SIXTH EDITION

Pediatric Dentistry

INFANCY THROUGH ADOLESCENCE

ARTHUR J. NOWAK
JOHN R. CHRISTENSEN
TAD R. MABRY
JANICE A. TOWNSEND
MARTHA H. WELLS

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Pediatric Dentistry: Infancy Through Adolescence

SIXTH EDITION

Arthur J. Nowak, DMD, MA

Professor Emeritus
Pediatric Dentistry and Pediatrics
University of Iowa
Iowa City, Iowa

Associate Editors:

John R. Christensen, DDS, MS (Ped Dent), MS (Ortho)

Adjunct Associate Professor
Department of Pediatric Dentistry
University of North Carolina at Chapel Hill School of Dentistry
Chapel Hill, North Carolina;
Durham Pediatric Dentistry and Orthodontics
Durham, North Carolina

Janice A. Townsend, DDS, MS

Associate Professor and Chair
Division of Pediatric Dentistry
The Ohio State University College of Dentistry;
Chief, Department of Dentistry
Nationwide Children's Hospital
Columbus, Ohio;
Clinical Associate Professor
Louisiana State University Health Sciences Center School of Dentistry
New Orleans, Louisiana

Tad R. Mabry, DDS, MS

Clinical Professor
Department of Pediatric Dentistry
University of Iowa College of Dentistry
Iowa City, Iowa

Martha H. Wells, DMD, MS

Associate Professor and Program Director
Department of Pediatric Dentistry
The University of Tennessee Health Science Center
Memphis, Tennessee

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1600 John F. Kennedy Blvd.
Ste 1800
Philadelphia, PA 19103-2899

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Contributors

Bhavini Acharya, BDS, MPH
Associate Professor/Program Director
Pediatric Dentistry
University of Texas Health Science Center at Houston
Houston, Texas
Chapter 5 Case Study

Abimbola O. Adewumi, BDS, MPed Dent RCS (Eng)
Clinical Associate Professor and Residency Program Director
Department of Pediatric Dentistry
University of Florida College of Dentistry
Gainesville, Florida
Chapter 29

Ola B. Al-Batayneh, BDS, MDSc, JDB
Associate Professor in Pediatric Dentistry
Department of Preventive Dentistry
Faculty of Dentistry
Jordan University of Science and Technology;
Consultant in Pediatric Dentistry
Head, Special Surgery-Pediatric Dental Surgery Division
King Abdullah University Teaching Hospital
Irbid, Jordan
Chapter 13 Case Study

Alexander Alcaraz, DMD
Assistant Professor of Clinical Dentistry
Pediatric Dentistry
Herman Ostrow School of Dentistry of USC;
Director of Clinical Education
Division of Dentistry
Children's Hospital Los Angeles
Los Angeles, California
Chapter 25 Case Study

Veerasathpurush Allareddy, BDS MBA, MHA, PhD, MMSc
Professor
Department of Orthodontics
Collegiate Director of Clinical Research
College of Dentistry and Dental Clinics
University of Iowa
Iowa City, Iowa
Chapter 5

Sahar M. Alrayyes, DDS, MS
Clinical Associate Professor and Clinic Director
Department of Pediatric Dentistry
University of Illinois at Chicago College of Dentistry
Chicago, Illinois
Chapter 41 Case Study

Homa Amini, DDS, MPH, MS
Professor of Clinical Dentistry and Program Director
Division of Pediatric Dentistry
The Ohio State University College of Dentistry
Columbus, Ohio
Chapter 11

Paul Andrews, DDS, MSc
Assistant Professor
Faculty of Dentistry
University of Toronto
Toronto, Ontario, Canada
Chapter 24 Case Study

Kay S. Beavers, DDS, RDH
Professor
Department of Diagnosis and Preventive Services
University of Oklahoma Health Sciences Center College of Dentistry
Oklahoma City, Oklahoma
Chapter 15

Erica Brecher, DMD, MS
Assistant Professor
Pediatric Dentistry
Virginia Commonwealth University
Richmond, Virginia
Chapter 38

R. John Brewer, EMT-P
Dental Education, Inc.
Bethel Park, Pennsylvania
Chapter 10

Jeffrey N. Brownstein, DDS
Clinical Instructor
Midwestern University College of Dental Medicine-Arizona
Glendale, Arizona;
Clinical Instructor
Department of Dental Anesthesiology
NYU Langone Medical Center
Brooklyn, New York;
Private Practice
Peoria, Arizona
Chapter 6

Paul S. Casamassimo, DDS, MS
Professor Emeritus
Pediatric Dentistry
The Ohio State University;
Department of Dentistry
Nationwide Children's Hospital
Columbus, Ohio
Chapter 1

Zafer C. Cehreli, DDS, PhD
Professor
Department of Pediatric Dentistry
Faculty of Dentistry
Hacettepe University
Ankara, Turkey
Chapter 39 Case Study
Chapter 40 Case Study

Donald L. Chi, DDS, PhD
Associate Professor, Department of Oral Health Sciences, School of Dentistry
Adjunct Associate Professor, Department of Pediatric Dentistry, School of Dentistry
Adjunct Associate Professor, Department of Pediatrics, School of Medicine
Adjunct Associate Professor, Department of Health Services, School of Public Health
University of Washington
Seattle, Washington
Chapter 11

John R. Christensen, DDS, MS (Ped Dent), MS (Ortho)
Adjunct Associate Professor
Department of Pediatric Dentistry
University of North Carolina at Chapel Hill School of Dentistry
Chapel Hill, North Carolina;
Durham Pediatric Dentistry and Orthodontics
Durham, North Carolina
Chapter 19
Chapter 27
Chapter 28
Chapter 28 Case Study
Chapter 31
Chapter 36
Chapter 38

Samuel J. Christensen, DDS, MS
Private Practice
Ankeny, Iowa
Chapter 27 Case Study

Matthew Cooke, DDS, MD, MPH
Associate Professor
Departments of Anesthesiology and Pediatric Dentistry
School of Dental Medicine
University of Pittsburgh
Pittsburgh, Pennsylvania;
Adjunct Clinical Assistant Professor
Department of Oral and Maxillofacial Surgery
School of Dentistry
Virginia Commonwealth University
Richmond, Virginia
Chapter 10

Marcio A. da Fonseca, DDS, MS
Chicago Dental Society Associate Professor and Head
Department of Pediatric Dentistry
University of Illinois at Chicago College of Dentistry
Chicago, Illinois
Chapter 4

William O. Dahlke, Jr., DMD
Associate Professor and Chair
Department of Pediatric Dentistry
Virginia Commonwealth University School of Dentistry
Richmond, Virginia
Chapter 33 Case Study

Kevin J. Donly, DDS, MS
Professor and Chair
Department of Developmental Dentistry
Professor
Department of Pediatrics
University of Texas Health Science Center at San Antonio
San Antonio, Texas
Chapter 21

Zameera Fida, DMD
Department of Dentistry
Boston Children's Hospital
Instructor, Developmental Biology
Harvard School of Dental Medicine
Boston, Massachusetts
Chapter 30

Henry Fields, DDS, MS, MSD
Professor and Division Chair
Orthodontics
The Ohio State University College of Dentistry;
Chief, Section of Orthodontics
Department of Dentistry
Nationwide Children's Hospital
Columbus, Ohio
Chapter 19
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Chapter 38

Catherine M. Flaitz, DDS, MS
Professor
Pediatric Dentistry
The Ohio State University College of Dentistry;
Associate Chief of Dentistry
Department of Dentistry
Nationwide Children's Hospital
Columbus, Ohio
Chapter 2

Fernando L. Esteban Florez, DDS, MS, PhD
Assistant Professor
Department of Dental Materials
University of Oklahoma College of Dentistry
Oklahoma City, Oklahoma
Chapter 21 (boxed material)

Suzanne Fournier, DDS
Postgraduate Program Director
Department of Pediatric Dentistry
Louisiana State University
New Orleans, Louisiana
Chapter 11 Case Study
Chapter 11 (boxed material)

Anna B. Fuks, DDS
Department of Pediatric Dentistry
Hadassah School of Dental Medicine
Hebrew University
Jerusalem, Israel;
Professor Emeritus
The Maurice and Gabriela Goldschleger School of Dental Medicine
Department of Pediatric Dentistry
Tel Aviv University
Tel Aviv, Israel
Chapter 23
Chapter 34

Matthew K. Geneser, DDS
Clinical Associate Professor
Department of Pediatric Dentistry
University of Iowa College of Dentistry
Iowa City, Iowa
Chapter 5

Gayle J. Gilbaugh, RDH
Specialized Care Coordinator/Dental Hygienist
Department of Pediatric Dentistry
University of Iowa College of Dentistry
Iowa City, Iowa
Chapter 20 (boxed material)

Elizabeth S. Gosnell, DMD, MS
Assistant Professor
Division of Pediatric Dentistry and Orthodontics
Cincinnati Children's Hospital Medical Center
Cincinnati, Ohio
Chapter 7
Chapter 8

Erin L. Gross, DDS, PhD, MS
Assistant Professor-Clinical
Division of Pediatric Dentistry
College of Dentistry
The Ohio State University
Columbus, Ohio
Chapter 13
Chapter 18

Steven H. Gross, CDT
President
SML Space Maintainers Laboratories
Headquarters
Chatsworth, California
Chapter 26 (boxed material)

Marcio Guelmann, DDS
Professor and Chair
Department of Pediatric Dentistry
University of Florida
Gainesville, Florida
Chapter 23

Kerrod B. Hallett, MDSc, MPH
Paediatric Dentist
Director, Department of Dentistry
Research Fellow, Clinical Sciences, Murdoch Children's Research Institute
Clinical Associate Professor, University of Melbourne
Melbourne, Victoria, Australia
Chapter 4 Case Study

Kimberly J. Hammersmith, DDS, MPH, MS
Nationwide Children's Hospital
Adjunct Assistant Professor
Division of Pediatric Dentistry
College of Dentistry
The Ohio State University
Columbus, Ohio
Chapter 3 Case Study
Chapter 18 Case Study

Kevin L. Haney, DDS, MS
Professor
Pediatric Dentistry
University of Oklahoma College of Dentistry
Oklahoma City, Oklahoma
Chapter 15

Brian D. Hodgson, DDS
Associate Professor, Program in Pediatric Dentistry
Department of Developmental Sciences
Marquette University School of Dentistry
Milwaukee, Wisconsin
Chapter 23 Case Study

Gideon Holan, DMD
Clinical Associate Professor
Pediatric Dentistry
The Hebrew University-Hadassah School of Dental Medicine
Jerusalem, Israel
Chapter 16

Cody C. Hughes, DMD, MSD
Program Director
Advanced Education Program in Pediatric Dentistry
UNLV School of Dental Medicine
Las Vegas, Nevada;
Pediatric Dentist
Valley Pediatric Dental
Mesquite, Nevada
Chapter 22 Case Study

Michael A. Ignelzi, Jr., DDS, PhD
Adjunct Professor
Department of Pediatric Dentistry
University of North Carolina at Chapel Hill
Chapel Hill, North Carolina;
Lake Jeanette Orthodontics and Pediatric Dentistry
Greensboro, North Carolina
Chapter 36 Case Study

Janice G. Jackson, DMD
Professor and Program Director
Department of Pediatric Dentistry
University of Alabama at Birmingham
Birmingham, Alabama
Chapter 35 Case Study

Faris Jamjoom, BDS, MS
Advanced Implantology Program
Department of Restorative and Biomaterial Sciences
Harvard School of Dental Medicine
Boston, Massachusetts
Chapter 3 Case Study

Michael J. Kanellis, DDS, MS
Associate Dean and Professor
Department of Pediatric Dentistry
University of Iowa College of Dentistry
Iowa City, Iowa
Chapter 22 (boxed material)

Piranit Nik Kantaputra, DDS, MS
Center of Excellence in Medical Genetics Research
Division of Pediatric Dentistry
Department of Orthodontics and Pediatric Dentistry
Chiang Mai University
Chiang Mai, Thailand
Chapter 17

Sharukh S. Khajotia, BDS, MS, PhD
Associate Dean for Research
Professor and Chair, Department of Dental Materials
University of Oklahoma College of Dentistry
Affiliate Associate Professor of Chemical, Biological, and Materials Engineering
University of Oklahoma College of Engineering
Oklahoma City, Oklahoma
Chapter 21 (boxed material)

Lisa Knobloch, DDS, MS
Professor
Division of Restorative Science and Prosthodontics
The Ohio State University College of Dentistry
Columbus, Ohio
Chapter 3 Case Study

Ari Kupietzky, DMD, MSc
Visiting Professor
Department of Pediatric Dentistry
Rutgers School of Dental Medicine
Newark, New Jersey;
Faculty Member
Department of Pediatric Dentistry
The Hebrew University-Hadassah School of Dental Medicine
Jerusalem, Israel
Chapter 23

Clarice S. Law, DMD, MS
Associate Clinical Professor
Sections of Pediatric Dentistry and Orthodontics
Division of Growth and Development
UCLA School of Dentistry
Los Angeles, California
Chapter 26
Chapter 27

Kecia S. Leary, DDS, MS
Associate Professor
Department of Pediatric Dentistry
University of Iowa College of Dentistry
Iowa City, Iowa
Chapter 32

Tad R. Mabry, DDS, MS
Clinical Professor
Department of Pediatric Dentistry
University of Iowa College of Dentistry
Iowa City, Iowa
Chapter 39

Cindy L. Marek, PharmD
Clinical Associate Professor
Department of Oral Pathology, Radiology, and Medicine
University of Iowa College of Dentistry
Department of Pharmacy Practice and Science
University of Iowa College of Pharmacy
Iowa City, Iowa
Chapter 9

Dennis J. McTigue, DDS, MS
Professor Emeritus
Pediatric Dentistry
The Ohio State University College of Dentistry
Columbus, Ohio
Chapter 16
Chapter 35

Beau D. Meyer, DDS, MPH
Research Assistant Professor
Pediatric Dentistry
University of North Carolina at Chapel Hill
Chapel Hill, North Carolina
Chapter 3
Chapter 31 Case Study

Travis Nelson, DDS, MSD, MPH
Clinical Associate Professor
Department of Pediatric Dentistry
University of Washington
Seattle, Washington
Chapter 22

Man Wai Ng, DDS, MPH
Associate Professor, Developmental Biology (Pediatric Dentistry)
Harvard School of Dental Medicine
Chief, Department of Dentistry
Boston Children's Hospital
Boston, Massachusetts
Chapter 30
Chapter 30 Case Study

Arthur J. Nowak, DMD, MA
Professor Emeritus
Pediatric Dentistry and Pediatrics
University of Iowa
Iowa City, Iowa
Chapter 13
Chapter 18
Chapter 20
Chapter 32

Eyal Nuni, DMD
Endodontist, Senior Member
Department of Endodontics
The Hebrew University-Hadassah School of Dental Medicine
Jerusalem, Israel
Chapter 34

Arwa I. Owais, BDS, MS
Associate Professor
Department of Pediatric Dentistry
University of Iowa School of Dentistry
Iowa City, Iowa
Chapter 20

Bhavna T. Pahel, BDS, DDS, MPH, PhD
Adjunct Assistant Professor
University of North Carolina at Chapel Hill
Chapel Hill, North Carolina;
Private Practice
Village Family Dental
Fayetteville, North Carolina
Chapter 19 Case Study

Rocio B. Quinonez, DMD, MS, MPH
Professor and Associate Dean of Educational Leadership and Innovation
Pediatric Dentistry and Academic Affairs
University of North Carolina at Chapel Hill
Chapel Hill, North Carolina
Chapter 19

Diana Ram, DMD
Associate Professor
Chair of the Department of Pediatric Dentistry
School of Dental Medicine
The Hebrew University-Hadassah School of Dental Medicine
Jerusalem, Israel
Chapter 7 Case Study

Steve K. Rayes, DDS, MS
Associate Director, Advanced Training in Pediatric Dentistry
Pediatric Dentistry
NYU-Lutheran;
Chief of Pediatric Dentistry
Department of Dentistry
Southcentral Foundation
Anchorage, Alaska
Chapter 14 Case Study

Issa S. Sasa, BDS, MS
Associate Clinical Professor
Developmental Dentistry
University of Texas Health Science Center at San Antonio
San Antonio, Texas
Chapter 21

Scott B. Schwartz, DDS, MPH
Assistant Professor
Division of Pediatric Dentistry and Orthodontics
Cincinnati Children's Hospital Medical Center
Cincinnati, Ohio
Chapter 31
Chapter 37 Case Study

N. Sue Seale, DDS, MSD
Clinical Professor
Department of Orthodontics and Pediatric Dentistry
University of Maryland School of Dentistry
Baltimore, Maryland
Chapter 22 (boxed material)

Rose D. Sheats, DMD, MPH
Adjunct Associate Professor
School of Dentistry
University of North Carolina at Chapel Hill
Chapel Hill, North Carolina
Chapter 36
Chapter 38

Jonathan D. Shenkin, DDS, MPH
Clinical Associate Professor
Health Policy, Health Services Research, and Pediatric Dentistry
Boston University School of Dental Medicine
Boston, Massachusetts;
Private Practice
Augusta, Maine
Chapter 11

Sujatha S. Sivaraman, BDS, DMD, MPH
Director of Pediatric Dentistry
Family Dental Center East
Columbia, Missouri
Chapter 20 Case Study

M. Catherine Skotowski, RDH, MS
Clinical Assistant Professor
Department of Pediatric Dentistry
College of Dentistry
University of Iowa
Iowa City, Iowa
Chapter 15 (boxed material)
Chapter 32 (boxed material)

