to as a bracket, but the term *tube* is a more useful description for this passive appliance. The appliance is a tube with tie-wings rather than a conventional bracket and, as such, has a static facial wall when the self-locking slide is closed. Vourdouris[5] compared the friction produced by passive and active conventional and self-locking brackets. With the use of a 0.019- × 0.025-inch stainless steel archwire, conventional brackets with elastomeric ties showed 400 (125.42 g/cm^2) to more than 600 (152.30 g/cm^2) times greater friction; steel ties showed 400 (97.07 g/cm^2) times greater friction; and active self-ligating brackets showed 216 (54.12 g/cm^2) times the friction of the *passive* self-ligating Damon tube (0.25 g/cm^2). With conventionally tied fixed appliances and active self-ligating brackets, the archwire is totally seated against the base of the bracket slot during some or all phases of treatment. The resulting binding and friction make closing spaces, leveling, archform changes, and closing of open bites, as well as the finishing, settling, and detailing of the occlusion, far more challenging. Passive self-ligation maintains play in the mechanical system during all stages of treatment. Figure 36-26 illustrates the degree of freedom of the initial round wire in a Damon tube. The small wire-to-lumen ratio is essential to leveling, aligning, and arch adaptation in a friction-free environment but *initially* limits rotational control. Rotational control is primarily achieved with the second, larger archwire, but the lack of friction and binding enables the overall correction to proceed faster. The short treatment times in the illustrative cases typify results obtained with consistent, biologically sensible forces. Figure 36-27 demonstrates that accurate tooth positioning, including rotational control, requires a full-depth archwire to fill the slot in a passive system. The play in the system when using 0.014- × 0.025-inch, 0.016- × 0.025-inch, 0.017- × 0.025-inch, and 0.019- × 0.025-inch rectangular wires in a 0.022-inch slot provides the needed rotational control and allows for 7 to 10 degrees of torque play in both directions with excellent rotational control later in the wire progression.

One must think of this new therapy as a complete system and not simply a new passive self-ligating bracket. With this new low-force, low-friction technology (tubes on each tooth instead of brackets with ligature ties and elastomerics), the clinician can apply very light forces that closely match normal biologic tooth-moving forces. *These properly attuned forces, not the clinician, allow the muscles of the face and tongue, bone, and soft tissue to determine the archform.*

An examination of what is known about the fragility of the vascular system and the bony architecture that support the dental arches is a necessary consideration for any orthodontic treatment.

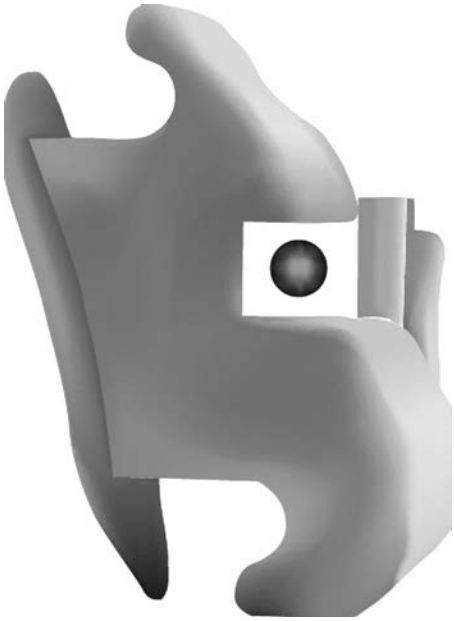


FIGURE 36-26 The small wire-to-lumen ratio of the Damon tube is essential to leveling, aligning, and arch adaptation in a friction-free environment.

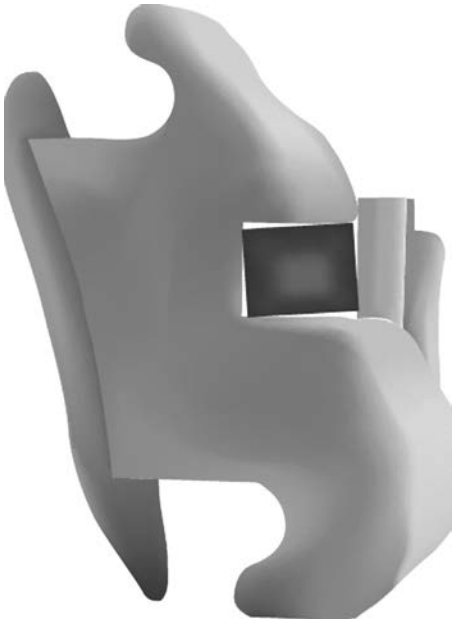


FIGURE 36-27 With larger archwires, the Damon conduit provides excellent torque and rotational control without lodging the system.

Case for Using Extremely Light Forces in Passive Tubes

Using scanning electron microscopy and the corrosion resin cast method, Brazilian researchers M.C. Kronka, I. Watanabe, and M.C. Pereira da Silva[6] have published images of the angioarchitecture of the palatine gingival in young rabbits. These photographs clearly illustrate the fragility of the vascular architecture entering the alveolar bone on one side of the periodontal space (Fig. 36-28, *A*) and the lacelike network on the root of the tooth on the other side (Fig. 36-28, *B*). Figure 36-28, *C*, illustrates an even closer view of the vascular network of the

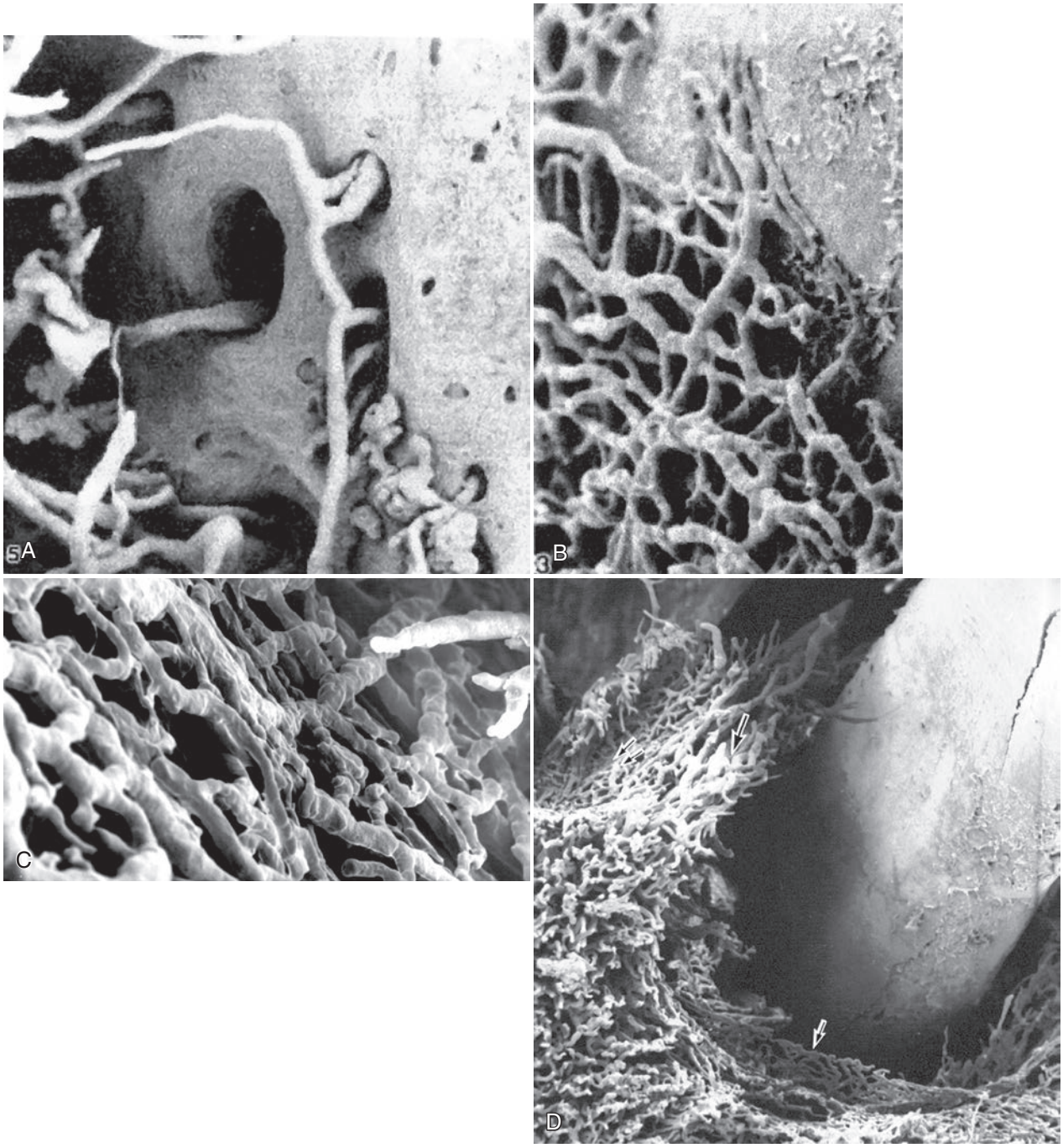


FIGURE 36-28 A, Lacelike vascular architecture of the tooth of a young rabbit reveals its fragility and susceptibility to destruction by high forces. B, Blood vessels are shown entering the alveolar bone. C, Image is a close up of the network. D, Tooth, sulcus, and vascular network are visualized.

periodontal ligament, with Figure 36-28, *D*, showing the tooth, sulcus, and vascular network. Such images should challenge all orthodontists to reevaluate their clinical force systems. Clinical mechanics and force systems are too often based on patient tolerance rather than on their impact on the periodontium and cellular biologic structure. Orthodontists continually ask the question, "What is the largest archwire I can get in?" If teeth do not move, then often orthodontists simply apply more force, yet they recognize the detrimental effects of excessive force on vascularity of the dentoalveolar complex. The cellular structure of orthodontically induced tooth movement is complex, and researchers are still challenged to unravel all the cellular mechanisms in play.

The modeling events of tooth movement are commonly referred to as areas of compression and tension within the periodontal ligament. Simply put, membranous bone growth is appositional with alveolar bone being resorbed on the moving front and new bone being laid down on the trailing side. Just more than 100 years ago, Carl Sandstedt[7] discovered the different responses of tissue to heavy and light orthodontic forces. He determined that on the tension side of the tooth, the responses to strong and weak orthodontic forces were

similar. Bone deposition occurred with spicules forming along the direction of the strained periodontal fibers. The old bone was unchanged in appearance, whereas the new bone growth was easy to recognize. Responses on the pressure side of the tooth varied significantly, however, between light and heavy forces. No root resorption occurred with light forces because new bone was equally resorbed along the entire socket surface. The response to heavy forces was notably different, which led Sandstedt to coin the often-heard phrase "undermining resorption." The underlying bone was not resorbed in areas where the periodontal ligament was overcompressed. Rather, active resorption of bone and tooth root took place in the still vital areas surrounding the damaged membrane. Sandstedt's findings have been well recognized in academia and in the literature but have been widely ignored in the clinic for more than a century.

In 1932, A.M. Schwarz[8] found that very light forces of 20 to 26 g/cm^2, the same pressure in the capillaries of the periodontal ligaments, afforded safe and effective tooth movement. The obvious conclusion is to not exceed the outward pressure of the blood vessel if one does not wish to collapse it. Anything greater strangles the capillaries and generates necrotic tissue at the pressure sites.

Perhaps the greatest contributions to understanding the mechanisms of structural changes and tissue behavior caused by excessive orthodontic forces were produced by K. Reitan.[9-13] His publications from the late 1940s through the early 1970s revealed the strangulation of periodontal ligament capillaries, damage to the tissues, and resulting necrosis of the periodontal ligament and root resorption caused by conventional orthodontic forces.

In 1973, P. Rygh[14,15] summed up the consequences of the conventional orthodontics (high force) of the day: With conventional forces, this destructive process occurs at each appointment, and the wire is activated through a distance wider than the periodontal ligament space. At each wire change, teeth slam against the wall of the socket, and the intricate network of blood vessels is crushed yet again. Each time this trauma occurs, it takes weeks for the periodontal ligament to revascularize at the cellular level.

In 1992, Tuncay and colleagues[16] observed that oxygen is the trigger mechanism for remodeling the periodontium. If vascularity is interrupted in the periodontal space between bone and teeth, then oxygen is no longer available and cellular activity is slowed or stopped. In 1993, W.R. Proffit and H.W. Fields[17] advocated that, "Optimum force levels for orthodontic tooth movement should be just high enough to stimulate cellular activity without completely occluding blood vessels in the periodontal ligament." The author has named this threshold force the *biozone* or *optimal force zone*. Proffit[9] also stated, "If the applied force is great enough to totally occlude blood vessels and cut off the blood supply, a hyalinized avascular necrotic area is formed. This area must revascularize before teeth start to move."

Regarding Rygh's and Reitan's findings, Bob Borkowski[18] stated, "This healing time is, in essence, a big timeout in the progress of treatment. Understanding this phenomenon makes me wonder if this healing time isn't the reason why, in conventional mechanics, adult treatment takes longer than adolescent treatment." Borkowski postulated that this difference is due to children healing from their wounds faster than adults.

The issue of oxygen supply raises questions about what is happening with the use of light, high-tech archwires in a passive tube where play between the wire and the lumen size of the tubes is considerable. The response to very light, high-tech wire used in low-friction, passive tubes is different from the use of the same high-tech wires in conventionally ligated or active self-ligating appliances. Certainly the adaptation phenomenon is occurring at the cellular level, and perhaps this phenomenon has something to do with oxygen supply. At this stage, the phenomenon is not clear. What orthodontists can verify is that the response is similar to that seen in patients treated with the Fränkel appliance.

Patients such as CB (see Case Study 36-1) have always prompted the author to question the cause of crowding. Given the cause, orthodontists can better assess what methods to use to stimulate the best possible resolution. As in most such cases, the shape and size of the body of the mandible is normal, but the alveolar process is severely constricted. The author's opinion is that this condition often results from lips and muscles of the face being out of balance with the tongue. The tongue is passive or sitting too low in its cavity, and without the exertion of the strong outward force of the tongue on the arches, the muscles of the face dominate, thus constricting the lower arch and allowing the maxilla to collapse inward. Fränkel[19] capably demonstrated that keeping the buccal tissues away from the dental arches fosters a redistribution of forces between the lips and muscles of the face and the tongue, generating greater arch length. Fränkel used buccal shields to increase arch width. The absence of cheek forces enabled the tongue to generate buccal drift of posterior teeth. Long-term studies on patients undergoing treatment with the Fränkel appliance have proved the results to be stable.

The Damon System, when properly applied, provides patients with the physiologic improvements possible with the Fränkel appliance and other muscular imbalance corrective appliances, plus the precision finishing attainable with preadjusted edgewise appliances. Because form follows function, the author considers many malocclusions to be *functional abnormalities*. Light wires acting in an almost friction-free environment in the Damon tube appear to be able to correct the functional imbalance and allow the alveolar process to create a new, individualized archform. The author's explanation for this phenomenon is that the friction and binding of the archwire in conventionally ligated appliances (and *active* self-ligating appliances) preclude functional adaptation. Orthodontists recognize the power of sliding mechanics to close space, with buccal tubes being essential to this process. Sliding mechanics also offers the most efficient means of aligning and leveling teeth in the early phases of treatment. Clinicians often use edgewise wires to unravel and level malposed teeth. Minimal play exists between the archwire and the bracket slot, and the wire becomes bound up in a tightly ligated system. In a nearly friction-free environment with the appropriate amount of play between the archwires (a 0.014-inch round nickel-titanium wire is essential to this process) and the passive bracket tube, teeth begin to level and unravel, and the arch begins to widen in the transverse. Alan Bagden[20] suggests that this beginning adaptation "wakes up the tongue." This phenomenon allows the tongue to seek a higher level in the oral cavity, thus instigating a new force interplay among the tongue, cheeks, and lips. With this gentle force of a small round wire in large fixed passive tubes, transverse arch adaptation occurs

similar to the Fränkel effect. Although the technique is too new to substantiate improved long-term stability, results to date have been encouraging. Apparently, moving teeth with high-tech archwires in passive self-locking tubes generates a healthy alveolar bone and periodontium support that is not as susceptible to relapse as the typical repositioning of the dentition with more traumatic forces.

After treating several thousand patients with this low-force tube system, the author noted the significant variation in the rate of tooth movement from patient to patient. In most patients the rapid rate of tooth movement with such light forces is impressive. Using high-force conventional mechanics for years, the author found the response among patients to be approximately the same, with the greatest variable being between adults and children. Now the slowest and most challenging cases are in those who have had previous extractions and conventional higher-force orthodontics. The alveolar bone appears to have been altered, which affects subsequent treatment planning and length of treatment.

COMPUTED TOMOGRAPHIC SCANS DEMONSTRATE HEALTHY BONE STRUCTURE AFTER TREATMENT WITH THE LOW-FORCE DAMON TUBE SYSTEM

Over the years, the author has observed that the kindest movers of teeth are normally functioning muscles of the face and tongue. Orthodontists have recognized the Fränkel effect in first-phase treatment using the 2 × 4 method (with fixed appliances only on the four anterior teeth and molars), which alters the impact of the facial musculature, allowing the premolars to move laterally. The author's experience has been that over several months, premolar width usually increases 6 to 8 mm with little tipping. This same type of bodily movement of teeth with extremely light, high-tech archwires in the passive Damon tube appliance has been verified on CT scans of patients who experienced posterior width change. The scans also document that the bone development tracked with the teeth movement and arch adaptation. Rather than using high-force expansion, this research suggests that trying to match this *natural force* adaptation with all clinical mechanics makes sense.

Until now, orthodontic research has been primarily limited to clinical observations and long-term retention studies, with conventional radiographs and periodontal findings serving as the evaluation technologies. With the technical advancements of three-dimensional CT scans for the midface, orthodontists now have the means to determine the posttreatment health of the alveolar bone. The author has spent hundreds of hours evaluating CT scans taken on 13 patients treated with low-force mechanics in a passive tube system where functional adaptation accommodated teeth in a crowded arch using nonextraction therapy. These findings are encouraging and should challenge clinicians everywhere to reevaluate their management of clinical forces and stimulate them to consider a paradigm shift of their treatment planning. These CT scans were taken on young and adult patients, from those just debonded to one patient in retention for longer than 5 years.

Most clinicians have believed that with clinical mechanics, at best, teeth can be tipped only in the transverse, not bodily moved as well. These CT scans conclusively show that alveolar bone can be altered and reshaped with low clinical forces. This low-force and passive tube technology and the clinical research that substantiates it call for changes in treatment planning and the mechanics of tooth movement. Other findings from the CT scans demonstrate how thin the alveolar bone is on the labial, buccal, and lingual aspects of the teeth that orthodontists are trying to move. These CT scans make it apparent that every effort should be made to take great care of the thin cortical plate. In viewing the scans of patients in long-term retention, the reader will note that the architecture and thickness of the alveolar bone appears to improve over time after low-force orthodontics. Recognizing that low-force orthodontics can have a *positive* impact on the periodontium is encouraging. The reason bone may regenerate in some areas of the mouth in periodontally challenged patients and not in others is not understood. The periodontist who conducted connective tissue grafts on patients during or after treatment with low-force and passive tube technology commented on observing an improved vascular environment. After having evaluated CT scans of several crowded cases in retention, the author recommends that orthodontists make every effort for early tooth guidance in children to keep permanent teeth from erupting through the labial, lingual, and buccal cortical plates. Obviously, teeth erupting in the center of the alveolar ridge usually have far improved bone on the labial and buccal aspects.

CASE PRESENTATIONS AND CLINICAL ANALYSES

The following cases graphically demonstrate how facial treatment planning should and can be the primary focus. The dentitions of these patients are obviously crowded. The dentitions beg the question, "Why are these patients so crowded?" Are the bones of the midface and body of the mandible smaller than normal, or has there been an adaptation of the alveolar process because of abnormal muscle forces affecting arch development? Most orthodontists have been trained to evaluate the patients only from head films. As mentioned in the case of CB, early in the author's career, extraction therapy would have been used to treat CB, despite the body of his mandible being exactly the same size as the author's, which is an exceedingly wide archform. To see that more and more orthodontists around the world realizing the impact that treatment decisions have on the maturing profile, for good or for ill, is exciting. With improvements in technique and technology, orthodontists now can plan and execute treatment by evaluating its long-term implications on profile (lateral view) and arch width and facial support (frontal view). In the author's estimation, designing treatment mechanics that *do not overpower the biologic system in any phase of treatment is critical*, and doing so requires the use of low-force archwires in a passive tube. In reviewing these case studies, the reader should note that great care has been given to using treatment forces that are "just high enough to stimulate cellular activity without overpowering the periodontium and orofacial muscular complex."[17] *If orthodontists maintain force levels in the optimal force zone, the alveolar bone and tissue can be moved with teeth*. With the Damon System, the art of clinical orthodontics is to match treatment mechanics with the natural low-force systems of the body and to learn how to read and react to how the alveolar process and muscles adapt.

CASE STUDY 36-2 Treatment with Nonextraction Therapy

Case 2 (AH) provides another example of how the transverse dimension and severe upper anterior crowding can be satisfactorily treated with simple low-force and tube mechanics. This case is a good example of facially driven treatment achieved by converting anterior crowding into posterior adaptation of bone, muscle, and soft tissues. Arch length gain is illustrated along with the correction of the Class II dentition. At 16 years, 5 months of age, AH had a Class II dental bilateral posterior crossbite and crowded maxillary arch. She was midface deficient but with a good profile and an aesthetically pleasing obtuse nasolabial angle.

The treatment objectives were as follows:

1. Gain maxillary arch width to support the midface.
2. Correct midface deficiency.
3. Maintain nasolabial angle while improving the midface.
4. Develop an ideal tooth-to-lip relationship.
5. Transpose the malpositioned upper right canine distolingually without damaging the root of the lateral directly lingual to the canine root.

Pretreatment records (Fig. 36-29) demonstrate the substantial Class II arch relationship and severely crowded maxillary dentition. Only low-force Damon System mechanics were used throughout treatment. No palatal expanders, distalizers, headgear, implants, lingual holding arches, or removable appliances were used. Using low-force mechanics, the result was far superior patient comfort throughout treatment and improved bone and tissue at the end of treatment.

Tom Pitts, a former member of Robert Ricketts' Bioprogressive Study Club, reported that in a study of the relationship between tooth-to-tooth gain in arch width and overall increase in arch length, Ricketts found specific

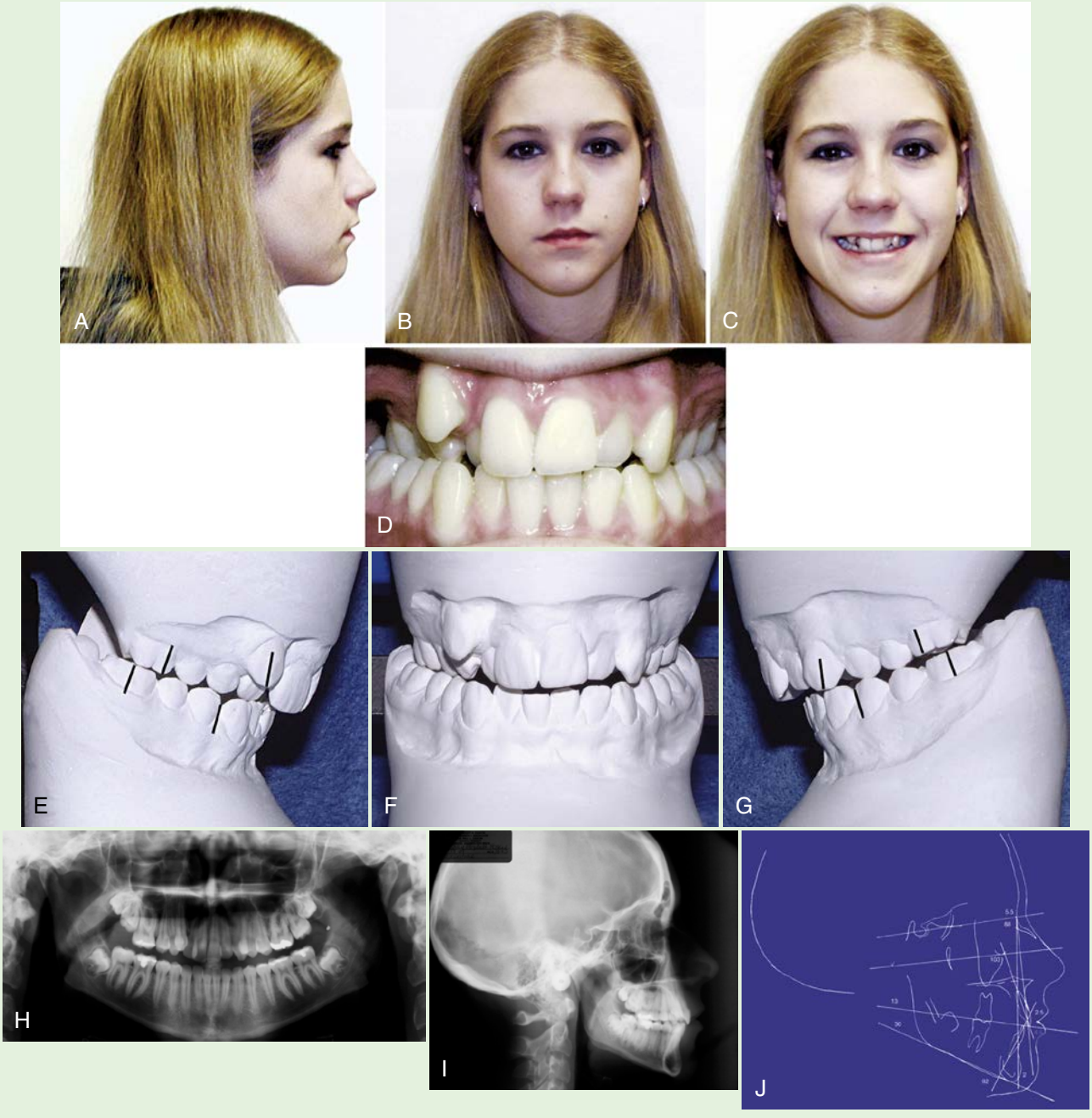


FIGURE 36-29 **A-J,** Pretreatment records for case 2 (AH, age 16 years, 5 months). Diagnosis: Class II with bilateral posterior crossbite and crowded maxillary arch and midface deficiency but good profile and obtuse nasolabial angle.

Continued

CASE STUDY 36-2 Treatment with Nonextraction Therapy—cont'd

ratios occurred with changing archform and orthodontic treatment.[21] This research indicates that a 1-mm gain in canine width, a 1.5-mm gain in first premolar width, a 2-mm gain in second premolar width, or a 4-mm gain in molar width each yields a 1-mm increase in arch length. The light-force Damon System maximizes the potential of each individual's anatomic and physiologic structure in delivering a natural archform. The system provides nature a second chance to correct inherent or habit-generated deficiencies. The crowding in this case indicates a tongue trapped low in the mouth and unable to provide the necessary facial thrust for normal maxillary arch formation (Fig. 36-30). The Damon System helps correct nature's muscle imbalances with gentle forces. The long intervals between appointments allow the light archwires to work to their full potential in the almost friction- and binding-free tubes.

Posttreatment records (Fig. 36-31) were taken after 22 months, 3 weeks of treatment in 11 appointments. Figure 36-32 shows that bodily tooth movement along with minimal corrective tipping created a 9- to 12-mm upper arch width adaptation and tremendous palatal change. The patient was happy to report that she could now breathe through her nose and sleep with her mouth closed. The intraoral photographs reveal healthy bone and tissue with significant alveolar improvement. Figure 36-33 compares the plaster models of the upper arches taken initially and the day of debonding with the CT scans taken 7 months after treatment. Initial and final comparisons of the plaster models clearly reveal the posterior arch widening, anterior unscrambling, and arch shape improvements. CT scans demonstrate healthy alveolar bone in all areas. Unfettered light wires worked to develop what nature and a trapped, underactive tongue failed to produce; that is, an appropriate archform for AH and a pleasing final result.

Archwires adapted to the initial and final upper archforms (Fig. 36-34) make apparent what happened to the anterior crowding. Adapting the final archwire to the initial archform demonstrates an arch length gain of 13 mm, yet the measurement distal of the first molars to the labial of the incisors at the beginning and end of treatment is only 1 mm, which explains why the existing attractive profile was maintained while the midface deficiency was improved. These measurements indicate that the anterior crowding was accommodated by transverse posterior adaptation. An observation in other patients is that a change in the posterior arch width of only 1 or 2 mm can have a favorable impact on the midface. The horizontal CT scans (Fig. 36-35) illustrate that the pleasant midface change is not simply a result of the expansion of the dental arch but the entire alveolar process high in the vestibule, which provides improved facial support.

Initial and final comparisons of the plaster models of the lower arch demonstrate the minimal change (0 to 2.5 mm) in posterior arch width (Fig. 36-36). The horizontal CT scan taken 7 months in retention (Fig. 36-37) demonstrates how the roots of the lower teeth sit in the cortical plate. Considering that the change in the lower arch width or anteroposterior tooth movement was small, it is interesting to note in the CT scans that so much of the tooth structure is sitting in cortical bone with low vascularity, again supporting Rygh's notion of low-force mechanics.

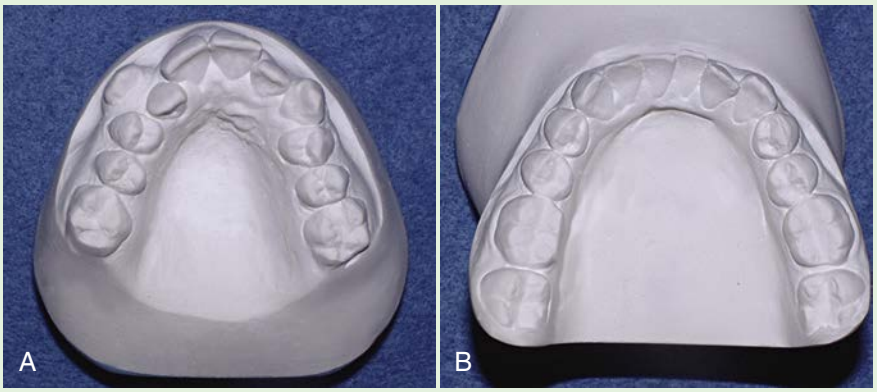


FIGURE 36-30 A, B, The trapped tongue is unable to normalize posterior maxillary arch width. The lower arch shape indicates low tongue posture.

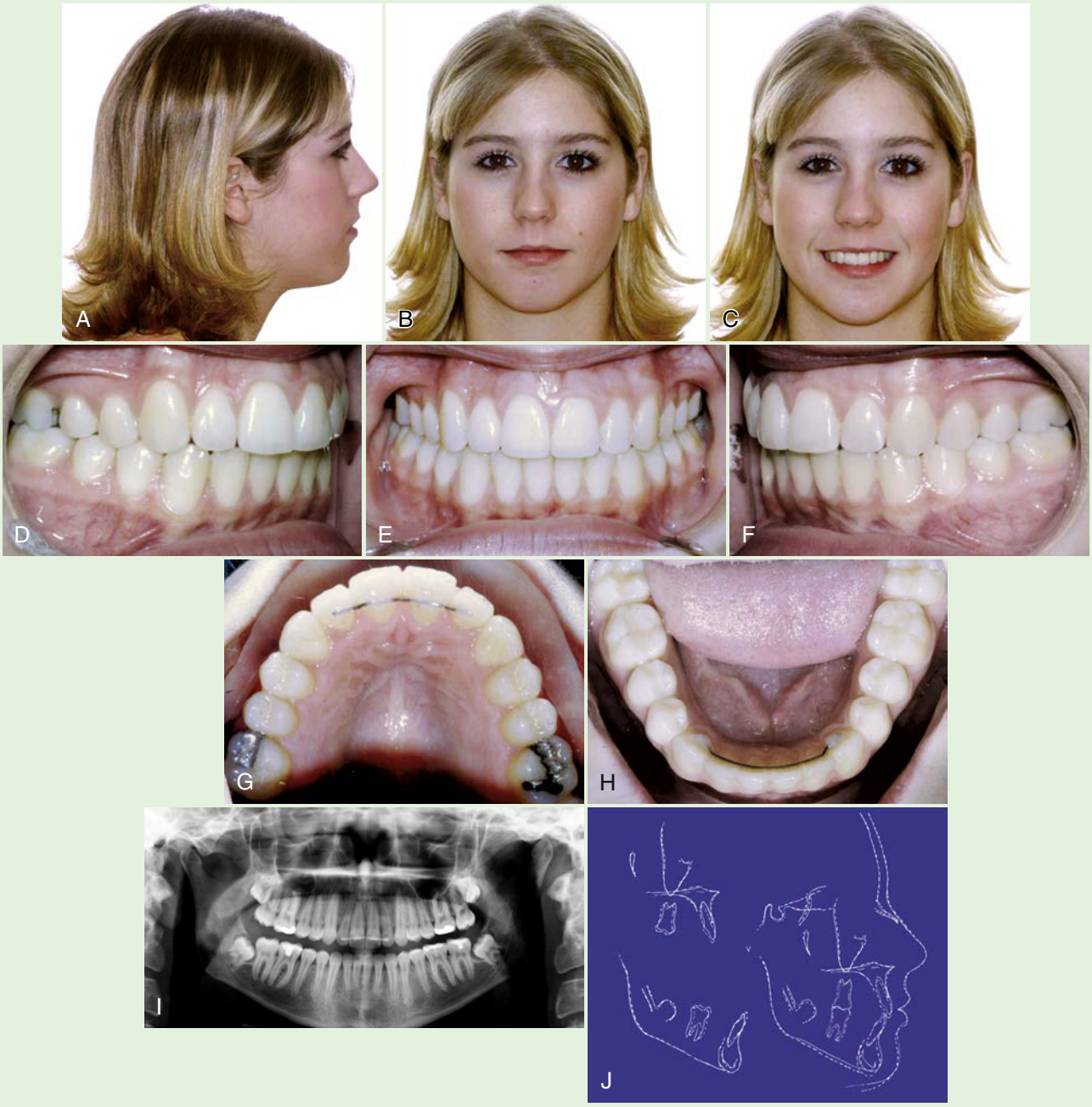


FIGURE 36-31 **A-J,** Posttreatment records of AH. Total treatment time: 22 months, 3 weeks with 11 appointments. Midfacial aesthetics improved while maintaining favorable profile. Minimal tipping with 9- to 12-mm upper posterior adaptation and improved tongue position are evident.

FIGURE 36-32 The pretreatment and posttreatment plaster models of AH demonstrate bodily tooth movement and tremendous palatal change with minimal corrective tipping that resulted in a significant improvement of articulation and easier breathing.

Continued

CASE STUDY 36-2 **Treatment with Nonextraction Therapy—cont'd**

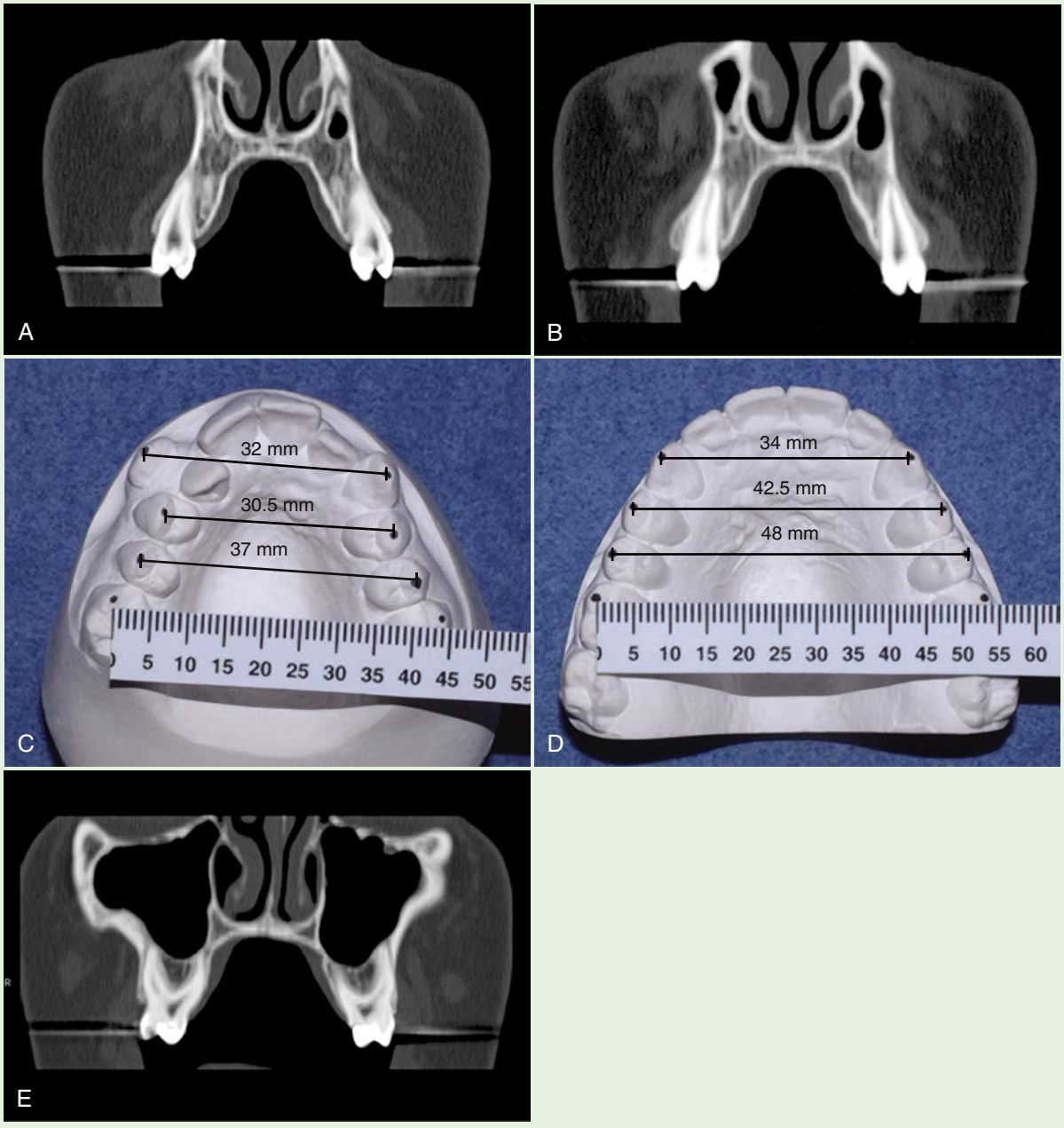


FIGURE 36-33 Comparisons of pretreatment and posttreatment upper arch plaster models for AH with computed tomographic scans. Initial and final models of upper arch show a 2-mm change in canine width, 12-mm change in first premolar width, 11-mm change in second premolar width, and 9-mm change in first molar width. Vertical scans taken 7 months into retention show healthy alveolar bone. **A,** Scan of upper first premolars at 7 months in retention. **B,** Scan of upper second premolars at 7 months in retention. **C,** Pretreatment. Canines: 32 mm; first premolars: 30.5 mm; second premolars: 37 mm; first molars: 44 mm. **D,** Posttreatment. Canines: 34 mm; first premolars: 42.5 mm; second premolars: 48 mm; first molars: 53 mm. **E,** Scan of upper first molars at 7 months in retention.

CASE STUDY 36-2 Treatment with Nonextraction Therapy—cont'd

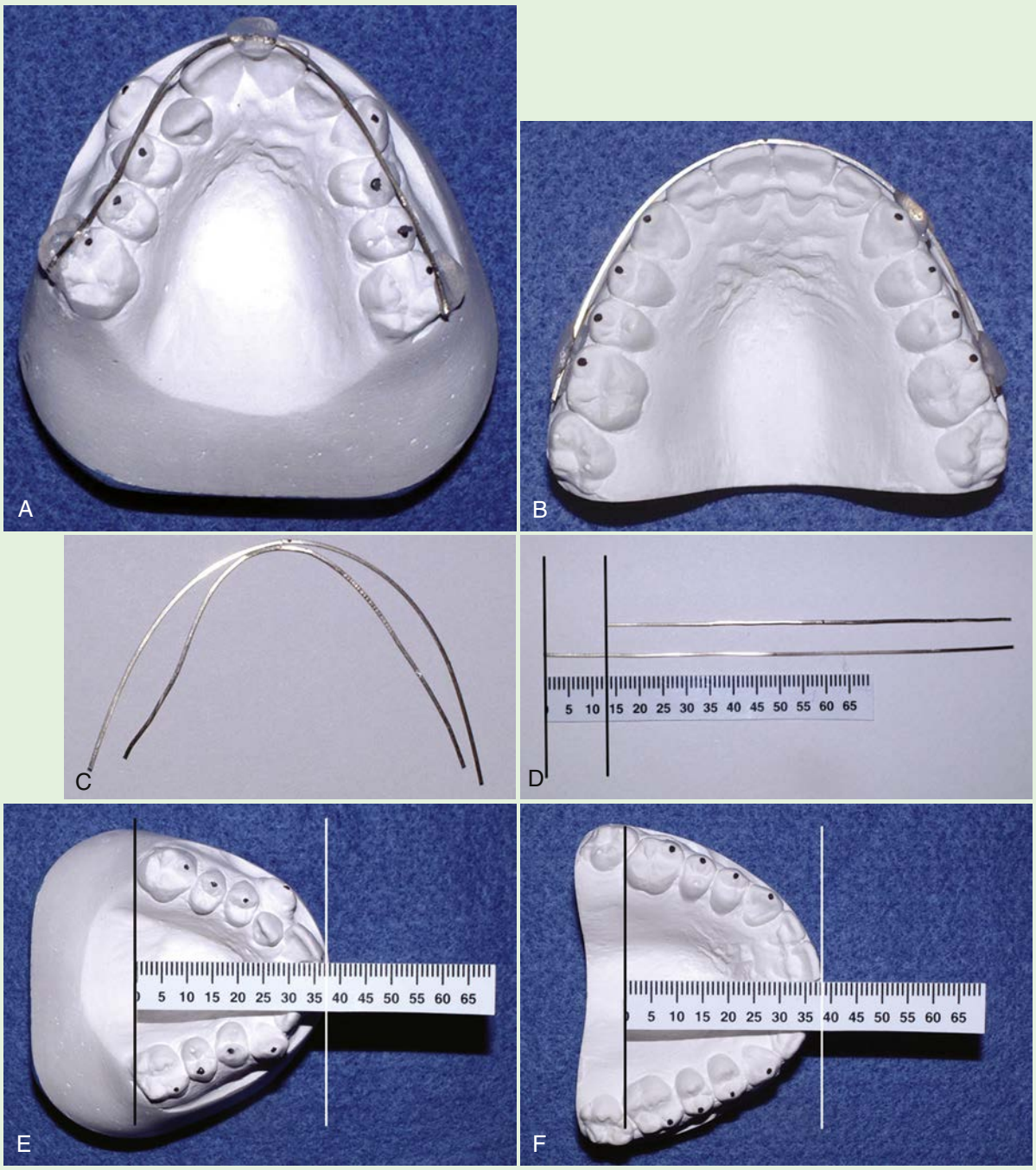


FIGURE 36-34 Adapting the final archwire to the initial archform demonstrates an arch length gain of 13 mm in AH, yet the measurement distal of the first molars to the labial aspect of the incisors at the beginning and end of treatment is only 1 mm, which explains why the existing attractive profile was maintained while the midface deficiency was improved. These measurements indicate that the anterior crowding was accommodated by transverse posterior adaptation. **A,** Final archwire adapted to the initial archform. **B,** Final archwire adapted to the final archform. **C,** Comparison of initial and final archwires. **D,** Comparison of initial and final archwires lengths shows 13 mm of change. **E,** Measurement from distal aspect of first molars to labial aspect of incisors of initial archform is 37 mm. **F,** Measurement from distal aspect of first molars to labial aspect of incisors of final archform is 38 mm.

Continued

CASE STUDY 36-2 Treatment with Nonextraction Therapy—cont'd

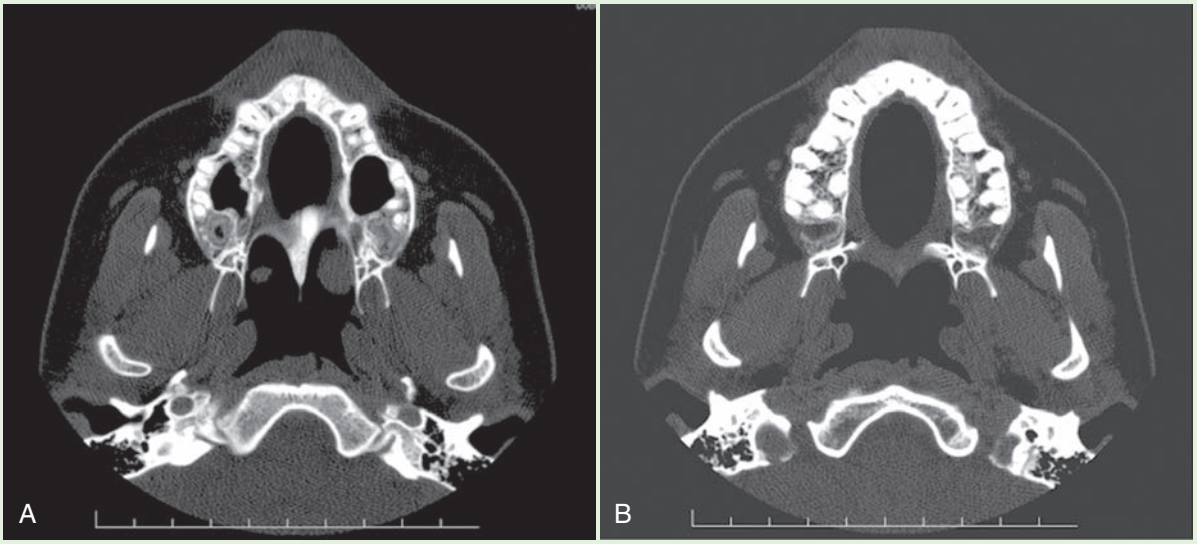


FIGURE 36-35 A, B, The horizontal computed tomographic scans of AH's upper arch illustrate that the pleasant midface change is not simply a result of the expansion of the dental arch, but it is also the result of the entire alveolar process high in the vestibule, which provides better facial support.

