Dentistry for the Child and Adolescent

RALPH E. McDONALD, DDS, MS
Dean Emeritus and Professor Emeritus
of Pediatric Dentistry
Indiana University School of Dentistry
Indianapolis, Indiana

DAVID R. AVERY, DDS, MSD
Ralph E. McDonald Professor and Director
of Pediatric Dentistry
Indiana University School of Dentistry
James Whitcomb Riley Hospital for Children
Indianapolis, Indiana

JEFFREY A. DEAN, DDS, MSD
Associate Professor of Pediatric Dentistry and Orthodontics
Director, Advanced Education Program in Pediatric Dentistry
Acting Chair, Department of Oral Facial Development
Indiana University School of Dentistry
James Whitcomb Riley Hospital for Children
Indianapolis, Indiana

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Contributors

CHRISTOPHER EDWARD BELCHER, MD
Director
Pediatric Infectious Diseases
Infectious Diseases of Indiana
Indianapolis, Indiana

DAVID BIXLER, DDS, PhD
Professor Emeritus of Oral Facial Genetics
and Medical and Molecular Genetics
Indiana University
Indianapolis, Indiana

DAVID T. BROWN, DDS, MS
Professor, Department of Restorative Dentistry
Indiana University School of Dentistry
Indianapolis, Indiana

DAVID A. BUSSARD, DDS, MS
Assistant Clinical Professor of Oral and Maxillofacial Surgery
Indiana University School of Dentistry
Private Practice of Oral and Maxillofacial Surgery
Indianapolis, Indiana

ROBERT L. CREEDON, DDS
Professor Emeritus
Department of Pediatrics/Dentistry
College of Medicine, University of Cincinnati
Director, Division of Pediatric Dentistry (retired)
Department of Pediatrics
Cincinnati Children's Hospital Medical Center
Cincinnati, Ohio

ROBERT J. CRONIN, Jr., DDS, MS
Professor and Director, Graduate Division
Department of Prosthodontics
The University of Texas Health Science Center at San Antonio Dental School
San Antonio, Texas

MURRAY DOCK, DDS, MSD, RPh
Associate Professor
Department of Pediatrics
College of Medicine, University of Cincinnati
Director, Residency Program in Pediatric Dentistry
Division of Pediatric Dentistry
Cincinnati Children's Hospital Medical Center
Cincinnati, Ohio

ROBERT J. FEIGAL, DDS, PhD
Professor and Chair, Department of Preventive Sciences
School of Dentistry, University of Minnesota
Minneapolis, Minnesota

DONALD J. FERGUSON, DMD, MSD
Professor and Chair, Department of Orthodontics
Director, Advanced Orthodontic Training Program
Henry M. Goldman School of Dental Medicine
Boston University
Boston, Massachusetts

CHARLES J. GOODACRE, DDS, MSD
Dean and Professor, Department of Restorative Dentistry, School of Dentistry
Loma Linda University
Loma Linda, California

ANN PAGE GRIFFIN, BA Clinical
Associate Professor Department
of Family Medicine
East Carolina University
Greenville, North Carolina
Faculty Associate, Department of Pediatric Dentistry
College of Dentistry, University of Tennessee
Memphis, Tennessee

LEE M. HARRISON, Jr., DDS, MS
Clinical Associate Professor
Department of Pediatric Dentistry
School of Dentistry, Louisiana State University
New Orleans, Louisiana
Private Practice of Pediatric Dentistry
Shreveport, Louisiana

JAMES K. HARTSFIELD, Jr., DMD, MS, MMedSci, PhD
Professor and Director of Oral Facial Genetics Professor of Orthodontics
Department of Oral Facial Development
School of Dentistry, Indiana University
Professor, Department of Medical and Molecular Genetics
School of Medicine, Indiana University
President, Meridian Orthodontics, PC
Indianapolis, Indiana

ROBERTA A. HIBBARD, MD
Professor, Department of Pediatrics
School of Medicine, Indiana University
Indianapolis, Indiana

RANDY A. HOCK, MD, PhD
Medical Director
Department of Pediatric Hematology/Oncology and Pediatric Hospice
St. Vincent Children's Hospital
Indianapolis, Indiana

DONALD V. HUEBENER, DDS, MS, MAEd
Professor, Plastic and Reconstructive Surgery
Department of Surgery, School of Medicine
Washington University, St. Louis, Missouri
Professor, Pediatric Dentistry
School of Dental Medicine
Southern Illinois University
Alton, Illinois

CHRISTOPHER V. HUGHES, DMD, PhD
Associate Professor and Chair
Department of Pediatric Dentistry
Henry M. Goldman School of Dental Medicine
Boston University
Boston, Massachusetts

JAMES E. JONES, DMD, MSD, PhD, EdD
Dean and Professor, School of Health Sciences
Indiana University Purdue University
Fort Wayne
Fort Wayne, Indiana
Professor of Pediatric Dentistry
Indiana University School of Dentistry
Indianapolis, Indiana

GEORGE E. KRULL, DDS
Private Practice of Pediatric Dentistry
Clarkston, Michigan

JOHN T. KRULL, DDS
Assistant Professor of Pediatric Dentistry
Indiana University School of Dentistry
Private Practice of Orthodontics
Indianapolis, Indiana

THOMAS H. LAPP, DDS, MS
Clinical Assistant Professor of Oral and Maxillofacial Surgery
Indiana University School of Dentistry
Private Practice of Oral and Maxillofacial Surgery
Indianapolis, Indiana

JASPER L. LEWIS, Jr., DDS, MS
Clinical Assistant Professor, Department of Pediatric Dentistry, College of Dentistry
University of Tennessee
Memphis, Tennessee
Clinical Professor, Department of Surgery
Chief of the Division of Dentistry
Clinical Assistant Professor
Department of Family Medicine
School of Medicine, East Carolina University
Private Practice of Pediatric Dentistry
Greenville, North Carolina

JAMES L. McDONALD, Jr., PhD
Professor of Oral Biology
Associate Dean for Dental Education
Indiana University School of Dentistry
Indianapolis, Indiana

JOHN S. McDONALD, DDS, MS, FACD
Volunteer Professor
Departments of Surgery and Anesthesia
Volunteer Associate Professor
Department of Pediatrics
Division of Pediatric Dentistry
College of Medicine, University of Cincinnati
Cincinnati, Ohio
Private Practice of Oral and Maxillofacial Pathology/Head and Neck Pain
Cincinnati, Ohio

DALE A. MILES, DDS, MS, FRCD(C)
Professor of Oral and Maxillofacial Radiology
Associate Dean for Clinical Affairs and Faculty Development
Arizona School of Dentistry and Oral Health
Mesa, Arizona

B. KEITH MOORE, PhD
Professor and Director of Dental Materials Graduate Program
Indiana University School of Dentistry
Indianapolis, Indiana

EDWIN T. PARKS, DMD, MS
Professor of Oral Pathology,
Medicine and Radiology
Indiana University School of Dentistry
Indianapolis, Indiana

ALAN MICHAEL SADOVE, MD
James Harbaugh Endowed Professor of Plastic Surgery
Indiana University School of Medicine
Chief, Plastic Surgery
James Whitcomb Riley Hospital for Children
Indianapolis, Indiana

BRIAN J. SANDERS, DDS, MS
Associate Professor of Pediatric Dentistry
Indiana University School of Dentistry
Director, Clinic Pediatric Dentistry
James Whitcomb Riley Hospital for Children
Indianapolis, Indiana

AMY D. SHAPIRO, MD
Medical Director
Indiana Hemophilia and Thrombosis Center, Inc.
Indianapolis, Indiana

GEORGE K. STOOKEY, MSD, PhD
Distinguished Professor Emeritus of Preventive and Community Dentistry
Indiana University School of Dentistry
Indianapolis, Indiana

JAMES A. WEDDELL, DDS, MSD
Associate Professor of Pediatric Dentistry
Indiana University School of Dentistry James
Whitcomb Riley Hospital for Children
Indianapolis, Indiana

GERALD Z. WRIGHT, DDS, MSD, FRCDIC)
Professor Emeritus
The University of Western Ontario
London, Ontario, Canada
Secretary General,
International Association of Paediatric Dentistry

KAREN M. YODER, MSD, PhD
Associate Professor and Director of Community Dentistry
Indiana University School of Dentistry
Indianapolis, Indiana

Preface

The eighth edition of *Dentistry for the Child and Adolescent* presents current diagnostic and treatment recommendations based on research, clinical experience, and current literature. This newest edition follows the same basic structure and format of the previous seven editions. The contributors who joined us in preparation of this latest revision express a coordinated philosophy in the approach to the most modern concepts of dentistry for the child and adolescent. The information contained herein is relevant to the contemporary science and practice of pediatric dentistry. This textbook is designed to help undergraduate dental students and postdoctoral pediatric dental students provide efficient and superior comprehensive oral health care to infants, children, teenagers, and medically compromised individuals. It also provides experienced dentists with reference information regarding new developments and techniques.

Chapter 2, Child Abuse and Neglect, includes an up-to-date overview of this topic and management of documentation and reporting requirements. In addition, the chapter contains several new photographs to help illustrate this societal problem. Chapter 5, Radiographic Techniques, includes new information on the use of digital radiography in pediatric dentistry. Chapter 6, Clinical Genetics for the Dental Practitioner, has a new lead author and provides updated information in several topic areas, particularly in genetic influence on external apical root resorption associated with orthodontic treatment. Theories relating to the molecular biology of tooth eruption are provided in Chapter 9. Chapter 10, Dental Caries in the Child and Adolescent, has been reformatted and presents much new information to reflect contemporary knowledge and management of the disease as well as an increased emphasis on early childhood caries. Chapter 13, Local Anesthesia for the Child and Adolescent, has been expanded to include a section on anxiety control, pain, and the use of analgesics in pediatric dentistry. Chapter 14, Pharmacologic Management of Patient Behavior, has a new lead author and has been thoroughly reviewed and updated to provide the latest information regarding sedation techniques for children. Chapter 17, Pit and Fissure Sealants and Preventive Resin Restorations, has undergone a major revision; a new co-author has provided fresh illustrations for the chapter as well. Chapter 18, Restorative Dentistry, provides an increased emphasis on the latest restorative materials and in particular on the "alternative restorative technique." Treatment modalities for pulp therapy and trauma have been incorporated in Chapters 19 and 21 that utilize the new science and information associated with molecular biology, in an attempt to maintain the vitality of the tissues associated with these treatments whenever possible. Chapter 23, Dental Problems of Children with Disabilities, and Chapter 24, Management of the Medically Compromised Patient, have been updated and revised to include information regarding the dental management of patients with sickle cell anemia. The latest information on the incorporation of early orthodontic treatment has been added to Chapter 27, Management of the Developing Occlusion, and a new co-author has helped with Chapter 28, Multidisciplinary Team Approach to Cleft Lip and Palate Management. In particular, the protocols developed by the American Cleft Palate—Craniofacial Association have been incorporated. And finally, Chapter 29, Practice Management, has been revised significantly and Chapter 30, Community Oral Health, has been completely rewritten.

Ralph **E. McDonald**
David R. Avery
Jeffrey A. Dean

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The faculties of pediatric dentistry and other disciplines at Indiana University have contributed substantially to this work in many ways. We truly appreciate their willingness to share information relevant to scientific accuracy of the manuscripts. In particular, we gratefully acknowledge Drs. Michael Baumgartner, Howard Eigen, Margherita Fontana, Richard Gregory, Michelle Howenstine, Vanchit John, Gopal Krishna, Chris Miller, John Rau, Paul Walker, and Susan Zunt. Many pediatric dentistry postdoctoral students and auxiliary staff have also assisted in numerous ways. The encouragement and support of all members of our families sustained our resolve to complete this task when it seemed that it would not get done. We extend our heartfelt thanks to all who played a role in helping us bring this project to a successful conclusion.

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CHAPTER

1

Examination of the Mouth and Other Relevant Structures

RALPH E. McDONALD
DAVID R. AVERY
JEFFREY A. DEAN

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CHAPTER OUTLINE

INITIAL PARENTAL CONTACT WITH THE DENTAL OFFICE
THE DIAGNOSTIC METHOD
PRELIMINARY MEDICAL AND DENTAL HISTORY
CLINICAL EXAMINATION TEMPOROMANDIBULAR EVALUATION UNIFORM DENTAL RECORDING
RADIOGRAPHIC EXAMINATION
EARLY EXAMINATION
INFANT DENTAL CARE
DETECTION OF SUBSTANCE ABUSE
    Etiologic Factors in Substance Abuse Specific Substances and Frequency of Use
SUICIDAL TENDENCIES IN CHILDREN AND ADOLESCENTS
INFECTION CONTROL IN THE DENTAL OFFICE
    Biofilm
EMERGENCY DENTAL TREATMENT

dentist is traditionally taught to perform a complete oral examination of the patient and to develop a treatment plan from the examination findings. Then the dentist makes a case presentation to the patient or parents, outlining the recommended course of treatment. This process should include the development and presentation of a prevention plan that outlines an ongoing comprehensive oral health care program for the patient and establishment of the "dental home."

The plan should include recommendations designed to correct existing oral problems (or halt their progression) and to prevent anticipated future problems. It is essential to obtain all relevant patient and family information, to secure parental consent, and to perform a complete examination before embarking on this comprehensive oral health care program for the pediatric patient. *Anticipatory guidance is* the term often used to describe the discussion and implementation of such a plan with the patient and/or parents. The American Academy of Pediatric Dentistry has published guide-lines concerning the periodicity of examination, preventive dental services, and oral treatment for children as summarized in Fig. 1-1.

Each pediatric patient should be given an opportunity to receive complete dental care. The dentist should not attempt to decide what the child, parents, or third-party agent will accept or can afford. If parents reject a portion or all of the recommendations, the dentist has at least fulfilled the obligation of educating the child and the parents about the importance of the recommended procedures. Parents of even moderate income will usually find the means to have oral health care completed if the dentist explains to them that the child's future oral health and even general health are related to the correction of oral defects.

INITIAL PARENTAL CONTACT WITH THE DENTAL OFFICE

The parent usually makes the first contact with the dental office by telephone. This initial conversation between the parent and the office receptionist is very important. It provides the first opportunity to attend to the parent's concerns by pleasantly and concisely responding to questions and by offering an office appointment. The receptionist must have a warm, friendly voice and the ability to communicate clearly. The receptionist's responses should assure the parent that the well-being of the child is the chief concern.

The information recorded by the receptionist during this conversation constitutes the initial dental record for the patient. Filling out a patient information form is a convenient method of collecting the necessary initial information (see Fig. 29-2). Additional discussion of the initial communication with parents is presented in Chapter 29.

THE DIAGNOSTIC METHOD

Before making a diagnosis and developing a treatment plan, the dentist must collect and evaluate the facts associated with the patient's or parents' chief concern and any other identified problems that may be unknown to the patient or parents. Some pathognomonic signs may lead to an almost immediate diagnosis. For example, obvious gingival swelling and drainage may be associated with a single, badly carious primary molar. Although the collection and evaluation of these associated facts are performed rapidly, they provide a diagnosis only for a single problem area. On the other hand, a comprehensive diagnosis of all the patient's problems or potential problems may sometimes need to be postponed until more urgent conditions are resolved. For example, a patient with necrotizing ulcerative gingivitis or a newly fractured crown needs immediate treatment, but the treatment will likely be only palliative, and further diagnostic and treatment procedures will be required later.

The importance of thoroughly collecting and evaluating the facts concerning a patient's condition cannot be overemphasized. Moskow and Barr' have described several examination methods that aid the dentist in this process.

The following list of methods is based largely on their chapter "Examination of the Patient" in *Current Therapy in Dentistry:*

- Medical and dental history taking
- Inspection
- Palpation
- Auscultation
- Exploration
- Radiography
- Percussion
- Transillumination
- Vitality tests
- Study casts
- Laboratory tests
- Photography

In certain unusual cases all of these diagnostic aids may be necessary to arrive at a comprehensive diagnosis. Certainly no oral diagnosis can be complete unless the diagnostician has evaluated the facts obtained by medical and dental history taking, inspection, palpation, exploration (if teeth are present), and often imaging (radiographs, etc.). For a more thorough review of evaluation of the dental patient, the reader is referred to the chapter by Glick, Siegel, and Brightman in the textbook *Burket's Oral Medicine.*

Guidelines on Periodicity of Examination, Preventive Dental Services, Anticipatory Guidance, and Oral Treatment for Children, Revised May 2000

Birth-12 Months

1. Complete the clinical oral assessment and appropriate diagnostic tests to assess oral growth and development and/or pathology.
2. Provide oral hygiene counseling for parents, guardians, and caregivers, including the implications of the oral health of the caregiver.
3. Remove supra- and subgingival stains or deposits as indicated.
4. Assess the child's systemic and topical fluoride status (including type of infant formula used, if any, and exposure to fluoridated toothpaste), and provide counseling regarding fluoride. Prescribe systemic fluoride supplements if indicated, following assessment of total fluoride intake from drinking water, diet, and oral hygiene products.
5. Assess appropriateness of feeding practices, including bottle feeding and breast-feeding, and provide counseling as indicated.
6. Provide dietary counseling related to oral health.
7. Provide age-appropriate injury prevention counseling for orofacial trauma.
8. Provide counseling for non-nutritive oral habits (e.g., digit, pacifiers).
9. Provice diagnosis and required treatment and/or appropriate referral for any oral diseases or injuries.
10. Provide anticipatory guidance for parent/guardian. 1 1. Consult with the child's physician as indicated.
12. Based on evaluation and history, assess the patient's risk for oral disease.
13. Determine interval for periodic reevaluation.

12-24 Months

1. Repeat birth to 12-month procedures every 6 months or as indicated by the individual patient's needs/ susceptibility to disease.
2. Review patient's fluoride status, including any child care arrangements that may affect systemic fluoride intake and provide parental counseling.
3. Provide topical fluoride treatments every 6 months or as indicated by the individual patient's needs.

2-6 Years

1. Repeat 12- to 24-month procedures every 6 months or as indicated by the individual patient's needs/susceptibility to disease. Provide age-appropriate oral hygiene instructions.
2. Complete a radiographic assessment of pathology and/or abnormal growth and development, as indicated by the individual patient's needs.
3. Scale and clean the teeth every 6 months or as indicated by the individual patient's needs.
4. Provide topical fluoride treatments every 6 months or as indicated by the individual patient's needs.
5. Provide pit and fissure sealants for primary and permanent teeth as indicated by the individual patient's needs.
6. Provide counseling and services (athletic mouth guards) as needed for or ofacial trauma prevention.
7. Provide assessment/treatment or referral of developing malocclusion as indicated by the individual patient's needs.
8. Provide diagnosis and required treatment and/or appropriate referral for any oral disease, habits, or injuries as indicated.
9. Assess speech and language development, and provide appropriate referral as indicated.

6-12 Years

1. Repeat 2- to 6-year procedures every 6 months or as indicated by the individual patient's needs/susceptibility to disease.
2. Provide substance abuse counseling (e.g., smoking, smokeless tobacco).

12-18 Years

1. Repeat 6- to 12-year procedures every 6 months or as indicated by the individual patient's needs/susceptibility to disease.
2. At an age determined by the patient, parent, and dentist, refer the patient to a general dentist for continuing oral care.

f I G. **1-1.** General oral health care guidelines for children.

PRELIMINARY MEDICAL AND DENTAL HISTORY

It is important for the dentist to be familiar with the medical and dental history of the pediatric patient. Familial history may also be relevant to the patient's oral condition and may provide important diagnostic information in some hereditary disorders. Before the dentist examines the child, the dental assistant can obtain sufficient information to provide the dentist with knowledge of the child's general health and can alert the dentist to the need for obtaining additional information from the parent or the child's physician. The form illustrated in Fig. 1-2 can be completed by the parent. However, it is more effective for the dental assistant to ask the questions informally and then to present the findings to the dentist and offer personal observations and a summary of the case. The questions included on the form will also provide information about any previous dental treatment.

Information regarding the child's social and psychologic development is important. Accurate information reflecting a child's learning, behavioral, or communication problems is sometimes difficult to obtain initially, especially if the parents are aware of their child's developmental disorder but are reluctant to discuss it. Behavior problems in the dental office are often related to the child's inability to communicate with the dentist and to follow instructions. This inability may be attributable to a learning disorder. An indication of learning

DATE_____

Child's name_____ Sex ____ Birth date_____ Place of birth _____
Last, first, nickname

Date of last medical examination _____ Child's physician/pediatrician _____ Telephone_____

Physician's address _____

MEDICAL HISTORY

GROWTH AND DEVELOPMENT Any learnig, behavioral, excessive nervousness, or communication problems?	No ( )	Yes ( )
Has child had psychological counseling or is counseling being considered for the near future?	No ( )	Yes ( )
Were there any complications during pregnancy or was child premature at birth?	No ( )	Yes ( )
Any problems with physical growth?	No ( )	Yes ( )
CENTRAL NERVOUS SYSTEM Any history of cerebral palsy, seizures, convulsions, fainting, or loss of consciousness?	No ( )	Yes ( )
Any history of injury to the head?	No ( )	Yes ( )
Any sensory disorders? (Seeing, Hearing)	No ( )	Yes ( )
CARDIOVASCULAR SYSTEM Any history of congenital heart disease, heart murmur, or heart damage from rheumatic fever?	No ( )	Yes ( )
Has any heart surgery been done or recommended?	No ( )	Yes ( )
Any history of chest pains or high blood pressure?	No ( )	Yes ( )
HEMATOPOIETIC AND LYMPHATIC SYSTEMS Has your child ever had a blood transfusion or blood products transfusion?	No ( )	Yes ( )
Any history of anemia or sickle cell disease?	No ( )	Yes ( )
Does your child bruise easily, have frequent nosebleeds, or bleed excessively from small cuts?	No ( )	Yes ( )
Is your child more susceptible to infections than other children are?	No ( )	Yes ( )
Is there any history of tender or swollen lymph nodes or glands?	No ( )	Yes ( )
RESPIRATORY SYSTEM Any history of pneumonia, cystic fibrosis, asthma, shortness of breath, or difficulty in breathing?	No ( )	Yes ( )
GASTROINTESTINAL SYSTEM Any history of stomach, intestinal or liver problems?	No ( )	Yes ( )
Any history of hepatitis or jaundice?	No ( )	Yes ( )
Any history of eating disorders, such as anorexia nervosa or bulimia (binge/purge)?	No ( )	Yes ( )
Any history of unintentional weight loss?	No ( )	Yes ( )
GENITOURINARY SYSTEM Any history of urinary tract infections, bladder or kidney problems?	No ( )	Yes ( )
Is the patient pregnant or possibly pregnant?	No ( )	Yes ( )
ENDOCRINE SYSTEM Any history of diabetes?	No ( )	Yes ( )
Any history of thyroid disorders or other glandular disorders?	No ( )	Yes ( )
SKIN Any history of skin problems?	No ( )	Yes ( )
Any history of cold sores (herpes) or canker sores (aphthae)?	No ( )	Yes ( )
EXTREMITIES Any limitations of use of arms or legs?	No ( )	Yes ( )
Any arthritis, joint bleeding, joint replacements or other joint problems?	No ( )	Yes ( )
Any problems with muscle weakness or muscular dystrophy?	No ( )	Yes ( )
ALLERGIES Is your child allergic to any medication?	No ( )	Yes ( )
Any hay fever, hives, or skin rashes caused by allergies?	No ( )	Yes ( )
Any other allergies?	No ( )	Yes ( )
MEDICATIONS OR TREATMENTS Is your child currently taking any medication (prescription or non-prescription medicine)?	No ( )	Yes ( )

If yes, Medication(s) Dose Times per day

_____ _____ _____
_____ _____ _____
_____ _____ _____

Has your child ever recieved radiation therapy (x-ray treatments) or is it planned?	No ( )	Yes ( )
Has your child ever recieved chemotherapy or is it planned?	No ( )	Yes ( )

HOSPITALIZATIONS

Has your child been hospitalized?

Hospital	(1)_____	(2)_____	(3)_____
Date	_____	_____	_____
Reason	_____	_____	_____

[I G **1-2.** Form used in completing the preliminary medical and dental history. *Continued*

disorders can usually be obtained by the dental assistant while asking questions about the child's learning process; for example, asking a young school-aged child how he or she is doing in school is a good lead question. Remember, though, to keep the questions age-appropriate to the child.

A notation should be made if a young child has been hospitalized previously for general anesthetic and surgical procedures. Shaw has reported that hospitalization and a general anesthetic procedure can be a traumatic psychologic experience for a preschool child and may sensitize the youngster to procedures that will

IMMUNIZATIONS: Is your child presently protected by immunization against

DPT: diphtheria, whooping cough (pertussis), tetanus?	No ( )	Yes ( )
IPV: polio or poliomyelitis?	No ( )	Yes ( )
MMR: measles (rubeola), mumps, and German measles (rubella)?	No ( )	Yes ( )
Hib (*Haemophilus b*)?	No ( )	Yes ( )
Pneumococcal pneumonia?	No ( )	Yes ( )
Hepatitis B	No ( )	Yes ( )

PLEASE CHECK ANY OF THE ILLNESSES THAT YOUR CHILD HAS NOW, HAS RECENTLY BEEN EXPOSED TO, OR HAS HAD IN THE PAST:

	Now	Exposed	Past
• Chickenpox (varicella)	☐	☐	☐
• Earache (otitis)	☐	☐	☐
• Eye infection (conjunctivitis)	☐	☐	☐
• German measles or 3-day measles (rubella)	☐	☐	☐
• Glandular fever or mono (infectious mononucleosis)	☐	☐	☐
• HIV/AIDS	☐	☐	☐
• Lead poisoning	☐	☐	☐
• Measles (rubella)	☐	☐	☐
• Mumps (parotitis)	☐	☐	☐
• Scarlet fever (scarlatina)	☐	☐	☐
• Sore throat (tonsillitis or pharyngitis)	☐	☐	☐
• Substance abuse, alcoholism, drug addiction	☐	☐	☐
• Tuberculosis	☐	☐	☐
• Upper respiratory infection (URI), or common cold (pharyngitis, rhinitis, sinusitis, or tonsillitis)	☐	☐	☐
• Venereal disease (genital herpes, gonorrhea, syphilis or other)	☐	☐	☐

DENTAL HISTORY

Does your child have a toothache or other immediate dental problem?	No ( )	Yes ( )
Has your child ever had a toothache?	No ( )	Yes ( )
Has your child had any injury to the mouth, teeth or jaws (fall, blow, etc.)?	No ( )	Yes ( )
Is this your child's first dental visit? If no:	No ( )	Yes ( )
Date: Dentist: Reason:		
Has your child ever had an unfavorable dental experience?	No ( )	Yes ( )
Is (was) your child nourished by nursing beyond one year of age? If yes:	No ( )	Yes ( )
Check: Breast _____ Nursing bottle _____ Both _____, and to what age? _____		
Does your child fail to eat a well-balanced diet? If yes, what foods or food groups are not adequate? _____	No ( )	Yes ( )
Does (or has) your child have (or had) sucking habit beyond one year of age? If yes:	No ( )	Yes ( )
Check: Thumb(s) _____ Finger(s) _____ Pacifier _____ Other: _____		
Does (or has) your child have (or had) any other oral habits beyond one year of age? If yes:	No ( )	Yes ( )
Check: Lip biting _____ Mouth breathing, _____ Nail biting _____ Teeth grinding _____ Other _____		
Does (or has) your child have (or had) difficulty opening his or her mouth, or does the child's jaw sometimes lock or stick in a certain position?	No ( )	Yes ( )
Does (or has) your child have (or had) popping or clicking noises or pain during chewing or yawning?	No ( )	Yes ( )
Does (or has) your child have (or had) frequent headaches or pain in or about the ears, eyes, or cheeks?	No ( )	Yes ( )

DENTAL DISEASE PREVENTION

How often does your child brush? _____ times per _____	No ( )	Yes ( )
Does your child use dental floss?	No ( )	Yes ( )
Does someone assist your child with brushing and cleaning the teeth?	No ( )	Yes ( )
Does someone inspect for thoroughness after the procedure?	No ( )	Yes ( )
Does your child use a fluoride toothpaste?	No ( )	Yes ( )
Has your child ever had a fluoride treatment?	No ( )	Yes ( )
Has your child ever taken fluoride supplement or vitamins with fluorides?	No ( )	Yes ( )

DRINKING WATER SOURCE

City water supply _____ Name of city _____
Private well or other than city _____ Has a fluoride analysis been done? _____
Date of analysis _____ Fluoride content _____

▶ Signature (Parent or guardian) ▶ Student Signature

DENTIST'S COMMENTS:

Medical consultation recommended? No _____ Yes _____ Date requested _____
Purpose for consultation: _____

SEMIANNUAL REVIEW OF MEDICAL-DENTAL HISTORY: If history remains essentially unchanged, sign below

Date _____ Parent _____ Student _____
Date _____ Parent _____ Student _____
Date _____ Parent _____ Student _____

A new history form should be completed at least every 2 years.

[I t. **1-2, confd,** Form used in completing the preliminary medical and dental history.

be encountered later in a dental office.' If the dentist is aware of previous hospitalization and the child's fear of strangers in clinic attire, the necessary time and procedures can be planned to help the child overcome the fear and accept dental treatment.

Occasionally, when the parents report significant disorders, it is best for the dentist to conduct the medical and dental history interview. When the parents meet with the dentist privately, they are more likely to discuss the child's problems openly and there is less chance for misunderstandings regarding the nature of the disorders. In addition, the dentist's personal involvement at this early time strengthens the confidence of the parents. When there is indication

of an acute or chronic systemic disease or anomaly, the dentist should consult the child's physician to learn the status of the condition, the long-range prognosis, and the current drug therapy.

Current illnesses or histories of significant disorders signal the need for special attention during the medical and dental history interview. In addition to consulting the child's physician, the dentist may decide to record additional data concerning the child's current physical condition, such as blood pressure, body temperature, heart sounds, height and weight, pulse, and respiration. Before treatment is initiated, certain laboratory tests may be indicated and special precautions may be necessary. A decision to provide treatment in a hospital and possibly under general anesthesia may be appropriate.

The dentist and the staff must also be alert to identify potentially communicable infectious conditions that threaten the health of the patient and others as well. Knowledge of the current recommended childhood immunization schedule is helpful. It is advisable to postpone nonemergency dental care for a patient exhibiting signs or symptoms of acute infectious disease until the patient has recovered. Further discussions of management of dental patients with special medical, physical, or behavioral problems are presented in Chapters 2, 3, 14, 15, 23, 24, and 28.

The pertinent facts of the medical history can be transferred to the oral examination record (Fig. 1-3) for easy reference by the dentist. A brief summary of important medical information serves as a convenient reminder to the dentist and the staff, because they refer to this chart at each treatment visit.

The patient's dental history should also be summarized on the examination chart. This should include a record of previous care in the dentist's office and the facts related by the patient and the parent regarding previous care in another office. Information concerning the patient's current oral hygiene habits and previous and current fluoride exposure helps the dentist develop an effective dental disease prevention program for the patient. For example, if the family drinks well water, a sample may be sent to a water analysis laboratory to determine the fluoride concentration.

CLINICAL EXAMINATION

Most facts needed for a comprehensive oral diagnosis in the young patient are obtained by a thorough clinical and radiographic examination. In addition to examining the structures in the oral cavity, the dentist may in some cases wish to note the patient's size, stature, gait, or involuntary movements. The first clue to malnutrition may come from observing a patient's abnormal size or stature. Similarly, the severity of a child's illness, even if oral in origin, may be recognized by observing a weak, unsteady gait of lethargy and malaise as the patient walks into the office. All relevant information should be noted on the oral examination record (see Fig. 1-3), which becomes a permanent part of the patient's chart.

The clinical examination, whether the first examination or a regular recall examination, should be all inclusive. The dentist can gather useful information while getting acquainted with a new patient. Attention to the patient's hair, head, face, neck, and hands should be among the first observations made by the dentist after the patient is seated in the chair.

The patient's hands may reveal information pertinent to the comprehensive diagnosis. The dentist may first detect an elevated temperature by holding the patient's hand. Cold, clammy hands or bitten fingernails may be the first indication of abnormal anxiety in the child. A callused or unusually clean digit suggests a persistent sucking habit. Clubbing of the fingers or a bluish color in the nail beds suggests congenital heart disease that may require special precautions during dental treatment.

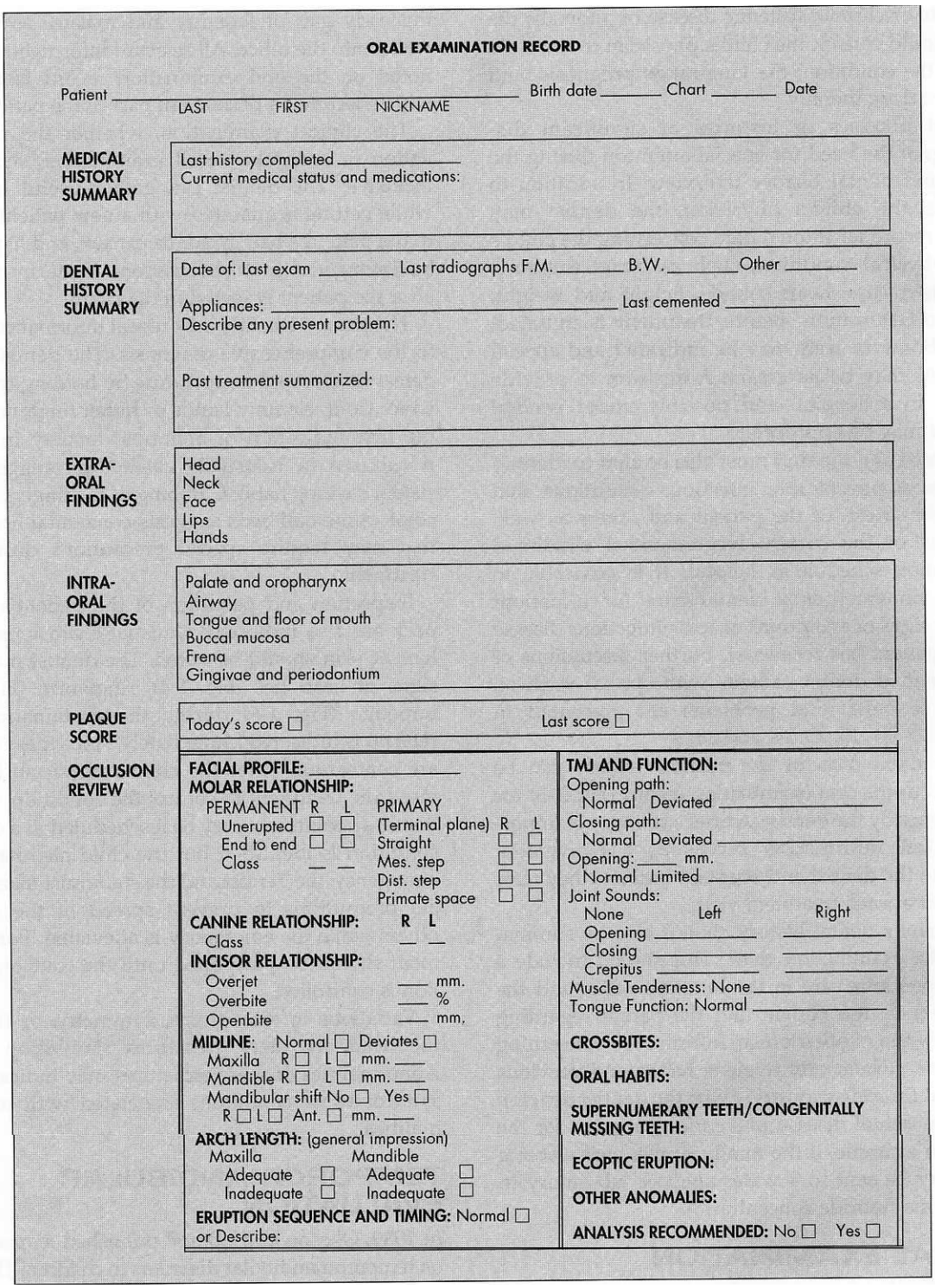
Inspection and palpation of the patient's head and neck are also indicated. Unusual characteristics of the hair or skin should be noted. The dentist may observe signs of head lice (Fig. 1-4), ringworm (Fig. 1-5), or impetigo (Fig. 1-6) during the examination. Proper referral is indicated immediately, since these conditions are contagious. After the child's physician has supervised the treatment to control the condition, the child's dental appointment may be rescheduled. If a contagious condition is identified but the child also has a dental emergency, the dentist and the staff must take appropriate precautions to prevent spread of the disease to others while the emergency is alleviated. Further treatment should be postponed until the contagious condition is controlled.

Variations in size, shape, symmetry, or function of the head and neck structures should be recorded. Abnormalities of these structures may indicate various syndromes or conditions associated with oral abnormalities.

TEMPOROMANDIBULAR EVALUATION

In 1989, Okeson and others' published a special report on temporomandibular disorders in children. The authors indicated that, although several studies include children 5 to 7 years of age, most observations have been made on the young adolescent. Studies have placed the findings into the categories of symptoms or signs—those reported by the child or parents and those identified by the dentist during the examination.

One should evaluate temporomandibular joint (TMT) function by palpating the head of each mandibular condyle and observing the patient while the mouth is



ORAL EXAMINATION RECORD

Patient _____ Birth date _____ Chart _____ Date
LAST FIRST NICKNAME

MEDICAL HISTORY SUMMARY

Last history completed _____
Current medical status and medications:

DENTAL HISTORY SUMMARY

Date of: Last exam _____ Last radiographs F.M. _____ B.W. _____ Other _____
Appliances: _____ Last cemented _____
Describe any present problem:

Past treatment summarized:

EXTRA-ORAL FINDINGS

Head
Neck
Face
Lips
Hands

INTRA-ORAL FINDINGS

Palate and oropharynx
Airway
Tongue and floor of mouth
Buccal mucosa
Frena
Gingivae and periodontium

PLAQUE SCORE

Today's score ☐ Last score ☐

OCCLUSION REVIEW

FACIAL PROFILE: _____
MOLAR RELATIONSHIP:

PERMANENT	R	L	PRIMARY (Terminal plane)	R	L
Unerupted	☐	☐	Straight	☐	☐
End to end	☐	☐	Mes. step	☐	☐
Class	__	__	Dist. step	☐	☐
			Primate space	☐	☐

CANINE RELATIONSHIP: R L
Class __ __
INCISOR RELATIONSHIP:
Overjet ___ mm.
Overbite ___ %
Openbite ___ mm.
MIDLINE: Normal ☐ Deviates ☐
Maxilla R ☐ L ☐ mm. ___
Mandible R ☐ L ☐ mm. ___
Mandibular shift No ☐ Yes ☐
R ☐ L ☐ Ant. ☐ mm. ___
ARCH LENGTH: (general impression)
Maxilla Adequate ☐ Inadequate ☐
Mandible Adequate ☐ Inadequate ☐
ERUPTION SEQUENCE AND TIMING: Normal ☐
or Describe:

TMJ AND FUNCTION:
Opening path:
Normal Deviated _____
Closing path:
Normal Deviated _____
Opening: ____ mm.
Normal Limited _____
Joint Sounds:

None	Left	Right
Opening	_____	_____
Closing	_____	_____
Crepitus	_____	_____

Muscle Tenderness: None _____
Tongue Function: Normal _____

CROSSBITES:

ORAL HABITS:

SUPERNUMERARY TEETH/CONGENITALLY MISSING TEETH:

ECOPTIC ERUPTION:

OTHER ANOMALIES:

ANALYSIS RECOMMENDED: No ☐ Yes ☐

FIG. 1-3. Chart used to record the oral findings and the treatment proposed for the pediatric patient.

closed (teeth clenched), at rest, and in various open positions (Fig. 1-7, A and B). Movements of the condyles or jaw that are not smoothly flowing or deviate from the expected norm should be noted. Similarly, any crepitus that may be heard or identified by palpation, or any other abnormal sounds, should be noted. Sore masticatory muscles may also signal TMJ dysfunction. Such deviations from normal TMJ function may require further evaluation and treatment. There is a consensus (American Academy of Pediatric Dentistry, Reference

CLINICAL	RADIOGRAPHIC	CLINICAL	RADIOGRAPHIC
1 2 3 4/A 5/B 6/C 7/D 8/E		16 15 14 13/J 12/I 11/H 10/G 9/F	
25/P 26/Q 27/R 28/S 29/T 30 31 32		24/O 23/N 22/M 21/L 20/K 19 18 17	

DIAGNOSTIC SUMMARY:

PERIODONTAL SCREENING AND RECORDING

Sextant			
Score			

TREATMENT PROPOSED		
PROPOSED APPLIANCES, REFERRALS, CONSULTATIONS, AND OTHER NOTATIONS:		

f G 1-3, cofIt'd, See legend on opposite page.



FIG . **1-4.** Evidence of head lice infestation. Usually the insects are not seen, but their eggs, or nits, cling to hair filaments until they hatch. *(Courtesy Dr. Hala Henderson.)*

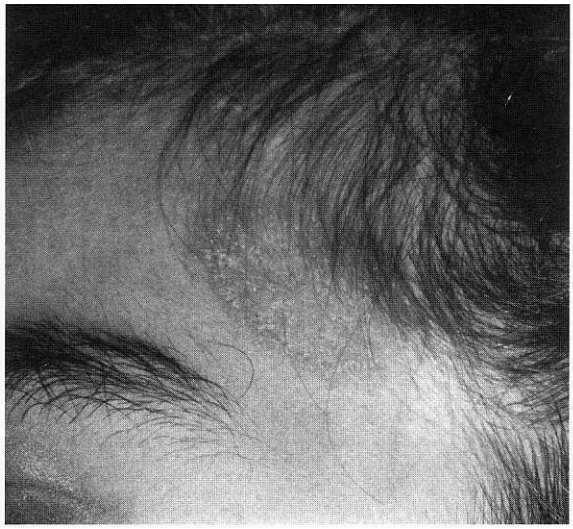


FIG. **1-5.** Lesion on forehead above left eyebrow is caused by ringworm infection. Several fungal species may cause the lesions on various areas of the body. The dentist may identify lesions on the head, face, or neck of a patient during a routine clinical examination. *(Courtesy Dr. Hala Henderson.)*

Manual 2002-2003) that temporomandibular disorders in children can be managed effectively by the following conservative and reversible therapies: patient education, mild physical therapy, behavioral therapy, medications, and occlusal splints.`'

Discussion of the diagnosis and treatment of complex TMJ disorders goes beyond the scope of this text. However, information on this subject is available from many sources. We suggest Okeson's *Management of Ten porornandibular Disorders and Occlusion* (2003) as an excellent textbook in this subject area.

The extraoral examination continues with palpation of the patient's neck and submandibular area (Fig. 1-7, C and D). Again, deviations from normal, such as unusual tenderness or enlargement, should be noted and follow-up tests performed or referrals made as indicated.

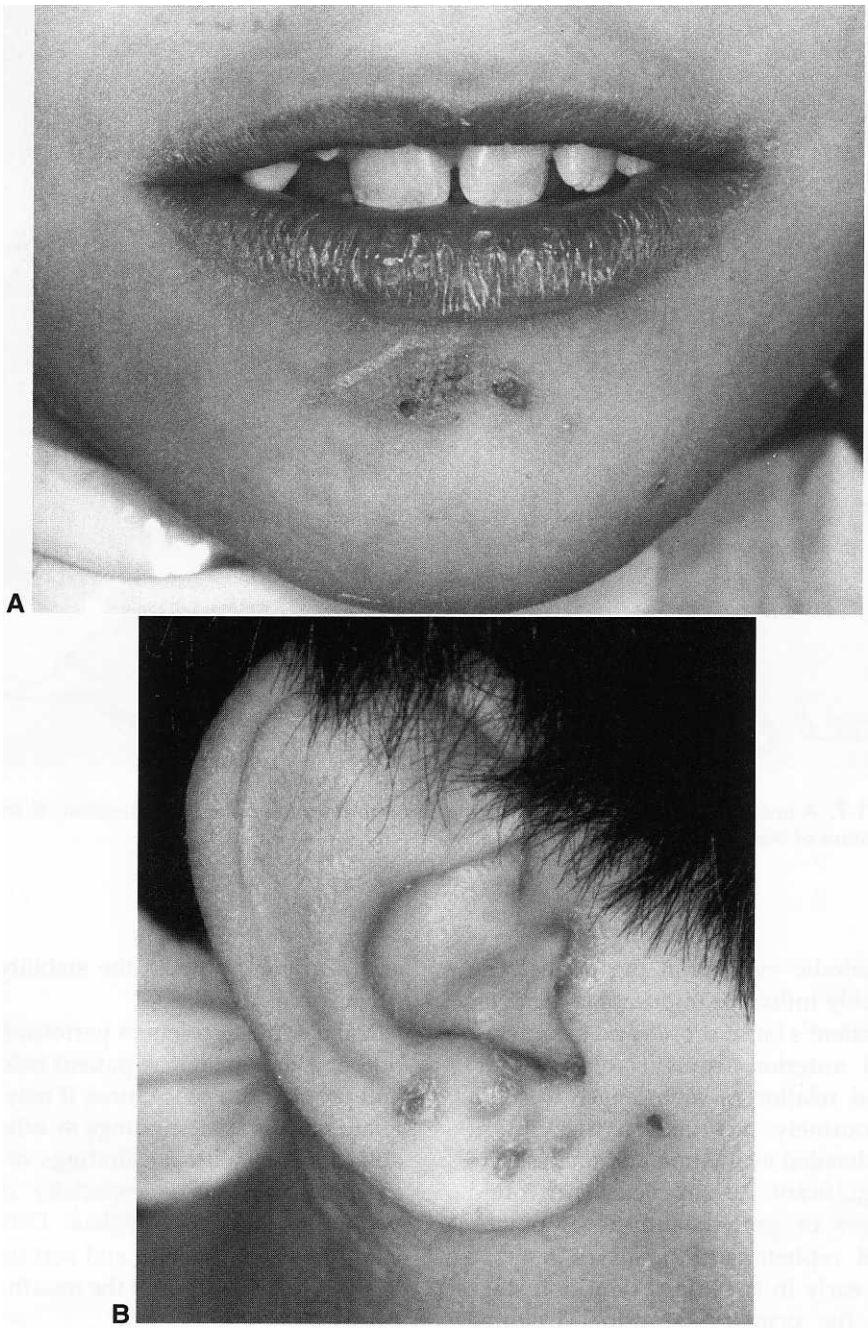
If the child is old enough to talk, speech should be evaluated. The positions of the tongue, lips, and paraoral musculature during speech, while swallowing, and while at rest may provide useful diagnostic information.

The intraoral examination of a pediatric patient should be comprehensive. There is a temptation to look first for obvious carious lesions. Certainly con-trolling carious lesions is important, but the dentist should first evaluate the condition of the oral soft tissues and the status of the developing occlusion. If the soft tissues and the occlusion are not observed early in the examination, the dentist may become so engrossed in charting carious lesions and in planning for their restoration that other important anomalies in the mouth are overlooked. Any unusual breath odors and abnormal quantity or consistency of saliva should also be noted.

The buccal tissues, lips, floor of the mouth, palate, and gingivae should be carefully inspected and palpated (Fig. 1-8). The use of the periodontal screening and recording program (PSR) is often a helpful adjunct in children. PSR is designed to facilitate early detection of periodontal diseases with a simplified probing technique and minimal documentation. Clerehugh and Tugnait[s] recommend initiation of periodontal screening in children following eruption of the permanent incisors and the first molars. They suggest routine screening in these children at the child's first appointment and at regular recare appointments so that periodontal problems are detected early and treated appropriately. Immunodeficient children are especially vulnerable to early loss of bone support.

A more detailed periodontal evaluation is occasionally indicated even in young children. Periodontal disorders of children are discussed further in Chapter 20.

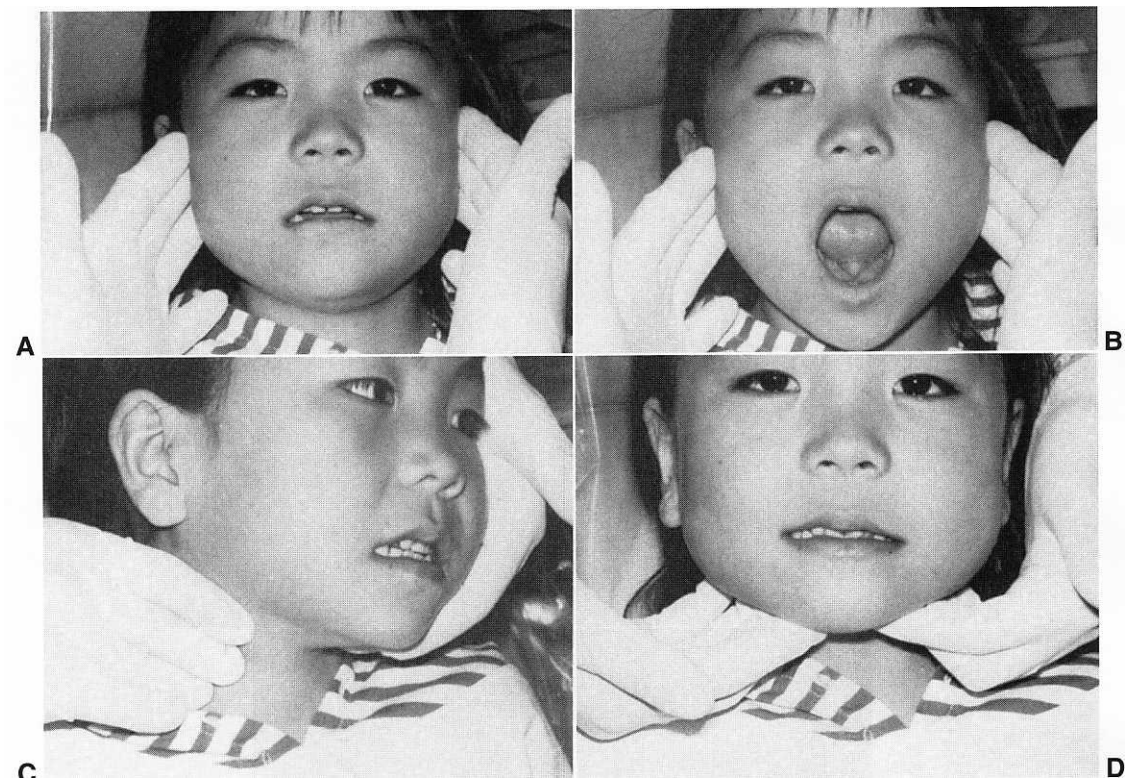
The tongue and oropharynx should be closely inspected. Enlarged tonsils accompanied by purulent exudate may be the initial sign of a streptococcal infection, which can lead to rheumatic fever. When streptococcal throat infection is suspected, immediate referral to the child's physician is indicated. In some cases it may be helpful to the physician and



[1 C **1-6.** Characteristic lesions of impetigo, on the lower face **(A)** and on the right ear (B). These lesions occur on various skin surfaces, but the dentist is most likely to encounter them on upper body areas. The infections are of bacterial (usually streptococcal) origin and generally require antibiotic therapy for control. The child often spreads the infection by scratching the lesions. *(Courtesy Dr. Halo Henderson.)*

convenient for the dentist to obtain a throat culture specimen while the child is still in the dental office, which contributes to an earlier definitive diagnosis of the infection. The diagnosis and treatment of soft tissue problems are discussed throughout this book, especially in Chapters 7, 8, and 20.

After thoroughly examining the oral soft tissues, the dentist should inspect the occlusion and note any dental or skeletal irregularities. The dentition and resulting occlusion may undergo considerable change during childhood and early adolescence. This dynamic developmental process occurs in all three planes of



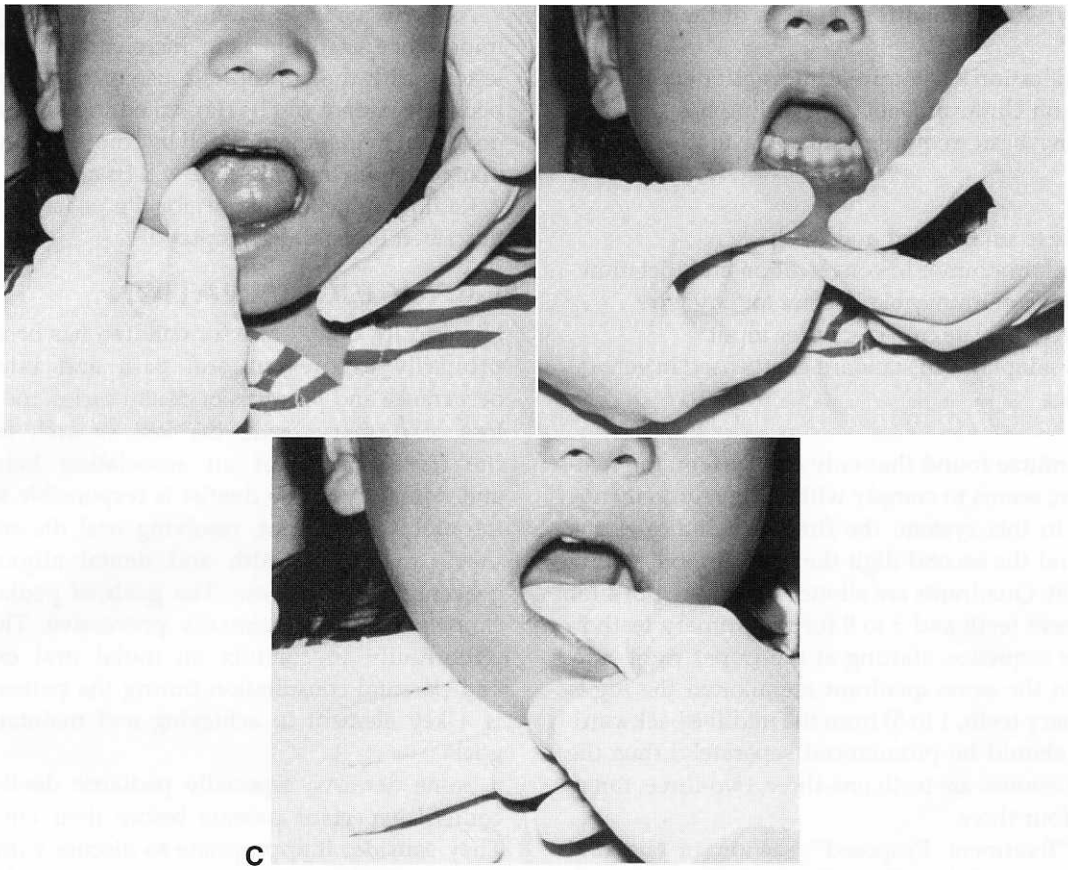
f **C 1-7. A** and **B,** Observation and palpation of temporomandibular joint function. C and **D,** Palpation of the neck and submandibular areas.

space, and with periodic evaluation the dentist can intercept and favorably influence undesirable changes. Monitoring of the patient's facial profile and symmetry; molar, canine, and anterior segment relationships; dental midlines; and relation of arch length to tooth mass should be routinely included in the clinical examination. More detailed evaluation and analysis are indicated when significant discrepancies are found during critical stages of growth and development. Diagnostic cast and cephalometric analyses may be indicated relatively early in the mixed dentition stage and sometimes in the primary dentition. Detailed discussions of analyses of developing occlusions and interceptive treatment recommendations are presented in Chapters 25 through 27.

Finally, the teeth should be inspected carefully for evidence of carious lesions and hereditary or acquired anomalies. The teeth should also be counted and identified individually to ensure recognition of supernumerary or missing teeth. Identification of carious lesions is important in patients of all ages but is especially critical in young patients because the lesions may progress rapidly in early childhood caries if not controlled. Eliminating the carious activity and restoring the teeth as needed will prevent pain and the spread of infection and also contribute to the stability of the developing occlusion.

If the dentist prefers to perform the clinical examination of a new pediatric patient before the radiographic and prophylaxis procedures, it may be necessary to correlate radiographic findings or other initially questionable findings with the findings of a second brief oral examination. This is especially true when the new patient has poor oral hygiene. Detailed inspection and exploration of the teeth and soft tissues cannot be performed adequately until the mouth is free of extraneous debris.

During the clinical examination for carious lesions each tooth should be dried individually and inspected under a good light. A definite routine for the examination should be established. For example, a dentist may always start in the upper right quadrant, work around the maxillary arch, move down to the lower left quadrant, and end the examination in the lower right quadrant. Morphologic defects and incomplete coalescence of enamel at the base of pits and fissures in molar teeth can often be detected readily by visual and explorer examination after the teeth have been cleaned and dried. The decision as to whether to place a sealant or to restore a defect depends on the patient's



[I G . **1-8.** Inspection and palpation of the buccal tissues IA), the lips (B), and the floor of the mouth (C).

history of dental caries, the parents' or patient's acceptance of a comprehensive preventive dentistry program (including dietary and oral hygiene control), and the patient's dependability in returning for recare appointments.

In patients with severe dental caries, caries activity tests and diet analysis may contribute to the diagnostic process by helping to define specific etiologic factors. These procedures probably have an even greater value in helping the patient or parents understand the carious disease process and in motivating them to make the behavioral changes needed to control the disease. The information provided to the patient or parents should include instruction in plaque control and the appropriate recommendations for fluoride exposure. Dental caries susceptibility, the caries disease process, caries activity tests, diet analysis, and caries control are discussed in Chapter 10. Plaque control procedures and instructions are presented in detail in Chapter 11.

The dentist's comprehensive diagnosis depends on the completion of a number of procedures but requires a thorough, systematic, and critical clinical examination. Any deviation from the expected or desired size, shape, color, and consistency of soft or hard tissues should be described in detail. The severity of associated problems and their causes must be clearly identified to the parents or the patient before success of a comprehensive oral health care program can be expected.

During the initial examination and also at subsequent appointments, the dentist and auxiliaries should be alert to signs and symptoms of child abuse and neglect. Because of the increasing prevalence of these problems and the important role the dentist can play in detecting their signs and symptoms, Chapter 2 has been devoted to this subject.

UNIFORM DENTAL RECORDING

Many different tooth charting systems are currently in use, including the universal system illustrated in the hard tissue examination section of Fig. 1-3. This system of marking permanent teeth uses the numbers 1 to 32, beginning with the upper right third molar (No. 1) and progressing around the arch to the upper left third molar (No. 16), down to the lower left third molar (No. 17), and around the arch to the lower right third molar (No. 32). The primary teeth are identified in the

universal system by the first 20 letters of the alphabet, A through T.

The Federation Dentaire International Special Committee on Uniform Dental Recording has specified the following basic requirements for a tooth charting system:

1. Simple to understand and teach
2. Easy to pronounce in conversation and dictation
3. Readily communicable in print and by wire
4. Easy to translate into computer input
5. Easily adaptable to standard charts used in general practice

The committee found that only one system, the two-digit system, seems to comply with these requirements. According to this system, the first digit indicates the quadrant and the second digit the type of tooth within the quadrant. Quadrants are allotted the digits 1 to 4 for the permanent teeth and 5 to 8 for the primary teeth in a clockwise sequence, starting at the upper right side; teeth within the same quadrant are allotted the digits 1 to 8 (primary teeth, 1 to 5) from the midline backward. The digits should be pronounced separately; thus the permanent canines are teeth one-three, two-three, three-three, and four-three.

In the "Treatment Proposed" section of the oral examination record (see Fig. 1-3), the individual teeth that require restorative procedures, endodontic therapy, or extraction are listed. Gingival areas requiring follow-up therapy are also noted. A check mark can be placed beside each listed tooth and procedure as the treatment is completed. Additional notations concerning treatment procedures completed and the date are recorded on supplemental treatment record pages.

RADIOGRAPHIC EXAMINATION

When indicated, radiographic examination for children must be completed before the comprehensive oral health care plan can be developed, and subsequent radiographs are required periodically to allow detection of incipient carious lesions or other developing anomalies.

A child should be exposed to dental ionizing radia-tion only after the dentist has determined the radio-graphic requirement, if any, to make an adequate diagnosis for the individual child at the time of the appointment.

Obtaining isolated occlusal, periapical, or bite-wing films is sometimes indicated in *very* young children (even infants) because of trauma, toothache, suspected developmental disturbances, or proximal caries. Carious lesions appear smaller on radiographs than they actually are.

As early as 1967, Blayney and Hill° recognized the importance of diagnosing incipient proximal carious lesions with the appropriate use of radiographs. If the pediatric patient can be motivated to adopt a routine of good oral hygiene supported by competent supervision, many of these initial lesions will be arrested.

Radiographic techniques for the pediatric patient are described in detail in Chapter 5.

EARLY EXAMINATION

Historically, dental care for children has been designed primarily to prevent oral pain and infection, the occurrence and progress of dental caries, the premature loss of primary teeth, the loss of arch length, and the development of an association between fear and dental care. The dentist is responsible for guiding the child and parent, resolving oral disorders before they can affect health and dental alignment, and preventing oral disease. The goals of pediatric dental care therefore are primarily preventive. The dentist's opportunity to conduct an initial oral examination and parental consultation during the patient's infancy is a key element in achieving and maintaining these goals.

Some dentists, especially pediatric dentists, like to counsel expectant parents before their child is born. They consider it appropriate to discuss with expectant mothers the importance of good nutrition during pregnancy and practices that can influence the expected child's general and dental health.

It is also appropriate to inquire about medication the expectant mother is taking. It is known that the prolonged ingestion of tetracyclines may result in discolored, pigmented, and even hypoplastic primary teeth.

The expectant mother should be encouraged to visit her dentist and to have all carious lesions restored. The presence of active dental caries and accompanying high levels of *Streptococcus rnntans* can lead to trans-mission by the mother to the infant and may be responsible for the development of carious lesions at a very early age.

It is not intended that the pediatric dentist should usurp the responsibility of the expectant mother's physician in recommending dietary practices, but instead that the dentist should reinforce good nutritional recommendations provided by medical colleagues.

INFANT DENTAL CARE

In 2002 the American Academy of Pediatric Dentistry revised the guidelines on infant oral health care.

The infant oral health care visit should be seen as the foundation on which a lifetime of preventive education and dental care can be built to help assure optimal oral

health into childhood. Oral examination, anticipatory guidance including preventive education, and appropriate therapeutic intervention for the infant can enhance the opportunity for a lifetime of freedom from preventable oral disease.

RECOMMENDATIONS:

1. Infant oral health care begins ideally with prenatal oral health counseling for parents. An initial oral evaluation visit should occur within 6 months of the eruption of the first primary tooth and no later than 12 months of age.
2. At the infant oral evaluation visit, the dentist should do the following:
   a. Record a thorough medical and dental history, covering the prenatal, perinatal, and postnatal periods
   b. Complete a thorough oral examination
   c. Assess the patient's risk of developing oral and dental disease, and determine an appropriate interval for periodic reevaluation based on that assessment
   d. Discuss and provide anticipatory guidance regarding dental and oral development, fluoride status, nonnutritive oral habits, injury prevention, oral hygiene, and effects of diet on dentition
3. Dentists who perform such services for infants should be prepared to provide therapy when indicated or should refer the patient to an appropriately trained individual for necessary treatment.

Thus it is appropriate for a dentist to perform an oral examination for an infant of any age, even a newborn, and an examination is recommended anytime the parent or physician calls with questions concerning the appearance of an infant's oral tissues. Even when there are no known problems, the child's first dental visit and oral examination should take place by at least 1 year of age. This early dental visit enables the dentist and parents to discuss ways to nurture excellent oral health before any serious problems have had an opportunity to develop. An adequate oral examination for an infant is generally quite simple and very brief, but it may be the important first step toward a lifetime of excellent oral health.

Some dentists may prefer to "preside" during the entire first session with the infant and parents. Others may wish to delegate some of the educational aspects of the session to auxiliary members of the office staff and then conduct the examination and answer any unresolved questions. In either case, it is sometimes necessary to have an assistant available to help hold the child's attention so that the parents can concentrate on the important information being provided.

It is not always necessary to conduct the infant oral examination in the dental operatory, but it should take place where there is adequate light for a visual examination. The dentist may find it convenient to conduct the examination in the private consultation room during the initial meeting with the child and the parents. The examination procedures may include only direct observation and digital palpation. However, if primary molars have erupted or if hand instruments may be needed, the examination should be performed in an area where instrument transfers between the dental assistant and the dentist may proceed smoothly.

The parents should be informed before the examination that it will be necessary to gently restrain the child and that it is normal for the child to cry during the procedure. The infant is held on the lap of a parent, usually the mother. This direct involvement of the parent provides emotional support to the child and allows the parent to help restrain the child. Both parents may participate or at least be present during the examination.

The dentist should make a brief attempt to get acquainted with the infant and to project warmth and caring. However, many infants and toddlers are not particularly interested in developing new friendships with strangers, and the dentist should not be discouraged if the infant shuns the friendly approach. Even if the child chooses to resist (which is common and normal), only negligible extra effort is necessary to perform the examination procedure. The dentist should not be flustered by the crying and resistant behavior and should proceed unhurriedly but efficiently with the examination. The dentist's voice should remain unstrained and pleasant during the examination. The dentist's behavior should reassure the child and alleviate the parents' anxiety concerning this first dental procedure.

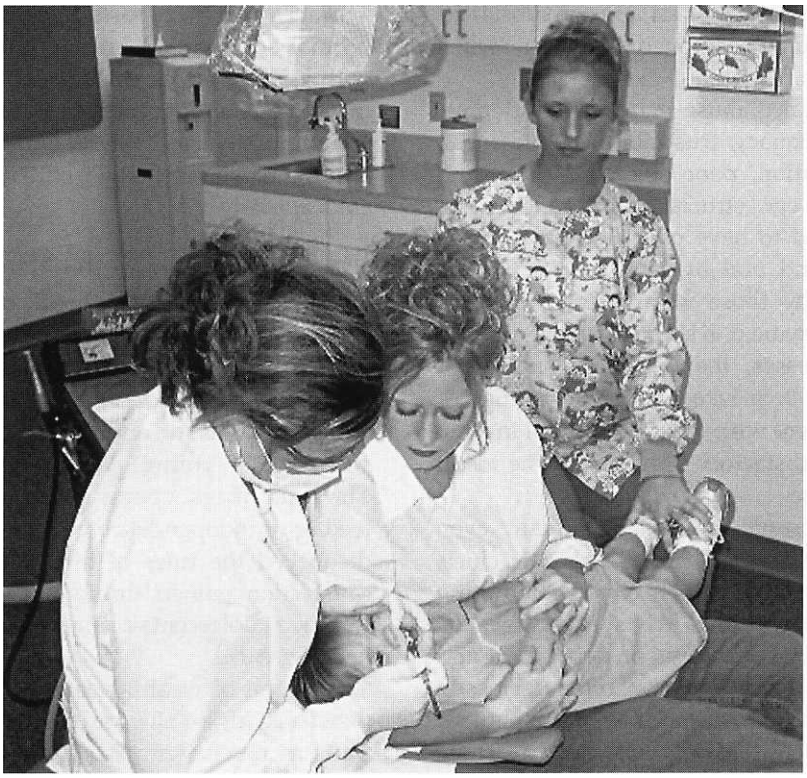
One method of performing the examination in a private consultation area is illustrated in Fig. 1-9, A. The dentist and the parent are seated face to face with their knees touching. Their upper legs form the "examination table" for the child. The child's legs straddle the parent's body, which allows the parent to restrain the child's legs and hands. An assistant is present to record the dentist's examination findings as they are dictated and to help restrain the child if needed. If adequate space is available in the consultation area, the approach illustrated in Fig. 1-9, B, may prove useful. The dental assistant is seated at a desk or writing stand near the child's feet. The dental assistant and the parent are facing the same direction side by side and at a right angle to the direction the dentist is facing. The dental assistant is in a good position to hear and record the dentist's findings as they are dictated, even if the child is crying loudly. These positions (see Fig. 1-9) are also convenient for demonstrating oral hygiene procedures to the parents.



H **E 1-9. A,** One method of positioning a child for an oral examination in a small, private consultation area. The dental assistant is nearby to record findings. **B,** If space allows three people to sit in a row, this method may be used to make it easier for the dental assistant to hear the findings dictated by the dentist. The dental assistant also helps restrain the child's legs.

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The positions of the dentist, parent, child, and dental assistant during the examination at the dental chair are illustrated in Fig. 1-10. The dental assistant is seated higher to permit good visibility and to better anticipate the dentist's needs. The assistant is also in a good position to hear and record the dentist's findings. The parent and the dental assistant restrain the child's arms and legs. The child's head is positioned in the bend of the parent's arm. The dentist establishes a chairside position so that not only the dentist's hands but also the lower arms and abdomen may be available for support of the child's head if necessary.



FIi. **1-10.** Oral examination of a very young child in the dental operatory.

The infant oral examination may often be performed by careful direct observation and digital palpation. The dentist may need only good lighting for visibility and gauze for drying or debriding tissues. Sometimes a tongue depressor and a soft-bristled toothbrush are also useful. At other times, as previously mentioned, the dentist will want the complete operatory available. The examination should begin with a systematic and gentle digital exploration of the soft tissues without any instruments. The child may find this gentle palpation soothing, especially when alveolar ridges in teething areas are massaged. The digital examination may help relax the child and encourage less resistance. If hand instruments are needed, the dentist must be sure to have a stable finger rest before inserting an instrument into the child's mouth.

Although there is little or no effective communication between the dentist and patient, the child realizes at the conclusion of the examination that nothing "bad" happened. The child also realizes that the procedure was permitted by the parents, who remained and actually helped with the examination. The child will not hold a lasting grudge against anyone, and the experience will not have a detrimental effect on future behavior as a dental patient. On the contrary, our experiences suggest that such early examinations followed by regular recall examinations often contribute to the youngsters' becoming excellent dental patients without fear at very young ages. These children's chances for enjoying excellent oral health throughout life are also enhanced.

DETECTION.OF SUBSTANCE ABUSE

It is within the scope of pediatric dentistry to be concerned with life-threatening habits and illnesses such as alcoholism and drug addiction that may occur in the older child.

Rosenbaum[7, 8] has reported that abusers in the teen years and younger are as common as adult addicts. Drug abuse problems interact directly with the dental care of a patient. Obtaining and maintaining a satisfactory history is important. The office health questionnaire, as presented in this chapter, must be worded to allow the patient or parent to give some indication of a drug problem. It is often difficult to detect addiction from casual observation. Therefore input from the patient giving an indication of addiction is needed. At subsequent visits, the dentist must also consider changes in the general health history as well as answers to specific questions.

It is also important to know if the patient is taking drugs at the time of the dental visit because there could be an interaction with drugs that may be given in the dental office, for example, nitrous oxide. If the

patient is under the influence of an abused substance, dental treatment should be postponed until a time when the patient is not "high."

Symptoms of substance abuse may include depression, feelings of inadequacy, frustration, helplessness, immaturity, self-alienation, poor object relations, and major deficiencies in ego structure and functioning. Heavy drug users tend to have poor impulse control. Hygiene in general and oral hygiene specifically are frequently neglected by those with a drug problem. In addition, because a patient is taking drugs that affect normal thought processes, the pain from untreated dental conditions may be masked. This combination of factors results in a patient with very little dental interest who is practicing unsatisfactory prevention. The result is increased oral disease.

Identification of substance abusers is difficult, even for an experienced observer. There are specific clues, however, that can be sought when one is attempting to draw general conclusions. Abrupt changes in behavior are common, as are signs of depression and moodiness. Interest in the opposite sex often decreases. Without any apparent consumption of alcohol, a drug-addicted person can appear intoxicated. There may be a desperate need for money, as well as a loss of appetite and loss of weight. The presence of scars along veins could be indicative of drug injection. Addicts frequently wear long-sleeved shirts, regard-less of the weather, in an effort to cover identifying scars.

In a group of addicted patients the following traits have been observed: deviant social background, school dropout, prostitution, broken relationships, inability to retain a job, low self-esteem, and impulsive behavior. Histories of hepatitis, poor diet, craving for sweets, vene real disease, and blackouts were reported.

In the past when many dentists thought about substance abuse, they envisioned the use of marijuana, alcohol, or amphetamines. Substances abused by children and adults today cover a much wider spectrum; they include solvents, inhalants, narcotics, stimulants, sedatives, hallucinogens, tranquilizers, and tobacco, perhaps the most addictive of all.

Hillis[9] reports that drug and alcohol abuse has changed from a personal adult problem to a medical, social, economic, and political issue. She raises this question:

> Ten years ago would you have expected to visit a third -grade class in your community and find little boys in Cub Scout uniforms and little girls in jumpers learning about drugs that even adults cannot handle? This scene is both terrifying and encouraging: terrifying in that drugs are such a problem that even third graders must be prepared to deal with them, and encouraging in that we finally are fighting back to prevent drugs from taking any more of our children.

MacDonald' reports that experimentation is a normal adolescent learning tool, but when combined with normal adolescent curiosity and fearlessness, it may be dangerous. Tobacco smoking is an example of a common teenage experiment. By their senior year, approximately one fourth, or 27.5%, of one class studied smoked daily.

ETIOLOGIC FACTORS IN SUBSTANCE ABUSE

Drug abuse in young people can be traced to many causes, the most important of which is considered to be rebellion against parents and society. Other factors may include a need to forget the pressures of daily living, a desire for pleasure, and a need to conform to the group with which young people want to be associated. Through drugs young people obtain a momentary feeling of independence and power because they have disobeyed the rules of their parents and society. The satisfaction gained through rebelling against parents can give adolescents a reinforcing motive for persisting in drug abuse.

Children of wealthy parents are increasingly recognized as a high-risk group for the development of such traits as narcissism, poor impulse control, poor tolerance of frustration, depression, and poor coping ability. Therefore it is not surprising that a large number of children within this group use drugs to cope with frustrations, boredom, anxiety, and depression.

Pallikkathayil and Tweed describe young drug users, especially those who are extensively involved in drug use, as less conforming than their peers, more tolerant of deviant behavior, and more likely to have a history of delinquent behavior before beginning to use drugs." In general, drug users have been found to be less interested in formal education, less involved in organized activities such as athletics, and less likely to have well-defined goals than youngsters who do not use drugs. Adolescents who use drugs heavily have been described as manifesting more psychologic problems than nonusers do. Significantly higher percentages of nonusers of drugs reported close relation-ships with their parents. Drug users are more often found to experience broken homes or the loss of a parent.

SPECIFIC SUBSTANCES AND FREQUENCY OF USE

Amphetamines are often used to ward off sleepiness, curb appetite, and relieve depression. Repeated consumption of these drugs results in the development of tolerance.

Benson'[2] has reported on the course and outcome of drug abuse in relation to medical and social conditions in select groups of young drug abusers in Sweden. Abuse of cannabis predominated 2 to 1 over

abuse of heavier drugs. In addition, emergency department data show that the use of the amphetamine derivative MDMA (ecstasy) in conjunction with marijuana increased from 8 cases in 1990 to 796 cases in 1999 (National Institute on Drug Abuse, http://www.drugabuse.gov). Black"' found that glue sniffing among adolescents was increasing in the United Kingdom, where it had rarely been reported before 1970. It has been a problem longer than that in the United States, where 7% to 12% of high school pupils reportedly have tried it at least once, with about 4% sniffing regularly. In the United Kingdom glue sniffing is now common among young people in inner-city areas, and it is suggested that these impoverished youngsters substitute it for alcohol. Glue sniffers are young adolescents, more often boys (although girls are increasingly represented), whose families have most of the marks of inner-city deprivation, such as poverty, overcrowding, unemployment, and delinquency. Most of these youth do not live with both of their natural parents. A small amount of glue is squeezed onto a rag or into a small bag and inhaled. The effect is sometimes enhanced by encasing the head in a larger plastic bag. Mild intoxication is produced and lasts for about 30 minutes.

Coulehan and others'[4] described the prevalence of gasoline sniffing and lead toxicity in Navaho Indian adolescents. Gasoline sniffing is the deliberate deep inhalation of gasoline vapors to achieve an altered mental state. Fifteen to 20 breaths may be sufficient to produce euphoria, ataxia, and disorientation lasting 5 or 6 hours. More massive exposure to these volatile hydrocarbons can lead to acute central nervous system depression and loss of consciousness, coma, and death. Adolescents who sniff gasoline tend to have other behavioral problems as well, including failure of school courses, suspension from school, and a record of being arrested. They are also more likely to use other drugs.

Inhalation abuse of various toxic agents continues to be a significant health problem among the younger members of our society. King, Smialek, and Troutman'' reported on the sudden death of adolescents resulting from the inhalation of typewriter correction fluid. A report describes four cases of sudden death in adolescents associated with recreational sniffing of typewriter correction fluid. The solvents used in most of these fluids are 1,1,1-trichloroethane and trichloroethylene, which are known to induce potentially fatal arrhythmias. Sniffing typewriter correction fluid poses a significant and underappreciated danger to the lives of these young abusers. Typewriter correction fluid, marketed as Liquid Paper, Wite-Out, and Sno-Paque, and commonly referred to as "white-out," is a readily available, inexpensive agent now being used by teenagers to obtain a rapid "high." The observation of dried white or colored chalky residue on the hands or face could be a clue to the presence of these volatile chemicals in the body.

Since the early 1970s there has been a great resurgence in the use of all forms of smokeless tobacco in the United States. Sales of smokeless tobacco have increased about 11% each year, with an estimated 22 million users in the United States.

Current advertising implies that smokeless tobacco habits are innocuous and safe; however, there is mounting evidence that the use of smokeless tobacco has the potential for causing cancer of the oral cavity, pharynx, larynx, and esophagus. A review of the medical and dental literature has identified many cases of cancer directly associated with smokeless tobacco use. Smokeless tobacco can produce significant effects on the soft and hard tissues of the mouth, including bad breath, discolored teeth and restorations, excessive wear, abrasion of the incisal and occlusal surfaces of the teeth, decreased ability to taste and smell, gingival recession, advanced periodontal destruction of the hard and soft tissues, leukoedema and erythema of the soft tissues, leukoplakia, and cancer. Smokeless tobacco can also pose a health hazard in certain medically compromised persons because its use results in increased blood pressure.

Suppose the dentist identifies a person who needs help. What can be done? Unless the dentist is exceptionally well qualified to handle problems of addiction, the answer is direct or indirect referral to a treatment center. If the person expresses a need, the dentist may directly inform that person or the parents about agencies in the area that provide assistance. However, addicts may react defensively, even with hostility, if a direct approach is used.

As with any problem related to general or dental health, preventive efforts must begin with the young. Children at a very young age need to be helped to develop a positive self-image, a sense of self-worth, and a separate identity.

SUICIDAL TENDENCIES IN CHILDREN AND ADOLESCENTS

During the examination of the child the pediatric dentist should be alert to signs and symptoms of suicidal tendencies. How prevalent is suicide in the young child and adolescent? According to Dr. Pamela Cantor, who spoke at the thirty-ninth annual meeting of the American Academy of Pediatric Dentistry (1986), suicide is now an epidemic, with the rate having increased 125% in the 30-year period from 1950 to 1980.°' There is

one adolescent suicide in the United States every 90 minutes and one attempted suicide every minute. The notion to kill oneself may be entrenched in early childhood and influenced by the society in which we live. There is evidence from many sources that suicide is second only to accidents as a cause of death among teenagers.

In 1980, 6000 teenagers killed themselves and an estimated 400,000 adolescents attempted suicide. MacDonald[17] has credited Dr. Perihan Rosenthal, head of child and adolescent psychiatry at Beth Israel Hospital in Newark, Ohio, with identifying the phenomenon of child suicide in 1979. Since then she has seen 30 suicidal children under 6 years of age. The youngest diagnosed was 2 1/2 years of age. Children under 13 years of age really do not have a firm grasp of what death means. The young child is still caught up in thinking that death can be partial or reversible. Young children will attempt suicide with the means available to them—jumping out of a window, running into traffic, or jumping in front of a car.

Suicidal tendencies follow a pattern and back-ground that can be observed by the astute professional or parent. According to the American Academy of Child Psychiatry, an estimated 10% of children in the United States suffer depression before 12 years of age. Depression in young people has been clearly linked to suicidal tendencies. It is important to be alert to the signs of suicidal tendencies, which are most often observed in the following individuals: one who is a female and a firstborn; one who shows depression with the addition of a second child; one who is very dependent on the mother but would be likely to experience parental conflicts during adolescence; one residing in a household in which there are struggles, conflicts, or divorce; one from a household in which the father is away from home a lot and, when present, is often overly critical; and one who feels that "the world would be better off without me." In addition, such tendencies may be seen in a girl who spends almost all of her time with a boyfriend and expects him to fill the role of an absent father; if the boyfriend rejects her, suicide or attempted suicide is frequent. Among those who are unable to derive satisfaction from living, who grow up too fast, who have low self-esteem and are bored with life, and who "keep things inside" and will not ask for help, suicide is also a problem. Some of the direct and indirect causes of suicide in children and adolescents according to Dr. Cantor are related to violence in the media, availability of guns, decreased employment opportunities for young people, alcohol and drug habits resulting in depression, and societal attitudes that teens are an inconvenience.' It should be recognized that the pediatric dentist and the orthodontist are in a unique position to recognize early warning signs of adolescent suicide. Loochtan and Cole's surveyed 1000 practicing orthodontists and 54 department chairs of postdoctoral programs. Of those surveyed, 50% had had at least one patient attempt suicide and 25% had had a young patient actually commit suicide.

INFECTION CONTROL IN THE DENTAL OFFICE

The dental team is exposed to a wide variety of microorganisms in the saliva and blood of their patients. These may include hepatitis B virus, herpes simplex viruses, cytomegalovirus, measles virus, mumps virus, chickenpox virus, human immunodeficiency virus type 1, *Mycobacterium tuberculosis,* streptococci, staphylococci, and numerous others that can infect the respiratory tract. Because it is impossible to identify all of those patients who may harbor dangerous microorganisms in their mouths, it is necessary to use standard precautions and practice infection control procedures routinely to avoid spread of disease. The following infection control procedures as described by Miller and Palenik[19] are based on those recommended for dentistry by the American Dental Association and by the Centers for Disease Control and Prevention (CDC) in the Public Health Service of the U.S. Department of Health and Human Services:

- Always obtain a thorough medical history, as discussed previously in this chapter, and include questions about medications, current illnesses, hepatitis, unintentional weight loss, lymphadenopathy, oral soft tissue lesions, or other infections.
- Clean all reusable instruments in an ultrasonic cleaner or washer/disinfector. Wear heavy rubber gloves, mask, and protective clothing and eyewear to protect against puncture injuries and splashing.
- Sterilize all reusable instruments that penetrate or come into contact with oral tissues or that become contaminated with saliva or blood. Metal or heat-stable instruments should be sterilized in a steam autoclave, a dry heat oven, or an unsaturated chemical vapor sterilizer. Heat-sensitive items may require up to 10 hours' exposure time for sterilization in a liquid chemical agent approved by the U.S. Food and Drug Administration as a disinfectant/sterilant, followed by rinsing with sterile water. High-level disinfection may be accomplished by submersion in the disinfectant/sterilant chemical for the exposure time recommended on the product label, followed by rinsing with water.
- Monitor the use and functioning of the sterilizer by using spore tests routinely (e.g., weekly for most dental offices). Chemical indicators may be used on the outside of packs.

- Insist that gloves be worn during all patient treatment procedures and when touching items contaminated with blood or saliva during operatory cleanup.
- Wear a surgical mask and protective eyewear when splatter or an aerosol of the patient's saliva or blood may be generated during treatment.
- Wear a protective gown or uniform when skin or clothing is likely to be soiled with blood or saliva. This attire should be changed at least daily or when visibly soiled.
- Use protective covers such as clear plastic wrap or paper backed with an impervious coating to protect operatory surfaces that are difficult to disinfect (e.g., light handles or x-ray unit heads).
- Use rubber dams whenever possible, along with high-speed evacuation, to reduce splatter and aerosols.
- Require vaccination against hepatitis B for all members of the dental team (dentists, hygienists, assistants, laboratory technicians). Blood tests should be given after vaccination to confirm response to the vaccine.
- Perform hand washing between patient contacts (after removing gloves) and after touching objects that may be contaminated with blood or saliva. An antimicrobial surgical hand scrub should be performed before gloving for surgical procedures. Hand washing with an antimicrobial or a plain, mild, nonirritating liquid soap is appropriate for examinations and nonsurgical procedures.
- Perform waste disposal functions in a way that protects those who must handle the waste. Use puncture-resistant containers marked with a biohazard label to dispose of needles, scalpel blades, and other sharp instruments immediately after use at chairside. To prevent needle stick injuries, needles should not be recapped by hand, bent, or broken before disposal. Nonsharp, regulated waste (e.g., cotton rolls or gauze pads saturated with blood or saliva) must be placed in sealed, sturdy, impervious bags to prevent leakage and marked as a biohazard through labeling or color coding. Liquid wastes may be poured down a drain connected to a sanitary sewer system.
- Place biopsy specimens in a sturdy container with a tight lid to prevent leaking during transport. Care should be taken not to contaminate the outside of the container.
- Clean and then disinfect contaminated operatory surfaces between patients. Wipe down the surfaces with absorbent toweling and disinfect with a 1:10 dilution of household bleach (for nonmetal surfaces) or with an Environmental Protection Agency–registered chemical germicide (e.g., an iodophor or phenolic).
- Use heat sterilization for all handpieces and air-water syringe tips before reuse. Water-cooled handpieces and the syringe should be activated and flushed with water for 3 minutes at the beginning of the day and for 30 seconds after completing care on each patient. They should be cleaned and sterilized according to the manufacturer's directions.
- Clean and disinfect impressions and intraoral appliances before handling, adjusting, or sending them to a dental laboratory They should also be cleaned and disinfected when they are received from the dental laboratory and before placement in the patient's mouth. It is important to consult with the manufacturer of the dental material regarding disinfection procedures. In addition, the most recent Centers for Disease Control Guidelines for Infection Control in Dental Health Care Settings—2003 update and revise previous guidelines and consolidate recommendations from other relevant CDC guidelines as well as those of other major infection control organizations.`° The document contains a review of the scientific evidence regarding dental infection control issues as well as consensus, evidence-based recommendations.

BIOFILM

A recent development in infection control involves microbial biofilm production inside the waterlines of dental units. The American Dental Association has stated that the microbiologic quality of dental unit water needs to be improved, and it has set a goal for dental unit manufacturers to make available dental units that will deliver treatment water with no more than 200 colony-forming units per milliliter (cfu/ml). Although municipal water supplies commonly are found to have an average of 5 to 50 cfu/ml, studies in the United States have shown the concentration of microorganisms in dental unit water to be from 20,000 to 500,000 cfu/ml. Currently, the options for improving dental unit water quality include use of separate water containers with periodic disinfection of the waterlines, use of in-line microbial filters, chemical or ultraviolet treatment of the water, and use of sterilizable water delivery systems. Research and development is ongoing in this area to verify the effectiveness of current approaches and to investigate new systems that may improve water quality.

EMERGENCY DENTAL TREATMENT

All too often a patient's initial dental appointment is prompted by an emergency situation. The diagnostic procedures necessary for an emergency dental appointment have already been outlined in this chapter. However, the emergency appointment tends to focus on

and resolve a single problem or a single set of related problems rather than provide a comprehensive oral diagnosis and management plan for the patient. Once the emergency problem is under control, the dentist should offer comprehensive services to the patient or parents.

The remainder of this book presents information for dentists and dental students to augment their diagnostic and management skills in providing oral health care services to children and adolescents during both emergency and preplanned dental visits.

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CHAPTER 7

Child Abuse and Neglect

ROBERTA A. HIBBARD

BRIAN J. SANDERS

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CHAPTER OUTLINE

IS IT CHILD ABUSE?
- Physical Abuse
- Sexual Abuse
- Neglect
- Emotional Abuse
- Factitious Disorder by Proxy

LEGAL REQUIREMENTS
WHO IS ABUSED?
IDENTIFICATION OF POSSIBLE CHILD ABUSE
- Physical Indicators
- Behavioral Indicators

EVALUATION
- Communication with the Patient
- Physical Examination

MANAGEMENT: DOCUMENTATION AND REPORTING
- Treatment
- Documentation
- Reporting
- Parental Concerns
- Obligation of the Dentist

Child abuse and neglect affect millions of children in the United States each year. Health care and dental professionals are in unique positions to identify the possibly abused child and must be knowledgeable in the recognition, documentation, treatment, and reporting of suspected child abuse cases. To appropriately intervene, professionals must be willing to consider abuse or neglect as a possibility—if it is not considered, it cannot be diagnosed. This chapter includes a discussion of the types of child maltreatment frequently encountered, the clinical presentation and management of such issues, and the documentation and reporting of suspected child abuse.

IS IT CHILD ABUSE?

Child abuse and neglect encompass a variety of experiences that are threatening or harmful to the child and are the result of acts of commission or omission on the part of a responsible caretaker. This includes physical or mental injury, sexual abuse, and negligent treatment or maltreatment of a child less than 18 years of age by a person responsible for the child's welfare. Many gray areas exist in the determination of threat or harm, and disagreements about the "abusive" nature of some experiences are common. No one individual is responsible for "deciding" what is abuse or neglect. Identification, treatment, and intervention are the tasks of professionals from multidisciplinary backgrounds working together to provide care and evaluation in the best interests of the child.

Maltreatment is not always willful; that is, the harm or injury inflicted is not always the intent of the act. Anger expressed actively or passively against the child is often unplanned, but nonetheless can result in significant injury or death. Education and prevention efforts may teach parents to redirect their actions and explore more appropriate discipline techniques and ways to manage anger or frustration.

PHYSICAL ABUSE

Physical abuse is often the most easily recognized form of child abuse. The battered child syndrome was initially described by Kempe et al in 1962 and elaborated further by Kempe and Helfer in 1972 as the clinical picture of physical trauma or failure to thrive in which the explanation of injury was not consistent with the severity and type of injury observed.[12] These injuries are inflicted and not accidental; some result from punishment that is inappropriate for the child's age, condition, or level of development. Some result from a parent's frustration and lack of control in acting out anger. Physical abuse is usually recognized by the pattern of injury and/or its inconsistency with the history related. Bruises, welts, fractures, burns, and lacerations are commonly inflicted physical injuries. Approximately 50% of physical abuse results in facial and head injuries that could be recognized by the dentist; 25% of physical abuse injuries occur in or around the mouth.

SEXUAL ABUSE

Sexual abuse and *sexual misuse* are frequently interchanged terms that denote any sexually stimulating activity that is inappropriate for the child's age, level of cognitive development, or role within the family. Many definitions incorporate the desire for sexual gratification on the part of one of the participants. In the spectrum of child sex play, sexual experimentation, and parent-child physical-sexual contact, it may be difficult to distinguish normal behavior from lustful intrusion. Sexually abusive acts may range from exhibitionism or kissing to fondling, intercourse, pornography, or rape. Trauma to the mouth may result from sexual contact.

NEGLECT

Inattention to the basic needs of a child, such as food, clothing, shelter, medical care, education, and supervision, may constitute neglect. While physical abuse tends to be episodic, neglect tends to be chronic. Determination of neglect also depends on the child's age and level of development as it may relate to periods of time without supervision, the parents' whereabouts, parental intention, and responsibilities of the child when the child is not supervised or not attending school. The American Academy of Pediatric Dentistry defines dental neglect as "willful failure of parent or guardian to seek and follow through with treatment necessary to ensure a level of oral health essential for adequate function and freedom from pain and infection."' Level of medical and dental care, adequate nutrition, and adequate food and clothing must be considered in light of cultural and religious differences, poverty, community requirements and standards, and the impact of such neglect on the physical well-being of the child.

EMOTIONAL ABUSE

Emotional abuse has been a concern for many years, but definitions and standards for identifying such abuse have been extremely difficult to establish. It is often difficult to demonstrate the direct or causal link between emotional and verbal abuse, and harm to the child. Such harm is usually seen as abnormal behaviors or mental health problems that are multifactorial in origin. Emotional and verbal abuse involve interactions or lack of interactions on the part of the caretaker that inflict damage on the child's personality, emotional well-being, or development. It must be an identifiable

act or omission that has demonstrable harm to the child and generally occurs in various ways over a prolonged period. Continuous isolation, rejection, degradation, terrorization, corruption, exploitation, or denial of affection are examples of behaviors that frequently have damaging effects on the child.

FACTITIOUS DISORDER BY PROXY

Perhaps the most difficult form of child maltreatment to identify and treat is a factitious disorder—factitious disorder by proxy, Munchausen syndrome by proxy, or pediatric condition falsification. These are conditions in which the perpetrator (usually the mother) relates a fictitious history, produces false signs or symptoms, and fabricates illnesses in the child that result in extensive medical evaluations, testing, and often prolonged hospitalizations. The fabrication may be deliberate to gain medical attention, the result of parental psychosis, or simply fraudulent to obtain money or services. Because health care providers are often dependent on the parental history of the child's illness, it takes some time for the practitioner to realize the inconsistencies and possibly fabricated or exaggerated nature of the complaints. These children present with persistent and recurrent illnesses that cannot be explained, signs and symptoms that do not make sense clinically, and problems that are rare, unusual, or bizarre. The bizarre nature of many of these cases makes them almost unbelievable to professionals involved, and an unbelieving social and legal system has considerable difficulty protecting a child.

LEGAL REQUIREMENTS

Every state has legal statutes requiring that suspected child abuse or neglect be reported to authorities. Statutes vary somewhat from state to state regarding detailed definitions of child abuse and neglect, but all states mandate that health care providers (including dentists) report child abuse or neglect when it is suspected. It is important to emphasize that one is required to report suspicions of child abuse and one need not have proof. It is the job of social and legal authorities to determine whether abuse has occurred and what intervention is legally necessary.

WHO IS ABUSED?

Children from all walks of life may be victims of child abuse or neglect—no age, race, gender, or socioeconomic level is spared. Statistics on child abuse reflect only those cases known or suspected, and all studies struggle with the component of the unknown. Approximately 50% to 65% of child maltreatment encompasses neglect and 25% involves physical abuse; sexual abuse and emotional abuse account for the majority of the remaining cases. Children who are victims of one form of maltreatment often are maltreated in other ways as well.

Sociodemographic characteristics of maltreated children vary somewhat by type of abuse or neglect. The average age of identification of maltreatment victims is 7. 4 years; 49% are male; and 68% are white, 21% are black, and 11% belong to other ethnic groups. Females are slightly overrepresented as abuse victims because sexual abuse is more prevalent among females. The youngest children (infants to 2 years) tend to be neglected most often and sexually or emotionally abused least often. Older children (12 to 17 years) are the least neglected, but the most sexually and emotion-ally abused; they are physically abused slightly more than average. Family characteristics overrepresented among families of maltreated children and therefore considered risk factors include female head of family, receipt of public assistance and thus lower income, presence of more children in the home, and presence of spousal abuse, drug or alcohol abuse, or significant health or economic stresses. Risk factors play a role, but ultimately every child is a potential victim.

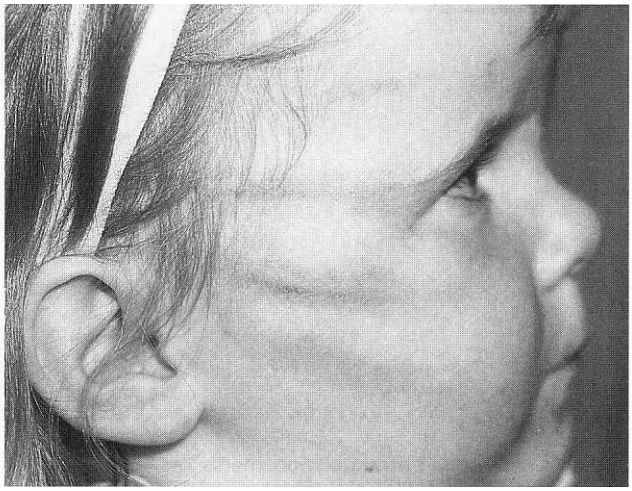
IDENTIFICATION OF POSSIBLE CHILD ABUSE

As stated earlier, child abuse and neglect are not identified if they are not considered as a diagnostic possibility. One must be willing to consider the diagnosis of abuse to make the diagnosis. A number of characteristics of the child, parent, or story given to explain the child's condition may lead a professional to suspect child maltreatment.

Indicators of child abuse and neglect are those signs or symptoms that should raise one's suspicions of the possibility of child maltreatment. The presence of such indicators does not "prove" maltreatment. Many of the signs or symptoms are nonspecific and may be present for a variety of reasons—child abuse is only one of those reasons. Indicators of abuse and neglect often depend on the child's age and developmental level and vary with the child's experiences and resiliency.

PHYSICAL INDICATORS

Situations raising the strongest suspicions and the most easily recognized maltreatment cases are those in which the pattern of injury is not consistent with the account explaining it. For example, a 3-month-old (nonambulatory) child is not going to sustain a spiral femur fracture from crawling. A bruise in the shape of a handprint on the cheek does not result from a fall down the stairs (Fig. 2-1). Accounts from two or more individuals (such as parents or a parent and child) that conflict with each other or that change over time are also very suspicious. Any significant injury that is reportedly "unwitnessed" should raise concerns of possible abuse.



f E. **2-1.** Bruise in the shape of a handprint on the cheek. *(From Hobbs CJ and Wynne JM: Physical signs of child abuse: a colour atlas, ed 2, WB Saunders [Harcourt Publishers Limited], London, 2001.)*



F **I G 2-2.** Intentional cigarette burn. *(From Hobbs CJ and Wynne JM: Physical* signs *of child abuse: a colour atlas, ed 2, WB Saunders [Harcourt Publishers Limited], London, 2001.)*

Physical indicators of child maltreatment tend to be somewhat more objective but must also be considered with the history. Unexplained bruises or welts in places not routinely subject to the child's rough-and-tumble lifestyle are those that become suspicious. Shin bruises and forehead bumps are expected in toddlers; bruises in the small of the back or torso are not. Unexplained injuries on the face, mouth, or lips; bruises clustering to form regular patterns or reflecting the shape of an article used to inflict the injury; and scattered significant bruises on different surface areas at various stages of healing are all very suspicious. Similarly, unexplained fractures of the head, multiple fractures in varying stages of healing, injuries to growth centers in the bone, and fractures in children younger than 2 years of age should raise concerns. Such injuries can be unintentional, but a clear explanation must be sought. Skull fractures may result from a fall from table to floor, but accompanying severe retinal hemorrhages or subdural hematoma with brain injury makes that explanation untenable. Burns are another form of recognizable child abuse; intentional cigarette (Fig. 2-2) and immersion burns should be readily distinguishable from accidental splash burns.

BEHAVIORAL INDICATORS

Significant behavioral changes often linked to child maltreatment include withdrawal, depression, poor school performance, regression in developmentally appropriate behavior, acting out, clinginess, and somatic complaints. Young maltreated children may show inappropriate affection toward others or may be extremely wary and distant in social interactions. Many children demonstrate affection toward an abusive parent; this should not be construed as evidence against maltreatment. Children who are afraid to go home, are frightened by their parents, or report injury by caretakers should be taken seriously. Extensive lists can be found describing behavioral indicators of possible maltreatment. These should be considered in light of the child's entire clinical history and presentation and not in isolation. When good explanations for such behaviors are not found, maltreatment is an appropriate consideration.

Behavioral indicators also may be present in caretakers. Lack of concern or inappropriately high levels of concern in relation to the severity of the child's injury are not unusual observations. Parents may be defensive and hostile when questioned or may refuse hospitalization and testing for the child. The explanation for the injury may be inconsistent with the pattern or the child's abilities, or the explanation may change when the perpetrator realizes that the first story is not believed. Poor judgment, jealousy or extreme protectiveness, child abandonment, violent behavior, or erratic behavior (which suggests drug or alcohol use, or psychiatric illness) are other clues to possible maltreatment.

Other indications of possible child abuse or neglect include a delay in seeking medical care for obvious injury, repeated ingestions of harmful substances, repeated hospitalizations, doctor or emergency department shopping, or excessive use of medical care for an apparently well child. Children whose basic needs for medical and dental care, food, clothing, shelter, or education are not being met may be victims of neglect.

EVALUATION

Trauma to the orofacial structures is a frequent manifestation of child abuse. Studies have indicated that the incidence is as high as 50% in child physical abuse.

The dentist who suspects child abuse or neglect needs to complete a thorough dental and general physical examination. Because abusive parents do not always show the same caution when visiting the dentist as when visiting the physician, the dental practitioner may be the first person to identify the abused child. Therefore the dentist must learn to recognize an abused child and make the appropriate referral.

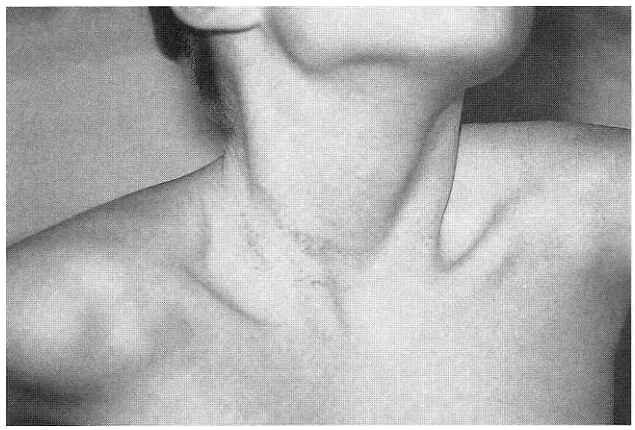
Any evaluation requires a medical history and physical examination. The combination of information is what influences or creates the suspicion of possible child maltreatment. The history should be a complete dental and medical history. Details regarding any trauma should be complete and obtained separately from more than one source (i.e., parent and child) if possible. Open-ended questions should be used; those with a yes-no response must be avoided. Details should include who witnessed the injury and who was with the child when it occurred, where the child and supervising adults were, and what exactly happened. Questions should include how and when the incident occurred. A description of present and past injuries, as well as the child's developmental abilities, may be helpful. Once enough information is obtained that the dentist is suspicious of child abuse or neglect, detailed questioning should be suspended.

COMMUNICATION WITH THE PATIENT

Professionals who are identifying and reporting suspected child maltreatment will have to talk to children in most circumstances to clarify a possible suspicion. They should not, however, be conducting investigative interviews of children to learn all the details or sort out the truthfulness of comments. A suggested guideline is the following: if based on your knowledge and experience you have reason to believe the child may have been abused or neglected, report it. Further detailed interviewing by a noninvestigating professional is neither necessary nor appropriate; that is the job of child protective service agencies. If, however, the child is talking and wants to disclose more, it is appropriate to listen and provide support.

PHYSICAL EXAMINATION

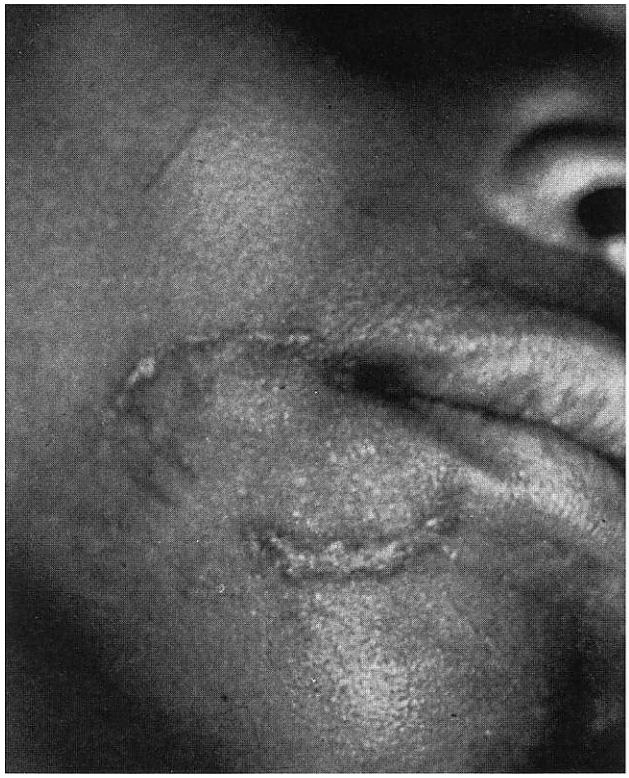
The examination of the patient by a dentist should include the entire body that is exposed without undressing the child. The examination begins before the patient is even back in the operatory. Observe the patient's posture, gait, and clothing. The dental staff should be trained in recognizing abuse and neglect so that they may alert the dentist of their suspicions. Inappropriate dress may be an indication of neglect and/or abuse. For example, a child who appears with a long-sleeved shirt in the middle of the hot summer may be dressed in this manner to cover old injuries. The child's behavior may also be inappropriate. A lack of spontaneous smiling and avoidance of eye contact may be indicators, as is being overly watchful and vigilant.



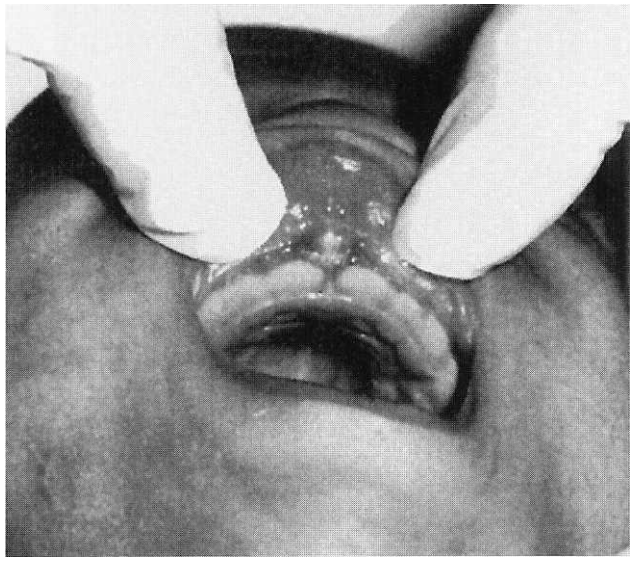
f **I G 2-3.** Attempted strangulation marks on the neck of an adolescent. *(From Hobbs CJ and Wynne JM: Physical signs of child abuse: a colour atlas, ed 2, WB Saunders [Harcourt Publishers Limited], London, 2001.)*

The dentist should start the examination at the top, beginning with the hair and scalp, and systematically work down. Alopecia without an underlying medical cause may be an indicator of malnutrition or hair pulling. Continue the examination by looking at the nose and nasal septum. A deviated septum or clotted blood may be an indicator of previous trauma. Look for any periorbital ecchymosis, ptosis, and deviated or unequal pupils, which indicate significant facial trauma. In cases of suspected head trauma, a neurologic assessment (history and physical) by the dentist can help so that the child receives the immediate attention that is necessary. If any question exists of possible abnormality, the child should be referred to a pediatrician or neurosurgeon familiar with child abuse as soon as possible.

Any bruise in the shape of an object, such as a belt, looped cord, handprint, or hanger, should alert the practitioner to inflicted trauma. The varying color of bruises should be particularly noted to identify the several stages of resolution that would indicate ongoing trauma. The neck should be examined for evidence of rope burns or bruises (Fig. 2-3) that may indicate attempted strangulation. Severe shaking can result in large bruises on the back of the neck that may also indicate brain damage. Physical trauma to the child's chest or ribs may elicit a painful response from the child if a lifting motion is used to slide the child up to the top of the dental chair during the examination. The presence of adult bite marks (Fig. 2-4) may be a sign of physical abuse, sexual abuse, or neglect. They may also be



[I G **2-4.** The presence of an adult bite mark may be a sign of physical or sexual abuse or neglect. *(Courtesy American Society of Dentistry for Children.)*



E I G **2-5.** Torn frenulum from blunt force trauma to mouth. Upon further investigation, this child was found to have 17 fractures.

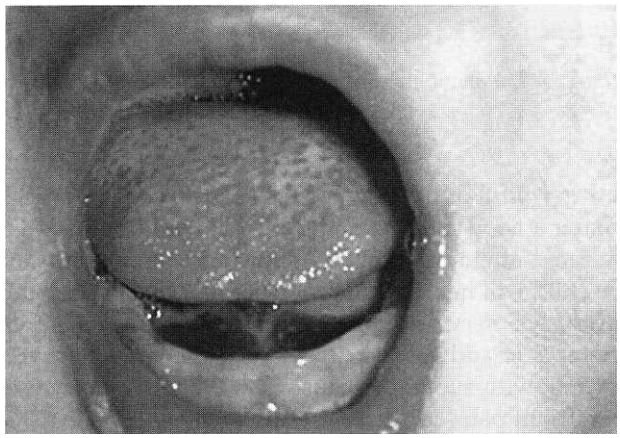


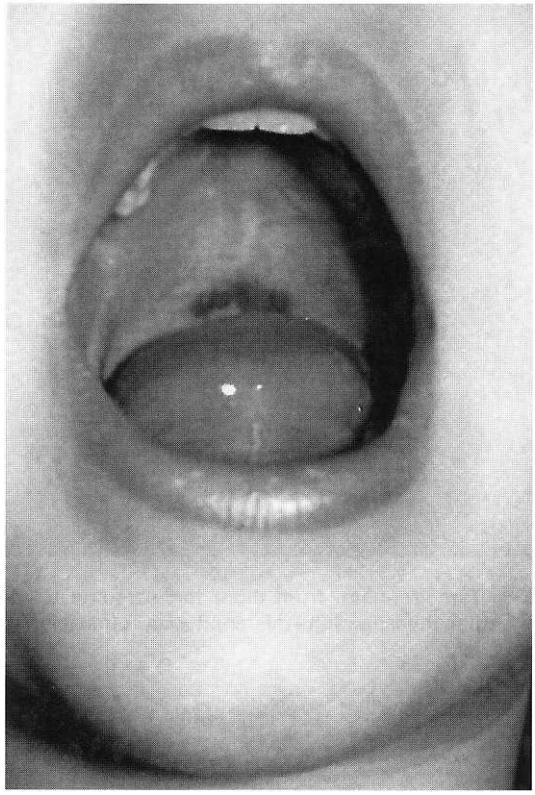
FIG **2-6.** Sublingual hemorrhage in an infant with signs of genital and abdominal trauma.

helpful in identifying the abuser. They need to be clearly documented and photographs taken, if possible, at the time they are first observed, since they tend to fade rapidly. A forensic dentist or bite mark expert should be consulted as soon as possible when adult bite marks are suspected. Any visible patterns of injury should be photographed if possible; most law enforcement agencies will dispatch a photographer if requested in child abuse cases.

On completion of the general physical examination, the dentist should examine the teeth and supporting structures. Note any missing teeth or previously traumatized teeth (avulsions, luxations, intrusions, or fractures) and pay especially close attention to any soft tissue injuries. The mandible should be examined for any deviation on opening, range of motion, trismus, and occlusion at rest. The maxilla should also be examined for any mobility indicating a facial fracture. Bleeding under the tongue may indicate a fracture of the body of the mandible.

Note the condition of the maxillary labial frenum and the lower lingual frenum. A torn maxillary frenum on a child who is too young to walk indicates possible trauma to the mouth from a slap, fist blow, or forced feeding. A torn lingual frenum could be indicative of sexual abuse or forced feeding (Figs. 2-5 and 2-6).

Bruising or petechia of the soft and hard palate may indicate sexual abuse in the form of oral penetration (Fig. 2-7). If any evidence of infection or ulceration is noted, specimens should be cultured for evidence of a sexually transmitted disease, such as gonorrhea, syphilis, or venereal warts. The child who presents with extensive, untreated dental caries, untreated infection, or dental pain may be considered a victim of physical neglect because the parents are not attending to the child's basic medical needs. Before filing a report, the dentist should determine if the failure to provide dental



f 11. **2-7.** Palatal hemorrhage from oral-genital contact.

care is willful or due to a lack of awareness or finances. Taking a good medical and dental history and making repeated attempts to obtain appropriate treatment for the child will help sort out these issues. A call to a child protective service agency is indicated if repeated attempts to address the cause of the dental neglect are not met with success, because dental neglect can cause significant pain, discomfort, and possible disability.

MANAGEMENT: DOCUMENTATION AND REPORTING

Clinical and medicolegal management of suspected child abuse and neglect involve several basic steps: medical and dental management, documentation (including photographs), and reporting.

As health care professionals, dentists should be especially sensitive to the need for protecting children from abuse or neglect. They must, of course, treat dental injuries. It is also important for dentists to know that they are legally mandated to report suspected child abuse or neglect. Reporting is initiated simply with a telephone call to the appropriate child protective service agency. The telephone call initiates a response by appropriately trained professionals, but the dentist should follow the call with a written report. Dentists are mandated to report based on "reasonable suspicion," and they are not responsible for any further investigation. Cases of abuse and neglect are often resolved without litigation, and the reporting dentist will not necessarily be called to testify even if the case goes to court.

TREATMENT

Any medical or dental treatment that is indicated by the child's condition should be provided. A referral for a complete pediatric history taking and physical examination will assist in identifying and treating other possibly associated conditions (failure to thrive, anemia, and so on). Medical evaluation should include assessment for medical conditions that can mimic or be confused with child abuse. A young child (younger than 18 months to 2 years) who has suffered a fracture should be examined by means of a skeletal survey to detect other fractures; children with bruising need to be examined for possible blood clotting disorders.

DOCUMENTATION

All data collected in the medical history and physical examination must be documented in a complete and objective manner. Pertinent positive and negative findings should be included. Actual comments and behaviors should be recorded; opinions about those behaviors should be avoided. For visible injuries, photographs should be taken if possible. The child's name and the date of the photograph should be included in the picture. Most law enforcement officials will take photo-graphs if requested to do so when suspected child abuse is reported. When suspected maltreatment is reported to authorities, the time, date, and method of reporting (phone or written report) should be documented in the medical and dental record.

REPORTING

The dentist is obligated by law to report suspected findings of child abuse to the appropriate authorities, that is, child protective service agencies and/or law enforcement officials. Failure to do so may result in the filing of civil or criminal charges against the dentist. With increased public awareness and inclusion of courses on child abuse in the dental curriculum, ignorance of the laws of child abuse is not an acceptable excuse.

PARENTAL CONCERNS

In most situations, parents should be told of the concerns about possible child abuse or neglect and the legal requirement to report it to local authorities. Health care professionals should not make any accusations about who may have caused the harm. Simple statements such as the following should be used: "Based on my training, I am concerned that this injury could not have happened this way. Because of this, I am required by law to make a report to Child Protective Services."

In those situations in which a child is suspected to have been significantly harmed in the home, in which the parent is expected to be violent, or in which possible retribution against the child for having told is a concern, it may be more prudent to contact authorities and have them present to protect the child before parents are told. The dental professional has no legal obligation to inform parents that abuse or neglect is suspected or will be reported; some situations may best be handled by not telling them at the time a report is filed.

The major concern must be for the welfare of the patient, and any concerns about losing a patient from a practice should be secondary. Individuals are protected from civil and criminal liability if the report is made in good faith. When the dentist's action is presented to parents as motivated by concern for the child and by an attitude of "let' s figure out what is going on," many parents are eventually appreciative and will continue to seek support and care from the reporting professional.

There should be no reluctance on the part of the dentist to report suspected child abuse because of concern that it will require a great deal of time. In most cases after the initial report has been filed no further involvement is necessary on the part of the dentist, and few cases require a court appearance. It is possible to report suspected child abuse anonymously, but it is preferred that you give your name so that the agency can contact you if there are any further questions.

OBLIGATION OF THE DENTIST

The privileged quality of communication between the caretakers or the patient and the practitioner is not grounds for excluding evidence in a judicial proceeding resulting from a report or for failing to make a report as required by law. Strict confidentiality of records is maintained. Reports and any other information obtained in reference to a report are confidential and available only to persons authorized to examine them by the juvenile code. Some state statutes stipulate that a mandated reporter who fails to make a report when abuse or neglect is suspected may be liable for proximate damages caused by the failure to report. Criminal liability is another possibility.

The health care professional must remember that it is suspicions of child abuse or neglect that must be reported; proof is not required. It is the responsibility of child protective service agencies and law enforcement officials to investigate suspicions and determine if intervention is necessary. The health care professional can assist by providing as much information as possible through communication and coordination. Investigating professionals cannot do their jobs if the health care professional does not share detailed information regarding why the suspicions exist. Health care professionals unhappy with the outcome of system intervention (e.g., that nothing was done) are usually those who would not or did not provide the information available that would assist authorities in making the best informed decisions. If the health care professional feels that a bad decision is being made, a follow-up phone call to the assigned caseworker or caseworker's super-visor to clarify concerns and interventions is appropriate. Many misperceptions exist about what interventions are possible legally. Communication and coordination can improve everyone's knowledge and understanding about a child's needs and what can be done to meet them.

Child abuse and neglect are identifiable in the dental office. Knowledgeable practitioners must be able and willing to identify, document, and report suspicions of child maltreatment. Awareness of local child protective community resources and professionals can facilitate interaction with the legal system and improve the ability to appropriately protect abused or neglected children.

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CHAPTER 3

Nonpharmacologic Management of Children's Behaviors

GERALD Z. WRIGHT

انتشارات
شایان نمودار

CHAPTER OUTLINE

PEDIATRIC DENTAL PATIENTS
- Variables Influencing Children's Dental Behaviors
  - *Maternal anxiety*
  - *Medical history*
  - *Awareness of dental problem*
- Classifying Children's Cooperative Behavior
- The Functional Inquiry

PARENTS OF PEDIATRIC PATIENTS

STRATEGIES OF THE DENTAL TEAM
- Preappointment Behavior Modification
- Fundamentals of Behavior Management
  - *The positive approach*
  - *The team attitude*
  - *Organization*
  - *Truthfulness*
  - *Tolerance*
  - *Flexibility*
- Communicating with Children
  - *Establishment of communication*
  - *Establishment of the communicator*
  - *Message clarity*
  - *Voice control*
  - *Multisensory communication*
  - *Problem ownership Active listening*
  - *Appropriate responses*
- Behavior Shaping
- Retraining
- Aversive Conditioning
- Practical Considerations
- *Scheduling*
  - *Parent-child separation*
  - *Tangible reinforcements*
  - *Dental attire*
- Limitations

The foundation of practicing dentistry for children is the ability to guide them through their dental experiences. In the short term, this ability is a prerequisite to providing for their immediate dental needs. More long-lasting beneficial effects also can result when the seeds for future dental health are planted early in life. A professional goal is to promote positive dental atti- tudes and improve the dental health of society. Logically, children are keys to the future.

A major difference between the treatment of children and the treatment of adults is the relationship. Treating adults generally involves a one-to-one relationship, that is, a dentist-patient relationship. Treating a child, however, usually relies on a one-to-two relationship among dentist, pediatric patient, and parents or guardians. Fig. 3-1, which illustrates this relationship, is known as the *pediatric dentistry treatment triangle*. Recently, society has been centered in the triangle. Management methods acceptable to society and the litigiousness of society have been factors influencing treatment modalities. Note that the child is at the apex of the triangle and is the focus of attention of both the family and the dental team. Although mothers' attitudes have been shown to significantly affect their children's behaviors in the dental office, the roles of families have been changing, and the entire family environment must be considered. Because changes are constantly occurring within each personality, one must remember that there is an ever-changing, dynamic relationship among the corners of the triangle—the child, the family, and the dental team. The arrows placed on the lines of communication also remind us that communication is reciprocal.

The importance of this unifying concept will become evident as management techniques are described.

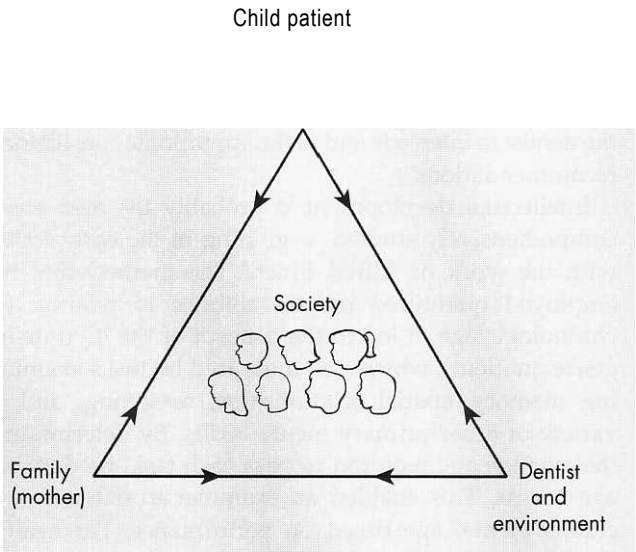


FIG . **3-1.** The pediatric treatment triangle illustrates basic relationships in pediatric dentistry.

However, this concept also serves as a means for organizing the present chapter, whose goal is to discuss the nonpharmacologic approaches to managing children's behavior in dentistry.

PEDIATRIC DENTAL PATIENTS

Child development involves the study of all areas of human development from conception through young adulthood. It involves more than physical growth, which often implies only an increase in size. Development implies a sequential unfolding that may involve changes in size, shape, function, structure, or skill.

Over the years, numerous child development theories have evolved. Summarizing them, Alpern stated that the most important general principle concerning development is that human development is not unitary.' He contended that there were several relatively important aspects of child development and that no single aspect could be used to assess development. He cautioned about relating to children through a single developmental label and suggested that a basic appreciation of child development knowledge could be helpful to the dentist.

Early child development study linked changes to specific chronologic ages. The initial work gathered age norms for physiologic developmental tasks. Eventually personality description principles also evolved. One of the pioneering and most notable groups, headed by Arnold Gesell, was at Yale University. Some of the typical personality characteristics related to specific chronologic ages that have relevance to dentistry are listed in Box 3-1. They can help guide management strategies. For example, if the dentist knows the limitation of a 2-year-old's vocabulary, it becomes apparent that communication must occur through the sense of touch and voice modulation rather than through the spoken word. Recognizing also the close symbiotic relationship with parents, dentists generally try to keep the parent-child pair intact.

Relating personality characteristics to chronologic ages has led to some interesting labeling. For example, a noncompliant 2-year-old often is referred to as in the stage of the *terrible twos*. Dentists sometimes refer to such children as being in the *precooperative stage*. Unfortunately, this has led in some instances to using the age of the child, rather than the child's ability, as a reason for noncompliance.

The broad area of physical development involves changes that occur in children's size, strength, motor coordination, functioning of body systems, and so forth. Thus the child's total physical growth and efficiency from the moment of conception until adulthood is termed *physical development*. Because a child's physical development is relatively independent of other major areas of development, subareas of physical development

BOX 3-1 Age-Related Psychosocial Traits and Skills for 2- to 5-Year-Old Children*

TWO YEARS

Geared to gross motor skills, such as running and jumping
Likes to see and touch
Very attached to parent
Plays alone; rarely shares
Has limited vocabulary; shows early sentence formation
Becoming interested in self-help skills

THREE YEARS

Less egocentric; likes to please
Has very active imagination; likes stories
Remains closely attached to parent

FOUR YEARS

Tries to impose powers
Participates in small social groups
Reaches out—expansive period
Shows many independent self-help skills
Knows "thank you" and "please"

FIVE YEARS

Undergoes a period of consolidation; deliberate
Takes pride in possessions
Relinquishes comfort objects, such as a blanket or thumb
Plays cooperatively with peers

*Based on the work of Dr. A. Gesell.

TABLE 3-1 Average Age and Age Range of Selected Physical Developmental Milestones

DEVELOPMENTAL TASK	AVERAGE AGE	NORMAL RANGE
Focuses on light	2 wk	1-4 wk
Lies on stomach, lifts chin	3 wk	1-10 wk
Birth weight doubles	6 mo	5-7 mo
Rolls from back to stomach	7 mo	5½ -11 mo
Sits alone	7 mo	6-11 mo
Stands with support	10 mo	7½ -14 mo
Stands alone	13½ mo	9-18 mo
Walks alone	14 mo	10-20 mo
Bowel control attained	18 mo	1-2½ yr
First menstruates	12 yr, 9 mo	10-17 yr

have to be relatively independent. A child's co-ordination cannot be judged by physical size, nor is physical strength related to dental development.

Relating physical changes to specific chronologic ages led to the establishment of developmental milestones that became a means of assessing individual children. Some of the classic developmental milestones are shown in Table 3-1. From these milestones, ranging from infancy through early childhood, two pieces of information are derived: (1) the average age at which a child acquires particular skills and (2) the normal range of ages at which a skill is acquired. A general principle is that, the earlier a skill emerges, the narrower is the range. On the other hand, developmental tasks tend to occur with wider ranges of normality as age increases. For the dentist, this holds practical importance. For example, consider the task of teaching children how to floss their teeth. Because the ability to floss occurs later in life (9 to 12 years of age), there is a wide performance range. Knowing the general developmental principle reminds the clinician to consider the ability or readiness of the individual to perform a given task.

Another area that has received great attention from psychologists is the socialization of children. As with physical development, age-specific skills have been derived for social development; these take into account both interpersonal relationships and independent functioning skills. Some of the key personality characteristics already have been identified in Box 3-1.

An important process for dentists is the child's growth toward independent functioning. For their survival, infants are dependent on others to clothe, feed, and nurture them. As children grow and their ability to care for themselves improves, they gain social independence. Recognizing that the change from functional dependency to functional autonomy is a normal process in social development can assist the dentist. Many young children, who lack digital dexterity, want to brush their own teeth. Parents, on the other hand, understand the lack of digital skills and often insist on attending to their children's oral health care. Appreciating that this tug-of-war is a normal part of social maturation allows the dentist to intercede and make appropriate conciliatory recommendations.

Intellectual development is probably the area most comprehensively studied, beginning in the early 1900s with the work of Alfred Binet.[2] The method that he employed quantified mental abilities in relation to chronologic age. It led to the concept of the IQ (intelligence quotient), which was measured by tasks examining memory, spatial relationships, reasoning, and a variety of other primary mental skills. By determining the average age required to pass each task, he derived age norms. This enabled an examiner to determine a child's mental age based on performance. For health care providers, viewing children in terms of their mental ages can be a helpful approach.

TABLE 3-2 Intelligence Quotient (IQ) Classification Guide

IQ RANGE	GENERAL CLASSIFICATION	PERCENT OF POPULATION
140 and up	Very superior	0.5
120-139	Superior	2.5
110-119	High average	14
90-109	Average	60
80-89	Low average	12
70-79	Borderline impaired	7
69 and below	Mentally retarded	4

The IQ formula used by Binet was the following:

$$IQ = \frac{\text{Mental age}}{\text{Chronologic age}} \times 100$$

Thus a child who performs tasks accomplished by a 10-year-old and who has the chronologic age of 8 years has an IQ of 125 (10/8 x 100 = 125). Quantification of intelligence has led to various classification guides. Table 3-2 shows the IQ ranges, the descriptive general terms, and the relative populations fitting into each category. An understanding of the levels of mental retardation can be helpful in treating young patients. Table 3-3 shows four IQ ranges and provides practical descriptions of the various groups.

Since the time of Binet, more than 300 tests have been devised to measure intellectual development. The best known and best standardized of these tests are the Wechsler intelligence scales as included in the text by Anastasi.[3] These are individualized tests, as opposed to group tests, and separate forms of the test are available for preschoolers (Wechsler Preschool and Primary Scale of Intelligence, or WPPSI), children (Wechsler Intelligence Scale for Children-Revised, or WISC-R), and adults (Wechsler Adult Intelligence Scale, or WAIS). The IQ scores that result indicate probable current levels of intellectual functioning for that person and have a high degree of reliability and validity.

At best, scores from all such tests, even the highly standardized ones, are only estimates and may not be a fair appraisal for a given child on a given day. The younger the child, the less reliable the test scores. The more delayed the child, the less reliable the test scores. The more an individual's cultural and educational opportunities differ from the norm, the less reliable and valid that test is for that individual. However, the information from psychometric assessments can alert a dentist to the possibility that a child may need an individualized approach in the dental office, as well as elsewhere.

TABLE 3-3 American Association of Mental Deficiency Classification Schema

IQ RANGE*	CLASSIFICATION	PRACTICAL DESCRIPTION
52-68	Mild	Can be educated in special classes to gain elementary school–level academic skills
36-51	Moderate	Can be trained to perform self-help skills and to achieve sheltered workshop employment
20-35	Severe	Abilities typically limited to simple language usage and the mastery of basic self-help skills
19 and below	Profound	Consistent custodial care required

IQ, Intelligence quotient.
*Based on Stanford-Binet intelligence test scores.

The environment is such a crucial factor in the development of a human being that it can be discussed as an independent factor only on the theoretic level. The fact that each child appears to have a characteristic temperament from his or her earliest age has been suggested by Sigmund Freud and by Gesell and I1g.[4 5] In recent years, however, some psychiatrists and psychologists have emphasized the influence of the child's early environment when discussing the origin of human personality. Whether personality is developed by "nature" (genetic influence) or "nurture" (environmental influence) is an age-old, unresolved question. However, if these two influences are in harmony, healthy development of the child can be expected; if they are dissonant, behavioral problems are almost sure to ensue.

VARIABLES INFLUENCING CHILDREN'S DENTAL BEHAVIORS

A fearful or anxious child who anticipates an unpleasant visit is more likely to have such an experience than is a child who has a low level of fear or anxiety. Anxiety or fearfulness affects a child's behavior and, to a large extent, determines the success of a dental appointment. The various schools of psychologic thought agree that anxiety is a personality trait, but they have various opinions concerning the origin of this trait. By the same token, dentistry has had some difficulty identifying the stimuli that lead to misbehavior in the dental office, although several variables in children's backgrounds have been related to it.

Maternal Anxiety. In past years, it has been customary for mothers, more often than fathers, to accompany children to the dental office. For this reason, the effect of maternal anxiety on children's dental visits has received considerable attention in the dental literature. Although in the near future this topic might be renamed "parental anx ieties," at the moment such a change would be premature.

With few exceptions, most investigations indicate a significant correlation between maternal anxiety and a child's cooperative behavior at the first dental visit. High anxiety on the part of parents tends to affect their children's behavior negatively. Although the scientific data reveal that children of all ages can be affected by their mothers' anxieties, the effect is greatest with those under 4 years of age. This might be anticipated because of the child-parent symbiosis that begins in infancy and gradually diminishes.

Medical History. The importance of the medical history, a highly complex variable, has been debated over the years. Some pediatric dentists believe that it has little bearing on a child's behavior in the dental office, whereas others consider it a major factor affecting children's cooperation. There is general agreement, however, that children who view medical experiences positively are more likely to be cooperative with the dentist. The emotional quality of past visits rather than the number of visits is significant.

Pain experienced during previous medical visits is another consideration in a child's medical history. The pain may have been moderate or intense, real or imaginary. Nonetheless, parental beliefs about past medical pain also are significantly correlated with their children's cooperative behavior in the dental environment. Study also has shown that previous surgical experiences adversely influence behavior at the first dental visit, but this was not the case in subsequent visits.

Awareness of Dental Problem. Some children may approach their dentist knowing that they have a dental problem. The problem may be as serious as a chronic dental abscess or as simple as extrinsic staining of the dentition. However, there is a tendency toward negative behavior at the first dental visit when the child believes that a dental problem exists. Such behavior may be the result of apprehension transmitted to the child by a parent. The significance of the variable provides the dentist with a good reason for educating the parents about the value of arranging a child's first dental visit before there are any dental problems.

CLASSIFYING CHILDREN'S COOPERATIVE BEHAVIOR

Numerous systems have been developed for classifying the behavior of children in the dental environment. An understanding of them holds more than academic interest. The knowledge of these systems can be an asset to the dentist in several ways: it can assist in directing the management method, it can provide a means for systematically recording behaviors, and it can assist in evaluating the validity of current research.

Wright's clinical classification places children in three categories":

- Cooperative
- Lacking in cooperative ability
- Potentially cooperative

During examination of a child, the cooperative behavior of the patient is taken into account because it is a key to rendering treatment. Most children seen in the dental office cooperate. Cooperative children are reasonably relaxed. They have minimal apprehension. They may be enthusiastic. They can be treated by a straightforward, behavior-shaping approach. When guidelines for behavior are established, they perform within the framework provided.

In contrast is the child lacking in cooperative ability. This category includes *very* young children with whom communication cannot be established and of whom comprehension cannot be expected. Because of their age, they lack cooperative abilities. Another group of children who lack cooperative ability is those with specific debilitating or disabling conditions. The severity of the child's condition prohibits cooperation in the usual manner. At times, special behavior management techniques are employed for these children. Although their treatment is accomplished, immediate major positive behavioral changes cannot be expected. Characteristically, the nomenclature applied to a potentially cooperative child is *behavior problem*. This type of behavior differs from that of children lacking cooperative ability because these children *have* the capability to perform cooperatively. This is an important distinction. When a child is characterized as potentially cooperative, clinical judgment is that the child's behavior can be modified; that is, the child can become cooperative.

The dental literature is filled with descriptions of potentially cooperative patients. Moreover, the adverse reactions have been given specific labels, such as uncontrolled, defiant, timid, tense-cooperative, and whining. Dentists often use these labels because they convey, in as few words as possible, the essence of the clinical problem.

Another system, which has been used in behavioral science research, is referred to as the Frankl Behavioral Rating Scale.' The scale divides observed behavior into four categories, ranging from definitely positive to definitely negative. Following is a description of the scale:

Rating 1: Definitely Negative. Refusal of treatment, forceful crying, fearfulness, or any other overt evidence of extreme negativism.

Rating 2: Negative. Reluctance to accept treatment, uncooperativeness, some evidence of negative attitude but not pronounced (sullen, withdrawn).

Rating 3: Positive. Acceptance of treatment; cautious behavior at times; willingness to comply with the dentist, at times with reservation, but patient follows the dentist's directions cooperatively.

Rating 4: Definitely Positive. Good rapport with the dentist, interest in the dental procedures, laughter and enjoyment.

Although the Frankl method of classification has been a popular research tool, it also lends itself to a shorthand form that can be used for recording children's behavior in the dental office. One can identify those children displaying a positive cooperative behavior by jotting down + or ++. Conversely, uncooperative behavior can be noted by – or - -. A shortcoming of this method is that the scale does not communicate sufficient clinical information regarding uncooperative children. If a child is judged as –, the user of this classification system must qualify as well as categorize the reaction. By recording "–, tearful," a better description of the clinical problem is made.

THE FUNCTIONAL INQUIRY

Before the dentist treats children, medical and dental histories are usually obtained. However, a functional inquiry, from a behavioral viewpoint, also should be conducted. During the inquiry, there are two primary goals: (1) to learn about patient and parental concerns and (2) to gather information enabling a reliable estimate of the cooperative ability of the child. Coupling the findings from the functional inquiry with the clinical experience, the dentist is in a much better position to meet the patient's needs and to apply appropriate management strategies to treat individual pediatric patients than by simply proceeding inadequately informed.

Usually, functional inquiries are conducted in two ways: (1) by a paper and pencil questionnaire completed by the parent and (2) by direct interview of child and parent. In some offices one method may predominate, whereas in others both techniques are used. Each method has specific merits.

Written questionnaires can be important tools for gaining information, because probing questions can uncover critical facts about child-rearing practices in the home, a child's school experiences, or the patient's developmental status. Four questions with clinical relevance that can be added to history forms are listed in Box 3-2.

The responses to these questions, originally from behavioral science research, can alert the clinician to a potential behavioral problem. If a parent responds negatively to more than one question, the chance of encountering a behavior problem rises considerably. An unlimited list of questions could, of course, be prepared for paper and pencil questionnaires, but from the dentist's viewpoint, a lengthy list is impractical.

BOX 3-2 Clinically Relevant Questions That Can Be Added to History Forms

	(CIRCLE ONE)
How do you think your child has reacted to past medical procedures?	Very well Moderately well Moderately poorly Very poorly
How would you rate your own anxiety (fear, nervousness) at this moment?	High Moderately high Moderately low Low
Does your child think there is anything wrong with his or her teeth, such as a chipped tooth, decayed tooth, gum boil?	Yes No
How do you expect your child to react in the dental chair?	Very well Moderately well Moderately poorly Very poorly

Most practicing dentists recognize the merits of personal contact with parents. For the personal interview to serve as an efficient functional inquiry tool, a structured framework is necessary. The paper and pencil questionnaire is a starting point. It provides general information or clues and helps guide the personal interview. Consider the following question:

Do you consider your child to be (check one):

advanced in learning?
progressing normally? a
slow learner?

If the parent has indicated that the child is a slow learner, more factual information is necessary. A leading question in the personal interview might be, "What school does your child attend?" A child may attend a special education class or a special school. Knowledge of the child's class or special school can offer a clue about the functional level of the patient. There is no limit to the depth of the personal interview, but if it is to be efficient, questioning must be thoughtful. Other avenues that can be explored include rewards and consequences used in the home environment. These provide insight into the type of behavior management techniques that would be acceptable to a parent.

PARENTS OF PEDIATRIC PATIENTS

From the moment of their children's birth, parents shape children's behaviors by selective encouragement and discouragement of particular behaviors, by their disciplinary techniques, and by the amount of freedom they allow. In early years, at least, it is mainly from parents that children learn what they are supposed to do and what behavior is forbidden.

Because North America is a cultural mosaic, understanding parents' dental behaviors is not always easy. Dental attitudes may be deeply rooted. Differences are revealed in intercountry studies. For example, consider digital sucking, a habit with dental relevance. Examining schoolchildren in Sweden, Larsen found a 12% prevalence of the habit, whereas Golden recorded a 56% prevalence in Israeli children residing on a kibbutz. [8'"]

The socioeconomic status of parents also has some bearing on their acceptance of management methods. Fields, Machen, and Murphy had 67 parents view videotapes of children's treatments and rate their acceptability of the techniques.[10] Although most parents favored commonly used behavior-shaping methods, socioeconomic status correlated negatively with the use of general anesthesia. Those in the higher socioeconomic group frowned on the use of general anesthesia. The reduced approval may result from the fact that the higher social group was more enlightened about the associated risks.

Societal changes in the latter part of the twentieth century have created dynamics that can indirectly affect the behavior of children in dental offices. For example, a center of social policy reported that one in ten children lives in a household not headed by a parent. Often these children are brought to dental offices by caretakers or child care workers who are unfamiliar with the children's histories. Reconstituted or blended families, same-sex families, and single-parent families all present with their special sets of circumstances. Considering 218 children of teenaged mothers from birth to 5 years, Stier et al found them at greater risk for maltreatment than children of older mothers."

Generalizations have been made, such as that future-oriented, conscientious, middle-class parents tend to seek dental care readily and cooperatively, and that they accept brushing and dental checkups as a matter of course. These generalizations must be approached cautiously. The dental team must recognize the individuality of parents. Some parents from the middle or upper socioeconomic strata may be well meaning and realize the importance of dentistry yet fail to follow through. Sometimes these parents are too busy with their everyday lives to cooperate with oral hygiene programs or even to bring the child to the dental office. Generalizations do not always hold true.

Despite the acknowledged importance of the parental role in the pediatric treatment triangle and the necessity of gaining parental cooperation, it is only recently that the dental literature has provided dentists with advice for dealing with parents. Good information can be gleaned from three studies by Fields, Machen, and Murphy, Murphy, Fields, and Machen, and Lawrence et al in which parents were shown videotaped scenes of different management techniques.[10-12•"] Four of the techniques were tell-show-do, positive reinforcement, voice control, and the use of a mouth prop. The six other techniques included various types of restraint methods, sedation, and general anesthesia. The techniques in this latter group were judged unacceptable by parents in the first two studies and acceptable by parents participating in the third study. The methodologic difference in the third study was the verbal explanation given to parents before showing them the videotapes. A comparison of these studies points out the importance of communicating with parents. Parents of pediatric patients often require understanding and have to be led through their children's dental experiences. We live in a litigious society, and it is critical to develop rapport with parents.

Casamassimo and Wilson reported on a survey of diplomates of the American Board of Pediatric Dentistry.' Most diplomates indicated that there has been a change in parental demographics. There are more single parents, increased mobility, and more dual-income families. The findings from this report found that children's behaviors in dental offices were strongly related to parenting styles, preferences, and demands. Failure of parents to set limits on their children's behaviors was the main parental child-rearing problem. They also found that parental expectations often were unattainable.

Dentists in practice have to anticipate these types of problems and learn to deal with them. The diplomates suggested several methods of coping with parent's and children's behaviors; however, one recommendation, the improvement of communication, dwarfed all others in frequency. Communication can mean many things, but in this instance it refers to the dentist's getting his or her message across to the parents and having them working with the dentist.

STRATEGIES OF THE DENTAL TEAM

A primary objective during dental procedures is to lead children step by step so that they develop a positive attitude toward dentistry. Fortunately, most children progress easily and pleasantly through their dental visits without undue pressure on themselves or the dental team. These successes can be attributed to a number of factors, such as a child's confident personality,

a parent's proper preparation of the child for the appointment, or a dental team's excellent communicative skills. On the other hand, some children's dental office experiences cause anxiety and the beginning of a negative dental attitude. Sometimes these controllable but apprehensive children are managed without medication, provided that appropriate nonpharmacologic psychologic techniques are employed.