Rebecca L. Slayton, DDS, PhD
Professor Emeritus
Department of Pediatric Dentistry
University of Washington School of Dentistry
Seattle, Washington
Chapter 17
Chapter 17 Case Study

Andrew Spadinger, DDS
Pediatric Dentist
Private Practice
Children's Dentistry and Orthodontics
Bridgeport, Connecticut
Chapter 41

Thomas R. Stark, DDS
Pediatric Dentistry and Orofacial Pain
Assistant Professor at Uniform Service Health Science University
Comprehensive Dentistry Program
Schofield Barracks, Hawaii
Chapter 38
Chapter 38 Case Study

William V. Stenberg, Jr., DDS, MPH, MS
Clinical Assistant Professor
Periodontics
Texas A&M University
Dallas, Texas
Chapter 25

Deborah Studen-Pavlovich, DMD
Professor and Graduate Program Director
Department of Pediatric Dentistry
University of Pittsburgh School of Dental Medicine
Pittsburgh, Pennsylvania
Chapter 37

Rosalyn M. Sulyanto, DMD, MS
Instructor of Developmental Biology (Pediatric Dentistry)
Harvard School of Dental Medicine
Associate in Dentistry
Boston Children's Hospital
Boston, Massachusetts
Chapter 30 Case Study

Thomas Tanbonliong, Jr., DDS
Associate Professor of Clinical Dentistry and Clinic Director
Division of Pediatric Dentistry and Dental Public Health
Herman Ostrow School of Dentistry of USC
Los Angeles, California
Chapter 8 Case Study

S. Thikkurissy, DDS, MS
Professor and Division Director
Director, Residency Program
Division of Pediatric Dentistry
Cincinnati Children's Hospital
University of Cincinnati
Cincinnati, Ohio
Chapter 7
Chapter 8

Sherry R. Timmons, DDS, PhD
Clinical Associate Professor
Department of Oral Pathology, Radiology, and Medicine
University of Iowa College of Dentistry
Iowa City, Iowa
Chapter 9

Norman Tinanoff, DDS, MS
Professor
Department of Orthodontics and Pediatric Dentistry
University of Maryland School of Dentistry
Baltimore, Maryland
Chapter 12

Janice A. Townsend, DDS, MS
Associate Professor and Chair
Division of Pediatric Dentistry
The Ohio State University College of Dentistry;
Chief, Department of Dentistry
Nationwide Children's Hospital
Columbus, Ohio;
Clinical Associate Professor
Louisiana State University Health Sciences Center School of Dentistry
New Orleans, Louisiana
Chapter 1
Chapter 24

Elizabeth Velan, DMD, MSD
Pediatric Dentist
Dental Department
Seattle Children's Hospital;
Affiliate Assistant Professor
Department of Pediatric Dentistry
University of Washington School of Dentistry
Seattle, Washington
Chapter 40

Adriana Modesto Vieira, DDS, MS, PhD, DMD
Professor and Chair
Department of Pediatric Dentistry
University of Pittsburgh School of Dental Medicine
Pittsburgh, Pennsylvania
Chapter 37

Craig V. Vinall, DDS, MDS
Predoctoral Program Director
Department of Pediatric Dentistry and Community Oral Health
The University of Tennessee Health Science Center
Memphis, Tennessee
Chapter 40 Case Study

William F. Waggoner, DDS, MS
Private Practice
Las Vegas, Nevada
Chapter 22

Jillian Wallen, BDS, MS
Associate Professor and Chair
Growth and Development
University of Nebraska Medical Center College of Dentistry
Omaha, Nebraska
Chapter 29 Case Study

Karin Weber-Gasparoni, DDS, MS, PhD
Professor
Department of Pediatric Dentistry
University of Iowa College of Dentistry
Iowa City, Iowa
Chapter 14

Martha H. Wells, DMD, MS
Associate Professor and Program Director
Department of Pediatric Dentistry
The University of Tennessee Health Science Center
Memphis, Tennessee
Chapter 24
Chapter 33

A. Jeffrey Wood, DDS
Professor and Chair
Department of Pediatric Dentistry
University of the Pacific Arthur A. Dugoni School of Dentistry
San Francisco, California
Chapter 37 Case Study

J. Timothy Wright, DDS, MS
Bawden Distinguished Professor
Department of Pediatric Dentistry
University of North Carolina at Chapel Hill
Chapel Hill, North Carolina
Chapter 3

Vajahat Yar Khan, BDS
Practicing Pediatric Dentist and Principal
Texas Family Dentistry, PLLC
DBA Creative Smiles and Kidzone Dental
Houston, Texas
Chapter 6 Case Study

Juan F. Yepes, DDS, MD, MPH, MS, DrPH
Associate Professor of Pediatric Dentistry
Department of Pediatric Dentistry
James Whitcomb Riley Hospital for Children
Indiana University School of Dentistry
Indianapolis, Indiana
Chapter 2 Case Study

Audrey Jung-Sun Yoon, DDS, MS
Lecturer, Clinical Faculty
Pediatric Dentistry and Orthodontics
UCLA School of Dentistry
Los Angeles, California
Chapter 26 Case Study

Feda Zawaideh, BDS, ADC (Vic), GradDipClinDent, DClinDent (Melb)
Assistant Professor in Paediatric Dentistry
Jordan University of Science and Technology;
Consultant in Pediatric Dentistry
King Abdullah University Hospital
Al Rabieh-Amman, Jordan
Chapter 9 Case Study

Foreword

Pediatric Dentistry: Infancy Through Adolescence was first published in 1988, has been translated into multiple foreign languages, and is now proceeding into another edition. When honored to write a foreword for this edition, I reviewed what was written in 1988 and realized that what was offered then was still applicable today. Citing some of the language of that preface:

> This book exists because the editors believe that a reference textbook on dentistry for children was needed that was, by design, developmentally organized. This book has been written in the belief that age is so incredibly relevant to pediatric dentistry that by presenting information according to a developmental organization, a student's appreciation of the science and techniques needed to effectively practice dentistry for children will be enlarged.

Those words are still critical for this new edition. In 1988 they seemed almost pioneering in that they allowed the book to use the word "infancy" in its title. It was felt by the editors that the next refinement in preventive dentistry for children had to embrace all children as early in age as possible. Prevention should not wait for the child's linguistic maturity. This customary age of 3 just did not make sense in terms of diet, home care, fluoride needs, habits, and other preventive dentistry concerns. Infancy has to be embraced by the contemporary dentist who values the prevention of dental disease in his or her child patients. Having a dental home cannot come too early for any child.

The developmental design also allows for a focus on the adolescent patient whose needs are unique when compared with children age 12 years and younger. Issues like periodontal disease, trauma, and esthetic dentistry, though important for children of all ages, often become pivotal concerns for these maturing patients.

The other two developmental divisions of the book are the primary dentition years (3 to 6) and the transitional years (6 to 12). All four age-related divisions have their own diagnoses and treatment planning as well as prevention chapters. This allows for the age-related focus that parallels the realities of clinical pediatric dental care.

At some point in the maturity of the clinician who treats children there is an appreciation of how dynamic the world of pediatric dentistry is when assessed by age. The 3-year-old presents an entirely different dentition than the 15-year-old. The panoramic x-ray of a 5-year-old is an image very different from that of a 10-year-old. The behavior of children changes from one recall appointment to the next. Their treatment needs tend to fit into different age zones. This dynamism of change is one of the challenges of pediatric dentistry and for many practitioners one of the joys of this area of clinical practice. This book honors that dynamic.

Jimmy R. Pinkham, DDS, MS
Emeritus Professor of Pediatric Dentistry
University of Iowa

Preface

Why a new edition now?

We live in a world of change. New information is constantly being reported. With the explosion of the internet into every office, home, and pocket, we are inundated with information on new discoveries, new drugs, and new procedures. Time-honored treatments are challenged and replaced with new evidence-based possibilities. And so it is in pediatric dentistry.

Since the 2013 edition, our understanding of the importance of oral health in systemic health has grown; children can grow and develop without cavities; the caries process is preventable; early stress in the infant's life can have profound effects on the child's growth, development, and behavior; common risk factors have been identified that lead to both obesity and cavities; we know more about the contribution of genetics to susceptibility or resistance to dental caries; infant oral health is embraced by many primary care providers, dentists, and parents; the importance of caries risk assessment early in the baby's life during the well-baby visits by primary care providers is now understood; fluoride content of community waters has been updated; fluoride varnish applications have become standard practice; use of esthetic crowns to restore badly destroyed primary teeth is now a common treatment option; use of medicinal agents to arrest the caries process is common; new treatments for vital and necrotic primary pulps are now available; retention of traumatized teeth has been extended; regenerative endodontic techniques for luxated teeth with open apices hold promise; and adolescent health, risky behaviors including smoking/vaping, bullying, and suicide, and transition from pediatric to adult dental supervision have become a part of our practice.

This sixth edition is intended for all audiences from the undergraduate dental student, to residents in training, and, finally, to seasoned practitioners. In addition, those in the allied health professions will also find the text an excellent reference for understanding the oral health needs of all children.

With this edition, we introduce four new associate editors whose enthusiasm and creativity were greatly appreciated by the senior editor. In addition, 35 new contributors have been invited to continue the work contributed by 57 colleagues in the first five editions.

The support this textbook receives is greatly appreciated. The text is recognized by educational programs throughout the world. All chapters have been comprehensively reviewed and updated with evidence-based references and guideline-supported recommendations.

What else is new? The text is now available electronically through the Expert Consult platform. Many chapters are accompanied by case studies and videos to enhance the learning experience.

Thirty years ago the first edition of the textbook was designed around developmental stages of childhood, whereby most textbooks were disease oriented. This format highlights the dramatic changes in the patients we treat as they develop from infancy through adolescence and has proven its benefit to learners.

The foundation for a lifetime of good oral health is established early in the child's life, maybe even before a child is born. We hope this new edition of *Pediatric Dentistry: Infancy Through Adolescence* will provide you with information and tools to provide optimal oral health care for all your pediatric patients.

Lastly, I would like to acknowledge the following individuals from Elsevier who helped produce this edition: Jennifer Flynn-Briggs, Senior Content Strategist; Kelly Skelton and Angie Breckon, Content Development Specialists; Rae Robertson, Senior Content Development Specialist; Brian Salisbury, Book Designer; and Kristine Feeherty, Book Production Specialist.

Arthur J. Nowak

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PART 1

Fundamentals of Pediatric Dentistry

Children are not small adults. Children have unique needs based on their ethnicity, stage of development, family composition, medical history, temperament, and mental well-being. Therefore this first part with 12 chapters provides information and themes pertinent to children of all ages. Unfortunately, children continue to be vulnerable to dental and oral diseases, and those from poor and minority families are the most at risk. This first part will further your understanding of children and set the stage for the remaining four age-related parts.

1

The Importance of Pediatric Dentistry

PAUL S. CASAMASSIMO AND JANICE A. TOWNSEND

CHAPTER OUTLINE

The world continues to change around us, and since the last edition of this textbook, changing social mores in this country and events around the world have shown that children, now more than ever, are vulnerable to oral disease and its complications. The dentist unquestionably has the responsibility to educate and advocate for a childhood free of pain and dental disease for all children regardless of nationality, ethnicity, or socioeconomic background. Fortunately science and technology have improved our ability to care for these children. This chapter attempts to place the changes affecting the oral health of children into a clearer picture for the general practitioner and pediatric dental specialist and create a broader concept of the dental home to best serve the interests of children.

Table 1.1 provides a timeline that depicts the evolution of dentistry for children. The historic mission of pediatric dentistry was threefold: stop the advance of early childhood caries (ECC) with restoration, pulp therapy, and extraction; establish prevention to stop recurrence of disease; and develop a regular care seeker to help ensure oral health into adulthood. In truth, the mission differed little from that for adults. It was not until the turn of the last century that the dental profession recognized the continuing epidemic of ECC and its disparate effect on the poor and underserved minority communities and a broader view of pediatric dentistry began to emerge.[1,2] Overlaying this continued epidemic was a changing social matrix that altered traditional doctor-directed care into a more complex mosaic that still accounted for management of dental caries but within a more complicated set of expectations and conditions.[3] Parents, society, the media, other professionals, and a host of environmental and scientific variables emerged to challenge traditional approaches to the tripartite mission of pediatric dentistry.[4] In Table 1.2, we attempt to lay out these changes, using the multidimensional model of Fisher-Owens et al.[5] The remainder of this chapter explains how these new variables impact the delivery of care to children and preview much of what will follow in more detail on current therapies used in pediatric dentistry.

Prevention and Diagnosis

For decades, our preventive arsenal remained the same, with a four-part message of drink fluoridated water, brush teeth with fluoridated dentifrice, eat wisely (which meant low sugar intake), and see a dentist twice a year. We have moved from a general blanket preventive message to one that is keyed to the individual child and the family's characteristics based on caries risk assessment (CRA), better diagnosis of caries, and an ever-increasing choice of preventive agents, including fluoride varnish and silver diamine fluoride (SDF).[6] Digital radiography and other electronic diagnostic tools offer better assessment of caries progression, adding to our ability to more conservatively and individually manage this disease.[7] Dental sealants have withstood critical review and achieved universal acceptance as a caries preventive technique promising a significant reduction in the lifelong caries experience of many Americans.[8] Also gaining universal acceptance is CRA, which promises to be a useful chairside diagnostic also supporting individualized, patient-centered care. More research is needed to achieve the sensitivity and specificity of a truly useful clinical tool. When CRA is matched with early intervention, the promise of a "cavity-free generation" may finally be realized.[9]

Behavior Guidance

Along with advances in dental science are changes in how children and families engage the dental profession in oral health care. Longstanding beliefs on how best to manage a child's in-office behavior and communicate with families have been reassessed as society changes and views on the value of oral health, acceptance of advice and trust of health professionals, and parental involvement in direct provision of care evolve. No doubt, the generational changes that affect both providers and parents have made interaction with children at chairside more challenging.[10,11] Similarly, the concentration of ECC in the poor and minority child populations and the emergence of cultural subgroups needing care have complicated a hierarchical approach to child behavior.[12,13] The application of basic behavior guidance techniques advocated by the American Academy of Pediatric Dentistry (AAPD) now must be meshed with our deeper understanding of adverse childhood experiences (ACEs) in the lives of those most affected with dental caries and, even more simply, the cultural overlays that, for example, limit touch and gender interactions in a clinical setting.[14] Parents

TABLE 1.1 Milestones in Dentistry for Children in the United States

1900	Few children are treated in dental offices. Little or no instruction in the care of "baby teeth" is given in the 50 dental schools in the United States.
1924	First comprehensive textbook on dentistry for children is published.
1926	The Gies Report on dental education notes that only 5 of the 43 dental schools in the United States have facilities especially designed for treating children.
1927	After almost a decade of frustration in getting a group organized to promote dentistry for children, the American Society for the Promotion of Dentistry for Children is established at the meeting of the American Dental Association (ADA) in Detroit.
1932	A report of the College Committee of the American Society for the Promotion of Dentistry for Children states that in 1928, 15 dental schools provided no clinical experience with children and 22 schools had no didactic information in this area.
1935	Six graduate programs and eight postgraduate programs exist in pedodontics.
1940	The American Society for the Promotion of Dentistry for Children changes its name to the American Society of Dentistry for Children.
1941	Children's Dental Health Day is observed in Cleveland, Ohio, and Children's Dental Health Week is observed in Akron, Ohio.
1942	The effectiveness of topical fluoride applications at preventing caries is described. The Council on Dental Education recommends that all dental schools have pedodontics as part of their curriculum.
1945	First artificial water fluoridation plant is begun at Grand Rapids, Michigan.
1947	The American Academy of Pedodontics is formed. (To a large degree, the start of the Academy was prompted by the need for a more scientifically focused organization concerned with the dental health of children.)
1948	The American Board of Pedodontics, a group formulated to certify candidates in the practice of dentistry for children, is formally recognized by the Council on Dental Education of the ADA.
1949	The first full week of February is designated National Children's Dental Health Week.
1955	The acid-etch technique is described.
1960	Eighteen graduate programs and 17 postgraduate programs in pedodontics exist.
1964	Crest becomes the first ADA-approved fluoridated toothpaste.
1974	The International Workshop on Fluorides and Dental Caries Reductions recommends that appropriate fluoride supplementation begin as soon after birth as possible. (This recommendation was later modified by authorities to start at 6 months of age.)
1981	February is designated National Children's Dental Health Month.
1983	A Consensus Development Conference held at the National Institutes of Dental Health endorses the effectiveness and usefulness of sealants.
1984	The American Academy of Pedodontics changes its name to the American Academy of Pediatric Dentistry.
1995	A new definition is adopted for the specialty of pediatric dentistry by the ADA's House of Delegates: *Pediatric dentistry* is an age-defined specialty that provides both primary and comprehensive preventive and therapeutic oral health care for infants and children through adolescence, including those with special health care needs.
2003	The AAP establishes "Policy Statement on Oral Health Risk Assessment Timing and Establishment of a Dental Home," and issuance of this policy statement will be manifested in several outcomes, including the need to identify effective means for rapid screening in pediatricians' offices, and the mechanisms for swift referral and intervention for high-risk children.
2011	The AAPD establishes the Pediatric Oral Health Research and Policy Center to inform and advance research and policy analysis to promote optimal oral health care for children.
2017	Pediatric Dentistry MATCH results show 676 applicants for 408 positions, which exceeds all other specialties.