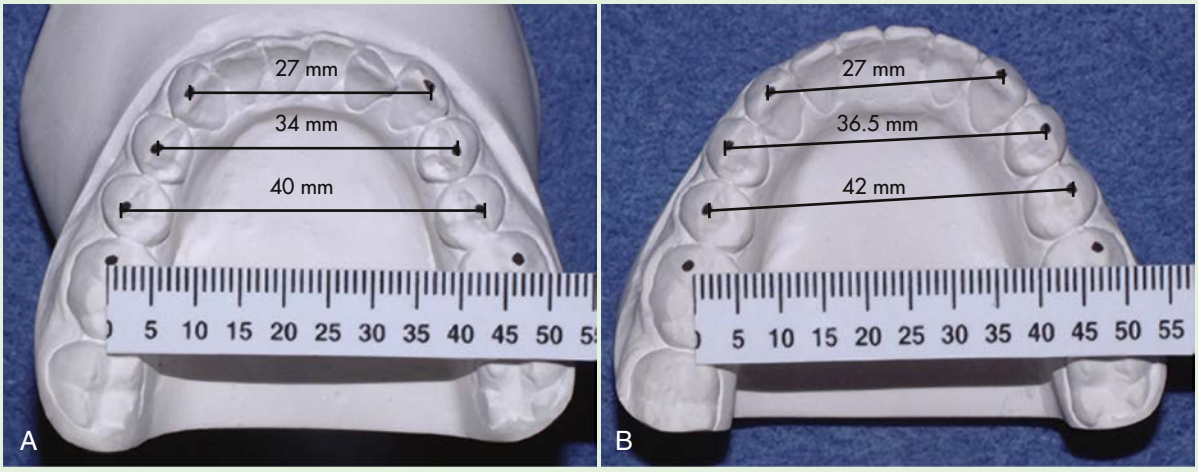


FIGURE 36-36 Comparisons of pretreatment and posttreatment lower arch plaster models for AH. Pretreatment and posttreatment models of the lower arch show no change in canine width, 2.5-mm change in first premolar width, 2-mm change in second premolar width, and 0.5-mm change in first molar width. **A,** Pretreatment. Canines: 27 mm; first premolars: 34 mm; second premolars: 40 mm; first molars: 46.5 mm. **B,** Posttreatment. Canines: 27 mm; first premolars: 36.5 mm; second premolars: 42 mm; first molars: 47 mm.

CASE STUDY 36-2 Treatment with Nonextraction Therapy—cont'd

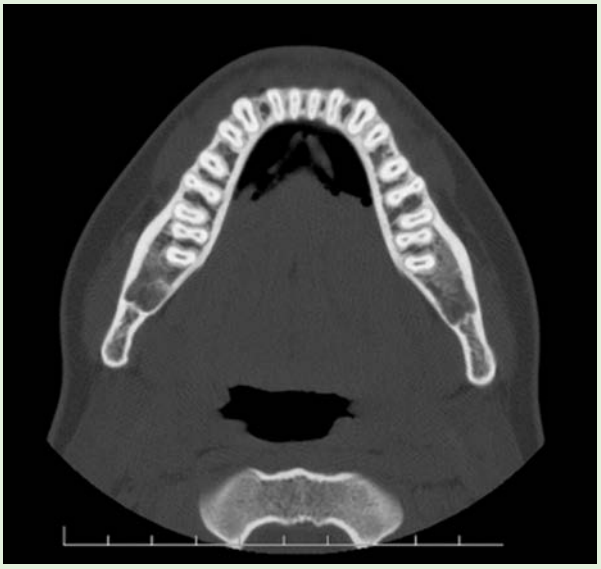


FIGURE 36-37 Horizontal computed tomographic scan of AH's lower arch taken at 7 months into retention shows healthy labial and buccal bone with minimal change in tooth position. The fact that so much tooth structure sits in cortical bone with low vascularity supports Rygh's notion of low-force mechanics.

CASE STUDY 36-3 Treatment with Extraction Therapy

Despite orthodontists' desire to treat with nonextraction therapy, it should be strongly emphasized that certain types of cases warrant extraction therapy. The philosophy of the Damon System is not founded on the idea of nonextraction therapy at any cost. The intent of the system is to achieve pleasing facial symmetry with a long-term healthy periodontium. Bimaxillary protrusive cases with full lip posture require extractions. In addition, on rare occasions, patients may not have the lip competence to keep the dentition from flaring forward with nonextraction therapy because the system heavily counts on the lip bumper or headgear effect. This lack of muscle competence must be recognized early in treatment, and extractions are recommended to avoid round tripping of the anterior teeth.

Case 3 (KH) was selected to demonstrate how the face determines treatment planning and that there is a time to extract. KH was 9 years, 8 months of age when she exhibited a Class II, Division 1 protrusive dentition with a severely deficient mandible, disproportionate lower facial height, and petite facial features. She had a small nose and a lack of lip seal. Her genetic potential was greatly limited from significant growth, the father's height being 5 feet 4 inches and the mother's height at 5 feet 1 inch. Both parents displayed refined features. Her dentition evaluation noted generalized spacing in the maxillary arch. Upper incisors were labially inclined, and the central incisors were large. She also had a significant overjet with a normal overbite.

The treatment objectives were as follows:

1. Achieve a Class I face, and then reevaluate protrusion and profile in an attempt to create facial balance and symmetry.
2. Anticipate the impact of treatment on the patient's maturing profile.
3. Leave the Herbst appliance on at least 16 to 18 months because of the severity of the Class II and the petite size of the slow-growing patient.
4. If necessary, extract four premolars before initiating phase 2 to achieve the best possible facial aesthetics.

Pretreatment records, which are shown in Figure 36-38, include tomograms taken before initiating phase 1 Herbst appliance treatment. In accord with the author's Herbst philosophy of slowly activating over a long time, the initial advancement was limited to 4 mm. The Herbst appliance treatment sequence (Box 36-2) reveals the small advancements that work along with the facial muscles in developing lateral upper arch expansion. Tomograms were taken again at the seventh appointment to check the condylar position. The Herbst appliance was removed on the eighth appointment, completing 18 months of functional Herbst appliance therapy. Progress records were taken at this time (Fig. 36-39). A comparison of the pre-Herbst and post-Herbst cephalometric tracings and facial photographs revealed an ANB (A point–nasion–B point) change of 3 degrees. Although the chin position improved with Herbst appliance treatment, this petite girl was still protrusive, and it continued to be a challenge for her to fit her lips over her teeth. In view of her genetic growth potential and the refined features of her parents, the girl was scheduled for four first premolar extractions.

The patient waited 5 months before starting phase 2 treatment. After the premolar extractions, the upper and lower arches were bonded 6 to 6 (second molars had not erupted). A special prescription of +7 degrees of torque was selected for the upper and lower canines, which is strongly recommended on all extraction cases to prevent lingual tipping of the clinical crowns during space closure. Maxillary and mandibular 0.014-inch Ni-Ti Align SE archwires were placed. Bonded treatment required 11 appointments and 21 months; the treatment sequence is shown in Box 36-3.

Figure 36-40 shows the placement of the 0.019- × 0.025-inch stainless steel archwires with Ni-Ti springs hooked on them distal to the first molars. The distal eyelets were bent 90 degrees before placing over the distal ends of the cut archwires. To avoid flaring the second molars to the buccal, springs were not attached to the second molar hooks. The preceding procedure should be followed in cases of minimum anchorage. In cases of maximum anchorage retraction, ligation of the first and second molars together while attaching the springs to the hooks of the first molars is recommended (Fig. 36-41).

KH demonstrates how simple mechanics work at their best in an unbound mechanical system with light application of force. Archwire posts evenly

Continued

CASE STUDY 36-3 **Treatment with Extraction Therapy—cont'd**

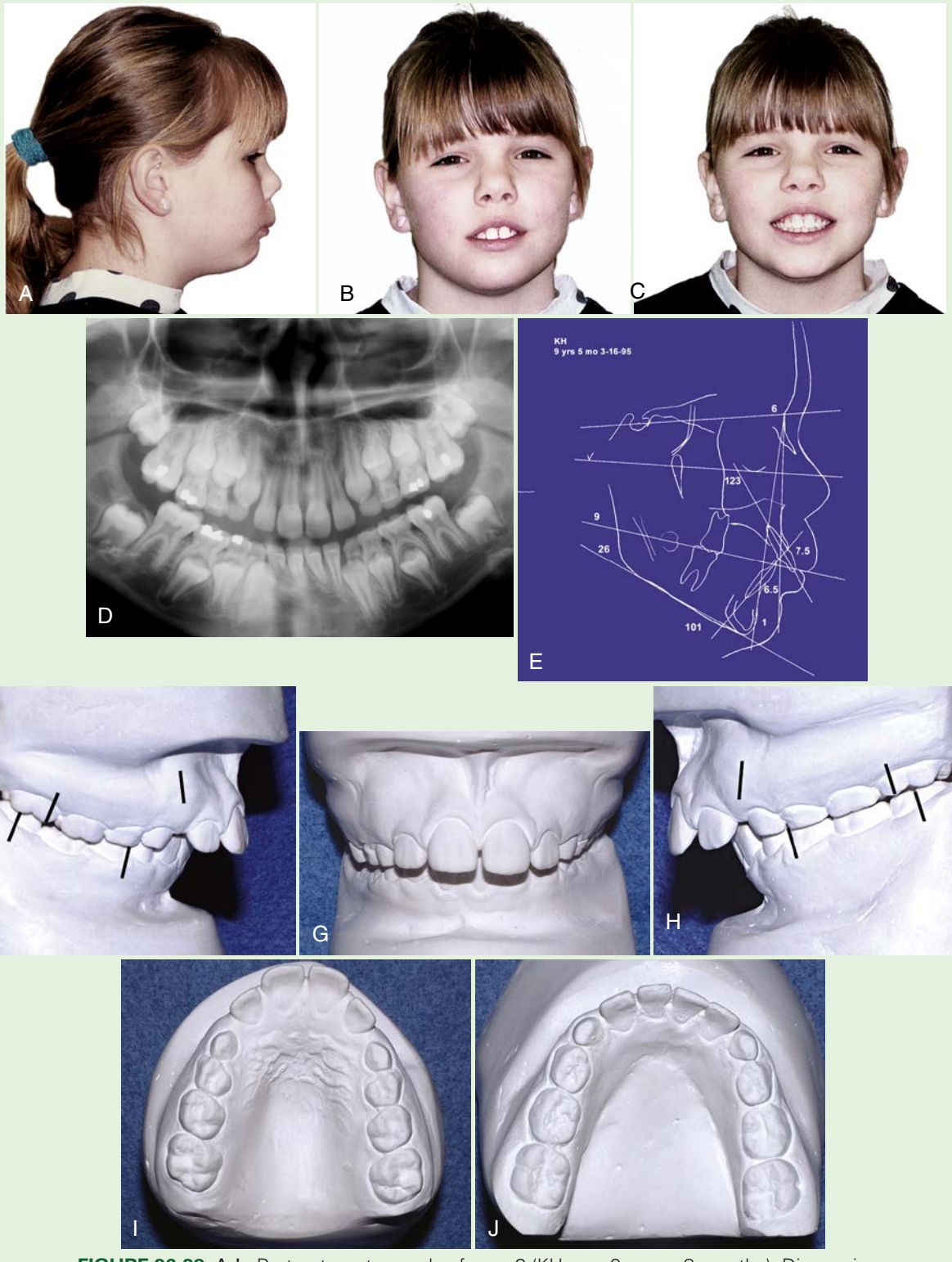


FIGURE 36-38 A-L, Pretreatment records of case 3 (KH, age 9 years, 8 months). Diagnosis: Class II, Division 1 bimaxillary protrusive with severely deficient mandible, disproportionate lower facial height, and petite facial features.

CASE STUDY 36-3 Treatment with Extraction Therapy—cont'd

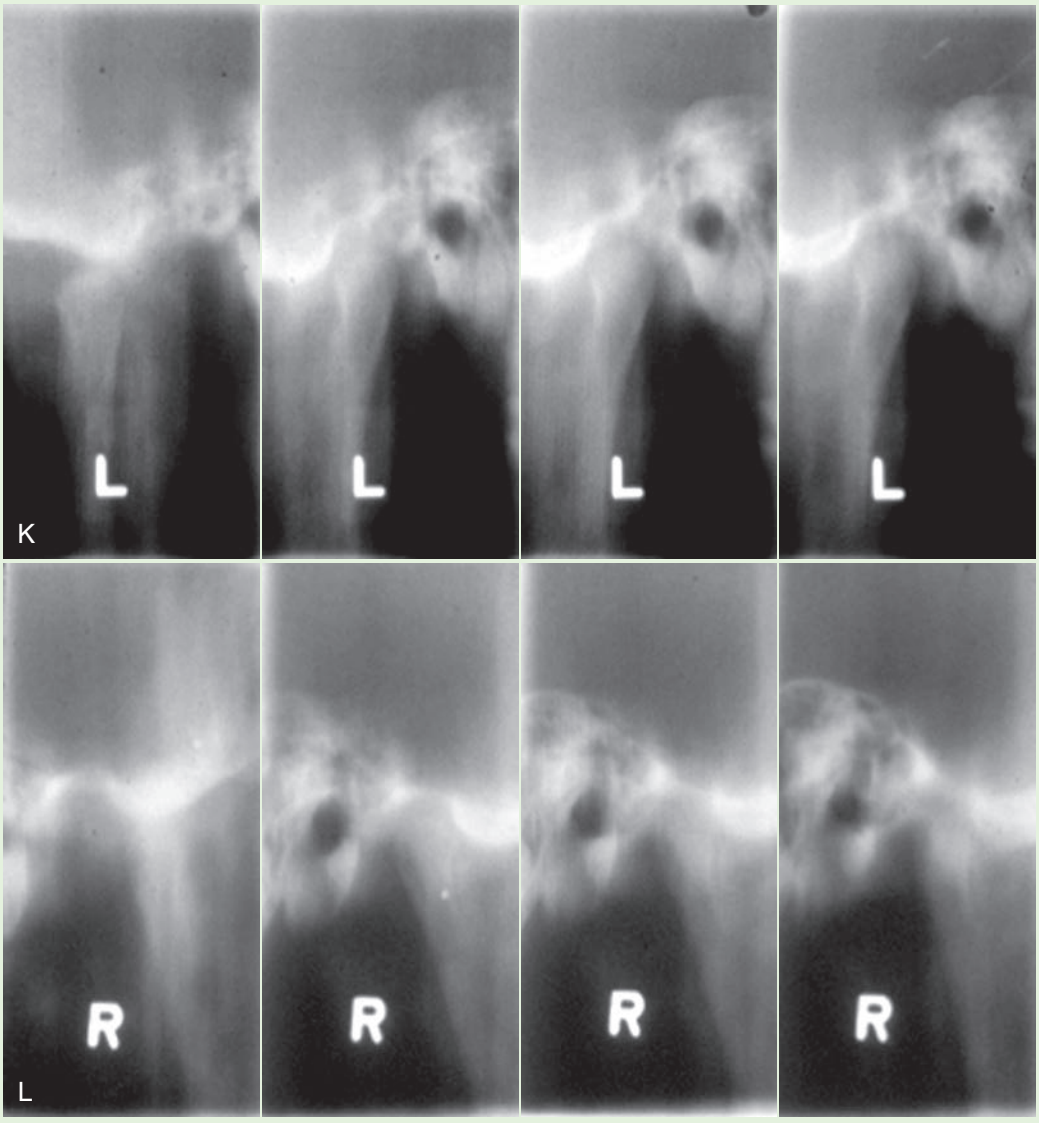


FIGURE 36-38, cont'd

BOX 36-2 Case 3 (KH) Treatment Sequence: Phase 1 Herbst Appliance

Start: Initial records, including tomograms, taken before beginning treatment.
First appointment: Placed Herbst appliance, and advanced 4 mm.
Second appointment at 2 months 2 weeks: Checked Herbst appliance.
Third appointment at 5 months: Advanced Herbst appliance 3 mm.
Fourth appointment at 7 months 2 weeks: Checked Herbst appliance.
Fifth appointment at 10 months 2 weeks: Checked Herbst appliance, and added 1-mm shim.
Sixth appointment at 13 months: Checked Herbst appliance.
Seventh appointment at 15 months: Checked Herbst appliance, and took tomograms.
Eighth appointment at 18 months: Removed Herbst appliance, took progress records, and scheduled four premolar extractions and full bonding (see Fig. 36-39).

Continued

CASE STUDY 36-3 **Treatment with Extraction Therapy—cont'd**

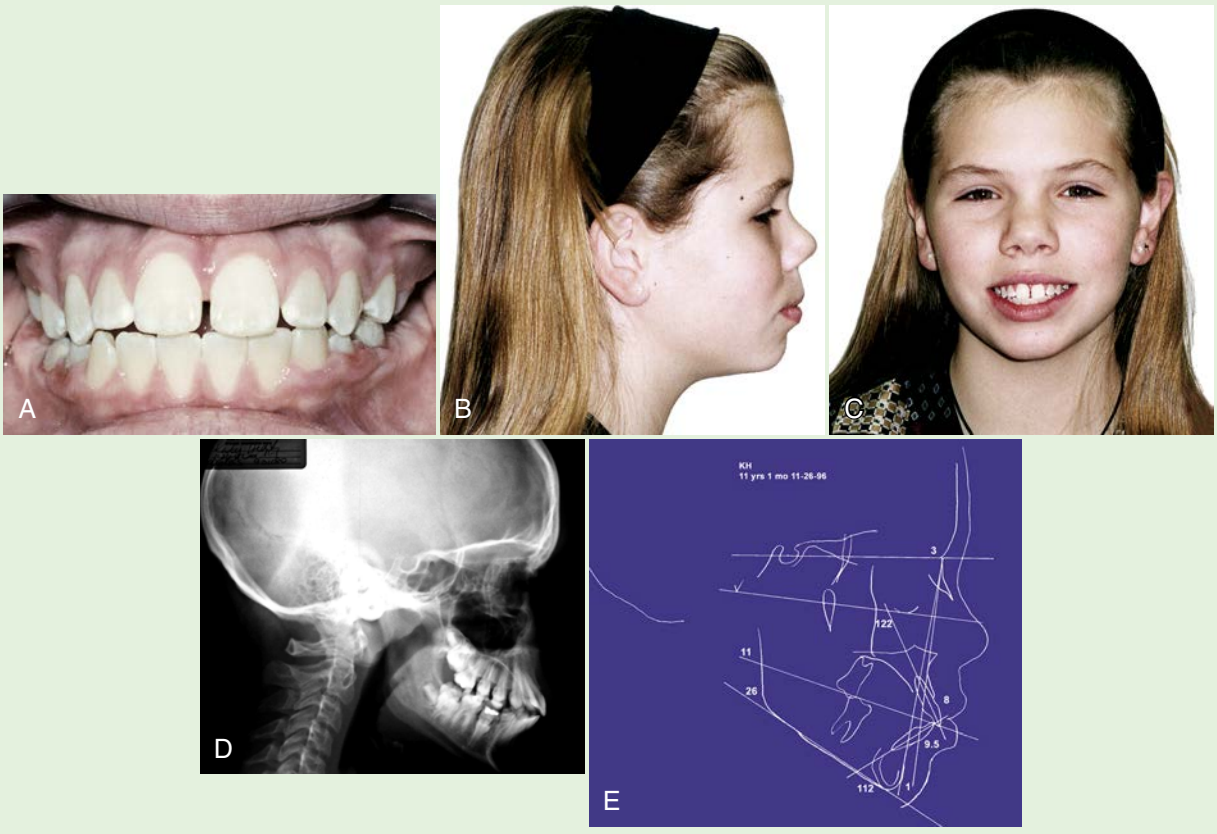


FIGURE 36-39 A-G, Phase 1 posttreatment records of KH with Herbst appliance (phase 1 treatment time: 18 months with eight appointments).

CASE STUDY 36-3 Treatment with Extraction Therapy—cont'd

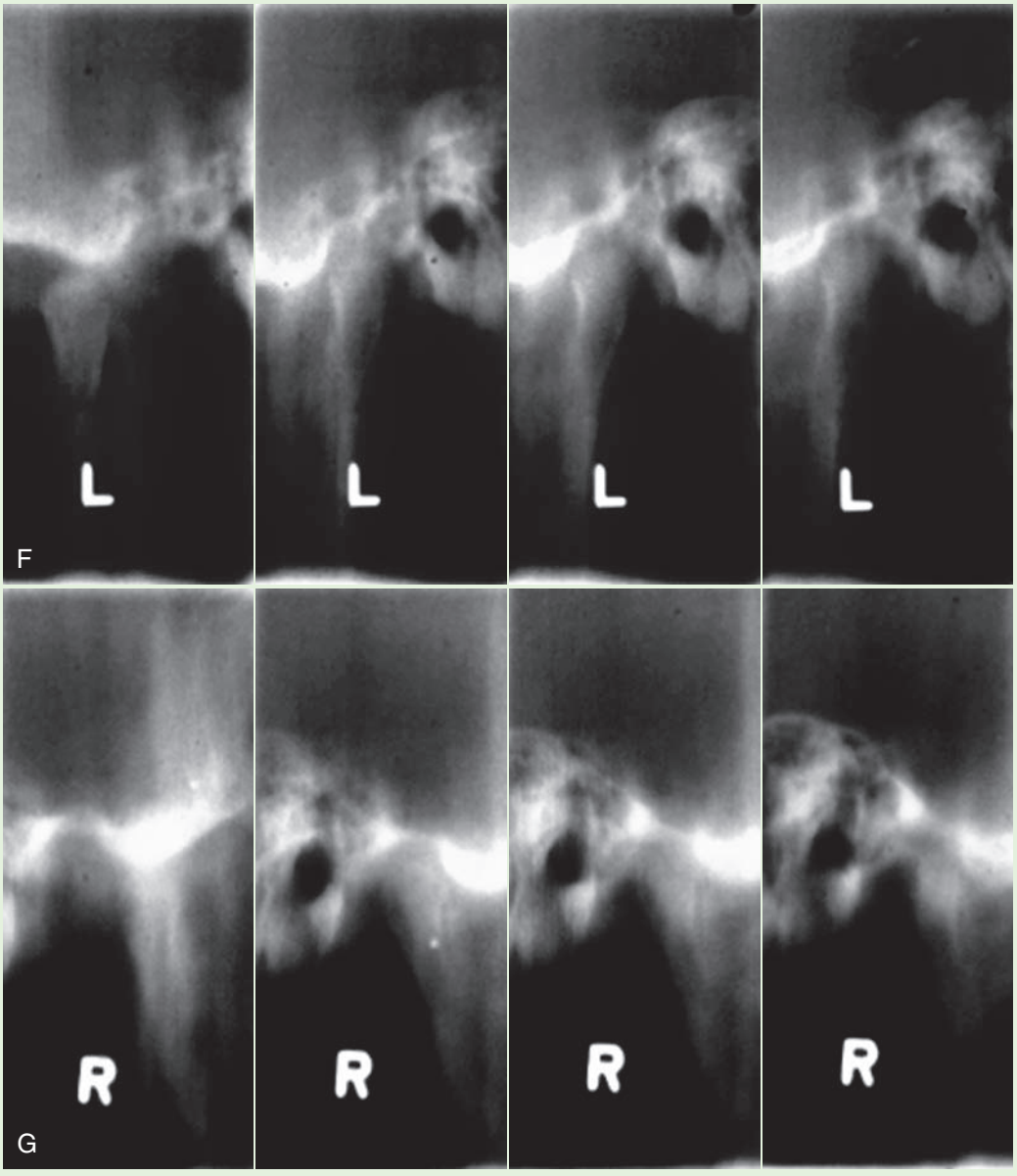


FIGURE 36-39, cont'd

BOX 36-3 Case 3 (KH) Treatment Sequence: Phase 2 Post-Herbst Appliance

Start: Extracted upper and lower first premolars.

First appointment: Bonded upper and lower arches 6 to 6. Used +7-degree torque on upper and lower canines. Placed 0.014-inch Ni-Ti Align SE maxillary and mandibular archwires.

Second appointment at 2 months 2 weeks: Placed maxillary and mandibular 0.016- × 0.025-inch Ni-Ti Align SE archwires.

Third appointment at 5 months: Placed maxillary and mandibular posted 0.019- × 0.025-inch stainless steel archwires with Ni-Ti closing springs hooked onto archwire distal to first molars(see Fig. 36-40).

Fourth appointment at 7 months, 1 week: Activated Ni-Ti springs, and clipped archwires distal to first molars (see Fig. 36-41).

Fifth appointment at 9 months, 3 weeks: Activated Ni-Ti springs, and started Class II elastics (Fig. 36-42).

Sixth appointment at 12 months: Bonded upper and lower second molars. Placed 0.016-inch Ni-Ti Align SE overlay archwire.

Seventh appointment at 14 months, 2 weeks: Adjusted maxillary archwire, and continued Class II elastics (night only), including anterior trapezoid.

Eighth appointment at 16 months: Adjusted both archwires. Started nighttime V-elastics and continued trapezoid elastics.

Ninth appointment at 17 months, 2 weeks: Adjusted maxillary archwire and continued V-elastics and anterior trapezoid elastics full time.

Tenth appointment at 19 months: Adjusted both archwires and continued V-elastics and anterior trapezoid elastics.

Eleventh appointment at 20 months, 2 weeks: Adjusted maxillary archwire. Continued V-elastics and anterior trapezoid elastics, and scheduled debonding.

Twelfth appointment at 21 months, 2 weeks: Debonded both arches (see Fig. 36-43). Bonded 0.016-× 0.022-inch Bond-a-Braid braided wire onto the maxillary teeth lateral to lateral incisor. Bonded 0.026-inch stainless steel round wire onto the mandibular teeth canine to canine.

Continued

CASE STUDY 36-3 Treatment with Extraction Therapy—cont'd

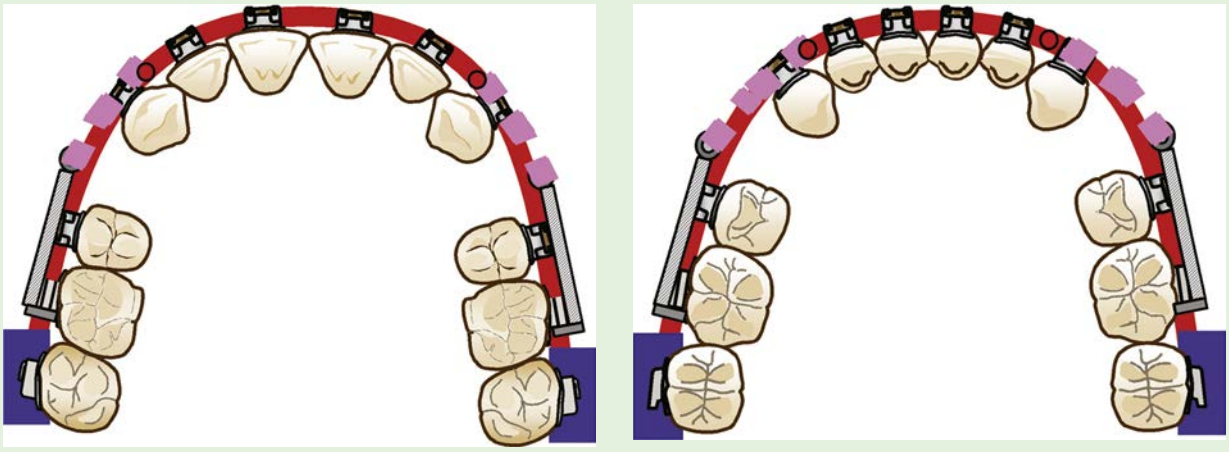


FIGURE 36-40 A, B, To close space, the distal eyelet of nickel-titanium (Ni-Ti) coil spring was bent 90 degrees before its placement over the distal end of the cut archwire on the first molars.

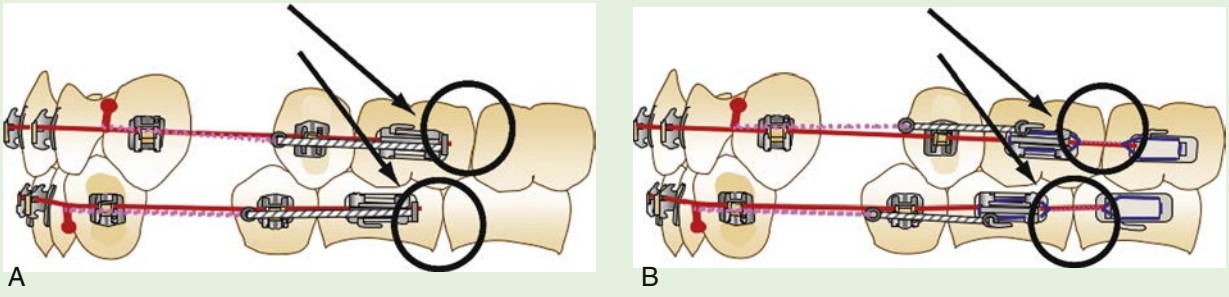


FIGURE 36-41 A, In minimum anchorage retraction cases, cutting the archwire distal to the first molar and attaching the spring to the distal end of the first molar tube are recommended. B, In maximum anchorage retraction cases, ligating the first and second molars together while attaching the spring to the hook of the first molar is recommended.

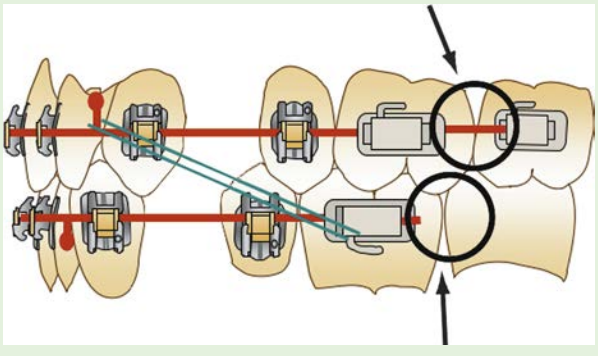


FIGURE 36-42 Tenth appointment at 19 months for KH. V-elastics and anterior trapezoid elastics are configured.

distribute forces over all teeth. Eliminating bracket hooks keeps the appliance clean and promotes healthy tissue response and patient comfort.

Posttreatment records were taken at 21 months, 2 weeks (Fig. 36-43). Figure 36-44 provides a summary of the wire progression of phase 2 treatment. The author does not see how this favorable of a result could have been achieved without the premolar extractions. Maxillary retention was achieved with 0.016-× 0.022-inch Bond-a-Braid braided wire (Reliance Orthodontic Products, Itasca, IL) bonded to each tooth lateral to lateral incisor. Mandibular retention was achieved with 0.026-inch stainless steel wire placed canine to canine and bonded to only the canines. Clear slip-cover retainers were made for both arches to help hold the extraction sites closed while the Class II splint was being fabricated (Fig. 36-45). To retain the Class II correction, the splint was worn nightly for 1 year, after which time the patient began wearing the slipcover retainers again only at night.

CASE STUDY 36-3 Treatment with Extraction Therapy—cont'd

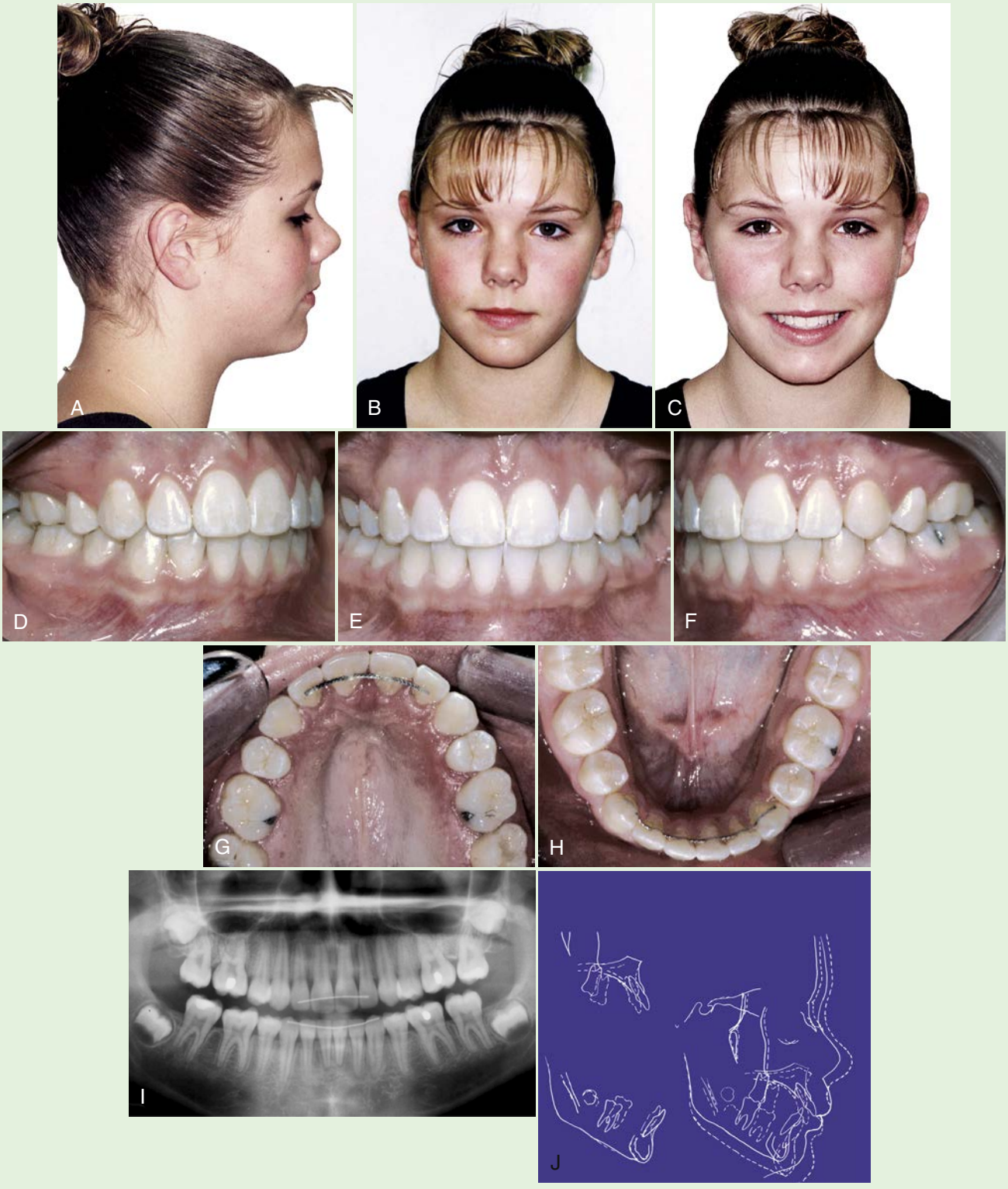


FIGURE 36-43 A-J, Posttreatment records for KH. Total treatment time: 18 months with 8 appointments of phase 1 treatment and 21 months, 2 weeks with 12 appointments of phase 2 treatment.

Continued

CASE STUDY 36-3 Treatment with Extraction Therapy—cont'd

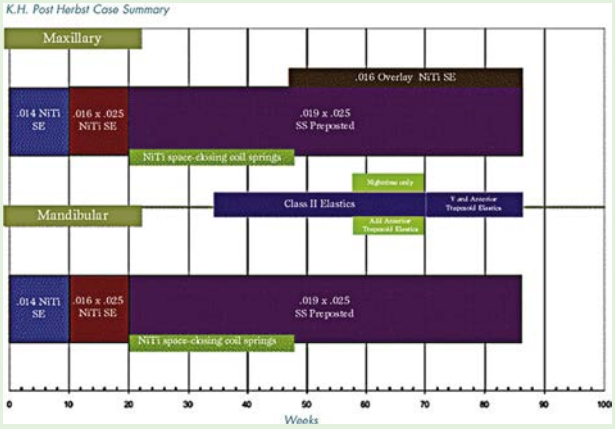


FIGURE 36-44 Summary of phase 2 wire progression treatment for KH is demonstrated.

FIGURE 36-45 Damon splint to retain Class II corrections (see description of appliance construction on page …)

CASE STUDY 36-4 Treatment with Elastics of Nongrowing Patients with Class II and III Dentitions

One of the greatest challenges in clinical orthodontics is planning the treatment of nongrowing patients with a Class II dentition. So often, these cases have significant overbite or overjet in which the profile and lateral facial support will be greatly affected by treatment planning decisions. Most adults seeking orthodontic treatment desire far more than simply straight teeth. They are keenly aware of what the aging process is doing to their bite and face. With this system, the clinician now has many more opportunities to meet this challenge without negatively affecting the profile and lateral facial support and can, in fact, sometimes improve it.

Some clinicians consider dental correction of Class II and III cases with nothing more than elastics impossible, but with the use of the tube system and high-tech archwires, orthodontists around the world are able to treat certain patients with Class II and III elastics that conventional mechanics precludes. Case 4 (TBM) was selected to demonstrate how the Damon System and Class II elastics can correct a nongrowing patient with a Class II dentition if certain principles are followed. Using a Herbst appliance attached to the archwire was obviously the author's first treatment choice, but the patient preferred to use elastics.

Patient TBM illustrates a combination of factors: a full Class II, Division 2 dentition; the necessity to level the curve of Spee; and moderate anterior crowding. Orthodontists using conventional mechanics would be necessarily apprehensive about treating such a case with Class II elastics for fear of severely dumping the lower anteriors. With traditional mechanics, the archwire is securely tied into each bracket and acts like a bridge beam. Each arch operates as a rigid unit against the other; any interference significantly slows anteroposterior correction. The Damon System functions similar to multiloop archwires, given the play between the archwire and the tube. With independent movement at every bracket, slight interferences do not shut down anteroposterior correction with Class II elastics. At the same time, using very low forces allows the lips and muscles of the face to act as a lip bumper, which precludes dumping the anteriors. Moreover, because second molars sit in cortical bone, the archwire is clipped distal to the first molars, allowing the second molars to drift independently. The gain in the transverse arch width not only helps accommodate the crowding, but it also helps correct the Class II dentition.

TBM was a 40-year-old patient exhibiting a Class II, Division 2 dentition with a pleasing upper lip–to–nose relationship and a strong chin button (Fig. 36-46). She had a 100% overbite with overerupted incisors with the upper central incisors too upright and labially inclined lower anterior teeth. The lower lip was slightly everted because of the position of the anterior dentition. She exhibited moderate crowding of both arches and a significant curve of Spee. Her lower left second premolar was congenitally missing, with a retained second primary molar occupying its space. Third molars had been extracted before treatment. The upper right central and lower right first molar had full porcelain crowns. Earlier in the author's career, the author would have tried to distalize the molars or would have extracted upper first premolars. Either of these treatment options would have had a significant detrimental impact on the profile with the loss of upper lip support.

The treatment objectives were as follows:

1. Correct Class II molar and canine, eliminate crowding, and level the curve of Spee without further dumping of the lower incisors.
2. Eliminate the option of extracting maxillary first premolars because of the negative impact on the face.
3. Establish archform to give lateral facial support.
4. Control torque of maxillary and mandibular anterior segments.
5. Maintain attractive nose-to-lip relationship.
6. Minimize everting of lower lip.

Box 36-4 outlines the treatment sequence. With this low-force and tube technology, the overbite and torque were corrected with the selection of high-torque anterior brackets and tubes and a reverse curve Ni-Ti archwire with 20 degrees of torque. These appliance choices saved considerable time and allowed the clinician to start Class II elastics as soon as a 0.019- × 0.025-inch stainless steel wire is placed in the upper arch. This size of stainless steel archwire is strongly recommended to maintain the vertical dimension in the maxillary arch when pulling Class II elastics. Figure 36-50 provides a summary of the wire progression. Correcting the Class II molar and canine relationships, eliminating the crowding in both arches, and leveling the curve of Spee without severely dumping the lower incisors were encouraging results. In this case, the lower incisors actually uprighted slightly with only minimal anterior bodily movement. Cutting the lower archwire distal to the first molars before placing Class II elastics is always important (see Case Study 3). In some cases, the patient will position the jaw forward when sleeping, minimizing the effect of elastic wear. If so, then instructing the patient to wear two elastics on each side during sleep is important.

CASE STUDY 36-4 Treatment with Elastics of Nongrowing Patients with Class II and III Dentitions—cont'd

Figure 36-51 illustrates comparisons of pretreatment and posttreatment upper arch plaster models, which show 3.5-mm change in canine width, 2-mm change in first premolar width, and 2.5-mm change in first molar width. Figure 36-52 illustrates comparisons of pretreatment and posttreatment models of lower arch plaster models, which show 1.5-mm change in canine width, 1.5-mm change in first premolar width, and 1.0-mm change in first molar width.

To be able to exceed this patient's expectations for orthodontics by completing treatment in 18 months, 1 week and with only 11 total appointments was gratifying (see Fig. 36-49). With such treatment, outstanding patient cooperation is obviously required. Figure 36-53 shows the facial and intraoral results taken 19 months into retention that illustrate excellent stability.

FIGURE 36-46 **A-K,** Pretreatment records for case 4 (TBM, age 40 years). Diagnosis: Class II, Division 2 dentally; pleasing upper lip–to–nose relationship and strong chin button; 100% overbite, overerupted incisors, upper central incisors too upright, and labially inclined lower incisors with everted lower lip; moderate crowding in both arches and significant curve of Spee; lower left second premolar congenitally missing with retained second primary molar. Upper right central and lower right first molar had full porcelain crowns. Third molars were extracted before treatment.

Continued

CASE STUDY 36-4 Treatment with Elastics of Nongrowing Patients with Class II and III Dentitions—cont'd

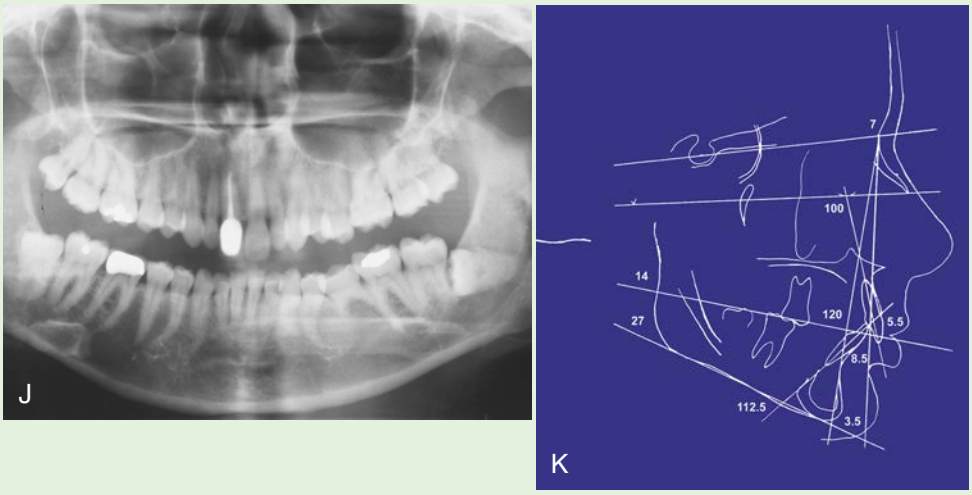


FIGURE 36-46, cont'd

BOX 36-4 Case 4 (TBM) Treatment Sequence

First appointment: Because this patient elected to wear Class II elastics rather than a Herbst appliance, she wore them for an extended period; consequently, high-torque brackets on the upper incisors (+17 degrees on the upper central incisors and +12 degrees on the upper lateral incisors) were selected to prevent the central incisors from uprighting too much. Low-torque brackets were selected for the lower incisors (–6 degrees) to keep them from flaring during extended Class II elastics wear. Bonded upper and lower arches 7 to 7, except for broken-down lower left first molar where a band was placed for support. Placed separators for band. Placed a 0.014-inch Ni-Ti Align SE archwire in the upper arch and a 0.014-inch Ni-Ti Align SE sectional archwire in the lower arch (the lower left first molar was not bondable). Made night-time soft splint for lower arch.

Second appointment at 2 months, 2 weeks: Placed a 0.017- × 0.025-inch Ni-Ti reverse curve with 20-degree torque in the upper arch. Banded lower left first molar. Placed a 0.016-inch Ni-Ti Align SE in the lower arch.

Third appointment at 5 months: Inserted a 0.018- × 0.025-inch Ni-Ti Align SE in the maxilla as a transitional wire between the 0.017- × 0.025-inch Ni-Ti reverse curve and the 0.019- × 0.025-inch stainless steel finishing archwire. Without this transitional wire, going from the reverse curve to the finishing archwire would have been too uncomfortable for the patient. (The reverse curve with +20-degree torque placed at the previous appointment worked exceptionally well in this instance. Sometimes a 0.019- × 0.025-inch reverse curve with torque is needed.) Placed a 0.016- × 0.025-inch Ni-Ti Align SE in the mandible.

Fourth appointment at 7 months, 1 week: Placed a 0.019- × 0.025-posted stainless steel archwire in both arches. Took panoramic radiograph. Started full-time Class II elastics (5/16-inch, 6-oz) wear. Clipped lower archwire distal to lower first molars. Doing so is critically important to be able to correct Class II dentitions with elastics because the second molar roots are usually positioned in cortical bone of low vascularity.

Fifth appointment at 9 months, 1 week: Continued Class II elastics. Recommend two elastics on each side at bedtime if patient postured mandible forward while sleeping.

Sixth appointment at 10 months, 3 weeks: Checked patient. Continued Class II elastics.

Seventh appointment at 12 months, 1 week: Adjusted maxillary archwire with tiebacks (Fig. 36-47). Continued Class II elastics.

Eighth appointment at 14 months: Placed a 0.014- × 0.025-inch Ni-Ti Align SE in the mandible with tiebacks. Used a 0.014- × 0.025-inch Ni-Ti Align SE to incorporate second molars. Left for one appointment. Continued Class II elastics.

Ninth appointment at 15 months, 2 weeks: Maintained the 0.019- × 0.025-inch posted stainless steel archwires in the maxilla. Placed a 0.016- × 0.025-inch posted stainless steel archwire in the mandible for desired play between the archwire and the bracket lumen to improve settling and finishing. Began full-time V-elastics. Changed Class II elastics wear from full time to night only.

Tenth appointment at 17 months: Adjusted maxillary and mandibular archwires (Fig. 36-48). Maintained same elastic wear. Prepared to debond.