Because management techniques are used daily and come naturally to many persons, their importance is sometimes overlooked or they are taken for granted. This increases the potential for avoidable behavior problems. However, fully understanding and consciously implementing strategies can lead to recognizable improvements in child management skills. Although this section heightens awareness of various techniques commonly used in dental offices today, it should be regarded as only a start to the study of behavior management strategies.

PREAPPOINTMENT BEHAVIOR MODIFICATION

Psychologists have developed many techniques for modifying patients' behaviors by using the principles of learning theory. These techniques are called *behavior modification.* Usually they are thought about in conjunction with dentist-patient intraoperatory relationships. However, *preappointment behavior modification,* as it is used here, refers to anything that is said or done to positively influence the child's behavior before the child enters a dental operatory. The merit of this strategy is that it prepares the pediatric patient and eases the introduction to dentistry. It has received a great deal of attention because the first dental visit is crucial in the formation of the child's attitude toward dentistry. If the first visit is pleasant, it paves the road for future successes.

Several methods of preappointment behavior modification are recognized. Films or videotapes have been developed to provide a model for the young patient. The goal is to have the patient reproduce behavior exhibited by the model. On the day of the appointment, or perhaps at a previous visit, the new pediatric patient views the presentation.

Most modeling studies indicate that there is merit in introducing children to dentistry in this way, but not all studies show statistically improved cooperative behavior on the part of the children. The lack of replica-tion may be the result of differences in experimental design, dental teams, or videotapes or films. It suggests a necessity for careful videotape or film selection for office use.

Preappointment behavior modification can also be performed with live patient models such as siblings, other children, or parents. Many dentists allow young children into the operatory with parents to preview the dental experience. Because the observing child likely will be initiated into dental care with a dental examination, a parent's recall visit offers an excellent modeling opportunity. On these occasions many young children climb into the dental chair after their parents' appointments. These previews should be selected carefully. Young children are some-times frightened by loud noises, as from a high-speed handpiece.

The merits of modeling procedures, commonly using audiovisual or live models, are recognized by psychologists. Rimm and Masters summarized them as follows: (1) stimulation of new behaviors, (2) facilitation of behavior in a more appropriate manner, (3) disinhibition of inappropriate behavior due to fear, and (4) extinction of fears.'[s] These procedures offer the practicing dentist some interesting ways to modify children's behavior before their dental visit.

Another behavior modification method involves preappointment mailings. Precontact with the parent can provide directions for preparing the child for an initial dental visit and therefore can increase the likelihood of a successful first appointment. Precontact offers a practical approach for teaching parents of new pediatric dental patients how to prepare their children for a first dental visit. Beneficial effects were demonstrated in a controlled study by Wright, Alpern, and Leake.'' Children seemed better prepared by their mothers, and the dentist saw more cooperative pediatric patients. Almost all parents understood the letter's contents, acknowledged the dentist's thought-fulness, and welcomed the concern for the proper presentation to their children. A similar letter is offered in Fig. 3-2.

Dentists using preappointment mailings should be selective. Correspondence has run the gamut from the simplest welcoming letter to a bombardment of mailings. Numerous mailings cause a reversal in parental attitude. Overpreparation could confuse a parent or provoke anxiety.

FUNDAMENTALS OF BEHAVIOR MANAGEMENT

Behavior management involves the total dental health team. Indeed, many dental auxiliaries are invaluable when it comes to dealing with children. All of the personnel have a stake in guiding a child through the dental experience.

Over the years, behavior management has meant different things to different people. In 1895 McElroy wrote, "Although the operative dentistry may be perfect, the appointment is a failure if the child departs in tears."' This was the first mention in dental literature of measuring the success or failure of a child's appointment by anything other than technical proficiency. Pediatric dentistry has progressed since that time, and a

Dear Parent:

Children who have pleasant dental appointments when they are very young are likely to have a favorable outlook toward dental care throughout life. The first appointment is very important in this attitude formation. That is the reason I am writing to you.

At our first appointment we will examine your child's teeth and gums and take any necessary x-ray films. For most children this will be an interesting and even happy occasion. All the people on our staff enjoy children and know how to work with them, but you, parents, play an important role in getting children started with a good attitude toward dental care. One of the useful things that you can do is to be completely natural and easygoing when you tell your child about the appointment with the dentist. This approach enables children to view their dental visit as an opportunity to meet some new people who want to help them stay healthy.

Your cooperation is appreciated. Remember, good general health depends partly on the development of good habits, such as sensible eating, sleeping routines, and exercise. Dental health also depends on good habits, such as proper toothbrushing, regular dental visits, and a good diet. We will have a chance to further discuss these points during your child's appointment.

Sincerely,

f G. **3-2.** Letter currently used in clinical practice to assist parents in preparing children for first dental visit. *(Adapted from Wright GZ, Alpern GD, Leake JL: ASDCJ* Dent Child *40:265-271, 1973.)*

currently adopted definition is the following:

Behavior management is the means by which the dental health team effectively and efficiently performs treatment for a child and, at the same time, instills a positive dental attitude.

Effectively in this definition refers to providing high-quality dental care. Efficient treatment is a necessity in private practice today. Quadrant dentistry, or perhaps half-mouth dentistry, using auxiliary personnel is vital in delivering efficient service to children. Finally, the development of a pediatric patient's positive attitude is an integral part of this definition. In the past many practitioners have considered "getting the job done" to be behavior management. The current definition suggests a great deal more.

What has been omitted from the definition of behavior management also is interesting. There is no mention of any specific techniques or modalities of treatment. The definition allows the exercise of individuality. The challenge to the dentist is to satisfy the elements of the definition as frequently as possible and as safely as possible for each child in a dental practice.

Although various methods in managing pediatric dental patients have evolved over the years, certain practices and concepts remain fundamental to good behavior management. These are basic to establishing good dental team–pediatric patient relationships. These practices increase the chances for success when dealing with children. They should be considered inviolate. The following fundamentals of behavior management center on the attitude and integrity of the entire dental team.

Positive Approach. There is general agreement that the attitude or expectations of the dentist can affect the outcome of a dental appointment. The child will respond with the type of behavior expected. In essence, the child fulfills the dentist's prophecy. Thus positive statements increase the chances of success with children. They are more effective than thoughtless questions or remarks. To obtain success with children, it is important to anticipate success.

Team Attitude. Personality factors such as warmth and interest that can be conveyed without a spoken word are critical when dealing with children. A pleasant smile tells a child that an adult cares. Children respond best to a natural and friendly attitude. Often this can be conveyed immediately to the pediatric patient through a casual greeting. Children also can be made to feel comfortable in the dental office by the use of nicknames, which can be placed on a patient's record. Noting school accomplishments or extracurricular activities such as Cub Scouts, baseball, hockey, or other hobbies helps in initiating future conversations and demonstrates a friendly, caring attitude to a pediatric patient.

Organization. Plans in the dental office have many dimensions, beginning, for example, with the reception

area. Who summons the new patient? The dentist, the dental assistant, the dental hygienist, or the receptionist? If a child creates a disturbance in the reception area, who will deal with the problem? Each dental office must devise its own contingency plans, and the entire office staff must know in advance what is expected of them and what is to be done. Such plans are key features of many pediatric dental offices because they increase efficiency and contribute to successful dental staff–pediatric patient relationships. Also, a well-organized, written treatment plan must be available for the dental office team. Delays and indecisiveness can build apprehension in young patients.

Truthfulness. Unlike adults, most children see things as either "black" or "white." The shades between are difficult for them to discern. To youngsters the dental health team is either truthful or not. Because truthfulness is extremely important in building trust, it is a fundamental rule for dealing with children.

Tolerance. A seldom-discussed concept, tolerance level varies from person to person. It refers to the dentist's ability to rationally cope with misbehaviors while maintaining composure. Recognizing individual tolerance levels is especially important when dealing with children. As well as varying from person to person, tolerance levels fluctuate for a given individual. For example, an upsetting experience at home can affect the clinician's mood in the dental office. Some people are in a better frame of mind early in the morning, whereas the coping abilities of others improve as the day progresses. Thus afternoon people should instruct receptionists not to book children with behavior problems the first thing in the morning. Learning to recognize factors that overtax tolerance levels is another fundamental, since it prevents loss of self-control.

Flexibility. Because children are children, lacking in maturity, the dental team must be prepared to change its plans at times. A child may begin fretting or squirming in the dental chair after half an hour, and the proposed treatment may have to be shortened. On the other hand, a dentist may plan an indirect temporary pulp treatment, but because the child is difficult, the plan may have to be altered to complete treatment at a single session. Many dentists, following accepted four-handed dentistry practices, work at the 11 o'clock or 12 o'clock position. Treatment of small children may demand a change in operating position. Thus the dental team must be flexible as the situation demands.

COMMUNICATING WITH CHILDREN

Several effective communication techniques can be suggested. These key points are guidelines, not inflexible rules, for in the unpredictable world of dealing with children, one must always be prepared to improvise as the situation requires.

Establishment of Communication. Previous editions of this textbook have stated that the first objective in the successful management of the young child is to establish communication. It is generally agreed that involving a child in a conversation not only enables the dentist to learn about the patient but also may relax the youngster. There are many ways of initiating verbal communication, and the effectiveness of these approaches differs with the age of the child. Generally, verbal communication with younger children is best initiated with complimentary comments, followed by questions that elicit an answer other than *yes* or no.

Establishment of the Communicator. Members of the dental team must be aware of their roles when communicating with a pediatric patient. Generally the dental assistant talks with the child during the transfer from reception room to operatory and during preparation of the child in the dental chair. When the dentist arrives, the dental assistant usually takes a more passive role, since the child can listen to only one person at a time. It is important that communication occur from a single source. When both dentist and dental assistant provide directions, the result may be a response that is undesirable simply because the child becomes confused.

Message Clarity. Communication is a complex, multisensory process. It includes a transmitter, a medium, and a receiver. The dentist or dental health team is the transmitter, the spoken word frequently is the medium, and the pediatric patient is the receiver. The message must be understood in the same way by both the sender and the receiver. As Chambers indicates, there has to be a "fit" between the intended message and the understood one.'

Very often, to improve the clarity of messages to young patients, dentists use euphemisms to explain procedures. For pediatric dentists, euphemisms or word substitutes are like a second language. Examples of word substitutes that can be used to explain procedures to children are given in Box 3-3.

It is important to be careful in selecting words and phrases used to indoctrinate the new pediatric dental patient, because for the young child, language labels are the basis for many generalizations. The classic example is the language label for "doctor," which confuses many youngsters. This is known as *mediated generalization*. Eventually, as a result of experiences, the child learns that the "dentist doctor" is different from the "physician doctor" and that the physician's office and the dentist's office are different environments. The process of sorting out such differences is referred to as *discrimination*.

Voice Control. Throughout the dental literature, reference is made to *voice control*. It is difficult to describe this effective communicative technique using

BOX 3-3 Word Substitutes for Explaining Procedures to Children

DENTAL TERMINOLOGY	WORD SUBSTITUTES
rubber dam	rubber raincoat tooth
rubber dam clamp	button
rubber dam frame	coat rack
sealant	tooth paint
topical fluoride gel	cavity fighter
air syringe	wind gun
water syringe	water gun
suction	vacuum cleaner
alginate	pudding statues
study models	whistle
high speed	motorcycle
low speed	

the written word. Sudden and firm commands are used to get the child's attention or to stop the child from whatever is being done. Monotonous, soothing conversation is supposed to function like music set to a mood. In both cases what is heard is more important because the dentist is attempting to influence behavior directly, not through understanding.

Although dentists have always recognized the merit of properly employing voice control when children's behaviors have been disruptive, Greenbaum et al have given it scientific credence.'" Considering the use of loud commands as a punishment technique, they compared the effects of loud and normal voice commands given to 40 children with potential behavior problems. Their findings demonstrated that loud commands reduced disruptive behaviors.

The theory of Chambers is that voice control is most effective when used in conjunction with other communications.'$ A sudden command to "stop crying and pay attention" may be a necessary preliminary measure for future communication. The same message spoken in a foreign language probably would be equally effective in stopping disruptive patient behavior that is preventing communication. Used properly in correct situations, voice control is an effective management tool.

Multisensory Communication. In verbal communications, the focus is on *what* to say or *hour* it is said. However, nonverbal messages also can be sent to patients or received from them. Body contact can be a form of nonverbal communication. The dentist's simple act of placing a hand on a child's shoulder while sitting on a chairside stool conveys a feeling of warmth and friendship. Greenbaum et al found that this type of physical contact helped children to relax, especially those 7 to 10 years of age."

Eye contact is also important. The child who avoids it often is not fully prepared to cooperate. Apprehension can be conveyed without a spoken word. Detecting a rapid heartbeat or noticing beads of perspiration on the face are observations that alert the dentist to a child's nervousness. When the dentist talks to children, every effort should be made not to tower above them. Sitting and speaking at eye level allows for friendlier and less authoritative communications.

Problem Ownership. In difficult situations, dentists sometimes forget that they are dealing with children. They begin by sending "you" messages. For example, " You must sit still!" These are negative messages and only undermine the rapport between a pediatric patient and dentist. "You" messages carry the implication that the child is wrong. An alternative is to send "I" messages. These messages establish the focus of the problem, such as "I can't fix your teeth if you don't open your mouth wide." This is one of the techniques discussed by Wepman and Sonnenberg that is particularly well suited to increase the flow of information between the dentist and the pediatric patient.'-° These techniques are incorporated into the Parent Effectiveness Training Program, which was popularized in the early 1970s.

Active Listening. Listening also is important in the treatment of children. However, listening to the spoken words may be more important in dealing with the older child than it is in dealing with the younger child, for whom attention to nonverbal behavior often is more crucial. Active listening is the second step in encouraging the kind of genuine communication cited by Wepman and Sonnenberg.-° The patient is stimulated to express feelings, and the dentist does the same, as necessary processes in communication.

Appropriate Responses. Another principle in communicating with children is that "the response should be appropriate to the situation_" The appropriateness of the response depends primarily on the extent and nature of the relationship with the child, the age of the child, and evaluation of the motivation of the child's behavior. An inappropriate response would be a dentist's displaying extreme displeasure with an anxious young child on the first visit when there has been insufficient time to establish a good rapport. On the other hand, if a dentist has made inroads with a child, who then displays unacceptable behavior, a dentist may well express disapproval without losing personal control. The response is then appropriate.

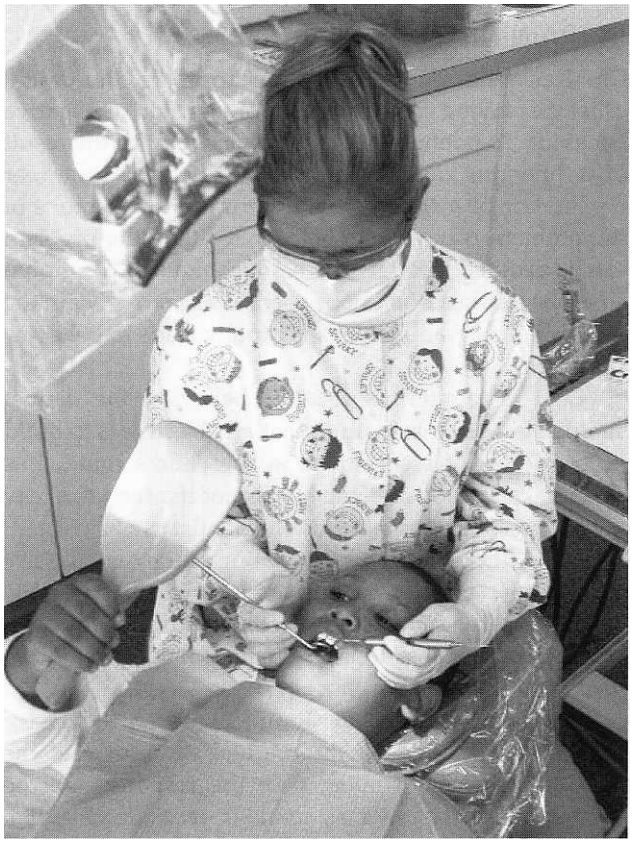
BEHAVIOR SHAPING

Behavior shaping is a common nonpharmacologic technique. It is a form of behavior modification; hence, it is based on the established principles of social learning. By definition, it is that procedure which very slowly

develops behavior by reinforcing successive approximations of the desired behavior until the desired behavior comes to be. Proponents of the theory hold that most behavior is learned and that learning is the establishment of a connection between a stimulus and a response. For this reason, it is sometimes called *stimulus-response (S-R) theory*.

When shaping behavior, the dental assistant or dentist is teaching a child how to behave. Young children are led through these procedures step by step. They have to be communicative and cooperative to absorb information that may be complex for them. The following is an outline for a behavior-shaping model:

1. State the general goal or task to the child at the outset.
2. Explain the necessity for the procedure. A child who understands the reason is more likely to cooperate.
3. Divide the explanation for the procedure. Children cannot always grasp the overall procedure with a single explanation; consequently, they have to be led through the procedure slowly.
4. Give all explanations at a child's level of understanding. Use euphemisms appropriately.
5. Use successive approximations. More than four decades ago, Addelston formalized a technique that encompasses several concepts from learning theory.[2'] It was called the *tell-show-do (TSD) techique*. Since its introduction in 1959, it has remained a cornerstone of behavior management. TSD is a series of successive approximations. It is a component of behavior shaping that should be routinely used by all members of the dental team who work with children. Dental assistants, dental hygienists, and dentists should demonstrate various instruments step by step before their application by telling, showing, and doing. When the dentist works intraorally, a pediatric patient should be shown as much of the procedure as possible. Only when the child has a view of the procedures being undertaken are successive approximations being performed properly (Fig. 3-3).
6. Reinforce appropriate behavior. Be as specific as possible, because specific reinforcement is more effective than a generalized approach. This advice is supported by the clinical research of Weinstein et al, who studied dentists' responses to children's behavior and found that immediate and specific reinforcements were most consistently followed by reductions in children's fear-related behaviors."
7. Disregard minor inappropriate behavior. Ignored minor misbehavior tends to extinguish itself when it is not reinforced.



f G **3-3.** Child views intraoral procedure with hand mirror. If mirror blocks light to oral cavity, smaller mirror is used. With fiberoptic handpieces, blockage of light is not a problem.

Behavior shaping is regarded as a learning model. A general rule about learning models is that *the most efficient learning models are those that follow the learning theory model most closely*. Deviations from the model create less efficiency in terms of learning. One way to improve consistency in this area is to record various clinical sessions with pediatric patients, using a tape recorder or videotape system, and then to review the tapes, keeping in mind the basics of the behavior-shaping learning model.

Although TSD is similar to behavior shaping, the two differ. As well as demanding the reinforcement of cooperative behavior, behavior shaping also includes the need to retrace steps if misbehavior occurs. For example, if a child is shown an instrument and looks away, the dentist must revert to the explanatory portion of the procedure. Behavior shaping requires that the "desired behavior" be observed along the way. If the dentist proceeds along the sequential steps and begins performing treatment when the desired behavior is not present, there is deviation from the learning model and a greater likelihood of increased misbehavior.

RETRAINING

Children who require retraining approach the dental office displaying considerable apprehension or negative behavior. The demonstrated behavior may be the result of a previous dental visit or the effect of improper parental or peer orientation. Determining the source of the problem is obviously helpful, for then the problem can be avoided through another technique or deemphasized, or a distraction can be used. These ploys begin the retraining program, which eventually leads to behavior shaping.

When encountering negative behavior, the dentist should always have in mind that an objective is to build a new series of associations in the child's mind. If a child's expectation of being hurt is not reinforced, a new set of expectations is learned. The dentist can be trusted! The child develops a new perception of the dental office and a new relationship to dentistry. Unacceptable behavior learned previously becomes extinguished. It is critical to remember that *the stimulus must be altered to elicit a change in the response*.

Individuals respond to stimuli to which they have been preconditioned. If the original stimulus and the new one are very similar, then the response will be similar. This is known as *stimulus generalization*. If a child has had an unpleasant experience in the dental office and then is taken to a different office where there is a different dentist and an entirely different staff and surroundings, the child still tends to generalize that an unpleasant event will occur in this new dental office. There are enough similar stimuli to produce this response. To offset the generalization, the dental team must demonstrate a "difference." This is one of the reasons why the use of nitrous oxide–oxygen sedation often works when retraining children. It offers a difference.

AVERSIVE CONDITIONING

The behavior modification method of aversive conditioning is also known as *hand-over-mouth exercise,* or by the acronym *HOME*. Its purpose is to gain the attention of a highly oppositional child so that communication can be established and cooperation obtained for a safe course of treatment. The technique fits the rules of learning theory: maladaptive acts (screaming, kicking) are linked to restraint (hand over mouth), and cooperative behavior is related to removal of the restriction and the use of positive reinforcement (praise).

It is important to stress that aversive conditioning is not used routinely but as a method of last resort, usually with children 3 to 6 years of age who have appropriate communicative abilities. Levitas emphasizes that for the very young, the immature, those with physical disabilities, or those who have mental or emotional disabilities, this behavioral approach is unacceptable.[3]

In recent years concern has been shown about the use of aversive conditioning. Some dentists have characterized the technique as punishment. Surveys of pedodontic diplomates in 1972 and 1981, however, demonstrated acceptance by a substantial portion of the profession. In both instances more than 80% of the pediatric dentists indicated that they used aversive therapy at times. Inquiring into acceptance of the technique by dental educators, Davis and Rombom obtained similar results; 83% of the respondents taught aversive conditioning in advanced training programs.' Some parents, too, have disapproved of the technique, and another concern has been a legal one. Bowers points out that acceptance of aversive conditioning by the dental profession is not an absolute assurance of the legality of the technique and that until a court rules on its legitimacy there remains some degree of uncertainty." He also contends that the dentist who uses aversive conditioning in accordance with the standards of the prudent dentist in the locale and who obtains the requisite consent to treatment should not fear liability for battery or malpractice.

Aversive conditioning can be a safe and effective method of managing a child with an extremely difficult behavior problem. However, any departure from the accepted application of aversive conditioning may expose the dentist to liability. Those dentists or dental students who contemplate using it should consult detailed writings beforehand. Hagan et al strongly suggest that dentists also should be aware of the accept-ability of the technique in their practice location.'"

PRACTICAL CONSIDERATIONS

Some procedural aspects of dental practice help to guide children through their dental experiences successfully. Like many of the techniques described previously, they have evolved over the years without experimental testing. Nevertheless, these practical considerations form an integral part of present-day dental practice. They also would be fruitful areas for future research.

Scheduling. Children are bundles of energy. Lacking the patience of adults, many children become restless and tired when faced with long delays in a reception area. This should be taken into account when designing an office schedule. A good general rule is that a child should not be kept waiting in the reception area and that every effort should be made to be on time.

Morning appointments have been suggested for children. It is a practice that has guided scheduling in many dental offices. The suggestion was based on the facts that children are more alert and the dental team is fresher in the morning. Many dentists also believe that

when age groups are kept together (preschoolers in the morning and older children in the afternoon), the peer group has a positive influence, with children serving as models for each other. Another advantage is that the dental office may run more smoothly with less psychologic change of pace.

Sometimes expediency rather than a realistic evaluation of a child's behavior may predispose the dentist toward morning appointments for preschool children. Frequently it is easier to persuade parents to take younger children out of nursery school or kindergarten than to arrange morning appointments for elementary or junior high school children. From a behavioral standpoint, other factors seem important when deciding the appointment time. Patient-related concerns include patient age, presence of a handicapping condition, and need for any sedation. The dentist's attitude also is important. Some dentists avoid seeing children with behavior problems first thing in the morning. A policy regarding scheduling should be formulated by the dentist, and scheduling should not be left to chance.

Another scheduling concern has been appointment length. Historically, writers have agreed that the nature of childhood precludes giving the sustained attention that may be required for long dental visits. Generally, a long visit is defined as any period in excess of half an hour. Most of these views were expressed more than 30 years ago, and appointments have increased in duration. Improved technology, the application of time and motion studies by efficiency experts, and the current trend toward quadrant or half-mouth dentistry have altered contemporary practices. Whether this affects children adversely remains a question for researchers. In Lenchner's study, the behavior of 43 children between 3 and $5^{1}/2$ years of age who were undergoing operative dentistry was examined in both short and long appointments.[27] Short appointments ranged from 16 to 30 minutes, whereas longer appointments were 48 to 125 minutes. The investigator found no significant difference in children's behavior during long dental appointments compared with shorter ones. On the other hand, Getz and Weinstein, studying videotapes of 36 children between 3 and 5 years of age who were undergoing restorative treatment, found the opposite.[28] The longer the restorative phase, the greater the likelihood of a stress-fear reaction. Thus current evidence on appointment duration is divided.

Parent-Child Separation. Excluding the parent from the operating room can contribute toward development of positive behavior on the part of the child. Over the years, Starkey has been one of the strongest advocates of separation of the child from the parent during treatment and has suggested that the policy of requiring the parent to remain in the reception room could be justified for many of the following reasons [29]:

- The parent often repeats orders, which creates an annoyance for both the dentist and the pediatric patient.
- The parent injects orders, becoming a barrier to development of rapport between the dentist and the child.
- The dentist is unable to use voice intonation in the presence of the parent because he or she may be offended.
- The child divides attention between the parent and dentist.
- The dentist divides attention between the parent and child.

There are other reasons for advocating a separation policy. Most dentists probably are more relaxed and comfortable when the parent remains in the reception area. Perhaps they then do not feel that they are "performing with an audience." As a consequence of their more relaxed manner, their actions are likely to have more positive effects on many children's behaviors.

When an office policy is formed, remember that many dentists who exclude parents from the operatory make exceptions. A parent can be a major asset in supporting and communicating with a disabled child, often providing important information and interpretation. Another important exception relates to age. Very young children (those who have not reached the age of understanding and full verbal communication) have a close symbiotic relationship with parents; consequently, they usually are accompanied by them. The age factor was studied by Frankl, Shiere, and Fogels, who investigated the effect of a parent's presence in the operatory? In their study the intact group (parent present with child) reacted more favorably than the separated group. It should be noted that children $3^{1}/2$ to 4 years of age appeared to benefit most from the parent's presence. Those older than 4 years demonstrated similar levels of response to dental care regardless of parental presence. Thus dentists who contemplate admitting only parents of infants and toddlers to the operatory might consider extending the age level somewhat.

The separation procedure warrants serious consideration. The dentist must develop an office policy, notify the office staff of it, and assume responsibility to train office personnel in reception room strategies. In this age of accountability, the dentist may also have to explain the policy to a parent. Establishment of the policy therefore should be based on a rationale that takes into account the benefits and drawbacks resulting from separation as well as the dentist's personal views.

Because some dentists become tense when parents are present and others enjoy having parents in the operatory, to some extent an office policy becomes an individual decision.

Tangible Reinforcements. Giving gifts to children has become a fact of commercial life in North America. There is general agreement on the merit of this practice in the dental office, for gift giving can serve as a reward. If the gift has a dental significance (such as a toothbrush kit), so much the better. In these situations the gift is also used as a reinforcement for dental health.

Various trinkets in a toy chest should be used as tokens of affection for children, not as bribes. Finn made the following distinction between rewards and bribes: "A bribe is promised to induce the behavior. A reward is recognition of good behavior after completion of the operation, without previously implied promise."[30] The gift-giving practice can have spectacular results. Many children who seem tense during operative procedures suddenly perk up on completion and scurry for a gift. These gifts provide a pleasant reminder of the appointment.

Dental Attire. Because fears are transferable from one situation to another, dentists have had some concern about wearing child-friendly apparel. If a child had poor experiences previously (whether with a physician or a barber), it is possible that these fears could be generalized to the dental situation, since a uniform can be common to all. Similarly, children who have been exposed to prior surgical procedures might be frightened by a face mask. Investigating this potential problem, Siegel et al suggested that wearing a mask during dental treatment represents a minimal stressor for the young child but recommended introducing the child to the dental environment and experience without the use of a protective mask.[31]

Opinions have differed on the relative importance of the dental team's attire. Research evidence from psychiatry and nursing has indicated few pervasive differences associated with the clothing worn. Similarly, research in dentistry has indicated that there were no significant preferences by patients. On the other hand, a survey of the Association of Pedodontic Diplomates suggested that enough children react unfavorably to white uniforms that many pediatric dentists have taken to wearing colored clothing.

Wearing apparel conceivably can influence both patients and professional staff. Less formal attire undoubtedly will have a beneficial effect on some children. It could also aid the staff by contributing toward a more natural and casual attitude. On the other hand, there are those who feel that street clothing might affect their professional image. Because conclusive evidence is lacking, it could be said that a dentist's attire remains a personal decision. Today, many practitioners and their staff have opted for wearing colorful scrubs and clinic gowns.

LIMITATIONS

Children today differ from those of 20 or 30 years ago. The child begins school earlier. Through the media, the child is more aware than children were years ago. We hear more of children facing poverty, experiencing learning disorders, and developing poor coping skills, and are more aware of children with eating disorders and drug usage. Children also have legal and social advocates who have influenced management techniques. Limitations on the dentist exist today that were unheard of previously.

Parenting also has changed. Much of the behavioral science research was done with traditional families in the 1960s and 1970s. Single-parent homes were less common, and terms such as *reconstituted families* and *partner relationships* were unknown. What about the child-rearing practices in these families? Two decades ago, when "father" came to the office, it usually meant that the child had a behavior problem and "father" was the enforcer. If a child misbehaved, the father might have spanked the child. Today, this is unacceptable in many families. With both parents working in many homes, it is not unusual for a father to accompany a child to the dental office. Have parental expectations changed in the dental office? Yes. This chapter discussed the reasons why parents might be excluded from the operatory, but it also urged reviewing the policy periodically. Years ago parents did not expect to enter the operatory. Currently, more and more parents insist on their right to accompany their child. Societal changes influence our management methods, and there is a need to review past research carefully and assess its applicability to the present.

The dentist has societal limitations, too, and they are changing approaches to management. Years ago the HOME technique might have been used to subdue a 3-year-old so that the dentist could perform an examination. This chapter did not describe the technique in detail because in today's society the dentist must have informed consent before using the HOME technique. It is also mandatory to be well trained in the use of the technique. Indeed, Carr et al report that the majority of pediatric dentists they surveyed use less aversive techniques (e.g., parents in the operatory, etc.) and had decreased or discontinued use of controversial techniques like HOME because of parental influences and legal and ethical concerns.[32] Dental students rarely have the opportunity to use the technique. Because dental practitioners are encouraged to perform behavior management consistent with their educational training and clinical experience, the technique may not belong in the armamentarium of most general practitioners.

The corners of the pediatric treatment triangle have been changing rapidly, and this influences the practice of dentistry for children. Recognizing these changing times, the Clinical Affairs Committee of the American Academy of Pediatric Dentistry has produced guide-lines for behavior management.[33] The techniques recommended in this chapter conform to these guide-lines. However, there are limitations to written standards; these standards change. Dental students and dentists must remember to keep abreast of the times in this highly dynamic area.

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CHAPTER

4

Development and Morphology of the Primary Teeth

RALPH E. McDONALD

DAVID R. AVERY

انتشارات
شایان نمودار

CHAPTER OUTLINE

LIFE CYCLE OF THE TOOTH
- Initiation (Bud Stage)
- Proliferation (Cap Stage)
- Histodifferentiation and Morphodifferentiation (Bell Stage)
- Apposition
- Calcification

EARLY DEVELOPMENT AND CALCIFICATION OF THE ANTERIOR PRIMARY TEETH

EARLY DEVELOPMENT AND CALCIFICATION OF THE POSTERIOR PRIMARY TEETH AND THE FIRST PERMANENT MOLAR

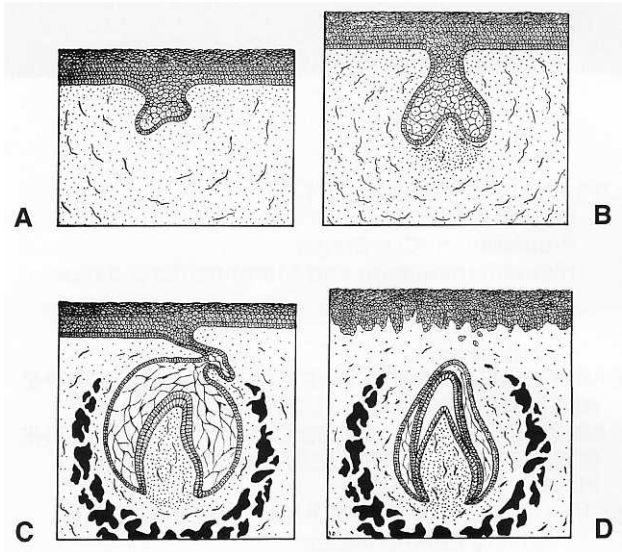
MORPHOLOGY OF INDIVIDUAL PRIMARY TEETH
- Maxillary Central Incisor
- Maxillary Lateral Incisor
- Maxillary Canine
- Mandibular Central Incisor
- Mandibular Lateral Incisor
- Mandibular Canine
- Maxillary First Molar
- Maxillary Second Molar
- Mandibular First Molar
- Mandibular Second Molar

MORPHOLOGIC DIFFERENCES BETWEEN PRIMARY AND PERMANENT TEETH

SIZE AND MORPHOLOGY OF THE PRIMARY TOOTH PULP CHAMBER

This chapter presents a brief review of the development of the teeth. An accurate chronology of primary tooth calcification is of clinical significance to the dentist. It is often necessary to explain to parents the time sequence of calcification in utero and during infancy. The common observation of tetracycline pigmentation, developmental enamel defects, and generalized hereditary anomalies can be explained if the calcification schedule is known. A brief discussion of the morphology of the primary teeth is also appropriate before considering restorative procedures for children.

A much more complete review is available in the reference texts on oral histology, dental anatomy, and developmental anatomy listed at the end of the chapter. Furthermore, contemporary scientists are rap-idly gaining knowledge of tooth development at the molecular level. We suggest that readers with a special interest in the molecular events of tooth development study the listed references by Ferguson et al and Sahlberg et al, and an extensive review article by Smith.[1-5]



(1 1 4-1. Life cycle of the tooth. A, Initiation (bud stage). **B,** Proliferation (cap stage). C, Histodifferentiation and morpho-differentiation (bell stage). **D,** Apposition and calcification. *(Adapted from Schour I, Massler M: Studies in tooth development: the growth pattern of human teeth,* J Am Dent Assoc *27:1785, 7940.)*

LIFE CYCLE OF THE TOOTH

INITIATION (BUD STAGE)

Evidence of development of the human tooth can be observed as early as the sixth week of embryonic life. Cells in the basal layer of the oral epithelium proliferate at a more rapid rate than do the adjacent cells. The result is an epithelial thickening in the region of the future dental arch that extends along the entire free margin of the jaws. This thickening is called the *primordium of the ectodermal portion of the teeth* and what results is called the *dental lamina.* At the same time, 10 round or ovoid swellings occur in each jaw in the position to be occupied by the primary teeth.

Certain cells of the basal layer begin to proliferate at a more rapid rate than do the adjacent cells (Fig. 4-1, A). These proliferating cells contain the entire growth potential of the teeth. The permanent molars, like the primary teeth, arise from the dental lamina. The permanent incisors, canines, and premolars develop from the buds of their primary predecessors. The congenital absence of a tooth is the result of a lack of initiation or an arrest in the proliferation of cells. The presence of supernumerary teeth is the result of a continued budding of the enamel organ.

PROLIFERATION (CAP STAGE)

Proliferation of the cells continues during the cap stage. As a result of unequal growth in the different parts of the bud, a cap is formed (Fig. 4-1, B). A shallow invagination appears on the deep surface of the bud. The peripheral cells of the cap later form the outer and inner enamel epithelium.

As with a deficiency in initiation, a deficiency in proliferation results in failure of the tooth germ to develop and in less than the normal number of teeth. Excessive proliferation of cells may result in epithelial rests. These rests may remain inactive or become activated as a result of an irritation or stimulus. If the cells become partially differentiated or detached from the enamel organ in their partially differentiated state, they assume the secretory functions common to all epithelial cells, and a cyst develops. If the cells become more fully differentiated or detached from the enamel organ, they produce enamel and dentin, which results in an odontoma (see Fig. 7-5) or a supernumerary tooth. The degree of differentiation of the cells determines whether a cyst, an odontoma, or a supernumerary tooth develops (see Figs. 27-50 to 27-52) .

HISTODIFFERENTIATION AND MORPHODIFFERENTIATION (BELL STAGE)

The epithelium continues to invaginate and deepen until the enamel organ takes on the shape of a bell (Fig. 4-1, C). It is during this stage that there is a differentiation of the cells of the dental papilla into odontoblasts and of the cells of the inner enamel epithelium into ameloblasts.

Histodifferentiation marks the end of the proliferative stage as the cells lose their capacity to multiply. This stage is the forerunner of appositional activity. Disturbances in the differentiation of the formative cells of the tooth germ result in abnormal structure of the dentin or enamel. One clinical example of the failure of ameloblasts to differentiate properly is amelogenesis imperfecta (see Fig. 7-31). The failure of the odontoblasts to differentiate properly, with the resultant abnormal dentin structure, results in the clinical entity dentinogenesis imperfecta (see Fig. 7-28).

In the morphodifferentiation stage the formative cells are arranged to outline the form and size of the tooth. This process occurs before matrix deposition. The morphologic pattern of the tooth becomes established when the inner enamel epithelium is arranged so that the boundary between it and the odontoblasts outlines the future dentinoenamel junction. Disturbances and aberrations in morphodifferentiation lead to abnormal forms and sizes of teeth. Some of the resulting conditions are peg teeth, other types of microdontia, and macrodontia.

APPOSITION

Appositional growth is the result of a layerlike deposition of a nonvital extracellular secretion in the form of a tissue matrix. This matrix is deposited by the formative cells, ameloblasts, and odontoblasts, which line up along the future dentinoenamel and dentinocemental junction at the stage of morphodifferentiation. These cells deposit the enamel and dentin matrix according to a definite pattern and at a definite rate. The formative cells begin their work at specific sites referred to as *growth centers* as soon as the blueprint, the dentinoenamel junction, is completed (Fig. 4-1, D).

Any systemic disturbance or local trauma that injures the ameloblasts during enamel formation can cause an interruption or an arrest in matrix apposition, which results in enamel hypoplasia (see Fig. 7-16). Hypoplasia of the dentin is less common than enamel hypoplasia and occurs only after severe systemic disturbances (see Fig. 7-15).

CALCIFICATION

Calcification (mineralization) takes place following matrix deposition and involves the precipitation of inorganic calcium salts within the deposited matrix. The process begins with the precipitation of a small nidus about which further precipitation occurs. The original nidus increases in size by the addition of concentric laminations. There is an eventual approximation and fusion of these individual calcospherites into a homogeneously mineralized layer of tissue matrix. If the calcification process is disturbed, there is a lack of fusion of the calcospherites. These deficiencies are not readily identified in the enamel, but in the dentin they are evident microscopically and are referred to as *interglobular dentin*.

EARLY DEVELOPMENT AND CALCIFICATION OF THE ANTERIOR PRIMARY TEETH

Kraus and Jordan have found that the first macroscopic indication of morphologic development occurs at approximately 11 weeks in utero. The maxillary and mandibular central incisor crowns appear identical at this early stage as tiny, hemispheric, moundlike structures.

The lateral incisors begin to develop morphologic characteristics between 13 and 14 weeks. There is evidence of the developing canines between 14 and 16 weeks. Calcification of the central incisor begins at approximately 14 weeks in utero, with the maxillary central incisor slightly preceding the lower central. The initial calcification of the lateral incisor occurs at 16 weeks and of the canine at 17 weeks.

It is interesting to note that the developmental dates listed precede by 3 to 4 weeks the dates that appear in the chronology of the human dentition, as developed by Logan and Kronfeld.[7] This observation has been confirmed by Lunt and Law[s]

EARLY DEVELOPMENT AND CALCIFICATION OF THE POSTERIOR PRIMARY TEETH AND THE FIRST PERMANENT MOLAR

The maxillary first primary molar appears macroscopically at 12'/2 weeks in utero. Kraus and Jordan' have observed that as early as $15^{1}/2$ weeks the apex of the mesiobuccal cusp may undergo calcification. At approximately 34 weeks the entire occlusal surface is covered by calcified tissue. At birth, calcification includes roughly three fourths of the occlusal gingival height of the crown.

The maxillary second primary molar also appears macroscopically at about $12^{1}/2$ weeks in utero. There is evidence of calcification of the mesiobuccal cusp as early as 19 weeks. At birth, calcification extends occlusogingivally to include approximately one fourth of the height of the crown.

The mandibular first primary molar initially becomes evident macroscopically at about 12 weeks in utero. Calcification may be observed as early as $15^{1}/2$ weeks at the apex of the mesiobuccal cusp. At birth, a completely calcified cap covers the occlusal surface.

The mandibular second primary molar also becomes evident macroscopically at $12^{1}/2$ weeks in utero. According to Kraus and Jordan, calcification may begin at 18 weeks.' At the time of birth the five centers have coalesced and only a small area of uncalcified tissue

remains in the middle of the occlusal surface. There are sharp conical cusps, angular ridges, and a smooth occlusal surface, all of which indicate that calcification of these areas is incomplete at birth. Thus there is a calcification sequence of central incisor, first molar, lateral incisor, canine, and second molar.

The work of Kraus and Jordan would indicate that the adjacent second primary and the first permanent molars undergo identical patterns of morphodifferentiation but at different times and that the initial development of the first permanent molar occurs slightly later. Their excellent research has also shown that the first permanent molars are uncalcified before 28 weeks of age; at any time thereafter calcification may begin. Some degree of calcification is always present at birth.

MORPHOLOGY OF INDIVIDUAL PRIMARY TEETH

MAXILLARY CENTRAL INCISOR

The mesiodistal width of the crown of the maxillary central incisor is greater than the cervicoincisal length. Developmental lines are usually not evident in the crown; thus the labial surface is smooth. The incisal edge is nearly straight even before abrasion becomes evident. There are well-developed marginal ridges on the lingual surface and a distinctly developed cingulum (Figs. 4-2 and 4-3). The root of the incisor is cone shaped with tapered sides.

MAXILLARY LATERAL INCISOR

The outline of the maxillary lateral incisor is similar to that of the central incisor, but the crown is smaller in all dimensions. The length of the crown from the cervical to the incisal edge is greater than the mesiodistal width. The root outline is similar to that of the central incisor but is longer in proportion to the crown.

MAXILLARY CANINE

The crown of the maxillary canine is more constricted at the cervical region than are the incisors, and the incisal and distal surfaces are more convex. There is a well-developed sharp cusp rather than a relatively straight incisal edge. The canine has a long, slender, tapering

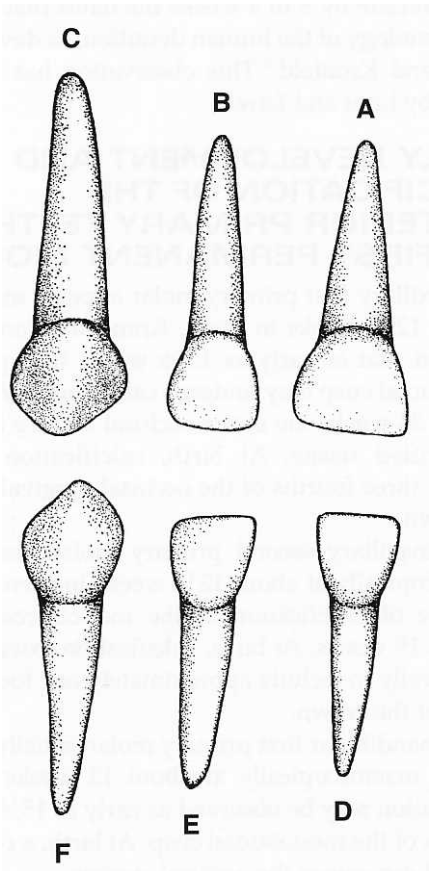


FIG 4-2. Primary right anterior teeth, labial aspect. **A,** Maxillary central incisor. **B,** Maxillary lateral incisor. C, Maxillary canine. D, Mandibular central incisor. **E,** Mandibular lateral incisor. **F,** Mandibular canine. *(From Ash MM, Nelson SJ: Wheeler's dental anatomy, physiology, and occlusion, ed 8, Philadelphia, 2003, WB Saunders.)*

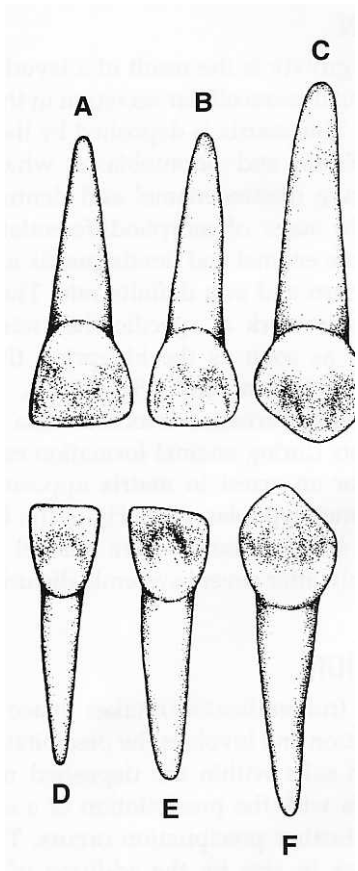


FIG 4-3. Primary right anterior teeth, lingual aspect. **A,** Maxillary central incisor. **B,** Maxillary lateral incisor. C, Maxillary canine. **D,** Mandibular central incisor. **E,** Mandibular lateral incisor. **F,** Mandibular canine. *(From Ash MM, Nelson SJ: Wheeler's dental anatomy, physiology, and occlusion, ed 8, Philadelphia, 2003, WB Saunders.)*

root that is more than twice the length of the crown. The root is usually inclined distally, apical to the middle third.

MANDIBULAR CENTRAL INCISOR

The mandibular central incisor is smaller than the maxillary central, but its labiolingual measurement is usually only 1 mm less. The labial aspect presents a flat surface without developmental grooves. The lingual surface presents marginal ridges and a cingulum. The middle third and the incisal third on the lingual surface may have a flattened surface level with the marginal ridges, or there may be a slight concavity. The incisal edge is straight, and it bisects the crown labiolingually. The root is approximately twice the length of the crown.

MANDIBULAR LATERAL INCISOR

The outline of the mandibular lateral incisor is similar to that of the central incisor but is somewhat larger in a dimensions except labiolingually. The lingual surfa may have greater concavity between the margin ridges. The incisal edge slopes toward the distal aspe of the tooth.

MANDIBULAR CANINE

The form of the mandibular canine is similar to that of the maxillary canine, with a few exceptions. The crown is slightly shorter, and the root may be as much as 2 mm shorter than that of the maxillary canine. The mandibular canine is not as large labiolingually as its maxillary opponent.

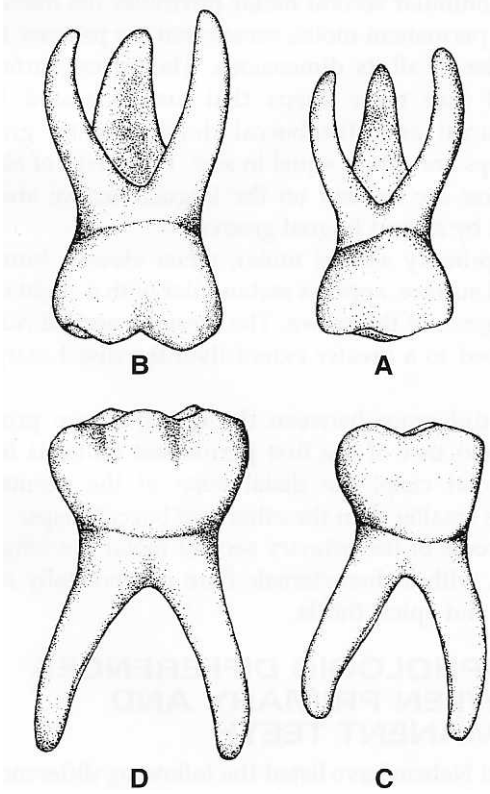
MAXILLARY FIRST MOLAR

The greatest dimension of the crown of the maxillary first molar is at the mesiodistal contact areas, and from these areas the crown converges toward the cervical region (Figs. 4-4 to 4-6).

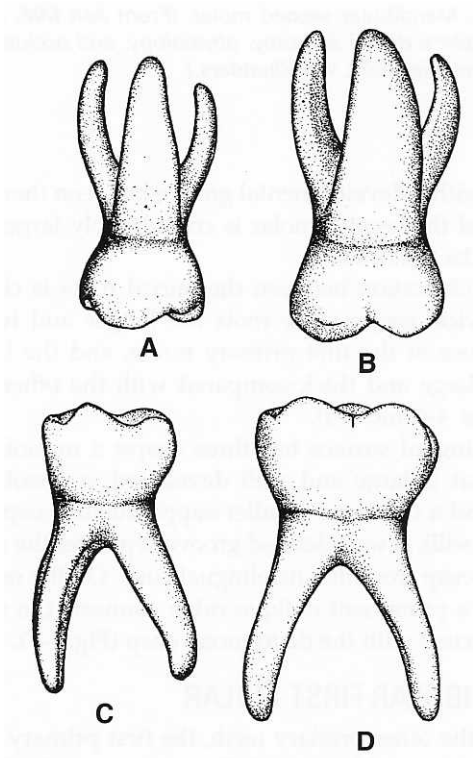
The mesiolingual cusp is the largest and sharpest. The distolingual cusp is poorly defined, small, and rounded. The buccal surface is smooth, with little evidence of developmental grooves. The three roots are long, slender, and widely spread.

MAXILLARY SECOND MOLAR

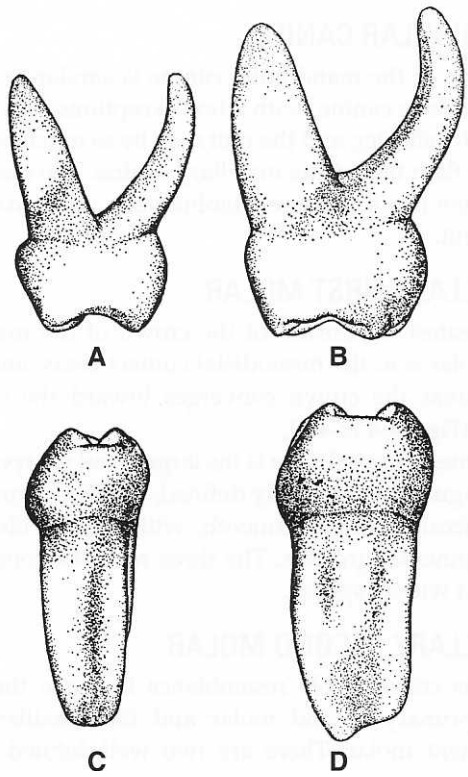
There is considerable resemblance between the maxillary primary second molar and the maxillary first permanent molar. There are two well-defined buccal



f I G . **4-4.** Primary right molars, buccal aspect. A, Maxillary first molar. B, Maxillary second molar. C, Mandibular first molar. D, Mandibular second molar. *(From Ash MM, Nelson SJ: Wheeler's dental anatomy, physiology, and occlusion, ed 8, Philadelphia, 2003, WB Saunders.)*



F **I G 4-5.** Primary right molars, lingual aspect. **A,** Maxillary first molar. **B,** Maxillary second molar. C, Mandibular first molar. D, Mandibular second molar. *(From Ash MM, Nelson SJ: Wheeler's dental anatomy, physiology, and occlusion, ed 8, Philadelphia, 2003, WB Saunders.)*



F I G 4-6. Primary right molars, mesial aspect. **A,** Maxillary first molar. **B,** Maxillary second molar. C, Mandibular first molar. D, Mandibular second molar. *(From Ash MM, Nelson SJ: Wheeler's dental anatomy, physiology, and occlusion, ed 8, Philadelphia,* 2003, *WB Saunders.)*

cusps, with a developmental groove between them. The crown of the second molar is considerably larger than that of the first molar.

The bifurcation between the buccal roots is close to the cervical region. The roots are longer and heavier than those of the first primary molar, and the lingual root is large and thick compared with the other roots (see Figs. 4-4 and 4-5).

The lingual surface has three cusps: a mesiolingual cusp that is large and well developed, a distolingual cusp, and a third and smaller supplemental cusp (cusp of Carabelli). A well-defined groove separates the mesiolingual cusp from the distolingual cusp. On the occlusal surface a prominent oblique ridge connects the mesiolingual cusp with the distobuccal cusp (Fig. 4-7).

MANDIBULAR FIRST MOLAR

Unlike the other primary teeth, the first primary molar does not resemble any of the permanent teeth. The mesial outline of the tooth, when viewed from the buccal aspect, is almost straight from the contact area to the cervical region. The distal area of the tooth is shorter than the mesial area.

The two distinct buccal cusps have no evidence of a distinct developmental groove between them; the mesial cusp is the larger of the two.

There is a pronounced lingual convergence of the crown on the mesial aspect, with a rhomboid outline present on the distal aspect. The mesiolingual cusp is long and sharp at the tip; a developmental groove separates this cusp from the distolingual cusp, which is rounded and well developed. The mesial marginal ridge is well developed, to the extent that it appears as another small cusp lingually. When the tooth is viewed from the mesial aspect, there is an extreme curvature buccally at the cervical third. The crown length is greater in the mesiobuccal area than in the mesiolingual area; thus the cervical line slants upward from the buccal to the lingual surface.

The longer slender roots spread considerably at the apical third, extending beyond the outline of the crown. The mesial root, when viewed from the mesial aspect, does not resemble any other primary root. The buccal and lingual outlines of the root drop straight down from the crown, being essentially parallel for over half their length. The end of the root is flat and almost square.

MANDIBULAR SECOND MOLAR

The mandibular second molar resembles the mandibular first permanent molar, except that the primary tooth is smaller in all its dimensions. The buccal surface is divided into three cusps that are separated by a mesiobuccal and distobuccal developmental groove. The cusps are almost equal in size. Two cusps of almost equal size are evident on the lingual surface and are divided by a short lingual groove.

The primary second molar, when viewed from the occlusal surface, appears rectangular with a slight distal convergence of the crown. The mesial marginal ridge is developed to a greater extent than the distal marginal ridge.

One difference between the crown of the primary molar and that of the first permanent molar is in the distobuccal cusp; the distal cusp of the permanent molar is smaller than the other two buccal cusps.

The roots of the primary second molar are long and slender, with a characteristic flare mesiodistally in the middle and apical thirds.

MORPHOLOGIC DIFFERENCES BETWEEN PRIMARY AND PERMANENT TEETH

Ash and Nelson have listed the following differences in form between the primary and permanent teeth`':

1. The crowns of the primary teeth are wider mesiodistally in comparison with crown length than are those of the permanent teeth.

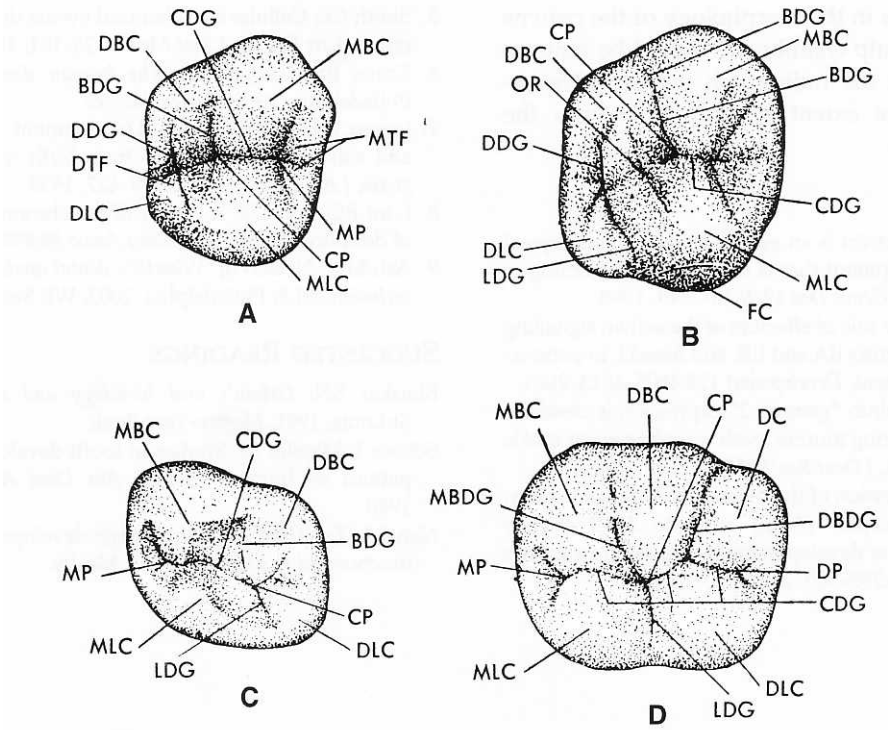


FIG. 4-7. Primary right molars, occlusal aspect. **A,** Maxillary first molar. **B,** Maxillary second molar. C, Mandibular first molar. D, Mandibular second molar. *BDG,* Buccal developmental groove; *CDG,* central developmental groove; *CP* central pit; *DBC,* distobuccal cusp; *DBDG,* distobuccal developmental groove; *DC,* distal cusp; *DDG,* distal developmental groove; *DLC,* distolingual cusp; *DP,* distal pit; *DTF,* distal triangular fossa; FC, fifth cusp; *LDG,* lingual developmental groove; *MBC,* mesiobuccal cusp; *MBDG,* mesiobuccal developmental groove; *MLC,* mesiolingual cusp; *MP,* mesial pit; *MTF,* mesial triangular fossa; OR, oblique ridge. *(From Ash MM, Nelson SJ: Wheeler's dental anatomy, physiology, and occlusion, ed 8, Philadelphia, 2003, WB Saunders.)*

2. The roots of primary anterior teeth are narrow and long compared with crown width and length.
3. The roots of the primary molars are relatively longer and more slender than the roots of the permanent teeth. There is also a greater extension of the primary roots mesiodistally. This "flaring" allows more room between the roots for the development of the premolar tooth crowns.
4. The cervical ridge of enamel at the cervical third of the anterior crowns is much more prominent labially and lingually in the primary than in the permanent teeth.
5. The crowns and roots of primary molars are more slender mesiodistally at the cervical third than are those of the permanent molars.
6. The cervical ridge on the buccal aspect of the primary molars is much more definite, particularly on the maxillary and mandibular first molars, than that on the permanent molars.
7. The buccal and lingual surfaces of the primary molars are flatter above the cervical curvatures than those of the permanent molars, which makes the occlusal surface narrower compared with that of the permanent teeth.
8. The primary teeth are usually lighter in color than the permanent teeth.

SIZE AND MORPHOLOGY OF THE PRIMARY TOOTH PULP CHAMBER

Considerable individual variation exists in the size of the pulp chamber and pulp canal of the primary teeth. Immediately after the eruption of the teeth the pulp chambers are large, and in general they follow the outline of the crown. The pulp chamber decreases in size as age increases and under the influence of function and of abrasion of the occlusal and incisal surfaces of the teeth.

No attempt is made here to describe in detail each pulp chamber outline; rather, it is suggested that the dentist examine critically the bite-wing radiographs of the child before undertaking operative procedures. Just as there are individual differences in the calcification time of teeth and also in eruption time, so are there

individual differences in the morphology of the crowns and the size of the pulp chamber. It should be remembered, however, that the radiograph will not demonstrate completely the extent of pulp horn into the cuspal area.

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CHAPTER 5

Radiographic Techniques

DALE A. MILES
EDWIN T. PARKS

انتشارات
شایان نمودار

CHAPTER OUTLINE

RADIATION SAFETY AND PROTECTION
SELECTION CRITERIA AND RADIOGRAPHIC EXAMINATIONS
- Criteria for Exposing of Radiographs in Asymptomatic Children
- Development of the Dentition as Criterion
  - *Primary dentition*
  - *Early transitional dentition*
  - *Early permanent dentition*
- Risk of the Patient for Dental Caries as Criterion
- Types of Findings Anticipated as Criteria
- Radiographic Examinations
  - *Four-film series*
  - *Eight-film survey*
  - *Twelve-film survey*
  - *Sixteen-film survey*

COMMONLY USED RADIOGRAPHIC TECHNIQUES
- Bite-Wing Technique Paralleling Technique
- Bisecting Angle Technique
- Specific Dental Projections
  - *Periapical*
  - *Molar projection*
  - *Premolar or primary molar projection*
  - *Permanent or primary canine projection*
  - *Permanent or primary incisor projection*
  - *Anterior maxillary occlusal technique*
  - *Posterior maxillary occlusal technique*
  - *Anterior mandibular occlusal technique*
  - *Occlusal*
- Panoramic Radiography

LOCALIZATION TECHNIQUES
DIGITAL RECEPTORS FOR PEDIATRIC PATIENTS
INTERPRETATION

Roentgen's discovery of the x-ray in 1895 provided one of the most important diagnostic aids in dentistry. Radiographs are essential if we are to treat children successfully. Evidence indicates that, unless carious lesions are discovered early, the primary teeth will not be retained until normal exfoliation.

Early diagnosis of caries prevents the pediatric patient from experiencing dental pain, extraction, and emotional stress. In addition, eruptive or developmental problems can be discovered with the use of radio-graphic images, and early treatment of these problems may reduce the need for prolonged orthodontic procedures. Some restorative procedures require an accurate registration of the pulpal outline that only a radiograph can reveal.

The selection of appropriate radiographs for the pediatric patient depends on the age of the child, the size of the oral cavity, and the level of patient cooperation. These are determined by a careful clinical examination of the patient before ordering the radiographic survey. The examination determines the need for radiographs and the type to be taken. Ideal technique should expose the patient to a minimum amount of radiation, require as few radiographs as possible, take as little time as possible, and provide a diagnostically accurate examination of the dentition and sup-porting structures. The child's cooperation is as essential to radiographic examination as is the selection of correct radiographic technique. Both increase the probability of success and reduce additional radiographic exposure.

Dental radiographic equipment can be threatening or can generate curiosity, depending on the child. It is wise to allow the patient to run a hand over the x-ray head and become acquainted with the "camera." The patient might hold one of the films and be shown where it will be placed. If it is a film that requires biting pressure, the patient should be shown how to bite on the film. "Show and tell" will go a long way in gaining cooperation. Careful wording of the description of the procedure is essential to gain patient cooperation. The potential cooperation of many a patient has been lost when the patient hears that you will be "shooting" a couple of films. Imaging the easiest region first may ensure success in other areas. This is particularly important if the child has an exaggerated gag reflex or objects to the placement of the film. The use of topical anesthetic agents may be beneficial in both situations.

The dentist should be patient with the child in obtaining radiographs. Repeated attempts at film placement may be necessary before the actual radiation exposure is made. If the child is uncooperative, firmness, voice control, and tender loving care are often effective. Emotionally, mentally, and physically disabled children require special handling.

RADIATION SAFETY AND PROTECTION

One characteristic of x-radiation is its ability to impart some of its energy to the matter it traverses. If that matter is living tissue, then some biologic injury may occur. Much information about high levels of radiation and subsequent damage is available. The effects of low levels of x-radiation (as used in diagnostic radiology) on biologic systems are virtually unknown. Our assumptions of damage are based on extrapolation of data from high levels to lower levels of radiation.

Still, dental health professionals must be concerned about any risk the patient may encounter during therapy. Concern is focused on three primary biologic effects of low-level radiation: (1) carcinogenesis, (2) teratogenesis (malformations), and (3) mutagenesis. Carcinogenesis and malformations are a response of somatic tissues and in most instances are believed to have a threshold response; that is, a certain amount of radiation is necessary before the response is seen. Mutation may occur as a response of genetic tissue (gonads) to x-radiation and is believed to have no threshold. In general, younger tissues and organs are more sensitive to radiation, with the sensitivity decreasing from the period before birth until maturity. It must also be recognized that far higher doses of radiation can be withstood by localized areas than by the whole body. We know that we live in a world that exposes us to natural background radiation averaging 360 mrem per year (3.60 millisievert) in the United States. Medical and dental radiographic examinations add to that exposure, and so must be ordered judiciously.

With regard to patient protection, evidence has shown that there are critical organs vulnerable to possible development of late effects. These organs should be shielded when possible. These critical organs and the associated adverse biologic effects are the following: (1) the skin (cancer), (2) red bone marrow (leukemia), (3) the gonads (mutation, infertility, and fetal malformations), (4) the eyes (cataracts), (5) the thyroid (cancer), (6) the breasts (cancer), and (7) possibly the salivary glands (cancer).

The practitioner and staff can physically protect the patient and indirectly protect themselves from unnecessary exposure to radiation by the use of correct technique. The most obvious method of protecting the patient is to shield those areas not being evaluated. This is easily accomplished using a lead apron and thyroid collar (Fig. 5-1).

The apron and collar may be incorporated as a single unit or used separately. The apron protects the gonads

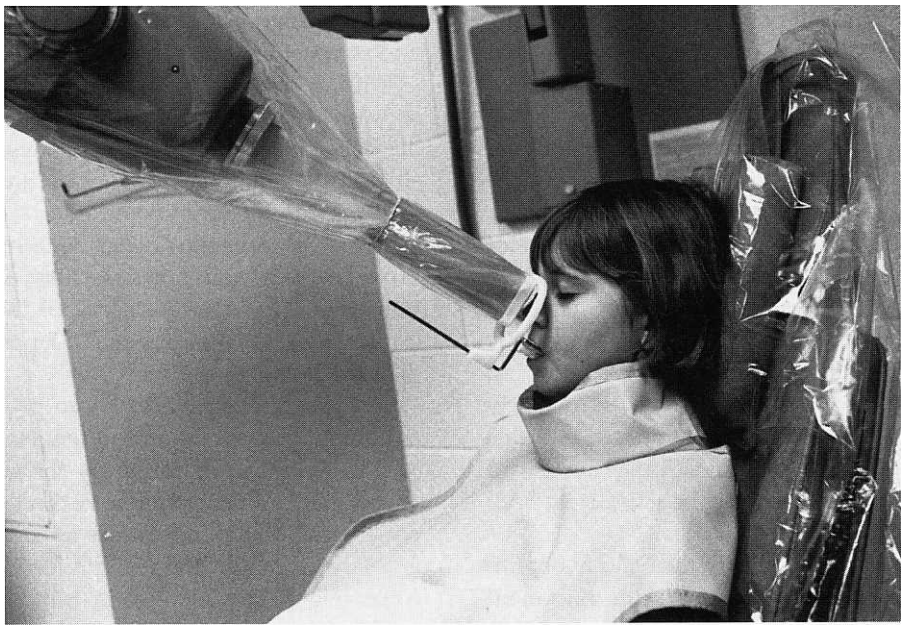


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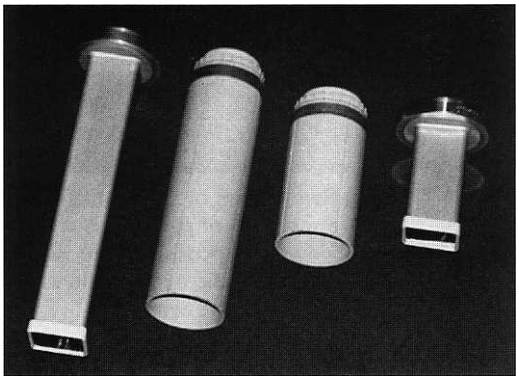
f I G. **5-1.** Lead apron and thyroid collar in place.

and chest from the primary beam and scatter radiation whereas the collar shields the thyroid. This method does not provide complete protection, particularly of the thyroid, but provides a great reduction in exposure. Aprons used in panoramic radiography have a front and back because the source of radiation is from the side and the rear of the patient.

Faster film speeds have contributed most significantly to the reduction in radiation to the patient. Film speeds of the "D," "E," and "F" groups are currently available for intraoral radiography. Faster film also reduces error from patient movement, a consideration with the pediatric patient. Extraoral (panoramic) radiography uses film-screen combinations that also have reduced exposure times. Recently, there has been an increased use of beam-positioning devices (Fig. 5-2) that have virtually eliminated some technical errors, particularly film cone cuts. The length and the shape of the



f I G. **5-2.** Use of the Rinn XCP positioning device.



f G **5-3.** Rectangular and round cones of various lengths.

cone (Fig. 5-3) have also aided in the reduction of patient radiation exposure. Use of a long rectangular collimator reduces the area unnecessarily exposed to radiation by almost 4 sq in compared with a round collimator. The use of higher kilovolt peak techniques reduces patient exposure to radiation and lowers contrast, thus increasing the number of shades of gray on the film. An additional benefit of using high kilovolt peak technique is that the exposure time is shortened, which potentially reduces retakes caused by patient movement.

Finally, quality control (and thus patient protection) begins in the darkroom. If the darkroom does not function at an optimal level, retakes will be required, necessitating additional patient exposure. The dentist must insist on time-temperature developing and the use of proper-strength chemicals if wet tanks are used. The darkroom should be clean and free of white light (light leaks). If automatic processing is used, chemical parameters must be continuously monitored and the unit must be cleaned weekly.

SELECTION CRITERIA AND RADIOGRAPHIC EXAMINATIONS

The decision to make radiographs is based on a thorough evaluation and examination of the patient. Radiographs should be made only when there is an expectation that disease is present or when an undetected condition left untreated could adversely affect the patient's dental health. Therefore, the decision to use ionizing radiation is based on professional judgment (Table 5-1).

Selection criteria are clinical signs or symptoms that allow the practitioner to identify patients who will benefit from a radiographic examination. Two important considerations when deciding whether to perform a radiographic examination for children are (1) the stage of dentition development and (2) the risk of dental caries.

The criteria for making radiographs, developed in part by the Conference on Radiation Exposure in Pediatric Dentistry, were summarized by Nowak et al[1] as follows.

CRITERIA FOR EXPOSING OF RADIOGRAPHS IN ASYMPTOMATIC CHILDREN

The criteria for exposing radiographs assume that the child is asymptomatic and that the dentist finds no specific clinical indications for radiographic examination. Exceptions to this rule include those conditions in which there is clinical evidence of injury, disease such as caries, pulpal pathosis, delayed or accelerated eruption or exfoliation of teeth, swelling, hemorrhage, pain, or ulceration, or those conditions in which there is a need to evaluate treatment. In such cases, taking appropriate radiographs is indicated to confirm the diagnosis and facilitate and evaluate treatment.

DEVELOPMENT OF THE DENTITION AS CRITERION

Dental radiographs are indicated in the following situations.

Primary Dentition. If the proximal surfaces of the primary teeth cannot be visually and tactilely inspected and the child can be expected to cooperate, then dental radiographs should be made to determine the presence of interproximal caries. If all surfaces of all primary teeth can be examined clinically because of open contact, then radiographs are not indicated.

If the child's behavior is such that obtaining films of adequate diagnostic quality is doubtful, then radiographs should be deferred until behavior improves.

Early Transitional Dentition. (After appearance of permanent first molars or permanent mandibular incisors, or both.) Radiographs are taken to evaluate the presence of interproximal caries, developmental anomalies of teeth, and pathologic conditions of the hard and soft tissues of the mouth, jaws, and associated structures.

Early Permanent Dentition. (After puberty; after patients have achieved most of their adult stature; late adolescence). Radiographs are taken to evaluate the same tissues as in the early mixed dentition and to check the position and developmental status of the third molars.

No other dental radiographs are routinely needed in children. Other radiographs would be prescribed for diagnostic purposes. As an example, diagnostic bite-wing radiographs would be taken to detect the presence of interproximal caries if the risk of caries activity were high for that individual. Occlusal caries, detected clinically, and discolored marginal ridges are indications of high risk of caries activity. Periapical films of the canine areas could be taken if these teeth were not clinically palpable by 9 years of age. The dentist must use clinical

TABLE 5-1 Guidelines for Prescribing Dental Radiographs

PATIENT CATEGORY	CHILD	
	PRIMARY DENTITION (PRIOR TO ERUPTION OF FIRST PERMANENT TOOTH)	TRANSITIONAL DENTITION (FOLLOWING ERUPTION OF FIRST PERMANENT TOOTH)
New patient* All new patients to assess dental diseases and growth and development	Posterior bite-wing examination if proximal surfaces of primary teeth cannot be visualized or probed	Individualized radiographic examination consisting of periapical/occlusal views and posterior bite-wings *or* panoramic examination and posterior bite-wings
Recall patient* Clinical caries or high-risk factors for caries[t]	Posterior bite-wing examination 6-month intervals or until no carious lesions are evident	
No clinical caries and no high-risk factors for caries'	Posterior bite-wing examination at 12- to 24-month intervals if proximal surfaces of primary teeth cannot be visualized or probed	Posterior bite-wing examination at 12- to 24-month intervals
Periodontal disease or a history of periodontal treatment	Individualized radiographic examination consisting of selected periapical and/or bite-wing radiographs for areas where periodontal disease (other than non-specific gingivitis) can be demonstrated clinically	
Growth and development assessment	Usually not indicated	Individualized radiographic examination consisting of a periapical/occlusal or panoramic examination

The recommendations contained in this table were developed by an expert dental panel comprised of representatives from the Academy of General Dentistry, American Academy of Dental Radiology, American Academy of Oral Medicine, American Academy of Pediatric Dentistry, American Academy of Periodontology, and American Dental Association under the sponsorship of the Food and Drug Administration (FDA). The chart is being reproduced and distributed to the dental community by Eastman Kodak Company in cooperation with the FDA.

ADOLESCENT	ADULT	
PERMANENT DENTITION (PRIOR TO ERUPTION OF THIRD MOLARS)	DENTULOUS	EDENTULOUS
Individualized radiographic examination consisting of posterior bite-wings and selected periapicals. A full mouth intraoral radiographic examination is appropriate when the patient presents with clinical evidence of generalized dental disease or a history of extensive dental treatment.		Full mouth intra-oral radiographic examination or panoramic examination
Posterior bite-wing examination at 6- to 12-month intervals or until no carious lesions are evident	Posterior bite-wing examination at 12- to 18-month intervals	Not applicable
Posterior bite-wing examination at 18- to 36-month intervals	Posterior bite-wing examination at 24- to 36-month intervals	Not applicable
Individualized radiographic examination consisting of selected periapical and/ or bite-wing radiographs for areas where periodontal disease (other than non-specific gingivitis) can be demonstrated clinically		Not applicable
Periapical or panoramic examination to assess developing third molars	Usually not indicated	Usually not indicated

***Clinical situations for which radiographs may be indicated include the following:**

A. **Positive Historical Findings**
- Previous periodontal or endodontic therapy
- History of pain or trauma
- Familial history of dental anomalies
- Postoperative evaluation of healing
- Presence of implants

B. Positive Clinical Signs/Symptoms
- Clinical evidence of periodontal disease
- Large or deep restorations
- Deep carious lesions
- Malposed or clinically impacted teeth
- Swelling
- Evidence of facial trauma
- Mobility of teeth
- Fistula or sinus tract infection
- Clinically suspected sinus pathology
- Growth abnormalities
- Oral involvement in known or suspected systemic disease
- Positive neurologic findings in the head and neck
- Evidence of foreign objects
- Pain and/or dysfunction of the temporomandibular joint
- Facial asymmetry
- Abutment teeth for fixed or removable partial prosthesis
- Unexplained bleeding
- Unexplained sensitivity of teeth
- Unusual eruption, spacing, or migration of teeth
- Unusual tooth morphology, calcification, or color
- Missing teeth with unknown reason

'Patients at high risk for caries may demonstrate any of the following:
- High level of caries experience
- History of recurrent caries
- Existing restoration of poor quality
- Poor oral hygiene
- Inadequate fluoride exposure
- Prolonged nursing (bottle or breast)
- Diet with high sucrose frequency
- Poor family dental health
- Developmental enamel defects
- Developmental disability
- Xerostomia
- Genetic abnormality of teeth
- IVlany multisurface restorations
- Chemo/radiation therapy

judgment and evaluate such factors as oral hygiene practices, diet, attitude and compliance, fluoride history, alignment of teeth, and morphology of teeth to determine the necessity for, the extent of, and the type and frequency of diagnostic radiographs.

RISK OF THE PATIENT FOR DENTAL CARIES AS CRITERION

A high risk of dental caries may be associated with poor oral hygiene, fluoride deficiency, prolonged nursing (bottle or breast), high-carbohydrate diet, poor family

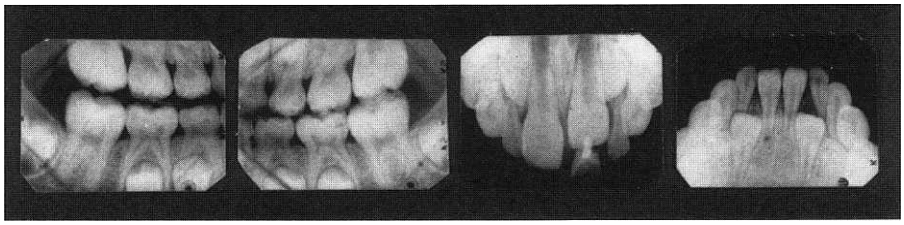


FIG **5-4.** Four-film series.

dental health, developmental enamel defects, developmental disability and acute or chronic medical history, and genetic abnormality.

The child with a high risk of dental caries should have bite-wing radiographs made as soon as posterior primary teeth are in proximal contact. The age of the patient is not an important variable. If interproximal caries is detected, then follow-up radiographs are indicated semiannually until the child is caries free and therefore is classified as having a low risk for dental caries.

A child who has a low risk for dental caries may be defined as a normal, healthy, asymptomatic patient exposed to optimal levels of fluoride (preferably since birth), who performs daily preventive techniques and consumes a diet with few exposures to retentive carbohydrates between meals. Posterior bite-wing radiographs should be made for the low-risk patient with closed proximal contacts. If no evidence of caries is found, then radiographs may be retaken every 12 to 18 months if primary teeth are in contact or after up to 24 months if permanent teeth are in contact. Bite-wing radiographs may be taken more frequently if the child enters the high-risk category.

The more rapid progression of caries in primary teeth should be considered in determining the time interval between bite-wing radiographs.

TYPES OF FINDINGS ANTICIPATED AS CRITERIA

Bite-wing radiographs are indicated when a clinical examination discloses posterior tooth contact. Bite-wing examinations are recommended at the first clinical evidence of caries. Bite-wing radiographs are usually taken every 12 to 18 months in the absence of dental caries with primary tooth contact or every 24 months with permanent tooth contact.

By the time the first permanent tooth has erupted (posterior or anterior), an anterior occlusal radiograph should be made. This allows detection of conditions such as supernumerary teeth, missing teeth, and dens in dente. A radiographic examination that includes the tooth-bearing areas of the mandible and maxilla is recommended at approximately the time of the early mixed dentition to assess the dental age of the patient and to aid in the early diagnosis of congenital and developmental anomalies.

The radiographic examination may consist of one of the following: posterior periapical radiographs, panoramic radiograph, or lateral jaw 45-degree projections. Another similar radiographic examination may be made within 2 years after the eruption of the permanent second molars.

To check on growth and development, a cephalometric radiograph may be prescribed by the practitioner who is providing the orthodontic diagnosis or treatment.

RADIOGRAPHIC EXAMINATIONS

When a new patient is seen at the dental office and no previous radiographs are available, it may be necessary to obtain a baseline series of radiographs. Again, we cannot overemphasize that the decision to make a radiographic examination is based on the criteria previously outlined. These examinations include the following.

Four-Film Series. This series consists of a maxillary and mandibular anterior occlusal and two posterior bite-wings (Fig. 5-4).

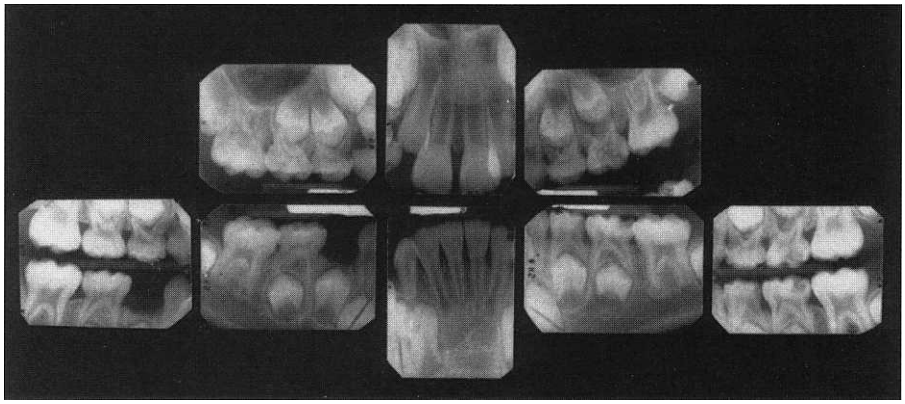
Eight-Film Survey. This survey includes a maxillary and mandibular anterior occlusal (or periapicals), a right and left maxillary posterior occlusal (or periapicals), right and left primary mandibular molar periapicals, and two posterior bite-wings (Fig. 5-5).

Twelve-Film Survey. This examination includes four primary molar-premolar periapical radiographs, four canine periapical radiographs, two incisor periapical radiographs, and two posterior bite-wing radiographs (Fig. 5-6).

Sixteen-Film Survey. This examination consists of the 12-film survey and the addition of 4 permanent molar radiographs (Fig. 5-7).

COMMONLY USED RADIOGRAPHIC TECHNIQUES

Several techniques are commonly used to radiograph a child's dentition. The technique used depends primarily on the size of the oral cavity, the number of teeth present, and patient cooperation. The procedures



f I G. **5-5.** Eight-film survey.

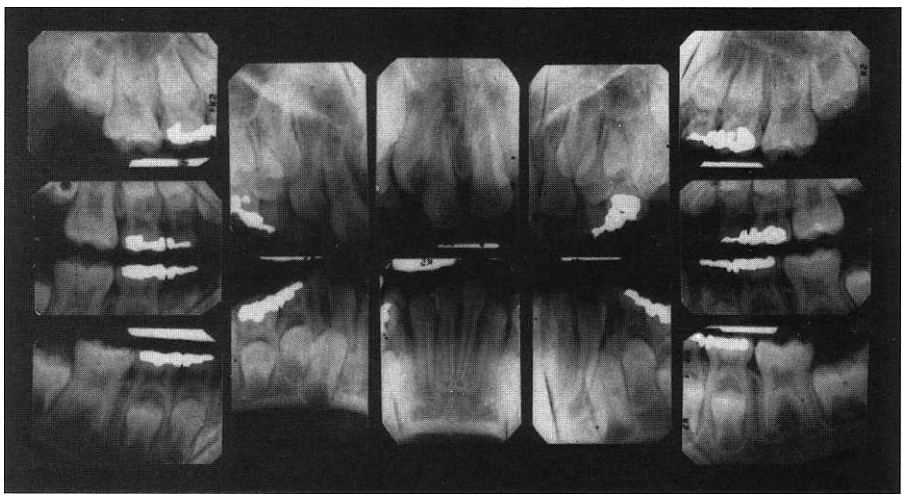
commonly used by the private practitioner include the following:

1. Bite-wing
2. Periapical
3. Occlusal
4. Panoramic

BITE-WING TECHNIQUE

A No. 0 bite-wing film is usually the most suitable size for the smaller patient. However, some children's mouths are large enough to receive a No. 2 bite-wing film. The head is positioned so that the midsagittal plane is perpendicular and the ala-tragus line is parallel to the floor. The inferior edge of the bite-wing film packet is placed in the floor of the mouth between the tongue and the lingual aspect of the mandible and the bite-tab or positioning device is placed on the occlusal surfaces of the mandibular teeth (Fig. 5-8). The anterior edge of the film packet is located as far anteriorly as possible in the region of the canine so that the distal aspect of the canine will be recorded. The lower anterior corner of the film packet is bent slightly toward the lingual to facilitate film placement and to decrease possible patient discomfort. The anteroinferior corner of the film packet usually lies near or at the attachment of the lingual frenum in the midline. In addition, the posterosuperior corner may be bent to prevent the gag reflex.

The dentist holds the bite-tab against the occlusal surfaces of the patient's mandibular teeth with an index finger, and the patient is instructed to "close slowly." The finger is rolled out of the way onto the buccal surfaces of the teeth as the patient closes in centric occlusion. The central ray enters through the occlusal plane at a point below the pupil of the eye. The vertical angle is +8 to +10 degrees. The horizontal diameter of the open-end cone is parallel to the end of the bite-tab



f **I G 5-6.** Twelve-film survey.

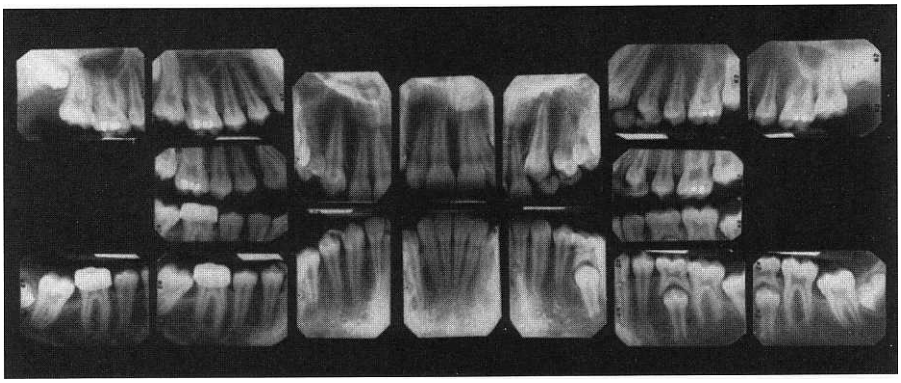
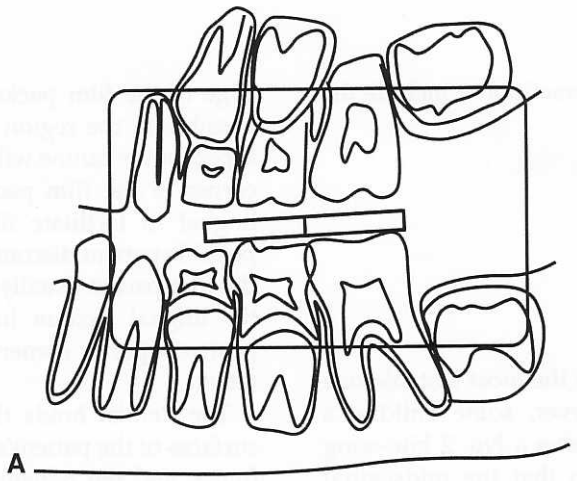


FIG. 5-77. Sixteen-film survey.



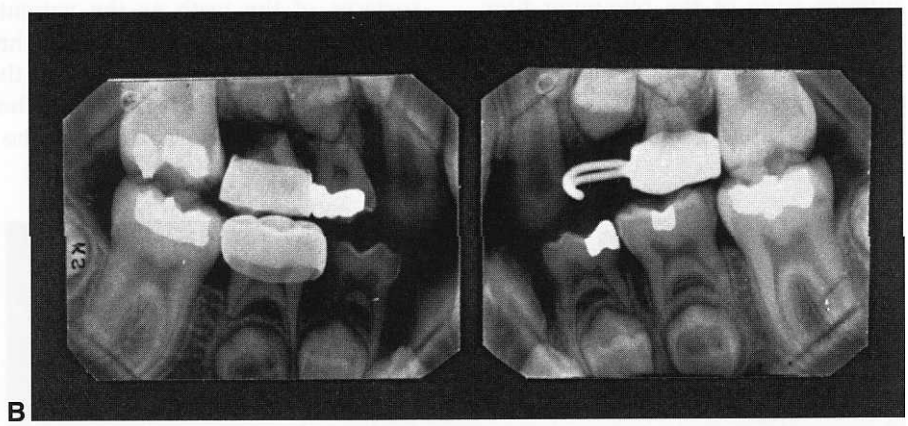
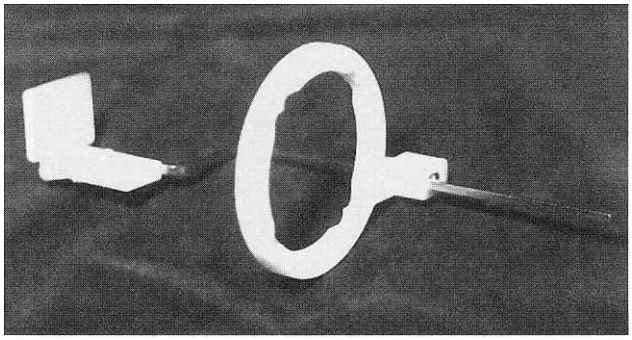


FIG. 5-8. Posterior bite-wing. A, Film placement. **B,** Radiograph.

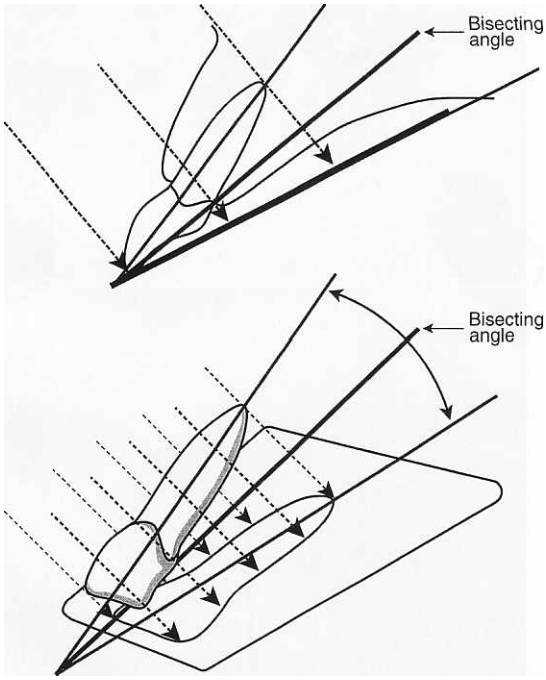
or to the mean tangent of the buccal surfaces of the posterior teeth being radiographed.

PARALLELING TECHNIQUE

In principle the paralleling technique requires the object (long axis of the tooth) and the film to be parallel in all dimensions. To achieve this, the film packet is placed farther away from the object, particularly the maxilla. This tends to magnify the image. This undesirable effect is offset when a longer cone is used, which thus reduces magnification. Use of a longer cone also increases image sharpness by decreasing the penumbra. Striving for true parallelism will enhance image accuracy.



f I G. **5-9.** Rinn XCP positioning *devices* and film holders cut down to accommodate child patient's mouth.



f **I G 5-10.** The bisecting angle. *(Courtesy Mr. Mark Dirlam.)*

Because the film is placed farther away from the object, a film holder is necessary (Fig. 5-9). Some of those holders also have beam-aligning devices to help ensure parallelism and reduce partial exposure of the film; thus unwanted cone cuts are eliminated. For the smaller child, the film holder may need to be reduced in size to accommodate the film and the child's mouth. Film-holding devices with cone alignment guides reduce operator error and thus reduce exposure of the patient to radiation. Because of the shallowness of the child's palate and floor of the mouth, film placement is somewhat compromised. Despite this, the resulting films are quite satisfactory.

BISECTING ANGLE TECHNIQUE

The bisecting angle technique is based on a principle called the *rule of isometry,* which basically states that two triangles are equal if they have two equal angles and a common side. The clinical application of this rule has the central ray directed perpendicularly to a plane that bisects the angle created by the long axis of the tooth and the film (Fig. 5-10). This technique is not as accurate as the paralleling technique and should be used as an accessory technique when paralleling technique is uncomfortable.

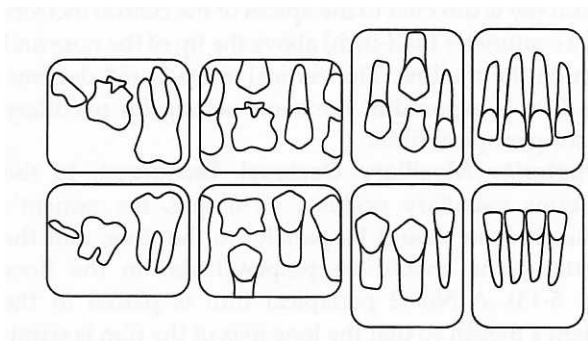
Positioning devices and film holders are also available for this technique. If the beam-aligning devices are not used, prescribed head positions are necessary, which may increase operator error. Even though there are limitations to this technique, in some situations it may be the only usable one.

SPECIFIC DENTAL PROJECTIONS

Periapical Technique. There are essentially two methods of taking periapical radiographs: paralleling and bisecting angle techniques. Each has benefits and limitations when used with the pediatric patient. It is not within the scope of this chapter to describe these techniques in detail. Thorough understanding will allow the practitioner to use them to the best advantage in any given situation.

Regardless of which technique is used, film positioning for the two techniques is identical. In general, films are positioned so that all areas of concern can be visualized, and there will usually be multiple views of a particular area. This is beneficial when minor technical errors exclude an area of importance on one film, since it can be visualized on another.

Regional film positions for the maxilla and mandible are identical; that is, film position for the maxillary molar projection is identical to that for the mandibular molar. Therefore only the regional positions are explained (Fig. 5-11). In all cases the identification dot is placed toward the occlusal surface.



f I G 5-11. Positioning landmarks for film placement. *(Courtesy Mr. Mark Dirlam.)*

Molar Projection. The unfolded film packet is positioned so that all of the third molar, the second molar, and all or part of the first molar are recorded. Thus the anterior edge of the film packet is usually located at the mesial contact of the first molar. The plane of the packet should be parallel to the buccal surfaces of the molars.

Premolar or Primary Molar Projection. The packet may be folded anteriorly for relief in the palate or floor of the mouth and positioned so that the first molar, the first and second premolars or first and second primary molars, and the distal surface of the canine are recorded. The film packet is placed parallel to the buccal surfaces of the premolars or primary molars.

Permanent or Primary Canine Projection. The film packet is usually bent, particularly for a younger child, by a long narrow bend of the part of the packet that will be toward the midline. The canine and lateral incisors are to be recorded in their entirety. The film should be positioned so that the central beam of x-rays is parallel to the proximal surfaces of the canine and lateral incisor and therefore perpendicular to the film.

Permanent or Primary Incisor Projection. If the film packet must be folded because of the narrowness of the arch, a $^{1}/_{s}$-inch bend should be made on each edge throughout the entire length of the film that parallels the long axis of the teeth. The film packet is positioned so that the central incisors are centered mesiodistally on the film. The central ray is parallel to the contacts of the proximal surfaces and perpendicular to the film.

Anterior Maxillary Occlusal Technique. In the anterior maxillary occlusal technique, the patient's occlusal plane should be parallel to the floor, and the sagittal plane should be perpendicular to the floor (Fig. 5-12). A No. 2 periapical film is placed in the patient's mouth so that the long axis of the film runs from left to right rather than anleroposleriorly and the midsagittal plane bisects the film. The patient is instructed to bite lightly to hold the film. The anterior edge of the film should extend approximately 2 mm in front of the incisal edge of the central incisors. The central ray is directed to the apices of the central incisors and a centimeter (half-inch) above the tip of the nose and through the midline. The vertical angle is +60 degrees. This film is exposed at the usual setting for maxillary incisor periapical films.

Posterior Maxillary Occlusal Technique. In the posterior maxillary occlusal technique, the patient's occlusal plane should be parallel to the floor, and the sagittal plane should be perpendicular to the floor (Fig. 5-13). A No. 2 periapical film is placed in the patient's mouth so that the long axis of the film is parallel to the floor. The anterior edge of the film should extend just mesial to the canine. The outer buccal edge of the film should extend approximately 2 mm beyond the primary molar crowns. The patient is instructed to bite lightly to hold the film. The central ray is directed toward the apices of the primary molars as well as interproximally. The vertical angle is +50 degrees. The film is exposed at the usual setting for maxillary premolar periapical films.



FIG 5-12. Anterior maxillary occlusal. A, Technique. B, Radiograph.

Anterior Mandibular Occlusal Technique. The film placement for the anterior mandibular occlusal technique is identical to that for the anterior maxillary

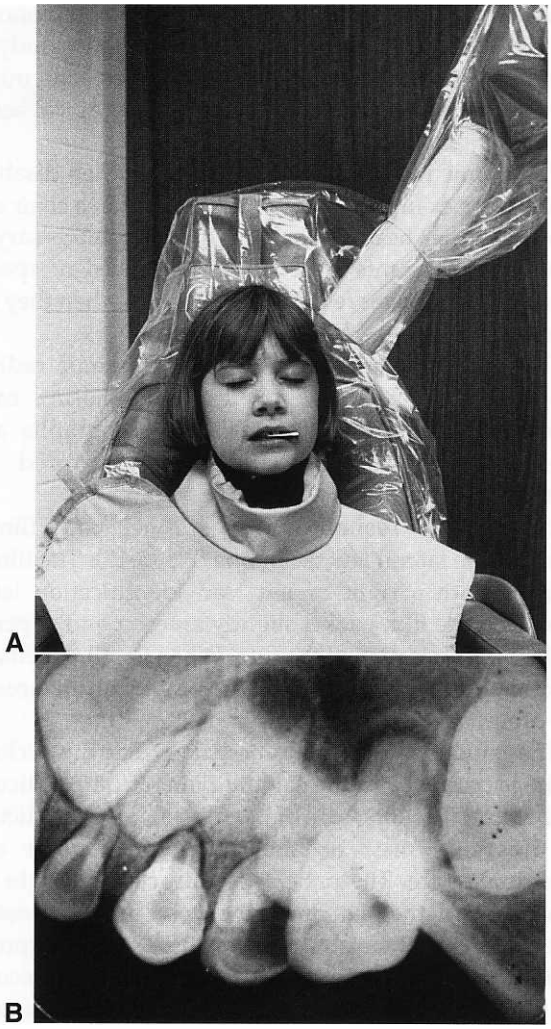
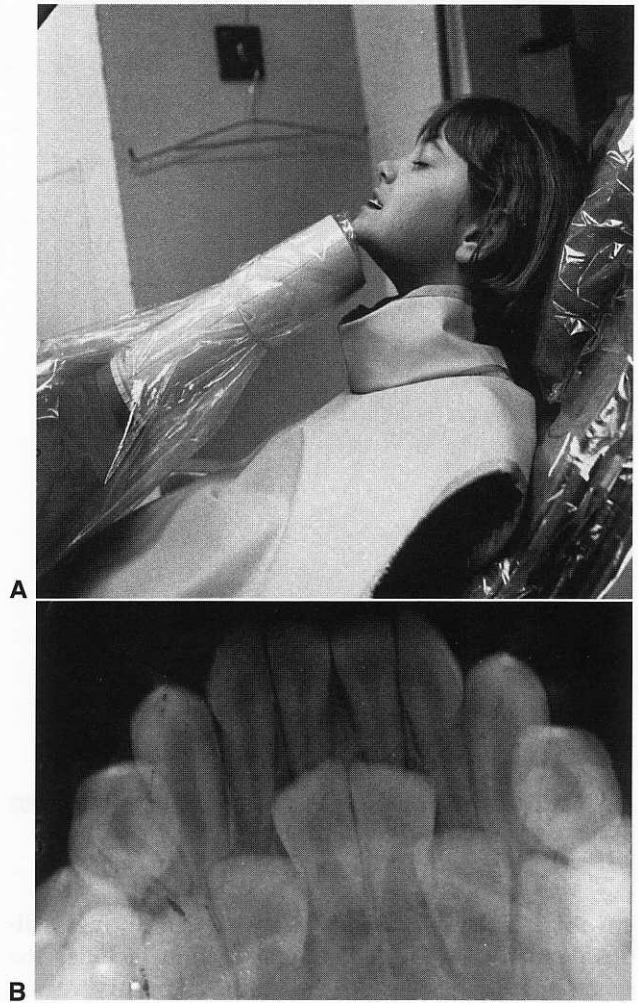


FIG **5-13.** Posterior maxillary occlusal. A, Technique. **B,** Radiograph.



F I G **5-14.** Anterior mandibular occlusal. **A,** Technique. **B,** Radiograph.

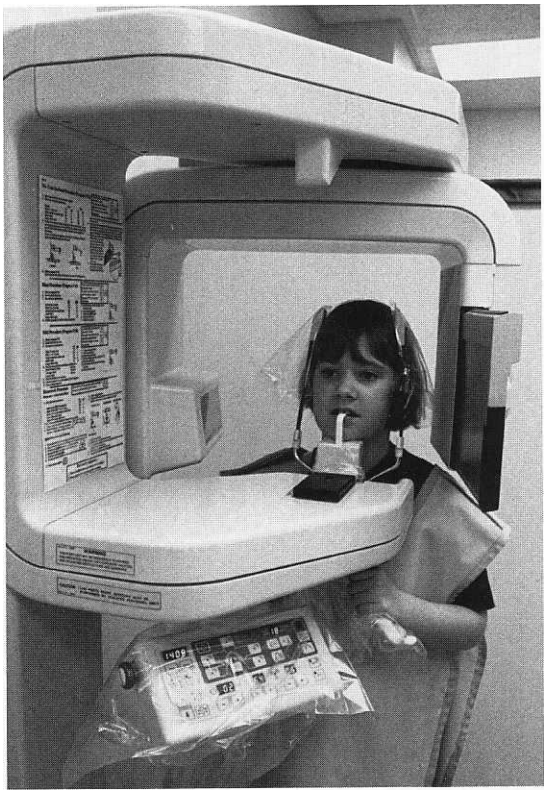
occlusal technique, except that the film must be placed so that the tube side faces the x-ray source (Fig. 5-14). In addition, when the patient occludes on the film, the anterior edge of the film is 2 mm beyond the incisal edge of the lower incisors. The patient's head is positioned so that the occlusal plane is at a -45-degree angle. The cone is then aligned at a -15-degree vertical angle, and the central ray is directed through the symphysis.

PANORAMIC RADIOGRAPHY

Numerous panoramic x-ray units are available to the dental profession. Fig. 5-15 illustrates the use of the Planmeca Proline PM2002 CC panoramic unit (Planmeca). Body-section radiography uses a mechanism by which the x-ray film and the source of the x-rays move simultaneously in opposite directions at the same speed. The reader is directed to textbooks by Langland et al (1989) and Miles et al (1999) for a more definitive discussion of the principles of panoramic imaging.

A panoramic radiographic unit can be used for examination of children. Since the examination is obtained without placement of the film in the mouth, it does not alarm the anxious child who may refuse an intraoral film. Moreover, the young patient may find the movement entertaining or fun. The diversion of being momentarily entertained usually invites cooperation. However, staying completely immobile for 15 seconds may not be possible for some very young children.

Although panoramic radiography is considered a supplement to, rather than a substitute for, the intraoral periapical radiographic series, it does provide excellent



f**IG. 5-15.** Patient positioned in a Planmeca Proline PM2002 CC panoramic unit.

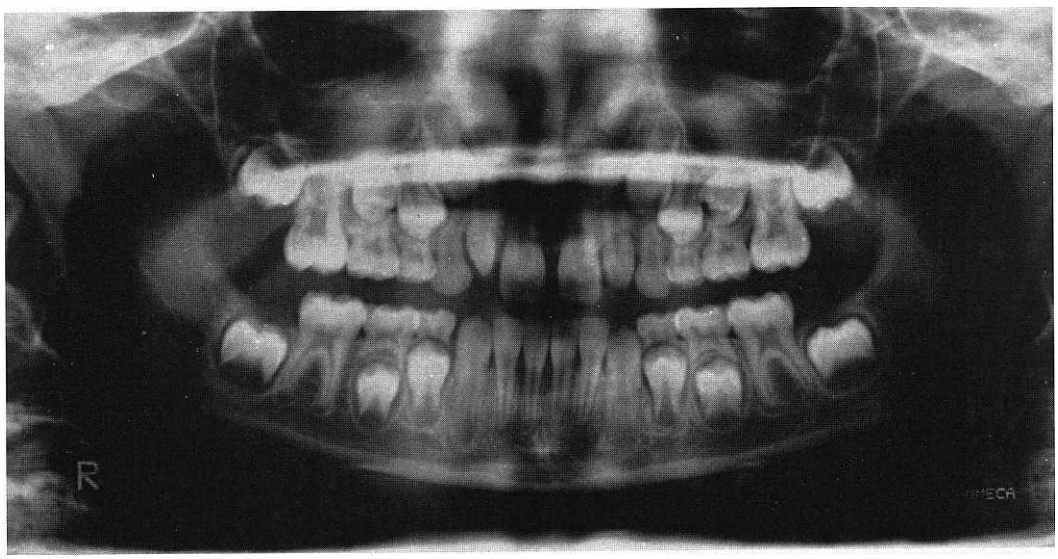
coverage of the structures that are viewed during pediatric dental diagnosis. A diagnostic film includes the teeth, the supporting structure, the maxillary region extending to the superior third of the orbit, and the entire mandible including the temporomandibular joint region. Pediatric dentists who have used panoramic radiographic technique have discovered condylar fractures, traumatic cysts, and anomalies that might have gone undetected with the routine periapical series of radiographs (Fig. 5-16).

Panoramic radiology can be valuable when disabled patients are examined if the patient can sit in a chair and hold his or her head in position. It may be necessary to administer relaxants or sedatives to palsied or spastic patients, who are more difficult to control when they are emotionally charged by the dental visit.

The only inherent drawback to panoramic radiography is lack of image detail for diagnosing early carious lesions. Adjunct bite-wing radiographs and selected periapical radiographs are required for that task.

Lateral Jaw Technique. A 5 x 7-inch x-ray film is used for the lateral jaw technique (Fig. 5-17). The film is marked with a right or left lead identification letter placed on the film packet slightly anterior and superior to the central portion of the film. When the film is finally positioned, this letter should be located in the area of the orbit.

The patient's head is positioned so that the occlusal plane is parallel and the sagittal plane is perpendicular to the floor. The long axis of the film, also perpendicular to the floor, rests on the patient's shoulder and against the face. The patient is instructed to rotate the head toward the film until the nose rests against it. Then the chin is raised and the head tilted approxi-mately 15 degrees toward the film. The patient secures the film with the palm of the hand and with fingers extended. The cone is positioned so that the central x-ray beam enters at a point a half-inch behind and



f**IG 5-16.** Panoramic radiograph. Notice the congenitally missing mandibular second premolars.

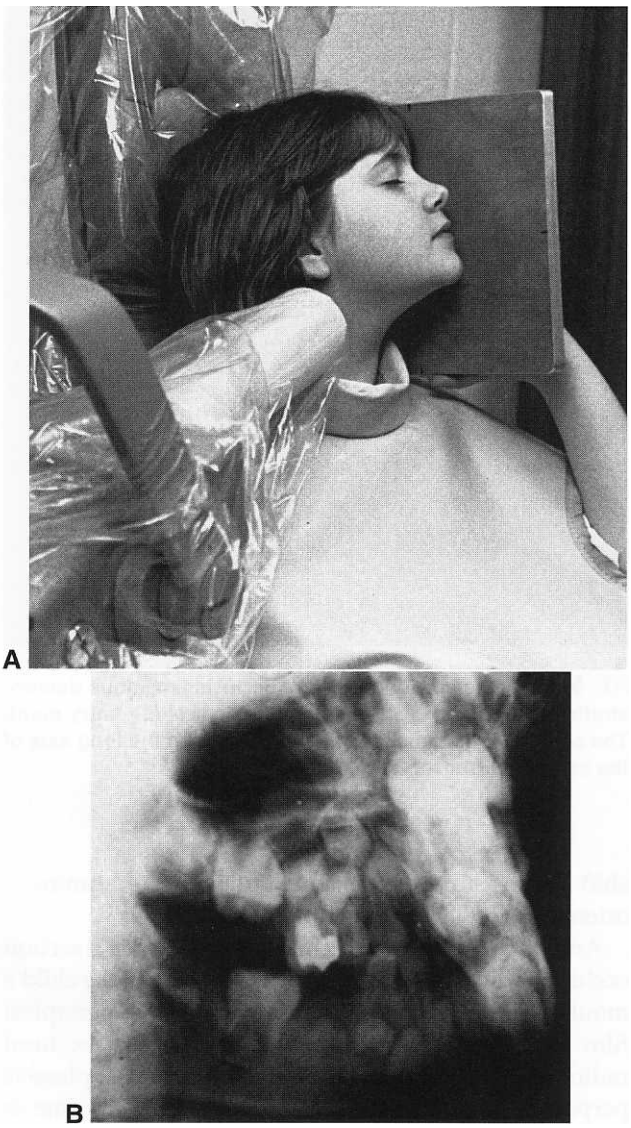


FIG **5-17.** Lateral view of jaw. **A,** Technique. **B,** Radiograph.

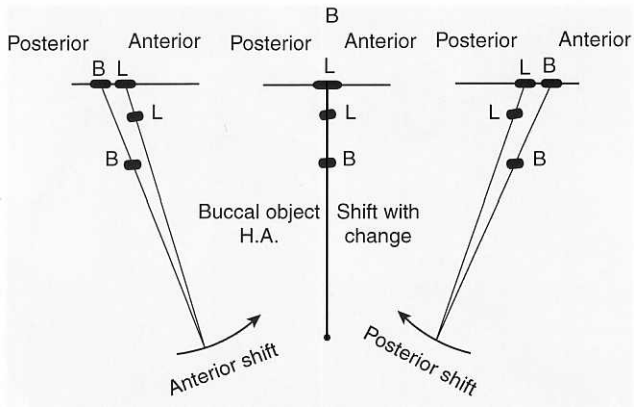
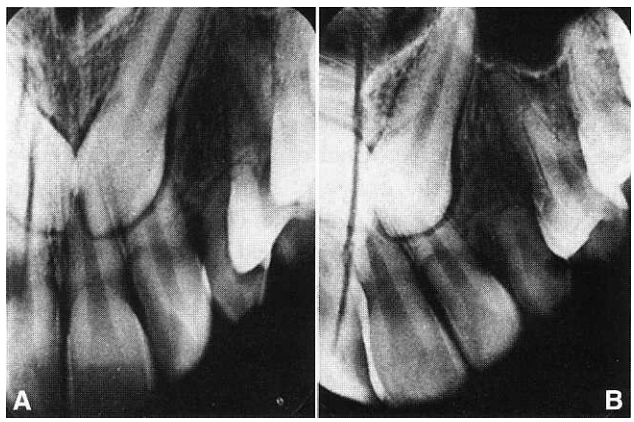


FIG **5-18.** The buccal object rule. B, Buccal; L, lingual; H.A., horizontal angle.



f **G . 5-19. A,** Shadow of the maxillary permanent canine is superimposed over the central incisor root. **B,** When the horizontal angle of the x-ray tube is shifted posteriorly, the crown of the unerupted canine appears to move posteriorly. The canine is lingually placed.

below the angle of the mandible on the side opposite the film. The vertical angle is -17 degrees. The central x-ray beam is perpendicular to the horizontal plane of the film.

LOCALIZATION TECHNIQUES

One method of localizing embedded or unerupted teeth uses the buccal object rule, which states that the image of any buccally oriented object appears to move in the opposite direction from a moving x-ray source. On the other hand, the image of any lingually oriented object appears to move in the same direction as a moving x-ray source (Fig. 5-18).

Using this principle for localization, the practitioner makes two radiographs of the unerupted tooth. The technique consists in positioning the patient's head so that the sagittal plane is perpendicular to the floor and the ala-tragus line is parallel to the floor. An intraoral periapical film is placed in the mouth and then exposed. A second film is placed in the mouth in the same position as the first film, with the patient's head position remaining the same. The second film is then exposed. The vertical angulation should be the same for each exposure. However, the horizontal angle is shifted either anteriorly or posteriorly, depending on the area being examined, for the second view.

Fig. 5-19, A, illustrates an unerupted maxillary permanent canine. The shadow of this tooth is superimposed over the central incisor root. When the horizontal angle



E **1 G 5-20. A,** Shadow of the maxillary permanent canine covers a small portion of the lateral incisor root on its distal aspect. **B,** When the horizontal angle of the x-ray tube is shifted posteriorly, the canine appears to move anteriorly. The canine lies buccal to the erupted teeth.

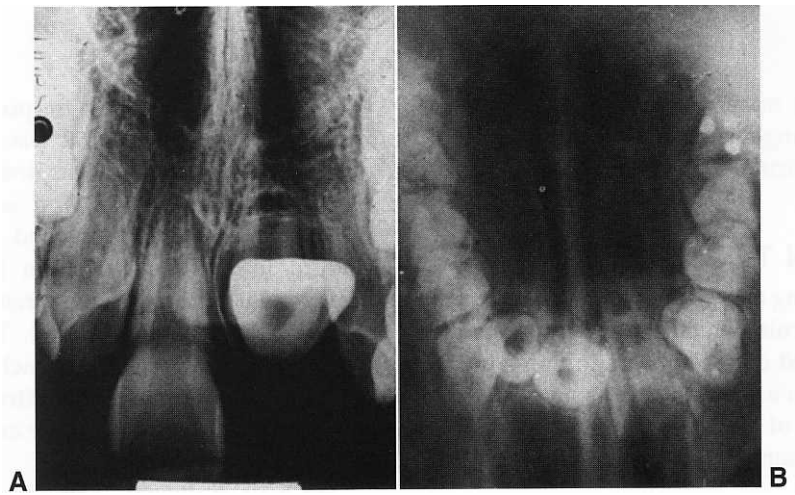
of the x-ray tube is shifted posteriorly (Fig. 5-19, *B),* the crown of the unerupted canine also seems to move posteriorly, and the image of the canine crown is no longer superimposed over the central incisor root. If the buccal object rule is applied, it can be seen that the embedded canine is oriented lingually to the erupted teeth.

Fig. *5-20, A,* illustrates an embedded maxillary permanent canine. The shadow of the crown of this tooth covers a small portion of the lateral incisor root on its distal aspect. When the horizontal angle is shifted posteriorly (Fig. *5-20, B),* the unerupted crown appears to move anteriorly or in a direction opposite to the shift of the x-ray source. Thus the unerupted canine is oriented buccally to the erupted teeth.

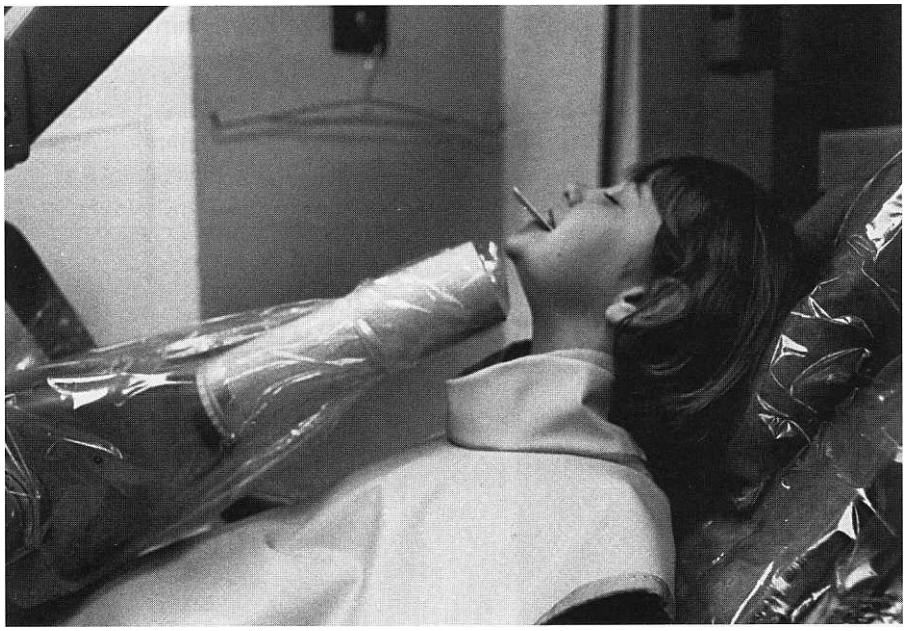


H G. **5-21.** Cross-section maxillary occlusal technique demonstrating the proper head position and central-ray entry point. The central ray is directed coincidentally with the long axis of the central incisor roots.

Another localization technique is the cross-section occlusal radiograph. Depending on the size of the child's mouth, either the adult occlusal or a No. *2* periapical film may be used. To obtain a cross-section occlusal radiograph of the maxilla, the patient's sagittal plane is perpendicular to the floor, and the ala-tragus line is



f **I G 5-22.** A, Dilacerated maxillary central incisor. **B,** The crown of the dilacerated central incisor lies labially.



f **I G 5-23.** Cross-section mandibular occlusal technique demonstrating the proper head position and vertical angulation of the x-ray head.

parallel to the floor. The patient is asked to occlude lightly on the film. The central ray is projected through the midsagittal plane and enters the skull 1 cm posterior to bregma. (Bregma is the point at which the sagittal suture meets the coronal suture.) The proper vertical angulation is determined when the central ray is directed through the long axis of the maxillary central incisor roots (Fig. 5-21).

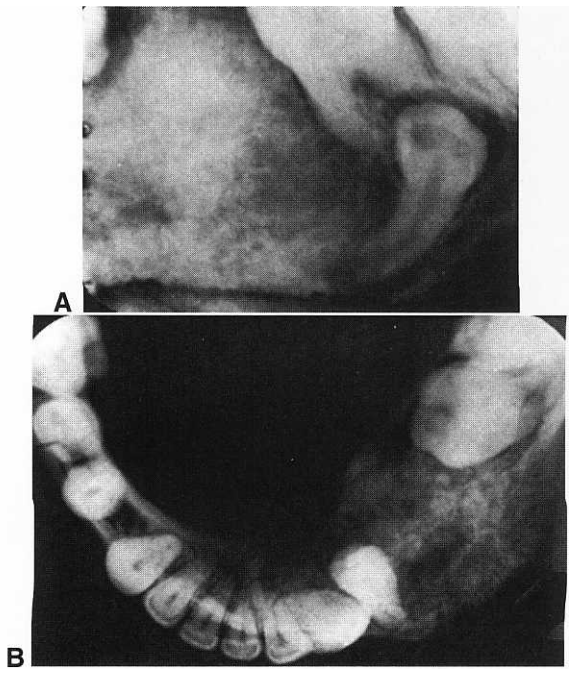
A maxillary central incisor periodical radiograph demonstrating a grossly dilacerated central incisor may be seen in Fig. *5-22, A.* It is important to determine, before a surgical procedure, if the crown of such a tooth is in a labial or a lingual position. In this instance, the crown is positioned labially (Fig. 5-22, *B).*

The cross-section occlusal film can be employed for localization of anomalies in the mandible. Either the adult occlusal or a No. 2 periapical film can be used. The patient's head is tilted backward sufficiently to direct the central ray through the long axis of the erupted teeth (Fig. 5-23). The patient's head must be tilted on its long axis to accommodate positioning of the x-ray tube. If determination of the buccal or lingual position of an impacted mandibular third molar is desired, the central ray should be projected through the long axis of the mandibular second molar. If it is necessary to localize an unerupted second premolar, the central ray should be directed through the long axis of the first premolar. Fig. *5-24, A,* reveals an unerupted mandibular premolar that has been displaced by an ossifying fibroma. In Fig. 5-24, *B,* a cross-section occlusal radiograph demonstrates that the crown of the premolar is lingually placed.

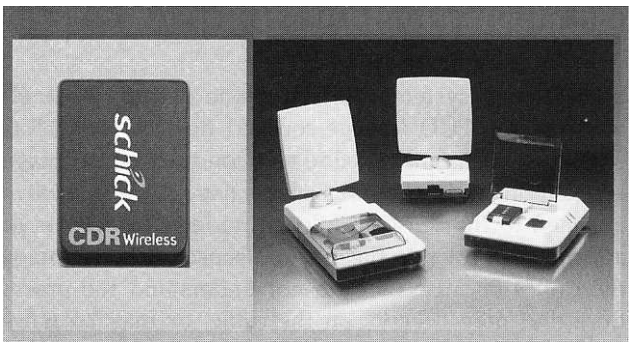
DIGITAL RECEPTORS FOR PEDIATRIC PATIENTS

Digital x-ray systems use solid-state detector technology such as CCD (charge-coupled devices) or CMOS (complementary metal oxide semiconductors) for image acquisition. Most of these systems have electronic wires attached to the sensor. Children under 4 or 5 years of age may not tolerate these wired sensors, may damage the cables because they do not understand the procedure and may "chew" on the cable, or may be more fearful just of the appearance of a wired system. For these reasons a phosphor-based digital x-ray system may be ideal for the pediatric patient. There is one " wireless" solid-state image detector from Schick Technologies, the Schick CDR Wireless sensor (Fig. 5-25). This device has just been introduced to the dental market, so there is not much experience with its use with children as yet.

Photostimulable phosphors (PSPs) or storage phosphors are used for digital imaging for image acquisition. Unlike panoramic or cephalometric screen materials, PSPs do *not* fluoresce instantly to produce light photons. Instead, these materials store the incoming x-ray photon information like a latent image in conventional film-based radiography until the plates are scanned by a laser beam in a drum scanner. The laser scanning excites the phosphor to give up



f I G . **5-24. A,** Unerupted mandibular premolar displaced by an ossifying fibroma. **B,** The crown of the unerupted premolar is lingually oriented.



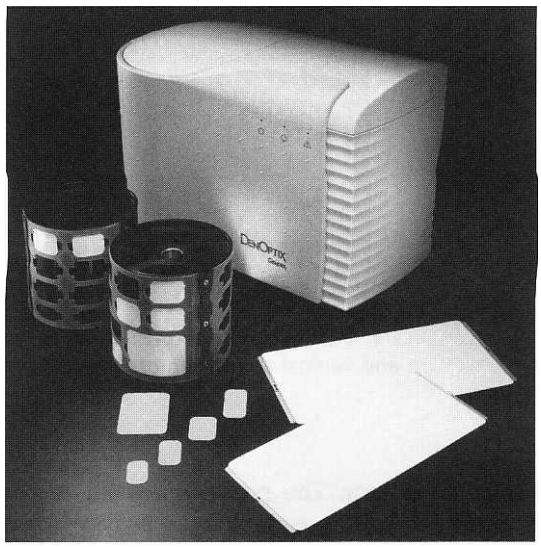
f **I G 5-25.** The Schick Technologies CDR Wireless system showing the sensor (left) and the charging unit and wireless RF antennas (right).

the stored energy as an electronic signal, which is then digitized, with various gray levels assigned to points on the curve to create the image information. The currently available phosphor imaging systems (Table 5-2) are from Soredex (Digora.), AirTechniques (ScanX) and Gendex (DenOptix).

These phosphor plates have no wire to the computer and resemble intraoral film in every way, including size, thickness, rigidity, and receptor placement. They are soft and flexible much like film, and as with

TABLE **5-2** Photostimulable Phosphor Systems

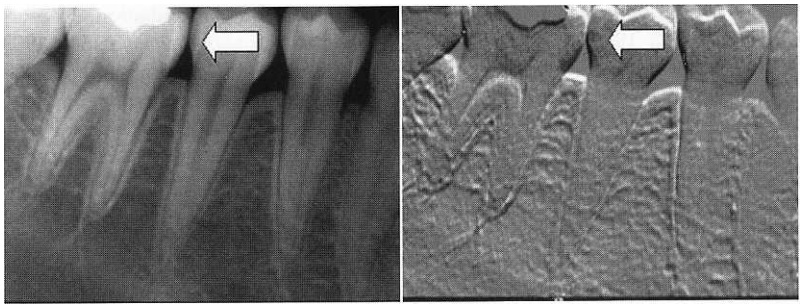
COMPANY	SYSTEM	PHOSPHOR TYPE	RESOLUTION (LP/mm)
Soredex	Digora	Agfa	>6
AirTechniques	ScanX	Fuji	6-8
Gendex	DenOptix	Fuji	~9



f **I G 5-26.** The Dentsply-Gendex DenOptix phosphor plate scanner.

conventional x-ray film techniques, infection control procedures must be employed. The plates must be wrapped, exposed, and unwrapped and then carefully shaken out of the barrier envelope onto a clean surface, wiped with a disinfectant, and fed into the laser scanner. After use, the plates must be exposed to light to eliminate any latent image before they are placed into new barrier envelopes for the next patient. Thus the total time to process each image can take several minutes. Unlike film, however, the plates should *not* be curved or bent, because a permanent defect or artifact will be created in the phosphor coating. The plate would then need to be replaced.

In some machines like the DenOptix system the plates are put into a template holder that is then placed into the drum scanner (Fig. 5-26). After the scanning process is completed, the resultant digital image can then be viewed on a monitor in about 30 seconds to 2.5 minutes depending on the system. This is called analog-to-digital conversion and it is how the phosphor imaging system becomes "digital." The technology is not new. This type of signal readout by laser scanning has been used in the laboratory industry



f **I C 5-27.** An interproximal carious lesion on the distal surface of tooth No. *29 (left)* made more apparent by electronic image processing *(right).*

for reading biologic fluid samples for decades. This " digital" imaging technique requires two steps to retrieve the final image, just like film. The technique also requires the same two-stage infection control procedures as film. But the final image is digital and can be subjected to electronic image processing to extract disease features more readily for treatment decision making (Fig. 5-27). An excellent explanation of the PSP process appears in the April 2000 issue of *Dental Clinics of North America.*

The advantage of these digital-like phosphors is really in the image processing that can be applied *after* the image is acquired. Although electronic image processing can be performed on conventional x-ray film, the film must first be converted to digital information by scanning the image. Storage phosphors (PSPs) and true digital images from solid-state detectors like CCD and CMOS receptors provide a virtually instantaneous digital image and require no additional data conversion prior to electronic image processing.

If the dentist wishes to employ these digital systems, either solid state or phosphor, he or she must use a good paralleling instrument system and paralleling technique to be successful.

INTERPRETATION

When interpreting radiographs, the dentist must develop a systematic approach so that no areas of the radiographs are missed. No one way is necessarily better than another, provided that the approach is consistently followed. Viewing conditions should be adjusted so that the maximum information can be obtained from the radiographs. The radiographs should be placed in a mount that does not allow light to be transmitted through the periphery, only through the film itself. Other extraneous light from around the mount should also be masked out. It is preferable for the room in which the radiographs are reviewed to have subdued lighting. Finally, it is recommended that a magnifying glass be used. If these viewing conditions are followed, more information will be obtained, and thus better care will be provided for the patient.

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CHAPTER

6

Clinical Genetics for the Dental Practitioner

JAMES K. HARTSFIELD, JR.

DAVID BIXLER

انتشارات
شایان نمودار

CHAPTER OUTLINE

Review of Genetic Principles
CELL DIFFERENTIATION AND DEVELOPMENTAL BIOLOGY
CHROMOSOMES
HEREDITARY TRAITS IN FAMILIES
DEVELOPMENTAL BIOLOGY OF ENAMEL
AUTOSOMAL DOMINANT INHERITANCE
AUTOSOMAL RECESSIVE INHERITANCE X-LINKED OR SEX-LINKED INHERITANCE
    X-Linked Dominant
    X-Linked Recessive
VARIATION IN GENE EXPRESSION
    Penetrance
    Expressivity
MULTIFACTORIAL INHERITANCE
    Multifactorial Inheritance in Human Diseases
Influence of Genetic Factors on Major Craniofacial, Oral, and Dental Conditions
GENETICS AND DENTAL CARIES
GENETICS AND PERIODONTAL DISEASE
    Early-Onset Periodontitis
GENETICS OF MALOCCLUSION
EXTERNAL APICAL ROOT RESORPTION
GENETICS OF CLEFT LIP AND PALATE

The purpose of this chapter is twofold: to review genetic principles and to mention a few examples of the influence of genetic factors on major craniofacial, oral, and dental conditions. As the basis for relatively rare developmental dysplasias, diseases, and syndromes which show a genetic cause or marked genetic influence becomes known, increasing attention is being paid to those genetic factors that influence (or are associated with) more common conditions. An increased appreciation of how genetic factors interact with environmental (nongenetic) factors to influence growth and pathology will lead to an increased understanding of pathogenesis and the recognition that some groups or individuals may be more susceptible.

Review of Genetic Principles

The genome contains the entire genetic content of a set of chromosomes present within a cell or an organism. Within the genome are genes that represent the smallest physical and functional units of inheritance that reside in specific sites (called loci, or locus for a single location). A gene can be defined as the entire DNA sequence necessary for the synthesis of a functional polypeptide molecule (production of a protein via a messenger RNA intermediate) or RNA molecule (transfer RNA and ribosomal RNA). Genotype generally refers to the set of genes that an individual carries and, in particular, usually refers to the specific pair of alleles (alternative forms of a particular gene) that a person has at a given location (locus) of the genome. In contrast, phenotype is the observable properties and physical characteristics of an individual, as determined by the individual's genotype and the environment in which the individual develops over a period of time.

Remarkable advances in the biochemical techniques that are used to study cell molecular biology and DNA have taken today's researcher to the threshold of understanding the regulation of cell functions. To illustrate, not so long ago DNA analyses were performed on minute amounts (picograms) of DNA. This limitation was necessary because there was so little DNA available for study in the samples. When the DNA polymerase enzyme was discovered that could replicate DNA through the polymerase chain reaction and make it by the gram, this sample problem disappeared. This advance facilitated completion of the human genome project, which resulted not only in definition of a single human genome sequence composed of overlapping parts from many humans, but also in an expanding catalogue of some 1.4 million sites of variation in the human genome sequence. These variations (or polymorphisms) may be used as markers to perform genetic analysis (including analysis of genetic-environmental interaction) in human beings. The genome varies from one individual to the next most often in terms of single base changes of the DNA, called *single nucleotide polymorplrisms* (SNPs, pronounced "snips"). The main use of this human SNP map will be to determine the contributions of genes to diseases (or nondisease phenotypes) that have a complex, multifactorial basis)

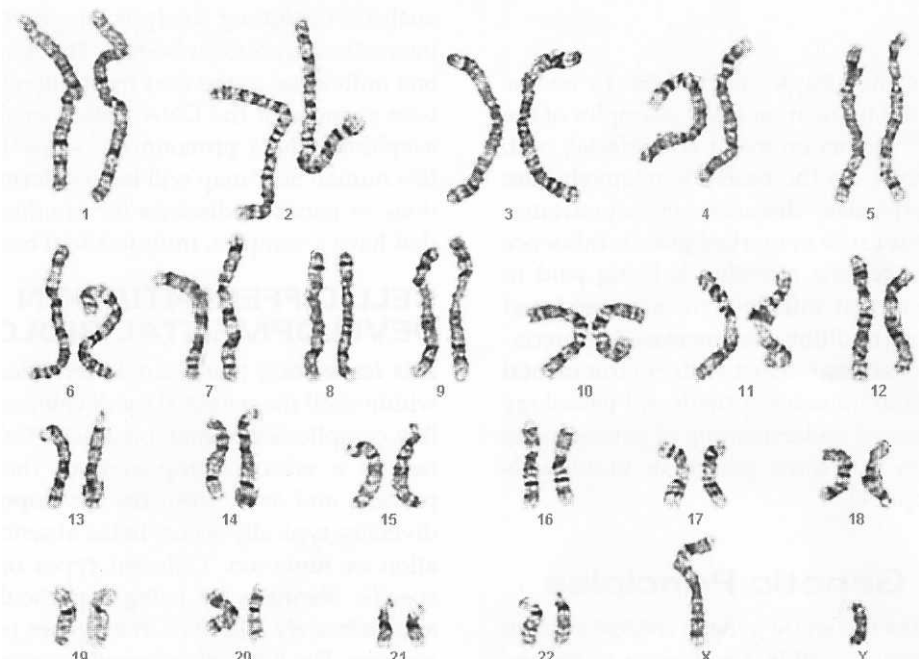
CELL DIFFERENTIATION AND DEVELOPMENTAL BIOLOGY

It is fascinating that a single fertilized ovum contains within itself the potential for development of the incredibly complicated human organism. Cellular differentiation is a critical component of this developmental process, and aside from the development of antibody diversity, typically occurs in the absence of genetic alteration or mutation. Different types of cells gain their specific identities by using a particular subset of the approximately 30,000 or more genes present within the genome. The types of polypeptides a cell can synthesize include enzymes, which catalyze various activities of cellular metabolism and homeostasis; structural proteins, which form the intracellular and extracellular scaffolding or cellular matrix; and regulatory proteins, which convey signals from the outside of the cell to the nucleus and modulate or control specific gene expression. In a developing embryo, cells reside in a three-dimensional environment and are responsive to signals from themselves (autocrine), from nearby sources (paracrine), and from anatomically distant sources (endocrine). Many of these signals are mediated by soluble molecules (either peptide or nonpeptide in origin) that bind to specific receptors (proteins) that are present on the surface or on the inside of cells. In addition to signals from soluble factors, cells can respond to cell-to-cell or cell-to-extracellular matrix signals.'

The action of "turning on" or "turning off" specific genes, referred to as regulation of gene expression, is care -fully orchestrated and remains a critical element in determining cell specificity and tissue morphogenesis. Transcription factors bind to DNA and either facilitate or suppress initiation of gene transcription, the most common control point of gene expression. In the development of the craniofacial complex there is increasing evidence for the role of homeobox-containing gene families that encode transcription factors. These then are critical for the control of complex interactions between genes that are subsequently expressed during development.'

In summary:

1. The genetic message lies in the DNA itself, which is coded and transmitted from cell generation to cell generation when these DNA molecules are replicated (or duplicated).



f C. **6-1.** Banded karyotypes of a normal human male *(left)* and female *(right).* Group designations according to Denver nomenclature are also indicated. *(Courtesy Cytogenetic Laboratories, Indiana University School of Medicine.)*

2. A given cell type and function is defined by what specific RNA molecules are made from the DNA master. These RNA molecular copies direct protein synthesis in the cell.
3. Transcription factors determine which genes are expressed through the production of the RNA and subsequent protein.
4. Development occurs through the action of specific transcription factors on specific genes that need to be expressed next in time.

CHROMOSOMES

DNA is grouped into units called chromosomes. Humans have 46 chromosomes that contain an estimated 30,000 genes, including numerous duplicates. Of the 46 chromosomes, the sex chromosomes are the X and Y, with the remaining 44 chromosomes referred to as autosomes. Each autosome has a paired mate that is referred to as its homologue. Therefore, with the exception of some of the genes on the X and Y chromosomes in males, there are at least two copies of each gene unless a piece of DNA is deleted. Thus the human chromosome complement consists of 23 pairs of chromosomes (one pair of sex chromosomes and 22 pairs of autosomes).

One area of special interest to the clinician is cytogenetics, the study of chromosomes. This interest has been stimulated by the development of techniques in which cells are grown in culture and the chromosomes are examined under a microscope for changes in size, shape, and fine structure. This is called *karyotyping.* Fig. 6-1 shows the karyotypes of a normal human male and female. By applying this technique, Lejeune, Turpin, and Gautier demonstrated that the fundamental cause in Down syndrome is the presence of an extra specific chromosome (number 21) in the affected individual's karyotype.[4] When an entire extra chromosome is present, the condition is called a trisomy of the chromosome in question, for example, trisomy 21 for Down syndrome. Fig. 6-2 shows the karyotype of a male who has Down syndrome. The extra chromosome in the group of number 21 and number 22 chromosomes is readily apparent.

Since this report in 1959, many disease states have been shown to be associated with an incorrect chromosome complement. By using this approach with considerable refinement, it was shown that alterations in the fine structure of chromosomes, as well as in their number, could be present. Monosomy of an autosome, or a missing autosomal chromosome, had not been believed to be compatible with life, but several monosomies in live-born children have now been reported. Monosomy of the sex chromosomes can be compatible with life and typically affects development of both internal and external sex organs of the individuals. The best-known example of this is Turner syndrome, which occurs in approximately 1 in every 5000 live female births. These persons are phenotypic females who are usually missing one of the X chromosomes and are

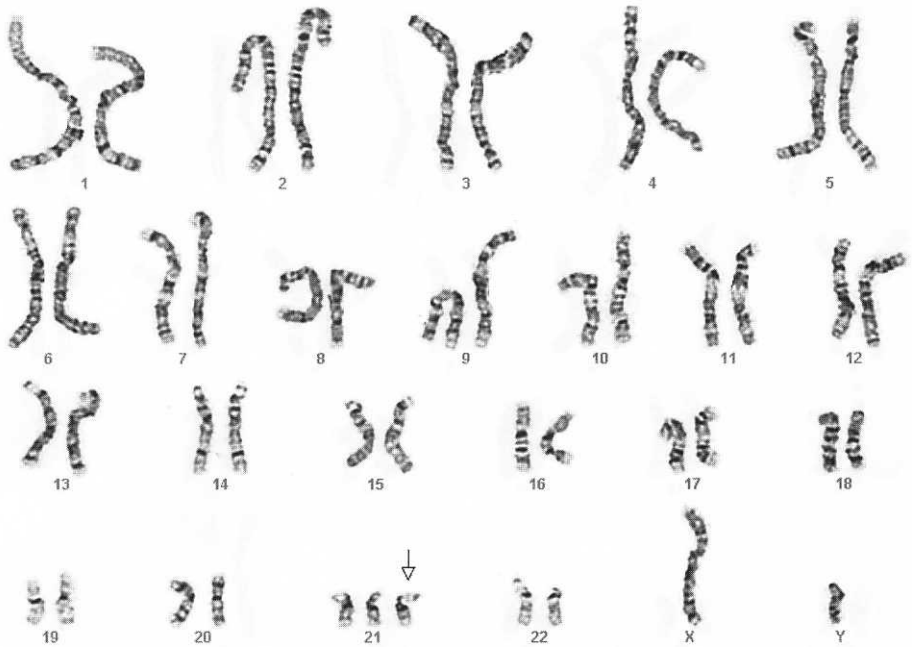


FIG 6-2. Banded karyotype of a male with trisomy of chromosome 21 (Down syndrome). *(Courtesy Cytogenetic Laboratories, Indiana University School of Medicine.)*

chromosomally designated as 45,X. Other aberrations of the X chromosome may also cause Turner syndrome. Affected individuals are typically short of stature, lack secondary sex characteristics, and are sterile. The Turner syndrome karyotype is shown in Fig. 6-3. Table 6-1 lists a few of the more common chromosomal aberrations that produce clinical disease, including examples of translocations (the attachment of a broken piece from one chromosome to another, but not homologous, chromosome), and deletions (the absence of a piece of chromosome).

Chromosome abnormalities are an important cause of spontaneous abortion. About 15% of all recognized pregnancies end in spontaneous abortion, and the incidence of chromosome abnormalities in such abortions is greater than 50%. Only 0.3% to 0.5% of all live-born infants have a chromosome abnormality. Clinicians agree that, even though these aberrations are relatively rare, they remain a significant cause of mental and physical abnormalities in humans.

HEREDITARY TRAITS IN FAMILIES

Heritability is the proportion of the total phenotypic variance in a **sample** that is contributed by genetic variance.' On an individual basis for a binary trait (i.e., a disease or trait that an individual either has or does not have), heritability is not the proportion of disease or the trait attributable to, or caused by, genetic factors. For a quantitative trait, heritability is not a measure of the proportion of an individual's score attributable to genetic factors.' A trait with a heritability of 1 is said to be expressed without any environmental influence, whereas a trait with a heritability of 0.5 has half its variability (from individual to individual) influenced by environmental factors and half by genotypic factors. Values over 1 may occur because the methodology provides an estimate of heritability under several simplifying assumptions that may be incorrect. Still, the estimation of heritability can provide an indication of the relative importance of genetic factors. Confirming that there is a certain degree of genetic influence on a trait is a preliminary step to performing further specific genetic linkage studies (using DNA markers) to deter-mine areas of the genome that appear to be associated with the characteristics of a given trait.'

When hereditary traits in families are to be studied, it is convenient to think of three classes of genetically influenced traits. These are (1) monogenic (2) polygenic, and (3) multifactorial. The following summarizes the characteristics of these three classes.