AAP, American Academy of Pediatrics; *AAPD*, American Academy of Pediatric Dentistry.

want to be present during treatment and will seek providers who will commit to that practice.[15]

Pharmacologic management of behavior continues to be a major consideration in pediatric dental care but has also seen dramatic change. Sedation deaths have prompted new guidelines, better training, and better patient monitoring and, of course, greater scrutiny of how this service is delivered.[16,17] Drugs such as chloral hydrate, long a staple of pediatric sedation, have largely been replaced by medications thought to be safer and reversible.[18] General anesthesia for dental care has seen a dramatic increase, in part due to the epidemic of ECC. In spite of that pharmacologic option being the top choice of parents, it too is challenged by cost, availability of surgical sites, and growing research on possible effects of anesthetics on early brain development.[19] Also driving the changes in behavior guidance is a greater recognition of the role of pain in behaviors, both chairside and in care seeking.[20]

TABLE 1.2 A Cross-Millennium View of the Changing Character of Pediatric Dentistry

Traditional Elements of Pediatric Oral Care for Children	Current and Future Directions and Their Drivers
Prevention	
• Diagnosis with traditional radiography and caries diagnostics	• Digital radiography and electronic caries detection
• A preventive arsenal composed largely of fluoride options including water fluoridation, fluoride dentifrice, office fluoride, fluoride supplements, and at home over-the-counter (OTC) fluoride rinses	• Prevention continues to emphasize most traditional modalities but now includes fluoride varnish, silver diamine fluoride (SDF), and a caries risk paradigm to the application of fluoride and other techniques, discontinuing supplementation
• Dental sealants to prevent occlusal and pit and fissure caries selectively applied	• Dental sealants now evidence-based and accepted universally as a primary preventive technique and may have therapeutic implications
• Caries risk assessment not considered essential to provision of preventive services	• Caries risk assessment now considered integral to preventive therapeutic plans and compensation for preventive services
Behavioral Guidance	
• Simplistic application of communicative and more advanced techniques based on chairside behaviors and special needs with dentist directing choice	• More sophisticated application of techniques with attention to chairside and other aspects of behavior with a strong parental advisement component
• Parental separation from clinical aspects of care	• Recognition of the changing parental attitudes toward restraint, pharmacologic management, and parental presence
• All children managed with the same paradigm and hierarchy of behavior techniques without regard to systemic, emotional, and other mitigating factors	• Recognition of the effects of poverty on child behavior from toxic stress and adverse childhood experiences (ACEs)
• Pain and anxiety addressed primarily as preoperative treatment need	• Greater understanding and subsequent management of pain and anxiety as factors in care avoidance, social and developmental behaviors, and intraoperative outcomes during dental treatment
• Largely office-based nonpharmacologic management of behavior	• Newer models of advanced behavior guidance using sedation, general anesthesia in-office with dental and medical anesthesiologists and surgery centers
• Simplistic vision of behavior in the dental office based on traditional family structure, majority social characteristics, and middle-class value system	• Recognition of the contribution of culture, poverty, and other nontraditional factors on behavior in the dental office
Treatment of ECC	
• Simplistic armamentarium of composite, amalgam, stainless steel crowns	• Fuller integration of preveneered crowns, zirconia crowns, resin infiltration
• Pulpotomy as a preferred therapy using formocresol, ferric sulfate	• Pulp therapy with fuller range options, including indirect techniques, mineral trioxide aggregate (MTA)
• Emphasis on immediate and primary tooth lifespan success in choice of materials and techniques	• Addition of safety and toxicity concerns in choices of restorative care
• Definitive treatment (restoration or extraction) in most cases of ECC	• Consideration of a range of treatment options including deferral of treatment, use of fluoride and other caries-static agents like SDF teamed with more frequent interventions
Dental Diseases Emphasis	
• Dental caries as the preeminent singular driver of care for children	• Greater recognition of esthetic concerns (fluorosis, tooth whiteness) in pediatric dentistry • Emergence of new conditions such as molar-incisal hypocalcification (MIH) and dental erosion as treatment considerations
Systemic Disease and Conditions and Oral Health	
• Traditional disease entities occurring in predictable patterns allowing application of consistent management	• New disease entities such as obesity and its management considerations; other eating disorders; increase in autism spectrum
• Predictable dental outcomes based on disease progression in special needs patients	• Lifespan elongation with complications of end-organ damage, effects of new medication and growing technology dependence to support life and function

TABLE 1.2 A Cross-Millennium View of the Changing Character of Pediatric Dentistry—cont'd

Traditional Elements of Pediatric Oral Care for Children	Current and Future Directions and Their Drivers
Practice Considerations	
• Paper-based records and office management	• Digitalization of records, billing, imaging, and laboratory procedures
• Simplistic safety orientation (OSHA, NIOSH, CDC)	• Office safety including risk mitigation, growing concerns about radiation exposure with introduction of CBCT and other digital advances; HIPAA changes, waterline management
• Global dental consent procedures	• Changing consent requirements based on procedures
• Regional and training-based care patterns	• Emergence of evidence-based guidelines for pediatric dental care

CBCT, Cone-beam computed tomography; *CDC,* Centers for Disease Control and Prevention; *ECC,* early childhood caries; *HIPAA,* Health Insurance Portability and Accountability Act of 1996; *NIOSH,* National Institute for Occupational Safety and Health; *OSHA,* Occupational Safety and Health Administration.

We more often confront a very young child who has been in pain from dental caries for many days, challenging the simple notion that pain control is confined to administration of local anesthesia.[21]

The science and clinical translation of anxiety, pain, and pharmacologic behavior guidance will likely continue. Our tools will also change as we add more powerful local anesthetics, such as articaine and intranasally administered local anesthesia, and as reversal agents like phentolamine sodium gain acceptance.[22,23] The subsequent chapters in this text address both longstanding and new approaches to management of pain, anxiety, and chairside behavior.

Treatment Options

The treatment of ECC continues to change with the science of materials and understanding of biology of the oral cavity and teeth. Today, clinicians can approach pulpal therapy with more choices and better outcomes. Mineral trioxide aggregate (MTA) and bioactive glass stand to revolutionize both permanent and primary teeth pulpal therapy.[24] Indirect pulp caps with various agents, including traditional calcium hydroxide and more recent glass ionomer cements, have challenged traditional thinking that a primary tooth with a large carious lesion is doomed to invasive pulp therapy.[25,26] Traumatic injuries now benefit from larger and longer studies that direct clinicians to better outcomes. Autotransplantation is now an accepted technique for hopeless permanent teeth due to caries or trauma.[27,28] Advances in regeneration techniques can now add vitality to immature teeth that traditionally had a poor long-term prognosis and may lead to full tooth regeneration in the future, as well as other systemic therapies.[29]

Restorative choices for primary teeth continue to increase. The strip crown, long a stalwart of anterior primary tooth restoration, has been joined by the preveneered stainless steel crown. Zirconia crowns, offering better esthetics, strength, and technique ease, are gaining favor, especially for posterior primary teeth.[30] The open-faced stainless steel crown has joined its less esthetic stainless steel crown in the annals of pediatric history. Bioactive restorative materials may lead to a class of restorative materials with intrinsic reparative abilities.[31]

Perhaps the most exciting advance in care of ECC, especially for very young children, is the expansion of our thinking about treatment urgency, restoration longevity, and adjunctive nonrestorative care. Restoration or extraction once dominated treatment of ECC, and application was often immediate, with accompanying use of behavioral techniques to accomplish care. Today, clinicians can reliably use nonrestorative techniques to stop ECC's advance, such as fluoride varnish and SDF, and avoid a costly general anesthetic that too often is just a precursor to another one later on.[32,33] A Hall technique crown can also be reliably and safely placed to stop caries and restore function, requiring minimal cooperation by the child.[34] The advances in treatment of ECC have made care safer, more effective, and more palatable to many families.

Dental Disease Emphasis and Systemic Disease

ECC has long been and remains the driver of attendance in a dental home, but other conditions have emerged as considerations in pediatric dental care, such as fluorosis, esthetic challenges, and management of molar-incisor hypocalcification.[35] Dental erosion has moved from an isolated condition suggestive of an eating disorder to a more widespread condition of children.[36] These conditions join inherited disorders of the dentition, such as amelogenesis imperfecta, intrinsic staining, and tooth number irregularities, as important intersection points in childhood oral assessment.

Systemic diseases affecting children fall into the purview of dentists caring for children by default. Caring for those children with special needs requires a working knowledge of medical, functional, and social and programming aspects of the lives of these children. Medical advances have prolonged life in many conditions but brought into play technology dependence, such as cerebral shunts and implanted devices, and organ effects from drugs, surgery, or the continued onslaught of the original condition. Care of these children requires the dentist to understand the disease, its treatment, and the effects on oral physiology and function, but more so today, the social and programmatic aspects of the lives of these children.[37] Although pediatric dentists continue to take the lead in caring for the very young with special needs, the intent of our health care system is to transition them to adult care as their dental needs grow outside the usual concerns of pediatric dentistry.[38,39]

Contemporary Practice and Care of Children

As all dental practice moves along the path of growing sophistication and digitalization, those areas addressing care of children follow. Paper-based offices are a thing of the past, and movement to the full electronic office means systems that can track caries risk, health histories, referrals, and serial disease manifestations and treatment. Models, radiographs, and analyses of the developing dentition are now electronic files.[40] As treatment techniques advance using lasers and other approaches, consent procedures for children become more complicated, especially with changing family structures. Office design, toys, and other aspects of dental offices have special considerations when children are a part of the patient family.

The concern of parents about safety of their children requires a more detailed attention to safety considerations, regulations, and office environment. Basic infection control remains a keystone of patient safety, but waterline safety, chemicals in dental materials, and radiation have emerged as special considerations in the dental care of children.[41] A growing area is interprofessional care, which suggests that dentists treating children need to heed obesity, immunizations, and other areas not necessarily considered in the realm of dentistry. Child abuse continues to be a required safety consideration for those treating children.

This Edition

Many of these topics are dealt with in depth in this latest edition. New chapters on management of patients with cleft lip and palate and cariology have been added to address the changing nature of oral health care for children. New online content has been added to enhance the understanding of basic principles and to address advanced topics in pediatric dentistry. The textbook retains its developmental view of pediatric dentistry, validating its changing science, including the benefits of early intervention and new age-related conditions and treatments. The text continues to offer in-depth chapters on many techniques and its age epoch diagnostic sections. Welcome to this sixth edition!

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2

Differential Diagnosis of Oral Lesions and Developmental Anomalies

CATHERINE M. FLAITZ

A wide variety of oral lesions and soft tissue anomalies are detected in children, but the low frequency at which many of these entities occur makes them challenging to clinically diagnose. The purpose of this chapter is to highlight selected oral lesions that are most commonly found in children and pathologic entities that primarily develop in this age group. In addition, oral lesions associated with several genetic disorders and specific malignancies, which may mimic benign or inflammatory conditions, are included to broaden the disease scope. The material is outlined in tables to make this comprehensive subject more succinct and easier to review. The brief description for each entity summarizes the most important clinical information that is relevant to the child patient. Representative examples of these conditions are included to illustrate the characteristic clinical or radiographic features.

Each oral lesion is described according to key points: (1) the most common pediatric age group affected and the gender predilection, (2) the characteristic clinical and radiographic findings of the lesion, (3) the most frequent location for the lesion, (4) the pediatric significance of the lesion, (5) the treatment and prognosis for the lesion, and (6) the differential diagnosis that is pertinent to this age group.

Except for the first table on selected developmental anomalies, the other tables are arranged to capture the primary clinical or radiographic characteristics for the purpose of comparison. The sequential headings for each of the tables include the following disease categories:

Developmental anomalies (Table 2.1, Fig. 2.1)
White soft tissue lesions (Table 2.2, Fig. 2.2)
- White surface thickening lesions
- White surface material lesions
- White subsurface lesions

Dark soft tissue lesions (Table 2.3, Fig. 2.3)
- Red or purple-blue lesions
- Brown-black lesions

Ulcerative lesions (Table 2.4, Fig. 2.4)
Soft tissue enlargements (Table 2.5, Fig. 2.5)
- Papillary lesions
- Acute inflammatory lesions
- Tumor and tumorlike lesions

Radiolucent lesions of bone (Table 2.6, Fig. 2.6)
Mixed radiolucent and radiopaque lesions of bone (Table 2.7, Fig. 2.7)
Radiopaque lesions of bone (Table 2.8, Fig. 2.8)

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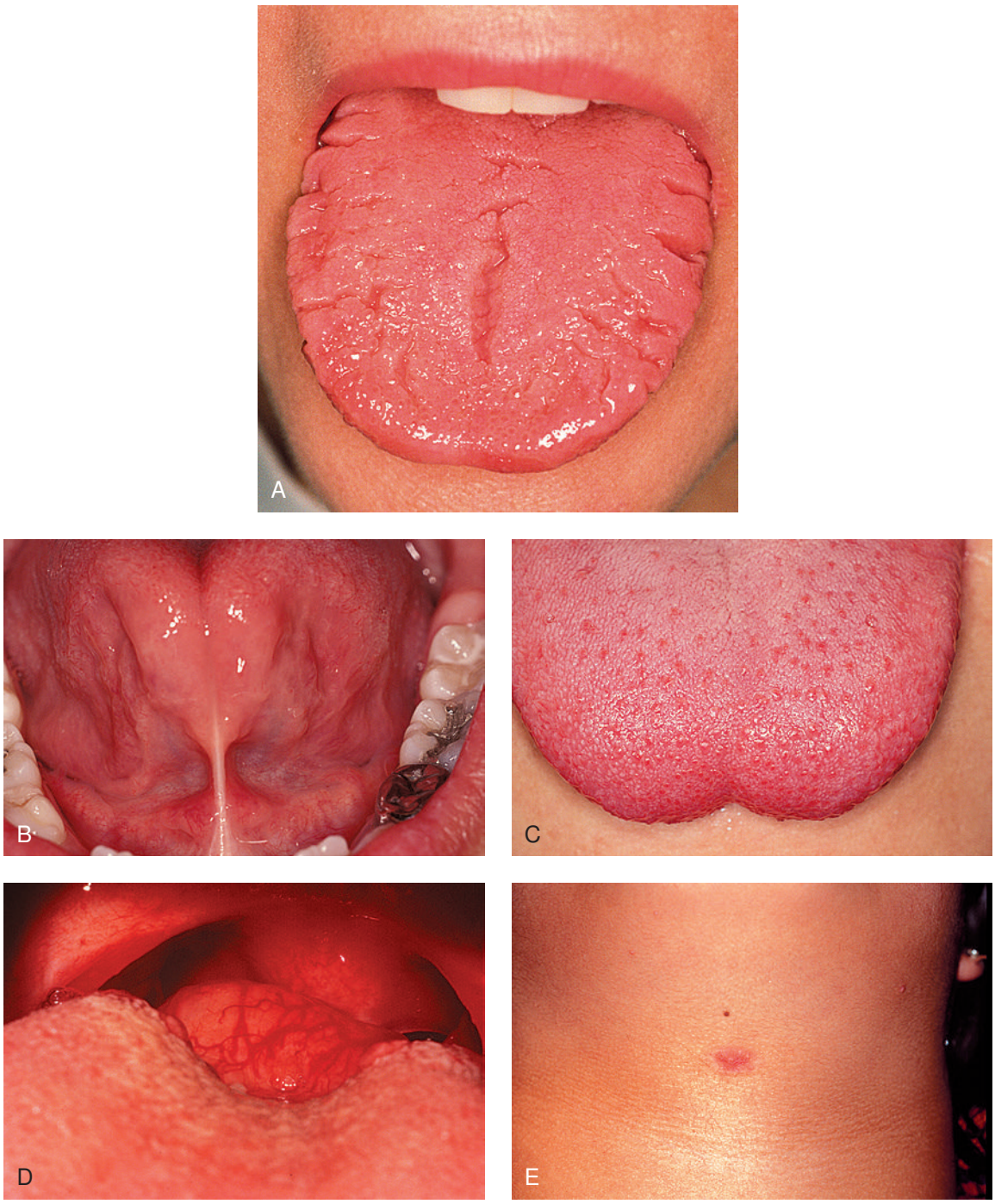
TABLE 2.1 Developmental Anomalies (see Fig. 2.1)