Eleventh appointment at 18 months, 1 week: Debonded both arches and placed retention wires (Fig. 36-49). Bonded a 0.016- × 0.022-inch Bond-a-Braid braided wire onto the maxillary teeth lateral to lateral incisor. Bonded 0.026-inch stainless steel round wire onto the mandibular teeth canine to canine. Prescribed the Damon splint be worn nightly for 10 to 12 months (length of time prescribed for each patient depends on the severity of the case). Also prescribed regular slip-cover retainers in addition to splint.

CASE STUDY 36-4 Treatment with Elastics of Nongrowing Patients with Class II and III Dentitions—cont'd

FIGURE 36-47 A-E, Seventh appointment for TBM at 12 months, 1 week.

Continued

CASE STUDY 36-4 Treatment with Elastics of Nongrowing Patients with Class II and III Dentitions—cont'd

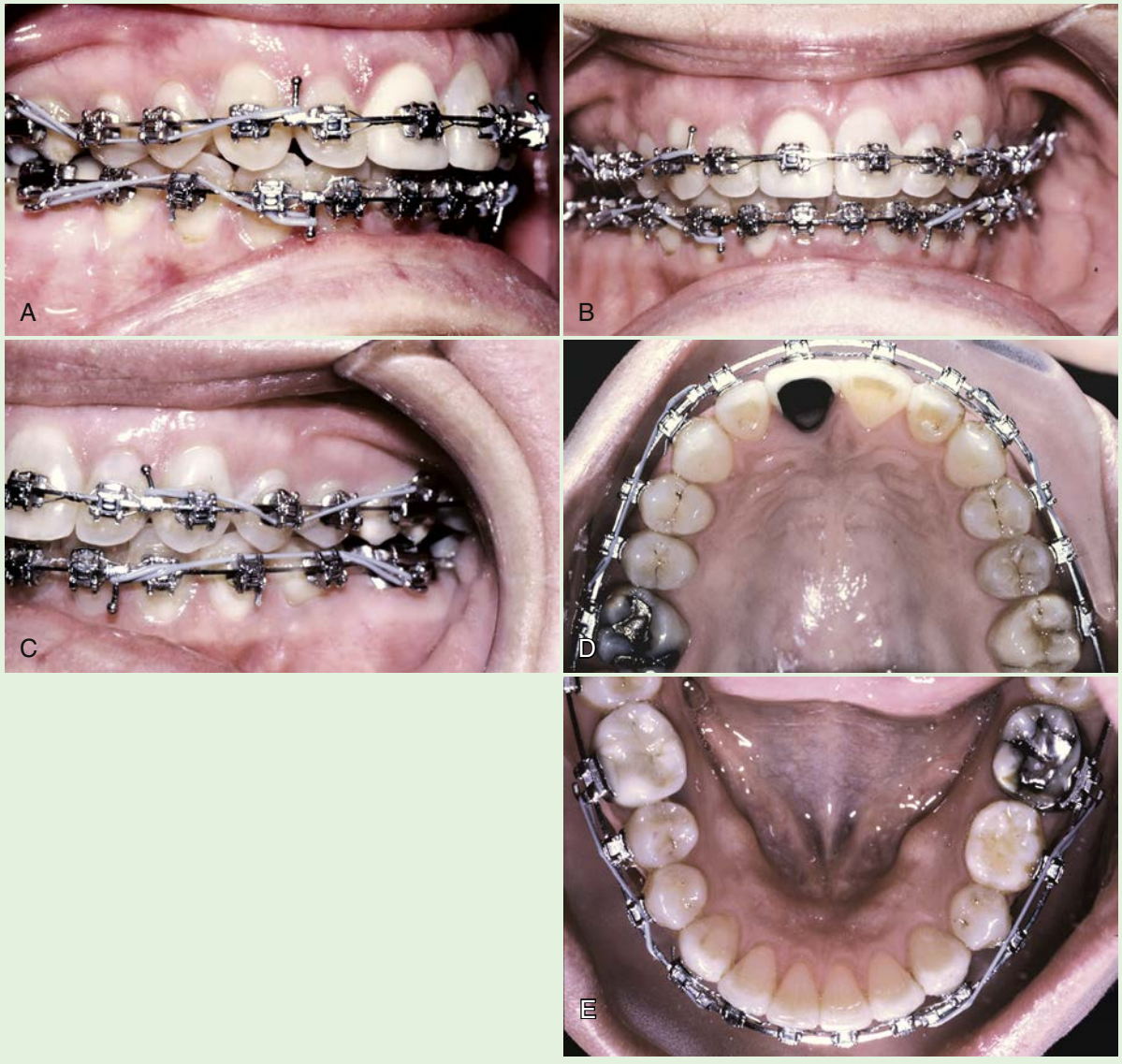


FIGURE 36-48 A-E, Tenth appointment for TBM at 17 months.

CASE STUDY 36-4 Treatment with Elastics of Nongrowing Patients with Class II and III Dentitions—cont'd

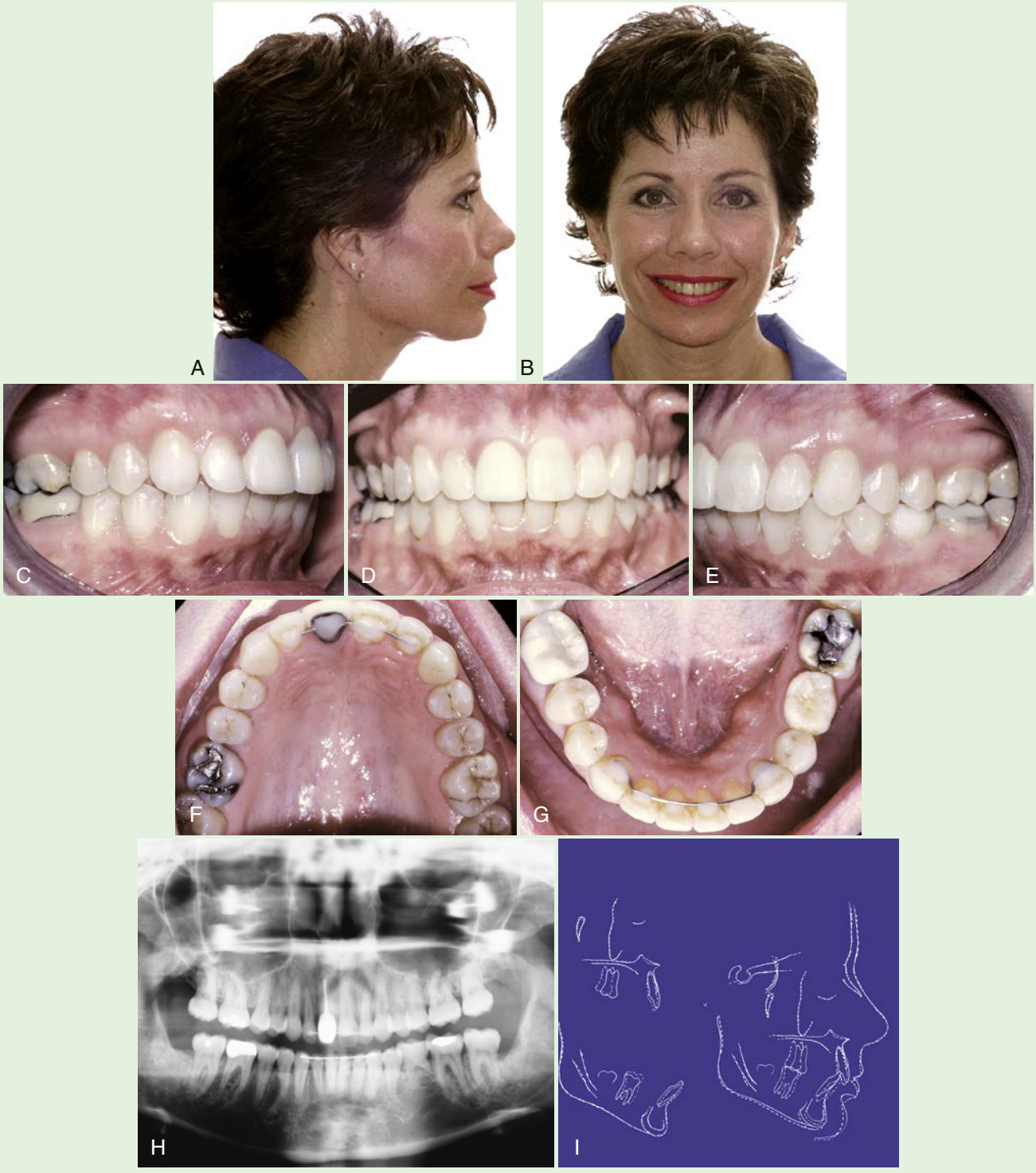


FIGURE 36-49 A-I, Posttreatment records for TBM. Total treatment time: 18 months, 1 week with 11 appointments.

Continued

CASE STUDY 36-4 Treatment with Elastics of Nongrowing Patients with Class II and III Dentitions—cont'd

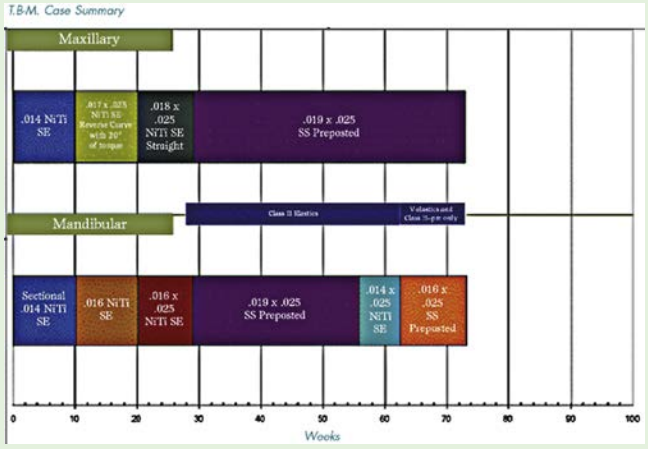


FIGURE 36-50 Archwire sequence for TBM is demonstrated.

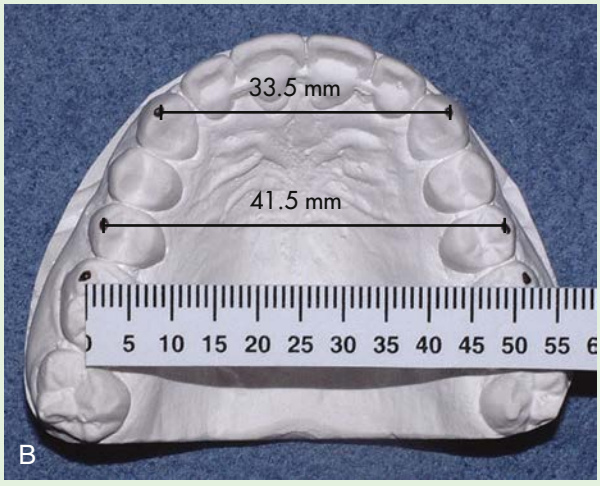
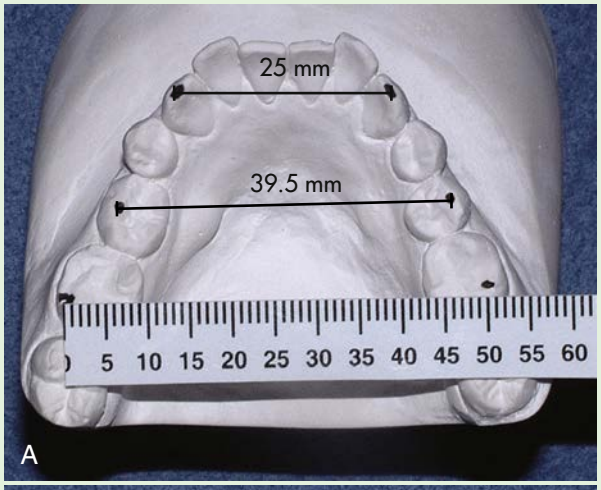


FIGURE 36-51 Comparisons of pretreatment and posttreatment upper arch plaster models for TBM. **A,** Pretreatment. Canines: 30 mm; first premolars: 39.5 mm; first molars: 49.0 mm. **B,** Posttreatment. Canines: 33.5 mm; first premolars: 41.5 mm; first molars: 51.5 mm. Pretreatment and posttreatment models of upper arch show 3.5-mm change in canine width, 2-mm change in first premolar width, and 2.5-mm change in first molar width.



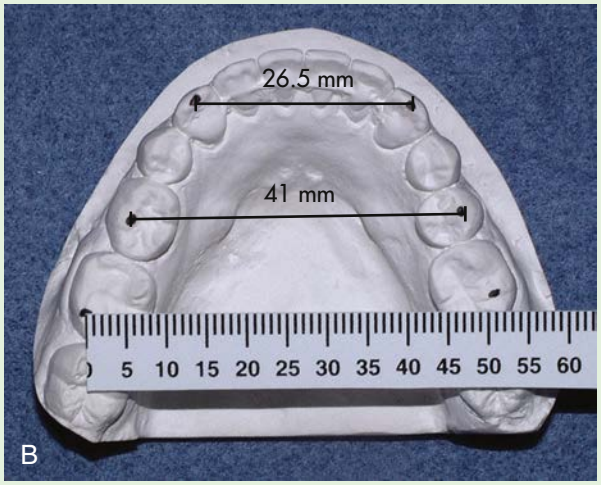


FIGURE 36-52 Comparisons of pretreatment and posttreatment lower arch plaster models for TBM. **A,** Pretreatment. Canines: 25 mm; first premolars: 39.5 mm: first molars: 50.0 mm. **B,** Posttreatment. Canines: 26.5 mm; first premolars: 41.0 mm; first molars: 51.0 mm. Pretreatment and posttreatment models of lower arch show 1.5-mm change in canine width, 1.5-mm change in first premolar width, and 1.0-mm change in first molar width.

CASE STUDY 36-4 Treatment with Elastics of Nongrowing Patients with Class II and III Dentitions—cont'd

FIGURE 36-53 A-H, Comparisons of facial and intraoral results at 19 months into retention for TBM demonstrate excellent stability.

CASE STUDY 36-5 Treatment of a Nongrowing Patient with a Class III Severe Posterior Crossbite and Anterior Open Bite

Case 5 (AB) was selected to show how low-force and tube treatment mechanics affect alveolar bone. The patient strongly opposed the surgical options presented. Before the advent of the Damon System technology, this patient could only have been treated surgically. CT scans taken the day of debonding clearly illustrate that if forces are biologically sensible, then the alveolar bone will follow the bodily movement of the teeth. AB was nearly 37 years old when he sought treatment after observing the results his brother experienced in the author's practice. He had Class III dentition with a severe open bite and a bilateral posterior crossbite (Fig. 36-54). He complained of airway problems and said he was challenged to enunciate clearly when making presentations. Both brothers were severe tongue thrusters, and to close the right posterior open bite was challenging for both of them. He is a vertical grower with a long lower facial height, long tooth roots, and midface deficiency. His upper and lower midline was off when placing the mandible in centric relation.

The treatment objectives were as follows:

1. Using low-force, low-friction mechanics, allow the orofacial musculature, bone, and soft tissue to establish a new physiologically determined tooth position that allows more room for the tongue.
2. Correct Class III molar relationship and bilateral posterior crossbite.
3. Increase maxillary posterior arch width to accommodate crowding.
4. Improve midface support by moving maxillary dentition forward.
5. Improve cant of occlusal plane, if possible.
6. Correct anterior open bite. Retain case to preclude tongue thrust from reopening bite.

Box 36-5 outlines the treatment sequence for this patient. Special torques were selected: +17 degrees for the upper central incisors, +10 degrees for the upper lateral incisors, and +7 degrees for the upper and lower canines. Figure 36-56 compares the plaster models of the upper arches taken initially and on the day of debonding with vertically cut

Continued

CASE STUDY 36-5 Treatment of a Nongrowing Patient with a Class III Severe Posterior Crossbite and Anterior Open Bite—cont'd

FIGURE 36-54 **A-K,** Pretreatment records for case 5 (AB, age 36 years, 11 months). Diagnosis: Class III dentally, severe open bite, bilateral posterior crossbite, midline off center with long lower facial height, long tooth roots, and midface deficiency; airway and enunciation problems. Patient desired an alternative to surgery.

CASE STUDY 36-5 Treatment of a Nongrowing Patient with a Class III Severe Posterior Crossbite and Anterior Open Bite—cont'd

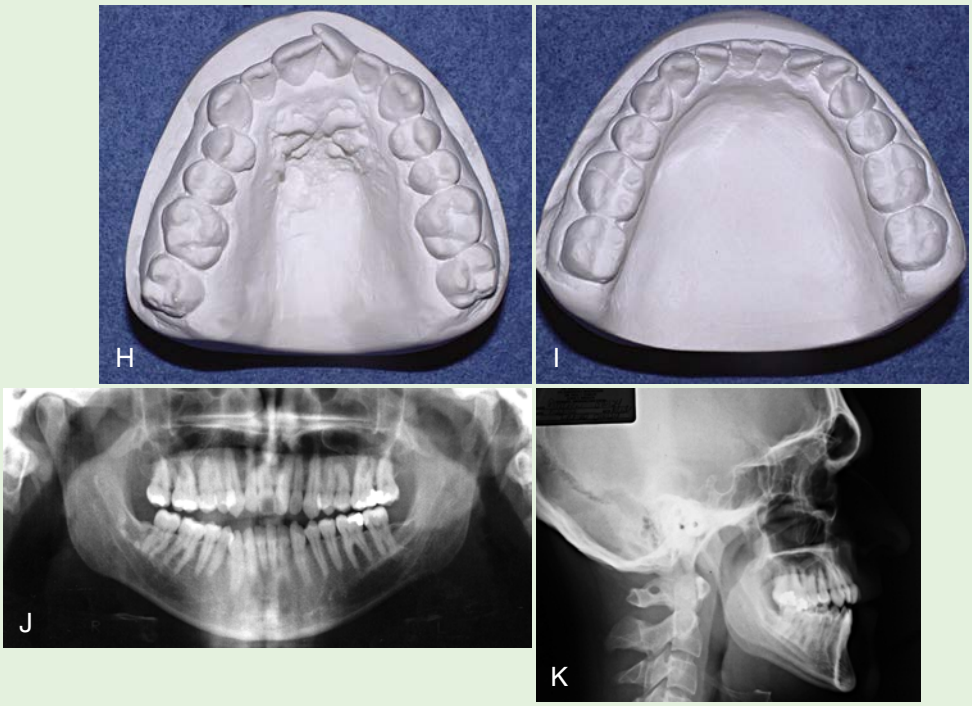


FIGURE 36-54, cont'd

BOX 36-5 Case 5 (AB) Treatment Sequence

First appointment: Bonded maxillary and mandibular arches 7 to 7. Placed 0.014-inch Ni-Ti Align SE in both arches.

Second appointment at 2 months, 2 weeks: Rebonded lower right lateral and replaced 0.014-inch Ni-Ti Align SE. Placed maxillary 0.014- × 0.025-inch Ni-Ti Align SE.

Third appointment at 4 months, 1 week: Placed mandibular 0.016-inch Ni-Ti Align SE. Patient missed appointments for the next several months.

Fourth appointment at 8 months 2 weeks: Placed maxillary 0.016- × 0.025-inch Ni-Ti Align SE. Placed mandibular 0.014- × 0.025-inch Ni-Ti Align SE.

Fifth appointment at 10 months, 2 weeks: Placed maxillary 0.019- × 0.025-inch posted stainless steel. Placed mandibular 0.016- × 0.025-inch stainless steel. Bonded lingual buttons to upper first molars. Started full-time posterior cross elastics and full-time Class III 5/16-inch, 6-oz elastics.

Sixth appointment at 13 months: Adjusted upper and lower archwire. Continued full-time wear of Class III elastics. Changed posterior cross elastics to nighttime wear.

Seventh appointment at 14 months, 2 weeks: Adjusted maxillary archwire. Started full-time 5/16-inch, 6-oz posterior V-elastics and anterior trapezoid elastics.

Eighth appointment at 16 months: Adjusted maxillary archwire. Continued same elastics wear.

Ninth appointment at 17 months, 3 weeks: Adjusted maxillary and mandibular archwires. Continued same elastics wear.

Tenth appointment at 19 months, 1 week: Adjusted maxillary and mandibular archwire. Continued same elastics wear.

Eleventh appointment at 20 months, 3 weeks (Fig. 36-55): Debonded both arches. Bonded 0.016- × 0.022-inch Bond-a-Braid braided wire onto the maxillary teeth lateral to lateral incisor. Bonded 0.026-inch stainless steel round wire onto the mandibular teeth canine to canine. Fabricated slipcover retainers for upper and lower arches. Fabricated Damon splint for nighttime wear.

CT scans also taken the day of debonding. The maxillary canine width increased 3 mm, the first premolar width increased 8 mm, the second premolar width increased 5 mm, and the first molar width increased 4.5 mm. The CT scans show positive alveolar bone response to significant lateral and vertical tooth movement, once again demonstrating that using biologically sensible forces will cause the alveolar bone to follow the movement of teeth in all planes of space with minimal tipping. (Note that the cut of the horizontal CT scans are tipped toward the anterior segment of the arch.) Using simple mechanics, this patient was treated in 20 months, 3 weeks with 11 office visits (see Fig. 36-55). Retaining these types of complex cases with a night muscle-training splint and lingually bonded wires on both the upper and lower anteriors is essential. Despite significant tooth movement, the photographs taken the day of debonding show a positive tissue response. The horizontal CT scan of the upper arch taken at debonding (Fig. 36-57) illustrates the well-shaped archform.

Continued

CASE STUDY 36-5 Treatment of a Nongrowing Patient with a Class III Severe Posterior Crossbite and Anterior Open Bite—cont'd

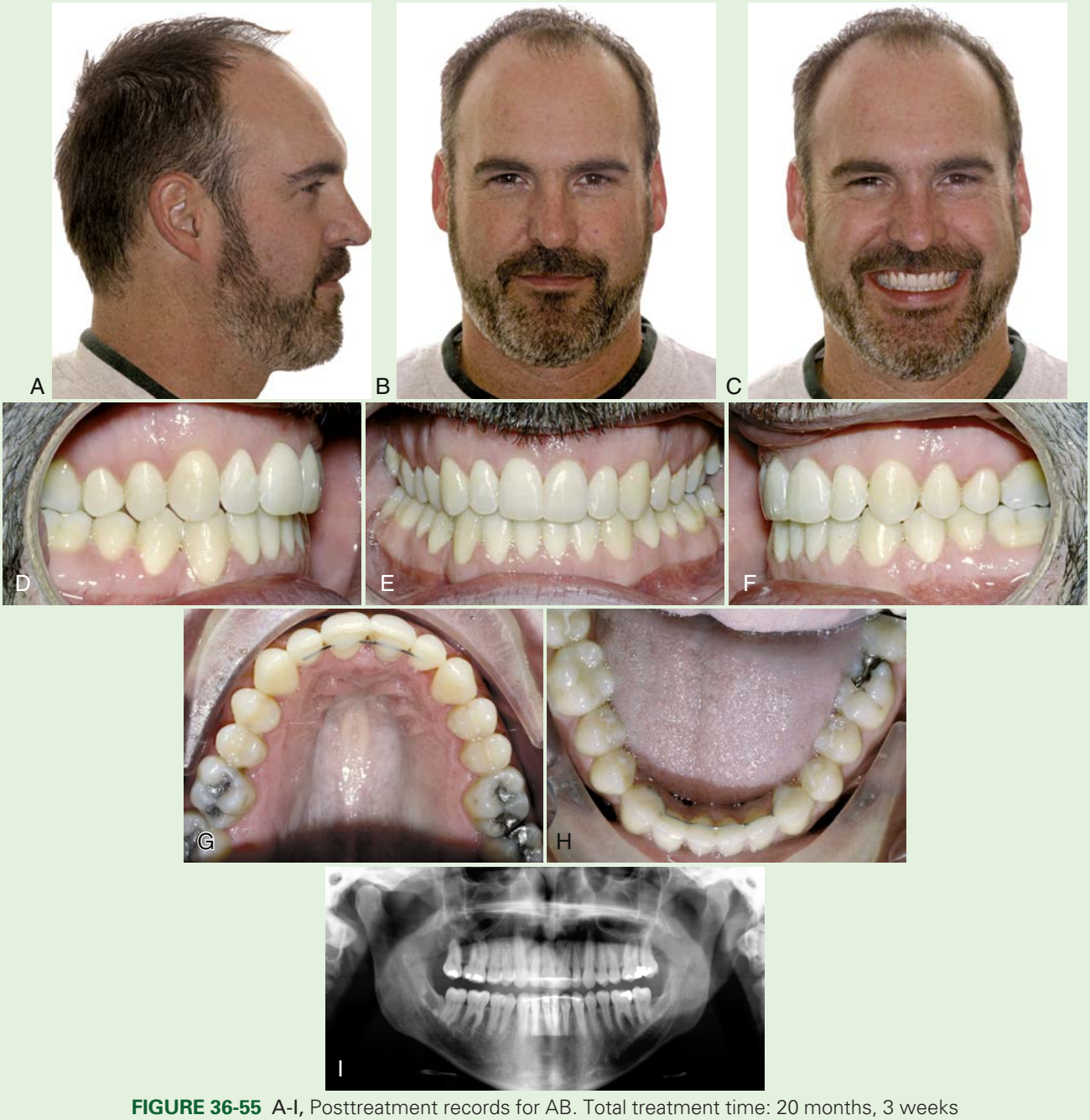


FIGURE 36-55 **A-I,** Posttreatment records for AB. Total treatment time: 20 months, 3 weeks with 11 appointments.

CASE STUDY 36-5 Treatment of a Nongrowing Patient with a Class III Severe Posterior Crossbite and Anterior Open Bite—cont'd

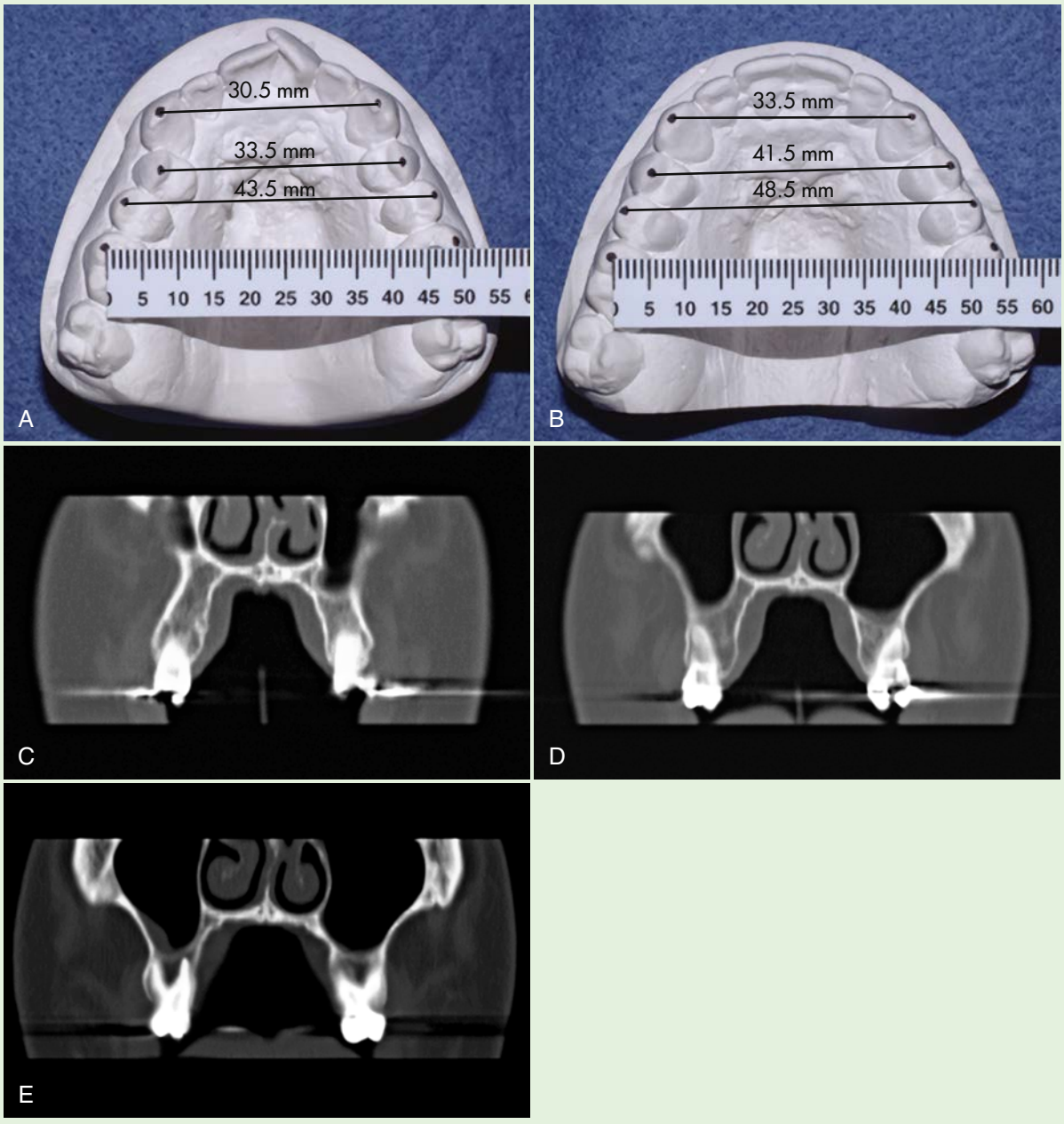


FIGURE 36-56 Comparisons of pretreatment and posttreatment upper arch plaster models for AB with computed tomographic scans. **A,** Pretreatment. Canines: 30.5 mm; first premolars: 33.5 mm; second premolars: 43.5 mm; first molars: 49 mm. **B,** Posttreatment. Canines: 33.5 mm; first premolars: 41.5 mm; second premolars: 48.5 mm; first molars: 53.5 mm. Initial and final models of upper arch show a 3-mm change in canine width, 8-mm change in first premolar width, 5-mm change in second premolar width, and 4.5-mm change in first molar width. Vertical computed tomographic scans taken the day of debonding show healthy alveolar bone. **C,** Scan of upper first premolars at debonding. **D,** Scan of upper second premolars at debonding. **E,** Scan of upper first molars at debonding.

Continued

CASE STUDY 36-5 **Treatment of a Nongrowing Patient with a Class III Severe Posterior Crossbite and Anterior Open Bite—cont'd**

FIGURE 36-57 The horizontal computed tomographic scan of upper arch at debonding illustrates the well-shaped archform in AB.

Treatment with the Herbst Appliance of Growing Patients with Skeletal Class II Dentition

This section includes some of the author's findings with the Herbst appliance after using it for longer than 24 years, treating more than 3000 patients, and following many of these patients for a number of years. Obviously, improvement in the technology continues to affect how orthodontists use and apply any appliance system. The author intends to give an overview of how this technology is used to treat some of the most challenging cases. (See also Chapter 16.)

The Herbst appliance is one of the most powerful treatment options in orthodontics. Combining the low-force and tube system with the Herbst appliance gives the clinician the opportunity to convert complex and long-term treatment situations into straightforward treatment using noncompliance mechanics for superior clinical results. After having the opportunity to lecture around the world, the author has found that many clinicians have a negative view of the Herbst appliance and its clinical application. Many of these views are justified when discussions center only around its impact on the A point–nasion–B point (ANB) angle. The author has observed great variation in what *does or does not happen* to the ANB angle with the use of the Herbst appliance. The author's interest in using the appliance focuses far more on its functional impact on the entire orofacial complex rather than its effect on the ANB. Although a positive response in the ANB angle is helpful, the ANB simply does not change in some cases. Although the ANB angle may not be positively affected, so many positive things do happen when the patient is given a second chance for the entire dentofacial complex to function in a more normal manner. If the mandible is slowly advanced over an extended period, then the impact can be dramatic *in growing and nongrowing patients*. Orthodontists have seen patients who had a narrow upper arch and a severe Class II skeletal pattern. This situation is an example of a functional adaptation of the alveolar process and dentition reacting to the musculature altered by the anteroposterior position of the mandible. If the mandibular position is slowly normalized, then clinicians often observe teeth and alveolar process responses even without orthodontic intervention.

Clinical Principles for Using the Herbst Appliance

Orthodontists know that growth of young patients occurs over a long period. Recognizing that the body needs time to react to a given functional change has always seemed logical. The form-follows-function adage is appropriate when using the Herbst appliance. The author's favorite time to start Herbst appliance treatment is when the young patient's growth starts to accelerate. Some exceptions are severe cases and those patients with self-esteem and special growth issues. The average Herbst appliance starting age for girls is 10½ to 11 years; boys are later at 11 to 11½ years of age.

The author strongly recommends advancing the Herbst appliance only 4.0 to 4.5 mm at the beginning of treatment. Slowly activating the appliance over a long period has many

advantages. The author prefers not to advance the condyle more than two thirds of the way down the articular eminence. This small advancement of the mandible lessens the Class II elastic effect on the musculature and thereby minimizes the forward proclination (dumping) of the lower incisors. With this small advancement, the author relies on the functional effect of the Herbst appliance and muscles to initiate lateral upper arch adaptation naturally, *negating the need for high-force palatal expansion before placing the Herbst appliance*. As the Herbst appliance advances the mandible, the combined impact of the V-shape of the mandible, the usual presence of an upper archwire connecting the four anteriors to the molars, and the Herbst rods foster a slow and natural lateral change in the maxilla with little dental tipping. *Surprisingly, this lateral adaptation of the palate is not age specific.*

The second Herbst appliance advancement of approximately 3 mm is performed 5 to 6 months into treatment. Once again, the activation is allowed an interval of approximately 5 months for the muscles to adapt. Using the same principles, activations thereafter are applied according to each patient's specific needs for skeletal correction. In most cases, activation is terminated when the upper and lower anterior teeth are end to end. The average length of treatment is 14 to 16 months. (In severe applications, treatment time is extended to 2 years or longer.) If a patient relapses during the full-bonded phase of treatment, then the author gives the Herbst appliance a second opportunity to work directly on the archwire. Some of the most successful cases the author has treated have resulted from more than one application of the Herbst appliance.

Patients treated with the Herbst appliance require special retention that is critical to long-term success. The length of time specified for a nightly splint to be worn after debonding in the case studies is an example. The activator-type retainer is most important to the success of this type of treatment.

Working together, the low-force and tube technology and the Herbst appliance provide a powerful combination that allows the clinician to simplify treatment mechanics and convert the most challenging cases to routine clinical orthodontics. Slowly advancing the Herbst appliance, allowing it work for a long period, and retaining it with a night splint are the key elements for success. Most of the Herbst appliance research to date has been on patients who have been rapidly activated, treated over a significantly shorter period, and released without anteroposterior retention.

Clinical Application of the Herbst Appliance

The author uses the Herbst appliance in several different ways in timing and treatment application:

- Mixed dentition Herbst appliance therapy with and without anterior brackets
- Archwire Herbst appliance therapy in patients with full dentition
- Archwire Herbst appliance therapy for patients with temporomandibular joint problems
- Archwire Herbst appliance therapy with coil springs as a supplement for Class II elastics

The following two cases demonstrate Herbst application in the author's practice.

CASE STUDY 36-6 Youth with Herbst Appliance Treatment Demonstrates Typical Response

Case 6 (KP) 12 years, 5 months of age was selected to demonstrate the typical response to standard Herbst appliance treatment. Pretreatment records (Fig. 36-58) indicate her to be a Class II, Division 1 case with a retrusive mandible and narrow midface. She exhibited a good chin button but with poor tissue contour under her chin. She demonstrated a moderate overeruption of lower incisors, flared upper incisors, and lingually inclined upper and lower canines with the typical narrowing of the anterior maxillary arch in patients with a Class II dentition.

The treatment objectives were as follows:

1. Achieve facial balance and symmetry of nose, lips, and chin.
2. Create maxillary and mandibular posterior arch width to support midface with low-force mechanics.
3. Establish maxillary and mandibular incisor position to give natural lip-to-tooth relationship.
4. Exert a positive impact on the archwires.

The pretreatment profile makes it apparent that the mandible is growing slower than the maxilla. Treatment plans that attempt to move the maxilla or dentition distally to meet a deficient mandible often leave patients with a prominent nose, lack of upper lip support, and a weak chin. In such cases, the author has always found it more conducive to create an environment for mandibular change. Box 36-6 outlines the phase 1 Herbst appliance treatment sequence. The impact Herbst appliance treatment had on this patient's facial balance and dentition is positive. Using Herbst appliance therapy before bonding means that the orthodontic treatment is initiated on a simple Class I case with the increased maxillary arch width, eliminating the need for high-force palatal expansion (see Fig. 36-61). An interesting note is that this patient's upper first premolar width change was less than is normally observed with Herbst appliance treatment. The tongue did not completely reposition itself until after fully bonded treatment was started, which added an additional 5-mm width gain after Herbst appliance treatment. Combining the Damon System with Herbst appliance therapy simplifies treatment planning, minimizes the need for patient cooperation, and yields rewarding results. Box 36-7 outlines the phase 2 posttreatment Herbst appliance sequence.

The combination of Herbst appliance therapy and Damon System mechanics achieved excellent facial balance (see Fig. 36-64). Figure 36-65 outlines the posttreatment sequence with the Herbst appliance archwire. Figure 36-66 illustrates the comparisons of the initial and final models of upper arch, which show 5.5-mm change in canine width, 10.5-mm change in first premolar width, and 7-mm change in first molar width. Figure 36-67 illustrates the comparisons of the initial and final models of lower arch, which show 0.5-mm change in canine width, 2-mm change in first premolar width, and 0.5-mm change in first molar width. One-year retention records illustrate good stability (Fig. 36-68). CT scans taken 2 years, 6 months into retention (Fig. 36-69) signify healthy alveolar bone and good archform.

Continued

CASE STUDY 36-6 Youth with Herbst Appliance Treatment Demonstrates Typical Response—cont'd

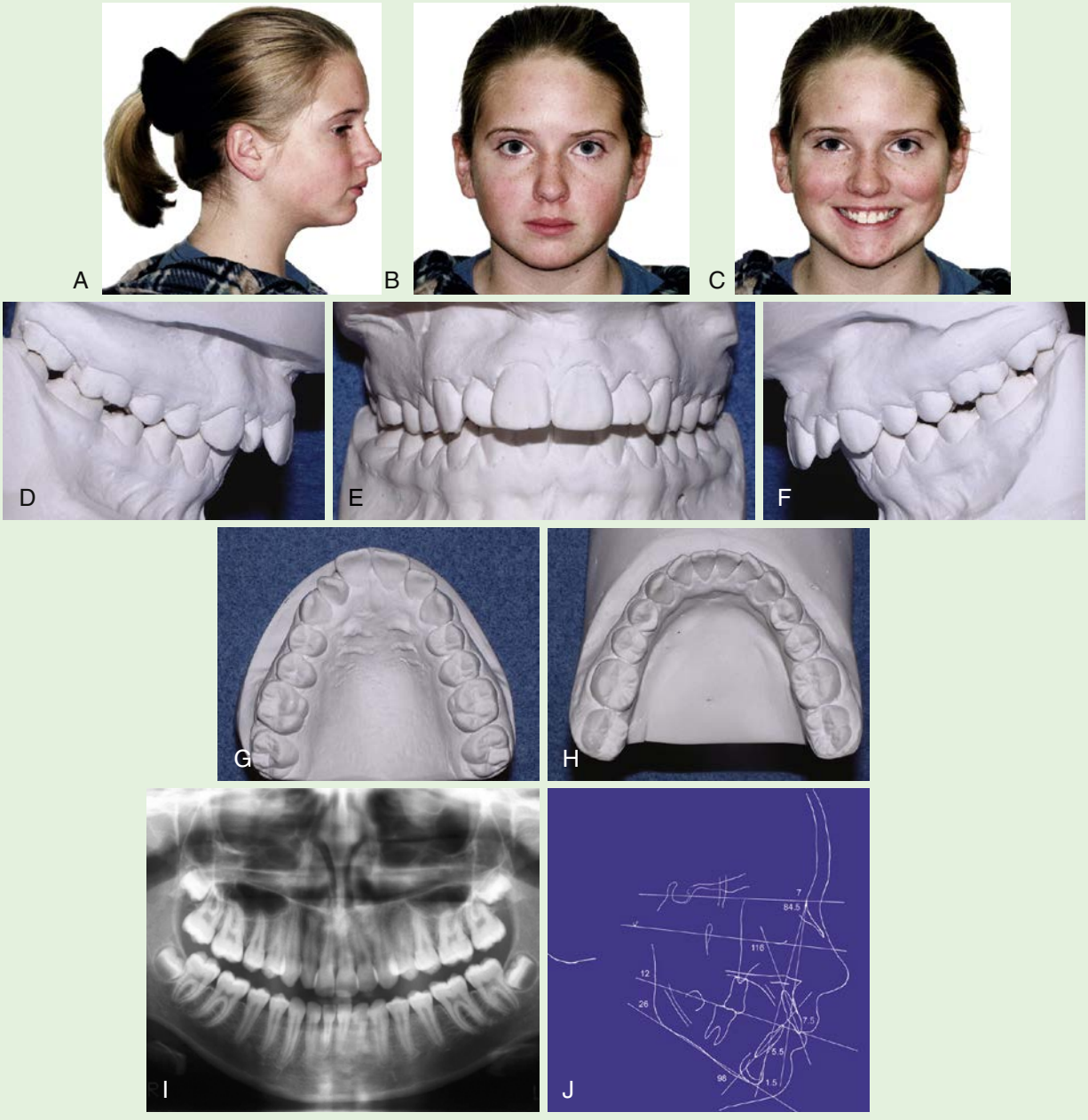


FIGURE 36-58 A-L, Pretreatment records for case 6 (KP, age 12 years, 5 months). Diagnosis: Class II, Division 1 with retrusive mandible, narrow midface, a good chin button but with excessive tissue under her chin; moderate over-eruption of lower incisors, flared upper incisors, and lingually inclined upper and lower canines with the narrowing of anterior maxillary arch that is typical in patients with Class II dentition.

CASE STUDY 36-6 Youth with Herbst Appliance Treatment Demonstrates Typical Response—cont'd

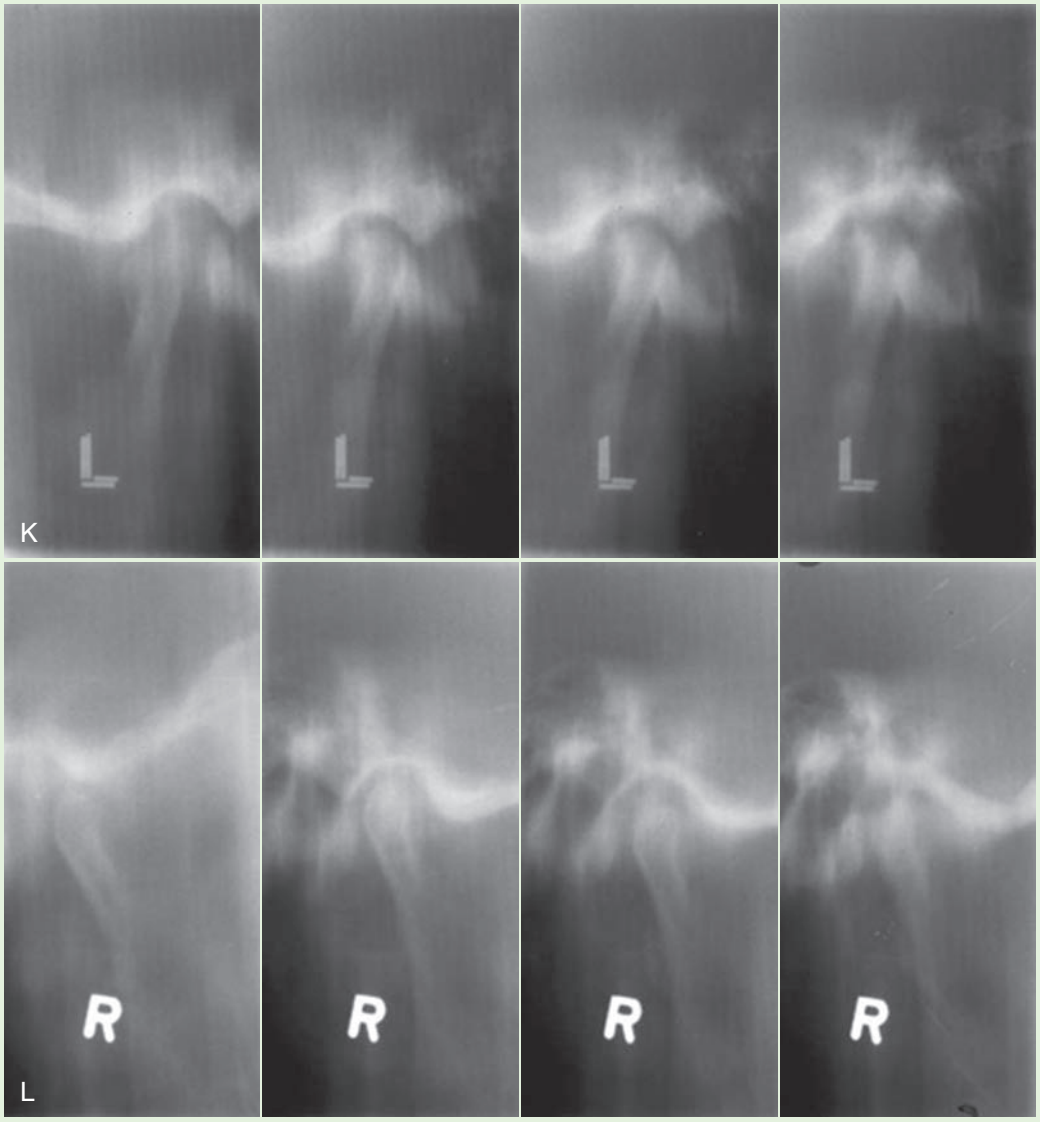


FIGURE 36-58, cont'd

BOX 36-6 Case 6 (KP) Treatment Sequence: Phase 1 Herbst Appliance

Start: Took tomograms.