Monogenic traits are traits produced and regulated by a single gene locus. Usually they are relatively rare in the general population (occurrence in fewer than 1 per 1000 individuals). However, if the appearance of an affected person is striking, there may be instant recognition of the disease, as with patients having albinism, achondroplasia, or neurofibromatosis. Monogenetic conditions often occur in families and show transmission

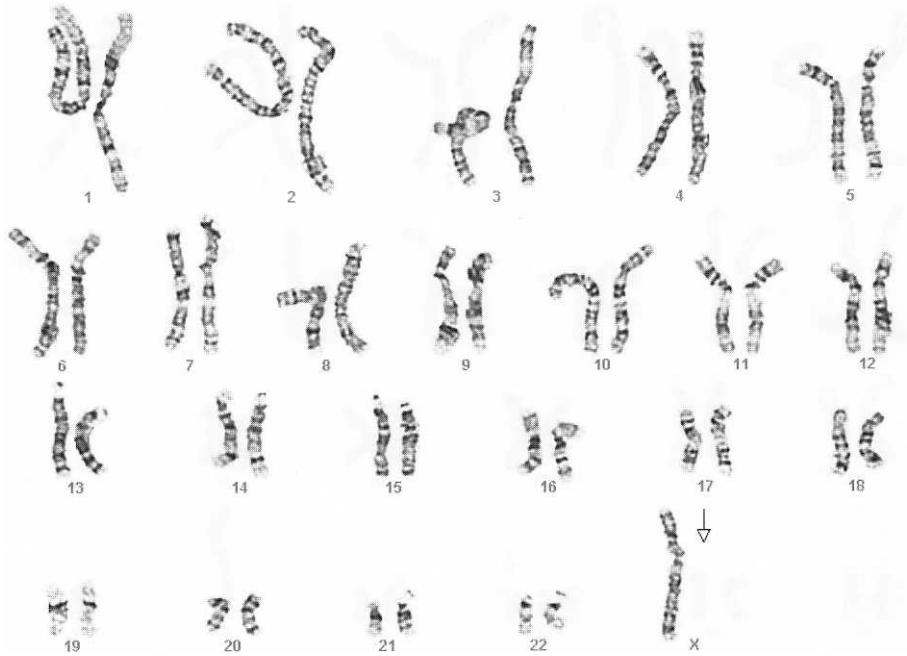


FIG **6-3.** Banded karyotype of a female with missing X chromosome (Turner syndrome). *(Courtesy Cytogenetic Laboratories, Indiana University School of Medicine.)*

TABLE **6-1** A Summary of the More Common Chromosomal Aberrations

TYPE	SPECIFIC ALTERATION	CLINICAL RESULT
Aneuploidy	Trisomy 21	Down syndrome
	Trisomy 18	Edwards syndrome
	Trisomy 13	Patau syndrome
	Extra X chromosomes	In females: XXX, XXXX, XXXXX syndromes In males: Klinefelter syndrome—XXY, XXXY, and XXXXY
	Monosomy, autosomal	Usually nonviable
	Monosomy, X chromosome	In females: Turner syndrome, 45,X In males: nonviable, 45,Y
Translocation	14/21, 21/21, or 21/22	Translocation carrier (normal phenotype) or Down syndrome
Deletion	Ring chromosome	Variable
	Short arm chromosone No. 5	Cri du chat syndrome
	Philadelphia chromosome (No. 22)	Chronic myeloid leukemia

characteristics of the mendelian (dominant or recessive) traits.

Polygenic traits, too, are hereditary and typically exert influence over rather common characteristics such as height, skin, and intelligence. This influence takes place through many gene loci collectively asserting their regulation of the trait. Although each gene involved has a minimal effect by itself, the effect of all the genes involved is additive. The associated phenotype is rarely discrete and is most commonly continuous or quantitative. Because these traits show a quantitative distribution of their phenotypes in a population, they do not show mendelian inheritance patterns. Specially developed mathematical models have been devised to analyze such traits in populations, but these are not discussed here. However, it is important to note that the very nature of their control (multiple genes each with a small additive effect) dictates that their environment may readily influence them. Monogenic traits are not readily amenable on a large scale to environmental

modification, although there can be variation, presumably secondary to other genetic and environmental factors. By contrast, one can easily think of a dozen environmental factors known to influence height and intelligence quotient.

Finally, the third etiologic group of gene-influenced conditions has been given the name *multifactorial* traits. These traits are controlled by multiple genes but differ significantly from polygenic traits in that control is achieved through an interaction of multiple genes and environmental factors and occurs when a liability threshold is exceeded. Although typically the number of genes involved is many, occasionally a few genes, sometimes only two or three, influence the trait. The effect of these genes on the phenotype is therefore a net effect, not a simple additive one. Furthermore, phenotypic expression approaches that of a discreet mendelian trait and therefore cannot be readily classed as a quantitative trait. Likewise, the effect of a gene influencing the phenotype may not be as great as that of a gene associated with a monogenic trait, but the gene may be referred to as having a major effect. Among the well-known hereditary types of conditions designated as multifactorial are many of the severe nonsyndromic congenital malformations such as cleft lip and palate (CLP), spina bifida-anencephaly, and hip dislocation. More discussion about multifactorial inheritance is given later.

The investigation of human heritable traits usually involves the observation of specific features in a family and the study of that family's pedigree. The affected individual in a family who first brings that family to the attention of the geneticist is called the *proband* or *propositus.* This individual is the index case. Brothers and sisters of the proband are siblings or sibs. Thus, a sibship consists of all the brothers and sisters in a nuclear family unit (parents and their offspring). The clinical appearance in an individual of a given trait, such as eye color or height, is that individual's phenotype, whereas the specific genetic makeup that controls that phenotype is the genotype.

In an earlier section, the point was made that the human chromosome complement has 22 homologous pairs of autosomes and one pair of sex chromosomes. Because of homologue pairing (excluding the X and Y chromosomes in the male), there are at least two copies of each gene, one located at the same position (locus) on each member of the homologous pair. Genes at the same locus on a pair of homologous chromosomes are alleles. When both members of a pair of alleles are identical, the individual is homozygous for that locus. When the two alleles at a specific locus are different, the individual is heterozygous for that locus.

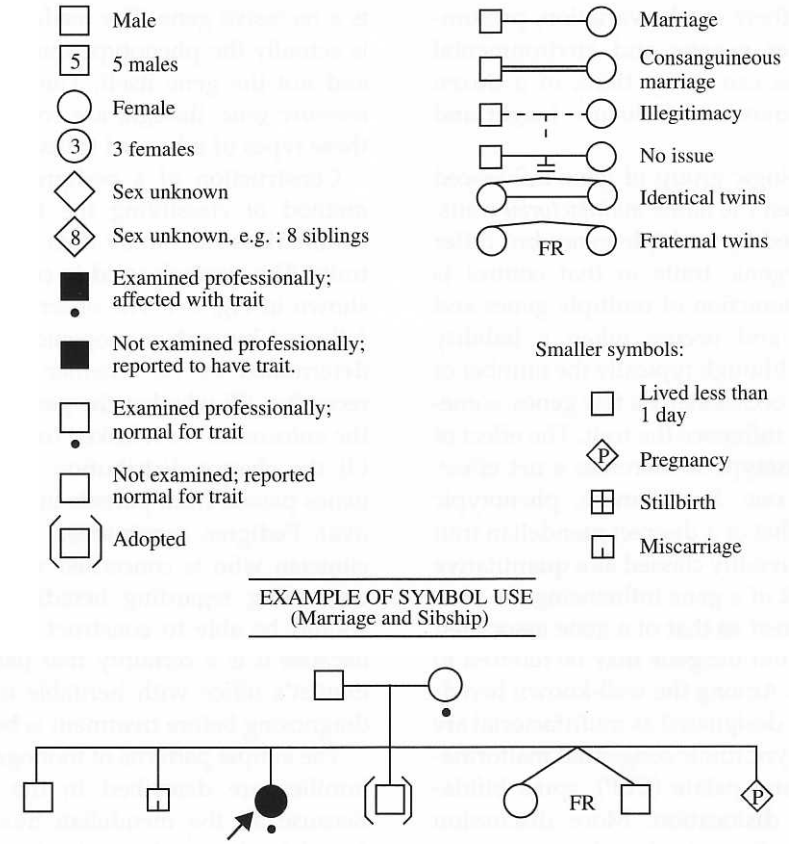
A gene that expresses a particular phenotype in single dose is a dominant gene. If the gene must be present in double dose (homozygous) to express the phenotype, it is a recessive gene. The reader should recognize that it is actually the phenotype that is dominant or recessive and not the gene itself. The terms *dominant gene* and *recessive gene,* though, are commonly used to describe these types of inherited traits in families.

Construction of a pedigree, which is a shorthand method of classifying the family data, conveniently summarizes the family data for the study of inherited traits. The symbols used in constructing a pedigree are shown in Fig. 6-4. The observable inheritance patterns followed by such monogenic traits within families are determined by (1) whether the trait is dominant or recessive, (2) whether the gene is autosomal (on one of the autosomes) or X linked (on the X chromosome), and (3) the chance distribution in the offspring of those genes passed from parents in their gametes (sperm and ova). Pedigree construction is a valuable tool for the clinician who is concerned with the diagnosis of and counseling regarding hereditary traits. Every dentist should be able to construct and interpret a pedigree, because it is a certainty that patients will come to the dentist's office with heritable oral diseases that need diagnosing before treatment is begun.

The simple patterns of monogenic inheritance seen in families are described in the following discussion. Because all the mendelian modes of inheritance are found in the amelogenesis imperfecta (AI) disorders, these are used to illustrate basic genetic principles.

DEVELOPMENTAL BIOLOGY OF ENAMEL

To enable understanding of how molecular biologists are attacking the problem of inherited disorders of teeth, a brief review of dental embryology is needed. The early stages of tooth development are characterized by a budding off of epithelium from the oral epithelium itself into the area of the future alveolar bone. This tooth precursor will give rise to both primary and permanent dentitions in the area (incisor, premolar, etc.). Only the inner layer of the double layer of cells (the inner enamel epithelium) has a functional tooth fate—it gives rise to the enamel. The sequence in which this occurs is now well documented and provides an excellent example of how embryonic tissues differentiate under the influence of adjacent but developmentally different cells. In this case, neural crest cells migrating into the dental lamina area from around the developing neural tube came to lie in an intimate but not touching relationship next to the inner enamel epithelium. These two developmentally different cell layers, inner enamel epithelium (enamel) and neural crest (dentin), are separated by an extracellular matrix.' Specific tooth development is then mutually dependent on reciprocal cell-to-cell signaling between these two developmentally different cell layers. '



Indicate index case (probands) with arrow.

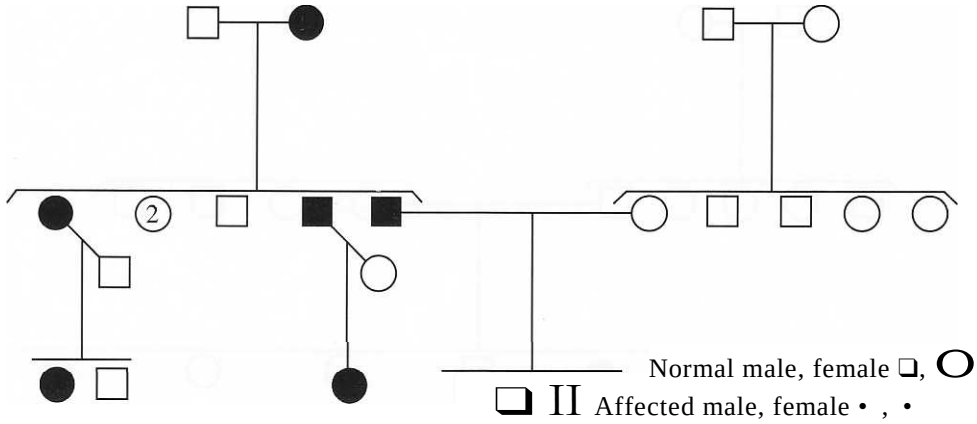
FIG. 6-4. Pedigree symbols used in family studies.

Molecular biologists working in this area are discovering structural proteins in enamel and dentin, as well as the genes that control the development of these tissues.[10] These genes are candidate genes for DNA mutation analysis, especially if they are in a chromosome location that has been linked to an inherited defect of enamel or dentin. The most intriguing dental research today is (1) the attempt to localize the genes for these proteins to a given chromosome and (2) the biochemical identification of a specific defect in the protein that prevents it from functioning normally. The following is a discussion of genetic principles best exemplified by the heritable disorders of enamel. Further comments on the molecular basis of the heritable disorders of dentin and enamel are given in Chapter 7.

Based on the clinical appearance, radiographic characteristics, and microscopic features, oral pathologists have recognized three major types of inherited enamel defects: hypoplasia, hypocalcification, and hypomaturation." These terms also provide us with the general description of the disease phenotypes. For example, in type 1, enamel hypoplasia, the enamel is hard and well calcified but defective in amount, so the teeth appear small. Two types of deficient enamel phenotypes are seen: generalized (all the enamel) and localized (pits and grooves in specific areas). Type 2, hypocalcification disorders, are those in which the enamel matrix is so drastically altered that normal calcification cannot occur, with the result that the clinical phenotype is a soft, mushy enamel that easily wears away. Type 3 defect, hypomaturation, involves the process of maturation of the enamel crystal. This occurs after an essentially normal enamel matrix has been established. The enamel is of normal thickness (not hypoplastic) and relatively normal hardness (slightly hypocalcified) with reduced radiographic density and discoloration.

From this collection of enamel diseases we can now draw out four examples of AI that illustrate the four major mendelian modes of inheritance: autosomal dominant (AD) and autosomal recessive (AR), and X-linked dominant (XLD) and X-linked recessive (XLR).

One characteristic of inherited dental defects is that both dentitions (primary and permanent) are affected. Occasionally, the defect is expressed differently in the two dentitions, as in the case of dentin dysplasia type II.[12] However, it is much more common to see the



[I C **6-5.** Autosomal dominant inheritance in hypocalcified amelogenesis imperfecta.

same clinical and radiographic picture in both dentitions. Both dentitions are affected in the AI disorders.

AUTOSOMAL DOMINANT INHERITANCE

From pedigrees such as that shown in Fig. 6-5, the following criteria for AD inheritance may be deduced:

1. The phenotype occurs in successive generations, that is, it shows vertical inheritance.
2. On the average, 50% of the offspring of an affected parent will also be affected.
3. Normal parents have normal offspring. The following causes of exceptions to this rule are worth noting:
   - Nonpenetrance of the trait (defined later).
   - A mutation in either the sperm or egg.
   - Germinal mosaicism. This is an increasingly invoked explanation for this situation. In this case, one of the parents is mosaic in the germ cell line and the sperm or eggs are of two types—one cell line with and one cell line without the mutation. Chance determines which sperm cell line will be selected. However, as the molecular basis of genetic traits becomes evident, mutation analysis may show that a parent believed to not be affected may actually also have the somatic mutation found in affected children.
   - Nonpaternity. Although this is not strictly a genetic problem, the illegitimacy rate in the U.S. population is high enough to make this a likely explanation when a normal couple has a child affected with a completely penetrant dominant trait.
4. Males and females are equally likely to be affected.

The hypocalcification type of AI provides an excellent example of AD inheritance. For diagnosing this trait, several criteria are employed. First, enamel matrix is susceptible to abrasion. The clinical picture is typical—gross accumulation of plaque on teeth that are hypersensitive because of the exposed dentin. Second, radiographs show enamel of varying thickness interproximally but with a "Swiss cheese" appearance because of loss of mineral. Thus, severe abrasion of this soft enamel is common.

AUTOSOMAL RECESSIVE INHERITANCE

Recessively inherited traits require that both genes of a given pair at a single locus be defective. Thus, of the two alleles at this genetic locus for Al, both must be mutants to show the trait. The following three gene pairs are recognized: AA—normal; Aa—heterozygote, showing an unaffected phenotype; and aa—homozygous-affected. The most common genetic situation producing an affected child is that in which both parents are heterozygous at this genetic locus (Fig. 6-6).

The following significant points about recessive inheritance must be noted:

1. The concept of a gene carrier is used here. The carrier is heterozygous for a recessive gene, and this single gene has only subtle, if any, expression. Parents of an affected child are typically heterozygous (carriers) and are then interpreted as being normal. Sometimes the carrier state can be detected, as in the case of phenylketonuria or Tay-Sachs disease. In these conditions a test is avail-able for carrier identification to detect the presence of the single mutant gene. This greatly improves the precision of genetic counseling.
2. The rarer the recessive gene, the more likely that normal parents who have an affected child will be blood relatives. This is a consanguineous mating. Given that both parents who produce an

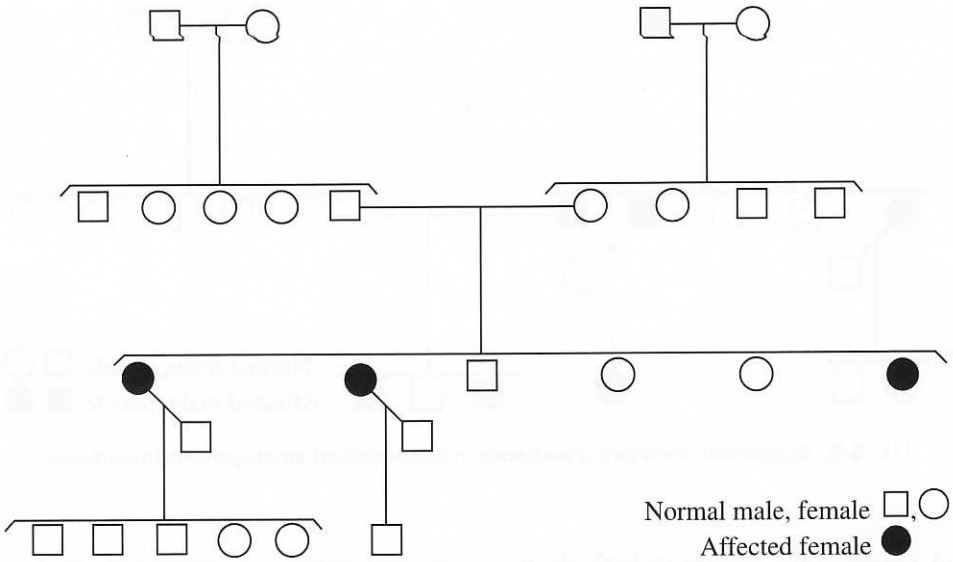


FIG 6-6. Autosomal recessive inheritance in pigmented amelogenesis imperfecta.

AR-affected child are heterozygotes, it is easy to see that only one out of the four possible combinations of parents' genes results in the homozygous-affected genotype. Hence, the recurrence risk for an affected child in this case is 25%. Note that transmission of the phenotype in a pedigree is horizontal (typically present only in sibs) and not vertical as with a dominant trait.

Of several AR types of AT, the one chosen for discussion here is the pigmented hypomaturation form. In this instance, the genetic defect probably lies in the protein needed in late tooth development to produce a mature, hard, and dense enamel. The defective enamel present is softer than normal but not nearly as soft and easily abraded as in the hypocalcification defect. Remarkably, a brown pigment is found in these outer layers of enamel that are formed last. The nature of this pigment is not known, but it imparts a dark brown, unsightly appearance demanding restorative treatment. A pedigree illustrating AR inheritance of this hypomaturation defect is shown in Fig. 6-6.

X-LINKED OR SEX-LINKED INHERITANCE

Genes on the sex chromosomes are unequally distributed to males and females. This inequality is the result of the following facts: (1) males have one X and one Y chromosome, whereas females have two X chromosomes and (2) the genes active on the Y chromosome are concerned with the development of the male reproductive system. For these reasons, then, males are hemizygous for X-linked genes, meaning that they have only half (or one each) of the X-linked genes. Since females have two X chromosomes, they may be either homozygous or heterozygous for X-linked genes, just as with autosomal genes.

Interesting genetic combinations are made possible by the male hemizygous condition. Because only one gene locus of each kind in the X chromosome is represented in the male, all recessive genes in single dose express themselves phenotypically and thereby behave as though they were dominant genes. On the other hand, XLR genes must be present in double dose (homozygous) in females to fully express themselves. Consequently, full expression of rare XLR diseases in practice is restricted to males and is seen infrequently in females.

To this point we have considered heritable defects in two of the three major types of enamel disorders. The third type—Al, hypoplastic type—shows both autosomal and X-linked modes of inheritance, but only one X-linked type is described here.

X-LINKED DOMINANT

Fig. 6-7 is the pedigree of a family with an X-linked form of Al, hypoplastic type.[13] The clinical features are diagnostic and in some females can be quite striking.

Once again, both dentitions are affected similarly. The surface defect has been described as being granular, lobular, or even pitted. Conceivably, all these different forms of expression are the result of the action of a single gene (or at least its alleles). The enamel is hard but because of its thinness is more susceptible to fracture and abnormal wear. Under the appropriate conditions this trait resembles a hypocalcification defect. However, radiographs quickly resolve this diagnostic problem and show enamel of normal density but with greatly reduced thickness.

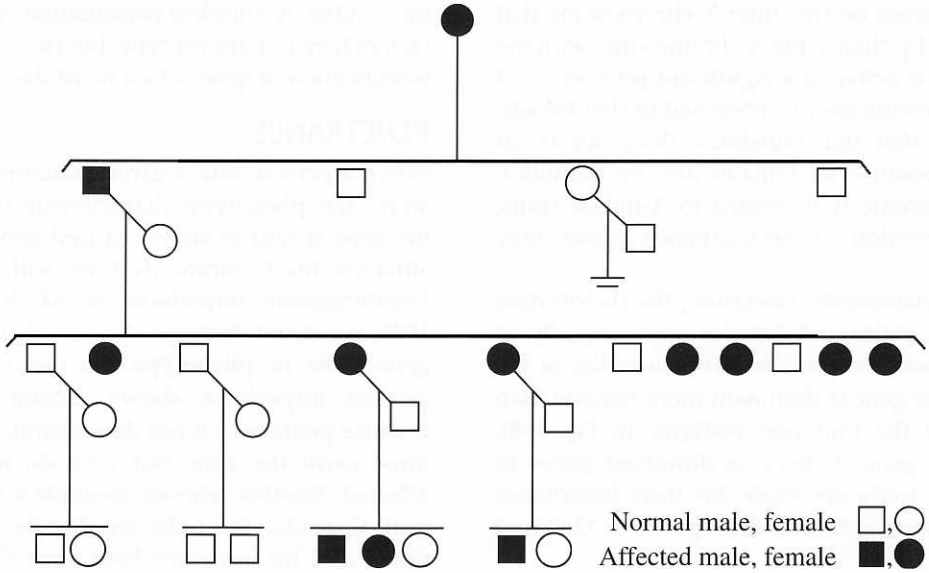


FIG. **6-7.** X-linked dominant inheritance in hypoplastic amelogenesis imperfecta.

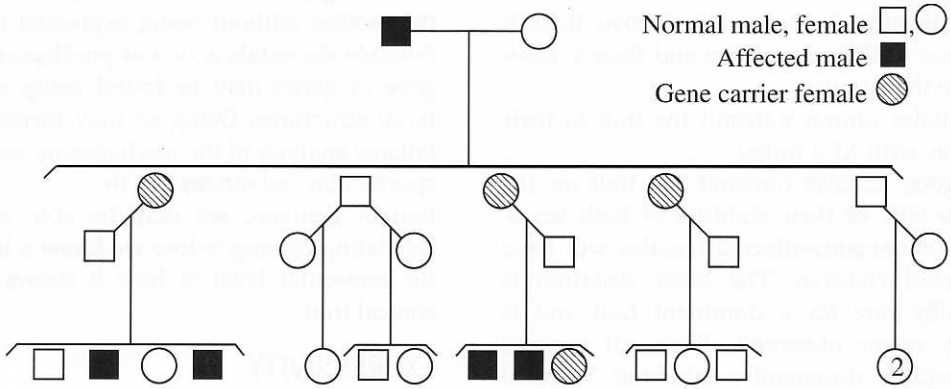


FIG. 6-8. X-linked recessive inheritance in hypomaturation amelogenesis imperfecta.

X-LINKED RECESSIVE

A pedigree of a family with the X-linked form of enamel hypomaturation is shown in Fig. 6-8. The genetic criteria for diagnosing an XLR trait are summarized as follows:

1. Because the gene cannot be passed from father to son, affected fathers almost never have affected sons. In rare situations, a son could be affected if the mother is a carrier of the XLR trait.
2. All daughters of an affected male receive his X-linked genes. Therefore, affected males transmit the trait to their grandsons through their daughters.
3. The incidence of the trait is much higher in males than in females. This is typified by the disease hemophilia, which is also caused by an XLR gene.

The clinical features of XLR hypomaturation type AI are most striking. The enamel has a somewhat reduced hardness but is not soft. However, the crowns of the teeth look like mountains with snow on them. Hence the name given has been "snow-capped teeth." Radiologically the enamel is hypomature; it shows a lack of contrast between enamel and dentin even though the enamel is of normal thickness.

It should be noted that heterozygous females occasionally show significant clinical expression of a single XLR gene. The reason for this apparent contradiction is the process of X-inactivation, termed *lyonization* after its discoverer, Mary Lyon. This occurs only in females. All female cells have two X chromosomes, but most of the genes on one of the two X chromosomes are inactivated at about the blastula stage of development. This has the effect of making the total number of active, X-linked genes about the same in both males and females. If the female is heterozygous for an X-linked trait, two populations of cells result. One cell population has genes on one X chromosome that are active, while the other cell

population has genes on the other X chromosome that are active. When by chance the X chromosome with the deleterious gene is active in a significant proportion of the cells, its expression may be observed in that female. Chance dictates that this imbalance does not occur frequently, but because all females are, by definition of lyonization, mosaic with regard to X-linked traits, phenotypic expression of heterozygous genes may occur in them.

The previous statements concerning the distribution of XLR genes in males and females apply equally as well to XLD genes. The principal difference lies in the fact that when the gene is dominant more females than males will show the trait (see pedigree in Fig. 6-8). Because all XLR genes behave as dominant genes in males, no new criteria are made for their inheritance in males. The following criteria distinguish an XLD trait in families:

1. Affected males must transmit the trait to all of their daughters (as with XLR traits), but all of them are affected, because fathers give their X chromosome to their daughters and their Y chromosome to their sons.
2. Affected males cannot transmit the trait to their sons (just as with XLR traits).
3. Heterozygous females transmit the trait on the average to 50% of their children of both sexes, whereas homozygous-affected females will have only affected children. The latter situation is exceptionally rare for a dominant trait and is practically never observed. Thus, all females affected with a dominantly inherited X-linked trait are considered to be heterozygotes until proven otherwise.

Two points are emphasized here. First, transmission of XLD genes by females follows a pattern indistinguishable from that of autosomal transmission. Thus, these two types of dominant inheritance can be differentiated only by observation of the offspring of affected males. Second, it was noted that XLR disorders are much less common in females than in males. The reverse is true for XLD traits. An XLD trait should appear about twice as often in females as in males, since the former have twice as many X chromosomes as males.

VARIATION IN GENE EXPRESSION

The patterns of inheritance shown in traits determined by genes at a single locus have been presented and discussed. Such patterns, if present, are usually easy to recognize. Unfortunately, many factors may modify the expression of a gene in a family in such a way that a typical monogenic pattern of inheritance is not discernible. A complete presentation of these modifying factors here is inappropriate, but two concepts related to modification of gene action merit discussion.

PENETRANCE

When a person with a given genotype fails to demonstrate the phenotype characteristic for the genotype, the gene is said to show reduced penetrance. This is a situation most commonly seen with dominant traits. Dentinogenesis imperfecta, an AD trait, is practically 100% penetrant, because all individuals who carry that gene show its phenotype. On the other hand, osteogenesis imperfecta shows incomplete penetrance, because pedigree studies demonstrate individuals who must carry the gene but who do not appear to be affected. Another relevant example is found in the CLP trait. Consider the following family history: a grandfather and his grandson both have CLP but the boy's mother (also the grandfather's daughter) does not. The probability is very high that her son's cleft liability came from his grandfather and therefore was passed through the mother without being expressed as an overt cleft. Possibly the subtle action or predisposition of a clefting gene or genes may be found using measurements of facial structures. Doing so may increase the power of linkage analysis of the predisposing genotype. With the spectacular advances in the understanding of the human genome, we may be able to locate a gene regulating clefting before we know what its action is at the molecular level or how it shows this action as a clinical trait.

EXPRESSIVITY

If a single gene trait can show different phenotypes in the affected members of a kindred, it is said to show variable expressivity. Osteogenesis imperfecta also provides an illustration of variable gene expression. The cardinal signs of this disease are (1) multiple fractures, (2) blue sclera, (3) dentinogenesis imperfecta, and (4) otosclerosis, which results in a hearing deficit. Affected persons in a single family may show any one or a combination of these signs, which illustrates the considerable variation in gene expression. The minimum expression of the gene observed in a family might then be only a blue color to the sclera, which could be unnoticed by the clinician. In this case highly variable gene expression may fade into nonpenetrance.

The craniosynostosis syndromes are AD traits associated with single gene mutations. They also provide good examples of how, even with the strong influence of a single gene, the phenotype can vary markedly. Although it was once thought that a particular mutation in a given gene would always result in a specific syndrome, several identical mutations in the fibroblast growth factor receptor 2 (FGFR2) gene have been found

in patients diagnosed with the three different clinical craniosynostosis syndrome entities of Crouzon, Pfeiffer, and Jackson-Weiss syndrome.[5,6,8]

Another example of the individual variability of these single gene mutation autosomal dominant phenotypes occurred when two individuals in the same family had the classic phenotypes of Pfeiffer and Apert syndrome. In addition, seven other family members had unusually shaped heads and facial appearance reminiscent of Crouzon syndrome.[7] The phenotype may be so variable that this individual may appear to be clinically normal, yet have the same gene mutation associated with Crouzon syndrome in three of his children and two of his grandchildren. Only through the analysis of radiographic measurements was a minimal expression of features suggestive of Crouzon syndrome evident."

MULTIFACTORIAL INHERITANCE

The following features typify multifactorial inheritance, in contrast to monogenic inheritance: (1) multiple genes (polygenes) at different loci are involved in expressing the phenotype, and (2) the phenotype produced is a summation of the effects of polygenes interacting with their environment. The phenotypic result is often a continuously varying spectrum of that trait (e.g., height) rather than presentation as a discrete (trait present or absent) phenotype.

Many common diseases, such as dental caries, have continuous variation with no sharp distinction between normal (average) and abnormal (extremes). However, there may be a specific measurement point beyond which that disease is arbitrarily regarded by the clinician as abnormal.

Multifactorial inheritance is troublesome to analyze genetically; in fact, geneticists often arrive at a diagnosis of multifactorial inheritance for a given trait only after the monogenic forms of inheritance have been considered and found to be unlikely. Certain techniques for studying it have been developed. The simplest is the method of resemblance between relatives, which states that the more closely related two individuals are, the more closely they resemble each other concerning the specific trait in question. It is important to stress, though, the continuous phenotypic variation that is characteristic of inheritance patterns resulting from polygenes.

This issue of continuous variation is emphasized because the most common diseases with which the dentist must deal (i.e., periodontal disease, dental caries, and malocclusion) are multifactorial traits. Only the extremes of variation are readily apparent to the dentist, such as in the child with rampant caries or the adult who is caries free. In this latter instance, if one did not understand the concept of multifactorial inheritance, one might conclude that such individuals represent a discrete phenotype influenced by a single gene in a mendelian manner. This is frequently not the case.

A most important feature of traits produced by polygenes is that they are susceptible to environmental modification. A phenotype resulting from the concerted action of 100 genes is much more likely to be altered and modified by the existing environment than a trait controlled by only one or even several genes. Still, this does not mean that a trait resulting from only one or even several genes cannot be influenced by environmental factors. The change in phenotype will depend on the individual's ability to respond to the environmental factor, which may be heavily influenced by the same gene(s) originally influencing the phenotype or by other genes.

An example of a polygenic trait that is markedly influenced by environmental factors is dental caries, which is the interaction product of three essential factors: a cariogenic diet, a caries-producing bacterial flora, and a susceptible tooth. These three factors encompass a variety of biologically complicated entities, such as saliva, plaque, tooth matrix formation, and crystallization. It should be easy to see that the development of these complex elements must involve a great number of genes. Environmental modification, such as properly timed systemic fluoride supplementation, produces a considerable alteration in the phenotype without changing the genetic constitution of the individual. The reader can probably think of additional environmental modifications that can produce a greatly altered dental caries experience without changing an individual's genes. Some conditions that we have attributed to a multi-factorial inheritance because they tend to occur in particular families may be greatly influenced by a gene or genes that predispose to the condition, depending on what other genetic or environmental factors are involved.

MULTIFACTORIAL INHERITANCE IN HUMAN DISEASES

For many common disorders, such as diabetes and hypertension, and even for the major common congenital malformations (i.e., spina bifida, hydrocephalus, and CLP) there is a definite familial tendency. This is shown by the fact that the proportion of affected near relatives is greater than the incidence in the general population. However, this proportion is much lower than what is expected for a monogenic trait, and the explanation most commonly offered for major congenital malformations is that they are multifactorial traits. As previously stated, one definition of a multifactorial trait is that it represents the summation of the effects of many genes (polygenes) interacting with the environment, hence the term *multifactorial*. Environment is defined as those nongenetic circumstances that render an individual more or less susceptible to a disease state. In contrast to monogenic traits, whose characteristics have been

summarized in preceding paragraphs, multifactorial diseases show the following characteristics:

Each person has a liability for a given disease, and that liability represents a sum of the genetic and environmental liabilities.

The multifactorial-threshold model is a mathematical way of expressing these liabilities. For polygenic traits the model is simply a gaussian curve. As already noted, for multifactorial traits a threshold must be added to allow the continuous polygenic model to be used in describing noncontinuous or discrete traits. For many human congenital malformations, a multifactorial model with threshold is appropriate for describing discrete traits such as CLP. Such a threshold means that all persons with sufficient gene dosage and environmental interaction will be above the threshold of expression and show the cleft lip. Those with less will not show a cleft lip. A graphic representation of this idea is shown in Fig. 6-9.

Because of the differing dosage of polygenes in groups that show a specific phenotype (e.g., CLP), the overall incidence of this trait will vary in near relatives of those affected. For example, a dominantly inherited trait has a gene dosage of 1 in 2 (50%). Assuming that several polygenes may be involved in CLP, this figure decreases at least tenfold to about 1% to 5%. Naturally, the incidence in a random population is even lower, or about 1 per 1000. Therefore, increasing gene dosage for a multifactorial trait in a family is associated with an increased incidence of that trait in near relatives of the affected individuals. The nature of this system with a threshold permits large numbers of persons at risk for showing that phenotype (CLP) to carry the liability for clefting without expressing it clinically. Based on current research findings regarding traits that are multifactorial with a threshold, it appears that it will be difficult to relate these mathematical observations to cellular biologic function.

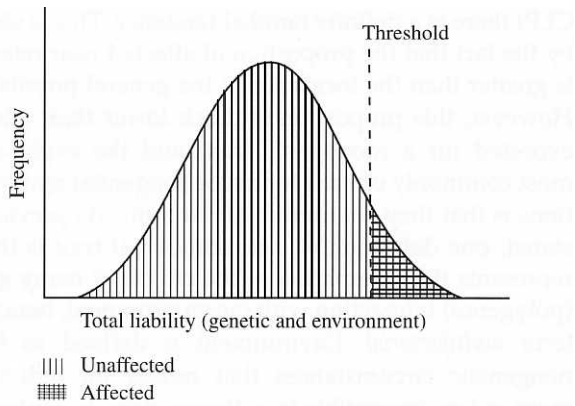


FIG 6-9. Multifactorial model for inheritance of cleft lip and palate.

Toward the end of the nineteenth century, Galton recognized that twins could be useful for evaluating the nature-nurture argument that was raging at that time. Interest in the twin method for study of the relative importance of heredity and environment in humans has been increasing. One explanation for this interest is that many human traits are multifactorial (polygenic), are susceptible to environmental modification, and there-fore are difficult to study by conventional methods. The twin method allows an approach to the study of such traits and is based on the principle that human twins are of two basic types: monozygotic (or identical) twins resulting from a single ovum fertilized by a single sperm, and dizygotic (or fraternal) twins resulting from fertilization of two ova by two sperm. It is axiomatic that monozygotic twins have identical genotypes, whereas dizygotic twins are no more closely related to each other than are any two siblings.[74] It also follows that differences between monozygotic twins result from environmental differences, whereas those between dizygotic twins result from differences in both heredity and environment.

To use the twin method, one must distinguish between the two types of zygosity. If both twins are identical for the trait in question (regardless of their zygosity), they are described as concordant. If they are unlike for the trait, they are discordant. Such intrapair differences are usually expressed in percentage figures for a group of twins being evaluated. For example, monozygous twins show a 33% concordance for CLP, whereas dizygous twins show only a 5% concordance.

Another method to estimate the heritability of a trait and to evaluate evidence of linkage of a phenotype with DNA polymorphisms is by sib-pair analysis. Heritability estimates can be generated from within- and between-sibship variance quantified by generalized linear models, with confounding factors controlled for where indicated. Polymorphic DNA markers may be tested for genetic linkage (proximity) to a gene influencing a particular phenotype by testing whether the magnitude of the phenotypic difference between two siblings is correlated with the alleles they share that are identical by descent (IBD). An allele is considered to be IBD if both members of a sibling pair inherited the same marker allele from the same parent. If a marker is linked to a gene contributing to the phenotype in question, then siblings with a similar (if quantitative) or the same (if discrete) phenotype would be expected to share more alleles IBD, whereas siblings with widely differing phenotypes would be expected to share few if any alleles IBD near any gene(s) influencing the phenotype.[75] In addition, another method of looking for DNA markers that are in linkage disequilibrium is to use the quantitative

transmission disequilibrium test. This analysis calculates the difference between the value of the quantitative trait in the offspring and the average value of the quantitative trait in all offspring in all families studied, while simultaneously considering the allele transmission from parent to offspring.[20] Thus, whereas the sib-pair linkage analysis involves two or more siblings, the quantitative transmission disequilibrium test involves trios of parents and one or more siblings.

Influence of Genetic Factors on Major Croniofacial, Oral, and, Dental Conditions

GENETICS AND DENTAL CARIES

It is clear from many dietary studies that variation in susceptibility to dental caries exists even under identical, controlled conditions.[21] This implies that, because of genetic differences, certain environmental factors are potentially more cariogenic for some people than for others. This is not to say that dental caries is an inherited disease; rather, genetic influences may modify the overt expression of this disease in the individual.

Fifty years ago, dental caries was presented to dental students as a disease that was so common that more than 99% of the general population was afflicted by it. Although it is still recognized as a common disease, the use of systemic and topical fluorides and persistence by organized dentistry to bring about changes in dietary habits and oral hygiene practices have contributed significantly to a remarkable decrease in the prevalence of this disorder, especially noted in children. Today, it is not unusual for a prepubertal-age child to be caries free.

Three essential interacting elements comprise the model system for dental caries that is most commonly used to discuss its etiology. These factors are microorganisms, substrate (fermentable carbohydrates), and host factors, such as tooth anatomy. It is in the last area of the host factors that genetics exerts a major influence on dental caries initiation.

Several investigators have studied the genetic aspects of dental caries in humans, using both the twin and the family pedigree approaches. Since dental caries is an age-dependent process, much of the reported data cannot be compared because of age differences in the various population groups studied. Nevertheless, the family observations by Klein and Palmer"- and Klein" are worth noting. Their findings indicated that children have a caries experience remarkably similar to that of their parents when the susceptibility of both parents is the same (either high or low). When caries susceptibility of the two parents is dissimilar, however, the children's susceptibility tends to be more like that of the mother than that of the father. This finding was particularly evident in daughters.

Since dental caries is an infectious communicable disease, however, familial clustering may to some degree reflect familial environmental contact, with transmission of cariogenic bacteria to children at certain ages. Li and Caulfield found that mothers are the principle source of mutans streptococci to their infants, with a greater rate of transmission to female than male infants.[24]

The more common a genetic trait is, the more difficult it is to demonstrate its genetic character. Several authors have attempted to do this for dental caries by the study of twins. Book and Grahnen attempted to maximize differences in caries experience within families by selecting caries-free, 20-year-old men and comparing caries experience within their families.[25] Results showed that parents and siblings of caries-free propositi had significantly lower rates of decayed, missing, and filled teeth than the control families. The authors concluded that the observed differences are hereditary and probably polygenic in nature.

Studies of twins by Dahlberg and Dahlberg,[26] Mansbridge,[27] Horowitz, Osborne, and De George,[2]" and Caldwell and Finn" indicated that genetic factors make a significant contribution to individual differences in caries susceptibility. However, most authors agree that this genetic component of dental caries is a minor one in comparison with the overall effect of environment. The conclusion from clinical twin and familial correlation studies and estimation of heritability studies regarding the degree of genetic influence on caries may be confounded by the already mentioned transmission of cariogenic bacteria within the family.

A review of inherited risks for susceptibility to caries found evidence of an association between altered dental enamel development in defined populations and an increased risk of caries, as well as a relationship between host immune complex genes and different levels of cariogenic bacteria and enamel defects.[3]" Thus the individual's genotype may influence the likelihood of intraoral colonization of cariogenic bacteria, which further exemplifies the complexity of caries development. To further delineate the familial aspect of caries that is bacterial from host genetic factors, studies relating caries susceptibility to specific biochemical differences in individuals may be undertaken. For example, Goodman et al reported significant differences in salivary flow, pH, and salivary amylase activity between monozygotic and dizygotic twins." In addition, genetic linkage studies on well-characterized populations with clearly defined caries experience will help define those host factors that have the greatest influence on the incidence of caries.'-'

Animal studies have been more productive. Hunt, Hoppert, and Rosen succeeded in establishing caries-resistant and caries-susceptible strains of rats using inbreeding techniques.[32] Although the resistant strain was challenged by oral inoculation of cariogenic bacteria,

the resistant phenotype was maintained. These were the first studies to confirm the presence of important genetics elements influencing dental caries susceptibility.

From this information, it is clear that heredity plays an important but obscure role in the cause of dental caries. Studies in the 1970s and 1980s examined the influence of saliva proteins on dental plaque formation. These studies suggested a possible mechanism of action for genes regulating this multifactorial trait. A group of saliva proteins designated as the proline-rich proteins (PRPs) because of their high content of the amino acid proline have been linked to early plaque and pellicle formation.[33'34] PRPs closely resemble enamel matrix proteins in both composition and structure, which accounts for why they bond so tightly to hydroxylapatite crystals. Furthermore, the ability to produce these several types of PRPs is inherited as a group of autosomal codominant traits. At least eight different polymorphic PRPs are known, and all these proteins are coded for by a block of genes called the salivary protein complex, located on the short arm of human chromosome 12 3[5.36] Only a few studies have attempted to associate these salivary protein phenotypes with oral disease states. Yu et al reported significant association between two specific PRPs phenotypes (Pa+ and Pr22) and an increase in dental caries scores in the permanent teeth of children 5 to 15 years of age.[37] This result suggested that persons with either or both of these two genotypes (Pa+ and/or Pr22) may be at significant risk for increased susceptibility to dental caries, whereas the allelic genes, Pa– and Pril or Pr12, appear to confer caries resistance. The mechanism of action could be related to the formation of a caries-susceptible plaque. Certainly, the most important result of an interaction between genetic and environmental factors is the pro-duction of dental plaque. It is through the plaque itself that dental caries is expressed as a disease process, and so plaque formation may be the critical element influenced by genetics in disease initiation.

In summary, susceptibility to human dental caries is influenced to a significant but minor degree by heredity. This genetic control is undoubtedly multifactorial in nature, and such a polygenic background strongly implies considerable environmental influence. Specific types of dental caries susceptibility representing the extremes of variation of this trait may ultimately prove to be monogenic traits, but at present the evidence is insufficient for a clear statement of such inheritance.

GENETICS AND PERIODONTAL DISEASE

The periodontal disease state is often described as a local inflammatory disease with possible underlying systemic factors. This disease is so widespread in human populations and has such widely varying clinicohistopathologic features that it seems certain that multiple diseases with multiple causes are being lumped together as a single entity. Periodontists suggest that there is evidence for the existence of several variant types of periodontal disease generally subclassified by the age of onset, severity of bone loss, oral hygiene status, and presence or absence of local factors. One might visualize a continuum of disease expression ranging from a localized gingivitis to a generalized peri-odontitis with severe bone and tooth loss. Such a complex disease shows both inflammatory and degenerative pathologic features.

It is easy to understand why genetic studies of this common problem have been neglected. As is true for dental caries, periodontal disease is common, occurs with a continuum of expressivity, and is greatly influenced by environmental conditions, such as diet, occlusion, and oral hygiene habits. All of these features fit the description of a multifactorial type of disease or at least of disease susceptibility.

Most periodontists believe that dental plaque formation is the critical first step in the initiation of chronic periodontal disease, and it is possible that the PRPs, which are influential in dental caries initiation through regulation of plaque formation, also play a role in the genetics of periodontal disease.

Most genetic studies of a trait make use of families with multiple affected individuals or twins. A carefully designed study of twins with periodontal disease by Ciancio, Hazen, and Curat was reported in 1969.[38] Using the Ramfjord index, which evaluates gingival inflammation, calculus formation, tooth mobility, and tooth loss in all four quadrants of the mouth, the authors examined 7 monozygotic and 12 dizygotic pairs of teenaged twins. They concluded that there was no evidence in these twins for significant heritability of any of these dental parameters.

Alternatively, Michalowicz et al published a large study of adult twins (mean age, 40 years) of which there were 63 monozygotic and 33 dizygotic pairs.[3y] Using elements of the Ramfjord index as criteria for diagnosis, heritability estimates were calculated. The authors state that from 38% to 82% of the periodontal disease identified in these twins was attributable to genetic factors.

Investigation by Kornman et al into the association of different polymorphisms of inflammation-mediating genes and periodontal disease in adult nonsmokers indicate interleukin la and 113 (IL-la and IL-1(3) genotype may be a risk factor.[:0] The IL-1(3 polymorphism was IL-1p+3953 and the IL-la polymorphism was IL-la-889. Nonsmokers aged 40 to 60 carrying the "2" allele (in either homozygous or heterozygous state) at both loci were observed to have nearly 19 times the risk of developing severe periodontitis of subjects

homozygous for the "1" allele at either or both of these loci. However, in a review of subsequent studies, Greenstein and Hart-" noted that the relationship of specific IL-1 genotypes and the level of crevicular fluid IL-1(3 is not clear, and that the ability of the genetic susceptibility test for severe chronic periodontitis based on the finding of Kornman et al-[4U] to forecast which patients will develop increased bleeding on probing, periodontitis, or loss of teeth or need for dental implants is ambiguous. It is evident that biochemical and molecular biologic techniques, in concert with clinical studies, will continue to be used in evaluating the heritable predisposing factors in periodontal disease.

Animal studies have not been a focus for examining the heredity of periodontal disease. Only the complex multifaceted periodontal disease of humans is available for study, and the variation in definitive diagnostic criteria that could be used to establish different types of periodontal disease increases the difficulty of studying this heterogeneous and complicated pathology. Early-onset periodontitis has been the subject of most family studies. Since several forms of early-onset periodontitis (e.g., localized prepubertal periodontitis, localized juvenile periodontitis [JP], and generalized JP) can be found in the same family, the expression of the underlying genetic defect appears to have the potential to be influenced by other genetic factors."

Progress has been made in the study of rare genetic conditions or syndromes that can predispose to periodontal disease or have periodontal disease as a relatively consistent component of their pleiotropic effect. For example, leukocyte adhesion deficiency (LAD), type I and type II, are autosomal recessive (AR) disorders of the leukocyte adhesion cascade.[3] LAD type I has abnormalities in the integrin receptors of leukocytes resulting from mutations in the 132 integrin chain (ITGB2) gene leading to impaired adhesion and chemotaxis, which results in an increased susceptibility for severe infections and early-onset (prepubertal) periodontitis.-",[45] LAD type II is also an AR disorder secondary to mutation in the gene encoding guanosine 5'-diphosphate–fucose transporter 1 (FUCT1). The infectious episodes and the severity are much milder than those observed in LAD type I, and the only persistent clinical symptom is chronic severe periodontitis. The exact defect in the system is absence of the stalyl Lewis x (SleX) structure antigens, which are important ligands for the selectin on the leukocyte, which leads to a profound defect in leukocyte rolling, the first step in the adhesion cascade. This causes a marked decrease in chemotaxis accompanied by pronounced neutrophilia. Apart from the leukocyte defect, these patients suffer from severe growth and mental retardation and exhibit the rare Bombay blood group type."

Ehlers-Danlos syndrome (EDS) is actually a collection of ten types distinguished on the basis of clinical symptoms and inheritance pattern. In addition to consistent early-onset periodontal disease, patients with EDS type VIII have variable hyperextensibility of the skin, ecchymotic pretibial lesions, minimal bruising, minimal to moderate joint hypermobility of the digits, and cigarette paper scars. Inheritance is AD. Early-onset periodontal disease may also be found in patients with EDS type IV. These individuals are usually characterized by type III collagen abnormalities with hyperextensibility of the skin, ecchymotic pretibial lesions, easy bruisability, cigarette paper scars, joint hypermobility of digits, pes planus, and, of greatest concern, arterial and intestinal ruptures. Like type VIII, type IV also has AD inheritance." The presence or absence of type III collagen abnormalities has been taken to be a differentiating factor between the two types, with EDS type IV showing abnormal type III collagen. The considerable overlap in phenotype of these two types warrants careful family and clinical evaluation, and biochemical studies of collagen, when a patient with features of EDS and periodontal disease is evaluated.'

Chediak-Higashi syndrome has frequently been linked with severe periodontitis.'[45] This rare AR disorder is characterized by oculocutaneous hypopigmentation, severe immunologic deficiency with neutropenia and lack of natural killer cells, a bleeding tendency, and neurologic abnormalities. It is caused by mutations in the CHSI/LYST gene.}[s]

Papillon-Lefevre syndrome and Haim-Munk syndrome are two of the many different types of palmoplantar keratoderma, differing from the others by the occurrence of severe early-onset periodontitis with premature loss of the primary and permanent dentition. Haim-Munk syndrome is characterized in addition by arachnodactyly, acroosteolysis, and onychogryphosis.[4`'] Hart et al[so] have shown that both of these AR syndromes are due to different mutations in the cathepsin C (CTSC) gene.

EARLY-ONSET PERIODONTITIS

Early onset of periodontitis may occur in the primary dentition (prepubertal periodontitis), may develop during puberty (JP), or may be characterized by exceedingly rapid loss of alveolar bone (rapidly progressive periodontitis). Along with hypophosphatasia, prepubertal periodontitis appears to be the most commonly encountered cause of premature exfoliation of the primary teeth, especially in girls.[46]

JP has the following features:

1. An early onset of the breakdown of periodontal bone. This bone loss is of two types: chronic

periodontitis in a generalized form affecting any dental area, and a localized form in which the molar or incisor regions of bone are the most severely affected.

Bone destruction that is rapid and vertical, with specific microorganisms associated with the periodontal lesion.

Familial aggregation, especially in the molar and incisor types. It seems probable that the generalized and localized types represent two different aspects of the same disorder, so this discussion will consider them as a complex entity that goes by the name familial JP.

At least three different modes of inheritance have been proposed for JR The early reports of Benjamin and Baer, and later that of Melnick et al, offered support for an XLD trait.[51,5'] This conclusion was based on the observation that twice as many females were observed to be affected as males (see preceding criteria for XLD inheritance). However, Hart et al have shown that females are twice as likely as males to seek out treatment.[53] When this biased ascertainment is allowed for, the male/female ratio is essentially unity. Saxen demonstrated that a clinical phenotype found in Finland closely resembling JP (if not actually JP) showed an AR mode of inheritance.[54] This disorder may be peculiar to Finland. Boughman et al reported linkage of a gene in a single large family for an AD form of JP on chromosome 4 to another dental trait (dentinogenesis imperfecta, Shields type III).[5,5] Hart et al, in a study of 19 unrelated families, strongly excluded linkage between an early-onset periodontitis susceptibility gene and chromosome region 4812-q13 assuming locus homo- geneity.[5t'] They concluded that the previous report of linkage was a false positive, or that there are two or more unlinked forms of JP, with the form located in 4g12-813 being less common. Dominant inheritance is probable for the type most prevalent in this country (Fig. 6-10).

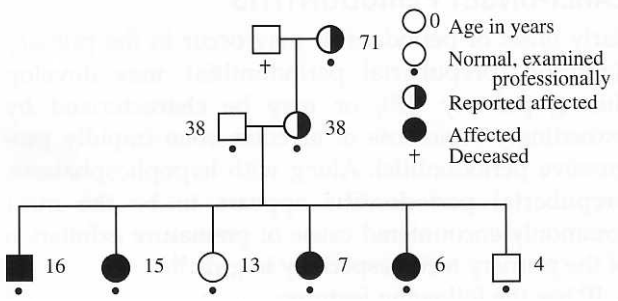


FIG. 6-10. Pedigree of family with juvenile periodontitis, a dominant trait.

Evaluation of the same IL-1a and IL-113 polymorphisms found by Kornman et al[l°] to be associated with periodontitis in adult nonsmokers was performed in black and white families with two or more members affected with early-onset periodontitis by Diehl et al.[5"] Interestingly, they found the IL-1 alleles associated with high risk of early-onset periodontitis to be the ones suggested previously to be correlated with low risk for severe adult periodontitis. They concluded that early-onset periodontitis is a complex, oligogenic disorder (i.e., involving a small number of genes), with IL-1 genetic variation having an important but not exclusive influence on disease risk.

GENETICS OF MALOCCLUSION

The study of occlusion pertains to relationships between teeth in the same dental arch, as well as between the two dental arches when the teeth come together. Many factors are involved in the definition of normal occlusion, and it is not possible in this chapter to discuss all of them. Some of the most important orofacial parameters of occlusion are airway function, soft tissue anatomy and function, size of the maxilla, size of the mandible (both rami and body), arch form, anatomy of teeth (including malformation), congenitally missing teeth, and rotation of teeth. All of these important elements must be included in the concept of occlusion.

Malocclusion is perhaps somewhat easier to define. One may simply say that malocclusion is a significant deviation from normal occlusion. However, this description is useful only if one considers the multiple aspects implicit in such a definition. Normal occlusion and malocclusion are dynamic concepts that involve the interrelationships of many factors, not a few of which have been shown to be influenced by genetic factors. For example, in a study of the association of the Pro561Thr (P56IT) variant in the growth hormone receptor (GHR) gene with craniofacial measurements on lateral cephalometrics radiographs by Yamaguchi et al, those who did not have the GHR P56IT allele had a significantly greater mandibular ramus length (condylion-gonion) than did those with the GHR P561T allele in a normal Japanese sample of 50 men and 50 women.[58] The average mandibular ramus height in those with the GHR P56IT allele was 4.65 mm shorter than the average for those without the GHR P561T allele. This significant correlation between the GHR P56IT allele and shorter mandibular ramus height was confirmed in an additional 80 women.

Theoretically, there are two general ways in which predisposing or causative factors for malocclusion could be due to heritable characteristics." One would be inheritance of a disproportion between the size of the

teeth and the jaws resulting in crowding or spacing, whereas the other would be inheritance of a disproportion in the position, size, or shape of the mandible and maxilla. As will be seen, genetic influences on each of these traits are rarely due to a single gene, which would be necessary for malocclusion to be due to the simple inheritance of discrete skeletal and dental characteristics. Instead they are often polygenic with the potential for environmental influence. Part of the practice of orthodontics is to use environmental (i.e., nongenetic) influences for the correction of malocclusion.

Dental anthropologists tell us that malocclusion is uncommon in pure racial populations. However, it has been debated whether this is due to the lack of procreation with other populations or the less refined diet often eaten by these typically isolated groups.

The experiments of Stockard et al in dogs have been cited as evidence that crossbreeding among inbred strains increases the incidence of malocclusion.[6"] However, the anomalies they produced may have been due in part to the influence of a major gene or genes that have been bred to be part of specific breeds. It seems improbable that racial crossbreeding in humans could resemble the condition of these experiments and thereby result in a synergistic increase of orofacial malrelations. An exception on an individual basis may be when an individual with a dominant trait or syndrome that results in a malocclusion has a child. Depending on whether it is autosomal or X-linked, the dominant gene, if transmitted to an offspring, may also affect the offspring's occlusal development in a similar fashion as in the affected parent.

Studies of occlusion in twins have also been made. Lundstrom performed an intensive analysis of specific dentofacial attributes in twins and concluded that heredity played a significant role in determining the following characteristics: tooth size, width and length of the dental arch, height of the palate, crowding and spacing of teeth, and degree of overbite!" Kraus, Wise, and Frei made a cephalometric study of triplets in an attempt to assign an inherited basis to specific craniofacial morphologic features.[62] The authors looked at the lateral profile of the head and cranial vault, the outline of the calvaria, the cranial base, and the facial complex, which included both the upper and lower face and the maxillomandibular relationship. In addition, they selected 17 individual measurements of single portions of a given bone (e.g., posterior border of rmus). They concluded that morphology of an individual bone is under strong genetic control but that the environment plays a major role in determining how various bony elements are combined to achieve a harmonious or disharmonious craniofacial skeleton. This observation at least partly explains the remarkable differences sometimes seen in the facial patterns of identical twins and emphasizes the important role of environment in their development.

Litton et al reported class III malocclusion to be a heritable trait that is probably polygenic in nature." Harris has shown that the craniofacial skeletal patterns of children with class II malocclusions are heritable and that there is a high resemblance to the skeletal patterns in their siblings with normal occlusion.[6"] From this he concluded that the genetic basis for this resemblance is probably polygenic. Interestingly, Harris used the family skeletal patterns as predictors for treatment prognosis of the child with a class II malocclusion."

King et al noted that many studies which estimate heritability of craniofacial structures may have a bias because they have generally involved subjects who had not undergone orthodontic treatment, and that often subjects judged to have an extreme malocclusion were excluded.[°'] They found that, in contrast to the relatively high heritability of cephalometric variables and low heritability of occlusal variables in subjects with naturally occurring good occlusion, the heritability estimates for craniofacial skeletal variables in subjects with overt malocclusions were significantly lower and the heritability estimates for occlusal variations were significantly higher. This observation supports the idea that everyone does not react to specific environmental factors in the same manner, although those who are related are more likely to react in a similar fashion. To quote King et al, "We propose that the substantive measures of intersib similarity for occlusal traits reflect similar responses to environmental factors common to both siblings. That is, given genetically influenced facial types and growth patterns, siblings are likely to respond to environmental factors (e.g., reduced masticatory stress, chronic mouth breathing) in similar fashions. Malocclusions appear to be acquired, but the fundamental genetic control of craniofacial form often diverts siblings into comparable physiologic responses leading to development of similar malocclusions."[6']

Although we have some information about genetic influence on specific traits (e.g., missing teeth, occlusal patterns, tooth morphology, and even mandibular prognathism), these cases are exceptions, and we do not have sufficient information to make accurate predictions about the development of occlusion simply by studying the frequency of its occurrence in parents or even siblings. Admittedly, family patterns of resemblance are frequently obvious, but predictions must be made cautiously because of the genetic and environ-mental variables and their interaction, which are unknown and difficult to evaluate.

Unfortunately, at this time the results of studies on the genetic and environmental factors that influence the

development of malocclusion are representative of the samples studied, not necessarily of any particular individual. In addition, the extent that a particular trait is influenced by genetic factors may have little if any effect on the success of environmental (treatment) intervention. Still, it may be that genetic factors that influenced a trait will also influence the response to intervention to alter that trait, or other genetic factors may be involved in the response. Therefore, the possibility of altering the environment to gain a more favorable occlusion theoretically exists even in individuals in whom the malocclusion does have a relatively high genetic influence. However, the question of how environmental and genetic factors interact is most relevant to clinical practice, because it may explain why a particular alteration of the environment (treatment) may be successful in one compliant patient and not in another.[bb]

EXTERNAL APICAL ROOT RESORPTION

Basic descriptors of root resorption are based on the anatomical region of occurrence; that is, designations are internal root resorption and external root resorption (cervical root resorption and external apical root resorption [EARRI). EARR is a frequent iatrogenic outcome associated with orthodontic treatment and may also occur in the absence of orthodontic treatment[67-68] Although orthodontic treatment is associated with some maxillary central incisor EARR in most patients, and more than one third of those treated experience greater than 3 mm of loss, severe EARR (more than 5 mm) occurs in 2% to 5%.[69,70]

Currently, there are no reliable markers to predict which patients will develop EARR nor how severe EARR will be following orthodontic tooth movement;' although the shape of the root does appear to be associated with the likelihood of EARR and is best examined on periapical rather than panoramic radiographs.[72] Even when duration of treatment is a factor, it along with several significant dentofacial structural measurements (such as overjet) do not account for enough of the observed variability to be useful as predictors of EARR by themselves."

The degree and severity of EARR associated with orthodontic treatment is multifactorial, involving host and environmental factors, with genetic factors accounting for at least 50% of the variation. A polymorphism in the IL-1(3 (IL-1B) gene in orthodontically treated individuals accounts for 15% of the variation in maxillary central incisor EARR. Individuals homozygous for the IL-1B allele 1 have a 5.6-fold (95% confidence interval, 1.9 to 21.2) increased risk of EARR greater than 2 mm compared with individuals who are not homozygous for the IL-1B allele 1.[73] Although this is the first genetic marker suggested to be associated with EARR, it accounts for too small an amount of the total variation to be predictive. Additional genetic studies such as the one that found genetic linkage for EARR with a marker on chromosome 18 near the RANK gene are needed to determine what other genes influence EARR.[7'] Future estimation of susceptibility to EARR will likely require the analysis of a number of genes, root morphology, dental and facial measurement values, and the amount of tooth movement planned for treatment.

GENETICS OF CLEFT LIP AND PALATE

Studies of the CLP phenotype in twins indicate that monozygous twins have a 35% concordance rate, whereas dizygous twins show less than 5% concordance.[75] Information from two sources (families and twins) then establishes a genetic basis for CLP, but despite many extensive investigations, no simple pattern of inheritance has been demonstrated. This has led to proposal of a variety of genetic modes of inheritance for CLP, including dominance, recessiveness, and sex linkage, and has led ultimately to the documentation of modifying conditions that may be present, such as incomplete penetrance and variable gene expressivity.[76] There are three important reasons for the failure to resolve the question of a hereditary basis for clefts:
(1) some clefts are of a nongenetic origin and should not be included in a genetic analysis; unfortunately, such cases are seldom recognized and are difficult to prove;
(2) individuals who have increased genetic liability for having a child with CLP often fail to be recognized, but because they do not have CLP themselves they cannot be identified with certainty; this latter situation defines the problem of nonpenetrance for genes that control CL P[77]; and (3) CLP, although sometimes appearing to be relatively simple in origin, is undoubtedly a complex of diseases with different etiologies lumped together because of clinical disease resemblance (they all show clefting).

There are two clearly recognized groups of etiologically different clefts—cleft lip either with or without cleft palate (CL[P]) and isolated cleft palate (CP). These two entities, CLIP) and CP, occur as single cases in a family and as multiple cases in a family. In the former they are called sporadic, and in the latter they are called familial or multiplex. Some researchers refer to multiplex cases as those individuals with findings in addition to an oral cleft, even if a specific syndrome is not recognized. It should also be noted that the isolated CP that occurs without a cleft of the lip is different from the palatal cleft that occurs as a part of CLP. The embryology and developmental timing are both different, and isolated CP is more commonly

TABLE 6-2 Potential Etiologic Groups Within Cleft Lip with or without Cleft Palate (CL[P]) and Cleft Palate (CP)

	SPORADIC CASES	FAMILIAL CASES	CLEFT SYNDROMES
CL(P)	74%	25%	1%
Recurrence risk	Less than 1%	16%	Risk varies from 0.1% to 50%
Isolated CP	80%	12%	8%
Recurrence risk	Less than 1%	16%	Risk varies from 0.1% to 50%

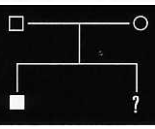
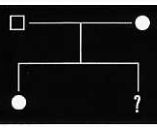
the result of an environmental teratogen than is CLP. When all potential study groups for CL(P) and CP are considered, the minimum number is six: three subgroups for CLIP) and three for CP. These three for each type of cleft are the sporadic and the familial groups and a small group of syndromes that feature CL(P) and/or CP. Although there are many such recognizable syndromes, they are relatively rare in occurrence and may not provide much information about the nonsyndromic clefting process. Thus, if these syndromic cases are omitted, the four groups that remain may yield useful information concerning possible genetic origin.

Table 6-2 gives a breakdown of the numbers to be found in these four groups. It can be seen that sporadic cases of both CL(P) and CP are in a large majority, comprising about three fourths of all cases. On the other hand, the familial cases, although not as common, still make up 12% to 20% of cleft cases. As noted earlier, it is probable that minor and subtle facial changes are more likely to produce the best-correlated phenotype needed to pinpoint the cleft genotype. Part of the reason for this view is the suspicion that certain facial shapes are more predisposed to developing CLP than others.[78] Although this approach seems best for producing an accurately generated clefting phenotype, as yet there are few data to substantiate it. This also means that we must go a lot farther in our study of the developmental anatomy of the head and face.

Recurrence risks for CL(P) and CP have been reported in which penetrance was not considered. These data are frequently cited by genetic counselors as illustrating the lesser risk associated with a multifactorial than with a monogenic trait. The reader will note that these data consider CL(P) and CP without regard to the three groups discussed previously. These data, therefore, are average risk figures (Table 6-3).

The published data on nonsyndromic cleft populations comes from around the world (Japan, China, Hawaii, Denmark, Sweden, Great Britain, and North America). These studies make it clear that both CL(P) and CP are heterogeneous diseases. That is, there are multiple causes for the single phenotypes, CL(P), and CP. To summarize the generally accepted hereditary basis for CL(P) and CP: Single, nonsyndromic cases of CLIP) and CP, or sporadic clefts, are believed to be the result of a complex interaction between multiple genetic and environmental factors. Hence, their etiology is multifactorial in the true sense of the word, and the chance that these multiple factors would interact to produce a cleft phenotype in relatives is small, probably less than 1%.

TABLE 6-3 Recurrence Risks for Cleft Lip with or without Cleft Palate (CL[P]) and Isolated Cleft Palate (CP)

		MATING TYPE 	
Fogh-Andersen[76]	4%	2%	14%
CLIP)	12%	7%	17%
CP*			
Curtis and Walker[79]	4%	4%	19%
CL(P)	2%	6%	14%
CP*			
Curtis, Fraser, and Warburton[80]	4%	—	17%
CLIP)	7%	—	15%
CP*			

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The other nonsyndromic group consists of multiple cases of clefts that occur in a single family. These are called familial (or multiplex) and have been viewed by researchers as the "true" genetic cases. Familial occurrences of CL(P) and CP seem most likely to be accounted for by the action of a single major gene, but the influence of multifactorial trait factors is difficult to rule out. Thus we are left with the idea that both multifactorial and single major gene elements may have a role in producing sporadic and familial cases of CL(P) and CP.

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CHAPTER

7

Acquired and Developmental Disturbances of the Teeth and Associated Oral Structures

RALPH E. McDONALD

DAVID R. AVERY

JAMES K. HARTSFIELD, JR.

انتشارات
شایان نمودار

CHAPTER OUTLINE

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- Trauma to the Tongue with Emphasis on Tongue Piercing

ABNORMAL LABIAL FRENUM
- Frenectomy

COMMON DISTURBANCES IN CHILDREN

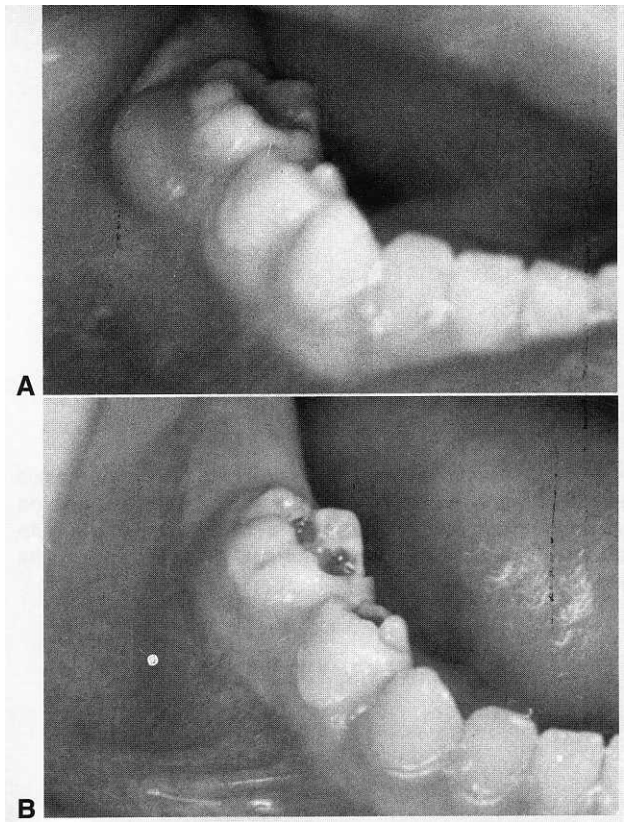
Dental care services have become more readily available to children, and caries prevention programs have become more effective. There has been a steady decline in the incidence and prevalence of dental caries in permanent teeth among U.S. children (see Chapter 10). However, according to the first-ever Surgeon General's report on oral health in America published in May 2000, dental caries is the single most common chronic childhood disease) Periodontal disturbances are also common. Although severe forms of periodontal disease are rare in children, all experience at least mild gingivitis on occasion. Both caries and periodontal disease are, for the most part, acquired and preventable disturbances of the teeth and jaws. Other chapters of this book are devoted to a more in-depth discussion of the cause, prevention, and management of dental caries (see Chapters 10, 17, 18, and 19) and periodontal disturbances (see Chapters 11 and 20). Injuries to the teeth and supporting tissues represent another large category of acquired disturbances (see Chapter 21).

Many children have orthodontic conditions that justify corrective treatment, and for some of them the condition is serious enough to be categorized as deforming or crippling. Approximately 1 in 1000 children in the United States is born with a cleft lip or palate. These conditions are primarily developmental disturbances and are discussed in greater detail in Chapters 6 and 25 to 28.

ALVEOLAR ABSCESS

During the examination procedure the dentist may observe evidence of an acute or chronic alveolar abscess. An alveolar abscess associated with the pulpless permanent tooth is usually a specific lesion localized by a fibrous capsule produced by fibroblasts that differentiate from the periodontal membrane. The primary tooth abscess is usually evident as a more diffuse infection, and the surrounding tissue is less able to wall off the process. The virulence of the microorganisms and the ability of the tissues to react to the infection probably determine whether the infection will be acute or chronic.

In the early stages the acute alveolar abscess can be diagnosed based on radiographic evidence of a thickened periodontal membrane. The tooth is sensitive to percussion and movement, and the patient may have a slight fever. The acute symptoms of an alveolar abscess can be relieved by using antibiotic therapy. Relief of the symptoms is more efficient if drainage is also established (Fig. 7-1). Drainage may be established through the pulp chamber of the tooth and/or the associated gingiva or by extraction of the tooth. If extraction is selected as the best choice of treatment at the emergency appointment and the patient has an unremarkable medical history, concomitant antibiotic therapy is not always necessary. During this acute infection phase, however, it may be impossible to establish effective pain control for the extraction procedure with conventional out-patient techniques.



E I G . 7-1. A, Acute alveolar abscess associated with a pulpless second primary molar. **B,** Removal of the roof of the pulp chamber to allow drainage resulted in immediate relief of pain. After the swelling has been reduced, one can decide whether the tooth is to be treated or extracted.

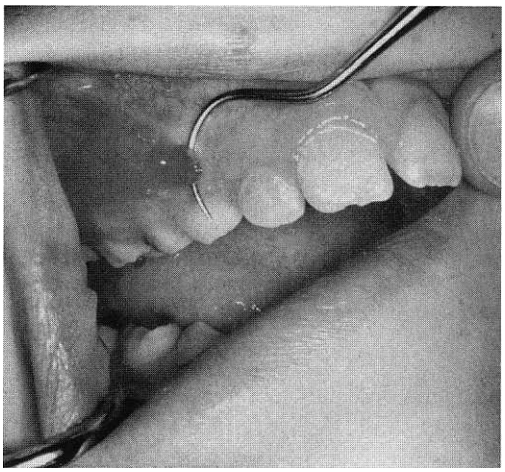
If establishing drainage through the pulp chamber is selected as part of the emergency treatment, a large opening should be made into the pulp chamber to permit drainage of the exudate. After initial debridement and rinsing, the opening to the chamber may be closed unless drainage persists to ooze indefinitely. Should pain occur during the excavation of tooth structure to establish drainage, the discomfort can be lessened if the tooth is stabilized by the dentist's fingers.

Warm saline mouth rinses often aid in localizing the infection and maintaining adequate drainage before endodontic treatment or extraction.

Chronic alveolar abscess, characterized by less soreness, is often a better-defined radiographic lesion. The patient will likely have some lymphadenopathy as well.



I G. **7-2.** Chronic alveolar abscess associated with a pulpless second primary molar that is also a candidate for incision and drainage, in addition to removal of the roof of the pulp chamber to initiate root canal therapy if the tooth is to be saved.



G I G. **7-3.** A pedunculated granulomatous lesion overlying the canine but associated with a chronic draining alveolar abscess of the maxillary right first primary molar.

Draining fistulas are also frequently associated with chronic alveolar abscesses. Usually, antibiotic therapy is unnecessary except in patients with an overriding systemic problem (e.g., patients susceptible to subacute bacterial endocarditis, patients with organ transplants, or those who are immunodeficient). Again, drainage and sterilization of the infected local area are necessary through root canal therapy for the involved tooth or through extraction. If the lesion has only recently passed the acute stage, there may be a pointing soft tissue abscess. In this situation, incising and draining the soft tissue may be indicated in addition to opening the tooth, especially if the tooth is to be treated endodontically (Fig. 7-2). If the lesion is in an advanced chronic stage, drainage may already be established as a natural reaction (Fig. 7-3).

CELLULITIS

Cellulitis is a diffuse type of infection of the soft tissues that may be caused by a pulpless primary or permanent tooth. It often causes considerable swelling of the face or neck, and the tissue appears discolored.

Cellulitis is a very serious infection. It can be life-threatening and is a potential complication of all acute dental infections. It is usually a result of severe untreated caries in patients who do not receive regular dental care or who may have had dental care only for treatment of dental emergencies. It is not unusual for the parents to take the child with dental cellulitis to the hospital emergency department. The child appears acutely ill and may have an alarmingly high temperature with malaise and lethargy.

If a maxillary tooth is the problem, the swelling and redness may involve the eye (Fig. 7-4, *A).* If cellulitis is treated too late, serious complications, such as involvement of the central nervous system or a cavernous sinus thrombosis, could occur. If cellulitis results from an infected mandibular tooth, the diffuse swelling and infection will spread to the floor of the mouth along fascial planes, nerves, and vessels. If the infection involves the submandibular, sublingual, and submental spaces, it is called *Ludwig angina.* In this condition the tongue and floor of the mouth become elevated to the extent that the patient's airway is obstructed and swallowing is impossible.

The establishment of drainage, if possible, by opening the pulp chamber of the affected tooth is helpful in reducing the acute symptoms of cellulitis. However, the child may have difficulty opening the mouth to permit the procedure. Incision of soft tissue to establish drainage is not indicated in the early stages of cellulitis because of the diffuse, nonlocalized nature of the infection.

The offending organisms in cellulitis from dental infections are usually capable of producing hyaluronidase and fibrinolysins. These agents break down the inter-cellular cementing substance (hyaluronic acid) and fibrin, which permits the rapid spread of the infection. One of the broad-spectrum antibiotics should be pre-scribed early to reduce the possibility of the infection localizing and draining on the outer surface of the face (Fig. 7-4, B). It should be emphasized to the parents or patient that antibiotics will not heal the condition completely and that follow-up treatment of the tooth is essential.

If the infection is already severe when the parent or patient seeks treatment, a blood culture or a culture of exudate may be performed to identify the infecting organisms. Then, if the infection does not respond to the initial antibiotic therapy, a second, more appropriate



[I C 7-4. **A,** Patient appears to be acutely ill because of an infected permanent molar and *resultant cellulitis.* ***B,*** *Use of broad-spectrum antibiotics reduced the acute symptoms of the* disease and prevented extraoral drainage.

antibiotic may be selected after the causative organisms have been identified. Molinari has emphasized the continuing emergence of antibiotic-resistant bacterial strains that renders many common antimicrobial agents ineffective.[2]

The child with cellulitis should be hospitalized if the clinical signs or symptoms warrant very close monitoring or if there is any question as to whether the parents or patient will follow through with the prescribed treatment. Hospitalization is recommended especially in the case of Ludwig angina, since maintenance of a patent airway may require the assistance of medical personnel. In these severe cases, parenteral administration of antibiotics is also recommended, at least initially.

DEVELOPMENTAL ANOMALIES OF THE TEETH

ODONTOMA

The abnormal proliferation of cells of the enamel organ may result in an odontogenic tumor, commonly referred to as an *odontoma.* An odontoma may form as a result of continued budding of the primary or permanent tooth germ or as a result of an abnormal proliferation of the cells of the tooth germ, in which case an odontoma replaces the normal tooth (Figs. 7-5 and 7-6). The anomaly is discussed in detail in Chapter 8. An odontoma should be surgically removed before it can interfere with eruption of teeth in the area. The presence of an odontoma should alert the practitioner to inquire about the concurrent presence of dysphagia or a family history of dysphagia that is perhaps due to hypertrophy of the smooth muscles of the esophagus as a part of the rare autosomal dominant odontoma-dysphagia syndrome.[3]

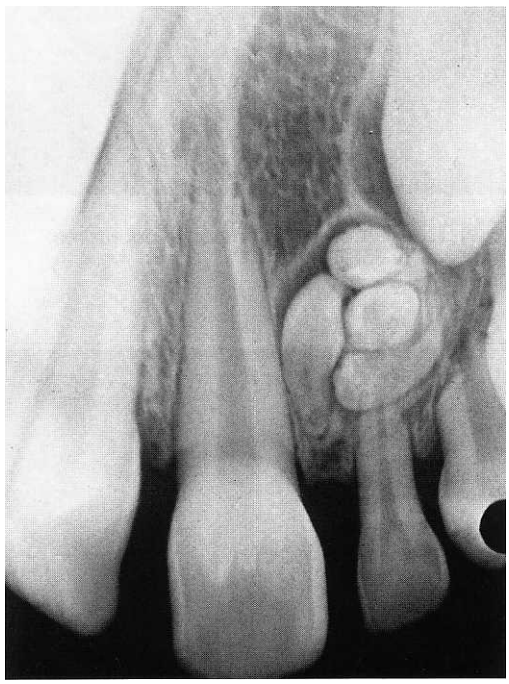
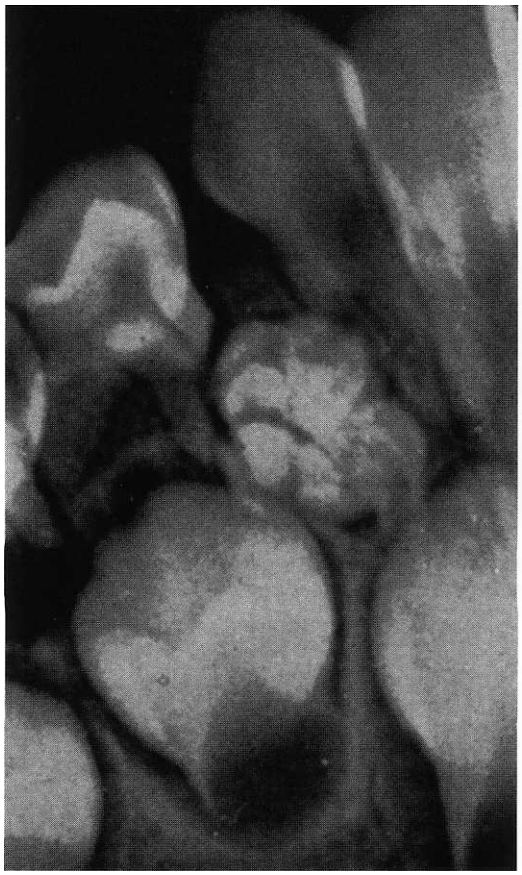


FIG **7-5.** Compound composite odontoma. The anomalous structure consists of small structures resembling teeth.

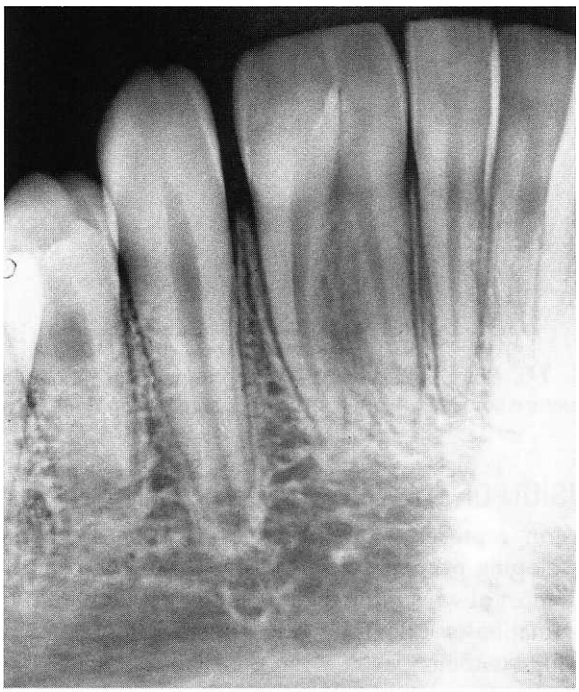
FUSION OF THE TEETH

Fusion represents the union of two independently developing primary or permanent teeth. The condition is almost always limited to the anterior teeth and, like gemination (see the following discussion), may show a familial tendency.

The radiograph may show that the fusion is limited to the crowns and roots. Fused teeth will have separate



f**I G 7-6.** Complex composite odontoma.



pulp chambers and separate pulp canals (Fig. 7-7). Dental caries may develop in the line of fusion of the crowns, necessitating the placement of a restoration. A frequent finding in fusion of primary teeth is the congenital absence of one of the corresponding permanent teeth.

Delaney and Goldblatt reported an interesting case that illustrates the multidisciplinary approach that may be indicated in the clinical management of certain problems associated with fused teeth.' The disciplines of pediatric dentistry, endodontics, surgery, restorative dentistry, and orthodontics were represented in the initial management of the case, and a post and core and a crown restoration were anticipated for the future. Yet excellent results were obtained in only 6 months with an organized approach to a complex problem that involved the fusion of a supernumerary tooth to a maxillary central incisor, severe crowding, and a palatally displaced lateral incisor. One root of the fused teeth was treated endodontically. The fused teeth were then hemi-sectioned, and the endodontically treated tooth was restored while the separated tooth was sacrificed. Orthodontic repositioning of the palatally displaced lateral incisor and alignment of the anterior segment concluded the management of the problem. A case of bilateral fusion of primary incisors has also been reported by Eidelman.[5]

The presence of a single primary and permanent maxillary central incisor may at first appear to be a product of fusion. However, if the single tooth is in the midline, and symmetric with normal crown and root shape and size, then it may be an isolated finding or may be part of the solitary median maxillary central incisor syndrome. This is a heterogeneous condition that may include other midline developmental abnormalities of the brain and other structures that can be due to mutation in the sonic hedgehog *(SHH)* gene, *SIX3* gene, or other genetic abnormality.' The development of only one maxillary central incisor is an indication for further evaluation for other anomalies.

GEMINATION

A geminated tooth represents an attempted division of a single tooth germ by invagination occurring during the proliferation stage of the growth cycle of the tooth. The geminated tooth appears clinically as a bifid crown on a single root (Fig. 7-8). The crown is usually wider than normal, with a shallow groove extending from the incisal edge to the cervical region. The anomaly, which may follow a hereditary pattern of occurrence, is seen in both primary and permanent teeth, though it probably appears more frequently in primary teeth.

The treatment of a permanent anterior geminated

f I G. 7-7. Fusion of a permanent central and lateral incisor. tooth may involve reduction of the mesiodistal width of



F I I. **7-8.** Gemination of a mandibular lateral incisor. The crown has a groove on the labial surface and is wider than normal.

the tooth to allow normal development of the occlusion. Periodic disking of the tooth is recommended when the crown is not excessively large, as is eventual preparation of the tooth for restoration if dentin is exposed. Secondary dentin formation and pulpal recession will follow judicial periodic reduction of crown size. Devitalization of the tooth and root canal therapy followed by the construction of a postcrown may be needed when the geminated tooth is large and malformed.

DENS IN DENTE (DENS INVAGINATUS)

The diagnosis of dens in dente (tooth within a tooth) can be verified by a radiograph. The developmental anomaly has been described as a lingual invagination of the enamel. This condition can occur in primary and permanent teeth. Unusual cases of dens invaginatus have been reported in a mandibular primary canine,' a maxillary primary central incisor[s] and a mandibular second primary molar.[9] Dens in dente is most often seen in the permanent maxillary lateral incisors. The condition should be suspected whenever deep lingual pits are observed in maxillary permanent lateral incisors. Grahnen, Lindahl, and Omnell found in a study of 58 families that a similar anomaly was present in more than one third of the parents of children with dens in dente.[10] Within the same family some had dens in dente and others had deep lingual pits, which indicates that the condition may be inherited as an autosomal dominant trait with variable expressivity and possibly incomplete penetrance (see Chapter 6 for definitions).

Anterior teeth with dens in dente are usually of normal shape and size. In other areas of the mouth, however, the tooth can have an anomalous appearance. A dens in dente is characterized by an invagination lined with enamel and the presence of a foramen cecum with the probability of a communication between the cavity of the invagination and the pulp chamber (Fig. 7-9).

Application of sealant or a restoration in the opening of the invagination is the recommended treatment to prevent pulpal involvement. If the condition is detected before complete eruption of the tooth, the removal of gingival tissue to facilitate cavity preparation and restoration may be indicated.

The advisability of performing endodontic procedures on such a tooth with pulpal degeneration depends on its pulp morphology and the restorability of the crown (Fig. 7-10).

EARLY EXFOLIATION OF TEETH

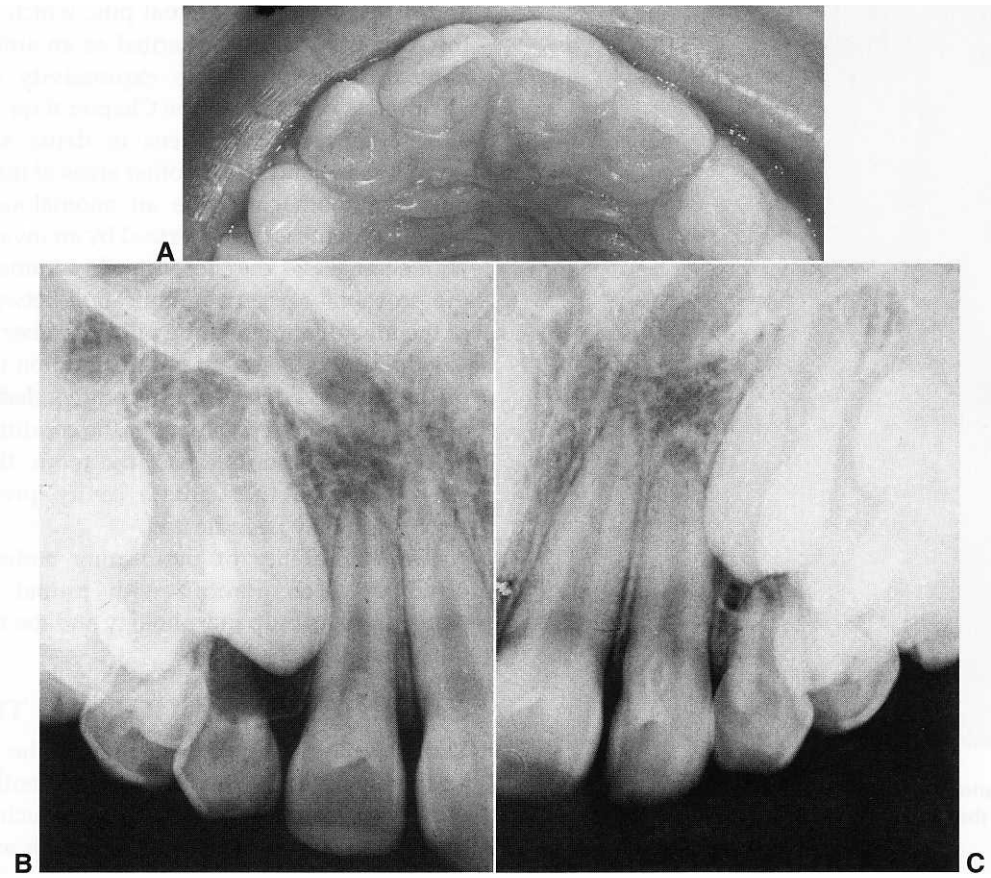
Variations in the time of eruption of the primary teeth and in the time of exfoliation are frequently observed in pediatric patients. A variation of as much as 18 months in the exfoliation time of primary teeth may be considered normal. However, this pattern must be consistent with other aspects of the dental development. Exfoliation of teeth in the absence of trauma in children younger than 5 years of age merits special attention because it can be related to pathologic conditions of local and systemic origin.

The early exfoliation of primary teeth resulting from periodontitis has been observed occasionally in young children, and this is discussed in Chapters 6 and 20. Along with hypophosphatasia, prepubertal periodontitis appears to be the most common cause of premature exfoliation of the primary teeth, especially in girls.

HYPOPHOSPHATASIA

The clinical dental finding diagnostic of hypophosphatasia in children is premature exfoliation of the anterior primary teeth associated with deficient cementum. The loss of teeth in the young child may be spontaneous or may result from a slight trauma. Severe gingival inflammation will be absent. The loss of alveolar bone may be limited to the anterior region. The disease is characterized by improper mineralization of bone caused by deficient alkaline phosphatase activity in serum, liver, bone, and kidney (tissue nonspecific). Increased levels of urinary phosphoethanolamine are also seen.

Ongoing study into the etiology of hypophosphatasia indicates that the usually lethal autosomal recessive



F **I G 7-9. A,** Small, "nonsticky" pits on the lingual surfaces of the maxillary lateral incisors are the only clues to the dens in dente condition of the teeth revealed radiographically in **B** and **C.**



[I G 7-10. Dens in dente in a maxillary lateral incisor. A communication between the invagination and the pulp chamber apparently caused necrosis of the pulp.

infantile type (I), as well as the usually autosomal recessive milder juvenile type (II) and the autosomal dominant adult type (III), are due to different mutations and heterozygosity versus homozygosity in the tissue-nonspecific alkaline phosphatase *(TNSALP* or *ALPL)* gene. Hu et al state that, as a general rule, the earlier the appearance of the disease the greater the severity." Early exfoliation of the primary teeth is usually associated with the juvenile type (II), although such a history may also be present in the adult type (III)." Diagnostic tests should include the determination of serum alkaline phosphatase levels for parents and siblings.

Pseudohypophosphatasia, first described by Scriver and Cameron, is a rare disorder in which the child has the phenotype of juvenile hypophosphatasia and elevated levels of urinary phosphoethanolamine but plasma alkaline phosphatase activity is normal.[12] However, the clinical findings are similar to those of juvenile hypophosphatasia.

CHERUBISM (FAMILIAL FIBROUS DYSPLASIA)

Cherubism is a rare childhood disease affecting jaw development. Cherubism is usually inherited as an autosomal dominant trait with somewhat reduced

penetrance in females. The expression may be so variable that a clinically normal-appearing parent may have a history of prominent facial swellings or radiographic evidence of abnormal bone pattern in the mandible. Although disease progression is expected to stabilize or even regress after puberty, a few very aggressive cases, sometimes producing morbid results, have been reported.[13-16] At least four cases of nonfamilial cherubism have been reported, which suggests occasional sporadic occurrences from spontaneous mutations.[17-19]

Two independent groups of investigators[20,21] have demonstrated that the gene for cherubism maps to chromosome 4p16. Follow-up work by Ueki et al found that mutations in the gene encoding c-Abl–binding protein SH3BP2 on chromosome 4p16.3 causes cherubism.[22] They found several different *SH3BP2* gene mutations in patients with cherubism, which probably resulted in a gain of function or dominant-negative effect, although some other gene(s) may also cause cherubism in other patients. They postulated that the onset of cherubism and its anatomically circumscribed characteristics may be related to dental developmental processes in children, when signals unique to the mandible and maxilla are transmitted through the extracellular matrix, triggered by the eruption of secondary teeth.

A symmetric or asymmetric enlargement of the jaws may be noted at an early age. Numerous sharp, well-defined multilocular areas of bone destruction and thinning of the cortical plate are evident in the radiograph (Fig. 7-11). Teeth in the involved area are frequently exfoliated prematurely as a result of the loss of support or root resorption or, in permanent teeth, as a result of an interference in the development of roots. Spontaneous loss of the teeth may occur, or the child may pick the teeth out of the soft tissue.

McDonald and Shafer reported a case in which the mandible and maxilla of a 5-year-old girl were symmetrically enlarged.[23] Radiographs showed multilocular cystic involvement of both mandible and maxilla. A complete skeletal survey failed to reveal similar lesions in other bones. Microscopic examination of a segment of bone showed a large number of multinucleated giant cells scattered diffusely throughout a cellular stroma. The giant cells were large and irregular in shape and contained 30 to 40 nuclei. During a 10-year observation period the bony lesions had not progressed appreciably.

The patient illustrated in Fig. 7-11 was followed into adulthood, and her mouth was restored in a very satisfactory manner. A comparison of the full-face photographs in Fig. 7-11 *(A* and I) illustrates that, as the face increases in height, the "cherubic" appearance caused by the bilateral bulging of the bone of the mandible is less apparent. Seven permanent teeth in the upper and lower arches were retained and prepared for Baker attachments. Complete dentures were constructed to restore function and improve appearance.

Pierce et al have reported their dental management of a mother and two daughters with inherited craniofacial fibrous dysplasia over a 15-year period." The daughters exhibited clinical and radiographic appearances similar to that of the patient just described, but the authors consider fibrous dysplasia and cherubism to be separate entities. Their treatment of the daughters, though more aggressive, resulted in nearly complete dentitions. Treatment included surgical autotransplantation of several teeth and bony recontouring. Orthodontic therapy was also provided for one child. Orthodontics was recommended for the other child but was declined; her dental alignment was acceptable 3 years after surgery.

Peters has reported a study of 20 cases of cherubism in one family that confirmed an autosomal dominant pattern of inheritance.[2'] The author preferred a conservative approach in managing the giant cell lesions, because they tend to resolve with maturity. Von Wowern has published an extensive review of the literature and a 36-year follow-up of families with cherubism.[26]

ACRODYNIA

The exposure of young children to minute amounts of mercury is responsible for a condition referred to as *acrodynia* or *pink disease*. Ointments and medications are the usual sources of the mercury. Weinstein and Bernstein recently reported on 20-month-old twin girls who presented to a hospital with the classic signs and symptoms of acrodynia.[27] Further investigation revealed that the girls had been receiving once- or twice-weekly doses of a mercury-containing teething powder during the preceding 4 months. Apparently such preparations are still available in some countries. Horowitz et al report on two young brothers diagnosed with acrodynia after playing repeatedly with a broken sphygmomanometer." Dental amalgam restorations do not cause acrodynia.

The clinical features of the disease include fever, anorexia, desquamation of the soles and palms (causing them to be pink), sweating, tachycardia, gastrointestinal disturbance, and hypotonia. The oral findings include inflammation and ulceration of the mucous membrane, excessive salivation, loss of alveolar bone, and premature exfoliation of teeth.

HYPOPHOSPHATEMIA (FAMILIAL OR X-LINKED HYPOPHOSPHATEMIC RICKETS OR VITAMIN D-RESISTANT RICKETS)

As reported by Hartsfield, hypophosphatemia is the most common inherited abnormality of renal tubular transport.[29] Clinical features become evident in the second year of life. They include short stature and

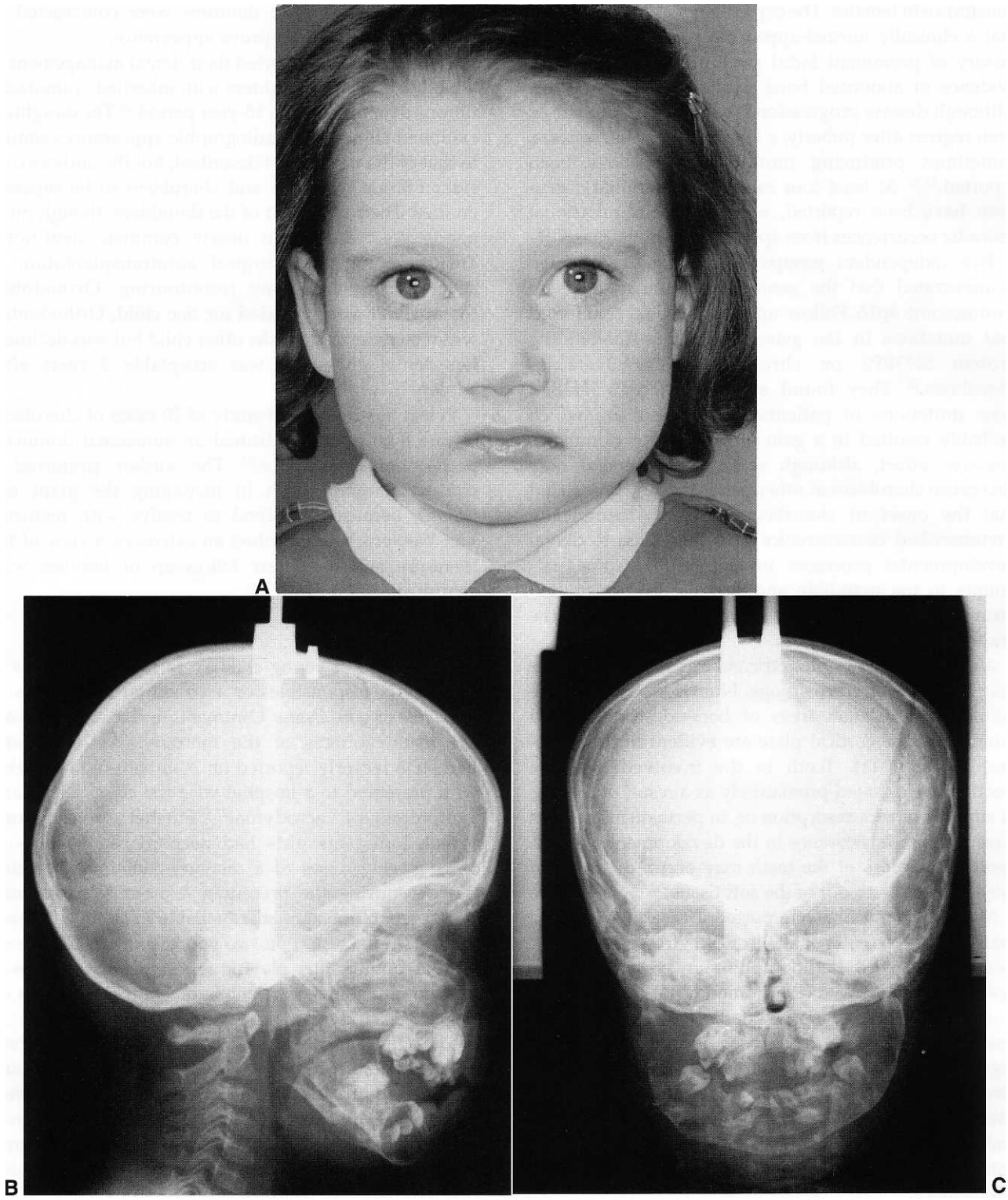


FIG. **7-11. A,** Enlargement of the cheeks caused by bilateral bulging of the bone of the mandible. **B** and C, lateral and anteroposterior cephalometric radiographs. Notice the displacement of the mandibular anterior teeth in a large area of bone destruction, the locular cystic involvement of the mandible and maxillae, and the number of missing teeth.

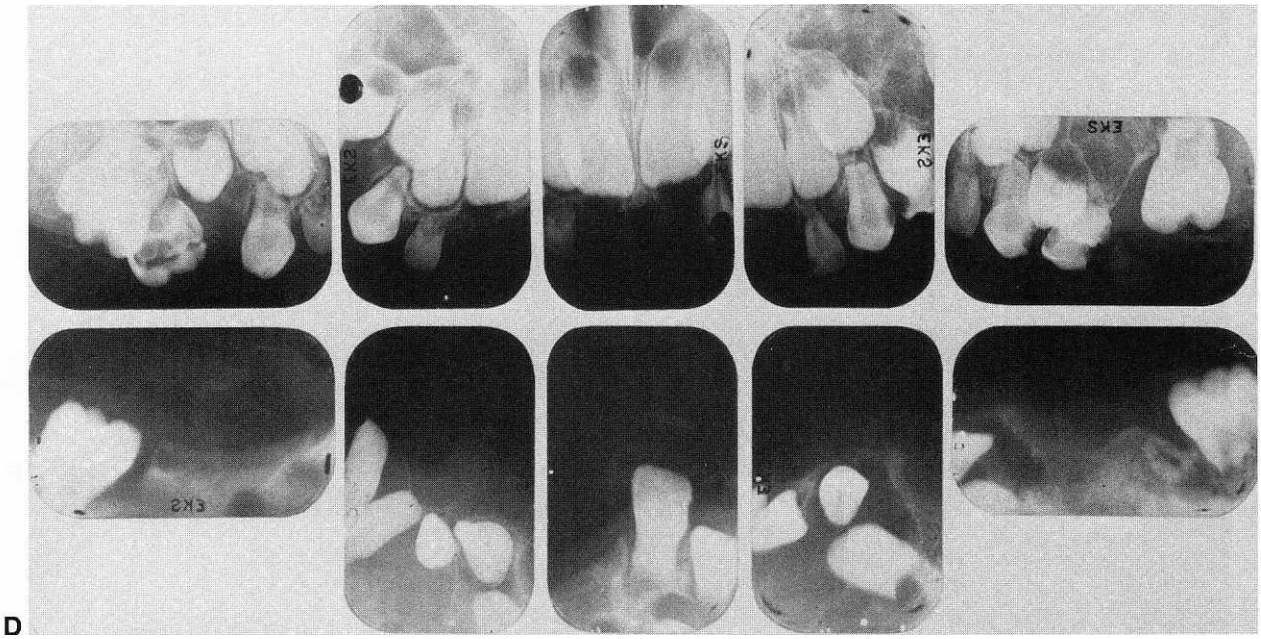


FIG 7-11, cont'd **D,** Full-mouth radiographs demonstrating large areas of bone destruction and several missing teeth.

Continued

bowing of the lower extremities in affected boys. Premature tooth exfoliation is sometimes also a feature. The inheritance pattern of the disease is usually X-linked dominant, and the disorder is twice as common in females as in males. The HYP Consortium of investigators found mutations in the *PHEX* (also called PEX) gene on the X chromosome in patients with this condition.[30]

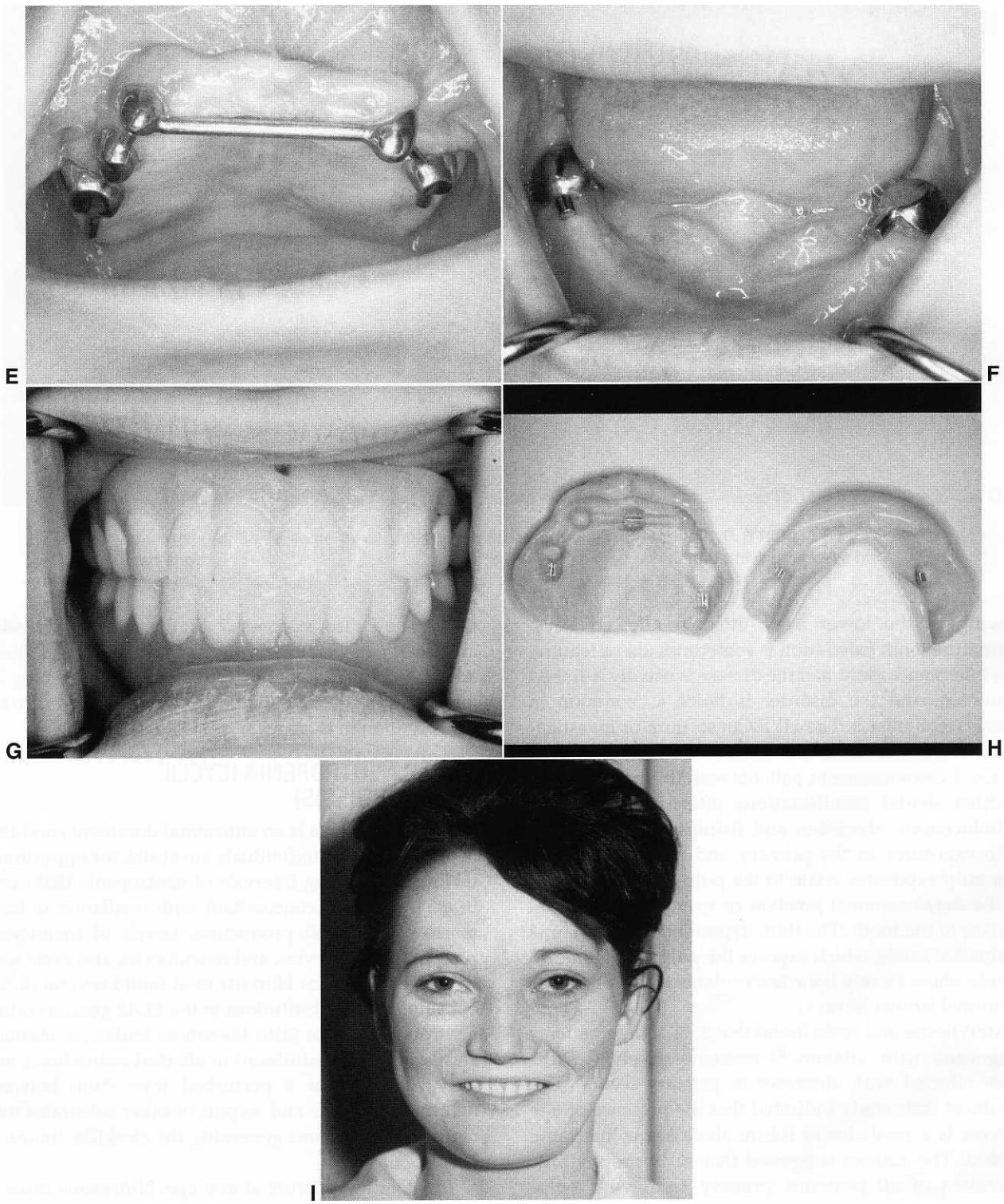
Other dental manifestations often include apical radiolucencies, abscesses, and fistulas associated with pulp exposures in the primary and permanent teeth. The pulp exposures relate to the pulp horns extending to the dentinoenamel junction or even to the external surface of the tooth. The thin, hypomineralized enamel is abraded easily, which exposes the pulp. Dental radio-graphs show rickety bone trabeculations and absent or abnormal lamina dura.

McWhorter and Seale found that 25% of their patients diagnosed with vitamin D–resistant rickets (VDRR) were affected with abscesses in primary teeth.[31] The results of their study indicated that the presence of one abscess is a predictor of future abscesses in the same patient. The authors suggested that early prophylactic treatment of all posterior primary teeth with pulpotomies and crown placement may be the most conservative therapy for a VDRR patient who develops a spontaneous abscess. However, a follow-up retrospective study by Shroff, McWhorter, and Seale found the success rate for prophylactic pulpotomies in these patients to be only 44%.[32] They concluded that prophylactic pulpotomy therapy cannot be recommended for patients with VDRR based on the currently available data. They also suggested that a more aggressive approach using prophylactic pulpectomy as previously advocated by Rakocz, Keating, and Johnson may be indicated in these patients and encouraged further investigation in this area.[33]

CYCLIC NEUTROPENIA (CYCLIC HEMATOPOIESIS)

Cyclic neutropenia is an autosomal dominant condition in which affected individuals are at risk for opportunistic infection during intervals of neutropenia that occur in a 21-day cycle concomitant with oscillation in bone marrow blood cell production. Levels of monocytes, platelets, lymphocytes, and reticulocytes also cycle with the same frequency. Horwitz et al found several different single-base substitutions in the *ELA2* gene encoding neutrophil elastase (also known as leukocyte elastase, elastase 2, and medullasin) in affected individuals, and hypothesized that a perturbed interaction between neutrophil elastase and serpins or other substrates may regulate mechanisms governing the clocklike timing of hematopoiesis.[34]

The condition occurs at any age. Numerous cases in children have been reported. The patients manifest a fever, malaise, sore throat, stomatitis, and regional lymphadenopathy, as well as headache, cutaneous infection, and conjunctivitis. Children exhibit a severe gingivitis with ulceration. With a return of the neutrophil count to normal, the gingiva may return to a nearly normal clinical appearance. Children experiencing repeated insults from the condition have a



f I C. 7-11, cont'd E, to **H,** When the patient was 18 years of age, the permanent teeth that had good support were prepared for Baker attachments, and complete dentures were constructed. I, The restored mouth and improved appearance of the adult. *(E to H courtesy Dr. Donald Cunningham.)*

considerable loss of supporting bone around the teeth. A case report by da Fonseca and Fontes describes a young woman who had suffered from poor oral health throughout her lifetime, and as she approached 21 years of age, all her remaining permanent teeth were finally removed.' Soon after the extractions the patient's blood counts improved to a level not previously seen by her hematologist.

OTHER DISORDERS

Premature exfoliation or marked mobility of teeth in childhood or adolescence has been associated in some cases with other systemic disorders, including acatalasia, Chediak-Higashi syndrome, Coffin-Lowry syndrome, Down syndrome, Ehlers-Danlos syndrome types IV and VIII, Hajdu-Cheney syndrome, hyperpituitarism, hyperthyroidism, juvenile diabetes, Papillon-Lefevre syndrome, progeria, Singleton-Merten syndrome, and some malignant diseases such as the histiocytosis X groups (see Chapter 8) and the leukemias (see Chapter 24).

ENAMEL HYPOPLASIA

Amelogenesis occurs in two stages. In the first stage, the enamel matrix forms, and in the second stage, the matrix undergoes calcification. Local or systemic factors that interfere with normal matrix formation cause enamel surface defects and irregularities called *enamel hypoplasia.* Factors that interfere with calcification and maturation of the enamel produce a condition termed *enamel hypocalcification.*

Enamel hypoplasia may be mild and may result in a pitting of the enamel surface or in the development of a horizontal line across the enamel of the crown. If ameloblastic activity has been disrupted for a long period, gross areas of irregular or imperfect enamel formation occur. Enamel hypoplasia is often seen as one component of many different syndromes.

Postnatal hypoplasia of the primary teeth is probably as common as hypoplasia of the permanent teeth, although the former usually does not occur in as severe a form. Hypoplasia of the primary enamel that forms before birth is rare, however (Fig. 7-12). In its mildest form, a prenatal disturbance is reflected as an accentuated neonatal ring in the primary tooth. In the severe type of neonatal disturbance, enamel formation is some-times arrested at birth or during the neonatal period (Fig. 7-13). Postnatal amelogenesis is confined to the portion of the crown located cervically from the enamel area present at birth (Fig. 7-14).

Seow et al have observed that enamel hypoplasia of the primary teeth is common in prematurely born, very-low-birth-weight children; its pathogenesis is not understood clearly.'" One likely mechanism is related to mineral deficiency, which may be diagnosed

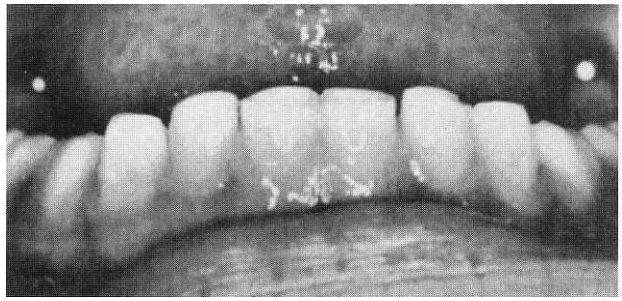
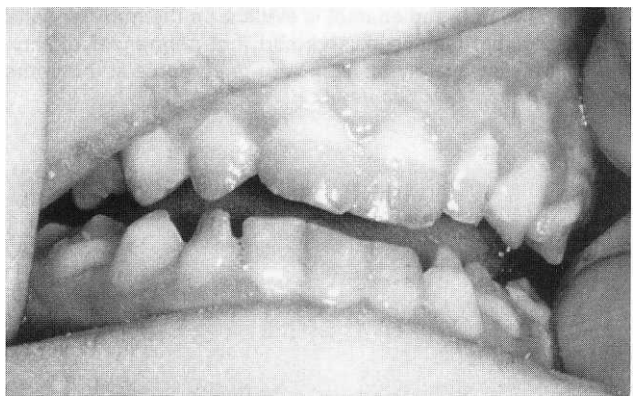


FIG **7-12.** Prenatal enamel hypoplasia. The medical history revealed that the patient suffered from cerebral palsy as a result of premature birth (gestation, 6 months; birth weight, 2 lb, 5 oz). *(Courtesy* Dr. *Stanley C. Herman.)*



f I G . **7-13.** Neonatal enamel hypoplasia. Only the most cervical parts of the intrinsically stained areas are hypoplastic. The child experienced severe nutritional deficiency during the first month of extrauterine life. *(Courtesy* Dr. *Stanley C.* Herman.)

radiologically as demineralization of long bones. These authors believe that both local and systemic factors are involved. An important local factor is trauma from laryngoscopy and endotracheal intubation, which usually results in localized enamel hypoplasia involving only the left maxillary anterior teeth. Slayton et al examined 698 well-nourished and healthy children 4 to 5 years of age and found that 6% had at least one primary tooth with enamel hypoplasia.[37]

HYPOPLASIA RESULTING FROM NUTRITIONAL DEFICIENCIES

Many clinical investigations have been undertaken to determine the relationship between hypoplastic defects of enamel and systemic disabilities. Relatively little importance has been placed on exanthematous fevers, but deficiency states, particularly those related to deficiencies in vitamins A, C, and D, calcium, and phosphorus, can often be related to the occurrence of enamel hypoplasia.

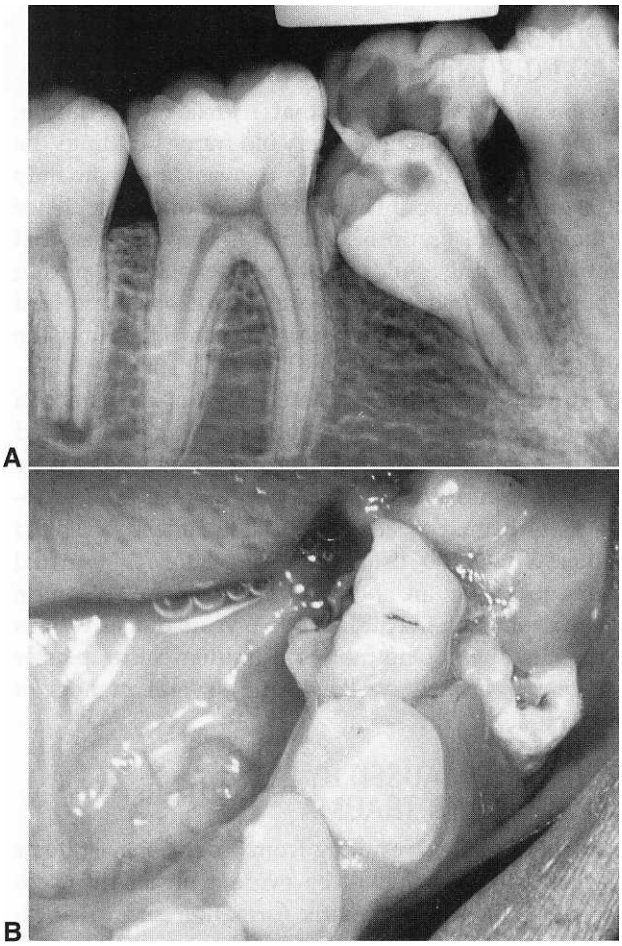
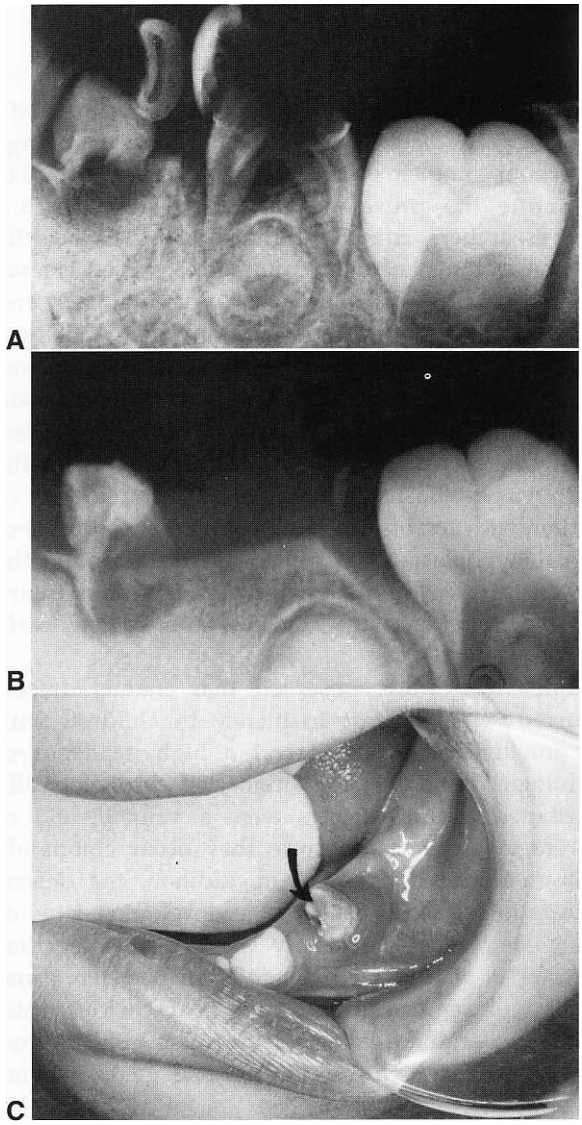


FIG. **7-17. A,** Infected mandibular second primary molar has caused hypoplasia of the second premolar and delayed eruption of the tooth. **B,** Hypoplasia is evident in the occlusal third of the second premolar.

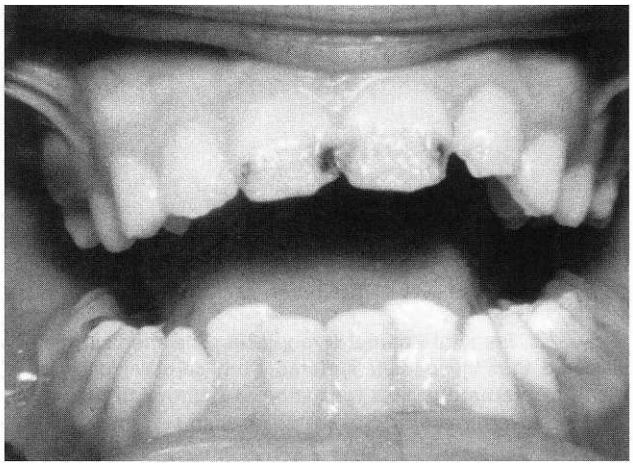


1 1G **7-18.** A, Only a root fragment remains as evidence of a pulpless first primary molar. The infection has affected the development of the first premolar. **B,** The second primary molar has been exfoliated prematurely. The first premolar is malformed as a result of the infection in the area. C, The malformed calcified mass (arrow) is surrounded by inflamed tissue.

A traumatic blow to an anterior primary tooth that causes its displacement apically can interfere with matrix formation or calcification of the underlying permanent tooth. The trauma or subsequent periapical infection frequently produces defects on the labial surface of the permanent incisor (Fig. 7-19). The retention of infected primary teeth, even if they are asymptomatic, is unjustifiable. The development of hypoplastic defects on the permanent tooth, its deflection from the normal path of eruption, and even death of the developing tooth may result.

HYPOPLASIA ASSOCIATED WITH CLEFT LIP AND PALATE

Mink studied the incidence of enamel hypoplasia of the maxillary anterior teeth in 98 patients with repaired bilateral and unilateral complete cleft lip and palate; the individuals ranged in age from $1^{1}/_{2}$ to 18 years.[51] Among patients in the repaired unilateral and bilateral complete cleft lip and palate group, 66% of those with maxillary anterior primary teeth had one or more primary teeth affected with enamel hypoplasia; 92% of those with erupted maxillary anterior permanent teeth had one or more permanent teeth affected with enamel hypoplasia. Mink concluded that the permanent teeth are in earlier stages of development at the time of the surgical procedure and are more subject to damage.[1] Dixon also attributed many of the defects of tooth structure and formation observed in patients with cleft palate to the reparative surgery.[J2] Vichi and Franchi, however, have suggested



f **G 7-14.** Enamel hypoplasia that occurred during infancy. A wide band of pitted enamel is evident on the maxillary and mandibular permanent incisors and first permanent molars. The child was severely affected with pneumonia at 6 months of age. (Courtesy Dr. Stanley C. Herman.)

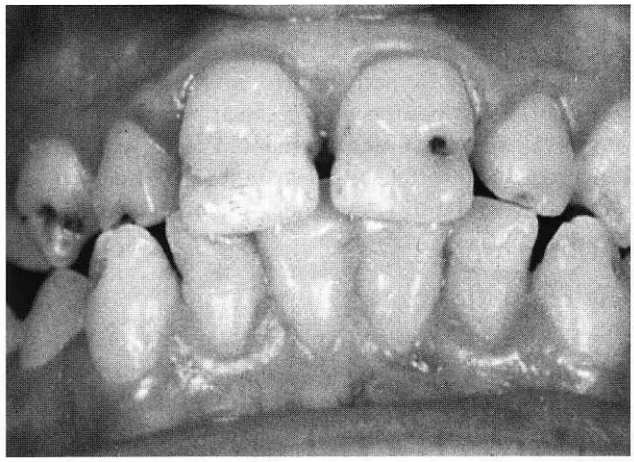


FIG. **7-16.** Enamel hypoplasia occurred during early childhood. Enamel formation on the incisal third of the lower incisors and the maxillary central incisors is normal.



E **I G 7-15.** Enamel hypoplasia that developed as the result of a nutritional deficiency during infancy. The first permanent molars, maxillary central incisors, and mandibular incisors show hypoplastic enamel and dentin.

Sarnat and Schour observed that, in a group of 60 children who had adequate medical histories, two thirds of the hypoplastic disturbances occurred during infancy (birth to the end of the first year)" (Fig. 7-15). Approximately one third of enamel hypoplasia was found in the portion of teeth formed during early childhood (13 to 34 months) (Fig. 7-16). Less than 2% of enamel defects found originated in late childhood (35 to 80 months).

Sheldon, Bibby, and Bales sought to determine whether defects in enamel were related to the occurrence of systemic ailments.' They examined ground sections of 95 teeth from 34 patients for whom detailed medical histories were available. In more than 70%0 of the individuals a positive correlation was established between the time of formation of a band of defective enamel and the existence of some systemic disability. However, defects in enamel occurred in 23% of patients who had no history of systemic conditions that might have produced enamel defects. No enamel changes occurred in 6% of patients who had histories of disabilities that had produced enamel changes in other patients. Deficiencies of vitamins A, C, and D, calcium, and phosphorus were the most common causes of defective enamel formation.

Purvis et al, in a study of 112 infants with neonatal tetany in Edinburgh, observed that 63 (56%) later showed severe enamel hypoplasia of the primary teeth.[0] Histologic examinations revealed a prolonged disturbance of enamel formation in the 3 months before birth. An inverse relationship was demonstrated between the mean daily hours of bright sunshine in each calendar month and the incidence of neonatal tetany 3 months later. This observation suggested that enamel hypoplasia and neonatal tetany can be manifestations of vitamin D deficiency during pregnancy and are most likely the result of secondary hyperparathyroidism in the mother. A significantly higher mean maternal age and a preponderance of lower social class was also seen in the mothers of those in the tetany group. Another study in Edinburgh indicated that only 1% of pregnant mothers took vitamin D supplements.

Apparently in some children a mild deficiency state or systemic condition without clinical symptoms can interfere with ameloblastic activity and can produce a permanent defect in the developing enamel.

HYPOPLASIA RELATED TO BRAIN INJURY AND NEUROLOGIC DEFECTS

Herman and McDonald studied 120 children with cerebral palsy between $2^1/_2$ and $10^1/_2$ years of age (for whom complete medical records were available) to determine the prevalence of dental hypoplasia."[1] The researchers compared them with 117 healthy children in the same age group and observed enamel hypoplasia in 36% of the group with cerebral palsy and in 6% of the group without the disorder. A definite relationship between the time of occurrence of the possible factors that could have caused brain damage and the apparent time of origination of the enamel defect (based on its location in the enamel on the crown of the tooth) was established for 70% of the affected teeth of children with cerebral palsy (see Fig. 7-12). Evidence of enamel hypoplasia is an aid to the clinician and the research worker in determining when brain injury occurred in patients in whom the cause is not clearly defined.

Cohen and Diner observed that enamel defects occurred with greatest frequency in children with low intelligence quotients and a high incidence of neurologic defects."- They found that chronologically distributed enamel defects were a valuable aid in neurologic diagnosis, because they occur commonly in brain-damaged children. In addition, the defects indicate the time of insult to the developing fetus or infant even when the history is reportedly negative. Martinez et al examined 170 children between 4 and 17 years of age (mean age, 12.03 years) with mental retardation and no history of dental trauma." They found that 37% of these children had dental enamel defects.

HYPOPLASIA ASSOCIATED WITH NEPHROTIC SYNDROME

Oliver and Owings observed enamel hypoplasia in permanent teeth in a high percentage of children with nephrotic syndrome and found a correlation between the time of severe renal disease and the estimated time at which the defective enamel formation occurred." Similarly, Koch et al found a high incidence of enamel defects in the primary teeth of children who were diagnosed with chronic renal failure early in infancy."

HYPOPLASIA ASSOCIATED WITH ALLERGIES

Rattner and Myers discovered a correlation between enamel defects of the primary dentition and the presence of severe allergic reactions." Enamel defects were present in 26 of 45 children with congenital allergies. The enamel lesions were localized in the occlusal third of the primary canines and first molars.

HYPOPLASIA ASSOCIATED WITH LEAD POISONING (PLUMBISM)

Lawson and Stout observed that in areas of Charleston, South Carolina, containing very old frame buildings the incidence of pitting hypoplasia was approximately 100% greater than published standards or the incidence in their control group of children." They suggest that dentists treating children with unexplained pitting hypoplasia should consider previous exposure to lead as a part of their health evaluation, particularly if the child is from a family in a low economic stratum.

Pearl and Roland have pointed out that the fetus of a lead-poisoned mother can be affected because lead readily crosses the placenta during pregnancy."[5] They observed significant delays in development and eruption of the primary teeth in the child of a lead-poisoned mother. They also listed pica (ingestion of unusual objects to satisfy an abnormal craving) as a common sign of plumbism in children 1 to 6 years old, as well as in their mothers. One mother admitted eating plaster from her apartment walls during several months of pregnancy.

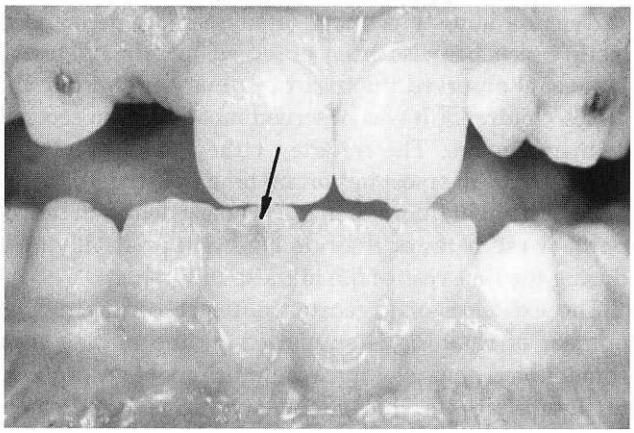
HYPOPLASIA CAUSED BY LOCAL INFECTION AND TRAUMA

Enamel hypoplasia resulting from a deficiency state or a systemic condition will be evident on all the teeth that were undergoing matrix formation and calcification at the time of the insult. The hypoplasia will follow a definite pattern. Individual permanent teeth often have hypoplastic or hypocalcified areas on the crown that result from infection or trauma (Figs. 7-17 and 7-18).

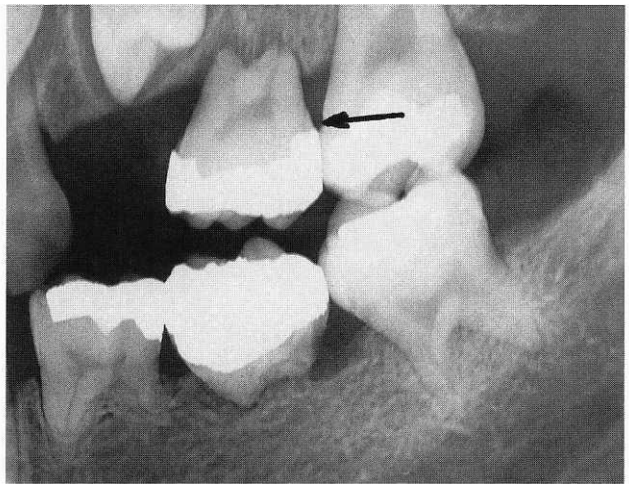
Turner first described this localized type of hypoplasia."[9] He noted defects in the enamel of two premolars and traced the defects to apical infection of the nearest primary molar. Enamel hypoplasia resulting from local infection is called *Turner tooth.*

Bauer concluded from a study of autopsy material that the periapical inflammatory processes of primary teeth extend toward the buds of the pertinent permanent teeth and affect them during their prefunctional stage of eruption.[50] The infection fails to stimulate the development of a fibrous wall that would localize the lesion. Instead, the infection spreads diffusely through the bone around the buds of the successors and thereby affects the important protective layer of the young enamel, the united enamel epithelium.

Bauer found that in some cases the united enamel epithelium was destroyed and the enamel was exposed to inflammatory edema and to granulation tissue.[5°] The granulation tissue later eroded the enamel and deposited a well-calcified, metaplastic, cementum-like substance on the surface of the deep excavation.



f1 6 **7-19.** Hypoplastic defect on the labial surface of a mandibular permanent central incisor *(arrow).* There was a history of trauma to the primary tooth.



F I G **7-20.** X-radiation caused hypoplastic defect on the crown of the first permanent molar *(arrow)* and stunting of root development.

that dental anomalies, including hypoplasia, probably result from multiple causes." They emphasize the difficulty in understanding the role played by genetic factors, postnatal environment, nutrition, and surgical influences in the development of the dental anomalies.

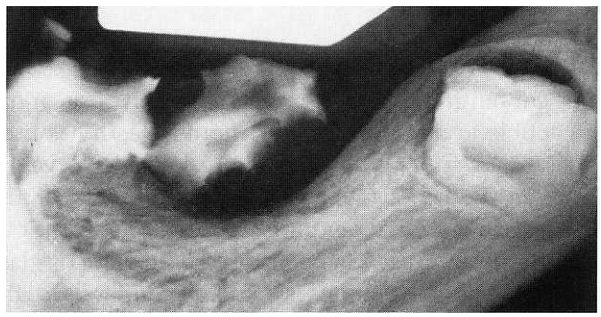
HYPOPLASIA CAUSED BY X-RADIATION AND CHEMOTHERAPY

Numerous dental abnormalities may result in surviving children who receive high-dose radiotherapy and chemotherapy during the time their teeth are forming. Kaste et al reviewed clinical and radiographic records of 423 survivors of acute lymphoblastic leukemia.[5)] Among these patients they observed root stunting in 24.4%, microdontia in 18.9%, hypodontia in 8.5%, taurodontia in 5.9%, and overretention of primary teeth in 4%. The patients who were younger than 8 years old at diagnosis or who received cranial irradiation (in addition to chemotherapy) developed more dental abnormalities than those older than 8 years at diagnosis and those who did not receive cranial irradiation. They also noted that the resulting dental defects may affect the survivors' quality of life.

Maguire and Welbury point out that, as survival rates for children with cancer improve, the emphasis in therapy has moved from saving children at all costs to saving children at the least cost to the child, and therapy protocols are continually reviewed with this goal in mind.[55]

Children who receive high-dose x-radiation in the treatment of a malignancy are at risk for developing rampant caries in the irradiated area. The cause is generally believed to be associated with changes in salivary gland function and is discussed in Chapter 10.

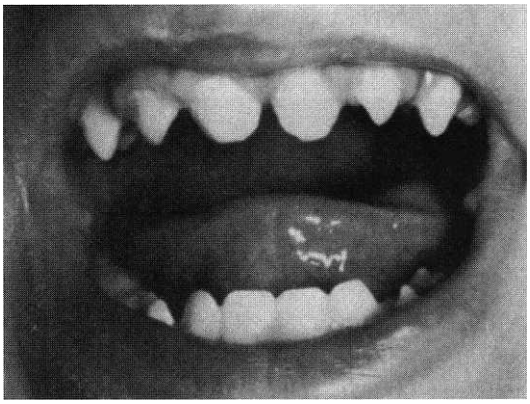
Ameloblasts are somewhat resistant to x-radiation. However, a line of hypoplastic enamel that corresponds to the stage of development at the time of therapy may be seen (Fig. 7-20). Radiotherapy will have a more severe effect on the development of the dentin, and root formation will be stunted. Occasionally, development of the permanent teeth will be arrested (Fig. 7-21).



H **G 7-21.** Absence of developing premolars and the malformed second permanent molar were caused by excessive x-radiation.

HYPOPLASIA RESULTING FROM RUBELLA EMBRYOPATHY

Musselman examined 50 children (average age, $2^{1}/_{2}$ years) with congenital anomalies attributed to in utero infection with rubella.[56] Enamel hypoplasia was found in 90% of the affected children, compared with only 13% of unaffected children in a control group. Tapered teeth also occurred in 78% of the children with a history of rubella (Fig. 7-22). Nine of the children in the study group had notched teeth, but this defect was not present in any of the children in the control group.



[I G. **7-22.** The mother of this child had rubella in the eighth week of pregnancy. The primary teeth were tapered and presented a rough hypoplastic surface. The child had a patent ductus arteriosus, pulmonary stenosis, and a cognitive disability. There was also a history of difficult feeding and dehydration at 2 months of age. *(Courtesy Dr. Robert Musselman.)*

TREATMENT OF HYPOPLASTIC TEETH

The contention that hypoplastic teeth are more susceptible to dental caries than normal teeth has little evidence to support it. Carious lesions do develop, however, in the enamel defects and in areas of the clinical crown where dentin is exposed. Small carious and precarious areas can be restored with resin or glass ionomer. The restoration is usually confined to the area of involvement. The occlusal third of the first permanent molar often shows gross evidence of hypoplasia, and treatment is necessary before the tooth fully erupts.

Hypoplastic primary and permanent teeth with large areas of defective enamel and exposed dentin may be sensitive as soon as they erupt. Satisfactory restoration may not be practical at this time. The topical application of fluoride has been found to decrease the sensitivity of the tooth. The application should be repeated as often as necessary to reduce sensitivity to thermal change and acid foods.

HYPOPLASIA CAUSED BY FLUORIDE (DENTAL FLUOROSIS)

Excess ingestion of fluoride can affect the ameloblasts during the tooth formation stage and can cause the clinical entity called *dental fluorosis* or *mottled enamel.* The appearance of enamel that is affected in its forma-tion by excessive fluoride varies considerably. Although the more severe cases of dental fluorosis are associated with a high level of fluoride consumption, there is apparently a great deal of individual variation. The affected enamel is often limited superficially to the most outer surface and presents a white or brown opaque and/or pitted appearance.

Dental fluorosis is most often seen in permanent teeth, but it has also been observed in primary teeth. Levy et al observed fluorosis of primary teeth in 12.1% of 504 children." It was observed most often on second primary molars. The middle of the first year of life seemed to be the most important time with regard to the development of fluorosis in the primary dentition based on their estimates of fluoride ingestion prenatally and during the first year of life in these children.

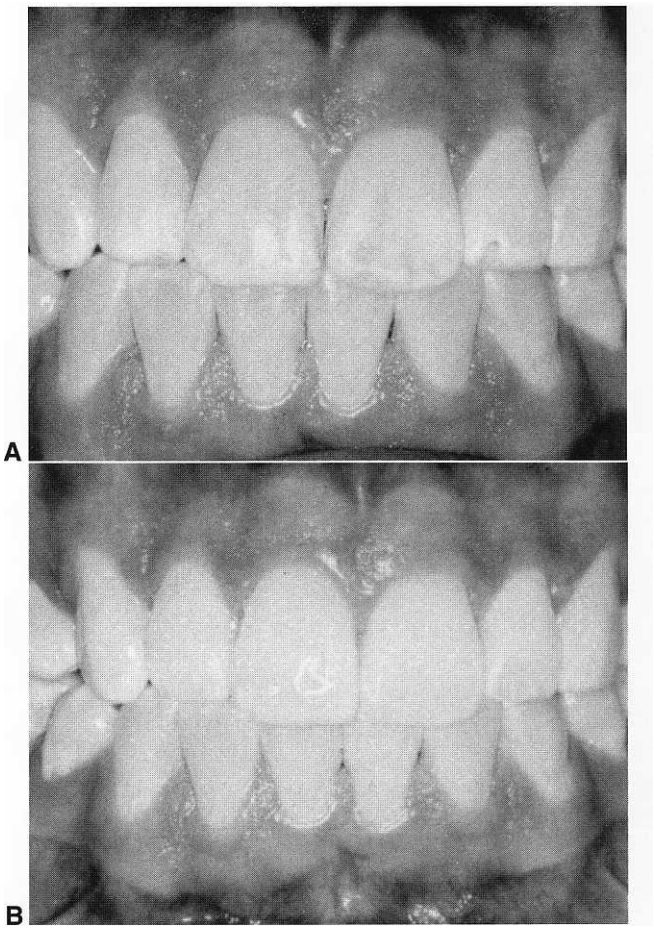
The existence of a genetic influence on the development of fluorosis is supported by the finding that some inbred strains of mice are much more susceptible to fluorosis than other strains who receive the same fluoride dosage under identical conditions. Studies of these differing mouse strains should identify candidate genes for study in human cases of dental and skeletal tluorosis.[5s]

ENAMEL MICROABRASION TO REMOVE SUPERFICIAL ENAMEL DISCOLORATIONS

For many years some dentists have advocated the application of hydrochloric acid as an effective method for destaining mottled enamel. McClosky described a technique, originally advocated by Kane, that used 18% hydrochloric acid on the affected enamel surfaces.[59] Croll and Cavanaugh advocated a modified procedure that they called *enamel color modification by controlled hydrochloric acid–pumice abrasion.*[60,6i] In their method, after the tooth or teeth are carefully isolated with a rubber dam and proper preparations have been made for safe use of the caustic agent, a slurry of fine pumice and 18% hydrochloric acid is applied under pressure and abrasion with a wooden stick. The slurry is rinsed away after each 5-second application until the desired color change has occurred. After a final rinsing with water, 1. 1% neutral sodium fluoride gel is applied for 3 minutes. Next a fine fluoridated prophylactic paste is applied with a rotating rubber cup. Finally the enamel is polished with an aluminum oxide disk.

Dalzell, Howes, and Hubler investigated the effects of the three variables of time, number of applications, and pressure on enamel loss when applying a slurry of 18% hydrochloric acid and pumice.[62] They found that enamel loss increased as the level of each variable increased and that a greater amount of enamel loss occurred when levels of two or three variables increased simultaneously. The combination of ten 10-second applications or fifteen 5-second applications with 20 g of pressure resulted in enamel loss of slightly less than 250 μm.

Croll proposed a modified procedure called *enamel microabrasion* in which a specially prepared abrasive



f **I G 7-23. A,** Mottled enamel. Because the brown pigmentation and white splotchy areas were objectionable, the teeth were treated by enamel microabrasion. **B,** Much of the pigment has been removed by the process.

compound (Prema*) is applied to the discolored enamel areas, similarly to prophylaxis paste, using a synthetic rubber applicator in a 10:1 gear-reduction handpiece.[68] Frequent water rinsings and reevaluation of the tooth for color correction are required. The instrumentation is continued until the undesirable coloration is removed or until a noticeable amount of enamel is being removed when the tooth is viewed from the incisal. Finally the abraded teeth are polished with a fine fluoridated prophylactic paste and given a 4-minute fluoride treatment (Fig. 7-23).

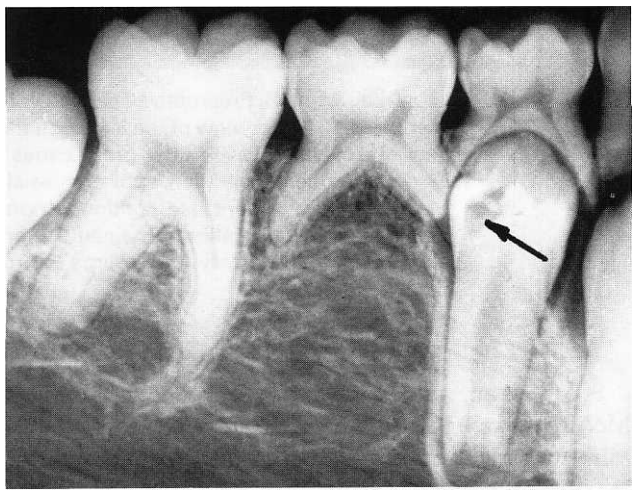
More recently Croll and Helpin have introduced a new delivery system for the microabrasion procedure.[6"] A viscous, water-soluble abrasion slurry containing hydrochloric acid and silicon carbide microparticles (Opalustre[t]) is conveniently applied to the tooth surface by a syringe. Vital tooth bleaching may be used in combination with enamel microabrasion to help remove deeper intrinsic discolorations. Bleaching is discussed later in this chapter.