Condition	Pediatric Age and Gender	Clinical Findings	Location	Pediatric Significance	Treatment and Prognosis	Differential Diagnosis
Fissured tongue (scrotal tongue)	First and second decades No gender predilection	Deep central groove; multiple, short furrows; tender, if irritated; may occur with *erythema migrans*	Dorsal and lateral tongue	Polygenic or autosomal dominant trait; occurs in *Down syndrome*, dry mouths, diabetes; detected in 1% of children; source of halitosis	Brush tongue; becomes more prominent with age	Erythema migrans Macroglossia with crenations Hemihyperplasia of tongue Orofacial granulomatosis
Ankyloglossia (tongue-tie)	Present at birth Male predilection	Short, thick lingual frenum or attachment to tip of tongue; may cause slight cleft at tip	Ventral tongue and floor of mouth	Occurs in 2%–11% of infants; rarely causes speech, feeding, swallowing or periodontal problems; multiple frenula associated with *oral-facial-digital* syndrome	Infrequently frenectomy is indicated; many self-correct with age	Bifid tongue Microglossia Palatoglossal adhesion (ankyloglossia superior) Tongue scar
Lingual thyroid	Second decade Female predilection	Nodular mass with pink or red, smooth surface; may cause dysphagia, dysphonia, or dyspnea	Midline base of tongue; *thyroglossal duct cyst* is variant that occurs in midline neck	Symptoms develop during puberty or pregnancy; normal thyroid absent in 70%; important cause of infantile hypothyroidism	Thyroid hormone therapy, excision or radioactive iodine ablation; carcinomas arise in <1%	Lymphoid hyperplasia Hemangioma Lymphangioma Epiglottis
Commissural lip pits	Second decade Male predilection	Unilateral or bilateral depressions or fistulas; fluid may be expressed	Corners of mouth	Occurs in <1% of children; an association with preauricular pits	None required	Paramedian lip pits Angular cheilitis
Paramedian lip pits (congenital lip pits)	Present at birth No gender predilection	Bilateral and symmetric depressions or swellings; fluid may be expressed	Adjacent to the midline of the lower lip vermilion	Autosomal dominant trait; associated with cleft lip and palate; van der Woude and other syndromes	None required; surgery, if cosmetic problem	Mucocele Soft tissue abscess Median lip fissure Double lip Lip piercing
Retrocuspid papilla	First and second decades Female predilection	Asymptomatic, pink, sessile papule or nodule; usually bilateral	Lingual attached gingiva, adjacent to mandibular canines	Very common in children and regresses with age	None required; normal anatomic variation	Irritation fibroma Giant cell fibroma Soft tissue abscess
Bifid uvula	Present at birth No gender predilection	Midline groove or splitting of uvula; may have speech impairment	Midline, posterior soft palate	Minimal expression of cleft palate; marker for submucous palatal cleft; associated with *Loeys-Dietz syndrome* and others	None required; genetic counseling may be indicated	Traumatic defect

Continued

TABLE 2.1 Developmental Anomalies (see Fig. 2.1)—cont'd

Condition	Pediatric Age and Gender	Clinical Findings	Location	Pediatric Significance	Treatment and Prognosis	Differential Diagnosis
Hyperplastic labial frenum	Present at birth No gender predilection	Thick triangular band of pink soft tissue; may be associated with gingival recession or diastema	Midline labial mucosa and gingiva; both maxillary and mandibular lip	Bleeds freely when lacerated; multiple frenula associated with *oral-facial-digital syndrome;* rare breastfeeding problems	None required; frenectomy for some large diastemas, gingival recession or lip mobility problems	Traumatic scar Frenal tag
Torus palatinus (palatal torus)	Second decade Female predilection	Bony hard mass that varies in size and shape; asymptomatic, unless traumatized; rarely seen as radiopacity on radiographs	Midline hard palate	Most tori in this age group are slightly elevated with a smooth surface; autosomal dominant inheritance or multifactorial influence	None required; will continue to grow during adulthood	Prominent median palatal raphe Palatal exostosis Median palatal cyst
Torus mandibularis (mandibular torus)	Second decade Male predilection	Bony hard mass that varies in size and shape; asymptomatic, unless traumatized; radiopacity may be superimposed over roots of teeth	Bilateral, lingual mandible	Less common than torus palatinus; genetic and environmental influence	None required; will continue to grow during adulthood	Exostosis Peripheral osteoma Proliferative periostitis Fibrous dysplasia Condensing osteitis Idiopathic osteosclerosis
Exostosis	Second decade No gender predilection	Single or multiple bony hard nodules; asymptomatic, unless traumatized; radiopacity may be superimposed over roots of teeth	Maxillary and mandibular alveolar ridge on the facial aspect; usually bilateral; may occur on the palate	Exostoses that are traumatized mimic odontogenic infection because of the location; may be tender to palpation in children	None required; will continue to grow during adulthood	Peripheral osteoma Proliferative periostitis Ectopic tooth eruption Condensing osteitis Idiopathic osteosclerosis



• **Figure 2.1** Developmental anomalies. (A) Fissured tongue. (B and C) Partial ankyloglossia with lingual frenum attachment at the tip of the tongue (B). Note the restricted mobility of the tongue with extension (C). (D) Lingual thyroid of the midline base of the tongue. (E) Thyroglossal duct cyst with sinus tract, midline neck.

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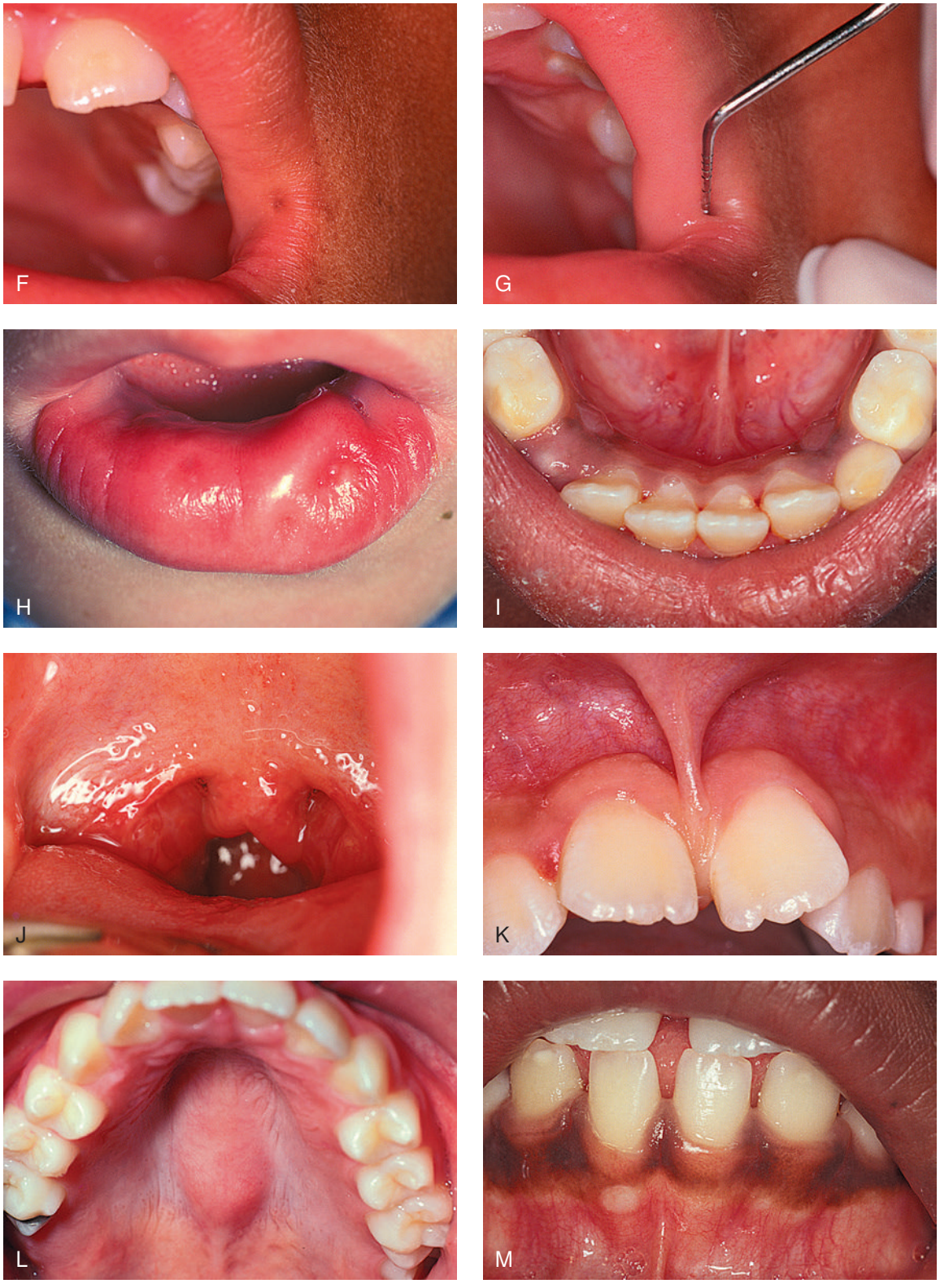


• **Figure 2.1, cont'd** (F and G) Commissural lip pit (F) with depth illustrated by periodontal probe (G). (H) Paramedian lip pits. (I) Retrocuspid papilla of the lingual mandibular gingiva. (J) Bifid uvula. (K) Hyperplastic maxillary labial frenum. (L) Torus palatinus of the midline hard palate. (M) Small exostosis of the anterior mandibular alveolus, facial aspect. ([D] Courtesy Dr. G.E. Lilly, University of Iowa College of Dentistry.)

TABLE 2.2 **White Soft Tissue Lesions (see Fig. 2.2)**

Lesion	Pediatric Age and Gender	Clinical Findings	Location	Pediatric Significance	Treatment and Prognosis	Differential Diagnosis
White Surface Thickening Lesions						
Frictional keratosis and Morsicatio mucosae oris	First and second decades No gender predilection	Localized to diffuse, white, rough or shredded patches; adherent; asymptomatic	Mucosa adjacent to occlusal plane, including buccal, labial mucosa, lateral tongue; attached gingiva	Caused by chronic nibbling habits *(morsicatio),* irritation from orthodontic appliances, fractured teeth, and improper tooth brushing	Elimination of cause; lesion regresses; acrylic splint therapy for severe cases of *morsicatio*	Leukoedema Linea alba Smokeless tobacco keratosis Cinnamon contact stomatitis Lupus erythematosus Hyperplastic candidiasis
Smokeless tobacco keratosis (tobacco pouch keratosis)	Second decade Male predilection	Diffuse, white, wrinkled patch; adherent; asymptomatic; gingival recession; tooth staining; caused by snuff or chewing tobacco	Vestibular, labial and buccal mucosa; usually mandibular site	Highly addictive habit; lesions develop after 1–5 years of use; increased risk for periodontal disease, dental caries, tooth sensitivity, and halitosis	Discontinuation of habit results in lesion reversal; biopsy of persistent lesions; low risk for malignant transformation	Leukoedema Frictional keratosis Cinnamon contact stomatitis Chronic hyperplastic candidiasis
Leukoedema	First and second decades No gender predilection	Widespread, filmy white, wrinkled mucosa; adherent; disappears when stretched	Bilateral buccal, labial mucosa and soft palate	Most prominent in black children; condition increases with age; more pronounced in cigarette smokers	None required; common variant of normal mucosa	Frictional keratosis Linea alba White sponge nevus
Cinnamon contact stomatitis	Second decade No gender predilection	Oblong to broadly linear, white plaques with a shaggy, thickened surface; diffuse erythema; tender	Gingiva, mucosa adjacent to occlusal plane, including buccal mucosa and lateral tongue	Cinnamon flavoring in candy, chewing gum, toothpaste, mouth rinses	Identify and discontinue use of offending product; lesions resolve within 1 week	Morsicatio mucosae oris Hyperplastic candidiasis Smokeless tobacco keratosis Hairy leukoplakia

Continued

TABLE 2.2 **White Soft Tissue Lesions (see Fig. 2.2)—cont'd**

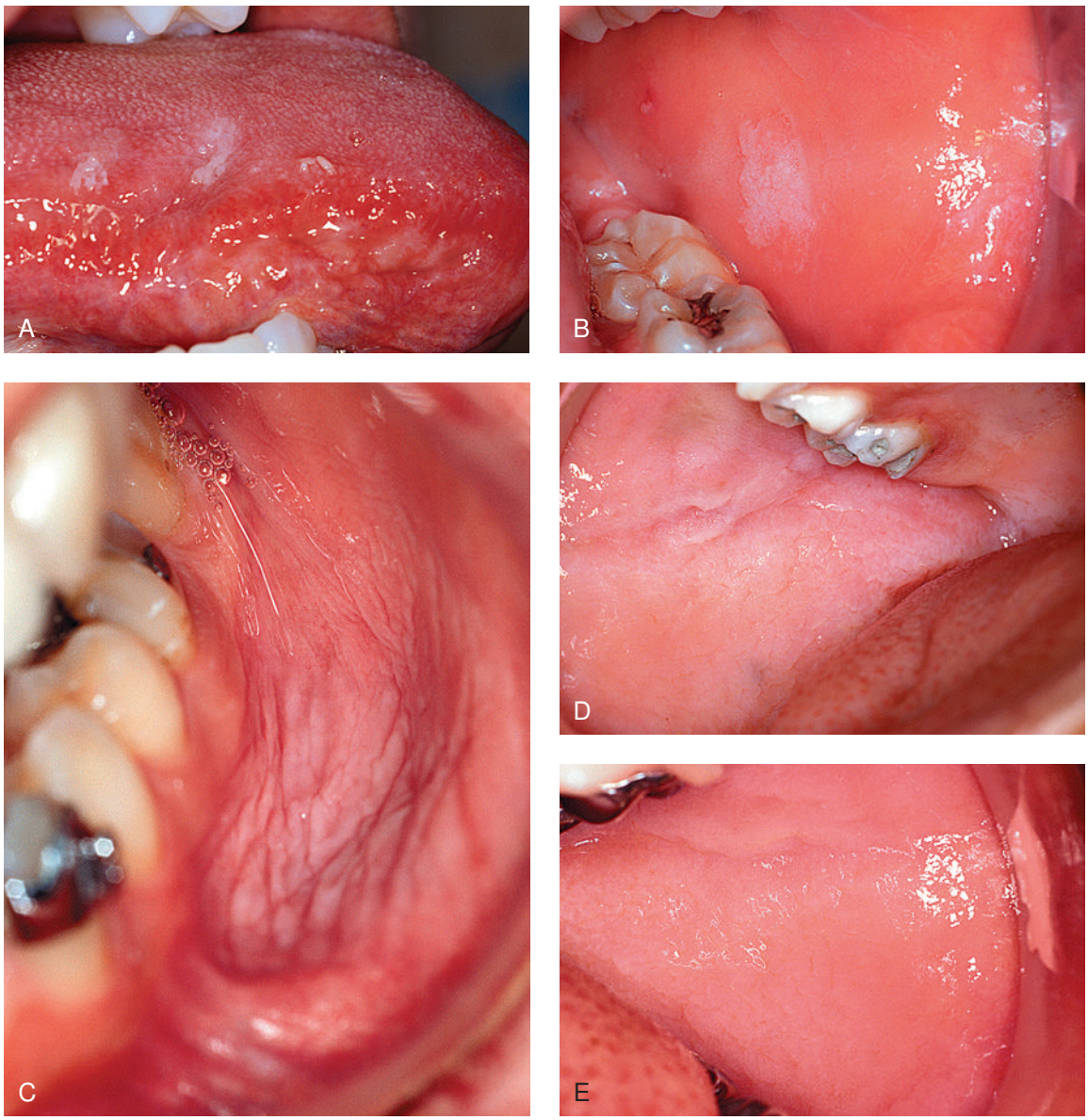
Lesion	Pediatric Age and Gender	Clinical Findings	Location	Pediatric Significance	Treatment and Prognosis	Differential Diagnosis
Linea alba	Any age following the eruption of teeth; female predilection	Smooth or shaggy white line; may be scalloped; asymptomatic	Bilateral buccal mucosa, along occlusal plane	Associated with biting irritation or sucking habit; may be associated with leukoedema	None required; may spontaneously regress	Cinnamon contact stomatitis Scar formation Morsicatio mucosae oris
Hairy tongue	Second decade No gender predilection	Cream to brown discoloration; diffuse elongation of filiform papillae	Dorsal tongue	Contributes to halitosis; associated with cigarette smoking, poor oral hygiene, antibiotics, dry mouth, overuse of mouth rinses; coated tongue is more common in children	Eliminate cause; brush tongue	Coated tongue Frictional keratosis Hyperplastic candidiasis
White sponge nevus	First decade, may be present at birth No gender predilection	Diffuse, symmetric, corrugated, or velvety white plaques: adherent; asymptomatic; persistent	Bilateral buccal mucosa is most common; also found on labial mucosa, ventral tongue, floor of mouth, and soft palate	Autosomal dominant skin disorder; defect in *keratin 4* and *keratin 13;* extraoral sites may be involved; reaches full expression during adolescence	None required; condition stabilizes in young adulthood	Leukoedema Hereditary benign intraepithelial dyskeratosis Frictional keratosis Hyperplastic candidiasis Syndrome-related leukoplakia
White Surface Material Lesions						
Pseudomembranous candidiasis (thrush)	Any age, especially infancy No gender predilection	Widespread, white plaques that wipe off leaving a normal or red, raw base; mild burning	Any mucosal site but common on buccal mucosa, tongue, and palate	Caused by *Candida albicans* and other species; contributing factors are antibiotics, steroids, immune suppression; infants may have diaper rash; pacifiers, orthodontic appliances, and toothbrushes may harbor fungus	Antifungal medication and proper oral hygiene; may recur if cause is not eliminated	Plaque Chemical burn Coated tongue Oral mucosal peeling Morsicatio mucosae oris Koplik spots of rubeola