First appointment: Placed Flip-Lock Herbst appliance (TP Orthodontics, Inc., La Porte, IN) (Fig. 36-59). Selected special high torques for upper central incisors (+17 degrees) and lateral incisors (+12 degrees). Bonded upper central and lateral incisors. Placed sectional 0.014-inch Ni-Ti Align SE extending from maxillary right canine to maxillary left lateral incisor with the ends of the wire heat treated and bent for comfort, leaving enough room for the anterior teeth to align. Initially activated Herbst appliance 4.5 mm.

Second appointment at 2 months, 3 weeks: Placed maxillary and mandibular 0.017- × 0.025-inch titanium molybdenum alloy (TMA) archwire with moderate intrusive bends anterior to the molar tubes. This wire was inserted in tubes soldered to the first molar Herbst crowns.

Third appointment at 6 months: Placed maxillary and mandibular 0.019- × 0.025-inch TMA archwire with moderate intrusive bends to intrude anterior teeth. This wire was inserted in the tubes of the first molar Herbst appliance crown. Added 2-mm shims.

Fourth appointment at 8 months, 2 weeks: Adjusted maxillary archwire. Added 1-mm shim.

Fifth appointment at 10 months, 3 weeks: Checked Herbst appliance.

Sixth appointment at 13 months, 2 weeks: Took tomograms and evaluated progress (Fig. 36-60). Scheduled HERBST appliance removal.

Seventh appointment at 16 months: Removed Herbst appliance (Fig. 36-61). Took progress records. First premolar width change with the Herbst appliance was 5.5 mm.

CASE STUDY 36-6 Youth with Herbst Appliance Treatment Demonstrates Typical Response—cont'd

FIGURE 36-59 Flip-Lock Herbst appliance used to treat KP.

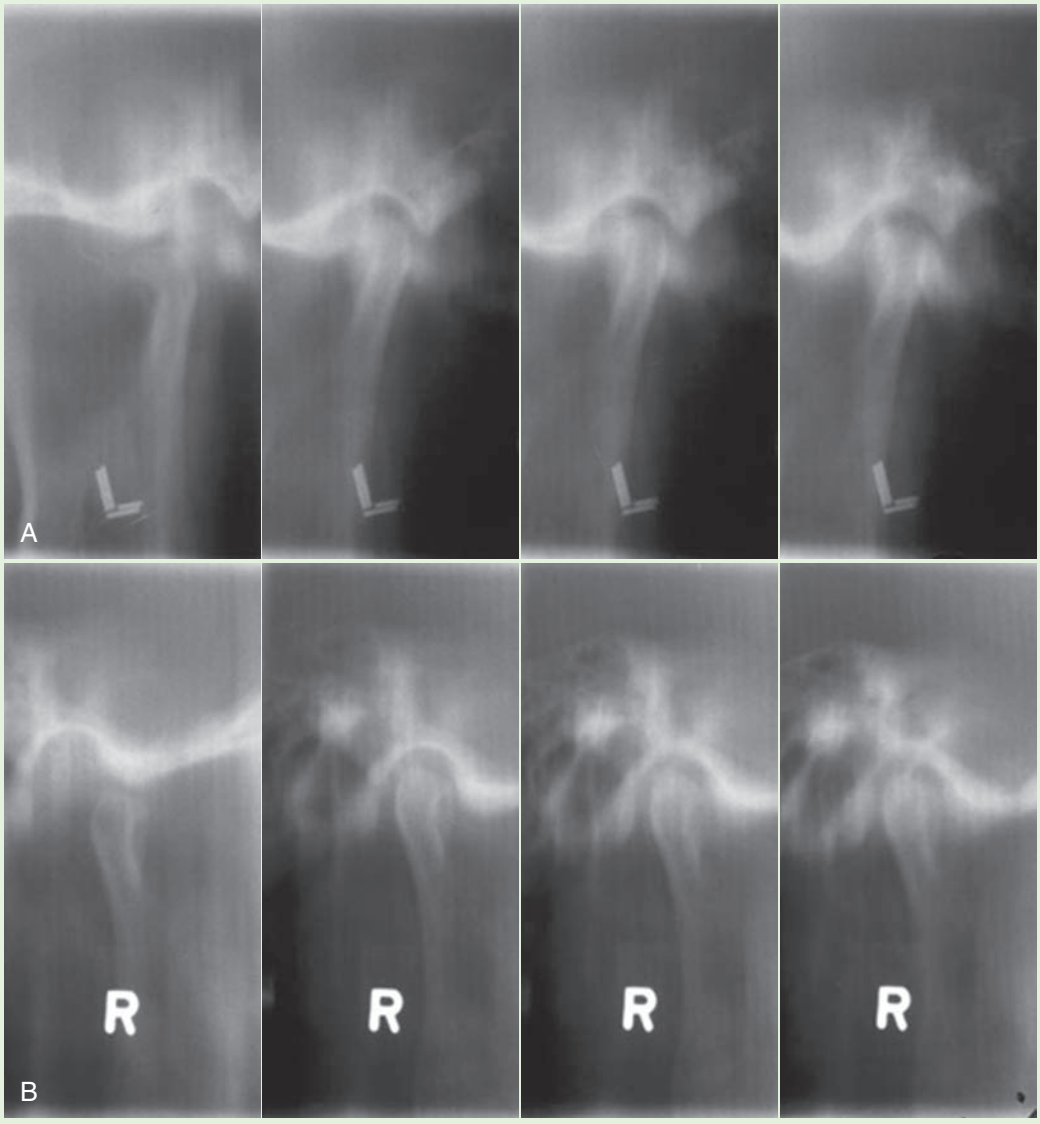


FIGURE 36-60 A, B, Tomograms taken at the sixth appointment of phase 1 Herbst appliance treatment for KP.

CASE STUDY 36-6 Youth with Herbst Appliance Treatment Demonstrates Typical Response—cont'd

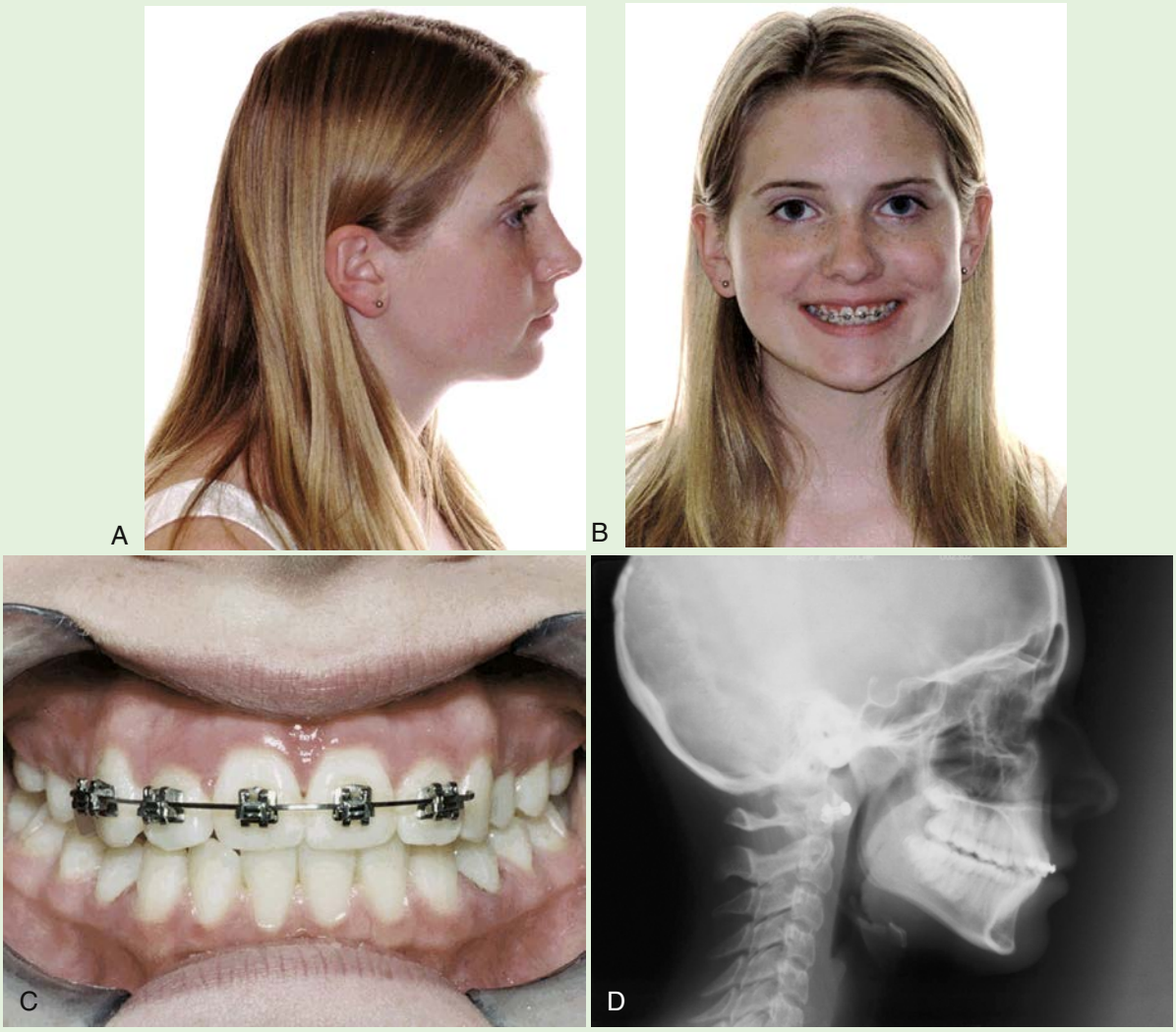


FIGURE 36-61 A-D, Phase 1 posttreatment records of KP with Herbst appliance (Herbst appliance treatment time: 16 months with seven appointments). Records taken after Herbst appliance treatment demonstrate Class I dentition and improved facial symmetry.

BOX 36-7 Case 6 (KP) Treatment Sequence: Phase 2 Posttreatment Herbst Appliance

This patient's canines were slightly toed in. Selected special torques (+7 degrees) for upper and lower canines to help upright them.

First appointment: Bonded maxillary and mandibular 7 to 7. Placed continuous maxillary and mandibular 0.014-inch Ni-Ti Align SE archwires with crimpable stops.

Second appointment at 2 months, 2 weeks: Placed upper 0.016- × 0.025-inch Ni-Ti Align SE. Placed lower 0.014- × 0.025-inch Ni-Ti Align SE.

Third appointment at 4 months, 3 weeks: Took panoramic radiograph to evaluate root angulations and bracket positions (Fig. 36-62).

Fourth appointment at 7 months, 2 weeks: Placed maxillary 0.019- × 0.025-inch posted stainless steel archwire with tiebacks. Placed mandibular 0.016- × 0.025-inch posted stainless steel archwire with tiebacks, which kept the play in the bracket tube to help eliminate binding and help close the posterior occlusion when trying to close the bite vertically. Started bilateral V-elastics.

Fifth appointment at 9 months, 3 weeks: Adjusted upper and lower archwires. Continued full-time V-elastics. Added Class II elastics for night wear only.

Sixth appointment at 12 months: Adjusted maxillary and mandibular archwires. Posterior occlusion was hard to close because of tongue repositioning.

Seventh appointment at 13 months, 2 weeks: Checked occlusion. Continued full-time elastics.

Eighth appointment at 15 months, 1 week: Adjusted maxillary and mandibular archwires. Continued full-time elastics.

Ninth appointment at 17 months: Debonded arches (Fig. 36-64). Initiated fixed retention by bonding 0.016- × 0.022-inch Bond-a-Braid braided wire onto the maxillary teeth lateral to the lateral incisor and bonding 0.026-inch stainless steel round wire onto the mandibular teeth canine to canine because of the severity of the initial crowding. The patient was instructed to wear clear plastic overlay retainers for upper and lower arches and the Damon splint for night retention for an activator type of effect until patient is finished growing.

Continued

FIGURE 36-62 Phase 2 Panorex for KP.

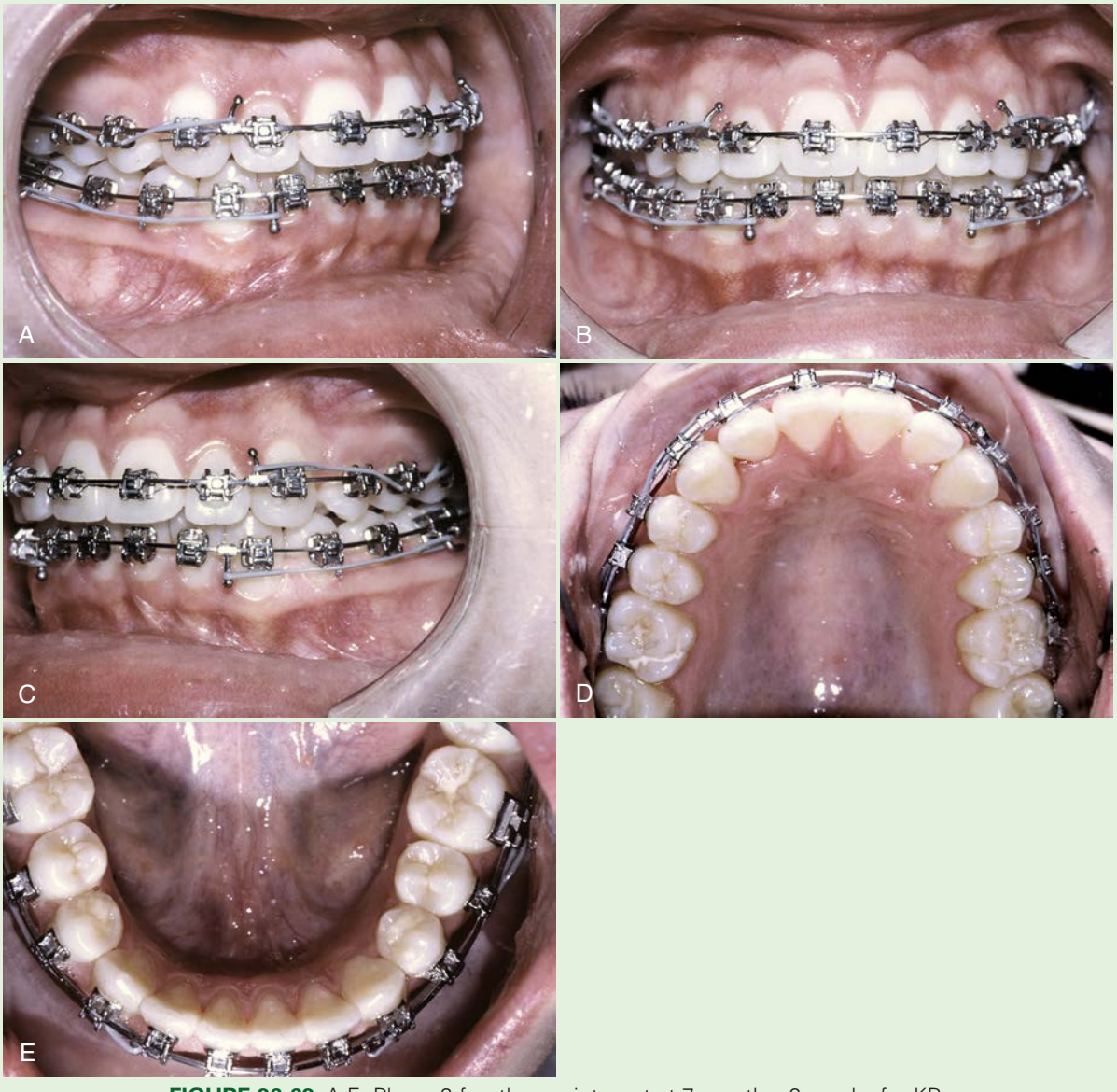


FIGURE 36-63 A-E, Phase 2 fourth appointment at 7 months, 2 weeks for KP.

CASE STUDY 36-6 **Youth with Herbst Appliance Treatment Demonstrates Typical Response—cont'd**

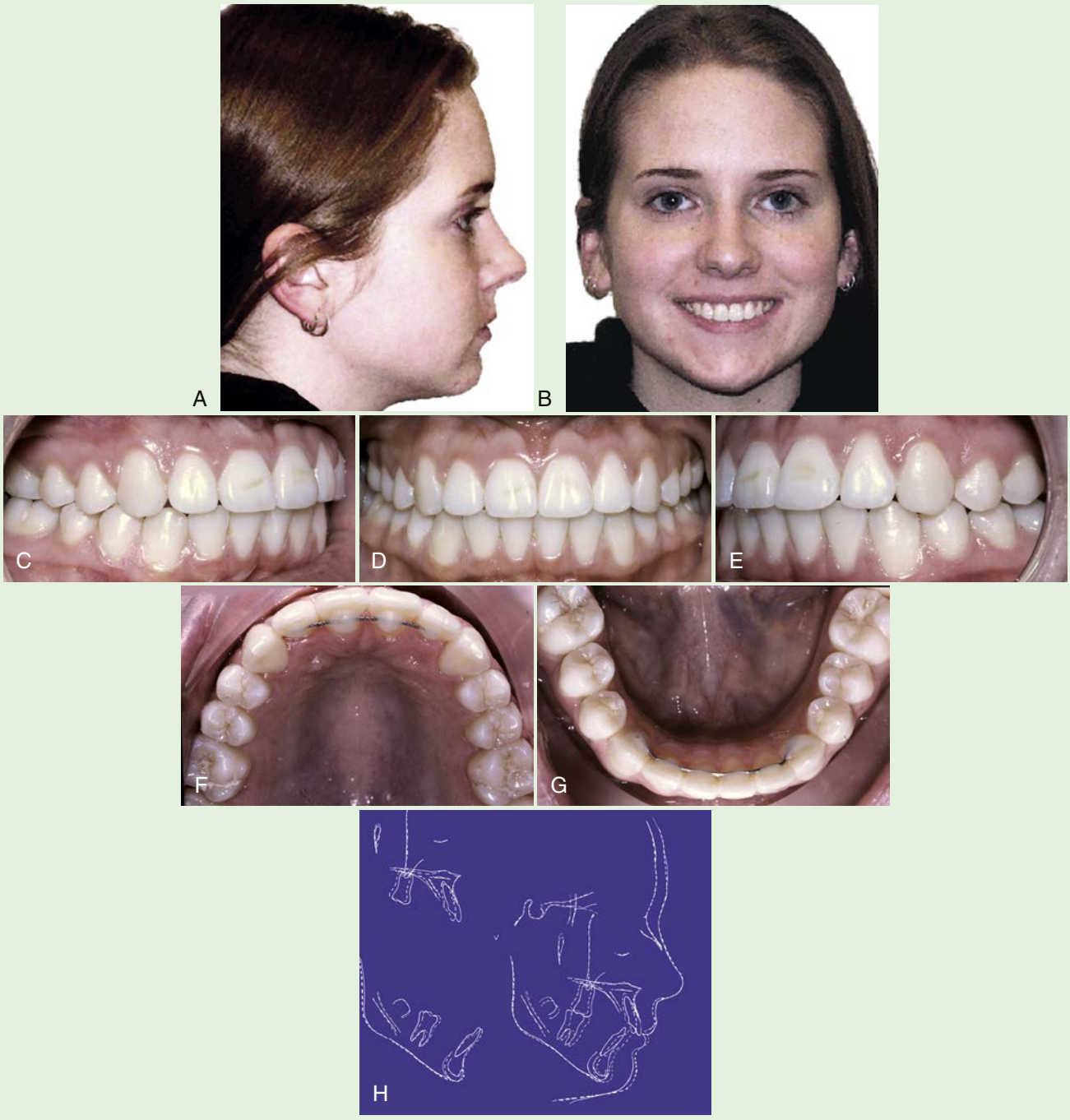


FIGURE 36-64 A-H, Posttreatment records for KP. Total phase 1 and phase 2 treatment time: 33 months with 16 appointments.

Continued

CASE STUDY 36-6 Youth with Herbst Appliance Treatment Demonstrates Typical Response—cont'd

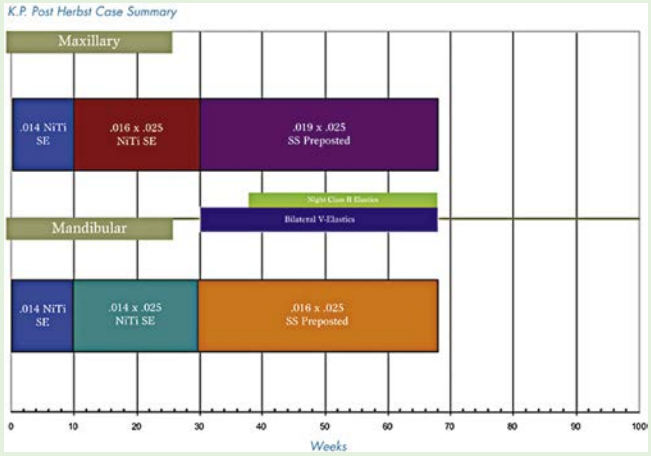


FIGURE 36-65 Post–Herbst appliance archwire sequence for KP is demonstrated.

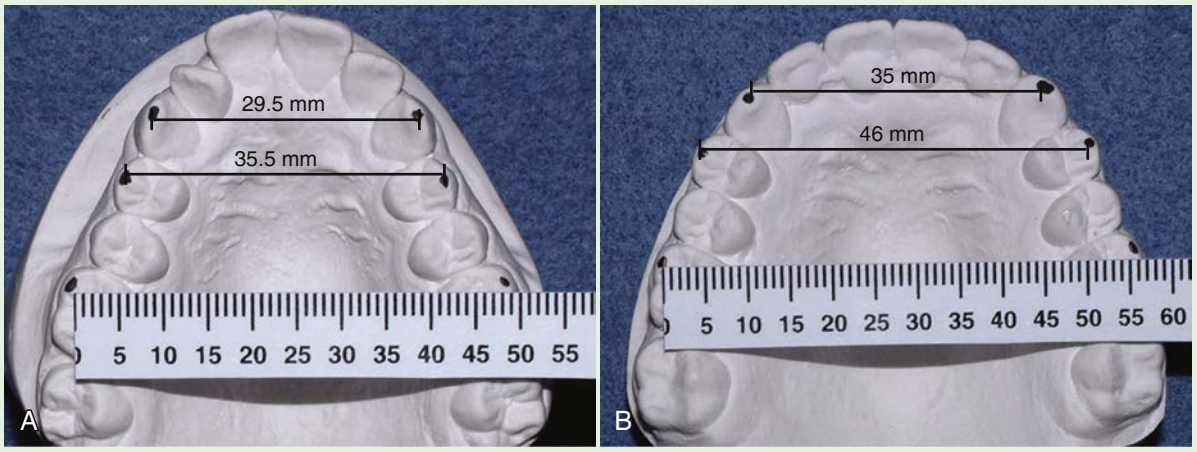


FIGURE 36-66 Comparisons of pretreatment and posttreatment upper arch plaster models for KP. **A,** Pretreatment. Canines: 29.5 mm; first premolars: 35.5 mm; first molars: 48.5 mm. **B,** Posttreatment. Canines: 35 mm; first premolars: 46 mm; first molars: 55.5 mm. Initial and final models of upper arch show 5.5-mm change in canine width, 10.5-mm change in first premolar width, and 7-mm change in first molar width.

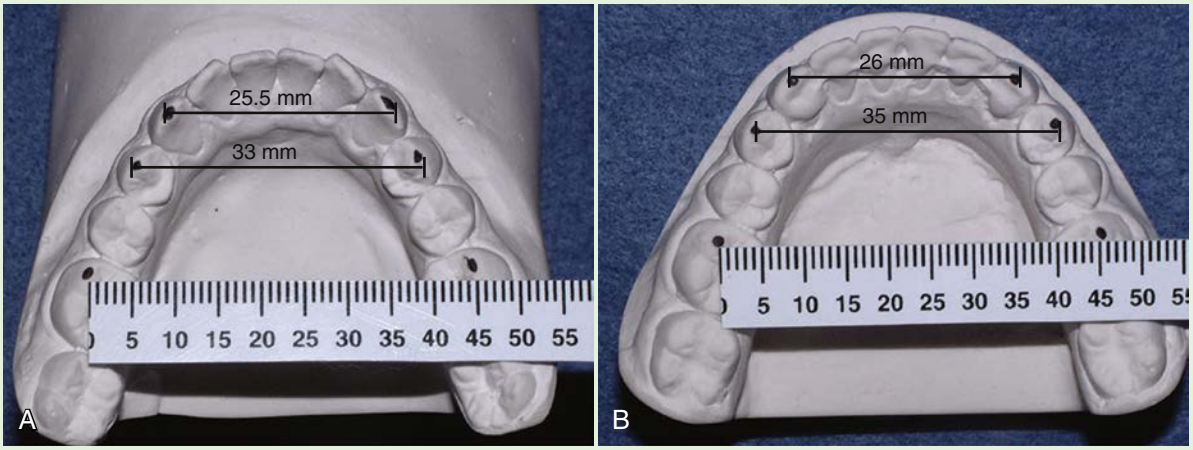


FIGURE 36-67 Comparisons of pretreatment and posttreatment lower arch plaster models for KP. **A,** Pretreatment. Canines: 25.5 mm; first premolars: 33 mm; first molars: 44.5 mm. **B,** Posttreatment. Canines: 26 mm; first premolars: 35 mm; first molars: 45.0 mm. Initial and final models of lower arch show 0.5-mm change in canine width, 2-mm change in first premolar width, and 0.5-mm change in first molar width.

CASE STUDY 36-6 Youth with Herbst Appliance Treatment Demonstrates Typical Response—cont'd

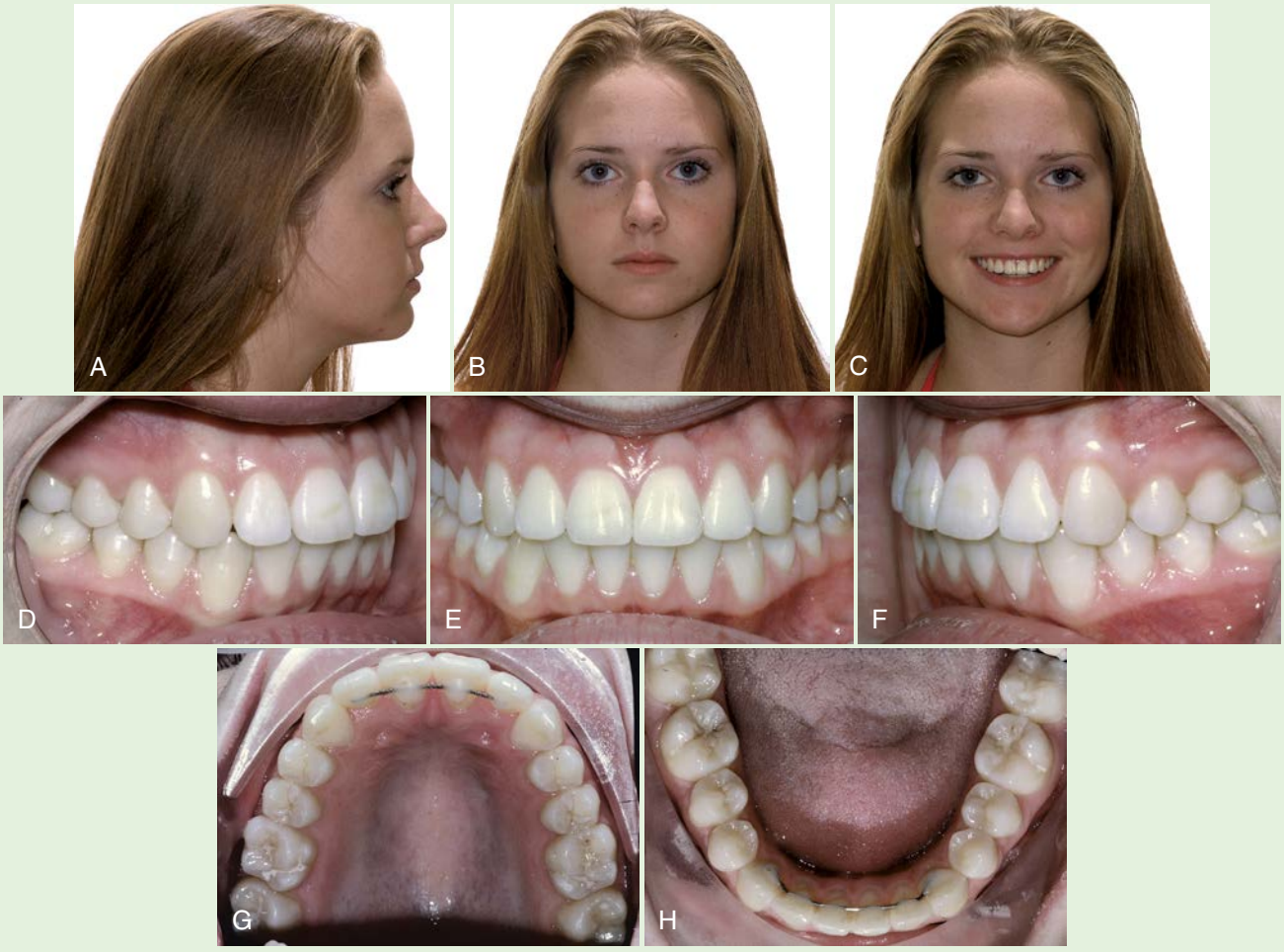


FIGURE 36-68 A-H, Retention records for KP at 1 year in retention.

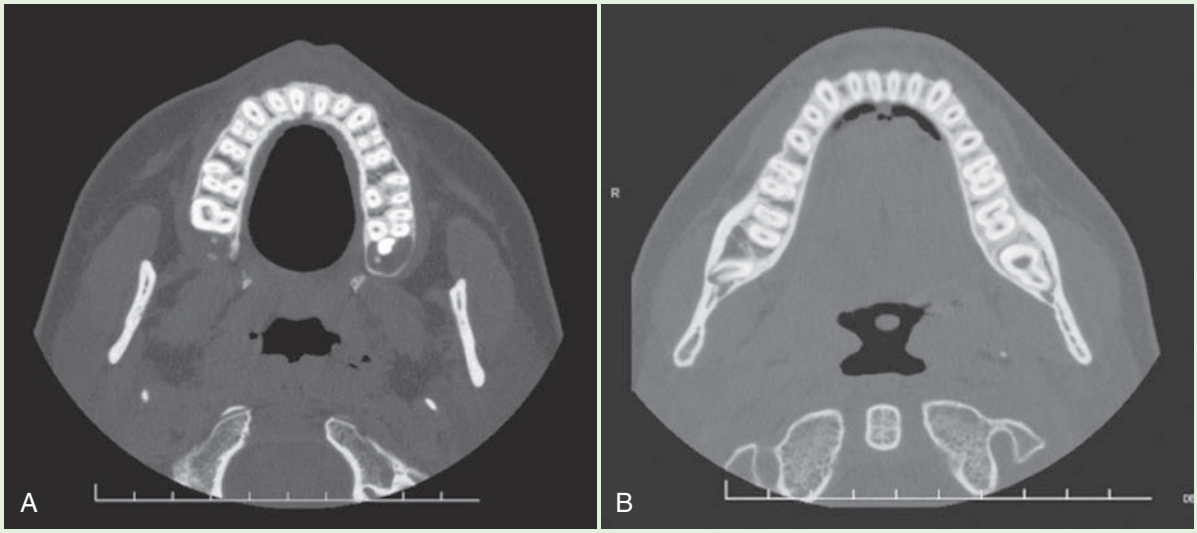


FIGURE 36-69 Horizontal computed tomographic scans of KP's upper arch (A) and lower arch (B) taken after 2 years, 6 months in retention.

CASE STUDY 36-7 Youth with Herbst Appliance Treatment Demonstrates the Definitive Response

This case was selected because it definitively demonstrates what the Damon System has to offer: a gold standard for gaining space in a full Class II occlusion. By simply normalizing the position of the mandible, the muscles of the face and tongue have a completely different impact on the surrounding structures, which gives the patient a second chance for normal physiologic adaptation to take place. This case is also a great example of form after function. What is exciting is that with a little time and effort, using simple mechanics, and allowing physiologic adaptation to occur, the patient can be treated with respect for the maturing face and profile.

Case 7 (KR) was 11 years, 6 months of age with a Class II, Division 2 crowded dentition (Fig. 36-70). She had a prominent nose but a good chin button. She lacked lateral facial support and arch length and width in both

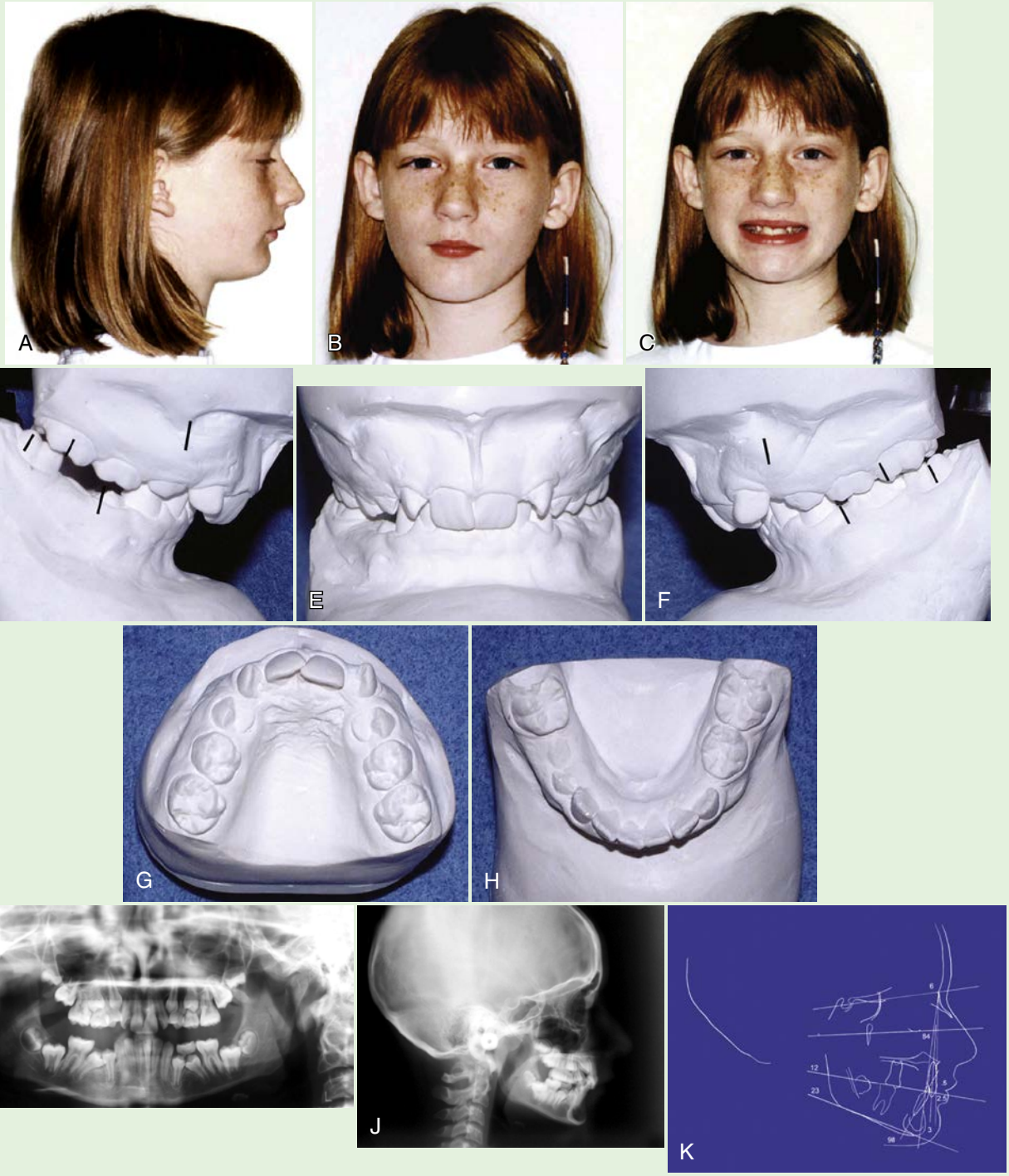


FIGURE 36-70 **A-M,** Pretreatment records for case 7 (KR, age 12 years, 5 months). Diagnosis: Class II, Division 2 crowded dentition; retrusive mandible and lacking lateral facial support, arch length and arch width in both arches; 100% overbite, overerupted upper and lower incisors; prominent nose and good chin button.

CASE STUDY 36-7 Youth with Herbst Appliance Treatment Demonstrates the Definitive Response—cont'd

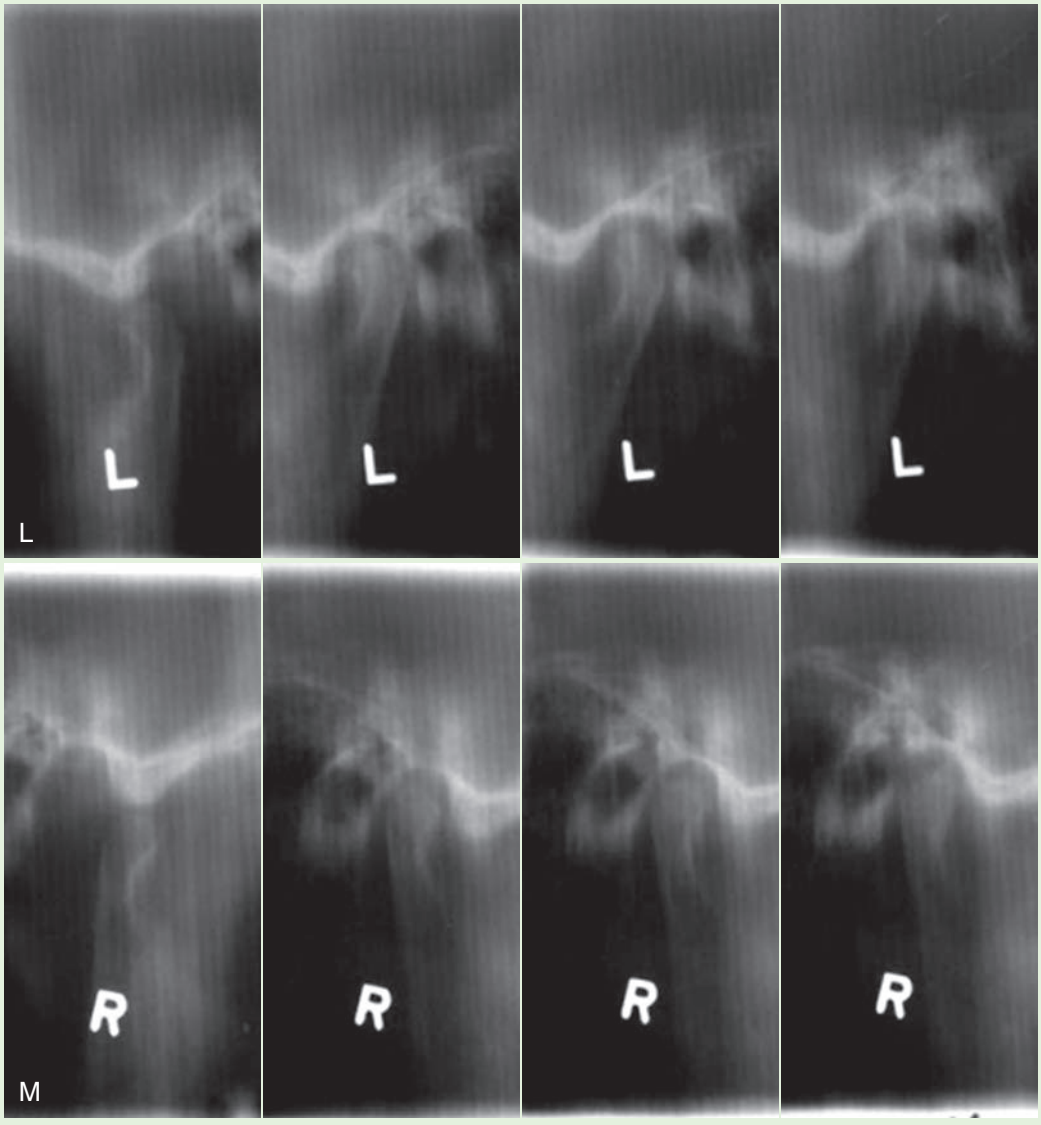


FIGURE 36-70, cont'd

arches. She had 100% overbite with overerupted upper and lower central and lateral incisors. Her retrusive mandible was likely to become more pronounced with maturity. What happens long-term to the maturing profile is well documented. This anticipated profile change calls for treatment mechanics that will move the upper incisors forward, giving increased dental support to the upper lip and, at the same time, helping minimize the prominence of the nose. As the nasolabial angle decreases, room is being made for the erupting upper canines. This anterior dental movement also allows room for Herbst appliance therapy to slowly position the mandible forward into a more normal anteroposterior position. In other words, the lower arch is fitted to the upper as much as growth and development will allow.

The treatment objectives were as follows:

1. Design treatment mechanics in anticipation of the long-term impact on the patient's face and profile.
2. Achieve facial balance and symmetry.
3. Establish maxillary anteroposterior position and posterior width to support the midface.
4. Gain maxillary arch length.
5. Establish ideal maxillary tooth-to-lip relationship.
6. Achieve physiologically sound tooth position.
7. Design treatment mechanics to position the deficient mandible to the maxilla as growth and development permit.

Tomograms were taken before starting phase 1 Herbst appliance therapy. Special high-torque central incisors (17 degrees) and lateral incisors (10 degrees) were selected. These central torques are selected for Division 2 cases. High torques are also the choice for central and lateral incisors for cases requiring Class II elastics to prevent loss of torque control from the elastic wear; they also are suitable for most extraction cases to prevent loss of torque control when retracting anterior teeth. At the start of treatment, a maxillary 0.014-inch Ni-Ti Align SE sectional archwire was placed lateral to lateral incisor, and the Herbst appliance was placed

Continued

CASE STUDY 36-7 Youth with Herbst Appliance Treatment Demonstrates the Definitive Response—cont'd

and advanced just short of the incisors. At the second appointment (2 months, 2 weeks), a 0.017- × 0.025-inch TMA archwire (Ormco Corporation, Orange, CA) with light intruding bends was placed. Light intrusion bends were used to minimize molar tube binding, and medium-light Ni-Ti coil springs were placed on the wire (engaging the molars) and activated twice the width of the bracket. Light C-chains were placed lateral to lateral incisor to consolidate space. Box 36-8 presents the phase 1 Herbst appliance treatment sequence.

Phase 1 therapy was completed in 18 months with nine appointments. KR had responded well with impressive lateral arch development resulting from the Fränkel effect of the Herbst appliance and archwire —a natural expander without force. Initial and after treatment, Herbst appliance upper model comparisons (see Fig. 36-74) reveal increases of 8 mm in first premolar width and 6 mm in first molar width, with Figure 36-75 illustrating the posttreatment Herbst appliance cephalometric tracing composite. Before the use of the Herbst appliance and tube technology, the author's usual treatment options were headgear, upper first premolar extractions, or distalizing upper molars to make room for blocked-out canines. These types of treatment had a detrimental long-term impact on the nasolabial angle and profile of the maturing face.

Phase 2 treatment was immediately initiated after phase 1, with full bonding of upper and lower arches and placement of 0.014-inch Ni-Ti Align SE archwires. Phase 2 treatment was completed in 14 months, 2 weeks with eight appointments. Box 36-9 shows the phase 2 Herbst appliance posttreatment sequence. Posttreatment records (see Fig. 36-76) include a cephalometric tracing composite and a panoramic radiograph. Comparing these records with pretreatment records reveals a pleasing result that accommodated the maturing profile changes seen in retention. Initial and final upper model comparisons (Fig. 36-77) reveal increases of 11 mm in first premolar width, 10 mm in second premolar width, and 7.5 mm in first molar width. Bodily movement and limited corrective tipping are apparent. Lower model comparisons (Fig. 36-78) show a 0.5-mm arch width increase in the canines and 5-mm width increase in the first molars.

With the Damon System, teeth can be moved and bone will follow; however, the muscles must be given time to adapt, which is why a splint was required for retention. The Damon splint (See Finishing Phase discussion) is made for nightly wear to provide an activator-type effect until the patient has completed growing. The author uses splints in 30% of all cases, 100% of Herbst appliance and Class II elastic cases, and for anyone with muscle dysfunction or temporomandibular joint problems. KR wore her splint at night for 12 months after debonding.

Maxillary retention was achieved with 0.016- × 0.022-inch Bond-a-Braid braided wire bonded to each tooth lateral to lateral incisor and left in place for approximately 2 years. Permanent mandibular retention was achieved with 0.026-inch stainless steel wire placed canine to canine and bonded to only the canines. Clear slipcover retainers were fabricated for use after nightly splint wear. Figure 36-79 shows facial photographs 20 months in retention. Horizontal CT scans of upper and lower arches (Fig. 36-80) were taken 3 years in retention. The healthy alveolar bone and excellent archforms are evident.

BOX 36-8 Case 7 (KR) Treatment Sequence: Phase 1 Herbst Appliance

Start: Took tomograms.
First appointment: Placed Herbst appliance, and bonded upper central and lateral incisors (high torques). Placed 0.014-inch Ni-Ti Align SE sectional archwire lateral to lateral incisor. Advanced Herbst appliance just short of incisors (Fig. 36-71).
Second appointment at 2 months, 2 weeks: Placed maxillary 0.017- × 0.025-inch TMA archwire with light intruding bends. Placed medium-light Ni-Ti coil springs and activated 2 times width of bracket. Placed light C-chain lateral to lateral incisor to consolidate space (Fig. 36-72).
Third appointment at 5 months: Activated Herbst appliance with shims just short of anterior teeth. Placed maxillary 0.019- × 0.025-inch TMA archwire with light intruding bends. Activated medium-light Ni-Ti coil springs.
Fourth appointment at 7 months, 2 weeks: Added 3-mm shims, and checked length of archwire in molar tube.
Fifth appointment at 10 months: Replaced maxillary 0.019 × 0.025 TMA with a longer one.
Sixth appointment at 12 months, 2 weeks: Added 3-mm shims.
Seventh appointment at 15 months: Checked appliance and increased intrusion bends in upper archwire.
Eighth appointment at 16 months, 2 weeks: Took tomograms and added 1-mm shims (Fig. 36-73).
Ninth appointment at 18 months: Removed Herbst appliance, took progress records (Figs. 36-74 and 36-75), conducted progress consultation, and arranged for full bonding.