*Premier Dental Products, King of Prussia, Pa. [t] Ultradent Products, South Jordan, Utah.

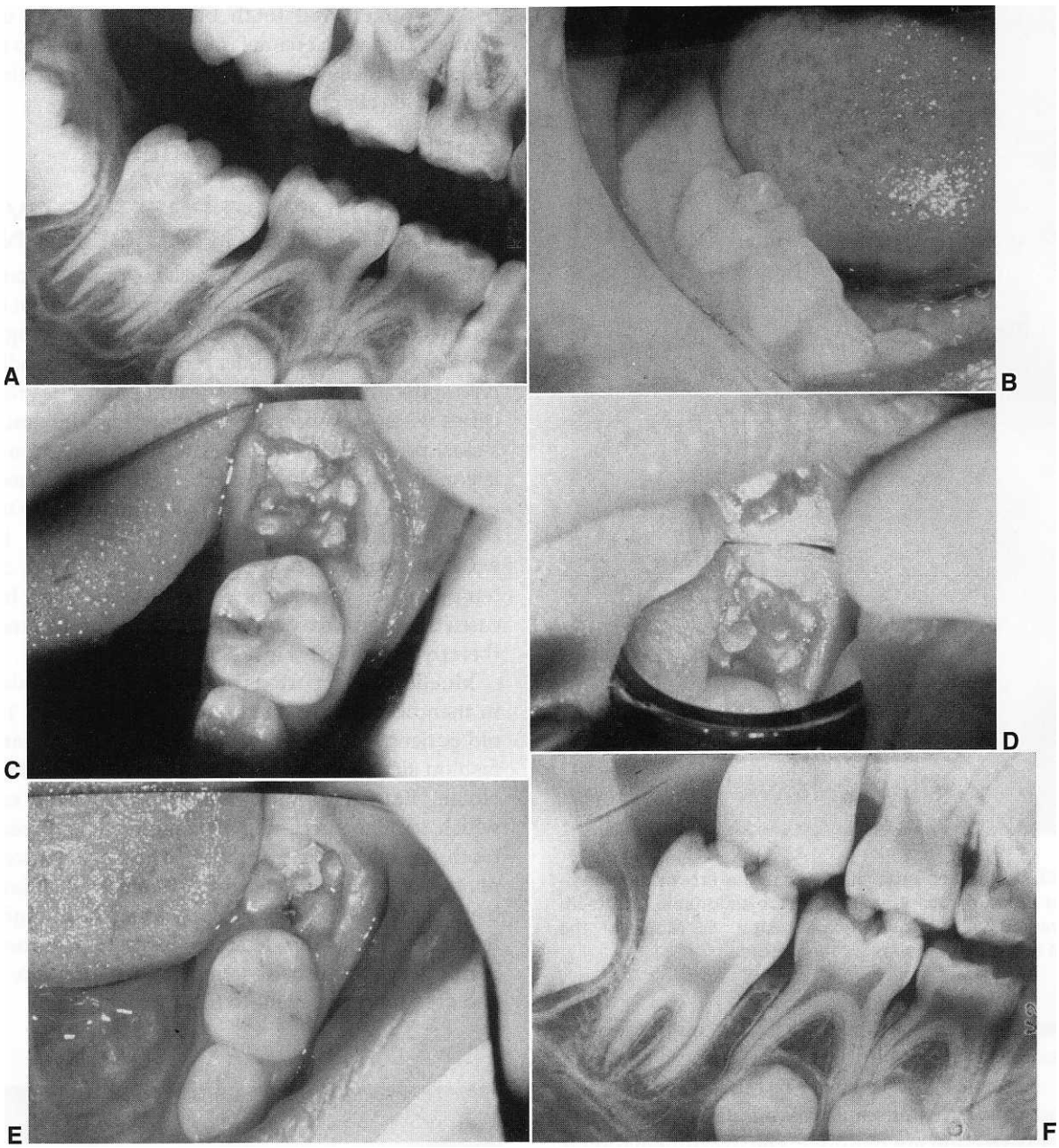
PREERUPTIVE "CARIES" (PREERUPTIVE CORONAL RESORPTION OR PREERUPTIVE INTRACORONAL RADIOLUCENCY)

Occasionally, defects on the crowns of developing permanent teeth are evident radiographically, even though no infection of the primary tooth or surrounding area is apparent (Fig. 7-24). Muhler referred to this condition as *preeruptive "caries."*[65] Such a lesion often does resemble caries when it is observed clinically, and the destructive lesion progresses if it is not restored. As soon as the lesion is reasonably accessible, the tooth should be uncovered by removal of the overlying primary tooth or by surgical exposure. The carieslike dentin is then excavated, and the tooth is restored with a durable temporary or permanent restorative material. In some cases the lesion may be so extensive that indirect pulp therapy is justified (Fig. 7-25).

Mueller et al reported carieslike resorption bilaterally in mandibular permanent second molars of a 12-year-old patient.[66] Both lesions were successfully treated in a fashion similar to the treatment illustrated in Fig. 7-25. Holan, Eidelman, and Mass reported three cases in which similar successful management of preemptive tooth defects was performed.[67] Their experience and a review of other reported cases suggest that impacted teeth or teeth delayed in eruption may be at higher risk for developing the lesions. Savage, Gentner, and Symons also published an informative literature review and case report relevant to this topic.[68]

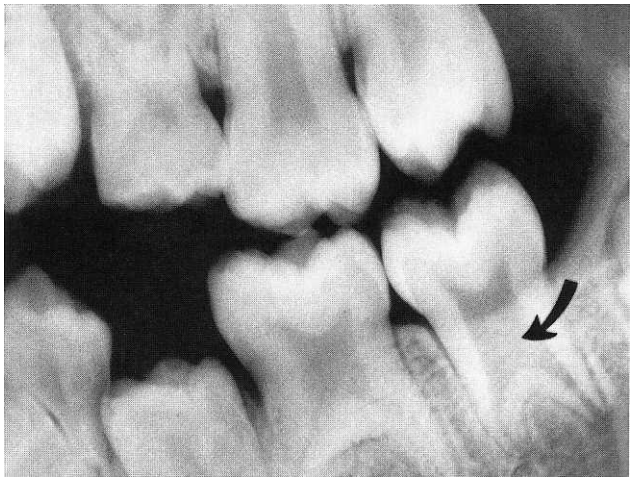


[I G **7-24.** Preeruptive "caries" on the crown of an unerupted first premolar (arrow).



f **I G 7-25. A** and **B,** Preeruptive "caries" in a mandibular right first permanent molar that is still unerupted. C, Mirror view of the lesion on the occlusal surface of the unerupted tooth. D, Mirror view of excavated cavity after gross caries removal. **E,** Mirror view of temporary restoration 1 week postoperatively (dark spot on mesial marginal ridge area is an artifact). F, Nine months postoperatively, patient had continued normal root development and eruption of the tooth. The temporary restoration remained 3 months before the tooth was reentered and restored with amalgam. *(Courtesy Drs. George E. Krull and James R. Roche.)*

Seow, Wan, and McAllan[b9] and Seow, Lu, and McAllan[70] have published retrospective studies to determine the prevalence of preemptive dentin radiolucencies in permanent teeth using bite-wing and panoramic radiographs, respectively. In the study using bite-wing radiographs, the authors examined a set of films from 1959 subjects and observed 9919 unerupted permanent teeth. They found 126 subjects (6%) with 163 teeth (2%) exhibiting preeruptive dentin radiolucencies. In the study using panoramic radiographs, the investigators examined 1281 films of individual patients with 11,767 unerupted permanent teeth (incisors excluded). This



f **I G 7-26.** Taurodontism. Notice the elongated pulp chamber and short root canals (arrow).

study identified 42 subjects (3%) with 57 teeth (0.5%) exhibiting preeruptive dentin defects. The authors emphasize the importance of carefully studying radiographic images of unerupted teeth so that early detection and treatment are possible.

TAURODONTISM

Lysell credits Keith with giving the name to the phenomenon known as *taurodontism."* This anomaly is characterized by a tendency for the body of the tooth to enlarge at the expense of the roots. The pulp chamber is elongated and extends deeply into the region of the roots (Fig. 7-26). A similar condition is seen in the teeth of cud-chewing animals such as the bull (Latin, *taurus).*

Jaspers and Witkop noted that taurodontism is found in about 2.5% of adult whites as an isolated trait, as well as in individuals with syndromes such as tricho-dento-osseous syndrome, otodental dysplasia, and X-chromosome aneuploidies.[72] Mena observed a mother and seven children, four of whom showed evidence of taurodontism in the permanent or primary teeth, or both." This was probably the first report of taurodontism of the primary dentition as a definite family trait in black children. Gedik and cimen reported taurodontism of six primary molars of a 7-year-old boy who had no syndromes or systemic disease." Other pedigrees have been consistent with autosomal dominant or autosomal recessive inheritance. The inheritance may also be polygenic.

The clinical significance of the condition becomes apparent if vital pulp therapy or root canal therapy is necessary.

INHERITED DENTIN DEFECTS

Two broad categories of heritable dentin defects, dentinogenesis imperfecta and dentin dysplasia, are identifiable, each with distinct subtypes.

DENTINOGENESIS IMPERFECTA (HEREDITARY OPALESCENT DENTIN)

Dentinogenesis imperfecta is inherited as an isolated autosomal dominant trait ("isolated" in this usage means that it occurs without other anomalies). Bixler, Conneally, and Christen observed this pattern in a six-generation family in which 34 members were studied.[75] There was 100% penetrance and consistent gene expression within a sibship. In a survey of 96,000 Michigan children, Witkop reported a prevalence of 1 in 8000 with the trait.[7h] The anomaly may be seen with osteogenesis imperfecta (Fig. 7-27). A decade later, Witkop suggested that there are two distinct diseases.[77] He recommended that the term *hereditary opalescent dentin* be used for the disease that occurs as an isolated trait and the term *dentinogenesis imperfecta* be used for that which occurs in conjunction with osteogenesis imperfecta. Shields, Bixler, and El-Kafrawy recognized the difference and proposed a new classification: the dentin defect that occurs in association with osteogenesis imperfecta is termed *(Shields) type I dentinogenesis imperfecta* and that which occurs as an isolated trait is termed *(Shields) type II dentinogenesis imperfecta.*[78] In addition, the dentin defects seen in the isolated Brandywine triracial population in southern Maryland was termed *(Shields) type III dentinogenesis imperfecta.* These latter defects consisted of variable expression of the features of (Shields) type I (without osteogenesis imperfecta) and type II, shell-like teeth, and multiple pulp exposures (Fig. 7-28). In this condition normal dentin formation is confined to a thin layer next to the enamel and cementum, followed by a layer of disorderly dentin containing a few tubules. The roots of shell teeth are short, and the primary teeth may be exfoliated prematurely.

Mutations in the *DSPP* gene, which codes for the two major noncollagenous dentin matrix proteins, dentin sialoprotein (DSP) and dentin phosphoprotein (also known as phosphophorin), have been found in patients with (Shields) type II dentinogenesis imperfecta by Zhang et al[l'] and by Xiao et al.[30] Sreenath et al noted in the *DSPP* knockout mouse that the teeth have a widened predentin zone and develop defective dentin mineralization similar in phenotype to that in human (Shields) type III dentinogenesis imperfecta; these findings imply that this gene may also be involved in the latter condition.[81]

The clinical picture of dentinogenesis imperfecta is one in which the primary and permanent teeth are a characteristic reddish brown to gray opalescent color. Soon after the primary dentition is complete, enamel often breaks away from the incisal edge of the anterior teeth and the occlusal surface of the posterior teeth. The exposed soft dentin abrades rapidly, occasionally to the

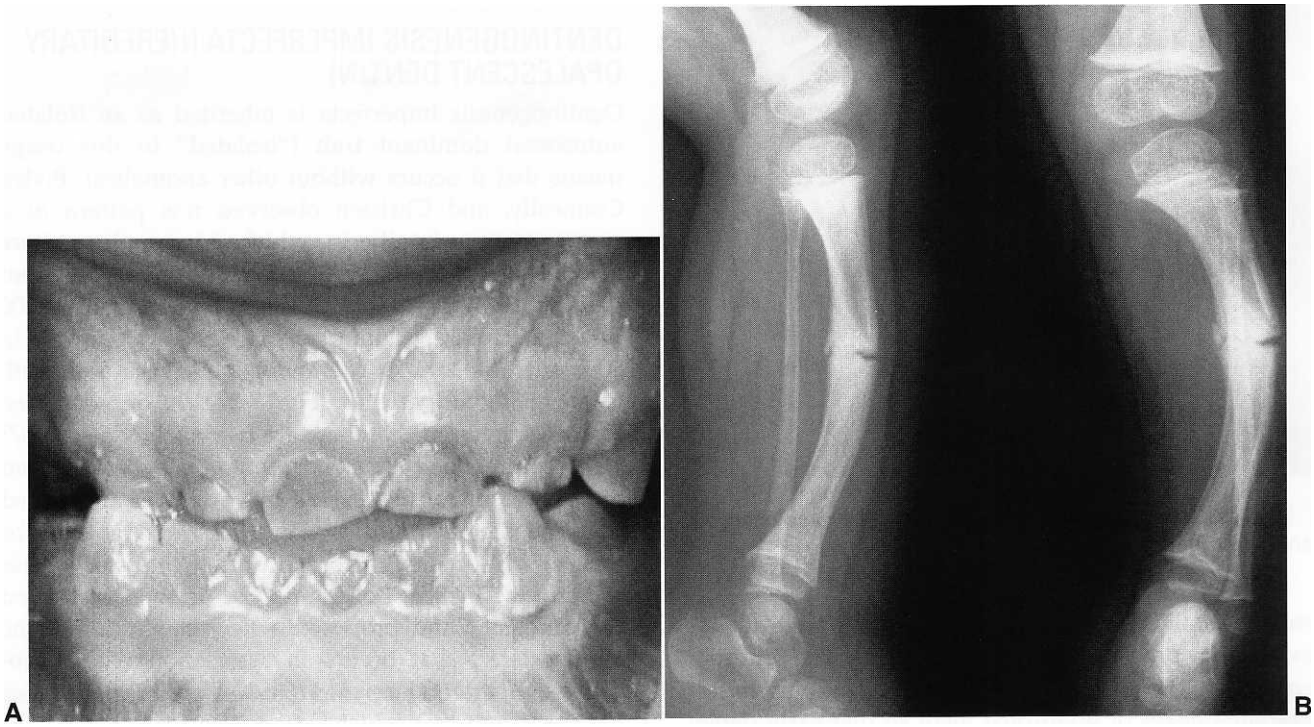


FIG. 7-27. **A,** Five-year-old girl with dentinogenesis imperfecta and osteogenesis imperfecta. The child had sustained numerous fractures of the long bones. **B,** A fracture of the tibia is evident in the radiograph.

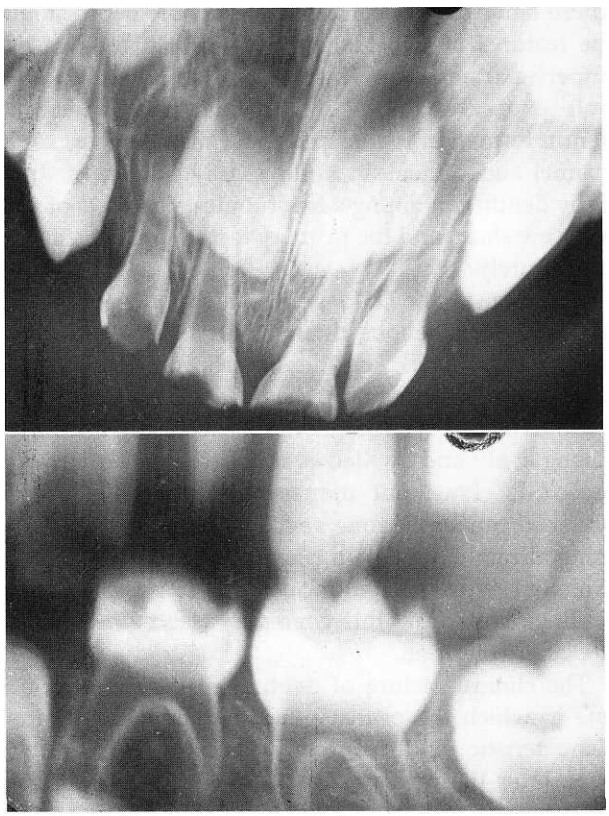
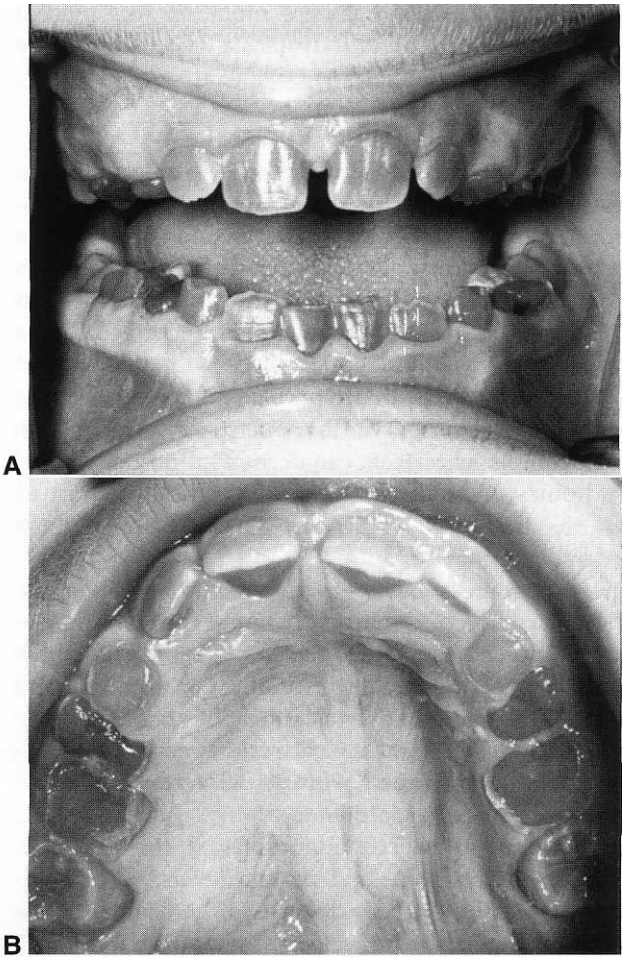


FIG 7-28. Shell teeth. The large size of the pulp cavities indicates the nonexistence of secondary dentin.

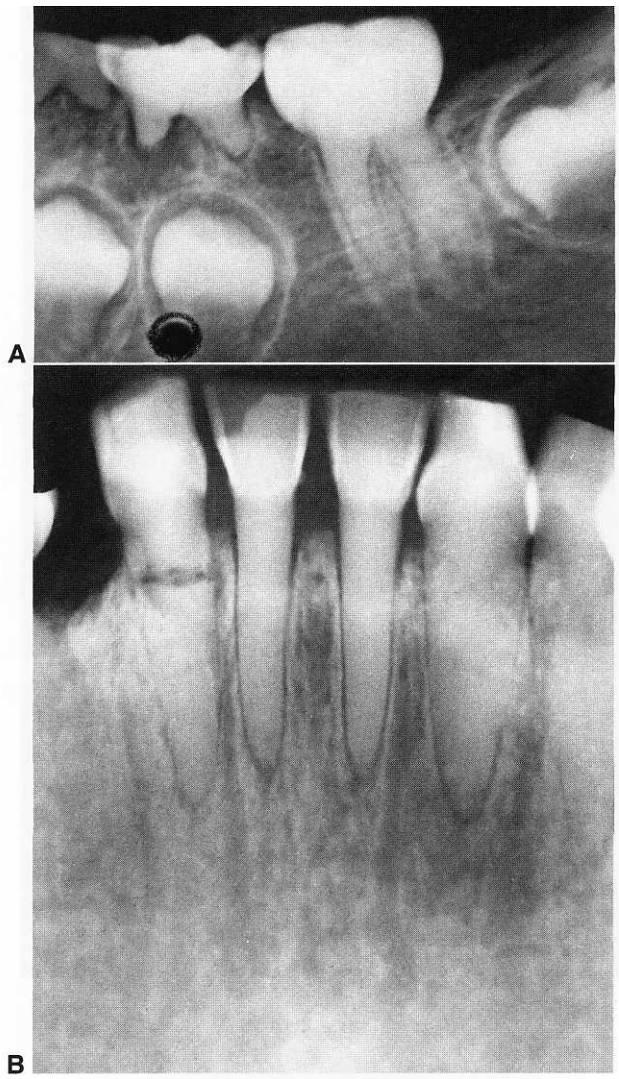
extent that the smooth, polished dentin surface is continuous with the gingival tissue (Fig. 7-29). Radiographs show slender roots and bulbous crowns. The pulp chamber is small or entirely absent, and the pulp canals are small and ribbonlike (Fig. 7-30). Periapical rarefaction in the primary dentition is occasionally observed. However, no satisfactory explanation has been offered, because the condition apparently is not related to pulp exposures and pulpal necrosis. Multiple root fractures are often seen, particularly in older patients. Crowns of the permanent teeth often seem to be of better quality and have less destruction. Occasionally they appear essentially normal clinically (Fig. 7-31).

The treatment of dentinogenesis imperfecta is difficult in both the primary and permanent dentitions. The placement of stainless steel crowns on the primary posterior teeth may be considered as a means of preventing gross abrasion of the tooth structure. Full-coverage restorations may be placed on the permanent teeth if the crowns need protection in late adolescence or young adulthood. Bonded veneer restorations on anterior teeth have also been used successfully for esthetic improvement in patients with dentinogenesis imperfecta when full-coverage restorations are unnecessary.

Teeth that have periapical rarefaction and root fracture should be removed. Extraction of the affected teeth is difficult because of the brittleness of the dentin.



[I G **7-29.** Dentinogenesis imperfecta. The primary teeth are severely abraded. Enamel is breaking away from the incisal edge of the lower permanent central incisors.



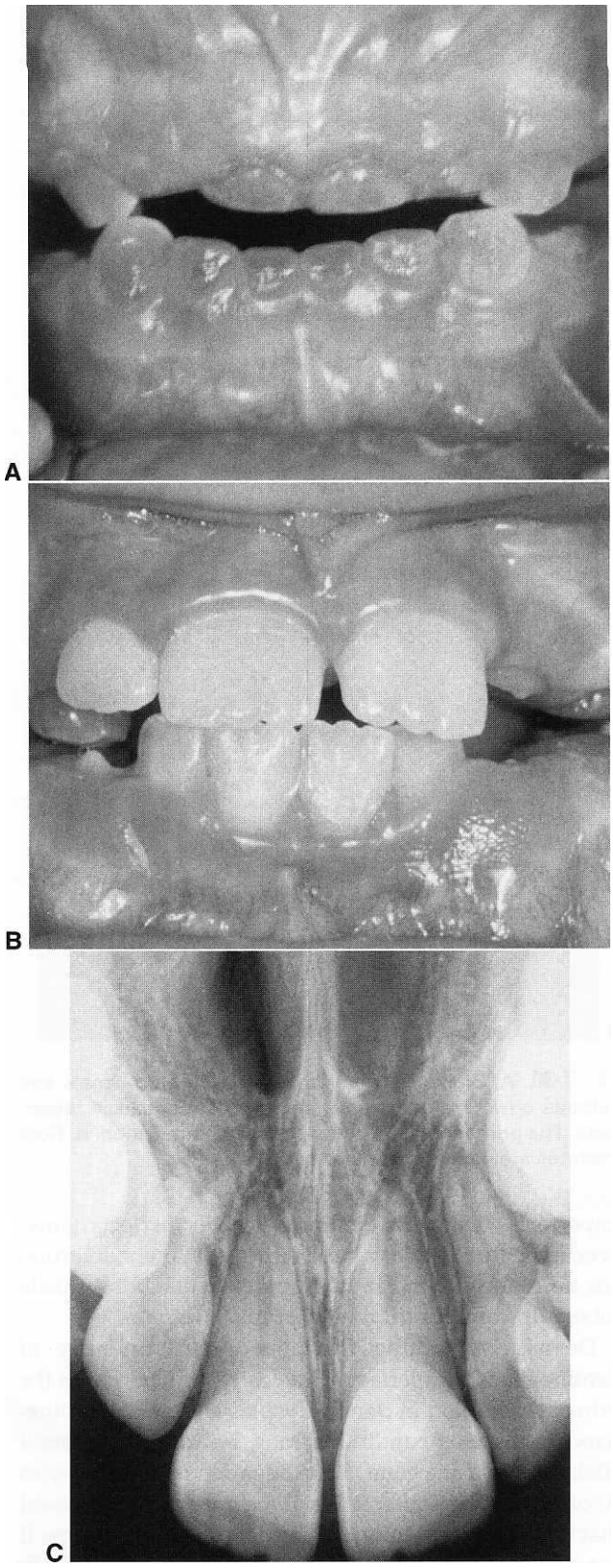
fl G **7-30. A,** Slender roots with ribbonlike pulp canals and bulbous crowns are characteristic of dentinogenesis imperfecta. The primary molars show periapical rarefaction. **B,** Root fractures are common in older patients.

DENTIN DYSPLASIA

Dentin dysplasia is a rare disturbance of dentin formation that Shields, Bixler, and El-Kafrawy have categorized into two types: radicular dentin dysplasia (type I) and coronal dentin dysplasia (type II).[s] Both primary and secondary dentitions are affected in dentin dysplasia type I, which is inherited as an autosomal dominant trait. Radiographically the roots are short and may be more pointed than normal. Usually the root canals and pulp chambers are absent except for a chevron-shaped remnant in the crown. The color and general morphology of the crowns of the teeth are usually normal, although they may be slightly opalescent and blue or brown. Periapical radiolucencies may be present at the apices of affected teeth.

Dentin dysplasia type II is inherited as an autosomal dominant trait in which the primary dentition appears opalescent and on radiographs has obliterated pulp chambers, similar to the appearance in dentinogenesis imperfecta. Unlike in dentinogenesis imperfecta, however, in dentin dysplasia type II the permanent dentition has normal color and radiographically exhibits a thistle tube pulp configuration with pulp stones.

Dean et al, noting the phenotypic similarity of dentinogenesis imperfecta, Shields type II, to that in the primary dentition in dentin dysplasia type II, hypothesized that these conditions may be due to different alleles of the same gene.[s'] Investigation of a family with 10 of 24 members affected in three generations showed that the candidate region for the dentin dysplasia type II gene overlaps the likely location of the gene for Shields type II dentinogenesis imperfecta. They suggested that a candidate gene for dentinogenesis imperfecta Shields type II and/or III should also be a candidate gene for dentin dysplasia type II. Subsequently Rajpar et al



f l **G 7-31. A,** Four-year-old child with dentinogenesis imperfecta. **B,** The permanent teeth, in contrast to the primary teeth, are normal in color. C, The radiograph shows the typical picture of dentinogenesis imperfecta.

showed that a DSPP missense mutation was present in a family with dentin dysplasia type II, thereby confirming the hypothesis of Dean et al [8,83]

AMELOGENESIS IMPERFECTA

As noted in Chapter 6, amelogenesis imperfecta is a developmental defect of the enamel with a heterogeneous etiology that affects the enamel of both the primary and permanent dentition. The anomaly occurs in the general population with an incidence of 1 in 14, 000 to 1 in 16,000. Amelogenesis imperfecta has a wide range of clinical appearances with three broad categories observed: the hypocalcified type, the hypomaturation type, and the hypoplastic type. Although amelogenesis imperfecta can occur as a part of several syndromes, Cartwright, Kula, and Wright confirmed that the trait itself could also be associated with a skeletal anterior open bite.[}]

Some progress has been made in unraveling the molecular basis of the myriad clinical forms of amelogenesis imperfecta. Aldred and Crawford discussed the limitations of the existing classification systems and proposed an alternative classification based on the molecular defect, biochemical result, mode of inheritance, and phenotype.' Hart et al recommended a standardized nomenclature for describing amelogenesis imperfecta that causes alterations at the genomic, complementary DNA, and protein levels."" In addition, two clinically distinct forms of autosomal dominant amelogenesis imperfecta—smooth hypoplastic amelogenesis imperfecta and local hypoplastic amelogenesis imperfecta—are associated with mutations in the enamelin *(ENAM)* gene located at 4q21. An X-linked form (AIH1) has been found to be associated with as many as 12 mutations in the amelogenin *(AMELX)* gene, located at Xp21. However, at least one family has had the trait linked to another location on the chromosome, Xg22-g28.[8]

The defective tooth structure is limited to the enamel. On radiographic examination the pulpal outline appears to be normal, and the root morphology is not unlike that of normal teeth. The difference in the appearance and quality of the enamel is thought to be attributable to the state of enamel development at the time the defect occurs. In the hypoplastic type the enamel matrix appears to be imperfectly formed; although calcification subsequently occurs in the matrix and the enamel is hard, it is defective in amount and has a roughened, pitted surface (Fig. 7-32). In the hypocalcified type, matrix formation appears to be of normal thickness, but calcification is deficient and the enamel is soft (Fig. 7-33). In both of these more common types of the defect, the enamel becomes stained because of the roughness of the surface and the increased permeability.

In still another variant of amelogenesis imperfecta a thin, smooth covering of brownish yellow enamel is



FIG 7-32. Both the primary and permanent teeth are affected by the hereditary anomaly amelogenesis imperfecta. The enamel is pitted but hard.

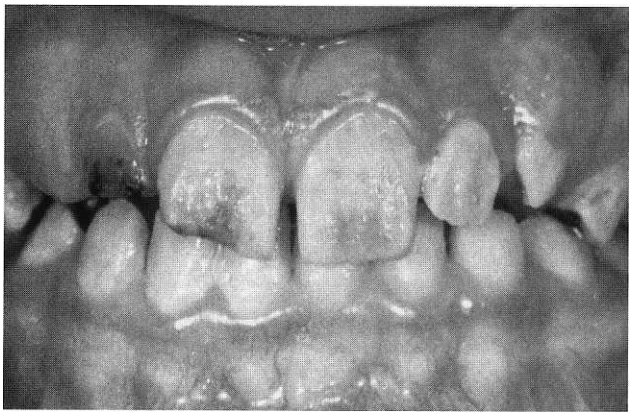


FIG 7-33. Hypocalcification type of amelogenesis imperfecta. The primary teeth were similarly affected. The enamel surface is soft.

present. In this type the enamel does not seem excessively susceptible to abrasion or caries (Figs. 7-34 and 7-35).

Congleton and Burkes reported three cases of amelogenesis imperfecta in which the patients also demonstrated taurodontism.[89] Others have identified cases with the clinical appearance of amelogenesis imperfecta and taurodontism along with strikingly curly hair and increased bone density (especially of the skull), and this has been identified as *tricho-dento-osseous syndrome.* Seow has suggested that some cases reported as amelogenesis imperfecta with taurodontism were actually cases of tricho-dento-osseous syndrome.[90] Price et al found that this autosomal dominant condition is caused by a mutation in the distal-less homeobox gene DLX3.[9'] Even though the taurodontism and amelogenesis imperfecta traits in this condition are fully penetrant in affected individuals, the osseous and hair features are variably expressed even when the same deletion is present in a family—findings which indicate that the variable expression is influenced by other genes and/or environmental factors. Further studies by Price et al have confirmed that tricho-dento-osseous syndrome and amelogenesis imperfecta of the hypomaturation-hypoplastic type with taurodontism are two genetically distinct conditions.[9"]

The treatment of teeth with amelogenesis imperfecta–like defects depends on its severity and the demands of aesthetic improvement. When indicated, the teeth can be prepared for full-coverage restorations. For some cases of the hypomaturation and hypoplastic types, bonded veneer restorations may offer a more conservative alternative for management of the aesthetic problem of the anterior teeth. Patel et al have reported successful treatment with porcelain laminate veneer restorations.[9"]

ENAMEL AND DENTIN APLASIA

Teeth that have some characteristics of both dentinogenesis imperfecta and amelogenesis imperfecta have been described in the literature. Chaudhry et al reported such a case and called the condition *odontogenesis imrrperfecta.'*

Schimmelpfennig and McDonald observed a similar dentition and termed it *enamel and dentin aplasia.*[95] The primary teeth were essentially devoid of enamel, and the smooth, severely abraded dentin was reddish brown. Radiographs showed normal alveolar bone around the roots of the teeth. Two teeth had pulp expo-sure and pulpal degeneration (Fig. 7-36). Radiolucent areas were present at the apices of the two primary teeth, with exposed and degenerated pulps. The pulp chambers and canals in all the primary teeth were extremely large, with no evidence that they were becoming obliterated. In ground sections of the primary teeth the dentinal tubules showed little evidence of a normal growth pattern. They were few and irregular, with a tendency toward branching. The cementum appeared normal and was acellular. No evidence of secondary dentin formation was found. A few fragments of enamel adhering to the dentin appeared thinner than normal, and few normal morphologic characteristics were present. The dentinoenamel junction was atypical in that it lacked the characteristic scalloping.

The permanent teeth, when they erupted, were partially covered with a thin, gray, poorly coalesced coating of enamel. Brown dentin could be seen on the labial aspect of the central incisors and at the base of the fissures of the first permanent molars. Stainless steel crown restorations were placed even before complete eruption to protect the teeth from continued abrasion.

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FIG 7-34. A, Case diagnosed as amelogenesis imperfecta. The permanent teeth have a thin covering of pigmented enamel. **B,** The radiographs show essentially normal root morphology. The crowns have a thin covering of enamel.

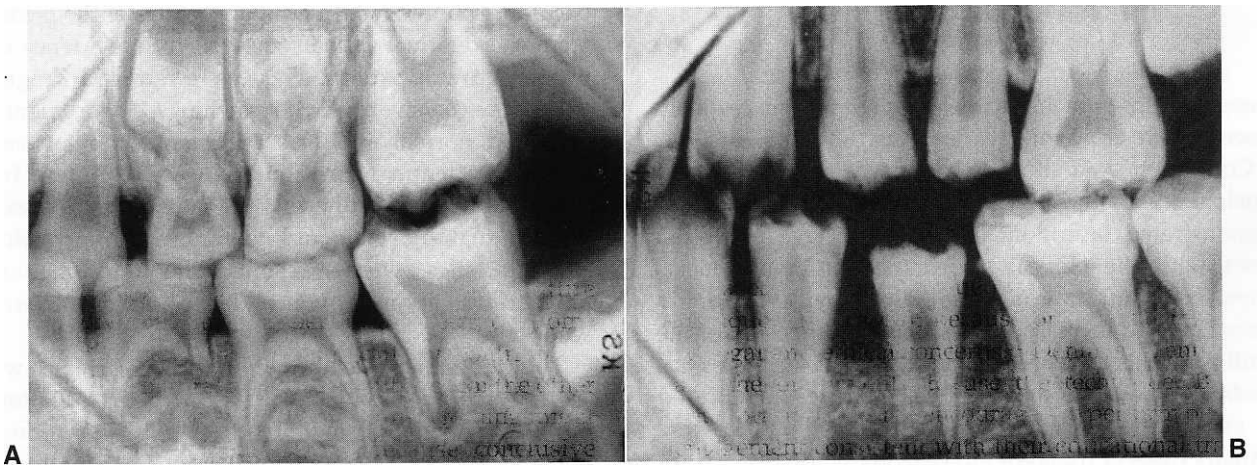
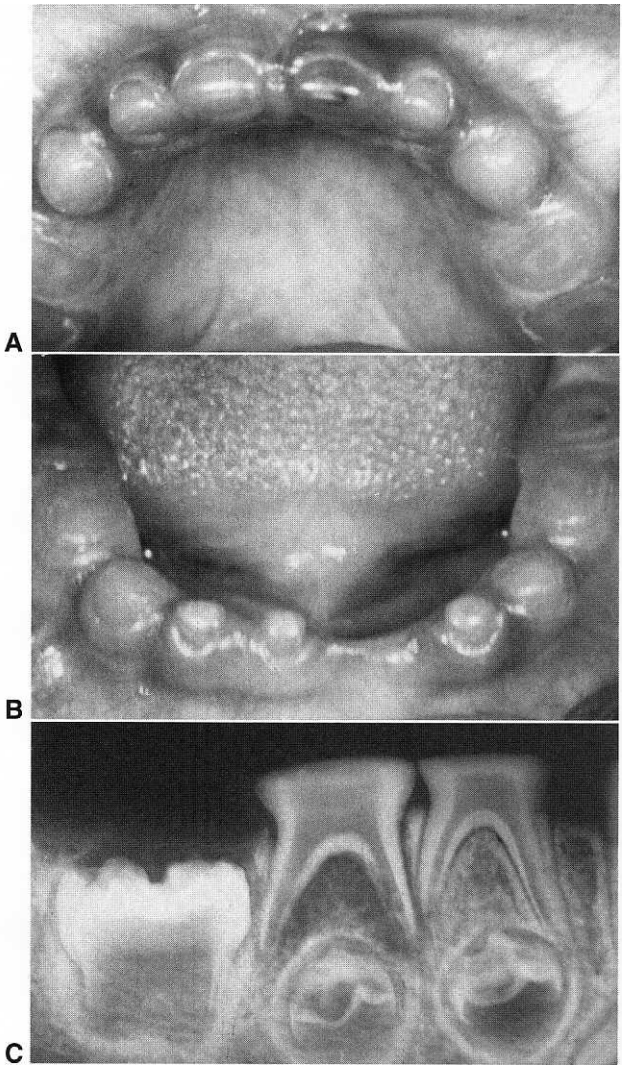


FIG 7-35. A and **B,** Left bite-wing radiographs of a patient with amelogenesis imperfecta. Radiograph in **B** was made 6 years after radiograph in **A** and demonstrates the maintenance of a caries-free dentition despite the thin enamel.



f G 7-36. A and **B,** Severely abraded teeth are almost entirely devoid of enamel. The outline of a large pulp chamber can be seen through a thin covering of dentin. The mandibular second primary molars have pulp exposure. C, Radiograph shows large pulp canals and large pulp chambers. Apical rarefaction is associated with pulp exposure of the second primary molar.

AGENESIS OF TEETH

ANODONTIA

Anodontia, which implies complete failure of the teeth to develop, is a rare condition. Although agenesis of permanent teeth is often referred to as *congenital absence*, they would not, of course, be expected in the oral cavity at birth anyway. Gorlin, Herman, and Moss noted that, when agenesis occurs as an isolated trait, the primary dentition is not affected, and the inheritance is autosomal recessive.[96] Swallow reported on an 11-year-old boy who had a complete primary dentition but no permanent dentition.[97] Schneider also observed a 7-year-old white female with primary teeth but missing permanent teeth.[9s] As is usually the case with presumed autosomal recessive inheritance, the hereditary background included no known consanguinity in the family or history of anodontia or ectodermal dysplasia in either the maternal or paternal lineages. Although consanguinity increases the likelihood that recessive traits or conditions will be expressed, most affected individuals do not have a family history of inbreeding. An overlay denture was constructed for the patient, which is often the treatment of choice. A comparable situation is shown in Fig. 7-37. Laird reported a similar patient in whom a complete primary dentition was present, but the only permanent teeth were maxillary first permanent molars.[99] Witkop studied two families in which both parents had peg or missing maxillary lateral incisors, which is an autosomal dominant trait with incomplete penetrance and variable expressivity, and concluded that agenesis of the permanent teeth can be an expression of the homozygous state of that gene.'°°

HYPODONTIA (OLIGODONTIA)

Agenesis of some teeth is referred to as *hypodontia,* which is preferable to the term *partial anodontia. Oligodontia is* sometimes used when only a few teeth develop. Hypodontia may occur without a family history of hypodontia, although it is often familial. It may also be found as a part of a syndrome, especially in an ectodermal dysplasia, although it usually occurs alone (isolated). Note that *isolated* in this usage means not occurring as part of a syndrome. It may still be familial.

Any one of the 32 permanent teeth may be missing. However, those most frequently missing in children are the mandibular second premolars, maxillary lateral incisors, and maxillary second premolars. This order of frequency has been confirmed in studies by Glenn[101] and by Grahnen.[1Q2] The absence of teeth may be unilateral or bilateral. Glenn observed during an examination of 1702 children that 5% had a missing permanent tooth other than a third molar.[101] In 97% of the children the formation of the second premolar could be detected radiographically at 5.5 years of age and that of the lateral incisor at 3.5 years of age.

In addition to the already mentioned autosomal dominant hypoplasia or agenesis of the maxillary lateral incisors, Vastardis et al found absence of second premolars in one family showing an autosomal dominant pattern of inheritance to be caused by a mutation in the MSXI gene located at 4pl6.1.[103] In contrast, evidence for the genetic heterogeneity of hypodontia is provided by the finding of a frameshift mutation (resulting in premature termination of translation and a shortened protein) in the human PAX9 gene in a large



f **G 7-37.** A complete primary dentition without evidence of permanent teeth in a 14-yearold girl.

family with autosomal dominant oligodontia.[m''] X-linked inheritance of oligodontia or hypodontia through four generations of a family was described by Erpenstein and Pfeiffer.[1''] Not only was there no maleto-male transmission, but males had oligodontia while females had hypodontia. Ahmad et al demonstrated that hypodontia inherited in an autosomal recessive mode and associated with malformation of teeth, enamel hypoplasia, and failure of eruption could be linked to 16g12.1.[10h]

Bennett and Ronk reported an unusual case of a 4-year-old boy with three missing primary teeth.[10i] The

mother related that the two missing mandibular central incisors erupted a few days after birth (neonatal teeth) and were removed because of a lack of root formation. The maxillary left first primary molar was congenitally missing as well. A panoramic radiograph of the patient also revealed the absence of multiple permanent tooth buds. The child did not exhibit any other signs or symptoms; he appeared normal in every other way, and there was no known familial history of missing teeth. A rare finding of nine congenitally missing primary teeth in a 3-year-old boy has been reported by Shashikiran, Karthik, and Subbareddy.[103] The boy's mother gave a history of consanguineous marriage. There was no known family history of missing teeth even among the patient's siblings. The primary teeth that were present appeared normal clinically and radiographically. The boy also exhibited ankyloglossia, and the frenum attachments were high in both arches. He did not show any evidence of other ectodermal disorders and otherwise appeared to be a normal young child.

Fleming, Nelson, and Gorlin have reported that a missing maxillary central incisor may occur as an isolated dental finding.[109] This anomaly has also been reported to occur in association with growth retardation with or without growth hormone deficiency, in association with autosomal dominant holoprosencephaly, and occasionally in association with other midline developmental defects. As already stated in the discussion of fusion of teeth, children with this dental anomaly should be referred for medical follow-up unless a comprehensive evaluation for a diagnosis has already been performed. Growth hormone therapy or some other form of treatment may be indicated.

ECTODERMAL DYSPLASIAS

The congenital absence of primary teeth is relatively rare. When a number of the primary teeth fail to develop, other ectodermal deficiencies are usually evident. There are more than 100 types of ectodermal dysplasia with varying anomalies of ectodermal derivatives, including both the primary and permanent teeth, hair, nails, and skin. Children with a number of missing primary and permanent teeth may have some or all of the signs of a type of ectodermal dysplasia and should undergo further evaluation.

One of the more common types of ectodermal dysplasia is X-linked recessive hypohidrotic ectodermal dysplasia (XLHED), also called *anhidrotic ectodermal dysplasia* and *Christ-Siemens-Touraine syndrome*. A rare autosomal recessive form also occurs that is clinically indistinguishable from XLHED. Hypodontia and dental hypoplasia, as well as hypotrichosis, hypohydrosis/anhidrosis, and asteatosis are characteristic of XLHED. Secondary characteristics include a deficiency in salivary flow, protuberant lips, and a saddle-nose appearance. The skin is often dry and scaly, and there is fissuring at the corners of the mouth. Mutations in the *EDA1* gene have been found in most patients with XLHED.[10]

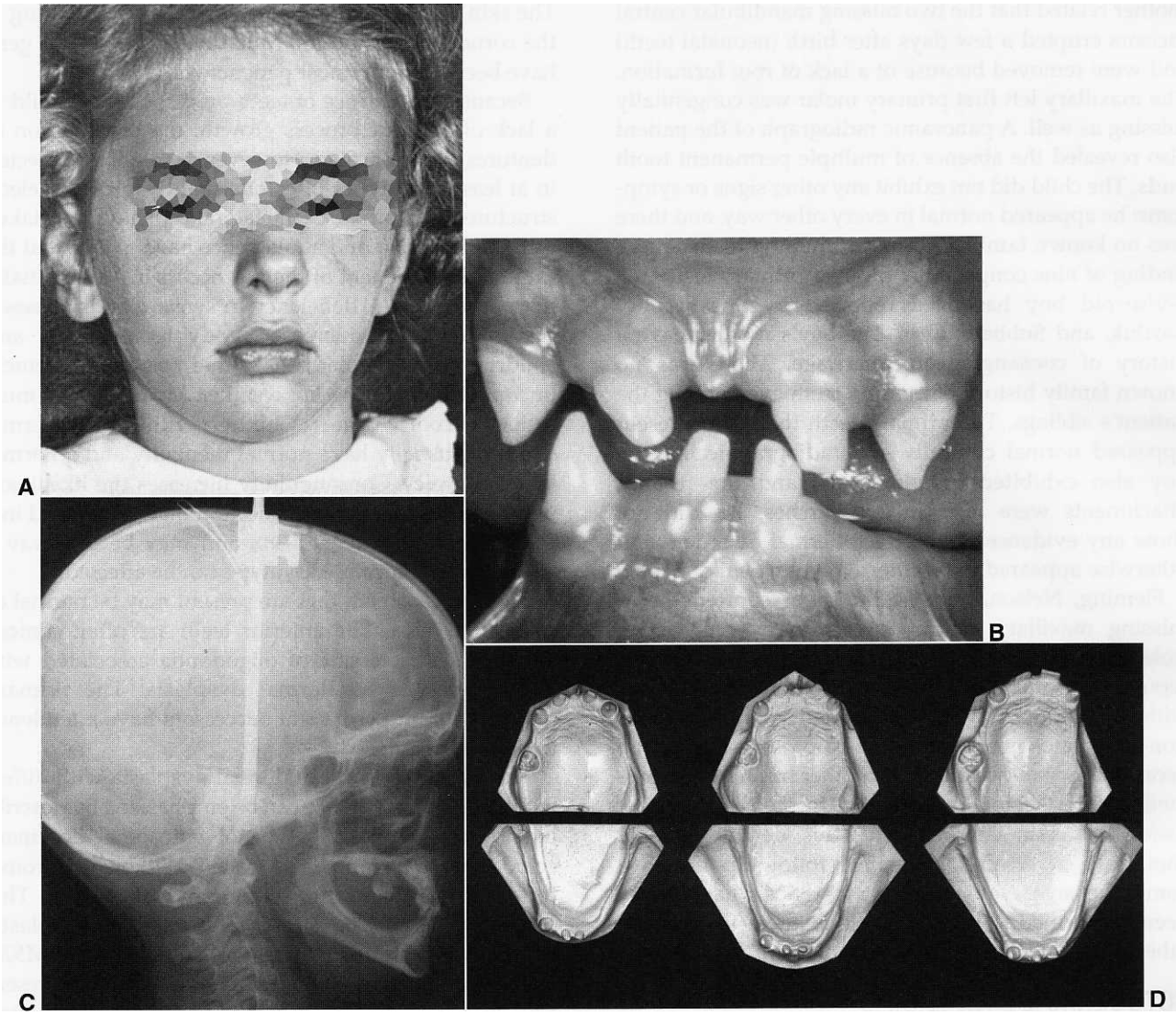
Because the absence of teeth predisposes the child to a lack of alveolar process growth, the construction of dentures is complicated. However, as might be expected in at least most types of ectodermal dysplasia, skeletal structures are normal. Serial lateral cephalograms taken during childhood and adolescence have shown that the sagittal development of the jaw occurs in an essentially normal manner. A deficiency in sweat glands causes a predisposition to increased body temperature, and children with hypohidrosis/anhidrosis are extremely uncomfortable during hot weather. Many of them must reside in cool climates. Children with an ectodermal dysplasia usually have normal mentality and a normal life expectancy. Consanguinity increases the likelihood of expression of a trait or condition that is inherited in a recessive manner (Fig. 7-38), and may be one way a female with a normal karyotype can be affected.

The primary teeth that are present may be normal or reduced in size. The anterior teeth are often conical, which is characteristic of oligodontia associated with many types of ectodermal dysplasia. The primary molars without permanent successors have a tendency to become ankylosed.

The many types of ectodermal dysplasia with different modes of inheritance can be emphasized by describing a "tooth and nail" type of autosomal dominant ectodermal dysplasia, also called Witkop syndrome, reported by Giansanti, Long, and Rankin.'" This ectodermal dysplasia is characterized by hypoplastic nails and hypodontia. A different mutation in the *MSX1* gene than that found when only hypodontia is present has been reported in a family with tooth and nail syndrome by Jumlongras et al."' In contrast to most nonsyndromic hypodontias, in which (excluding third molars) premolars and maxillary lateral incisors are most often missing, the mandibular incisors, second molars, and maxillary canines are most frequently absent. Overall, the teeth are generally not affected to the extent seen in XLHED, and there can be little involvement of the hair and sweat glands.[13]

For children with a large number of missing primary teeth, partial dentures can be constructed at an early age. Two-year-old and 3-year-old children have worn partial dentures successfully. Their ability to masticate food increases, and their nutritional status may improve. A partial denture can be adjusted or remade at intervals to allow for the eruption of permanent teeth. Denture construction at an early age may also reduce the psychologic problem of the child's feeling "different" (Fig. 7-39).

If the permanent teeth erupt in good position and in favorable relationship to each other, partial dentures

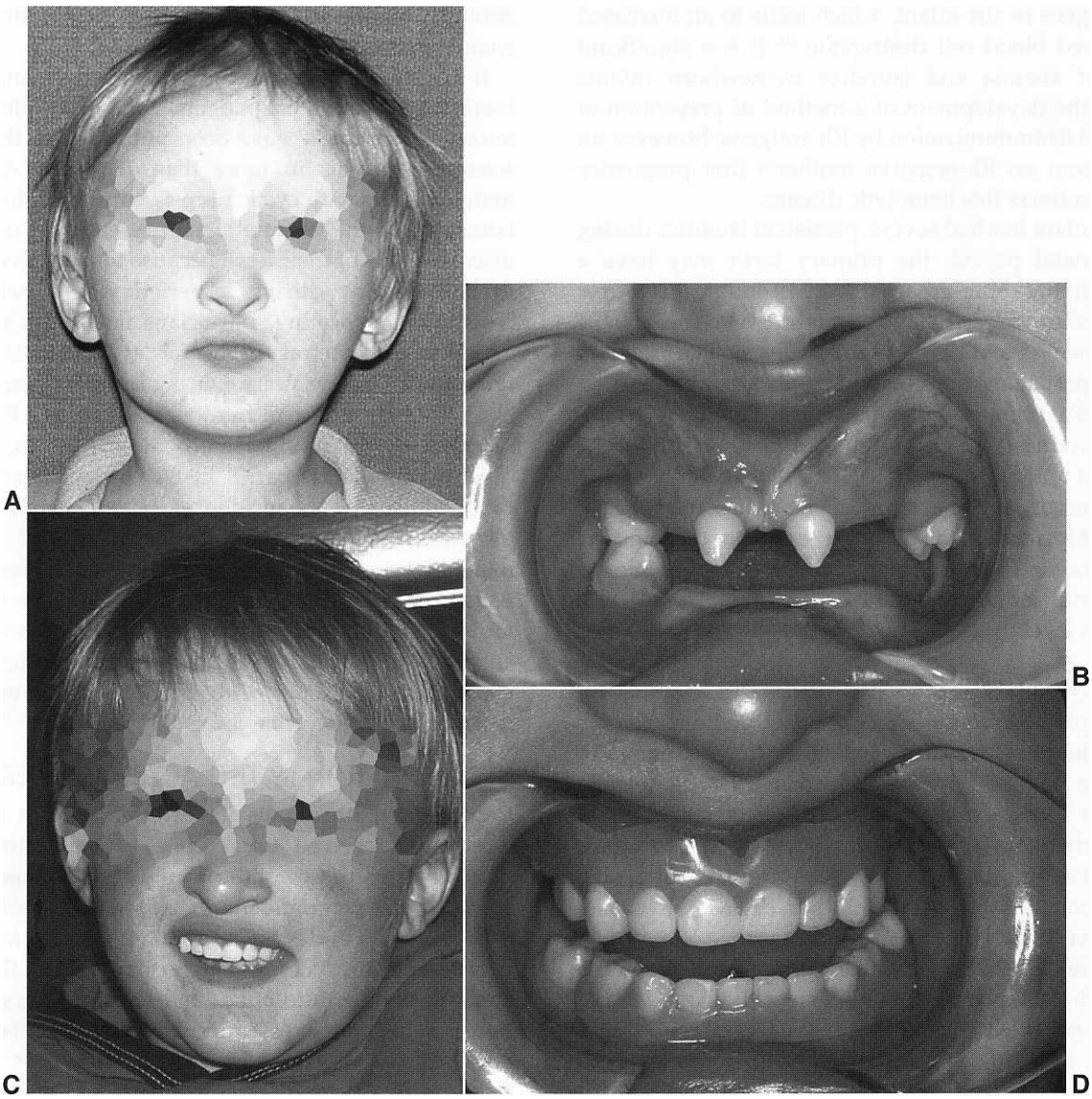


f C. 7-38. **A,** Four-year-old girl with many features of ectodermal dysplasia. There was a history of consanguinity. **B,** The anterior teeth were small and conical in shape. The lack of development of the alveolar process is evident in the photograph. C, The facial pattern was good even though many primary and permanent teeth were missing. D, Partial dentures were constructed, modified, and remade as additional teeth erupted. The models show how growth has occurred in the mandible and maxilla.

may serve until the child is old enough for implants or fixed bridgework as described in Chapter 22. Orthodontic and surgical procedures may be necessary before the prosthodontic treatment.

Bonding techniques have improved the ability to provide aesthetic interim restorations and greater function for patients with conical teeth with or without oligodontia or hypodontia. Nunn et al published a series of five papers (monthly, beginning in the March 2003 of the *British Dental Journal)* that outline the management of patients with hypodontia by a coordinated interdisciplinary team of dentists and demonstrate the advantages of this approach."' Several dental specialties are represented on the team. Ideally, the initial responsibility for oversight and coordination of the patient's care begins in infancy with the services of a pediatric dentist. As the patient grows and develops through puberty, a general family practitioner may assume the oversight and coordination responsibilities.

When maxillary lateral incisors are missing, the occlusion and arches must be analyzed carefully to determine whether there is sufficient room within the arch to maintain space and to provide fixed bridgework. If space for a normal-sized lateral incisor replacement is



f **G 7-39. A,** Four-year-old boy with characteristics of ectodermal dysplasia. Many primary and permanent teeth are congenitally missing. The skin is dry, and the hair is sparse. **B,** The anterior primary teeth are typically conical. C and **D,** A full maxillary overdenture and partial mandibular denture were constructed.

insufficient, the clinician may sometimes choose to move the canine forward into the lateral position and reshape it to make it appear more like a permanent lateral incisor.

INTRINSIC DISCOLORATION OF TEETH (PIGMENTATION OF TEETH)

The primary teeth occasionally have unusual pigmentation. Certain conditions arising from the pulp can cause the whole tooth to appear discolored. Factors causing these conditions include bloodborne pigment, blood decomposition within the pulp, and drugs used in procedures such as root canal therapy. Color changes in relation to trauma are discussed in Chapter 21.

DISCOLORATION IN HYPERBILIRUBINEMIA

Excess levels of bilirubin are released into the blood in a number of conditions."[5] If teeth are developing during periods of hyperbilirubinemia they may become intrinsically stained. The two most common disorders that cause this intrinsic staining are *erythroblastosis fetalis* and *biliary atresia.* Other less common causes are premature birth, ABO incompatibility, neonatal respiratory distress, significant internal hemorrhage, congenital hypothyroidism, biliary hypoplasia, tyrosinemia, a,-antitrypsin deficiency, and neonatal hepatitis.

Erythroblastosis fetalis results from the transplacental passage of maternal antibody active against red blood

cell antigens of the infant, which leads to an increased rate of red blood cell destruction." It is a significant cause of anemia and jaundice in newborn infants despite the development of a method of prevention of maternal isoimmunization by Rh antigens; however, an infant from an Rh-negative mother's first pregnancy rarely contracts this hemolytic disease.

If an infant has had severe, persistent jaundice during the neonatal period, the primary teeth may have a characteristic blue-green color, although in a few instances brown teeth have been observed. The color of the pigmented tooth is gradually reduced. The fading in color is particularly noticeable in the anterior teeth.

Cullen has reported the occurrence of erythroblastosis fetalis produced by Kell immunization.'[17] In utero, the maternal antibodies coat the fetal red blood cells and cause hemolysis. The fetus develops anemia with a resultant increase in the bilirubin content of the amniotic fluid. The newborn appears pale and anemic. Shortly after birth, jaundice occurs as a result of the high bilirubin levels.

DISCOLORATION IN PORPHYRIA

The porphyrias are inherited and acquired disorders in which the activities of the enzymes of the heme biosynthetic pathway are partially or almost completely deficient."[8] As a result, abnormally elevated levels of porphyrins and/or their precursors are produced, accumulate in tissues, and are excreted. Congenital erythropoietic porphyria (Gunther disease) is a rare autosomal recessive form of the disease.

Children with congenital erythropoietic porphyria have red-colored urine, are hypersensitive to light, and develop subepidermal bullous lesions when their skin is exposed to sunlight. Their primary teeth are purplish brown as a result of the deposition of porphyrin in the developing structures. The permanent teeth also show evidence of intrinsic staining but to a lesser degree.

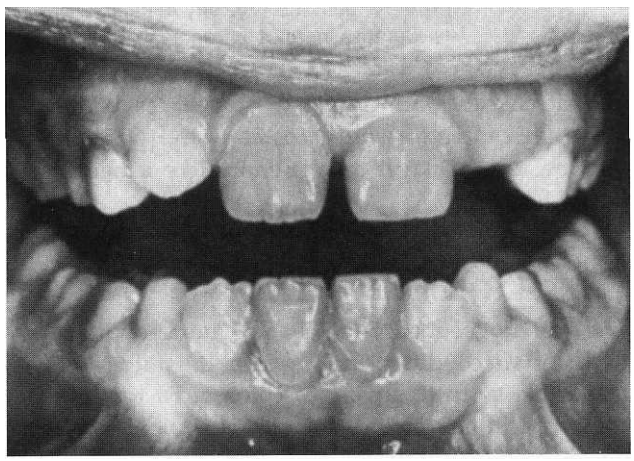
DISCOLORATION IN CYSTIC FIBROSIS

Cystic fibrosis is an inherited, chronic, multisystem, life-shortening disorder characterized primarily by obstruction and infection of the airways and poor digestion. It is autosomal recessive and is caused by mutations in both copies of the cystic fibrosis transmembrane regulator gene *(CFTR).* Zegarelli et al have suggested that tooth discoloration in persons with cystic fibrosis is a result of the disease alone; of therapeutic agents, especially tetracyclines; or of a combination of the two factors.[11`'] The possibility that there is at least in part an intrinsic developmental enamel abnormality secondary to the disease is supported by the investigations of Wright, Hall, and Grubb, who found abnormal enamel in the incisors of homozygous *CFTR* knockout mice.[120] Further studies by Arquitt, Boyd, and Wright also strongly suggest that *CFTR* plays an important role in enamel formation.'[2']

It is interesting to note that no papers attributing tooth discoloration in patients with cystic fibrosis to tetracycline therapy have been published in the major scientific journals in more than 10 years. Although many patients with cystic fibrosis who lived during the latter half of the twentieth century endured unsightly discolorations of their teeth because they received tetracycline therapy during a period when their tooth crowns were forming, physicians nowadays rarely, if ever, prescribe tetracyclines for any patients during their tooth-forming years. During the era when tetracycline therapy for children was common, Primosch reported on tetracycline tooth discolorations, enamel defects, and dental caries in 86 young patients with cystic fibrosis.[12']- The incidence of dental caries in these patients was compared to that in control subjects matched for sex, race, exposure to optimally fluoridated water, chronologic age, and dental age. The findings indicated a high prevalence of tooth discolorations and enamel defects but a significantly reduced caries experience in the patients with cystic fibrosis who had received the tetracycline drugs.

DISCOLORATION IN TETRACYCLINE THERAPY

Dentists and physicians have observed that children who have received tetracycline therapy during the period of calcification of the primary or permanent teeth show a degree of pigmentation of the clinical crowns of the teeth. As Van der Bijl and Pitigoi-Aron pointed out, because the tetracyclines chelate calcium salts, the drugs are incorporated into bones and teeth during calcification.'" The crowns of affected teeth are discolored, ranging from yellow to brown and from gray to black (Fig. 7-40). Currently most, if not all, infections in



[I G . **7-40.** Pigmentation in tetracycline therapy. The perma nent incisors that have erupted show a yellowish brown color.

children can be treated effectively with antibiotics that do not cause tooth discoloration. Consequently this previously common problem is now rarely encountered.

Tetracycline is deposited in the dentin and to a lesser extent in the enamel of teeth that are calcifying during the time the drug is administered. The location of the pigment in the tooth can be correlated with the stage of development of the tooth and the time and duration of drug administration. The tetracyclines, which are yellow, fluoresce under ultraviolet light. When tetracyclines in the dental structures darken from yellow to brown, the fluorescence diminishes because of the destruction of the fluorophore.

The exposure of the teeth to light results in slow oxidation, with a change in color of the pigment from yellow to brown. The larger the dose of drug relative to body weight, the deeper the pigmentation. The duration of exposure to the drug may be less important than the total dose relative to body weight.

Because tetracyclines can be transferred through the placenta, the crowns of the primary teeth may also show noticeable discoloration if tetracyclines are administered during pregnancy. Moffitt et al have observed that the critical period for tetracycline-related discoloration in the primary dentition is 4 months in utero to 3 months postpartum for maxillary and mandibular incisors and 5 months in utero to 9 months postpartum for maxillary and mandibular canines.[1] The sensitive period for tetracycline-induced discoloration in the permanent maxillary and mandibular incisors and canines is 3 to 5 months postpartum to about the seventh year of the child's life. The maxillary lateral incisors are an exception because they begin to calcify at 10 to 12 months postpartum.

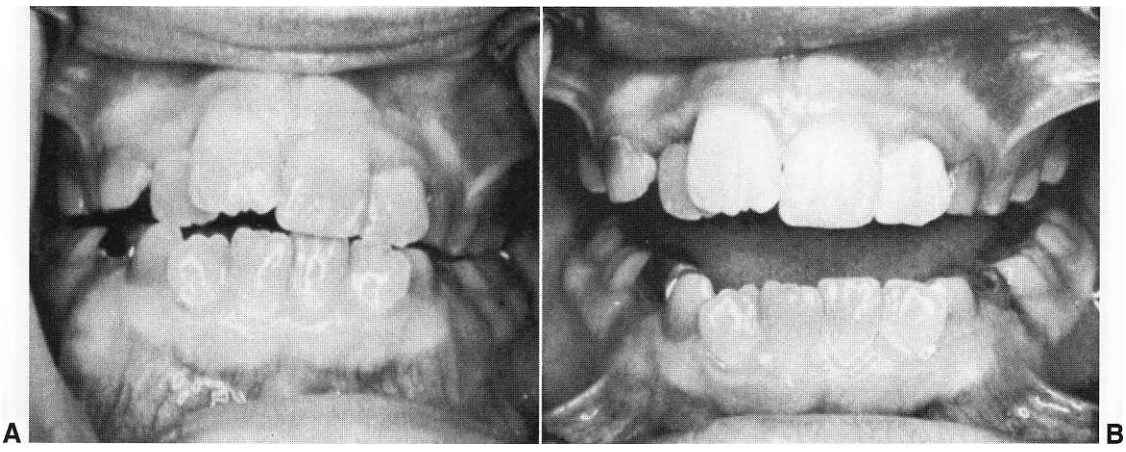
BLEACHING OF INTRINSIC TOOTH DISCOLORATION

Vital bleaching of intrinsically discolored teeth became a very popular dental cosmetic procedure during the late twentieth century (Fig. 7-41). Several safe techniques are available to achieve tooth bleaching. The accepted procedures incorporate the use of a peroxide compound placed on the tooth surface that bleaches the intrinsic tooth pigments to a lighter hue. Adding energy to the peroxide compound, as in the form of heat, light, or laser radiation, may accelerate this process.

Although many dilute tooth-bleaching products are available over the counter, the most efficient and effective systems are provided or prescribed by a dentist. To be safe, bleaching procedures must be carefully performed and monitored. Although these procedures are usually performed on permanent teeth, Brantley, Barnes, and Haywood have reported successfully bleaching discolored primary teeth in a 4-year-old girl.[1]

It is beyond the scope of this textbook to describe the various bleaching techniques in detail. The reader is referred to current textbooks on endodontics or cosmetic dentistry for more information. Another valuable resource with considerable detailed information is the Special Supplement of the *Journal of the American Dental Association,* volume 128, April 1997. This supplement provides reports by numerous recognized bleaching experts presented during an international symposium on the subject of nonrestorative treatment of discolored teeth.

If the tooth discoloration is severe and bleaching does not adequately improve the condition, the dentist may consider masking the visible surfaces with bonded veneer restorations similar to those discussed in



[I G **7-41. A,** Tetracycline-pigmented teeth. **B,** The maxillary incisors have been bleached; the lower mandibular incisors are untreated.

Chapter 18. Bleaching and enamel microabrasion may be used in combination for certain types of discoloration, and these procedures may also be used as adjunctive steps before placement of veneer restorations.

MICROGNATHIA

Micrognathia is usually considered a congenital anomaly; however, the condition may be acquired in later life. The mandible is most often affected (Fig. 7-42). The etiology of congenital micrognathia appears to be heterogeneous. Deficient nutrition of the mother and intrauterine injury resulting from pressure or trauma have been suggested as possible causes. In addition, micrognathia may be part of the Robin sequence, which also includes cleft palate (especially in the posterior with a rounded distal edge) and glossoptosis. Although this developmental sequence may be sporadic, it is often the pleiotropic expression of a gene for a condition such as Stickler syndrome or velocardiofacial syndrome and warrants a clinical genetic evaluation.[126]

Infants with mandibular micrognathia have difficulty breathing and experience episodes of cyanosis, and must be kept in a ventral position as much as possible. The anterior portion of the mandible is positioned so that the tongue has little if any support and can fall backward, causing an obstruction.

Based on longitudinal growth studies Pruzansky and Richmond reported that, in most instances of congenital micrognathia, the increment in mandibular growth as related to total facial growth during infancy and early childhood is sufficient to overcome the extreme recessiveness of the chin at birth.'-" This is often referred to as "catch-up growth." Daskalogiannakis, Ross, and Tompson performed 29 cephalometric measurements, taken at three different ages, on 96 patients with Robin sequence and compared them to similar measurements in 50 patients with isolated clefting of the palate (control group).'" They found that patients with Robin sequence had significantly smaller mandibles than the control group from approximately 5.5 years of age to 17 years of age. This finding suggests that the mandibles of patients with Robin sequence do not really grow proportionately more than those in others not affected by the disorder.

The nursing bottle may be used in the treatment of congenital micrognathia to help promote adequate function of the mandible. Because the infant should be made to reach for the nipple of the nursing bottle, the bottle should never be allowed to rest against the mandible. The parent is instructed to sit the infant on his or her lap in an upright position and place gentle forward pressure on the child's ramus with the thumb and fingers while offering the bottle. Thus the infant must extend the mandible to feed. Mandibular advancement by orthopedic force is sometimes recommended by Strohecker and Lahey.'[29] Surgical mandibular



[I C 7-42. **A,** Micrognathia in a girl 1 month of age. The chin was noticeably recessive. **B,** The radiograph shows the extent of the development of the dentition at birth. When the patient was 1 year of age, the micrognathia was less noticeable.

reconstruction in some cases is another option proposed by James and Ma.[13o]

Hotz and Gnoinsky have described the use of a special palatal obturator appliance to help infants born with Robin sequence.[13'] In addition to providing coverage to seal the cleft palate, the appliance includes a posterior extension to simulate the missing soft palate structures including the uvula. The appliance seems to stimulate the infant to maintain a more normal tongue position. The improved tongue position significantly reduces the tendency for the infant to experience life-threatening apneic episodes. Dean et al also reported successful use of similar appliances in infants with Robin sequence.[13'] Their study included 22 infants with severe airway obstruction. Their results supported the use of the obturator, but they also concluded that further study of treatment with these appliances was needed.

Acquired micrognathia may develop gradually and may not be evident until 4 to 6 years of age. This anomaly in growth is usually related to heredity. Ankylosis of the jaw caused by a birth injury or trauma in later life may result in an acquired type of micrognathia. Infection in the temporomandibular joint area can also cause arrested growth at the head of the condyle and lead to development of the acquired pattern of micrognathia. In cases of true ankylosis of the mandible, arthroplasty should be recommended.

ANOMALIES OF THE TONGUE

Pediatric patients with unremarkable medical histories rarely complain of symptomatic tongue lesions. However, the tongue should be inspected carefully during the examination procedure. Several benign conditions may be evident that should be brought to the attention of the parents.

Burket described four main types of papillae of the tongue.[133] Large circumvallate papillae, 10 to 15 in number, may be found on the posterior border of the dorsum. These papillae have a blood supply and are the site of numerous taste buds. Fungiform (mushroom-shaped) papillae may be distributed over the entire dorsum of the tongue; however, they are present in greater numbers at the tip and toward the lateral margins of the tongue. Inflammatory and atrophic changes occurring on the dorsum of the tongue may involve the vascularized fungiform papillae. The most numerous papillae of the tongue are the filiform papillae, which are thin and hairlike and evenly distributed over the dorsal surface. The filiform papillae are without a vascular core, and their continuous growth is slight. The foliate papillae represent a fourth type and are arranged in folds along the lateral margins of the tongue; the taste sensation is associated with these papillae.

MACROGLOSSIA

Macroglossia refers to a larger than normal size of the tongue. This condition may be either congenital or acquired. Congenital macroglossia, which is caused by an overdevelopment of the lingual musculature or vascular tissues, becomes increasingly apparent as the child develops.

An abnormally large tongue is characteristic of hypothyroidism, in which case the tongue is fissured and may extend from the mouth. Macroglossia is also commonly observed with type 2 glycogen-storage disease, neurofibromatosis type 1, and Beckwith-Wiedemann syndrome. It can be an isolated and sporadic (nonfamilial) trait or a familial (autosomal dominant) trait as studied by Reynoso et al.[13j] Macroglossia has also been cited as a characteristic of Down syndrome. Occasionally an allergic reaction will cause a transitory enlargement of the tongue (angioneurotic edema). Both allergic reaction and injury can cause such severe enlargement of the tongue that a tracheotomy is necessary to maintain a patent airway.

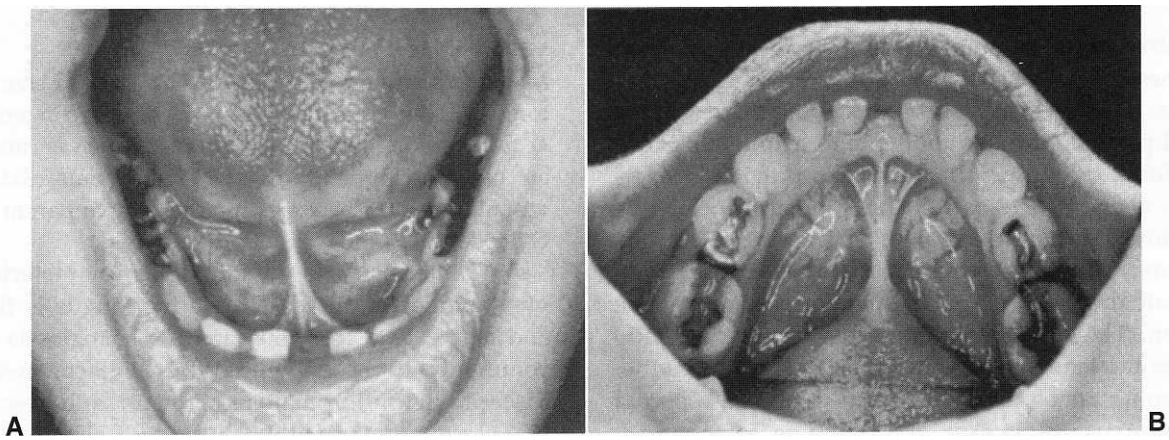
A disproportionately large tongue may cause both an abnormal growth pattern of the jaw and malocclusion. Flaring of the lower anterior teeth and an Angle class III malocclusion are occasionally the result of macroglossia.

The treatment of macroglossia depends on its cause and severity. Surgical reduction of a portion of the tongue is occasionally necessary.

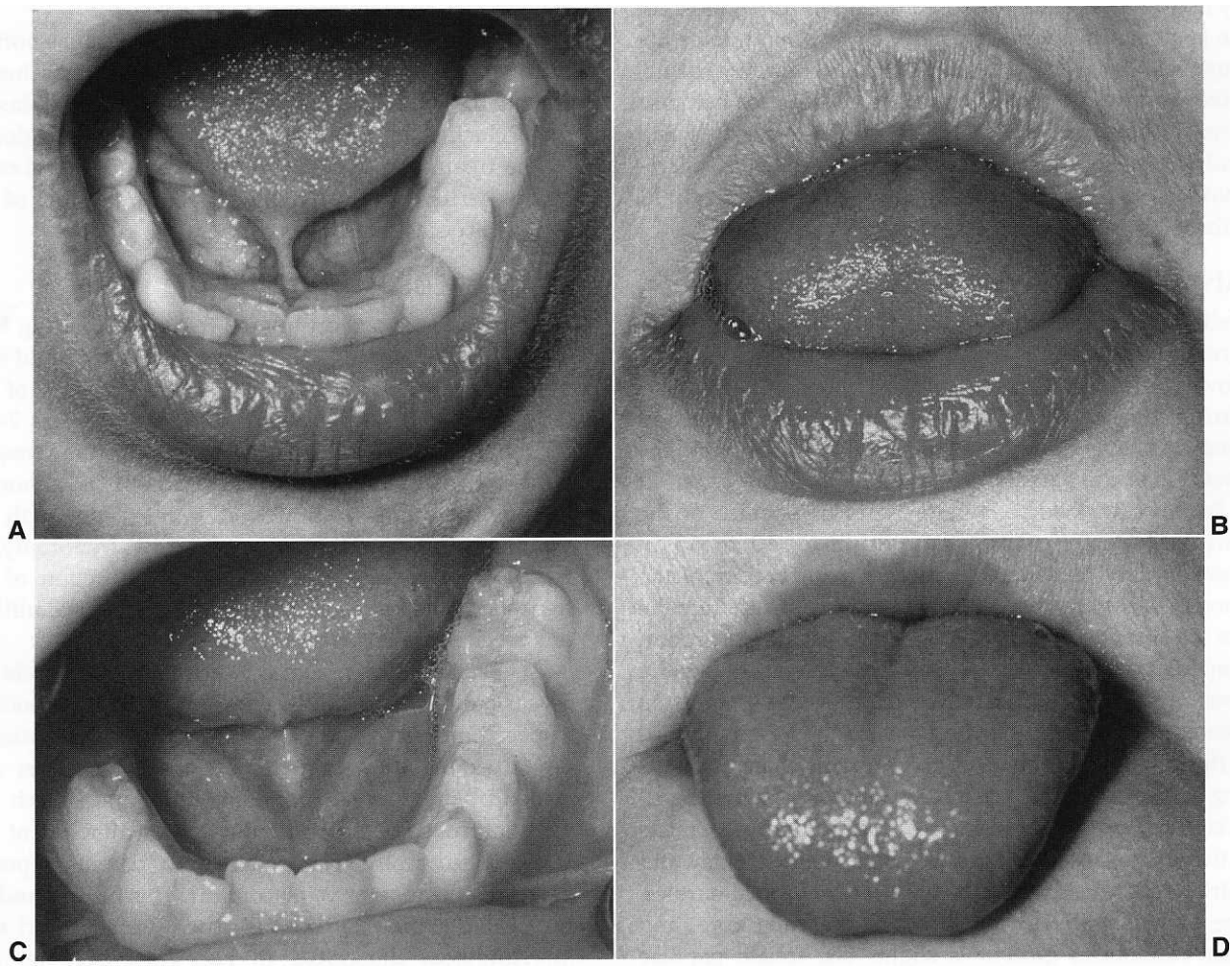
ANKYLOGLOSSIA (TONGUE-TIE)

In ankyloglossia a short lingual frenum extending from the tip of the tongue to the floor of the mouth and onto the lingual gingival tissue limits movements of the tongue and causes speech difficulties (Fig. 7-43). Stripping of the lingual tissues may occur if the tongue-tie is not corrected. Surgical reduction of the abnormal lingual frenum is indicated if it interferes with the infant's nursing (lingual frenectomy, frenotomy, or frenuloplasty). In the older child a reduction of the frenum should be recommended only if local conditions or speech problems warrant the treatment.

Ayers and Hilton reported a case of ankyloglossia in a 7-year-old boy who had been evaluated at his school for a speech problem.[j35] The patient previously had routine dental examinations, but no treatment had been suggested by the dentist. Tongue mobility and speech patterns improved greatly after the frenum attachment was released surgically. The patient and parents reported very little postoperative discomfort. This history and the results are similar to those of the 6-year-old girl with ankyloglossia illustrated in Fig. 7-44. Messner and Lalakea, after studying 30 children aged 1 to 12 years with ankyloglossia, concluded that tongue mobility and speech improve significantly after frenuloplasty.[136]



I **G 7-43. A,** Ankyloglossia (tongue-tie). A short, heavy lingual frenum extends from the top of the tongue to the floor of the mouth and onto the lingual tissue. **B,** A mirror view of the abnormal frenum.



F I G. 7-44. **A,** Ankyloglossia in a 6-year-old girl. **B,** Patient had limited tongue mobility and speech problems. C, Two weeks after surgical release. D, Tongue mobility and speech improved spontaneously.

FISSURED TONGUE AND GEOGRAPHIC TONGUE (BENIGN MIGRATORY GLOSSITIS)

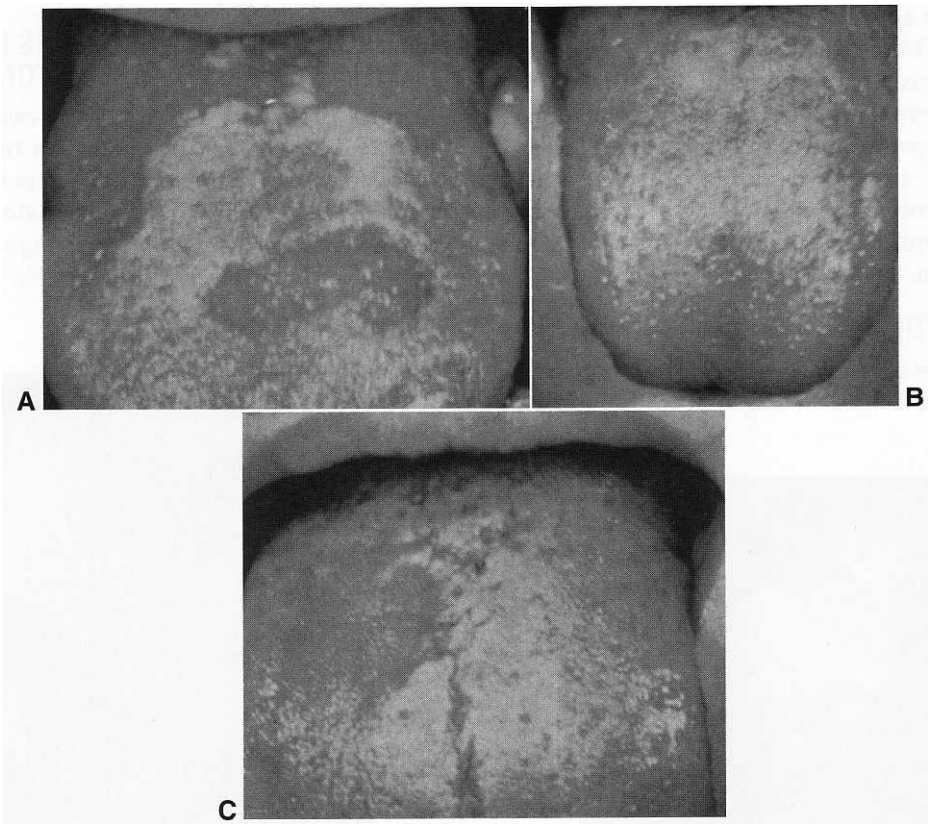
A fissured tongue is seen in a small number of children and may be of no clinical significance, although it is sometimes associated with hypothyroidism and Down syndrome. The fissures on the dorsum of the tongue usually have a symmetric pattern and may be longitudinal or at right angles to the margin of the tongue. Vitamin B—complex deficiency may be associated with the fissuring. Treatment of the fissured tongue is generally unnecessary unless a mild inflammation develops at the base of the fissures from an accumulation of food debris. Brushing of the tongue and improved oral hygiene aid in reducing the inflammation and soreness.

A wandering type of lesion and probably the most common tongue anomaly is known as *geographic tongue.* Rahamimoff and Muhsam observed a 14% prevalence of migratory glossitis in 5000 children 2 years old and younger.[137] Kleinman, Swango, and Pindborg reported a prevalence of geographic tongue of 0.6`7, in 39,206 children aged 5 to 17 years.' Meskin, Redman, and Gorlin have observed the incidence of geographic tongue among college students to be 1.1 % 139

Kullaa-Mikkonen studied the inheritance of fissured tongue in 31 families and concluded that fissured tongue with smooth-surfaced papillae is transmitted as an autosomal dominant trait with incomplete penetrance and is preceded by geographic tongue.[140] The severity of fissured tongue increased with age. Tongue fissuring with normal-appearing filiform papillae was found not to be familial and was not associated with geographic tongue.

Geographic tongue is often detected during routine dental examination of pediatric patients who are unaware of the condition. Red, smooth areas devoid of filiform papillae appear on the dorsum of the tongue. The margins of the lesions are well developed and slightly raised. The involved areas enlarge and migrate by extension of the desquamation of the papillae at one margin of the lesion and regeneration at the other (Fig. 7-45). Every few days a change can be noted in the pattern of the lesions. The condition is self-limited, however, and no treatment is necessary.

Banoczy, Szabo, and Csiba have indicated that gastrointestinal disturbances associated with anemia may be related to migratory glossitis.'`" In addition, a psychosomatic disorder should be considered as a



f c 7-45. **A,** Geographic tongue. The smooth areas are devoid of filiform papillae. **B,** The pattern observed at the initial visit is indistinguishable 4 weeks later. C, In 1 year a new pattern is developing on the dorsum of the tongue.

possible etiologic factor. Histologically the process appears to be superficial, with desquamation of the keratin layers of papillae and inflammation of the corium.

COATED TONGUE

A white coating of the tongue is usually associated with local factors. The amount of coating on the tongue varies with the time of day and is related to oral hygiene and the character of the diet. The coating consists of food debris, microorganisms, and keratinized epithelium found on and around the filiform papillae (Fig. 7-46).

Children who have a congenital or acquired deficiency in salivary flow may have a coated tongue, occasionally to the extent that a dry crust appears on the dorsum of the tongue. Frequent rinsings with artificial saliva will palliate the condition. Systemic disease with associated fever and dehydration may also cause a coating, which is usually white but may become stained with foods or drugs. Increased ingestion of liquid is appropriate to alleviate this situation. Brushing the tongue with a toothbrush and dentifrice will reduce the coating.

WHITE STRAWBERRY TONGUE

An enlargement of the fungiform papillae extending above the level of the white desquamating filiform papillae gives the appearance of an unripe strawberry. The condition has been observed in cases of scarlet fever and Kawasaki disease in young children. During the course of scarlet fever and other acute febrile conditions the coating disappears, and the enlarged red papillae extend above a smooth denuded surface, which gives the appearance of a red strawberry or raspberry. The tongue will return to normal after recovery from the systemic condition.

BLACK HAIRY TONGUE

Black hairy tongue is rarely seen in children but occurs in young adults and has been related to the oral and systemic intake of antibiotics, smoking, and excessive ingestion of dark drinks such as coffee and tea (Fig. 7-47). Accumulations of keratin on the filiform papillae in the middle third of the tongue become elongated into hairlike processes, sometimes as long as 2.5 cm (1 inch). Neville et al note that the cause is uncertain but that an apparent increase in keratin production or a decrease in normal keratin desquamation results.[1'5] It is a benign condition with no serious sequelae. Rigorous hygienic procedures such as brushing and scraping the tongue may help control it. When the condition appears during antibiotic therapy, it usually disappears again without specific treatment after the antibiotics have been discontinued.

INDENTATION OF THE TONGUE MARGIN (CRENATION)

During the examination of the pediatric patient the dentist may notice a scalloping or crenation along the lingual periphery. Careful examination will reveal the markings to be caused by the tongue's lying against the lingual surfaces of the mandibular teeth. Although usually no significance can be attached to these crenations, they have been related to pressure habits, macroglossia, vitamin B–complex deficiency, and systemic disease that causes a reduction of muscle tone.

MEDIAN RHOMBOID GLOSSITIS (CENTRAL PAPILLARY ATROPHY OF THE TONGUE)

Median rhomboid glossitis is an oval, rhomboid, or diamond-shaped reddish patch on the dorsal surface of the tongue immediately anterior to the circumvallate papillae. Flat, slightly raised, or nodular, it stands out distinctly from the rest of the tongue because it has no filiform papillae. This atrophic area is usually



FIG **7-46.** White coating of the tongue is usually associated with local factors.

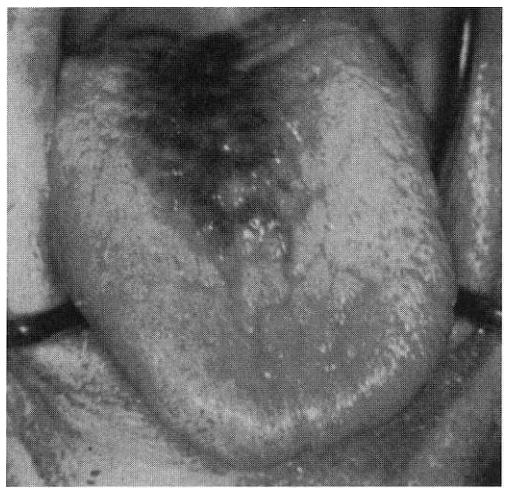


FIG. **7-47.** Black hairy tongue. This condition usually has no clinical significance.

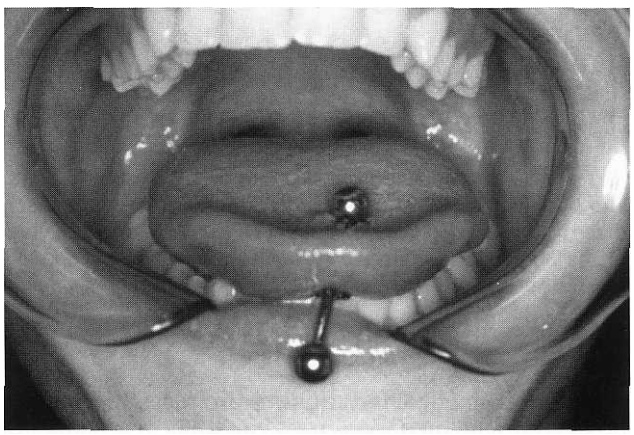
asymptomatic. Long believed to be a developmental anomaly, the condition is now recognized almost exclusively to represent a chronic, localized, and mild candidal infection, as proposed by Cooke.[142]

Although median rhomboid glossitis occurs more often in adults, it is sometimes seen in teenagers and even occurs infrequently in younger children. It has been observed with high prevalence in HIV-positive children by Barasch et al.[14]" Treatment with topical antifungal agents is appropriate.

TRAUMA TO THE TONGUE WITH EMPHASIS ON TONGUE PIERCING

A child may bite his or her tongue as a result of a traumatic blow or fall. The dentist may inadvertently traumatize the tongue with a cutting instrument during operative procedures. Deep laceration of the tongue requires suturing to minimize the scarring and to aid in hemorrhage control. In cases of severe injury the tongue should be examined carefully to detect any enlargement that might interfere with the maintenance of an open airway.

Tongue piercing, a deliberate trauma, is one of the popular types of body piercing occurring in all parts of the world today, especially among teenagers and young adults (Fig. 7-48). Tongue piercing is of particular interest in dentistry because it carries a high risk of adverse intraoral sequellae and can have significant systemic effects as well. The scientific literature has exploded with documented cases of complications following piercing procedures. Fractured teeth, dental abrasion, and gingival recession are reported to be common sequelae.[744'46] Other observed complications with life-threatening potential include brain abscess,[147] cephalic tetanus,[148] endocarditis,[748] Ludwig angina,'[so] and upper airway compromise.'" Whenever appropriate, dentists should counsel their patients and other members of the community about the serious risks associated with this form of body art. If patients insist on wearing tongue ornaments and other such body jewelry, they should be advised at least to remove them during athletic activities in which the risk of injury is very high."[2]



F **I G 7-48.** An example of a tongue ornament very popular among teenagers and young adults.

ABNORMAL LABIAL FRENUM

A maxillary midline diastema is frequently seen in preschool children and in those in the mixed dentition stage. It is important to determine whether the diastema is normal for that particular time of development or is related to an abnormal maxillary labial frenum.

A midline diastema may be considered normal for many children during the time of eruption of the permanent maxillary central incisors. When the incisors first erupt, they may be separated by bone, and the crowns incline distally because of the crowding of the roots. With the eruption of the lateral incisors and the permanent canines, the midline diastema will be reduced, and in most cases normal contact between the central incisors will develop (see Fig. 27-55).

Insufficient tooth mass in the maxillary anterior region, the presence of peg lateral incisors, or the congenital absence of lateral incisors may cause a diastema. Other factors and anomalies, including a midline supernumerary tooth, an oral habit, macroglossia, and abnormally large mandibular anterior teeth, should be considered as possible causes (in addition to an abnormal labial frenum) of the midline diastema.

The labial frenum is composed of two layers of epithelium enclosing a loose vascular connective tissue. Muscle fibers, if present, are derived from the orbicularis oris muscle.

The origin of the maxillary frenum is at the midline on the inner surface of the lip. The origin is often wide, but the tissue of the frenum itself narrows in width and is inserted in the midline into the outer layer of periosteum and into the connective tissue of the internal maxillary suture and the alveolar process. The exact attachment site is variable. It can be several millimeters above the crest of the ridge or on the ridge, or the fibers may pass between the central incisors and attach to the palatine papilla.

Many dentists delay considering an abnormal labial frenum as the cause of a diastema until all the maxillary permanent anterior teeth, including the canines, have erupted. This approach may be considered generally correct. However, other diagnostic points should be kept in mind.

One can carry out a simple diagnostic test for an abnormal frenum during mid to late mixed dentition by observing the location of the alveolar attachment when intermittent pressure is exerted on the frenum. If a



[I G **7-49.** Abnormal labial frenum. There is blanching of the free marginal tissue between the central incisors and also of the palatine papilla. A frenectomy is indicated.

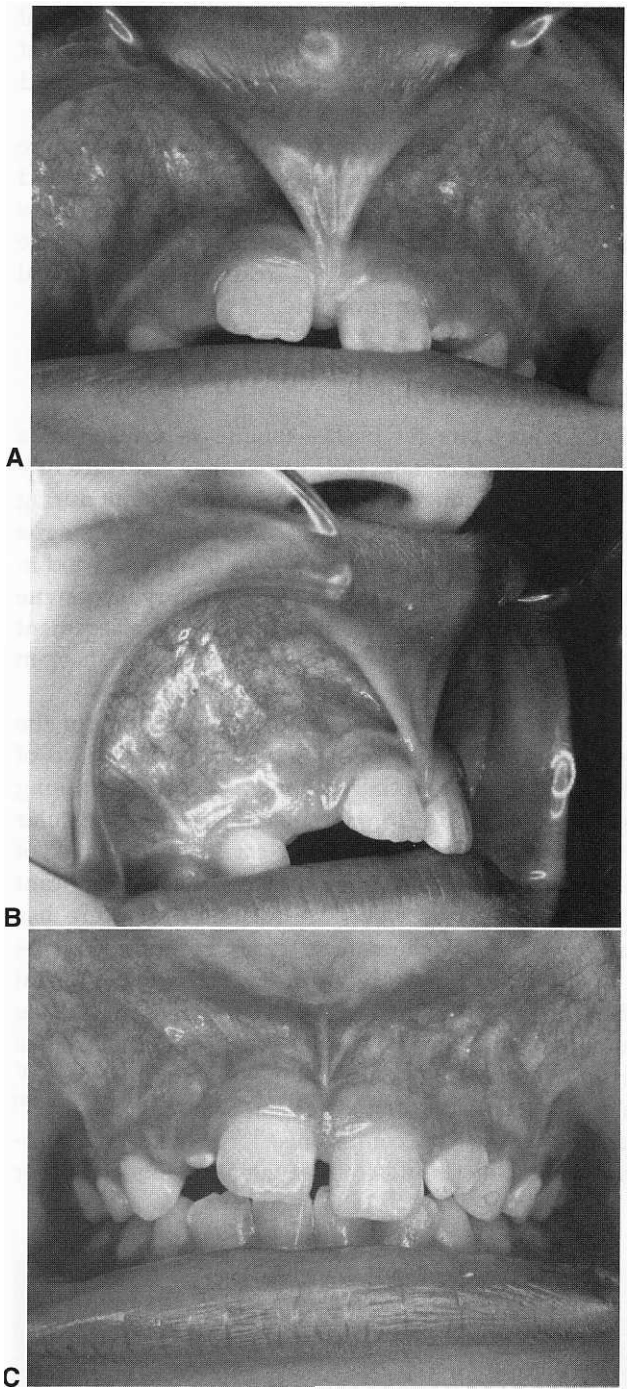
heavy band of tissue with a broad, fanlike base is attached to the palatine papilla and produces blanching of the papilla, it is safe to predict that the frenum will unfavorably influence the development of the anterior occlusion (Fig. 7-49).

The abnormal labial frenum, in addition to causing a midline diastema, can produce other undesirable clinical conditions. The heavy band of tissue and low attachment can interfere with toothbrushing by making it difficult to place the brush at the proper level in the vestibule to brush in the conventional manner. If fibers of the frenum attach into the free marginal tissue, stretching of the lip during mastication and speech may cause stripping of the tissue from the neck of the tooth. Such attachment may also cause the accumulation of food particles and eventual pocket formation. The abnormal frenum may restrict movements of the lip, may interfere with speech, and may produce an undesirable cosmetic result (Fig. 7-50).

FRENECTOMY

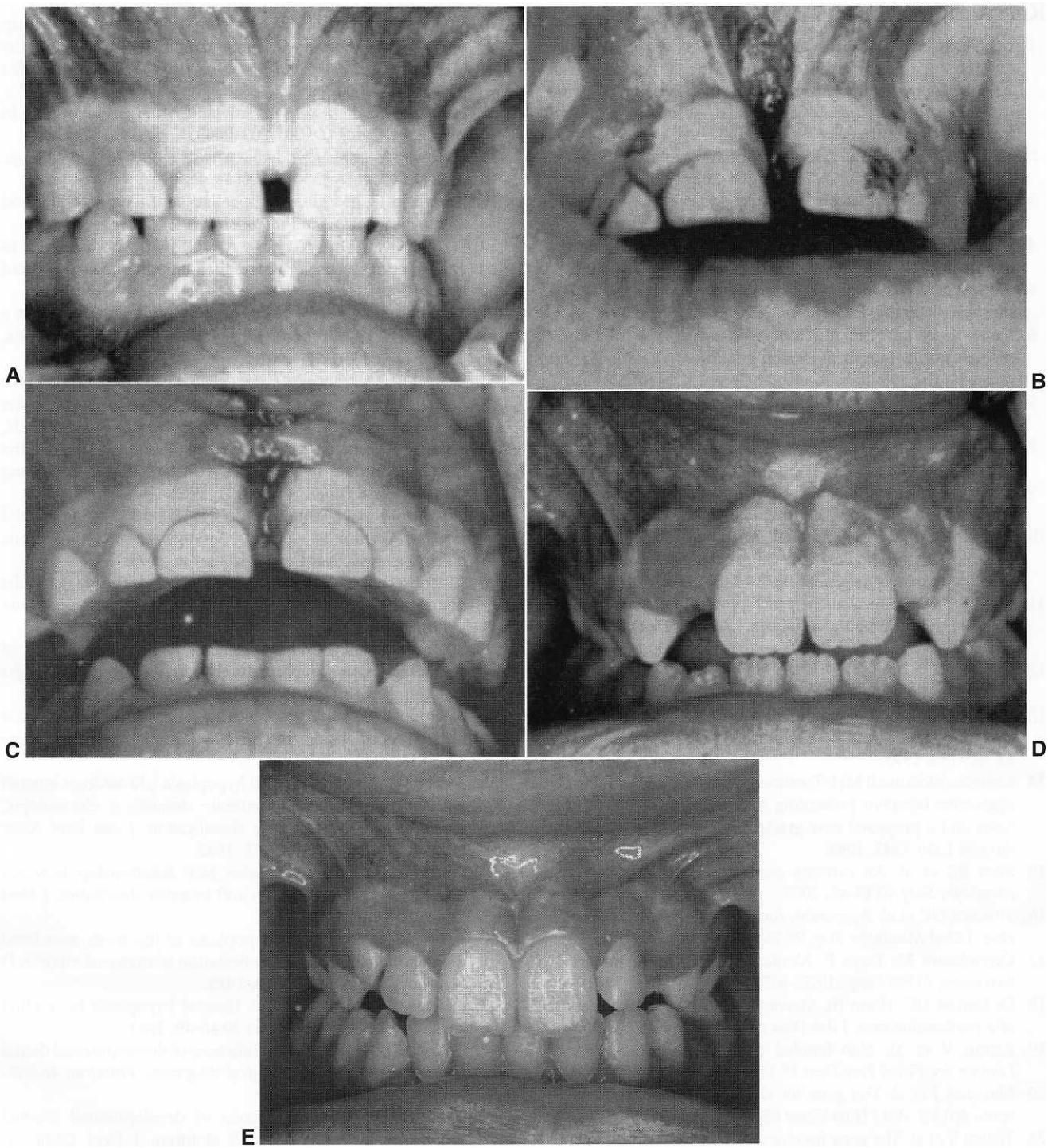
The decision regarding treatment of the labial frenum should be made only after a careful evaluation to determine whether the result will be undesirable if the condition is allowed to remain.

In the surgical technique, a wedge-shaped section of tissue is removed, including the tissue between the central incisors and the tissue extending palatally to the nasal palatine papilla (Figs. 7-50 and 7-51). Lateral incisions are made on either side of the frenum to the depth of the underlying bone. The free marginal tissue on the mesial side of the central incisors should not be disturbed. The wedge of tissue can be picked up with tissue forceps and excised with tissue shears at an area close enough to the origin of the frenum to provide a



f **G 7-50. A,** Abnormal labial frenum in an 8-year-old child. The heavy, fan-shaped band of tissue interfered with speech and presented an undesirable appearance. **B,** An oblique view of the abnormal frenum. C, A much more desirable appearance is observed 6 months after the frenectomy.

desirable cosmetic effect. Sutures are placed inside the lip to approximate the free tissue margins. It is generally unnecessary to suture or pack the tissue between the incisors.



f i i **7-51. A,** Abnormal labial frenum observed in a preschool child is causing a diastema between the primary central incisors and is interfering with normal movement of the upper lip. **B,** A wedge-shaped section of tissue including the frenum has been removed. C, Two sutures have been placed to approximate the tissue margins. **D,** The permanent incisors have erupted. No reattachment of the frenum fibers has occurred. **E,** A desirable result of the frenectomy is evident 5 years after the surgical procedure.

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CHAPTER

8

Tumors of the Oral Soft Tissues and Cysts and Tumors of the Bone

JOHN S. McDONALD

انتشارات
شایان نمودار

CHAPTER OUTLINE

BENIGN TUMORS OF THE ORAL SOFT TISSUE
- Squamous Papilloma and Verruca Vulgaris
- Fibroma
- Pyogenic Granuloma, Peripheral Ossifying Fibroma, and Peripheral Giant Cell Granuloma
- Neurofibroma and Neurofibromatosis (von Recklinghausen's disease)
- Hemangioma
- Lymphangioma
- Congenital Epulis (Gingival Granular Cell Tumor) of the Newborn
- Mucocele
- Ranula

BENIGN TUMORS OF BONE
- Fibro-Osseous Lesions of the Jaws
- Fibrous Dysplasia
- Ossifying and Cementifying Fibroma
- Juvenile Active Ossifying Fibroma
- Central Giant Cell Granuloma

ODONTOGENIC CYSTS
- Primordial Cyst
- Dentigerous Cyst
- Eruption Cyst or Eruption Hematoma
- Odontogenic Keratocyst
- Nevoid Basal Carcinoma Syndrome (Gorlin's Syndrome)

ODONTOGENIC TUMORS Ameloblastoma
- Adenomatoid Odontogenic Tumor
- Odontogenic Myxoma Ameloblastic
- Fibroma Ameloblastic Fibro-
- Odontoma
- Odontoma

MALIGNANT TUMORS
- Fibromatosis and Fibrosarcoma in Infancy and Childhood
- Malignant Lymphoma
  - *Hodgkin's lymphoma (Hodgkin's disease)*
  - *Non-Hodgkin's Lymphomas*
- Rhabdomyosarcoma
- Osteogenic Sarcoma Ewing's
- Sarcoma
- Langerhans' Cell Histiocytosis (Histiocytosis X)

Not only is it the responsibility of the dental practitioner to oversee the care and maintenance of the teeth and periodontium of each patient, but also it is at least as important to monitor and help maintain the overall health and well-being of that individual as manifested in the orofacial and head and neck structures that are so obviously within the purview of the dental profession. It is all too easy to focus strictly on the dental needs of the patient and remain oblivious to subtle or even not so subtle lumps, bumps, swellings, or changes in texture or color that may signify the presence of a reactive or a hamartomatous overgrowth of tissue or a benign or malignant disease. Oral tumors occur too often for the dentist to take the attitude that these lesions happen only in someone else's patients.

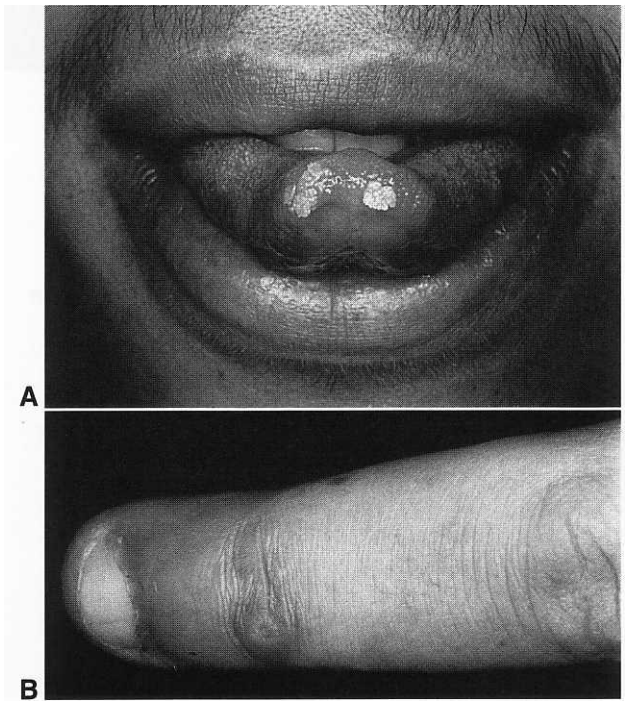
The purpose of this chapter is not to present a comprehensive treatise on oral tumors and cysts in children but to provide an overview of some of the more frequently encountered ones that may be found in the oral soft tissues and bone in the pediatric age group.

BENIGN TUMORS OF THE ORAL SOFT TISSUE

SQUAMOUS PAPILLOMA AND VERRUCA VULGARIS

The squamous papilloma is a relatively common, benign neoplasm that arises from the surface epithelium. It is typically an exophytic lesion whose surface may vary from cauliflower-like to fingerlike in appearance, and although it is generally a pedunculated lesion, it may arise from a sessile base. Although the average age of occurrence is in the fourth decade of life, nearly 20% of cases have been noted by Abbey, Page, and Sawyer and by Greer and Goldman to occur before 20 years of age.'" The most common sites of occurrence appear to be the tongue and palatal complex, followed by the buccal mucosa, gingiva, and lips. This lesion is also seen with some frequency on the alveolar ridge, floor of the mouth, and retromolar pad regions.

Oral verruca vulgaris, or oral warts, are exophytic papillomatous lesions indistinguishable clinically from oral squamous cell papillomas. Like their skin counterpart, the common wart (verruca vulgaris), they are a viral disorder associated with the human papillomavirus (HPV) and may be spread to the oral cavity in children through autoinoculation by finger or thumb sucking (Fig. 8-1). According to Praetorius's review of HPV-associated diseases of oral mucosa, types 2 and 57 were most commonly found.' In addition, DNA types 6, 11, and 16 have been demonstrated in oral verrucae as reported by Naghashfar et al as well as by Zeuss, Miller, and White.[45]



f**IG 8-1. A**, Verruca vulgaris on the anterior tip of the tongue. This is an example of autoinoculation from finger sucking. **B**, Notice the wart on this patient's finger from which he inoculated his tongue. (Courtesy Dr. Mark L. Bernstein.)

Although the histopathologic differences between squamous papilloma and verruca vulgaris are subtle, these lesions are distinguishable from one another, and HPV has been detected in some squamous papillomas. In one study by Jenson et al, HPV was found in two of five multiple papillomas, and in another study by Loning et al, it was reported that five of six clinically and morphologically defined papillomas tested positive for HPV-associated antigens.'" Syrjanen, Syrjanen, and Lamburg used the in situ DNA hybridization technique and reported the presence of HPV antigens in 57% of squamous papillomas studied.' The types of HPV identified in the study of Syrjanen et al and in a subsequent report by Eversole and Laipis were HPV-6 and HPV-11.[9] Ward et al detected the presence of HPV DNA types 6 and 11 in 68% of clinically diagnosed and histologically confirmed oral squamous cell papillomas analyzed by the highly sensitive polymerase chain reaction technique followed by dot blot hybridization of the polymerase chain reaction product with digoxigenin-labeled, type-specific oligonucleotide probes for HPV DNA types 6, 11, 16, and 18.[10]

Histologically, the papilloma is seen as a proliferation of the spinous cell layer in a papillary pattern, often with hyperkeratosis, acanthosis, and basilar hyperplasia. Mitotic figures may be prominent. The supporting fibrous connective tissue stroma often contains

prominent numbers of small blood vessels as well as an inflammatory cell infiltrate. The presence of a coarse keratohyalin granular cell layer and vacuolated cells with pyknotic nuclei (koilocytes) may be used to differentiate verruca vulgaris from a squamous papilloma. Treatment of either the oral squamous papilloma or verruca vulgaris is best accomplished by complete surgical excision of the lesion, including the base.

FIBROMA

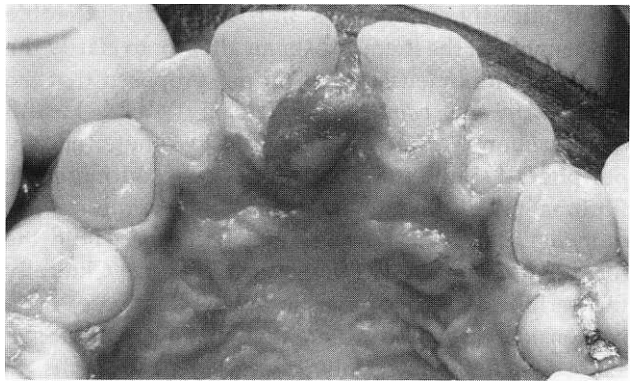
The fibroma is the most common benign soft tissue tumor found in the oral cavity. It is characteristically a dome-shaped lesion with a sessile base and a smooth surface that is usually the color of the surrounding mucosa. It may vary from firm to flaccid in texture and most commonly occurs in sites predisposed to irritation or trauma, such as the buccal mucosa, lip, tongue, gingiva, and hard palate. It may occur at any age. Sometimes termed *focal fibrous hyperplasia,* a fibroma occurring in the oral cavity is reactive in nature, being basically either a reactive type of fibrous hyperplasia or in some cases a healed pyogenic granuloma that has undergone sclerosis.

Histologically the fibroma is a dome-shaped lesion composed of a fibrous connective tissue stroma that may vary from loose and delicate to quite dense in its appearance, with an overlying layer of stratified squamous epithelium. Treatment consists of simple surgical excision. There is little propensity for recurrence.

PYOGENIC GRANULOMA, PERIPHERAL OSSIFYING FIBROMA, AND PERIPHERAL GIANT CELL GRANULOMA

The pyogenic granuloma is a relatively common soft-tissue tumor that arises from the fibrous connective tissue of the skin or mucous membranes. Originally believed to be a botryomycotic infection, it is now known to be a reactive inflammatory process in which an exuberant fibrovascular proliferation of the connective tissue occurs secondary to some low-grade, chronic irritation.

Clinically the pyogenic granuloma is a raised lesion on either a sessile or a pedunculated base. Its surface may have a smooth, lobulated, or, occasionally, warty appearance that is erythematous and often ulcerated (Fig. 8-2). Depending on the age of the lesion, it will vary in texture from soft to firm and is suggestive of an ulcerated fibroma. Because of the pronounced vascularity of these lesions, *they* often bleed easily when probed. A review of pyogenic granulomas of the oral cavity by Angelopoulos revealed a 65% to 70% incidence of occurrence on the gingiva, most commonly the maxillary anterior labial gingiva, followed by the lips, tongue, buccal mucosa, palate, mucolabial or mucobuccal fold, and alveolar mucosa of edentulous areas."



[I G **8-2.** Pyogenic granuloma arising from the interdental papilla between the maxillary central incisors. The surface is lobulated with erythema and ulceration. There was poor oral hygiene with heavy accumulation of plaque on the lingual surfaces of the teeth.

Twenty-seven percent of cases in his series of 46 patients were in individuals younger than 20 years of age. Lawoyin, Arotiba, and Dosumu reviewed 38 cases from Ibadan, Nigeria, and reported an age range of 5 to 75 years (mean age, 33 years) with the main site of occurrence on the gingiva (74%), which closely approximates the findings of Angelopoulos.[11,12]

Histologically the pyogenic granuloma presents as a remarkable proliferation of plump fibroblasts and endothelial cells with the formation of prominent numbers of thin-walled, endothelium-lined vascular channels. A polymorphous inflammatory cell infiltrate is present, and the overlying surface epithelium is often ulcerated. Treatment consists of surgical excision, with care being taken to completely remove any local irritant that may still be present that would predispose to recurrence of the lesion.

In addition to an ulcerated fibroma, which in fact may itself be a nearly healed or sclerosed pyogenic granuloma, both the peripheral ossifying fibroma and peripheral giant cell granuloma must be considered in the differential diagnosis of pyogenic granuloma because these lesions are clinically indistinguishable from it. The peripheral ossifying fibroma, which has also been called the *peripheral odontogenic fibroma, is* a reactive lesion believed to be of periodontal ligament origin that occurs exclusively on the gingiva. In the largest series of cases, reported by Cundiff, 50% of the lesions were noted to occur in individuals between 5 and 25 years of age, with the peak incidence at 13 years." The lesions were approximately equally divided between the maxilla and the mandible, with more than 80% of the lesions in both jaws occurring anterior to the molar area.

Over time, the terms *peripheral ossifying fibroma* and *peripheral odontogenic fibroma* have been used

synonymously, which has introduced considerable confusion into the literature. In an attempt to clarify the confusion in nomenclature, Gardner proposed that the term *peripheral odontogenic fibroma* (World Health Organization [WHO] type) be used to denote the rare peripheral counterpart of the central odontogenic fibroma (WHO type) and that the term *peripheral ossifying fibroma* be retained and used to denote the relatively common reactive gingival lesion.[4] Clinically the peripheral odontogenic fibroma (WHO type) must be considered in the differential diagnosis of dome-shaped or nodular, usually nonulcerated, growths on the gingiva.

Histologically the peripheral ossifying fibroma demonstrates a proliferation of plump fibroblasts in a characteristic stroma of delicate, interlacing collagen fibrils. Osteoid and calcified material varying from dystrophic calcification to spicules of lamellar bone may be found in the lesion. The surface epithelium is often ulcerated. Although simple surgical excision is the treatment of choice, recurrences are not uncommon and were reported by Cundiff and by Eversole and Rovin in 16% and 20% of cases, respectively.[11]

The peripheral giant cell granuloma, like the peripheral ossifying fibroma, is a lesion unique to the oral cavity, occurring only on the gingiva or, in the case of the peripheral giant cell granuloma, also on the alveolar mucosa of edentulous areas. Like the pyogenic granuloma and peripheral ossifying fibroma, the peripheral giant cell granuloma may represent an unusual response to tissue injury. It is distinguishable from the pyogenic granuloma and peripheral ossifying fibroma only on the basis of its unique histomorphology, which is essentially identical to that of the central giant cell granuloma that is discussed later in this chapter. As reported by Giansanti and Waldron in their review of 720 cases, 33% were seen in patients under 20 years of age, which concurs with the findings of a study by Andersen, Fejerskov, and Philipsen, who reported 33 of 97 cases occurring in individuals between 5 and 15 years of age.[17] There is a nearly 2:1 predilection of females to males, with the mandible being involved more often than the maxilla. Nedir, Lombardi, and Samson point out that although it is quite rare, root resorption may be associated with peripheral giant cell granuloma."

The peripheral giant cell granuloma is best treated by complete surgical excision, with care taken to excise it at its base. Little tendency for recurrence has been noted.

NEUROFIBROMA AND NEUROFIBROMATOSIS (VON RECKLINGHAUSEN'S DISEASE)

Neurofibroma is a benign neural neoplasm of Schwann cell origin of which several clinical forms are recognized. The solitary neurofibroma may present in the skin or oral mucous membranes as a soft tumor with a sessile or pedunculated base. Some neurofibromas are diffuse, presenting as a soft, nonspecific tissue mass or swelling. Although patients with neurofibromatosis may have either solitary or diffuse neurofibromas, the presence of either lesion does not in itself herald the diagnosis of this syndrome. The presence of a third form, the plexiform neurofibroma, is, however, considered to be diagnostic of neurofibromatosis. The plexiform neurofibroma differs from either the solitary or diffuse form in that it remains confined to the perineurium, presenting as a tortuous, fusiform enlargement of a nerve.

Neurofibromatosis type 1 (NF1), known as von Recklinghausen's disease, is a relatively common disease showing autosomal dominant inheritance with complete penetrance, variable clinical expressivity and pleiotropy, and age-dependent expression of clinical manifestations. It occurs in approximately 1 in 3000 live births with an equal sex predilection. It is carried on chromosome 17, and there is growing evidence that the tumor-suppressing properties of neurofibromin (the NF1 gene product) may be impaired or lost. It is said to be the most common single-gene disorder to affect the human nervous system. The criterion for diagnosis is the presence of two or more of the following features: six or more cafe-au-lait spots (1.5 cm or larger in postpubertal individuals, 0.5 cm or larger in prepubertal individuals), two or more neurofibromas of any type or one or more plexiform neurofibromas, freckling in the axillary or inguinal region, optic glioma, two or more pigmented iris hamartomas or Lisch nodules, dysplasia of the sphenoid bone or dysplasia or thinning of long bone cortex (pseudarthrosis), and a first-degree relative with NF1.

The NF1 gene is a tumor-suppressor gene, and thus patients with neurofibromatosis are at increased risk of developing benign and malignant tumors. in a study by Young, Hyman, and North, 5% of 495 adults and children diagnosed at their clinic had neoplasms, including both central nervous system and non–central nervous system neoplasms.[19] Poyhonen, Niemela, and Herva found the risk of malignancy in NF1 in Finland to be 8%, with the most common being malignant peripheral nerve sheath tumors.`°

hhtraorally, neurofibromas may present as nodular lesions on either a sessile or pedunculated base, often with a normal, pink mucosal color (Fig. 8-3); as a diffuse, ill-defined swelling with a firm to doughy consistency; or as a diffuse, noncompressible mass. They are most frequently found on the tongue and buccal mucosa but occasionally present as intraosseous lesions, which occur most commonly in the posterior mandible (Fig. 8-4).

The most common radiographic findings according to Lee, Yan, and Pharoah include an increase in bone

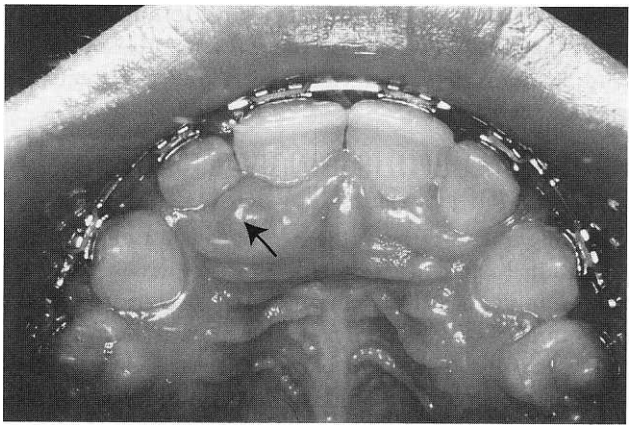


FIG. **8-3.** A 14-year-old boy undergoing orthodontic therapy was referred for evaluation of gingival hyperplasia on the lingual surfaces of the maxillary incisors and a firm swelling on the palatal side of tooth number 9 that was found after biopsy to be a neurofibroma (arrow).

density, enlarged mandibular foramen, lateral bowing of the mandibular ramus, increase in dimensions of the coronoid notch, and decrease in the mandibular angle.[27]

In six cases for which computed tomographic scans were available, enlargement of the mandibular foramen and concavity of the medial surface of the ramus were seen.

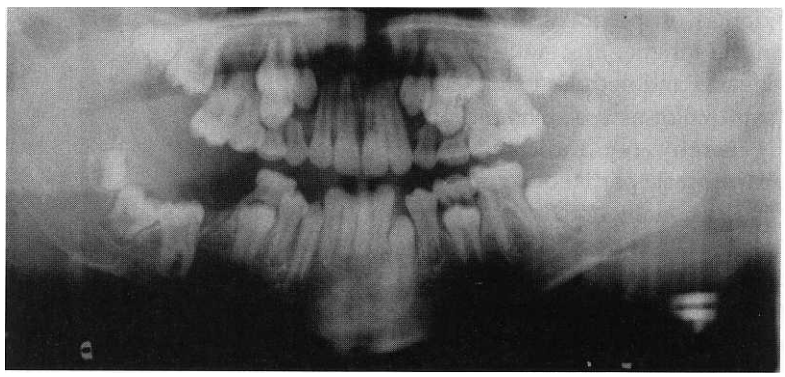
A great deal of histomorphologic variability may be noted, from relatively circumscribed but nonencapsulated solitary neurofibromas to diffuse and plexiform neurofibromas. The diffuse neurofibroma is characterized by its infiltrative growth pattern along connective planes and its Wagner and Meissner tactile corpuscles. Plexiform neurofibromas are characterized by fascicles of neoplastic Schwann cells and collagen within a myxoid matrix that is confined to the perineurium. Mast cells are common findings within neurofibromas.

Solitary soft lesions are best treated by simple surgical excision and show little propensity for recurrence. Surgical excision of diffuse neurofibromas is difficult because of their diffuse, infiltrating nature. Plexiform neurofibromas are also difficult to treat as they have a tendency to grow along the course of a nerve, which results in frequent recurrences. According to Needle et al, children younger than 10 years of age; children with plexiform neurofibromas in the head, face, and neck; and those with tumors that cannot be almost completely removed are at particular risk for progression of their lesions.[22]

HEMANGIOMA

The hemangioma is a common, benign, vasoformative tumor that frequently occurs in the head and neck in children. It is generally believed to be hamartomatous rather than neoplastic in nature. Most hemangiomas are either present at birth or develop within the first year of life. It is believed that even those lesions that do not appear until adult life may have been present but not clinically evident until they began to enlarge. The classification of hemangiomas arising within the head and neck is confusing at best, and a discussion of it is not within the scope of this chapter.

Hemangiomas arising in the oral soft tissues most commonly affect the tongue, lips, and buccal mucosa. Clinically, they may be flat or raised and may vary from deep red to blue in color. Their histologic classification is based on the size of the vascular spaces. A capillary hemangioma is composed of many tiny capillaries with a pronounced endothelial cell proliferation. A cellular hemangioma consists principally of a proliferation of endothelial cells with only small numbers of discernible capillaries. A cavernous hemangioma is characterized by large, blood-filled sinusoidal spaces lined by endothelial cells and supported by a fibrous stroma. Hemangiomas may also occur centrally within either the mandible or maxilla.



F **I G 8-4.** Neurofibroma involving the molar region and ramus of the right mandible in a child with neurofibromatosis. *(Courtesy Dr. Robin Cotton.)*

Central hemangiomas of bone may be asymptomatic, picked up only as incidental radiographic findings, or they may cause pain and swelling. They are typically well circumscribed, radiolucent lesions with no characteristic radiographic pattern to denote their underlying nature. Occasionally, loose or displaced teeth may be seen. The central hemangioma of bone bears a radiographic similarity to any number of other relatively well-circumscribed unilocular or multilocular radiolucent lesions in the jaws. Therefore it is advisable to aspirate such lesions with a needle before surgery or dental extraction to avoid the possibility of severe blood loss or exsanguination caused by the inability to control the resultant profuse bleeding.

The treatment for hemangiomas varies with the type, location, and size of the lesion involved. Many lesions spontaneously involute with age, especially those that are noted early and cease growing during the first year of life. Others require no treatment because of their small size and innocuous nature.

LYMPHANGIOMA

Lymphangiomas are thought to arise as a benign hamartomatous proliferation of sequestered lymphatic rests. Forming along tissue planes or penetrating adjacent tissue, they become canalized and, in the congenital absence of venous drainage, accumulate fluid. The majority becomes clinically evident early in life; however, a small number may not be manifest for a number of years. Lymphangiomas are classified on a histologic basis into three types: capillary lymphangiomas, cavernous lymphangiomas, and cystic hygroma. The capillary lymphangioma is typically composed of a proliferation of thin-walled, endothelium -lined channels primarily devoid of erythrocytes. The cavernous lymphangioma is characterized by the presence of dilated sinusoidal endothelium-lined vascular channels devoid of erythrocytes. The cystic hygroma is a macroscopic form of the cavernous lymphangioma, with large sinusoidal spaces lined with a single layer of endothelial cells that form multilocular cystic masses of varying sizes. Lymphangiomas of the oral soft tissues occur most commonly on the tongue, lips, and buccal mucosa. They are often elevated and nodular in appearance and may have the same color as the surrounding mucosa. Treatment is generally not indicated for small oral mucosal lymphangiomas; partial or complete spontaneous involution is occasionally noted.

Although cystic hygromas may be found in sites in the oral cavity, such as the tongue, they most frequently appear as a mass in the neck, occasionally extending into the mediastinum. Most commonly presenting as an asymptomatic soft tissue mass, they are usually slow growing; however, they may undergo sudden enlargement in the presence of trauma, inflammation, internal hemorrhage, or respiratory tract infection. Large cystic hygromas may encroach on the airway and esophagus, leading to difficulty in swallowing and even causing airway obstruction.

CONGENITAL EPULIS (GINGIVAL GRANULAR CELL TUMOR) OF THE NEWBORN

The congenital epulis of the newborn is a rare lesion of uncertain histogenesis that occurs exclusively in newborn infants, chiefly on the maxillary anterior alveolar ridge and less commonly on the mandibular anterior alveolar ridge. Although usually solitary lesions, they may be multiple, most often affecting both the maxilla and mandible. Clinically the lesion presents at birth as a pink, smooth to lobulated, pedunculated mass that may vary in size from a few millimeters to more than 7 cm in diameter. More than 90% of cases occur in girls (Fig. 8-5).

Ugras et al point out that, while typically considered a lesion of uncertain histogenesis, immunohistochemical studies have revealed strong and diffuse cytoplasmic staining for neuron-specific enolase and vimentin, which suggests that the congenital epulis may be derived from uncommitted nerve-related mesenchymal cells [23]

Although histologic similarities to the granular cell myoblastoma have long been noted, the epithelium over the congenital epulis is generally thin without rete ridge formation, whereas the giant cell myoblastoma is characterized by pseudoepitheliomatous hyperplasia of

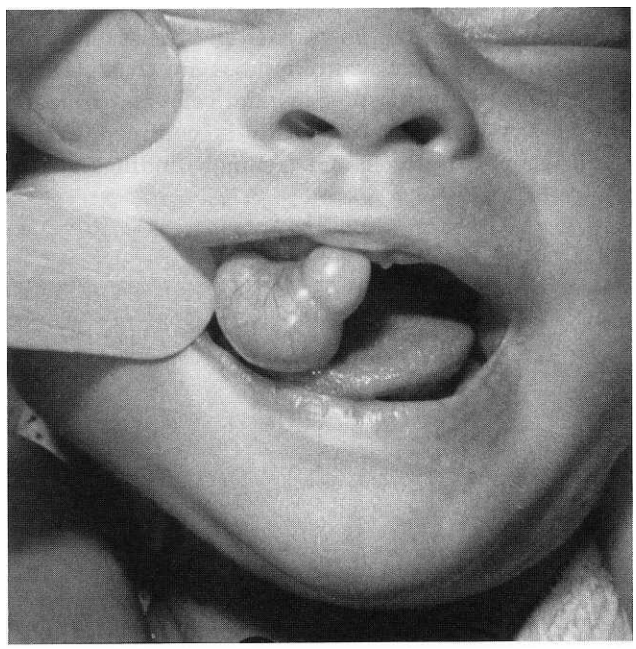


FIG **8-5.** Congenital epulis of the newborn. (Courtesy *Dr. Robin Cotton.)*

the overlying epithelium; distinct differences are obvious on both ultrastructural and immunohistochemical evaluation.

Although the clinical presence of the congenital epulis may frighten parents, it ceases to grow following birth and is entirely benign, with some cases under-going spontaneous involution. The usual treatment is simple surgical excision, with care taken not to interfere with the developing dentition. There is no propensity for recurrence, even in those cases in which the lesion is incompletely removed.

MUCOCELE

The mucocele, or *mucus retention phenomenon,* as it is often called, is a salivary gland lesion of traumatic origin that forms when the main duct of a minor salivary gland is torn with subsequent extravasation of mucus into the fibrous connective tissue so that a cystlike cavity is produced. The wall of this cavity is formed by compressed bundles of collagen fibrils, and its lumen contains inspissated mucin.

Mucoceles are noted to occur most commonly on the lower lip, with the floor of the mouth and buccal mucosa being the next most frequent sites of involvement. Mucoceles are only rarely seen on the upper lip, retromolar pad, or palate. Although they may occur at any age and have been reported to be present at birth, they tend to be noted most frequently in the second and third decades of life. No obvious sex predilection is noted.

A mucocele may be located either in the superficial mucosa, where it is typically seen as a fluid-filled vesicle or blister (Fig. 8-6), or deep within the connective tissue as a fluctuant nodule with the overlying mucosa normal in color. There may be spontaneous drainage of the inspissated mucin with temporary resolution, especially in superficial lesions, and subsequent recurrence as the mucous saliva continues to drain into the connective tissue at the site of the torn duct. Fibrosis may be observed over the surface of long-standing lesions where chronic periodic drainage has taken place.

Treatment is by surgical excision, with removal of the involved accessory salivary gland. Marsupialization will only result in recurrence.



f **I G 8-6.** Mucocele on the lower lip. Notice the small white area of fibrosis where the mucocele has spontaneously drained with healing on the roof of the lesion, only to have the mucocele recur.

RANULA

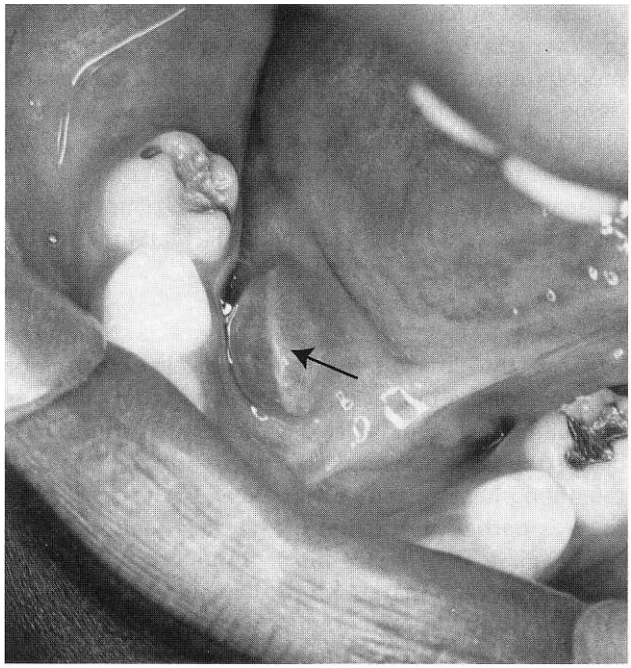
Ranula is the clinical term for a mucocele occurring on the floor of the mouth after trauma to components of the sublingual glands. Two varieties of ranula exist: cystic (mucus retention cyst) and pseudocystic (mucus retention phenomenon or mucocele). In the cystic type, there is partial obstruction of the distal end of a sub-lingual gland duct that results in a small epithelial-lined cyst usually less than 1 cm in diameter. However, the most common variety of ranula (which is pseudocystic) forms as a result of extravasation of mucus into the fibrous connective tissue after a tear in a sublingual gland duct and, as in the case of a mucocele, arises as a result of the escape of mucus into the adjacent connective tissue. The clinical term *plunging ronnie is* used when extravasated mucus dissects through the mylohyoid muscle and along the fascial planes of the neck, producing a swelling evident in the floor of the mouth.

Ranulas are typically slowly enlarging, fluctuant masses occurring to one side of the midline of the floor of the mouth and are so named because of their resemblance to the bloated underside of a frog's belly (Fig. 8-7). Lesions are usually treated by marsupialization, with occasional recurrence being noted. Chronic recurrence may require excision of the entire involved gland.

BENIGN TUMORS OF BONE

FIBRO-OSSEOUS LESIONS OF THE JAWS

The fibro-osseous lesions of the jaws include a diverse group of lesions sharing as a common denominator the replacement of normal bone architecture by a benign fibrous stroma containing varying amounts of mineralized material, including woven bone, lamellar bone, and curvilinear trabeculae or spherical calcifications. The mineralized material may histologically resemble bone and/or cementum. Because of the similar histomorphology of these lesions, diagnosis is best made on the basis of distinguishing clinical and radiographic as well as histologic features. This group of lesions includes fibrous dysplasia (considered a developmental process), reactive or dysplastic processes grouped under the collective term *cemento-osseous dysplasia,* and ossifying fibroma, which is a benign neoplasm. Because of the frequency of occurrence of fibrous dysplasia and



f **G . 8-7.** Ranula on the floor of the mouth (

ossifying fibroma of bone in the jaws of children, and the relative lack of occurrence of the cemento-osseous dysplasia group of lesions, only the former two are discussed here.

FIBROUS DYSPLASIA

Fibrous dysplasia of the jaws is a distinct clinicopathologic entity that is generally considered to be a nonneoplastic developmental lesion of bone. One of a variety of disease entities included in the spectrum of benign fibro-osseous lesions of the jaws, it is distinguished from the others by its clinical and radiographic features. In the child and adolescent, it has most often been confused with the ossifying and cementifying fibroma, which is a benign neoplasm believed to be of periodontal ligament origin. Fibrous dysplasia is caused by a somatic activating mutation of the alpha subunit of the G protein (Gsalpha) that ultimately results in abnormalities of osteoblast differentiation and therefore of bone matrix. Progression of the lesion results from an increase in osteoblastic bone resorption.

Two forms of fibrous dysplasia exist, the relatively rare polyostotic form of the disease and the consider-ably more common monostotic fibrous dysplasia. A severe form of polyostotic fibrous dysplasia called *McCune-Albright syndrome is* associated with cafe-au-lait macules on the skin and a variety of endocrine disorders, including Cushing's syndrome, hyperthyroidism, hyperparathyroidism, diabetes mellitus, and precocious puberty.

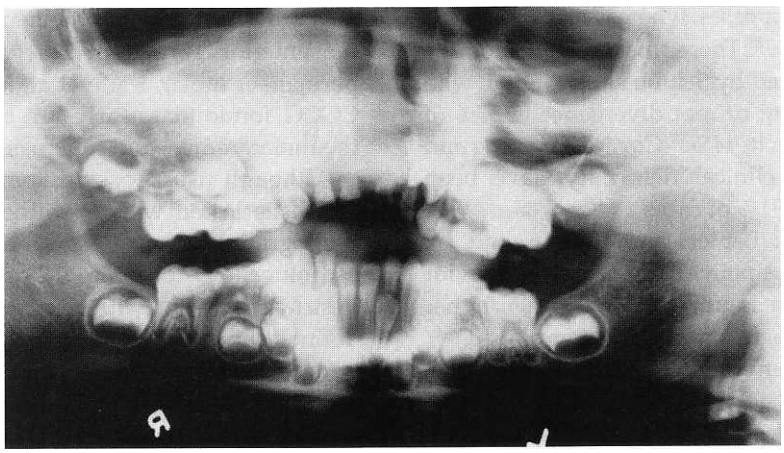
Monostotic fibrous dysplasia is seen with approximately equal frequency in males and females, with the maxilla being involved more frequently than the mandible. Maxillary involvement can be an especially serious form of the disease, frequently involving contiguous bones across suture lines, including the maxillary sinus, floor of the orbit, sphenoid bone, base of the skull, and occiput. This form of the disease has been called *craniofacial fibrous dysplasia* and is not truly monostotic in its nature.

Monostotic fibrous dysplasia tends to develop early in life. Of 22 cases reported in one study by Waldron and Giansanti, 9 patients were initially diagnosed as having fibrous dysplasia before 20 years of age, with a reliable clinical history of occurrence in the first or second decade of life in an additional 6 cases.[24] The most common clinical manifestation is a painless swelling of the jaws characterized by a smooth, uniform, fusiform expansion of the involved alveolar ridge. Obliteration of the mucobuccal or mucolabial fold is a common feature, with the overlying mucosa being normal in appearance. When the maxilla is involved, elevation of the eye may be noted.

Radiographically, maxillary lesions most often show a ground-glass appearance of the bone, whereas mandibular lesions usually show either a ground-glass or a mixed radiolucent-radiopaque appearance (Fig. 8-8). Their borders are typically poorly defined, except for the anterior portion of some maxillary lesions, which may appear to be well circumscribed. Divergence of roots may be noted, and in children in whom developing permanent teeth are present there may be displacement of teeth with noneruption (see Fig. 8-8). Other potential distinguishing radiographic findings as reported by Petrikowski et al include superior displacement of the mandibular canal and a fingerprint bone pattern as well as displacement of the maxillary sinus cortex, alteration of the lamina dura because of the abnormal bone pattern, and narrowing of the periodontal ligament space."

Histologically there is a benign fibrous stroma with bony trabeculae varying from woven to lamellar in appearance, proportionate to the relative maturity of the lesion.

Monostotic fibrous dysplasia of the jaws is typically a slow-growing, painless, progressive enlargement of bone whose growth pattern often stabilizes with time as maturation in skeletal growth is reached. Because of the benign nature of this lesion, as well as the fact that surgically its margins are ill defined and blend into the surrounding normal bone, conservative therapy is indicated. Surgery, chiefly in the form of osseous recontouring, should be considered only in those cases in which there is functional or significant cosmetic deformity and then usually only after stabilization of the



II G **8-8.** Panelipse radiograph for a child with fibrous dysplasia presenting as a uniformly radiodense lesion crossing the midline with poorly defined borders blending into the surrounding bone. Notice the displacement of the developing teeth.

disease process. Because this is a benign lesion of bone, radiation therapy is contraindicated because of the possibility of development of a postradiation sarcoma in the area.

OSSIFYING AND CEMENTIFYING FIBROMA

The ossifying fibroma is a benign neoplasm of bone grouped with fibrous dysplasia and other benign nonodontogenic tumors under the broad category of benign fibro-osseous lesions of the jaws. As with other benign fibro-osseous lesions, it is characterized histologically by a benign fibrous stroma, with formation of variable amounts of woven-appearing bone, lamellar bone, and spherical to annular to amorphous cementum-like calcifications. Although it was traditionally considered to be an odontogenic neoplasm of periodontal ligament origin affecting the tooth-bearing areas of the jaws, the occurrence of histologically identical neoplasms in the temporal, frontal, ethmoid, and sphenoid bones leaves this concept in doubt. When the predominant calcified component is bone, the term is *ossifying fibroma.* When cementum or cementum-like calcifications predominate, the term is *cementifiying fibroma.* When both bone and cementum-like tissues are present, the lesions are termed *cemento-ossifying fibromas.* Although there is a predilection for occurrence in the third and fourth decades of life, the ossifying fibroma is found with some frequency in patients younger than 20 years of age. The mandible is involved more often than the maxilla, with the molar and premolar region of the mandible being the most common site of occurrence.

The ossifying fibroma may be entirely asymptomatic, being discovered on routine radiographic examination, or may present as a painless expansion of bone. Radiographically the ossifying fibroma is a well-circumscribed lesion, often with a well-demarcated sclerotic border (Fig. 8-9). Beyond this, the radiographic features are quite variable, as demonstrated by Eversole, Leider, and Nelson. [26] It most frequently presents as a unilocular radiolucency with or without radiopaque foci, which may be superimposed over teeth, may be interposed between contiguous teeth, or may reside in edentulous regions. Aggressively expansile lesions with or without radiopaque foci as well as multilocular expansile lesions may also be noted. Divergence of roots of teeth and/or root resorption may be noted with varying degrees of frequency. Eversole, Leider, and Nelson reported root

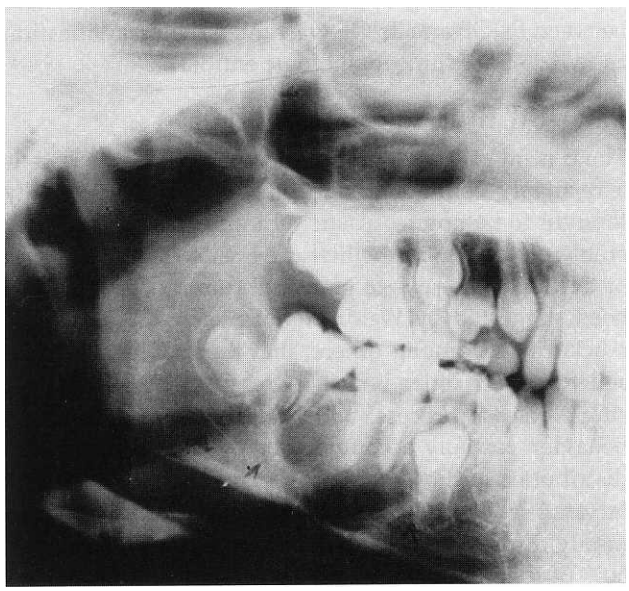


FIG . **8-9.** Ossifying fibroma in a 10-year-old boy. The radiograph shows a well-circumscribed, expansile, radiolucent lesion with thinning of the inferior cortical plate of bone. *(Courtesy Dr. Mark L. Bernstein.)*

divergence in 17% of their cases, whereas root resorption was encountered in 11%.[2'] These rates are considerably lower than those observed by Sciubba and Younai, who noted tooth displacement or root divergence in 33% of cases and root resorption in 44%.[227] Cortical thinning and bony expansion with clinical deformity have been reported in as many as 91% of cases.