Chemical burn	First and second decades No gender predilection	Localized or widespread, white nonadherent plaques, erosions or ulcers; tender to painful; sudden onset	Any site but common on lips, tongue, buccal mucosa, and gingiva	Multiple chemicals and drugs may cause this reaction, including those used in dentistry—inappropriate use of mouth rinses, topical anesthetics, phenol, formocresol	Identify and remove cause; ask about homeopathic remedies; symptomatic relief management	Thermal burn Pseudomembranous candidiasis Coated tongue Oral mucosal peeling Mucous patch of syphilis
Coated tongue (furred tongue)	First and second decades No gender predilection	White or yellow nonadherent coating on tongue; asymptomatic; may be source of halitosis	Dorsal tongue	Common condition associated with mouth breathing, febrile illnesses, dehydration, poor oral hygiene; source of halitosis	Brushing tongue and adequate hydration; tends to recur	Pseudomembranous candidiasis Hairy tongue White strawberry tongue
Oral mucosal peeling	Second decade No gender predilection	Translucent to white strips of mucosa that peel off; stringy and slimy in texture; may burn	Buccal and labial mucosa, tongue	Associated with detergents and other ingredients in toothpastes and mouth rinses	Identify and discontinue the oral hygiene product; resolves spontaneously	Plaque Pseudomembranous candidiasis Allergic contact stomatitis Thermal/chemical burn
White Subsurface Lesions						
Scar formation (cicatrix)	First and second decades No gender predilection	White or pale pink line or irregular patch with smooth surface; cross-hatch or starburst pattern; asymptomatic	Any site but common on labial mucosa, lip vermilion, tongue	History of oral trauma or surgery; may represent child abuse or self-mutilation	None required; scar revision if cosmetic concern or if restricts function	Linea alba Mucosal graft Lichen planus
Fordyce granules	Second decade Male predilection	Small, yellow-white, multifocal papules; discrete or clustered; asymptomatic	Bilateral buccal mucosa, retromolar pad and upper lip vermilion	Oral sebaceous glands occur in 20%–30% of children; puberty stimulates development	No treatment is necessary; may increase in size; laser treatment for cosmetics	Frictional keratosis Scar formation Pustules Milia

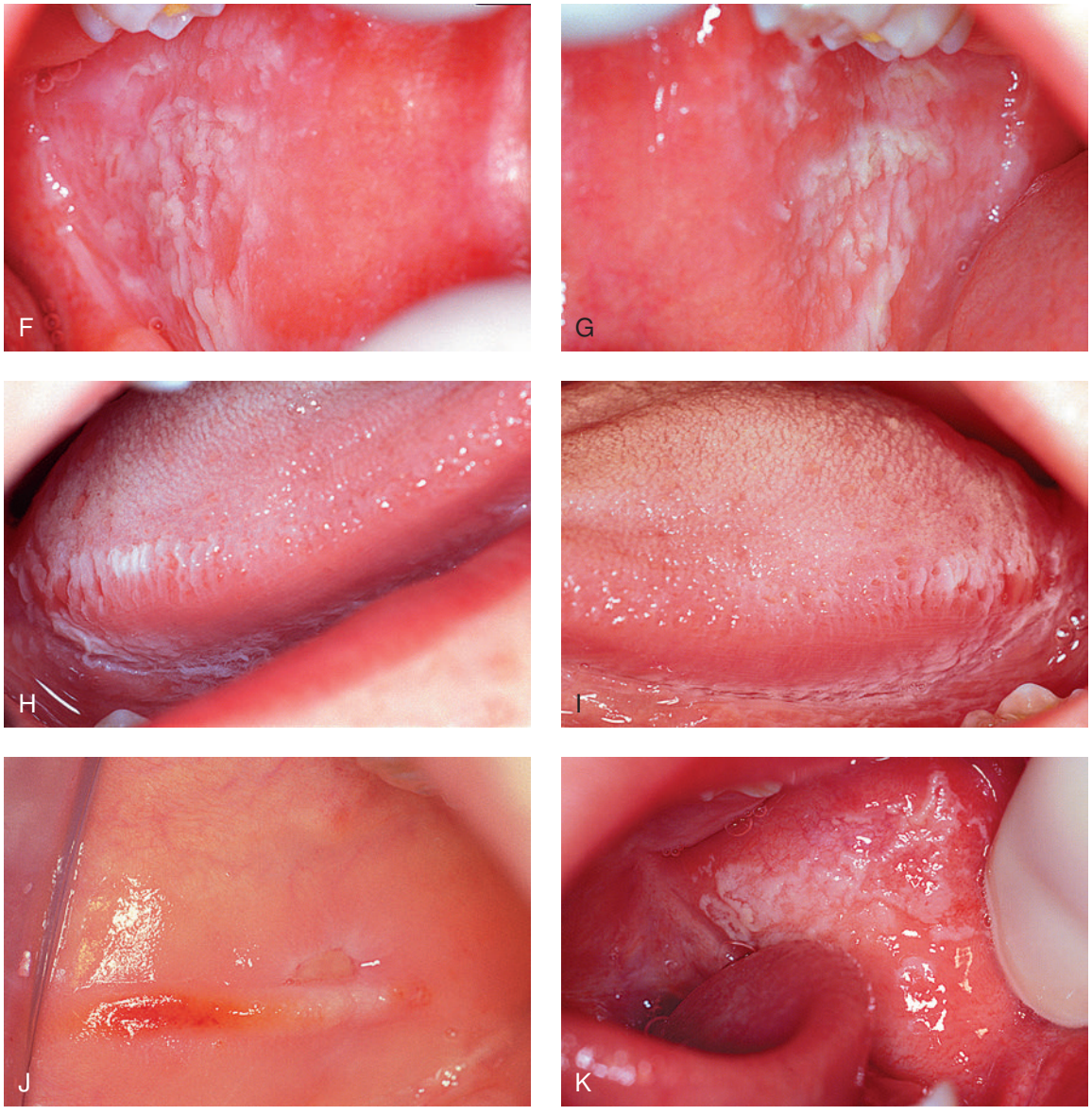
Continued

TABLE 2.2 **White Soft Tissue Lesions (see Fig. 2.2)—cont'd**

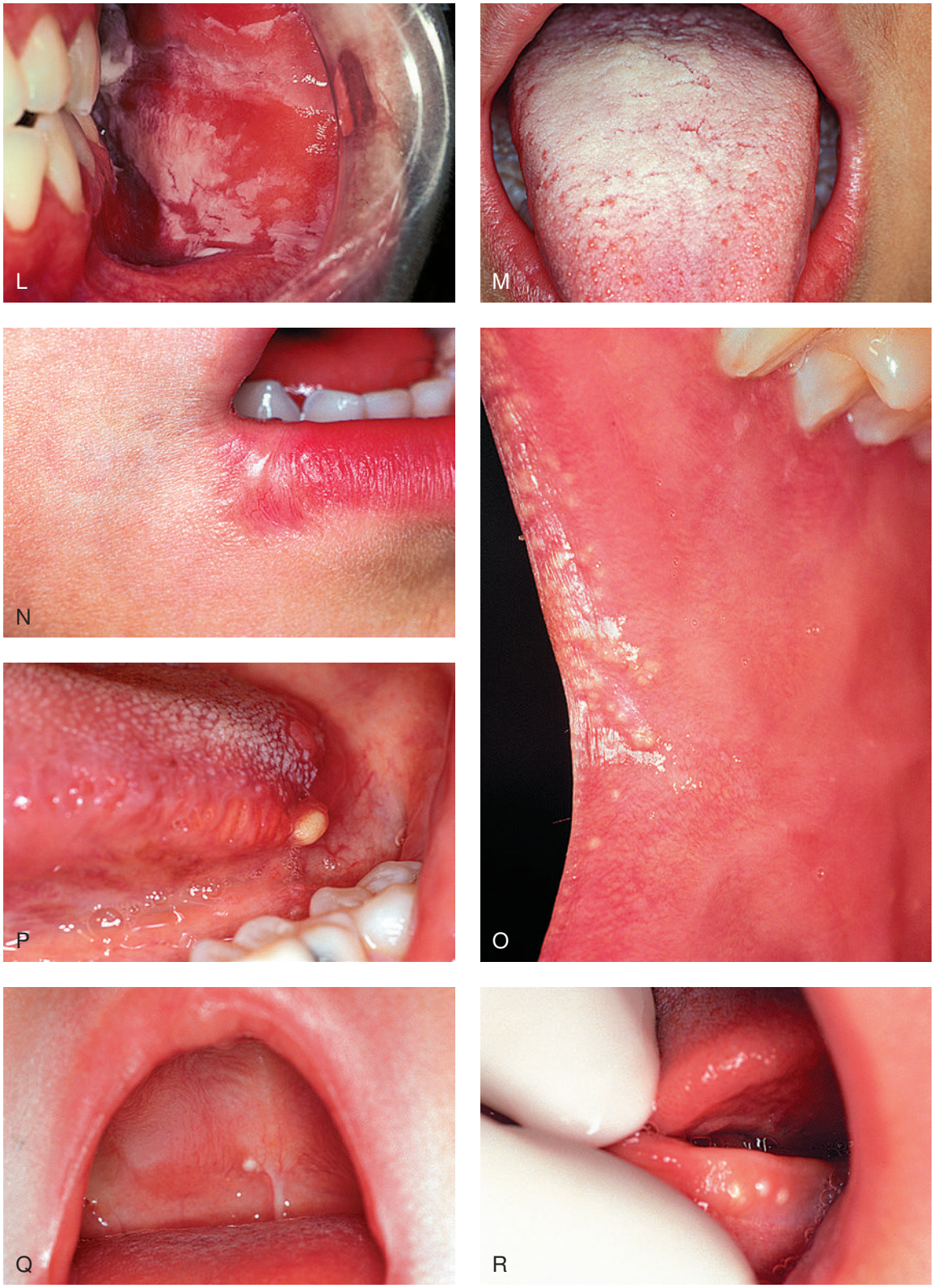
Lesion	Pediatric Age and Gender	Clinical Findings	Location	Pediatric Significance	Treatment and Prognosis	Differential Diagnosis
Oral lymphoepithelial cyst	Second decade No gender predilection	Solitary, soft, pinkish white nodule with superficial fine vascular pattern; usually nontender	Posterior lateral tongue, floor of mouth, soft palate	Mimics an abscess because it may fluctuate in size and discharge contents	Excisional biopsy; does not recur	Soft tissue abscess Lipoma Sialolith Tonsillolith Hyperplastic lymphoid aggregate
Sialolithiasis	Second decade No gender predilection	Solitary or multiple, hard, yellowish white globular mass; episodic pain and swelling when eating; obstructive disease of the duct	Usually floor of mouth within Wharton duct, submandibular gland	Occlusal or panoramic radiograph may assist with diagnosis; circular calcified mass	Massage of gland, surgical removal of stone and sometimes the gland; lithotripsy; stone may recur	Soft tissue abscess Oral lymphoepithelial cyst Epidermoid cyst Calcified lymph nodes
Palatal cysts of the newborn	Neonates No gender predilection	Solitary or multiple, discrete or clustered papules with a smooth pearly white surface; usually 1–3 mm in size, asymptomatic	*Epstein pearls:* median palatal raphe *Bohn nodules:* lateral hard and soft palate and junction	Cysts occur in up to 85% of neonates	None required; keratin-filled cysts that spontaneously rupture within first month	Soft tissue abscess Oral lymphoepithelial cyst
Gingival cysts of the newborn (dental lamina cysts)	Neonates No gender predilection	Solitary or multiple, discrete or clustered papules with a smooth translucent to pearly white surface; usually 1–3 mm in size, asymptomatic	Alveolar mucosa, especially maxillary mucosa	Cysts occur in up to 50% of neonates	None required; spontaneously rupture within first 3 months	Natal/neonatal teeth Soft tissue abscess Neonatal alveolar lymphangioma



• **Figure 2.2** White soft tissue lesions. (A and B) Frictional keratosis of the lateral tongue (A) and buccal mucosa (B) from chronic biting of the tissues. (C) Smokeless tobacco keratosis of the posterior mandibular vestibule. (D and E) Leukoedema of the buccal mucosa, bilaterally. *Continued*



• **Figure 2.2, cont'd** (F–I) White sponge nevus of the buccal mucosa (F and G) and lateral tongue (H and I). (J) Ulcerated linea alba from aggressive sucking habit. (K) Pseudomembranous candidiasis of the buccal mucosa.

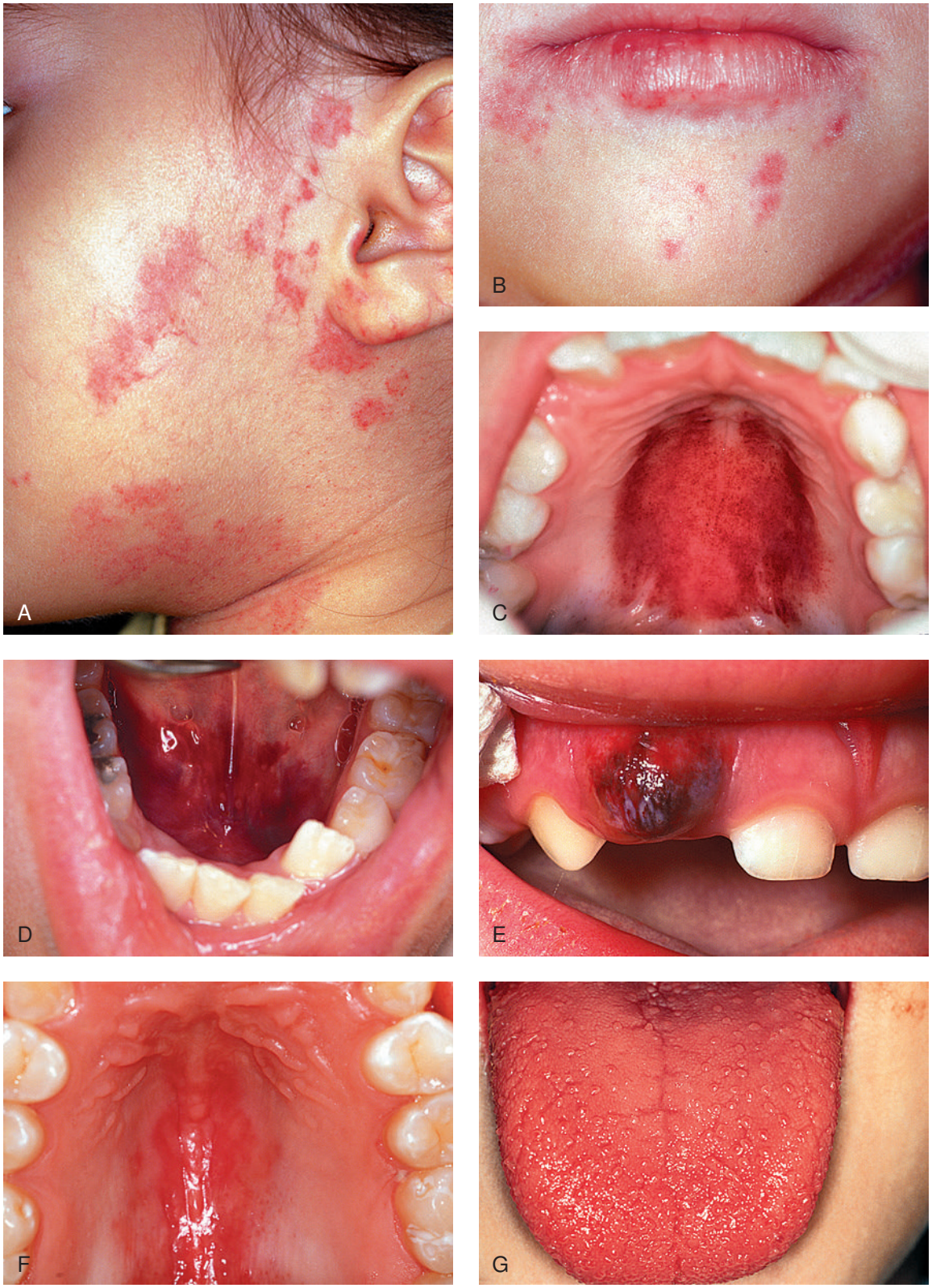


• **Figure 2.2, cont'd** (L) Chemical burn from overuse of a topical anesthetic. (M) Coated tongue in a child who is mouth breathing. (N) Fan-shaped scar at the corners of the mouth due to an electrical burn. (O) Cluster of Fordyce granules of the anterior buccal mucosa. (P) Oral lymphoepithelial cyst of the posterior lateral tongue. (Q) Single palatal cyst of the newborn on the midline hard palate. (R) Cluster of gingival cysts of the newborn on the mandibular alveolar mucosa.