FIGURE 36-71 First appointment. Herbst appliance disarticulated and bonded upper anteriors are used to start KR in phase 1 treatment.

FIGURE 36-72 Second appointment at 2 months, 2 weeks for KR.

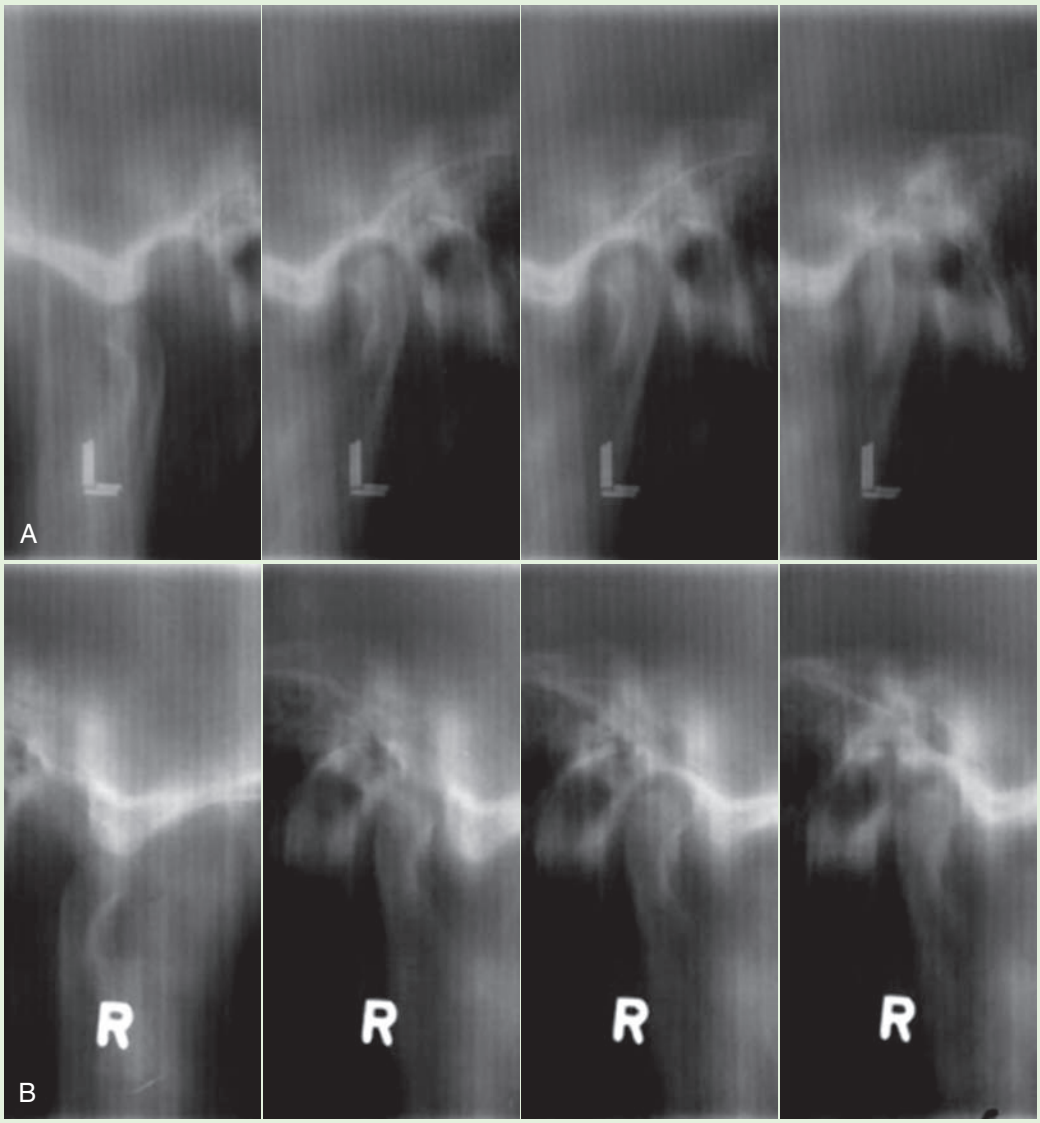


FIGURE 36-73 A, B, Tomograms taken during the eighth appointment at 16 months, 2 weeks for KR.

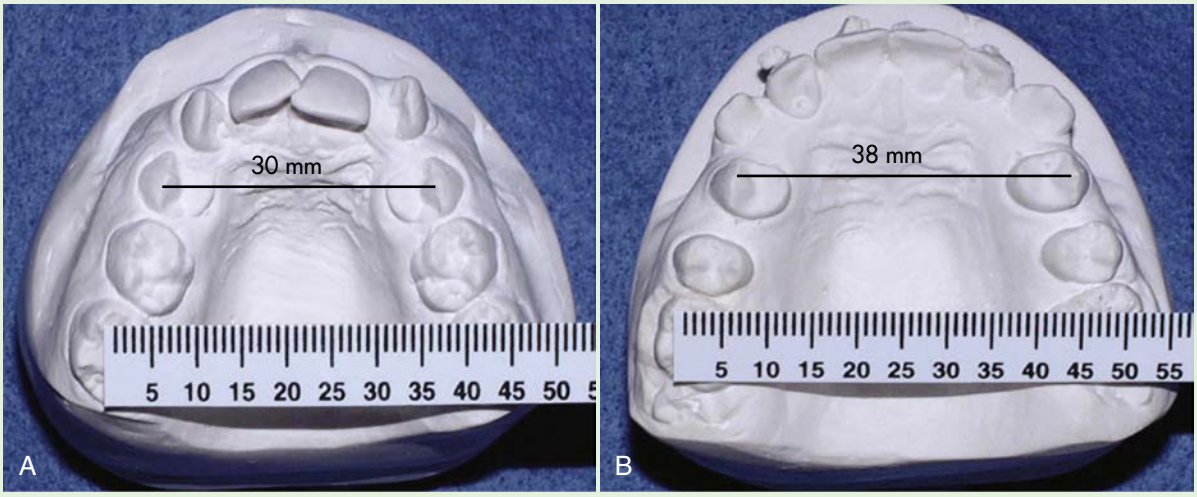


FIGURE 36-74 Comparisons of pretreatment and posttreatment with Herbst appliance upper arch plaster models for KR. A, Pretreatment. First premolars: 30 mm; first molars: 45 mm. B, Posttreatment. First premolars: 38 mm; first molars: 51 mm. Before and after treatment with Herbst appliance plaster models of upper arch show 8-mm change in first premolar width and 6-mm change in first molar width.

Continued

CASE STUDY 36-7 Youth with Herbst Appliance Treatment Demonstrates the Definitive Response—cont'd

FIGURE 36-75 Cephalometric tracing composite at posttreatment with Herbst appliance for KR.

BOX 36-9 Case 7 (KR) Treatment Sequence: Phase 2 After Herbst Appliance Treatment

First appointment: Fully bonded upper and lower arches, and placed 0.014-inch Ni-Ti Align SE archwires.

Second appointment at 2 months, 2 weeks: Placed maxillary 0.016- × 0.025-inch and mandibular 0.014- × 0.025-inch Ni-Ti Align SE archwires.

Third appointment at 5 months: Placed maxillary 0.019- × 0.025-inch posted stainless steel archwire and mandibular 0.018- × 0.025-inch Ni-Ti Align SE archwire. Took panoramic radiograph and checked root position and bracket placement.

Fourth appointment at 7 months, 2 weeks: Placed mandibular 0.019- × 0.025-inch posted stainless steel archwire, and started full time Class II elastics.

Fifth appointment at 10 months: Adjusted maxillary archwire, and continued full time Class II elastics.

Sixth appointment at 11 months, 2 weeks: Adjusted archwires, and cut Class II elastic wear to half time.

Seventh appointment at 13 months: Adjusted maxillary archwire torque, and added bilateral V-elastics.

Eighth appointment at 14 months, 2 weeks: Debonded both arches. Bonded 0.016- × 0.022-inch Bond-a-Braid braided wire onto the maxillary teeth lateral to lateral incisor and placed a 0.026-inch stainless steel wire onto the mandibular teeth canine to canine, bonding only the canines. Made upper and lower slipcover retainers for full-time wear and Damon splint for nightly wear (Fig. 36-76).

CASE STUDY 36-7 **Youth with Herbst Appliance Treatment Demonstrates the Definitive Response—cont'd**

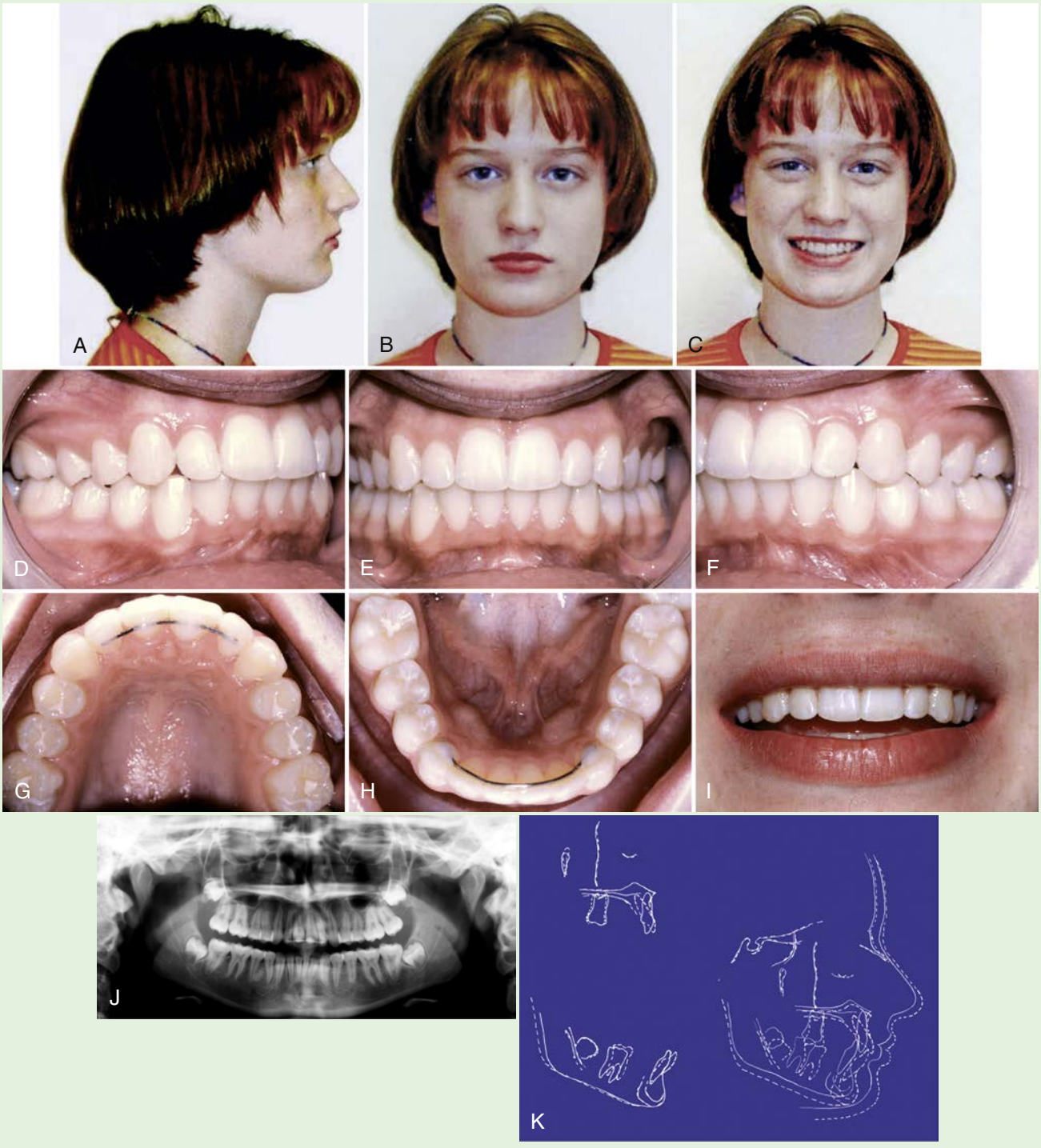


FIGURE 36-76 A-K, Posttreatment records for KR. Total treatment time: 14 months, 2 weeks with eight appointments.

Continued

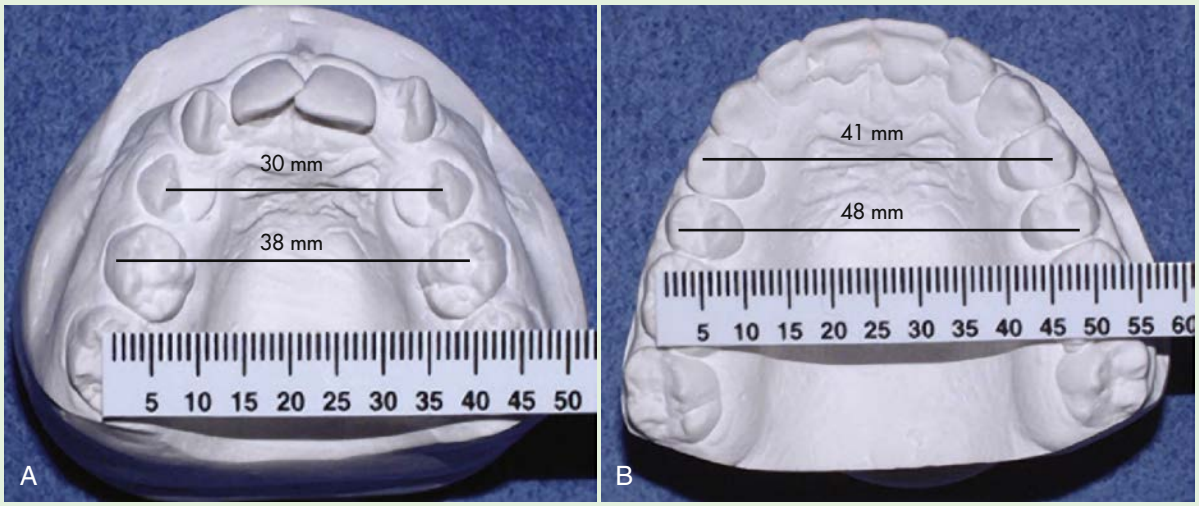


FIGURE 36-77 Comparisons of pretreatment and posttreatment upper arch plaster models for KR. **A,** Pretreatment. First premolars: 30 mm; second premolars: 38 mm; first molars: 45 mm. **B,** Posttreatment. First premolars: 41 mm; second premolars: 48 mm; first molars: 52.5 mm. Pretreatment and posttreatment plaster models of upper arch show 11-mm change in first premolar width, 10-mm change in second premolar width, and 7.5-mm change in first molar width.

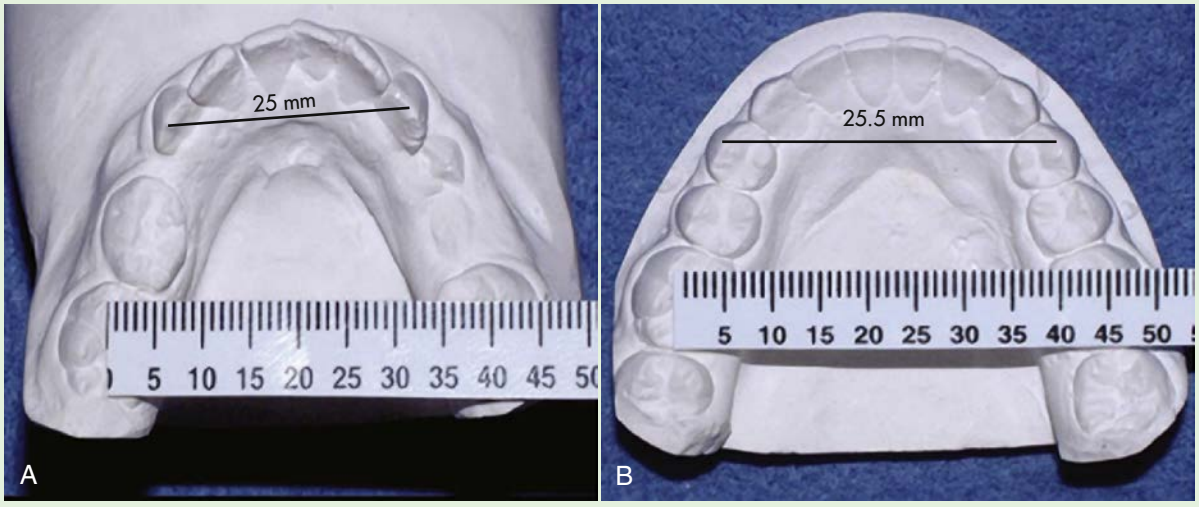


FIGURE 36-78 Comparisons of pretreatment and posttreatment lower arch plaster models for KR. **A,** Pretreatment. Canines: 25 mm; first molars: 41 mm. **B,** Posttreatment. Canines: 25.5 mm; first molars: 46 mm. Pretreatment and posttreatment plaster models of lower arch show 0.5-mm change in canine width and 5-mm change in first molar width.

FIGURE 36-79 **A-C,** KR at age 16 years, 3 months with longer than 20 months in retention.

CASE STUDY 36-7 **Youth with Herbst Appliance Treatment Demonstrates the Definitive Response—cont'd**

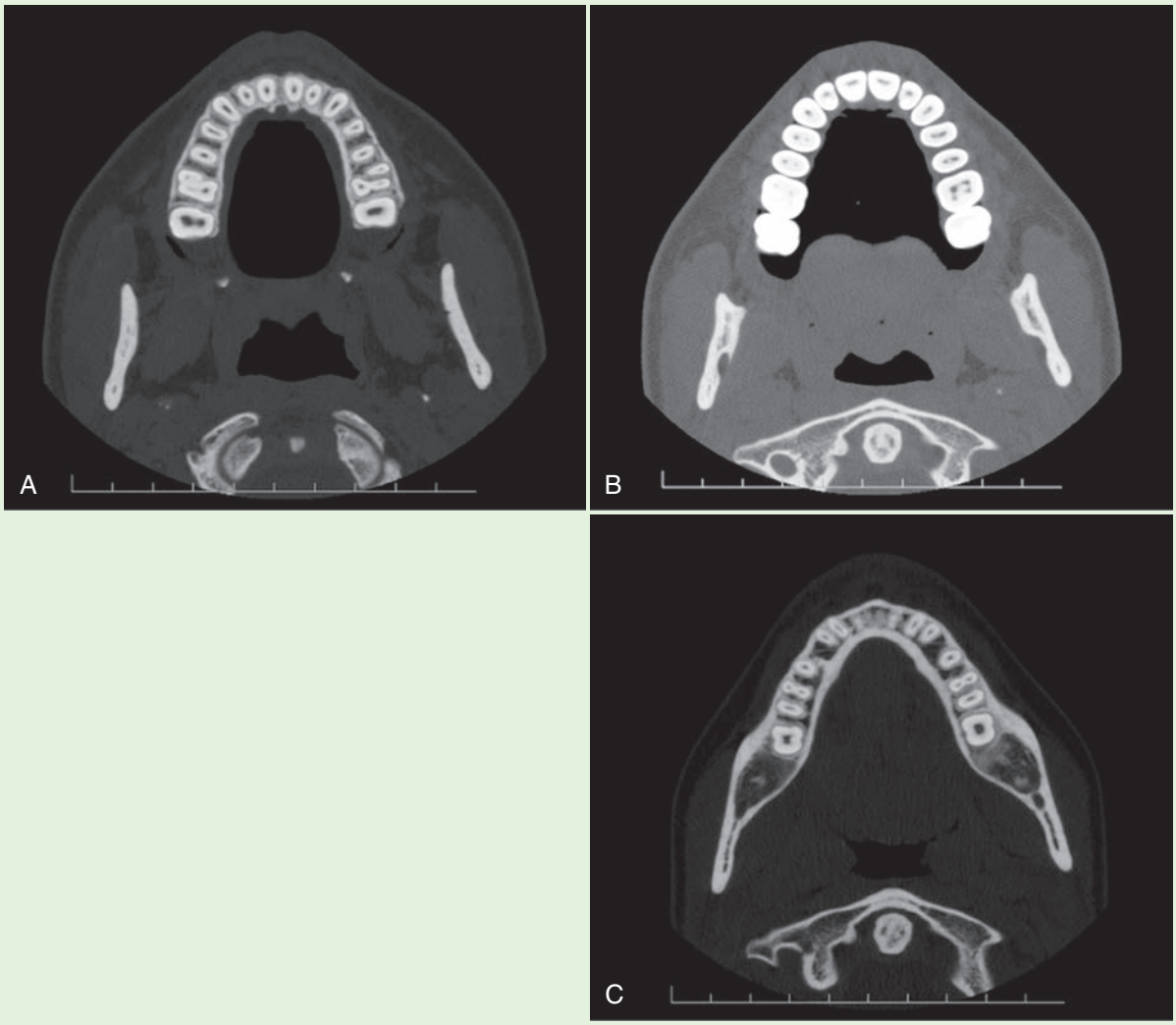


FIGURE 36-80 A-C, Computed tomographic scans of KR's upper and lower arches 3 years in retention. The healthy medullary bone and excellent archforms are evident.

DAMON SYSTEM ESSENTIALS

Damon System Appliance

The preadjusted Damon appliance (Fig. 36-81) is available in 0.022- and 0.018-inch slots. The author prefers the 0.022-inch dimension because the larger slot allows greater flexibility in the selection of archwire sizes and materials. Considering the archwire progression possibilities for each size, the author believes that the 0.022-inch slot maximizes the potential for keeping forces in the optimal force zone. A small wire in a large lumen is most favorable for tooth movement because it diminishes the divergence of angles in the archwire slot, allowing freer movement. Those who prefer the 0.018-inch slot to work with lighter forces will find that with the Damon System, the 0.022-inch slot affords that advantage. When used with the recommended archwire progression, the greater play allows faster, unrestricted tooth movement and a more comfortable patient.

The Damon tube is manufactured by metal injection molding, which is the most precise process today to manufacture metal brackets and tubes. The metal injection molding process makes it possible to manufacture exceedingly small, accurate parts that allow movement of the slide and to provide the close tolerances of the archwire slot. Opening the slide in the latest D3 version is achieved with an opening tool, whereas closing requires only finger pressure. Upper tubes open incisally and lower tubes open gingivally to provide the best visibility when checking archwire placement.

Damon Standard Prescription

The Damon standard prescription is recommended for all molars and premolars, all incisors and canines in good position, and labially inclined canines (Table 36-1). Careful selection of specific torque prescriptions is strongly encouraged before starting treatment.

A B

FIGURE 36-81 The Damon 3 tube and bracket in the (A) open and (B) closed positions.

TABLE 36-1 Standard Torques for the Damon Prescription

	Torque (in degrees)	Tip (in degrees)	Rotation (in degrees)
Upper Arch			
Central incisors	+12	+5	0
Lateral incisors	+8	+9	0
Canines	0	+6	0
First premolars	−7	+2	0
Second premolars	−7	+2	0
First molars	−9	0	10
Second molars	−9	0	5
Lower Arch			
Central incisors	−1	+2	0
Lateral incisors	−1	+2	0
Canines	0	+5	0
First premolars	−12	+2	0
Second premolars	−17	+2	0
First molars	−30	+2	0
Second molars	−10	0	5

High-Torque and Low-Torque Alternatives to the Damon Standard Prescription

Incisors and canines are available with torque variances for specific malalignments to shorten treatment times through greater torque control.

Upper central incisors

- +17-degree torque, +5-degree tip, 0-degree rotation: Division 2 cases, cases requiring extensive Class II elastics, and most extraction cases
- +7-degree torque, +5-degree tip, 0-degree rotation: central incisors requiring extensive uprighting, cases requiring extensive Class III elastics, extremely crowded cases combined with anterior tongue thrusting or thumb or finger habit, and when extensive arch length needs to be gained and the incisors have near normal angulation

Upper lateral incisors

- +10-degree torque, +9-degree tip, 0-degree rotation: Division 2 cases, cases requiring extensive Class II elastics, and most extraction cases
- +3-degree torque, +9-degree tip, 0-degree rotation: lateral incisors requiring extensive uprighting, blocked lateral incisors in lingual crossbite that will have too much torque from standard prescription as they move into normal position, cases needing extensive Class III elastics, extremely crowded cases combined with anterior tongue thrusting or thumb/finger habit, and where extensive arch length needs to be gained and the incisors have near normal angulation

Upper and lower canines

- +7-degree torque, +6-degree tip, 0-degree rotation: canines needing coronal uprighting and most extraction cases requiring first premolar space closure

Lower central and lateral incisors

- −6-degree torque, +2-degree tip, 0-degree rotation: extreme crowding in lower anterior segment, cases needing extensive Class II elastics, any lower incisor initially positioned lingual with labial root position, and cases needing a Herbst appliance on the archwire

Damon System Archwires and Archwire Sequencing

The Damon System has been carefully designed and developed to take advantage of play in the mechanical system and yet maintain superb control of teeth in the working and finishing stages of treatment. The amount of play between the bracket tube and archwire is critical and must be understood to take advantage of this new technology. Archwire sequencing is a critical element in managing clinical forces. In the early aligning phase, minimization of binding and friction is important and accomplished by placing a small high-tech archwire in a large lumen or tube of the bracket. This method is particularly significant in the lower anterior segment where teeth are usually crowded and the interbracket distance is minimal. The applied force can be less if the divergence of the bracket slots is minimized when a small flexible archwire is positioned on either side of the large bracket tubes. This concept is similar to driving a car down a winding highway by using both sides of the road to minimize the curves and to allow for faster travel. This mechanical advantage allows the clinician significant advantages to apply a force just high enough to stimulate cellular activity without crushing the lacelike network of blood vessels around the roots in the periodontal ligament and surrounding alveolar bone. One of the key mistakes clinicians make when starting to use this technology is not allowing enough time for

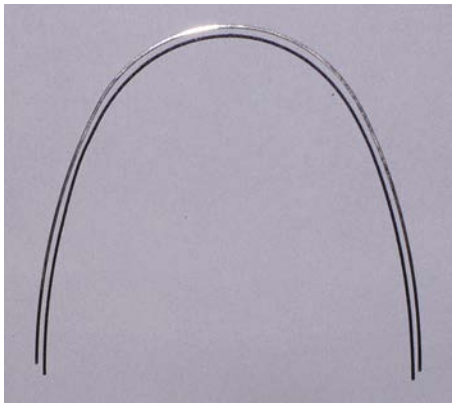


FIGURE 36-82 The shape of the working and final archform are copied in stainless steel from the lower archform that results from the high-tech archwire edgewise phase of treatment.

these light archwires to work. The author encourages clinicians to resist the temptation to change archwires constantly. Proffit stated, "Activating an appliance too frequently, short circuiting the repair process, can produce damage to the teeth or bone that a longer appointment cycle would have prevented or at least minimized."[17] Observing the root proximity in or adjacent to the alveolar cortical plate on lower CT scans makes a strong case for using biologically sensible forces and paying close attention to how forces are applied. Most clinicians using traditional mechanics select archwires based on patient tolerance rather than selecting forces based on biologic principles. Those clinicians who use conventional archwire sequencing or force archwire changes in the bracket tubes *severely compromise the clinical response of this new technology*.

The Damon archform has been developed over many years but is used in a single form and size only during the first two phases of high-tech archwires. The first two high-tech archwire phases are intended to give the body a second chance to find a new physiologically determined tooth position and archform dictated by the muscles of the face and tongue, bone, and soft tissue. This is a functional or natural archform, not a forced archform. The art of new clinical orthodontics is to learn how to read and react to how the body responds to these very light forces. The teeth are not being driven into a predetermined archform. *The shape of the working and final archform is copied from the lower arch after the high-tech edgewise phase* (Fig. 36-82). *Instead of treating to a small, medium, and large archform, each archform is patient specific* (Fig. 36-83). *After using this new technology for more than 8 years, the author realized that predicting the shape and size of the final archform is impossible before treatment*. This is a significant advantage over traditional mechanics and other current treatment techniques. (Note that the Ni-Ti wires specified throughout the cases presented are Ni-Ti Align SE wires. The author has worked with Ormco to develop Copper Ni-Ti archwires for the initial light round wire and high-tech edgewise phases that the author now uses.)

Light Round Wire Phase

By the end of the light round wire phase, the orthodontist will have achieved significant leveling and aligning, begun rotational control, and begun arch development.

FIGURE 36-83 Examples of two cases treated with the same high-tech archform.

The orthodontist should consider the following:

- Use a 0.014-inch Copper Ni-Ti initial archwire to start tooth movement, level, begin archform development, and prepare for the next archwire.
- Use a 0.016-inch Copper Ni-Ti archwire for a second archwire in severely crowded adult patients who are not ready for the second phase archwire.

High-Tech Edgewise Phase

By the end of the high-tech edgewise phase, the orthodontist will have completed leveling and aligning, completed rotational control, begun torque control, begun space consolidation, and continued arch development.

The orthodontist should consider the following:

- Place 0.016- × 0.025-inch Copper Ni-Ti wires in primarily young patients with well-prepared upper arches.
- Place 0.014- × 0.025-inch Copper Ni-Ti when archwire is too difficult to engage in the upper arch. In all lower arches, because of interbracket distance, 0.014- × 0.025-inch Copper Ni-Ti is strongly recommended. In all adults, 0.014- × 0.025-inch Copper Ni-Ti is recommended as the second archwire in upper and lower arches. Space is consolidated in the anterior segments.
- Use a 0.018- × 0.025-inch Copper Ni-Ti archwire after a 0.014- × 0.025-inch Copper Ni-Ti archwire. The archwire is well suited for preparing for the insertion of the working stainless steel wire.
- A 20-degree pretorqued reverse curve 0.017- × 0.025-inch Copper Ni-Ti archwire serves well as a Division 2 second wire. Use the same wire without the torque if only intrusion is needed. A 0.019- × 0.025-inch version of the same wire is available for more challenging cases or a follow-up archwire.
- Take a panoramic radiograph, and assess root positions after the use of high-tech edgewise wire to determine the need to reposition brackets.

Major Mechanics Phase

By the end of the major mechanics phase, the orthodontist will have completed or nearly completed torque control, completed space consolidation, corrected buccolingual discrepancies, and finalized the patient-specific archform.

A 0.019- × 0.025-inch posted stainless steel archwire is used to maintain arch integrity and should be used during anteroposterior correction and closure and also to maintain the anterior vertical and posterior buccolingual.

Finishing Phase

By the end of the finishing phase, the orthodontist will have conducted final detailing.

In most cases the working archwires are kept in place to finish the case. If more bending or torquing of archwires is needed to finish, then the orthodontist should use 0.019- × 0.025-inch or 0.017- × 0.025-inch TMA archwire.

Tieback Usage with the Damon System

Once all the spaces are closed, modules, elastic chains, or ligature wires are typically used in the major mechanics and final stages of treatment to keep all spaces closed. Available in a variety of sizes and strengths, tieback modules are extended intraarch from the posted hook on a 0.019- × 0.025-inch archwire, occlusally passing beneath the first premolar tie-wings to the hook of the first molar in the same arch. Tiebacks are strongly recommended in this low-force, low-friction system because space easily opens.

Using Elastics with the Damon System

Many ways exist to achieve good clinical results with elastic wear. The elastics most frequently used in the author's clinic have proved effective in the passive self-locking system. The author keeps treatment mechanics simple and has been impressed with what can be achieved with elastic wear when biologically sensible forces are applied to a mechanical system with greatly reduced friction and binding. Posts on the archwires used for elastic wear are preferred so as to evenly distribute force over all teeth. Eliminating bracket hooks keeps the appliance clean and more comfortable and promotes healthy tissue response. Because the bracket is self-ligating, the tie-wings are free for elastic attachment.

Lingual Retainer Wire and Splint Retainer

Using this tube technology and low-force, low-friction mechanics, clinicians now are able to treat many patients who, in the past, would have been scheduled for surgery. To retain these challenging patients who have had severe muscle imbalances, the author strongly recommends the placement of temporary upper and permanent lower lingual retainer wires and nightly use of a splint.

As an outstanding muscle trainer, the splint not only maintains the alignment of teeth and dental arches, but it also helps maintain the orientation of the occlusion. Approximately 30% of the author's patients today are retained with this special splint for at least 1 year or longer. *Without the use of the splint, the long-term stability of many cases would be compromised.* Surprisingly, patients adapt well to splints, and many mention an improvement in nightly sleep patterns.

Splints are recommended in patients with:

1. Severe muscle dysfunction (buccolingual coordination challenges)
2. Herbst appliance
3. Severe posterior crossbite
4. Lateral tongue-thrust
5. Severe Class II or III dentitions corrected with elastics
6. Temporomandibular joint disorders
7. Some sleep apnea

For maximum retention, the orthodontist places lingual retention wires after debonding and bonds a 0.016- × 0.025-inch Bond-a-Braid braided wire to each upper tooth lateral to lateral incisor. (The orthodontist removes the upper retention wire 2 to 3 years into retention, depending on the severity of the original anterior tooth position and patient stability in retention.) The orthodontist takes an impression of the lower arch. The orthodontist fabricates a canine-to-canine lingual retainer on the model with a 0.026-inch round stainless steel wire. In normal cases the orthodontist bonds the retainer to only the canines. In severe cases the orthodontist bonds the retainer to each tooth canine to canine. (The lower lingual retainer is permanently left in.)

After the lingual bonded retainers are in place, the orthodontist takes impressions of the upper and lower dental arches. Thick pink base plate wax is heated in a hot water bath and folded end to end twice to create four thicknesses of wax. (The orthodontist must make certain that the wax is soft to prevent distortion of the condyle when taking a bite registration.) The orthodontist instructs the patient to bite slowly into the wax, carefully aligning the midlines and leaving 3 to 4 mm of bite opening for airway and thickness of the upper and lower retainer materials. In all Class II retention cases, the orthodontist takes the bite registration in a slightly overcorrected mandibular position. Figure 36-84 illustrates the fabrication and seating of this valuable retention splint.

FIGURE 36-84 Fabrication of the splint retainer. **A,** Trim models and remove any occlusal bubbles. Cut groove in base of models with acrylic bur in alveolar ridge area *(arrows)*. **B,** Apply sticky wax to the models together with three toothpicks. Paint Al-Cote separating agent (Dentsply, York, PA) on both model bases. **C,** Mount upper and lower models on simple articulator. **D,** Carefully separate models from their bases at the separating agent joint. **E,** Remove plaster from the center of both models, and use vacuum application of Essex A+ (Raintree Essix, Metairie, LA) or Biocryl (Great Lakes Orthodontics, Tonawanda, NY). Block out undercuts with Wonderfill (Dental Creations, Waco, TX). **F,** Trim retainer material on the model, and place the models back on the articulator with sticky wax. **G,** Mix acrylic, and place it between the models from middle canine to posterior canine on the buccal and lingual aspects. **H,** Leave airway in anterior area canine to canine. Amount of airway is determined by the needs of the patient. **I,** Place acrylic beyond the tip of the upper canine. Leave it short of the cusp tip *(arrow)*. A fracture line sets up if patients clench their teeth. **J,** Relieve any undercuts with acrylic bur on upper canine. **K,** Note how tongue is contained *(arrows)*.

SUMMARY

The philosophy underlying the intended use of the Damon System is to approximate the forces that normal functioning muscles have on tooth position and the developing archform. The system is a blend of high-technology archwires and passive self-ligating tubes (brackets) carefully matched to produce great finishes while maintaining an appreciable amount of play in the mechanical system, especially in the initial stages of treatment. This new way of thinking about clinical mechanics is based on trying to achieve the following two clinical principles:

1. Do not overpower the biologic system with high-force mechanics during any phase of treatment.
2. Work with the orofacial musculature, allowing it to determine the new physiologically based tooth position and archform balanced among the facial muscles, tongue, bone, and soft tissue.

For many years, clinicians have based most of their treatment planning decisions on maintaining the patient's original archform. Determining the appropriate force level was often based on patient tolerance rather than its impact on the biologic cellular composition of bone and tissue. Conventional thinking also ascribed stable results to the elimination of tooth mass to maintain the original archform. Long-term retention studies have not shown a statistically significant correlation between tooth extraction and stability. For years, orthodontists have observed that many malocclusions are simply the result of functional abnormalities. With muscle-imbalance corrective appliances, Fränkel demonstrated that the posterior archform could be altered long-term stability. The reason why the Damon System appears to have an effect similar to the Fränkel effect with its concomitant impact on the physiologic adaptation of the muscles is not completely understood. With the use of very light, biologically sensible forces to start to unravel crowding, the changing archform appears to "wake up the tongue" and, in most patients, creates a new balance of the orofacial musculature. This newly created balance of the muscles, bone, and soft tissue is what determines the new tooth position and archform. With the Damon System and the philosophy of low-force mechanics, the art of clinical orthodontics is learning how to read and react to what the body determines for each individual patient. Extensive clinical results indicate that complete dentitions can be maintained, even in many crowded arches. In most cases, the body—not the clinician—determines the final archform.

The most significant impact of this new, passive technology is the myriad of additional options available to the clinician for facially driven treatment planning. Clinicians around the world are acutely aware of the long-term impact of traditional extraction therapy on the maturing face. (The philosophy of the Damon System still strongly recommends extraction therapy in bimaxillary protrusive cases.) By designing new treatment mechanics that consider the biologic impact of low-force treatment over the lifetime of a patient, orthodontists now have the opportunity to consider what the patient will look like at 50 years of age and design treatment plans in which most complete dentitions are preserved.

CT scans taken of patients just debonded and compared with scans taken 5 years and longer in retention show promising results from the low-force therapy offered via the Damon System and its underlying treatment philosophy. The following observations were made from studying the results of Damon System therapy on 13 patients ranging in ages from 13 years, 6 months to 56 years of age:

1. Alveolar bone can be altered and reshaped with low clinical forces.
2. With low-force, low-friction orthodontics via the Damon System, the alveolar bone will allow the movement of teeth with minimal tipping in all planes of space.
3. Alveolar bone is exceedingly thin on the labial, buccal, and lingual aspects of teeth that clinicians are trying to move. This observation calls for low-force therapy that respects the delicate nature of this bony architecture.
4. In many lower arches, pretreatment roots are routinely positioned within the cortical plate. This observation mandates the deployment of extremely low clinical force systems that take into account the low vascularity of cortical plate.
5. The architecture and thickness of alveolar bone appears to improve over time in retention after low-force orthodontics.
6. The low-force mechanics used through the Damon System indicate improved bone response for teeth that have not been allowed to erupt through the facial or lingual cortical plate. For this reason, early tooth guidance is strongly encouraged.
7. Low-force orthodontics *can* have a positive impact on the bone of periodontal patients.
8. Several hundred thousand patients have now been treated worldwide with the Damon System technology. In compromised dentitions, periodontists following complex cases treated with this light-force system have been impressed with the positive bone and tissue response. Although the clinical results are promising, further research and long-term retention studies are encouraged.

Acknowledgment

The author would like to acknowledge Robert Borkowski for his input about the physiologic functioning of tooth movement and contribution to this chapter in that regard, much of which comes from his postgraduate orthodontic thesis.

REFERENCES

1. Pollard A. Capturing the essence of the Damon approach. *Clin Impressions*. 2003;12(2):4–11.
2. Whitehall W. *Personal communication*; March 2004 (Whitehall is former executive vice president of Unitek.).
3. Andreiko C. *Personal communication*; March 2004.
4. Morrow R. *Personal communication*; March 2004.
5. Voudouris JC. Interactive edgewise mechanisms: form and function comparison with conventional edgewise brackets. *Am J Orthod Dentofacial Orthop*. 1997;111:119–140.
6. Kronka MC, Watanabe IS, da Silva MC. Scanning electron microscopy of angioarchitecture of palatine gingiva in young rabbits. *Braz Dent J*. 2001;12(3):163–166.
7. Sandstedt C. Einige Beiträge zur Theorie der Zahnregulierung. *Nord Tandlaek Tidskr*. 1904;5:236–256.

8. Schwarz AM. Tissue changes incidental to orthodontic tooth movement. *Int J Orthod Oral Surg Radiogr*. 1932;18:331–352.
9. Reitan K. Continuous bodily tooth movement and its histological significance. *Acta Odontol Scand*. 1947;7:115–144.
10. Reitan K. The initial tissue reaction incident to orthodontic tooth movement as related to influence of function. *Acta Odontol Scand Suppl*. 1951;6:1–240.
11. Reitan K. Tissue behavior during orthodontic tooth movement. *Am J Orthod*. 1960;46:881–900.
12. Reitan K. Initial tissue behavior during apical root resorption. *Angle Orthod*. 1974;44:68–82.
13. Reitan K, Kvam E. Comparative behavior of human and animal tissue during experimental tooth movement. *Angle Orthod*. 1971;41:1–14.
14. Rygh P. Elimination of hyalinized periodontal tissues associated with orthodontic tooth movement. *Scand J Dent Res*. 1974;82:57–73.
15. Rygh P, Reitan K. Ultrastructural changes in periodontal ligament incident to orthodontic tooth movement. *Trans Eur Orthod Soc*. 1972:393–405.
16. Tuncay OC, Ho D, Barker MK. Oxygen tension regulates osteoblast function. *Am J Orthod Dentofacial Orthop*. 1994;105:457–463.
17. Proffit WR, Fields Jr HW. The biological basis of orthodontic therapy. In: Profitt WR, Fields Jr HW, eds. *Contemporary Orthodontics*. 3rd ed. St Louis: Mosby; 2000:296–325.
18. Borkowski RN. The biologically based case for truly light-force mechanics. *Clin Impressions*. 2004;13(1):19–22.
19. Fränkel R, Müller M, Falck F. The uprighting effect of the Fränkel appliance on the mandibular canines and premolars during eruption. *Am J Orthod Dentofacial Orthop*. 1987;92:109–116.
20. Bagden A. *Personal communication*; March 2004 (Bagden is a practicing orthodontist in Springfield, VA.).
21. Pitts T. *Personal communication*; March 2004 (Pitts is a practicing orthodontist in Reno, NV.).

INDEX

A
AAPD. *see* American Academy of Pediatric Dentistry (AAPD)
Abuse, and dental avulsion, 885
Abutment teeth, parallelism of, adult interdisciplinary therapy in, 571, 573f
AcceleDent device, 918–919, 919f
  effect of, on alignment of lower anterior teeth, 919
  patient's comfort and ease of using, 920
AcceleDent Type I appliance, 93
Acetaminophen, for micro-osteoperforations, 915–916
Acid etching, conventional, for enamel conditioning, 819–820
  alternatives to, 820
  amount of enamel removed by, 820
  gels for, 820
  optimal time for, 820
  prolonged, with fluoride pretreatment, 820
  surfaces for, 820
  on teeth with internal white spots, 820
Acidogenic plaque, reducing amount of, 889–890
  chemical, 890
  mechanical, 890
Ackerman-Proffit classification, 226–229, 230f
Acoustic rhinometry, for upper airway, 327–329, 328f
ACPA. *see* American Cleft Palate-Craniofacial Association (ACPA)
Acquired deformities, patients with, 299
α-Actinin-2, 39
α-Actinin-3, 39
Activa brackets, 732, 732f
Activation moment, 181
Active archwires, friction with
  thick, stiff, 736–737
  thin, flexible, 737–738, 737f–738f
Active clips, 740–741
Active members, of orthodontic appliance, 164
Active self-ligating brackets, new prescriptions for, 479
ADA. *see* American Dental Association (ADA)
Adaptive prescription, in targeted therapeutics, 946–947, 949f
Additive bending, in targeted therapeutics, 951, 953f
Adenoid facies, 320
Adenoidectomy, 343
Adenotonsillar hypertrophy, 335
Adenta, 735
Adequate tools, 397
Adhesives, 193–195, 813–817
  biological properties of, 194–195
  for bonded fixed retainer, 840–841, 843f
  composite resins as, 813–816
  excess, removal of, 830
  for fixed retention, 197b
  glass ionomer cements as, 817, 818f
  grinding of, 195, 195f
  light-polymerized, 814, 815f
  no-mix, 814
  orthodontic resins in, cytotoxicity of, 816
  primers as, 814–815
  residual, removal of, 834–837, 835f–837f
Adjacent tissues, newly positioned teeth and, 982–983
Adolescent patient
  lingual treatment of, 773f–777f, 773b
  management and motivation for, 395–402
    behavior guidance of, 395–396
    communication guidance in, 396–397
    extraoral appliances, 398
Adolescent patient *(Continued)*
    first impressions of, 395–396
    headgear for class II correction, 398
    at home responsibilities, 397–398
    intraoral elastics, 400
    orthodontic appointments, 399–400
    pain management of, 395
    removable intraoral appliances, 398–399
    removable orthodontic appliances, 398
    removable retainers, 400–401
Adrenal insufficiency, 695t
Adult interdisciplinary therapy, 569–620
  in adult orthodontic conditions, diagnosis of, 583–589, 586f
    periodontal differential, 586–589
    skeletal differential, 583–586, 587f–588f, 599f–600f, 602f–605f
    temporomandibular joint differential, 589, 589t, 590f
  clinical management of, 589–596
    biomechanical considerations in, 591–594
  communication in, 614
  current status of, 569–570, 570f–572f, 572t
  in dentoalveolar collapse, 597–606, 598f, 600f, 602f–606f, 602b
  goals of, 570–583
    additional adult treatment, objectives in, 571–583, 573f–582f, 573b, 584f–585f
    in ideal orthodontic treatment, adult patient and, 570–571
    individualized, objectives of, 571
  in orthodontic tooth movement, 594–595
  orthodontist and staff preparation for, behavioral management of, 595–596
  risk management in, 613–615, 615f–618f
  sequence of, 596–597
  stability and retention individualized for, 570f, 578f–580f, 606–613, 606f, 608f–614f
    communicating in, with team members, 613
  summary of, 617
Adult orthodontic conditions, diagnosis of, 583–589, 586f
  periodontal differential, 586–589
  skeletal differential, 583–586, 587f–588f, 599f–600f, 602f–605f
  temporomandibular joint differential, 589, 589t, 590f
Adult patients
  changes, in craniofacial growth and development, 25
  lingual treatment of, 768f–772f, 768b
  treatment objectives, in adult interdisciplinary therapy, 571–583, 573f–582f, 573b, 584f–585f
Aerosol, 195
Aesthetic considerations, in periodontal-orthodontic interrelationships, 642–643, 646f
Aesthetic line, of the dentition, 230f
Aesthetic self-ligating brackets, 735–736
Aesthetic/functional improvement, adult interdisciplinary therapy in, 583, 584f
Agenesis, dental, 42–43
Aggressive periodontitis, 624, 625f
AHI. *see* Apnea-Hypopnea Index (AHI)
Aligning, with lingual appliances, 764–765
  type 1, 765, 765f
  type 2, 765, 766f
  type 3, 765, 767f
Alignment, of active clips and passive slides, 741
Alleles, 34
Allergy-immune problems, 695t
Alloy soldering, 188–189
Alloys
  basic behavior of, 166–167
  beta-titanium (TMA), 168
  elastic limit of, 167
  modulus of elasticity in, 167
  nickel titanium (nitinol), 167
  orthodontic, ideal, 168
  selection of, 169–173
  shape-memory, 167–168
  steel, 167
  stiffness of, 172t
  stress-strain relationship of, 166f
  titanium niobium, 168
Alveolar bone, 54, 56f
  bone resorption and, 54, 56f, 126
  cells of, 92t
  in decortication, 661–662
  decortication of, 479
  depository side of, 54–55
  reduced height of, tooth movement into, 73, 74f–75f
  resorptive surface of, 54–55
  shape of, as indication for micro-osteoperforations, 916–917
Alveolar bone grafting, intermediate/secondary, 378–381
  sequencing, 380–381, 381f
  surgical technique, 381
  timing, 378–379, 380f
Alveolar decortication, 661–665
  alveolar bone biological response in, 661–662
  patient management in, 663
  surgical technique in, 662–663, 662f
  treatment indications and limitations of, 663–665, 663f–664f
Alveolar fracture, 881
Alveolar process, density of, *versus* maxillary molars, 131–132
Amalgam, bonding to, 827–829, 827f–828f
  procedures for, 828, 829f
American Academy of Pediatric Dentistry (AAPD), 395
American Cleft Palate-Craniofacial Association (ACPA), 367
American Dental Association (ADA), 200–201
  Center for Evidence-Based Dentistry, 200–201, 205
Analysis system, differential diagnosis of, 493, 494t
Anchorage
  anterior, increase of, 470, 473f–478f
  appliance, Goshgarian's, 456
  differential, 131–132, 137f
  endosseous implants for, 133
  maximum, 444
  medium, 444
  minimum, 444
  retromolar implant, 138–140, 140f
Andrews, Lawrence, 437, 457
Androgens, in calcium conservation, 117
Anecdotal, evidence in orthodontics, 202, 202f
Anesthetic, for micro-osteoperforations, 914
Angiogenic capillary budding process, in sutures, 119–120, 123f–124f
Angle, Edward Hartley, 200, 481, 482f, 730
  classification system created by, 695
Angle classification, 226, 227f
Angle system, 481–482
Angle's standard appliance, basic components of, 482f
Animal models
  for bone adaptation, remodeling, and modeling, 143–145, 144f–147f
  expedited tooth movement from, 148–149

Page numbers followed by *b, t,* or *f* refer to boxes, tables, or figures, respectively.