Studies by Eversole, Leider, and Nelson and by Sciubba and Younai have pointed out that the behavior of the ossifying fibroma is unpredictable when either radiographic or histologic criteria are employed.[26'27] Initial treatment is by enucleation or curettage where possible, with the frequency of recurrence varying from 0% as reported by Waldron to 28% in a series reported by Eversole, Leider, and Nelson.[2',2s]

JUVENILE ACTIVE OSSIFYING FIBROMA

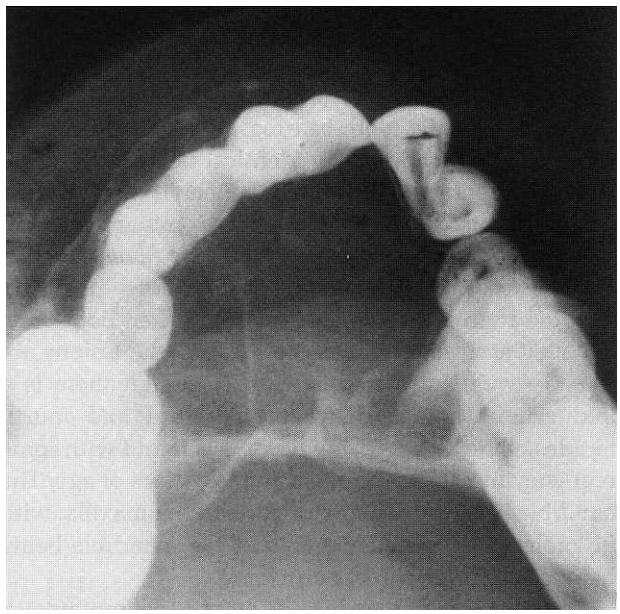
The term *juvenile active ossifying fibroma* °OF) encompasses two distinct histopathologic variants of an aggressive form of ossifying fibroma involving the craniofacial bones: the psammomatoid juvenile ossifying fibroma (PsJOF) and trabecular juvenile ossifying fibroma (TrJOF). El-Mofty, in a review of the literature, reported six new cases of JOF, three of the psammomatoid type and three of the trabecular variant. [729] The PsJOF was noted to occur predominantly in the sinonasal and orbital bones in patients with a mean age range of 16 to 33 years (range, 3 months to 72 years), whereas the TrJOF primarily affected the jaws in patients with a mean age range of 8[1]/2 to 12 years (range, 2 to 12 years). Aggressive growth was noted to occur in some, but not all, cases of both types. El-Mofty proposed that these histologic, demographic, and clinical differences between PsJOF and TrJOF warranted their classification as two distinct clinicopathologic entities.

CENTRAL GIANT CELL GRANULOMA

The central giant cell granuloma (CGCG) of the jaws is a relatively common lesion of bone that is generally thought to be nonneoplastic in nature. There is considerable discussion in the literature as to the relationship of CGCG of the jaws to giant cell tumor of bone. It has been suggested by Waldron and Shafer, as well as by Auclair et al, among others, that the CGCG and giant cell tumor of other bones represent a spectrum of a single disease process, with the differences in clinical behavior partially accounted for by differences in age distribution and by the site of occurrence, among other factors.[30'31] The CGCG of the jaws occurs most commonly during the first 30 years of life, with more than 60% of lesions noted before 20 years of age and nearly 50% occurring in patients younger than 16 years of age. The CGCG occurs more often in females than in males. It may be found in either jaw, but the mandible is involved twice as often as the maxilla. Although it is generally accepted that the majority of lesions occur in the anterior portion of the jaw, anterior to the first permanent molar, Kaffe et al, in a study of 80 cases of CGCG, found that nearly 50% of their cases occurred in the molar, ramus, and tuberosity regions.[32]

Although usually asymptomatic, CGCG may be an expansile lesion with or without divergence of teeth. Radiographically the CGCG may vary from a unilocular radiolucency to a multilocular expansile bone-destructive lesion with faint trabeculation and an ill-defined border (Fig. 8-10). Both displacement and noneruption of teeth may be noted. Histologically the CGCG is composed of a delicate, fibrous connective tissue stroma, interspersed with multinucleated giant cells, plump fibroblasts, and capillaries. Prominent numbers of extravasated red blood cells with associated hemosiderin pigment are often scattered throughout the fibrous connective tissue. Varying amounts of osteoid and trabeculae of bone are also noted.

Traditionally, treatment of CGCG has consisted of complete curettage or conservative surgical excision. Auclair et al, however, have pointed out that the recurrence rate may be higher than previously suggested in the literature." In their series, 5 of the 25 patients for whom follow-up information was available developed recurrence. The average age of these five patients was 11 years compared with an average age of 29 years for patients without recurrence. All five of the patients with recurrence were under the age of 17 years, and these patients accounted for 45% of all those in this age group



[I I **G . 8-10.** Central giant cell granuloma involving the anterior mandible presenting radiographically as a multilocular, radiolucent lesion with expansion and thinning of the cortical plates. Notice also the faint trabeculation within the lesion.

with follow-up. Minic and Stajcic studied 31 cases of CGCG of the jaws in 29 patients.[33] Seven lesions demonstrated cortical perforation and soft tissue extension. Lesions recurred in five cases, all of which showed microscopically confirmed cortical perforation at the time of first occurrence. Hence, despite the good prognosis for patients with this condition, close long-term follow-up would seem to be indicated, particularly in those cases in which cortical perforation is noted. In cases in which less radical nonsurgical treatment options are desirable, especially to avoid disfigurement, intralesional injections with triamcinolone acetonide have been used successfully to stop lesional growth, possibly effecting some regression in size and providing bony filling of the osteolytic lesion. Radiation therapy is contraindicated.

ODONTOGENIC CYSTS

PRIMORDIAL CYST

Traditionally the primordial cyst was considered to be a relatively uncommon type of odontogenic cyst formed by cystic degeneration of the enamel organ (primordium) before the formation of enamel or dentin. Primordial cysts are usually asymptomatic and are most likely to be found on routine radiographic examination. Because of this, although they begin to evolve when the enamel organ is developing before the formation of the enamel or dentin, they may not be noticed or become clinically evident for many years.

Radiographically, the primordial cyst may be a well-circumscribed, unilocular- or multilocular-appearing radiolucent lesion in a location where a permanent tooth failed to develop and where none has been extracted. Such cysts are more commonly found in the third molar region but may occur in any location where a permanent tooth would have formed.

Although more typically lined by nonkeratinizing, stratified squamous odontogenic cyst-lining epithelium with a fibrous connective tissue wall, the primordial cyst may present as a histologically distinct type of odontogenic cyst, the odontogenic keratocyst. In one large series of cases reported by Brannon, 60 of 135 (44.4%) of primordial cysts were histologically odontogenic keratocysts.[34] Some authors have concluded that all primordial cysts are odontogenic keratocysts and use the two terms synonymously. As shown by Brannon, however, this practice ignores the histologic evidence to the contrary.

Appropriate treatment for the primordial cyst is surgical removal unless an extremely large bone-destructive lesion is present, in which case cystotomy with placement of a polyethylene drain followed by cystectomy when the cyst has reached a manageable size is the treatment of choice. If the lesion is found histologically to be an odontogenic keratocyst, the chance for recurrence is high.

DENTIGEROUS CYST

The dentigerous cyst is a relatively common type of odontogenic cyst associated with the crown of an impacted, embedded, or otherwise unerupted tooth. Although these lesions are typically asymptomatic and are usually found on routine radiographic examination, they may be large, destructive, expansile lesions of bone. The radiographic appearance is that of a well-defined radiolucent lesion that may be unilocular or multilocular in its appearance. The lesion may vary in size from one in which the differentiation from an enlarged dental follicle is, at best, arbitrary to a large, bone-destructive lesion that may resorb the roots of primary teeth, cause the divergence of or completely displace permanent teeth, and produce large areas of bone destruction in either the mandible or the maxilla (Fig. 8-11).

In addition to its potential for bone destruction and because of the multipotential nature of this epithelium derived from the dental lamina, several entities may arise in or be associated with the wall of a dentigerous cyst. In a study by Brannon, 8.5% of clinical dentigerous cysts were found histologically to be odontogenic keratocysts.[34] Although ameloblastomas are relatively uncommon in children, McMillan and Smillie believe that the majority of ameloblastomas associated with dentigerous cysts arise in children or adolescents." Studies by Leider, Eversole, and Barkin and by Gardner and Corio have shown that in 50% to 80% of cases cystic ameloblastoma appeared radiographically as dentigerous cysts.[36,37] Also, based on histopathologic features,

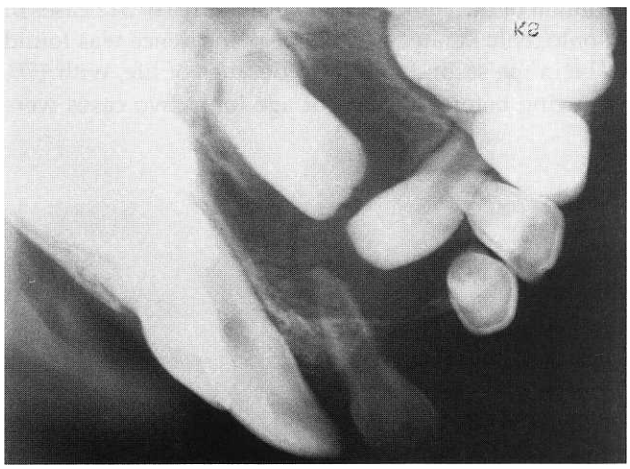


FIG. **8-11.** A dentigerous cyst in an 8-year-old girl associated with the developing maxillary central incisor with bone destruction and expansion, as well as displacement of the lateral incisor.

Leider, Eversole, and Barkin concluded that these ameloblastomatous changes occurred in a preexisting cyst rather than representing cystic degeneration of a solid tumor.'"" Mucus-secreting cells, and more significantly mucoepidermoid carcinoma, may also arise in the wall of a dentigerous cyst.

The treatment of choice for dentigerous cysts is surgical removal. Large bone-destructive lesions are, however, best treated initially by cystotomy and the placement of a polyethylene drain in the lesion; this will facilitate shrinkage with healing around the periphery to the point where the lesion becomes amenable to more simple surgical removal. Because of the potential for occurrence of an odontogenic keratocyst or the development of an ameloblastoma or mucoepidermoid carcinoma, all such lesions, when removed, should be submitted for histopathologic evaluation.

ERUPTION CYST OR ERUPTION HEMATOMA

The eruption cyst or eruption hematoma is a type of dentigerous cyst associated with an erupting primary or permanent tooth in its soft tissue phase after erupting through bone. The lesion is usually a translucent, smooth, painless swelling over the erupting tooth. If bleeding occurs into the cystic space, it may appear blue to blue-black and is then called an *eruption hematoma.* In most cases, no treatment is indicated because the tooth will usually erupt into the oral cavity undelayed and in a normal fashion.

ODONTOGENIC KERATOCYST

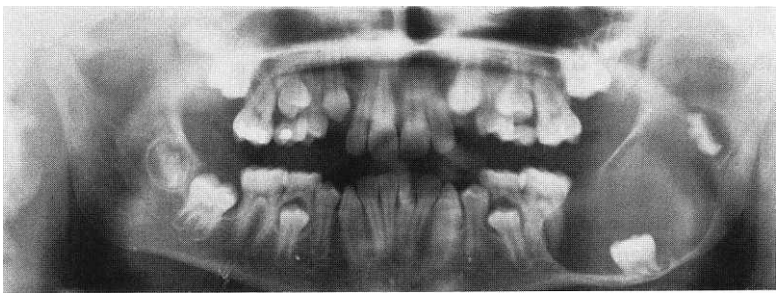
The odontogenic keratocyst is a distinct clinicopathologic entity with a clearly identifiable histologic appearance. Clinically it is characterized by a high rate of recurrence after treatment and the potential to become an aggressive, bone-destructive lesion. In a study by Brannon of the clinicopathologic features of 312 cases of odontogenic keratocysts, the peak incidence was found to be in the second and third decades of life, with 17% occurring before 20 years of age (only two cases were noted in children younger than 10 years of age).[34] The mandible is involved twice as frequently as the maxilla, with the most common site of origin being the mandibular third molar region and ramus of the mandible, followed by the maxillary third molar region, mandibular first and second molar area, maxillary canine area, and mandibular premolar region. In this series, half of the patients were symptomatic, with swelling and drainage being the most common clinical findings.

Radiographically, the odontogenic keratocyst may be a unilocular or multilocular lesion, almost always with a well-defined sclerotic border (Fig. 8-12). Tooth displacement, particularly of unerupted teeth, as well as mobility, root resorption, root divergence, and extrusion of erupted teeth may be seen. Although radiographic evaluation typically includes a panoramic radiograph, in the case of large lesions, especially those in the maxilla, computed tomographic studies should be performed preoperatively to accurately define margins and soft tissue extension that may be present.

The histologic appearance is pathognomonic. The lesion has a thin, generally uniform lining epithelium, usually 6 to 10 cells in thickness, with palisading and hyperchromasia of the basal layer of cells and a corrugated surface; often parakeratotic squames are shed into the lumen of the cyst cavity.

There is no uniform agreement as to the most appropriate method of treatment of odontogenic keratocysts. Recommendations vary from enucleation, with or without adjunctive therapy such as application of Carnoy solution, to marsupialization or decompression, to marginal or segmental resection. This is partly because of their variable clinical presentation, which includes differences in size and the multilocular nature of many odontogenic keratocysts, as well as their benign nature coupled with the high frequency of recurrence that has been recorded for these lesions.

Eyre and Zakrzewska studied 22 reports citing 1592 cases of odontogenic keratocysts with a recurrence rate



f G 8-12. An odontogenic keratocyst in a 9-year-old girl involving the angle and ramus of the left mandible with displacement of the developing second and third molar teeth. (Courtesy Dr. Steven Reubel.)

varying from 3% to 62% and an average rate of recurrence of 30.8%." As shown by Woolgar, Rippin, and Browne, 70% of recurrences take place within 5 years.'" Although these lesions should be surgically excised, enucleation is difficult because the cyst lining is often extremely thin and friable. Anand, Arrowood, and Krolls suggested that disruption of the epithelial lining in the resected specimen may be a primary determinant of recurrence, and Forsell, Forsell, and Kahnberg found that odontogenic keratocysts that were completely enucleated recurred significantly less often than those removed piecemeal.'0'" Forsell, Forsell, and Kahnberg, and Meara et al also point out that patients with documented or suspected nevoid basal cell carcinoma syndrome had a higher rate of recurrence than patients without the syndrome."'" As pointed out in the review by Blanas et al, enucleation followed by application of Carnoy solution, and decompression followed by enucleation were associated with low recurrence rates and resulted in significantly less morbidity than resection."[3] The radiograph in Fig. 8-13 shows a large odontogenic keratocyst in which decompression was performed with placement of a polyethylene drain, followed by cystectomy, as the lesion became more surgically manageable.

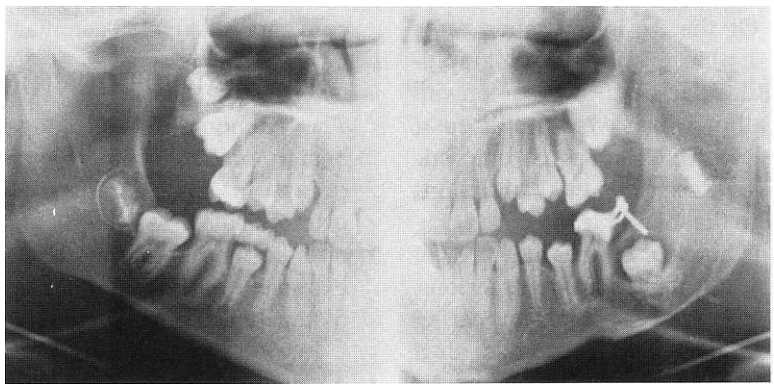
NEVOID BASAL CARCINOMA SYNDROME (GORLIN'S SYNDROME)

The nevoid basal carcinoma syndrome is a trait with autosomal dominant inheritance showing high penetrance and variable expressivity. It is caused by a mutation of the PTCH gene, the human homologue of the patched gene originally identified as a *Drosophila* segment polarity gene, which is thought to be crucial for proper embryonic development and for tumor suppression. Traditionally characterized by a symptom complex of one or more odontogenic keratocysts, basal cell carcinomas of the skin, and a bifidrib, a variety of other abnormalities may accompany this disorder. Patients may have characteristic facies with frontal and parietal bossing with overdevelopment of the supraorbital ridges, hypertelorism, and a broad nasal root and mild mandibular prognathism. Calcification of the falx cerebri is evident radiographically in the majority of adult patients. Milia, epidermoid cysts, chalazia, comedones, and palmar and plantar pits are commonly seen, in addition to the basal carcinomas. Cleft lip and cleft palate and various ocular defects, including blindness, strabismus, and nystagmus, may be present. A variety of benign and malignant neoplasms have been reported to occur, including medulloblastoma, meningioma, ovarian fibroma, and fetal rhabdomyoma and rhabdomyosarcoma. Neurologic abnormalities, including cognitive disabilities, may be noted, and some patients may be hyporesponsive to parathyroid hormone. Skeletal abnormalities may include marfanoid habitus, bifid ribs, spina bifida occulta, and abnormalities of the hands or feet, including the presence of a short fourth metacarpal bone. Because of the association of the odontogenic keratocyst with the basal cell nevus syndrome, all patients with single or multiple odontogenic keratocysts should be evaluated medically with this condition in mind. Also, as previously pointed out, there is an apparent increase in the rate of recurrence of odontogenic keratocysts in patients with the syndrome.

ODONTOGENIC TUMORS

AMELOBLASTOMA

The ameloblastoma is a benign odontogenic neoplasm of ectodermal origin that has traditionally been characterized as persistent and aggressive in nature. Usually an asymptomatic lesion, ameloblastoma may be discovered on routine radiographic evaluation or may show features such as bony expansion, mobility, or divergence



[I G 8-13. Same patient as in Fig. 8-12, 9 months after marsupialization and placement of a drain. Notice shrinkage of the cyst with bone fill in the area. Notice also inferior displacement of the inferior alveolar nerve. (Courtesy Dr. Steven Reubel.)

of teeth. The average age of patients with ameloblastoma is said to be approximately 36 years, with more than 80% of the lesions occurring in the mandible and more than 70% occurring in the molar-ramus area.

At first glance, with the average age of occurrence of ameloblastomas in the fourth decade of life, the discussion of this entity in a pediatric context would not appear to be particularly relevant to this chapter. In 1977, however, Robinson and Martinez described the unicystic ameloblastoma, which they felt to be a prognostically distinct entity.[" In the 20 patients they studied, the average age of occurrence was 21 years, and half of their patients were below the age of 20 years. Clinically and radiographically the lesions were described as having the features of a nonneoplastic cyst, with the majority mimicking dentigerous cysts.

Philipsen and Reichert presented a critical review of 193 cases of unicystic ameloblastoma in the literature.[45] They found that the mean age at the time of diagnosis of unicystic ameloblastoma was most closely related to one clinical feature: the presence of an impacted tooth. They termed cases associated with an impacted tooth the dentigerous type and those not associated with an impacted tooth the nondentigerous type. The mean age at diagnosis of the dentigerous variant was 16.5 years, whereas that of the nondentigerous type was 35.2 years.

Kahn published a clinicopathologic analysis of 38 cases of ameloblastoma (37 intraosseous, 1 peripheral) in individuals younger than 20 years of age."[F] In the 19 years and under age group, the average age at diagnosis was 10.4 years with no obvious sex predilection. All of the 37 intraosseous cases reported in this study occurred in the mandible, with 80% occurring in the molar-ramus area. The most common presenting symptom was swelling, noted in 54% of cases. Sixty-nine percent of cases for which information was available were found to be associated with impacted teeth. In 29% of cases, teeth were displaced. In cases available for review radiographically, 72% of lesions were unilocular with a well-demarcated border and 28% were multi-locular in appearance. Of the 37 intraosseous cases reported in this study, 31 were unicystic (radiographically unilocular or multilocular) and 5 were conventional ameloblastomas, all of which were multilocular radiographically. In a series of 21 cases of unicystic ameloblastoma reported by Rosenstein et al, all lesions were also reported to be in the mandible."

As alluded to previously, ameloblastomas may present as either unilocular or multilocular radiolucent lesions, with or without bony expansion. Cystic-appearing spaces may be compartmentalized by separate, distinct septa of bone. Unicystic ameloblastoma, which is considered a distinct prognostic entity, occurs almost exclusively in the mandible, predominantly in the posterior part. Although most commonly found in association with the crowns of impacted teeth, unicystic ameloblastomas may be seen in interradicular, periapical, or edentulous regions.

Ameloblastomas are distinguished by their histologic resemblance to the enamel organ. Regardless of age or location, ameloblastomas demonstrate singly or in combination a variety of distinct histologic patterns. They are frequently characterized by discrete islands of neoplastic odontogenic epithelium with a peripheral layer of columnar to cuboidal epithelial cells that are palisaded in their appearance with polarization of the nuclei. The central portions of these islands of neoplastic odontogenic epithelium are composed of cells that resemble the stellate reticulum. In some lesions, these central areas may take on an acanthomatous or granular appearance. The plexiform pattern is most frequently encountered in the pediatric age group, with arrangement of tumor cells as a network of interconnecting strands of cells. An important histologic finding in the unicystic form, other than the observation of ameloblastic change in the cyst lining itself, is the presence of a significant luminal and/or mural component characterized by a proliferation of ameloblastic odontogenic epithelium into the lumen of the cyst, the fibrous cyst wall, or both. Mural invasion of the cyst wall portends a higher risk of recurrence for this lesion.

Therapy is by surgical removal, the method of which will vary according to the location and clinical and radiographic extent of the lesion. Although a recurrence rate of 55% to 90% has been found for ameloblastomas of all types that have been treated by curettage, the cystic form, which is the type found most commonly in children, was noted in a study by Gardner and Corio to recur only 11% of the time after the same treatment.[37] After a mean follow-up of 5.2 years, Kahn found recurrence in 7% of cases in which lesions were initially treated by enucleation and/or curettage (cauterized)." It has been recommended, however, that unicystic ameloblastomas with proliferation of epithelium into the fibrous wall of the cyst be treated as aggressively as conventional ameloblastomas.

ADENOMATOID ODONTOGENIC TUMOR

The adenomatoid odontogenic tumor (AOT) is a benign, probably hamartomatous, epithelial tumor that occurs in two intraosseous forms (follicular and extrafollicular) as well as a peripheral variant. It has also been known as adenoameloblastoma, which is extremely misleading because it behaves clinically in a distinctly different fashion than does ameloblastoma.

In 1998 Philipsen and Reichart presented an update on data collected since Philipsen et al reviewed the biologic profile of 499 cases of AOT in 1991, with approximately 250 more cases added since that time.[8,49] The AOT can be subclassified into three variants based

on clinical and radiologic findings. Two of these are central or intraosseous variants. The first is the follicular (dentigerous) type in which the tumor is found in association with the crown of an impacted tooth, with the most likely provisional diagnosis being that of a dentigerous cyst. The second and less commonly reported intraosseous variant of the AOT is the extrafollicular type in which there is no association with the crown of an impacted tooth and with the most likely provisional diagnosis being that of a residual, radicular, globulomaxillary, or lateral periodontal cyst, depending upon its location. The third variant of AOT is the peripheral or extraosseous variant, which may appear clinically as a fibroma. Approximately 95% of AOTs are reported as being the intrabony or central type, with the follicular variant accounting for 71%. Although most often asymptomatic, the tumor frequently causes a painless swelling exhibiting slow but progressive growth.

AOTs occur most commonly in the second decade of life. Nearly 70% of patients are younger than 20 years of age and more than 50% of cases occur in the teenage years. Females are involved twice as often as males, and the intraosseous lesions have been noted to occur nearly twice as frequently in the maxilla as in the mandible, with a noticeable predilection for occurrence in the canine and incisor regions. The rare peripheral type is found almost entirely in the anterior maxillary region.

The most common radiographic finding is that of a unilocular radiolucent lesion, which as previously stated may appear radiographically as a dentigerous cyst (Fig. 8-14) or as a residual, radicular, globulomaxillary, or lateral periodontal cyst, depending on its location. Radiopacities of varying size and density are often present. Because these are space-occupying lesions, divergence of roots and displacement of teeth may be noted.

Histologically the AOT is composed of neoplastic odontogenic epithelium with a distinct histologic appearance: an encapsulated proliferation of swirling strands of spindle-shaped or polygonal epithelial cells within which are nodules of ductlike or rosettelike structures composed of a row of definite cuboidal to columnar epithelial cells; these may be empty or may contain variable amounts of an amorphous eosinophilic material, which may become calcified in some areas.

Because these lesions are in almost all cases well encapsulated and show an identical benign biologic behavior, conservative surgical enucleation and curettage is the treatment of choice. There is no propensity for recurrence.

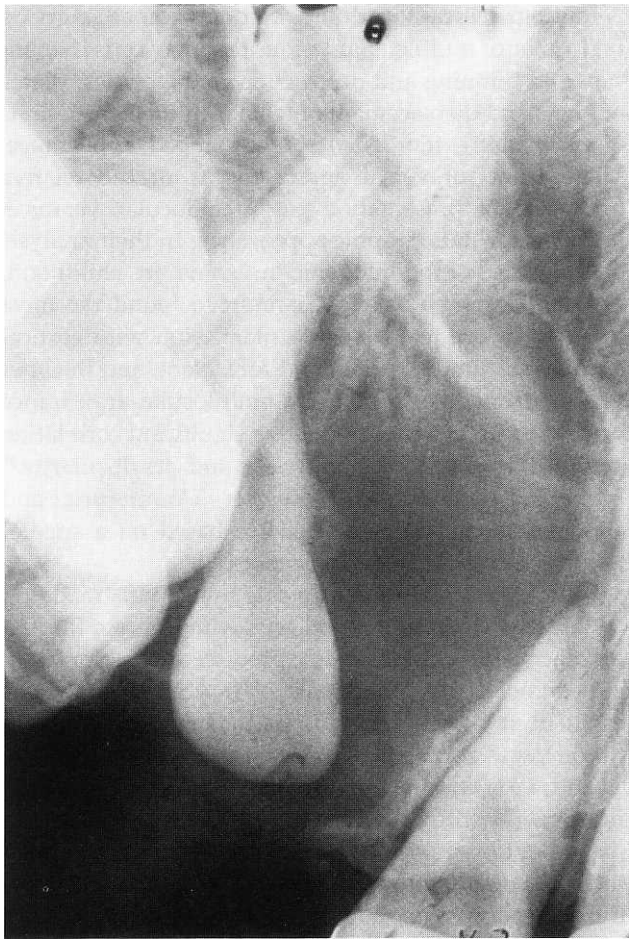


FIG. **8-14.** Adenomatoid odontogenic tumor in a 12-year-old girl presenting as an expansile radiolucent lesion in the right maxilla associated with the lateral incisor. The lesion was curetted, and the lateral incisor was allowed to erupt and was orthodontically moved into proper alignment. (Courtesy Dr. Dan J. Crocker.)

ODONTOGENIC MYXOMA

The odontogenic myxoma is an uncommon benign mesodermal neoplasm of the jaws that is thought to arise from the mesenchymal portion of the odontogenic apparatus. Kaffe, Naor, and Buchner reviewed the English-language literature on odontogenic myxoma published between 1965 and 1995." Their review included 162 cases from the literature and 2 new cases. Two thirds of the cases involved the mandible and one third of the cases involved the maxilla, with the molar and premolar region being the most common site of occurrence. Although the majority of cases were diagnosed in the second to fourth decades, 34% of the cases were in patients 20 years of age or younger.

Several cases have been reported as occurring in association with impacted or missing teeth; however, at least in the case of unerupted teeth, this is an uncommon finding. In addition, several cases have been noted to occur in non–tooth-bearing areas, such as the condyle and the condylar neck. Clinically, these are usually painless lesions that may show mobility and divergence of teeth.

Radiographically, odontogenic myxomas may be unilocular or multilocular lesions that may cause expansion and thinning and destruction of the cortical plates of bone, and displacement of teeth. Multilocular lesions often exhibit a mottled or honeycombed appearance. There is no universal agreement as to the relative frequency of presentation as a unilocular versus a multilocular radiographic appearance. In their analysis of 10 cases of odontogenic myxoma in childhood, Keszler, Dominguez, and Giannunzio found the most frequent image to be a unilocular lesion with cortical expansion." In the review by Kaffe, Naor, and Buchner of cases from all age groups, a multilocular appearance was noted in 55% of cases, with a significant correlation between the size of the lesion and its locularity.[50] Perhaps the findings by Keszler, Dominguez, and Giannunzio can be accounted for based on a smaller lesional size in their pediatric cases.[51]

Histologically, the odontogenic myxoma is made up of stellate to spindle-shaped cells with delicate fibrillar interlacing processes, which produce a loose myxoid appearance. Occasional nests of inactive odontogenic epithelium may be noted, interspersed within this fibromyxoid stroma.

The appropriate treatment for the odontogenic myxoma is still a matter of debate and is certainly dependent on its size and location. The preferred treatment is complete surgical excision, which may prove difficult because of infiltration and expansion of the tumor into bone, and the absence of a true capsule. Treatment is complicated by the understandable reluctance to perform wide surgical excision of a benign lesion, especially in a child. Periodic follow-up to check for recurrence is important. In Keszler, Dominguez, and Giannunzio's series of 10 cases, two patients experienced recurrence within the first year after surgery?'

AMELOBLASTIC FIBROMA

The ameloblastic fibroma is a true mixed neoplasm of odontogenic origin characterized by the proliferation of both odontogenic epithelium and mesenchymal tissue without the formation of enamel or dentin. It is generally believed to be a less aggressive lesion than the ameloblastoma. The average age of occurrence is approximately 14 years with no obvious sex predilection noted. The lesion occurs in the mandible in approximately 70% of cases, primarily in the molar region. Its initial clinical presentation is most often swelling; however, it is not uncommonly asymptomatic, being found on routine radiographic examination. Radiographically, it may be a unilocular or multilocular radiolucent lesion, usually with well-defined, often sclerotic borders, and may be found in association with unerupted or displaced teeth.

The ameloblastic fibroma has a characteristic histologic appearance, with nests and strands of cuboidal to columnar epithelial cells proliferating along with a primitive-appearing mesenchymal component that resembles the developing dental papilla.

Treatment is by surgical removal that is complete but conservative compared with that advocated for the ameloblastoma. At one time, simple surgical excision was advocated, because there was believed to be little chance for recurrence. Recent evidence, however, indicates that the recurrence rate is higher than originally suspected, and somewhat more aggressive surgical therapy is indicated than was previously recommended.

AMELOBLASTIC FIBRO-ODONTOMA

As reviewed by Philipsen, Reichart, and Praetorius, the WHO defines this lesion as "a lesion similar to ameloblastic fibroma, but also showing inductive changes that lead to the formation of both dentine and enamel."[52] Philipsen, Reichart, and Praetorius updated the review by Slootweg, bringing the total number of cases in the literature to 86.[52,53] The average age of recurrence has been reported as 9 years. The majority of cases are found in the posterior mandible, and the ratio of lesions in this location to those in both the anterior and posterior maxilla is 2.4:1. Described as a painless, slow-growing, and expanding tumor, the lesion is associated with unerupted teeth in 83%, of cases, which often leads to its diagnosis. Size may vary from lesions evident only microscopically to a large mass up to 6 cm or more in its greatest dimension.

Histologically the epithelial and mesenchymal components are those of an ameloblastic fibroma, with the formation of osteodentin or dentin-like material and enamel matrix.

Philipsen, Reichart, and Praetorius agreed with Slootweg that the ameloblastic fibro-odontoma is a hamartomatous lesion but also proposed that it is a stage preceding the development of a complex odontoma.52, [53] In his review, Takeda argued that ameloblastic fibro-odontoma should not be considered a hamartoma because rare cases showing true neoplastic behavior exist and because the existence of a malignant variant has been reported." Conservative surgical enucleation is considered to be the treatment of choice, and recurrence is unlikely.

ODONTOMA

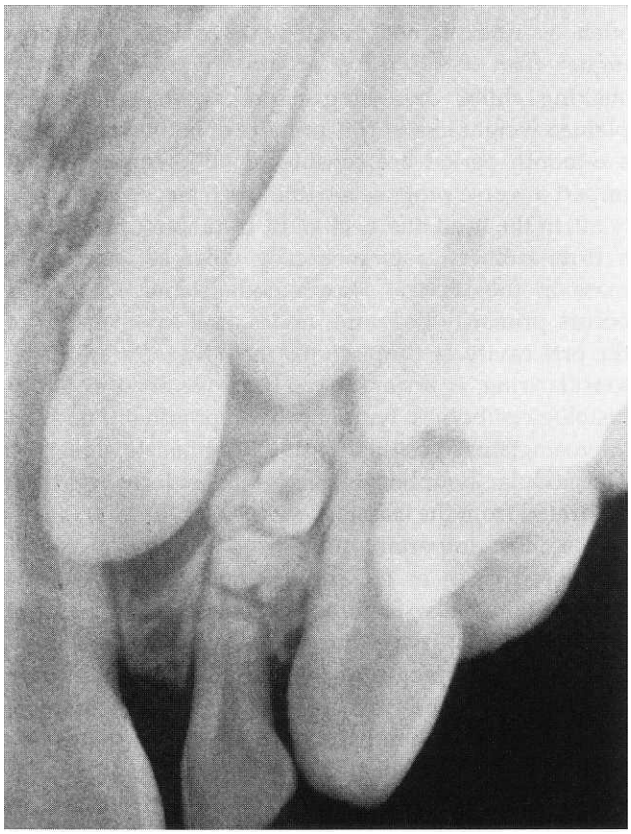
Odontomas are mixed odontogenic tumors in which both the epithelial and mesenchymal components have undergone functional differentiation to the point that both enamel and dentin are formed. The most common of the odontogenic tumors, odontomas are believed to be hamartomatous rather than neoplastic in nature. The WHO has classified odontomas into two types

depending on their degree of morphodifferentiation. The compound odontoma is a lesion in which all the dental tissues are represented in an orderly fashion so that there is at least superficial anatomic resemblance to teeth. In a complex odontoma, on the other hand, although all the dental tissues are represented, they are formed in such a rudimentary fashion that there is little or no morphologic similarity to normal tooth formation (Fig. 8-15).

Budnick, in a review of 65 cases from the literature and 84 from the Emory University School of Dentistry Pathology Department, reported that compound odontomas have a propensity for occurrence in the canine and incisor region, being found more often in the maxilla than in the mandible, whereas complex odontomas show a predilection for occurrence in the posterior jaws." In a review, Philipsen, Reichart, and Praetorius also found the posterior mandible to be the most frequent site of occurrence followed by the anterior maxilla." Compound odontomas have been reported by Slootweg as having a mean age of occurrence of 14.8 years compared to 20.3 years of age for complex odontomas, possibly because the odontogenic tissue in the anterior jaws where the compound odontoma predominantly occurs has finished. its differentiation earlier than tissues in the posterior part of the jaw." Budnick and Kaugars, Miller, and Abbey did not differentiate between clinical types of odontomas; however, they noted a mean age of detection of 14.8 and 16 years of age, respectively."'" Kaugars, Miller, and Abbey have argued that, because the radiographic and histologic distinction between the compound and complex odontomas is poorly defined and because no appreciable clinical difference separates them, differentiation of these two types of odontomas cannot be justified given the obliquity of the diagnostic criteria."' Philipsen, Reichart, and Praetorius take exception to this concept, holding the view that complex and compound odontomas are pathogenetically different."- They believe that complex odontomas are the terminal stage in the series of hamartomatous lesions that they termed the developing complex odontoma line and that includes the ameloblastic fibro-dentinoma, the ameloblastic fibro-odontoma, and what they call the hamartomatous type of ameloblastic fibroma (they argue that ameloblastic fibromas occurring after the age of 20 years represent a benign neoplasm, whereas those occurring during childhood [i.e., during the tooth-development age group] are likely to be nonneoplastic hamartomatous lesions). They feel that the compound odontoma represents a malformation with a high degree of histomorphologic differentiation similar to the process producing supernumerary teeth, "multiple schizodontia," or locally conditioned hyperactivity of the dental lamina.

Although odontomas are usually asymptomatic, they may be the cause of noneruption or impaction of teeth and retained primary teeth (see Fig. 8-15). In the series of cases by Kaugars, Miller, and Abbey, odontomas were found to be in association with an unerupted tooth in 48% of cases and in conjunction with a dentigerous cyst in 28% of cases."

Odontomas are most commonly found on routine radiographic examination, presenting as an irregular radiopaque mass or as small, toothlike structures. The lesions are typically asymptomatic, and the most common presenting symptom is usually lack of eruption of a permanent tooth or bony expansion or swelling. The recommended treatment for an odontoma is conservative surgical excision, with care taken to remove the surrounding soft tissue. No propensity for recurrence has been noted.



[I i **8-15.** Complex composite odontoma delaying eruption of the lateral incisor.

MALIGNANT TUMORS

FIBROMATOSIS AND FIBROSARCOMA IN INFANCY AND CHILDHOOD

As reviewed by Fisher, these neoplasms are part of a large group of benign and malignant lesions that present as subcutaneous or deep tumor masses, often with alarmingly rapid growth.[57] The fibromatoses are

characterized by a proliferation of spindle-shaped or oval bland and uniform cells without pleomorphism or giant cells. They are marked by their propensity for local infiltration and recurrence. Although they may kill by local extension, technically they are benign, because they do not metastasize.

The common or desmoid type of infantile fibromatosis is found from birth to 5 years (usually before 2 years of age) in the shoulder and upper arm, thigh, or head and neck region.[5i] The literature suggests that conservative surgery with the goal of achieving a wide lesion-free margin should be attempted as a primary means of control. Radiation therapy should be used only as a last step in the young patient because of the risk of disturbance of skeletal growth and potential for radiation-induced sarcoma. Chemotherapy may have a role in the treatment of children with inoperable disease.

Fibrosarcoma is a malignant neoplasm of fibroblastic and myofibroblastic differentiation. According to Fisher, congenital and infantile fibrosarcoma is a relatively rare tumor that usually is manifest in the first year of life, especially in the first 3 months, and almost always before 4 years of age.[57] It is distinguished from fibromatosis by cellular pleomorphism, increased mitotic activity, and its potential for metastasis. Less cellular examples may also occur in older children and resemble adult-type desmoids. Fibrosarcoma that occurs in children younger than 4 years of age is much less likely to metastasize than it is in adults and has more in common with infantile aggressive fibromatosis than it does with the adult form of the disease. Although recurrence is possible, metastases are rare, and the majority of cases are cured by wide local excision. Adjuvant radiation therapy and chemotherapy are reserved for inoperable, recurrent, or metastatic cases.

MALIGNANT LYMPHOMA

Malignant lymphomas are neoplasms of lymphoreticular origin that are divided into two main categories: non-Hodgkin's lymphoma (NHL) and Hodgkin's lymphoma (HL, or Hodgkin's disease). As a group, malignant lymphomas represent the most frequently encountered malignant tumors in the head and neck in children and are approximately equally divided in their frequency between HL and NHL.

Hodgkin's Lymphoma (Hodgkin's Disease). Hodgkin's lymphoma (HL) is a malignant neoplasm of lymphoreticular origin distinguished from NHL by diverse but distinctive morphologic features with one common denominator, the presence of Reed-Sternberg cells or their variants, which are widely accepted as the neoplastic cells in this disease. HL is characterized by a bimodal age distribution with the first peak incidence at approximately 25 years of age and the second peak at approximately 65 years of age. Although the first peak incidence is noted in adolescence and young adulthood, HL may be found even in very young children. Males are affected more often than females.

The staging of Hodgkin's disease is anatomically based because of it's propensity to spread contiguously from one lymph node group to the next. The Ann Arbor staging classification was initially reported in 1971 by Carbone et al.[5$] In 1989 the Ann Arbor staging was modified at a meeting in Cotswolds, England.[5"] The Cotswolds modifications validated the use of computed tomographic scanning for the identification of intraabdominal disease, formally introduced bulk of disease into the staging system, and included the concept of unconfirmed complete remission. According to these modifications of the Ann Arbor staging system, stage I HL signifies disease involving one lymph node region or lymphoid structure (e.g., spleen, thymus, Waldeyer ring); stage II signifies involvement of two or more lymph node regions on the same side of the diaphragm, most often in the neck and mediastinum; stage III indicates disease on both sides of the diaphragm (III1 with or without involvement of splenic, hilar, celiac, or portal nodes [upper abdomen] and I1I2 with involvement of paraaortic, iliac, and mesenteric nodes [lower abdomen]); and stage IV indicates the presence of involvement of extralymphatic sites, most often the liver and/or lungs, bone marrow, or bone. Symptoms such as unexplained fever with oral temperatures higher than 38° C for 3 or more consecutive days, shaking chills, drenching night sweats, and unexplained weight loss of 10% or more of body weight over a 6-month period are considered "B" symptoms that impart a worse prognosis within each stage.

HL in the head and neck most commonly presents as a firm, rubbery, asymmetrically enlarged, nontender mass in the cervical lymph node chain. The lesion occurs primarily in lymph nodes, and involvement of the oral cavity or oropharynx, including the Waldeyer tonsillar ring, is uncommon. HL is classified into four histologic subtypes: lymphocyte predominant, nodular sclerosing, mixed cellularity, and lymphocyte depletion. The nodular lymphocyte–predominant form has been separated from the classic disease.

The most important prognostic variable in newly diagnosed cases of HL is the clinical stage, and the strongest predictive factor for patients with relapse after initial treatment is the duration of the initial response. Most treatment programs for limited-stage HL include chemotherapy and involved-field radiation therapy. With advances in therapy, current 5-year recurrence-free survival has been achieved by *95%* or more of patients with stage I or II nonbulky disease and absence of systemic B symptoms (mediastinal lymphadenopathy is considered bulky). Five-year survival rates for patients with bulky disease, B symptoms, and/or advanced

stage III or IV disease are between 80% and 94%. For patients who have experienced relapse after initial combined-modality therapy and those who experience multiple relapses, hematopoietic stem cell transplantation may offer hope of long-term remission.

Non-Hodgkin's Lymphomas. The NHLs are a heterogenous group of neoplasms of the lymphoid system that may arise in the head and neck in the lymph nodes (nodal), in extranodal lymphatic areas (the Waldeyer tonsillar ring), or in extranodal, extralymphatic tissue, such as the mandible, salivary glands, pharynx, deep fascial spaces, paranasal sinuses, the orbit, and other areas. As pointed out by Miller, Young, and Novakovic, Wright, McKeever, and Carter, and Skarin and Dorfman, the classification of NHL has changed substantially in the past 25 years."[0-"z] Childhood NHL differs from adult NHL in that the majority of childhood cases are high grade, demonstrate a diffuse growth pattern, and commonly involve extranodal sites.

The majority of cases fall into one of three types: small, noncleaved cell lymphoma, Burkitt's type (some authors use the terms Burkitt's lymphoma and small, noncleaved lymphoma synonymously; some use Burkitt's lymphoma and non—Burkitt's lymphoma as subtypes of small, noncleaved cell lymphoma; and others speak of the two as "related" entities); lymphoblastic lymphoma; and anaplastic large cell lymphoma. The separation of each of these can be made on the basis of morphologic and immunohistochemical features. Unlike HL, which spreads to contiguous lymph node groups, NHL spreads in an unpredictable fashion, so that most patients present with stage III or IV disease.

Ribeiro et al studied 87 consecutively treated children with NHL arising in the head and neck region.[63] Thirty-one children had primary nodal NHL, whereas 56 had extranodal NHL. Their data indicate that, unlike adults, children whose primary head and neck NHLs were extranodal had a significantly worse treatment outcome than those whose primary tumors were nodal. Children whose tumors were extranodal and extralymphatic had an even worse outcome than the others. Owing to the anatomical sites of origin of NHL in childhood, the majority of patients do not present with peripheral lymphadenopathy. When lymphadenopathy is present, it may appear as a firm, nontender swelling.

The use of effective risk-adapted multiagent chemotherapy along with improvements in supportive care have resulted in marked improvements in cure rates for all stages and subtypes of NHL in children, with event-free survival ranging from 70% to 90% depending on the stage and subtype of disease. Stage of disease and tumor burden are now used to select the intensity and length of therapy rather than serving as a major indicator of outlook for survival.

RHABDOMYOSARCOMA

Rhabdomyosarcoma, a malignant neoplasm of skeletal muscle origin, is the most common soft tissue sarcoma in children, accounting for more than half of all such lesions occurring in childhood. There are two key age ranges for the occurrence of rhabdomyosarcoma in children: 2 to 6 years of age and adolescence. According to Miser and Pizzo, the early peak is due primarily to occurrences in the head and neck region and genitourinary tract."}

Three histologic subtypes are recognized: embryonal and its botryoid variant, alveolar, and pleomorphic. The embryonal subtype accounts for most rhabdomyosarcomas in infancy and childhood that occur within the deep soft tissues or along mucosal surfaces.

The most common clinical finding is a mass occurring in any region of the head and neck where striated muscle or its mesenchymal progenitor cells exist. There are three primary sites of involvement for rhabdomyosarcoma in the head and neck in children: the eyelid and orbit, parapharyngeal sites, and remaining head and neck sites. The parapharyngeal sites include the pterygopalatine and infratemporal fossa, nasal cavity, nasopharynx, paranasal sinuses, and middle ear and mastoid. Orally, the tonsil and soft palate, tongue, and cheek are most frequently involved. Typically a rapidly growing, often nonulcerated soft tissue mass, rhabdomyosarcoma typically metastasizes by hematogenous routes to lungs, bone, and brain but may also disseminate via the lymphatics or by direct extension.

As discussed by Kraus, Saenz, and Gallamudi, rhabdomyosarcomas arising in the head and neck region are characterized by a wide variation in survival rate depending on the site, with orbital lesions associated the highest survival rates of any site in the body and parapharyngeal tumors having a worse prognosis." Age, sex, resectability, and tumor-node-metastasis (TNM) stage are also important prognostic variables. In their review, Simon et al pointed out that children 11 years of age or younger with a tumor 5 cm or smaller had the best survival, whereas patients older than 11 years with a tumor larger than 5 cm had the worst survival outcome.[bb] A multimodality approach to therapy consisting of multiagent chemotherapy, surgery, and external-beam radiotherapy has improved survival. In the study by Kraus, Saenz, and Gallamudi, overall 5-year survival was 74%, with each variable contributing to the TNM stage being statistically significant in determining survival.[65]

OSTEOGENIC SARCOMA

Osteogenic sarcoma is an uncommon, highly malignant, primary neoplasm of bone with a soft tissue counterpart with a similar histomorphology. As pointed out by

Meyers and Gorlick, its peak age of occurrence is the second decade of life, with the modal age of incidence being 16 years for girls and 18 for boys."' The most common site of involvement is the distal femur followed by the proximal tibia and proximal humerus.

Osteogenic sarcoma involving the jaws accounts for approximately 6.5% of all cases of this disease. A review of 56 cases of osteogenic sarcoma of the jaws by Garrington et al showed a median age of occurrence of 27 years, with 34% of mandibular cases but only 12% of maxillary cases occurring before 20 years of age.[68] The neoplasm was noted to occur approximately twice as frequently in the mandible as in the maxilla, with swelling and pain being the most frequently described early symptoms. A review of 66 cases of osteogenic sarcoma of the jaws by Clark et al concurred that swelling and pain are the most frequent presenting complaints.[69] However, they found approximately equal involvement in the mandible and maxilla, and listed the mandible, maxillary antrum, and alveolar ridge of the maxilla as the most frequent sites of occurrence. More recently, Gadwal et al presented a clinicopathologic study of 22 cases of osteosarcoma of the head and neck along with a review of the literature.[i0] They found the primary occurrence of pediatric osteogenic sarcoma in the head and neck not associated with previous radiation therapy or an unknown syndrome to be rare. There was no sex predilection in their patients aged 1 to 18 years (mean age, 12.2 years). The primary symptoms were painless swelling, loss of teeth, headaches, or a mass lesion, with an average duration of symptoms of 5.9 months. Nineteen cases (86%) occurred in the mandible, two cases in the sphenoid sinus, and one in the maxilla. These researchers found that, despite indications of invasiveness and malignancy by radiologic and/or histologic examination, and even with multiple local recurrences, these tumors tended to behave as low-grade neoplasms, and long-term prognosis was good.

The radiographic findings are typically those of a poorly defined, bone-destructive lesion suggestive of malignancy. It may be osteoblastic or osteolytic or may have a mixed radiographic appearance. Clark et al have reported that most of the lesions on the maxilla were osteoblastic, whereas most of those on the mandible were osteolytic."" A frequently described radiographic feature is a "sun-ray" appearance with delicate, hairlike trabeculae radiating in a sunburst fashion away from the peripheral surface of the lesion. This has been reported to occur in approximately 25% of cases. An additional radiographic finding in early osteogenic sarcoma of the jaws is symmetrical widening of the periodontal ligament space around one or more of the teeth in the area of the lesion.

Histologically the osteogenic sarcoma is characterized by the production of tumor osteoid forming a malignant stroma that often appears fibroblastic. These tumors are typically classified histologically into osteoblastic, chondroblastic, or fibroblastic subtypes according to the dominant histologic pattern. In the cases of primary osteosarcoma of the head and neck reported by Gadwal et al were osteoblastic osteosarcomas except one, which was a chondroblastic subtype.[70]

Although almost all patients with osteosarcoma of the long bones have at least microscopic metastatic disease at the time of diagnosis, the review by Mardinger et al of osteosarcoma of the jaw in all age groups found the incidence of metastatic disease at the time of diagnosis to be no higher than 18%.[71] Gadwal et al noted no metastases in their series of pediatric patients.[7"] It would appear from the literature that osteosarcoma in the head and neck in pediatric patients differs from osteosarcoma of the long bones in its biologic behavior despite having the same histologic appearance. Complete surgical resection is the treatment of choice followed by appropriate adjuvant therapy for osteosarcoma.

EWING'S SARCOMA

After osteosarcoma, the Ewing's sarcoma family of tumors (Ewing's sarcoma along with peripheral primitive neuroectodermal tumor [pPNET]) is the second most common primary malignancy of bone in children and adolescence. Ewing's sarcoma of bone and soft tissue, as well as pPNET, are small round cell tumors, the genetic hallmarks of which are the presence of a translocation of chromosomes 11 and 22 (t[11;22] [g24; g12]) and in most cases overexpression of the MIC2 gene surface marker (CD99), which differentiates them from the majority of other small round cell tumors. Lombart-Bosch et al in their review point out that the histologic and electron microscopic features of Ewing's sarcoma and pPNET, as well as expression of antigen of the MIC2 gene, provide the basis for characterizing them as neuroectodermally derived neoplasms.[72]

Siegal et al have reported on primary Ewing's sarcoma involving the bones of the head and neck for the Intergroup Ewing's Sarcoma Study." Their data indicated that approximately 1% to 4% of primary cases of Ewing's sarcoma arising in bone occurred in the bones of the head and neck, with the skull being the most frequent site of involvement and the mandible being the most commonly involved gnathic bone. Vaccani et al reported their experience with 70 consecutively presenting cases of Ewing's sarcoma, of which 5 (7.1%) involved the head and neck." One of their cases, however, was metastatic to the mandible. The age of their patients at presentation was 7.5 to 14 years, and an enlarging mass in the mandible was the mode of presentation in three of five patients. Wood et al reported a case and reviewed the literature on Ewing's

sarcoma involving the jawbones.[75] Their review of 105 cases showed the average age at diagnosis to be 15.9 years with an age range of 2 to 44 years at presentation. There was a slight predilection for occurrence in males, with a male-to-female ratio of 1.5:1. The mandible was involved more than twice as often as the maxilla, with a predilection of 4:1 for occurrence in the posterior aspect of the jaws over other aspects.

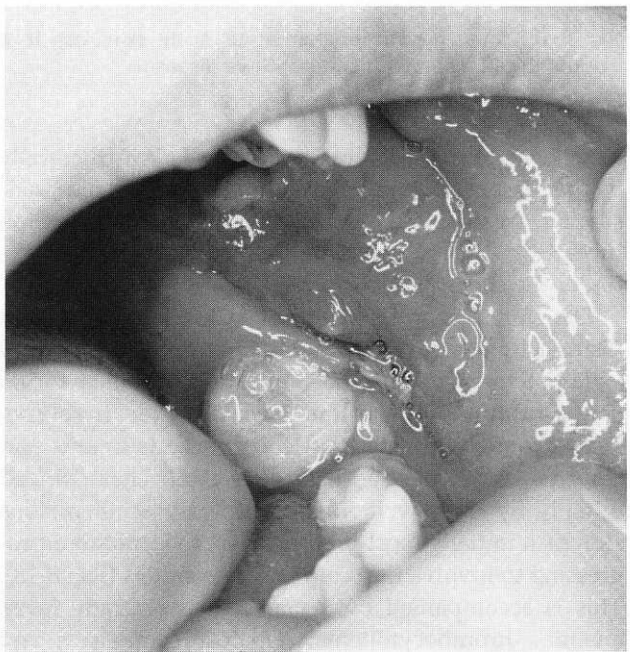
Raney et al reported on 130 patients with Ewing's sarcoma of soft tissue in childhood.[76] Fifty-four percent were boys, and 33% of cases involved the head and neck. Siebenrock et al (1996) compared 21 patients with soft tissue or extraskeletal Ewing's sarcoma with 13 patients with pPNET.[77] There were no significant differences in patient age, gender, tumor location, or stage at presentation between patients with Ewing's sarcoma and those with pPNET of the soft tissue.

Localized swelling and pain are the most frequent complaints at the time of presentation, although loose teeth may also be a presenting symptom. The soft tissue overlying the lesion may be erythematous and warm to the touch, and thus more suggestive of an inflammatory process than a neoplasm (Fig. 8-16). In addition, the patient may have fever, elevated erythrocyte sedimentation rate, increased serum lactic dehydrogenase level, anemia, and leukocytosis.

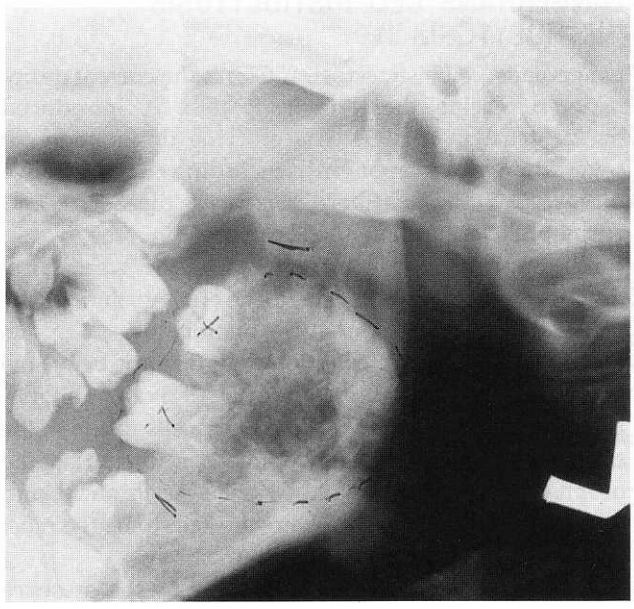
The radiographic features are those of a diffuse bone-destructive lesion, appearing as an irregular, somewhat mottled, radiolucent lesion that may resemble an osteomyelitis (Fig. 8-17). Although reduplication or lamination of the periosteum has been considered a common radiologic sign of Ewing's sarcoma, the review by Wood et al reported that, of 86 cases in the literature for which the radiographic features were presented, only one showed true periosteal lamination and two others showed periosteal lamination with "sun-ray" spicules.[75] Eggli et al pointed out that computed tomography and magnetic resonance imaging are invaluable in further delineating the extent of disease not readily visible on plain radiographs." Gallium scintigraphy and gadolinium-enhanced magnetic resonance images are said to be best for following the response to therapy.

Histologically, Ewing's sarcoma appears as sheets of cells with small, dark-staining nuclei and poorly defined cytoplasmic outlines. Mitotic figures are prominent, and necrosis is a common feature. Sheets of cells may be separated by vascular connective tissue septa. Intracytoplasmic glycogen is present, and positive results on periodic acid–Schiff staining are an essential finding unless there is documented glycogen content on electron microscopy. Detection of MIC2 antigens along with the use of molecular techniques to detect the t(11; 22) (q24;q12) translocation have generally improved the ability to diagnose these tumors.

In the past, Ewing's sarcoma as a whole has been associated with an almost uniformly poor prognosis,



f I **G 8-16.** Radiograph for a 6-year-old boy with Ewing's sarcoma who had a 1-month history of swelling on the left side of his face. Clinically there was swelling with mucosal ulceration in the mucobuccal fold in the first molar area. *(Courtesy Drs. Richard L. Miller and William Epstein.)*



E **I** G **8-17.** Radiograph of the same patient as in Fig. 8-16 with Ewing's sarcoma involving the left angle of the mandible. Notice the mottled radiolucent appearance. *(Courtesy Drs. Richard L. Miller and William Epstein.)*

with nearly all patients having micrometastatic disease at the time of diagnosis. In the Intergroup Ewing's Sarcoma Study as reported by Siegal et al, the prognosis for head and neck Ewing's sarcoma was noted to be significantly better than that for Ewing's sarcoma overall.[73] Of their patients who had been followed in the study for more than 3 years, 80% were alive and well without known progressive recurrent or metastatic disease. Of the 10 patients who had survived 5 years or longer, none had subsequently died, and of the 5 patients in their study who had died, none had gnathic involvement.

Therapy consists of systemic multiagent chemotherapy along with local control, which may consist of surgery, radiation therapy, or a combination of the two, depending on the age of the patient, the location of the primary tumor, and the functional consequences of therapy. Because of the potential for postradiation sarcomas, particularly bone sarcoma in the field of radiation treatment, the importance of local control of the disease with surgery and safe local control with radiation is now emphasized. Patients with clinically detectable metastases at the time of diagnosis and those with relapse after initial therapy have a significantly poorer prognosis. As pointed out by Rodriguez-Galindo, Spunt, and Pappo in their overview, chemotherapy dose intensification and the use of megatherapy with autologous hematopoietic stem cell transplantation are routinely used at a number of treatment centers for these high-risk patients.[7"]

LANGERHANS' CELL HISTIOCYTOSIS (HISTIOCYTOSIS X)

Langerhans' cell histiocytosis (LCH) is the current designation replacing the term *histiocytosis X* introduced by Lichtenstein in 1953 as a unifying designation for several previous eponyms, including Letterer-Siwe disease, Hand-Schuller-Christian disease, and eosinophilic granuloma (LCH of bone).[80]

Although clinical forms of LCH were first described at least a century ago, the etiology and pathogenesis of Langerhans' cell histiocytosis remains obscure, with the one common denominator being the Langerhans' cell, a bone marrow—derived, antigen-presenting dendritic cell. Langerhans' cells reside in the skin, thymus, and mucosal epithelium, including the oropharynx and nasopharynx, esophagus, bronchi, and cervix and, as reviewed by Lam, are said to be the most potent antigen-presenting cells in the body.[3']

Histologically, LCH is characterized by the presence of uniform sheets of large round Langerhans' histiocytes with a homogenous pink cytoplasm when stained with hematoxylin and eosin, interspersed with variable numbers of eosinophils, lymphocytes, plasma cells, and multinucleated giant cells, particularly in lesions of bone. Langerhans' cells are characterized ultrastructurally by the presence of Langerhans' or Birbeck granules, rod-shaped organelles that may have a vesicular portion imparting the so-called tennis racquet appearance under the electron microscope.

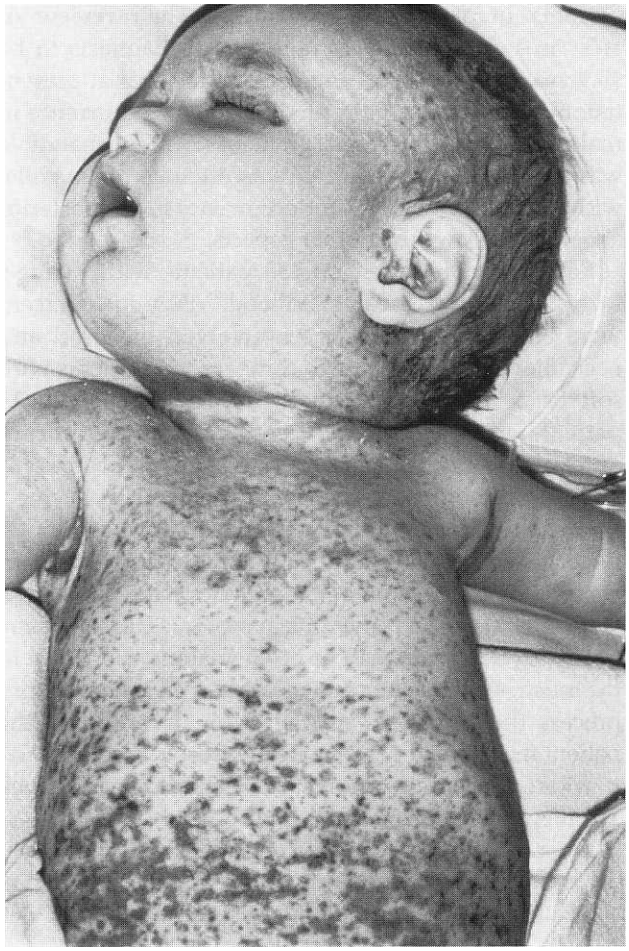


FIG . **8-18.** Characteristic erythematous scaly skin rash in a 5 1/2-month-old infant with Letterer-Siwe disease.

The clinical manifestations of LCH are variable and historically were divided into distinct entities. The term *Letterer-Siwe disease* has been used as a moniker for an acute fulminating proliferative disorder involving Langerhans' histiocytes that chiefly affects infants and children under the age of 3. It is often characterized initially by the development of a scaly erythematous skin rash initially most prominent on the trunk but progressing to involve the scalp and extremities (Fig. 8-18). This is accompanied by a persistent low-grade fever, anemia, thrombocytopenia, hepatosplenomegaly, and lymphadenopathy.

Bony involvement, indistinguishable from that found in Hand-Schuller-Christian disease, and eosinophilic granuloma of bone may be noted (Fig. 8-19). Oral lesions with pain, swelling, ulceration, gingival necrosis, and

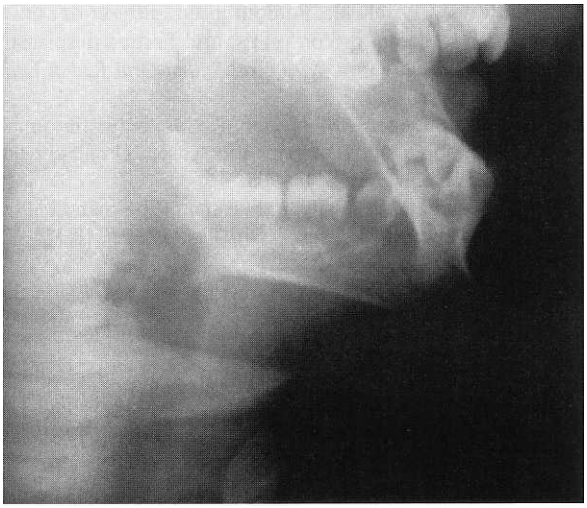
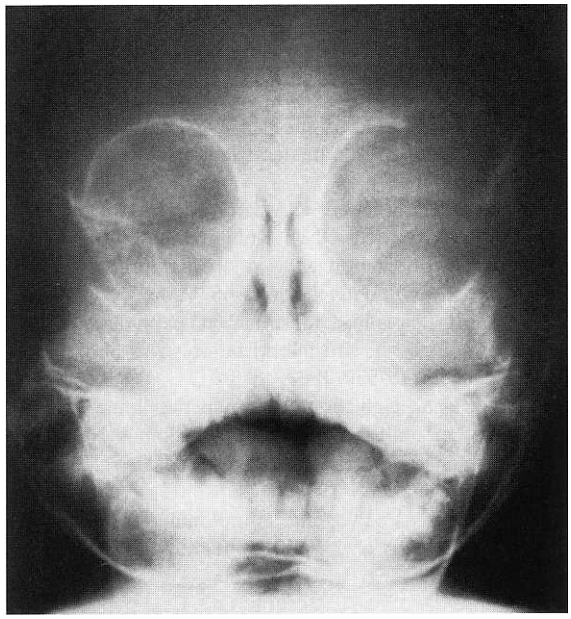


FIG **8-19.** Diffuse destruction of maxillary and mandibular alveolar bone around the developing primary teeth in the same patient as in Fig. 8-18. Notice also destruction of the left angle and ramus of the mandible.



I I I **8-20.** Radiograph for a 12-month-old girl with Hand-Schuller-Christian disease. There is an expansile, radiolucent lesion in the right body of the mandible as well as destruction of the greater wing of the sphenoid and supraorbital rim on the left.

destruction of alveolar bone with premature exfoliation of the teeth may be an early manifestation of Letterer-Siwe disease. Although multiagent chemotherapy may result in regression of the disease in some cases, patients with this form of the disease have a relatively poor prognosis.

The term *Hand-Schuller-Christian disease* has been used to describe the chronic, disseminated form of LCH, which is characterized by the development of multifocal eosinophilic granulomas of bone, lymphadenopathy, and visceral involvement, especially hepatosplenomegaly (Fig. 8-20). The classical clinical triad that has often been described for this disease is the occurrence of punched-out—appearing radiolucent defects in membranous bones, exophthalmos, and diabetes insipidus. Chronic otitis media is also a frequent symptom. Although usually diagnosed early in the first decade of life, Hand-Schiiller-Christian disease has been reported by McDonald et al to occur as late as the sixth decade.[8]

The treatment varies according to the extent of the disease, with surgical curettage or radiation therapy being used to treat focal disease. Multiagent chemotherapy has proved to be relatively successful in the long-term control of disseminated disease. *Eosinophilic granuloma* is the most common and also least severe form of LCH. It is characterized by single or multiple usually well-defined radiolucent bony lesions, most often accompanied by pain and swelling. Older children and young adults most commonly display this form of the disease. In addition to the mandible and skull, the femur, humerus, ribs, and pelvis are also frequently involved. The maxilla is involved less frequently than the mandible, with the midposterior mandible being the most common site of occurrence. Skin and visceral involvement such as that described for Hand-Schuller-Christian and Letterer-Siwe diseases are absent. A variety of options exist for patients with this form of the disease. After confirmation of the diagnosis by biopsy, the lesion may be left alone for observation, it may be surgically curetted or excised, intralesional injections of corticosteroids may be given, or low-dose radiotherapy may be used. As noted in the review by Ardekian et al, the recurrence rate of eosinophilic granuloma ranges from 1.6% to 25%.[83] Their study was the first to use a standardized treatment regimen of surgery and radiation therapy. A 7.3% recurrence rate was reported.

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CHAPTER 4

Eruption of the Teeth: Local, Systemic, and Congenital Factors That Influence the Process

RALPH E. McDONALD
DAVID R. AVERY
JEFFREY A. DEAN

انتشارات
شایان نمودار

CHAPTER OUTLINE

CHRONOLOGIC DEVELOPMENT AND ERUPTION OF THE TEETH
- Influence of Premature Loss of Primary Molars on Eruption Time of Their Successors
- Variations in the Sequence of Eruption

LINGUAL ERUPTION OF MANDIBULAR PERMANENT INCISORS

TEETHING AND DIFFICULT ERUPTION
- Eruption Hematoma (Eruption Cyst)
- Eruption Sequestrum
- Ectopic Eruption

NATAL AND NEONATAL TEETH

EPSTEIN PEARLS, BOHN NODULES, AND DENTAL LAMINA CYSTS

LOCAL AND SYSTEMIC FACTORS THAT INFLUENCE ERUPTION
- Ankylosed Teeth
- Ankylosis of Primary Molars with Absence of Permanent Successors
- Ankylosed Permanent Teeth
- Trisomy 21 (Down Syndrome [DS])
- Cleidocranial Dysplasia (CCD)
- Hypothyroidism
  - *Congenital hypothyroidism (cretinism)*
  - *Juvenile hypothyroidism (acquired hypothyroidism)*
- Hypopituitarism
- Achondroplastic Dwarfism
- Other Causes

CHRONOLOGIC DEVELOPMENT AND ERUPTION OF THE TEETH

A variety of developmental defects that are evident after eruption of the primary and permanent teeth can be related to systemic and local factors that influence matrix formation and the calcification process. Thus it is important that the dentist be able to explain to the parents the time factors related to the early stages of tooth calcification both in utero and during infancy.

Lunt and Law made a careful review of the literature on the calcification of the primary teeth.' They compared their findings with the values in Table 9-1 showing Logan and Kronfeld's chronology of human dentition, which has been an accepted standard for many years.' They offered a revised table that establishes earlier ages than those previously accepted for initial calcification (Table 9-2). A similar review was carried out for the ages at which the primary teeth erupt.[3]

Lunt and Law concluded after reviewing the work of Kraus and Jordan (1965) and of Nomata (1964) that Table 9-1 should be modified.[s] The sequence of calcification of the primary teeth should be changed to central incisor, first molar, lateral incisor, canine, and second molar. They determined that the times of initial calcification of the primary teeth are 2 to 6 weeks earlier than those given in Table 9-1. They also concluded that the maxillary teeth are generally ahead of the mandibular teeth in development. Exceptions are the second molars, which generally are advanced in the mandible, and the lateral incisors and canines, which at times may be ahead in the mandible.

Lunt and Law also believe that the lateral incisor, first molar, and canine tend to erupt earlier in the maxilla than in the mandible; the Logan and Kronfeld table suggests that eruption in the mandible is generally ahead of that in the maxilla. The ages at which primary teeth erupt are 2 months or more later than suggested in the Logan and Kronfeld table.

It should be remembered that the time of eruption of both primary and permanent teeth varies greatly. Variations of 6 months on either side of the usual eruption date may be considered normal for a given child. Interestingly, a study by Parner et al compared the well-known general acceleration of the physical development of children over the past century with their own observations of the emergence of permanent teeth." They found that the emergence of permanent teeth has not been subject to a similar acceleration; in fact, the mean age of eruption has increased slightly, but only by a few days per year. They conclude that the age of eruption of the permanent teeth is a much more stable phenomenon than other aspects of physical development of children.

Numerous in vivo animal experiments and human radiographic studies have been done to better understand the process of tooth eruption. Although many theories have been advanced, the factors responsible for the eruption of the teeth are not fully understood. The factors that have been related to the eruption of teeth include elongation of the root, forces exerted by the vascular tissues around and beneath the root, growth of the alveolar bone, growth of dentin, growth and pull of the periodontal membrane, hormonal influences, presence of a viable dental follicle, pressure from the muscular action, and resorption of the alveolar crest.

A series of experiments by Cahill and Marks have established that a viable dental follicle is required for tooth eruption.' Further studies by Marks and Cahill resulted in the conclusion that "tooth eruption is a series of metabolic events in alveolar bone characterized by bone resorption and formation on opposite sides of the dental follicle and the tooth does not contribute to this process."'

Baume, Becks, and Evans reported evidence that tooth eruption is influenced by pituitary growth hormone and thyroid hormone.`" Philbrick et al have demonstrated that parathyroid hormone—related protein is required for tooth eruption.[10]

Shumaker and El Hadary observed in a radiographic study that each tooth starts to move toward occlusion at approximately the time of crown completion." The interval from crown completion and the beginning of eruption until the tooth is in full occlusion is approxi-mately 5 years for permanent teeth.

Cron observed in her study of 874 Boston children that tooth emergence appeared to be more closely associated with the stage of root formation than with the chronologic or skeletal age of the child."-- By the time of clinical emergence approximately three fourths of root formation had occurred. Teeth reach occlusion before the root development is complete.

Demirjian and Levesque presented a large sample of 5437 radiographs from a homogeneous (French-Canadian) population." They used this sample to investigate the sexual differences in the development of permanent mandibular teeth from the early stages of calcification to closure of the apex. The analysis of the developmental curves of individual teeth shows a common pattern, namely, the similarity in timing between the sexes for the early stages of development. For the first stages of crown formation, which they refer to as A, B, and C, there was no difference between boys and girls in the chronology of the dental calcification in the majority of teeth. For the fourth stage, D, which represents the completion of crown development, girls were more advanced than boys by an average of 0.35 year for four teeth. For the stages of root development the mean difference between the sexes for all teeth was 0. 54 year; the largest difference was for the canine

TABLE 9-1 Chronology of the Human Dentition

TOOTH	HARD TISSUE FORMATION BEGINS	AMOUNT OF ENAMEL FORMED AT BIRTH	ENAMEL COMPLETED	ERUPTION	ROOT COMPLETED
DECIDUOUS DENTITION					
Maxillary					
Central incisor	4 mo in utero	Five sixths	1 1/2 mo	7 1/2 mo	1 1/2 yr
Lateral incisor	4 1/2 mo in utero	Two thirds	2 1/2 mo	9 mo	2 yr
Cuspid	5 mo in utero	One third	9 mo	18 mo	3 1/4 yr
First molar	5 mo in utero	Cusps united	6 mo	14 mo	2 1/2 yr
Second molar	6 mo in utero	Cusp tips still isolated	11 mo	24 mo	3 yr
Mandibular					
Central incisor	4 1/2 mo in utero	Three fifths	2 1/2 mo	6 mo	1 1/2 yr
Lateral incisor	4 1/2 mo in utero	Three fifths	3 mo	7 mo	1 1/2 yr
Cuspid	5 mo in utero	One third	9 mo	16 mo	3 1/4 yr
First molar	5 mo in utero	Cusps united	5 1/2 mo	12 mo	2 1/4 yr
Second molar	6 mo in utero	Cusp tips still isolated	10 mo	20 mo	3 yr
PERMANENT DENTITION					
Maxillary					
Central incisor	3-4 mo		4-5 yr	7-8 yr	10 yr
Lateral incisor	10-12 mo		4-5 yr	8-9 yr	11 yr
Cuspid	4-5 mo		6-7 yr	11-12 yr	13-15 yr
First bicuspid	1 1/2-1 3/4 yr		5-6 yr	10-11 yr	12-13 yr
Second bicuspid	2-2 1/4 yr		6-7 yr	10-12 yr	12-14 yr
First molar	At birth	Sometimes a trace	2 1/2-3 yr	6-7 yr	9-10 yr
Second molar	2 1/2-3 yr		7-8 yr	12-13 yr	14-16 yr
Third molar	7-9 yr		12-16 yr	17-21 yr	18-25 yr
Mandibular					
Central incisor	3-4 mo		4-5 yr	6-7 yr	9 yr
Lateral incisor	3-4 mo		4-5 yr	7-8 yr	10 yr
Cuspid	4-5 mo		6-7 yr	9-10 yr	12-14 yr
First bicuspid	1 3/4-2 yr		5-6 yr	10-12 yr	12-13 yr
Second bicuspid	2 1/4-2 1/2 yr		6-7 yr	11-12 yr	13-14 yr
First molar	At birth	Sometimes a trace	2 1/2-3 yr	6-7 yr	9-10 yr
Second molar	2 1/2-3 yr		7-8 yr	11-13 yr	14-15 yr
Third molar	8-10 yr		12-16 yr	17-21 yr	18-25 yr

From Kronfeld R: *Bur* 35:18-25, 1935 (based on research by WHG Logan and R Kronfeld); adapted by Kronfeld R, Schour I: *J ihn Dent Assoc 26:* 18-32, 1939; further adapted by McCall JO, Wald SS: *Clinical dental roentgenology: technic and interpretation including roentgen studies of the child and young adult,* Philadelphia, 1940, WB Saunders.

(0.90 year). The data from Demirjian and Levesque show the importance of sexual dimorphism during the period of root development rather than during the period of crown development.[13]

The tooth eruption process is clearly quite complex, and many different mechanisms are undoubtedly involved. Some of the leading scientists who are contributing to a better understanding of the tooth eruption process have written review articles to help consolidate the facts and theories associated with this process. A review article by Wise et al focuses on the molecular signals that initiate tooth eruption.[14] These researchers state that tooth eruption is a complex and tightly regulated process that involves cells of the tooth organ and the surrounding alveolus. Mononuclear cells (osteoclast precursors) must be recruited into the dental follicle prior to the onset of eruption. These cells, in turn, fuse to form osteoclasts that resorb alveolar bone, creating an eruption pathway for the tooth to exit its bony crypt. In recent years, knowledge of the biology of tooth eruption has greatly increased. What has emerged is the realization that interactions of osteoblasts, osteoclasts, and dental follicles involve a complex interplay of regulatory genes that encode various transcription

TABLE 9-2 Modification of the Table "Chronology of the Human Dentition" Suggested by Lunt and Law for the Calcification and Eruption of the Primary Dentition

TOOTH	HARD TISSUE FORMATION BEGINS	AMOUNT OF ENAMEL FORMED AT BIRTH	ENAMEL COMPLETED	ERUPTION (±1 SD)	ROOT COMPLETED
DECIDUOUS DENTITION					
Maxillary					
Central incisor	14 (13-16) wk in utero	Five sixths	1 1/2 mo	10(8-12) mo	1 1/2 yr
Lateral incisor	16 (14 2/3-16 1/2) wk in utero	Two thirds	2 1/2 mo	11(9-13) mo	2 yr
Canine	17 (15-18) wk in utero	One third	9 mo	19(16-22) ma	3 1/4 yr
First molar	15 1/2 (14 1/2-17) wk in utero	Cusps united; occlusal completely calcified plus half to three fourths crown height	6 mo	16(13-19) mo boys, 16 (14-18) mo girls	2 1/2 yr
Second molar	19 (16-23 1/2) wk in utero	Cusps united; occlusal incompletely calcified; calcified tissue covers one fifth to one fourth crown height	11 mo	29(25-33) mo	3 yr
Mandibular					
Central incisor	14 (13-16) wk in utero	Three fifths	2 1/2 mo	8 (6-10) mo	1 1/2 yr
Lateral incisor	16 (14 2/3-) wk in utero	Three fifths	3 mo	13 (10-16) mo	1 1/2 yr
Canine	17 (16-) wk in utero	One third	9 mo	20 (17-23) mo	3 1/4 yr
First molar	15 1/2 (14 1/2-17) wk in utero	Cusps united; occlusal completely calcified	5 1/2 mo	16 (14-18) mo	2 1/4 yr
Second molar	18 (17-19 1/2) wk in utero	Cusps united; occlusal incompletely calcified	10 mo	27 (23-31) mo boys, 27 (24-30) mo girls	3 yr

From Lunt RC, Law DB: *J Am Dent Assoc 89:8720-879,* 1974.
SD, Standard deviation.

factors, protooncogenes, and soluble factors. For the clinician faced with treating both simple and complex dental complications arising from abnormal tooth eruption, knowledge of the basic molecular mechanisms involved is essential (Fig. 9-1). A paper by Kardos reviews the evidence for a tooth-eruptive force as well as its direction and source.[15] Finally, an extensive review by Marks and Schroeder analyzes experimental data to identify the basic principles of tooth eruption and offers their guiding theories of the process.[16] Readers who wish to obtain more information about the details of the tooth eruption process are referred to these review articles.

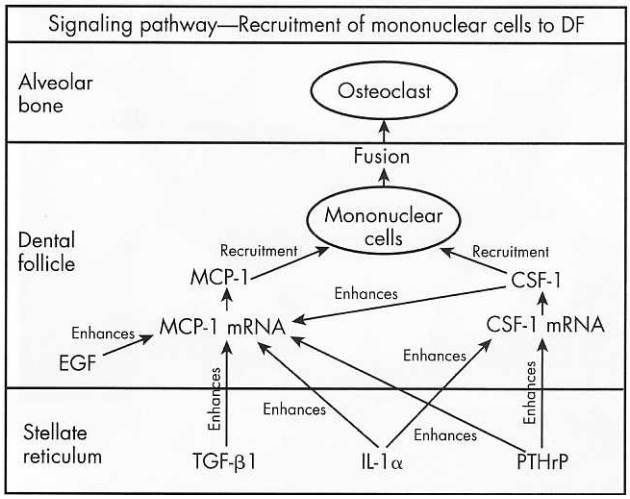
INFLUENCE OF PREMATURE LOSS OF PRIMARY MOLARS ON ERUPTION TIME OF THEIR SUCCESSORS

Posen, after reviewing the records of children in the Burlington study who had undergone unilateral extraction of primary molars, came to the following conclusions: Eruption of the premolar teeth is delayed in children who lose primary molars at 4 or 5 years of age and before.' If extraction of the primary molars occurs after the age of 5 years, there is a decrease in the delay of premolar eruption. At 8, 9, and 10 years of age, premolar eruption resulting from premature loss of primary teeth is greatly accelerated. Hartsfield stated that premature loss of teeth associated with systemic disease usually results from some change in the immune system or connective tissue.[13] The most common of these conditions appears to be hypophosphatasia and early-onset periodontitis.

VARIATIONS IN THE SEQUENCE OF ERUPTION

The mandibular first permanent molars are often the first permanent teeth to erupt. They are quickly followed by the mandibular central incisors. Lo and Moyers found little or no clinical significance to the eruption of the incisors before the molars."[N]

After analyzing serial records of 16,000 children in Newburgh and Kingston, New York, Carlos and Gittelsohn concluded that the average eruption time of the lower central incisors was earlier than that of the first molars by about 1 1/2 months in both boys and girls.[2D] Of considerable interest was the gender difference in the



[I **9-1.** Paracrine signaling between the stellate reticulum and dental follicle (DF) results in the synthesis and secretion of chemotactic molecules, colony-stimulating factor 1 (CSF-1) and monocyte chemotactic protein 1 (MCP-1) for recruitment of mononuclear cells. *EGF,* Epidermal growth factor; IL-la, interleukin 1a.; PTHrP, parathyroid hormone-related peptide; *mRNA,* messenger RNA; *TGF-J31,* transforming growth factor (l1. *(Reprinted with permission from Wise GE et al: Crit Rev Oral Bio! Med 12141:323-335, 2002.)*

eruption sequence of permanent teeth. The mandibular canine erupted before the maxillary and mandibular first premolars in girls. In boys the eruption order was reversed—the maxillary and mandibular first premolars erupted before the mandibular canine.

Moyers stated that the most common sequence of eruption of permanent teeth in the mandible is first molar, central incisor, lateral incisor, canine, first premolar, second premolar, and second molar.[1] The most common sequence for the eruption of the maxillary permanent teeth is first molar, central incisor, lateral incisor, first premolar, second premolar, canine, and second molar (Fig. 9-2). He identified these common sequences in each arch to be favorable for maintaining the length of the arches during the transitional dentition.

It is desirable that the mandibular canine erupt before the first and second premolars. This sequence aids in maintaining adequate arch length and in preventing lingual tipping of the incisors. Lingual tipping of the incisors not only causes a loss of arch length but also allows the development of an increased overbite. An abnormal lip musculature or an oral habit that causes a greater force on the lower incisors than can be compensated by the tongue allows a collapse of the anterior segment. For this reason use of a passive lingual arch appliance is often indicated when the primary canines have been lost prematurely or when the sequence of eruption is undesirable.

A deficiency in arch length can occur if the mandibular second permanent molar develops and erupts before the second premolar. Eruption of the second permanent molar first encourages mesial migration or tipping of the first permanent molar and encroachment on the space needed for the second premolar. The importance of maintaining the second primary molar until its replacement by the second premolar is discussed in Chapter 27.

In the maxillary arch the first premolar ideally should erupt before the second premolar, and they should be followed by the canine. The untimely loss of primary molars in the maxillary arch, which allows the first permanent molar to drift and tip mesially, results in the permanent canine's being blocked out of the arch, usually to the labial side.

The position of the developing second permanent molar in the maxillary arch and its relationship to the first permanent molar should be given special attention. Its eruption before the premolars and canine can cause a loss of arch length, just as in the mandibular arch.

The eruption of the maxillary canine is often delayed because of an abnormal position or devious eruption path. This delayed eruption should be considered along with its possible effect on the alignment of the maxillary teeth. The significance of the sequence of the eruption of permanent teeth is considered further in Chapter 27.

LINGUAL ERUPTION OF MANDIBULAR PERMANENT INCISORS

The eruption of mandibular permanent incisors lingual to retained primary incisors is often a source of concern for parents. The primary teeth may have undergone extensive root resorption and may be held only by soft tissues. In other instances the roots may not have undergone normal resorption and the teeth remain solidly in place. It is common for mandibular permanent incisors to erupt lingually, and this pattern should be considered essentially normal. It is seen both in patients with an obvious arch length inadequacy (Fig. 9-3) and in those with a desirable amount of spacing of the primary incisors (Fig. 9-4). In either case the tongue and continued alveolar growth seem to play an important role in influencing the permanent incisors into a more normal position with time. Although there may be insufficient room in the arch for the newly erupted permanent

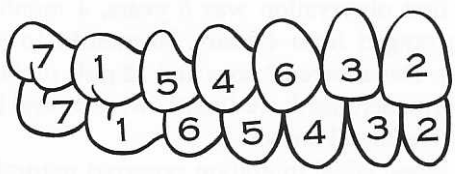
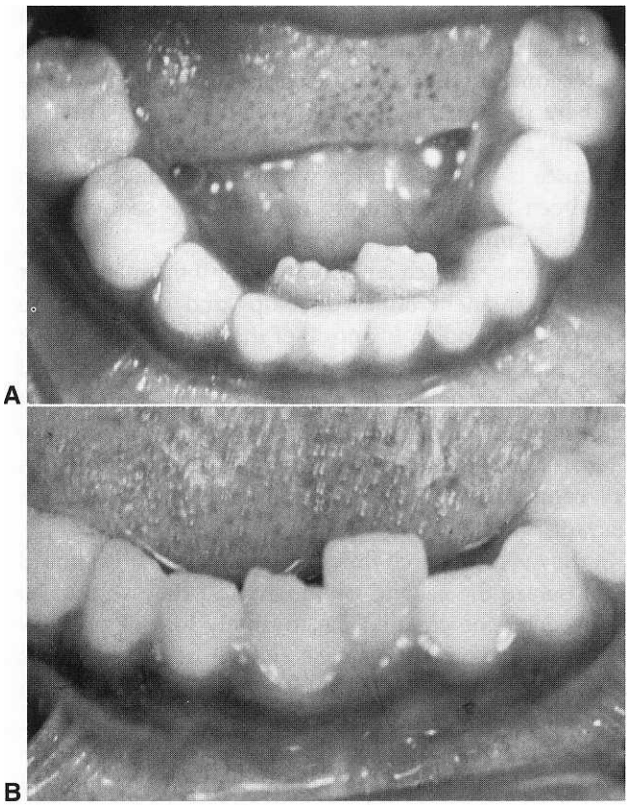


FIG **9-2.** Desirable eruption sequence for the permanent teeth.



f I G. **9-3. A,** The permanent central incisors are erupting lingual to the retained primary central incisors, which were extracted. **B,** The arch length is inadequate to accommodate the permanent incisors. However, they have moved forward into a more favorable position as a result of the force exerted on them by the tongue.

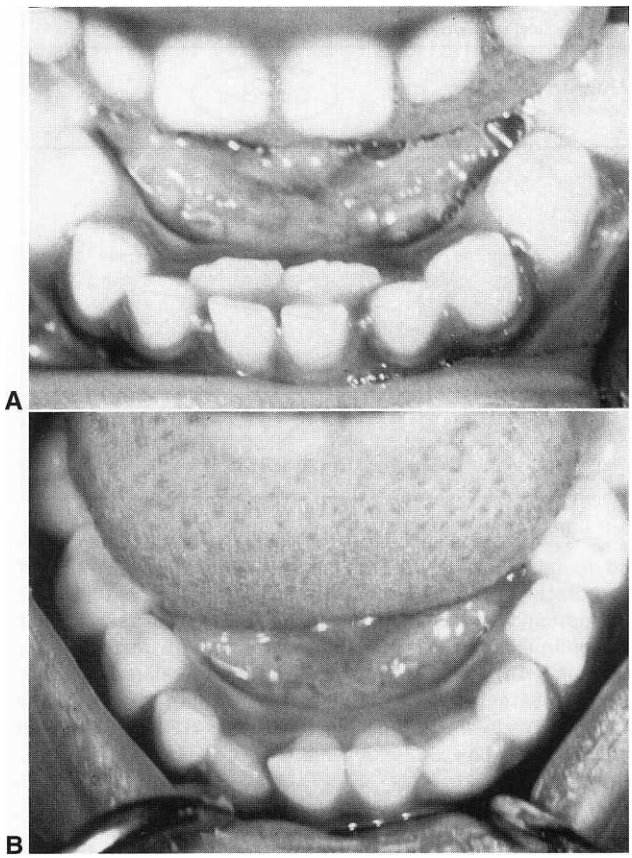


FIG . **9-4. A,** Primary teeth are desirably spaced with sufficient room for the permanent central incisors. However, the permanent teeth erupted lingually to the primary teeth. **B,** Extraction of the primary central incisors resulted in a desirable positioning of the permanent teeth, but given enough time this condition probably would have been self-correcting.

tooth, its position will improve over several months. In some cases there is justification for removal of the corresponding primary tooth. Extraction of other primary teeth in the area is not recommended, however, because it will only temporarily relieve the crowding and may even contribute to the development of a more severe arch length inadequacy.

Gellin has emphasized the anxiety created when the parents discover a double row of teeth. He suggested that if the condition is identified before 7'/2 years of age it is unnecessary to subject the child to the trauma of removing the primary teeth because the problem is almost always self-correcting within a few months." However, he warned that when lingually erupted permanent mandibular incisors are seen in an older child and the radiograph shows no root resorption of the primary teeth, self-correction has not been achieved and the corresponding primary teeth should be removed.

Gellin and Haley conducted a clinical study to determine if removal of the corresponding primary tooth is necessary when the lingual eruption pattern of the mandibular permanent incisor is identified.[2]" They monitored 57 lingually positioned mandibular permanent central or lateral incisors in 44 children. The children were selected for the study if they had one or more permanent mandibular incisors erupting immediately lingual to the corresponding primary incisor.

Other criteria required for entry into the study included the presence of both primary mandibular canines, the absence of any other anomalies of the mandibular primary or permanent incisors, and the absence of severe crowding of the permanent mandibular incisors characterized by eruption of the lateral incisors directly behind the central incisors. The mean age of the children at their first observation was 6 years, 4 months, and the ages ranged from 4 years, 10 months to 8 years, 8 months. There were 22 boys and 22 girls in the study. Of the permanent teeth studied, 47 were central incisors and 10 were lateral incisors.

In all cases, labial migration occurred naturally, and extraction of the corresponding primary incisor was unnecessary. Gellin and Haley reported that spontaneous

correction of lingually erupted mandibular permanent central incisors occurred by age 8 years, 2 months in 95% of the cases that met the criteria of their study.'-' They also observed that spontaneous correction of lingually erupted lateral incisors occurred by at least 8 years, 4 months of age. Although the sample of the lateral incisors was too small to draw specific conclusions, correction occurred in all cases, and central incisors migrated labially at an earlier age. Gellin and Haley recommended a conservative approach of waiting and periodic observation to spare the child a surgical procedure. They suggested that if labial migration of the permanent incisor had not occurred by 8.2 years for central incisors and 8.4 years for lateral incisors, overretention of the primary incisor should be suspected and removal of the primary tooth considered. However, they recommended removal only if the primary incisor remained firm and the root had failed to resorb.

We recognize that spontaneous correction of lingually erupted permanent incisors is likely to occur given enough time, particularly in cases in which there is not severe crowding. A watchful waiting approach may be justified, especially when the patient is first seen in the dentist's office for this specific problem. Removal of a tooth during the first dental appointment of a child 6 to 8 years old probably compromises the dentist's ability to develop rapport with the child. However, the extraction procedure in such cases is quite simple, and we believe there may be many times when it is appropriate. The parents' feelings should not be ignored in the decision; even a 95% chance that correction will occur may not satisfy all parents. The dentist may find that some dental patients prefer to have the primary tooth extracted and the problem laid to rest. Although monitoring the condition without extraction is accept-able under the conditions outlined by Gellin and Haley, we know of no significant contraindications to early removal of the offending primary incisor even in spaced dentitions, if specific conditions warrant its consideration.

Even when mandibular permanent incisors erupt uneventfully, they often appear rotated and staggered in position. The molding action of the tongue and the lips improves their relationship within a few months.

TEETHING AND DIFFICULT ERUPTION

In most children the eruption of primary teeth is preceded by increased salivation, and the child will want to put the hand and fingers into the mouth. These observations may be the only indication that the teeth will soon erupt.

Some young children become restless and fretful during the time of eruption of the primary teeth. Many conditions, including croup, diarrhea, fever, convulsions, primary herpetic gingivostomatitis, and even death have been incorrectly attributed to eruption. In the nineteenth century, complications from teething were believed to be a very frequent cause of infant mortality. Both King and Dally have reported that 5016 deaths in England and Wales were attributed to teething in 1839.[42']

Illingworth made a thorough search of the world literature and failed to produce evidence that teething causes fever, convulsions, bronchitis, or diarrhea.[26] His findings are supported by Tasanen's unique study of teething, in which 192 tooth eruptions were observed in 126 infants and 107 controls.' All the babies were seen on the day of the eruption of the tooth, and records were kept of the temperature, incidence of infection, erythrocyte sedimentation rate, white blood cell count, behavior (including sleep), color of the mucosa, sensitivity of the tissue covering the erupting tooth, and pain resulting from pressure on the tooth. Tasanen concluded that teething does not increase the incidence of infection, does not cause any rise in temperature, erythrocyte sedimentation rate, or white blood cell count, and does not cause diarrhea, cough, sleep disturbance, or rubbing of the ear or cheek, but that it does cause daytime restlessness, an increase in the amount of finger sucking or rubbing of the gum, an increase in drooling, and possibly some loss of appetite. In one third of the children there was no change in the color of the mucosa in the area of the erupting tooth, in one third of the children the change was slight, and in the remaining one third of the children there was a pronounced change in the mucosa, often with small hemorrhages.

A later study of 46 healthy infants conducted by Jaber, Cohen, and Mor demonstrated a small increase in body temperature in 20 (43%) of the infants on the day of emergence of their first tooth.[2R] However, the authors emphasized a danger in attributing fever to teething. Macknin et al have confirmed these results." Leung has reported that serious mistakes have been made in the care of infants and toddlers when their symptoms were ascribed to teething without completion of a thorough diagnostic evaluation and resulted in the overlooking of significant systemic disturbances.[30] Swann observed 50 children who were hospitalized after their symptoms were initially attributed to teething by parents or physicians.[3] After a careful medical evaluation, an organic cause of illness was identified in 48 of the 50 patients.

Because the eruption of teeth is a normal physiologic process, the association with fever and systemic disturbances is not justified. A fever or respiratory tract infection during this time should be considered coincidental to the eruption process rather than related to it.

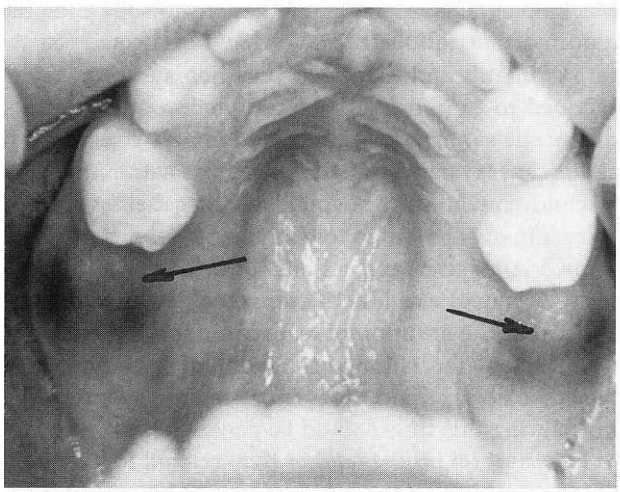
Inflammation of the gingival tissues before complete emergence of the crown may cause a temporary painful condition that subsides within a few days. The surgical

removal of the tissue covering the tooth to facilitate eruption is not indicated. If the child is having extreme difficulty, the application of a nonirritating topical anesthetic may bring temporary relief. The parent can apply the anesthetic to the affected tissue over the erupting tooth three or four times a day. Several low-dose products specifically formulated for infants are available without prescription. Caution must be exercised, however, when one is prescribing topical anesthetics, especially for infants, because systemic absorption of the anesthetic agent is rapid, and toxic doses can occur if it is misused. The parent must clearly understand the importance of using the drug only as directed. The eruption process may be hastened if the child is allowed to chew on a piece of toast or a clean teething object.

ERUPTION HEMATOMA (ERUPTION CYST)

A bluish purple, elevated area of tissue, commonly called an *eruption hematoma,* occasionally develops a few weeks before the eruption of a primary or permanent tooth. The blood-filled cyst is most frequently seen in the primary second molar or the first permanent molar regions. This fact substantiates the belief that the condition develops as a result of trauma to the soft tissue during function (Fig. 9-5). Usually within a few days the tooth breaks through the tissue, and the hematoma subsides. Because the condition is almost always self-limited, treatment of an eruption hematoma is rarely necessary. However, surgically uncovering the crown may occasionally be justified.

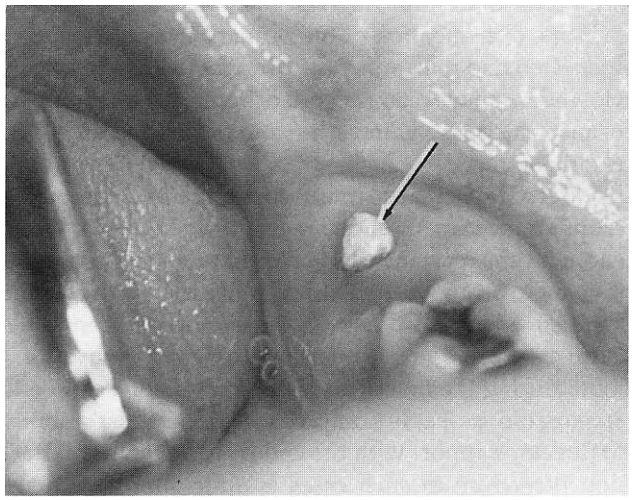
When the parents discover an eruption hematoma, they may fear that the child has a serious disease such as a malignant tumor. The dentist must be understanding and sensitive to their anxiety while reassuring them that the lesion is not serious.



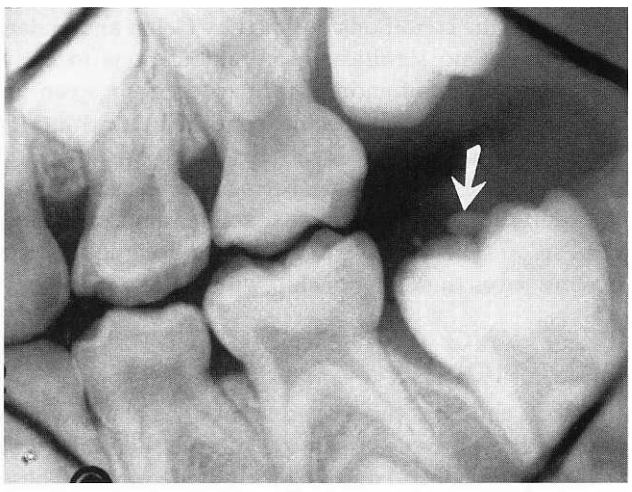
[I G . **9-5.** Eruption hematomas (arrows) have developed before the eruption of the second primary molars.

ERUPTION SEQUESTRUM

The eruption sequestrum is seen occasionally in children at the time of the eruption of the first permanent molar (Figs. 9-6 and 9-7). Starkey and Shafer have described the sequestrum as a tiny spicule of nonviable bone overlying the crown of an erupting permanent molar just before or immediately after the emergence of the tips of the cusps through the oral mucosa."- Watkins reported one case of eruption sequestrum composed of dentin and cementum.[33] Priddy and Price described two cases and offered evidence to support their claim that an



F I G **9-6.** Arrow points to an eruption sequestrum in a girl 5 years, 11 months of age. It appears clinically as a white spicule of hard tissue overlying the central fossa of a mandibular first permanent molar, which is just beginning to erupt through the *mucosa.* (Courtesy Drs. Paul E. Starkey and William G. Shafer.)



[I G **9-7.** Radiographic appearance of an eruption sequestrum (arrow) in a child 6 years, 9 months of age. No treatment is required unless symptoms develop. (Courtesy Drs. Paul E. Starkey and William G. Shafer.)

eruption sequestrum is composed of cementum-like material formed within the follicle." These various reports suggest that eruption sequestra may develop from either osteogenic or odontogenic tissue. Regardless of its origin, the hard tissue fragment is generally over-lying the central fossa of the associated tooth, embedded, and contoured within the soft tissue. As the tooth erupts and the cusps emerge, the fragment sequestrates.

Eruption sequestra are usually of little or no clinical significance. It is probable that some of these sequestra spontaneously resolve without noticeable symptoms. However, after an eruption sequestrum has surfaced through the mucosa, it may easily be removed if it is causing local irritation. The base of the sequestrum is often still well embedded in gingival tissue when it is discovered, and application of a topical anesthetic or infiltration of a few drops of a local anesthetic may be necessary to avoid discomfort during removal.

ECTOPIC ERUPTION

Arch length inadequacy, tooth mass redundancy, or a variety of local factors may influence a tooth to erupt or try to erupt in an abnormal position. Occasionally this condition may be so severe that actual transposition of teeth takes place. Several problems associated with ectopic eruption of teeth and the management of these problems are presented in Chapter 27.

NATAL AND NEONATAL TEETH

The prevalence of natal teeth (teeth present at birth) and neonatal teeth (teeth that erupt during the first 30 days) is low. Leung conducted a retrospective study of hospital records of 50,892 infants born in Calgary, Alberta.[3S] These records identified 15 infants found to have natal teeth, a prevalence of 1 in 3392 births. In another survey Kates, Needleman, and Holmes found the calculated prevalence of natal teeth to be 1 in 3667 among 11,000 infants when the survey information was obtained indirectly; however, in a group of 7155 infants actually examined, the prevalence was found to be 1 in 716.[3"]

Zhu and King conducted an extensive review of the literature of reported cases of natal and neonatal teeth.[37] They found that about 85% of natal or neonatal teeth are mandibular primary incisors, and only small percentages are supernumerary teeth. It is common for natal and neonatal teeth to occur in pairs. Natal and neonatal molars are rare: Zhu and King found only 20 reported cases dating from 1897. They also reported that most premature tooth eruption seems to occur in otherwise normal infants, with or without a family history of the condition. In some infants, however, the presence of such teeth may be a localized manifestation of an environmental cause or an underlying syndrome, and this underscores the importance of thorough diagnostic evaluations of infants with natal or neonatal teeth.

Spouge and Feasby believe that the terms *natal teeth* and *neonatal teeth* constitute a relatively artificial distinction and should be further qualified to provide a more practical clinical meaning.[3"] They have suggested that the terms *mature* and *immature* are more in keeping with the varying prognoses associated with such teeth.

Early eruption of primary teeth seems to be familial. Many parents volunteer the information that their teeth erupted early. Bodenhoff and Gorlin found that 15% of the children with natal or neonatal teeth had parents, siblings, or other near relatives with a history of such teeth.[3o]

A radiograph should be made to determine the amount of root development and the relationship of a prematurely erupted tooth to its adjacent teeth. One of the parents can hold the x-ray film in the infant's mouth during the exposure.

Most prematurely erupted teeth (immature type) are hypermobile because of the limited root development. Some teeth may be mobile to the extent that there is danger of displacement of the tooth and possible aspiration, in which case the removal of the tooth is indicated. In some cases, the sharp incisal edge of the tooth may cause laceration of the lingual surface of the tongue (Riga-Fede disease), and the tooth may have to be removed.

Zhu and King were unable to find any reported cases of aspirated natal or neonatal teeth. Extraction of such a tooth, if necessary, is a simple procedure but is emotionally difficult for the parents.[37] After the tooth is removed, careful curettage of the socket is indicated in an attempt to remove any odontogenic cellular remnants that may otherwise be left in the extraction site. Such retained remnants may subsequently develop atypical toothlike structures that require additional treatment, as reported by Nedley, Stanley, and Cohen.'"

The preferable approach, however, is to leave the tooth in place and to explain to the parents the desirability of maintaining this tooth in the mouth because of its importance in the growth and uncomplicated eruption of the adjacent teeth. Within a relatively short time the prematurely erupted tooth will become stabilized, and the other teeth in the arch will erupt (Fig. 9-8).

Eruption of teeth during the neonatal period presents less of a problem. These teeth can usually be maintained even though root development is limited (Figs. 9-9 and 9-10).

A retained natal or neonatal tooth may cause difficulty for a mother who wishes to breast-feed her infant. If breast-feeding is too painful for the mother initially, the use of a breast pump and bottling of the milk are recommended. However, the infant may be conditioned not to "bite" during suckling in a relatively short time if the mother persists with breast-feeding. It seems that the infant senses the mother's discomfort and learns to avoid causing it.

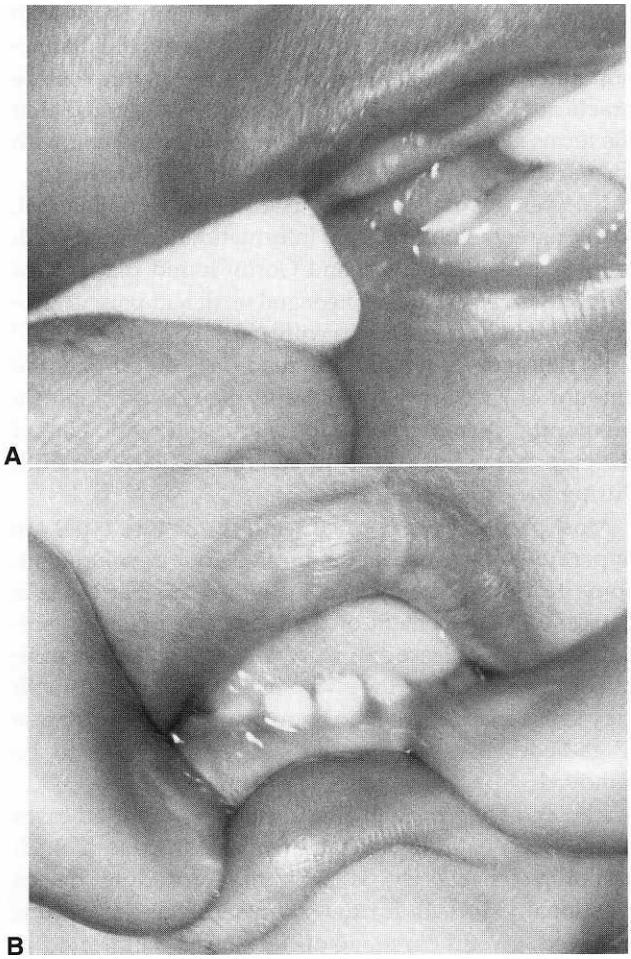
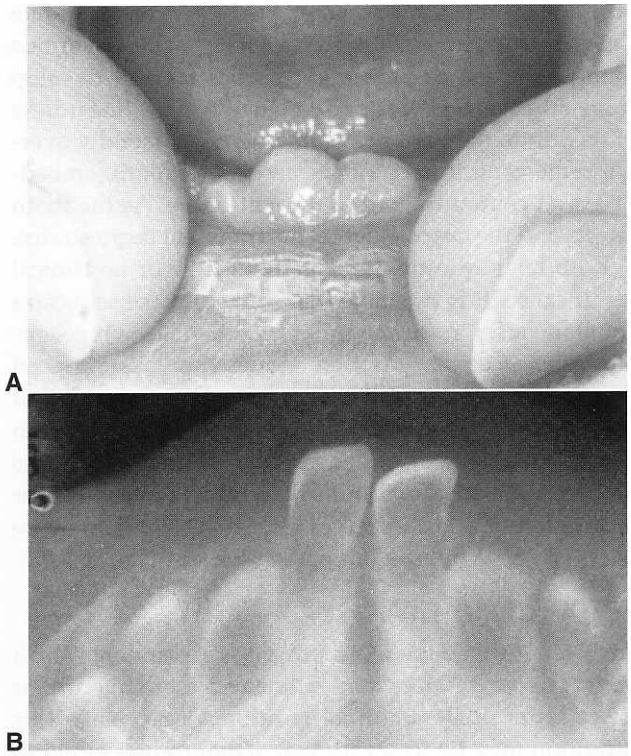


FIG . **9-8. A,** Natal tooth in a 3-day-old infant. Because the tooth was not excessively mobile, there was no reason to recommend its removal. **B,** Within 2 months other teeth in the mandibular anterior region erupted. *The ungloved fingers in the photographs are those of* the *infant's* parent.



f **I G 9-9.** A, Parents of a 3-week-old infant were concerned about the elevated mass of tissue on the lower ridge. **B,** Radiograph revealed two primary central incisors that would soon erupt. The ungloved fingers in the photograph are those of the infant's parent.

EPSTEIN PEARLS, BOHN NODULES, AND DENTAL LAMINA CYSTS

Small, white or grayish white lesions on the alveolar mucosa of the newborn may on rare occasions be incorrectly diagnosed as natal teeth. The lesions are usually multiple but do not increase in size (Fig. 9-11). No treatment is indicated, since the lesions are spontaneously shed a few weeks after birth.

Fromm reported that clinically visible cysts were found in 1028 of 1367 newborn infants.'He noted and classified the following three types of inclusion cysts:

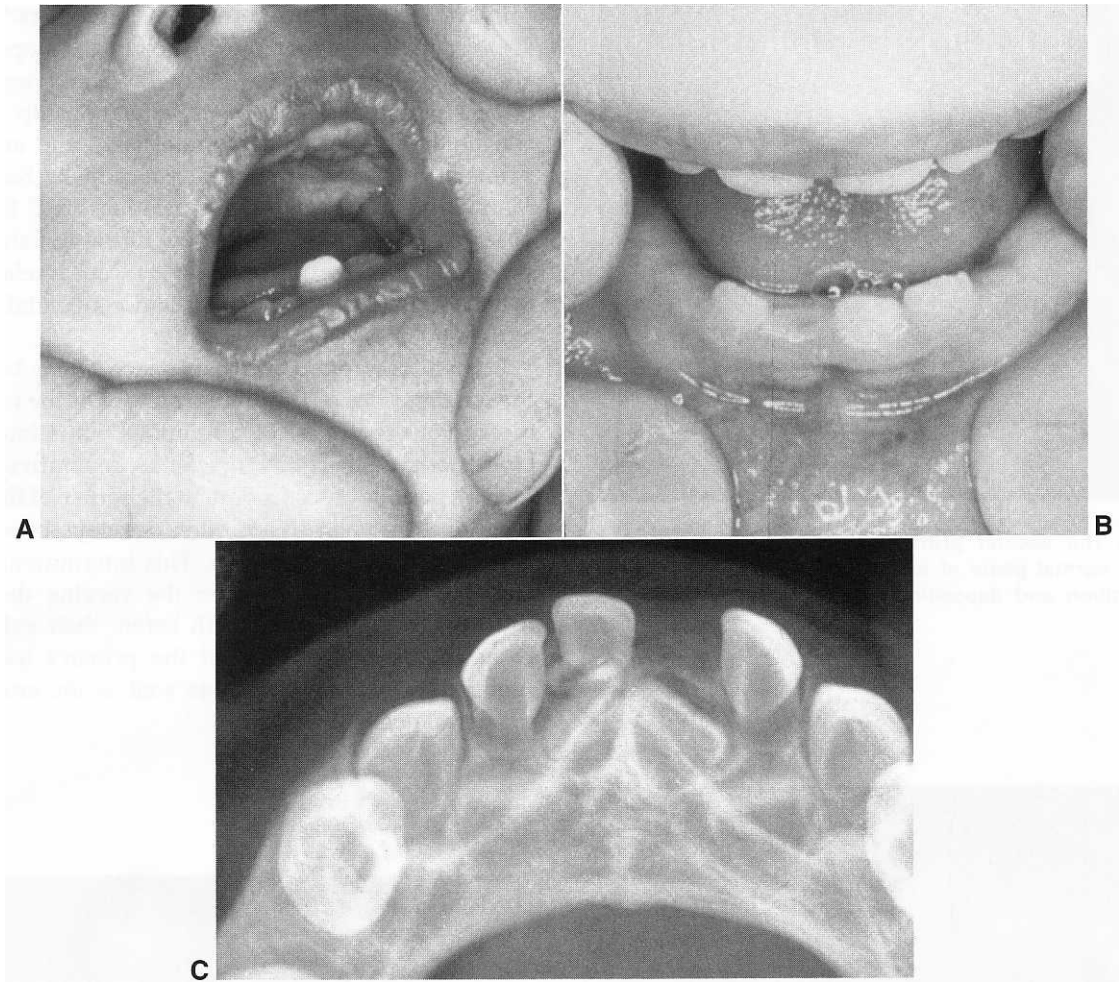
1. Epstein pearls are formed along the midpalatine raphe. They are considered remnants of epithelial tissue trapped along the raphe as the fetus grew.
2. Bohn nodules are formed along the buccal and lingual aspects of the dental ridges and on the palate away from the raphe. The nodules are considered remnants of mucous gland tissue and are histologically different from Epstein pearls.
3. Dental lamina cysts are found on the crest of the maxillary and mandibular dental ridges. The cysts apparently originated from remnants of the dental lamina.

LOCAL AND SYSTEMIC FACTORS THAT INFLUENCE ERUPTION

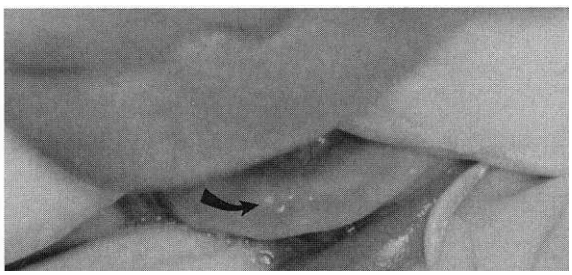
ANKYLOSED TEETH

The problem of ankylosed primary molars deserves much attention by dentists. Application of the term *submerged molar* to this condition is inaccurate, even though the tooth may appear to be submerging into the mandible or maxilla. This misconception results from the fact that the ankylosed tooth is in a state of static retention, whereas in the adjacent areas eruption and alveolar growth continue. The term *infraocclusion,* although commonly used today, in the authors' opinions is not preferable to ankylosis.

Henderson pointed out that ankylosis should be considered an interruption in the rhythm of eruption and further observed that a patient who has one or two



f IG 9-10. **A,** Eruption of one of the primary central incisors occurred at 4 weeks of age. The tooth was mobile because of limited root formation, but it was not extracted. **B,** One of the prematurely erupted central incisors was subsequently lost as the result of a fall, but the other was retained. C, Dilacerated root formation on one of the neonatal teeth. The ungloved fingers in the photographs are those of the infant's parent.



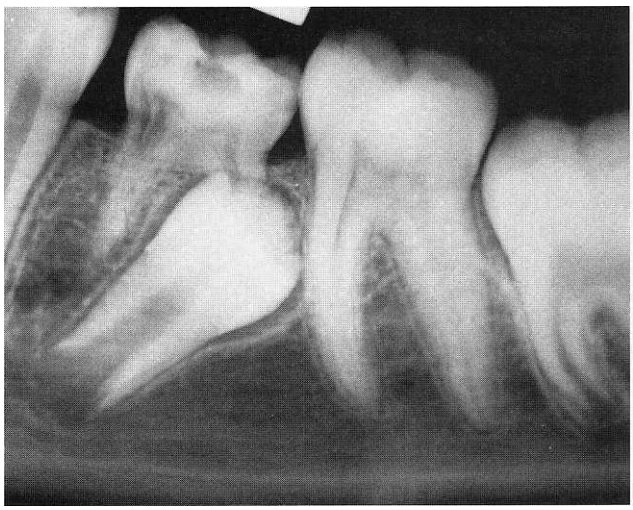
I IG 9-11. Dental lamina cyst (arrow). No treatment is indicated; such lesions disappear within a few weeks after birth.

ankylosed teeth is more likely to have other teeth become ankylosed.` [2]

The mandibular primary molars are the teeth most often observed to be ankylosed (Figs. 9-12 and 9-13). In unusual cases all the primary molars may become firmly attached to the alveolar bone before their normal exfoliation time. Such a case was reported by Alexander et al, and a similar case is illustrated in this chapter (see Fig. 9-17). ' Ankylosis of the anterior primary teeth does not occur unless there has been a trauma.

The cause of ankylosis in the primary molar areas is unknown, but at least three theories have been proposed. The observation of ankylosis in several members of the same family lends support to the theory that it follows a familial pattern. Via has reported that the condition occurs more frequently among siblings of children with the characteristics." The occurrence is noted to have a familial tendency and is probably a non–sex-linked trait. Krakowiak has observed the prevalence of ankylosis to be much lower among black children (0.93%) than among white children (4.10%).[5]

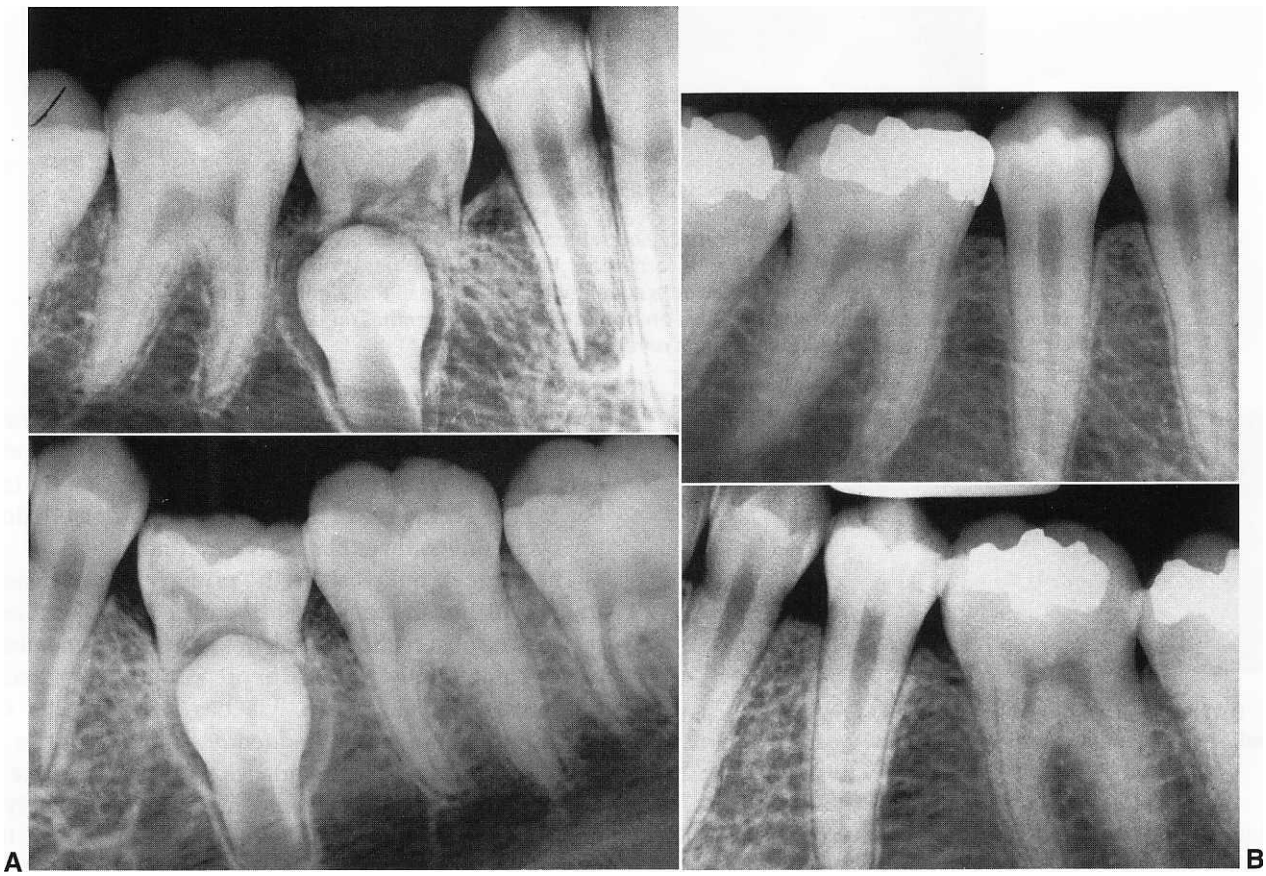
Darling and Levers observed that, in a group of children with 108 ankylosed teeth, 21 of the affected



F **I G 9-12.** The second primary molar is ankylosed and below the normal plane of occlusion. There is evidence of root resorption and deposition of bone into the resorbed areas.

primary teeth had no permanent successors." Brown also reported a higher prevalence of developmentally absent premolar teeth in patients with ankylosis." It is often suggested that there is a relationship between congenital absence of permanent teeth and ankylosed primary teeth. Steigman, Koyoumdjisky-Kaye, and Matrai have discounted this relationship." Based on observation and a careful review of the literature, they reported that there appears to be no causal relationship between ankylosed precursors and congenital absence of their successors.

Normal resorption of the primary molar begins on the inner surface or the lingual surface of the roots. The resorption process is not continuous but is interrupted by periods of inactivity or rest. A reparative process follows periods of resorption. In the course of this reparative phase a solid union often develops between the bone and the primary tooth. This intermittent resorption and repair may explain the varying degrees of firmness of the primary teeth before their exfoliation. Extensive bony ankylosis of the primary tooth may prevent normal exfoliation, as well as the eruption of the permanent successor.



f **I G 9-13.** A, Bilateral ankylosis of second primary molars. **B,** The ankylosed molars were eventually shed, and the second premolars erupted into good occlusion. Frequently the ankylosed teeth must be removed surgically.



FIG **9-14.** Ankylosed second primary molar with a carious lesion in the occlusal surface. This tooth probably became ankylosed soon after root resorption began.

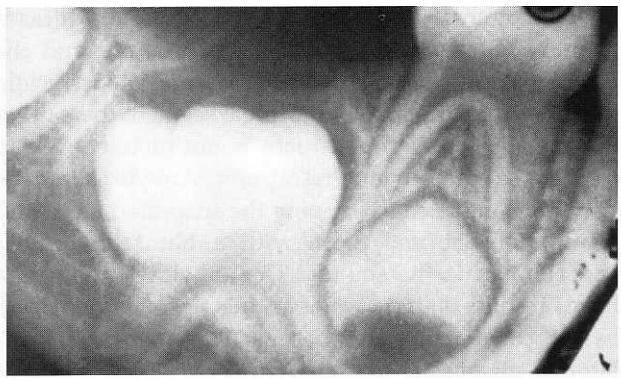


FIG **9-15.** An ankylosed, deeply embedded second primary molar. Surgical removal of this tooth is indicated.

Ankylosis of the primary molar to the alveolar bone does not usually occur until after its root resorption begins. If ankylosis occurs early, eruption of adjacent teeth may progress enough that the ankylosed tooth is far below the normal plane of occlusion and may even be partially covered with soft tissue (Fig. 9-14). An epithelium-lined track will, however, extend from the oral cavity to the tooth. Ankylosis may occasionally occur even before the eruption and complete root formation of the primary tooth (Fig. 9-15). Tsukamoto and Braham reported a case of apparent early ankylosis of a mandibular second primary molar that was not diagnosed until the patient was 10 years of age; at that time the succedaneous second premolar was lying malposed but occlusal to the unerupted primary molar." Ankylosis can also occur late in the resorption of the primary roots and even then can interfere with the eruption of the underlying permanent tooth (Fig. 9-16).

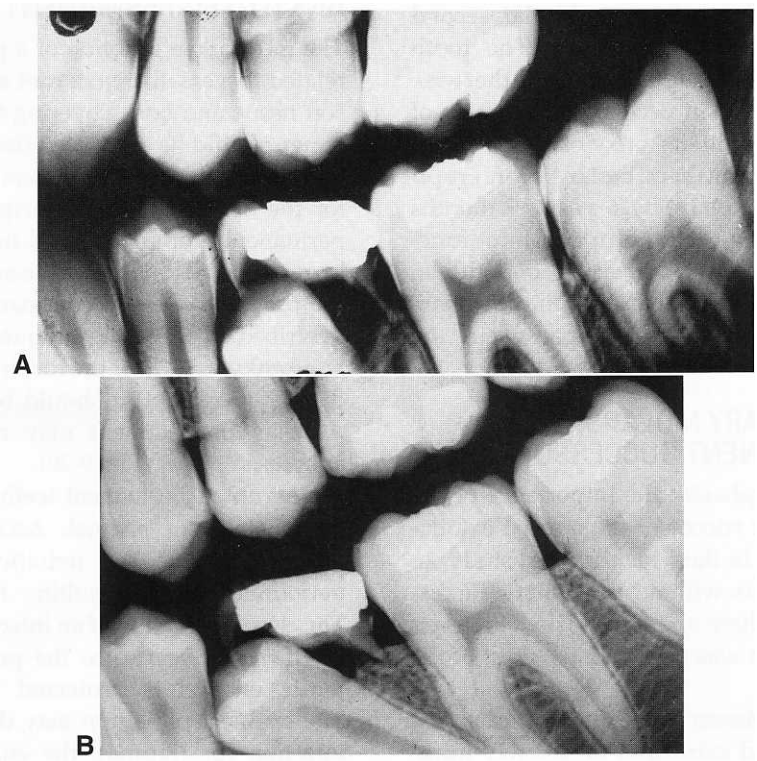


FIG **9-16. A,** A small spicule of root of the primary tooth is ankylosed to the alveolar bone. This was overlooked at the time of the routine examination. **B,** One year later the second primary molar is still retained, and the second premolar has moved into a more unfavorable position.

The diagnosis of an ankylosed tooth is not difficult to make. Because eruption has not occurred and the alveolar process has not developed in normal occlusion, the opposing molars in the area seem to be out of occlusion. The ankylosed tooth is not mobile, even in cases of advanced root resorption. Ankylosis can be partially confirmed by tapping the suspected tooth and an adjacent normal tooth with a blunt instrument and comparing the sounds. The ankylosed tooth will have a solid sound, whereas the normal tooth will have a cushioned sound because it has an intact periodontal membrane that absorbs some of the shock of the blow.

The radiograph is often a valuable aid in making a diagnosis. A break in the continuity of the periodontal membrane indicating an area of ankylosis is often evident radiographically.

In the management of an ankylosed tooth, early recognition and diagnosis are extremely important. The eventual treatment may involve surgical removal (Fig. 9-17). However, unless a caries problem is unusual or loss of arch length is evident, the dentist may choose to keep the tooth under observation. A tooth that is definitely ankylosed may at some future time undergo root resorption and be normally exfoliated. When patient cooperation is good and recall periods are regular, a watchful waiting approach is best.

Belanger, Strange, and Sexton reported a case in which even early ankylosis of a mandibular second primary molar spontaneously resolved.5[3] The tooth was discovered to be unerupted in an otherwise complete primary dentition in occlusion at 3 years of age. The tooth remained in its ankylosed condition until the adjacent first permanent molar began erupting through the gingival tissue. By 6 years, 9 months of age the primary molar had erupted into functional occlusion and a normal-appearing periodontal ligament space could be seen radiographically even though no space was previously apparent in the furcation.

ANKYLOSIS OF PRIMARY MOLARS WITH ABSENCE OF PERMANENT SUCCESSORS

Kurol and Thilander emphasize the importance of the presence of a permanent successor for normal exfoliation of a primary molar. In their longitudinal study no ankylosed primary molars without permanent successors were found to exfoliate spontaneously. However, very slow root resorption was observed for most of the ankylosed teeth.[S1]

The observations of Messer and Cline indicate that failure to carry out timed extraction of severely infraoccluded molars results in reduced alveolar bone support for the premolars.[52] However, Kurol and Olson suggest that infraocclusions and ankylosis of primary molars does not constitute a general risk for future alveolar bone loss mesial to the first permanent molars.[53] In their study of 119 infraoccluded primary molars next to permanent first molars, all but two of the first permanent molars showed a normal alveolar bone level mesially. Therefore, the general treatment recommendation to await normal exfoliation and eruption of successors remains valid in their opinion. They suggest that, in patients in whom there is an abnormality associated with a succedaneous tooth (such as agenesis, ectopic eruption, etc.), early intervention will most likely be required. In situations in which permanent successors of ankylosed primary molars are missing, attempts have been made to establish functional occlusion using stainless steel crowns, overlays, or bonded composite resins on the affected primary molars. Currently, bonded restorations would be the preferred choice. Williams, Zwemer, and Hoyt reported excellent success with two bonded composite crowns on ankylosed mandibular second primary molars that were well below the occlusal plane.[5"] At the time of their report the restorations had been functional for more than 7 years in a young adult patient. This treatment is successful only if maximum eruption of permanent teeth in the arch has occurred. If adjacent teeth are still in a state of active eruption, they will soon bypass the ankylosed tooth (Fig. 9-18).

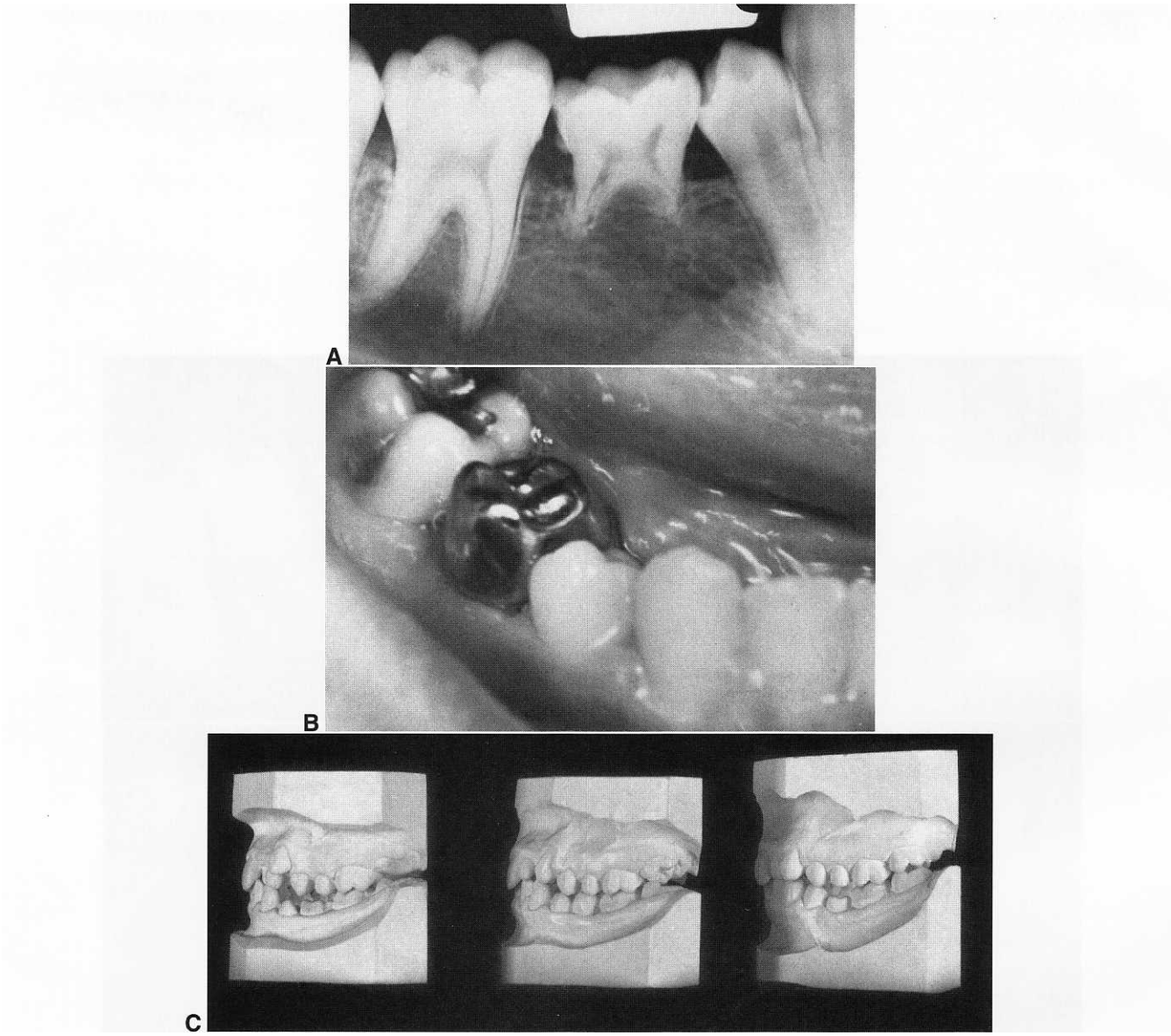
ANKYLOSED PERMANENT TEETH

The incomplete eruption of a permanent molar may be related to a small area of root ankylosis. The removal of soft tissue and bone covering the occlusal aspect of the crown should be attempted first, and the area should be packed with surgical cement to provide a pathway for the developing permanent tooth (Fig. 9-19). If the permanent tooth is exposed in the oral cavity and at a lower occlusal plane than the adjacent teeth, ankylosis is the probable cause. Biederman and also Skolnick have described a luxation technique effective in breaking the bony ankylosis.[55.56] If the rocking technique is not immediately successful, it should be repeated in 6 months. A delay in treatment may result in a permanently ankylosed molar (Fig. 9-20).

Unerupted permanent teeth may become ankylosed by inostosis of enamel. According to Franklin, the process follows the irritation of the follicular or periodontal tissue resulting from chronic infection." The close association of an infected apex to an unerupted tooth may give rise to the process. In the unerupted tooth, enamel is protected by enamel epithelium. The enamel epithelium may disintegrate as a result of infection (or trauma), the enamel may subsequently be resorbed, and bone or coronal cementum may be deposited in its place. The result is solid fixation of the tooth in its unerupted position (Fig. 9-21).



f I G. **9-17**. **A,** All eight of the primary molars were ankylosed. Continuing eruption of the adjacent teeth has caused a loss of arch length. **B,** Radiographs aided in the diagnosis of the ankylosed primary molars. The recommended treatment was surgical removal of the ankylosed teeth. C, Space maintainers were constructed after removal of the ankylosed teeth and were worn until the permanent teeth erupted. D, Ideal occlusion was achieved as a result of early diagnosis and the removal of the ankylosed teeth at the proper time.



f1 6 **9-18. A,** Ankylosed primary molar without a permanent successor. **B,** Mesiodistal width of the primary molar was reduced to allow the premolar to erupt, and an overlay was constructed to establish occlusion with the opposing teeth. C, Models at the left show the original condition. Center models show the occlusion at the time the overlay was placed on the ankylosed tooth. Models at the right show the continued eruption of the adjacent teeth that occurred in the subsequent 18-month period.

TRISOMY 21 SYNDROME (DOWN SYNDROME)

Trisomy 21 syndrome (Down syndrome [DS]) is one of the congenital anomalies in which delayed eruption of the teeth frequently occurs. The first primary teeth may not appear until 2 years of age, and the dentition may not be complete until 5 years of age. The eruption often follows an abnormal sequence, and some of the primary teeth may be retained until 15 years of age. A study of 127 males and 128 females with DS by Ondarza et al found that, on average, 6 primary teeth were delayed in eruption in boys and 11 primary teeth were delayed in girls.[58] A similar study of 116 males and 124 females with DS by Jara et al showed delayed eruption of 13 permanent teeth in boys and 8 permanent teeth in girls.[5"] These studies seem to confirm that delayed tooth eruption is common but sporadic in children with DS.

Earlier literature refers to DS as "mongolism," but the use of this term is inappropriate according to Schreiner and it may be insulting to the affected families.[60] DS occurs very early in embryonic development, possibly



fl G. 9-19. Series of radiographs demonstrating the successful treatment of delayed eruption of a first permanent molar. A, The first permanent molar has erupted on the right side. **B,** The left first permanent molar remains embedded in bone and is probably ankylosed. C, Soft tissue and bone have been removed, and surgical cement has been placed over the unerupted tooth. D, Within 3 months the first permanent molar has moved occlusally. **E,** The lingual arch and distal extension hold the surgical cement in position and prevent continued eruption of the opposing molar.

Continued

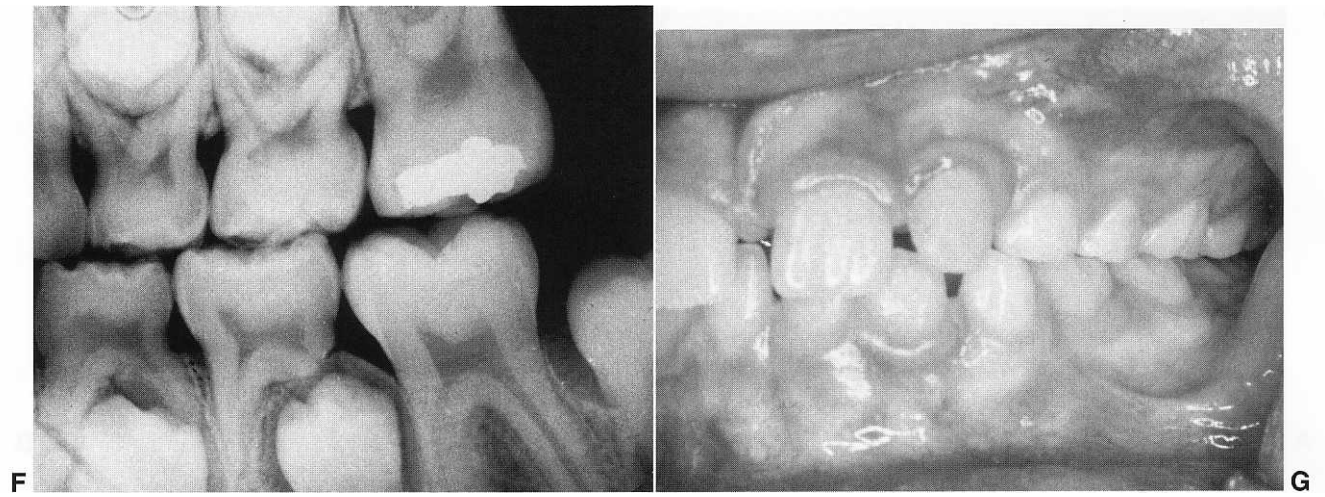


FIG 9-19, tout[**F** and **G,** The first permanent molar has erupted, and the occlusion is good. Notice the progressive resorption of the distal root of the mandibular second primary molar.

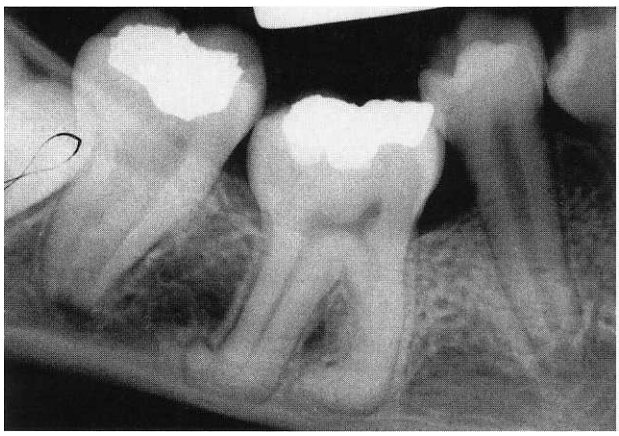


FIG . 9-20. Ankylosed first permanent molar.

during the first cell divisions. Anomalies of the eye and external ear are seen, and congenital heart defects are often present. The occurrence of DS is frequently related to maternal age. Benda reported the frequency of DS to be approximately 1.5 per 1000 births for mothers in the 18- to 29-year-old age group." The frequency increases for maternal ages of 30 years and higher, reaching 29 per 1000 in the 40-year-old and older age group and a high of 91 per 1000 in the 44-year-old age group.

The cause of DS is trisomy 21, that is, the presence of three number 21 chromosomes rather than the normal two (diploid). The diagnosis of DS in a child is not usually difficult to make because of the characteristic facial pattern (Fig. 9-22). The orbits are small, the eyes slope upward, and the bridge of the nose is more depressed than normal. Cohen, in a study of 194 children with DS, reported that 54% demonstrated anomalies in the formation of the external ear, characterized by outstanding "lap" ear with flat or absent helix." Mental retardation is another characteristic finding, with most children in the mild to moderate range of disability (see Table 3-3).

Landau made a cephalometric comparison of children with DS and their normal siblings."[3] Retardation in the growth of the maxillae and mandible was evident in those with DS. Both the maxillae and mandible were positioned anteriorly under the cranial base. The upper facial height was found to be significantly smaller. The midface was also found to be small in the vertical and horizontal dimensions. The smaller jaws contribute to a tendency for protrusion of the tongue and dental crowding, both of which may compromise good occlusion development. The tongue also tends to be larger than normal.

Many children with DS have chronic inflammation of the conjunctiva and a history of repeated respiratory tract infections. The use of antibiotics has reduced the incidence of chronic infection and has resulted in fewer deaths from infection.