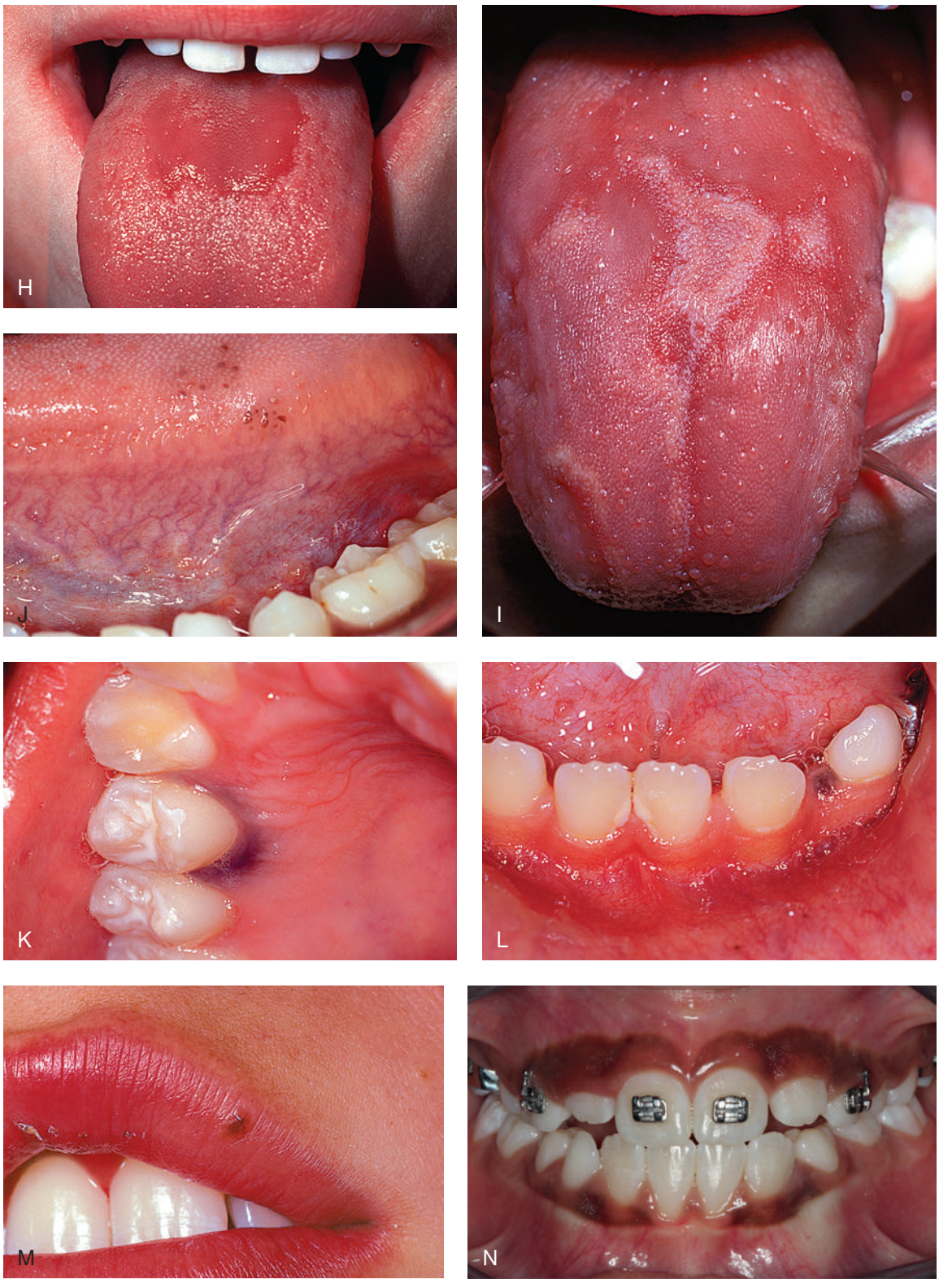
TABLE 2.3 **Dark Soft Tissue Lesions (see Fig. 2.3)**

Lesion	Pediatric Age and Gender	Clinical Findings	Location	Pediatric Significance	Treatment and Prognosis	Differential Diagnosis
Red or Purple-Blue Lesions						
Port-wine stain (capillary vascular malformation)	Infancy No gender predilection	Localized to diffuse, red to purple macular lesions; variable blanching; bleeds freely; gingival and bony enlargement; grows with child	Face, along distribution of trigeminal nerve, is most common site; may have lip and oral mucosal involvement	Occurs in approximately 1% of newborns; may be sign of *Sturge-Weber syndrome;* bleeding is complication; possible neurologic disease; gingival lesion mimics *pyogenic granuloma*	Laser treatment; persistent lesion that may become darker in color and nodular with age	Hemangioma Venous and arteriovenous malformation Ecchymosis Hereditary hemorrhagic telangiectasia
Submucosal hemorrhage, including petechiae, ecchymosis, and hematoma	First and second decades No gender predilection	Localized to diffuse, pinpoint spots, patches or swellings with smooth surface; early lesions are red; late lesions are blue-black; may be tender	Buccal mucosa, lips, lateral tongue, and soft palate; may develop concurrently on skin	If multiple lesions, need to exclude child abuse, factitial injury, infectious diseases, such as infectious mononucleosis, and blood disorders including leukemia, thrombocytopenia, anemia, and hemophilia	Identify the cause; no treatment for lesions; spontaneously resolve	Amalgam/graphite tattoo Blue nevus Hemangioma Vascular malformation Erythematous candidiasis Blood dyscrasia
Erythematous candidiasis	First and second decades No gender predilection	Multiple red macules to diffuse red patches; depapillation of tongue; burning sensation; may have *angular cheilitis*	Palate, buccal mucosa, dorsal tongue	Caused by *Candida albicans* and other species; contributing factors are antibiotics, immunosuppression, xerostomia, pacifier, and palatal coverage appliances	Antifungal medication and proper oral hygiene; may recur if cause is not eliminated or managed	Contact allergy Traumatic erythema Erythema migrans Thermal burn Palatal petechiae Anemia Scarlet fever (strawberry tongue)
Median rhomboid glossitis	First and second decades No gender predilection	Localized red, depapillated patch; oval to rhomboid in shape with smooth or lobulated surface; asymptomatic	Midline posterior dorsal tongue	Caused by candidal infection; localized palatal erythema or "kissing lesion" may be present	Antifungal medication and proper oral hygiene	Erythema migrans Traumatic erosion Contact allergy Hemangioma Vascular malformation Lingual thyroid
Erythema migrans (benign migratory glossitis)	First and second decades Female predilection	Multiple oval or circular red patches with white scalloped borders; loss of filiform papillae; pattern changes; may burn	Dorsal and ventrolateral tongue; rarely at other mucosal sites	More common in children than adults; increased risk in atopic children; may occur with *fissured tongue* and *transient lingual papillitis*	None required; avoidance of hot, spicy, or acidic foods; topical coating agents or steroids in symptomatic cases	Median rhomboid glossitis Contact allergy Erythematous candidiasis Transient lingual papillitis Lichen planus

Eruption hematoma and cyst	First and second decades No gender predilection	Localized patch or swelling; amber, red, or blue in color; overlying an erupting tooth; usually nontender	Alveolar mucosa	Eruption cyst is soft tissue counterpart of *dentigerous cyst;* infrequently delays tooth eruption; minimal bleeding may occur at this site	No treatment is usually necessary; resolves with tooth eruption; uncover tooth if symptomatic	Hemangioma Neonatal alveolar lymphangioma Pyogenic granuloma Amalgam tattoo
Brown-Black Lesions						
Physiologic (racial) pigmentation	First and second decades No gender predilection	Gray, brown, or black patches with smooth surface; patchy to generalized distribution	Any location but attached gingiva is most common	Pigmentation increases with age of child; common in dark-complexioned skin types	None required; common variant of normal mucosa	Postinflammatory pigmentation Drug-induced pigmentation Smoker's melanosis Lead poisoning
Amalgam tattoo	Second decade No gender predilection	Gray-blue, black macule with smooth surface and well-defined to irregular margins; radiographs may show opaque fragments	Gingiva, alveolar mucosa, buccal mucosa	Graphite tattoo is found on palate from self-inflicted wound; intentional tattooing rarely observed on lower labial mucosa	None required unless melanocytic neoplasm cannot be excluded; permanent discoloration	Melanotic macule Graphite tattoo Melanocytic nevus Varix Late ecchymosis
Oral melanotic macule (focal melanosis)	First and second decades Female predilection	Brown, gray, or black oval macule with smooth surface, well-defined margins; single or multiple	Lower lip vermilion, buccal mucosa, gingiva	Most common oral pigmentation of fair-complexioned children; multiple lip macules in *Peutz-Jeghers syndrome*	None required unless a melanocytic neoplasm cannot be excluded; no malignant transformation	Amalgam tattoo Graphite tattoo Melanocytic nevus Smoker's melanosis Late ecchymosis Drug-induced melanosis
Melanocytic nevus	Second decade Female predilection	Brown, blue, or black well-defined nodule or macule with smooth surface	Lip vermilion, palate, gingiva	Oral lesions are uncommon but head and neck skin is frequently involved	Excisional biopsy; low risk of malignant transformation on skin but uncertain risk on oral mucosa	Amalgam tattoo Graphite tattoo Oral melanotic macule Melanoacanthoma Melanoma



• **Figure 2.3** Dark soft tissue lesions. (A and B) Vascular malformation on the side of the face (A) and around the lips (B). (C) Ecchymosis of the hard palatal mucosa from sucking aggressively on a lollipop. (D) Hematoma of the floor of the mouth following trauma to the chin, which is frequently associated with fracture of the condyles. (E) Eruption hematoma of the maxillary alveolar mucosa. (F and G) Erythematous candidiasis of the hard palatal mucosa (F) and dorsal tongue (G).



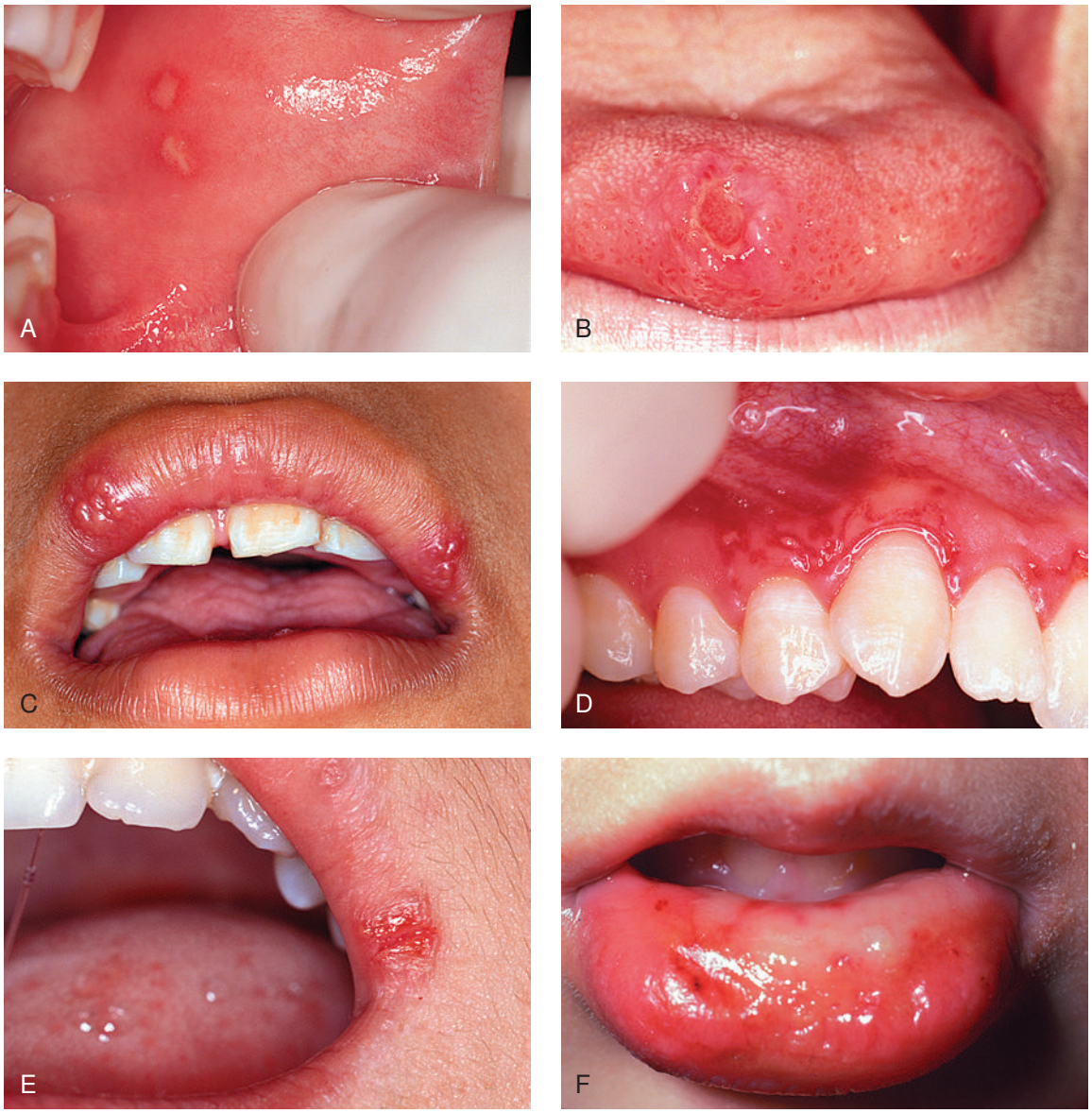
• **Figure 2.3, cont'd** (H) Median rhomboid glossitis of the dorsal tongue. (I) Erythema migrans of the dorsal tongue. (J) Postinflammatory pigmentation of fungiform papillae on the lateral tongue (papillary tip melanosis). (K) Amalgam tattoo of the maxillary palatal gingiva adjacent to the first premolar. (L) Oral melanotic macule of the mandibular gingiva in a child with a history of oral melanoma. (M) Compound nevus on the vermilion of the maxillary lip. (N) Physiologic pigmentation of the attached gingiva.

TABLE 2.4 Ulcerative Lesions (see Fig. 2.4)

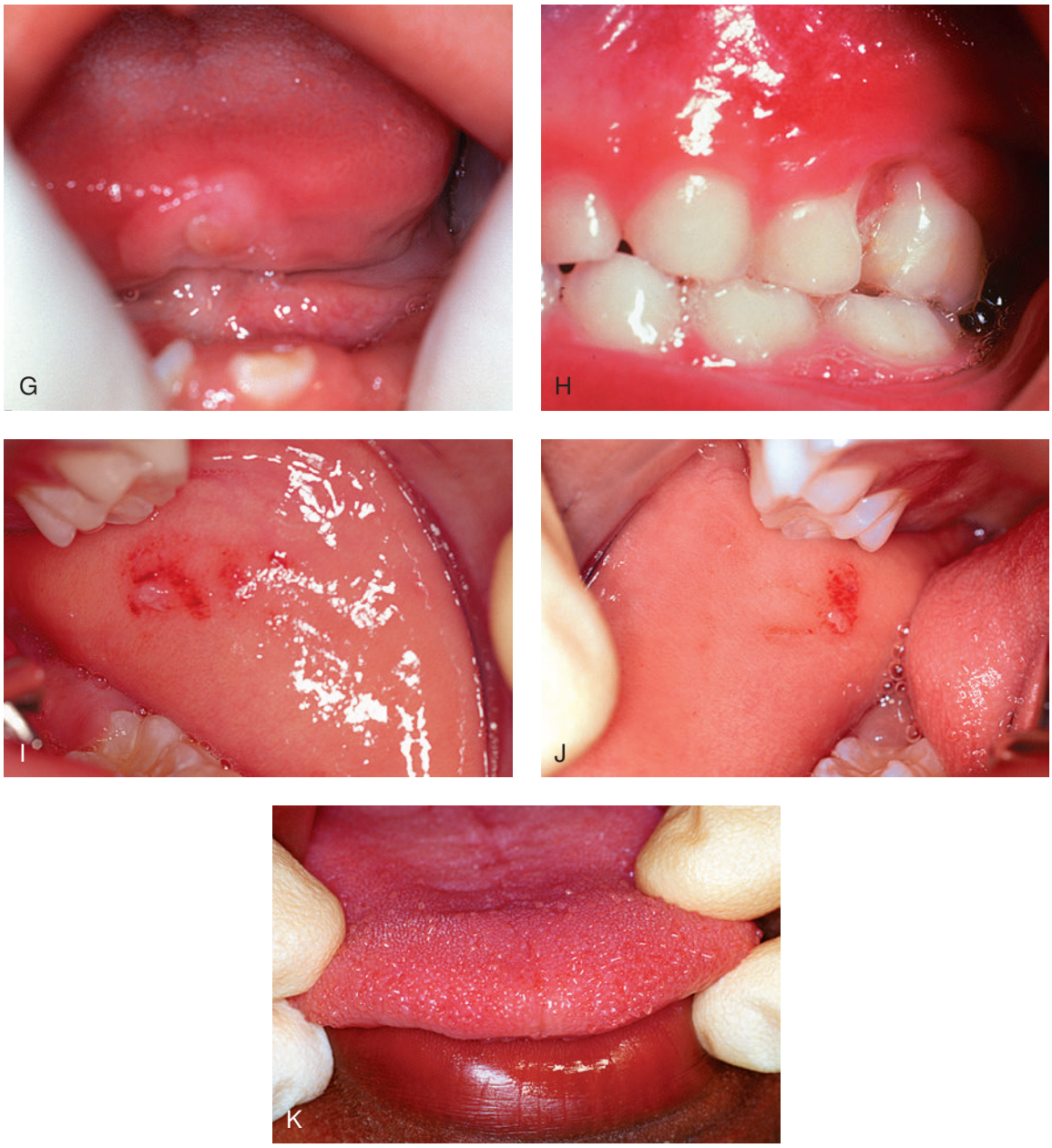
Lesion	Pediatric Age and Gender	Clinical Findings	Location	Pediatric Significance	Treatment and Prognosis	Differential Diagnosis
Aphthous ulcer	First and second decades Female predilection	Recurrent, painful ulcers *Minor variant:* 1–5 superficial oval ulcers <1 cm; resolves in 7–10 days *Major variant:* multiple, deep ulcers >1 cm; resolves in 2–6 weeks *Herpetiform variant:* showers of multiple small ulcers	Buccal, labial mucosa, and ventral tongue are most common; primarily occurs on nonkeratinized mucosa	Occurs in 20%–30% of children; T cell–medicated immune reaction; trauma and orthodontic appliances are important factors in children; genetic predisposition; associated with several systemic diseases, food sensitivities, nutritional deficiencies	Topical anesthetics and coating agents for symptomatic relief; topical and systemic steroids, chlorhexidine oral rinse, laser treatment, nutritional supplements Major variant heals with scarring	Traumatic ulcer Secondary herpetic ulcer Transient lingual papillitis Crohn disease Behçet syndrome Celiac disease Neutropenic ulcer PFAPA syndrome Gastroesophageal reflux disease
Secondary herpetic ulcer	First and second decades No gender predilection	Multiple, recurrent, small ulcers; painful; preceded by vesicles; clustered pattern; prodromal burning sensation; heals in 7–14 days	Herpes labialis on lip vermilion and perioral skin; intraoral herpetic ulcers on hard palate, attached gingiva; herpetic whitlow on fingers, especially with digit sucking habit	Reactivation of HSV; occurs in one-third of children; ultraviolet light, systemic diseases, trauma, stress, menses are triggering factors	Sunscreen for prevention; topical anesthetics for symptomatic relief; topical antiviral agents, systemic acyclovir, valacyclovir; immunocompromised children should be treated	Traumatic erosion Aphthous ulcer Angular cheilitis Impetigo Contact allergy Transient lingual papillitis Herpes zoster
Angular cheilitis	First and second decades No gender predilection	Red fissures that bleed and may ulcerate; scaling and crusted surface; burning sensation; may be recurrent	Commissures of mouth; may be associated with concurrent oral candidal infection	Caused by *Candida* species and staphylococci; lip incompetence, licking of lips and drooling are aggravating factors	Lubrication of lips, antifungal, antifungal/steroid ointments; recurring lesions may require oral antifungal treatment	Secondary herpetic ulcer Impetigo Exfoliative cheilitis Traumatic erosions Contact allergy Anemia