Ankyloglossia, 218
Ankylosed primary teeth, 218
Ankylosis, 880
  in unerupted teeth, ectopically positioned and, 650–657, 658f–661f
Anorexia nervosa, 293
Antegonial notching, 229
Anterior anchorage, increase of, 470, 473f–478f
Anterior bite plates, 261
Anterior crossbite, 373–374
Anterior facial height, definition of, 490
Anterior open bite, skeletal tendency toward, 225
Anterior retraction, in extraction treatment, 541
  biomechanics in, 541, 544f–545f
  decision making in, 541
Anterior space analysis, dental disharmony and, 492
Anterior teeth
  crowding of, Schwarz appliance for, 414
  display, 228–229
  size discrepancies and, 983. *see also* Tooth-size discrepancies
Anteroposterior dimensions, 229
Anteroposterior movement, of incisors, 714t
Antibiotics, for dental avulsion, 884
Anticipatory orthodontics, in proactive decision making, 944, 948f
AO-BO relationship, 489
Apical base school, 981–982
Apical root displacement, in external apical root resorption (EARR), 900, 901f–905f
Apnea, 333–334
Apnea-Hypopnea Index (AHI), 334
Appliance
  edgewise, 482
    malocclusion correction with, 502b–506b
    Tweed-Merrifield, 493–495, 494f
  extraoral, 398
  fixed, 592–594
  Goshgarian's anchorage, 456
  improvement efficiency and, 473f–478f, 479
  modification of, in adult interdisciplinary therapy, 596
  night crib, 607–609
  optimal adaptation of, 467
  orthodontic. *see* Orthodontic appliance
  phase I, 592
  removable, 591–592
  sagittal, 591
  selection of, 633f
  sequential, placement, Tweed-Merrifield edgewise appliance and, 495
  stainless steel labially affixed, 592–594
  therapy, arch alteration with, 983
  Tweed-Merrifield edgewise appliance and, 493
  variations of, Tweed-Merrifield edgewise appliance and, 495
Arch
  alteration with appliance therapy and, 983
  depth of, mandibular and maxillary, 23–24
  development of, craniofacial, 23–25, 23f–24f
  ideal dental, 763, 763f
  leveling of, 719
  perimeter of, 23–24, 24f
  width dimensions, 985f
Arch expansion, 233, 412–416
  acrylic splint expander for, 412f, 413, 414f
  lower arch of, 414
  mandibular adaptations of, 414
  mandibular dental uprighting and expansion appliances, 414–416
  maxillary adaptations, 413–414, 413f–414f
  orthodontic expansion, 412
    rationale for early, 412–413
  orthopedic expansion and, 412, 412f
  passive expansion, 412
  permanent dentition, 413
Arch expansion *(Continued)*
  protocols, 413–414
  self-ligating bracket benefits for, 743, 744f
  types of, 412
Arch form, 273, 275f
  selection, in targeted therapeutics, 947
Arch length
  measurements, 985f
  posterior, 493
  width and, 986, 987f
Archwires, 192–193
  bending of, 946–947, 949f, 954–957
    robot for, 927
  design, 937–939, 940f
  first-order compensations using, 763–764, 763f
  malocclusion correction with edgewise appliance and, 507f
  NiTi, clinical parameters on, 193b
  optimization of selection for, in targeted therapeutics, 954–957
  replacement of, minimal, in targeted therapeutics, 947–950, 951f
  robot-assisted modified, 942
  secure engagement of, 736
  self-ligation bracket effects on insertion and removal of, 736
  stainless steel, 189
  Tweed-Merrifield edgewise appliance and, 493
Area of maximum constriction, 323, 326–327
Aromatase, in estrogen biosynthesis, 41
Arousal index, 334
Attached gingiva, 52
Attachment, 788–791, 789f–791f
  beveled, 790–791, 790f–791f, 794–795
  rectangular, 790, 790f
Attention-deficit/hyperactivity disorder, 291
Augmentation grafting, 661–665
  alveolar bone biological response in, 661–662
  patient management in, 663
  surgical technique in, 662–663, 662f
  treatment indications and limitations of, 663–665, 663f–664f
Austenitic active, NiTi archwires, 192–193
Australian didgeridoo, 344
Autoradiography, in bone physiology, 104
Autosomal dominant inheritance, 35, 35f
Autosomal recessive inheritance, 35
Autosomal recessive traits, 36f, 37
Autosomes, 32–33, 33f
Auxiliaries, Tweed-Merrifield edgewise appliance and, 495
Auxiliary appliances, 950
Avulsion, in permanent dentition
  abuse and, 885
  management of, 880–886
    with closed apex, 883
    with open apex, 883–884
  prevention of, 884–885
Axial inclinations, 989
Axial loading, of orthodontic appliance, 165, 165f
Axis inhibitor 2 gene, mutations of, 42–43

B

Backscatter emission, 106
Banner advertising, in orthodontics, 205
Bariatric surgery, 343
Basal bone
  discrepancy, 703
  lower incisors placement in, 983
Basal plate, development of, 8, 8f
Base-wing joint, of stainless steel brackets, 188–189, 189b
BDD. *see* Body dysmorphic disorder (BDD)
Beam filtration, 304
Beck Depression Inventory, 295
Begg, Raymond, 730
Begg brackets, 739
Begg mechanics, with straight-wire control, 757, 758f–759f
Begg pins, 730
Behavior guidance, for child and adolescent patient, 395–396
Behavior management, differences between pediatric dentistry and orthodontics, 396–397
Behavioral disorders, 695t
Bending, 165
Berlin questionnaire, 338, 339t
Beta-titanium (TMA), 168, 169f
Bilateral symmetry, environmental and genetic influences on, 44
Biodigital orthodontics, 926–980
  case study for, 959f–979f, 959b
  in clinical practice, 930–942
  craft-based care model in, 926–927, 928f
  efficacy and effectiveness of, 957
  integrated digital technology platform in, 927–930
  patient-centered model of care, 927, 929f
  proactive decision making and, 942–944
  professional-based model of care, 927, 929f
  and targeted therapeutics, 946–957
Biological factors, of radiation dose risk, 303
Biological responses, by orthodontic forces, 85–87
Biomechanical considerations, in temporary anchorage devices, 511–568, 517f–518f. *see also* Temporary anchorage devices (TADs)
Biomechanics, straight wire, contemporary, 437–454
Biotype, in periodontal-orthodontic interrelationships, 626, 626f
Bipolar disorder, 292–293
Birthday party air balloon, for air volume, 326–327, 327f
bis-GMA/TEGDMA mixture, 193–194
Bisphenol A-glycidyl methacrylate (bisGMA), 813–814
Bisphenol-A (BPA), 195
Bite plane therapy, 592
Bite splint, 706t
Biting strength, of human masticatory apparatus, 119
Bleeding disorders, 695t
Blue light
  biological properties of, 194–195
  effects, 194–195
Bodily movement
  into defects, 637–640, 643f
  tooth movement and, 69, 69f, 76f
Body dysmorphic disorder (BDD), 216–217, 292, 292b
Boil-and-bite guards, 884–885
Bonded retainers, 840–855
  fixed
    adhesives for, 840–841, 843f
    materials for, 840, 842f
    periodontal health with, 841–842, 844f–845f
Bonding, 812–867
  advantages of, 812–813
  to amalgam, 827–829, 827f–828f
    procedures for, 828, 829f
  applications of, 855–861
  area, exposure of, 764
  to artificial tooth surfaces, 825–829, 825f
  bleached teeth and, 820
  to composite restoratives, 829
  conclusions in, 832–833, 862
  for deciduous teeth, 820
  to enamel, 819–824, 822f
    cleaning in, 819
    curing in, 824, 824f
    enamel conditioning in, 819–821
    excess in, removal of, 823–824, 823f
    fitting in, 823
    positioning in, 823
    premedication for, 819
    procedure for, 823
    sealing and priming in, 821, 822f
    transfer in, 823
  to gold, 829, 830f

Bonding *(Continued)*
indirect, 829–831, 831f
advantages of, 830
clinical procedure for, 830, 832f
with composite custom bracket base, 830–831
of large acrylic appliance, 855
for deciduous or mixed, 859–860, 861f
for permanent dentition, 859
technical procedures for, 859–860
materials and devices used in, 813–818
adhesives as, 813–817
brackets as, 813
fluoride in, 893–894, 893f–894f
light sources as, 817–818
light-emitting diodes as, 818, 819f
of molars, 832–833
for occlusal buildup of posterior teeth, 860–861
to porcelain, 825–827
procedures for, 826, 826f–827f
strength of, 833
Bone
alveolar, decortication of, 479
biomechanics of, 118–119, 118f–121f
biotype, 626, 626f
cancellous, 381
contact, of implant, 142
density, and tooth movement, 916–917
harvesting, morbidity of, 381
metabolism, 116–117, 117f
newly positioned teeth and, 982–983
in orthodontic practice, 99–153
physiology of, 104–108
principles of, in alveolar bone, 597, 600f
specific assessment methodology for, 104–106
response, to vibration, biologic mechanism of, 921–923
Bone adaptation, 108–111. *see also* Bone modeling; Bone remodeling
animal models for, 143–145, 144f–147f
Bone grafting, alveolar, intermediate/secondary, 378–381
sequencing, 380–381, 381f
surgical technique, 381
timing, 378–379, 380f
Bone modeling, 110–111, 111f–112f
animal models for, 143–145, 144f–147f
mechanotransduction and, 87–88, 89f
Bone remodeling, 99–100, 100f, 108–110
animal models for, 143–145, 144f–147f
biomechanical control of, 113
in cortical bone, 108–110, 108f
cutting/filling cones in, 110, 110f, 112f
miniscrew implants and, 142
in trabecular bone, 108–110, 109f
types of, 110, 110f
Bone resorption
alveolar bone and, 54, 56f, 126
intermittent *versus* continuous mechanics, 131, 136f–137f
osteoclast recruitment and, 130–140, 136f
periodontal ligament and, 126
tooth movement and, 124–125, 133f
Bone tissue, classification of, 107–108, 107f
bundle bone, 107f, 108
composite bone, 107f, 108
lamellar bone, 107–108, 107f–109f
woven bone, 107, 107f
Bone-anchored maxillary protraction, 431–432
Bone-anchored rapid palatal expansion, in periodontal-orthodontic interrelationships, 629–633, 631f–633f
Borderline cases
in orthognathic surgery, 712
therapeutic diagnosis of, 705–706, 706t, 707f
Brachyfacial, patients, 228–229
Brackets, 721f, 813
active self-ligating, new prescriptions for, 479
base thickness, compensation by adjusting, 763–764, 763f–764f
Brackets *(Continued)*
ceramic, 191–192, 735–736, 813, 813f
design of, 176
extraction, 437
final position of, in target setup, 942, 942f
flared upper incisor (brown), 158, 160f
force system in, path and, 158–159
friction force produced by, 183
metal, 813, 814f
placement, 281–282, 282f
bracket positioning in, 281–282, 283f
stainless steel, 187–190
straight wire appliance and, 437
strategic placement of, and digital diagnostic simulation model, 930–934
titanium, 191
translation, 437
Tweed-Merrifield edgewise appliance and, 493
Braided wires, 171, 172t
Brain injury, acquired deformities and, 299
Brazing alloys, 188–189
Breathing, sleep-disordered, 333–344
classifications of, 334–335
central sleep apnea syndrome as, 335
obstructive sleep apnea (OSA) as, 320, 334
sleep hypoventilation syndromes as, 335
upper airways resistance syndrome as, 335
clinical prediction of, 336
clinical presentation of, 335–336
definitions and testing reports in, 333–334
epidemiologic factors in, 335
pathophysiologic factors in, 335
physical characteristics of, 336
preventative management in, 344
tests and questionnaires in, 336–340
Berlin questionnaire as, 338, 339t
cephalometric analysis as, 338
Epworth Sleepiness Scale as, 338
Friedman classification as, 336–337
Kushida Index as, 338
Mallampati score method as, 337
Sleep Disorders Questionnaire as, 340t
STOPBang questionnaire as, 338, 339t
treatment modalities in, 340
treatment options of, 340–344
lifestyle and behavioral modification as, 341
oral appliance as, 341–343
oropharyngeal exercises as, 343–344
positive airway pressure as, 341
surgical treatment as, 343
upper airway electrical neurostimulation as, 344
Broadbent/Bolton template, 691–694
Broadbent-Bolton transparencies, in cephalometric analysis, 595–596
Bruxism
CNS-induced, 362
sleep-related, 364
Buccal corridor width, of smile, 272–273, 274f
Buccal segments, distalizing, sagittal appliance for, 591
Buccal-root torque, 458
Buccolingual crown inclination, 190
Bulimia nervosa, 293
Bundle bone, 54, 56f, 107f, 108
Bypass arch, 178
canine, 179f
cuspid, 179f
incisor, 180f

C

Calcium
conservation, 117
homeostasis, 116
increasing availability of, for remineralization, 895
metabolism, 116, 117f
Calcium hydroxide, for dental avulsion, 884
Camouflage treatment
checklist for class II patients, 705b
contraindications for, 705
indications for, 703–705, 704f, 705b
Camphorquinone, 194
Cancellous bone, 54, 56f, 381
Cancellous compaction, 108
Canine
displaced, 876
impaction and/or displacement of, 43
maxillary, impaction of, 870
retraction
frictional forces in, 181–182, 183f
of Tweed-Merrifield edgewise, 497
root formation, permanent, 378–379
straight wire appliance and, 439
Canine-to-canine lingual retainer bar, 842–847, 846f–847f
bonding of, 845–847, 848f–849f
failure analysis and long-term experience with, 847
retention period for, 847
Cantilever testing, of archwires, 193
α-Cardiac muscle isoforms, 39
Cardiovascular computed tomography (cine CT), 326
Caries Balance concept, 888–889, 892f
Carrier, 35f
CAS. *see* Computer-aided surgery (CAS)
Case report, 202
Case/control study, in orthodontics, 202
Casein phosphopeptide-amorphous calcium phosphate (CPP-ACP), 895
for demineralized white lesions, 896
Casein phosphopeptides (CPPs), 895
Casts
analysis, for diagnosis, in orthodontics, 223
posttreatment, 490f, 504f, 508f
pretreatment, malocclusion correction with edgewise appliance and, 502f, 506f
CBCT. *see* Cone beam computed tomography (CBCT)
CBT. *see* Cognitive-behavioral therapy (CBT)
Cell kinetics, in bone physiology, 106
Cementum, root, 53, 55f
Centers of rotation, 156–158, 159f
lingual force in, 156, 156f
moment-to-force (M/F) ratio against, 157, 158f
pure moment in, 156
three-dimensional finite element method for, 158, 159f
for translation, 156, 156f
Central fifth, of face, 262
Central hypopneas, 334
Central incisors, maxillary, impacted, 870
Central nervous system, increase peripheral input to, 362
Central sleep apnea syndrome, 335
Centric relation (CR), 442
habitual occlusion and, in debonding or debanding, 602
Cephalogram, 223, 255
for upper airway, 326
Cephalometric analysis, 338, 383–387
for diagnosis, in orthodontics, 223–226, 233f
Cephalometric head-holders, 253
Cephalometric tracing
with all values and planes, 491f
malocclusion correction with edgewise appliance and, 503f
posttreatment, 490f, 505f, 508f
pretreatment, malocclusion correction with edgewise appliance and, 503f, 507f
Cephalometric values, pretreatment and posttreatment average
in female group, 464t
in male group, 464t
Cephalometry, contemporary applications of, 254–255
Ceramic brackets, 191–192, 813, 813f
brittleness of, 191, 192f
fracture of, 191
grain size effect in, 192

Ceramic brackets *(Continued)*
large-grained, 192
polycrystalline, 192
reconditioning of, 832
removal of, 834, 834f–835f
in wet environments, 191–192
Cervical bodies, 405–406
Cervical headgear, 261, 263f
upper airway and, 331
Cervical stage
first (C1), 405–406, 406f
second (C2), 406, 407f
third (C3), 406, 407f–408f
fourth (C4), 406, 407f–408f
fifth (C5), 406–408, 408f
six (C6), 408f
Cervical traction headgear, 398
Cervical vertebral maturation method, 405–408
schematic representation of, 405f
Cetlin approach, 455–456
Cetlin mechanics, 468
Charge-coupled device (CCD) video camera, 888
Chart, in adult interdisciplinary therapy, 596
Chewing gum, 895
Cheyne-Stokes respiration, 335
Child Oral Health Impact Profile scale, 299
Child patient, management and motivation for, 395–402
behavior guidance of, 395–396
extraoral appliances, 398
first impressions of, 395–396
headgear for class II correction, 398
at home responsibilities, 397–398
intraoral elastics, 400
orthodontic appointments, 399–400
pain management of, 395
removable intraoral appliances, 398–399
removable orthodontic appliances, 398
Children, lingual treatment of, 773f–777f, 773b
Chin cup, 706t
Chin deficiency, throat form and, 265
Chin projection, 265
Chlorhexidine mouth rinse, for acidogenic plaque, 890
Chondrocranium, 4
Chromosomes, human, 32–33, 32f
Chronic obstructive sleep apnea, 320
Chronic periodontitis, 623
Circular fibers, 52, 53f
Circummaxillary suture system, 12, 12f, 78
Class I malocclusion, 209
Class II correction, headgear for, 398
Class II dental relationship, orthodontic correction of, 499–500
Class II division 2 (II/2) malocclusion, 42
Class II elastics, 796, 796f–799f, 810f
nonextraction case with use of, 773f–777f, 773b
Class II force system, 499
of Tweed-Merrifield edgewise, 498–499
Class II malocclusion, 209, 261, 262f, 273, 275f, 409, 671f
camouflage treatment for, 705b
cephalometric analysis of, 224
components of, 419
family data and, 40
orthognathic surgery for, 681f, 709f
patients and, 416–418, 417f
prevalence of, 676t
spontaneous improvement of, after rapid maxillary expansion, 416–418, 417f
treatment of, 419–427
additional comments regarding, 426–427
appliance selection, 423
available strategies of, 419–422
extraoral traction, 419–420, 420f
facebow, 419
FR-3 Appliance of Fränkel, 429, 429f
functional jaw orthopedics, 422–426
Class II malocclusion (*Continued*)
headgear, 420f
Herbst appliance, 423–424, 423f–424f
mandibular anterior repositioning appliance, 425, 425f
maxillary distalization, 419
maxillary molar distalization, 420–421
Pendex appliance, 421–422, 422f
Pendulum appliance, 421–422, 421f–422f
summary of, 432–433
timing, 426
twin block appliance, 425–426, 425f–426f
Class II mandibular deficiency, diagnosis of, 695, 696f–701f
Class II skeletal patterns, 224, 323
Class III deep bite cases, muscle fibers of, 39
Class III elastics, 468
Class III malocclusion, 41–42, 209
cephalometric analysis of, 224
components of, 427
FR-3 appliance of Fränkel, 429
mandibular growth effects in, 984
maxillary skeletal retrusion associated with, 427
spontaneous improvement of, after rapid maxillary expansion, 418–419
timing of treatment of, 409
treatment of, 427–432
additional comments regarding, 432
appliance selection, 427–432
chin cup, 429–432, 430f
orthognathic surgery, 433
orthopedic facial mask, 427–429, 428f
strategies for, 427
Class III mandibular excess, diagnosis of, 695, 696f–701f
Class III skeletal patterns, 211
Cleaning, in bonding to enamel, 819
Clear aligner treatment, 778–811
appliances used in, 778, 783
biomechanics of, 783–786
brief history of, 778–780, 779t–780t
case study of, 800b, 801f–810f, 805b, 807b
ClinCheck and, 782–783, 783t, 784f
diagnosis and treatment planning for, 782
difficult movements using, 785–786, 785f–786f
interproximal tooth reduction and, 778
low-level mechanical vibrations and, 921, 921f–922f
periodontal considerations of, 800
simple movements using, 785–786, 785f–786f
simultaneous staging of, 786, 786f–788f
staging of, 786–788, 786f–788f
terminology associated with, 778–780
torque control and, 791–792, 792f
virtual models and, 789f
Cleft lip/palate, 298–299
complete unilateral, 372f
isolated, 368
prenatal diagnosis of, 368
Cleft palate-craniofacial team
diagnostic considerations of, 368
advantages of, 368
disadvantages of, 368
incidence, 368
limitations in, 368
prenatal, 368
ultrasound technique, 368
management of missing lateral incisor space, 390–392, 392f
mixed dentition stage (7 to 12 years of age), 375–382, 377f–380f
nasomaxillary complex, 372
neonate and infant (birth to 2 years of age), 369–372
orthodontist's role in, 369–390
permanent dentition stage, 382–390, 382f–383f
primary dentition stage (2 to 6 years of age), 372–375, 373f–376f
surgical lip adhesion, 371–372
team approach to, 368–369
Cleidocranial dysplasia, 869, 870f
ClinCheck, 782–783, 783t, 784f
Clinical cephalometrics, development of, 223
Closed eruption exposure technique, for impaction, 874, 874f
Closest point measurements, in CBCT, 309, 309f, 312, 312f–313f
Close-up images
with black background, for microaesthetics, 252, 254f
of posed smile, in facial photographs, 248, 249f
Cochrane Collaboration, for orthodontics, 205
CODA. *see* Council on Dental Accreditation (CODA)
Coffin loop, 456, 457f, 462f
Cognitive awareness, TMJ and, 362
Cognitive-behavioral therapy (CBT), 292
Cohort study, in orthodontics, 202, 202f
systematic review and, 204, 204f
Coil activation, Niti thermal-activated, 444
Collagen fibers, in gingiva, 52, 53f
Color-coded maps, in CBCT, 311, 311f–312f
Columella, 264
Combination facebow headgear, 419
Commissure height, 270, 271f
Common coordinate system, in CBCT, 310–311
Communication
in adult interdisciplinary therapy, 614
guidance, for child and adolescent patient, 396–397
between orthodontist and patient, 289
reports used in, 673–674
Complementary force system, in targeted therapeutics, 950, 954, 958f
Completely customized lingual appliances, 761, 762f
innovation in, 764
Completion conference report, in adult interdisciplinary therapy, 618f
Complex adaptive system, 947
Complex traits, 37, 38f
Composite bone, 107f, 108
Composite resins, 813–816
flowable, 841
Composite restoratives, bonding to, 829
"Compound contour" base, straight wire appliance design and values and, 437
Compound loading, of orthodontic appliance, 165, 165f
Comprehensive database, 229–230
Computed tomography, cone-beam, 442
Computer imaging
in contemporary treatment planning, 255, 257f
for diagnosis, in orthodontics, 220
Computer-aided surgery (CAS), 306
Concurrent mechanics, in targeted therapeutics, 950, 952f
Concussion, 881
Condylar cartilage, 79, 81f
Condylar growth, 79
retardation of, 80
Condylar position, alteration of, 362
Condylar process, 18
Condyle-disc complexes, 355
Cone beam computed tomography (CBCT), 220, 221f, 302–318, 303f, 432f, 442, 871, 872f
in biotype, 626, 626f
challenges to advance understanding of growth and treatment responses in, 307–309
clinical applications, and potential limitations of, 304–306
airway assessment in, 305–306, 306f
craniofacial anomalies in, 306, 307f
dentofacial deformities in, 306, 307f
temporomandibular joint health and disease in, 304–305, 305f–306f
tooth morphology in, 304, 304f
for facial structure, 691
image analyses in, 307–313
three-dimensional surface models and superimpositions in, 307–313
of TMJ degeneration, 101–102, 104f–105f
for upper airway, 326–327
Consanguineous mating, 37

Consistent force systems, in targeted therapeutics, 954, 954f–957f
Consonant smile arc, 270
Constraint recognition and management
in proactive decision making, 944, 947f
in targeted therapeutics, 950, 951f
Consultation, in adult interdisciplinary therapy
devices, 596
techniques, 595
Contemporary straight wire biomechanics, 437–454
anchorage
maximum, 444
medium, 444
minimum, 444
arch coordination, 441, 441f
bracket
extraction, 437
placement, 438–439, 439f
straight wire appliance and, 437
translation, 437
case study of, 446f–453f, 446b, 448b, 450b, 452b
closing extraction spaces, 443–444
design and values of, 437
double keyhole loop activation, 444
finishing stage, 445
future directions of, 445–454
intermaxillary elastics, 444–445
leveling and aligning of, 439–440
stage and, 440f
maximum anchorage, 444
medium anchorage, 444
minimum anchorage, 444
Niti thermal-activated coil activation, 444
optimal bracket placement, 438–439, 439f
overbite correction using, 441–443, 442f
overjet correction using, 441–443, 442f
self-ligation and, 438–445
stage 1 of treatment, types of wires, size, and sequence suggested for
in cases with mild crowding, 441t
in cases with moderate to severe crowding, 441t
stage 2 of treatment, types of wires, size, and sequence suggested for
in extraction cases, 445t
in nonextraction cases, 445t
stage 3 of treatment, types of wires, size, and sequence suggested for, 445t
treatment mechanics, 439–445
working stage and, 440–445
Continuous arch, forces from, 177–179
Continuous force, 900
Continuous positive airway pressure (CPAP), 341
Conventional ligation, force levels with, 746
Conventional mechanics, TAD mechanics and, 511, 516–517, 517f–518f, 519
Corpus, of mandible, 18
Cortical bone, 54, 56f
bone remodeling in, 107f–108f, 108–110
growth and maturation of, 111–116, 113f–115f
structural and metabolic fractions of, 113–116, 116f
in temporary anchorage device, 522
Corticision, 93
Corticotomy, 470
facilitates tooth movement, 478–479
have precise indications, 479
limited effect in space, 478
for malocclusion, 661
proper orthodontic management, 479
proper surgical management, 479
Council on Dental Accreditation (CODA), 201
Couple-to-force ratio, 157, 157f
Coupling factors, of bone modeling, 111, 112f
CPAP. *see* Continuous positive airway pressure (CPAP)
CPP-ACP. *see* Casein phosphopeptide-amorphous calcium phosphate (CPP-ACP)
CPPs. *see* Casein phosphopeptides (CPPs)
CR. *see* Centric relation (CR)
Craft-based care model, 926–927, 928f
Cranial base, 8–12
development of, 8, 8f
postnatal growth of, 10–12, 11f
Cranial disharmony, 488–492
craniofacial analysis and, 490–492
Cranial vault, 5–8
brain expansion and, 7, 7f
development of, 5–6, 5f
postnatal growth of, 7–8
Craniofacial analysis, 491, 492t
cranial disharmony and, 490–492
horizontal skeletal component of, 492
skeletal analysis values and, 488–490
Craniofacial anomalies, 306, 307f
team approach to patients with, 370f
Craniofacial complex, 3–4, 3f
functional units of, 4, 4f
structural units of, 3–4
Craniofacial deformities, patients with, 298–299
Craniofacial growth and development, 1–17
adult changes in, 25
arch development, 23–25, 23f–24f
cranial base, 8–12. *see also* Cranial base
cranial vault, 5–8. *see also* Cranial vault
craniofacial complex, 3–4, 3f
functional units of, 4, 4f
structural units of, 3–4
midface/nasomaxillary complex, 12–16
modification of, 404
molecular basis of, 5
postnatal interrelationships during, 25–27, 26f–27f
significance of, for orthodontics, 27–28
somatic growth, 1–3
tooth migration, and eruption, 23–25, 24f–25f
Craniosynostosis, 6–7
Crib appliance, 607–609, 612f
Critical point, in patient cooperation, 404
Critical sections, 175
Crossbite
anterior, 373–374
unilateral, 458
Crouzon syndrome, and upper airway, 332f
Crowded lower arch, treated without extraction, 747f–751f, 747b–748b
Crowding
alleviation, of NiTi archwires, 193
case study of, 747f–751f, 747b–748b
management of, in orthodontics, 232–233
self-ligation for, 746–756
treated with lingual appliance treatment, 765
Crown tipping, 164
Crown-to-root ratio, adult interdisciplinary therapy in, 574–575, 581f
Curve of Wilson, 441
Custom milling, in orthodontics, 236
Customized lingual appliance, 761–762
decalcification risk reduced with, 762
illustration of, 762f
innovation in, 764
target setup model system in, 761
treatment goals realized with, 761–762
Cutting/filling cones, 113–116
in bone remodeling, 110, 110f, 112f
perivascular process and, 122, 125f
CYP19A1, 41
Cytokines, in tooth movement, 914
Cytotoxicity, of orthodontic resins, 816

D

DAC. *see* Dentoalveolar collapse (DAC)
Damon 2 brackets, 733, 733f
Damon 3 brackets, 733, 733f
Damon D3 MX bracket, 733, 733f
Damon Q bracket, 733, 734f
Damon SL brackets, 732, 732f
Database programs, application of, to clinical information, 255–259, 258f
Debonding, 833–840
amount of enamel lost in, 837
clinical procedure in, 833–834
removal of ceramic brackets in, 834, 834f–835f
removal of steel brackets in, 833–834, 833f
decalcification in, prevention and reversal of, 838–840, 838f
in dentoalveolar collapse
coordination of, with other treatment providers, 606
evaluation before, 606f
enamel tearouts and cracks (fracture lines) in, 834, 835f
operator safety during, 837–838
residual adhesive, removal of, 834–837, 835f–837f
Decalcification
prevention and reversal of, 838–840, 838f
microabrasion for, 838–840, 839f
resin infiltration for, 840, 840f–841f
reduced risk of, 762
Decision making
proactive, 942–944
process, in orthodontics, 208–244
problem-oriented approach in, 215
Decortication, 662
alveolar, 661–665, 662f
Decortication-bone grafting, 914
Deep overbite
intrusion for, 173, 174f
overcorrection of, 982
Deficiency
mandibular, 672f, 676t, 677f, 695, 696f–698f, 711
maxilla, 680f–681f, 708
Definitive lip repair, 372
Degeneration, in hyalinization phase, 60, 61f
Degree of cure-conversion, 816
DeLaire face mask, 706t
Delayed healing, 720–722
Delayed pain, 162–163
Demand, *versus* need, in orthognathic surgery, 672–676
Demineralization
prevention of, 889–892
tooth movement and, 147
Demineralized white lesions
detection and measurement of, 888
with quantitative light-induced fluorescence, 888, 890f–891f
with quantitative light-induced fluorescence-digital, 888, 891f
with transverse microradiography, 888, 889f
loss of mineral from enamel (demineralization) in, prevention of, 889–892
prevalence of, 887–888
prevention and management of, 887–896, 888f
promoting uptake of mineral into enamel (remineralization) in, 892–895
treatment of, 895–896
with casein phosphopeptide-amorphous calcium phosphate, 896
with fluoride, 896
with resin infiltration, 896
Dental agenesis, 42–43
Dental alignment, analysis of, 229
Dental compensations, 210–211, 211f, 237b–239b
Dental crown morphology, 42
Dental disharmony, 492–493
Dental eruption problems, 43–44
Dental luxation
extrusive, 881–883
intrusive, 883
in permanent dentition, management of, 880–886
prevention of, 884–885

Dental materials, in orthodontics, 187–199
adhesives for, 193–195
archwires for, 192–193
blue light in, 194–195
mechanics of, appliance properties on, 187–192
NiTi archwires, 192–193
photocuring in, 193–195
retainers for, fixed, 195–197
stainless steel brackets for, 187–190
summary in, 197
Dental microaesthetics, 276–282
bracket placement in, 281–282, 282f
gingival shape and contour in, 276–281, 279f–281f
Dental midlines, 229
Dental proportions, 261–262
Dental protrusion, lip projection and, 265, 267f
Dental Technology Services (DTS) model, 942
Dental trauma, 639f
Dentition
in craniofacial complex, 4
dimensions of, 484–485
mixed, 462f
open bite in, 463f
partially edentulous, 73, 75f
space maintenance during transition of, 409
Dentition stage
mixed, 375–382, 377f–380f
permanent, 382–390, 382f–383f
primary, 372–375, 373f–376f
Dentoalveolar collapse (DAC), 597–606, 598f
cases of, requirement in, 602
characteristics of, 602b
conclusion for, 602
debonding or debanding in
coordination of, with other treatment providers, 606
evaluation before, 606f
etiology of, 602b
principles in, application of, 600–602, 602f–605f
Dentoalveolar compensation, 699f
Dentoalveolar region, trauma to, 880
Dentoalveolar widening, 467f
Dentofacial appearance, evaluation of, 228–229, 231f
Dentofacial deformities, 306, 307f
orthognathic surgery in, 295
Dentofacial imbalances, 673b
Dentofacial orthopedic treatment timing, 403–436
cervical vertebral maturation method of, 405–408
serial extraction of, 410–412, 411f–412f
timing of treatment intervention of, 404–405
intervene, 408–409
modification of craniofacial growth, 404
patient cooperation, 404
practice management, 404–405
sagittal malocclusions, spontaneous improvement of, 416–419
tooth size-arch size discrepancy problems, 409–410
Dentofacial orthopedics, 51
Dentogingival fibers, 52, 53f
Dentoperiosteal fibers, 52, 53f
Denture completion, of Tweed-Merrifield edgewise, 500–501, 501f
Denture correction, of Tweed-Merrifield edgewise, 497–500, 497f, 499f–500f
Denture preparation, of Tweed-Merrifield edgewise, 496–497, 496f–497f
Denture recovery, of Tweed-Merrifield edgewise, 501–502
Deoxyribonucleic acid (DNA), 32–33
codes, 32–33
variations of, 34
Dependent clinger, 294
Depression, 293
Desmocranium, 3–4
Destructive lesion, 623–624
Diabetes mellitus, 695t
Diagnosis, in orthodontics, 208–244
Angle classification in, 226
camouflaging discrepancies on, 210–211
cephalometric analysis for, 223–226
class II malocclusion for, evaluation of, 224
classification in, 226–229
clinical evaluation for, 217–219
face in, 217
facial proportions in, 217
intraoral examination in, 217–219
jaw function in, 218–219
soft tissue function in, 218
TM joints in, palpated, 219
concepts of, 208–215
database for, 226–229
dental compensations in, 210–211
first contact interview for, 216
interview at first appointment for, 216
interview data for, collection of, 216–217
modern health care paradigm of, 208–209
goals of, 209
patient evaluation for, 215–230
patient meeting for, 216–217
problem-list as, 215
prioritizing, 230–232
problem-oriented approach to, 215, 230–236
problem-oriented treatment planning in, 246, 246f–247f, 258
profile for, 233
quality of life in, 208–209
records needed for, 219–222
skeletal compensations in, 210–211
special considerations in, 245–288
speech evaluation for, 218
summary of, 229–230
systematic description for, 226–229
application of, 228–229
template-guided, 694–695, 696f–702f
therapeutic, 705–706, 706t, 707f
therapeutic modifiability, 234–235
treatment planning for, 230–236
Diagnostic facial triangle, Tweed's, 484f
Diagnostic records, in orthodontics, 219–222
additional, 219
analysis of, 222–226
cast, 223
cephalometric, 223–226
class II malocclusion, 224
class III malocclusion, 224
clinical cephalometrics in, 223
skeletal and dental units in, relationships of, 223, 224f
vertical dental problems in, 225–226
vertical skeletal problems in, 224–225
computer imaging in, 220
cone beam computed tomography in, 220
data from, 215f
head orientation, for cephalograms, 219
intraoral scanning in, 220–222
photography in, 219–220
physical dental casts in, virtual *versus*, 219
technologic advances in, 219–222
3D facial photography in, 220
videography in, 219–220
Diagnostically driven, robotic-assisted (DDRA) orthodontics, 926
Diet
fluoride in, 894–895
for patient motivation, 397–398
Differential anchorage, 131–132, 137f
Differential diagnosis, 485–493
analysis system, 493, 494t
cranial disharmony of, 488–492
dental disharmony, 492–493
facial disharmony, 485–488
Merrifield's contributions, 485
in orthognathic surgery, 712, 717–718
Differential scanning calorimetry (DSC), 193
Differential space closure, 181, 182f
"Difficult" patients, 294
Digital diagnostic model, for SureSmile, 930–934, 931f–937f
Digital diagnostic simulation (DDS) model, 930
Digitization, 780–782
Dilantin, in gingival hyperplasia, 218
Diode laser, 279f–280f, 280–281
Direct-bonded labial retainers, 854–855
long-term results of, 855, 859f–860f
technical procedure for, 855
Directional force, 495, 496f
Disarticulation, in adult interdisciplinary therapy, 591, 592f–593f
Disc displacement
joint sounds caused by, 361
without reduction, 711
Displacement, lateral, 881
Distalization
en masse, 528, 541f
molar, temporary anchorage devices in, 515
archform control in, 538f–540f
biomechanics in, 528
decision making in, 528, 531f
mechanical efficiency in, 528, 532f–536f
mesiodistal axis control in, 537f
methods for, 522f
three-dimensional control in, 528
torque control in, 538f
vertical control in, 539f–540f
Distalizing buccal segments, sagittal appliance for, 591
Distalizing forces, molar resistance to, reduction of, 470–479
Distalizing plate, 455
clinical management of, 464–466, 466f
removable, 464, 466f
Distraction osteogenesis, 122
in permanent dentition stages, 388–390, 390f–391f
Distraction osteogenesis (DO), 331
Distribution, of teeth, adult interdisciplinary therapy in, 571, 574f
Diverse methods, using temporary anchorage devices, 523f
DNA. *see* Deoxyribonucleic acid (DNA)
DO. *see* Distraction osteogenesis (DO)
Dolichofacial, patients, 228–229
Dominant gene/dominant allele, 35
Double keyhole loop activation, 444
Double-blind molar uprighting study, 635
DSC. *see* Differential scanning calorimetry (DSC)
Dutch Inter-center prospective, 371
Dutchcleft, 371

E

Early postretention stability, 989
EARR. *see* External apical root resorption (EARR)
Eating disorders, 293
EBD. *see* Evidence-based dentistry (EBD)
Ectomeninx, 5
EDA gene, 42
Edema control, in orthognathic surgery, 676
Edentulous dentitions, partially, 73, 75f
Edgelok brackets, 731, 731f
Edgewise appliance, 482
malocclusion correction with, 502b–506b
Tweed-Merrifield, 493–495, 494f
Education, in adult interdisciplinary therapy, 595
for patient, 596
Effective dose, 302–303
Elastic limit, of alloys, 167
Elastics
class III, 468
fluoride in, 894, 894f
intermaxillary, 444–445
Elastomeric ligatures, 190, 729–730, 730f, 742–743
Elastomeric rings, 739

Electromagnetic tracking, tracking technology in, 315
Embrasure space, adult interdisciplinary therapy in, 571, 576f
En masse distalization, 528, 541f
Enamel
  bonding to, 819–824, 822f
    cleaning in, 819
    curing in, 824, 824f
    enamel conditioning in, 819–821
    excess in, removal of, 823–824, 823f
    fitting in, 823
    positioning in, 823
    premedication for, 819
    procedure for, 823
    sealing and priming in, 821, 822f
    transfer in, 823
  conditioning of, before bonding, 819–821
    enamel pretreatment in, 819–821
    moisture control and, 819, 819f
  debonding and, amount lost during, 837
  sealing of, 892
  tearouts and cracks, in debonding, 834, 835f
Endocrinology, of calcium conservation, 117
Endosseous implants, 136–140, 139f–140f, 391–392
Endothelial nitric oxide synthase (eNOS), 914
Enlow's classic diagram, 223, 224f
eNOS. *see* Endothelial nitric oxide synthase (eNOS)
Entitled demanders, 294
Envelope of discrepancy, 213–214, 213f
  for maxillary and mandibular arches, 607, 611f
Epigenetic landscape, 38
Epworth Sleepiness Scale, 338
ERT. *see* Estrogen replacement therapy (ERT)
Eruption. *see also* Extrusion.
  primary failure of, 43–44
Eruptive wire mechanics, 261, 263f
Estrogen
  in calcium conservation, 117
  facial growth and, 41
Estrogen replacement therapy (ERT), 117
Estrogenic action, 195, 195b
Evidence-based care, 201, 201f
Evidence-based clinical recommendations, in orthodontics, 205
Evidence-based dentistry (EBD), 200–201
Evidence-based journals, for orthodontics, 205
Evolution bracket, 735
Evolvers, 296
Excess
  mandibular, 679f, 695, 696f–701f, 705–706, 708–711, 709f–710f, 711b, 713, 715f–716f, 719f
  maxillary. *see* Maxillary excess
Excessive lip projection, *versus* inadequate lip projection, 265, 266f–267f
Excessive submental fat, throat form and, 265
Excessive vertical disproportionality, 260, 261f
Excursive movements, in debonding or debanding, 605
Expedited tooth movement, 145–149
  from experimental studies on rodents and canines, 148–149
External apical root resorption (EARR), 897, 897f
  apical root displacement in, 900, 901f–905f
  demographic factors in, 897–898
  diagnostic factors, 897–899
    patient medical history and habits in, 899
    tooth anatomy in, 897, 898f–899f
  early treatment as, 900
  etiologic factors of, 896–897
  expansion in, 900
  extractions and, 900
  genetic factors and, 44–45, 45f
  intermittent *versus* continuous force in, 900
  low-level mechanical vibrations and, 919–920
  magnitude of applied force in, 900
  malocclusion factors in, 898
    root canal treatment and, 898, 899f
External apical root resorption *(Continued)*
  management of, 901–908, 901b
    history in, 901
    imaging in, 901
    progress review in, 902
    root resorption, detection of, at progress, 902, 906f–907f
  mechanical, 900
  prolonged treatment time for, 900
  risk factors for, 897–900, 901b
  start of occurrence of, 902–903
  stopping of occurrence, 903
  and teeth with short roots, 903–904, 907f
  treatment factors for, 900
External distractor, 331, 332f
External inflammatory root resorption, 896
External resorption, 896
External root resorption, in unerupted teeth, ectopically positioned and, 650–657, 658f–661f
Extraction brackets, 437
Extraction gaps, closure of, 83, 84f
Extraction spaces, closing, 443–444
Extraction treatment, anterior retraction in, 541
  biomechanics in, 541, 544f–545f
  decision making in, 541
Extractions
  case study of, 747f–751f, 747b–748b
  evaluations before, 708
  self-ligation effects on, 746–756, 747f–751f, 747b–748b
  serial, 410–412, 411f–412f
  upper airway and, 329–330
Extraoral appliances, 398
Extraoral forces, 456
  headgear and, 462
Extrinsic causes, in orthodontic problems, 211–213
Extruded posterior segments, temporary anchorage devices for, 571–573
Extrusion
  correction, in Invisalign, 795–796, 795f–796f
  mandibular molar, maxillary molar intrusion with, 559b, 560f–562f
  of teeth, 634–636, 639f
  as tooth movement, 70–71, 70f
Extrusive luxation, 881–883