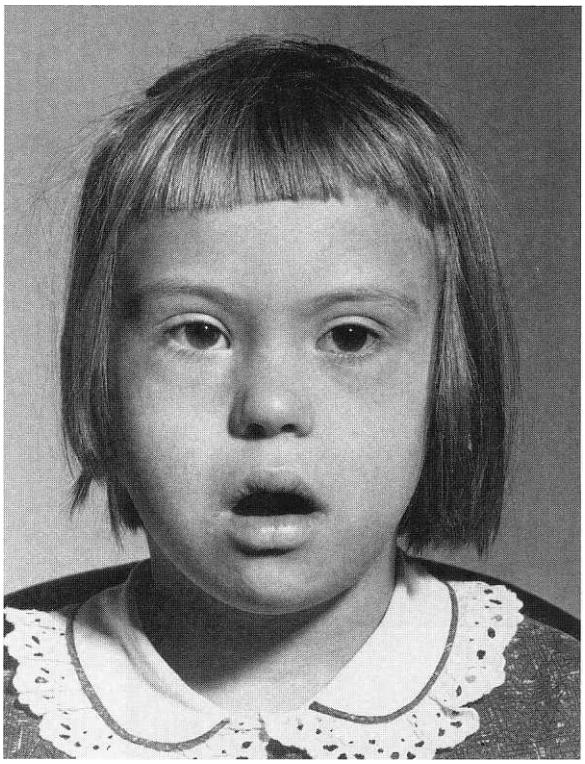
Tannenbaum and also Baer and Benjamin observed that the prevalence and severity of periodontal disease in children with DS are much higher than the norm."'" A high prevalence of necrotizing ulcerative gingivitis was also observed. After reviewing the available literature Cichon, Crawford, and Grimm concluded that individuals with DS have a higher prevalence of periodontal disease than otherwise normal, age-matched control groups and other mentally disabled patients of similar age distribution.`" Furthermore the reports of exaggerated immunoinflammatory responses of the tissues in DS patients cannot be explained by poor oral hygiene alone and may be the result of impaired cell-mediated and humoral immunities and deficient



[III Ankylosis by inostosis. A, A mesiodens has delayed the eruption of the maxillary right permanent central incisor. **B,** The primary incisors and the mesiodens were removed. During the surgical removal of the mesiodens, there was apparently damage to the enamel epithelium. C, There is evidence of resorption of the enamel of the unerupted incisor and ankylosis of the tooth. **D,** The left central incisor crown sustained a fracture and pulp exposure. A calcium hydroxide pulpotomy was successfully performed, which resulted in continued root development.

phagocytic systems. Cichon et al's study of 10 DS patients aged 20 to 31 years demonstrated that the young age of onset, the severe destruction, and the pathogenesis of disease in the periodontal tissues were consistent with a juvenile periodontitis disease pattern.

Morinushi, Lopatin, and Van Poperin obtained blood samples and conducted gingival health assessments of 75 individuals with DS aged 2 to 18 years." The extent of gingival inflammation and the antibody titers of the DS subjects suggested that colonization of certain pathogenic organisms for periodontal disease had occurred



[I **G 9-22.** Child with Down syndrome, 8 years of age. *(Courtesy Dr. Mace Landau.)*

before 5 years of age. The prevalence and extent of gingivitis was significantly higher than in normal children. The antibody titers also suggested that colonization of additional pathogenic organisms increased with age. The authors believe that there are abnormalities in the systemic defenses that are responsible for the early onset of disease in the DS subjects. Similarly, Carlstedt et al have demonstrated significantly higher oral colonization with *Candida albicans in* DS children compared with an age- and sex-matched control group." They believe that abnormalities of the immune response in DS children are responsible for their greater susceptibility to oral mucosal disease.

Dental caries susceptibility is usually low in those with DS. This finding has been reported by Johnson, Young, and Gallios, who noted a much lower dental caries incidence in both the primary and the permanent dentition.[69] Brown and Cunningham found in a study of DS children that 44% were caries free.[70] Shapira and Stabholz successfully demonstrated caries reduction and improved periodontal health during a 30-month period after initiating a comprehensive preventive oral health program for 20 children with DS.[71]

Although some children with low cognitive ability are unmanageable for dental procedures, most are pleasant, cheerful, affectionate, and well behaved. They can often be managed in the dental office in a conventional manner. The possibility of reduced resistance to infection should be considered in the dental management of the child with DS.

CLEIDOCRANIAL DYSPLASIA

A rare congenital syndrome that has dental significance is cleidocranial dysplasia (CCD), which has also been referred to as *cleidocranial dysostosis, osteodentin dysplasia, mutational dysostosis,* and *Marie-Sainton syndrome.* Transmission of the condition is by either parent to a child of either sex, so that the disorder thus follows a true mendelian dominant pattern. CCD can also occur sporadically with no apparent hereditary influence and with no predilection for race. The diagnosis is based on the finding of an absence of clavicles, although there may be remnants of the clavicles, as evidenced by the presence of the sternal and acromial ends. The fontanels are large, and radiographs of the head show open sutures, even late in the child's life. The sinuses, particularly the frontal sinus, are usually small.

Richardson and Deussen performed cephalometric analyses of 17 patients with CCD.'- They found that, on average, the patients exhibited mandibular prognathism caused by increased mandibular lengths and short cranial bases. The maxillae tended to be short vertically but not anteroposteriorly. Somewhat similar findings have been reported by Jensen and Kreiborg in their study of 22 children with CCD."

The development of the dentition is delayed. Complete primary dentition at 15 years of age resulting from delayed resorption of the deciduous teeth and delayed eruption of the permanent teeth is not uncommon (Fig. 9-23). One of the important distinguishing characteristics is the presence of supernumerary teeth. Some children may have only a few supernumerary teeth in the anterior region of the mouth; others may have a large number of extra teeth throughout the mouth. Even with removal of the primary and

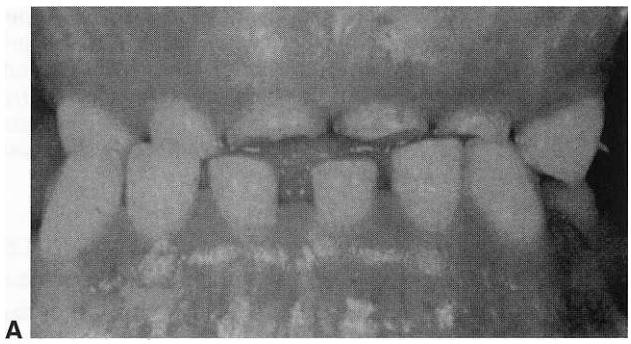


FIG **9-23.** Cleidocranial dysplasia. **A,** A Primary dentition is still present at 15 years of age. *Continued*



!!U S-23 cont'd B, Delayed dentition and the presence of many supernumerary teeth. C, Removal of supernumerary teeth in the maxillary arch caused irregular and delayed eruption of some of the permanent teeth.

supernumerary teeth, eruption of the permanent dentition, without orthodontic intervention, is often delayed and irregular. Other reports by Jensen and Kreiborg, based on their experiences and longitudinal study of 19 patients with CCD, provide information to help clinicians predict the location and time of onset of formation of supernumerary teeth. This information should help the clinician optimally time the surgical treatment(s).[74'"]

Hutton, Bixler, and Garner have reported the successful dental management of a patient with CCD over a 15-year period.'[6] The patient was first seen at 2 years of age. Treatment consisted of timed extractions of primary and supernumerary teeth and conservative uncovering of the permanent teeth. The surgical procedures were planned according to progressive radio-graphic evidence of the development of the permanent teeth. This management results in a nearly normal but slightly delayed eruption sequence. Orthodontic treatment was begun at 14 years of age, and by 16 years of age the patient displayed acceptable occlusion and normal vertical dimension, root development, and periodontal bone support.

Learning from their experiences with the long-term management of 16 patients with CCD, Becker et al advocate cooperative efforts by clinicians from the disciplines of pediatric dentistry, oral and maxillofacial surgery, and orthodontics and dentofacial orthopedics.' The pediatric dentist serves as the coordinator of over-all oral health care and disease prevention during an extended treatment regimen that usually includes two surgical interventions and three stages of orthodontic surgery.

Delayed eruption has also been reported in other forms of osteopetroses.

HYPOTHYROIDISM

Hypothyroidism is another possible cause of delayed eruption. Patients in whom the function of the thyroid gland is extremely deficient have characteristic dental findings.

Congenital Hypothyroidism (Cretinism). Hypothyroidism occurring at birth and during the period of most rapid growth, if undetected and untreated, causes mental deficiency and dwarfism. This condition was referred to as *cretinism* in earlier medical and dental literatures. Congenital hypothyroidism is the result of an absence or underdevelopment of the thyroid gland and insufficient levels of thyroid hormone (Fig. 9-24). Today it is routinely diagnosed and corrected at birth because of mandatory blood screening of newborn infants. An inadequately treated child with congenital hypothyroidism is a small and disproportionate person, with abnormally short arms and legs. The head is disproportionately large, although the trunk shows less deviation from the norm. Obesity is common.

Without adequate hormonal therapy the dentition of the child with congenital hypothyroidism is delayed in all stages, including eruption of the primary teeth, exfoliation of the primary teeth, and eruption of the permanent teeth. The teeth are normal in size but are crowded in jaws that are smaller than normal. The tongue is large and may protrude from the mouth. The abnormal size of the tongue and its position often cause an anterior open bite and flaring of the anterior teeth. The crowding of the teeth, malocclusion, and mouth breathing cause a chronic hyperplastic type of gingivitis.

Although untreated congenital hypothyroidism is rare, even in developing countries, Loevy, Aduss, and Rosenthal published a case report documenting the condition discovered in a 19-year-old boy." The patient presented with a complete caries-free primary dentition and partially erupted maxillary first permanent molars. All primary teeth showed some abrasion. At a subsequent oral examination 1 year and 9 months after appropriate L-thyroxine therapy was initiated, several primary teeth had exfoliated, permanent incisors and first molars had erupted, and radiographs showed additional development of other permanent teeth.

Juvenile Hypothyroidism (Acquired Hypothyroidism). Juvenile hypothyroidism results from a malfunction of the thyroid gland, usually between 6 and 12 years of age. Because the deficiency occurs after the period of rapid growth, the unusual facial and body pattern characteristic of a person with congenital hypothyroidism is not present. However, obesity is evident to a lesser degree. In the untreated case of juvenile hypothyroidism, delayed exfoliation of the primary teeth and delayed eruption of the permanent teeth are characteristic. A child with a chronologic age of 14 years may have a dentition in a stage of development comparable with that of a child 9 or 10 years of age (Fig. 9-25).

HYPOPITUITARISM

A pronounced deceleration of the growth of the bones and soft tissues of the body will result from a deficiency in secretion of the growth hormone. Pituitary dwarfism is the result of an early hypofunction of the pituitary gland. Again, early diagnosis is routine because of the mandatory blood screening of newborn infants for congenital hypothyroidism.

An individual with pituitary dwarfism is well proportioned but resembles a child of considerably younger chronologic age (Fig. 9-26). The dentition is essentially normal in size.

Delayed eruption of the dentition is characteristic. In severe cases the primary teeth do not undergo

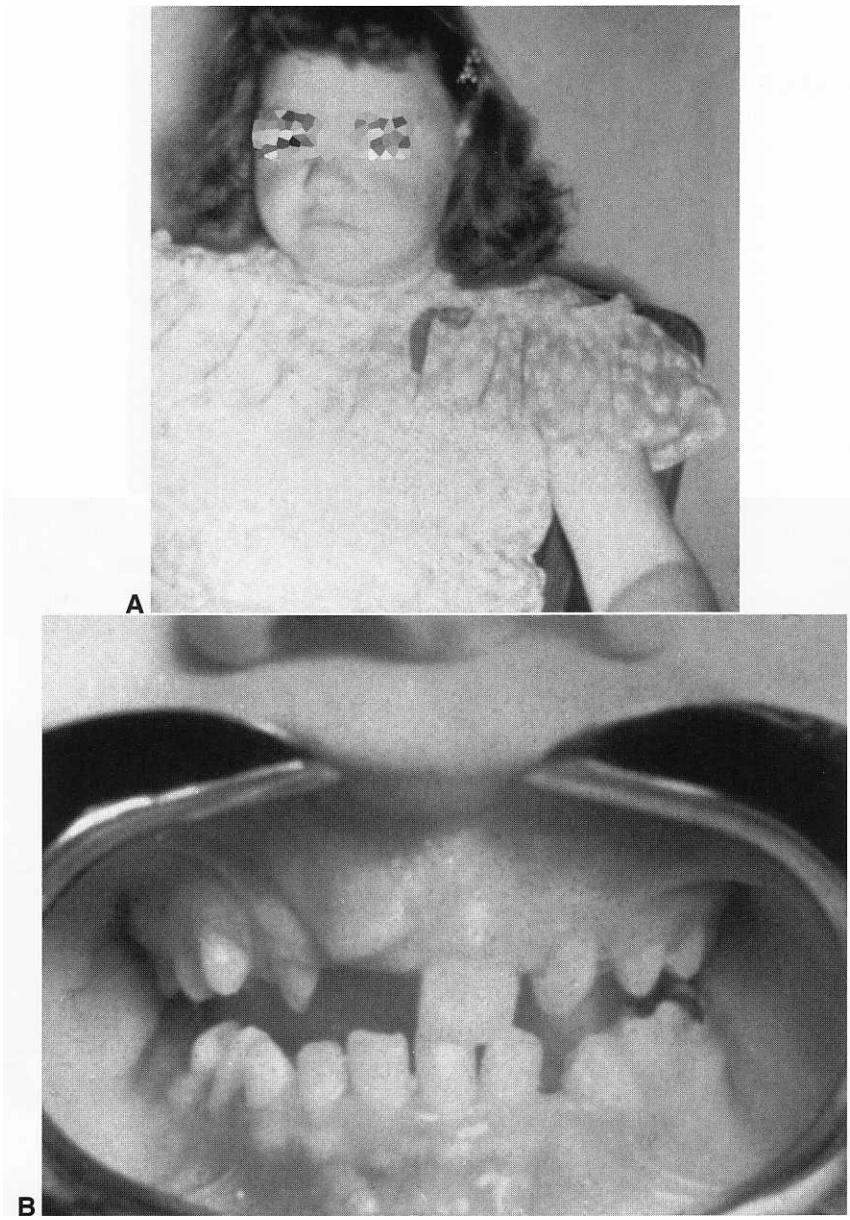


FIG 9-24. **A,** A 24-year-old patient with congenital hypothyroidism. **B,** Dentition is greatly delayed. With the administration of thyroxine, the eruption of the permanent teeth was accelerated. *(Courtesy Dr. David F. Mitchell.)*

resorption but instead may be retained throughout the life of the person. The underlying permanent teeth continue to develop but do not erupt. Extraction of the deciduous teeth is not indicated, since eruption of the permanent teeth cannot be assured. Some degree of cognitive disability often occurs.

ACHONDROPLASTIC DWARFISM

Achondroplastic dwarfism, also diagnosed at birth, demonstrates a few characteristic dental findings. Growth of the extremities is limited because of a lack of calcification in the cartilage of the long bones. Stature improvements have been reported with surgical lengthening of the limbs and also with growth hormone therapy. The head is disproportionately large, although the trunk is normal in size. The fingers may be of almost equal length, and the hands are plump. The fontanels are open at birth. The upper face is underdeveloped, and the bridge of the nose is depressed.

Although the etiology of achondroplastic dwarfism is unknown, it is clearly an autosomal dominant disorder although sporadic spontaneous mutations occur.



FIG 19-25. **A,** A 14-year-old girl who was diagnosed as having juvenile hypothyroidism. **B,** The occlusion was essentially normal but was delayed in its development. C, Delayed development of the teeth in juvenile hypothyroidism. The maxillary midline supernumerary tooth is a coincidental finding.

There is some evidence that the condition is more likely to occur when the ages of the parents differ significantly. In contrast to DS, the increased age of the father may be related to the occurrence of the condition.

Deficient growth in the cranial base is evident in many individuals with achondroplastic dwarfism. The maxilla may be small, with resultant crowding of the teeth and a tendency for open bite. A chronic

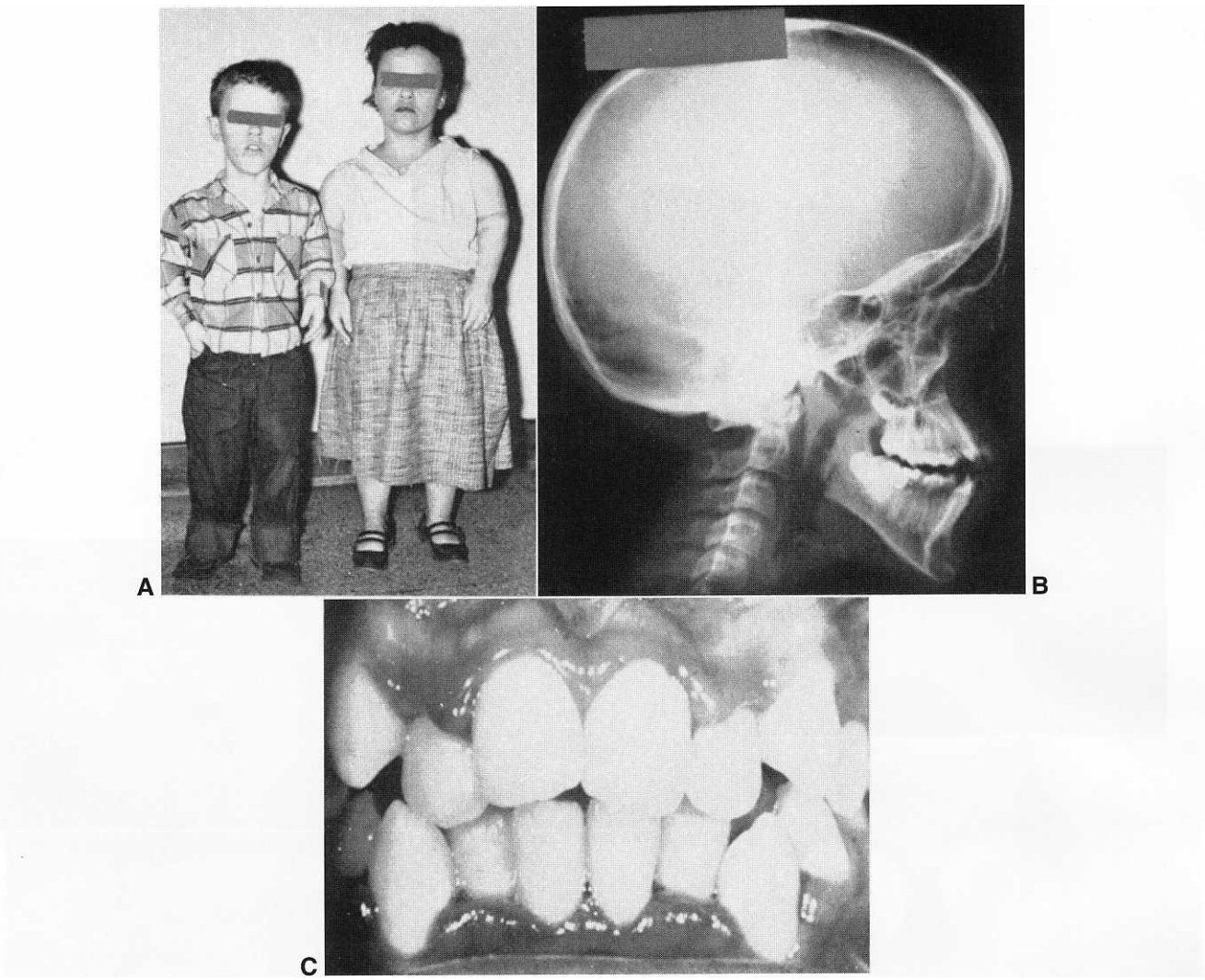


l **I G 9-26.** A 28-year-old woman diagnosed as having hypopituitary dwarfism. **A,** Complete primary dentition at 28 years of age. The first permanent molars have erupted. **B,** The roots of the primary teeth have not been resorbed to an appreciable degree, though some permanent teeth show complete development.

gingivitis is usually present. However, this condition may be related to the malocclusion and crowding of the teeth. In the patient shown in Fig. 9-27, the development of the dentition was slightly delayed.

OTHER CAUSES

Delayed eruption of the teeth has been linked to other disorders, including fibromatosis gingivae (see Chapter 20), Albright hereditary osteodystrophy,



[I G. **9-27. A,** A 14-year-old boy with achondroplastic dwarfism and his mother. Growth of the extremities is limited in both. **B,** The upper face is greatly underdeveloped. C, The arch length is inadequate, and the teeth are crowded. (A *and* **B** *courtesy Dr. Ralph E. McDonald. C from Shafer W, Hine MK, Levy BM: A textbook of oral pathology, Philadelphia, 1958, WB Saunders.)*

chondroectodermal dysplasia (Ellis—van Creveld syndrome), de Lange syndrome, frontometaphyseal dysplasia, Gardner syndrome, Goltz syndrome, Hunter syndrome, incontinentia pigmenti syndrome (Bloch-Sulzberger syndrome), Maroteaux-Lamy mucopoly-saccharidosis, Miller-Dieker syndrome, progeria syndrome (Hutchinson-Gilford syndrome), and familial hypophosphatemia.

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CHAPTER

10

Dental Caries in the Child and Adolescent

RALPH E. McDONALD

DAVID R. AVERY

GEORGE K. STOOKEY

انتشارات
شایان نمودار

CHAPTER OUTLINE

ETIOLOGY OF DENTAL CARIES
CARIES PREVALENCE IN PRESCHOOL CHILDREN
CARIES PREVALENCE IN SCHOOLCHILDREN
RAMPANT DENTAL CARIES
EARLY CHILDHOOD CARIES, SEVERE EARLY CHILDHOOD CARIES, NURSING CARIES, BABY BOTTLE TOOTH DECAY
ADDITIONAL FACTORS KNOWN TO INFLUENCE DENTAL CARIES
- Saliva
  - *Salivary deficiency*
  - *Determination of salivary flow*
  - *Viscosity of saliva*
- Socioeconomic Status
- Anatomic Characteristics of the Teeth
- Arrangement of the Teeth in the Arch
- Presence of Dental Appliances and Restorations
- Hereditary Factors

EARLY DETECTION OF DISEASE ACTIVITY Infrared
- Laser Fluorescence (DIAGNOdent) Digital
- Imaging Fiber Optic Transillumination (DIFOTI)
- Quantitative Light Fluorescence
  - *Applications*

PREDICTION OF PATIENTS' RISK OF FUTURE DISEASE (RISK ASSESSMENT)
CONTROL OF DENTAL CARIES
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- Reduction of Dental Plaque (and Microorganisms) with Good Oral Hygiene Procedures
- Use of Fluorides and Topical Antimicrobial Agents
  - *Communal water fluoridation Fluoride-containing dentifrices Topical fluorides in the dental office Topical fluorides for the general anesthesia*
    - *patient*
  - *Home fluoride mouthrinses and gels*
  - *Dietary fluoride supplements*
  - *Combinations of fluoride therapies*
  - *Chlorhexidine and thymol*
  - *Povidone iodine*
  - *Xylitol*
- Restorative Dentistry in the Control Program
- Pit and Fissure Sealants
- Caries Vaccine
- Dental Caries Activity Tests

DENTIST'S ROLE IN THE CARIES CONTROL PROGRAM

We know that good oral health is an integral component of good general health. Although enjoying good oral health includes more than just having healthy teeth, many children have inadequate oral and general health because of active and uncontrolled dental caries. According to the first-ever United States Surgeon General's report on oral health in America published in May 2000, dental caries is the single most common chronic childhood disease.[1] Dental caries is five times more common than asthma and seven times more common than hay fever. Furthermore, as Edelstein and Douglass noted, dental caries is not self-limiting, like the common cold, nor amenable to treatment with a simple course of antibiotics, like an ear infection.[1] After analyzing the National Health Interview Survey data from 1993 through 1996, Newacheck et al concluded that dental care is the most prevalent unmet health need among American children.[1] Much other available data verify that we learned a great deal during the twentieth century about preventing dental caries, but other variables that contribute to the spread of the disease among many people in the world continue to thwart our efforts to eliminate this major health problem. Although effective methods are known for prevention and management of the disease, the unmet need for treatment, especially in children, does not seem to be diminishing. Gift has estimated that 51 million school hours per year are lost in the United States, because of dental-related illness.[1]

The National Institutes of Health sponsored a Consensus Development Conference on Diagnosis and Management of Dental Caries Throughout Life in March 2001.[5] Thirty-four papers were presented by recognized experts on dental caries. The October 2001 issue of the *Journal of Dental Education* published the entire proceedings of that conference. Only a few of the papers from the conference are specifically cited in the references for this chapter. However, the complete journal issue provides many good updates on the diagnosis and management of dental caries.

ETIOLOGY OF DENTAL CARIES

For as long as the science of dentistry has existed, there has been theorizing about the cause of dental caries. Today, all experts on dental caries generally agree that it is an infectious and communicable disease and that multiple factors influence the initiation and progression of the disease. The disease is recognized to require a host (tooth in the oral environment), a dietary substrate, and aciduric bacteria.[1] The saliva (also considered a host component), the substrate, and the bacteria form a biofilm (plaque) that adheres to the tooth surface. Over time the presence of the substrate serves as a nutrient for the bacteria, and the bacteria produce acids that can demineralize the tooth. The flow, dilution, buffering, and remineralizing capacity of saliva are also recognized to be critical factors that affect, and in some ways regulate, the progression and regression of the disease. If the oral environment is balanced and favorable, saliva can contribute to strengthening of the tooth by supplying the components known to help build strong apatite structure. If the oral environment is unfavorable (too much acid is produced too often), an adequate flow of saliva can help dilute and buffer the acid, and thus slow the rate of damage to the tooth or even repair it. The critical pH for dissolution of enamel has been shown to be about 5.5. Once the process reaches dentin, dissolution can occur at a considerably higher pH. In addition, we know of many anatomic, behavioral, dietary, genetic, social (and cultural), socioeconomic, and therapeutic variables that can significantly influence the level of caries activity favorably or unfavorably.

We recognize dental caries as a preventable disease. Furthermore we know that the disease typically begins in enamel and progresses slowly in the early stages of the process. Rampant caries is an exception to the typical course and is discussed later. Cavitation of the tooth structure is quite a late stage of the disease. Prior to cavitation, the progress of the disease may be arrested and/or reversed if a favorable oral environment can be achieved. Even after cavitation occurs, if the pulp is not yet involved and if the cavitated area is open enough to be self-cleansing ("plaque-free"), the caries process can halt and become an "arrested lesion." Arrested lesions typically exhibit much coronal destruction, but the remaining exposed dentin is hard and usually very dark, there is no evidence of pulpal damage, and the patient has no pain. We also must emphasize that treating a carious tooth by providing a restoration does not cure the disease. If the unfavorable oral environment that caused the cavity persists, so will the disease, and more restorations will be required in time. Treating the oral infection by reducing the number of cariogenic microorganisms and establishing a favorable oral environment to promote predominantly remineralization of tooth structure over time will stop the caries process and cure the disease. Curing the disease currently requires modifications by the patient and/or caretaker and relies on their compliance in making the necessary modifications. Research efforts are on-going to find a feasible method of achieving caries immunity that would be far less dependent on patient compliance.

Studies by Orland[1] and by Fitzgerald, Jordan, and Achard[11] demonstrated that dental caries will not occur in the absence of microorganisms. Animals maintained in a germ-free environment did not develop caries even when fed a high-carbohydrate diet. However, dental

caries did develop in these animals when they were inoculated with microorganisms from caries-active animals and then fed cariogenic diets.

A number of microorganisms can produce enough acid to decalcify tooth structure, particularly aciduric streptococci, lactobacilli, diphtheroids, yeasts, staphylococci, and certain strains of sarcinae. *Streptococcus mutans* has been implicated as one of the major and most virulent of the caries-producing organisms. Consequently *S. mutans* has been targeted in a large share of research. Loesche conducted an extensive review of the literature regarding the etiology of caries. He concluded that the evidence suggests that S. *mutans,* possibly *Streptococcus sobrinus,* and lactobacilli are human odontopathogens. He stated that aciduricity appears to be the most consistent attribute of S. *mutans* and is associated with its cariogenicity. He also observed that other aciduric species such as *S. sobrinus* may be more important in smooth-surface decay and are perhaps associated with rampant caries. Loesche concluded the review with the suggestion that treatment strategies that interfere with the colonization of S. *mutans* may have a profound effect on the incidence of caries in humans.' As caries research proceeds, there seems to be increasing evidence that disease may result from a group of microbial species in the tooth-adhering biofilm. Currently, which combinations of organisms are most blameworthy is not clear.

Wan et al have published a series of three papers that report on 111 infants whom they observed to 2 years of age. They found S. *mutans* colonization in infants as young as 3 months, and over 50% of the predentate infants were infected by 6 months of age. By 24 months of age, 84% of the children harbored the bacteria.[70-'2] Investigations by Davey and Rogers'' and by Berkowitz and Jones'[s] have confirmed that S. *mutans is* transmitted orally from mother to infant, whereas Brown, Junner, and Liew'' have demonstrated a relationship between the numbers of S. *mutans* present in mothers and infants. Research by Kohler, Andreen, and Jonsson demonstrated that reducing the numbers of oral *S. mutans* in mothers delayed the colonization of the organisms in the mouths of their children.'[6] Their findings also showed that 52% of the children who carried S. *mutans* at 3 years of age had caries, whereas only 3% of the children without demonstrable S. *mutans* had caries at the same age. In 1988, these same investigators reported that, the earlier the colonization of *S. mutans* in the mouths of children, the higher the caries prevalence at 4 years of age.'' Caufield, Cutter, and Dasanayake suggested the possibility of a "window of infectivity" between 19 and 33 months of age during which most children acquire the cariogenic organisms.'[8] The mother was the most common source of transmission of the bacteria to the child. In a group of 122 children 6 to 24 months of age, Mohan et al found oral mutans streptococci colonization in 20% of the children under 14 months of age.'`'' In addition, logistic regression models that controlled for both age and number of teeth indicated that children who consumed sweetened beverages in their baby bottle were four times more likely to have mutans streptococci than children who only consumed milk. Although investigations to elucidate how and when mutans streptococci are transmitted to children are continuing, essentially all experts agree that the earlier transmission occurs, the higher the caries risk.

The acids that initially decalcify the enamel have a pH of 5.5 to 5.2 or less and are formed in the plaque material, which has been described as an organic nitrogenous mass of microorganisms firmly attached to the tooth structure. This film, which exists primarily in the susceptible areas of the teeth, has received a great deal of attention. Considerable emphasis is currently being given to plaque and its relationship to oral disease. Methods of chemical plaque control are being investigated. The method that has received the most attention during the past decade is the use of antimicrobial agents whose action is selective against certain types of microorganisms, including S. *mutans.* Chlorhexidine and other agents are available in antimicrobial oral rinse solutions. Another approach involves the use of monomolecular layers on the tooth surface that prevent the adherence of microorganisms. Perhaps we will learn how to make enamel resistant to bacterial colonization (plaque formation) and consequently reduce both caries and gingival disease.

The acids involved in the initiation of the caries process are normal metabolic by-products of the microorganisms and are generated by the metabolism of carbohydrates. Because the outer surface of enamel is far more resistant to demineralization by acid than is the deeper portion of enamel, the greatest amount of demineralization occurs 10 to 15 μm beneath the enamel surface (Fig. 10-1). The continuation of this process results in the formation of an incipient subsurface enamel lesion that is first observed clinically as a so-called white spot. Unless the demineralization is arrested or reversed (remineralization) the subsurface lesion will continue to enlarge, with the eventual collapse of the thin surface layer and the formation of a cavitated lesion.

Remineralization of incipient subsurface lesions may occur as long as the surface layer of the enamel remains intact. Saliva, which is supersaturated with calcium and phosphate and has acid-buffering capability, diffuses into plaque, where it neutralizes the microbial acids and repairs the damaged enamel. The time required for remineralization to replace the hydroxyapatite lost during demineralization is determined by the age of the plaque, the nature of the carbohydrate consumed, and the presence or absence of fluoride. For example, it has

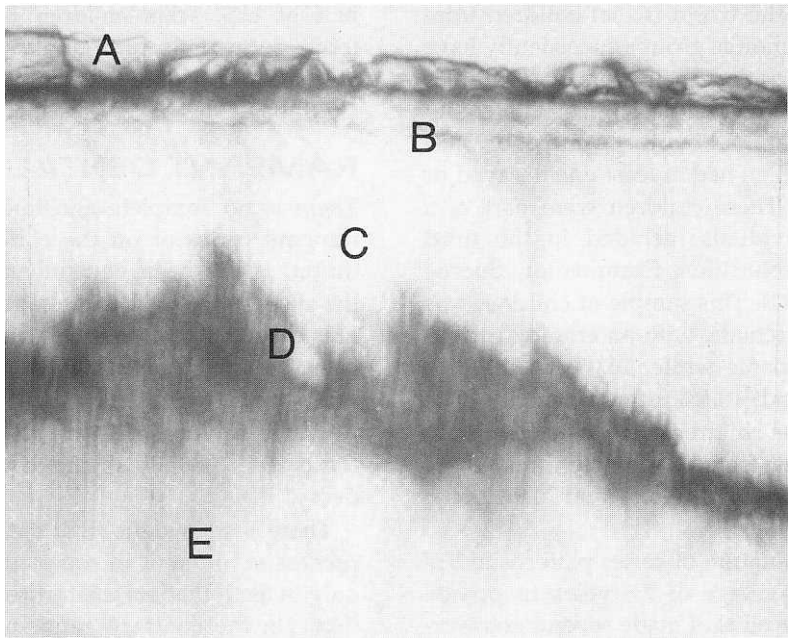


FIG **10-1.** Polarized light appearance of natural subsurface caries lesion that has imbibed quinoline (100x). A, Calculus, **B,** Intact surface area (surface zone IV). C, Demineralized area (body of lesion III). D, Remineralized area (dark zone II). **E,** Advancing front (translucent zone I)
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been suggested that, in the presence of dental plaque that has developed for 12 hours or less, the enamel demineralization resulting from a single exposure to sucrose will be remineralized by saliva within about 10 minutes. In contrast, a period of at least 4 hours is required for saliva to repair the damage to enamel resulting from a similar exposure to sucrose in the presence of dental plaque that is 48 or more hours old. As is noted later in this chapter, the presence of fluoride has a profound effect on the remineralization process; not only does fluoride greatly enhance the rate of remineralization of enamel by saliva but it also results in the formation of a fluorhydroxyapatite during the process, which increases the resistance of the remineralized enamel to future attack by acids. Fluoride also has antimicrobial effects.

Thus the development of dental caries may be considered as a continuous dynamic process involving repeating periods of demineralization by organic acids of microbial origin and subsequent remineralization by salivary components (or therapeutic agents), but in which the overall oral environment is imbalanced toward demineralization. Several factors influence the degree of vulnerability of the tooth.

CARIES PREVALENCE IN PRESCHOOL CHILDREN

Weddell and Klein examined 441 children who ranged in age from 6 to 36 months and resided in a community with water fluoridation.[20] They found dental caries in 4.2% of the children 12 to 17 months of age, 19.8% of those 24 to 29 months of age, and 36.4% of those 30 to 36 months of age. Children in the middle and middle-low socioeconomic groups showed a trend toward higher caries frequencies. Edelstein and Tinanoff found that 30. 5% of 200 preschool children had caries detectable by visual or radiographic examination.[21] These children were recruited from a private pediatric dental office and ranged in age from 5 months to 5 years 11 months (mean, 3 years 8 months).

Douglass et al determined the caries prevalence of 3- to 4-year-old children from a fluoridated community in Connecticut who were enrolled in the same Head Start program.[22] They compared the caries prevalence in 517 children enrolled in 1999 with that in 311 children enrolled in 1991. They found a caries prevalence of 38% in the children enrolled in 1999 and 49% in those enrolled in 1991. They noted, however, that the children enrolled in 1999 had a greater severity of maxillary anterior caries. Tang et al performed dental caries examinations on 5171 preschool children recruited from public health assistance programs in Arizona.[23] They found caries in 6.4% of 1-year-olds, nearly 20% of 2-year-olds, 35% of 3-year-olds, and 49% of 4-year-old children in the study.

In general, other reports of caries prevalence among children in various parts of the world show rates that seem to be comparable to those cited here. Another common element of caries prevalence in the United

States and throughout the world is that children from families in low socioeconomic groups consistently have greater caries prevalences than their peers from families at a higher socioeconomic level. Vargas, Crall, and Schneider reported that 27.4% of a sample of 3889 children 2 to 5 years of age had at least one decayed or filled primary tooth.- These children were part of a larger sample of individuals included in the third National Health and Nutrition Examination Survey (NHANES III, 1988-1994). This sample of children was 51.4% male and 48.6% female, with an ethnic distribution of 64.1% non-Hispanic white, 16.0% black, 9.5% Mexican American, and 10.4% other ethnicity. The family incomes for these children were distributed into four groups categorized from low to high, and these groups comprised 27.9%, 25.5%, 21.6%, and 24.9% of the sample, respectively.

In a longitudinal evaluation of caries patterns in 317 children followed an average of 7.8 years in private dental practices, Greenwell et al made several noteworthy discoveries." They found that 84% of the children who were caries free in the primary dentition remained caries free in the mixed dentition. Children with pit and fissure caries in the primary dentition were more likely to develop smooth-surface caries of primary teeth than the caries-free children. Fifty-seven percent of the children with proximal lesions in primary molars in the primary dentition developed additional primary molar proximal lesions in the mixed dentition. Children with faciolingual decay (nursing caries) were at the highest risk of any group for developing additional carious lesions. These investigators also discovered levels of caries susceptibility in children that can be characterized as caries free, pit and fissure caries, and proximal molar caries patterns.

CARIES PREVALENCE IN SCHOOLCHILDREN

The report by Vargas, Crall, and Schneider provides additional representative data for schoolchildren as well.' Their report revealed that 61.0% of the sample of children 6 to 12 years of age had at least one decayed or filled primary tooth. Furthermore, in the sample of 4116 children 6 to 14 years of age, 40.0% had at least one decayed or filled permanent tooth. Of the 1383 children 15 to 18 years of age, 89.8% had at least one decayed or filled permanent tooth. The ethnic and family income distributions for children in these different age groups were comparable to those outlined in detail for the preschool children. This information, along with that in many other published reports, clearly indicates that managing the disease of dental caries among children remains a formidable task despite the advances we have made in our various preventive programs. Edelstein and Douglass have noted, "The popular statement that half of U.S. schoolchildren have never experienced tooth decay fails profoundly to reflect the extremity and severity of this still highly prevalent condition of childhood."'

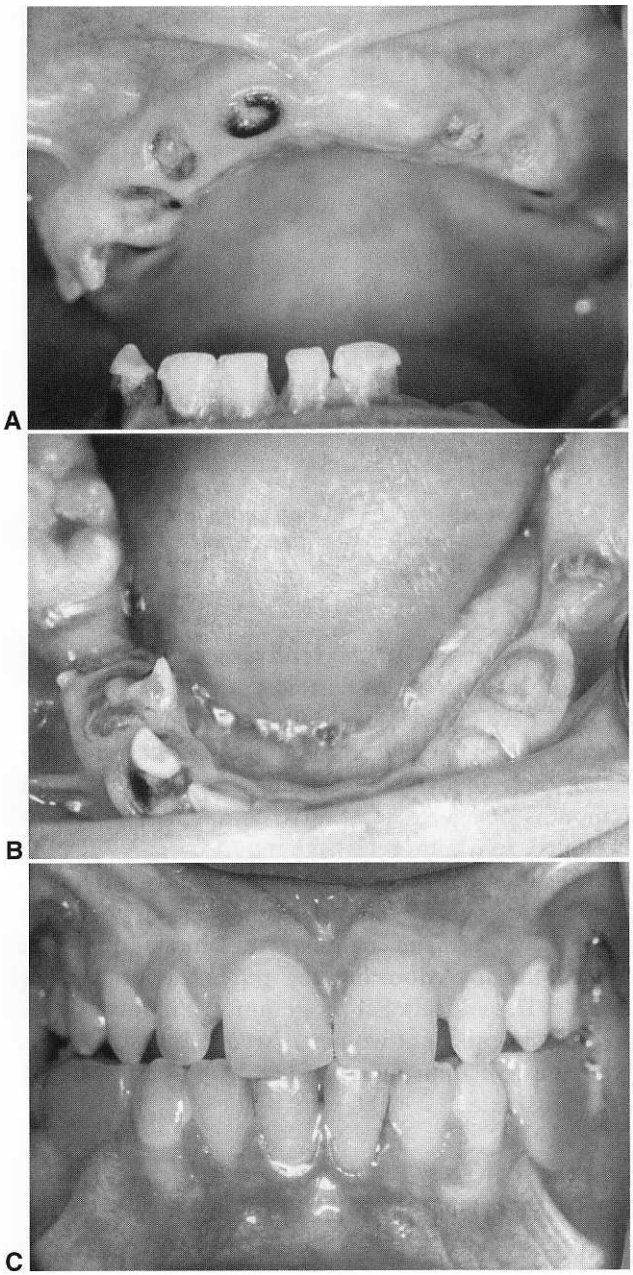
RAMPANT DENTAL CARIES

There is no complete agreement on the definition of rampant caries or on the clinical picture of this condition. It has been generally accepted, however, that the disease referred to as rampant caries is, in terms of human history, relatively new. Rampant caries has been defined by Massler as a "suddenly appearing, widespread, rapidly burrowing type of caries, resulting in early involvement of the pulp and affecting those teeth usually regarded as immune to ordinary decay."'"'

There is no evidence that the mechanism of the decay process is different in rampant caries or that it occurs only in teeth that are malformed or inferior in composition. On the contrary, rampant caries can occur suddenly in teeth that were previously sound for many years. The sudden onset of the disease suggests that an overwhelming imbalance of the oral environment has occurred, and some factor(s) in the caries process seems to accelerate it so that it becomes uncontrollable; it is then referred to as *rampant caries.*

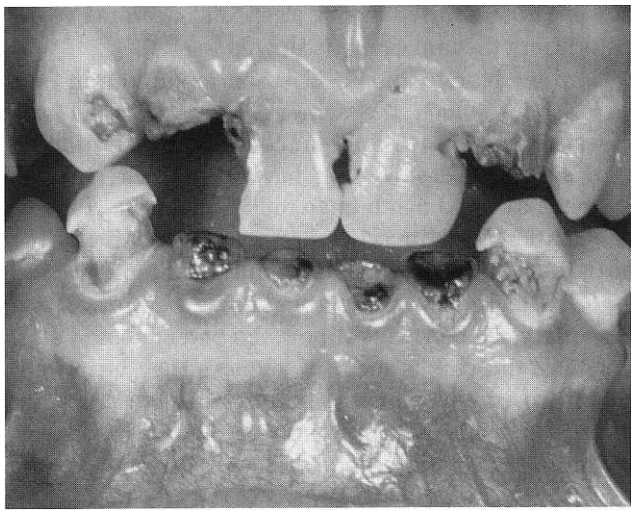
When a patient has what is considered an excessive amount of tooth decay, one must determine whether that person actually has a high susceptibility and truly rampant caries of sudden onset or whether the oral condition represents years of neglect and inadequate dental care. Young teenagers seem to be particularly susceptible to rampant caries, though it has been observed in both children and adults of all ages (Figs. 10-2 and 10-3).

There is considerable evidence that emotional disturbances may be a causative factor in some cases of rampant caries. Repressed emotions and fears, dissatisfaction with achievement, rebellion against a home situation, a feeling of inferiority, a traumatic school experience, and continuous general tension and anxiety have been observed in children and adults who have rampant dental caries. Because adolescence is often considered to be a time of difficult adjustment, the increased incidence of rampant caries in this age group lends support to this theory. An emotional disturbance may initiate an unusual craving for sweets or the habit of snacking, which in turn might influence the incidence of dental caries. On the other hand, a noticeable salivary deficiency is not an uncommon finding in tense, nervous, or disturbed persons. Indeed, various forms of stress in both children and adults, as well as various medications (such as tranquilizers and sedatives) commonly taken to help persons cope with stress, are associated with decreased salivary flow and decreased caries resistance



I **I G 10-2. A,** Rampant dental caries and evidence of dental neglect in preschool child. There has been gross destruction of the clinical crowns of the primary teeth. **B,** Recently erupted first permanent molar with an extensive carious lesion. The destruction process evident in the primary teeth at an early age would be expected to continue in the permanent teeth. C, Same patient at 10 years of age. A preventive and corrective program has maintained the permanent teeth.

caused by impaired remineralization. It is well known that radiation therapy to the head and neck often results in significantly diminished salivary function and may place patients at high risk for severe caries development.



F I G **10-3.** Teenaged patient with rampant caries. Occasionally a full-mouth extraction must be recommended if there is gross destruction with pulpal involvement.

EARLY CHILDHOOD CARIES, SEVERE EARLY CHILDHOOD CARIES, NURSING CARIES, BABY BOTTLE TOOTH DECAY

The American Academy of Pediatric Dentistry (AAPD) defines early childhood caries (ECC) as the presence of one or more decayed (noncavitated or cavitated), missing (due to caries), or filled tooth surfaces in any primary tooth in a child 71 months of age or younger. The Academy also specifies that, in children younger than 3 years of age, any sign of smooth-surface caries is indicative of severe early childhood caries (S-ECC).[27]

For many years it has been recognized that, after eruption of the primary teeth begins, excessively frequent bottle feedings and/or prolonged bottle or breast feedings is often associated with early and rampant caries. The clinical appearance of the teeth in S-ECC in a child 2, 3, or 4 years of age is typical and follows a definite pattern." There is early carious involvement of the maxillary anterior teeth, the maxillary and mandibular first primary molars, and sometimes the mandibular canines (Fig. 10-4). The mandibular incisors are usually unaffected. A discussion with the parents often reveals an inappropriate feeding pattern: the child has been put to bed at afternoon naptime and/or at night with a nursing bottle holding milk or a sugar-containing beverage. The child falls asleep, and the liquid becomes pooled around the teeth (the lower anterior teeth tend to be protected by the tongue). It would seem that the carbohydrate-containing liquid provides an excellent culture medium for acidogenic microorganisms. Salivary

flow is also decreased during sleep, and clearance of the liquid from the oral cavity is slowed.

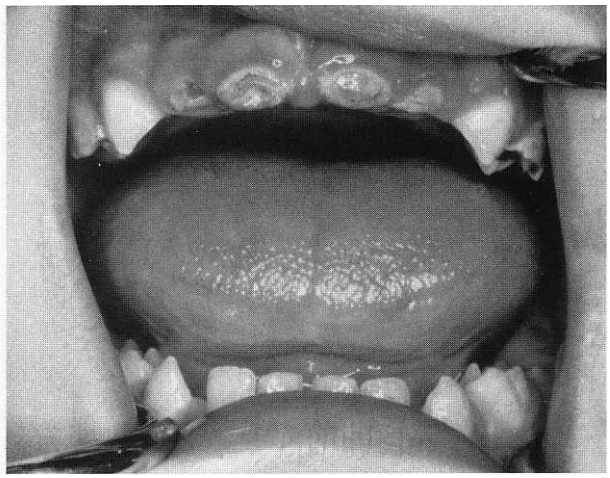
Gardner, Norwood, and Eisenson reported four case histories in which the same pattern of caries was observed, and in each child the condition was attributed to a specific breast-feeding habit.'[L] In each case the mother explained that human milk was the main source of nutrition. The investigators recommend that from birth the infant should be held while feeding. The child who falls asleep while nursing should be burped and then placed in bed. In addition, the parent should start brushing the child's teeth as soon as they erupt and should discontinue nursing as soon as the child can drink from a cup—at approximately 12 months of age.

The AAPD endorses the policy statement of the American Academy of Pediatrics (AAP) on breast-feeding and the use of human milk.[30] The AAP statement includes the acknowledgment that "breast-feeding ensures the best possible health as well as the best development and psychosocial outcomes for the infant." However, both organizations discourage extended or excessive frequency of feeding times (from the breast or bottle) and encourage appropriate oral hygiene measures for infants and toddlers.

Dilley, Dilley, and Machen observed a large number of children with prolonged nursing habit caries and concluded that there was no association between the nursing habit and family background, except that the families were predominantly from lower socioeconomic groups." All subjects demonstrated prolonged breast-feeding or bottle feeding, with milk reported to be the liquid most often used in the bottle. Parents indicated that they did not know when weaning should occur and when oral hygiene should be instituted. The authors also observed nearly symmetric caries patterns.

Hallonsten et al screened 3000 children 18 months of age for dental caries and ongoing breast-feeding.[3"] Twelve (19.7%) of the 61 children still being breast-fed had caries, while 51 (1.7%) of the 2939 children not being breast-fed had caries. The authors found that children who experience prolonged breast-feeding tend to develop unsuitable dietary habits that put them at risk for caries at an early age.

There is considerable scientific evidence from experiments in vitro and in animal models to suggest that some dairy products such as bovine milk and cheese as well as human breast milk are not cariogenic and may actually be protective to tooth structure and promote remineralization under certain conditions. Similar experiments show that many infant formulas, with refined food additives, do promote caries. These issues are discussed in more detail later. Suffice it to say here that we have much yet to learn about caries progression in both the more typical disease and this rampant form. It is prudent to counsel parents to practice good oral hygiene measures for the child and to avoid inappropriate feeding habits that are associated with S-ECC.


A

f **G 10-4. A,** Severe early childhood caries in a 20-month-old child. There is extensive carious involvement of the maxillary primary incisors and first molars.

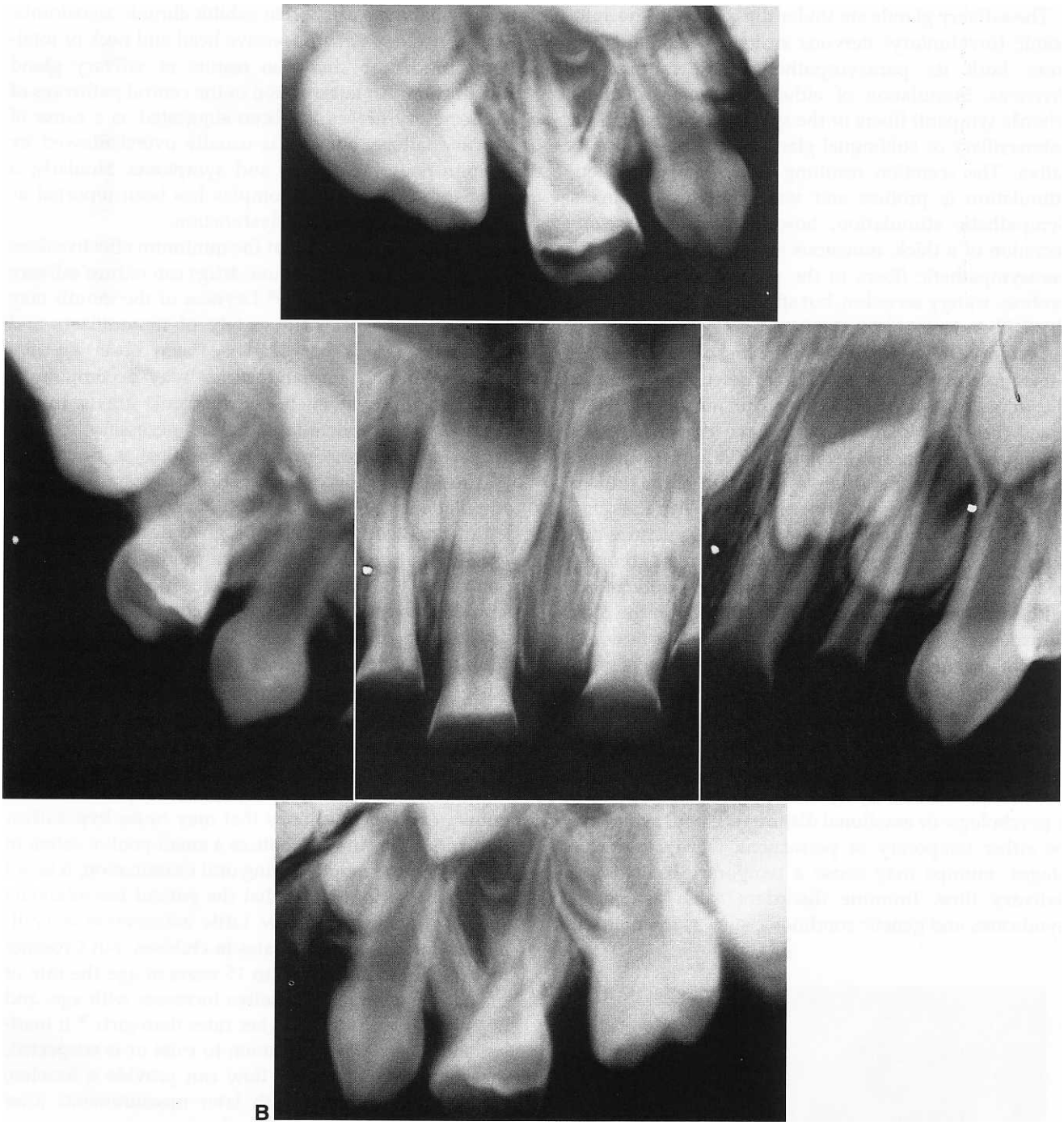
S-ECC may be prevented by early counseling of the parents. This is one reason for suggesting that children receive their first dental examination between 6 and 12 months of age, when S-ECC is not likely to have developed. In a comprehensive report prepared for the Oral Health Subcommittee of the Healthy Mothers–Healthy Babies Coalition, Ripa states, "Priority needs to be given to a major national educational program directed toward educating the public about nursing caries.' The educational program must involve direct contact with pregnant women, parents, and other caregivers in population subgroups with a high prevalence of nursing caries.

ADDITIONAL FACTORS KNOWN TO INFLUENCE DENTAL CARIES

SALIVA

Although saliva was identified in the etiology section earlier as part of the host component and thus a primary part of the caries process, the role of saliva overall is so unique and special that further discussion is warranted here regarding its influence on several aspects of the caries process that may help produce favorable environments to combat the process. Any patient with a salivary deficiency, from any cause, is at higher risk for caries activity.

It is generally accepted that the dental caries process is controlled to a large extent by a natural protective mechanism inherent within the saliva. Many properties of saliva have been investigated to learn their possible role in the caries process. Considerable importance has been placed on the salivary pH, the acid-neutralizing



f I i 10-4, contd. **B,** Radiographs of the maxillary arch. Maxillary incisors are indicated for extraction. Notice the deep caries in the first primary molars. The primary second molars have not erupted.

power, and the calcium, fluoride, and phosphorus content. It has long been suggested that in addition to these properties the rate of flow and the viscosity of saliva may influence the development of caries. The normal salivary flow aids in the solution of food debris on which microorganisms thrive. In addition, the saliva manifests a variety of antibacterial and other antiinfectious properties. All known characteristics of saliva seem somehow relevant to the process of dental caries.

Saliva is secreted by three paired masses of cells—the submaxillary, sublingual, and parotid glands. Small accessory glands are also scattered over the oral mucous membranes. Each of these has its own duct.

The salivary glands are under the control of the autonomic (involuntary) nervous system, receiving fibers from both its parasympathetic and sympathetic divisions. Stimulation of either the parasympathetic (chorda tympani) fibers or the sympathetic fibers to the submaxillary or sublingual gland causes a secretion of saliva. The secretion resulting from parasympathetic stimulation is profuse and watery in most animals. Sympathetic stimulation, however, causes a scanty secretion of a thick, mucinous juice. Stimulation of the parasympathetic fibers to the parotid gland causes a profuse, watery secretion, but stimulation of the sympathetic fibers causes no secretion.

Salivary Deficiency. One of the first descriptions of a severe salivary deficiency with its deleterious effect on the dentition was reported by Hutchinson in 1888.' Since that time many reports have emphasized the importance of a normal flow of saliva in preventing a breakdown of the dentition. A reduction in the salivary flow may be temporary or permanent. When the quantity is only moderately reduced, the oral structures may appear normal. A pronounced reduction or complete absence of saliva, however, will result in a septic mouth with rampant caries (Fig. 10-5). In addition to the rapid destruction of the teeth, there may be dryness and cracking of the lips, with fissuring at the corners of the mouth, burning and soreness of the mucous membranes, crusting of the tongue and palate, and sometimes paresthesia of the tongue or mucous membrane.

There are many reasons for a reduction in salivary flow. Acquired salivary dysfunction may be the result of a psychologic or emotional disturbance and, again, may be either temporary or permanent. During the acute stages, mumps may cause a temporary reduction in salivary flow. Immune disorders, such as Sjogren syndrome, and genetic conditions, such as hypohidrotic ectodermal dysplasia, often exhibit chronic xerostomia. Many oncology patients receive head and neck or total-body irradiation that also results in salivary gland dysfunction. An interruption in the central pathways of the secretory nerves has been suggested as a cause of salivary failure, but this is usually overshadowed by definite neurologic signs and symptoms. Similarly, a deficiency of vitamin B complex has been reported as a cause of salivary gland dysfunction.

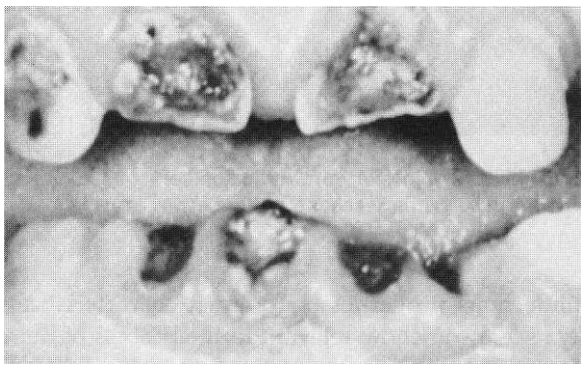


FIG **10-5.** Rampant dental caries in a 9-year-old boy. There was a septic dry mouth caused by the congenital absence of salivary glands.

One study indicated that the minimum effective dose of many of the antihistaminic drugs can reduce salivary flow by as much as 50%.[3'] Dryness of the mouth may occur after the use of a variety of tranquilizers and antihistamines. It has likewise been observed that dry mouth and rampant caries may accompany a systemic condition, such as myasthenia gravis. In this disease the acetylcholine that is necessary for the proper transmission of nerve impulses is destroyed; as a result the salivary glands do not receive adequate stimulation.

Previous work has shown a great deal of individual variation in the amount of saliva produced by stimulation of the glands. The range is from less than a measurable amount in patients with acquired or congenital dysfunction of the salivary glands to 65 ml during a 15-minute period of stimulation and collection. Patients with deficient salivary flow often have excessive or rampant caries. In contrast, patients with greater than average salivary flow are usually relatively free from dental caries.

Determination of Salivary Flow. If a patient has no known existing conditions that may cause hyposalivation and if the clinician notices a small pool of saliva in the floor of the mouth during oral examination, it is not unreasonable to assume that the patient has adequate salivary quantity and flow. Little information is available about salivary flow rates in children, but Crossner reports that in children 5 to 15 years of age the rate of mixed whole stimulated saliva increases with age, and boys have consistently higher rates than girls.[3"] If inadequate salivary flow is known to exist or is suspected, measurement of salivary flow can provide a baseline useful for comparing with later measurements after implementation of adjunctive therapy.

To evaluate the adequacy of salivary flow, Zunt recommends establishing the unstimulated salivary flow (USF) rate.[3j] The USF rate is measured after a period of 1 hour without eating, drinking, chewing gum, or brushing the teeth. Sitting in the "coachman" position, on the edge of the dental chair, the patient passively drools into a funnel inserted into a graduated cylinder for 5 minutes. The eyes should remain open except for blinking during the 5-minute collection period. The head and neck should be bent, and the arms should rest comfortably on the thighs or knees.

The volume of saliva collected in the cylinder after 5 minutes is divided by 5 to determine the USF. A USF rate of less than 0.1 ml per minute is diagnostic of salivary gland hypofunction. If the USF rate is less than 0.1 ml per minute, the next step is to measure the stimulated salivary flow (SSF). The patient should chew unflavored paraffin for 45 chews or 1 minute and expectorate into a funnel inserted into a graduated cylinder. The SSF rate should be 1 to 2 ml per minute; less than 0.5 ml per minute is scored as an abnormal rate. A convenient alternative method for measuring USF is the modified Schirmer technique, which uses a calibrated paper test strip to collect saliva in the floor of the mouth.

In patients who are known or suspected to have salivary deficiency, it is not unusual to find a salivary flow ranging from slightly below normal to practically a dry mouth. If there is a deficiency of saliva or a dry mouth, the cause should be sought. Sometimes the cause is readily determinable; sometimes it is obscure. An emotional disturbance should not be overlooked as a cause in a patient of any age. Psychotherapy may be helpful in these cases. If the cause cannot readily be determined, perhaps it should be assumed that the sparse flow is related to inadequacies in the diet, particularly a vitamin deficiency or excessive sugar consumption to the exclusion of needed foods. Monthly quantitative analyses of the saliva should be performed to determine whether dietary improvement is accompanied by an increased flow.

If the salivary glands have not undergone degenerative or metaplastic change and if the nerve pathways between the central nervous system and the salivary glands are still intact, salivary stimulants may be recommended. If dryness of the mouth is attributable to dehydration, increased fluid intake should be recommended. The use of gustatory stimulants (sugar-free candy) or masticatory stimulants (xylitol gum) has been suggested as an adjunct to encourage salivation. Prescription sialagogue medications, also known as secretagogues, such as pilocarpine and cevimeline may be of benefit in improving the salivary flow rate in patients with Sjogren syndrome or with radiation damage to salivary glands. The use of sialagogue medications has not been studied in pediatric populations, but these agents are considered very safe for most adult patients and have been used successfully in older children. The use of salivary substitutes has been suggested by Shannon, McCrary, and Starcke as helpful in preventing soft tissue problems associated with dry mouth." Saliva substitutes, as well as fluoride and chlorhexidine rinses, are also reported to enhance remineralization and promote resistance to demineralization of tooth surfaces, and may help prevent radiation-induced caries.

Viscosity of Saliva. It has long been suggested that the viscosity of saliva is related to the rate of dental decay. Both thick, ropy saliva and thin, watery saliva have been blamed for rampant dental caries. Previous work conducted at Indiana University has shown a statistically significant direct relationship between the viscosity of saliva and the number of decayed, missing, and filled teeth.[39] This relationship held true for all members of the observation group, regardless of age. Patients with thick, ropy saliva invariably had poor oral hygiene. The teeth were covered with stain or plaque, and the rate of dental caries ranged from greater than average to rampant.

No evidence exists that viscosity changes with age under normal conditions. This property of the saliva is governed not only by the particular set of glands stimulated but also by the type of nervous stimulation and the amount of mucin (glycoprotein) present.

We have observed that children who consume excessive amounts of carbohydrates often have not only a sparse flow but also a viscous saliva. Even minimal doses of some antihistaminic drugs will result in a greatly increased viscosity of saliva in some persons.[35]

There are apparently only a limited number of ways to alter the viscosity of saliva. Reduction of refined sugar intake may be effective in some patients.

Although relatively little information specific to salivary function, flow, and viscosity in children is available, an excellent review article by Leone and Oppenheim provides much additional information regarding the relationship between saliva and dental caries for the interested reader."

SOCIOECONOMIC STATUS

The Surgeon General's report of 2000 indicated that one in four American children is born into poverty' It noted that children and adolescents living in poverty suffer twice as much tooth decay as their more affluent peers and that their disease is more likely to go untreated. The report also mentioned that, although continuing reductions of dental caries in permanent teeth have been achieved, caries prevalence in primary teeth has stabilized or possibly increased in some population groups. A Census Bureau report published in March 2003 showed that the poverty rate for children in the United States rose in 2002, whereas it dropped for people 65 years and older." Nearly half of the 35 million people living in poverty were children. These are alarming data calling attention to a huge unmet oral health care need in our country.

Following the report of the Surgeon General, Edelstein pointed out that, paradoxically, children living in poverty also have the highest rates of dental insurance coverage, largely through the Medicaid program and the State Children's Health Insurance

Program.[41] Yet Medicaid-eligible children who have cavities have twice the number of carious teeth and twice the number of visits for pain relief but fewer total dental visits than children in families with higher incomes. He also notes that these disparities continue into adolescence and young adulthood but to a lesser degree. Because practitioners have the opportunity to assess the oral health of poor children individually, they will identify some patients at low risk for dental caries. However, the available data confirm that, from a demo-graphic perspective, economically poor children are at high risk for dental caries.

ANATOMIC CHARACTERISTICS OF THE TEETH

Certain teeth of many patients, particularly permanent teeth, seem vulnerable to dental caries as they emerge and, in caries-active mouths, they may show evidence of the attack almost coincident with their eruption into the oral cavity. Because enamel calcification is incomplete at the time of eruption of the teeth and an additional period of about 2 years is required for the calcification process to be completed by exposure to saliva, the teeth are especially susceptible to caries formation during the first 2 years after eruption. Permanent molars often have incompletely coalesced pits and fissures that allow the dental plaque material to be retained at the base of the defect, sometimes in con-tact with exposed dentin. These defects or anatomic characteristics can readily be seen if the tooth is dried and the debris and plaque removed. In addition to occlusal surfaces, lingual pits on the maxillary permanent molars, buccal pits on the mandibular permanent molars, and lingual pits on the maxillary permanent lateral incisors are vulnerable areas in which the process of dental caries can proceed rapidly.

ARRANGEMENT OF THE TEETH IN THE ARCH

Crowded and irregular teeth are not readily cleansed during the natural masticatory process. It is likewise difficult for the patient to clean the mouth properly with a toothbrush and floss if the teeth are crowded or over-lapped. This condition therefore may contribute to the problem of dental caries.

PRESENCE OF DENTAL APPLIANCES AND RESTORATIONS

Partial dentures, space maintainers, and orthodontic appliances often encourage the retention of food debris and plaque material and have been shown to result in an increase in the bacterial population. Few patients keep their mouths meticulously clean, and even those who make an attempt may be hampered by the presence of dental appliances that retain plaque material between brushings. Patients who have had moderate dental caries activity in the past might be expected to have increased caries activity after the placement of appliances in the mouth unless they practice unusually good oral hygiene.

Rosenbloom and Tinanoff evaluated the S. *mutans* level of patients before, during, and after orthodontic treatment.[43] S. *mutans* levels were significantly elevated during active treatment. When samples were taken 6 to 15 weeks into the retention phase of treatment, how-ever, the microbial levels were found to have decreased significantly to levels comparable to those of untreated children.

Dentists have known for many years that the tooth structure at the interface with restorative material is especially vulnerable to recurrent caries. Clinical studies suggest that dentists and their patients should not expect successful restorative treatment to reduce a patient's risk for future development of carious lesions. Tinanoff, Siegrist, and Lang found higher numbers of salivary S. *mu tans* in patients after they received restora-tive treatment.[44] Wright et al observed significant reduc-tions in the number of mutans streptococci and lactobacilli immediately after restoration; however, mutans streptococci returned to prerestoration levels in many of their subjects.[45] Gregory, El-Rahman, and Avery observed that postoperative counts of salivary streptococci essentially equaled the preoperative counts after all restorative work had been completed in their patients.[46] Effective prevention programs are required to protect the patient from additional caries and to better justify the investment in restorative care.

HEREDITARY FACTORS

Although parents of children with excessive or rampant caries tend to blame the condition on hereditary factors or tendencies, and some scientific evidence as reviewed in Chapter 6 acknowledges certain genetic influences on the caries process, most authors agree that genetic influences on dental caries are relatively minor in com-parison with the overall effect of environmental factors. The fact that children acquire their dietary habits, oral hygiene habits, and oral microflora from their parents makes dental caries more an environmental than a hereditary disease. Although several hereditary factors identified in Chapter 6 may be influential in promoting or preventing dental caries activity, available effective preventive therapies along with proper dietary and plaque control measures can override the hereditary factors that contribute to caries development.

EARLY DETECTION OF DISEASE ACTIVITY

Traditionally, dentists have relied upon a visualtactile-radiographic procedure for the detection of dental caries. This procedure involves the visual identification of demineralized areas (typically white spots)

or suspicious pits or fissures and the use of the dental explorer to determine the presence of a loss of continuity or breaks in the enamel and assess the softness or resilience of the enamel. Carious lesions located on interproximal tooth surfaces have generally been detected with the use of bitewing radiographs. These procedures have been used routinely in virtually every dental office in the United States for the past 50 years.

Because the reversal of the caries process depends on an intact surface layer of the lesion and the typical use of the dental explorer to probe the suspicious areas often results in the rupture of the surface layer covering early lesions, the use of the dental explorer to probe enamel is no longer recommended. The recommended use of the dental explorer is to judiciously remove plaque and debris to permit visual inspection of pits and fissures.

This understanding of the nature of the caries process also emphasizes the need for alternative methods for the early detection of dental caries. Lesions detected on radiographs have generally progressed to the initial involvement of dentin. Thus, the increased desire of dental professionals and patients for more conservative restorative procedures as well as the implementation of measures to control and reverse the caries process led to significant efforts to develop technologies for the early detection of dental caries. Although a variety of new technologies are being explored for this purpose, only three of these technologies are currently available for clinical dental practice.'

INFRARED LASER FLUORESCENCE (DIAGNOdent)

An instrument designed to facilitate the detection of dental caries, DIAGNOdent,* has recently become available in several countries. This instrument was developed for the detection and quantification of dental caries of occlusal and smooth surfaces. It uses a diode laser light source and a fiber-optic cable that transmits the light to a hand-held probe with a fiber-optic eye in the tip. The light is absorbed and induces infrared fluorescence by organic and inorganic materials. The emitted fluorescence is collected at the probe tip, transmitted through ascending fibers, and processed and presented on a display window as an integer between 0 and 99. Increased fluorescence reflects carious tooth substance, particularly for numerical values higher than about 20. What material is responsible for the fluorescence is still under investigation, but it appears to be bacterial metabolites, particularly the porphyrins.

An appreciable number of in vitro studies and a few in vivo (clinical) studies of the performance of this instrument have been reported in the past 5 years.

*Kaltenbach & Voigt GmbH & Co., Bismarckring 39.

The results of the various in vitro studies have indicated that the DIAGNOdent instrument is capable of detecting relatively advanced carious lesions, and DIAGNOdent readings show a very good correlation with histologic evidence of caries but not with the depth of the lesions into dentin. These studies have also demonstrated the instrument to exhibit excellent reproducibility and good to excellent sensitivity. However, the results of the in vitro studies have also indicated that the readings are influenced by several variables, including the degree of dehydration of the lesion, the presence of dental plaque, and the presence of various types of stain in occlusal fissures.

The results of clinical investigations have shown significant differences in readings between different DIAGNOdent instruments with regard to occlusal caries, which raises questions regarding the selection of a value of 20 or 25 to indicate the presence of caries. For a given instrument, intraoperator reliability was generally very good and interoperator reliability typically ranged from good to very good. Further, DIAGNOdent readings increased linearly with clinical histologic measurements, and therefore the instrument was reported to be able to distinguish with good sensitivity between sound tooth structure or shallow enamel caries and deeper carious lesions in enamel or lesions extending into dentin. Recent studies, however, have not been able to confirm the narrow range of values related to lesion extent reported previously. The instrument is very good at indicating the presence of deeper lesions in enamel or into dentin that may not be apparent yet on radiographs but is unable to reliably indicate the depth of a dentinal lesion.

The DIAGNOdent instrument appears to be particularly useful for confirming the presence of occlusal caries. Especially with the judicious use of the dental explorer to remove oral debris from occlusal pits and fissures, the DIAGNOdent is capable of confirming the presence or absence of caries in otherwise suspicious areas. Instrument readings higher than 20 or 25 reflect the likely presence of caries, and higher readings generally reflect more extensive lesion progression, although there does not appear to be a linear relation between the readings and the extent of the lesions. Prudent use of the instrument can identify early lesions that should be considered for preventive rather than restorative treatment.

DIGITAL IMAGING FIBER-OPTIC TRANS-ILLUMINATION (DIFOTI)

Conventional clinical caries examinations routinely use transillumination to identify lesions located on the interproximal surfaces of the anterior teeth. For at least 30 years a fiber-optic transillumination (FOTI) instrument has been available for clinical use. It provides an

intense light beam that is transmitted through a fiber-optic cable to a specially designed probe to permit the use of transillumination on the proximal surfaces of posterior teeth. Repeated improvements have been made in the instrument so that it may be used on occlusal as well as proximal tooth surfaces, and the instrument is commonly used, often in place of radio-graphs, in private practices in Europe. Numerous studies have compared the use of FOTI to visual and radiographic examinations, and a recent investigation indicated that dental practitioners were able to detect more proximal lesions with FOTI than with a visual examination with or without the use of radiographs.

Digital Imaging Fiber Optic Trans-Illumination (DIFOTI[*]) is a further advancement of this technology in which the visually observed images are captured using a digital charged-coupled device (CCD) camera and sent to a computer for analysis using dedicated algorithms. This instrument has become commercially available relatively recently, and very few investigations of its capabilities and limitations have been reported. The results of an in vitro study involving the imaging of extracted teeth indicated that the instrument had greater sensitivity for the detection of caries on all tooth surfaces than did radiologic imaging. However, a more recent in vitro study in which artificial lesions developed over 14 weeks underwent intermittent imaging and radiography at 2-week intervals indicated that the DIFOTI system, unlike radiography, was not able to determine the depth of lesions. Although at least one clinical investigation of this instrument is in progress, there are no published reports of the clinical use of the instrument demonstrating its usefulness in practice at the time of this writing. Nevertheless, based on prior reports of the use of FOTI, it is reasonable to expect that DIFOTI should be at least as good as radiography for detection of caries on interproximal tooth surfaces.

QUANTITATIVE LIGHT FLUORESCENCE

Without question the most extensively investigated technique available for the early detection of dental caries is quantitative light fluorescence (QLF). This methodology began with the observation in 1978 by *a* dental scientist in Sweden that the use of a laser light of selected wavelength markedly enhanced the visibility of early noncavitated lesions. Subsequent investigations by this group confirmed the value of this approach for the early detection of caries clinically. Further studies established a relationship between the loss of fluorescent intensity and increasing mineral loss from the lesions compared with sound enamel. Numerous additional in vitro and in situ studies confirmed this important correlation between the amount of observed fluorescence and the mineral content of the lesions and made possible the development of a system that could truly assess changes in either the progression or regression of carious lesions.

To facilitate clinical investigations as well as the use of the device in clinical practice, a small portable system[*] was developed in which the laser source was replaced by a regular light source and filter system. The light illuminating the tooth is transported through a liquid-filled light guide. The fluorescent filtered images are captured using a color CCD camera and a frame grabber. Data are collected, stored, and analyzed by custom software. Since the first attempts at in vivo quantification of mineral changes in the enamel, the QLF method has continued to undergo further development with respect to both software and hardware. A number of clinical investigations of this newer system have been conducted and have convincingly demonstrated the ability of the QLF instrument to detect early carious lesions and to accurately monitor either lesion progression or regression. The first of these studies monitored the changes in white spot lesions in orthodontic patients following the removal of the appliances and the institution of improved oral hygiene and use of a fluoride dentifrice; the remineralization of the lesions was impressively demonstrated within a few weeks. Another study of school-aged children showed that the QLF instrument detected 5 to 10 times more early lesions than conventional detection methods, was particularly useful for examination of occlusal pits and fissures, and gave reproducible results. A clinical study of white spot lesions in caries-active children in Sweden demonstrated significant remineralization of the lesions within 6 months after treatments with a fluoride varnish. An additional clinical study by this group involving multiple examiners demonstrated the reproducibility of clinical QLF measurements. A team of Japanese scientists conducted a clinical trial using QLF to monitor changes in white spot lesions of children brushing with a fluoride dentifrice and reported a significant decrease in the size of the lesions within 3 months of the 1-year test period. From these clinical studies it is apparent that QLF enhances the early detection of dental caries and is uniquely useful in monitoring the progression or regression of lesions.

Numerous investigations have demonstrated the practical usefulness of QLF for the early detection of dental caries on occlusal and smooth tooth surfaces as well as for the quantification of lesion changes related to treatment procedures and environmental factors such as oral hygiene. The only significant limitation to this instrument is its inability to detect or monitor interproximal lesions.

'Electro-Optical Sciences, Inc., Irvington, NY 10533.

*Omnii Oral Pharmaceuticals, West Palm Beach, FL 33409.

Applications. Caries detection is entering a new era with new technologies capable of detecting lesions at an earlier stage of development and quantifying the impact of noninvasive professional fluoride treatments such as fluoride varnishes. DIAGNOdent appears to be most useful in confirming the presence of caries in suspicious occlusal pits and fissures and detecting deep dentinal lesions of the occlusal surface (so-called hidden caries). DIFOTI, pending further study, may have greatest usefulness as a potential replacement for bitewing radiography in the detection of caries on interproximal surfaces. QLF has been shown to be most useful for the early detection of dental caries on occlusal, buccal, and lingual surfaces and the quantification of changes in early lesions associated with various preventive treatments. Such early identification will provide evidence to guide the dental professional in implementing various measures for the reversal and control of these lesions.

PREDICTION OF PATIENTS' RISK FOR FUTURE DISEASE (RISK ASSESSMENT)

In contemporary health care practices, caries-risk assessment is now recognized as a useful component in providing appropriate care for children and adolescents. The AAPD has adopted a policy statement on caries-risk assessment and a Caries-Risk Assessment Tool (CAT). The CAT is illustrated in Table 10-1. The policy statement, almost in its entirety except for references, follows":

Purpose

The American Academy of Pediatric Dentistry (AAPD) recognizes that caries-risk assessment is an essential element of contemporary clinical care for infants, children and adolescents.

Background

...While assessment of caries risk undoubtedly will benefit from emerging science and technologies, the AAPD believes that sufficient evidence exists to support the creation of a framework for classifying caries risk in infants, children and adolescents based on a set of physical, environmental and general health factors.

The table [Table 10-11...represents a first step toward incorporating available evidence into a concise, practical tool to assist both dental and nondental health care providers in assessing levels of risk for caries development in infants, chil dren and adolescents. The AAPD intends this to be a dynamic instrument that will be evaluated and revised periodically as new evidence warrants.

Clinicians using this tool should:

1. be able to visualize adequately a child's teeth and mouth and have access to a reliable historian for non-clinical data elements;
2. assess all 3 components of caries risk—clinical conditions, environmental characteristics and general health conditions;
3. be familiar with footnotes that clarify use of individual factors in this instrument;
4. understand that each child's ultimate risk classification is determined by the highest risk category where a risk indicator exists (ie, the presence of a single risk indicator in any area of the "high-risk" category is sufficient to classify a child as being at "high risk"; the presence of at least 1 "moderate-risk" indicator and no "high-risk" indicators results in a "moderate-risk" classification; and a child designated as "low risk" would have no " moderate-risk" or "high-risk" indicators).

Users of the AAPD caries-risk assessment tool (CAT) must understand the following caveats:

1. CAT provides a means of classifying dental caries risk at a point in time and, therefore, should be applied periodically to assess changes in an individual's risk status.
2. CAT is intended to be used when clinical guidelines call for caries-risk assessment. Decisions regarding clinical management of caries, however, are left to qualified dentists (ideally, the dentist responsible for the child's "dental home").
3. CAT can be used by both dental and nondental personnel. It does NOT render a diagnosis. However, clinicians using CAT must be familiar with the clinical presentation of dental caries and factors related to caries initiation and progression.
4. Since clinicians with various levels of skill working in a variety of settings will use this instrument, advanced technologies such as radiographic assessment and micro -biologic testing (darker shaded areas in Table 10-1) have been included but are not essential for using this tool.

Policy statement

The AAI'D:

1. encourages both dental and nondental health care providers to use CAT in the care of infants, children and adolescents;
2. encourages dentists to use advanced technologies such as radiographic assessment and microbiologic testing with CAT when assessing an individual's caries risk;
3. recognizes the need to evaluate CAT periodically and revise the tool as new science and technologies warrant.

Zero, Fontana, and Lennon presented an extensive review of the literature to determine the predictive validity of available multivariate caries-risk assessment strategies for children and adults." Some of their conclusions were the following:

- The predictive validities of the models reviewed depended strongly on the caries prevalence and characteristics of the population for which they were designed.
- Many models included similar categories of risk indicators but provided very different outcomes depending on the study population.
- In many instances, the use of a single risk indicator gave results that were equally as good as those obtained using a combination of indicators.

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Any condition, treatment, or process

- No combination of risk indicators was consistently considered a good predictor when applied to different populations, across different age groups. In general, however, the best indicators of caries risk were easily obtained from dental charts and did not require additional testing.
- Previous caries experience was an important predictor for primary, permanent, and root surface caries in most models tested.
- Most of the research in this area has been done in children, for both primary and permanent teeth.

It is also obvious from their report that the caries-risk assessment process is very much a work in progress at this time. Further refinements will continue for some time in the future.

Of course, accurate caries-risk assessments of patients can guide clinicians and health care facilities toward better allocation of their time and resources for their high-risk patients. As our accuracy and efficiency in identifying patients with active disease or high potential to develop disease improves and as parents and patients (and, it is hoped, health care insurance plans) accept this newer approach to care, the standard "6-month recall visit" for children may change to a more customized plan for individual patients or groups of patients. Children who are at low risk for caries and do not present with other oral conditions that need frequent monitoring may not require oral health care visits as often as those at high risk (with or without active disease), whereas compliant high-risk patients may, at times, require frequent visits and multiple forms of caries control therapies in addition to their voluntary modification of caries-promoting dietary and behavioral habits.

CONTROL OF DENTAL CARIES

Many practical measures for the control of dental caries are applicable to private practice. Most practitioners have tried control measures with varying degrees of success. One cannot emphasize too strongly, however, that no single measure for the control of dental caries will be entirely satisfactory. At the present time all possible preventive measures and approaches must be considered in the hope of successfully controlling and preventing the caries process.

Pediatric dentists who see patients on a referral basis may hear a parent remark, "My child has so many cavities that my dentist doesn't know where to start." Although it is true that the problem may at first seem overwhelming, a systematic, understanding approach will often result in a gratifying response. An outline of procedures for the control of active and/or rampant caries in cooperative and communicative patients follows. With this approach and with patient cooperation, the problem can usually be explained and brought under control. The successful management of active dental caries, however, depends on the parents' and/or patient's interest in maintaining the patient's teeth and their cooperation in a customized and specific caries control program.

CONTROL OF ALL ACTIVE CARIOUS LESIONS

When rampant caries occurs, the first steps are to initiate treatment of all carious lesions to stop or at least slow the progression of the disease and to identify the most important causes of the existing condition. Next, and even simultaneously, if possible, the practitioner begins working with the parents and/or the patient to achieve the appropriate behavioral modifications required to prevent recurrence. The problem may then be approached in a systematic manner. Invariably modifications in oral hygiene procedures and dietary habits will be necessary. Often, achieving patient compliance with the recommended modifications is the greatest challenge of all.

If the initial restorative treatment is to be done in one appointment under general anesthesia or in one or two appointments with sedation, control of the existing lesions will be definitive at that time. If the restorative care is to be performed over several visits in the outpatient setting, gross caries excavation as an initial approach in the control of rampant dental caries has several advantages. The removal of the superficial caries and the filling of the cavity with a glass ionomer material or zinc oxide–eugenol cement (IRM*) will at least temporarily arrest the caries process and prevent its rapid progression to the dental pulp. Gross caries removal can usually be accomplished easily in one appointment. If there are many extensive carious lesions, however, a second appointment may be necessary.

An alternative approach for some compliant children (with compliant parents) old enough to rinse and expectorate and for compliant adolescents is to initiate intensive and multiple antimicrobial and topical fluoride therapies in conjunction with the necessary behavioral life-style modifications, and then to proceed systematically with restorations and other indicated therapies.

REDUCTION IN THE INTAKE OF FREELY FERMENTABLE CARBOHYDRATES

Some excellent studies have been reported that show a relationship between diet and dental caries. As a result of these studies, considerable emphasis has been given to this phase of the caries control program. There is also much evidence to confirm that between-meal snacking and the frequency of eating and drinking are related to dental caries incidence.

*Intermediate Restorative Material, LD Caulk Co., Milford, DE, 19963.

Gustafsson et al conducted a well-controlled study of dental caries, now considered a classic, and observed that a group of patients whose diet was high in fat, low in carbohydrates, and practically free of sugar had low caries activity.[50] When refined sugar was added to the diet in the form of a mealtime supplement, there was still little or no caries activity. However, when caramels were given between meals, a statistically significant increase in the number of new carious lesions occurred. It was concluded from these studies that dental caries activity could be increased by the consumption of sugar if the sugar were in a form easily retained on the tooth surface. The more frequently this form of sugar was consumed between meals, the greater was the tendency for an increase in dental caries.

Weiss and Trithart reported additional evidence for the relationship between the incidence of dental caries and between-meal eating habits." In a group of preschool children it was found that most between-meal snacks were of high sugar content or were high in adhesiveness. The children who did not eat between meals had 3.3 decayed, extracted, or filled primary teeth (deft), whereas those who ate four or more items between meals had a deft rate of 9.8.

As mentioned earlier, sweetened liquids provided to young children in nursing bottles can have enormous cariogenic potential. Likewise, the carbonated soft drinks and other sweetened drinks so popular with older children and adolescents are readily available today. Frequent ingestion of these drinks is another form of snacking that can promote and accelerate caries progression.

In view of the results of these and many other subsequent well-controlled studies, another consideration in the clinical control of dental caries could be a determination of eating habits by having the patient keep a 7-day record of all food eaten at mealtime and between meals. The record should then be evaluated to determine the adequacy of the diet and the amount of freely fermentable carbohydrates. The number of servings of food in each of the basic five food groups should be determined and compared with the recommended number of servings outlined in Figs. 10-6 and 10-7, and Table 10-2. The basic diet analysis form (Box 10-1) has been found to be helpful in evaluating the adequacy of the diet.

Figs. 10-6 and 10-7 and Table 10-2 provide appropriate guidelines to assist in assessing the adequacy of the patient's diet. Fig. 10-6 illustrates the Food Guide Pyramid, which presents the recommendations of the National Academy of Sciences and the U.S. Department of Agriculture. Fig. 10-7 shows the Guide to Good Eating, which presents the recommendations of the National Dairy Council (NDC). These two guides are designed for individuals who are school-aged and older. The NDC has also proposed guidelines for preschool children. The NDC guidelines for children are summarized in Table 10-2.

An evaluation of 500 consecutive records of children coming to the Indiana University Clinic has demonstrated that only a small percentage has adequate diets. The majority of children have noteworthy deficiencies in one or more of the basic five food groups.

Investigations by Schachtele and Jensen[52] as well as by Park, Ashmore, and Stookey[S3] have indicated that the acidity of plaque located in interproximal areas, which generally have less exposure to saliva, may remain below the critical pH for periods in excess of 2 hours after carbohydrate ingestion. Because foods containing sugars in solution as well as retentive sugars are included in the diet analysis, 20 minutes may be considered as the minimal time each exposure permits acid concentrations to be available in the bacterial plaque (Fig. 10-8).

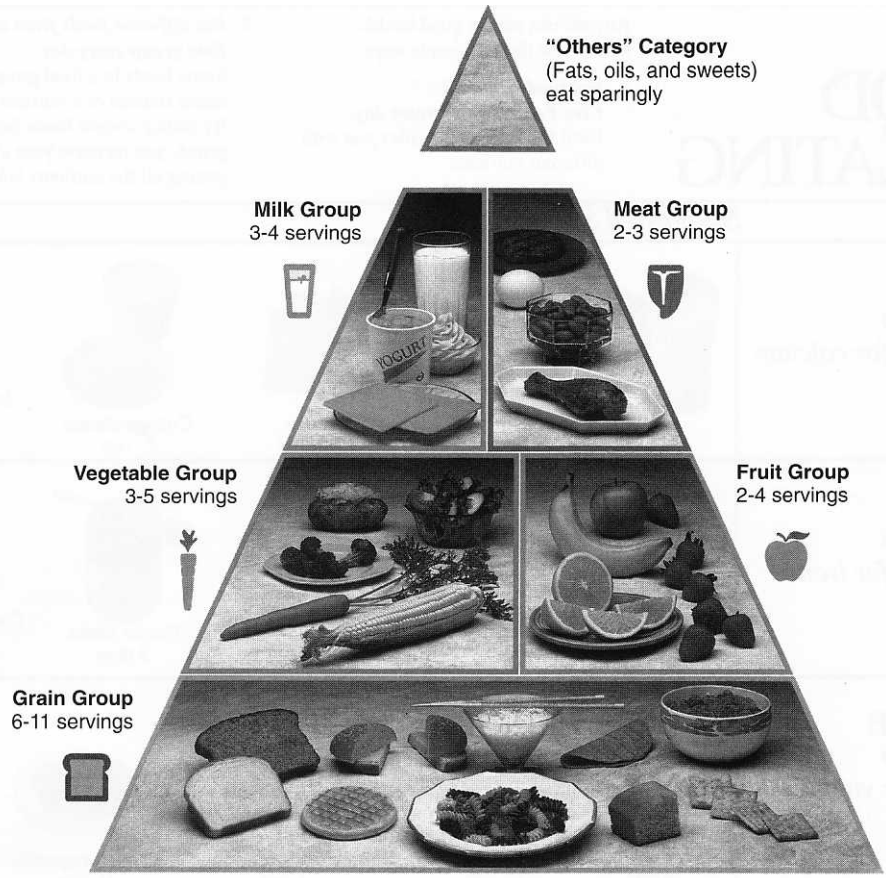
The following can be used in explaining the dental caries process to a parent or child:

Fermentable carbohydrate + Oral bacteria within plaque Acid within plaque
Acid + Susceptible tooth Tooth decay

Foods in the five basic groups should not be included in the fermentable carbohydrate calculation (with some exceptions) unless free sugar is added during their preparation. One exception is dry cereal, because it is considered a refined food. Cereals may be retained on the teeth for long periods of time; in addition, many of them are sugar coated and some are high in sucrose content. Although the cariogenicity of a food may he related to its sucrose content, its consistency, the time of consumption, and the conditions under which it is eaten, dietary counseling should involve particular consideration of sucrose consumption between meals.

Dried fruits, including raisins, prunes, peaches, apricots, and dates, have a high sugar content; therefore their sugar equivalents should also be included in the diet analysis. These are foods commonly eaten by children between meals, and there is no reason to believe that they are any less cariogenic than refined foods. Although honey is considered a natural food, its sugar equivalent should be considered, in addition to that of syrups and other spreads for bread.

Rather than insist that the patient follow a strict dietary routine severely restricted in carbohydrates, the dentist should suggest a basic diet. The purpose of limiting a diet to the basic five food groups is twofold. An adequate diet is fundamental to general good health and is, of course, essential during the period of tooth formation to help ensure the development of normal



How many servings are right for me?
The pyramid shows a range of servings for each food group. The number of servings that are right for you depends on how many calories you need, which in turn depends on your age, sex, size, and activity level. Almost everyone should have at least the lowest number of servings in the ranges. The following calorie level suggestions are based on recommendations of the National Academy of Sciences and on calorie intakes reported by people in national food consumption surveys.

For adults and teens

1600 calories is about right for many sedentary women and some older adults.

2200 calories is about right for most children, teenage girls, active women, and many sedentary men. Women who are pregnant or breastfeeding may need somewhat more.

2800 calories is about right for teenage boys, many active men, and some very active women.

Sample Diets for a Day at Three Calorie Levels

	Lower about 1600	Moderate about 2200	Higher about 2800
Bread group servings	6	9	11
Vegetable group servings	3	4	5
Fruit group servings	2	3	4
Milk group servings	2-3[1]	2-3[1]	2-3[1]
Meat group (ounces)	5	6	7
Total fat (grams)	53	73	93

[1] Women who are pregnant or breastfeeding, teenagers, and young adults to age 24 need 3 servings.

f **C 10-6.** Food Guide Pyramid. *(From US Department of Agriculture, 7992.)*

tooth structure. In addition, however, it is a well-established fact that if children and adults closely follow the recommended basic diet and consume a diet containing adequate amounts of protein, fresh fruit, and vegetables, the appetite for snacks between meals will be decreased.

REDUCTION OF DENTAL PLAGUE (AND MICROORGANISMS) WITH GOOD ORAL HYGIENE PROCEDURES

The next chapter of this text discusses the importance of good oral hygiene in more detail, but it must be mentioned here as a critically important component of

GUIDE TO GOOD EATING

Anyone can eat for good health. Just follow these 2 simple steps:

1. ***Eat foods from all Five Food Groups every day.*** Each food group provides you with different nutrients.
2. ***Eat different foods from each food group every day.*** Some foods in a food group are better sources of a nutrient than others. By eating several foods from each food group, you increase your chance of getting all the nutrients you need.

Every day eat:	*Suggested Serving Sizes*
MILK *Group for calcium* **3-4 servings**	Milk 1 cup; Yogurt 1 cup; Cheese 1½–2 oz; Cottage cheese ½ cup; Ice cream, ice milk, frozen yogurt ½ cup
MEAT *Group for iron* **2-3 servings**	Cooked, lean meat 2-3 oz; Cooked, lean poultry, fish 2-3 oz; Egg 1; Peanut butter 2 tbsp; Cooked, dried peas, dried beans ½ cup
VEGETABLE *Group for vitamin A* **3-5 servings**	Juice ¾ cup; Raw vegetable ½ cup; Raw leafy vegetable 1 cup; Cooked vegetable ½ cup; Potato 1 medium
FRUIT *Group for vitamin C* **2-4 servings**	Juice ¾ cup; Raw, canned, or cooked fruit ½ cup; Apple, banana, orange, pear 1 medium; Grapefruit ½; Cantaloupe ¼
GRAIN *Group for fiber* **6-11 servings**	Bread 1 slice; English muffin, hamburger bun ½; Ready-to-eat cereal 1 oz; Pasta, rice, grits, cooked cereal ½ cup; Tortilla, roll, muffin 1

Some foods do not have enough nutrients to fit in any of the Five Food Groups; these foods are called "Others". These foods are okay to eat in moderation. They should not replace foods from the Five Food Groups.

"OTHERS" *category*	
Fats and oils, sweets, salty snacks, alcohol, other beverages, and condiments	

f I G 10-7. Guide to Good Eating. *(Reprinted by permission of the National Dairy Council, Rosemont, III.)*

TABLE 10-2 Recommended Food Servings for Children

FOOD GROUP	PRESCHOOL	6-12 YEARS	TEEN GIRLS	TEEN BOYS
Milk	3	3-4	4	4
Meat	2	2-3	2-3	2-3
Vegetables	3	3-4	3-4	4-5
Fruit	2	2-3	2-3	3-4
Grain	4	6-9	6-9	9-11

Adapted from the Guide to Good Eating, ed 6, and Food Models Teacher/Leader Guide for Early Childhood Educators, Rosemont, III, National Dairy Council.

any caries control program. Berenie, Ripa, and Leske studied the relationship between frequency of toothbrushing, oral hygiene, gingival health, and caries in 384 children, 9 to 13 years of age, who resided in a fluoride-deficient western New York community" Of the children studied, 37% brushed their teeth once a day, 37% brushed twice a day, and 13% brushed less than once a day. The remaining children in the group, approximately 13%, brushed their teeth three or more times each day. A trend toward decreased scores for decayed, missing, or filled permanent teeth (DMFT) and decayed, missing, or filled permanent tooth surfaces (DMFS) accompanied increased daily brushing. The increased frequency of daily toothbrushing had its most significant positive effect on the level of oral hygiene.

Beal et al studied the caries incidence and gingival health of children who were 11 to 12 years old at the start of the study.5S The children's dental cleanliness was evaluated at yearly examinations for a 3-year period. The children whose dental cleanliness was consistently good had lower caries increments than those whose dental cleanliness was consistently bad. Horowitz et al have demonstrated the benefits of a school-based plaque removal program in a 3-year study of children in grades 5 to 8.[5"] At the end of the study the adjusted mean DMFS scores were 13% lower in the supervised plaque removal group than in the control group.

The difference between groups was accounted for entirely by a 26% difference in affected mesial and distal surfaces, a figure that approached statistical significance (p = 0.07). Similarly, Tsamtsouris, White, and Clark have demonstrated that supervised toothbrushing with instruction produces significantly and consistently lower plaque scores, even in preschool children, than were achieved through a control test of the same children when they were neither supervised nor instructed.[57] The investigators concluded that constant reinforcement is necessary to maintain effective plaque control in preschool children.

Wright, Banting, and Feasby conducted a clinical study to evaluate the effect of frequent interdental flossing on the incidence of proximal dental caries.[5'] Schoolchildren from a fluoride-deficient area were studied after clinical and radiographic examinations were performed. Based on the observations of this study, the authors concluded that frequent interdental flossing resulted in a 50% reduction in the incidence of proximal caries in primary teeth during a 20-month period. The longer the period of interdental flossing, the greater the benefit; however, there was little residual effect after flossing was discontinued.

USE OF FLUORIDES AND TOPICAL ANTIMICROBIAL AGENTS

Without doubt the repeated use of fluorides is of critical importance for the control and prevention of dental caries in both children and adults. Numerous controlled clinical investigations have consistently demonstrated the cariostatic properties of fluoride provided in a variety of manners. These studies have also shown that the maximal benefit from fluoride is achieved only through the use of multiple delivery systems.

Existing evidence indicates that the cariostatic activity of fluoride involves several different mechanisms. The ingestion of fluoride results in its incorporation into the dentin and enamel of unerupted teeth; this makes the teeth more resistant to acid attack after eruption into the oral cavity. In addition, ingested fluoride is secreted into saliva. Although it is present in low concentrations, the

BOX 10-1 Basic Diet Analysis Form

WEEKLY REQUIREMENT	TOTAL SERVINGS CONSUMED	ACTUAL DEFICIENCY	PERCENT OF TOTAL
Bread, cereal, rice, and pasta group			
Vegetable group			
Fruit group			
Milk, yogurt, and cheese group			
Meat, poultry, fish, dry beans, eggs, and nuts group			

Fermentable carbohydrate exposures										
Form	*Day* of *week*									*Total* number of *exposures*
Sugar in solution	During meal									
	End of meal									
	Between meals									
Solid and retentive sugar-containing foods	During meal									
	End of meal									
	Between meals									

Grand total:

Total exposures x 20 min. _

Which specific food items are the offenders?

1.

2.

3.

4.

Suggested substitutes:

1.

2.

3.

4.

F **I G . 10-8.** Form for analysis of ingested fermentable carbohydrates.

fluoride is accumulated in plaque, where it decreases microbial acid production and enhances the remineralization of the underlying enamel. Fluoride from saliva is also incorporated into the enamel of newly erupted teeth, thereby enhancing enamel calcification (frequently called enamel maturation), which decreases caries susceptibility. As a topically applied therapeutic agent, fluoride is effective in preventing future lesion development, in arresting or at least slowing the progression of active cavitated lesions, and in remineralizing active incipient lesions. Topical fluoride also has some antimicrobial properties.

The exposure of the teeth to fluoride through professional application of fluoride solutions, gels, foams, and varnishes plus exposure from dentifrices and other fluoride preparations used at home engages almost all of the foregoing mechanisms except the preemptive incorporation into enamel. Although it is difficult to separate the benefits of the different mechanisms of action of fluoride, research has suggested that the predominant mechanism is the impact of fluoride on the remineralization of demineralized enamel. Numerous studies have shown that the presence of fluoride greatly enhances the rate of remineralization of demineralized enamel and dentin. Moreover, tooth structure remineralized in the presence of fluoride contains increased concentrations of fluorhydroxyapatite, which makes the remineralized tissue more resistant to future attack by acids than was the original structure. In view of fluoride's multiple mechanisms of action, it is not surprising that treatment with fluoride through multiple delivery systems has additive benefits. This supports the recommendation that frequent exposure to fluoride is beneficial for maximal caries prevention and control.

Communal Water Fluoridation. Research studies and observations in private practice continue to support the contention that fluoridation of the communal water supply is the most effective method of reducing the dental caries problem in the general population.

In 1998, Stookey noted that approximately half of the population of the United States enjoys the benefits of fluoridated communal water supplies.[5`']

Cohen has cited observations in Philadelphia, the first city with a population over 1 million to fluoridate its water supply.(" The reduction in DMFT has averaged 75% at 6 years of age, 54.5% at 8 years, 42.6% at 12 years, and 46.7% at 14 years. A 50% reduction in the decay rate has been noted in the primary teeth.

Murray and Rugg-Gunn reviewed 94 studies, conducted in 20 countries, to help clarify varying reports on the benefits to primary teeth of communal water fluoridation.[6'] A thorough review of the data clearly showed that water fluoridation provides protection for primary teeth against dental caries but to a somewhat lesser degree than for permanent teeth. The caries reduction benefits to primary teeth ranged between 40% and 50%, whereas the range for permanent teeth was between 50% and 60%.

Carmichael et al[62] and Rock, Gordon, and Bradnock[63] have reported data in separate studies comparing the caries incidence in children living in two fluoridated communities with that in children living in two nonfluoridated communities in England. The role of fluoridation in reducing dental caries is obvious in both studies. The study by Carmichael et al also demonstrated that children in lower social classes gain an even greater caries prevention benefit than children in higher social classes. The reason is that, as a group, the children in the lower social classes have a higher prevalence of proximal carious lesions, and proximal tooth surfaces derive the greatest benefit from fluoridation.

The protection afforded by the ingestion of fluoridated water persists throughout the lifetime of the person. Investigations by Russell and Elvolve[61] and by Englander, Reuss, and Kesel,[65] as well as other studies, have shown that the continuous ingestion of fluoridated water during adulthood decreases the prevalence of dental caries by about the same magnitude observed in children. In addition, Stamm and Banting have reported a 56% decrease in the prevalence of root-surface caries in adults who lived continuously in a fluoridated community.[`']

The posteruptive benefits associated with the ingestion of fluoridated water also have been demonstrated. Arnold et a1[6i] as well as Hayes, Littleton, and White[68] have reported that the posteruptive ingestion of fluoridated water can result in decreases in caries prevalence as great as 30%. Similarly, Hardwick, Teasdale, and Bloodworth have reported a 27% reduction in caries prevalence after 4 years of ingestion of fluoridated water by teenagers who were 12 years of age when fluoridation was initiated." These observations are consistent with the multiple mechanisms of action of fluoride cited earlier and support the significant contribution of the exposure of the teeth to fluoride even in the very low concentrations present in fluoridated drinking water.

More recent studies concerning the reduced prevalence of dental caries associated with the presence of fluoride in communal water supplies have demonstrated appreciably lesser benefits, typically ranging between 18% and 30%.[70-'- This decrease in attributable benefit is due to the so-called halo effect associated with the preparation of numerous foods and beverages in fluoridated communities and their consumption in nonfluoridated communities. Recent reports have attempted to quantify this halo effect by measuring the fluoride intake of children residing in communities that do not have a fluoridated communal water supply and have shown that fluoride ingestion is nearly 70% that of residents of optimally fluoridated communities.[7-75] Thus it is not surprising that only modest differences in caries prevalence rates are noted between children residing in fluoridated and nonfluoridated communities.

When fluoridation is discontinued in a community, an increase in the dental caries incidence will follow. Way has reported that, after a 2-year lapse of drinking fluoride-free water in Galesburg, Illinois, children experienced as much as a 38% increase in tooth decay.[7E] Lemke, Doherty, and Arra have reported that in Antigo, Wisconsin, a city of 9600, tooth decay rose 92% among kindergarten children, 183% among second-graders,

and 100% among fourth-graders when fluoridation was discontinued."

Attwood and Blinkhorn reported on the dental health of schoolchildren in Stranraer, Scotland, 5 years after fluoridation of their water supply had ended." They found that, among 5-year-old children, the dmft score had increased to 3.08 compared with 2.48 among the same age group 5 years earlier. After 5 years of no fluoridation, the scores for decayed, missing, or filled primary teeth (draft) and DMFT were quite similar to the scores of children from another city in the same region (Annan, Scotland) that had never had fluoridated water. Most of the 44% difference in dmft scores and the 50% difference in DMFT scores that previously favored the Stranraer children was lost 5 years after the fluoride had been removed from their water. The authors point out that, although there is a general decline in dental caries because of the almost universal use of fluoride toothpaste (the Annan children also had lower draft and DMFT scores at the end of the study), fluoridation of water has a significantly beneficial added effect.

Eichenbaum, Dunn, and Tinanoff reported interesting information related to the long-term impact of communal fluoridation on the private practice of pediatric dentistry.[79] A survey conducted from 1948 to 1950 showed that 86% of the pediatric patients in a private pediatric dental practice needed restorative treatment, and nearly half of these children required pulp therapy. The results of this survey encouraged the city health officials to implement dental health education and preventive programs that included communal water fluoridation. A survey of the same practice almost 30 years later (1977 to 1979) revealed a dramatic change in the restorative needs of the children. The majority of children needed no restorations, and the number of teeth with pulp involvement was negligible.

In 1986, Smith estimated that the average annual cost of fluoridating communal water supplies is approximately 25 cents per person.[80] Gish pointed out in 1979 that the annual cost varies with the size of the community and may range from approximately $1.50 per person in very small communities to as low as 10 cents per person in larger metropolitan areas.[8'] These estimates are still valid, with just upward adjustment for inflation. Communal water fluoridation remains by far the most cost-effective caries prevention measure available.

Horowitz, Heifetz, and Law have reported findings that have relevance for the millions of Americans who are deprived of the benefits of community water fluoridation because they live in areas not served by central water supplies.[42] The water supply of a rural school was fluoridated for 12 years at a level of 5 ppm, which is 4.5 times the optimum level for community fluoridation in the area. In the final survey, children who had attended the school continuously during the study had 39% fewer DMFT than did their counterparts who had not attended the school continuously. Late-erupting teeth (canines, premolars, and second molars) demonstrated twice as much caries protection as early-erupting teeth (incisors and first molars). In both categories of teeth the greatest benefits were observed on proximal surfaces, with as much as 69% less caries for late-erupting teeth.

Considerable research related to the effect of school water fluoridation on dental caries has been conducted using even higher concentrations of fluoride. Heifetz, Horowitz, and Brunelle completed another 12-year study in Seagrove, North Carolina, where school water is fluoridated at the level of 6.3 ppm, or approximately seven times the optimum recommended for community water fluoridation.[SB] Observations after 12 years show only a slight improvement in DMFS reduction compared with the earlier 12-year study using 5 ppm (or 4.5 times the optimum). The researchers concluded that there is little justification for recommending the higher fluoride level for school water fluoridation programs.

When these investigations were conducted, it was thought that school water fluoridation would provide a mechanism for children in rural, nonfluoridated areas to receive the public health benefits of fluoride. At its peak, school water fluoridation programs were in operation in 13 states, serving about 170,000 children in 470 schools. However, numerous problems associated with equipment maintenance and monitoring coupled with the recently recognized impact of increased amounts of fluoride in the food chain (i.e., the halo effect) has led the Centers for Disease Control and Prevention (CDC) to discontinue promoting these programs in the United States. In 2001 the Task Force on Community Preventive Services of the CDC strongly recommended community water fluoridation and school-based or school-linked pit and fissure sealant delivery programs for prevention and control of dental caries.[8"] However, the Task Force found insufficient evidence of effectiveness for the remaining interventions reviewed, including school water fluoridation.

Fluoride-Containing Dentifrices. Extensive research initiated in the early 1950s ultimately resulted in the identification of the first fluoride-containing dentifrice* capable of decreasing the incidence of dental caries. This dentifrice contained stannous fluoride (SnF_2) in combination with calcium pyrophosphate as the cleaning and polishing system and in 1964 was accepted as the first therapeutic dentifrice by the Council on Dental Therapeutics of the American Dental Association (ADA) based on more than 20 clinical trials. The significance of this original development has been profound;

*Crest, Procter & Gamble, Cincinnati, Ohio.

in fact, a review by Jenkins concluded that the general decline in caries prevalence in Britain and other developed countries appears to be attributable in large part to the widespread use of effective fluoride-containing dentifrices." Caretakers should be counseled so they know that no more than a small, pea-sized amount of fluoridated toothpaste should be used when they brush the teeth of infants and other very young children.

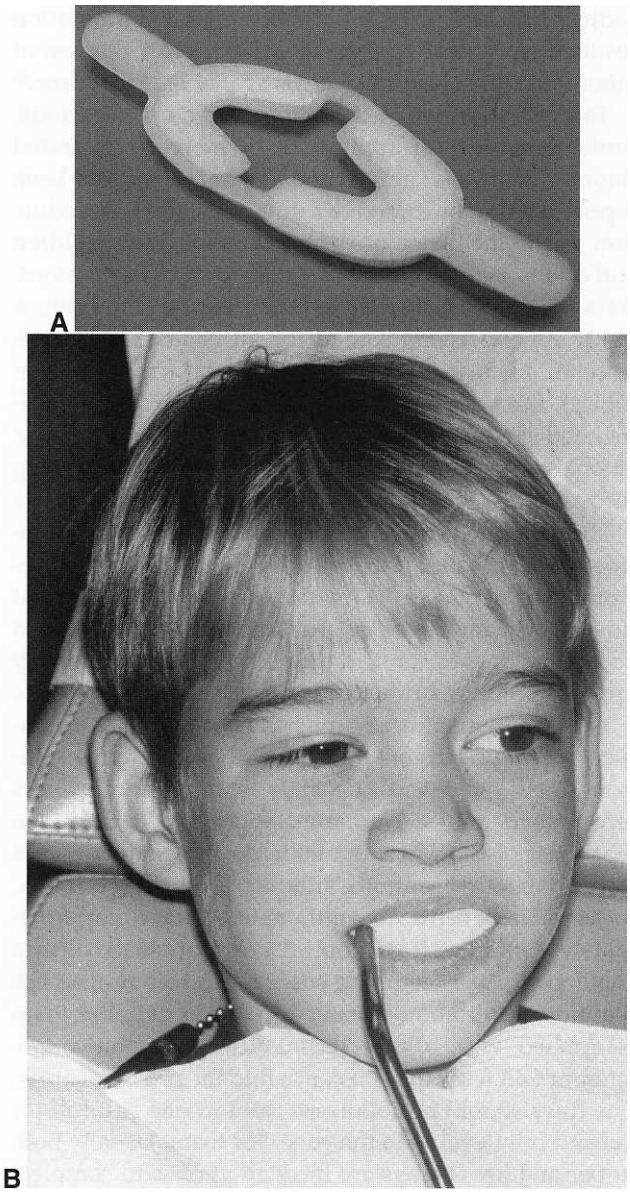
For nearly three decades after the completion of the first successful clinical trial in 1954, this stannous fluoride dentifrice served as a standard of reference for the development of additional fluoride dentifrices as well as for efforts to identify even more effective compositions. An extensive review of the literature by Stookey in 1985 indicated that more than 140 clinical trials of fluoride dentifrices had been reported." These investigations identified several fluoride dentifrice systems with demonstrated cariostatic activity and verified the commercial availability of many such systems. In the United States at the present time, 49 dentifrice formulations from 15 manufacturers have received the Seal of Approval of the ADA. These ADA-accepted dentifrices now comprise approximately 90% of all dentifrices sold in the United States. Fluoride dentifrices similarly represent 85% to 90% of the dentifrice sales in Britain as well as several in other countries.

Before 1981, attempts to identify a fluoride dentifrice system significantly more effective than the original stannous fluoride formulation were unsuccessful. However, in 1981 the results of two clinical studies demonstrated the superiority of a sodium fluoride composition.[$'•$S] A clinical study of 3 years' duration was conducted by Beiswanger, Gish, and Mallatt[S'] to determine the effect of a sodium fluoride—silica abrasive dentifrice on dental caries. The dentifrice, containing 0. 243% sodium fluoride, was compared with stannous fluoride in a study group of 1824 schoolchildren 6 to 14 years of age in Indiana cities where water supplies were fluoride deficient (containing less than 0.35 ppm fluoride). After 3 years the group brushing with the sodium fluoride dentifrice had significantly lower DMFT and DMFS increments than the group brushing with the stannous fluoride dentifrice. Two independent examiners found that the reductions were 14.8% and 10.5% for DMF1' and 16.4% and 13.1% for DMFS. These results are consistent with those reported by Zacherl in which the sodium fluoride dentifrice resulted in a 40.7% decrease in DMFS compared with a 23.4% decrease observed with the stannous fluoride dentifrice." Similarly, studies conducted by Gerdin[n'] and by Edlund and Koch[90] indicated that the use of sodium fluoride dentifrices by children resulted in significantly fewer caries than the use of dentifrices containing sodium monofluorophosphate. The results of these studies and several similar reports led Stookey to conclude in 1985 that the use of sodium fluoride in a highly compatible formulation resulted in greater cariostatic activity than the use of other fluoride dentifrice systems available at that time."

Topical Fluorides in the Dental Office. The periodic professional topical application of more concentrated fluoride solutions, gels, foams, or varnishes has been repeatedly demonstrated to result in a significant reduction in the incidence of dental caries in both children and adults as well as the arrestment of incipient lesions. As a result, professional topical fluoride applications are routinely recommended for all children and adolescents. Even in the absence of dental caries activity, topical fluoride applications to children are recommended as a means of raising the fluoride content of the enamel of *newly* erupted teeth and thereby increasing the resistance of these teeth to caries formation.

Historically the periodic topical application of fluoride was first demonstrated to be effective for the prevention of dental caries in the early 1940s. Since that time, many hundreds of publications have provided additional data to confirm the efficacy of professionally applied topical fluoride treatments for caries prevention.

A 4-minute treatment time has been typically recommended for professionally applied topical fluoride solutions, gels, or foams. Recently some manufacturers recommend only a 1-minute application. It is known that most of the fluoride uptake in the enamel occurs during the first minute after application. However, measurable benefits do continue to accrue for approxi-mately 4 minutes if the topical preparation remains in contact with the teeth. We continue to recommend the 4-minute application whenever possible. If gel or foam is applied with a tray technique, use of an ample amount will force the substance into the proximal areas. The trays should be about one third to one half full for gel and full (level with the edge) for foam. Usually both upper and lower trays are inserted at once to complete the topical fluoride treatment in one 4-minute application. Some trays are supplied as a connected double set (Fig. 10-9). The patient sits in an upright position with head tipped slightly forward to allow excess saliva and fluoride to flow toward the lips. Patients who follow instructions well may be provided with the high-velocity evacuator tip to help control the drooling themselves, or they may be given a plastic "drool bag" that enables them to tip the head forward even more and catch the drooled liquid in the bag, which is later discarded. The dentist or appropriate office staff should supervise the treatment and provide assistance as needed. If necessary the auxiliary may manipulate the evacuator tip or help hold the patient's head forward and the trays in place over the drool bag. Patients requiring assistance also often need positive reinforcement during the procedure. Extra caution and special application techniques are required when topical solutions, gels, or foams are



f l **G 10-9.** A, Sufficient topical fluoride foam is placed in the upper and lower trays so that each tray is filled approximately level with the edge of the tray. **B,** The teeth are dried and the trays are inserted in the patient's mouth to provide a complete-mouth topical fluoride treatment in a single 4-minute application. The evacuator tip is positioned to remove the excess saliva and foam as the liquids flow toward the lips.

placed in the mouths of young children (around 4 years old and younger). The agent is usually brushed on to the teeth in small amounts and the excess is wiped away with gauze.

The results of independent clinical trials have seriously questioned the need for dental prophylaxis before the topical application of acidulated phosphate fluoride (APF). Ripa et al compared the caries incidence during a 3-year period in children given semiannual topical applications of APF after different cleaning procedures."' Before each fluoride treatment the children either received conventional prophylaxis using a nonfluoride prophylactic paste, performed supervised toothbrushing and flossing, or rinsed their mouths with water. Caries increments after 3 years were essentially identical in all the treatment groups, a finding which indicates that the manner of cleaning the teeth before the APF treatment may not influence the cariostatic activity of the fluoride applications. Similarly designed clinical trials were conducted by Houpt, Koenigsberg, and Shey[92] and by Katz et al"; in both instances the results were comparable to those observed by Ripa et al."' An additional study by Bijella et al likewise demonstrated that the presence or absence of a prophylactic procedure did not alter the efficacy of a topical APF application." Collectively these studies indicate that prophylaxis before an APF topical application may be an optional procedure with regard to caries reduction.

Beginning with reports by Ekstrand et al" and by LeCompte and Whitford," several investigators have expressed concern regarding the amount of fluoride swallowed by children during a topical fluoride application. These reports indicated that, depending on the manner of application, 15 to 31 mg of fluoride may be swallowed during the treatment. Stookey et al explored the feasibility of permitting patients to rinse after a topical fluoride application as a means of reducing fluoride ingestion."' However, they observed that rinsing with water after a topical fluoride application significantly decreased fluoride deposition in incipient carious lesions. A report by LeCompte and Doyle indicated that one could drastically reduce the amount of fluoride ingested during topical fluoride application to about 1.6 mg by using only the necessary amount of fluoride gel, having the patient seated in an upright position, using high-velocity saliva evacuation during the treatment, and asking the patient to expectorate thoroughly for 1 minute after completion of the treatment."' Thus the latter procedures are recommended to minimize fluoride ingestion during the application, particularly in younger children, and the patient should be encouraged not to eat, drink, or rinse for 30 minutes after the treatment to maximize fluoride uptake in enamel.

Fluoride-containing varnishes have been widely used in Europe and other parts of the world for approximately 40 years but were not available in the United States until 1994. The first fluoride varnish was introduced in Europe in 1964 and contained 5.0% sodium fluoride (or 2.26% fluoride). A second product was introduced in Europe in 1975 and contained 0.9% silane fluoride (or 0.1% fluoride). Much more research has been conducted using the sodium fluoride system, and it is the most widely accepted.

Petersson," Horowitz and Ismail,[100] and Petersson et al [101] have reviewed the numerous controlled clinical trials of fluoride varnishes and concluded that these materials are equally as effective as professional topical fluoride applications for the prevention of dental caries in children. Seppa et al investigated the effect of the sodium fluoride varnish in children with a high past caries experience and found that this measure resulted in numerically fewer new caries lesions than was achieved with semiannual applications of an APF gel during a 3-year study period.[1Q7]- These investigators also noted that the data for use of this varnish satisfied the criteria of the ADA for the claim of being "at least as good as" professionally applied topical fluoride gels. Helfenstein and Steiner performed a statistical meta-analysis of the data from a number of clinical trials and found that use of the sodium fluoride varnish resulted in an overall reduction in caries of 38% in the permanent teeth.[103] In general, slightly lower average benefits of about 30% have been observed in studies of varnish use on primary teeth.

The sodium fluoride varnish is particularly recommended for use in preschool-aged children because of its ease of application and equal efficacy to APF systems.[S9] Before application of the varnish, the teeth receive prophylaxis or are brushed with a dentifrice to remove plaque and oral debris. The varnish is then applied with a soft brush, with reapplications recommended at 4- to 6-month intervals. A more intensive annual treatment regimen consisting of three applications within a 10-day period has been investigated, and this regimen was observed to be as effective as applications every 4 months.[104-106] Furthermore, single annual applications have been found to be without clinical benefit. The varnish has a brown color but is lost from the tooth surfaces within 24 to 48 hours. Because less than a milliliter of varnish is used for a professional treatment in preschool-aged children, the amount of fluoride that will ultimately be ingested when the varnish is lost from the tooth surfaces is less than 3 mg. Thus there are no practical concerns regarding safety, and this procedure is frequently recommended for use in young children in place of the traditional topical fluoride gel application.

Topical Fluorides for the General Anesthesia Patient. The application of fluoride to the teeth of children receiving dental care under a general anesthetic, after the placement of restorations, is certainly recommended. Thorough prophylaxis should precede the placement of the rubber dam for a quadrant of restorations. The fluoride should be applied after the restorative work has been completed for that quadrant but before removal of the rubber dam.

Home Fluoride Mouthrinses and Gels. The use of dilute oral fluoride rinses and/or gels as an additional dental caries control measure has become another helpful adjunct. Children under 4 years of age may not have full control over their swallowing reflexes; therefore, caution should be exercised in recommending these products for home use for this age group. However, some small children can expectorate rinses quite reliably under proper supervision, or the parent can brush on the gel and wipe away the excess. In non-fluoridated areas it may be appropriate to have the child swallow the rinse after "swishing" it to gain the systemic fluoride effect as well. Some rinses (acidulated or aqueous solutions that do not contain alcohol) are pre-pared to provide the necessary supplemental systemic dose of fluoride if the child does not drink fluoridated water. However, current Food and Drug Administration (FDA) regulations specify that fluoride rinses not be recommended for children under 6 years of age unless they are intended for use as a systemic fluoride supplement.

Radike et al observed schoolchildren who rinsed their mouths once each school day for 2 school years with a stannous fluoride mouthrinse containing 250 ppm fluoride (about 0.1% stannous fluoride).[107] There was a significant reduction in dental caries at the end of the first and second school years. Two independent examiners found caries reductions of 33% and 43% in DMFS scores. The anticaries benefit from the stannous fluoride mouthrinse was especially encouraging because the children already were receiving the optimum benefit of water fluoridation.

APF mouthwashes in concentrations of either 100 or 200 ppm fluoride ion used twice daily have been shown by Finn et al to be effective.[10"] They reported that during a 26-month period the APF rinses containing relatively small amounts of fluoride were effective in reducing dental caries.

Miller made a preliminary report on results of the National Caries Program sponsored by the National Institute of Dental Research.[1°J] A nationwide school-based fluoride mouthrinse program that included 85,000 children in 17 states and Guam produced encouraging results. The schoolchildren rinsed their mouths for 60 seconds once weekly with 5 ml of a 0.2% neutral sodium fluoride solution. Children in the 7-, 11-, and 13-year-old groups experienced a reduction in dental decay. There was also evidence of caries decrease in the primary teeth.

Leske et al[IIO] and Leske, Ripa, and Green' have reported on results of a fluoride rinsing program that was begun in 1975 in the Three Village Central School District of Long Island, New York. The program was one of 17 demonstration projects sponsored by the National Institute of Dental Research, which initially enrolled approximately 4500 elementary schoolchildren (kindergarten through the sixth grade), who rinsed once a week with a 0.2% neutral sodium fluoride solution

under the supervision of homeroom teachers. The prevalence of caries in the children who participated in the rinse program for 2 to 4 years was 20.4% DMFT and 24.4% DMFS lower than that of children who had never rinsed. The reduction in proximal caries was 49.6%. The reductions in occlusal and buccolingual caries were 21. 0% and 18.8%, respectively. The examination of exfoliated teeth also indicated that fluoride rinsing may produce a residual benefit. The later report on children who had participated in the program for 7 years demonstrated caries prevalence reductions of 55.7% DMFI and 60.9% DMFS, with 85.4% reduction of proximal surface caries.

Extensive field research has been conducted on the use of fluoride mouthrinses. Most studies incorporate the use of a 0.2% sodium fluoride rinse once weekly or a 0.05% sodium fluoride rinse once daily. As does the work previously summarized, these other studies show unquestionable caries prevention benefits of the regular use of self-administered fluoride rinses when properly supervised. These benefits accrue to primary and permanent teeth, and seem to be helpful both in fluoridated areas and in areas with nonfluoridated water. The studies of Heifetz[12] and of Ripa and Leske"[3] are cited in the references listed at the end of this chapter as good examples of clinical research with fluoride rinses. Based on numerous studies indicating the efficacy of fluoride rinses, some rinses were approved by the FDA in 1974 and by the ADA Council on Dental Therapeutics in 1975. Studies supporting the use of the dilute gels are not as numerous as those for the rinses, but some gels have also subsequently been approved.

Dietary Fluoride Supplements. A review of the literature on the value of fluorides administered during pregnancy fails to disclose any valid evidence to sup-port such use even in nonfluoridated areas. In 1966 the FDA banned the manufacturers of fluoride supplements from marketing products bearing the claim that dental caries would be prevented in the offspring of women who used such products during pregnancy. The FDA took this action because of the insufficiency of clinical evidence to substantiate such a claim. There was no question of safety. Although some medical and dental practitioners have continued to prescribe dietary fluoride supplements for pregnant women since the FDA's advertising ban, generally there is unanimity among most research experts and public health officials that fluoride ingestion by gravid women does not benefit the teeth of their offspring, at least not the permanent teeth.

Participants in a symposium concerning the use of prenatal fluorides agreed that transfer of fluoride does occur from the mother to the fetus through the placenta.' `' Only in recent years has such a transfer been generally acknowledged. How much fluoride is transferred from the blood of the mother to the blood of the fetus, however, remains uncertain. Nevertheless, the members of the symposium generally agreed that some benefit of caries prevention accrues to the primary teeth when the fetus is subjected to fluoride. There was considerable doubt about the benefit to the permanent teeth. The symposium discussions seemed to lead to the conclusion that additional well-controlled research efforts are needed to define more clearly the possible benefits of prenatal fluoride administration. Some research into this issue continues, as does the controversy.

A study by Katz and Muhler suggested that the effect of fluoride on primary teeth is mainly postnatal.'" In a study to determine the effect of waterborne fluoride on dental caries in primary teeth, 890 children from 4 to 7 years of age were examined in one Indiana city having a communal water supply with only 0.05 ppm fluoride and in three cities having a supply with a concentration of 1 ppm fluoride. Children living in the cities with fluoridated water had between 35% and 65% fewer dental caries in their primary teeth than those living in the fluoride-deficient city. Comparisons of dental caries incidence in the primary teeth of children living in the same city who were exposed either prenatally or postnatally or exclusively postnatally showed no difference between the groups.

Hennon, Stookey, and Muhler conducted a clinical study that included 815 children between 18 and 39 months of age residing in three fluoride-deficient Indiana communities.'" The children received chewable tablets containing vitamins, vitamins plus fluoride, or fluoride alone (1 mg as sodium fluoride) and were examined for dental caries initially and at 6-month intervals. The findings during the first 2 years of the 5 '/2-year study indicated a significant reduction of about 37% in the incidence of dental caries after 6 months in the children ingesting either the fluoride or vitamin-fluoride supplements.'" This degree of protection increased to about 55% and 63%, respectively, after the children had used the supplement for 1 and 2 years. The important finding was the significant reduction in the incidence of dental caries after use of the tablets for only 6 months. This observation indicates a highly effective topical benefit of chewing the tablets, because most of the primary teeth had already erupted when the study began.

Many other studies have demonstrated the anticariogenic effectiveness of fluoride supplements. If children do not have the benefit of drinking water containing an optimum fluoride concentration, supplements should be prescribed in accordance with the dosages recommended in Table 10-3. The natural fluoride content of the water should first be determined. If the natural fluoride content is 0.6 ppm or higher, supplements should not be administered. If the fluoride content is below 0.6 ppm, the administration of fluoride

TABLE 10-3 Dietary Fluoride Supplementation Schedule

AGE	LESS THAN 0.3 ppm F	0.3-0.6 ppm F	MORE THAN 0.6 ppm F
Birth-6 mos	0	0	0
6 mos-3 yrs	0.25 mg	0	0
3 yrs-6 yrs	0.50 mg	0.25 mg	0
6 yrs up to at least 16 yrs	1.00 mg	0.50	0

From American Academy of Pediatric Dentistry: Guideline on fluoride therapy, *Pediatr Dent* 24(7 suppl):66, *2002* (special issue: Reference manual 2002-2003).

supplements should commence as indicated in Table 10-3 and should continue through the time of the eruption of the second permanent molars.

A number of studies report the caries-preventive effects of adding fluoride to a variety of foods and beverages. Fluoride has been used as a caries-preventive additive in salt, milk, and even sugar. Some suppliers now offer controlled concentrations of fluoride in bottled water. Numerous reports show that these prod- ucts can have measurable and favorable results when used as intended. Such products are designed for use by specific and targeted population groups. These products have not yet had widespread popularity, although there is growing interest in this concept and public acceptance seems to be growing for fluoridated bottled water for children who either do not have or do not use community fluoridated water.

Combinations of Fluoride Therapies. There is considerable evidence to suggest that using combinations of therapeutic fluoride agents often produces additive anticariogenic effects. The evidence also indicates that the earlier fluoride therapy is initiated in children the more effective the caries control will be. However, one must use caution in prescribing multiple therapies in *very* young children to avoid excessive fluoride ingestion.

Fluoride is the most effective caries-prevention agent commercially available today. Except in a patient who has a fluoride allergy (very rare), it is considered completely safe when properly used. The ingestion of high concentrations can lead to nausea, vomiting, dental fluorosis (mottling), or, in extreme cases, even death, especially in children. Extreme care must be used to safeguard the agents from inappropriate and/or inadvertent ingestion. It is imperative that the dental profession have full awareness of the hazards accompanying its use and yet be prepared to use it to the patient's maximum advantage through careful consideration of each patient's individual situation.

Chlorhexidine and Thymol. As an oral antimicro- bial, chlorhexidine has been used in oral rinses, dentifrices, chewing gum, varnish, and gel. In the United States it is used most often in the form of a prescription oral rinse. Many children object to the taste of these products, but they have been shown to be effective against microorganisms causing both caries and periodontal disease. Thymol has also been included with chlorhexidine in some varnish preparations. To date, these products have not shown superior caries prevention results when compared to multiple fluoride therapies and they may require more frequent application to be effective. However, they should provide an additive effect and may be used in combination with fluoride, particularly in high-risk patients. In the rare situation in which a provider is trying to control or prevent caries in a patient with fluoride allergy, they could be very important therapies.

Povidone Iodine. Considerable data exist from laboratory and animal studies to confirm the dramatic suppression of mutans streptococci by iodine. Several studies reconfirm this observation in humans as well. Lopez et al published two human studies demonstrating that topical application of povidone iodine at 2-month intervals in dentate infants is effective in preventing S-ECC in children at high risk for the disease.[118,19] The authors called for more and larger in-depth clinical trials. If such trials prove as successful as these initial reports, they could hopefully lead to wide-spread implementation of an effective defense against this devastating disease for which minimal parental compliance is required.

Xylitol. Xylitol is a low-calorie sweetener that inhibits the growth of S. *mutons.* Numerous studies seem to confirm its anticariogenic capability. Xylitol has been tested as an additive to a variety of foods and to dentifrice. However, the vast majority of published data come from studies in which xylitol was incorporated into chewing gum. Makinen has reported numerous studies on the topic, most of them performed with many different coworkers in different parts of the world. In 2000, he published a concise summary titled "The Rocky Road of Xylitol to Its Clinical Application." [120] The available data not only show that xylitol chewing gum reduces caries activity but also provide evidence that it decreases the transmission of S. mittens Irom gum-chewing mothers to their children. The use of xylitol chewing gum seems to be gaining popularity as another caries prevention strategy. It should be readily accepted by many children.

RESTORATIVE DENTISTRY IN THE CONTROL PROGRAM

Excellent restorative dentistry is also valuable in the dental caries control program. For patients who do not comply with nonrestorative caries control recommendations (e.g., use of fluorides and/or antimicrobials, appropriate diet, and plaque control), the restorative

care will be more aggressive than it would otherwise need to be. Further relevant discussion of appropriate selection of restorative materials and procedures can be found in Chapters 16, 17, and 18.

PIT AND FISSURE SEALANTS

For many years, attempts have been made to eliminate pit and fissure caries. In 1923 Hyatt recommended that all pits and fissures be opened with a bur and filled with amalgam.[12'] Attempts have been made to prevent pit and fissure caries by applying a variety of different chemical solutions, including silver nitrate and potassium ferrocyanide with zinc chloride, and by covering the depressions with various types of dental cement. None of these methods proved successful over time.

Pit and fissure sealants have been used successfully by many dentists. Because many recent clinical studies have shown that the placement of pit and fissure sealants is an effective dental caries—preventive mea-sure, the subject is covered in detail in Chapter 17.

CARIES VACCINE

A vaccine to prevent the disease of dental caries has been an anticipated scientific breakthrough since at least the early 1940s. Most recent research efforts assume that *S. rnutans* is the principal etiologic organism of dental caries, and the development of a method of immunization specifically targeted at neutralizing S. *rnutans* has been a major thrust of caries vaccine research. Bowen reported that monkeys remained caries free for more than 6 years after the animals received intraoral injections of killed *S. mutans,* even though the monkeys were fed highly cariogenic diets and had severe malocclusion that would predispose them to caries.[122]

Most current research is being directed toward a greater understanding of the immune system and specifically of immune responses to mutans streptococci. The route of administration of the vaccine is usually mucosal absorption by intraoral or intranasal tissues. It is beyond the scope of this chapter to present detailed information regarding this complex subject area. A review article by Michalek, Katz, and Childers is cited in the references for the reader who would like more information about this fascinating area.'[23]

DENTAL CARIES ACTIVITY TESTS

For the larger part of a century, dental scientists have been trying to develop a convenient method of measuring quantitatively the degree of dental caries activity in individual patients. Techniques requiring laboratory procedures to determine oral bacterial counts or their aciduric potential have been developed and used. More recently, convenient paper test strips to gauge salivary microbial density in patients have been tested. No truly convenient and efficient test method has yet been developed that has sufficient accuracy to be a reliable caries activity indicator. Research continues in this area because having an accurate, convenient, and efficient test to measure early caries activity and its level of potential, especially in young children, would be a very useful diagnostic tool for private practitioners and public health assistance providers.

DENTIST'S ROLE IN THE CARIES CONTROL PROGRAM

The success of a dental caries control program depends to a great extent on the interest and cooperation of the patient and/or the patient's caretakers. Rampant caries should not be viewed as a hopeless problem. Diagnostic, therapeutic, and preventive measures are available to control it. In the clinical management of rampant caries, the dentist's role consists of seeking and eliminating the cause to the extent possible. This includes trying to correct inappropriate habits or deficiency states that may be contributing factors, restoring the salvageable teeth to good form and function, and, finally, making use of all available therapeutic preventive and control measures in an established, ongoing manner.

Successful management of all active dental caries problems also requires careful and complete dental and medical history taking, the use of currently accepted diagnostic aids, initiation of a comprehensive preventive program, the application of sound principles of restorative dentistry, and establishment of regular recall schedule for maintenance and reemphasis of the preventive procedures. The recall appointment should be set at each visit based on the clinician's judgment of the patient's risk for future disease at that time.

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CHAPTER

11

Mechanical and Chemotherapeutic Home Oral Hygiene

JEFFREY A. DEAN

CHRISTOPHER V. HUGHES

انتشارات
شایان نمودار

CHAPTER OUTLINE

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- Powered Mechanical Plaque Removal
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- Disclosing Agents
- Other Adjuncts for Plaque Control
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- Visual-Motor Skill Mastery
- Time Considerations

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- Preschoolers (3 to 6 Years Old)
- School-Aged Children (6 to 12 Years Old)
- Adolescents (12 to 19 Years Old)
- In-Office Oral Hygiene Programs

As the technological level of health care increases, it is important not to lose sight of the basics of patient care. In dentistry, this means establishing and maintaining effective preventive habits in our patients. No matter how sophisticated our dental techniques and procedures have become, preventive dentistry is the foundation on which all oral health care must be built.

In 1960 McDonald discussed how pediatric medicine had changed in the previous 30 years (1930 to 1960) from 90% treatment and 10% prevention to just the reverse.' He stated that preventive measures for dentistry were available and remained to be applied, as they had been in pediatrics. With this preventive philosophy, dentistry, particularly dentistry for children, has come a long way toward reaching this ratio of 90% prevention to 10% treatment.

At the core of this preventive foundation is home oral hygiene and plaque control. The area of oral hygiene has undergone recent developments that have turned a mundane subject into a field of surprising growth and research. Modern biology has made new inroads in the area of plaque control and will continue to exert a strong influence on how we look at oral hygiene and plaque in the future. The traditional focus of oral hygiene has been and will continue to be the control of the two most prevalent oral diseases, caries and periodontal disease. Although plaque control is essential to oral hygiene, it is important to realize that, unlike with periodontal disease, no clear relationship exists between plaque control and the prevention of caries. As discussed in Chapter 10, the complex etiology of decay centers on the following factors: tooth susceptibility, bacterial plaque, refined carbohydrates, and time. Many other variables, such as oral sugar clearance and salivary flow and pH, add to the complexity of this process. This complex etiology may help explain the difficulty in demonstrating a relationship between oral hygiene practices and caries prevention.

Despite this ambiguity, plaque control remains an essential element for oral health. Although Marsh has shown that the natural oral microflora confers several benefits on the host, in the absence of oral hygiene, dental plaque accumulates, which leads to shifts in bacterial populations away from those associated with health.' Treatment should therefore be designed to control rather than to eliminate dental plaque.

Not only have there been advances in biology, but the public's consciousness regarding home oral hygiene has been raised to new levels by the advertising of home health care products. The oral care market in the United States is expected to reach nearly $8 billion by 2007. Health and cosmetic awareness by patients is possibly at an all-time high; they are willing to pay for the best in health products.

This chapter addresses the broad area of home oral hygiene for the child and adolescent, from the biology of plaque development to plaque removal techniques and patient motivation. Dental health care professionals need to make home oral hygiene the core of their preventive foundation.

MICROBIAL ASPECTS OF ORAL HYGIENE AND PLAQUE FORMATION

Although Miller proposed in the late nineteenth century that microorganisms play a role in dental disease; definitive evidence of the microbial etiology of dental caries and periodontal diseases did not appear until three quarters of a century later with the work of Keyes`' and of Loe, Theilade, and Jensen.' Since these seminal studies were performed, the major focus of dental research has been to define the specific microorganisms in dental plaque that mediate these diseases. Although great progress has been made in identifying these pathogens, our primary tools in preventing dental diseases remain mechanical removal of plaque and promotion of the remineralization of the tooth surface. Therefore the following brief review of the timing, mechanisms, and biology of plaque formation provide a scientific rationale for any clinical program of oral hygiene and prevention.

Moore has demonstrated that the oral cavity naturally contains more than 300 bacterial species and numerous distinct bacterial habitats. Interestingly, only a limited number of species are found in high numbers in dental plaque.' These species are uniquely suited to this habitat. The formation of plaque on the tooth surface is characterized by progression from a limited number of pioneer species (mainly streptococci and other gram-positive organisms) to the complex flora of mature dental plaque. This maturation involves initial adherence of bacteria to the salivary pellicle and subsequent formation of a complex multispecies biofilm. Most oral bacteria have evolved specific adherence mechanisms that enable them to colonize the tooth surface. In addition, bacteria undergo a number of phenotypic changes as they initiate the formation of a biofilm. The molecular mechanisms that underlie these processes have been intensively studied. Kolenbrander has provided reviews of these areas.' Although their reports offer the possibility of new methods of plaque control, mechanical plaque removal with supplementation by chemotherapeutic agents currently offers the most practical method of controlling plaque.

Not only do microbial changes occur as plaque matures on the tooth surface, but mature dental plaques

associated with oral diseases appear to differ from those associated with oral health. Many studies have demonstrated that, in dental caries, the pathogenicity of plaque is related to the numbers of *Streptococcus mutans* and related species present (see Balakrishnan et an. In contrast, the plaques associated with gingival inflammation are characterized by a predominance of gram-negative bacteria rather than the predominantly gram-positive flora found in oral health. This transition seems to coincide with inflammatory changes that occur at the gingival margin. Plaque control efforts should be directed toward two goals: (1) limiting the numbers of mutans streptococci in dental plaques for prevention of caries by mechanical elimination of supragingival plaque and limitation of dietary sucrose, and (2) maintaining the predominantly gram-positive flora associated with gingival health by mechanical removal of plaque from the subgingival area on a regular basis. The use of chemotherapeutic agents, particularly chlorhexidine, can also play a role in maintenance of gingival health. The incorporation of these methods into the daily routines of patients and their parents is perhaps the greatest challenge facing the dentist.

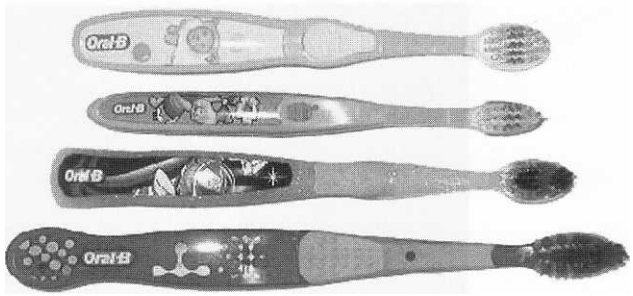
MECHANICAL METHODS OF PLAQUE CONTROL

Mechanical methods of plaque control are the most widely accepted techniques for plaque removal. Toothbrushing and flossing are the essential elements of these mechanical methods; adjuncts include disclosing agents, oral irrigators, and tongue scrapers.

MANUAL TOOTHBRUSH

The toothbrush is the most common method for removing plaque from the oral cavity. A number of variables enter into the design and fabrication of toothbrushes. These include the bristle material; length, diameter, and total number of fibers; length of brush head; trim design of brush head; number and arrangement of bristle tufts; angulation of brush head to handle; and handle design. In addition, many features, such as the use of neon colors or familiar cartoon caricatures, are designed to attract the attention of potential purchasers (Fig. 11-1).