Traumatic ulcer	First and second decades No gender predilection	Usually single ulcer; variable shape with irregular margins; shallow or deep; painful; typically heals in 1–3 weeks	Lateral tongue, buccal mucosa, lips and gingival; *Riga-Fede disease* occurs in infants on ventral tongue from rubbing against lower incisors	Most common oral ulcer; may indicate child abuse, neurologic impairment, or factitial injuries when persistent and recurrent	Symptomatic relief; eliminate cause; factitial ulcers are diagnostic problem; may heal with scarring	Aphthous ulcer Mucosal burn Secondary herpetic ulcer Contact allergy
Contact allergy	First and second decades No gender predilection	Focal or widespread erythema, vesicles, and ulcers; swelling, burning sensation and pain; if chronic, then white plaques may develop	Any mucosal site that comes in contact with allergen, especially lips, buccal mucosa, and gingiva	Wide variety of allergens including foods, dental materials, oral hygiene products, topical medications, cosmetic products	Identify and eliminate allergen; patch testing helpful in older children; topical steroids to reduce symptoms; lesions recur with reexposure to allergen	Mucosal burn Secondary herpetic ulcer Aphthous ulcer Angular cheilitis Erythema multiforme
Erythema multiforme	Second decade Male predilection	Widespread, painful, red macules, vesicles, bullae, and ulcers; blood-crusted lesions on lips; target lesions on skin; acute onset; fever, malaise	Oral lesions on lips, tongue, buccal mucosa, and soft palate Skin lesions on extremities and head and neck region	Common precipitating factors include HSV and medications; minor and major forms of the disease	Withdrawal of medication; lubrication of lips, symptomatic relief; hospitalization if severe; recurrences are common if triggered by HSV	Primary herpetic gingivostomatitis Necrotizing ulcerative gingivitis Hand, foot, and mouth disease Chemical burn
Primary herpetic gingivostomatitis	Usually first decade No gender predilection	Fever, irritability, pain, lymphadenopathy, drooling, multiple vesicles, and ulcers; diffuse erythema; sudden onset; resolves in 7–10 days	Widespread oral and perioral involvement; gingival lesions are usually chief complaint; pharyngeal involvement in adolescents	Caused by HSV; high fever and dehydration are serious complications in children; digital and ocular lesions may occur	Supportive care includes antipyretics, analgesics, palliative oral rinses, hydration; systemic acyclovir may be indicated	Necrotizing ulcerative gingivitis Erythema multiforme Herpangina Hand, foot, and mouth disease

HSV, Herpes simplex virus; *PFAPA*, periodic fever, aphthous-stomatitis, pharyngitis, adenitis syndrome.



• **Figure 2.4** Ulcerative lesions. (A) Aphthous minor ulcer of the posterior buccal mucosa. (B) Aphthous major ulcer of the anterior dorsal tongue. (C) Herpes labialis of the vermilion of the maxillary lip. (D) Secondary herpetic ulcers of the maxillary attached gingiva. (E) Angular cheilitis. (F) Diffuse traumatic ulcer from biting the lip following local anesthesia for restorative treatment.



• **Figure 2.4, cont'd** (G) Riga-Fede disease of the ventral tongue in a child with neonatal teeth. (H) Erythema and recession of the attached gingiva between the primary first and second maxillary molars from picking the tissues with the fingernails. (I and J) Bilateral erosions of the buccal mucosa (I and J) and tenderness of the fungiform papillae at the tip of the tongue (K) from a toothpaste hypersensitivity.

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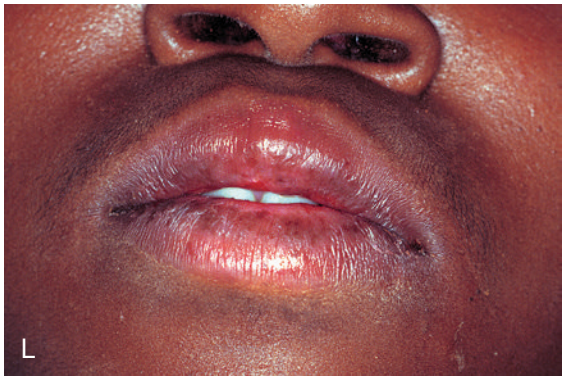
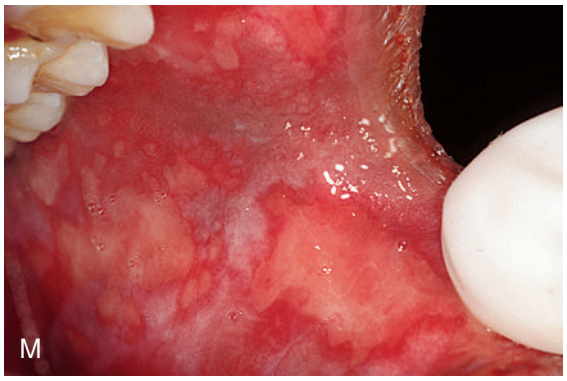
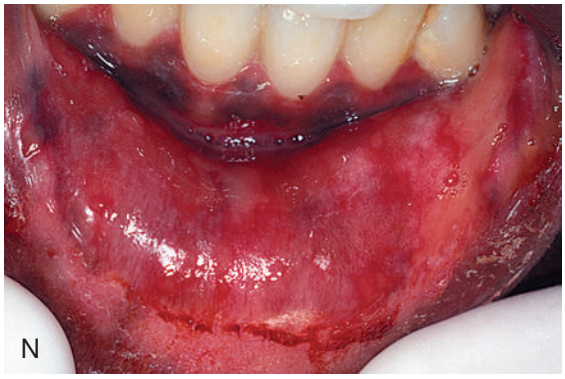
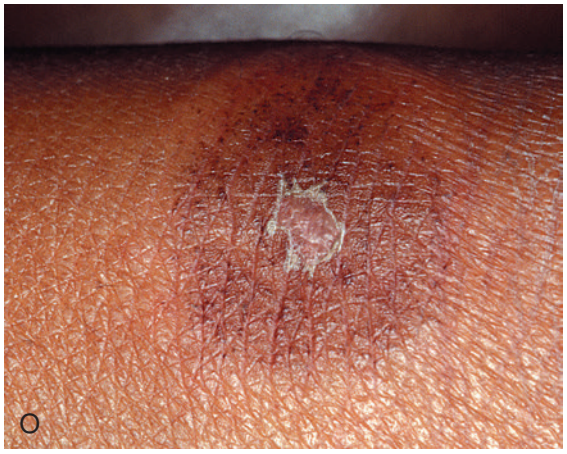
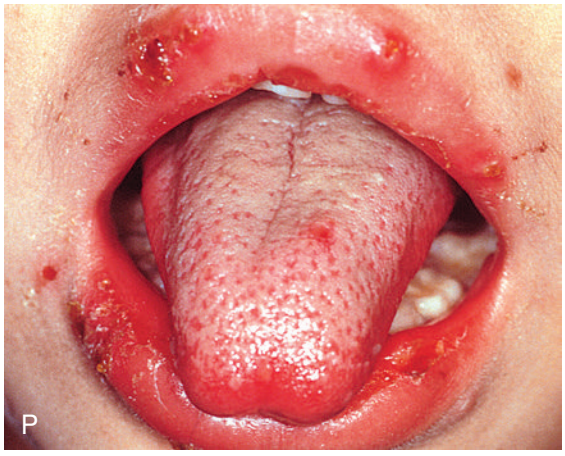
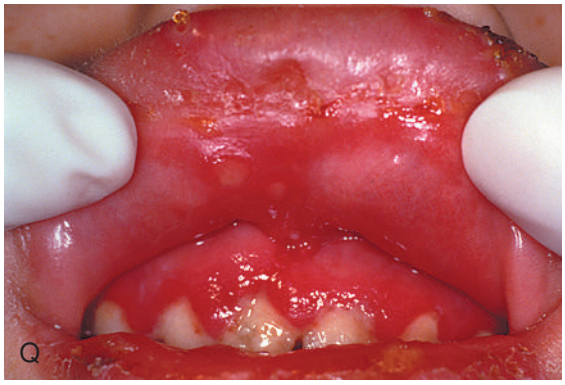


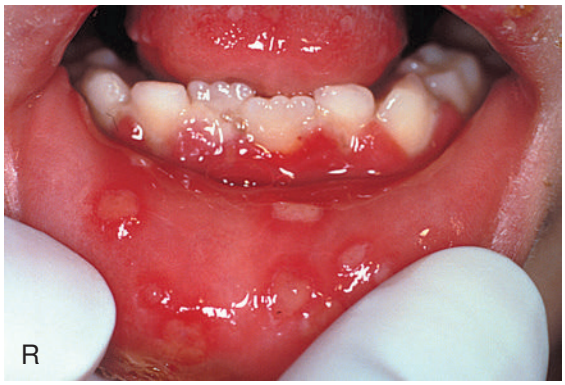


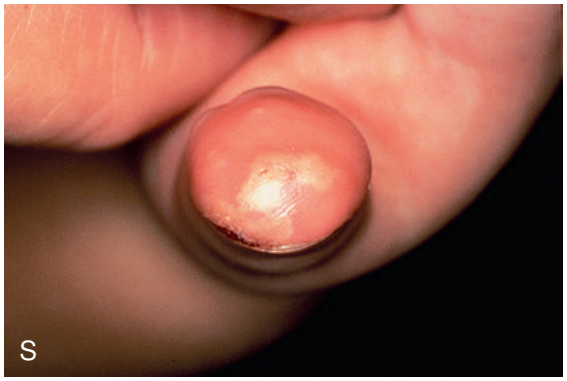




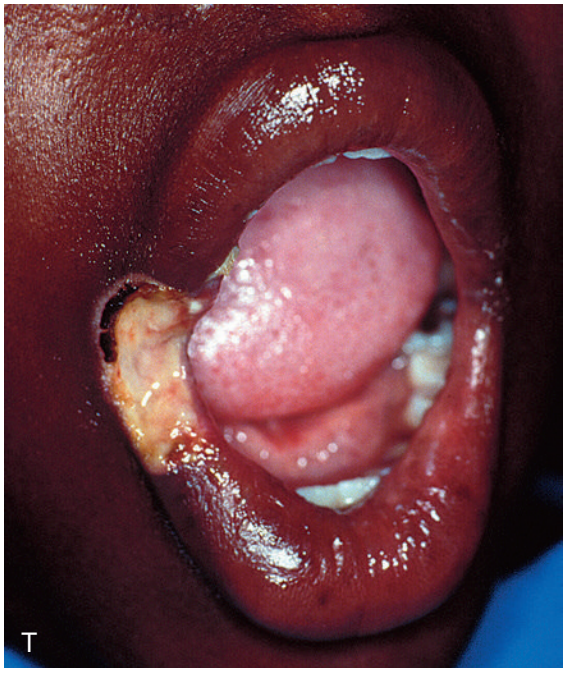


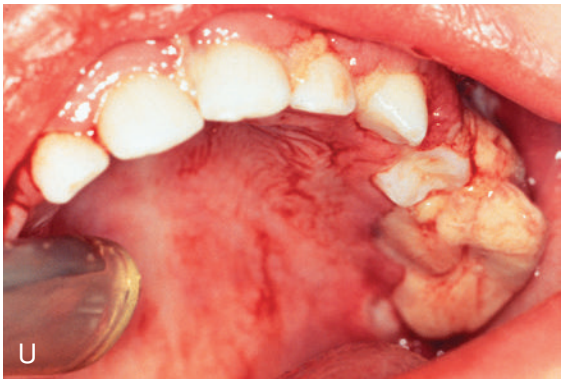


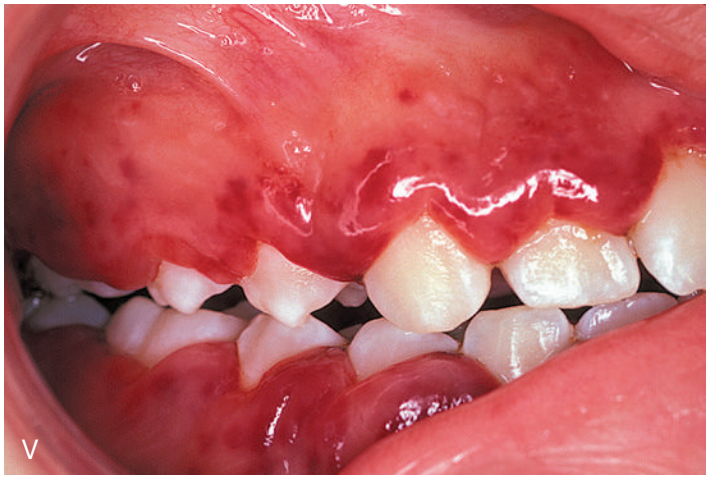


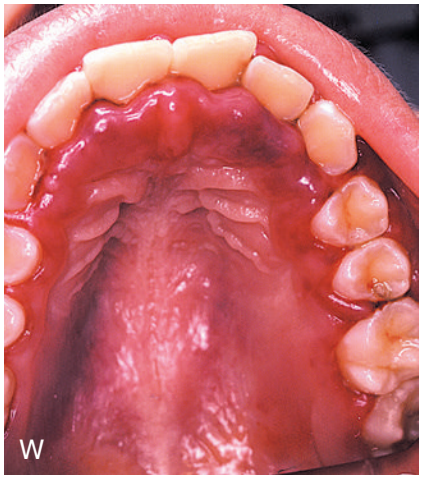


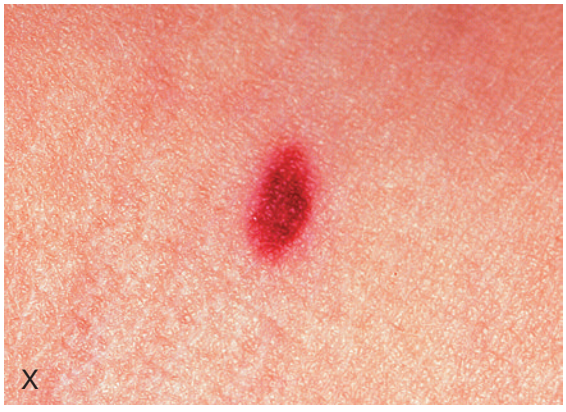
• **Figure 2.4, cont'd** (L–O) Drug-induced erythema multiforme with swelling of the lips (L), erythema and ulcerations of the buccal mucosa (M) and labial mucosa (N), and target lesions on the skin (O). (P–S) Primary herpetic gingivostomatitis of the tongue and lips (P), maxillary gingiva and labial mucosa (Q) and mandibular gingiva and labial mucosa (R), and vesicles on the thumb (S).











• **Figure 2.4, cont'd** (T) Electrical burn of the lip. (U) Necrotizing gingival ulcer with a thick pseudomembrane in a child with acute lymphoblastic leukemia. (V–X) Red-purple enlargements of the buccal gingiva (V), palatal mucosa (W), and skin (X) that represent leukemic infiltrates in a child with acute myeloid leukemia.