F

Face, 217
  central fifth of, 262
  in craniofacial complex, 4
  height
    decreased, 260–261
    increased, 260
  medial two-fifths of, 262
  outer two-fifths of, 262
  overall proportionality of, 261, 264f
  protrusive, profile line, 486f
  vertical thirds of, 259f, 261
Face-bow, 175, 175f
Facial asymmetry, management of, 711
Facial cancer, acquired deformities and, 299
Facial disharmony, 485–488
Facial exercises, for OSA, 344
Facial growth
  genetics of, 41–42
  pattern of, 383
Facial height
  anterior, definition of, 490
  change ratio, definition of, 490
  index, definition of, 490
  posterior, definition of, 490
Facial photographs, 248–251
  frontal, 248, 249f
  oblique (three-quarter, 45-degree), 248–250, 250f
  posttreatment, 488, 489f, 491f
    malocclusion correction with edgewise appliance and, 504f, 508f
Facial photographs *(Continued)*
  pretreatment, 489f, 506f
    malocclusion correction with edgewise appliance and, 502f
  profile, 250–251
Facial profile, profile line in, 486f
Facial proportions
  clinical examination of, 255–259
  evaluation of, 259–282
    frontal vertical facial relationships in, 259–261, 259f
  transverse, 261–262
Facial soft tissues, 372–373
Facial symmetry, clinical examination of, 255–259
  application of database programs in, 255–259, 258f
Facial trauma, acquired deformities and, 299
Faciolingual relationships, of posterior teeth, 229
Family data, for growth prediction, 40
Family-based (familial) traits, 35–36
Female, maxillary postnatal growth in, 16, 16f
Fiberotomy, 644, 646f
Fiber-reinforced composite, in wires, 182, 183f
Fibroblast, in gingiva, 52
Field of view, in CBCT, 303–304
Fillers, 194, 194f
Final occlusion, of Tweed-Merrifield edgewise, 501f
Finite element modeling, 106
First-order bends, in Tweed-Merrifield edgewise appliance, 493
First-order compensations, using lingual appliance treatment, 763–764, 763f–764f
Fixation, skeletal, after orthognathic surgery, 684–689, 690f–691f
Fixed appliances, 706t
  in adult interdisciplinary therapy, 592–594
Fixed orthodontic appliances, white spot lesions associated with, 762
Fixed prostheses, 391
Flash-free product, 824, 824f
Flat smile arc, 270, 272f
Flat wire, 169
Flexible spiral wire retainers, 847–854, 850f
  bonding of, 849–852
    direct, 849, 851f–854f
    indirect, 849–852
  failure analysis and repair of, 852, 855f–858f
  indications for, 849
  long-term experience with, 852–854
Fluctuating asymmetry, 44
Fluorapatite, 895
Fluorescent labels, as bone markers, 104
Fluoride
  for demineralized white lesions, 896
  increasing availability of, for remineralization, 892–895
  professional application of, 838, 839f
Fluoride-containing glass bead, 895f
FMA. *see* Frankfort mandibular plane angle (FMA)
FMH. *see* Functional matrix hypothesis (FMH) of Moss
FMIA. *see* Frankfort mandibular incisor axis angle (FMIA)
Fontanels, 7, 8f
Force indentation depth loading-unloading curves, 196f
Force magnitude, in tooth movements, 159–162
Force systems, 499
  class II, 499
    of Tweed-Merrifield edgewise, 498–499
  complementary, in targeted therapeutics, 950, 954, 958f
  consistent, in targeted therapeutics, 954, 954f–957f
  inconsistent, 954, 954f
  upward and forward, 496f
  in wire, 177, 177t
Force-driven mechanics, 522, 523f, 524
Forestadent brackets, 735
Forward-resting tongue posture problem, 609–610
Fracture
  alveolar, 881
  lines, in debonding, 834

Frankfort mandibular incisor axis angle (FMIA), 486–488, 487f–488f
probability index after pretreatment range for, 492t
Z angle and, 486f
Frankfort mandibular plane angle (FMA), 485, 488
Frankfort plane, 219
Free gingiva, 52, 52f
FRESH acronym, 669, 670b
Friction
with active archwires
thick, stiff wires, 736–737
thin, flexible wires, 737–738, 737f–738f
of active clips and passive slides, 741
low
clinical significance of, 739–740, 740f
ligation and, 739, 739f
role of, 182–183
self-ligation and, 736–739
of stainless steel brackets, 189–190, 189b
in vivo: occlusal and masticatory forces, 738–739
Friedman classification, of OSA, 336–337
Frontal at rest, in frontal photographs, 248, 249f
Frontal cephalogram, 690f, 692f
Frontal dynamic (smile), in frontal photographs, 248, 249f
Frontal photographs, 248, 249f
Frontal vertical facial relationships, 259–261
Frontal view with teeth in maximal intercuspation, in facial photographs, 248
Frontonasal prominence, 5
Functional matrix hypothesis (FMH) of Moss, 598, 669

G

GCF. *see* Gingival crevicular fluid (GCF)
Genes, 33, 33f
key, in craniofacial growth and development, 5, 5.e1t
Genetic effects, types of, 34–37
Genetic syndrome, 36
Genetics
background and basic definitions of, 32–34
etiology and, 31–32
of facial growth, 41–42
nature *versus* nurture, 38–40
orthodontics and, 31–50
of tooth size and agenesis, 42–43
Genome, 32–33
Genotype, 34
Gingiva, 52, 52f
lacerated, 881
Gingival biotype, 626
Gingival clefts, 218
Gingival crevicular fluid (GCF), 904–908
Gingival display, of smile, 270, 271f
Gingival hyperplasia, in periodontal-orthodontic interrelationships, 642
Gingival margins, of canines, 271–272, 273f
Gingival recession
alveolar bone dehiscences with, 661
in periodontal-orthodontic interrelationships, 626
Gingival retention, periodontal-orthodontic interrelationships, 642–643
Gingival tissue, in orthodontic therapy, 595
Gingival trauma, OSA and, 345, 346f
Gingival zenith, 276–280
Gingivectomy, 279f–280f, 280–281
Gingivitis, 623
in children, 218
Glass ionomer cements, 817, 818f
Gnathostatics, 226
Gold, bonding to, 829, 830f
Gold-based brazing materials, 189
Goldenhar syndrome, 390, 391f
Goshgarian's anchorage appliance, 456
GRADE. *see* Grading of Recommendation Assessment, Development and Evaluation (GRADE)
Grading of Recommendation Assessment, Development and Evaluation (GRADE), 204
Grafting
augmentation, 661–665
before orthodontic treatment, diagnostic considerations in, 644b
particulate, 662–663, 662f
Great Extraction Debate, 329
Grinding, selective, in adult interdisciplinary therapy, 591
Growth
centers, skeletal, 10
considerations, in permanent dentition stages, 382–383, 384f–387f
corrections carried out during periods of, 983
craniofacial, modification of, 404
differences in, during puberty, 41
face, 714t
facial, pattern of, 383
factors, 718, 984
family data for, 40
mandibular
in boys, 711
in girls, 711
sites, 10
spurts, 2, 3f
Growth treatment response vector (GTRV), 711
GTRV. *see* Growth treatment response vector (GTRV)

H

Hahn, George, 482
Hanks Balanced Salt Solution (HBSS), 883
Hapsburg jaw, 41
Hard palate, isolated clefts of, 368
Hard tissue biotype, 626, 626f
Hard tissues, intraoral examination of, 217–218
Hardness, of dental materials, 188f, 188t, 189
Harvesting bone, morbidity of, 381
Hawley appliance, removable, 398–399
Hawley bite plane, 571
for disarticulation, 577f, 591, 595
Hawley retainer, 991–992, 991f–992f
HBSS. *see* Hanks Balanced Salt Solution (HBSS)
Head orientation, for cephalograms, 219
Headgear
clinical management of, 462
high-pull, 462, 463f, 465f
interlandi-type, 419–420, 421f
low-pull, 462, 465f
Height measurements, maturation and, 3
Helical coils, in wire, 173, 174f
vertical loop with, 176, 176f
Hemophilia, 695t
Herbst appliance, 331
Heritability, estimation of, 39–40
HGP. *see* Human Genome Project (HGP)
Hierarchy of evidence, in orthodontics, 201–203
High filling effect, of occlusion, 210
High-efficiency mechanics, in temporary anchorage devices, 511–513, 512f
High-pull headgear, 462, 463f, 465f
orthopedic effect, 706t
Histone deacetylase 4 *(HDAC4)*, 39
Histones, 32–33
H-MP. *see* Hyoid to mandibular plane (H-MP)
Homeobox genes, in craniofacial growth and development, 5
Homologous chromosomes, 32–33
Homologous pairs, of chromosomes, 32–33
Hormones, in tooth movement, 914
Human Genome Project (HGP), 32–33
Hyalinization phase, 59–64, 60f–62f
Hyalinized zones, 60, 61f
root resorption and, 60–64, 62f–65f
Hydrofluoric acid, as etchant, 825–826
Hygienist, of orthodontist, in adult interdisciplinary therapy, 596
Hyoid bone, position and morphologic features of, 322–323
Hyoid to mandibular plane (H-MP), 322
Hyperactivity, during orthodontic treatment, 291
Hyperplasia, gingival, in periodontal-orthodontic interrelationships, 642
Hyperthyroidism, 695t
Hypodontia, 42
Hypopharynx, 320, 321f
Hypopnea, 334
Hypotensive anesthesia, in orthognathic surgery, 676
Hyrox-type palatal expander, dental tipping with, 612f

I

ICRR. *see* Invasive cervical root resorption (ICRR)
Ideal occlusion, normal *versus*, 209–210
Image registration, in CBCT, 307–308, 310
Image segmentation, 310
Immediate pain, 162–163
Impactions
attachments in, 875–876, 875f
causes of, 868–869, 868f–870f
diagnosis of, 869–870, 870f
failure in
orthodontist-dependent factors, 876–877, 877f
patient-dependent factors, 876, 876f
surgeon-dependent factors, 877
management of, 868–879
positional diagnosis of, 870–871
clinical examination in, 870
plain-film radiography in, 870, 871f
three-dimensional imaging in, 871, 872f
prevalence of, 870, 871f
resolving, 873
surgical exposure of, 873
surgical options for, 873–874
traction mechanisms in, 876
treatment planning for, 871–873
choosing impacted tooth in, 871–872
extraction of impacted tooth in, 871
prevention and interception in, 871
resolution in, 872
Implants
endosseous, 136–140, 139f–140f
miniscrew
bone contact of, 142
bone remodeling and, 142
current status of, 140–143
failure of, 142–143
rigidity of, 143, 143f
retromolar, anchorage, 138–140, 140f
Inadequate lip projection, *versus* excessive lip projection, 265, 266f–267f
Incisal forces, redistribution of, adult interdisciplinary therapy in, 571, 575f
Incisal guidance
in debonding or debanding, 605
at vertical dimension, adult interdisciplinary therapy in, 571, 577f
Incisor alignment, orthodontic considerations associated with secondary bone grafting, 381
Incisor crowding, 211
Incisor impactions, in unerupted teeth, ectopically positioned and, 650, 656f–657f
Incisor mandibular plane angle, 488
Incisor repositioning, for camouflage, 233
Incognito appliances, 735
Incomplete penetrance, 35f, 36
Inconsistent force systems, 954, 954f
Incorrect torque control, 764, 764f
Index of Treatment Need (IOTN), 211
Infant, in cleft lip/palate, 369–372
Infection, in orthognathic surgery, 676–677
Inflammation control, in orthodontic therapy, 595
Inflammatory disease, 623–624
Inflammatory resorption, 880

Infrabony defects, movement into, 637
Infrared (IR) optical tracking devices, tracking technology in, 315
Inheritance, modes of, 34–37
Initial phase, of tooth movements, 159–160
Injury-facilitated acceleration, of tooth movement, 93
In-Ovation, 733–734, 734f
Integrated digital technology platform, 927–930
  technology of, 927
Interarch relationship, 752f–756f, 752b
Intercanine width, 986, 987f
Interdisciplinary therapy, in orthognathic surgery, 712
Interlabial gap, 270
Interlandi-type headgear, 419–420, 421f
Intermaxillary elastics, 444–445
Intermediate alveolar bone grafting, 378–381
Intermittent force, 900
Internal resorption, 881, 896
Interproximal tooth-size reduction, 983
  clear aligner treatment and, 778
Intra-arch symmetry, analysis of, 229
Intracortical compartment, of cortical bone, 108–111
Intracortical secondary osteonal bone remodeling, 108–110
Intraoral appliances, removable, 398–399
Intraoral elastics, 400
Intraoral examination, for diagnosis, in orthodontics, 217–219
Intraoral photographs, 252, 254f, 674f–675f
Intraoral scanning, for diagnosis, in orthodontics, 220–222
Intrusion
  of maxillary and mandibular dentitions, temporary anchorage device in, 550b, 551f–558f, 554b–555b
  maxillary molar, with mandibular molar extrusion, temporary anchorage device in, 559b, 560f–562f
  molar, temporary anchorage device in, 524–541
    archform control during, 525f
    biomechanics of, 525–526, 541
    decision making in, 524–525, 528–541
    general effects of, 524f
    local effects of, 524f
    mandibular, 528
    maxillary, 526
    mechanical efficiency in, 525–526
    posterior torque during, 525f
    rotation control in, 542f
    three-dimensional control in, 526, 526f–527f
    treatment mechanics in, 526–528
    vertical control in, 542f
  of tooth, 636–637, 640f–642f
  as tooth movement, 70f–71f, 71
Intrusion arches, 174f
Intrusive luxation, 883, 884f
Intrusive mechanics, in temporary anchorage devices, 511, 512f
Invasive cervical root resorption (ICRR), 869, 869f, 904
Invisalign, 921, 921f–922f
  attachment in, 788–791, 789f–791f
    beveled, 790–791, 790f–791f, 794–795
    rectangular, 790, 790f
  auxiliaries and, 796–797, 796f–799f
  biomechanics of, 783
  ClinCheck and, 782–783, 783t, 784f
  definition of, 778
  digital manufacturing of, 780
  extrusion correction in, 795–796, 795f–796f
  history of, 778, 779t–780t
  midcourse correction and, 778–780
  power ridges and pressure areas in, 791–792, 792f
  refinement and, 778–780
  root control in, 792–794, 793f–794f
  rotation correction in, 794–795, 795f
  teen, 797–800, 799f
  terminology associated with, 778–780
  virtual set up for, 782
IOTN. *see* Index of Treatment Need (IOTN)
Irregular canine tooth, full ligation of, 738f
Irregularity Index scores, 985–986, 986f
Irreversible lesion, 623–624
Isolated cleft lip, 368
Isotretinoin, 216
ITK-SNAP software, in CBCT, 310

J
Jacobson template method
  in cephalometric analysis, 595–596
  of superimposition, 584–586
Jaw function, 218–219
Jaw rehabilitation, 689, 691f
J-hook headgear, 419
Joint
  sounds, 360
    clinical significance of, 361–362
  symptoms, in debonding or debanding, 605
  temporomandibular
    anatomic structures of, 353
    condyles by, 354f
    major muscles that stabilize, 353–354
    muscle palpation of, 358, 359f
    occlusal evaluation, 360–362
    pain in, 358
    palpation of, 358–360, 359f
    range of mandibular movement, 360, 360f
    sounds, clinical significance of, 361–362
    tenderness of, 358
Journal of Clinical Orthodontics, in alveolar collapse, 602
Juvenile periodontitis, 218

K
Keyhole loop activation, double, 444
Kushida Index, 338

L
Labial techniques
  lingual techniques difference between, 762–765
  torque control using, 764, 764f
Labiolingual rotation, of tooth, 196–197, 196f
Labiomental sulcus, 265
  effects on, 265, 268f
Lacunae, resorption of, 61, 64f–65f
Lag phase, of tooth movements, 159–161
Lamellar bone, 107–108, 107f–109f
Lamellated bone, 54
Lamps, for photocuring, 194
Large-grained ceramics, 192
Laryngopharynx, 320, 321f
Laser etching, for enamel conditioning, 820–821, 821f
Laser holography, 158
Laser irradiation-induced acceleration, of tooth movement, 93–94
Laser lights, 194
Laser welding, 191
Lasers, for ceramic brackets removal, 834, 835f
Latent transforming growth factor beta binding protein 3 (*LTBP3;* OMIM 602090) gene, 42–43
Lateral cephalometric films, 253
Lateral cephalometric radiograph, 219, 253
Lateral dimensions, in systematic description, 229, 232f
Lateral displacement, 881, 882f
Lateral incisor space, missing, management of, 390–392, 392f
Laterotrusive contacts, 355
Leaf gauge, 355–356, 357f
LeFort I osteotomy, complete, 389–390
Leveling, with lingual appliances, 764–765
  type 1, 765, 765f
  type 2, 765, 766f
  type 3, 765, 767f
Levels of evidence, 201f, 202
Life-long retention, 989
Ligation
  force levels with, 746
  low friction and, 739, 739f
  self-ligation. *see* Self-ligation
Ligatures
  elastomeric, 729–730, 730f, 742–743
  stainless steel, 729
Light intensity, of photocuring, 193–194
Light sources, 817–818
Light-cured adhesives, 193, 814
Light-emitting diodes (LEDs), 817–818, 819f
  curing units, 194
Light-polymerized adhesives, 814, 815f
Line of occlusion, 209
Lingual appliance treatment, 761–777, 762f
  bonding area exposure using, 764
  case study of, 768f–777f, 768b, 773b
  crowding treated with, 765
  customized, 761–762
    illustration of, 762f
    innovation in, 764
  decalcification risk reduced with, 762
  efficient leveling and aligning with, 764–765
    aligning type 1, 765, 765f
    aligning type 2, 765, 766f
    aligning type 3, 765, 767f
  first-order compensations using, 763–764, 763f–764f
  labial techniques difference between, 762–765
  nonextraction case of, 773f–777f, 773b
  target setup model system in, 761
  third-order prescription using, 762–763, 762f
  torque control using, 764
  treatment goals realized with, 761–762
Lingual arch, 410
  lower, 410f
Lingual bonded retainers, 991f
Lingual cleats, 493
Lingual force, 156
Lingual orthodontic appliances, state-of-the-art, 764
Lingual root torque, 157
Lingual self-ligating brackets, 735
Lingual tooth surfaces, anatomic variations of, 762–763, 762f
Lingual-root torque, 458
Lip(s)
  cleft
    complete unilateral, 372f
    prenatal diagnosis of, 368
  lower, facebow lie on, 462
  procumbency, 485–486
  upper, 485, 485f
Lip bumper, 416, 416f
  activating, 468
  characteristics of, 467, 468f
  clinical management of, 468
  definition of, 416
  effect, 744
  fitting, 467–468
  with loops, 468f
  lower arch space gaining using, 466–468
  occlusal view of, 467f
  reactivation of, 456
Lip competency, support and, adult interdisciplinary therapy in, 574, 580f
Lip projection, 264–265
Lip repair, definitive, 372
Lip thickness
  lip projection and, 265
  upper, 485, 485f
Load-bearing capacity, of temporary anchorage device, 517
Load-deflection rate, 164
  of coil spring, 166, 166f
  factors influencing, 170t
  helical coils used to reduce, 173
  wire and, 171

Loading, in orthodontic appliance
direction of, 175–176
manner of, 165–166
Local biological mediators, of orthodontic tooth movements, 88–89, 90f, 91t
Locus, of gene, 34
Long face problem, 607, 610f
Long face syndrome, 320
Long-face, 676t
Long-term stability, treatment timing associated with, 988
Loops
Coffin, 456, 457f, 462f
mesiodistal position of, 181
rectangular, 177–178
T-loops, 181
vertical, 175
in wire, 174, 177–178
Low friction
clinical significance of, 739–740, 740f
ligation and, 739, 739f
Low hyoid bone position, throat form and, 265
Lower airway, 320
Lower arch
expansion of, 214, 214f
space gaining and, 466–468
Lower face, 217
Lower incisors, placement in basal bone, 983
Lower lingual arch, 410, 410f
Lower lip, facebow lie on, 462, 465f
Lower lip procumbency, throat form and, 265
"Low-force technique", self-ligation as, 746
Low-level mechanical vibrations, 918–923
bone response to, biologic mechanism of, 921–923
clinical studies for, 918–921, 919f–920f
and treatment with clear aligners, 921, 921f–922f
Low-pull headgear, 462, 465f
Lyonization, 37
Lysine acetyltransferase 6B, 39

M

Macroaesthetics, 259–282
MACROS memory mnemonic, 937
Magnetic resonance imaging, for upper airway, 323–326
Male, maxillary postnatal growth in, 16, 16f
Mallampati score method, 337
Malocclusion, 254–255
with class I molar, 460f
with class II molar, 458f, 470f–472f
elimination of cause of, 982
etiology of, 31–32
factors of, 31
Hapsburg jaw and, 41
heritability studies in, 31
overcorrection of, 982
ranges of correction in, 213–214
reassessment of, in debonding or debanding, 605, 606f
skeletal, 673–676, 673b, 674f–675f, 676t
skeletal component of, in adult interdisciplinary therapy, 586f, 596–597
Mandible, 16–23
bending and torsion in, 100–101, 102f
development of, 16–18, 17f
differential osteology, 100–101, 101f
functional units of, 18, 18f
postnatal growth of, 20–23, 21f–23f
Mandibular advancement, 714t
Mandibular anchorage preparation, Tweed-Merrifield edgewise appliance and, 498f
Mandibular arch, alteration with appliance therapy and, 983
Mandibular condyle, 18
age-related changes in, 19
articular layer of, 19
growth layer of, 19
growth of, 18–20
mechanism of, 19–20, 20f
Mandibular condyle *(Continued)*
histomorphology of, 18–19, 18f, 18.e1t
zone of endochondral ossification, 19
zone of hypertrophy, 19
zone of maturation, 19
Mandibular deficiency, 224, 672f, 676t, 677f, 695, 696f–698f, 711
Mandibular displacement and rotation, 26, 26f
Mandibular distraction osteogenesis, upper airway and, 332, 333f
Mandibular incisor school, 982
Mandibular inferior border repositioning, 714t
Mandibular intercanine width, 23–24, 23f
Mandibular midline frenum, in periodontal-orthodontic interrelationships, 640
Mandibular molar extrusion, maxillary molar intrusion with, 559b, 560f–562f
Mandibular molar intrusion, 528, 528f–530f
Mandibular molars, mesial movement of, *versus* maxillary molars, 131–132
Mandibular neck, 79, 82f
Mandibular prognathism, 41–42
Mandibular retrusion, 80
Mandibular setback, 714t
Mandibular splint retainer, 990f
Manic-depressive disorder. *see* Bipolar disorder
Manipulative help-rejecters, 294
Manual segmentation, 326
Marginal gingivitis, 218
Martensitic active, NiTi archwires, 192–193
Martensitic stabilized, NiTi archwires, 192–193
mAs, in CBCT, 304
Masseter muscle, muscle fibers of, 39
Masticatory forces, friction in vivo and, 738–739
Material stiffness numbers, 171, 172f
Maturation, craniofacial
differential development and, 2, 2f
growth rates during, 2–3, 3f
Maxilla
differential osteology, 100–101, 101f
postnatal growth of, 14–15, 14f–15f
Maxillary advancement, 331, 714t
Maxillary canine, eruption of, orthodontic considerations associated with secondary bone grafting, 381–382
Maxillary deficiency, 680f–681f, 708
Maxillary excess, vertical, 678f, 698f–699f
Class III mandibular excess and, 705–706
orthognathic surgery for, 678f
Maxillary incisors
central, mesial tipping of, 414
lateral, mesial tipping of, 414
Maxillary inferior positioning surgery, 684–685
Maxillary intercanine width, 23–24, 23f–24f
Maxillary intermolar width, 413
Maxillary midline frenum, in periodontal-orthodontic interrelationships, 640–642, 645f
Maxillary molar intrusion, 526
with mandibular molar extrusion, 559b, 560f–562f
Maxillary molars
first
banding of, 832–833
transpalatal arch and, 409
mesial movement of, mandibular molars *versus*, 131–132
mesial tipping of, after premolar extraction, 794f
Maxillary orthopedics, neonatal, 369–371
Maxillary palatal impactions, in unerupted teeth, ectopically positioned and, 647–650, 653f
Maxillary premolar extractions, 214
Maxillary protraction, 122
bone-anchored, 431–432
Maxillary setback, 714t
Maxillary skeletal excess, 224
Maxillary stabilization plate, 416f
Maxillary transverse deficiency, 718
Maxillomandibular protrusion, lip projection and, 265
Maximal elastic load, 164, 173f–174f
factors influencing, 170t
of wire, 168
Maximal elastic moment, of orthodontic appliance, 164
Maximal stress, sections of, 175
Maximum anchorage, of straight wire appliance, 444
Maximum intercuspal position, 218–219
Maximum intercuspation (MIC), 442, 443f
Maximum mouth opening, 360f
McNamara's airway analysis, 338, 340f
McNamara's method of cephalometric evaluation, 224
Mean, regression to, TMJ and, 362–363
Mechanical loads, bone modeling response to, 88
Mechanical properties, of dental materials, 188t
Mechanics, of temporary anchorage devices
centered approach, shifting from, 516
characteristics of, 511–513
clinical significance of, 513–516, 546b, 547f–549f, 550b, 551f–558f, 554b–555b, 559b, 560f–562f
considerations for, 516–519
force-driven, 522, 523f, 524
high-efficiency, 511–513, 512f
intrusive, 511, 512f
shape-driven, 522, 523f, 524
side effects related to, 519, 519f–521f
Mechanotherapy
modification of, in adult interdisciplinary therapy, 591
plan, 236
temporary anchorage devices in
design, 522–524, 523f
orthodontic, 515–516, 515f–516f
Mechanotransduction
molecular mechanisms of, 88
in orthodontic tooth movement, 87–88, 89f
Meckel's cartilage, 16, 17f
Medial pterygoid muscles, 353–354
Medial two-fifths, of face, 262
Medium anchorage, of straight wire appliance, 444
Melanocortin 1 receptor gene, 34–35
Mendelian traits, 35, 36f
Merrifield, Levern, 484, 484f
Merrifield's contributions, differential diagnosis, 485
Mesenchymal stem cells, 914
Mesh pad, 813
Mesial drift, 986
Mesiodistal position, of loop, 181
Metaanalyses, in orthodontics, 203
Metabolic bone disease, 117–118
osteopenia and, 119, 120f
Metal brackets, 813, 814f
loose, rebonding of, 831
Metal injection molding (MIM), 189
Metals, mechanical properties of, 166–168
Metamorphosizers, 296
Methylation, 38
MIC. *see* Maximum intercuspation (MIC)
Microabrasion, 838–840, 839f
Microaesthetics
close-up images with black background for, 252, 254f
dental, 276–282
bracket placement in, 281–282, 282f
gingival shape and contour in, 276–281, 279f–281f
smile refinement in, 284b, 285f–287f
Microcomputed tomography, in bone physiology, 106
Microdamage, in bone remodeling, 110
Microelectrodes, in bone physiology, 106
MicroEtcher, 825, 825f
Microindentation testing, in bone physiology, 106
Micro-osteoperforations, 913–918
anesthetic for, 914
biologic mechanism of, 913–914, 915f
effect of, on tooth movement, 916
perforations in, distribution of, 915, 916f
technique and indications for, 914–918, 916f–918f

Microradiography
for bone mineralization, 104
transverse, and demineralized white lesions, 888, 889f
Midarch space analysis, dental disharmony and, 492–493
Midcourse correction, 778–780
Midface, 12–16, 12f
deficiency of, 372–373, 373f
development of, 12–13, 13f
postnatal growth of, 13–16, 14f–16f
protrusion, 224
Midline frenum
mandibular, 640
maxillary, 640–642, 645f
Milk, as storage medium, 883
MIM. *see* Metal injection molding (MIM)
Mineralized sections, for bone physiology, 104
Miniaesthetics, 265–273
smile refinement in, 284b, 285f–287f
Minimally invasive approach, in adult interdisciplinary therapy, 583, 585f
Minimum anchorage, of straight wire appliance, 444
Miniscrew implants
bone contact of, 142
bone remodeling and, 142
current status of, 140–143
failure of, 142–143
rigidity of, 143, 143f
Miniscrew-type, of temporary anchorage device, 517
Mirroring techniques, 314f
Missing lateral incisor space, management of, 390–392, 392f
Mixed apnea, 334
Mixed dentition, 462f
arch expansion in, rationale for, 413
open bite in, 463f
patient cooperation issues, 404
tooth size-arch size discrepancy problems and, 409
Mixed dentition protocol, 409
Mixed dentition stage, cleft palate-craniofacial team and, 375–382, 377f–380f
MMT. *see* Modified Mallampati test (MMT)
Mobility, tooth
in adult interdisciplinary therapy, 594–595
force magnitude and, 162–163
induced alveolar remodeling, 598
Modified Mallampati test (MMT), 336–337, 338f
Modulus of elasticity, 167
of dental materials, 188t
Moisture, control of, in binding to enamel, 819
Moisture-insensitive primers, 815
Molar(s), 461f
bonding to, 832–833
class I, malocclusion with, 460f
class II, malocclusion with, 458f, 470f–472f
class III, mechanics of, 468
constriction of, 458
distalization of, 457
eruption of, 458
expansion of, 458
rotation, 457–458
second, upper, 458
self-ligation and, 438–445
straight wire appliance and, 439
third, impaction of, 870
upper right, 459f
upper second, 458
vertical control and, 458–462
Molar distalization, temporary anchorage devices in, 515
archform control in, 538f–540f
biomechanics in, 528
decision making in, 528, 531f
en masse distalization and, 528, 541f
mechanical efficiency in, 528, 532f–536f
mesiodistal axis control in, 537f
methods for, 522f
Molar distalization, temporary anchorage devices in *(Continued)*
three-dimensional control in, 528
torque control in, 538f
vertical control in, 539f–540f
Molar extrusion, mandibular, maxillary molar intrusion with, 559b, 560f–562f
Molar intrusion, temporary anchorage device in, 524–528
archform control during, 525f
biomechanics of, 525–526
decision-making in, 524–525
general effects of, 524f
local effects of, 524f
mandibular, 528
maxillary, 526
with mandibular molar extrusion, 559b, 560f–562f
mechanical efficiency in, 525–526
posterior torque during, 525f
three-dimensional control in, 526, 526f–527f
treatment mechanics in, 526–528
Molar protraction, 148–149
temporary anchorage device in, 528–541
biomechanics in, 541, 543f
decision making in, 528–541
rotation control in, 542f
using asymmetric mechanics, 546b, 547f–549f
vertical control in, 542f
Molar translation, rates of, 133
Molar-to-molar smile, 272–273
Moments (couples), in sign conventions, 155
Moment-to-force (M/F) ratio, in orthodontic appliance, 164
Monoamine oxidase (MAO) A, 293
Monogenic traits, 35–37, 36f
Monomers, in orthodontic resins, 816
Mosaic, 36
Moss' functional matrix theory, 320
Mouth breathing, 320
in periodontal-orthodontic interrelationships, 644–645, 647f
Mouth guards, 884–885
Mouth opening
maximum, 360f
measuring, 360f
restricted, TMJ and, 360
Mouth rinses, fluoride in, 892
Mucogingival considerations, in periodontal-orthodontic interrelationships, 640–645, 644f, 644b
aesthetic considerations and, 642–643, 646f
fiberotomy, 644, 646f
gingival hyperplasia, 642
gingival retention, 642–643
mandibular midline frenum, 640
maxillary midline frenum, 640–642, 645f
mouth breathing, 644–645, 647f
Mucogingival defects, adult interdisciplinary therapy in, 575–583, 582f
Multifactorial traits, 34, 35f
Multiple provider team, in orthognathic surgery, 712
Muscle fiber, composition of, 39
Muscles
pterygoid, medial, 353–354
temporalis, palpation of, 358f
Musculature school, 982
Musculoskeletally stable (MS) position, 353
finding of, 355–357, 356f
location of, 355
"Mushroom technique," 764
Mutation, in craniofacial growth and development, 5
Myosin 1H gene, 39
Myosin heavy chain (MHC) protein isoforms, 39

N

Nance holding arch, 422, 422f
Naproxen, 819
Nasal dorsum, 264
Nasal obstruction, 319–320
Nasal proportions, evaluation of, 264, 264f
Nasal tip, 264
Nasoendoscopy, for upper airway, 326
Nasolabial angle, 264
Nasomaxillary complex, 12–16. *see also* Midface
growth of, 370
Nasopharynx, anatomy of, 320, 321f
National Center for Biotechnology Information (NCBI), 34
National Dental Practice-based Research Network, 205–206, 206f
National Health and Nutrition Examination Survey (NHANES) data, 427
Natural head position (NHP), 217
Nature, *versus* nurture, 38–40
Nausea, 676
NCBI. *see* National Center for Biotechnology Information (NCBI)
Necrosis, of pulp, 880–881
Necrotizing periodontal diseases, 622f–623f, 624
Need, *versus* demand, in orthognathic surgery, 672–676
Negative space, on smile, 272–273, 274f
Neonatal maxillary orthopedics, 369–371
Neonate, in cleft lip/palate, 369–372
Neurocranium, 4
Neuropeptides, orthodontic tooth movements and, 89
NHP. *see* Natural head position (NHP)
Nickel titanium (NiTi) alloys, 167
activation and deactivation curves for, 168f
Nickel-titanium archwire, 192–193, 876
hardness of, 189
"Nickel-titanium" dilemma, 742
Nickel-titanium wires, 742
Night crib appliance, 607–609
Niti thermal-activated coil activation, 444
Nitinol alloys, 167
Nitinol archwires, 947–950, 954
Nitric oxide, 320
No-mix adhesives, 814
Nonextraction treatment, 455–480
Cetlin approach and, 455–456
evolution of technique, 468–479
reduction of patient's compliance, 468–470, 470f–472f
phase I: space-gaining phase, 456–468
phase sequence of, 456
planning of, 455
space-utilization phase and, 456, 468
Nonhealing, 720–722
Nonpenetrance, 36
Nonsteroidal antiinflammatory drugs (NSAIDs), for micro-osteoperforations, 915–916
Nonsuperelastic Niti archwires, 193
Normal occlusion, 209, 209f–210f
Nose, in orthodontics, 264
Novel orthodontic appliance systems, RCTs of, 742
Nuclear volume morphometry, in bone physiology, 106
Nurture, nature *versus*, 38–40

O

Oblique at rest, in oblique photographs, 248–249, 250f
Oblique close-up smile, in oblique photographs, 250, 250f–251f
Oblique on smile, in oblique photographs, 249–250, 250f
Oblique (three-quarter, 45-degree) photographs, 248–250, 250f
Obsessive-compulsive disorder, 291–292
Obstructive hypopneas, 334
Obstructive sleep apnea (OSA), 320, 334
in adults, 336
in children, 335
diagnostic testing of, 340
in women, 335
Occipital-pull chin cup, 430, 430f
Occlusal adjustment, 984
Occlusal condition, alteration of, 362

Occlusal control, in adult interdisciplinary therapy, 577f, 594
Occlusal disharmony, 492
Occlusal forces
  friction in vivo and, 738–739
  redistribution of, adult interdisciplinary therapy in, 571, 575f
Occlusal landmark relationship, adult interdisciplinary therapy in, 573–574, 578f–580f
Occlusal plane
  adult interdisciplinary therapy in, 571, 577f
  changes, in orthognathic surgery, 682
  definition of, 490
Occlusal prematurities, 119, 121f
Occlusal school, 981
Occlusal traits, genetic variation of, 40
Occlusal trauma, tooth movement and, 124–125
Occlusion
  final, of Tweed-Merrifield edgewise, 501f
  proper, 982
Occlusogingival levels, 162f
ODI. *see* Oxygen desaturation index (ODI)
Odontomes, 868, 869f
Old Glory, 202, 202f
OMIM. *see* Online Mendelian Inheritance in Man (OMIM)
1000Genomes Browser, 34
Online Mendelian Inheritance in Man (OMIM), 34
Open eruption exposure technique, for impaction, 873, 873f
OPG, in bone modeling, 111, 112f
Optimal aesthetics, bracket positioning for, 281–282
Optimal bracket placement, 438–439, 439f
Optimal oral hygiene requires professional instructions, 397
Optimal time, for class III treatment regimen, 409
Optimum joint relationship, TMJ and, 354
Optimum orthopedic stability, 355
Optional submental view, 251, 252f
Oral apparatus, 23–24
  in craniofacial complex, 4
Oral appliances, 341–343, 341f–342f
Oral function, in orthognathic surgery, 297
Oral hygiene, for patient motivation, 397–398
OraMetrix Digital Laboratory and Manufacturing, 930
OraScanner, 934
Original edgewise brackets, 482f
Oronasal fistulae, closure of, 378
Oropharynx, 320, 321f
Orthodontic alloys, ideal, 168
Orthodontic appliance, 164–184
  active members of, 164
  alloys in
    basic behavior of, 166–167
    ideal, 168
    modulus of elasticity of, 167
    selection of, 169–173
    shape-memory, 167–168
  attachment of, 176–177
  axial loading of, 165
  compound loading of, 165
  critical sections in, 175
  design of, 184
  fiber-reinforced composite in, 182
  forces delivered by, 155
  friction in, 182–183
  iatrogenic effects of, 887–912
  load-deflection rate in, 164
  loading in
    direction of, 175–176
    manner of, 165–166
  maximal elastic moment of, 164
  metals in, mechanical properties of, 166–168
  moment-to-force ratio in, 158f, 164
  reactive members of, 164
  removable, 398
  spring characteristics of, 164
Orthodontic appliance *(Continued)*
  summary in, 184
  3D biomechanics in, 183–184
  wire cross section in, 168–169
Orthodontic considerations, associated with secondary bone grafting, 381–382
Orthodontic diagnosis
  assessment of radiation dose risk in, 302–304
    factors that influence dose and risk estimation in, 303–304
  computer-assisted surgery in, treatment planning with, 313–315
    surgical navigation in, 314–315, 315f
    surgical planning and simulation in, 313–314
  final considerations in, 315
  treatment planning and, 302–318
Orthodontic flagellation, 942
Orthodontic force system, 522, 523f
Orthodontic forces
  biological responses by, 85–87
  bone modeling response to, by mechanotransduction, 87–88, 89f
  Sharpey fibers and, 57–58, 58f
  tissue reaction and, 57–58
  transmission of, into cellular reaction, 74, 76f
  types of, 57, 58f
Orthodontic intervention, timing of, 408–409
Orthodontic mechanotherapy, temporary anchorage devices in, 515–516, 515f–516f
Orthodontic practice, biological techniques into, 94
Orthodontic problems, 211–213, 212f, 396
Orthodontic retention and relapse, tissue reactions in, 81–83, 83f–84f
Orthodontic retreatment, 673–676, 673b, 674f–675f, 676t, 692b, 715f–716f
Orthodontic Simulator (OSIM), 737f
Orthodontic therapy, 356
  managing TMD symptoms during, 363–364
Orthodontic tooth movement, 57–58, 123–127, 126f–127f. *see also* Tooth movement
  biology of, physical methods of, 93–94
  local biological mediators of, 88–89, 90f, 91t
  neuropeptides and, 89
  periodontal management during, 594–595
Orthodontic treatment
  limitations of, 213–215
  purposes and goals of, 246–255
Orthodontics
  behavior management and, 396–397
  clinical practice of, 484–485
  cone-beam computed tomography in, 304–306
  dental materials science in, 187–199
  evidence in, 200–207
    conclusions in, 206
    future of evidence-based, 205–206
    hierarchy of evidence in, 201–203
    how much of, 203–205
    levels of, 202
    metaanalyses of, 203
    systematic reviews of, 203
    where to find, 205
  genetics and, 31–50
  importance of smile in, 265–268
  optimizing, 403–436
  personalized-precision, 45
  presurgical, 369–370
  quality of life and, 246
Orthodontist
  as bioengineer, 598
  as "gatekeeper," 600
  hygienist of, in adult interdisciplinary therapy, 596
  in treating adults, 570
Orthodontist's role
  in cleft palate-craniofacial team, 369–390
    neonate and infant (birth to 2 years of age), 369–372
  in permanent dentition stages, 388, 389f
Orthognathic surgery, 331–332, 343, 387–388, 427
  applied concepts for types of cases in, 712–713, 714t, 715f–716f
  diagnostic considerations for, 691–695, 692f–694f, 695t, 696f–703f
  emotional well-being affected by, 722–724
  gingival deficiency with recession and, 725
  goals of, 669–672, 670b, 671f–672f
  hypotensive anesthesia in, 676
  infection secondary to, 676–677
  integrated treatment plan for, 706–711, 708b
  lower jaw skeletal-dental problem and, 705–706
  mandibular deficiency corrected with, 679f, 711
  mandibular excess corrected with, 679f
  mandibular sagittal split and, 677–682, 684f–689f
  maxillary horizontal deficiency corrected with, 680f, 708
  medical advances in, 676–677
  nausea secondary to, 676
  need *versus* demand in, 672–676
  objectives of, 670, 670b
  oral health complications and sequelae of, 724–725
  orthodontic aspects of, 669–728
  for patient treatment, 691–711, 692b
  patients with, 294–298
    expectations in, 296
    psychological status and motivation in, 294–295
    satisfaction in, 296–298, 297f–298f
  planning in
    milestones in, 708b
    options for, 695–705
  presurgical orthodontic preparation in, 719
  psychological considerations in, 713t
  pulpal changes secondary to, 725
  range of motion affected by, 722–724, 722t
  risks of, 720–725, 720b, 721f, 723f
  secondary surgery after, 722, 723f
  self-esteem affected by, 724
  sequence of treatment for, 711–712
    importance of, 712
    step 1: multiple provider team selection (interdisciplinary therapy), 712
    step 2: goal clarification, 712
    step 3 through 16, 712, 713t
    step 17: treatment experience, 712
  sinus complications of, 722
  skeletal fixation after, 684–689, 690f–691f
  stability optimization in, 713–720
    orthodontic considerations in, 714
    surgical considerations in, 714–720, 717f, 719f
  steps involved in, 708b
  stereolithographic models in, 691, 693f–694f
  stomatognathic system function affected, 722
  surgical advances in, 677–684, 677f–690f
  surgical treatment objective of, 706
  technologic advances in, 676–689
  template-guided diagnosis and, 694–695, 696f–702f
  temporomandibular dysfunction symptoms reduced with, 722
  timing of, 708
  tooth loss secondary to, 725
  transverse skeletal component and, 695, 703f
  treatment planning and, 387–388
  upper jaw skeletal-dental problem and, 705–706
  vertical maxillary excess corrected with, 678f
Orthopedic expansion, of palate, 629
Orthopedic stability, concept of, 353–355
Orthopedically stable joint position, 354
OSA. *see* Obstructive sleep apnea (OSA)
OSIM. *see* Orthodontic Simulator (OSIM)
Osseointegration, 141–142, 141f
Osseous defects, adult interdisciplinary therapy in, 575–583, 582f
Osseous surgery, in orthodontic therapy, 595
Osteoarthritis, 695t

Osteoblasts, 54, 914
histogenesis and bone formation, 130, 134f–135f
in osteogenesis, 91, 92t
Osteoclastogenesis, RANK/RANKL/OPG system for, 89–91
Osteoclasts, 54, 56f
in osteogenesis, 91, 92t
recruitment, bone resorption and, 130–140, 136f
Osteocytes, response to vibration of, 921–923
Osteogenesis
biological control of, 91–93, 92t
distraction
benefit of, 389–390
in permanent dentition stages, 388–390, 390f–391f
at tension side, 92, 93f
Osteoid, 54, 56f
Osteology, 100–104
differential, of maxilla and mandible, 100–101, 101f–102f
temporomandibular articulation in, 101–104, 102f–105f
Osteons
primary, 108
secondary, 108–110
Osteopenia, metabolic bone disease and, 119, 120f
Osteoporosis, 117–118
Osterix (Osx), in osteogenesis, 92
Outer two-fifths, of face, 262
Overbite correction, straight wire appliance using, 441–443, 442f
Overjet correction, straight wire appliance using, 441–443, 442f
Oxygen desaturation index (ODI), 334
Oxygen saturation, reporting, 334
Oyster brackets, 735–736