Today, most commercially available brushes are manufactured with synthetic (nylon) bristles. Park, Matis, and Christen identify the bristle and head of the toothbrush as the most important part of the brush, and note that the length of most bristles is 11 mm.[9] Brushes are classified as soft, medium, or hard based on the diameter of these bristles. The diameter ranges for these classifications are 0.16 to 0.22 mm for soft, 0.23 to 0.29 mm for medium, and 0.30 mm and higher for hard. In addition to the bristle diameter, the bristle end has been studied to determine the most beneficial



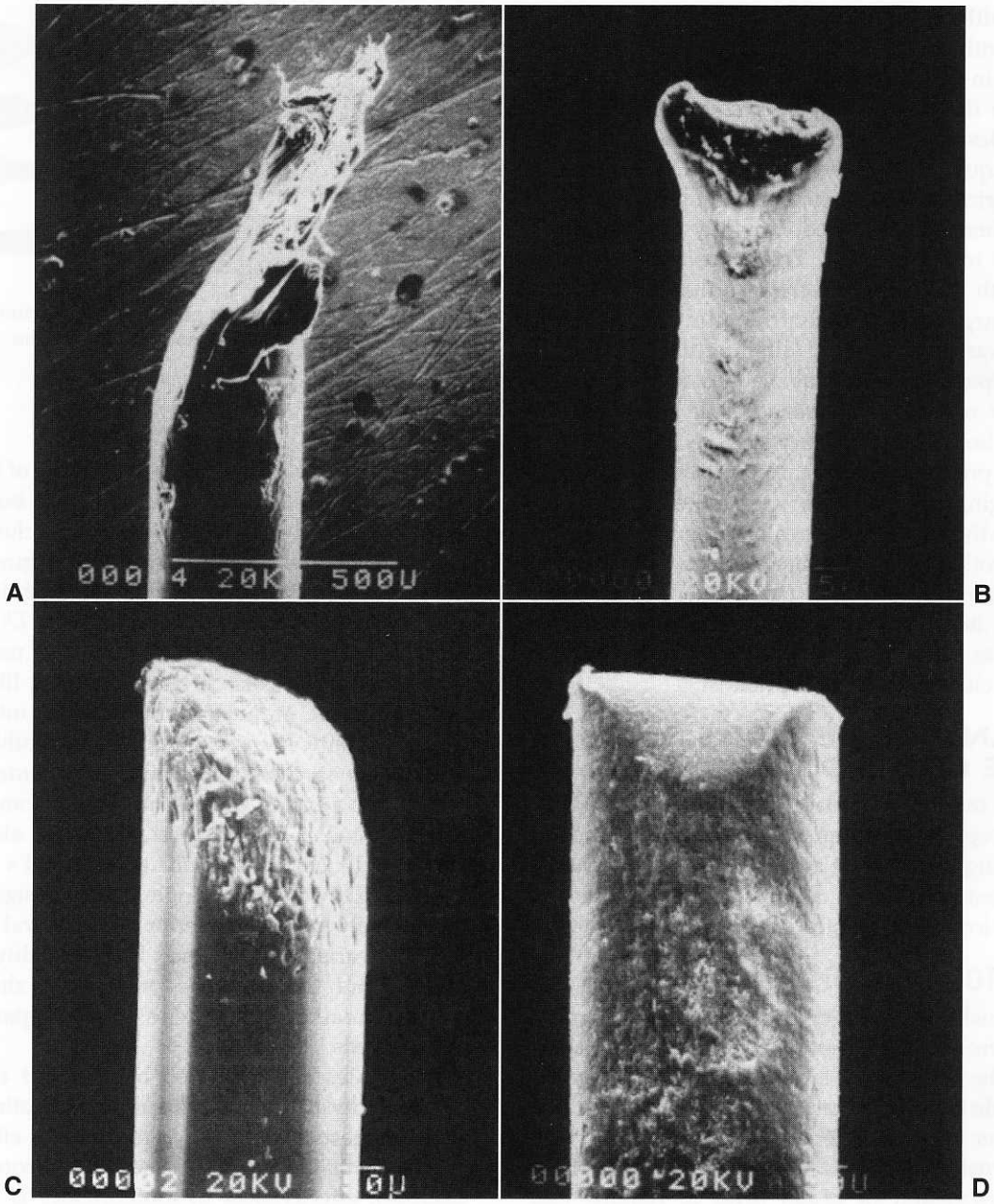
I I I . 11-1. Features such as neon colors or cartoon characters on toothbrushes are designed to attract the attention of purchasers.

type for plaque control. Of the three types of bristle ends (Fig. 11-2, A to C), coarse-cut, enlarged bulbous, and round, the round end is the bristle type of choice because it is associated with a lower incidence of gingival tissue irritation. However, even the coarse-cut bristles round off eventually with normal use (Fig. 11-2, D).

The soft brush is preferable for most uses in pediatric dentistry because of the decreased likelihood of gingival tissue trauma and increased interproximal cleaning ability. In evaluating the best toothbrush head and handle for children, Updyke concludes that it is best to use a brush with a smaller head and a thicker handle than on the adult-size brush to aid in access to the oral cavity and facilitate the child's grip of the handle.[10] However, no single brush design has been scientifically proved superior for removal of plaque. Multiple variables influence a brush's ability to remove plaque, and therefore the practitioner should make recommendations only after assessing a patient's individual needs.

Wear rates of toothbrush bristles and their subsequent ability to remove plaque raise another concern. Dean suggests that toothbrushes remain effective well after patients identify a brush as being worn out." The cleansing effectiveness of toothbrushes is maintained until pronounced toothbrush wear has occurred. This implies that patients are much more likely to dispose of a brush well before its clinical usefulness actually ends than to continue to use a toothbrush that no longer cleans effectively. In this regard, one manufacturer claims that their commercial toothbrush* indicates when the brush should be replaced by means of centrally located tufts of bristles dyed with food colorant. When the blue band fades to halfway down the bristle, it is time to replace the brush (Fig. 11-3). The company states that on average this occurs after 3 months but that the time varies depending on the individual's brushing habits.

*Oral-B Indicator, Oral-B Laboratories, Inc., Belmont, Calif.



f **I C 11-2.** Scanning electron micrographs of toothbrush bristles manufactured by different processes. A, Coarse-cut bristle end, probably the result of an incomplete single-blade cut.

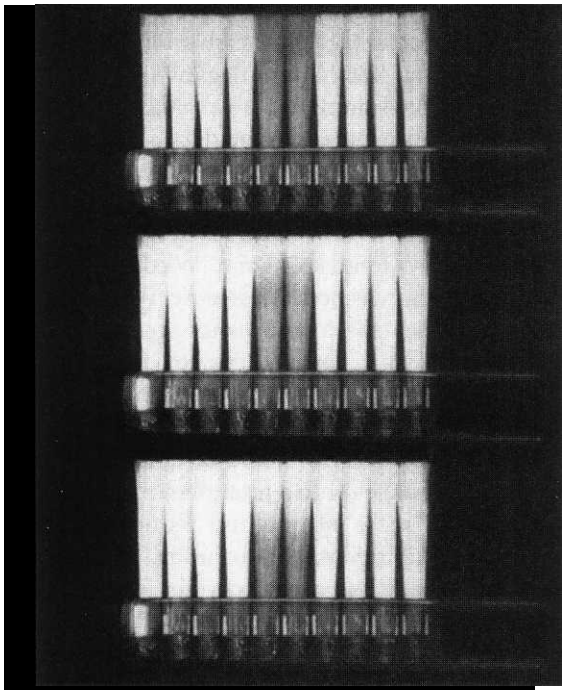
Slightly enlarged, bulbous nylon bristle end, resulting from a double-blade or scissor cut.

Tapered or round-end nylon toothbrush bristle produced by heat or a mechanical polishing process. D, Scrubbing, mechanical action of a toothbrush wear machine has nicely rounded off this bristle removed from a brush that was originally coarse-cut. *(From Park KK, Matis BA, Christen AG:* Clin Prev Dent 7(4):5-70, 7985.)

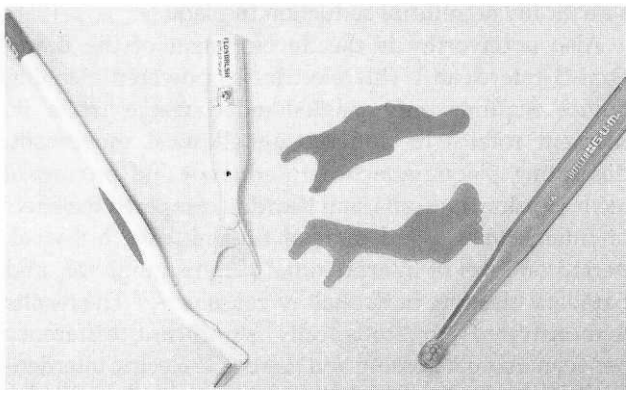
Parents frequently ask how often *they* should change a child's brush. Based on the studies just mentioned, the best advice is to replace the brush when it appears well worn. This can present some problems for parents, because some children, especially toddlers, chew their brushes when brushing, which rapidly gives the bristles a well-worn appearance.

FLOSS

Although toothbrushing is the most widely used method of mechanical plaque control, toothbrushing alone cannot adequately remove plaque from all tooth surfaces. In particular, it is not efficient in removing interproximal plaque, which means that interproximal cleaning beyond brushing is necessary. Many devices

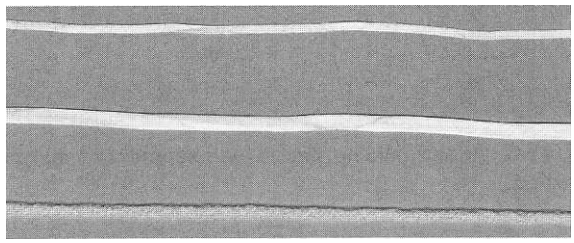


[IG **11-3.** Blue dye in the center bristle tufts of this brush fades down from the end with use. When the dye reaches the half-way point *(bottom brush),* the manufacturer suggests replacing the brush.



f **I** G . **11-4.** Several different methods for interproximal cleaning. *Left to right,* Interdental brush, Y-shaped floss holder, disposable floss holders, and end tuft brush.

have been suggested for interproximal removal of plaque, such as interdental brushes, floss holders and floss, and end tuft brush (Fig. 11-4). According to Mauriello et al, there appears to be no substantial difference between these devices in their ability to remove plaque and their tendency to produce gingival inflammation effects when they are used properly; however, floss is the standard device to which other devices are most often compared." The other devices are more often recommended



F **I G 11-5.** Several different types of dental floss are available: thin *(top),* tape *(middle),* and meshwork *(bottom).*

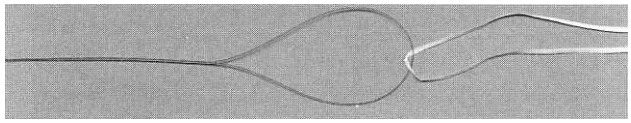
in certain unique circumstances, for example, the interdental brush may be recommended for orthodontic patients. Unfortunately, regular flossing does not occur daily in most households. Chen and Rubinson demonstrated that daily flossing was practiced by only 20% of mothers, 12% of fathers, and 6% of children within families.[13] In addition, 28% of mothers, 45% of fathers, and 48% of children never floss their teeth. This low compliance rate, particularly in children, is a problem requiring our attention as practitioners.

Several different types of floss are available: flavored and unflavored, waxed and unwaxed, and thin, tape, and meshwork* (Fig. 11-5). Almost all commercially available floss is made of nylon, although floss† made of Teflon material (polytetrafluoroethylene) is also available. The manufacturer claims that, because the material has a lower coefficient of friction than nylon, this floss does not shred, slides easily between tight contacts, and minimizes snapping of the floss.

Based on the work of Bass, unwaxed nylon-filament floss has generally been considered the floss of choice because of the ease of passing the floss between tight contacts, the lack of a wax residue, the squeaking sound effect produced by moving the floss over a clean tooth, and the fiber spread, which results in increased surface contact and greater plaque removal.[1]" However, more recent work indicates clearly that individual patient needs and preferences should be taken into account before floss selection recommendations are made. Carr et al tested four different types of floss: waxed, unwaxed, woven, and shred resistant." They studied the reductions in interproximal plaque scores for 24 dental hygiene students. Statistical analysis revealed no significant differences between floss types in cleaning efficacy, comfort of use, or the ease of use.

With these results kept in mind, it may prove beneficial in making floss recommendations to parents for their children to consider both the parent's and child's preferences and individual needs. From the perspective of patient acceptance, flavored waxed floss may be the

*Super Floss, Oral-B Laboratories, Inc., Belmont, Calif. '
Glide, W.L. Gore and Associates, Inc., Flagstaff, Ariz.



f 11. **11-6.** Floss-threading device with segment of thin floss attached.

most effective type. In addition, many parents complain that their fingers are too large for their child's mouth. Floss-holding devices (see Fig. 11-4) are an excellent alternative for parents when this complaint is voiced or when the dexterity of the parent or child prevents hand-holding of floss. For orthodontic patients, the use of Super Floss or a floss threader (Fig. 11-6) is helpful in negotiating the floss under the archwires to allow for interproximal cleaning. For orthodontic patients flossing is a tedious process but is nonetheless essential to maintenance of oral health.

POWERED MECHANICAL PLAQUE REMOVAL

The use of powered or electric toothbrushes has received considerable attention since the 1960s. The rationale for using powered brushes is that many patients remove plaque poorly because they lack adequate manual dexterity in manipulating the brush. The powered brushes should decrease the need for dexterity by automatically including some movement of the brush head. However, initial studies into the plaque removal effectiveness of powered toothbrushes failed to demonstrate greater efficacy for powered than for manual toothbrushes. Although improvement was seen initially, over time the level of cleaning achieved with power toothbrushes declined to the same level obtained using manual toothbrushes. Kerlinger refers to this as the Hawthorne effect: almost any change or experimental manipulation will induce an improvement in behavior, apparently because of a novelty effect.'" The introduction of powered toothbrushes caused an initial increase in use, and therefore plaque and gingivitis were controlled. Over time, however, the results became comparable to those with manual brushes.

Despite these previous findings, use of the latest power brushes, such as the Sonicare* or the Braun Oral-B Kids' Power Toothbrush (D10),t may prove to be more beneficial than use of other brushes. The Sonicare uses sonic technology in the form of acoustic energy to improve the plaque removal ability of traditional toothbrush bristles. The brush has an electromagnetic device that drives the bristles' motions at 261 Hz, or 31,320 brush strokes per minute. Ho and Niederman found that the Sonicare brush was significantly more effective than the manual brush in reducing the plaque index, gingival index, percentage of sites that bled on probing, pocket depth, and total gram-negative bacteria in a subgingival plaque sample." Nowak et al have demonstrated a 40% improvement in the debris index component of the Simplified Oral Hygiene Index in children aged 4 to 9 years who were using the Braun Oral-B Kids' Power Toothbrush (D10).'s Studies by Grossman and Proskin[19] and by Jongenelis and Wiedemann[20] also compared the effectiveness of electric versus manual toothbrushes when the toothbrushes were specifically designed for children. Both studies concluded that the powered toothbrushes removed significantly more plaque than the manual toothbrushes for children. Finally, Heanue et al performed a meta-analysis showing that power brushes with a rotation-oscillation action design removed more plaque and reduced gingivitis more effectively than manual brushes in both the short and the long term.[21] No other powered brush designs were consistently superior to manual toothbrushes.

The Toothbrush Acceptance Program Guidelines of the American Dental Association (ADA) Council on Scientific Affairs list several requirements for powered toothbrushes that are not imposed on manual toothbrushes.[L2] Among these are evidence that the product can be employed under unsupervised conditions by the average layperson to provide a 15% statistically significant reduction over baseline in gingivitis and a statistically significant reduction in plaque.

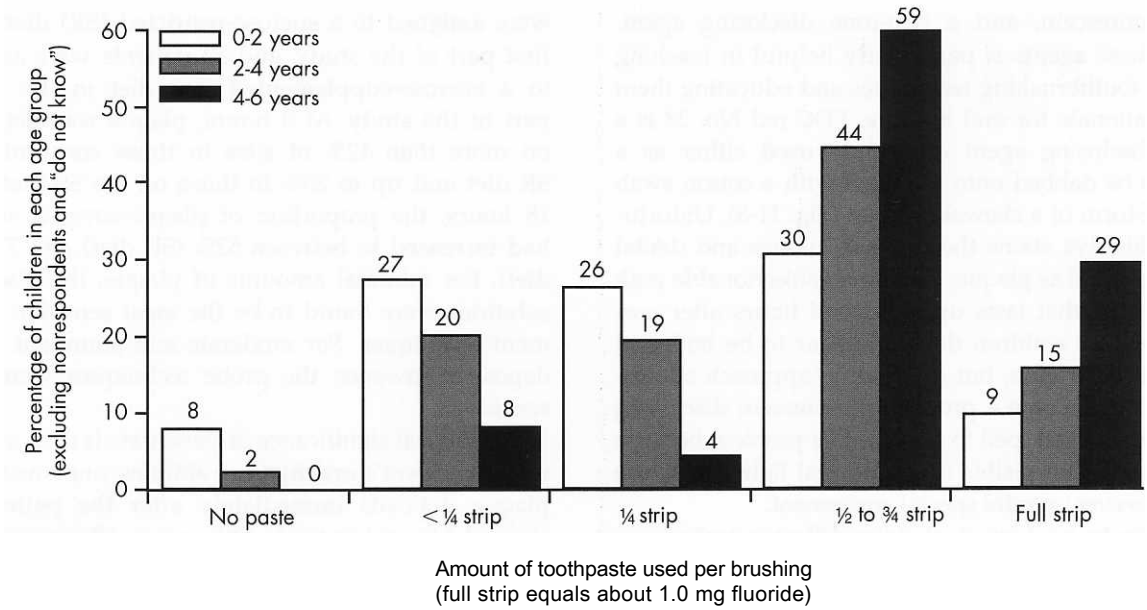
Also noteworthy is the development of the Braun Oral-B Interclean.* This electrically powered cleaning device requires only single-handed usage while its filament rotates to undergo an elliptical movement, disrupting plaque attached to adjacent and proximate teeth. Gordon, Frascella, and Reardon compared the effect of Interclean use and manual flossing over a 4-week period on level of interproximal plaque, gingivitis, and papillary bleeding in 52 healthy volunteers.- The results demonstrated no statistically significant difference between manual flossing and use of the electric interdental cleaning device. Results from the personal preference phase of the study demonstrated that significantly more subjects preferred the interdental cleaning device (69.5%) to floss (24.5%). This new type of interdental cleaning device may be particularly helpful for adolescent orthodontic patients, and the promise for its future is bright.

DENTIFRICES

Dentifrices serve multiple functions in oral hygiene through the inclusion of a variety of agents. They act as plaque- and stain-removing agents through the use of abrasives and surfactants. Pleasant flavors and colors

*Philips Oral Healthcare, Inc, Snoqualmie, Wash. '
Oral-B Laboratories, Inc., Belmont, Calif.

"Braun GmbH, Kronberg, Germany.



f I G. **11-7.** Quantity of toothpaste used by children from birth to 6 years of age. *(From Levy SM, Zarei-M Z:* J Dent Child *58(6):467-473, 1991.)*

encourage their use. They have tartar control properties because of the addition of pyrophosphates. Finally, dentifrices have anticaries and desensitization properties through the action of fluoride and other agents. A child's dentifrice should contain fluoride, rank low in abrasiveness, and carry the ADA seal of acceptance. In 2003, 36 different fluoride-containing dentifrices were listed as accepted dental therapeutic products by the Council on Scientific Affairs (http://ada.org/ ada.seal. index.asp). Many of the 36 dentifrices are specifically designed and flavored to catch the attention of children.

These formulations are useful because a child is more likely to practice oral hygiene procedures if the tools to be used are pleasing to the child. Although the caries-preventive efficacy of fluoride toothpastes in children has been well documented, the impact of dentifrices on the total fluoride intake in children must be considered. Adair, Picitelli, and McKnight-Hanes confirmed that children tend to use larger amounts of dentifrice, brush for a longer period, and rinse and expectorate less when using a children's dentifrice than when using an adult dentifrice.[2"] Levy and Zarei-M studied toothbrushing habits and quantities of toothpaste used on brushes in children from birth through 6 years. Fig. 11-7 shows their results.[25] This study did not quantify the amount of toothpaste, and therefore of fluoride, ingested from the use of this much paste on the brush. However, the investigators suggest that ingestion was likely a substantial source of systemic fluoride for these children during the years when a risk of dental fluorosis is present. It is interesting to note that many toothpaste advertisements show children with large amounts of toothpaste on their brushes. Clearly, this is not the perception dentists want the public to have when regarding the use of fluoridated toothpastes in young children.

Simard et al concluded from their study of 12- to 24-month-old children that 20% of the children ingested more than 0.25 mg of fluoride per day by toothbrushing alone.[26] They suggest the following to reduce the chance of dental fluorosis in children secondary to toothpaste ingestion. Manufacturers should market a low-fluoride dentifrice for infants or reduce the diameter of the tube orifice. Parents should be advised to delay the use of fluoride dentifrice until the child is older than 36 months and to use small, pea-sized quantities of toothpaste. Pediatricians should take into consideration all sources of fluoride before prescribing supplements. In the wake of these recommendations, one manufacturer has developed a dentifrice for children called Baby Orajel Tooth and Gum Cleanser.* The manufacturer states that it is nonabrasive, nonfoaming, without fluoride, safe for infants, and ideal for babies aged 4 months to 3 years. It contains a mild surfactant and simethicone, is sugar-free, and comes in vanilla and fruit flavors.

DISCLOSING AGENTS

In an effort to increase the patient's ability to remove plaque, several agents have been developed to allow for patient visualization of plaque. These include iodine, gentian violet, erythrosin, basic fuchsin, fast green, food

*Del Pharmaceuticals, Inc., Plainview, N.Y.

dyes, fluorescein, and a two-tone disclosing agent. Use of these agents is particularly helpful in teaching children toothbrushing techniques and educating them on the rationale for oral hygiene. FDC red No. 28 is a plaque-disclosing agent commonly used either as a liquid to be dabbed onto the teeth with a cotton swab or in the form of a chewable tablet (Fig. 11-8). Unfortunately, this dye stains the oral soft tissues and dental pellicle, as well as plaque, leaving an objectionable pink discoloration that lasts up to several hours after use. Most younger children do not appear to be bothered by the discoloration, but as children approach adolescence it can become a problem. Fluorescein disclosing agents were developed to address this problem because fluorescein is not visible under normal light. Their use does, however, require special equipment.

In a study by Lim et al, four different techniques were compared for clinically detecting plaque during different dietary regimes." Subjects ranging in age from 18 to 27 years had their plaque levels assessed using a caries probe, a plaque-detection probe, erythrosin, and a two-tone disclosing agent at 3, 6, and 18 hours after thorough cleaning of their teeth. Thirty-eight patients were assigned to a sucrose-restricted (SR) diet in the first part of the study, and 32 patients were assigned to a sucrose-supplemented (SS) diet in the second part of the study. At 3 hours, plaque was detectable on more than 12% of sites in those consuming the SR diet and up to 23% in those on the SS diet. After 18 hours, the proportion of plaque-covered surfaces had increased to between 52% (SR diet) and 73% (SS diet). For minimal amounts of plaque, the disclosing solutions were found to be the most sensitive assessment techniques. For moderate and abundant plaque deposits, however, the probe techniques were more sensitive.

The clinical significance of these data is that, in measuring a patient's oral hygiene abilities, one must assess plaque deposits immediately after the patient has cleaned his or her teeth. Otherwise, allowances must be made for factors such as the time elapsed since the teeth were cleaned and the patient's diet. If a patient is seen several hours after the teeth have been cleaned, the quality of plaque control may be deemed unsatisfactory regardless of the quality of the patient's performance. It is also interesting to note that disclosing

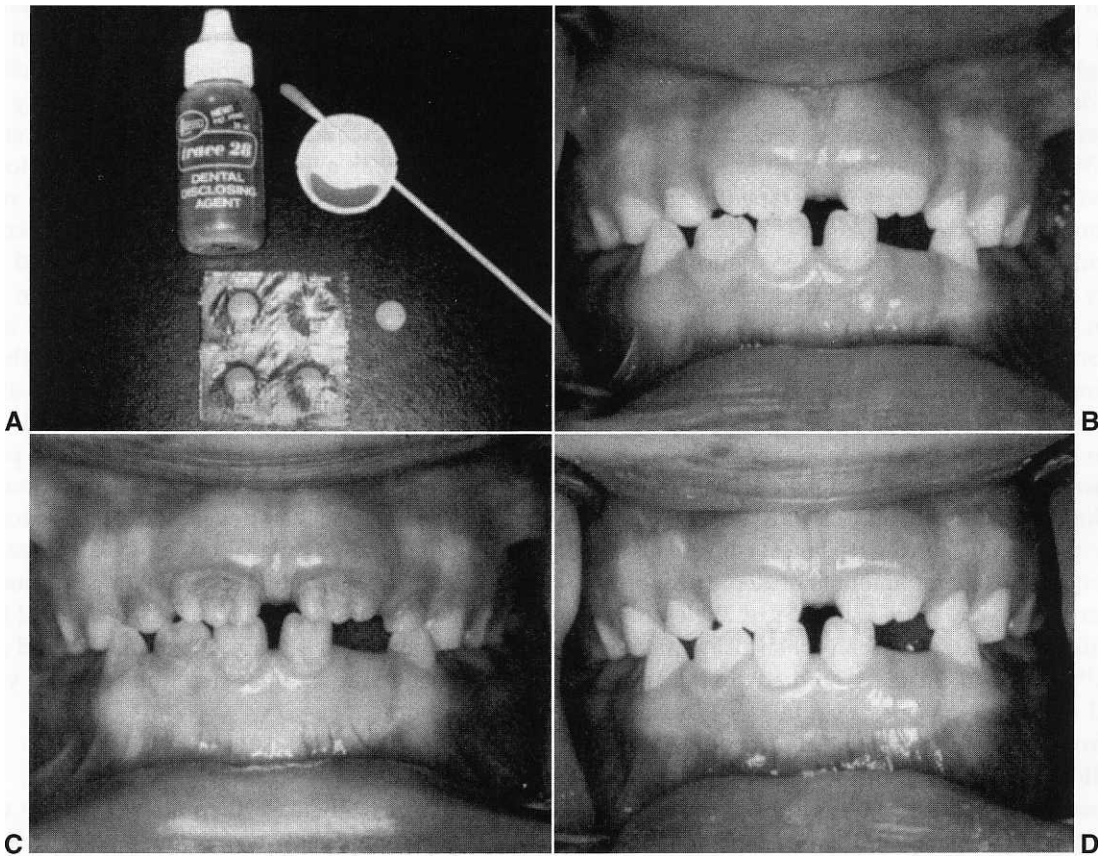


FIG 11-8. Plaque-disclosing procedure. **A,** Two common forms of FDC red No. 28 disclosing agent: a liquid that is dabbed on with a cotton swab and a chewable tablet. **B,** Mixed dentition in a patient before oral hygiene and disclosing. C, Patient before oral hygiene but after disclosing. D, Patient after oral hygiene and disclosing.

agents have some antimicrobial activity, according to Baab, Broadwell, and Williams.[1-4] Although short-term quantitative inhibition of plaque growth has not been observed clinically, long-term home use of disclosing agents may contribute to qualitative differences in plaque composition. Further studies are needed to measure the long-term in vivo effect of these agents.

OTHER ADJUNCTS FOR PLAQUE CONTROL

Several other devices, such as oral irrigators and tongue scrapers, have been suggested for routine oral hygiene. Oral irrigators use pulsed water or chemotherapeutic agents to dislodge plaque from the dentition. Tongue scrapers, which are flat, flexible plastic sticks, are used to remove bacterial and food deposits that accumulate within the rough dorsal surface of the tongue. In addition, gauze or special dental washcloths are useful in infants to massage the gums and to remove plaque on newly erupted teeth. Although these adjuncts add to our basic hygiene tools, toothbrushes and floss remain the most effective means of mechanical plaque removal. Professional recommendation of these adjuncts should be to suggest them as supplements to and not substitutes for the basic tools and should take into consideration the patient's and caregiver's individual needs, abilities, and preferences.

TECHNIQUES

As with toothbrush design, several different types of toothbrushing techniques for children have been advocated over the years. The more predominant techniques are the roll method, the Charters method, the horizontal scrubbing method, and the modified Stillman method. Anaise, in his study of the effectiveness of these four techniques in children 11 to 14 years of age, describes them as follows[1-9]:

Roll Method. The brush is placed in the vestibule, the bristle ends directed apically, with the sides of the bristles touching the gingival tissue. The patient exerts lateral pressure with the sides of the bristles, and the brush is moved occlusally. The brush is placed again high in the vestibule, and the rolling motion is repeated. The lingual surfaces are brushed in the same manner, with two teeth brushed simultaneously.

Charters Method. The ends of the bristles are placed in contact with the enamel of the teeth and the gingiva, with the bristles pointed at about a 45-degree angle toward the plane of occlusion. A lateral and downward pressure is then placed on the brush, and the brush is vibrated gently back and forth a millimeter or so.

Horizontal Scrubbing Method. The brush is placed horizontally on buccal and lingual surfaces and moved back and forth with a scrubbing motion.

Modified Stillman Method. The modified Stillman method combines a vibratory action of the bristles with a stroke movement of the brush in the long axis of the teeth. The brush is placed at the mucogingival line, with the bristles pointed away from the crown, and moved with a stroking motion along the gingiva and the tooth surface. The handle is rotated toward the crown and vibrated as the brush is moved.

Anaise concluded that the horizontal scrubbing method exhibited a more significant plaque-removing effect than the roll, Charters, and modified Stillman methods.[5-9] This finding supports the work done by McClure[1] and by Sangnes, Zachrisson, and Gjermo.[9]

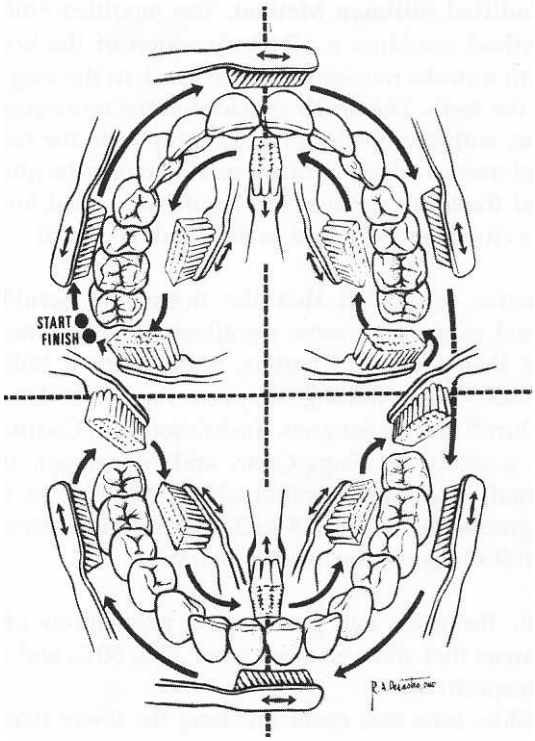
In a study by Rugg-Gunn and Macgregor, uninstructed videotaped toothbrushing behavior in three age groups (5, 11, and 18 to 22 years) was analyzed.[32] The following conclusions were drawn:

- In the three age groups, the proportions of the areas that were brushed were 25%, 50%, and 67%, respectively.
- More time was spent brushing the lower than the upper teeth.
- The contralateral side (the left side in right-handed persons) was brushed more than the ipsilateral side in children, but equally in adults.
- Less than 10% of the time was spent brushing lingual areas.
- The most popular brushing strokes were the horizontal stroke in children and the vertical stroke in adults; the roll and circular strokes were seldom used.
- Eighty-four percent of subjects used more than one type of stroke.

The horizontal scrub technique removes as much or more plaque than the other techniques, regardless of how old the child is and whether the brushing is performed by the parent or the child. In addition, it is the technique most naturally adopted by children. Therefore, in most situations the horizontal scrubbing method can be recommended for brushing children's teeth. Regardless of the brushing method used, a systematic approach to brushing as advocated by Starkey should be employed.[1] By following a systematic approach, as shown in Fig. 11-9, the child or parent can help ensure that all areas of the mouth are cleaned. Notice also on this figure the positioning of the brush head on the lingual surfaces of the anterior teeth and on the distal aspect of the most posterior tooth in each quadrant.

For flossing, the following technique is recommended (Fig. 11-10):

1. A 46- to 61-cm (18- to 24-inch) length of floss is obtained, and the ends are wrapped around the



[**I C 11-9.** Systematic approach to brushing the teeth begins with the buccal aspects of the teeth in the maxillary right quadrant and follows the arrows. Bristles are held at a 45-degree angle to the long axis of the teeth and are directed to the gum line. Short back-and-forth strokes are used, allowing bristles to remain in the same place. The handle of the brush is placed parallel to the biting surfaces except when brushing the lingual aspects of the anterior teeth and the posterior aspects of the last tooth in each quadrant, when a heel-toe direction of brushing is used. *(Courtesy Dr. Paul Starkey.)*

patient's or parent's middle fingers. Floss should be long enough to allow the thumbs to touch each other when the hands are laid flat.

2. The thumbs and index fingers are used to guide the floss as it is gently sawed between the two teeth to be cleaned. Care must be taken not to snap the floss down through the interproximal contacts to avoid gingival trauma.
3. The floss is then manipulated into a C shape around each tooth individually and moved in a cervical-occlusal reciprocating motion until the plaque is removed. In between cleaning each pair of teeth the floss is repositioned on the fingers so that fresh, unsoiled floss is used at each new location.

Learning a flossing technique is difficult and takes some practice. Some children and their parents prefer to make a loop of floss. Tying the two ends of the floss together, instead of wrapping it around their fingers, assists them in holding and controlling the floss. However, Rodrigues et al demonstrated that, even when the looped floss technique is used, a training program is required for children $6^1/2$ to $7^1/2$ years of age to accomplish significant reduction in proximal surface dental plaque indices.[34]

VISUAL-MOTOR SKILL MASTERY

Several attempts have been made to develop specific recommendations for when children can begin performing oral hygiene procedures themselves with adequate effectiveness. Terhune stated that the variables of age, gender, and eye-hand coordination could not precisely predict when particular children were ready to learn an effective dental flossing technique.[3'] However, all 8- to 11-year-old children in his study learned how to use dental floss effectively within 10 days. Mescher, Brine, and Biller found that hand function was an age-related factor in children's ability to perform sulcular toothbrushing, but that hand function test scores were not accurate predictors of an individual's toothbrushing ability.[3"]

Preisch, however, did find a significant relationship between developmental age and oral hygiene scores using a visual-motor integration developmental test.[37] With the use of this test, significant correlations have been shown between the ability of children to copy geometric forms and their academic achievement and motor skill level. Higher levels of thinking and behavior require integration among sensory inputs and motor action. A child can have well-developed visual and motor skills but may be unable to coordinate the two. Although both chronologic and developmental ages were found to be predictors of plaque removal ability, only developmental age demonstrated statistically significant predictive power. Unfortunately, because of the complexity of this test, we are left with-out a practical method for making recommendations to parents as to when their child can begin brushing on their own. As Preisch laments, many dentists use anecdotal accounts and tell parents to supervise their children's brushing until the children can color within the lines, tie their own shoelaces, or cut through a tough piece of meat.[37] However, this may still be our best practical recommendation.

TIME CONSIDERATIONS

Another of the important questions regarding home oral health care involves time considerations in oral hygiene practices. How often should patients brush and floss their teeth and for how long? In discussing frequency of oral hygiene procedures, Loe suggests that oral cleanliness should be regarded as a defined state in which all surfaces of all teeth are plaque free.[3b] He states that it may not be surprising to find that complete removal of plaque once daily or every second day, or

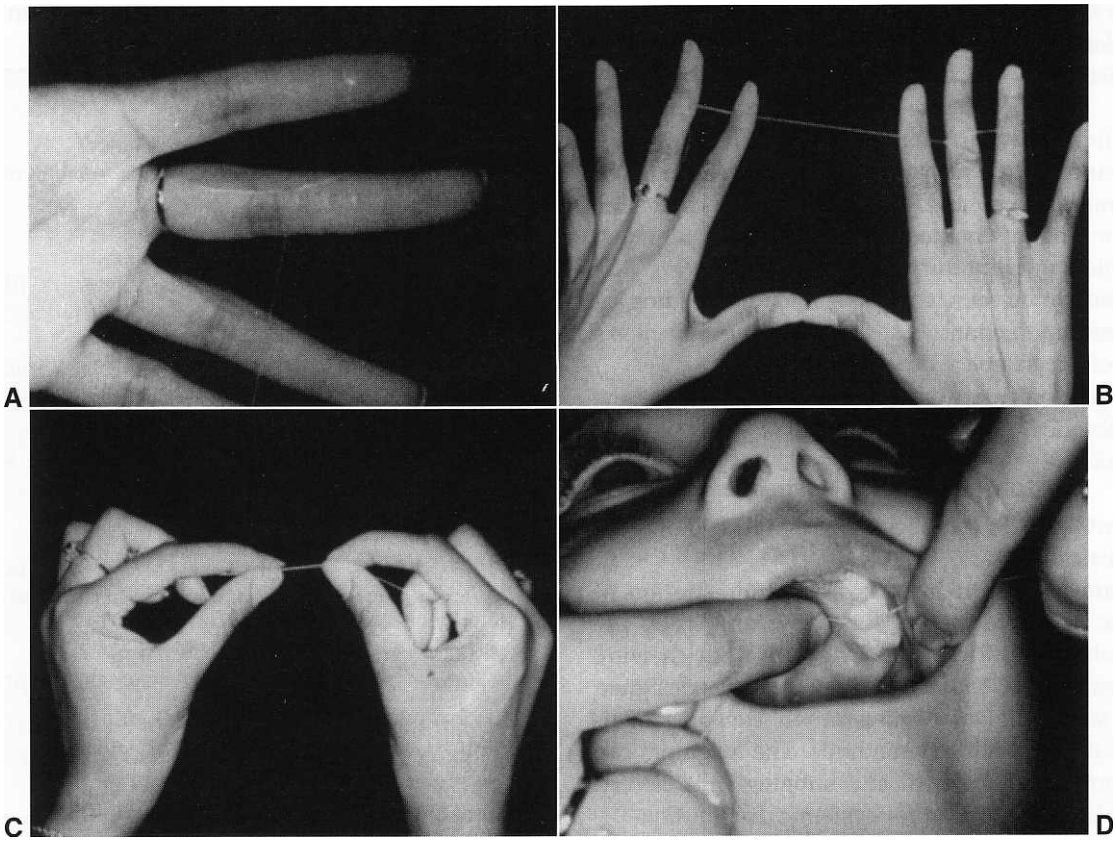


FIG **11-10**. Flossing technique. A, The length of floss is wrapped around the middle fingers of each hand. **B,** Enough floss should be left between the middle fingers to allow the thumbs to touch when the hands are laid flat. C, The index fingers and thumbs are used to manipulate the floss. D, The floss is carefully placed in a C shape between the interproximal contacts and gently sawed up and down until each tooth surface is clean.

possibly even once every third day, is more valuable in preventing dental disease than performing two or three inadequate brushings per day. Indeed, Lang, Cumming, and Loe observed that completion of effective oral hygiene procedures at intervals of up to 48 hours is compatible with gingival health." Studies addressing the relation between the frequency of hygiene procedures and caries experience in children have yielded inconclusive results.

In addition to optimal brushing frequency, the most efficacious length of brushing time has been investigated. In a study by Hodges, Bianco, and Cancro, 84 children aged 5 to 15 years brushed their teeth with a fluoridated dentifrice for 30, 60, 120, and 180 seconds."[0] The results of the study suggest that, statistically, a 1-minute brushing period provides the greatest plaque removal benefit of all time periods tested.

The following recommendations are made based on the preceding information. In children, thorough oral hygiene procedures should be performed at least once daily, preferably twice, with parental super-vision. Teeth should be brushed for at least 1 minute with a fluoridated dentifrice; flossing and other plaque removal activities are added to this time. If oral hygiene is accomplished only once per day, it should be the last thing the child does before bedtime at night. Because the flow of saliva and its buffering capacity are reduced during sleep, it is advantageous to remove plaque before bedtime. In addition, the development in children of a learned behavior per-formed at a specific time of day, each and every day, will prove beneficial throughout childhood and into adulthood.

CHEMOTHERAPEUTIC PLAQUE CONTROL

Although the use of mechanical therapy for plaque control can provide excellent results, it is clear that many patients are unable, unwilling, or untrained to practice routine effective mechanotherapy. In addition, certain patients with dental diseases (e.g., periodontitis) or medical diseases (e.g., immunocompromised conditions) require additional assistance beyond mechanotherapy to maintain a normal state of oral health. Because of this, chemotherapeutic agents have been developed as adjuncts in plaque control.

Van der Ouderaa has stated that the ideal chemotherapeutic plaque control agent should have the following characteristics':

- Specificity only for the pathogenic bacteria
- Substantivity, the ability to attach to and be retained by oral surfaces and then be released over time without loss of potency
- Chemical stability during storage
- Absence of adverse reactions, such as staining or mucosal interactions
- Toxicologic safety
- Ecologic safety so as not to adversely alter the microbiotic flora
- Ease of use

No agent has yet been developed that has all of these characteristics.

There are several main routes of administration of antiplaque agents designed for home use. They are mouthwashes, dentifrices, gels, irrigators, floss, chewing gum, lozenges, and capsules. All of these are designed for local, supragingival administration, except the irrigator and capsule delivery methods. The irrigators can provide both supragingival and subgingival delivery. The capsules are designed for systemic distribution.

Both van der Ouderaa[4'] and Mandel[42] have provided excellent reviews of the various chemotherapeutic agents and their uses. Box 11-1 is adapted from those reviews. Space does not allow a complete discussion of the agents listed in this box; however, a few pertinent subjects are addressed.

BOX 11-1 Chemotherapeutic Plaque Control Agents

ANTISEPTIC AGENTS

Positively Charged Organic Molecules:

Quaternary ammonium compounds—cetylpyridinium chloride

Pyrimidines—hexedine

Bis-biguanides—chlorhexidine, alexidine

Noncharged Phenolic Agents: Listerine (thymol, eucalyptol, menthol, and methylsalicylate), triclosan, phenol, and thymol

Oxygenating Agents: Peroxides and perborate

Bis-Pyridines: Octenidine

Halogens: Iodine, iodophors, and fluorides

Heavy Metal Salts: Silver, mercury, zinc, copper, and tin

ANTIBIOTICS

Niddamycin, kanamycin sulfate, tetracycline hydrochloride, and vancomycin hydrochloride

ENZYMES

Mucinases, pancreatin, fungal enzymes, and protease

PLAQUE-MODIFYING AGENTS

Urea peroxide

SUGAR SUBSTITUTES

Xylitol, mannitol

PLAQUE ATTACHMENT INTERFERENCE AGENTS

Sodium polyvinylphosphonic acid, perfluoroalkyl

ANTISEPTIC AGENTS

The antiseptic agents used in chemotherapeutic plaque control have been shown to exhibit little or no oral or systemic toxicity in the concentrations used. Virtually no drug resistance is induced, and in most instances these agents have a broad antimicrobial spectrum. Chlorhexidine, a positively charged organic antiseptic agent, has received considerable attention and study because of its ability to reduce plaque and gingivitis scores. It has strong substantivity, binding well to many sites in the oral cavity and maintaining an ongoing antibacterial presence. Chlorhexidine binds with anionic glycoproteins and phosphoproteins on the buccal, palatal, and labial mucosa and the tooth-borne pellicle. Its antibacterial effects include binding well to bacterial cell membranes, increasing their permeability, initiating leakage, and precipitating intracellular components.

Grossman et al conducted a 6-month clinical trial in which 430 adults rinsed twice daily with either 0.12% chlorhexidine or a placebo.[43] Gingivitis and plaque scores were lower in the chlorhexidine group (34% to 41%) than in the placebo group (61%). Several studies have demonstrated the use and efficacy of chlorhexidine therapy in children as young as 8 years of age. Studies have examined its use in the form of a rinse, a spray, a varnish, and a chlorhexidine gel used in flossing.

Lang et al investigated the effects of supervised rinsing with chlorhexidine in 158 schoolchildren, aged 10 to 12 years.[`'] The children were divided into four groups. Group A rinsed with a 0.2% solution of chlorhex idine digluconate (CHX) six times weekly. Group B rinsed with 0.2% CHX two times weekly. Group C rinsed with a 0.1% CHX solution six times weekly. Group D rinsed six times weekly with a placebo solution. All rinsing was performed under supervision, and no effort was made to change the children's oral hygiene habits. Fig. 11-11 shows the results of the study. All three experimental groups, A, B, and C, exhibited statistically significant reductions in the gingival index compared with the control group, Group D. The investigators concluded that gingivitis can be controlled successfully in children by regular rinsing with a chlorhexidine solution over an extended period.

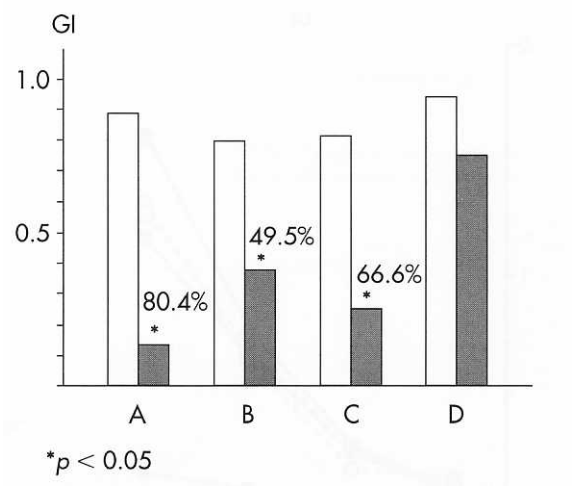


Fig. 11-11. Mean gingival index (GI) in four groups of schoolchildren rinsing with chlorhexidine digluconate (CHX) or placebo solution for 6 months under supervision. Clear bars, Before treatment; *screened* bars, after treatment. Group A: 0.2% CHX 6 times weekly; Group B: 0.2% CHX 2 times weekly; Group C: 0.1% CHX 6 times weekly; Group D: Placebo 6 times weekly. (From *Lang NP* et *al:* J Periodontal Res 17:101-111, 1982.)

Choorhexidine spray has stimulated interest regarding its use in disabled populations because of its effectiveness and ease of administration. Burtner et al demonstrated a 35% reduction in plaque levels with use of the spray compared with placebo use in a study of 16 institutionalized adult males with severe and profound mental retardation.[45] Chikte et al conducted a 9-week, double-blind, randomized crossover clinical trial involving 52 institutionalized mentally disabled individuals 10 to 26 years of age. By the end of the trial, plaque and gingival indices had been reduced by 48% and 52%, respectively, in the group treated with a stannous fluoride spray.'" In the group treated with chlorhexidine spray, reductions in plaque and gingival indices were 75% and 78%, respectively.

In addition to its use in institutionalized patients with mental retardation, chlorhexidine has been studied for its use in immunocompromised patients. Ferretti et al found that the prophylactic use of chlorhexidine mouthrinse produced reductions in gingivitis and mucositis and oral microbial burden in patients undergoing bone marrow transplantation." Raether et al, however, studying a similar patient population, found no significant difference between the group receiving chlorhexidine and that given a placebo in terms of number of oral ulcerations, development of bacteremia, and length of hospital stay.[18] Despite these findings, they suggested the use of a chlorhexidine mouthrinse as an antiplaque and antigingivitis agent in bone marrow transplant patients to augment their oral hygiene. Finally, chlorhexidine varnish has been shown by Fennis-le et and by Petersson et a1[50] to suppress the level of mutans streptococci. However, recent clinical studies have failed to demonstrate a transfer of this effect into a decrease in caries on either smooth enamel surfaces or occlusal pits and fissures. Additional work is needed to conclusively demonstrate the value of chlorhexidine varnishes in reducing dental caries.

The use of positively charged antiplaque agents has been hampered by adverse reactions such as staining of teeth, impaired taste sensation, and increased supragingival calculus formation. Different attempts have been made to decrease these side effects, such as alteration of dietary habits, increase in mechanical plaque removal efforts, and use of hydrogen peroxide solutions in conjunction with the antiseptic agent. Continued research is needed to find methods to limit these adverse reactions.

The most widely known noncharged phenolic antiseptic agent is Listerine.* It has demonstrated a long history of efficacy and was among the original anti-septic agents studied by W.D. Miller in 1890.[5] In addition, it was the first over-the-counter mouthrinse to be accepted by the Council of Dental Therapeutics for its help in controlling plaque and gingivitis."- Despite its long history of use, studies by Clark et a1[5'] and by Brownstone et a1[52] have shown chlorhexidine to be significantly more effective than Listerine in reducing plaque and gingivitis indices. Listerine tends to give patients a burning sensation, and it has a bitter taste. Lang and Brecx have summarized the changes in plaque index, gingival index, and discoloration index scores resulting from the use of four well-known chemotherapeutic plaque control agents (Fig. 11-12)." The effects of two daily 10-mL rinses with either 0.12% chlorhexidine digluconate, the quaternary ammonium compound cetylpyridinium chloride, the phenolic compound Listerine, or the plant alkaloid sanguinarine were compared with those of rinses with a placebo. All rinses were supervised by registered dental hygienists during these 21-day studies. The subjects were divided into five groups of eight individuals each and were instructed to refrain from oral hygiene during the 21 days. Although the sanguinarine, Listerine, and cetylpyridinium chloride inhibited plaque formation to some extent, they did not prevent gingivitis significantly more than the placebo. The chlorhexidine, how-ever, maintained the preexperimental gingival index scores throughout the 21 days. Unfortunately, all of the antiseptics demonstrated higher discoloration index scores than the placebo. As can be seen in Fig. 11-12, C, chlorhexidine had the second highest discoloration score of the four agents.

*Pfizer Warner Lambert Division, Morris Plains, N.J.

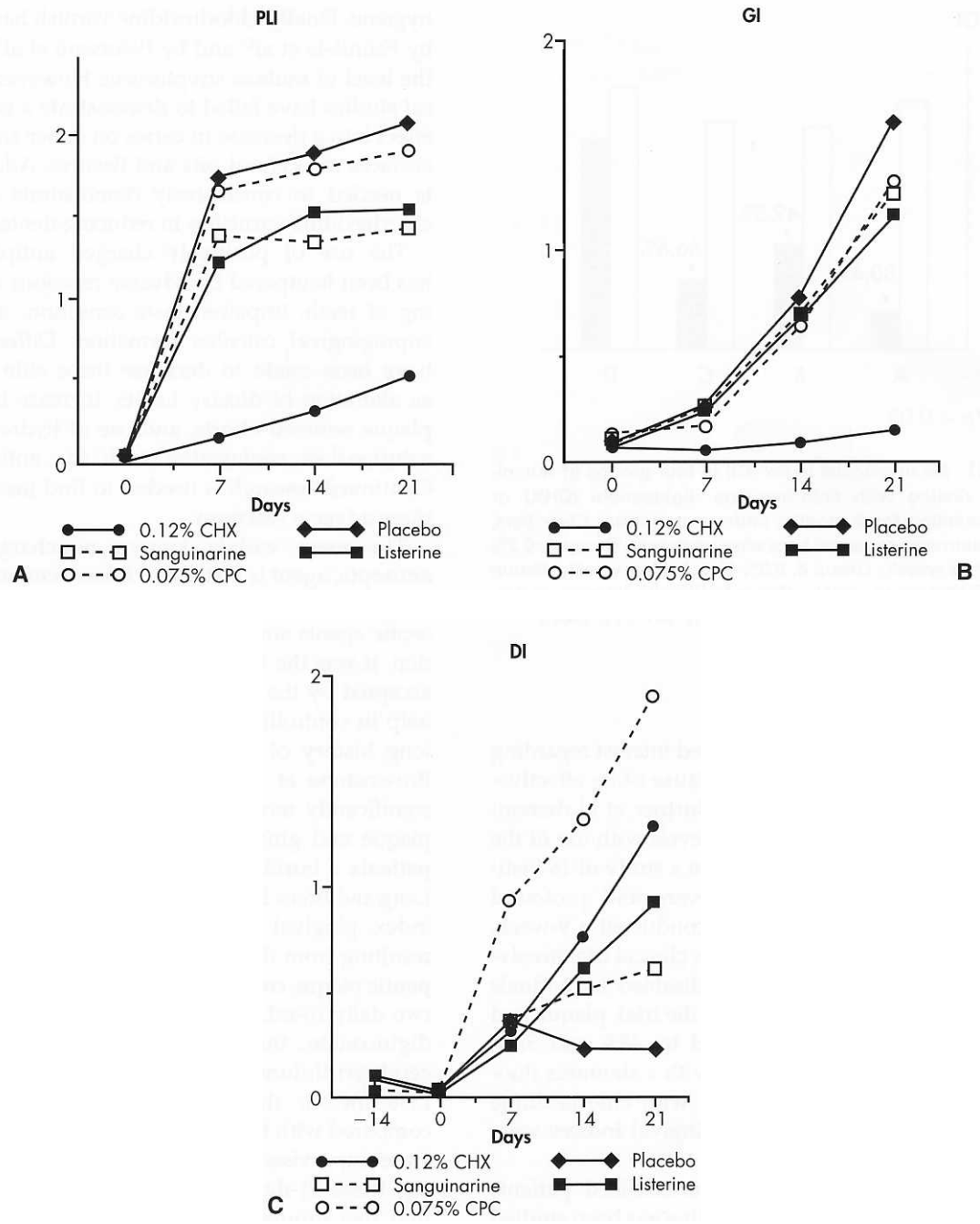


FIG. 11-12. Mean indices in five groups of eight individuals refraining from oral hygiene for 21 days rinsing with either 0.12% chlorhexidine digluconate (CHX), 0.075% cetylpyridinium chloride (CPC), Listerine, sanguinarine, or placebo. A, Mean plaque index (PLI). **B,** Mean gingival index (GI). C, Mean discoloration index (DI). *(From Lang NP, Brecx MC: J* Periodontal Res *16 (21 suppl):74-89, 1986.)*

Listerine has one of the highest alcohol contents of any mouthwash, approximately 25%. The alcohol content of some mouthwashes has been the cause of some concern. Although the development of oral and pharyngeal cancer with long-term mouthwash use has been investigated, alcohol intoxication is more relevant to pediatric dentistry. In addition, the relation-ship of alcohol-containing mouthwashes to oral carcinomas is equivocal. Alcohol intoxication of children and adolescents from mouthwashes is a concern because of the products' availability. Most parents do not recognize the potential harm from these rinses. Selbst, DeMaio, and Boenning reported the case of a 4-year-old boy who died secondary to consuming approximately 12 oz of a 10% alcohol mouthwash.'' They urge for stronger legislation that would restrict

the level of alcohol in substances that might be avail-able to children and for continued education of practitioners and parents regarding the potential lethality of most mouthwashes so that accidental ingestions are prevented. One consumer advocacy group states that it is inconsistent for cough and cold products with 12% alcohol to have child-resistant tops whereas some mouthwashes with an even higher alcohol content have designer shot-glass tops. The ADA Council on Dental Therapeutics requires any mouthrinses that carry the ADA seal of acceptance and contain more than 5% ethyl alcohol to be packaged in bottles with child-resistant caps.

A few comments regarding the use of fluoride as a halogen antiseptic plaque control agent are appropriate here, although its use in dentistry is discussed in other portions of the text. The fluoride ion inhibits carbohydrate utilization of oral organisms by blocking enzymes involved in the glycolytic pathway; however, at preventive-use levels it probably does not alter the plaque ecosystem. As mentioned earlier, stannous fluoride can produce reductions in plaque and gingivitis scores approaching those of chlorhexidine, but this effect is caused by the tin content of this salt, not the fluoride content. It is interesting to note that two anti-septic agents, chlorhexidine and triclosan, have been incorporated into dentifrice formulations.

ENZYMES, PLAGUE-MODIFYING AGENTS, AND PLAGUE ATTACHMENT INTERFERENCE AGENTS

Enzyme systems intended to alter plaque architecture and adherence, as well as enzymes designed to generate antibacterial products, have been investigated. However, problems associated with the long-term stability of enzyme molecules in environments with potentially high concentrations of alcohol or surfactants have yet to be addressed. The use of urea peroxide as a plaque-modifying agent has been investigated because of its increased stability over hydrogen per-oxide and the protein denaturation effect of urea. Unfortunately, limited success has been demonstrated. The use of agents designed to interfere with the initial adherence of bacteria to the salivary pellicle or the subsequent accumulation by growth and interbacterial adherence seems encouraging. Delmopinol, derived from orpholinoethanol, exerts its affects by binding to salivary proteins and altering the cohesiveness and adhesiveness properties of the films formed.

Although these areas may hold promise for future chemotherapeutic control of plaque, additional research is needed.

SUGAR SUBSTITUTES

The use of sugar substitutes such as xylitol, mannitol, sucralose, and aspartame has been advocated. Although Park et al have shown that sugar substitutes can have a positive influence on plaque pH, the intrinsic antiplaque activity is much lower than that of other plaque control agents." These agents have been suggested for use in chewing gum to decrease plaque accumulation and pH. Advocating the use of chewing gum as a preventive technique is not without controversy, however. Hoerman et al demonstrated that in a no-oral-hygiene environment plaque accumulation was lower when gum with sucrose or sorbitol was chewed than when gum was not chewed.[i6] In addition, Isokangas et al carried out a 2-year study of 11- and 12-year-old children with moderate and decreasing caries prevalence." They demonstrated that the combination of xylitol gum chewing and fluoride usage resulted in a significantly lower incidence of caries than fluoride usage alone. Continued research into the use of sugar substitutes as plaque control agents is needed.

In closing this section, one final comment is in order. Because of conflicting results published on the effectiveness of the commercially available prebrushing rinse containing sodium benzoate, it is not included in the list of chemotherapeutic plaque control agents. In addition, it is not accepted by the ADA. O'Mullane suggests that the positive results found for this pre-brushing rinse may stem purely from the advantage of rinsing with water before brushing.[$] Indeed, an in vitro study by Jakober and Perritt supports this idea.[59] They demonstrated that brushing with hot water (40° to 43° C) was more efficient at removing simulated plaque on ceramic tiles than brushing alone or brushing with toothpaste. They also showed that flowing hot water was more effective at removing the simulated plaque than flowing cold water (30° to 35° C). The idea of using water to help remove plaque is not new. The "swish-and-swallow" method of removing material from the mouth immediately after eating in circum-stances where brushing is impractical has been advocated for a long time. Ciancio recommends that, when a product is selected for a patient, consideration be given to necessity, efficacy, adverse effects, and cost effectiveness.'i[0]

AGE-SPECIFIC HOME ORAL HYGIENE INSTRUCTIONS

The appropriateness and effectiveness of home oral hygiene procedures change throughout childhood. Specific age-related home oral hygiene recommendations are described in the following sections. It is necessary to involve the parent at some level of the oral hygiene procedure for each of the age categories.

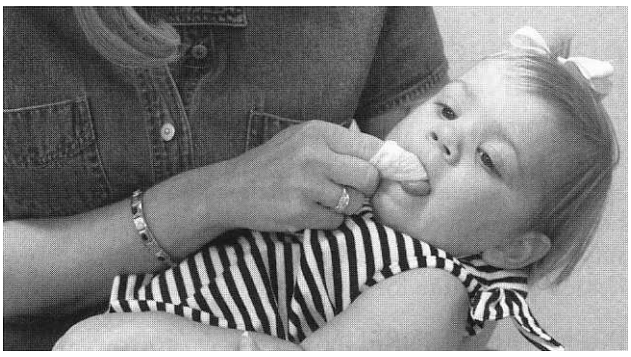
PRENATAL COUNSELING

The best time to begin counseling parents and establishing a child's dental preventive program is actually before the birth of the child. It is beneficial to begin at this time for numerous reasons. For an expectant

couple, particularly if the child is their first, this is a time in their lives when they are most receptive to preventive health recommendations. These parents-tobe become acutely aware of their child's dependence on them for all of the child's nurturing and health care needs. Parents have a strong instinct to provide the best that they can for the child. Counseling them on their own hygiene habits and the effect they can have on their children as role models will aid in improving both the parents' and child's oral health. Discussing pregnancy gingivitis with the mother-to-be and dispelling some of the myths about childbirth and dental health can prove beneficial. In addition, a review of infant dental care (see next section) is useful for the expectant parents.

INFANTS (0 TO 1 YEAR OLD)

It is important that a few basic home oral hygiene procedures for the child begin during the first year of life. There is general agreement that plaque removal activities should begin on eruption of the first primary teeth. Some practitioners recommend cleaning and massaging of the gums before this to help in establishing a healthy oral flora and to aid in teething. This early cleaning must be done totally by the parent. It can be accomplished by wrapping a moistened gauze square or washcloth around the finger and gently massaging the teeth and gingival tissues. The child can be positioned in numerous ways during this procedure, but cradling the child with one arm while massaging the teeth with the hand of the other may be the simplest and provides the infant with a strong sense of security (Fig. 11-13). This procedure should be performed once daily. Generally, other plaque removal techniques are not necessary. The introduction of a moistened, soft-bristled, child- or infant-sized toothbrush during this age is advisable only if the parent feels comfortable using the brush. The use of a dentifrice is neither necessary nor advised as the foaming action of the paste tends to be objectionable



f r **11-13.** Arm-cradled position of child for effective cleansing of the oral cavity. This figure shows the use of a gauze square for wiping the child's dentition and gingival tissues.

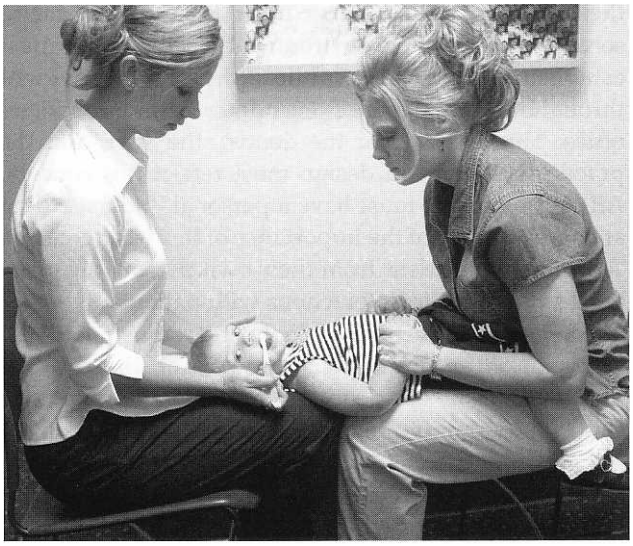
to the infant. Because fluoride ingestion is possible, however, as mentioned earlier, use of a nonfluoridated tooth and gum cleanser may prove beneficial.

The child's first visit to the dentist should take place during this period. The American Academy of Pediatric Dentistry recommends that children have their first dental visit at approximately the time of eruption of the first tooth or, at the latest, by the age of 12 months." When the child has special dental needs, such as medical problems or trauma, this visit can be sooner. Several objectives are accomplished at this visit. Certainly, instruction of the parents in the use of the oral hygiene practices just mentioned is necessary. In addition, an infant dental examination and fluoride status review should be accomplished, and dietary issues related to nursing and bottle caries as well as other health concerns are addressed. These subjects are discussed in more detail in other sections of this text. These first dental visits are also a time for the child to become familiar with the dental environment and the dental staff and the dentist, which makes any future dental treatment less anxiety provoking.

TODDLERS (1 TO 3 YEARS OLD)

During toddlerhood, the toothbrush should be introduced into the plaque removal procedure if this was not accomplished previously. Because of the inability of children in this age group to expectorate and the potential for fluoride ingestion, only a nonfluoridated dentifrice should be used. Most children enjoy imitating their parents and will readily practice toothbrushing. Unfortunately, adequate plaque removal is not usually accomplished by the child alone. Although the child should be encouraged to begin rudimentary brushing, the parent remains the primary caregiver in these hygiene procedures. The use of additional instruments for plaque control is generally unnecessary; however, flossing may be needed if any interproximal contacts are closed. The use of a flossing aid may also be indicated.

Positioning of the child and parent is again important. Although most children enjoy brushing their own teeth, many are resistant to allowing anyone else to do the brushing. Several positions can be used by the parent, but the lap-to-lap position, as shown in Fig. 11-14, allows one adult to control the child's body movements while the other adult brushes the teeth. Notice how the child's arms and legs are controlled with the hands and elbows of the adult responsible for body movements. The parents should be encouraged to make this a special time for the child and to praise the child as much as possible. For single-parent households, a one-adult position often becomes necessary. In this situation the parent sits on the floor with his or her legs stretched out in front and the child is positioned between the legs. The child's head is placed between the thighs of the parent, with the



f I **G 11-14.** Lap-to-lap position of child. Two adults sit with knees touching, using their laps as a table on which to rest the child. The adult on the right holds the child's legs and arms while the adult on the left performs the oral hygiene procedures.

child's arms and legs carefully controlled by the legs of the parent. This position is a little awkward, but for a young child resistant to oral hygiene, it does allow these procedures to be accomplished.

PRESCHOOLERS (3 TO 6 YEARS OLD)

Although children in the preschool age range begin to demonstrate significant improvements in their ability to manipulate the toothbrush, it is still the responsibility of the parent to be the primary provider of oral hygiene procedures. All too often, parents of these children feel that the child has adequately achieved the skills necessary to clean the teeth. It is important to stress to the parents that they must continue to brush their child's teeth. A fluoride dentifrice can be introduced at 3 years of age. Although fluoride ingestion remains a concern for this age group, during this time, most children develop the skills to expectorate toothpaste adequately. Until this occurs, it is important for parents to continue to use only a pea-sized amount of toothpaste on the child's brush. In addition, it is during this age that flossing is most likely to begin. As mentioned previously, if the interproximal contacts are closed, the parent must begin flossing procedures. In the primary dentition, the posterior contacts may be the only areas where flossing is needed. The closure of the spaces between the primary molars tends to occur somewhere near the start of this age range. If any interproximal area has tooth-to-tooth contact, however, daily flossing of that area becomes necessary.

Proper positioning of the child continues to be useful for this age group in performing oral hygiene. One method advocated is that in which the parent stands behind the child and both face the same direction. The child rests his or her head back into the parent's nondominant arm. With the hand of this arm the cheeks can be retracted, and the other hand is used to brush. This position is also appropriate for flossing. To brush their child's teeth many parents use a frontal approach, which is awkward and provides little head support. This positioning technique should be discouraged.

It is also during this stage that fluoride gels and rinses for home use may be introduced. Because of the risk of ingestion, however, these agents should be employed in small quantities and their use should be limited to those patients demonstrating a moderate to high risk of caries. The use of other chemotherapeutic plaque control agents is generally not recommended.

SCHOOL-AGED CHILDREN (6 TO 12 YEARS OLD)

The 6- to 12-year stage is marked by acceptance of increasing responsibilities by the children. The need to assume responsibility for homework and household chores tends to occur during this time. In addition, the child can begin to assume more responsibility for oral hygiene. Parental involvement is still needed. However, instead of performing the oral hygiene, parents can switch to active supervision. By the second half of this stage, most children can provide their basic oral hygiene (brushing and flossing). Parents may find they only need to brush or floss their child's teeth in certain difficult-to-reach areas of the mouth or if there is a compliance problem. Parents do need to actively inspect their child's teeth for cleanliness on a regular basis. One helpful adjunct is the use of a disclosing agent. After the child has brushed, flossed, and used the disclosing agent on his or her teeth, the parent can easily visualize any remaining plaque and assist the child in removing it.

By this age, ingestion of fluoridated materials, such as dentifrices, gels, or rinses, is not as pronounced a concern because the children can now expectorate well. Certainly the use of fluoridated dentifrices is essential; however, fluoridated gels and rinses can be reserved for those children at risk for caries. In addition, the use of chlorhexidine or Listerine can be introduced to those at risk for periodontal disease and caries. Unfortunately, some children who might benefit from these chemotherapeutic agents will find their use objectionable.

Because early treatment of malocclusions has increased, this age group has undergone more of this treatment and experienced its accompanying increased risk for caries and periodontal disease. Special attention to oral hygiene is necessary for these patients. Increased frequency and adequacy of brushing and flossing becomes necessary. Although fluoridated dentifrices provide cost-efficient fluoride exposure, the use of fluoridated gels or rinses is strongly encouraged. In addition,

as with other patients at risk for caries and periodontal disease, the use of chemotherapeutic agents and adjuncts such as oral irrigators is recommended. Feil et al published an interesting study on the intentional use if the Hawthorne effect to improve oral hygiene compliance in orthodontic patients.[62] Forty orthodontic adolescent patients with histories of poor oral hygiene were assigned to one of two groups. The experimental subjects were presented with a situation that stimulated participation in an experiment, whereas the control subjects had no knowledge of study participation. Although there were no statistically significant differences between the control and the experimental group at baseline, the experimental group showed significantly lower plaque scores at 3 months and again at 6 months. The experimental subjects had significantly improved oral hygiene, which suggests that the Hawthorne effect (participating in an " experimental study") caused the adolescent patients to pay more attention to oral hygiene and therefore to do a better job.

ADOLESCENTS (12 TO 19 YEARS OLD)

Although the adolescent patient usually has developed the skills for adequate oral hygiene procedures, compliance is a major problem during this age period. Griffin and Goepferd point out that motivating an adolescent to assume responsibility for personal oral hygiene may be complicated by reactions of rebellion against external authority and some incapacity to appreciate long-term consequences.[63] Macgregor and Balding's survey of 4075 children 14 years old suggests a positive relationship between self-esteem and toothbrushing behavior and motivation for mouth care in adolescents.[6"] Because self-esteem declines between the ages of 11 and 14 and then shows a gradual improvement into adulthood, it is not hard to understand why plaque control in these patients declines. In addition, poor dietary habits and pubertal hormonal changes increase adolescents' risk for caries and gingival inflammation.

Therefore, it is important for practitioners and parents to continually help and guide adolescents as they progress through this difficult stage. Stressing their increased responsibility as young adults without appearing authoritarian can aid them in accepting their new role. The parents must be ready to adapt to their child's changing personality and to continue to rein-force the need for oral health care and hygiene. Increasing adolescents' knowledge regarding plaque control and oral diseases, as well as appealing to their appearance, may also help in motivating these patients.

IN-OFFICE ORAL HYGIENE PROGRAMS

As mentioned at the beginning of this chapter, preventive dentistry is the foundation on which all oral health care must be built. In establishing this foundation for their patients, practitioners must first look at themselves and their office environment. Each practice must establish a preventive philosophy that is evident throughout the patient's encounter with the dental office. This means that the dentist, the staff, and the practice systems and design must reflect this concept. All staff members must have a personal understanding and appreciation of the importance of this basic concept. This must be evident from their own personal hygiene and in their routine interactions with patients.

After this introspective look and adjustment, the practitioner can now turn to the patient directly. Ong discusses several basic concepts for developing a plaque control program in the dental office.[65] Gathering information from the child and parent is necessary for the practitioner to understand what their concerns are and to let them know that he or she understands these concerns. By discussing the patient's and parents' needs, and listening to and observing their reactions, the practitioner can gauge their readiness to begin the plaque control program. Dental education of the parent and child should be accomplished next with tailoring to the patient's individual problem. Describing exactly why oral hygiene is important in the patient's particular case can help with motivation. The information should be delivered in simple terms and with enthusiasm and conviction. It also needs to be conveyed to the child in age-appropriate language.

When specific age-appropriate oral hygiene instructions are given, it is important to be positive and reassuring, not critical. Use phrases like "Let me show you how to improve," rather than saying, "You're doing it all wrong." Be gentle but firm, and enlist the parent and patient's help in the treatment plan and therapy. Setting goals and complimenting achievements will assist in keeping the parent and patient's attitudes positive. It is very useful to be open to parental and patient feedback regarding their priorities and progress. As with many long-term commitments, cyclic participation can be expected and accepted to a certain extent. However, the parents and patient must know the consequences of neglect. Finally, establishment of a regular maintenance schedule is imperative. Along with prophylaxis, reinstruction and remotivation in the plaque control program is a necessary element for success. Recare intervals should be personalized to the individual patient's needs, with consideration of factors such as caries and periodontal disease risk; restorative, orthodontic, and prosthetic concerns; and individual patient and parental dental education and skill levels. It is the responsibility of every dental practitioner to make oral hygiene and prevention the core of his or her practice. By listening to, educating, adapting to, and motivating our patients and their parents, we can make our preventive practices successful and enjoyable.

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CHAPTER 12

Nutritional Considerations for the Pediatric Dental Patient

JAMES L. McDONALD, JR.

انتشارات
شایان نمودار

CHAPTER OUTLINE

FOOD GUIDE PYRAMID
MALNUTRITION AMONG U.S. CHILDREN
DIETARY PATTERNS
PROBLEMS OF UNDERCONSUMPTION
- Iron Deficiency
- Calcium Inadequacy
- Vitamin D Deficiency
- Vitamin B_{12} Deficiency

PROBLEMS OF OVERCONSUMPTION
- Obesity and Physical Activity Levels

EATING DISORDERS
- Anorexia Nervosa
- Bulimia

NUTRITIONAL CONSIDERATIONS FROM INFANCY THROUGH ADOLESCENCE
- Infant and Toddler (0 to 3 Years Old)
- Preschooler (3 to 6 Years Old)
- School-Aged Child (6 to 12 Years Old)
- Adolescent (12 to 18 Years Old)
- Drug and Alcohol Use in Adolescence

When the leading causes of death in the United States are tabulated (Table 12-1), the list is headed by heart disease and cancer, with stroke a distant third. When the focus is instead on the root causes of death, three major underlying factors related to lifestyle choices can be identified: cigarette smoking, a sedentary lifestyle, and inappropriate dietary choices. It has become increasingly clear that what we eat (and don't eat) is a major factor in determining both the quantity and quality of our lives. The basis of our dietary choices, and thus our nutritional status, is established early in life. It follows that food choices and patterns of eating initiated in childhood affect our health and well-being at every stage of life. There are many ways in which health professionals can promote the health of their patients. A major one is to educate them regarding the importance of following good nutritional principles. This chapter focuses on those nutritional factors that have the greatest potential influence on the health of the pediatric dental patient.

Several decades ago it was recognized that our health is profoundly affected by our dietary choices; since then, an evolution has occurred in efforts to promote healthy food choices in the United States. In 1977, the Senate Select Committee on Nutrition and Human Needs first published the *Dietary Goals for the United States.* This was followed in 1979 *by Healthy People: The Surgeon General's Report on Health Promotion and Disease Prevention* and again in 1988 by *The Surgeon General's Report on Nutrition and Health.* This latter document concluded that 5 of the 10 leading causes of death in the United States were related to poor dietary choices. In 1990, the U.S. Department of Health and Human Services released *Healthy People 2000,* a report that described the general goals for increasing life span, reducing health disparities, and achieving better access to preventive services for all Americans over the following decade. Numerous nutritional goals were outlined in this report. The latest report, *Healthy People 2010,* was released in January 2000. Its overall nutritional focus is to promote health and to reduce chronic disease associated with diet and weight. Chapter 19 of the report discusses a number of these objectives, many of which are related to pediatric nutrition; the latter are summarized in Table 12-2, which shows both the current status and target level for each.

Other nutrition-related objectives for children include reducing sodium consumption, increasing calcium intake, and reducing iron-deficiency anemia. As can be noted from Table 12-2, some of the nutrition objectives (19-5, 19-6, 19-7) are particularly ambitious and require major changes in eating behavior. Whether these objectives realistically can be met is another issue.

The *Dietary Guidelines for Americans* (the ABCs) promulgated by the U.S. Department of Agriculture (USDA) include the following recommendations and support the objectives in *Healthy People 2010:*

Aim for fitness.

- Aim for a healthy weight.
- Be physically active each day.

Build a healthy base.

- Let the Food Guide Pyramid guide your food choices.
- Choose a variety of grains daily, especially whole grains.
- Choose a variety of fruits and vegetables daily.
- Keep food safe to eat.

Choose sensibly.

- Choose a diet low in saturated fat and cholesterol and moderate in fat.
- Choose beverages and foods so as to moderate your intake of sugars.
- Choose and prepare foods with less salt.
- If you drink alcoholic beverages, do so in moderation.

TABLE 12-1 Leading Causes of Death in the United States (Final 2000 Data)

CAUSE	NUMBER OF DEATHS
Heart disease	710,760
Cancer	553,091
Stroke	167,661
Chronic lower respiratory disease	122,009
Accidents	97,900
Diabetes	69,301
Pneumonia/influenza	65,313
Alzheimer disease	49,558
Nephritis, nephritic syndrome, and nephrosis	37,251
Septicemia	31,224

From US Department of Health and Human Services, Centers for Disease Control and Prevention: *Nail Vital Stat* Rep 50(16), 2002.

FOOD GUIDE PYRAMID

The Food Guide Pyramid (see Fig. 10-6 on p. 221) was introduced by the USDA in 1992 as a pictorial representation for the USDA's Daily Food Guide. It is a tool commonly used to help plan a healthful diet. It organizes the foods we eat into five major food groups and one miscellaneous category. The latter category includes

TABLE 12-2 *Healthy People 2010* Selected Nutritional Goals

NUMBER	OBJECTIVE	CURRENT	TARGET
19-3	Reduce the proportion of children and adolescents who are overweight or obese	11%	5%
19-4	Reduce growth retardation among low-income children under 5 years of age	8%	5%
19-5	Increase the proportion of persons aged 2 and older who consume at least two daily servings of fruit	28%	75%
19-6	Increase the proportion of persons aged 2 and older who consume at least three daily servings of vegetables, with at least one third being dark green or deep yellow vegetables	3%	50%
19-7	Increase the proportion of persons aged 2 and older who consume at least six daily servings of grain products, with at least three being whole grains	7%	50%
19-8	Increase the proportion of persons aged 2 and older who consume less than 10% of calories from saturated fat	36%	75%
19-9	Increase the proportion of persons aged 2 and older who consume no more than 30% of calories from fat	33%	75%
19-15	Increase the proportion of children and adolescents age 6 to 19 whose intake of meals and snacks at schools contributes proportionally to good overall dietary quality	*	*

No target established.

TABLE 12-3 Number of Servings of Major Food Groups Needed Each Day

	CHILDREN (2-6 YEARS)	OLDER CHILDREN OR TEENAGED GIRLS	TEENAGED BOYS
1. Milk and milk products	2	3	3
2. Meat and meat alternatives	2	2	3
3. Vegetables	3	4	5
4. Fruits	2	3	4
5. Bread and cereals	6	9	11

fats, oils, and sweets, all of which are to be used sparingly. The major food groups and recommended daily servings for children and adolescents are presented in Table 12-3.

Two cautions should be kept in mind in using the Food Guide Pyramid. One caution is that teens, young adults, pregnant and nursing women, and women concerned about osteoporosis prevention need at least four daily servings from the milk products group or additional calcium from alternative sources. The second caution is that "one serving" does not equal the exaggerated serving sizes often presented at commercial eating establishments, but rather represents a smaller, more conservative food serving size. If one were to use as the standard the large serving sizes typical of today's American culture, then it would be impossible to meet these recommendations without consuming a large excess of calories.

MALNUTRITION AMONG U. S. CHILDREN

Malnutrition may take the form of either undernutrition (inadequate intake of nutrients that potentially leads to deficiency diseases) or overnutrition (excessive dietary intake of energy, fat, or cholesterol that predisposes individuals to chronic diseases later in life). The latter excessive consumption pattern is probably quantitatively more relevant to overall mortality and morbidity rates in contemporary U.S. society than are nutrient deficiencies.

In March 2002, the U.S. Department of Health and Human Services released the infant mortality data as of 2000. These data showed some positive gains compared to the past. The rate at which infants die before their first birthday reached a historic low of 6.9 deaths per 1000 live births. Moreover, the proportion of mothers receiving early prenatal care is at a record high. Malnutrition as measured by low weight for age and low growth rates has also declined. Some of this decline has been attributed to better nutrition. By the same token, the U.S. infant mortality rate continues to rank among the highest of the industrialized nations. The infant mortality rate among black children is more than double that among white children. Also, more than 13 million children in the United States are estimated to

be living below the poverty level, and some estimates indicate that approximately 10% suffer to some degree from clinical malnutrition. Certainly, all poor children are at risk for the long-term consequences of malnutrition.'

DIETARY PATTERNS

Numerous national surveys measuring nutritional status and dietary patterns of children and adolescents were conducted in the 1970s and 1980s. More recent information shows some intriguing trends. In comparing food intake trends among children aged 6 to 11 over approximately a 20-year period from 1977 through 1998, Enns, Mickle, and Goldman reported increases in consumption of soft drinks, grain products as a whole, grain mixtures (crackers, popcorn, pretzels, corn chips), fried potatoes, noncitrus juices and nectars, cheese, candy, and fruit drinks and ades.[2] Decreases were noted in intake of milk in general and whole milk, various vegetables and legumes, beef, pork, and eggs. For any given pyramid group, less than one half of the children consumed the recommended number of servings, and their intakes of discretionary fat and added sugars were much higher than recommended.

Similar findings were reported by Cullen et al, who evaluated the intake of soft drinks, fruit-flavored beverages, and fruits and vegetables by children in grades 4 through 6.[3] Lower parental education level was associated with higher consumption of soft drinks and sweetened beverages, and students who had a high consumption of sweetened beverages reported low fruit and high calorie intakes. Troiano et al have also reported that beverages contribute 20% to 24% of energy intake among youth aged 2 to 19 years and that soft drinks provide 8% of energy intake among adolescents.'

An additional disturbing trend is an increase in away-from-home eating as well as expanding portion sizes in the U.S. marketplace. Again, in examining the changes in dietary intake patterns between 1977 and 1996, Guthrie, Lin, and Frazao reported that food prepared away from home increased from 18% to 32% of total calories consumed.[5] Both meals and snacks prepared away from home contained more calories per eating occasion. Food eaten outside the home was also higher in both total and saturated fat on a percentage basis, and contained less dietary fiber, calcium, and iron per calorie. Based on this information, eating away from home is associated with a compromised quality of nutritional intake.

As touched upon earlier, another major trend being reported is that most marketplace portions of foods exceed standard serving sizes by at least a factor of 2 (bagels and sodas) and sometimes by a factor of 8 (cookies)." Fast-food chains offer larger sizes of hamburgers, sodas, and french fries. The current sizes are often two to five times larger than the original size marketed. These changes in dietary patterns parallel the progressive increase in obesity seen in the United States.

PROBLEMS OF UNDERCONSUMPTION

IRON DEFICIENCY

Although the prevalence of iron deficiency has declined in recent years, it remains an important pediatric public health problem in the United States. Many of the adverse consequences of iron deficiency are associated with its most severe form, iron-deficiency anemia.[?-9] However, iron deficiency without anemia has been linked to negative impacts on cognitive development in children and adolescents.[10] Typically, the high iron needs for growth, when combined with a low dietary intake, produce a low iron status in children. Iron deficiency early in life appears related to behavioral problems in infants who score significantly lower on various tests measuring intellectual and motor functioning. It has long been recognized that toddlers and adolescent females are among the most susceptible groups. The prevalence of iron deficiency has been estimated to be higher among non-Hispanic black and Mexican-American females than among non-Hispanic white females. To prevent iron deficiency, vulnerable populations should be encouraged to eat iron-rich foods and breast-feed or use iron-fortified formula for infants. Iron is found primarily in meat, poultry, and fish. However, other foods such as beans, lentils, fortified cereal grain products, and certain vegetables can make a significant contribution to dietary intake of iron.

Clinical signs of iron-deficiency anemia may include weakness, fatigue, pallor, and numbness and tingling of the extremities. Common oral manifestations are glossitis and fissures at the corners of the mouth *(angular cheilitis)* . The papillae of the tongue may be atrophied, which gives the tongue a smooth, shiny, red appearance.

CALCIUM INADEQUACY

Osteoporosis is a bone disease of older individuals and is primarily seen in postmenopausal women. It is characterized by a reduction in the quantity of skeletal tissue, and thus is often considered to be a geriatric disorder. Education for its prevention, however, is legitimately within the domain of pediatricians and pediatric dentists. Childhood and adolescence are crucial times for development of the skeletal system, and the dietary requirement for calcium peaks during the teenage years. The Food and Nutrition Board of the Institute of Medicine recommends an intake of 1300 mg per day of calcium during adolescence. This equals roughly the amount of calcium present in $4^{1}/_{3}$ cups of milk, so this is not a particularly easy recommendation to meet.

Achieving a high peak bone mass is the first line of defense against osteoporosis. Low calcium intake, particularly in combination with low levels of physical activity, may compromise the attainment of optimal peak bone mass. This is a particularly important consideration for adolescent girls, because almost half of the adult skeletal mass is formed during the second decade of life and calcium accumulation normally triples during the pubertal growth spurt.' Unfortunately, this is the very age group that is at highest risk for low calcium intakes. Only 30% of adolescent girls reach 75% of the recommended daily allowance for calcium, and calcium intake appears to be declining among 6- to 11-yearolds." This problem may be alleviated by educating youth to select more calcium-rich foods (dairy products, fortified breakfast cereals, fortified orange juice concentrates) or to consider using calcium supplements. Calcium carbonate has a good absorption rate and has been characterized as a relatively inexpensive supplement containing a high percentage level of calcium."

VITAMIN D DEFICIENCY

Fortified milk serves as the primary dietary source of vitamin D in the United States. Few other commonly consumed foods contain appreciable quantities of this vitamin. Exposure to sunlight is another important source of vitamin D, because ultraviolet rays from the sun trigger vitamin D synthesis in the skin. Vitamin D is an essential nutrient for proper bone growth, and children who receive too little can develop the hone disease rickets. During the first half of the twentieth century, thousands of cases of vitamin D–deficiency rickets were reported in the United States, particularly in the northern climates during the winter months when exposure to sunlight was minimal. This disease was virtually eradicated once vitamin D began to be added to milk. In recent years, however, somewhat of a resurgence of rickets has been seen, particularly in dark-skinned, breast-fed babies. There appear to be two major reasons for this resurgence. First, heavily pigmented skin is less efficient than is light skin in synthesizing vitamin D from sunlight. Second, many nonwhite people are unable to efficiently digest the lactose in milk, which leads to a significant reduction in milk intake and consequently in vitamin D levels. O'Dell et al reported that, of 1546 black women studied, 42% had blood vitamin D levels below the value considered to reflect a vitamin D deficiency.' In contrast, only 4% of white women demonstrated blood concentrations below that threshold. Thus, vita-min D deficiency appears to be a major unrecognized disease in certain populations of adult women of child-bearing age. The recommended intake of vitamin D for adults who do not receive sufficient exposure to sunlight should be reevaluated in light of these findings.

VITAMIN 13_{12} DEFICIENCY

Vitamin B_{12} is one of the B-complex vitamins, and it contains cobalt in the molecule; thus it is the only vitamin that contains a mineral. Vitamin B_{12} is essential for the synthesis of red blood cells and for myelin synthesis in the nervous system. This vitamin is not thought to be present in plant foods, and as a result strict vegetarians are considered to be at risk for a dietary deficiency of this vitamin. During 2001, neuro-logic impairment resulting from a vitamin B_{17} deficiency was reported in two children breast-fed by mothers who followed vegetarian diets (consumption of limited food of animal origin)." In one of these cases, the diagnosis was made at 15 months, and vitamin B,, therapy was initiated. At age 28 months, the child's developmental skill levels ranged from 9 months for fine motor skills to 18 months for gross motor skills. Her expressive language was at a 10-month level. Health care providers should be alert to the possibility of $B,_2$ deficiency under these circumstances. Patients following vegetarian diets should ensure an adequate intake of vitamin $B,_2$. The only reliable unfortified sources of this vitamin are animal products, including meat, dairy products, and eggs. Plant foods fortified with this vitamin, such as some cereals, meat analogues, soy or rice beverages, and nutritional yeast, can be reliable and regular sources.

PROBLEMS OF OVERCONSUMPTION

For most children and adolescents in the United States, compromised health brought on by malnutrition is much more likely to be related to overconsumption than to underconsumption. The eating environment in contemporary America has been characterized as consisting of convenient, relatively inexpensive, highly palatable foods served in large portions.'[6] The chronic adherence to this type of eating pattern from childhood through adult life contributes to obesity and to numerous diseases, including diabetes, hypertension, coronary heart disease, and certain types of cancer. Those diseases that become evident during adulthood but have their origins in childhood and adolescence provide the health care professional with a potential avenue for preventive medicine. Atherosclerotic heart disease is such a case. Modifiable risk factors can be identified and addressed in the pediatric population, with the goal of preventing or ameliorating heart disease in later life. Thus, prevention of coronary heart disease is a pediatric health issue, as is prevention of the other diseases mentioned earlier.

OBESITY AND PHYSICAL ACTIVITY LEVELS

Data from a variety of sources strongly indicate that we are in the midst of an obesity epidemic among children, adolescents, and adults in the United States. Obesity

results from a chronic imbalance between energy intake and energy expenditure. Although obesity is defined as the excessive accumulation of fat in the body, it is typically diagnosed based on weight and weight-for-height measures. As mentioned previously, a major focus of the nationwide health promotion and disease prevention agenda in *Healthy People 2010 is* to reduce the proportion of children and adolescents who are overweight or obese. Unfortunately, a large disparity is found between the existing prevalence rates of obesity and the prevalence rate goals targeted in *Healthy People 2010."* The percentage of overweight young people in the United States has doubled since 1980, and currently one in seven children and adolescents is affected.[18]

The consequences of obesity are many and are both immediate and long-term. The current epidemic of type 2 diabetes in children and adolescents is associated with obesity and a persistently elevated body mass index (BMI). The BMI is calculated by dividing the individual's weight in kilograms by the square of height in meters. A BMI of 30 or higher generally is considered to indicate obesity and may be a critical etiologic factor in the high prevalence of type 2 diabetes among an increasingly younger population. A recent study found that 25% of obese children (aged 4 to 10 years) were glucose intolerant.[19] This figure is noteworthy because glucose intolerance is a precursor to diabetes. Other obesity-related disorders, such as obstructive sleep apnea, have been observed in as many as one in six obese children. This condition can lead to daytime somnolence, neurocognitive abnormalities, and impaired learning.

Aside from the obvious health risks associated with obesity in childhood and adolescence, there are also significant psychologic and quality-of-life issues to be considered. Ackard et al reported that objective overeating with loss of control in adolescents was associated with lower scores on measures of body satisfaction and self-esteem and higher scores on a measure of depressive mood.[20] Overeating was also associated with suicide risk. Thus, objective overeating among adolescents is linked to a number of adverse behaviors and negative psychologic experiences. It remains to be determined whether objective overeating is an early warning sign of psychologic distress or rather a potential consequence of compromised psychologic health.

Schwimmer, Burwinkle, and Varni compared the health-related quality of life of obese children and adolescents with that of both their healthy, nonobese counterparts and a cohort of children and adolescents diagnosed as having cancer.[18] They found that quality-of-life ratings of severely obese children and adolescents were lower than those of children and adolescents who are healthy and similar to those of children and adolescents diagnosed with cancer. An impaired self-image and perception of low quality of life in this population group is not terribly surprising, because obesity is one of the most stigmatizing and least socially acceptable conditions in childhood. These and similar findings emphasize how critical it is for health professionals, teachers, and parents to be aware not only of the medical risks of obesity in children but also of the potential psychologic significance of this condition.

Because obese children tend to become obese adults, the potential impact of childhood obesity on the health care system is enormous. It has been suggested that the increased medical care costs associated with obesity may be greater than those associated with smoking and drinking.[1-] Severe obesity has long been thought to reduce life expectancy. A recent report states that white men aged 20 years with BMI greater than 45 (extreme obesity) are estimated to lose 13 years of life because of their obesity." If one assumes a life expectancy of 78 years, this translates into *a 22%* reduction in remaining years of life for these individuals. Despite several decades of various public health efforts to educate the population regarding the dangers of obesity, the trend toward progressively increasing body weight has not been reversed or even slowed. The time has clearly come for identifying new strategies to deal with this issue. To focus on reversing this alarming obesity trend in children and adolescents and devising more effective strategies for dealing with it, all the major etiologic factors must be identified.

One such factor is the possible influence of the mass media on childhood health behaviors. Sometimes the media appear to receive the blame for almost every ill present in contemporary society. Nevertheless, the fact remains that a typical teenager's life today is increasingly saturated with the mass media, which often depict an array of unhealthy behaviors, including physical aggression, unprotected sex, smoking, drinking, and poor nutritional choices." Chronic behaviors involving poor nutritional choices inevitably will lead to obesity, high disease risks, or eating disorders such as anorexia nervosa and bulimia.

Clearly more teens than ever are obese, primarily because they eat food of low nutritional density at the expense of fruits and vegetables, and they do not get enough exercise. A 2000 report to the President from the Secretary of Health and Human Services and the Secretary of Education describes our nation's youth as " in large measure, inactive, unfit, and increasingly overweight. This increasingly sedentary lifestyle threatens to reverse the decades of progress achieved in reducing deaths from cardiovascular diseases." Physical activity is a major determinant of both morbidity and mortality. It has been identified as a national priority area for promoting the health of the U.S. population. In spite of the documented benefits of an active lifestyle, however, many Americans, including children and adolescents,

do not meet the recommendations for physical activity. This fact is particularly sobering because the adolescent years are thought to be the period during which adult health-related behaviors such as dietary and physical activity patterns begin to develop. Thus, childhood may be a critical time for promoting physical activity. Studies indicate that the current levels of physical activity in children and adolescents have declined in recent years. For example, walking and bicycling among 5- to 15-year-olds decreased by 40% between 1977 and 1995. The reduction in total physical activity that occurs from mid to late adolescence appears to be related more to a reduction in the number of activities in which adolescents choose to participate than in a decline in the time spent on each activity.' This finding supports other evidence that physical activity declines during adolescence.

The following are some general findings concerning physical activity and health that have emerged over the past decade or two:

People who tend to be inactive can improve their health and well-being by becoming even moderately active on a regular basis.
Physical activity need not be strenuous to provide some health benefits.
Greater health benefits can be achieved by increasing the intensity, frequency, or duration of the physical activity.

Current physical activity recommendations generally state that children and adults should strive for at least 30 minutes daily of physical activity of moderate intensity. The approximate energy expenditure of various physical activities is shown in Table 12-4. An alternate approach might be to engage in separate 5- to 10-minute periods of moderately intense activity throughout the day for a total of at least 30 minutes for adolescents and 60 minutes for children. Walking briskly, biking, swimming, engaging in games and sports, participating in physical education classes, and doing tasks at home or in the garden may all contribute to accumulated physical activity. Walking programs are increasingly being promoted for our youth in selected school systems. Many of these programs are using pedometers to register the number of steps being taken. These small units, which clip onto waistbands, resemble tiny pagers and are reasonably accurate in counting all the steps taken over a given period of time. For some programs, the goal is to reach 12,000 steps per day, but for all the pro-grams, a general objective is to motivate the person wearing the pedometer to progressively increase the step count over time and perhaps to maintain a minimally acceptable level of steps per day.

It seems intuitive that the more time children (and adults for that matter) spend watching television and playing video games, the more likely they are to be obese. Research shows that the prevalence of obesity is lowest among children watching 1 or fewer hours of television a day and highest among those watching 4 or more hours of television a day.'[s] Not only will sedentary children expend less energy, but they will invariably be increasing their consumption of high-fat, high-sugar, high-caloric snack foods during these sedentary periods. Research from more than a decade ago indicates that food (typically sweet snacks) is consumed or referred to three to five times per half-hour on prime-time programming." Also, the majority of commercials shown during children' s programming promote foods with low nutritional value, including candy, soft drinks, sugared cereals, and potato chips, as well as other high-salt, high-fat snacks.'

TABLE 12-4 Approximate Energy Expenditure of Various Types of Exercise

TYPE OF EXERCISE	CALORIES USED PER 30 MINUTES*
Raking leaves	100
Walking (15-minute miles)	**200**
Playing singles tennis	210
Stationary cycling	300
Stair climbing	300
Jogging (10-minute miles)	330
Shoveling snow	350
Rowing	**350**

Data from National Institute of Sports Medicine.
*"These values will be higher for larger individuals, lower for smaller individuals.

EATING DISORDERS

Looking good is certainly a high priority for most teenagers, and for some adolescent girls in particular, getting "thin enough" can become an obsession. Estimates are that 5 million Americans suffer from eating disorders and that 5% of female and 1% of male Americans have anorexia nervosa, bulimia nervosa, or binge-eating disorder. Because women and men are often secretive about their eating disorders, existing epidemiologic studies may underestimate the true prevalence of these conditions." Some personality traits, such as perfectionism and concern with weight and shape, may cluster in families of women with eating disorders. Perfectionism may be an environmentally transmitted or genetically mediated trait that flows down to offspring.[29]

Again, the mass media are viewed by many as a major contributor to the mixed messages regarding desirable eating behaviors and desirable looks. Given

all the unhealthy food consumption depicted in the media, how is it that most of the people in the media, especially white women, are so thin and apparently healthy? Content analyses show that more than two thirds of the women characters on television, but only 18% of the men, are thin. Analyses of body measurements of 500 models listed on modeling agency web sites and of *Playboy* centerfolds from 1985 to 1997 showed that nearly all the centerfolds and three quarters of the models had BMIs of 17.5 or below, a figure that meets the American Psychological Association's criterion for anorexia nervosa.[23] Although boys may be increasingly influenced by the portrayals of muscular men in the media, most research has focused on the effects of media portrayals on girls' eating and dietary habits. Teenaged girls are most at risk for developing eating disorders as they struggle with bodies that are getting larger in a culture that simultaneously attempts to sell them junk food and tells them that they should be thin. For some girls, bulimia must appear as a rational response to these contradictory messages: splurge and then purge.[23]

ANOREXIA NERVOSA

Preoccupation with appearance and body weight during adolescence often leads to restriction in food intake. Anorexia nervosa results when this behavior is carried to the point of starvation. This illness, which is much less common in males and is also less common than bulimia nervosa, is characterized by self-imposed weight loss, amenorrhea, and a distorted attitude toward eating and body weight. In some instances this behavior is used as a means of establishing a sense of identity and control. Anorexia nervosa has captured the interest of psychoanalysts, behavior therapists, family therapists, nutritionists, and endocrinologists alike because of the interplay between the powerful psycho-logic and physiologic components of the disease. Anorexia nervosa rarely begins before puberty and probably is manifest across a wide range of severity levels. Affected individuals often lack the ability to recognize that their emaciated bodies are too thin. Despite their advanced state of wasting, they may continue to believe that they are overweight. The four diagnostic criteria for anorexia nervosa are the following[30]:

- Refusal to maintain a body weight equal to or greater than 85% of that expected for the patient's age and height.
- An intense fear of gaining weight or becoming fat, even though the individual is underweight.
- A distorted view of one's body weight, size, or shape; the emaciated anorexic individual actually feels fat.
- In postmenarcheal women and girls, the absence of at least three consecutive menstrual cycles.

A wide range of complications, including many of the consequences of starvation, is possible in anorexia nervosa. Fat depletion is the most obvious physical consequence. Qualitative deficiencies in the diet may lead to anemia, to hypoproteinemia, and sometimes to vita-min deficiencies. Serious electrolyte imbalances, notably hypokalemia, can occur when vomiting or laxative or diuretic abuse are practiced. Anorexia may be accompanied by enlargement of the parotid glands, edema of the legs, increased facial hair, and reductions in blood pressure and pulse rate. Nutritional deficiencies may lead to a reduction in the amount and pH of the saliva and an increase in dental caries susceptibility. Gingivitis may also be noted. Long-term studies have demonstrated diverse outcomes ranging from full recovery to chronicity and death. Outpatient treatment is preferred for most individuals. If the illness is severe and family and environmental circumstances are too damaging or if there is little response to outpatient treatment, then hospitalization is indicated. Fluoxetine hydrochloride appears to help control the obsessive-compulsive behavior involved in both anorexia nervosa and bulimia. This drug raises the brain levels of serotonin, and as a consequence the urge to binge and the preoccupation with food appears to lessen.

BULIMIA

Another eating disorder, bulimia nervosa, is characterized by binge eating and invariably by self-induced vomiting. It also is more prevalent in young women and is more common than is anorexia nervosa. It usually begins during late adolescence or early adult life. Its prevalence among males is probably vastly underestimated because of underreporting. The American Psychiatric Association diagnostic criteria for bulimia nervosa are the following[3D]:

- Consumption of an unusually large amount of food in a discrete time period (within 2 hours)
- A perceived lack of control over eating during an episode
- Compensatory behavior to rid the body of excess calories and prevent weight gain
- The occurrence of binge eating and compensatory behaviors at least twice a week for 3 months
- A persistent concern with body shape and size

Bulimia nervosa is associated with significant health consequences, although it is more medically benign than is anorexia nervosa. Approximately half of patients with this disorder have fluid and electrolyte abnormalities. A small percentage develop hypokalemia. Enlargement of the parotid glands, esophagitis, and gastric necrosis may also occur. Enamel erosion is common among bulimia nervosa patients because of the exposure

of the tooth surfaces to the highly acidic regurgitated gastric contents. The degree of enamel damage can be extensive. Although unanimity of opinion does not exist, the suggestion has been made that toothbrushing after vomiting actually promotes enamel loss and that, instead, patients should be instructed to rinse with an alkaline solution such as sodium bicarbonate dissolved in water. Other suggestions include use of liquid sugar-free antacids, water, or milk. Fluoride treatment should be considered because of its potential for remineralizing previously demineralized areas of the dentition. Daily rinses with 0.5% sodium fluoride and administration of a 1.1% neutral fluoride gel in custom trays can be recommended.

Most bulimic patients can be treated effectively as outpatients. Multidisciplinary treatment approaches seem indicated, although indications exist that antidepression medications may be helpful in some cases. There is some evidence that individuals who eat dinner with their families regularly score significantly lower on the bulimia-risk scale.[37] Family meals may serve a protective function against disordered eating as well as other problems by acting as a forum for working through various issues.

As health care professionals deal with eating disorders in their patients, they recognize that primary prevention combined with early detection and treatment clearly helps reduce morbidity and mortality in affected adolescents."

NUTRITIONAL CONSIDERATIONS FROM INFANCY THROUGH ADOLESCENCE

During postnatal life, childhood exposure to environmental factors, primarily via dietary intake, will slowly begin to condition adult susceptibility to diseases both positively and negatively." Some examples of dietary factors include calories, saturated fat, sodium, calcium, and antioxidants. Although dietary intervention for disease prevention is possible in adult life, it is difficult and the beneficial results are sometimes limited. A better option is to promote intake of a diet with high nutritional quality early in life; this can reduce risk factors and potentially accomplish major reductions in the incidence of several diseases of adults.

INFANT AND TODDLER (0 TO 3 YEARS OLD)

Except for prenatal existence, the period of most rapid growth in humans occurs during the first 6 months of life. Thus, energy and nutrient requirements are high during this time. A full-term infant is capable of digesting and absorbing protein, a moderate amount of fat, and simple carbohydrates. Starch presents some digestive challenges, however, because amylase, the starch-splitting enzyme, is not produced in significant quantities until approximately the age of 3 months. Moreover, the immature kidneys of an infant cannot concentrate waste efficiently. As a result, the infant must excrete relatively more water than does an adult to eliminate a comparable amount of waste. When dealing with infants, one must always be on guard against dehydration, which has potentially very serious consequences.

Liquid or semiliquid foods are the choice until the teeth begin to erupt. Breast-feeding continues to be the best overall method of infant feeding, and breast milk could well be the infant's only food source for the first 4 to 6 months. Observational studies in affluent populations generally show no difference in weight or length gain between exclusively breast-fed and partially breast-fed infants during the first 4 to 6 months of life. Thereafter, milk can be supplemented with various pureed foods, either homemade or commercially prepared. Breast milk complemented by the infant's own internal stores will meet most of the nutrient needs until the first 6 months have elapsed. Supplements of substances such as vitamin D, iron, and fluoride should be considered after consulting with the child's pediatrician and pediatric dentist. Evidence suggests that low tissue levels of iron adversely affect brain and intellectual development and performance. Whether breast-fed infants should receive iron supplements is still the subject of debate. Although infants who are exclusively breast-fed appear to be iron sufficient at 6 months of age, some breast-fed infants who do not receive iron supplements are in negative iron balance between the ages of 3 and 6 months. Therefore, it may be desirable to begin iron supplementation (often with ferrous sulfate) for the breast-fed infant at about 4 months of age. If the baby is formula-fed, the makeup of the formula determines what additional supplementation is indicated. If an iron-fortified formula is not used, iron supplements again may be recommended after 4 months of age. The use of infant fluoride supplements to maximize caries resistance is discussed elsewhere in this text. The pediatrician and pediatric dentist may be consulted regarding specific situations and local circumstances.

There is no nutritional need for introducing solid foods to infants prior to 6 months of age." Earlier use may contribute to the development of allergies or increased risk of obesity. New foods should be introduced singly to permit detection of allergies. For the first 6 months of life, the optimal single food for the infant is human milk, or alternative formula when indicated. Some concerns have been expressed over inclusion of egg yolk when solid foods begin to be added to the weanling diet because of the potential for elevation of blood cholesterol levels. However, a recent study indicates that egg yolk may be safely introduced into the weanling diet with no elevation in plasma cholesterol level nor increase in the prevalence of egg allergies.[35]

Regular unmodified cow's milk is not considered suitable for infants. It is an insufficient source of vitamin C and iron. Moreover, it may cause gastrointestinal bleeding, and its solute load is too heavy for the infant's renal system to handle. In addition, low-fat milks should not be used by infants because of their insufficient energy provision and their lack of essential fatty acids.

When the infant becomes a toddler and the rapid growth rate of the first year declines, parents are frequently concerned about a very noticeable reduction in appetite. This is a normal occurrence. Although fewer calories may be needed for growth at this stage, the dietary needs for protein and minerals to promote muscle and skeletal development remain high. Thus, a variety of foods should be offered in smaller amounts several times a day to provide these key nutrients. Attractive, brightly colored foods seem to be particularly appealing to children.

PRESCHOOLER (3 TO 6 YEARS OLD)

Physical growth occurs in spurts between 3 and 6 years of age. The child is not growing as rapidly as in the first years of life. Thus, fewer calories are required but relatively high protein and mineral needs remain. A variety of foods should be offered but in lesser amounts. Parents sometimes provide adult-sized food servings to a child who may be only one fifth the size of an adult. Care must be taken not to turn mealtimes into major struggles for control over what to eat, how much to eat, and when to eat.

Although it is important to keep the total fat, saturated fat, and cholesterol at recommended levels in the diet, care must be taken not to overdo this approach. With insufficient intake of calories and nutrients, the child cannot grow and develop to full potential. The child of this age should be helped to grow out of fatness by increasing physical activity rather than by severely restricting calories. Providing wholesome, nutritious snacks can promote adequate intake of essential nutrients without adding excessive calories.

SCHOOL-AGED CHILD (6 TO 12 YEARS OLD)

The 6- to 12-year stage is generally accompanied by a reduced rate of growth, which results in a decline in food requirements per unit of body weight. As a result, there is some need to be more discriminatory in food selection, with emphasis on high nutrient density (foods having a high ratio of nutrients to calories). Vegetables are generally among the least-favored foods in this age group, but fruits are usually liked and provide many of the same nutrients. In this age group, regular eating patterns should be established; at the same time, consumption of nutritious snacks should be stressed and the use of foods, particularly sweets, as rewards should be minimized. Children should be encouraged to eat breakfast. This long-standing advice delivered by mothers from time immemorial seems consistent with the research literature. Reddan, Wahlstrom, and Reicks found in a study of fourth- through sixth-graders in a school with a universal school breakfast program that the majority of students felt that eating breakfast provides benefits of increased energy and ability to pay attention in schoo1.[36] Barriers to eating breakfast cited by students were not having time and not being hungry in the morning. A focus on eating foods with high nutrient value and maintaining sufficient physical activity levels is important throughout this age range, because obesity problems often begin in this period.

ADOLESCENT (12 TO 18 YEARS OLD)

The nutritional requirements of adolescents are influenced primarily by the onset of puberty and the final growth spurt of childhood. This profound increase in growth rate is accompanied by increased needs for energy, protein, vitamins, and minerals. In general, girls consume far less food than do boys and must meet their needs for individual nutrients within a smaller range of caloric intakes. As a result, adolescent females are at high risk for nutritional inadequacies. In addition, girls often encounter significant social and peer pressure to restrict food intake for weight-control purposes. A multitude of fad weight-loss diets may be tried at this time. Some adolescent girls also turn to cigarette smoking to help control their body weight. The nutritional status of adolescent girls remains of concern because of low dietary intakes and attendant marginal iron, calcium, and folic acid status. Generally, as girls progress through adolescence, their average intake of vitamins and minerals and calories declines, while their nutritional requirements actually increase. Particularly among adolescent female athletes in sports that emphasize leanness, research indicates that reported energy and nutrient intakes are generally well below recommended levels and lower than those of typical teenage girls.[37]

Teenagers, particularly boys, receive a greater proportion of their total energy intake from snacks than do other population groups. Although these snack items may have significant nutritional value, more often than not they are high in fat and sugar and thus contribute relatively low quantities of essential nutrients compared with their caloric content. The result may be the establishment of lifetime eating patterns that promote future risk of heart disease, obesity, hypertension, and cancer.

Table 12-5 summarizes some major gender differences in adolescent nutrition issues.

DRUG AND ALCOHOL USE IN ADOLESCENCE

The teenage years are typically a period of search for identity, independence, and peer acceptance. Enmeshed

TABLE 12-5 Gender Differences in Adolescent Nutritional Issues

FEMALES	MALES
Lower energy needs	Higher energy needs
Thinness considered important	Strength considered important
Concern about peak bone mass	Less concern about peak bone mass
Higher risk for eating disorders	Lower risk for eating disorders
Higher risk for nutritional deficiencies	Lower risk for nutritional deficiencies

within this search are a myriad of personal choices to be made, including decisions concerning the use of drugs, alcohol, and tobacco. If the choice is made to use these types of substances, the individual should recognize that all have significant negative impacts on fitness and health. Aside from the dangers of establishing a lifetime addiction and increasing the risk of numerous diseases, the use of alcohol, nicotine, and other drugs has a significant negative impact on the nutrient status of the affected individual.

Nicotine is the psychoactive substance in tobacco products. Based on the fact that 90% of tobacco users become addicted to the use of tobacco, nicotine may be considered one of the most addictive substances known. Cigarette smoking affects nutrition in numerous ways. Studies have shown that smokers have a lower intake than nonsmokers of numerous essential nutrients, including vitamins C and A, 3-carotene, folic acid, and dietary fiber. Moreover, several of these nutrients have been associated with a reduced risk of lung cancer, a disease for which cigarette smokers are at high risk. Therefore, the typical smoker is receiving less of the very nutrients most needed. Evidence also exists that smokers metabolize vitamin C more rapidly than do nonsmokers. The result is that smokers require approximately twice as much vitamin C as do nonsmokers to maintain similar blood levels. Smoking also influences both hunger and body weight, tending to postpone feelings of hunger and to reduce weight. In spite of the fact that smokers as a group weigh less than nonsmokers, there are strong indications that smoking is associated with greater fat accumulations in the central portion of the body (a higher waist-to-hip ratio).

Tobacco use in youth has long been considered to be a gateway to the subsequent introduction and use of other drugs. For example, in a study of 20,000 children and adolescents, those who were pack-a-day smokers were more likely to drink alcohol, were 7 times more likely to use smokeless tobacco, and were 10 to 30 times more likely to use illicit drugs than were nonsmokers." Moreover, there was a strong dose-dependent relation-ship between smoking behavior and binge drinking, as well as between smoking and the use of alcohol and illicit drugs.

Alcohol is a very popular substance among many of the nation's youth and is not difficult to obtain for the vast majority of them. Some adolescents, as well as adults, use alcohol as an inappropriate way to cope with problems and stress. Nutritionally speaking, alcohol is a relatively high energy substance (7 kcal/g versus 4 kcal/g for carbohydrates) that is devoid of any nutritional value. Alcohol use displaces the intake of essential nutrients. With chronic, excessive use it adversely affects the absorption, transport, and metabolism of nutrients, which sometimes leads to serious nutritional inadequacies.

The long-term use of other drugs, such as cocaine and heroin, often produces numerous adverse physiologic effects, many of which compromise the nutritional state of the user and may result in multiple nutritional problems. Chronic abuse of these drugs may lead to eating disorders, in part because of chronic loss of appetite produced by the drugs. Often, during the "highs" produced by drugs, the individual loses interest in food, eating, and nutrient intake. The nutritional quality of the diet suffers under these circumstances.

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CHAPTER 13

Local Anesthesia and Pain Control for the Child and Adolescent

RALPH E. McDONALD
DAVID R. AVERY
JEFFREY A. DEAN

انتشارات
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CHAPTER OUTLINE

TOPICAL ANESTHETICS
JET INJECTION
LOCAL ANESTHESIA BY CONVENTIONAL INJECTION
ANESTHETIZATION OF MANDIBULAR TEETH AND SOFT TISSUE
- Inferior Alveolar Nerve Block (Conventional Mandibular Block)
- Lingual Nerve Block
- Long Buccal Nerve Block
- Infiltration Anesthesia for Mandibular Primary Molars
- Infiltration for Mandibular Incisors
- Mandibular Conduction Anesthesia (Gow-Gates Mandibular Block Technique)

ANESTHETIZATION OF MAXILLARY PRIMARY AND PERMANENT INCISORS AND CANINES
- Supraperiosteal Technique (Local Infiltration)

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ANESTHETIZATION OF MAXILLARY PERMANENT MOLARS
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SUPPLEMENTAL INJECTION TECHNIQUES
- Infraorbital Nerve Block and Mental Nerve Block
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- Intraosseous Injection, Interseptal Injection, and Intrapulpal Injection
- Computer-Controlled Local Anesthetic Delivery System (Wand)

COMPLICATIONS AFTER A LOCAL ANESTHETIC
- Anesthetic Toxicity
- Trauma to Soft Tissue

ELECTRONIC DENTAL ANESTHESIA
ANALGESICS

t is generally agreed that one of the most important aspects of child behavior guidance is the control of pain. If children experience pain during restorative or surgical procedures, their future as dental patients may be damaged. Therefore it is important at each visit to reduce discomfort to a minimum and to control painful situations. There are many pharmacologic pain control strategies to help children cope with these situations, both preoperatively and postoperatively. Most of these strategies involve the use of local anesthetics and/or analgesics.

Because there is usually some discomfort associated with the procedure, use of a local anesthetic is generally indicated when operative work is to be performed on the permanent teeth, and the same is true of cavity preparations in primary teeth. Dental procedures can be carried out more effectively if the child is comfortable and free of pain. The local anesthetic can prevent discomfort that may be associated with placing a rubber dam clamp, ligating teeth, and cutting tooth structure. Even the youngest child treated in the dental office normally presents no contraindications for the use of a local anesthetic.

Investigators have found that injection is the dental procedure that produces the greatest negative response in children. Responses become increasingly negative over a series of four or five injections. Venham and Quatrocelli[i] have reported that a series of dental visits sensitized children to the stressful injection procedure while reducing their apprehension toward relatively nonstressful procedures. Thus dentists should anticipate the need for continued efforts to help the child cope with dental injections.

TOPICAL ANESTHETICS

Topical anesthetics reduce the slight discomfort that may be associated with the insertion of the needle before the injection of the local anesthetic. Some topical anesthetics, however, present a disadvantage if they have a disagreeable taste to the child. Also, the additional time required to apply them may allow the child to become apprehensive concerning the approaching procedure.

Topical anesthetics are available in gel, liquid, ointment, and pressurized spray forms. However, the pleasant-tasting and quick-acting liquid, gel, or ointment preparations seem to be preferred by most dentists. These agents are applied to the oral mucous membranes with a cotton-tipped applicator. A variety of anesthetic agents have been used in topical anesthetic preparations, including ethyl aminobenzoate, butacaine sulfate, cocaine, dyclonine, lidocaine, and tetracaine.

Ethyl aminobenzoate (benzocaine) liquid, ointment, or gel preparations are probably best suited for topical anesthesia in dentistry. They offer a more rapid onset and longer duration of anesthesia than other topical agents. They are not known to produce systemic toxicity as oral topical anesthetics, but a few localized allergic reactions have been reported from prolonged or repeated use. Examples of commercially available products are Hurricaine,[*] Topicale,[†] and Gingicaine.[‡] All three products are available in gel form. Gingicaine is also available in liquid and spray forms, and Hurricaine is available as a liquid. Topicale is available in ointment form.

The mucosa at the site of the intended needle insertion is dried with gauze, and a small amount of the topical anesthetic agent is applied to the tissue with a cotton swab. Topical anesthesia should be produced in approximately 30 seconds.

During the application of the topical anesthetic, the dentist should prepare the child for the injection. The explanation should not necessarily be a detailed description but simply an indication that the tooth is going to be put to sleep so that the treatment can proceed without discomfort.

A more recently developed product for achieving topical anesthesia is known as DentiPatch (lidocaine transoral delivery system). This system seems to be designed primarily for situations in which superficial oral tissue anesthesia is desired for several minutes rather than the shorter time required for local anesthetic injections. The use of this product has not yet been shown to be convenient or efficacious in young children.

JET INJECTION

The jet injection instrument is based on the principle that small quantities of liquids forced through very small openings under high pressure can penetrate mucous membrane or skin without causing excessive tissue trauma. One jet injection device, the Syrijet Mark II II holds a standard 1.8-ml cartridge of local anesthetic solution. It can be adjusted to expel 0.05 to 0.2 ml of solution under 2000 psi pressure.

Jet injection produces surface anesthesia instantly and is used by some dentists instead of topical anesthetics. The method is quick and essentially painless, though the abruptness of the injection may produce momentary anxiety. This technique is also useful for obtaining gingival anesthesia before a rubber dam clamp is placed for isolation procedures that otherwise do not require local anesthetic. Similarly, soft tissue anesthesia may be obtained before band adaptation of

*Beutlich L.P. Pharmaceuticals, Inc., Chicago, Ill. †Premier Dental Products, Inc., Plymouth Meeting, Penn. ‡Gingi-Pak, Inc., Camarillo, Calif.
§Noven Pharmaceuticals, Inc, Miami, Fla.
‖Mizzy, Inc., Cherry Hill, N.J.

partially erupted molars or for the removal of a very loose (soft tissue–retained) primary tooth. O'Toole has reported that the Syrijet may be employed in place of needle injections for nasopalatine, anterior palatine, and long buccal nerve blocks.[1]

A more recently developed jet injection device reported by Duckworth et al delivers a dose of dry powdered anesthetic to the oral mucosa.[2] In this study, an initial trial with 14 adult subjects, successful topical analgesia without tissue damage was reported. Of course, more clinical trials are required before substantial claims can be made regarding how efficacious the technique is and whether its routine use for topical anesthesia is warranted.

LOCAL ANESTHESIA BY CONVENTIONAL INJECTION

Wittrock and Fischer[1] and later Trapp and Davies[5] demonstrated that human blood can be readily aspirated with the smaller-gauge needles. Trapp and Davies reported positive aspiration through 23-, 25-, 27-, and 30-gauge needles without a clinically significant difference in resistance to flow. Malamed recommends the use of larger-gauge needles (i.e., 25 gauge) for injection into highly vascular areas or areas where needle deflection through soft tissue may be a factor.[6] Regardless of the size of the needle used, it is generally agreed that the anesthetic solution should be injected slowly and that the dentist should watch the patient closely for any evidence of an unexpected reaction.

The injections that are most commonly used in the treatment of children are described in the following sections.

ANESTHETIZATION OF MANDIBULAR TEETH AND SOFT TISSUE

INFERIOR ALVEOLAR NERVE BLOCK (CONVENTIONAL MANDIBULAR BLOCK)

When deep operative or surgical procedures are undertaken for the mandibular primary or permanent teeth, the inferior alveolar nerve must be blocked. The supraperiosteal injection technique may sometimes be useful in anesthetizing primary incisors, but it is not as reliable for complete anesthesia of the mandibular primary or permanent molars.

Olsen reported that the mandibular foramen is situated at a level lower than the occlusal plane of the primary teeth of the pediatric patient.[1] Therefore the injection must be made slightly lower and more posteriorly than for an adult patient. An accepted technique is one in which the thumb is laid on the occlusal surface of the molars, with the tip of the thumb resting on the internal oblique ridge and the ball of the thumb resting in the retromolar fossa. Firm support during the injection procedure can be given when the ball of the middle finger is resting on the posterior border of the mandible. The barrel of the syringe should be directed on a plane between the two primary molars on the opposite side of the arch. It is advisable to inject a small amount of the solution as soon as the tissue is penetrated and to continue to inject minute quantities as the needle is directed toward the mandibular foramen.

The depth of insertion averages about 15 mm but varies with the size of the mandible and its changing proportions depending on the age of the patient. Approximately 1 ml of the solution should be deposited around the inferior alveolar nerve (Figs. 13-1 and 13-2).

LINGUAL NERVE BLOCK

One can block the lingual nerve by bringing the syringe to the opposite side with the injection of a small quantity of the solution as the needle is withdrawn. If small amounts of anesthetic are injected during insertion and withdrawal of the needle for the inferior alveolar nerve block, the lingual nerve will invariably be anesthetized as well.

LONG BUCCAL NERVE BLOCK

For the removal of mandibular permanent molars or sometimes for the placement of a rubber dam clamp on these teeth, it is necessary to anesthetize the long buccal nerve. A small quantity of the solution may be deposited in the mucobuccal fold at a point distal and buccal to the indicated tooth (Fig. 13-3).

All facial mandibular gingival tissue on the side that has been injected will be anesthetized for operative procedures, with the possible exception of the tissue facial to the central and lateral incisors, which may receive

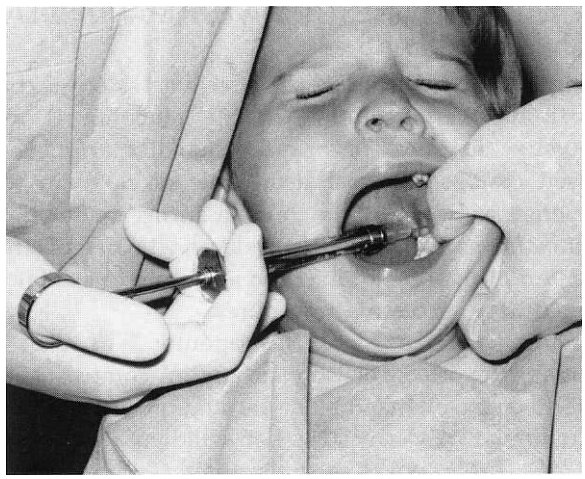


FIG **13-1.** The mandible is supported by the thumb and middle finger, while the needle is directed toward the inferior alveolar nerve.

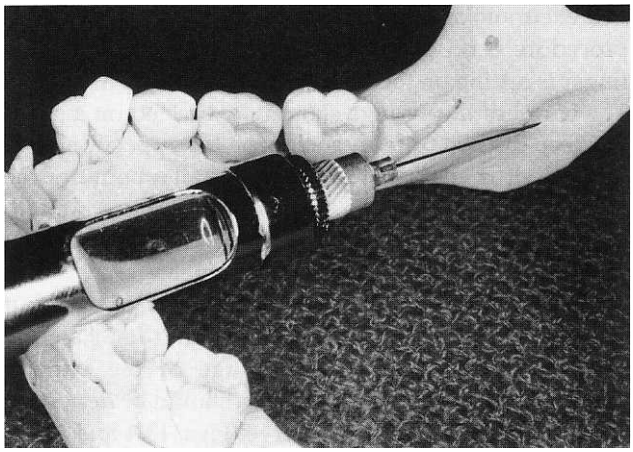


FIG 13-2. Anesthetic solution is deposited around the inferior alveolar nerve.

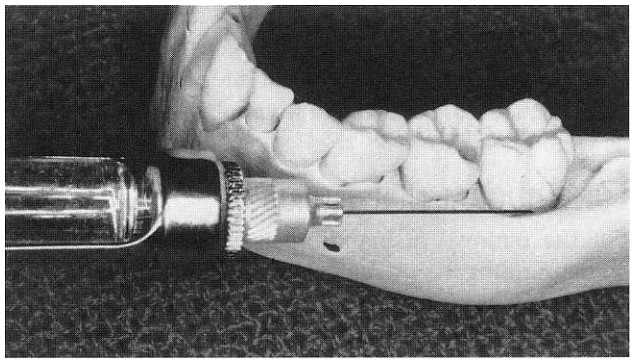


FIG 13-3. In anesthetizing the long buccal nerve, a small quantity of the solution may be deposited in the mucobuccal fold adjacent to the first permanent molar.

innervation from overlapping nerve fibers from the opposite side.

INFILTRATION ANESTHESIA FOR MANDIBULAR PRIMARY MOLARS

Wright et al have studied the effectiveness of injecting local anesthetic solution in the mucobuccal fold between the roots of primary mandibular molars.' Based on observational data, little or no pain was experienced by 65% of the children during cavity preparation. It was also noted that children who demonstrate comfort at the time of injection are likely to exhibit no pain during successive procedures.

Oulis, Vadiakas, and Vasilopoulou,' as well as Sharaf,[1] ° reported very similar studies comparing the effectiveness of mandibular infiltration anesthesia to mandibular block anesthesia in children 3 to 9 years of age for restorative pulpotomy and extraction therapies in primary mandibular molars. Eighty-nine and 80 children, respectively, were included in their studies. Oulis, Vadiakas, and Vasilopoulou found that the two anesthesia techniques were equally effective for restorative procedures, but the mandibular infiltration technique was less effective than mandibular block for extraction and pulpotomy.' Sharaf reported that the infiltration anesthesia was as effective as block anesthesia for all the performed therapies except for pulpotomy in mandibular second primary molars.[10] From these reports, one may infer that mandibular block anesthesia produces more profound anesthesia of mandibular primary molars, but infiltration may produce adequate anesthesia in mandibular primary molars for most restorative procedures.

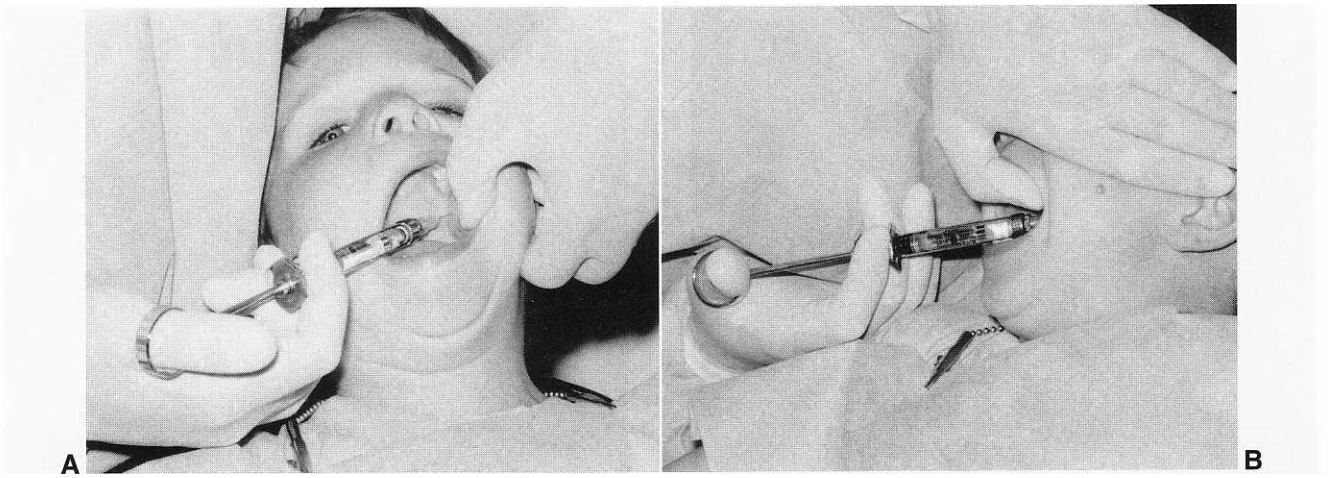
INFILTRATION FOR MANDIBULAR INCISORS

The terminal ends of the inferior alveolar nerves cross over the mandibular midline slightly and provide conjoined innervation of the mandibular incisors. Therefore a single inferior alveolar nerve block may not be adequate for operative or surgical procedures on the incisors, even on the side of the block anesthesia. The labial cortical bone overlying the mandibular incisors is usually thin enough for supraperiosteal anesthesia techniques to be effective.

If only superficial caries excavation of mandibular incisors is needed or if the removal of a partially exfoliated primary incisor is planned, infiltration anesthesia alone may be adequate. Incisor infiltration is most useful as an adjunct to an inferior alveolar nerve block when total anesthesia of the quadrant is desired. In this case the infiltration injection is made close to the mid-line on the side of the block anesthesia, but the solution is deposited labial to the incisors on the opposite side of the midline. For example, if block anesthesia is used for the mandibular right quadrant, anesthetic solution is infiltrated over the left mandibular incisors by insertion of the needle just to the right of the midline diagonally toward the left incisors.

MANDIBULAR CONDUCTION ANESTHESIA (GOW-GATES MANDIBULAR BLOCK TECHNIQUE)

In 1973 Gow-Gates introduced a new method of obtaining mandibular anesthesia, which he referred to as *mandibular conduction anesthesia."* This approach uses external anatomic landmarks to align the needle so that anesthetic solution is deposited at the base of the neck of the mandibular condyle. This technique is a nerve block procedure that anesthetizes virtually the entire distribution of the fifth cranial nerve in the mandibular area, including the inferior alveolar, lingual, buccal, mental, incisive, auriculotemporal, and mylohyoid nerves. Thus with a single injection the entire right or left half of the mandibular teeth and soft tissues can be anesthetized, except possibly the mandibular incisors, which may receive partial innervation from the incisive nerves of the opposite side. Gow-Gates suggested that, once the



f **I G 13-4. A,** Injection site for the Gow-Gates mandibular block technique. B, Barrel of the syringe is aligned with a line from the corner of the mouth to the intertragic notch.

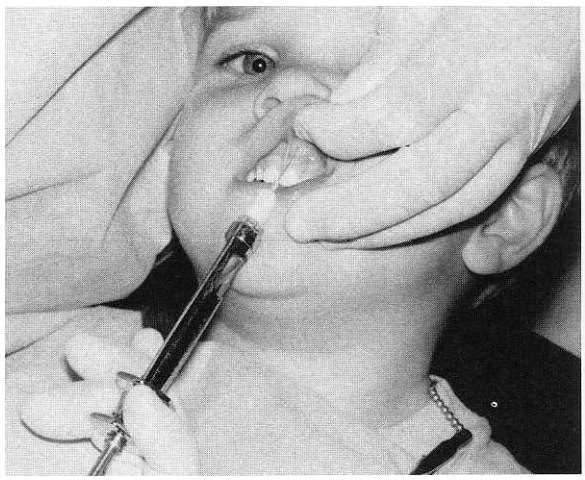
technique is learned properly, it rarely fails to produce good mandibular anesthesia." He had used the technique in practice more than 50,000 times. The technique has become increasingly popular and is often referred to as the *Gow-Gates technique.*

The external landmarks to help align the needle for this injection are the tragus of the ear and the corner of the mouth. The needle is inserted just medial to the tendon of the temporal muscle and considerably superior to the insertion point for conventional mandibular block anesthesia. The needle is also inclined upward and parallel to a line from the corner of the patient's mouth to the lower border of the tragus (intertragic notch). The needle and the barrel of the syringe should be directed toward the injection site from the corner of the mouth on the opposite side (Fig. 13-4).

ANESTHETIZATION OF MAXILLARY PRIMARY AND PERMANENT INCISORS AND CANINES

SUPRAPERIOSTEAL TECHNIQUE (LOCAL INFILTRATION)

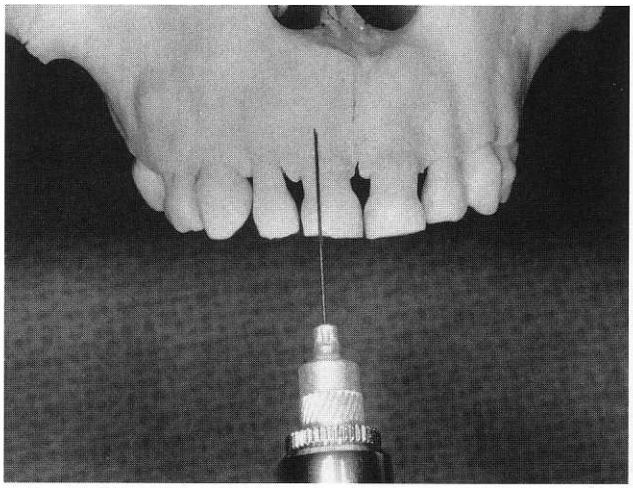
Local infiltration (supraperiosteal technique) is used to anesthetize the primary anterior teeth. The injection should be made closer to the gingival margin than in the patient with permanent teeth, and the solution should be deposited close to the bone. After the needle tip has penetrated the soft tissue at the mucobuccal fold, it needs little advancement before the solution is deposited (2 mm at most) because the apices of the maxillary primary anterior teeth are essentially at the level of the mucobuccal fold. Some dentists prefer to pull the upper lip down over the needle tip to penetrate the tissue rather than advancing the needle upward. This approach works quite well for the maxillary anterior region (Figs. 13-5 to 13-7).



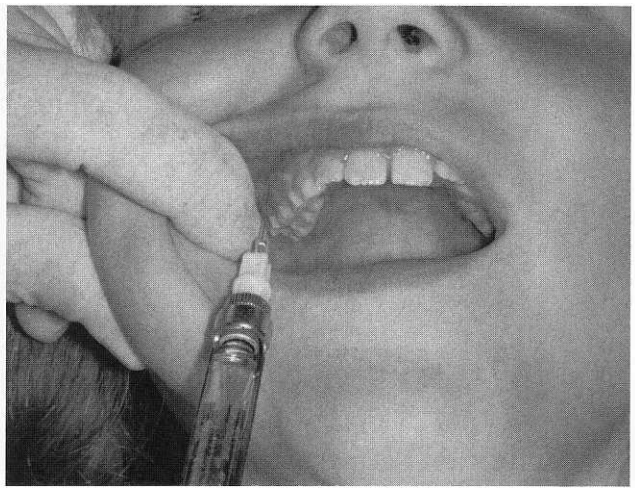
13-5. Anesthetizing a primary central incisor. The supraperiosteal injection should be close to the bone and adjacent to the apex of the tooth.

In anesthetizing of the permanent central incisor teeth the puncture site is at the mucobuccal fold, so that the solution may be deposited slowly and slightly above and close to the apex of the tooth. Because nerve fibers may be extending from the opposite side, it may be necessary to deposit a small amount of the anesthetic solution adjacent to the apex of the other central incisor to obtain adequate anesthesia in either primary or permanent teeth. If a rubber darn is to be applied, it is advisable to inject a drop or two of anesthetic solution into the lingual free marginal tissue to prevent the discomfort associated with the placement of the rubber dam clamp and ligatures.

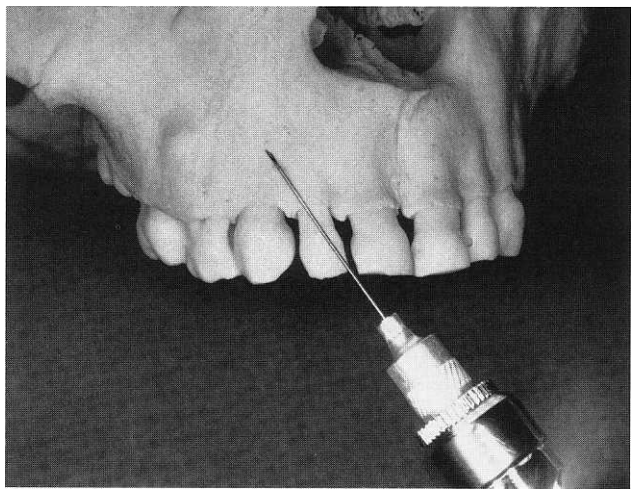
Before extraction of the incisors or canines in either the primary or permanent dentition, it will be necessary to anesthetize the palatal soft tissues. The nasopalatine injection provides adequate anesthesia for the palatal



H G. **13-6.** Needle point is opposite the apex of the maxillary primary incisor.



f **I G 13-8.** Injection of the anesthetic solution to anesthetize the maxillary first primary molar for operative procedures.



f **I G 13-7.** Position of the needle for anesthetizing a maxillary primary canine.

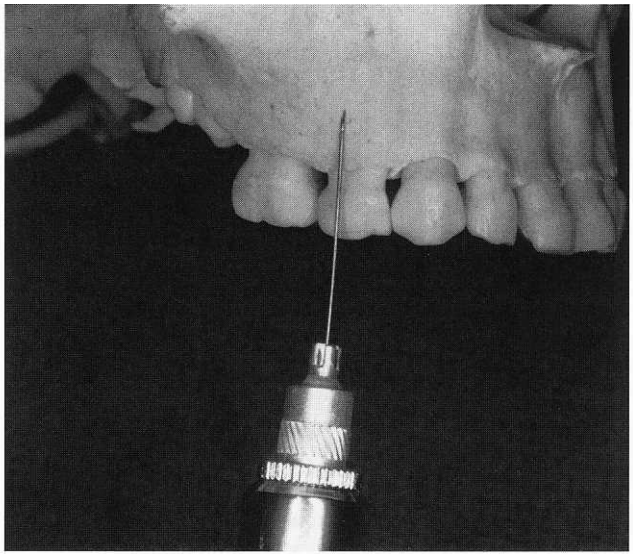
tissues of all four incisors and at least partial anesthesia of the canine areas. Nerve fibers from the greater (anterior) palatine nerve usually extend to the canine area as well. If only a single anterior tooth is to be removed, adequate palatal anesthesia may also be obtained when anesthetic solution is deposited in the attached palatal gingiva adjacent to the tooth to be removed. If it is observed that the patient does not have profound anesthesia of anterior teeth during the operative procedures with the supraperiosteal technique, a nasopalatine injection is advisable.

ANESTHETIZATION OF MAXILLARY PRIMARY MOLARS AND PREMOLARS

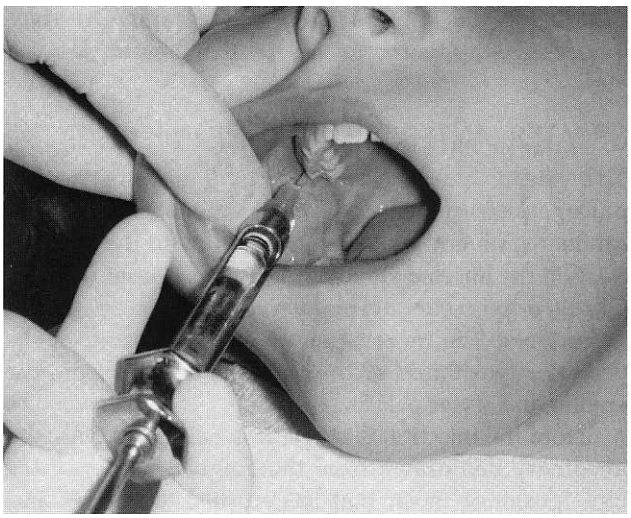
Traditionally, dentists have been taught that the middle superior alveolar nerve supplies the maxillary primary molars, the premolars, and the mesiobuccal root of the first permanent molar. There is no doubt that the middle superior alveolar nerve is at least partially responsible for the innervation of these teeth. However, Jorgensen and Hayden have demonstrated plexus formation of the middle and posterior superior alveolar nerves in the primary molar area on child cadaver dissections.[12] The role of the posterior superior alveolar nerve in innervating the primary molar area has not previously received adequate attention. In addition, Jorgensen and Hayden have demonstrated maxillary bone thickness approaching 1 cm overlying the buccal roots of the first permanent and second primary molars in the skulls of children.[12]

The bone overlying the first primary molar is thin, and this tooth can be adequately anesthetized by injection of anesthetic solution opposite the apices of the roots (Figs. 13-8 and 13-9). However, the thick zygomatic process overlies the buccal roots of the second primary and first permanent molars in the primary and early mixed dentition. This thickness of bone renders the supraperiosteal injection at the apices of the roots of the second primary molar much less effective; the injection should be supplemented with a second injection superior to the maxillary tuberosity area to block the posterior superior alveolar nerve as has been traditionally taught for permanent molars (Figs. 13-10 and 13-11). This supplemental injection helps compensate for the additional bone thickness and the posterior middle superior alveolar nerve plexus in the area of the second primary molar, which compromise the anesthesia obtained by injection at the apices only.