TABLE 2.5 Soft Tissue Enlargements (see Fig. 2.5)

Lesion	Pediatric Age and Gender	Clinical Findings	Location	Pediatric Significance	Treatment and Prognosis	Differential Diagnosis
Papillary Lesions						
Squamous papilloma	Second decade No gender predilection	Single, pedunculated nodule with fingerlike projections; pink to white; soft and nontender	Any oral site but predilection for the tongue, lips, and soft palate	Caused by HPV, especially types 6, 11; low virulence and infectivity rate	Excisional biopsy; recurrence is rare; no evidence of malignant transformation	Verruca vulgaris Condyloma acuminatum Giant cell fibroma Localized juvenile spongiotic gingival hyperplasia
Verruca vulgaris (common wart)	First and second decades No gender predilection	Multiple sessile or pedunculated papules and nodules with rough, pebbly or papillary surface; white; nontender	Common on skin of hands and face; infrequently found on the lip vermilion, labial mucosa, and anterior tongue	Caused by HPV, especially type 2; spread by autoinoculation to oral site by sucking on fingers or nail biting	Excisional biopsy of oral warts; low risk for recurrence for oral lesions; spontaneous resolution may occur for skin lesion; no risk for malignant transformation	Squamous papilloma Verruca plana Condyloma acuminatum Giant cell fibroma Molluscum contagiosum Frictional keratosis
Condyloma acuminatum (venereal wart)	Second decade No gender predilection	Multiple, discrete, sessile nodules with blunted papillary surface; pink; nontender	Usually anogenital lesions; oral sites include labial mucosa, soft palate, and ventral tongue	Oral lesions caused by HPV 6, 11, 16, 18; oncogenic HPV types 16, 18; autoinoculation, vertical or sexual transmission; may indicate child abuse	Excisional biopsy, laser ablation of oral warts; highly contagious; frequently recur; oral HPV 16, 18 associated with oropharyngeal carcinoma; HPV vaccine available	Squamous papilloma Multifocal epithelial hyperplasia Inflammatory papillary hyperplasia Giant cell fibroma Superficial lymphangioma Linear epidermal nevus Focal dermal hypoplasia
Giant cell fibroma	Second decade Female predilection	Solitary, sessile, or pedunculated nodule with a pebbly surface; pink; nontender	Attached gingiva, dorsal tongue, and hard palate	Fibrous lesion of unknown cause that has a predilection for children	Excisional biopsy; recurrence is rare	Squamous papilloma Retrocuspid papilla Irritation fibroma
Multifocal epithelial hyperplasia (Heck disease)	First and second decades No gender predilection	Multifocal, sessile, papules and nodules with pink grainy to stippled surface; lesions coalesce, display cobblestone appearance; nontender	Usually located on labial and buccal mucosa and tongue	Caused by HPV 13, 32; familial tendency, genetic susceptibility Other risk factors include poor oral hygiene, crowded living conditions, nutritional deficiencies	Excisional biopsy; laser ablation; recurrence is common; spontaneous regression may occur; no malignant transformation potential	Verruca vulgaris Condyloma acuminata Multiple hamartoma syndrome Multiple endocrine neoplasia syndrome 2B

Localized juvenile spongiotic gingival hyperplasia	Second decade Female predilection	Isolated red, velvety to papillary patch or enlargement; bleeds freely; persistent; does not respond to oral hygiene measures	Anterior facial gingiva, usually maxillary gingiva; represents transplanted crevicular or junctional epithelium	Contributing factors appear to be mouth breathing, anterior crowding, orthodontic appliances; used to be diagnosed as puberty gingivitis	Excisional biopsy; may spontaneously resolve; recurs up to 16%	Pyogenic granuloma Inflamed squamous papilloma Giant cell fibroma Superficial gingival lymphangioma
Inflammatory papillary hyperplasia	Second decade No gender predilection	Multiple, clustered papules and nodules with pink to red granular surface; cobblestone appearance; nontender	Hard palatal mucosa	Caused by continuous wear of palatal coverage appliance; other factors include mouth breathing and high palatal vault; candidal infection may be present	Remove and clean appliance; reline appliance if needed; antifungal therapy; excisional biopsy of persistent lesions	Condyloma acuminata Multifocal epithelial hyperplasia Erythematous candidiasis Early nicotine stomatitis
Acute Inflammatory Lesions						
Soft tissue abscess (parulis)	First and second decades No gender predilection	Solitary pinkish white or deep red nodule; purulence; fluctuates in size; tender to painful; may progress to cellulitis	Gingiva and alveolar mucosa are most common sites	Usually caused by odontogenic infection or entrapped foreign body; *pericoronitis* is a gingival abscess associated with erupting molars	Manage source of infection; local debridement; usually antibiotics are not indicated; recurs if infection is not eliminated	Pyogenic granuloma Oral lymphoepithelial cyst Sialolithiasis Tonsilithiasis Gingival cysts of newborn
Cellulitis	First and second decades No gender predilection	Diffuse erythematous swelling of sudden onset; soft to board-like; warm and painful tissues; fever, headache, airway obstruction, and leukocytosis may be present	Upper or lower face and neck	Caused by odontogenic infection, facial or oral lacerations, insect bites, peritonsillar abscesses, jaw fractures, sialadenitis, sinusitis, and bacteremia	Manage source of infection; antibiotic therapy; incision and drainage in severe cases *Ludwig angina* and *cavernous sinus thrombosis* may be life threatening	Facial hematoma Plunging ranula Emphysema Obstructive sialadenitis Angioedema Acute sinusitis Acute lymphadenitis
Angioedema	First and second decades No gender predilection	Diffuse swelling of sudden onset; soft and nontender; may be associated with respiratory and gastrointestinal problems	Lips, tongue, soft palate and face, and other cutaneous sites	Acquired form is caused by allergic reaction to foods, plants, drugs, insect bites, cold, heat, latex, pressure, stress, and infections; most hereditary forms are caused by C1-INH deficiency	Allergic forms are treated by antihistamines, steroids, or epinephrine; other drugs are used for the hereditary forms; may be life threatening	Cellulitis Emphysema Traumatic edema Contact allergy Orofacial granulomatosis

Continued

TABLE 2.5 **Soft Tissue Enlargements (see Fig. 2.5)—cont'd**

Lesion	Pediatric Age and Gender	Clinical Findings	Location	Pediatric Significance	Treatment and Prognosis	Differential Diagnosis
Mucocele	First and second decades No gender predilection	Fluid-filled nodule with a smooth, translucent, red or blue surface; sudden onset; fluctuates in size; tender if traumatized; periodically drains	Lower labial mucosa, buccal mucosa, and anterior ventral tongue	Most common lip swelling in children; may be associated with trauma and orthodontic appliances; rare cases are congenital	Excisional biopsy with removal of underlying minor salivary glands; may recur with incomplete removal or repeated trauma	Lymphangioma Hemangioma Hematoma Soft fibroma Soft tissue abscess Salivary duct cyst
Ranula	First and second decades No gender predilection	Fluid-filled swelling with smooth, translucent to blue surface of recent onset; fluctuates in size; mildly tender; periodically drains; may elevate tongue	Floor of mouth, lateral to midline; plunging variant results in diffuse swelling of the submandibular region and neck	Usually associated with sublingual gland; rare cases are congenital and caused by aplasia of submandibular excretory duct	Excisional biopsy of sublingual gland or marsupialization; recurrences are common with marsupialization	Lymphangioma Hemangioma Mucoepidermoid carcinoma Obstructive sialadenitis Salivary duct cyst Dermoid cyst
Tumor and Tumorlike Lesions						
Irritation fibroma	First and second decades No gender predilection	Nodule with pink smooth surface; firm and nontender; limited growth potential	Buccal and labial mucosa, tongue, and attached gingiva	Common reactive hyperplastic lesion caused by chronic trauma and mimics a tumor	Conservative excisional biopsy; may recur if irritation continues	Fibrosing mucocele Giant cell fibroma Fibrosing pyogenic granuloma Benign submucosal neoplasm
Peripheral ossifying fibroma	Second decade Female predilection	Nodule with pink to red surface; frequently ulcerated; firm and nontender; may resorb alveolar bone; limited growth potential	Emanates from interdental papilla of attached gingiva; most common site is anterior region	Reactive hyperplastic lesion with mineralized product from cells of periosteum or periodontal ligament; may displace teeth	Excisional biopsy down to periosteum and remove local irritation; 16% recurrence rate	Irritation fibroma Peripheral giant cell granuloma Giant cell fibroma Pyogenic granuloma Peripheral odontogenic fibroma
Peripheral giant cell granuloma	Second decade Female predilection	Nodule with red or purple-blue surface; may be ulcerated; firm and nontender; resorb alveolar bone; limited growth potential	Attached gingiva or alveolar mucosa	Reactive hyperplastic lesion caused by irritation; may displace teeth	Excisional biopsy to periosteum and remove local irritation; 10%–18% recurrence rate	Pyogenic granuloma Ulcerated irritation fibroma Peripheral ossifying fibroma Hemangioma Foreign body granuloma

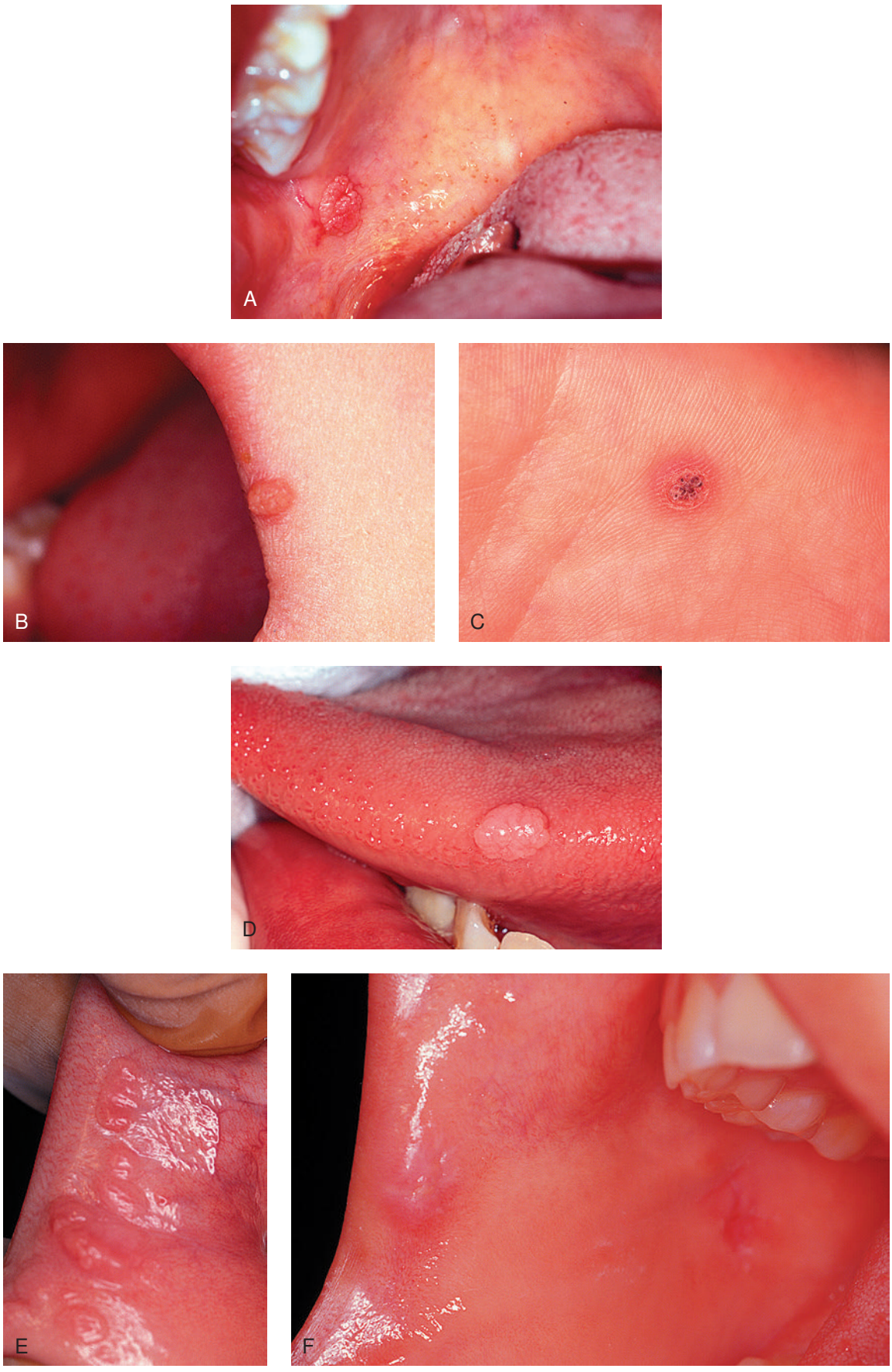
Pyogenic granuloma	First and second decades Female predilection	Nodule with smooth to irregular, red surface; usually ulcerated; bleeds freely; soft and friable; nontender; limited growth potential	Most occur on attached gingiva; other sites include lip, tongue, and buccal mucosa; also occurs on skin	Reactive hyperplastic lesion caused by irritation and poor oral hygiene; may be associated with pregnancy *(pregnancy tumor)* or may develop at extraction site because of bony sequestra *(epulis granulomatosa)*	Excisional biopsy and remove local irritation; recurrence rate is 3%–15%	Ulcerated irritation fibroma Peripheral ossifying fibroma Peripheral giant cell granuloma Soft tissue abscess Hemangioma Localized, juvenile spongiotic gingival hyperplasia
Gingival fibromatosis	First and second decades No gender predilection	Localized or generalized gingival enlargements; pink, smooth to stippled surfaces; firm and nontender; affects both dentitions	Attached gingiva and maxillary tuberosity	May be familial or idiopathic; associated with several syndromes; interferes with eruption of teeth; displacement of teeth	Gingivectomy and good oral hygiene; high recurrence rate	Drug-induced gingival overgrowth Mouth breathing gingivitis Chronic hyperplastic gingivitis Leukemic gingival infiltrates Scorbutic gingivitis
Hemangioma	Infancy Female predilection	Localized to diffuse, red, blue, or purple lesion, flat or nodular, soft and compressible; may blanch; bleeds freely; 20% are multiple	60% occur in head and neck region; lips, tongue, and buccal mucosa are most common sites; rarely occurs in jaws	Hemorrhage is potential complication; may cause malocclusion; scarring is common with involution	Involution of lesion within first decade; surgery for select cases and scar revision, laser ablation, corticosteroids, propranolol; does not recur	Vascular malformation Pyogenic granuloma Lymphangioma Eruption cyst/hematoma Mucocele
Lymphangioma (lymphatic malformation)	Infancy; most detected by 2 years No gender predilection	Localized to diffuse, translucent to red or purple swelling; smooth or pebbly surface; soft and compressible; crepitus may be palpated	Up to 75% occur in head and neck; common oral sites include the tongue, lip, and buccal mucosa	May cause malocclusion, dysphagia, and respiratory problems; *cystic hygroma* and *neonatal alveolar lymphangioma* are variants	Surgical excision; recurrences are common; airway obstruction and death may occur with large neck or tongue lesions	Hemangioma Squamous papilloma Lingual papillitis Mucocele Plunging ranula Parotitis Branchial cleft cyst
Congenital epulis	Infancy Female predilection	Pedunculated or sessile nodule; pink to red smooth surface; may be ulcerated; 10% are multiple	Anterior alveolar ridge; usually maxilla	May cause feeding problems; usually reaches maximum size at birth	Surgical excision; occasional spontaneous regression; no recurrence; normal tooth development	Hemangioma Pyogenic granuloma Neonatal alveolar lymphangioma Neuroectodermal tumor of infancy

Continued

TABLE 2.5 Soft Tissue Enlargements (see Fig. 2.5)—cont'd

Lesion	Pediatric Age and Gender	Clinical Findings	Location	Pediatric Significance	Treatment and Prognosis	Differential Diagnosis
Neurofibroma	Second decade No gender predilection	Single or multiple nodules with smooth surface; discrete or diffuse; soft to firm on palpation; nontender	Tongue, buccal mucosa, and palate; rarely within mandible; syndromic lesions occur at any site, especially skin	*Neurofibromatosis* is autosomal dominant condition with neurofibromas, café-au-lait macules, axillary freckling, and Lisch nodules on iris	Surgical excision if solitary lesion; selective excision of syndrome type; 5% malignant transformation of syndrome type	Schwannoma Mucosal neuroma Irritation fibroma Benign submucosal neoplasm Salivary gland neoplasm
Mucosal neuromas (multiple endocrine neoplasia syndrome, type 2B)	First decade No gender predilection	Multiple, pink papules and nodules; soft and nontender; marfanoid body type; narrow face with full lips	Labial and buccal mucosa, anterior tongue, gingiva; also on conjunctiva and eyelid	Autosomal dominant syndrome; other stigmata include pheochromocytoma and medullary carcinoma of thyroid gland	Surgical excision of neuromas for cosmetics; aggressive thyroid cancer develops in second decade	Neurofibromatosis Multifocal epithelial hyperplasia Multiple hamartoma syndrome
Pleomorphic adenoma (benign mixed tumor)	Second decade Slight female predilection	Pink, dome-shaped enlargement with smooth surface; slowly growing; firm and nontender	Parotid gland is most common site; palate is most common oral site	Most common benign salivary gland neoplasm; *mucoepidermoid carcinoma* is most common malignant salivary gland tumor in this age group	Surgical excision with adequate margins; recurrence is low; malignant transformation rate of <4%	Neurofibroma Schwannoma Mucoepidermoid carcinoma Irritation fibroma
Juvenile aggressive fibromatosis	First and second decades No gender predilection	Rapidly growing, pink, firm mass with an irregular surface; may be ulcerated; painless; large in size; facial disfigurement; destruction of adjacent bone	Head and neck region; paramandibular soft tissues are common intraoral sites	Rare, locally aggressive and destructive lesion that mimics a malignancy; associated with *familial adenomatous polyposis, Gardner syndrome*	Surgical excision with wide margins; adjunctive chemotherapy and radiotherapy may be indicated; high recurrence rate	Large pyogenic granuloma Fibrosarcoma Rhabdomyosarcoma Metastatic disease
Rhabdomyosarcoma	First and second decades Male predilection	Rapidly growing, infiltrative and destructive mass; painless	Head and neck region is the most common site; face, orbit, nasal cavity, maxillary sinus, palate	Skeletal muscle malignancy; one of the most common sarcomas in children	Surgical excision, multiagent chemotherapy with or without radiation therapy Pediatric prognosis is 70% 5-year survival rate	Desmoidlike fibromatosis Lymphoma Neuroblastoma Malignant salivary gland tumor Retinoblastoma Juvenile nasopharyngeal angiofibroma

C1-INH, C1-esterase inhibitor; *HPV,* human papillomavirus.



• **Figure 2.5** Soft tissue enlargements. (A) Squamous papilloma of the soft palate. (B and C) Verruca vulgaris of the lip vermilion (B) and hand (C). (D) Giant cell fibroma of the lateral tongue. (E and F) Focal epithelial hyperplasia of the buccal mucosa displaying a cobblestone appearance (E) or a small clustered pattern (F).

Continued