P

P2RX7 protein, in external apical root resorption, 44–45
P2X7 receptor, in bone biomechanics, 118
Pain, and force magnitude, 162–163
Paired box 9, mutations of, 42–43
Palatal bar, 456–462
bite closed after treatment with, 463f
clinical use of, 456–462
with distal force, 460f
expanded, 459f
lingual sheath holds, 457f
Palatal height, implant-supported crown and, 83, 86f–87f
Palatal root torque, 464f
Palatally displaced canines (PDC), 43
Palate, orthopedic expansion of, 629
Panic disorder, 293
PAOO. *see* Periodontally Accelerated Osteogenic Orthodontics (PAOO)
PAP. *see* Positive airway pressure (PAP)
Parallelism
of abutment teeth, adult interdisciplinary therapy in, 571, 573f
of root, in debonding or debanding, 602
Particulate grafting, 662–663, 662f
Passive slides, 740–741
Patient
comfort, self-ligation and, 742
compliance
impaction and, 876
reduction of, 468–470, 470f–472f
educating and informing, in adult interdisciplinary therapy, 596
input, in debonding or debanding, 605
motivation, 397
Patient care team, 345
Patient-centered model of care, 927, 929f
Patient-parent conference, in orthodontics, 235, 240b–243b
PCSI. *see* Primary Self-concept Inventory (PCSI)
PDC. *see* Palatally displaced canines (PDC)
PDL. *see* Periodontal ligament; Periodontal ligament (PDL)
Peak height velocities (PHV), 2
Pediatric dentistry, behavior management and, 396–397
Pedigrees, 35–36, 35f–36f
Peel force, 833–834
Pendulum, 470
with skeletal anchorage, 469f
Peptide hormones, 117
Perceptometrics technique, 289–290, 290f
Pericytes, 119–120
Periodontal biotype, 626
Periodontal considerations, reevaluation of, in debonding or debanding, 605
Periodontal differential diagnosis, of adult orthodontic conditions, 586–589
Periodontal health, self-maintenance of, adult interdisciplinary therapy in, 582f, 583
Periodontal ligament (PDL), 52–53, 54f, 99, 154, 881
bone resorption and, 126
response of, 127–130, 131f–133f
sutures and, 119–120, 121f–122f
Periodontal preparation
in adult interdisciplinary therapy, 597
before orthodontic therapy, 595
Periodontal probe, for gingival evaluation, 218
Periodontally Accelerated Osteogenic Orthodontics (PAOO), 93, 470, 594
Periodontal-orthodontic interrelationships, 621–668, 622f–623f
alveolar decortication in, augmentation grafting and, 661–665
early treatment in, 633–634, 636f
high-risk factors of, 624–633
biotype in, 626, 626f
bone-anchored rapid palatal expansion in, 629–633, 631f–635f
gingival recession in, 626
transverse dimension in, 626–633, 627f–630f
inflammatory disease in, 623–624
gingivitis, 623
periodontitis, 623–624, 625f
mucogingival considerations in, 640–645, 644f, 644b
aesthetic considerations and, 642–643, 646f
fiberotomy, 644, 646f
gingival hyperplasia, 642
gingival retention, 642–643
mandibular midline frenum, 640
maxillary midline frenum, 640–642, 645f
mouth breathing, 644–645, 647f
in tooth movement, tissue response to, 634–640, 637f–638f
bodily movement, into a defect, 637–640, 643f
extrusion (eruption), 634–636, 639f
intrusion, 636–637, 640f–642f
tipping, 637
unerupted teeth in, ectopically positioned and, considerations with, 645–657, 648f
ankylosis in, 650–657, 658f–661f
external root resorption in, 650–657, 658f–661f
incisor impactions in, 650, 656f–657f
maxillary palatal impactions in, 647–650, 653f
second molar impaction in, 650, 654f–655f
second premolar impaction in, 650, 654f–655f
surgical technique in, 647, 649f–652f
Periodontitis, 623–624
orthodontics and, 135–136, 138f
Periosteum, 54, 55f
Permanent canine root formation, 378–379
Permanent dentition
arch expansion and, 413
dental luxation and avulsion injuries in, management of, 880–886
stage, cleft palate-craniofacial team and, 382–390, 382f–383f
Personality disorders, 294
Personalized-precision orthodontics, 45
PFE. *see* Primary failure of eruption (PFE)
Pharyngeal critical (Pcrit) pressure, 323
Pharyngometry, for upper airway, 328f, 329
Phase I appliances, 592
Phenotypes, 34
Philippe bracket, 735
Philtrum height, 270, 271f–272f
Phosphate, increasing availability of, for remineralization, 895
Photocuring, 193–195
PHV. *see* Peak height velocities (PHV)
Physical dental casts, virtual *versus*, 219
Physiologic tooth migration, 54–55, 57f, 83
PICO (population, intervention, comparison/control group, and outcome) format, 203
Pierre Robin sequence, 390
Piezocision, 93
Pins, Begg, 730
Placebo effect, in TMJ, 362
Plain-film radiography, for impaction, 870, 871f
Plasma lamps, 194
Plaster models, of teeth, 220
Pliers, debracketing with, 833, 833f
Polarized light birefringence, 104
Polycrystalline ceramic brackets, 192
Polysomnography (PSG), 333
POMR. *see* Problem-oriented medical record (POMR)
Population Reference Bureau, 569–570
Porcelain, bonding to, 825–827, 826f
procedure for, 826, 826f–827f
Porphyromonas gingivalis, in chronic periodontitis, 623
Position, of root, adult interdisciplinary therapy in, 571
Positional therapy, for sleep-disordered breathing, 341
Positioner, in retention planning, 994, 994f
Positioning, of temporary anchorage device, 516–517
Positive airway pressure (PAP), 341
Posterior arch length, 493
Posterior facial height, definition of, 490
Posterior open bite, 226
Posterior space analysis, dental disharmony and, 493
Posterior teeth, occlusal buildup of, 860–861
technical procedure for, 861, 862f
Posteroanterior cephalogram tracing, with skeletal landmarks, 636f
Postlag phase, of tooth movements, 159–160
Postnatal growth
of cranial base, 10–12, 11f
of mandible, 20–23, 21f–23f
of maxilla, 14–15, 14f–15f
of midface, 13–16, 14f–16f
Postretention changes, 985–986, 985f–986f
Postretention irregularity, 986f
Postretention period, changes during, 83, 85f–87f
Postretention registry, 984–989, 985f–988f
Posttreatment stability, 81–83
in orthodontic retention and relapse, 81–83, 83f–84f
postretention period and, changes during, 83, 85f–87f
Power ridges, 791–792, 792f
Practice management perspective, 405
Pragmatists, 296
Pregnancy, 695t
Premolars
extraction of, maxillary molar tipping after, 794f
lateral movement of, 458
straight wire appliance and, 439
Preparation, of temporary anchorage devices, 513, 513f–514f
Pre-pregs, 182
President's Council of Advisors on Science and Technology, on personalized-precision orthodontics, 45
Presurgical nasoalveolar molding, 371f
Pretreatment irregularity, 986f
Pretreatment-posttreatment smiling photographs, 505f
Pretreatment-posttreatment superimpositions, 505f, 509f
Primary alveolar bone grafting, mixed dentition stage and, 377
Primary cementum, 53

Primary dentition
class III malocclusion and, 409
stage, cleft palate-craniofacial team and, 372–375, 373f–376f
Primary failure of eruption (PFE), 43–44, 226, 869
Primary Self-concept Inventory (PCSI), 298–299
Primers, for bonding, 814–815
moisture-insensitive, 815
self-etching, 815, 816f–818f
Principal fibers, of periodontal ligament, 52
Proactive decision making, 942–944
anticipatory orthodontics in, 944, 948f
constraint recognition and management in, 944, 947f
risk analysis in, 944, 946f
smart orthodontics, principle of, 942, 943f–945f
Problem lists
for database programs, 259
as diagnosis, in orthodontics, 215, 215f
Problem-oriented approach, 670
in orthodontics, 215, 230–236
Problem-oriented medical record (POMR), in adult orthodontic conditions, 583
Professional-based model of care, 927, 929f
Profile at rest, in profile photographs, 250, 251f
Profile photograph, 250–251
Profile smile, in profile photographs, 251, 251f
Profound gel, 914
Progress conference report, 674
Progress report, in adult interdisciplinary therapy, 614, 616f
Prolonged treatment time, 913
Propel device, 914
Protraction, 78
maxillary, 122
molar, 148–149
temporary anchorage device in molar, 528–541
biomechanics in, 541, 543f
decision making in, 528–541
rotation control in, 542f
using asymmetric mechanics, 546b, 547f–549f
vertical control in, 542f
Protraction facemask, upper airway and, 331
Protrusion
facial balance affected by, 485
management of, 232–233
Protrusive face, profile line, 486f
PSG. *see* Polysomnography (PSG)
Psychological aspects, of diagnosis, 289–301
patient perceptions in, 289–291
patients having orthognathic surgery in, 294–298
expectations in, 296
psychological status and motivation in, 294–295
satisfaction in, 296–298, 297f–298f
patients with craniofacial deformities in, 298–299
patients with psychological disorders in, 291–294
attention-deficit/hyperactivity disorder, 291
bipolar disorder, 292–293
body dysmorphic disorder, 292
depression, 293
eating disorders, 293
obsessive-compulsive disorder, 291–292
panic disorder, 293
personality disorders, 294
summary in, 299
Psychological disorders, 291–294
attention-deficit/hyperactivity disorder, 291
bipolar disorder, 292–293
body dysmorphic disorder, 292
depression, 293
eating disorders, 293
obsessive-compulsive disorder, 291–292
panic disorder, 293
personality disorders, 294
Psychological issues, in craniofacial deformities, 298
Pterygoid muscles, medial, 353–354
Puberty, growth differences during, 41
PubMed, for orthodontics, 205
Pulp, response of, to trauma, 880–881
pulp canal obliteration as, 881
pulp necrosis as, 880–881
Pumice prophylaxis, 819
Pure moment, 156

Q

QLF. *see* Quantitative light-induced fluorescence (QLF)
Qualification, Angle's system of, 695
Quality of life, in orthognathic surgery, 295
Quantification, Angle's system of, 695
Quantitative light-induced fluorescence (QLF), and demineralized white lesions, 887–888, 890f–891f
digital, 888, 891f
Quantitative measurements, in CBCT, 308–309, 311–313
Quick brackets, 733–734, 734f

R

Radiation dose risk, assessment of, 302–304
Radiofrequency ablation, 343
Radix, 264
Raloxifene (Evista), 117
Ramus
condylar growth and, 79
of mandible, 18
Randomized controlled trials (RCTs), in self-ligation, 742
suggested treatment protocol for, 742
Randomized trials, in orthodontics, 203, 203f
observational studies, many case series and, 204, 204f
systematic review and, 204–205, 204f
RANK
in bone biomechanics, 118–119
in bone modeling, 111, 112f
RANKL
in bone biomechanics, 118–119
in bone modeling, 111, 112f
RANK/RANKL/OPG system, for osteoclastogenesis and tooth movement, 89–91
RAP. *see* Regional acceleratory phenomenon (RAP)
Rapid maxillary expansion (RME), 330, 404
in children, 633–634
Rapid palatal expansion (RPE), 119–120
bone-anchored, 629–633, 631f–633f
RDI. *see* Respiratory Disturbance Index (RDI)
Reactive force management, in targeted therapeutics, 954, 954f–957f
Reactive members, of orthodontic appliance, 164
Rebonding, 831–832
Receptor technology, 303–304
Recession, gingival
alveolar bone dehiscences with, 661
in periodontal-orthodontic interrelationships, 626
Recessive gene/recessive allele, 35
Records, orthodontic, 248–255
cephalometric radiographs as, 253–254
computer imaging in contemporary treatment planning in, 255, 257f
contemporary applications of cephalometry in, 254–255
digital video technology in, 251–252, 252f–253f
facial photographs as, 248–251
intraoral photographs as, 252
Rectangular loops, 177–178, 178f
Rectangular wires, 172–173
thick, 741
RED. *see* Rigid external distraction (RED)
Refinement, 778–780
Regional acceleratory phenomenon (RAP), 93, 113, 132–133, 147, 470, 661–662
Reitan, Dr. Kaare, 125, 127f
Relapse, 981–997
recovery after, 994–995, 995f
Remineralization, 892–895
Removable appliances, in adult interdisciplinary therapy, 591–592
Removable distalizing plate, 464, 466f
Removable intraoral appliances, 398–399
Removable orthodontic appliances, 398
Repetitive RERAs, 334
Replacement resorption, 880
Repositioning, 881, 882f
RERAs. *see* Respiratory effort-related arousals (RERAs)
Residual moment, 181
Resin infiltration, 840, 840f–841f
for demineralized white lesions, 896
Resins, properties of, for fixed retainers, 196–197
Resolution, in CBCT, 304
Respiratory Disturbance Index (RDI), 334
Respiratory effort-related arousals (RERAs), 334
Respiratory tract, 320
Restorative commitment, reaffirmation of, in debonding or debanding, 605
Restricted mouth-opening, TMJ and, 360
Retainers
bonded, 840–855
canine-to-canine lingual, 842–847, 846f–847f
direct-bonded labial, 854–855
fixed, 195–197
flexible spiral wire retainers, 847–854, 850f
Retention, 981–997
appliances for, 991–994, 991f–994f
clinical applications of, 989–991, 990f–991f
duration of, 984, 994
gingival, periodontal-orthodontic interrelationships, 642–643
history of, 981–982
apical base school, 981–982
mandibular incisor school, 982
musculature school, 982
occlusal school, 981
other factors related to, 983–984
problems, in debonding or debanding, 605
report, in adult interdisciplinary therapy, 614–615, 617f
theories of, 982–983
Retention conference report, 674
Retraction
anterior, in extraction treatment, 541
biomechanics in, 541, 544f–545f
decision making in, 541
of maxillary and mandibular dentitions, 550b, 551f–558f, 554b–555b
Retreatment, 673–676, 673b, 674f–675f, 676t, 692b, 715f–716f
Retromolar implant anchorage, 138–140, 140f
Retropositioned mandible, throat form and, 265
Retrospective studies, in self-ligation, 741–742
Reverse curve of Spee, 176, 176f, 261
Reversible lesion, 623
Rheumatic heart disease, 695t
Rheumatoid arthritis, 695t
Riedel, Richard, 984–985, 985f
Rigid anchorage, in temporary anchorage devices, 511
Rigid external distraction (RED), 389–390
Rigid fixation, 684–685
Rigid registration, 307–308
Risk analysis, in proactive decision making, 944, 946f
RME. *see* Rapid maxillary expansion (RME)
Robotic wire bending, in orthodontics, 236
Robustness, in active clips and passive slides, 741
Root
control of, 792–794, 793f–794f
remodeling/repair of, in tooth movement, 125
resorption of
impaction and, 876
tooth movement in, 124–125, 133f
Root canal treatment, 898, 899f
Root cementum, 53, 55f
Root injuries, in temporary anchorage devices, 519, 519f–521f
Root parallelism, in debonding or debanding, 602

Root position, adult interdisciplinary therapy in, 571, 576f
Root resorption, 880, 896
  detection of, 904–908
    at progress, 902, 906f–907f
  external apical, genetic factors and, 44–45, 45f
  hyalinized zones and, 60–64, 62f–65f
Root torque, palatal, 464f
Rotations
  as tooth movement, 69–70, 70f
  toward initial positions, 986–988, 987f–988f
"Roth setup," 437
Roughness, of dental materials, 188, 188f, 188t
Round wire, 169
RPE. *see* Rapid palatal expansion (RPE)
Rummaging exposure, 877
Runt-related transcription factor 2 (Runx2), in osteogenesis, 92
Runt-related transcription factor 2 gene *(RUNX2)*, 39

S

Sagittal appliance, 591
Sagittal malocclusions, spontaneous improvement of, 416–419, 417f–418f
Saliva, increase in pH of, for remineralization, 895
Salivary flow, increase in, for remineralization, 895
SARPE. *see* Surgically assisted rapid palatal expansion (SARPE)
Sassouni, in cephalometric analysis, 225, 225f
"Scammon's curve," 2, 2f
Scanning, 780–782
Schwartz plate, 591
Schwarz appliance, 414–416, 415f
Scintigraphy analysis, for condylar hypertrophy, 711
Sealants
  in bonding, 815
  hydrophilic resin, 815
Second molars
  impaction, in unerupted teeth, ectopically positioned and, 650, 654f–655f
  upper, 458
Second premolar impaction, in unerupted teeth, ectopically positioned and, 650, 654f–655f
Secondary alveolar bone grafting, 378–381
  mixed dentition stage and, 377–382
Secondary bone grafting, orthodontic considerations associated with, 381–382
Secondary cartilage, of mandible, 17–18
Secondary cementum, 53
Second-order bends, in Tweed-Merrifield edgewise appliance, 494
Segmentation, in CBCT, 307, 308f, 326
Selective grinding, in occlusal control, 591
Self-destructive deniers, 294
Self-etching primers, 815, 816f–818f
Selfies, 248, 248f
Self-ligating bracket (SLB) system, 438
  active, new prescriptions for, 479
  aesthetic, 735–736
  biomechanics of, 729–760
  design features in, 438f
  practical consequences for clinical technique in, 756–757
    choice of, 756
    practical tips in, 756–757, 758f–759f
  proposed core advantages of, 736–740
Self-ligation, 731
  current hypotheses in, 743–746, 743f–746f
  effectiveness of torque control and, 742–743
  factors hindering adoption of, 731–735, 731f–735f
  force levels with, 746
  friction and, 736–739
  historical context of, 729–731
  patient comfort and, 742
  psychological factors and adoption of, 735
Self-ligation *(Continued)*
  straight wire appliance and, 438–445
  treatment efficiency and, 741–742
    randomized controlled trials in, 742
    retrospective studies in, 741–742
Sella-nasion-subspinale angle (SNA), 488
Sella-nasion-supramentale angle, 488
Semiautomatic segmentation, 326
Seminars in Orthodontics, 106
Sequential appliance placement, Tweed-Merrifield edgewise appliance and, 495
Sequential mandibular anchorage preparation, of Tweed-Merrifield edgewise appliance and, 495, 498
Sequential tooth movement, Tweed-Merrifield edgewise appliance and, 495
Serial extraction protocol, 410f–412f
"Servosystem hypothesis of mandibular growth," 19–20, 20f
Setup prescription, 934–937, 938f
Severe skeletal discrepancies in the primary dentition, 374–375
Sex chromosomes, 32–33, 32f
SFOT. *see* Surgically facilitated orthodontic therapy (SFOT)
Shape correspondence measurements, in CBCT, 309, 312, 314f
Shape-driven mechanics, 522, 523f, 524
Shape-memory alloys, 167–168
Sharp bend, in wire, 175
Sharpey fibers, 53, 108
  orthodontic forces and, 57–58, 58f
Shedders, 296
SHH. *see* Sonic hedgehog (SHH)
Sickle cell anemia, 695t
Sign conventions, in tooth movements, 155, 155f
Single nucleotide polymorphism, 34, 34f
6-mm specialty, of orthodontics, 203
Skeletal adaptation, 108–111
Skeletal analysis values, 488–490
Skeletal anchorage, 236
  pendulum with, 469f
Skeletal anchors, 140–141
Skeletal compensations, 210–211
Skeletal differential diagnosis, of adult orthodontic conditions, 583–586, 587f–588f, 599f–600f, 602f–605f
Skeletal fixation, after orthognathic surgery, 684–689, 690f–691f
Skeletal landmarks, posteroanterior cephalogram tracing with, 636f
Skeletal limitations, of orthodontic treatment, 607, 610f
Skeletal malocclusions, 673–676, 673b, 674f–675f, 676t
Skeletal maturity, biological indicators of, 405
Skeletal open bite, 225
Skeletal pattern, airway morphologic structure and, 323, 324f–325f
Skeletal-facial considerations, in permanent dentition stages, 383–387, 388f
Skull, frontal section of, 100, 100f–101f
Sleep apnea, 319–352
  central, 335
  chronic obstructive, 320
  mixed, 334
  obstructive, 320, 334
Sleep Disorders Questionnaire, 340t
Sleep hypoventilation, 334
Sleep hypoventilation syndromes, 335
Sleep-disordered breathing, 333–344
  classifications of, 334–335
    central sleep apnea syndrome as, 335
    obstructive sleep apnea (OSA) as, 320, 334
    sleep hypoventilation syndromes as, 335
    upper airways resistance syndrome as, 335
  clinical prediction of, 336
  clinical presentation of, 335–336
  definitions and testing reports in, 333–334
  epidemiologic factors in, 335
  pathophysiologic factors in, 335
  physical characteristics of, 336
  preventative management in, 344
Sleep-disordered breathing *(Continued)*
  tests and questionnaires in, 336–340
    Berlin questionnaire as, 338, 339t
    cephalometric analysis as, 338
    Epworth Sleepiness Scale as, 338
    Friedman classification as, 336–337
    Kushida Index as, 338
    Mallampati score method as, 337
    Sleep Disorders Questionnaire as, 340t
    STOPBang questionnaire as, 338, 339t
  treatment modalities in, 340
  treatment options of, 340–344
    lifestyle and behavioral modification as, 341
    oral appliance as, 341–343
    oropharyngeal exercises as, 343–344
    positive airway pressure as, 341
    surgical treatment as, 343
    upper airway electrical neurostimulation as, 344
Slicer software, in CBCT, 310–311, 311f
Slow palatal expander, 591
Smart Bracket Placement, image-guided, in targeted therapeutics, 947, 950f
Smart orthodontics, in proactive decision making, 942, 943f–945f
SmartClip, 734–735, 735f
Smile, 228–229
  analysis of, 268–269
  asymmetry, 273
  commissure height of, 270, 271f
  crown height and width of, 270, 271f
  dental microaesthetics and, 276–282
  diagnostic analysis of, 270–271
  evaluation of, 265–273
  gingival display of, 270, 271f
  incisor display at rest in, 270, 271f
  incisor display on, 270, 271f
  nonposed, 268–269, 270f
  oblique characteristics of, 276, 276f–278f
  philtrum height of, 270, 271f–272f
  posed, 268–269, 270f
  refinement, 284b, 285f–287f
  social, 270
  transverse characteristics of, 272–273
    arch form in, 273, 275f
    buccal corridor width in, 272–273, 274f
    transverse cant in, 273, 275f
  vertical characteristics of, 271–272
Smile arc, 209f, 270, 271f
Smoking cessation, in orthodontic therapy, 595
SNA. *see* Sella-nasion-subspinale angle (SNA)
Social smile, 270
"Socially debilitating" malocclusions, 409
Sodium fluoride solution, daily rinsing with, 838
Soft palate exercise, for OSA, 343
Soft tissue
  changes, simulation of, 314
  facial, 372–373
  function, 218
    treatment of, limitations in, 214
  intraoral examination of, 217–218
Soft tissue stretching hypothesis, 323
Somatic growth, 1–3
Sonic hedgehog (SHH), 42
SORT. *see* Strength of Recommendation Taxonomy (SORT)
Space, inadequacy of, and impaction, 868
Space analysis
  anterior, dental disharmony and, 492
  posterior, dental disharmony and, 493
Space closure, of Tweed-Merrifield edgewise, 497f
Space gaining, 456
  lower arch, 466–468
  modalities used in, 456
  objectives of, 456
  phase, 456–468
  in sagittal dimension, 456

Space gaining *(Continued)*
sequence, 456
upper arch, 456–466
anchorage, 462
cervical force for, 462, 465f
extraoral force, 462
high-pull headgear, 458
molar distalization, 458
occipital force, 462, 465f
palatal bar, 456–462
removable distalizing plate, 464, 466f
torque, 462
Space-utilization phase, 456, 468
Spatial relationships, of jaw and teeth, 226, 228f–229f
Speech evaluation, 218, 219t
Speed brackets, 731, 732f
Spheno-occipital synchondrosis, 10, 11f
Spherical harmonic description (SPHARM), in CBCT, 309
Splanchnocranium, 4
Spring
characteristics, of orthodontic appliance, 164
design principles of, 179–182
Spring clips, 740
aging of, 741
Spring retainers, 995, 995f
Square wire, 172–173
Stability, 981–997
of molar intrusion, 525
in orthognathic surgery, 713–720
orthopedic, 353–355
of temporary anchorage device, 516
temporomandibular joint, 718
Stabilization report, 674
in adult interdisciplinary therapy, 614–615, 617f
Stainless steel archwires, 189, 195–196
heat treatment of, 197
Stainless steel brackets, 187–190
base-wing joint of, 188–189
friction of, 189–190
hardness of, 189
material properties of, 190
roughness of, 188
stiffness of, 187–188, 188b
torque of, 190, 191b
Stainless steel labially affixed appliances, pretorqued and preangulated, 592–594
Stainless steel ligatures, 729
Stainless steel wires, 437
Standard edgewise, 481–510
historical perspective of, 481
Standardized sequencing, in orthognathic surgery, 712
Statistically computed difficulty factors, probability index variables with, 491t
Steel alloys, 167
Steel brackets, removal of, 833–834, 833f
Steel wires, stainless, 437
Stereolithographic models, 691, 693f–694f
printing and, in orthodontics, 236
Steroid hormones, 117
Stiffness
of stainless steel brackets, 187–188
of wires, 171, 196–197
STO. *see* Surgical treatment objective (STO)
Stochastic effects, 302–303
Stochastic remodeling, of bone, 110
Stomatognathic functions exercises, for OSA, 344
Stomatognathic system, 669
imbalances of, 672
STOPBang questionnaire, 338, 339t
Straight archwire concept, 946
Straight wire appliance (SWA), 178, 178f, 437
anchorage
maximum, 444
medium, 444
minimum, 444
Straight wire appliance *(Continued)*
arch coordination, 441, 441f
biomechanics, contemporary, 437–454
bracket
extraction, 437
placement, 438–439, 439f
straight wire appliance and, 437
translation, 437
case study of, 446f–453f, 446b, 448b, 450b, 452b
closing extraction spaces, 443–444
design and values of, 437
double keyhole loop activation, 444
edgewise appliance and, 742
finishing stage, 445
future directions of, 445–454
intermaxillary elastics, 444–445
leveling and aligning of, 439–440
stage and, 440f
maximum anchorage, 444
medium anchorage, 444
minimum anchorage, 444
Niti thermal-activated coil activation, 444
optimal bracket placement, 438–439, 439f
overbite correction using, 441–443, 442f
overjet correction using, 441–443, 442f
self-ligation and, 438–445
stage 1 of treatment, types of wires, size, and sequence suggested for
in cases with mild crowding, 441t
in cases with moderate to severe crowding, 441t
stage 2 of treatment, types of wires, size, and sequence suggested for
in extraction cases, 445t
in nonextraction cases, 445t
stage 3 of treatment, types of wires, size, and sequence suggested for, 445t
treatment mechanics, 439–445
Tweed-Merrifield edgewise appliance, 495
working stage and, 440–445
Straight-pull facebow headgear, 419
Straight-wire control, Begg mechanics with, 757, 758f–759f
Straightwire technique, 191b
Strength of Recommendation Taxonomy (SORT), 204
Stress distribution, 162, 163f
Stress raisers, in wire, 174–175
Stress-strain relationship, of alloys, 166f
Subluxation, 881
Subspinale-nasion-supramentale angle, 488–489
Subtractive bending, in targeted therapeutics, 951, 953f
Sugar, reducing consumption of, 890–892
Superelastic Niti archwires, 193
Superelastic NiTi (nickel titanium) wires, 168
Superior repositioning splint, 706t
SureSmile technology, 222, 761, 926–930, 929f–930f
in clinical practice, 930–942
custom-designed wire insertion in, 939, 941f
digital diagnostic model for, 930–934, 931f–937f
effectiveness of, 957
outcome evaluation phase of, 942, 942f–943f
pre-SureSmile therapeutic phase in, 930–934, 931f–937f
therapeutic phase, 934–942, 938f–941f
Surface resorption, 880, 896
Surgery, orthognathic, 387–388
treatment planning, 387–388
Surgical lip adhesion, 371–372
Surgical navigation, 314–315, 315f
Surgical splints, 691
Surgical technique, in unerupted teeth, ectopically positioned and, 647, 649f–652f
Surgical treatment objective (STO), 706
Surgically assisted rapid palatal expansion (SARPE), 330, 683f, 705–706
Surgically facilitated orthodontic therapy (SFOT), 684, 690f
Surgical-orthodontic protocol, 470
Sutures, 119–122
angiogenic capillary budding process in, 119–120, 123f–124f
brain expansion and, 7, 7f
expansion of, 121–122, 124f–125f
fontanels and, 7, 8f
growth of, 5–6, 6f
mechanisms of, 6–7, 6f
molecular basis of, 6–7, 7f
healing of, 78
periodontal ligament and, 119–120, 121f–122f
perivascular process in, 122, 125f
response to orthodontic/orthopedic forces, 77–78, 79f–80f
structures of, 76–77, 77f–78f
tissue response in, 74–78, 76f
SWA. *see* Straight wire appliance (SWA)
Synchondrosal growth, mechanism of, 8–10
Synchondroses, 8, 9f
growth of, mechanism of, 8–10
spheno-occipital, 10, 11f
Syndrome, defined, 36
Synostosis, 7–8
Systematic reviews, in orthodontics, 203
observational data, metaanalyses and, 204f, 205
Systemic diseases, periodontitis and, 624

T

TADs. *see* Temporary anchorage devices (TADs)
Tamoxifen, 117
Tannerella forsythensis, in chronic periodontitis, 623
Target setup model system, 761
Targeted remodeling, of bone, 110
Targeted therapeutics, principles of, 946–957
adaptive prescription, 946–947, 949f
additive or subtractive bending in, 951, 953f
complementary force system in, 954, 958f
concurrent mechanics, 950, 952f
consistent force systems in, 954, 954f–957f
constraint management in, 950, 951f
image-guided Smart Bracket Placement and Archform selection in, 947, 950f
minimal archwire replacement, principle of, 947–950, 951f
optimization of archwire and material selection in, 954–957
preferential staging in, 951, 953f
reactive force management in, 954, 954f–957f
shaping in, 951, 953f
Team approach, to cleft palate-craniofacial team, 368–369
Teamwork, for CPAP, 345, 346f
Te-bisphosphate, 113
Technical factors, of radiation dose risk, 303–304
Teeth
eruption of, 54
of original malocclusion, 496
Telephone, first contact by, 216
Tell-show-do, in orthodontics, 396–397
Template-guided diagnosis, treatment planning and, 584–586, 599f–605f
Temporalis muscles
palpation of, 358f
TMJ and, 353–354
Temporary anchorage devices (TADs), 900, 902f–905f
in adult interdisciplinary therapy, 594
in anterior retraction, extraction treatment, 541
biomechanics in, 541, 544f–545f
decision making in, 541
biologic considerations in, 517
biomechanical applications of, 519
general principles of, 519–541
biomechanical considerations in, 511–568, 517f–518f
characteristics of, 511–519
clinical significance of, 511–519
diverse methods using, 523f

Temporary anchorage devices *(Continued)*
for extruded posterior segments, 571–573
geometric positions of, 540f
increased treatment efficiency in, 513–515, 515f–516f
individualized treatment plan in, 519–524
load-bearing capacity of, 517
mechanics
centered approach, shifting from, 516
characteristics of, 511–513
clinical significance of, 513–516, 546b, 547f–549f, 550b, 551f–558f, 554b–555b, 559b, 560f–562f
considerations for, 516–519
high-efficiency, 511–513, 512f
intrusive, 511, 512f
side effects related to, 519, 519f–521f
mechanotherapy design in, 522–524
miniscrew-type, 517
in molar distalization, 515, 528
biomechanics of, 528
decision making in, 528, 531f
en masse distalization and, 528, 541f
mechanical efficiency of, 528, 532f–536f
methods for, 522f
off-center distal placement of, 535f
three-dimensional control in, 528, 537f–540f
in molar intrusion, 524–528
archform control during, 525f
biomechanics of, 525–526
decision-making in, 524–525
general effects of, 524f
local effects of, 524f
mandibular, 528, 528f–530f
maxillary, 526
mechanical efficiency in, 525–526
posterior torque during, 525f
three-dimensional control in, 526, 526f–527f
treatment mechanics in, 526–528
in molar protraction, 528–541
biomechanics in, 541, 543f
decision making in, 528–541
rotation control in, 542f
vertical control in, 542f
optimal treatment plan in, 519–524
in orthodontic mechanotherapy, range of, 515–516
positioning of, 516–517
rigid anchorage in, 511
selection of, 519–522
anatomic factors in, 521f, 522
biomechanical factors in, 522
clinical factor in, 522
simple anchorage preparation in, 513, 513f–514f
stability of, 516
treatment strategy in, 522, 522f
Temporary titanium screws/plates, 236
Temporomandibular articulation, 101–104, 102f–105f
Temporomandibular discrepancies, 105f–106f, 105b
Temporomandibular disorder (TMD), 210
evaluating patient for, 357–362
musculoskeletal stable position, finding of, 355–357, 356f
orthodontic therapy
managing symptoms during, 363–364
in patient with, 353–366
screen examination, 358–362
screen history of, 358
symptoms of, 357–358
treatment plan for, 362–363
Temporomandibular joint (TMJ)
age-related changes of, 101–102, 103f
anatomic structures of, 353
condyles by, 354f
degeneration of, 101–102, 104f–105f
differential diagnosis, of adult orthodontic conditions, 589, 589t, 590f
dysfunction of, differential diagnosis of, 589
formation of, 16–17
Temporomandibular joint *(Continued)*
health and disease, 304–305, 305f–306f
major muscles that stabilize, 353–354
muscle palpation of, 358, 359f
occlusal evaluation, 360–362
pain in, 358
palpated, 219
palpation of, 358–360, 359f
range of mandibular movement, 360, 360f
response to orthopedic forces, 79–80
sounds, clinical significance of, 361–362
stability of, 718
structure of, 79–80
condylar cartilage, 79, 81f
mandibular neck, 79, 82f
temporal component of, 19, 79, 82f
tenderness of, 358
tissue response in, 78–80, 81f
Tension, area of, in molar, 638f
Tentative treatment plan, 235–236
commercialization in, 236
computer image predictions in, 235–236
consent in, 235–236
consent sequence in, 235
custom milling in, 236
final step in, 236
mechanotherapy plan, 236
patient-parent conference in, 235
robotic wire bending in, 236
skeletal anchorage in, 236
stereolithographic models and printing in, 236
treatment response in, 236
Testosterone, facial growth and, 41
Testosterone/estrogen (T/E) ratio, 41
Tetanus prophylaxis, for dental avulsion, 884
Textbooks, for orthodontics, 205
The Angle Orthodontist, 607, 609–610
Theoretic mechanics, 154
Therapeutic aids, for orthognathic surgery, 706t
Therapeutic diagnosis, 705–706, 706t, 707f
Therapeutic model, 934–937, 939f–940f
Therapeutic modifiability, 234–235
Therapeutic scan, 934
Thick rectangular wires, 741
Thick stiff wires, 736–737
Thick-flat biotype, 626
Thin flexible wires, 737–738, 737f–738f
Thin-scalloped biotype, 626
Third molars
extraction of, 989
tooth-size discrepancies and, 984
Third-order bends, in Tweed-Merrifield edgewise appliance, 494–495
3D angular measurements, in CBCT, 309, 312–313, 314f
3D biomechanics, in orthodontic appliance, 183–184
3D components, of CBCT, 309, 312
3D diagnostic records, acquisition of, 309–310
3D facial photography, for diagnosis, in orthodontics, 220
3D linear surface distances in triangular meshes, 308–309, 311–312, 312f
3D surface models, conversion to, in CBCT, 308f, 310
3D tooth movement software, 220, 222f
3D volumetric files, construction of, 310
3D volumetric label maps (segmentation), construction of, 307, 308f
Three-dimensional image analysis procedures, 309–313
Three-dimensional (3D) radiographic imaging, in skeletal differential diagnosis, 583
Throat form, 265, 269f
Thrombocytopenia, 695t
Tip-back bend, 176, 176f
Tipping
crown, 164
in tooth, 637
as tooth movement, 65, 67f–68f
Tissue
deficiency of, 376–377
reactions
orthodontic forces and, 57–58
in orthodontic retention and relapse, 81–83, 83f–84f
in orthodontics, 51–98
summary, 84
response
in periodontium, 59–74
in sutures, 74–78, 76f
in temporomandibular joint, 78–80, 81f
Titanium brackets, 191, 191b
Titanium niobium, 168
T-loops, 181, 182f
TMA cantilever root-spring, 178, 178f
TMA transpalatal arch, 179, 180f
TMD. *see* Temporomandibular disorder (TMD)
TMJ. *see* Temporomandibular joint (TMJ)
"*Tobacco Use and the Periodontal Patient*", 589
Tongue
appliance, 607–609, 612f
evaluation of, 218
exercises, for OSA, 343–344
Tonsil classification, 336–337, 337f
Tonsillectomy, 343
Tooth bud, primary displacement of, 868, 868f
Tooth crowns, traits of, 222
Tooth displacements, 156, 159, 161
Tooth Masseuse device, 93, 920
Tooth migration
eruption and, 23–25, 24f–25f
physiologic, 54–55, 57f, 83
Tooth mobility
in adult interdisciplinary therapy, 594–595
induced alveolar remodeling, 598
Tooth movement, 160f
biological control in, 91–93, 92t
biomechanics of, 155–164
bone density and, 916–917
at cellular and molecular levels, 85–94
centers of rotation on, 156–158
cytokines and hormones in, 914
displacement of, 156
effect of micro-osteoperforations on, 916
force magnitude in, 159–162
frictional forces in, 181–182
general theory of, 157, 158f
initial period of, 59, 59f
initial phase of, 159–160, 913
injury-facilitated acceleration of, 93
in labial and buccal direction, 71, 72f–73f
lag phase of, 159–160, 913–914
laser irradiation-induced acceleration of, 93–94
load-deflection rate in, 164
minimally and non-invasive approaches to, 913–925
moment-to-force ratio for, 164
orthodontic, 123–127, 126f–127f, 913
demineralization and, 147
expedited, 145–149
of growing children, 135
induction response of, 125–127, 127f–130f
occlusal trauma and, 124–125
rate of, 132–135, 138f
remodeling/repair of root in, 125
root resorption and, 124–125, 133f
pattern of, 744, 745f
postlag phase of, 159–160
RANK/RANKL/OPG system for, 89–91
rate of, 159–162, 161f
into reduced alveolar bone height, 73, 74f–75f
response of, in forces, 155
return to former position, 982
secondary period of, 64–65, 66f–67f
sequential, Tweed-Merrifield edgewise appliance and, 495

Tooth movement *(Continued)*
sign conventions in, 155
stress in, optimal force and, 163–164
tissue response to, 634–640, 637f–638f
bodily movement, into a defect, 637–640, 643f
extrusion (eruption), 634–636, 639f
intrusion, 636–637, 640f–642f
tipping, 637
types of, 65–71
bodily movement, 69, 69f, 76f
extrusion, 70–71, 70f
intrusion, 70f–71f, 71
rotation, 69–70, 70f
tipping, 65, 67f–68f
torque, 65–69, 68f–69f
vibration-induced acceleration of, 93
Tooth size, 42–43
Tooth size-arch size discrepancy problems, treatment of, 409–410
Tooth surfaces, lingual, anatomic variations of, 762–763, 762f
Tooth-borne (Hyrax) expander, tooth-tissue-borne and, comparison of, 629
Tooth-borne maxillary expansion appliances, 376
Toothpaste, fluoride in, 892
Tooth-size discrepancies
growth factors and posttreatment change and, 984
interproximal reduction for, 983
reevaluation of, in debonding or debanding, 605
retention affected by, 983
third molars effect on, 984
Tooth-supporting tissues, 52–54
alveolar bone, 54, 56f
gingiva, 52, 52f
periodontal ligament, 52–53, 54f
root cementum, 53, 55f
Tooth-tissue-borne expander
bonded, bone-anchored and, comparison of, 630
tooth-borne (Hyrax) expander and, comparison of, 629
Torque
of stainless steel brackets, 190
as tooth movement, 65–69, 68f–69f
Torque control
clear aligner treatment and, 791–792, 792f
incorrect, 764, 764f
labial appliances and, 764, 764f
lingual appliances and, 764
self-ligating bracket effects on, 742–743
Torsion, in orthodontic appliance, 165, 165f
Torsional stiffness, of dental materials, 190, 190f
Total dentition difficulty analysis, malocclusion correction with edgewise appliance and, 503f
Total dentition space analysis, dental disharmony and, 492
Total facial aesthetic planning, role of orthodontics in, 246–248
Total space analysis, 492, 492t
TPA. *see* Transpalatal arch (TPA)
Trabecular bone, bone remodeling in, 108–110, 109f
Tracheostomy, 343
Tracking technologies, 315
Traction mechanisms, in impaction, 876
inefficient, 877
Trait, 34, 35f
Transabdominal ultrasonography, cleft lip/palate and, 369f
Transcription factors, in craniofacial growth and development, 5
Transformation, in CBCT, 307–308
Transforming growth factor β, in bone modeling, 111, 112f
Transitional occlusion, of Tweed-Merrifield edgewise, 501f
Translation
of canines, 180, 181f
center of rotation for, 156
pure, 161–162, 162f
Translation brackets, 437
Transpalatal arch (TPA), 405, 410f
treatment of tooth size-arch size discrepancy problems, 409
Transpalatal bar
with U-loop positioned generally, 457f
unilateral crossbite and, 461f
Transseptal fibers, 52, 53f
Transverse cant, 273, 275f
Transverse dimension
orthodontic considerations associated with secondary bone grafting, 381
in periodontal-orthodontic interrelationships, 626–633, 627f–630f
treatment envelope of, 633f
Transverse facial proportions, 261–262
Transverse microradiography, and demineralized white lesions, 888, 889f
Transverse plane of space, 229
Transverse smile dimension, 273, 274f
Trauma
as cause of impaction, 868, 869f
to dentoalveolar region, 880
management of, and immediate sequelae for, 881–883
pulpal response to, 880–881
root resorption and, 880
tissue response to, 880
Treatment completion, in adult interdisciplinary therapy, 615, 618f
conference report, 614, 615f, 674
Treatment goals, in orthognathic surgery, 712
Treatment optimization, 255–258
Treatment planning, in orthodontics, 208–244
with computer-assisted surgery, 313–315
considerations in, 232–235
crowding in, management of, 232–233
extraction in, 232–234
incisor repositioning in, 233
orthodontic diagnosis and, 302–318
patient cooperation in, 235, 237f–238f
patient-parent conference in, 235
potential solutions in, 235, 240f–242f
problem list on, 230–232
protrusion in, management of, 232–233
tentative treatment plan for, 235–236
therapeutic modifiability in, 234–235
visual treatment objectives in, 233–234
Treatment planning conference report, 674
Treatment time, prolonged, 913
Treponema denticola, in chronic periodontitis, 623
"T-Rex" configuration of Pendex, 421–422, 422f
Tubes, Tweed-Merrifield edgewise appliance and, 493
Tumor necrosis factor-alpha (TNF-α), and tooth movement, 914
Tungsten carbide, dome-tapered, for removal of excess adhesive, 834–835, 835f–837f
Tweed, Charles, 482–484, 483f
Tweed-Merrifield edgewise appliance, 493–495, 494f
treatment with, 495–496
steps of, 496–502
timing of, 496
Tweed's diagnostic facial triangle, 484f
Typical serial extraction protocol, 410

U

UCLP. *see* Unilateral cleft lip and palate (UCLP)
UDDN. *see* Unilateral disc displacement without reduction (UDDN)
U-loop positioned generally, transpalatal bar with, 457f
Ultrasonography, tracking technology in, 315
Unerupted teeth in, ectopically positioned and, considerations with, 645–657, 648f
ankylosis in, 650–657, 658f–661f
external root resorption in, 650–657, 658f–661f
incisor impactions in, 650, 656f–657f
maxillary palatal impactions in, 647–650, 653f
Unerupted teeth in, ectopically positioned and, considerations with *(Continued)*
second molar impaction in, 650, 654f–655f
second premolar impaction in, 650, 654f–655f
surgical technique in, 647, 649f–652f
Unilateral cleft lip and palate (UCLP), 371
Unilateral disc displacement without reduction (UDDN), 711
Upper airway, 319–352
anatomy and growth of, 320–323, 321f–322f
complications, far-reaching effects of, 319–320
influence of orthodontic treatment on, 329–332
functional orthopedic appliances in, 330–331
orthognathic surgery in, 331–332
rapid maxillary expansion in, 330
summary of orthodontic treatment effects in, 332
treatment including extractions in, 329–330
measurements and imaging in, 323–329, 327f
acoustic rhinometry for, 327–329
cone-beam tomography for, 326–327
pharyngometry for, 329
multidisciplinary approach in, 345
segmentation, 326
sleep-disordered breathing and, 333–344. *see also* Sleep-disordered breathing
summary and conclusions in, 347
Upper airway bypass procedure, 343
Upper airway electrical neurostimulation, 344, 345f
Upper airways resistance syndrome, 335
Upper incisors, proclination of, 745f
Upper lateral incisor, straight wire appliance and, 439
Upper lip thickness, 485, 485f
Upper second molars, 458
"Upper stomion concept," 443–444, 444f
Uvulopalatopharyngoplasty, 343

V

Variable expressivity, 35f–36f, 36–37
Variable-modulus principle, 172–173
Varnish, fluoride in, 892–893
VAS. *see* Visual analog scale (VAS)
Venn diagram, 226–228
Vertebral bodies, of C3 and C4, 407f
Vertical dental problems, 225–226
Vertical dimension, 229
incisal guidance at, adult interdisciplinary therapy in, 571, 577f
increase in, 362
Vertical growers, 323, 325f
Vertical loops, in wire, 175, 175f
Vertical maxillary deficiency, 260, 262f, 383
Vertical maxillary excess (VME), 260, 260f, 678f, 698f–699f
Class III mandibular excess and, 705–706
nonsurgical correction of, 550b
orthognathic surgery for, 678f
Vertical movement, of incisors, 714t
Vertical skeletal problems, 224–225
Vertical thirds, of face, 259f, 261
Vertical-pull chin cup, 430, 431f
VHN. *see* Vickers hardness number (VHN)
Vibration-induced acceleration, of tooth movement, 93
Vickers hardness number (VHN), 189
Videography, for diagnosis, in orthodontics, 219–220
Virtual dental models, 220, 222f
Virtual osteotomies, 314
Viscerocranium, 4
Vision, of evidence-based dentistry, 200–201
Visual analog scale (VAS), 920
Visual treatment objective (VTO), 233–234, 234f, 256f, 444
computer imaging and, 255
questions regarding, 708
VME. *see* Vertical maxillary excess (VME)
Volume, in CBCT, 308, 311
von Willebrand disease, 695t
Voxel-based registration, for longitudinal assessments, 310
VTO. *see* Visual treatment objective (VTO)

W
Websites for various dental specialty organizations, for orthodontics, 205
Wedge of cheese, 405–406, 406f
Wet environments, ceramics in, 191–192
White spot lesions (WSLs), 838, 838f, 887
  associated with fixed orthodontic appliances, 762
Width, of bracket, 176
WIN System, 761
Wind instrument, for OSA, 344
Wire. *see also* Archwires
  amount of, 173–174
  braided, 171
  cross section of, 168–169, 170t
  fiber-reinforced composite in, 182
  flat, 169
  geometry of, 177f
  gingival amount of, 180, 181f
  helical coils in, 173
  larger than 0.018 inch in diameter, 741
  length, 173
  load-deflection rate and, 171
Wire *(Continued)*
  loops in, 174
  material stiffness numbers for, 171
  maximal elastic load of, 168
  maximal stress in, sections of, 175
  nickel-titanium, 742
  normal forces in, 184f
  properties of, for fixed retainers, 196–197, 197b
  rectangular, 172–173, 741
  round, 169
  selection of, 169–173
  sharp bend in, 175
  smaller than 0.018 inch in diameter, 740
  square, 172–173
  stiffness of, 170, 171f
  straight, 178
  stress raisers in, 174–175
  vertical loops in, 175
Wire retainers. *see* Flexible spiral wire retainers
Wire stiffness (W_s), 171, 172f
Wits cephalometric analysis, 225
Wits differential, 705b
Wnt pathway, in osteoblast differentiation, 92
Wolff's law, 597
Woven bone, 107, 107f
W_s. *see* Wire stiffness (W_s)
WSLs. *see* White spot lesions (WSLs)

X
X inactivation, 37
XEDAR, 42
X-linked traits, 36f, 37

Z
Z angle
  in craniofacial analysis, 492
  facial disharmony analysis, 486
  Frankfort mandibular incisor axis angle (FMIA) and, 486f
ZA. *see* Zone of acceptability (ZA)
Zone of acceptability (ZA), 289–291
Zone of endochondral ossification, mandibular condyle, 19
Zone of hypertrophy, mandibular condyle, 19
Zone of maturation, mandibular condyle, 19