To anesthetize the maxillary first or second premolar, a single injection is made at the mucobuccal fold to allow the solution to be deposited slightly above the apex of the tooth. Because of the horizontal and vertical



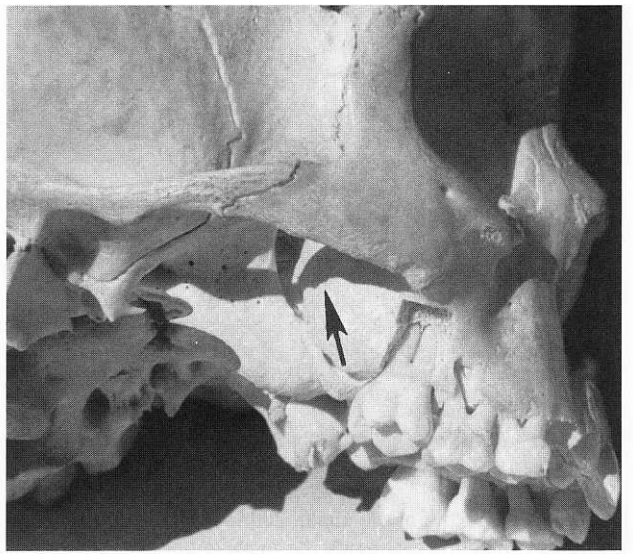
[I G **13-9.** Anesthetic solution is injected opposite the apices of the buccal roots of the first primary molar.



[I G **13-10.** Posterior superior alveolar injection for maxillary permanent molars and second primary molar.

growth of the maxilla that has occurred by the time the premolars erupt, the buccal cortical bone overlying their roots is thin enough to permit good anesthesia with this method. The injection should be made slowly, and the solution should be deposited close to the bone; these recommendations hold true for all supraperiosteal and block anesthesia techniques in dentistry.

Before operative procedures for maxillary primary molars and maxillary premolars, the appropriate injection technique(s) for the buccal tissues, as just described, should be performed. If the rubber dam clamp impinges on the palatal tissue, injection of a drop or two of the anesthetic solution into the free marginal tissue lingual to the clamped tooth will alleviate the discomfort and



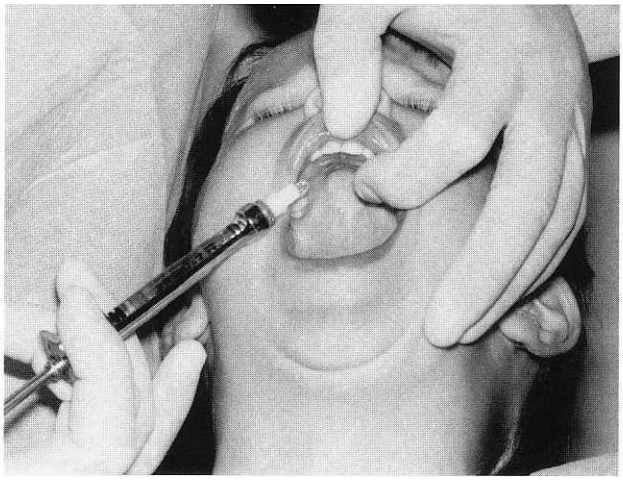
IIG. **13-11.** Maxillary tuberosity area (arrow) for posterior superior alveolar injection. (Courtesy Dr. Paul E. *Starkey.)*

will be less painful than the true greater (anterior) palatine injection. The greater palatine injection is indicated if maxillary primary molars or premolars are to be extracted or if palatal tissue surgery is planned.

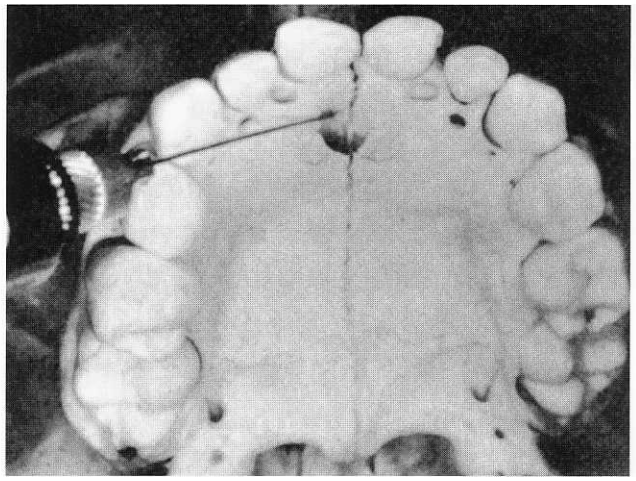
ANESTHETIZATION OF MAXILLARY PERMANENT MOLARS

To anesthetize the maxillary first or second permanent molars, the dentist instructs the child to partially close the mouth to allow the cheek and lips to be stretched laterally. The tip of the dentist's left forefinger (for a right-handed dentist) will rest in a concavity in the mucobuccal fold and is rotated to allow the fingernail to be adjacent to the mucosa. The ball of the finger is in contact with the posterior surface of the zygomatic process. Bennett suggests that the finger be on a plane at right angles to the occlusal surfaces of the maxillary teeth and at a 45-degree angle to the patient's sagittal plane. [3] The index finger should point in the direction of the needle during the injection. The puncture point is in the mucobuccal fold above and distal to the distobuccal root of the first permanent molar. If the second molar has erupted, the injection should be made above the second molar. The needle is advanced upward and distally, depositing the solution over the apices of the teeth. The needle is inserted for a distance of approximately 3/4 inch (2 cm) in a posterior and upward direction; it should be positioned close to the bone, with the bevel toward the bone (see Figs. 13-10 and 13-11).

To complete the anesthesia of the first permanent molar for operative procedures, the supraperiosteal injection is made by insertion of the needle in the mucobuccal fold and deposition of the solution at the apex of the mesiobuccal root of the molar.



f I 13-12. Blocking of the nasopalatine nerve may be accomplished by injecting alongside the incisive papilla.



f I G 13-13. The needle is directed upward into the incisive canal when anesthetizing the nasopalatine nerve.

ANESTHETIZATION OF THE PALATAL TISSUES

Anesthesia of the palatal tissues can be one of the more exquisitely painful procedures performed in dentistry. Ramirez, Lee, and Takara discuss methods for achieving profound anesthesia with minimal pain in the palatal and lingual aspects." After buccal infiltration, they suggest interdental (interpapillary) infiltration, with slow injection of the anesthetic solution as the needle is penetrating the papilla. The interdental infiltration allows diffusion of the anesthetic to the palatal aspect via the craterlike area of the interproximal oral mucosa joining the lingual and buccal interdental papillae, known as the col. Blanching of the area is indicative of sufficient anesthesia of the superficial soft tissues; however, additional palatal infiltration may be given as needed.

NASOPALATINE NERVE BLOCK

Blocking the nasopalatine nerve will anesthetize the palatal tissues of the six anterior teeth. If the needle is carried into the canal, it is possible to anesthetize the six anterior teeth completely. However, this technique is painful and is not routinely used before operative procedures. If the patient experiences incomplete anesthesia after supraperiosteal injection above the apices of the anterior teeth on the labial side, it may be necessary to resort to the nasopalatine injection. The path of insertion of the needle is alongside the incisive papilla, just posterior to the central incisors. The needle is directed upward into the incisive canal (Figs. 13-12 and 13-13). The discomfort associated with the injection can be reduced by deposition of the anesthetic solution in advance of the needle. When anesthesia of the canine area is required, it may be necessary to inject a small amount of anesthetic solution into the gingival tissue adjacent to the lingual aspect of the canine to anesthetize overlapping branches of the greater palatine nerve.

GREATER (ANTERIOR) PALATINE INJECTION

The greater palatine injection will anesthetize the mucoperiosteum of the palate from the tuberosity to the canine region and from the median line to the gingival crest on the injected side. This injection is used with the middle or posterior alveolar nerve block before surgical procedures. The innervation of the soft tissues of the posterior two thirds of the palate is derived from the greater and lesser palatine nerves.

Before the injection is made, it is helpful to bisect an imaginary line drawn from the gingival border of the most posterior molar that has erupted to the midline. Approaching from the opposite side of the mouth, the dentist makes the injection along this imaginary line and distal to the last tooth (Figs. 13-14 and 13-15). In the child in whom only the primary dentition has erupted, the injection should be made approximately 10 mm posterior to the distal surface of the second primary molar. It is not necessary to enter the greater palatine foramen. A few drops of the solution should be injected slowly at the point where the nerve emerges from the foramen.

SUPPLEMENTAL INJECTION TECHNIQUES

INFRAORBITAL NERVE BLOCK AND MENTAL NERVE BLOCK

The infraorbital nerve block and the mental nerve block are two additional local anesthetic techniques used by many dentists. The infraorbital nerve block anesthetizes

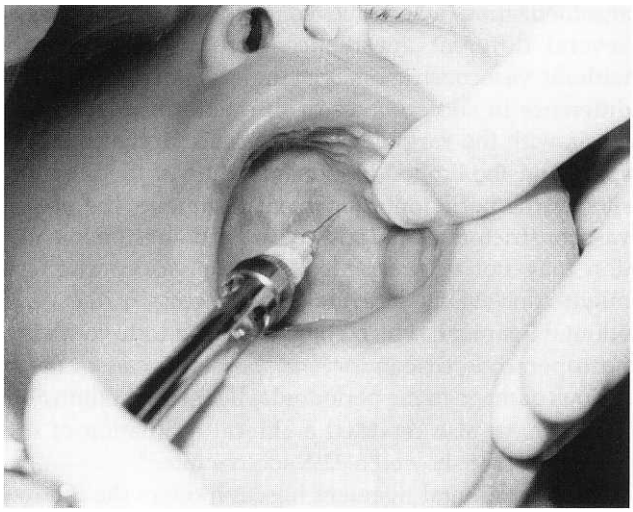


FIG 13-14. Greater palatine injection is used in conjunction with the middle or posterior alveolar nerve block before removal of a maxillary primary molar.

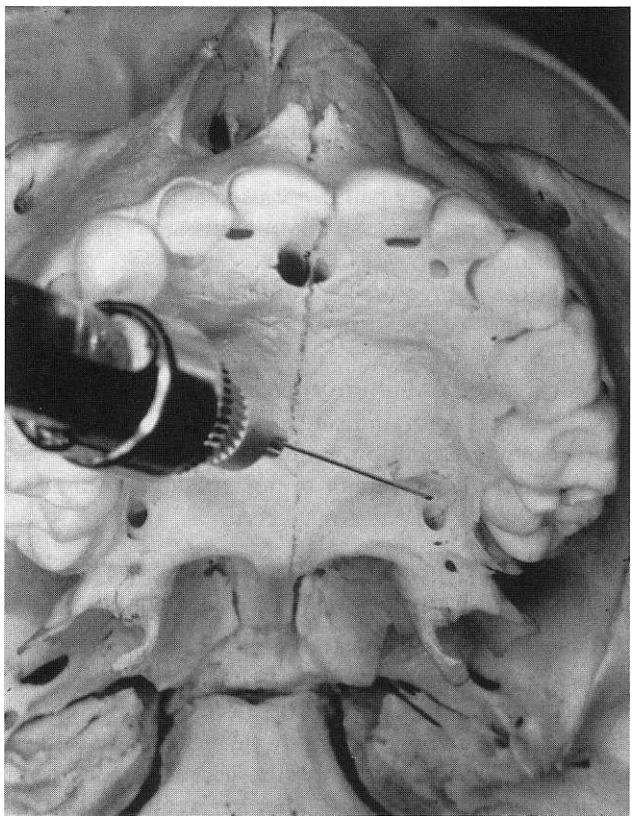


FIG 13-15. The needle is inserted approximately 10 mm posterior to the distal surface of the second primary molar.

the branches of the anterior and middle superior alveolar nerves. It also affects innervation of the soft tissues below the eye, half of the nose, and the oral musculature of the upper lip on the injected side of the face. This leaves the child with a feeling of numbness above the mouth similar to that below the mouth when an inferior alveolar nerve is blocked. In addition, there is temporary partial oral paralysis. These effects do not contraindicate use of the technique when it is truly needed. However, we find its use difficult to justify in routine operative and extraction procedures for teeth innervated by the anterior and middle superior alveolar nerves, since the supraperiosteal techniques are more localized and just as effective. The infraorbital block technique is preferred when impacted teeth (especially canines or first premolars) or large cysts are to be removed, when moderate inflammation or infection contraindicates use of the supraperiosteal injection site, or when longer duration or a greater area of anesthesia is needed.

The mental nerve block leaves the patient with essentially the same feelings of numbness as the inferior alveolar nerve block. Blocking the mental nerve anesthetizes all mandibular teeth in the quadrant except the permanent molars. Thus the mental nerve block makes it possible to perform routine operative procedures on all primary teeth without discomfort to the patient. However, we believe that the inferior alveolar nerve block should be favored unless there is a specific contraindication to its use at the inferior alveolar nerve injection site. The mental nerve block is no more comfortable for the patient, and the technique puts the syringe in clear view of the patient, whereas the inferior alveolar nerve block may be performed with the syringe out of the child's direct vision.

The reader is referred to the textbooks by Jorgensen and Hayden' and by Malamed[6] listed at the end of this chapter for more detailed information concerning the infraorbital block, the mental block, and other local anesthetic techniques.

PERIODONTAL LIGAMENT INJECTION (INTRALIGAMENTARY INJECTION)

The periodontal ligament injection has been used for many years as an adjunctive method of obtaining more complete anesthesia when supraperiosteal or block techniques failed to provide adequate anesthesia. This technique has also gained credibility as a good method of obtaining primary anesthesia for one or two teeth.

The technique is simple, requires only small quantities of anesthetic solution, and produces anesthesia almost instantly. The needle is placed in the gingival sulcus, usually on the mesial surface, and advanced along the root surface until resistance is met. Then approximately 0.2 ml of anesthetic is deposited into the periodontal ligament. For multirooted teeth, injections are made both mesially and distally. Considerable pressure is necessary to express the anesthetic solution.

A conventional dental syringe may be used for this technique. However, the great pressure required to express the anesthetic makes it desirable to use a syringe with a closed barrel to offer protection in the unlikely event that the anesthetic cartridge should break. Some syringes are equipped with a metal or Teflon sleeve that encloses the cartridge and provides the necessary protection should breakage occur.

Syringes designed specifically for the periodontal ligament injection technique have been developed. One syringe, the Peri-Press, is designed with a lever-action " trigger" that enables the dentist to deliver the necessary injection pressure conveniently. The Peri-Press syringe has a solid metal barrel and is calibrated to deliver 0.14 ml of anesthetic solution each time the trigger is completely activated.

There are some possible psychologic disadvantages to use of the periodontal ligament injection technique, especially for the inexperienced pediatric patient. The technique provides the patient with an opportunity to see the syringe and to watch the administration of the anesthetic. This may not be a significant problem for the experienced, well-adjusted dental patient, but it may contribute to the anxiety reaction of the new or anxiety-prone patient. In addition, the very design of the Peri-Press (which resembles a handgun) probably has some adverse psychologic effects.

There are two types of syringes designed specifically for intraligamentary injections. One is gunlike and the other is penlike. They both have the additional disadvantage of being quite expensive compared to a good, conventional aspirating syringes. The penlike syringe would be preferred in pediatric dentistry, but unfortunately it is even more expensive than the gunlike instrument.

Nevertheless, the periodontal ligament injection technique seems to offer a valuable adjunctive method of achieving dental anesthesia.

Malamed has reported a clinical study in which impressive results were obtained for certain procedures when the periodontal ligament injection technique was used.[75] The sample size was small for some procedures, and he pointed out that additional research was warranted. However, seven periodontal procedures (curettage and root planing) were performed with 100% effective anesthesia, and two teeth were extracted with 100% effective anesthesia (injections were administered to the mesial, distal, buccal, and lingual areas for these procedures). Seventy-one routine restorative procedures were performed under periodontal ligament anesthesia, with 91.5% effectiveness. The technique proved 66.6% effective for crown preparation procedures on 12 teeth, and for eight endodontic procedures adequate anesthetization was achieved only 50% of the time. Several different anesthetics were used, with and without vasoconstrictors, yet there seemed to be little difference in success rates or duration of pulpal anesthesia with the various agents. Because of the confined space and the limited blood circulation at the injection site for the periodontal ligament technique, the use of vasoconstrictors as an additive to the anesthetic solution may not be warranted. In fact, vasoconstrictors might conceivably contribute to ischemia of the periodontal ligament, which could at least add to localized postoperative discomfort or possibly cause more serious damage to the periodontal ligament. Walton and Abbott have also reported a clinical evaluation of the technique that showed a 92% success rate."

The periodontal ligament injection offers the following advantages for either primary or adjunctive anesthesia:

1. It provides reliable pain control rapidly and easily.
2. It provides pulpal anesthesia for 30 to 45 minutes, long enough for many single-tooth procedures without an extended period of postoperative anesthesia.
3. It is no more uncomfortable than other local anesthesia techniques.
4. It is completely painless if used adjunctively.
5. It requires very small quantities of anesthetic solution.
6. It does not require aspiration before injection.
7. It may be performed without removal of the rubber dam.
8. It may be useful in patients with bleeding disorders that contraindicate use of other injections.
9. It may be useful in young or disabled patients in whom the possibility of postoperative trauma to the lips or tongue is a concern.

INTRAOSSEOUS INJECTION, INTERSEPTAL INJECTION, AND INTRAPULPAL INJECTION

Intraosseous, interseptal, and intrapulpal injection techniques have been known for many years, but they have recently received renewed attention. The intrapulpal injection is an adjunctive anesthesia technique designed to obtain profound pulpal anesthesia during direct pulp therapy when other local anesthesia attempts have failed. The intrapulpal injection often provides the desired anesthesia, but the technique has the disadvantage of being painful initially, although the onset of anesthesia is usually rapid.

Intraosseous injection techniques (of which the interseptal injection is one type) require the deposition of local anesthetic solution in the porous alveolar bone. One may do this by forcing a needle through the cortical plate and into the cancellous alveolar bone, or a small,

*University Dental Implements, Fanwood, N.J.

TABLE 13-1 Maximum Recommended Doses of Local Anesthetics

DRUG	PROPRIETARY NAME	PERCENT OF LOCAL ANESTHETIC	VASOCONSTRICTOR	DURATION OF ANESTHETIC	MAXIMUM RECOMMENDED DOSE (MALAMED)
Lidocaine	Xylocaine	2	Epinephrine 1:100,000	Pulpal: 60 min Soft tissue: 3-5 hr	4.4 mg/kg
Mepivacaine	Carbocaine	3		Pulpal: 20-40 min Soft tissue: 2-3 hr	4.4 mg/kg
Prilocaine	Citanest Forte	4	Epinephrine 1:200,000	Pulpal: 60-90 min Soft tissue: 3-8 hr	6.0 mg/kg
Bupivacaine	Marcaine HCl	**0.5**	Epinephrine **1:200,000**	Pulpal: 90-180 min Soft tissue: 4-9 hr	1.3 mg/kg

Adapted from Malamed SF: *Handbook of local anesthesia,* ed 4, St Louis, 1997, Mosby.

round bur may be used to make an access in the bone for the needle. A small, reinforced intraosseous needle may be used to penetrate the cortical plate more easily. This procedure is not particularly difficult in children because they have less dense cortical bone than adults. The intraosseous techniques have been advocated for both primary anesthesia and adjunctive anesthesia when other local injections have failed to produce adequate anesthesia. These techniques have been reported by Lilienthal to produce profound anesthesia.' They do not seem to offer any advantages over the periodontal ligament injection except when use of the latter is contraindicated by infection in the periodontal ligament space.

COMPUTER-CONTROLLED LOCAL ANESTHETIC DELIVERY SYSTEM (WAND)

Reports by Friedman and Hochman' and by Krochak and Friedman" emphasize the advantages of a computer-controlled local anesthetic delivery system known as the Wand.* The system includes a conventional local anesthetic needle and a disposable, wandlike syringe held by a pen grasp when used for oral local anesthetic injections. A microprocessor with a foot control regulates the delivery of anesthetic solution through the syringe at a precision-metered flow rate, constant pressure, and controlled volume. The system includes an aspiration cycle for use when necessary. Block, infiltration, palatal, and periodontal ligament injections are all reported to be more comfortable for the patient with the Wand than with conventional injection techniques. In a randomized clinical trial comparing the Wand with the traditional anesthetic delivery system, Allen et al demonstrated that use of the Wand led to significantly fewer disruptive behaviors ($p < 0.1$) in preschool-aged children.[20] None of the preschool-aged children exposed to the Wand required restraint during the initial interval while nearly half of the children receiving a traditional injection required some type of immediate restraint.

'"Milestone Scientific, Livingston, N.J.

COMPLICATIONS AFTER A LOCAL ANESTHETIC

ANESTHETIC TOXICITY

Systemic toxic reactions from the anesthetics are rarely observed in adults. However, young children are more likely to experience toxic reactions because of their lower body weight. Young children are also often sedated with pharmacologic agents before the treatment. The potential for toxic reactions increases when local anesthetics are used in conjunction with sedation medications. Aubuchon found a direct lineal relationship between the number of cartridges of local anesthetic administered and the frequency of severe reactions.[21] It is most important for dentists who treat children to be acutely aware of the maximum recommended dosages of the anesthetic agents they use, because allowable dosages are based on the patient's weight (Table 13-1). For example, the toxic dose of lidocaine would be attained if hardly more than P/2 cartridges (3 ml) of 2% lidocaine with 1:100,000 epinephrine were injected at one time in a patient weighing 14 kg (30 lb). Yet $5^1/_2$ cartridges of the same anesthetic agent would be required to reach the toxic level in an adolescent patient weighing 46 kg (100 lb).

Because there is the possibility of toxic reaction to local anesthetic in some children, Wilson et al have studied and reported on the clinical effectiveness of 1% and 2% lidocaine.[22] They found that 1% and 2% lidocaine were equally effective when performing minor procedures on primary molars.-[2] The 1% lidocaine had a slightly lower effectiveness for major procedures, including pulpotomies and extractions.

TRAUMA TO SOFT TISSUE

Parents of children who receive regional local anesthesia in the dental office should be warned that the soft tissue in the area will be without sensation for a period



[I G **13-16. A,** Child who has chewed his lip after an inferior alveolar nerve block for operative procedures. B, Twenty-four hours after the initial trauma, a large ulcerated area is evident.

of 1 hour or more. These children should be observed carefully so that they will not purposely or inadvertently bite the tissue. Children who receive an inferior alveolar injection for routine operative procedures may bite the lip, tongue, or inner surface of the cheek. Sometimes a parent calls the dentist's office an hour or two after a dental appointment to report an injury to the child's oral mucous membrane. The parent may wonder if the accident occurred during the dental appointment; in all probability the child has chewed the area, and the result 24 hours later is an ulceration, often termed a *traumatic ulcer* (Fig. 13-16). Complications after a self-inflicted injury of this type are rare. However, the child should be seen in 24 hours, and a warm saline mouth rinse is helpful in keeping the area clean.

In a prospective study, College et al evaluated unilateral versus bilateral mandibular nerve block anesthesia with regard to postoperative soft tissue trauma and other complications in a pediatric population.[23] Their results showed that after unilateral and bilateral blocks, 13% of patients experienced postoperative soft tissue trauma, with the younger patients (less than 4 years of age) experiencing over twice as many problems as the older patients (over 12 years of age). Interestingly, the study showed that, in the group younger than age 4, patients receiving the unilateral nerve blocks had a statistically significantly higher incidence of trauma than the patients receiving the bilateral nerve blocks (35% versus 5%). Although the use of bilateral mandibular nerve blocks has been discouraged in the past, College et al concluded that there is no contraindication to the use of bilateral mandibular block anesthesia in pediatric patients.

ELECTRONIC DENTAL ANESTHESIA

Although an excellent record of effective and safe local anesthesia has been achieved in dentistry and medicine by injection, it is common knowledge that many children and adults have acquired a significant aversion to any form of therapy requiring injection with a needle. This aversion has prompted the investment of much time, energy, and resources in the search for effective and practical local anesthesia techniques that do not require the use of needles. During the late twentieth century, renewed interest in electronic dental anesthesia (EDA) as a viable alternative to conventional local injections occurred. At least six clinical studies have been completed to evaluate the efficacy of EDA in children [2a-29] In general, these studies show that EDA can produce sufficient oral local anesthesia and analgesia to comfortably allow many invasive dental procedures in children. However, the procedures cannot be performed as efficiently, and the effectiveness of anesthesia is not consistently as reliable as with conventional techniques. Further research on EDA and the refinement of EDA delivery systems are required before EDA can be recommended as a superior substitute for conventional procedures.

ANALGESICS

In addition to local administration of anesthetics to aid in pain control, occasionally systemic administration of analgesics is necessary to help control pain. These analgesics may be needed in instances of moderate to severe pain associated with trauma or infectious processes such as abscessed teeth, or they may be administered preoperatively or postoperatively in association with a dental procedure that may cause pain for the child. The rationale for preoperative administration of analgesics draws on the theory that giving the drug before the procedure provides effective analgesia because it precedes the inflammatory response and subsequent pain incurred during the operative procedure. This theory is not without controversy, however, and there are conflicting reports within the literature as to the efficacy of this technique.

TABLE 13-2 Medications and Dosages for Oral Pediatric Postoperative Pain Management

MEDICATION	AVAILABILITY	DOSAGE	40-LB CHILD	80-LB CHILD
Acetaminophen	Elixir: 160 mg/5 ml Tablets: 325 mg tab Chewable: 160 mg	10-15 mg/kg/dose given at 4-6 hr intervals	160 mg = 1 tsp 160 mg = 1 chewable	**325** mg = 1 tablet 320 mg = 2 chewables
Ibuprofen	Suspension: 100 mg/5 ml Tablets: 200, 300, 400, **600, 800** mg	4-10 mg/kg/dose given at **6-8** hr intervals	100 mg = 1 tsp	**200** mg = 2 tsp 200 mg = 1 tablet
Tramadol	Tablets: **50, 100** mg	1-2 mg/kg/dose given at 4-6 hr intervals maximum 100 mg	25 mg = 1/2 tablet	**50** mg = 1 tablet
Codeine and acetaminophen	Suspension: 12 mg/5 ml	0.5-1.0 mg codeine/kg/dose given at 4-6 hr intervals	12 mg = 1 tsp	24 mg = 2 tsp
Meperidine	Syrup: 50 mg/5 ml Tablets: 50 mg, 100 mg	1-2 mg/kg/dose given at 4-6 hr intervals	25 mg = 1/2 tsp	50 mg = 1 tsp

Adapted from Tate *AR, Acs G: Dent Clin North Am* 46:707-717, 2002.

Tate and Acs suggest that the selection and dosages of analgesics vary because of the change in body weight and composition that occur throughout childhood.[30] Of course, the first choice in most cases is the least potent analgesic with the fewest side effects. Table 13-2, adapted from Tate and Acs, shows common pediatric pain management agents and their appropriate dosage schedules.[30] Tate and Acs suggest that only rarely does the recommended dosage of acetaminophen or non-steroidal antiinflammatory drugs fail to control the dental pain. In the rare cases in which acetaminophen or nonsteroidal antiinflammatory drugs are not sufficient to manage the pain, the combination of codeine and acetaminophen provides the needed pain relief. Finally, in cases of severe pain in which codeine and acetaminophen are not effective, meperidine may be indicated.

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CHAPTER

14

Pharmacologic Management of Patient Behavior

MURRAY DOCK

ROBERT L. CREEDON

انتشارات
شایان نمودار

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FUNDAMENTAL CONCEPTS

To perform the highest quality dental care for the pediatric patient, the practitioner may need to use pharmacologic means to obtain a quiescent, cooperative patient. Techniques that use drugs to induce a cooperative yet conscious state in an otherwise uncooperative child are most commonly referred to as techniques of conscious sedation.

The goals of conscious sedation for the pediatric patient are (1) to facilitate the provision of quality care, (2) to minimize the extremes of disruptive behavior, (3) to promote a positive psychologic response to treatment, (4) to promote patient welfare and safety, and (5) to return the patient to a physiologic state in which safe discharge, as determined by recognized criteria, is possible. Conscious sedation techniques produce in a patient a minimally depressed state or level of consciousness in which the patient retains the ability to maintain a patent airway independently and continuously and is able to respond appropriately to physical stimulation and/or verbal commands. This means that the patient can in some way purposefully acknowledge a request to move or open the eyes, or react to an uncomfortable applied stimulus. All reflexes are essentially intact. If the patient is incapable of response by virtue of being very young or severely disabled, one should exercise care not to depress that particular patient to a point where such a determination is difficult to make. The techniques and drugs used to produce this state should possess a margin of safety of ample width to preclude unintended loss of consciousness.

In contrast to this awake state, deep sedation and general anesthesia are defined as conditions of the patient characterized by an incomplete, partial, or total loss of protective reflexes. In addition, there may be a partial or complete loss of the ability to independently and continuously maintain a patent airway. Under these circumstances, the patient is noninteractive and does not respond purposefully to physical stimulation or verbal command.

Occasionally the sedated patient will drift into normal sleep. When this occurs, it becomes the practitioner's responsibility to be assured that what is being observed is a normal sleep state by frequently arousing the patient. This may prove to be somewhat frustrating, because the objective is to produce a patient who is totally cooperative and the sleeping patient is just that.

Often, deep sedation and general anesthesia are differentiated; however, for the purposes of describing what is required to manage the patient in this condition in terms of training, monitoring, facilities, and personnel, the two conditions are treated as a single physiologic state of the patient. The requirements for management of the unconscious patient are much more stringent and address a greater concern for the safety of the patient.

A caution for the practitioner should be to avoid thinking of the patient's state in terms of the techniques employed rather than the level of consciousness. Historically, many sedation techniques have been defined by the route of administration. It is not the technique of administration but the drugs or agents employed that produce the effect observed in the patient. For example, one can produce an unconscious or fully anesthetized patient using the oral route.

Several requisites should be satisfied when the use of conscious sedation for pediatric dental patients is considered. They are the following:

1. The practitioner should possess a thorough knowledge of the agent or agents to be used and should be formally trained in the proper method of their administration. When sedating pediatric patients, practitioners must adhere to the guidelines published by the American Dental Association as well as by the American Academy of Pediatric Dentistry for the use of conscious sedation, deep sedation, and general anesthesia."[2] It is unwise and no longer acceptable to learn conscious sedation by experimenting in one's own practice setting.
2. There should be a carefully planned and documented rationale for the use of sedation for each patient. The decision should be made based on a careful analysis of the behavioral profile of the patient, the nature and extent of the treatment required, the risk-to-benefit ratio with regard to the physical status of the patient, the capability of the family to meet the demands of an extensive treatment plan, and the economic feasibility of alternative choices.
3. The patient should be carefully evaluated to ensure that no condition exists that might alter the expected response to the sedative agent or technique and pose added risk to the patient.
4. There should be a well-documented informed consent. No sedation technique should be attempted unless the parent or guardian has participated in a thorough consultation on the alternatives to sedation and foreseeable risks, knows what is expected in the postsedation period, and willingly agrees to proceed.
5. The office facilities should be such that a comfortable experience can be assured. There should be no physical barriers or lack of proper equipment to complicate the management of any emergency that might arise. Personnel should be thoroughly trained in techniques of monitoring a sedated

TABLE 14-1 Vital Signs at Various Ages

AGE (YR)	HEART RATE (BEATS/MIN)	BLOOD PRESSURE MM/HG	RESPIRATORY RATE (BREATHS/MIN)
1-3	70-110	90-105/55-70	20-30
3-6	65-110	95-110/60-75	20-25
6-12	60-95	100-120/60-75	14-22
12	55-85	110-135/65-85	12-18

Adapted from Behrman, et al: *Nelson textbook of pediatrics,* ed 17, Philadelphia, 2004, Elsevier Science.

patient. These personnel should be trained and frequently rehearsed in patient resuscitation.

6. Mobile emergency medical services should be readily available. The capabilities of these facilities and their personnel should be appraised and their response time to the office known before reliance on them is considered a part of the emergency protocol.

ANATOMIC AND PHYSIOLOGIC DIFFERENCES

When a regimen of conscious sedation for the pediatric patient is prepared, it is important to consider the differences between the adult patient and one in the pediatric age group. Differences in size, weight, and age as a measure of maturation of systems are obvious. Less obvious, but equally important, are the differences in basal metabolic rate between the adult and pediatric patient. Basal metabolic activity is greater in children, which ultimately affects not only drug response but also important physiologic parameters as well. Because oxygen demand is greater but the alveolar system is less mature, the respiratory rate is far higher in children than in adults (Table 14-1). This is an important consideration when drugs that depress the respiratory system are administered.

Airway management requires different consideration in the pediatric patient because of anatomic variations. The narrow nasal passages and glottis, combined with hypertrophic tonsils and adenoids, enlarged tongue, and greater secretions, produce a much greater risk of airway obstruction. The airway of all patients should be examined prior to sedation (Fig. 14-1). Patients with tonsillar tissue that occupies more than 50% of the pharyngeal space are at increased risk of respiratory obstruction, and alternative treatment options should be considered.' Children demonstrate a reduced tolerance to respiratory obstruction. Thus, sudden apnea is a greater concern in the pediatric age group. Because the thorax is smaller, with less expansion capability, children have less functional reserve. Consequently they are more prone to rapid desaturation on obstruction or respiratory depression. For this reason children with sleep apnea are not good candidates for conscious sedation.

Cardiovascular parameters are different for children. The heart rate is faster and blood pressure is lower than in the adult. Children are more susceptible to bradycardia, decreased cardiac output, and hypotension. Unlike in the adult population, heart rate is the primary determinate of blood pressure in children. Compensatory mechanisms to maintain adequate blood pressure when the heart rate is depressed are not as well developed in children. Thus a decrease in heart rate leads to a corresponding decrease in blood pressure and tissue oxygenation. This concept must be well appreciated when giving drugs that depress the heart rate in the pediatric age population.

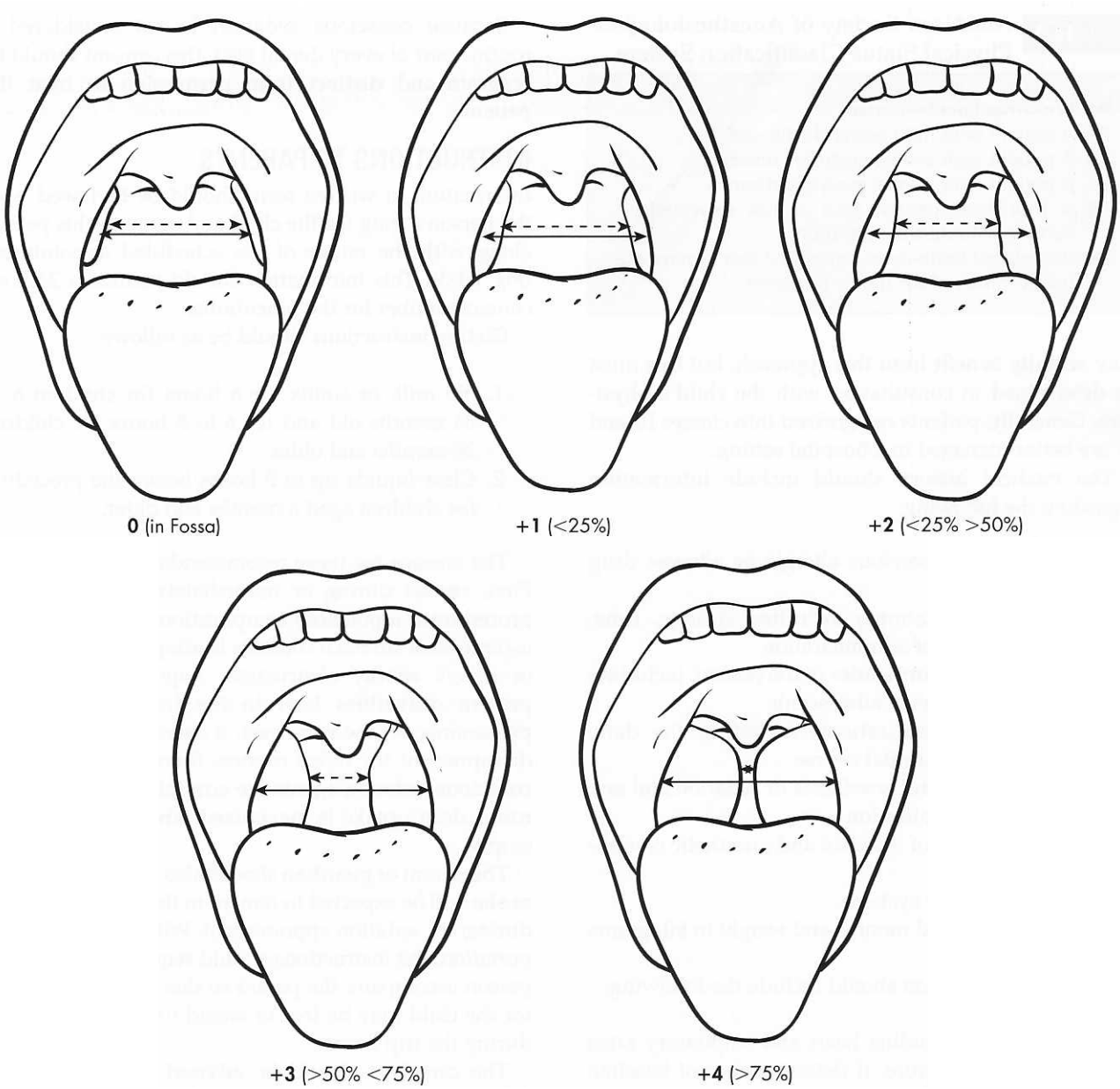
The effect and duration of action of drugs is much more variable in children. For agents that are more lipophilic retention may be prolonged, especially in children who are obese. For some types of patients, drug metabolism may be increased. Because of better peripheral perfusion in children, the onset of intramuscularly administered drugs may be more rapid.

The anatomic and physiologic differences between children at different age levels and adults lead one to the conclusion that dosage is not simply an application of a formula for derivation of a percentage of the adult dosage of any agent. Drug dosages for children should be carefully individualized for each patient following established guidelines.

PATIENT SELECTION AND PREPARATION

INDICATIONS

Because conscious sedation embodies a group of techniques designed to alter patient behavior, the practitioner should have a rationale for making the choice as to which patients will most likely benefit from their use. The indiscriminate application of these techniques to all patients should be avoided. Several behavioral or anxiety-assessment profiles have been developed that can be of great help to the practitioner as the various techniques are introduced into a practice.[4,5] As one gains experience, this decision becomes more one of clinical judgment and intuition as to which approach produces the most successful results for specific types of patients for that individual practitioner. No one technique or agent, or combination of agents, should be expected to



f **I G . 14-1.** Standardized system for evaluation of tonsillar size. Classification of tonsil size should be completed for all patients prior to sedation. Patients classified as +3 or greater (having more than 50% of the pharyngeal area occupied by tonsils) are at increased risk of developing airway obstruction. *(From Brodsky L:* Pediatr Clin North Am *36:1551-1569, 1989; and Cote CJ et al.* A practice of anesthesia for infants and children, *Philadelphia, 1993, WB Saunders, pp 313-314.)*

be successful every time. There are also degrees of success, and total immobilization and near unconsciousness of a patient should not necessarily be equated with the most successful sedation. One should choose the agent and technique that best fits the patient type as well as the nature of what needs to be accomplished.

A thorough medical history is required to determine if a patient is suitable for sedative procedures. This, along with a recent physical examination, constitute a risk assessment or physiologic status evaluation. This health evaluation should be used to place the patient in one of the categories set forth by the American Society of Anesthesiologists (http://www.asahq.org/clinical/physicalstatus.htm) and should be documented in the record (Box 14-1).

Only patients who are categorized into ASA class I are routinely acceptable as candidates for conscious sedation. Some children assigned to ASA class II or III

BBR 14-1 American Society of Anesthesiologists' Physical Status Classification System

P1 A normal healthy patient
P2 A patient with mild systemic disease
P3 A patient with severe systemic disease
P4 A patient with severe systemic disease
P5 A moribund patient who is not expected to survive without the operation
P6 A declared brain-dead patient whose organs are being removed for donor purposes

may actually benefit from this approach, but this must be determined in consultation with the child's physician. Generally, patients categorized into classes III and IV are better managed in a hospital setting.

The medical history should include information regarding the following:

1. Allergies and previous allergic or adverse drug reactions
2. Current medications, including dosage, time, route, and site of administration
3. Diseases or abnormalities in the patient, including pregnancy status of adolescents
4. Previous hospitalizations, including the date, purpose, and hospital course
5. History of general anesthesia or sedation and any associated complication
6. Family history of diseases and anesthetic complications
7. Review of body systems
8. Age in years and months and weight in kilograms

The physical evaluation should include the following:

1. Vital signs, including heart and respiratory rates and blood pressure. If determination of baseline vital signs is prevented by the patient's physical resistance or emotional condition, the reason(s) should be documented.
2. Evaluation of airway patency.
3. ASA classification (see Box 14-1).

INFORMED CONSENT

The parent or legal guardian must be agreeable to the use of conscious sedation for the child. These individuals are entitled to receive complete information regarding the reasonably foreseeable risks and the benefits associated with the particular technique and agents being used, as well as any alternative methods available. Therefore the explanation should be in clear, concise terms that are familiar to them (Fig. 14-2). The consent form can be on or part of a sedation record with space provided for the signatures of all parties.

Because conscious sedation is not considered a routine part of every dental visit, this consent should be separate and distinct from permission to treat the patient.

INSTRUCTIONS TO PARENTS

Information in written form should be reviewed with the person caring for the child and given to this person along with the notice of the scheduled appointment (Fig. 14-3). This information should include a 24-hour contact number for the practitioner.

Dietary instructions should be as follows:

1. No milk or solids for 6 hours for children 6 to 36 months old and for 6 to 8 hours for children 36 months and older
2. Clear liquids up to 3 hours before the procedure for children aged 6 months and older.

The reasons for these recommendations are twofold. First, emesis during or immediately after a sedative procedure is a potential complication that can result in aspiration of stomach contents leading to laryngospasm or severe airway obstruction. Aspiration may even present difficulties later in the form of aspiration pneumonia. At the very least, it creates an unfavorable disruption of the office routine. Second, because most conscious sedation agents are administered by the oral route, drug uptake is maximized when the stomach is empty.

The parent or guardian should also be advised that he or she will be expected to remain in the area of the office during the sedation appointment. With regard to transportation, the instructions should request that a second person accompany the parent so that the person caring for the child may be free to attend to the child's needs during the trip home.

The caregiver should be advised that, on arriving home, the child may sleep for several hours and may be drowsy and irritable for up to 24 hours after the sedation. It is important to stress the need for frequent observation if the child is sleeping, to ensure an open airway. Activity should be restricted to quieter pursuits and closely supervised for the remainder of the day.

Following treatment the child should first be offered clear liquids and may advance to solid foods as tolerated. Once solids are tolerated, there are no dietary restrictions other than those imposed as a result of the dental procedure performed. Nausea and vomiting may occur, especially when narcotics have been used, which may thus prolong the delay before beginning solid food intake. In this event, special attention should be paid to the fluid intake to ensure adequate hydration.

Knowledge on the part of the parent of what to expect is the most reliable way to ensure a calm, comfortable,

CONSENT FOR THE USE OF SEDATION OR GENERAL ANESTHESIA FOR PEDIATRIC DENTAL TREATMENT

I _____, as the legally responsible parent or guardian of _____, give my consent to the use of local anesthetics, sedative drugs, or general anesthetic agents that Dr(s). _____ may deem necessary on the child's examination chart, as previously explained to me, and any other procedure deemed necessary or advisable as a corollary to the planned treatment for _____, except for: (if none, so state) _____.

I have been informed and understand that occasionally there are complications of the treatment, drugs, or anesthetic agents, including but not limited to: numbness, infection, swelling, bleeding, discoloration, nausea, vomiting, allergic reactions, brain damage, stroke, or heart attack. I further understand and accept that complications may require hospitalization and may even result in death.

Dr(s). _____ discussed with me, to my satisfaction, these complications. I acknowledge the receipt of and understand the preoperative and postoperative instructions. The treatment and sedation or anesthesia procedures have been explained to me, to my satisfaction, along with possible alternative methods and their advantages and disadvantages, risks, consequences, and probable effectiveness of each, as well as the prognosis if no treatment is provided.

I have read this consent and understand, to my satisfaction, the procedures to be performed and accept the possible risks.

Legally responsible parent or guardian: _____ Date: _____

Address: _____

Witness: _____

I certify that I explained the above procedures to the parent or guardian before requesting his or her signature.

_____ Date: _____

Signature of dentist

f**IG 14-2.** Example of form for informed consent. *(Courtesy Dr. Kenneth C. Troutman)*

and uncomplicated postsedation period. Therefore these instructions and recommendations should be in written form and should be reviewed again with the person responsible for the patient and given to this person at the time of discharge from the office (Fig. 14-4).

DOCUMENTATION

Meticulous and accurate documentation of the sedation experience is imperative. In the event of an adverse reaction, the best insurance is an accurate, clear, continuous, documented account of what occurred before, during, and after the encounter.

Preprocedural records should document (1) proper adherence to food and liquid intake restrictions; (2) the preoperative health evaluation, including the patient's health history and a complete physical assessment along with the patient's current weight, age, and baseline vital signs; (3) name and address of the physician who usually cares for the child; (4) a note as to why the particular method of management was chosen; (5) the presence of informed consent; and (6) the delivery of instructions to the caregiver.

Intraoperatively the appropriate vital signs should be recorded as they are assessed (Table 14-2). Timed notations regarding the patient's appearance should be included. The type of drug, the dose given, and the route, site, and time of administration should be clearly indicated. If a prescription is used, either a copy of the prescription or a note as to what was prescribed should also be a part of the permanent record.

INSTRUCTIONS TO FOLLOW BEFORE YOUR CHILD'S SEDATION

EATING AND DRINKING

1. No milk or solid foods 6 hours before the sedation appointment.
2. Clear liquids such as water, clear juices, gelatin, Popsicles, or broth, may be given up to 3 hours before the appointment.
3. Let everyone in the home know the above information, because siblings or others living in the home often unknowingly feed the child.

ACTIVITY

1. Plan the child's sleep and awakening times to encourage the usual amount of sleep the day before the sedation appointment.
2. Please arrive on time for your scheduled appointment. This is a long appointment, and you may be here for several hours.
3. The legal guardian must accompany the child to the sedation appointment.
4. A second responsible adult must join you and your child at the time of discharge. This enables one adult to drive the car while the second adult focuses attention on your child after the treatment is completed. The child should be carefully secured in a car seat belt during transportation.
5. Make sure your child uses the restroom before the sedation.

ACTIVITY AFTER THE SEDATION

1. Your child may take a long nap. He/she may sleep from 3 to 8 hours and may be drowsy and irritable for up to 24 hours after sedation. When your child is asleep, you should be able to awaken him/her easily.
2. Your child may be unsteady when walking or crawling and will need support to protect him/her from injury. An adult must be with the child at all times until the child has returned to his/her usual state of alertness and coordination.
3. Closely supervise any activity for the remainder of the day.

CHANGE IN HEALTH

It is important that you notify the office of the development of a cold, cough, fever, or any illness within 14 days before the sedation appointment. For your child's safety, the sedation may need to be rescheduled.

f **I G 14-3.** Example of presedation instructions to the parent or caregiver.

After completion of treatment, the patient should be continuously observed in an appropriately equipped recovery area. The patient should remain under direct observation until respiratory and cardiovascular stability have been ensured. The patient should not be discharged until the presedation level of consciousness or a level as close as possible for that child has been achieved (Box 14-2). At the time of discharge, the condition of the patient should be noted.

SEDATION TECHNIQUES

A variety of methods are available for producing sedation or alteration of mood in the pediatric patient. These systemic procedures are based on the thoughtful use of various drugs that produce sedation as one of their principal effects, as well as the use of differing routes of administration. Sedative drugs may be administered by inhalation or by oral, rectal, submucosal, intramuscular, or intravenous routes. The use of combinations of drugs and specific selection of routes of administration to maximize effect and increase safety, as well as maximize patient acceptability, are common. Inhalation of a nitrous oxide—oxygen mixture is often coupled with administration of an agent by any of the other routes.

Another very practical and efficient technique is the timed sequencing of a combination of drugs. The only classification of sedative drugs acceptable for administration preprocedurally outside of the treatment facility is the minor tranquilizers (e.g., diazepam, hydroxyzine). Minor tranquilizers do not include chloral hydrate or narcotics.` These agents can be given well before the actual procedure by the parent at home, or on arrival at the office to calm the apprehensive patient. Doing so may aid in the administration of other agents as the procedure begins. This sequencing of drugs may in fact reduce the quantity of subsequently administered

CARE OF YOUR CHILD AFTER SEDATION

Today your child had dental treatment under conscious sedation.

He/she received the following medicine(s) for sedation:

☐ Chloral hydrate ☐ Meperidine (Demerol) ☐ Hydroxyzine (Vistaril)
☐ Diazepam (Valium) ☐ Midazolam ☐ Other_____

Children repond to sedation in their own way, but the following guidelines will help you know what to expect at home:

GOING HOME

1. Your child will not be able to walk well, so we suggest that you carry your child or use a wheelchair to transport your child to the car.
2. Young children must be restrained in a car safety seat and older children must be restrained with a seat belt during transportation.

ACTIVITY

1. Your child may take a long nap. He/she may sleep from 3 to 8 hours and may be drowsy and irritable for up to 24 hours after sedation. When your child is asleep, you should be able to awaken him/her easily.
2. Your child may be unsteady when walking or crawling and will need support to protect him/her from injury. An ADULT must be with the child at all times until the child has returned to his/her usual state of alertness and coordination.
3. Your child should not perform any potentially dangerous activities, such as riding a bike, playing outside, handling sharp objects, working with tools, or climbing stairs until he/she is back to his/her usual alertness and coordination for at least 1 hour.
4. We advise you to keep your child home from school or daycare after treatment and possibly the next day if your child is still drowsy or unable to walk well. Your child should return to his/her usual state of alertness and coordination within 24 hours.

EATING AND DRINKING INSTRUCTIONS

Begin by giving clear liquids such as clear juices, water, gelatin, Popsicles, or broth. If your child does not vomit after 30 minutes, you may continue with solid foods.

REASONS TO CALL THE DOCTOR

1. You are unable to arouse your child.
2. Your child is unable to eat or drink.
3. Your child experiences excessive vomiting or pain.
4. Your child develops a rash.

FOR THESE OR ANY OTHER CONCERNS about your child's sedation, please contact our office at_____.

f I G. **14-4.** Example of postsedation instructions to the parent or caregiver.

agents. In some instances, this preprocedural administration may be all that is required for a particular patient.

The selection of technique is often made as a matter of clinical judgment. It is a question of successfully matching a method to a specific objective.

The primary goal of these techniques is to produce a quiescent patient to ensure the best quality of care. Another goal might be to accomplish a more complex or lengthy treatment plan in a shorter period by lengthening appointment times and thereby reducing the number of repeat visits required. Children presenting with a dental injury or acute pain may require sedation for completion of treatment as well as postoperative analgesia. In fact, the reduction in anxiety may reduce the amount of analgesia required. Conscious sedation may also allow for more comfortable and acceptable treatment for physically impaired or cognitively disabled patients. Often these patients may benefit from parenteral sedation.

TABLE 14-2 Template of Definitions and Characteristics for Levels of Sedation and General Anesthesia

	FUNCTIONAL LEVEL OF SEDATION			
	CONSCIOUS SEDATION			DEEP SEDATION
	MILD SEDATION (ANXIOLYSIS)	INTERACTIVE	NONINTERACTIVE/AROUSABLE WITH MILD/MODERATE STIMULUS	NONINTERACTIVE/NON-AROUSABLE EXCEPT WITH INTENSE STIMULUS
	(Level 1)	**(Level 2)**	**(Level 3)**	**(Level 4)**
Goal	Decrease anxiety; facilitate coping skills	Decrease or eliminate anxiety; facilitate coping skills	Decrease or eliminate anxiety; facilitate coping skills; promote sleep	Eliminate anxiety; coping skills overridden
Responsiveness	Uninterrupted interactive ability; totally awake	Minimally depressed level of consciousness; eyes open or temporarily closed; responds appropriately to verbal commands	Moderately depressed level of consciousness; state mimics physiologic sleep (vital signs not different from those during sleep); eyes closed most of time; may or may not respond to verbal prompts alone; responds to mild/moderate stimuli (e.g., repeated trapezius pinching or needle insertion in oral tissues elicits reflex withdrawal and appropriate verbalization [complaint, moan, crying]); airway only occasionally may require readjustment via chin thrust	Deeply depressed level of consciousness; sleeplike state, but vital signs may be slightly depressed compared with physiologic sleep; eyes closed; does not respond to verbal prompts alone; reflex withdrawal with no verbalization when intense stimulus occurs (e.g., repeated, prolonged, and intense pinching of the trapezius); airway expected to require constant monitoring and frequent management
Personnel required	2	2	2	3
Monitoring equipment	Clinical observation*	PO; precordial recommended'	PO, precordial, BP; capno desirable'	PO and capno, precordial, BP, ECG; defibrillator desirable
Monitoring information	None	HR, RR, O_2 before, during (ql5min), after, as needed	HR, RR, O_2, BP; [CO_2] if available; before, during (g10min), after until stable/discharge criteria met	HR, RR, O_2, [CO_2], BP before, during (q5min), after until stable/discharge criteria met

American Academy of Pediatric Dentistry: Guidelines on the elective use of conscious sedation, deep sedation and general anesthesia in pediatric dental patients, *Pediatr Dent* 24(7):74-80, *2003.*

BP, Blood pressure; *cap»o,* capnography; ECG, electrocardiography; *HR,* heart rate; *PO,* pulse oximetry; *precordial,* precordial (pretracheal) stethoscope; *RR,* respiratory rate.

*It should be noted that clinical observation should accompany any level of sedation and general anesthesia.

"Recommended" and "desirable" should be interpreted as not a necessity but an adjunct in assessing patient status.

Although the presence of a compromising medical condition is generally a contraindication to sedation, some *patients in this category may in fact* benefit *from its use.*[6] This would, of course, include patients for whom stress reduction would reduce the likelihood of complication. These children should be managed in close cooperation with the physicians who regularly cares for them.

NITROUS OXIDE AND OXYGEN SEDATION

Eighty-five percent of pediatric dentists use nitrous oxide and oxygen for sedation of patients. This makes it the most frequently used sedative agent.

Nitrous oxide is a slightly sweet-smelling, colorless, inert gas. It is compressed in cylinders as a liquid that vaporizes on release. This is an endothermic reaction

BOX 14-2 Discharge Criteria

1. Cardiovascular function is satisfactory and stable.
2. Airway patency is uncompromised and satisfactory.
3. Patient is easily arousable and protective reflexes are intact.
4. State of hydration is adequate.
5. Patient can talk, if applicable.
6. Patient can sit unaided, if applicable.
7. Patient can ambulate, if applicable, with minimal assistance.
8. If the child is very young or disabled, incapable of the usually expected responses, the presedation level of responsiveness or the level as close as possible for that child has been achieved.
9. Responsible individual is available.

that pulls heat from the cylinder and environment; consequently the cylinder will become cool or even cold when in use. The gas is nonflammable but will support combustion.

Pharmacokinetics. Nitrous oxide is slightly heavier than air, with a specific gravity of 1.53, and has a blood to gas partition coefficient of 0.47. Because of its low solubility in blood, it has a very rapid onset and recovery time. Nitrous oxide will become saturated in blood within 3 to 5 minutes following administration and is physically dissolved in the serum fraction of the blood. There is no biotransformation, and the gas is rapidly excreted by the lungs when the concentration gradient is reversed. Very small amounts may be found in excreted body fluids and intestinal gas.

A phenomenon termed *diffusion hypoxia* may occur as the sedation is reversed at the termination of the procedure. The nitrous oxide escapes into the alveoli with such rapidity that the oxygen present becomes diluted; thus the oxygen–carbon dioxide exchange is disrupted and a period of hypoxia is created. However, this phenomenon is reported not to occur in healthy pediatric patients.' Nonetheless, to minimize this effect, the patient should be oxygenated for 3 to 5 minutes after a sedation procedure, if for no other reason than to allow for proper nasal hood evacuation of the exhaled gas.

Pharmacodynamics. Nitrous oxide produces nonspecific central nervous system (CNS) depression. Although it is classed with inhalational general anesthetics, it produces limited analgesia, and thus surgical anesthesia is unlikely unless concentrations producing anoxia are reached. Nitrous oxide is the weakest of all inhalation agents, with a minimum alveolar concentration of 105. The minimum alveolar concentration of an inhalation agent is a measure of its potency. It is the concentration required to produce immobility in 50% of patients. At concentrations between 30% and 50%, nitrous oxide will produce a relaxed, somnolent patient who may appear dissociated and easily susceptible to suggestion. Amnesia may occur in some patients, but there is little alteration of learning or memory. At concentrations greater than 60%, patients may experience discoordination, ataxia, giddiness, and increased sleepiness. It is not recommended that concentrations greater than 50% be used in dental practice.

Nitrous oxide reduces hypoxic-driven ventilation and has minimal effect on the hypercapnic respiratory drive. When used as a single agent, nitrous oxide will not cause hypoxemia. It should be avoided, however, in patients who rely significantly on hypoxia-driven ventilation, in whom exposure to high levels of oxygen can result in respiratory depression. When combined with other agents that depress respiration, nitrous oxide decreases the body's normal response to low oxygen tension. These effects are usually negligible, however, because of the high concentration of oxygen administered with the combination. Nitrous oxide slightly increases the respiratory minute volume. As the patient becomes more relaxed from the effects of nitrous oxide, the respiratory rate may decrease slightly. The gas is nonirritating to the respiratory tract and can be given to patients with asthma without fear of bronchospasm. Problems can arise, however, from the added respiratory effects when it is given in combination with narcotics or other CNS depressants.

Cardiac output is decreased and peripheral vascular resistance is increased when nitrous oxide is used. This is generally of insignificant degree and is a consideration only in patients with severe cardiac disease. The respiratory and cardiac effects may be secondary to the high concentration of oxygen administered in conjunction with nitrous oxide.

Adverse Effects and Toxicity. Nausea and/or vomiting is the most common adverse effect experienced with nitrous oxide sedation. The incidence increases significantly with concentrations in excess of 50%, with lengthy procedures, with rapid fluctuations in concentrations, and with rapid induction and reversal.

Nitrous oxide will become entrapped in gas-filled spaces such as the middle ear, sinuses, and the gastrointestinal tract. Middle ear pressure will increase significantly, and although it is of little significance in a patient with normal patency of eustachian tubes, it can induce pain in patients with acute otitis media. Because of this, use of nitrous oxide should be avoided in patients with acute otitis media.

Other contraindications include severe behavioral problems and emotional illness, uncooperativeness, fear of "gas," claustrophobia, maxillofacial deformities that prevent nasal hood placement, nasal obstruction

(e.g., upper respiratory infection, nasal polyps, deviated septum), chronic obstructive pulmonary disease, pregnancy, and situations in which high oxygenation is inadvisable (e.g., bleomycin therapy).

In clinical use for sedation at proper concentrations of medical-quality nitrous oxide and oxygen, the gas has no toxic effects. The greatest concern regarding toxicity centers on exposure of dental personnel to high ambient air levels of the gas for periods of time during its use for patient sedation. Chronic exposure to nitrous oxide, including recreational abuse, can over time produce neurotoxicity, impotence, and renal and/or liver toxicity. The rate of spontaneous abortion is known to be higher in operating room personnel and in the spouses of operating room personnel.' In addition, chronically exposed individuals may experience a decrease in fertility.' An increase in hepatic disease was found in dentists and dental personnel exposed to high levels of nitrous oxide for periods longer than 3 hours per week. Because of these facts, leakage from open systems, such as those used in the dental office, should be reduced as much as possible. This can be accomplished by limiting the amount of mouth breathing by the patient and using an efficient scavenging system. The installation of laminar air-flow systems might also be considered in new office constructions when it is known that considerable amounts of nitrous oxide will be in use. In addition, the office as well as office personnel should be periodically monitored for exposure to unscavenged nitrous oxide. A variety of units are available that use infrared spectrophotometry to measure ambient levels. These machines can detect levels as low as 1 ppm and are particularly useful for uncovering leaks around tanks and flowmeters. A less costly and more practical approach is the use of dosimetry badges that are worn by office personnel when using nitrous oxide. These units are generally worn for an 8-hour period and report exposure as time-weighted averages. Exposure-limiting methods are listed in Box 14-3.

Equipment. Several manufacturers produce machines for the safe delivery of nitrous oxide–oxygen mixtures for use in conscious sedation in the dental office. The machine should be of the continuous-flow design, with flowmeters capable of accurate regulation. A fail-safe mechanism that provides automatic shutdown if oxygen falls below 25% and audible and visual alarms that are activated by oxygen failure are important design features. There should be a flush lever for easy and immediate flushing of the system with 100% oxygen.

Mobile, self-contained units are available, as well as those operating from a central supply. A major safety consideration with either type is the presence of a good pin-indexed yoke system to absolutely prevent crossover of the cylinder hookup. One should be continuously aware of the danger of crossed lines or cylinders. Such crossover becomes possible when office renovations are done and when fittings wear with age and use.

An efficient scavenger system is an important component of any hose-mask system. The double-mask type is the most efficient type of scavenger (Fig. 14-5). These systems exhaust into the vacuum waste system, which should be vented to the outside to prevent dispersal of gases to other areas of the office or building. Nasal hoods should be of good design and should be available in pediatric and adult sizes to ensure adequate fit, which further reduces leakage (Fig. 14-6).

BOX 14-3 Controlling Ambient Nitrous Oxide Levels

VENTILATION

- Operatories must have good cross ventilation.
- Exhaust vents should preferably be located in the ceiling as close to the head of the chair as possible.
- Nitrous oxide exhaust must be vented to the outside.
- Room air exchanges of 10 or more per hour are recommended.

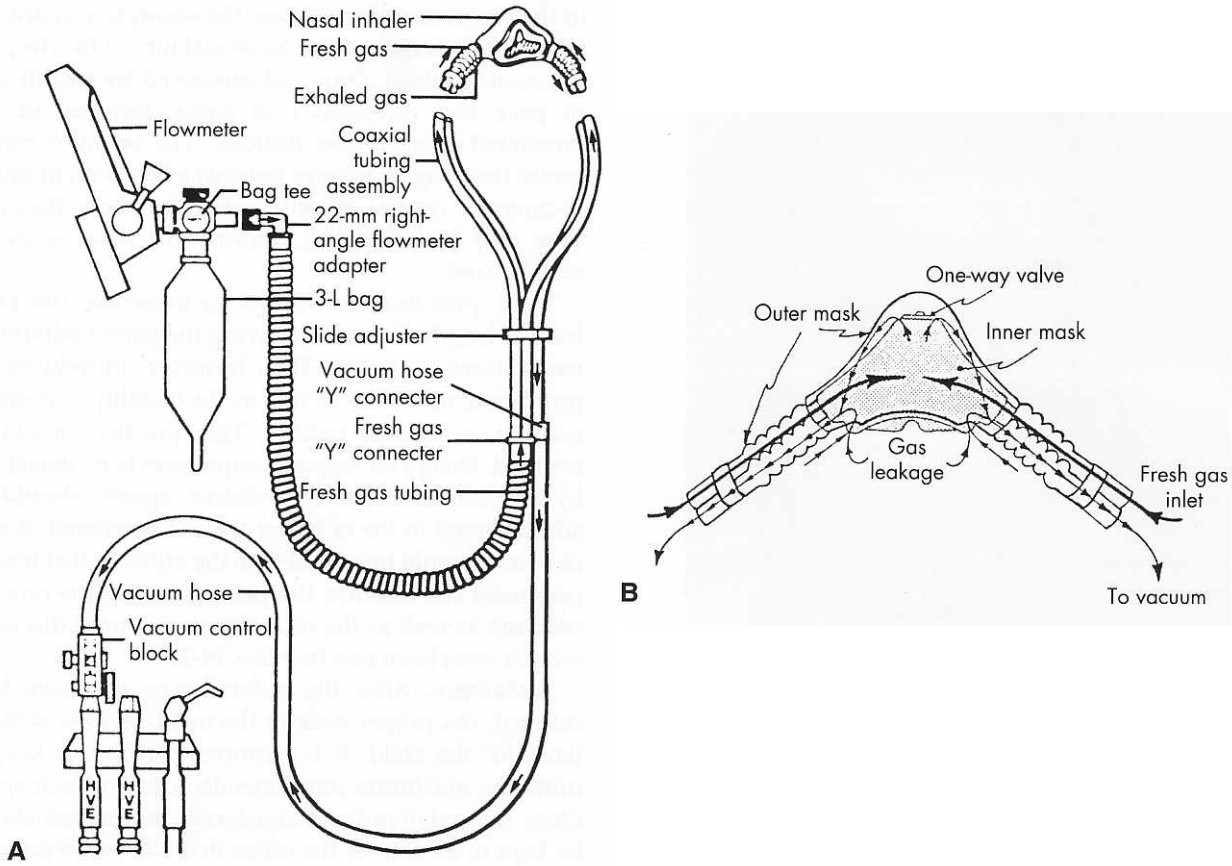
WORK PRACTICES

- Inspect equipment each day to ensure that all connectors are tight and that tubing and bags are free of holes and cracks.
- Always use a scavenger system when administering nitrous oxide and oxygen. Adjust the scavenger flow rate to 45 L/min.
- Select an appropriately sized mask to ensure a sealed but comfortable fit.
- Instruct patients to refrain from mouth breathing and talking during the procedure.
- Adjust the nitrous oxide and oxygen flow rate to keep the bag from overfilling. The bag should collapse and expand as the patient breathes. After administration, flush the patient and system by administering 100% oxygen to the patient for at least 5 minutes.

MAINTENANCE

- Schedule periodic inspections every 3 months of all aspects of the system, with particular attention to areas of potential leaks.
- Document results of inspections as well as all corrective actions taken.
- Ensure that repairs and modifications are performed only by authorized dealers.
- Consider periodic personal sampling of dental personnel with a dosimeter.

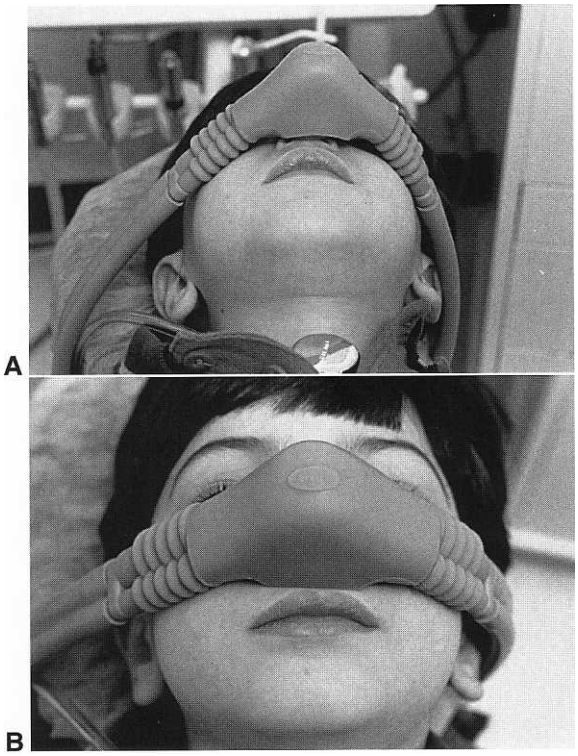
Adapted from American Dental Association Council on Scientific Affairs, Council on Dental Practice: *J Am Dent Assoc* 128:364-365, 1997; and Howard *1 R : JAm Dent Assoc* 128:356-360, 1997.



f **C 14-5. A,** Schematic drawing illustrating the components of a nitrous oxide-oxygen delivery and scavenging system. **B,** Components of the system's nasal hood. *(Courtesy Porter instrument Co., Hatfield, Pa.)*

Technique. After a thorough inspection of the equipment, the mask should be introduced to the patient with an explanation delivered at the appropriate level of understanding, and then the mask should be carefully placed over the nose. The delivery tubes are tightened behind the chair back in a comfortable position. The bag is filled with 100% oxygen and delivered to the patient for 2 or 3 minutes at an appropriate flow rate, typically between 4 and 6 L per minute. With an appropriate flow rate, slight movement of the mixing bag should be apparent with each inhalation and exhalation. With too high a flow rate, the bag will be overinflated, movement will not be seen with each breath, and leakage will occur from around the mask. In this instance, the flow rate should be adjusted downward. Too low a flow rate will deplete the bag of mixed gases. Once the proper flow rate is achieved, the nitrous oxide can be introduced by slowly increasing the concentration at increments of 10% to 20% to achieve the desired level. The operator should encourage the patient to breathe through the nose with the mouth closed. The sensations should be explained as they begin to be felt. They are best described as a floating, giddy feeling with tingling of the digits. The eyes will take on a distant gaze with sagging eyelids. When this state is reached, the local anesthetic may be given. Once this is completed, the concentration can be reduced to 30% nitrous oxide and 70% oxygen or lower. The patient can now be maintained and monitored, and the contemplated procedure carried out. The dentist should communicate with the patient throughout the procedure, paying particular attention to the maintenance of an open, relaxed airway. The level of nitrous oxide may be periodically reduced to determine the minimum level required for that patient. This should be recorded for future reference. An emesis basin should be readily available, and in the event that vomiting does occur, the head should be rotated to the side. However, the laryngeal reflex is not obtunded with nitrous oxide, and so aspiration of vomitus is unlikely.[r0]

Recovery can be achieved quickly by reverse titration. Once the sedation is reversed, the patient should be allowed to breathe 100% oxygen for 3 to 5 minutes. Oxygenation will purge the patient and the nitrous



[I [. **14-6.** A, Poorly fitting mask with leakage under nares. **B,** Well-fitting mask.

oxide system of residual gas. The patient should be allowed to remain in the sitting position for a brief period to ensure against dizziness on standing. The patient is now ready for discharge. Even though psychomotor effects return to normal within 5 to 15 minutes, it is not advisable to allow teenage patients to drive themselves home from the appointment.

ORAL SEDATION

By far the most universally accepted and easiest method of drug administration is the oral route. From the patient' s point of view, this is certainly true, particularly in pediatrics, because there is no discomfort. That is not to say that all patients will readily accept orally administered drugs. The taste may be quite objectionable, especially to very young children. This can usually be overcome when the drug is mixed with a palatable liquid. It has been suggested that, for children who object, a syringe be used to squirt the solution into the oral cavity. Caution is advised, however, since there are reported cases of aspiration of the drug with too forceful an effort. " Although complications can and do arise when drugs are administered by the oral route, their frequency and severity are usually reduced.

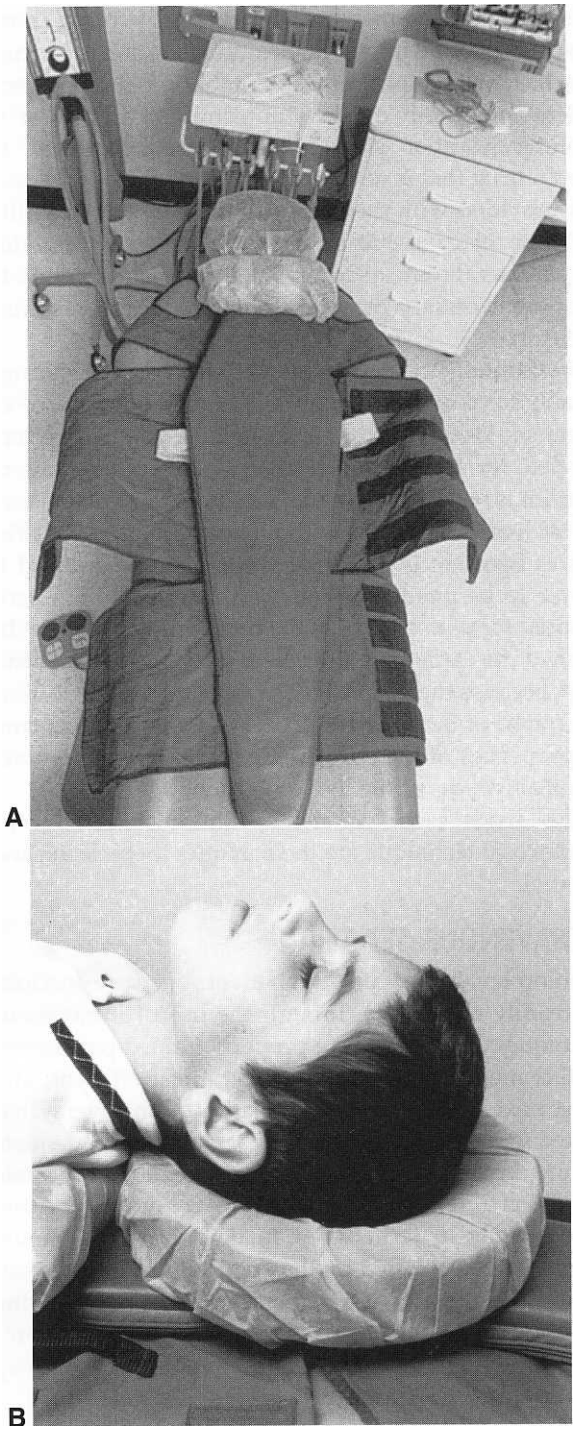
The oral route of administration is the most variable. The method is dependent on absorption through the gastrointestinal mucosa. This means that the condition of the stomach and intestines, the absorption characteristics of the drug, and the bioavailability of the drug are all considerations. Drugs administered by mouth vary in peak and consistency of effect. Reversal of any unwanted effect is also difficult. The operator cannot titrate the drug to achieve optimal effect with momentto-moment control as with other methods. Recovery time may be prolonged, because the drug is slowly metabolized.

Many practitioners attempt to overcome the problems of length of onset by having the parent administer medications at home. This, however, introduces the problem of reliability as well as the inability to properly monitor a sedated patient. This practice should be avoided. Except for minor tranquilizers (e.g., diazepam, hydroxyzine), all other sedative agents should be administered in the office by trained personnel. A suitable area should be available in the office so that trained personnel can monitor the patient during the onset of sedation as well as the recovery period until discharge criteria have been met (see Box 14-2).

Technique. After the sedative agent(s) have been selected, the proper dose of the medication(s) is calculated for the child. It is important always to keep in mind the maximum recommended dose of each agent. Once the sedative is administered, the patient should be kept in an area of the office that allows for continuous monitoring. When the desired effect is observed (usually after a period of 30 to 60 minutes), the patient is transferred to the operating area and placed in the chair. For small children or children who may become physically combative, placing the patient in a reclining restraint (e.g., a Papoose Board* [Fig. 14-7, AI) is advisable. The arms, legs, and midbody should be secured; however, the chest and diaphragm must not be restrained to avoid obstruction of respiratory muscles. A shoulder roll should be placed to keep the head slightly rolled up and back, to allow for a more patent airway (Fig. 14-7, B). Nitrous oxide and oxygen may be started at this time for added effect, as well as for oxygenation. If the patient is not adequately sedated to the point that treatment can be accomplished, the attempt should be aborted and considered a sedation failure. The patient should be given a reappointment for another attempt, in which either the dose or the technique is altered. Increments of medication should not be administered to avoid the risk of an overdose caused by variable and unpredictable absorption. Titration is dangerous and should be reserved for intravenous techniques that do not rely on variable absorption.

The patient should be monitored carefully during the procedure to ensure responsiveness (see Table 14-2).

*Olympic Medical Corp., Seattle, Wash.



[I I; 14-7. A, Papoose Board. **B,** Patient positioning on Papoose Board. The shoulder roll functions to keep the head rolled up and back, which allows for a more patent airway.

Discharge to the parent should occur only after minimal discharge criteria have been met (see Box 14-2). Full oral and written instructions on postsedation care should be carefully reviewed with the caregiver and the written information given to that person for easy reference at home (see Fig. 14-4).

Oral sedation may be achieved by using a combination of drugs. This is done either to achieve a combined effect or, more often, to reduce the amount required of drugs with a greater potential for problems at higher dosages.

INTRAMUSCULAR SEDATION

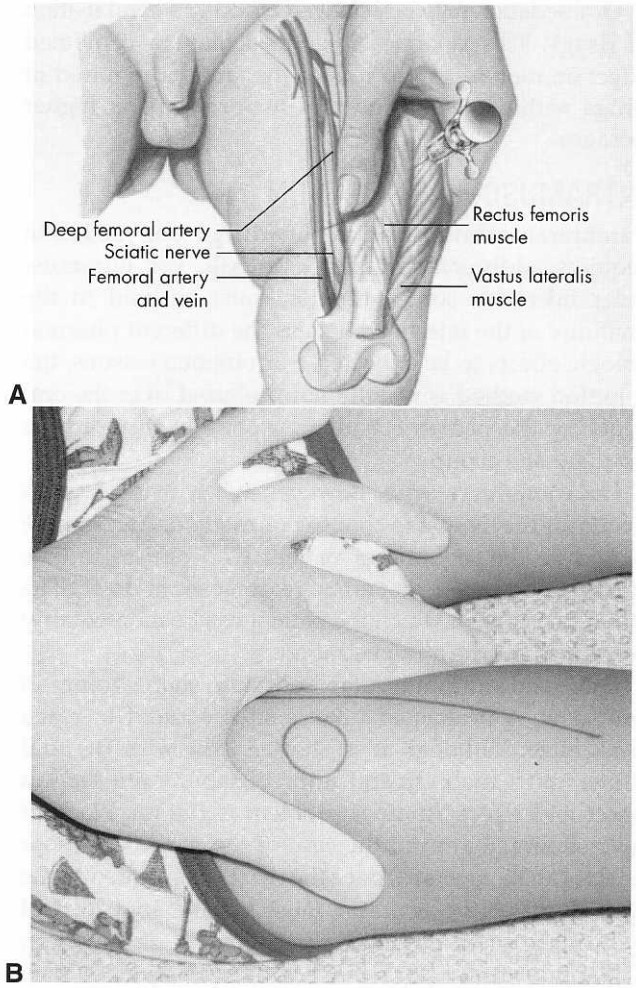
Parenteral administration of drugs for sedation requires additional training and skill. For intramuscular injections some attention must be paid to the anatomy of the injection site and the different pharmacologic effects to be expected. For obvious reasons, the injection method is usually not preferred over the oral route by the pediatric patient, especially those in the younger age group.

Occasions may arise, however, when it is advantageous for the dentist to choose this method. An example would be the sedation of patient who refuses to take medication orally or for some reason cannot do so. The situation is one in which the patient can be momentarily restrained for the administration.

Oral and intramuscular sedation share common problems. A prolonged time is still required to reach peak effect, although it is shorter than with the oral route, and variability and unpredictability are seen in onset and effect. Another problem is the total lack of reversibility. Except in the case of the narcotics whose effects can be reversed, once the drug is deposited in the muscle mass, the operator must be prepared to deal with unexpected problems for the extent of the effect. The opportunity for idiosyncratic reaction is also greater.

Anatomic Considerations. Injection-site selection is a very important aspect of the intramuscular drug administration. The primary considerations are the presence of adequate tissue for deposition of the drug volume and reduction of the risk of injury from needle penetration. Use of the vastus lateralis muscle on the anterior thigh is the safest for small children. This is a rectangular area over the anterior lateral aspect of the thigh (Fig. 14-8). The upper outer quadrant of the gluteus maximus muscle and the middle of the posterior lateral aspect of the deltoid muscle are other acceptable sites provided muscle development is adequate.

Once the medication has been injected, if the desired effect is not achieved in a reasonably expected time of 20 to 30 minutes, either the treatment should be performed under existing circumstances or the patient should be given a reappointment at which either an increased dose or an alternative method of sedation is used. As with oral administration, titration to a desired level of sedation by injection of incremental amounts of medication is dangerous and must be avoided.



C **14-8. A,** Anatomy of the anterior thigh of a child. **B,** Demonstration of patient injection site. *(A courtesy Wyeth Laboratories, Philadelphia;* **B** *courtesy Dr. David R. Avery.)*

SUBMUCOSAL SEDATION

Submucosal sedation involves the deposition of the drug beneath the mucosa. Its use is almost exclusively the province of dentistry. Although the effectiveness of oral local anesthetics is best if they are placed subperiosteally, most infiltrations are in fact submucosal injections. As is the case with local anesthetics, care must be taken that sedatives are not injected into any of the muscles of the face and jaw lest the absorption and effect of the drug be unduly and unexpectedly prolonged. The site usually chosen for injection is the buccal vestibule, particularly in the area of the maxillary primary molar or canine teeth.

The term *submucosal* is often used interchangeably with *subcutaneous*. This usage may be erroneous, because research has shown the serum level of a drug to rise very quickly after injection into the vessel-rich areas just described. The response time probably should be considered to be somewhere between those of the intramuscular and the intravenous administration of a drug. This fact makes the method suitable for the pediatric patient for whom quick administration and onset is desirable. Caution should be exercised, however, to select a drug that is not irritating to tissue. The information provided with the drug will indicate its suitability for tissue injection. It is important to remember that this is a parenteral route of administration, and adherence to all of the necessary precautions and requirements must be ensured.

Technique. The technique requires only a syringe suitably sized for the volume of drug affixed with a 27- or 30-gauge, '/z-inch needle. A tuberculin syringe is recommended for volumes less than 1 ml. Some patient restraint is usually required. Placement of a small quantity of local anesthesia without vasoconstrictor in the area of injection is preferred if the drug being used is known to be painful on injection. The local anesthetic required for pain control of the procedure should not be given in the same area after the injection of the sedative drug, because the action of the vasoconstrictor will slow the uptake of the sedative drug. Also, the physiochemical properties of the sedative agent may interfere with the effectiveness of the local anesthetic agent.

The drugs most commonly administered by the submucosal technique are the narcotics meperidine and fentanyl.

INTRAVENOUS SEDATION

Sedation levels in which the patient remains conscious are readily achievable through the use of intravenous techniques. In the hands of properly trained professionals this method can be the easiest, most efficient, and safest next to sedation by inhalation. Controversy has existed for several years over what constitutes adequate training in the use of this modality. The individual state boards are resolving this controversy by writing requirements in terms of didactic and clinical hours required to be certified by the board to use intravenous sedation. Generally, these requirements are following those set forth by the American Dental Association's Guidelines for the Use of Conscious Sedation, Deep Sedation, and General Anesthesia for Dentists.'

The use of intravenous conscious sedation in pediatric dentistry is somewhat restricted to certain types and ages of patients. Venipuncture is difficult to accomplish in the very young or the combatant child. Such difficulty is attributable to smaller vein size and availability together with the need to restrain the patient. Because of this, the technique is often more suitable for the apprehensive preteen and adolescent patient.

The attribute that makes intravenous sedation desirable for some patients also makes it undesirable for others in the hands of the untrained practitioner. In very

young children, the incidence of untoward effects is increased. The onset of action of the drug or drugs used is approximately 20 to 25 seconds, which is the most rapid among all techniques. Thus the opportunity to render the patient unconscious unintentionally is much greater. A practitioner with limited training in management of the airway or of the unconscious patient should never consistently crowd this line between consciousness and unconsciousness.

The possibility of phlebitis is also a disadvantage, particularly with drugs insoluble in water. Hematoma at the site of venipuncture is a complication. There is also an increased need to monitor the patient more closely on a continuous basis, and this usually means the use of an assistant who has had extensive training in the monitoring of sedated patients. In fact, current sedation guidelines require the presence of a third person for the sole purpose of monitoring.

With the intravenous method of sedation one must also consider that, although the onset of effect is very rapid, the reversal of effect is not.

Techniques. There are basically two techniques for using intravenous sedation. The first employs a single drug, usually a benzodiazepine, whereas the second requires a sophisticated combination of several drugs, usually including a narcotic. The single-drug technique is probably the most adaptable to pediatric practice. Unless extensive training opportunities exist, the multiple-drug method should not be considered.

COMBINATIONS OF METHODS AND AGENTS

It is often advantageous to consider combining methods and agents to produce a state of balanced conscious sedation. A goal of combining agents should be to establish a balance between sedation, analgesia, and amnesia, all while minimizing adverse effects and maintaining physiologic homeostasis. When agents are combined, often one drug will be potentiated by another. Their combination thus enables the operator to reduce the dosage of a stronger drug, such as a narcotic, and thus reduce the possibility or degree of an adverse effect, such as severe respiratory depression. Another goal in combining agents might be to quiet the behavior of the patient for the introduction of a method requiring more patient cooperation, such as placement of an intravenous line.

Conscious sedation is usually most effective when combined with the use of local anesthesia. Sedation techniques are not pain-control techniques, and the sedative effects are often overridden when intraoperative pain is experienced by the patient. To overcome this circumstance with the use of sedative agents alone requires the use of very high doses or the addition of a narcotic to the regimen; this produces a deeper level of sedation than might be required and increases the possibility of adverse effects. Sedation techniques should not be used simply to escape the need to inject a local anesthetic. One should also remember that local anesthetic agents are CNS depressants, and effects will be additive. Occasionally a drug with a known capacity to improve postoperative comfort also may be used, particularly if surgical procedures are required.

Inhalation sedation is the next most frequently combined technique. Nitrous oxide and oxygen can be combined with all other methods of conscious sedation. Not only does this combination provide increased sedation, but it also increases the availability of oxygen for the patient. In fact, inhalation sedation is so often used in combination that one should be reminded that nitrous oxide is also a CNS depressant and should be the first agent to be reduced or reversed when the sedation level becomes deeper than desired.

The orally administered sedatives are the next most commonly employed in combination. Most frequently combined include sedative hypnotics, narcotics, and/or antihistamines. A great deal of care must be taken when combining oral agents to avoid deepening the patient's state from conscious sedation to unconscious sedation or general anesthesia. It is important to appreciate that oral agents are potent and that combinations are capable of deepening the patient's sedation to the point that protective reflexes may be lost.

The oral premedication of the patient with minor tranquilizers in advance of the use of inhalation or parenteral sedation methods is a common practice. This technique works especially well to calm the patient for a smoother beginning of a sedative appointment. When parenteral agents are used, unless the result of admixture is definitely known, it is not good practice to mix the agents before administration to the patient, because they may be incompatible in mixed solution. Combining drugs complicates their use because of their additive, synergistic, or potentiating effects. This makes recognition of the causative agent of the untoward effect and subsequent efficient management very difficult. The combination of methods or agents should be viewed as a balancing technique and should always be manipulated so as to produce a maximum result with minimum risk of complication.

Multiple-drug use is also common with intravenous methods of conscious sedation. All the previous cautions pertain in these situations as well. Because of the great potential for swiftly occurring, serious adverse reactions, these multiple-drug intravenous techniques should *never* be used by anyone who is not specifically trained in their use.

Regardless of the method of administration, the combining of agents should be approached with great caution because of the possibility of additive effects. Individual drug doses should be reduced by 20% to 50% when the drugs are given in combination.

FACILITIES AND EQUIPMENT

The primary requisite for successful sedative management of the pediatric patient is a comfortable, quiet environment. All necessary supplies and equipment should be readily available to ensure a minimum amount of disturbance once the procedure is under way. Although not necessary, a comfortable, single-patient, quiet room is helpful. The facility should also be equipped with adequate suction, monitoring devices, and a positive-pressure oxygen delivery system that is capable of sustaining greater than 90% oxygen at a flow of 10 L per min for at least 60 minutes (650-L E cylinder). When a self-inflating bag valve mask device is used for delivering positive-pressure oxygen, a 15 L per minute flow is recommended. A functional suction apparatus with appropriate suction catheters and a sphygmomanometer with cuffs of appropriate size for pediatric patients must be immediately available. All equipment must be adaptable to children of all ages and sizes.'

As previously stated, all inhalation sedation equipment must have the capability for immediate delivery of 100% oxygen with a minimum delivery of 25%. The apparatus must have a fail-safe system that accomplishes complete shutdown when the oxygen supply drops below the 25% level. This system must be checked and calibrated annually.

Emergency drugs and equipment needed to resuscitate a nonbreathing, unconscious patient must be close at hand and kept up to date and functional. Portability is essential so that a patient can be supported during transfer to a medical facility if necessary. The best way to provide a portable, reliable oxygen system is with an E-sized (650-L) cylinder to which a self-inflating bag and mask, commonly called an Ambu bag, can be attached (Fig. 14-9). With this apparatus, oxygen from the cylinder mixed with room air can be delivered to the patient. The device is equipped with a valve to prevent rebreathing and can be used alone to supply room air to the patient during movement from one area to another.

Establishing an open airway and administering oxygen should be the first thought in any emergency related to conscious sedation. These situations almost always are respiratory in nature at the outset. If respiratory problems are ignored, other systemic problems will rapidly ensue. Quick action is the means to preventing deterioration of adverse reactions.

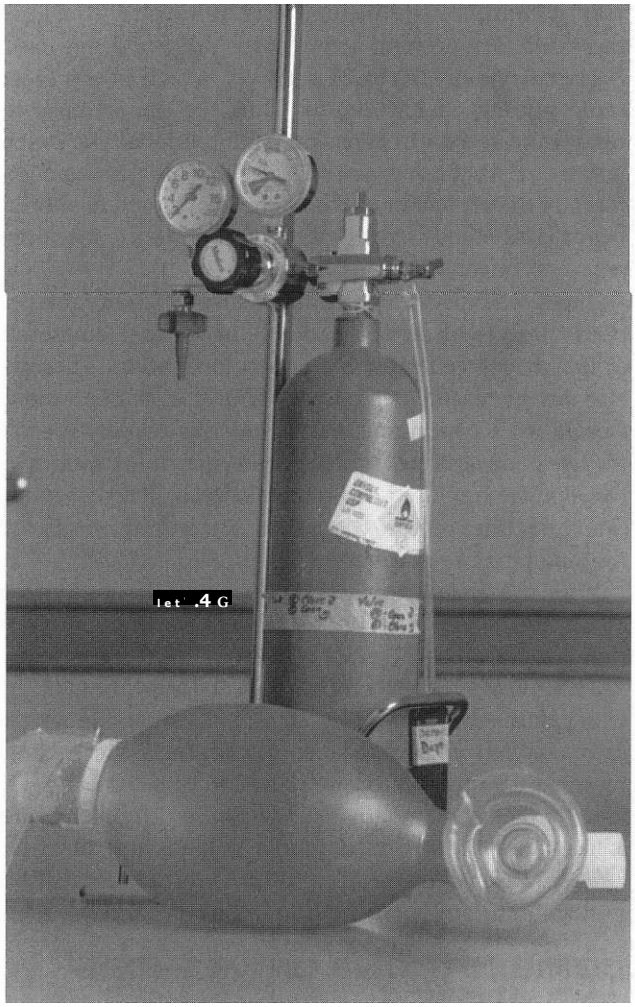


FIG. 14-9. Bag-mask-valve device for assisting respiration.

COMMON AGENTS EMPLOYED

GASES

As previously mentioned, only two gases are used in conscious sedation techniques—nitrous oxide and oxygen. The pharmacology of nitrous oxide is discussed in the section on inhalation sedation and need not be repeated here. Oxygen requires no discussion, because its sole purpose is to avoid anoxia, which is produced if nitrous oxide is used alone.

ANTIHISTAMINES

■ Hydroxyzine (Atarax, Vistaril)

Rapidly absorbed from the gastrointestinal tract. Clinical effect seen in 15 to 30 minutes, with peak levels at 2 hours and a mean half-life of 3 hours. Administration is preferably by the oral route. Intramuscular injections must be deep in a large muscle mass. Care must be exercised in using this drug in a small child. The drug should not be injected subcutaneously or intravenously because of potential tissue necrosis and hemolysis.

ADVERSE REACTIONS: Extreme drowsiness, dry mouth, and hypersensitivity

DOSAGE: Oral—1 to 2 mg/kg
Intramuscular (IM)—1.1 mg/kg

SUPPLIED:

Atarax
Tablets—10, 25, 50, and 100 mg
Syrup—10 mg/5 ml
Vistaril
Suspension—25 mg/5 ml
Injectable—25 or 50 mg/ml

■ *Promethazine* (Phenergan)

A phenothiazine with sedative and antihistaminic properties. Well absorbed after oral ingestion. Onset is within 15 to 60 minutes, with a peak at 1 to 2 hours and a duration of 4 to 6 hours. Phenothiazines should be used with caution in children with a history of asthma, sleep apnea, or a family history of sudden infant death syndrome. Phenothiazines lower the seizure threshold and should be avoided in seizure-prone patients.

INTERACTIONS: Potentiates other CNS depressants ADVERSE REACTIONS: Dry mouth, blurred vision, thickening of bronchial secretions, mild hypotension, extrapyramidal effects
DOSAGE: Oral/IM—0.5 to 1.1 mg/kg
SC—not recommended
Maximum recommended single dose is 25 Ing
SUPPLIED: Tablets—12.5, 25, and 50 mg
Syrup-6.25 and 25 mg/ml
Injectable—25 and 50 mg/ml ampules

■ Diphenhydramine (Benadryl)

An antihistamine with sedative properties. Rapidly absorbed through the gastrointestinal tract, with maximum effect in 1 hour and a duration of 4 to 6 hours. Metabolized by the liver and completely excreted in 24 hours. Produces a mild sedative effect but has additive effects with other CNS depressants.

ADVERSE REACTIONS: Disturbed coordination, epigastric distress, and thickening of bronchial secretions
DOSAGE: Oral, IM, or intravenous (IV)—1.0 to 1.5 mg/kg
Maximum single dose is 50 mg
SUPPLIED: Capsules—25 and 50 mg
Elixir—12.5 mg/5 ml
Injectable—50 mg/ml

BENZODIAZEPINES

■ *Diazepam* (Valium)

A benzodiazepine that is lipid soluble and water insoluble. It is rapidly absorbed from the gastrointestinal tract, reaching peak levels at 2 hours. Biotransformation of the drug occurs quite slowly and it has a half-life of 20 to 50 hours. The drug has three active metabolites, one of which is also very lipophilic and has a half-life of 96 hours. These metabolites are more anxiolytic than sedative.

After intravenous administration, diazepam is redistributed within 30 to 45 minutes, and the patient seems not to be sedated although free from anxiety. The patient should not be considered recovered from the drug. It has simply been redistributed. In fact, stored drug can be redistributed to the CNS by a fatty meal consumed some time later and the patient will suddenly feel resedated. This is referred to as the *rebound effect.*

Diazepam has strong anticonvulsant activity and provides some prophylaxis against this adverse reaction of other drugs during the operative procedure.

Diazepam can be administered orally, rectally, or parenterally. If the intravenous route is selected, use of a large vein and slow administration is recommended because of the drug's propensity to cause irritation of the vein, with resultant thrombophlebitis. In addition, rapid administration may result in apnea.

Ataxia and prolonged CNS effects are the only common adverse reactions that can be anticipated when diazepam is used for conscious sedation.

DOSAGE: Oral or rectal—0.2 to 0.5 mg/kg to a maximum single dose of 10 mg
IV—0.25 mg/kg
SUPPLIED: Tablets-2, 5, and 10 mg
Suspension—5 mg/ml

■ Midazolam (Versed)

A significant advantage of midazolam over diazepam is its high water solubility. Consequently, the possibility of thrombophlebitis is reduced to a minimum. Midazolam is packaged in an acidic solution at a pH of 3.3, a state in which the drug is water soluble. Once the drug enters the blood, which has a pH of 7.4, the chemical structure changes to an active form with high lipid solubility. In fact, its lipid solubility is among the highest of all benzodiazepines.

After intravenous administration, sedation occurs in 3 to 5 minutes. Recovery occurs in 2 hours but is variable and may require up to 6 hours for complete return to baseline values. There is no rebound phenomenon from metabolites. Midazolam can also be given effectively intramuscularly.

Midazolam is effective when given orally and is available as a syrup. Approximately 50% to 65% of the oral dose will go through first-pass hepatic metabolism. Onset of action when given orally is between 20 and 30 minutes with an elimination half-life of 1 to 4 hours, which provides approximately 30 minutes of working time. Oral administration is indicated primarily for anxious patients requiring relatively short dental procedures. As a single agent, it should not be used to control combative behavior.

Midazolam may produce respiratory depression with higher doses. There is also a dose-related risk of apnea,

which is believed to be influenced by the rapidity with which the drug is administered."- Apnea is more likely to occur when midazolam is used with narcotics.' Hypotension also occurs more frequently with this combination. Doses should be adjusted downward when midazolam is used with any other CNS depressant, especially the narcotics. These effects are not usually seen if the drug is appropriately titrated.

Compared with diazepam, midazolam produces better anxyolysis and amnesia. In fact, between 75% and 90% of patients will experience retrograde amnesia for up to 4 hours when given midazolam. Midazolam is three to four times more potent than diazepam and has twice the affinity for the benzodiazepam receptor.

DOSAGE: Oral—0.25 to 1.0 mg/kg to a maximum single dose of 20 mg
IM—0.1 to 0.15 mg/kg to a maximum dose of 10 mg
IV—slow IV titration; see manufacturer's recommended dosage guidelines
SUPPLIED: Syrup—2 mg/ml
Injectable—1 mg/ml and 5 mg/ml vials

BENZODIAZEPINE ANTAGONIST

■ *Flumazenil* (Romazicon)

Flumazenil is a benzodiazepine receptor antagonist. The drug selectively inhibits the CNS effects of the benzodiazepines by a competitive, high-affinity interaction with benzodiazepine receptors. It has no antagonistic properties against the opioids. When carefully titrated, flumazenil has been shown to be effective in reversing the sedative effects but not necessarily the amnesic or anxiolytic qualities of benzodiazepines.

Flumazenil is well tolerated in patients having no dependence on or increased tolerance to benzodiazepines. In patients with benzodiazepine dependency, withdrawal symptoms will occur when flumazenil is given. Induced seizure activity will occur in patients receiving benzodiazepines for control of convulsive disorders when flumazenil is used. The drug is recommended for intravenous use only and is not recommended for use in children younger than 18 years of age.

For reversal of conscious sedation, the initial dose should be 0.2 mg given over 15 seconds. Should the desired level of consciousness not occur after waiting an additional 45 seconds, another dose of 0.2 mg should be administered and dosing repeated at 60-second intervals to a maximum total dose of 1 mg. Most patients will respond to doses in the range of 0.6 to 1.0 mg. A series of injections is preferable to a single bolus to titrate to a desired end point and thus manage the problem with the minimally effective amount of drug. Onset of reversal is usually seen within 1 to 2 minutes. The duration and degree of reversal are related to dose and plasma concentration of the sedating benzodiazepine, as well as that of the antagonist given. This, coupled with the fact that the duration of effect is shorter for flumazenil than for most benzodiazepines, means that resedation can occur. Patients should be carefully monitored for resedation and respiratory depression throughout the period of reversal. The longer the period of sedation, the longer the period required for monitoring and surveillance for resedation. If resedation should occur, repeated doses of flumazenil at no less than 20-minute intervals may he used.

DOSAGE: IV—as just described
SUPPLIED: 5- and 10-ml multiple-use vials containing 0.1 mg/ml in boxes of 10

SEDATIVE HYPNOTICS

■ Barbiturates

Barbiturates can produce all levels of CNS depression, ranging from mild sedation to general anesthesia and deep coma. Their use has fallen out of favor, however, with the availability of sedatives and hypnotics with fewer adverse effects. Consequently, barbiturates are of very limited value for pediatric patients.

■ *Chloral Hydrate* (Noctec, Aquachloral Supprettes)

Chloral hydrate is an extremely well known and widely used drug for pediatric sedation. It has an onset of action of 30 to 60 minutes when given orally. It has a duration of action between 4 and 8 hours and a half-life of 8 to 11 hours as a result of the formation of active metabolites. The primary metabolite of choral hydrate is trichloroethanol, which is responsible for most of the CNS effects that occur.

Chloral hydrate is irritating to gastric mucosa and unless diluted in a flavored vehicle will frequently cause nausea and vomiting. Children given chloral hydrate often enter a period of excitement and irritability before becoming sedated. The drug causes prolonged drowsiness or sleep and respiratory depression. In large doses it produces general anesthesia. Large doses also depress the myocardium and can produce arrhythmias, and thus should be avoided in patients with cardiac disease.[4]''' The lethal dose of chloral hydrate is stated to be 10 g in adults, yet ingestion of 4 g has caused death.

Because the drug does not reliably produce sedation of a degree to permit operative procedures at lower doses, the tendency is to push the dosage higher to achieve the necessary sedation. With such a wide range of reported toxicity, this drug may be an unwise choice for many pediatric patients. It is recommended that young children receive not more than 1 g as a total dose. Risks are increased when chloral hydrate is combined with nitrous oxide, narcotics, or local anesthetic agents.

At higher doses and in combination with other agents, loss of a patent airway is a common problem.'

DOSAGE: Must be individualized for each patient
RECOMMENDED: 25 to 50 mg/kg to a maximum of 1 g
SUPPLIED: Oral capsules—500 mg
Oral solution—250 and 500 mg/5 ml
Rectal suppositories—324 and 648 mg

NARCOTICS

Narcotics are the "heavy artillery" of pediatric conscious sedation. They are not employed with any great consideration for their analgesic properties. They do produce sedation and euphoria to a greater degree in children than in adults. Local anesthesia is still required for intraoperative pain control. One should remember that local anesthetics are also CNS depressants.

A significant drug-drug and drug-physiologic interaction can occur when narcotics or other drugs that depress respiration are combined with local anesthetics. In usual doses, local anesthetics are CNS depressants and will provide additive depression when combined with other CNS depressants. In addition, when drugs that depress respiration are used (particularly narcotics), varying degrees of hypercarbia can occur, with a resultant decrease in serum pH. As the respiratory depression continues to deepen, metabolic acidosis will result in an increase in the availability of lidocaine to the CNS. This occurs as a result of less serum protein binding of lidocaine along with central vasodilation and an increase in blood flow to the CNS in an acidotic state. Consequently the threshold for CNS lidocaine toxicity is lowered. Lidocaine toxicity will result in CNS excitation and seizures and ultimately coma and death. As a result, *the maximum dosage of local anesthetic must be reduced when used in combination with a CNS and/or respiratory depressant.* This very important and significant interaction is often overlooked and in fact is the cause of many of the adverse incidents reported in pediatric sedation. It is important to remember that the maximum local anesthetic dose in chil dren may allow for the use of only one or two dental cartridges, which is quite different than for adult patients.

Combination with other sedative drugs, including nitrous oxide–oxygen, will reduce the need for larger doses of narcotics and thus reduce the potential for unwanted effects from these potent drugs. A practitioner employing the narcotics should be thoroughly familiar with their actions and interactions and should have had some supervised experience in their use as well as in management of the airway and patient resuscitation procedures.

■ *Meperidine* (Demerol)

A synthetic opiate agonist. It is very water soluble but is incompatible with many other drugs in solution.

Meperidine may be administered orally or by subcutaneous, intramuscular, or intravenous injection. It is least effective by mouth. It is very bitter and requires masking by a flavoring agent. By the oral route, peak effect occurs in 1 hour and lasts about 4 hours. Parenteral administration shortens the time of onset and duration. High doses that lead to an accumulation of normeperidine, a primary metabolite of meperidine, have resulted in seizures. Meperidine should be used with extreme caution in patients likely to accumulate or be sensitive to this metabolite (e.g., patients with hepatic or renal disease, or history of seizures).

DOSAGE: Oral, SC, or IM—1.0 to 2.2 mg/kg, not to exceed 100 mg
SUPPLIED: Oral tablets—50 and 100 mg
Oral syrup—50 mg/5 ml
Parenteral solution—25, 50, 75, and 100 mg/ml

■ *Fentanyl* (Sublimaze)

A synthetic opiate agonist in the same chemical class as meperidine. It is a very potent narcotic analgesic. A dose of 0.1 mg is approximately equivalent to 10 mg of morphine or 75 mg of meperidine. Fentanyl has a rapid action, and after a submucosal or intramuscular injection the onset will occur in 7 to 15 minutes; duration of effects is 1 to 2 hours. The drug is metabolized by the liver and is excreted in the urine.

Respiratory depression is the same as with other narcotics. Respiratory rate decreases but returns to near normal quite rapidly. Tidal volume, however, remains depressed. The response to carbon dioxide can be depressed for extended periods, long after the analgesic effect has disappeared. It is believed that this characteristic is attributable to redistribution of the drug. When fentanyl is used, one should be attentive to and competent in airway management. With higher doses administered rapidly by vein, rigidity of skeletal muscle has been reported. Apnea has also occurred under these circumstances. This effect can be reversed by administration of naloxone along with a skeletal muscle relaxant and/or managed by assisted or controlled ventilation. Bradycardia has been reported. Atropine can be used to normalize heart rate.

Fentanyl produces little histamine release and has much less emetic effect than morphine or meperidine.

Fentanyl can be administered by the intramuscular, intravenous, or submucosal route. When it is used with other CNS depressants, the dose should be reduced. The drug works well with orally administered diazepam and nitrous oxide–oxygen. It is not recommended for use in children under 2 years of age.

DOSAGE: 0.002 to 0.004 mg/kg
SUPPLIED: 0.05 mg/ml in 2- and 5-ml ampules

NARCOTIC ANTAGONIST

■ *Naloxone* (Narcan)

A semisynthetic opiate antagonist used for the sole purpose of reversing the effects of narcotic drugs. Naloxone is a pure antagonist, with no agonist activity even in large doses. It acts in 2 to 5 minutes after subcutaneous or intramuscular injection and 1 to 2 minutes intravenously. After intravenous administration the duration of reversal is about 45 minutes; it is slightly longer when the drug is administered intramuscularly or subcutaneously This is an important difference, because the duration of effect of the opiate is in all likelihood longer than that of the antagonist. Because of this, patients undergoing reversal of sedation with naloxone should be kept under continual surveillance until it has been determined that the narcotic will not produce a rebound effect. The time period will vary depending on the duration of action of the narcotic. Repeated doses of naloxone may be necessary to establish patient stability. If the decision has been made to administer an antagonist, other resuscitative measures must be available and must be used as necessary. Naloxone administration should never take precedence over basic resuscitative measures. There is no evidence to support the contention that naloxone will reverse respiratory depression but not the sedative action of the opiate.

Adverse reactions include nausea, vomiting, sweating, hypotension, hypertension, ventricular tachycardia and fibrillation, and pulmonary edema. None of these effects, however, has been reported with its use in pediatric conscious sedation.

DOSAGE: IV, SC, IM—initial dose: 0.01 mg/kg; subsequent doses: 0.1 mg/kg (2 mg maximum) every 2 to 3 minutes

SUPPLIED: Parenteral solution—0.02, 0.4, 1.0 mg/kg

MONITORING

For successful monitoring of the vital physiologic parameters of a patient receiving treatment with the aid of conscious sedation, baseline values taken preoperatively at a time when the patient is calm are extremely helpful. Admittedly this is not possible in all circumstances, but the attempt should be made to establish these values.

INTRAOPERATIVE MONITORING

Intraoperative monitoring must include an assessment of oxygenation, ventilation, and circulation. The depth of sedation will dictate the degree and frequency of monitoring required (see Table 14-2).[2]

As the treatment progresses, the state of consciousness should be evaluated frequently by verbal communication with the patient. How often this is done is related directly to the level of sedation at any one time. As the patient becomes quieter with deeper levels of sedation, more vigilance to all physiologic parameters is required. If the patient is responding to questions and commands, the patient is conscious by definition. For those patients incapable of communication either because of age or disability, some means of evoking a response should be used. For the very lightly sedated patient capable of verbal response, this may be the extent of the monitoring required.

Another area of assessment is the patient's appearance. The oral mucosa, the nail beds, and the complexion of the skin provide indications of perfusion of the patient. This should be done at intervals throughout the procedure and documented in the record. If restraining devices that cover the patient are used, a hand or foot should be exposed. These devices should be carefully applied to the sedated patient to ensure that there is no restriction of the chest.

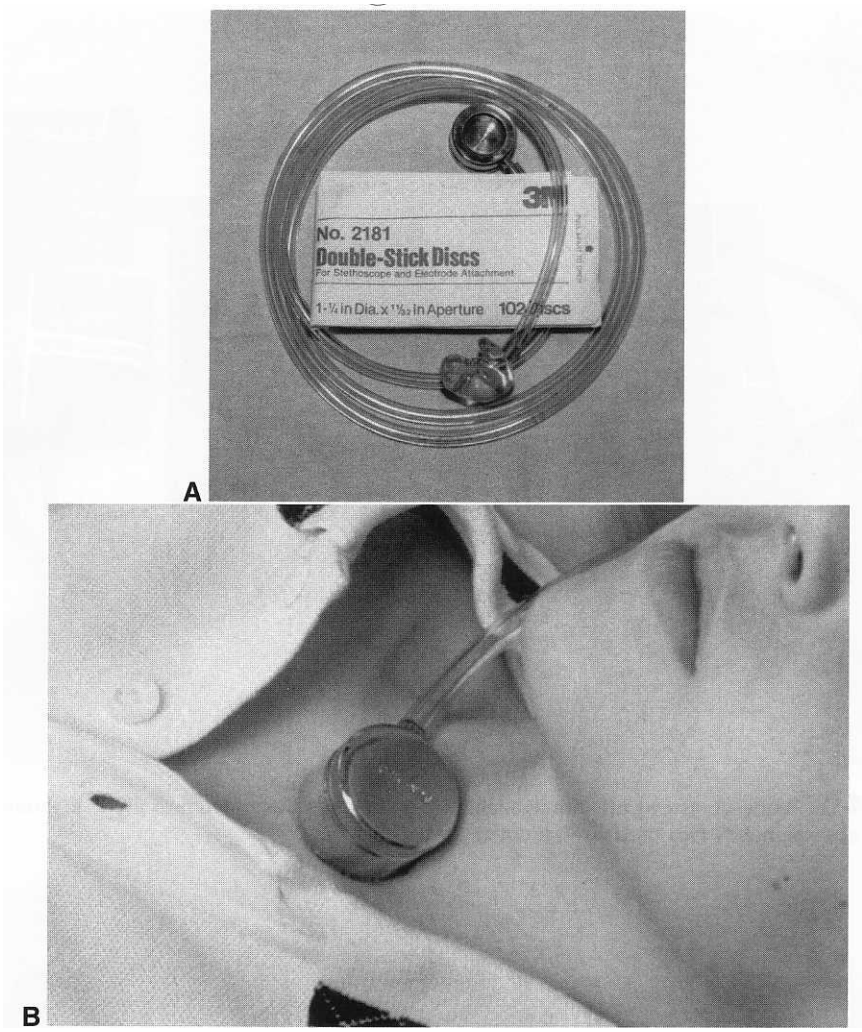
The heart and respiratory rates can be continuously monitored by means of a stethoscope with a custom-fitted earpiece. The bell of the instrument is taped over the trachea in the suprasternal notch. This piece of equipment is referred to as a precordial or, more accurately, a pretracheal stethoscope (Fig. 14-10).

In addition to the rate, the quality of heart and breath sounds should be assessed and recorded. The patient should have a strong, regular pulse and an airway without noisy movement of air.

The excursion of the chest is important also, because the rate of respiration can remain steady while the depth becomes increasingly shallow. Shallow respiration is a reduction in tidal volume, and the exchange of oxygen is correspondingly reduced.

For deeper levels of conscious sedation, such as with the use of narcotics, it is desirable to monitor blood pres sure at frequent intervals. Again, the frequency is determined by the depth of the sedation (see Table 14-2). It is important to remember, however, that physiologic deterioration occurs long before changes in blood pressure are detected.

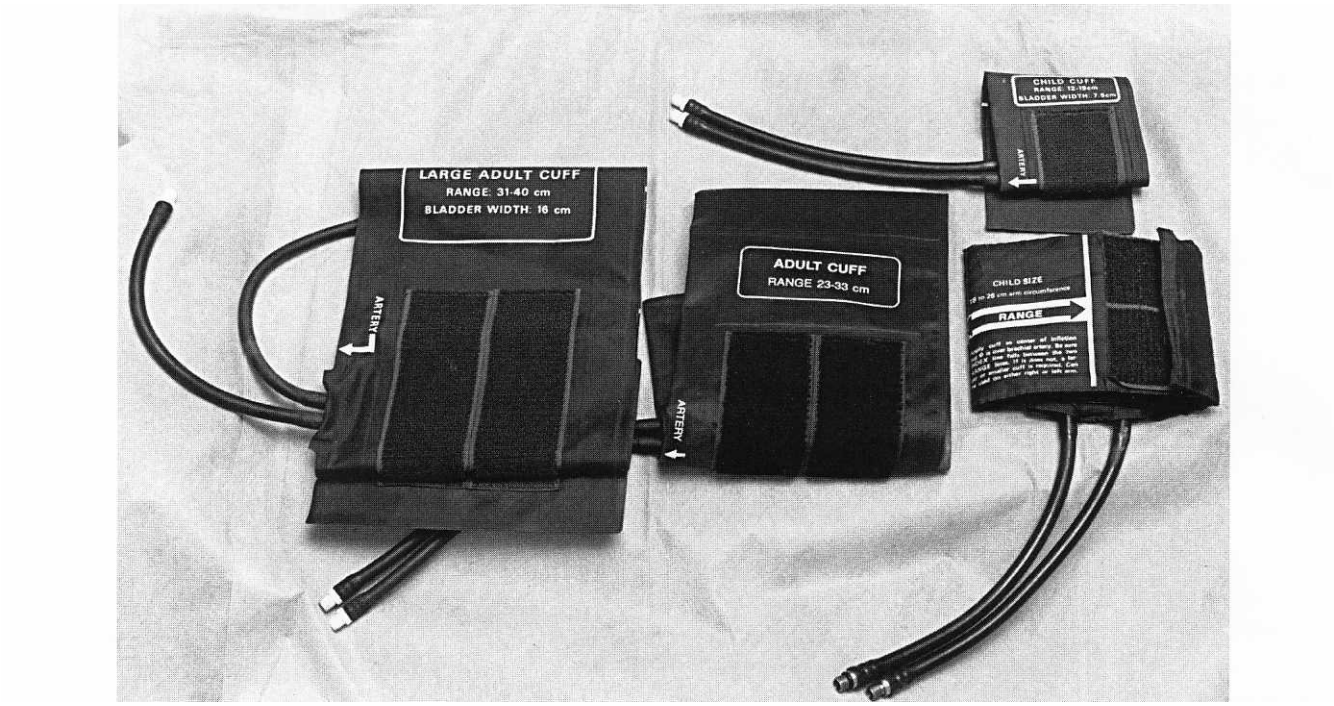
Although it is acceptable to measure physiologic parameters using the basic methods of auscultation and an aneroid sphygmomanometer with a manually inflated cuff, numerous electronic, automatic, continuous-monitoring devices are available. These range from the uncomplicated and reasonably priced to the elaborate and expensive. In all instances the cuff should be of the appropriate size for the patient (Fig. 14-11). Pulse monitors are available that attach to the finger or earlobe and produce both visual and audible signals. There are variable-cycled blood pressure monitors with automatically inflating cuffs (Fig. 14-12). These are monitors in which both blood pressure and pulse rate are continuously assessed together with the mean arterial pressure and automatically recorded on a paper strip. The device carries adjustable visual and audible alarms.



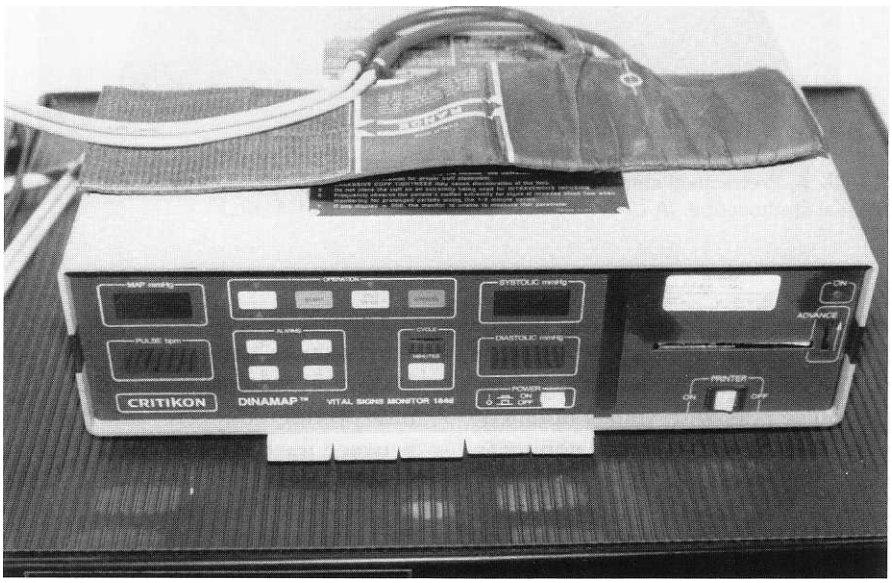
f **I G . 14-10. A,** Pretracheal stethoscope with fitted earpiece. Double-stick disks. **B,** Placement of pretracheal stethoscope. *(A Courtesy 3M Corporation, St. Paul, Minn.)*

One of the most valuable pieces of electronic monitoring equipment is the pulse oximeter (Fig. 14-13). This device continuously assesses arterial hemoglobin oxygen saturation and pulse rate, with values updated with every heartbeat. An oxisensor is attached noninvasively to a digit on the hand or foot or to the earlobe and consists of a light-emitting diode and light-detecting diode (Fig. 14-14). The light-emitting diode emits both red and infrared wavelengths of light, and the light-detecting diode detects light transmitted through the tissue. Red wavelengths are absorbed primarily by oxygenated hemoglobin, whereas infrared wavelengths are absorbed primarily by deoxygenated hemoglobin. The device's processor then calculates the percent oxygenation of hemoglobin and the results are conveyed both audibly and visually. A delay of over a minute occurs before respiratory compromise is reflected in oxygen saturation levels as determined by pulse oximetry.

There is a correlation between the percent oxygen saturation of hemoglobin (SaO_2) and oxygen tension in arterial blood (PaO_2) that must be appreciated when interpreting oxygen saturation. The relationship between the two parameters is plotted on the oxyhemaglobin dissociation curve (Fig. 14-15). It is the unbound oxygen dissolved in blood that produces the tension (PaO_2) required to drive oxygen into tissues. Hypoxemia occurs when oxygen saturation drops to 95%, corresponding to an oxygen tension of 80 mm Hg. Once the arterial saturation levels drop to 90% (PaO_2 = 60 mm Hg), the patient will begin to desaturate very rapidly, and the cells of vital organs will begin to be deprived of oxygen. The patient's oxygen saturation level should be as close to 100% as possible during the sedation. A number of conditions can lead to false oximeter readings, including (1) failure to place diodes directly opposite each other, (2) interference from ambient light, (3) placement



f **I G 14-11.** Blood pressure cuffs are available in various sizes. The width of the cuff should cover approximately two thirds of the upper arm.



[I G **14-12.** Automated vital signs monitor. *(Courtesy Dinamap by Critikon, Inc., Tampa, Fla.)*

of diodes on the same limb as a blood pressure cuff, (4) fingernail polish on the digit to which the sensor is attached, (5) cold limbs, (6) profound tissue pigmentation, (7) reuse of disposable oxisensors, and (8) motion artifact. Sensor displacement is the most common cause for false readings in children and can be minimized by using a sensor with adhesive tabs rather than a clip-on sensor. Securing the sensor with additional tape and using a toe rather than a finger may also help minimize displacement.

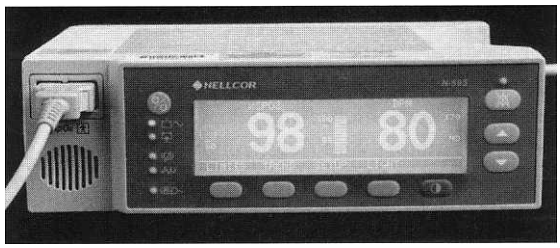


FIG. 14-13. Pulse oximeter. *(Courtesy Nellcor, Pleasanton, Calif.)*

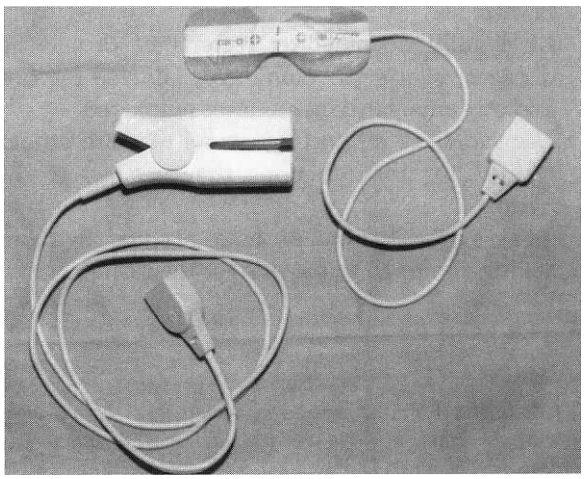


FIG. 14-14. Two types of optical sensors. Sensors with adhesive tabs are less likely to dislodge in pediatric patients than are clip-on sensors.

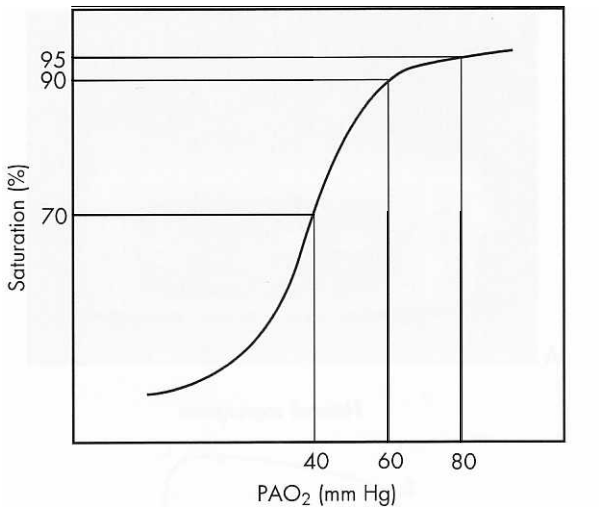


FIG. 14-15. Oxyhemoglobin dissociation curve, which shows the saturation of hemoglobin with oxygen ($Sa0_2$) relative to the oxygen tension (PaO_2). Hypoxemia is defined as PaO_2 below 80 mm Hg (95% $Sa0_2$). *(From Dionne R, Phero, J, Becker D:* Management of pain and anxiety in the dental office, *Philadelphia, 2002, WB Saunders.)*

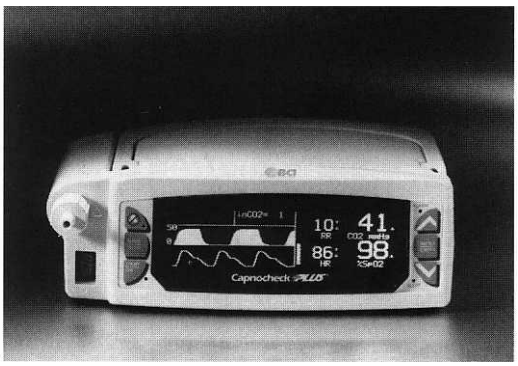
Knowing the peripheral oxygen saturation from moment to moment is important to detect sudden deterioration of the patient's physiologic status during conscious sedation. Hypoxia is almost always the primary complication with these techniques of patient management. Oxygen saturation measures oxygenation, the transport of oxygen to metabolically active tissues. It does not, however, reflect ventilation—the movement of gases from the atmosphere to alveoli. Thus, oxygenation represents only half of the results of ventilation. Ventilation must be evaluated independently from oxygenation." Methods used to monitor ventilation include visual monitoring for chest wall movement, listening for breath sounds with a precordial stethoscope, determination of respiratory rate, and capnography. Capnography is considered the gold standard for monitoring ventilatory status and will reveal respiratory compromise within 15 seconds.

The capnography monitor (Fig. 14-16, A), detects both the presence and the quality of ventilation by analyzing the concentration of carbon dioxide in the exhaled gasses through differential infrared absorption. The end-tidal carbon dioxide concentration is the concentration of carbon dioxide measured at the terminal portion of the exhalation curve (Fig. 14-16, B).[15] The sampling line is placed either in the nostril or in close approximation to the nose or mouth and allows for suctioning of a sample of exhaled air into the unit. There are limitations to the accuracy of readings that must be kept in mind, especially when the device is used in children. Head movement, mouth breathing, crying, and tube blockage by mucous all result in inaccurate readings. Technology is being developed to overcome these obstacles. In the future, capnography will become the most important monitoring instrument when patient sedation is used.

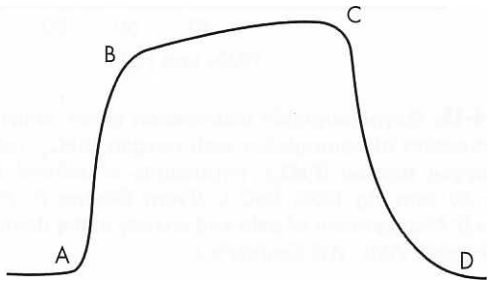
The availability of appropriately trained personnel to evaluate the patient and interpret monitoring results is just as important as using the correct monitoring devices. In addition to the operating practitioner, an individual trained to monitor appropriate physiologic parameters must be present during conscious sedation. Both individuals must be certified in basic life-support techniques and must be familiar with the office emergency cart or kit.

POSTOPERATIVE MONITORING

Before any patient receiving conscious sedation is discharged, all vital signs must be stable. The child must be reasonably alert, able to talk, ambulating with minimal assistance, and sitting unaided. For the very young or disabled patient who might be incapable of the usually expected responses, a level of awareness that is as close to the usual state for that person must be achieved before discharge (see Box 14-2).



A



A: Exhalation begins
B-C: Plateau = outflow of alveolar gas
B D: End-tidal CO_2

[I li **14-16.** Capnography. **A,** The monitor produces a waveform by the continuous analysis of expired gas for carbon dioxide. **B,** The presence of a waveform implies exhalation of gases from the lungs. The end-tidal carbon dioxide concentration *(point* C) corresponds to the concentration of alveolar gas, which correlates closely with the arterial partial pressure of carbon dioxide. *(A courtesy Capnocheck by Pryon Inc., Beaverton, Ore.* **B** *from Anderson J, Vann W: Respiratory monitoring during pediatric sedation: pulse oximetry and capnography,* Pediatr Dent *10(2): 94-101, 1988.)*

These patients should be monitored at frequent and regular intervals, and the condition of these children should be fully documented before they are discharged to the care of the parent or guardian.

RISK MANAGEMENT

To minimize the risk of adverse events' occurring during the treatment of a patient under conscious sedation, the practitioner should consider the following recommendations set forth in a policy statement of the American Dental Association:

- Use only drugs and techniques that are thoroughly understood with regard to indications, contraindications, adverse reactions and their management, drug interactions, and proper dosage for the desired effect.
- Limit the use of these modalities to patients who require them because of factors such as the extent and type of operative procedure, psychologic need, or medical status.
- Conduct comprehensive preoperative evaluation of each patient, including recording of a complete medical history and determination of current physical and psychologic status, age, and preference for and past experience with sedation and anesthesia.
- Conduct continuous physiologic and visual monitoring of the patient from onset through recovery.
- Have available appropriate emergency drugs, equipment, and facilities and maintain proficiency in their use.
- Keep fully documented records of drugs used, dosages, vital signs monitored, adverse reactions, and any emergency procedures employed.
- Use sufficient support personnel who are properly trained for the functions they are assigned to perform.
- Treat high-risk patients in a hospital or similar setting equipped to provide for their care.

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CHAPTER

15

Hospital Dental Services for Children and the Use of General Anesthesia

JAMES A. WEDDELL

JAMES E. JONES

انتشارات
شایان نمودار

CHAPTER OUTLINE

OBTAINING HOSPITAL STAFF PRIVILEGES
INDICATIONS FOR GENERAL ANESTHESIA IN THE TREATMENT OF CHILDREN
PSYCHOLOGIC EFFECTS OF HOSPITALIZATION ON CHILDREN
OUTPATIENT VERSUS INPATIENT SURGERY
MEDICAL HISTORY AND PHYSICAL EXAMINATION
ADMISSION TO THE HOSPITAL
OPERATING ROOM PROTOCOL
PROPERTIES OF INHALATION GENERAL ANESTHETICS
ANESTHETIC PREPARATION OF THE CHILD
PERIORAL CLEANING, DRAPING, AND PLACEMENT OF PHARYNGEAL THROAT PACK
RESTORATIVE DENTISTRY IN THE OPERATING ROOM
COMPLETION OF THE PROCEDURE POSTANESTHESIA CARE UNIT
POSTOPERATIVE CARE

Dentists can provide essential services to patients within an operating room setting in addition to providing consultative and emergency services. Staff membership is necessary. National commissions such as the Joint Commission on Accreditation of Healthcare Organizations (JCAHO) issue the standards for hospital governance for all hospital services.

In recent years, with the increasing number of general practice residencies and postdoctoral specialty programs, the qualified dentist finds that hospital staff privileges are a necessity. Active involvement in hospital dentistry has added a rewarding component to the practice of many dentists. *Many* hospitals have incorporated not only dental specialties but also general dental services, providing a comprehensive health care facility in which to serve the community.

OBTAINING HOSPITAL STAFF PRIVILEGES

Requirements for obtaining hospital staff privileges *vary* among institutions. The dentist must fulfill the following three basic requirements to become a hospital staff member:

1. The applicant must have graduated from an accredited dental school.
2. The applicant must be licensed to practice dentistry in the state in which the facility is located.
3. The applicant must have high moral and ethical standards.

Additional requirements may have to be met to obtain staff privileges. Many hospitals ask staff members to sign a "Delineation of Privileges" form indicating the procedures that staff members are qualified to perform and that are accepted by the governing body of the hospital. In addition, the applicant must show proof of professional liability insurance, and membership in the American Dental Association is desirable.

In a children's hospital, dentists might be required to have adequate advanced training to treat and manage children in the hospital. The requirements may include a dental residency of 1 to 4 years in a teaching hospital in which the dentist (1) gains experience in recording and evaluating the medical history and current medical status of children; (2) receives instruction in physical examination techniques and in recognition of conditions that may influence dental treatment decisions; (3) learns to initiate appropriate medical consultations when a problem arises during treatment; (4) learns the procedure for admitting, monitoring, and discharging children; and (5) develops proficiency in operating room protocol. A rotation in which the dental resident was actively involved in administering general anesthetics to children is highly desirable. Current certification in basic cardiopulmonary resuscitation should be maintained by all members of the hospital's professional staff, including dentists.

As active members of the hospital staff, dentists should be aware of the hospital's bylaws, rules, regulations, and meetings. A copy of the bylaws should be obtained for easy reference. Fully understanding the responsibilities of staff membership will enable dentists to treat their patients within the established protocol of the institution. Most important, dentists should endeavor to provide the highest quality care within the specialty area for which they are trained. The American Academy of Pediatric Dentistry encourages the participation of pediatric dentistry practitioners on hospital medical-dental staffs, recognizes the American Dental Association as a corporate member of the JCAHO, and encourages hospital member pediatric dentists to maintain strict adherence to the rules and regulations of the policies of the hospital medical staff.

INDICATIONS FOR GENERAL ANESTHESIA IN THE TREATMENT OF CHILDREN

The use of general anesthesia for dental care in children is sometimes necessary to provide safe, efficient, and effective care. Depending on the patient, this will be done in an ambulatory care setting or inpatient hospital setting. It should be only one component of the dentist's overall treatment regimen. Oral hygiene and preventive care must be implemented at the onset of treatment with parents or guardians and patients to eliminate the cause of the dental problem.

The safety of the patient and practitioner, as well as the need to diagnose and treat, must justify the use of general anesthesia. All available management techniques, including acceptable restraints and sedation, should be considered before the decision is made to use a general anesthetic. Crespi and Friedman cite several authors who agree in recommending that at least one or two attempts be made using conventional behavior management techniques or conscious sedation before general anesthesia is considered.'

Parental or guardian written consent must be obtained before the use of general anesthesia. Documentation regarding dental treatment needs, unmanageability in the dental setting, and contributory medical problems must be included in the patient's hospital record. Records must be clearly written so others are able to read and understand them. Review organizations examine dental admissions for proper documentation in the hospital chart for insurance payment and quality assurance purposes.

Patients for whom general anesthesia has been the management technique of choice include the following:

1. Patients with certain physical, mental, or medically compromising conditions
2. Patients with dental restorative or surgical needs for whom local anesthesia is ineffective because of acute infection, anatomic variations, or allergy
3. The extremely uncooperative, fearful, anxious, physically resistant, or uncommunicative child or adolescent with substantial dental needs for whom there is no expectation that the behavior will soon improve
4. Patients who have sustained extensive orofacial and/or dental trauma
5. Patients with immediate comprehensive oral or dental needs who otherwise would not receive comprehensive dental care
6. Patients requiring dental care for whom the use of general anesthesia may protect the developing psyche and/or reduce medical risks

Patients for whom general anesthesia is usually contraindicated include those with a medical contraindication to general anesthesia and healthy and cooperative patients with minimal dental needs.

PSYCHOLOGIC EFFECTS OF HOSPITALIZATION ON CHILDREN

Hospitalization is a frequent source of anxiety for children. According to King and Nielson, 20% to 50% of children demonstrate some degree of behavioral change after hospitalization.' Separation of the child from the parent appears to be a significant factor in posthospitalization anxiety, although other causes are also documented. Allowing the parent to stay with the child during the hospitalization, and especially to be present when the child leaves for and returns from surgery, can reduce anxiety for the child and parent alike.

According to Camm et al, postoperative behavioral changes reported by mothers in a limited sample of children who received dental treatment with general anesthesia in a hospital were similar to those observed in children who received treatment under conscious sedation in a dental clinic.' Mothers of children receiving dental treatment with general anesthesia in a hospital setting were found to experience more stress during the procedure. Ways to decrease these stresses include the following: providing a prior tour of the operating room facility, informing the parents of the status of the child during the procedure, and letting them know that "everything is all right." Seventy-five percent of the children receiving general anesthesia exhibited some type of behavioral change. Positive changes included less fuss about eating, fewer temper tantrums, and better appetite. Negative changes included biting the fingernails, becoming upset when left alone, being more cautious or avoiding new things, staying with the parent more, needing more attention, and being afraid of the dark. Ways to minimize negative changes include (1) involving the child in the operating room tour, (2) allowing the child to bring along a favorite doll or toy, (3) giving preinduction sedation, (4) providing a nonthreatening environment, (5) giving postprocedure sedation as needed, and (6) allowing parents to rejoin their children as early as possible in the recovery area.

To limit the severity and duration of psychologic disturbances, the dentist should strive to reduce parental apprehension concerning the operative procedure. Because children often sense apprehension in their parents, effectively reducing the parents' anxiety will put the child more at ease. Thoroughly explaining the procedure, describing the normal postanesthetic side effects, and familiarizing the child and parents with the hospital can reduce postoperative anxiety.

Peretz et al concluded that children treated for early childhood caries under general anesthesia or under conscious sedation at a very young age behaved similarly or better in a follow-up examination approximately 14 months after treatment than at their pretreatment visit, as measured by the Frankl scale and by the "sitting pattern."'

OUTPATIENT VERSUS INPATIENT SURGERY

During the past 25 years the popularity of outpatient anesthesia and surgery has continually increased. Currently more than 70% of all pediatric surgical and diagnostic procedures are performed on an *outpatient* basis. The criteria for and advantages of ambulatory general anesthesia procedures are well recognized. The increasing cost of inpatient hospital care, advances in anesthetic management, and quality assessment of patient care have led to changes in preoperative and postoperative management of many surgical procedures done under general anesthesia that were previously assumed to be possible only on an inpatient basis. Ambulatory care is more expeditious, better tolerated both by family and hospital teams, and less traumatic for the patient. Development of freestanding ambulatory care surgical centers (i.e., same-day surgery centers) and hospital ambulatory surgical care areas has cut health care costs for consumers and third-party providers. The advances in perioperative anesthesia care are related to the wider availability of more highly qualified anesthesia care providers (board-certified anesthesiologists with subspecialty training) and the availability of modern, safer short-acting anesthetic and adjuvant drugs and monitoring equipment. A number of studies have reported a significant decrease in anesthesia-related

morbidity and mortality in children during the past two decades.

Good patient selection is an important criterion of a successful *outpatient* surgery program. A young child or adolescent who requires a general anesthetic and is free of any significant medical disorders (i.e., is categorized as class I or II on the American Society of Anesthesiologists (ASA) physical status classification—see Box 14-1) can be considered a candidate for outpatient surgery. Certain patients with well-controlled chronic systemic diseases such as asthma, diabetes, and congenital heart disease can also be considered for outpatient anesthesia following prior consultation with an anesthesiologist.

When the outpatient surgery is planned, the child undergoes a complete preoperative evaluation, including a comprehensive medical history and physical examination, anesthesia assessment, and limited hematologic evaluation. Manv medical facilities allow this preadmission preparation to be performed outside of the medical outpatient treatment facility. Biery, Shamaskin, and Campbell suggest that routine laboratory tests, such as urinalysis and complete blood count with indices and electrolyte levels, are not cost effective nor are they necessary for patients categorized as ASA class I in whom the prior complete medical history and physical examination was unremarkable.[5]

As an outpatient, the child should be brought by the parents to the hospital at least $1^{1}/_{2}$ hours before the dental surgery. The nursing staff will verify that all preoperative instructions have been followed and that the appropriate laboratory tests have been performed. Several hours after the procedure is completed, the patient is released to the parent or guardian. Postoperative instructions are given, and a follow-up appointment is scheduled.

It should be recognized that the dentist will be more responsible for team communication, physical assessment, management, and postoperative evaluation for outpatient procedures under general anesthesia than for inpatient procedures. Ferretti reported that pediatric outpatient general anesthesia patients must have reliable parents or guardians to qualify for treatment.' For example, the parents must have transportation available to return the child to the hospital should post-operative complications develop at home.

The child should be treated as an *inpatient* if a medical condition exists that requires close follow-up, if the child lives outside the general area of the hospital, or if the parents demonstrate questionable ability to comply with preoperative or postoperative instructions. In many instances, medically or developmentally disabled patients with multiple problems requiring lengthy dental treatment are not good candidates for ambulatory care using general anesthesia. However, even some of these patients can be managed in an ambulatory setting when they are properly assessed and when no postoperative complications are anticipated.

MEDICAL HISTORY AND PHYSICAL EXAMINATION

Once the decision has been made that a general anesthetic would be preferable for a pediatric patient, the dentist should evaluate the child's medical history, the current medical status, and the possibility of complications resulting from the procedure. This risk assessment process is discussed in Chapter 14, and the patient-classification categories are shown in Box 14-1. The parents should be told of any potential complications, and their informed consent must be obtained (Fig. 15-1).

Intraoperative medical complications of dental patients with and without disabilities undergoing general anesthesia have been reported at 0% to 1.4%. In a survey of 200 pediatric dental general anesthesia cases, Enger and Mourino indicated that the most common postoperative complications following general anesthesia in children under the age of 5 were vomiting, fever, and sore throat.' Treatment of complications consisted of administration of antiemetic medications for nausea with vomiting, ice chips for sore throat, and acetaminophen (Tylenol) for fever postoperatively. Libman, Coke, and Cohen reported the most frequent postoperative general anesthetic complications of dental patients with disabilities to be hyperthermia.' Bradley and Lynch found that no significant long-term complications resulting from anesthesia or operative procedures were observed in 100 disabled and nondisabled patients.`'

The JCAHO requires that all patients admitted to a hospital have a physical examination performed by a physician or qualified dentist. The child's physician must therefore be consulted for the completion of a comprehensive medical history and physical examination (Box 15-1). Tf the physician is not a member of the hospital staff, a staff physician should complete the medical history and physical examination before admission. The dentist should perform a thorough intraoral examination and submit a record of the findings together with a summary of the child's dental history and the reason for admission (Box 15-2). The hospital must be notified to reserve an appropriate surgical suite and a bed for the child. Two weeks before admission, a letter containing general instructions concerning the procedure, results of the dental examination, and pertinent dates and times should be mailed to the parents (Fig. 15-2).

Obviously, before the administration of general anesthesia, a dental procedure may be canceled due to patient illness; productive cough, rhinitis, or wheezing; failure to comply with preoperative instructions such as abstention from eating or drinking; or risks related to coexisting diseases.

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Clarian Health
Methodist · IU · Riley

CONSENT FOR PROCEDURE

☐ Inpatient ☐ Outpatient

By signing this form, I agree that _____, M.D., his or her associates, members of the Clarian medical staff and appropriately licensed personnel of Clarian Health may perform the following operations or procedure. I understand that Clarian Health provides educational training for health care professionals and agree that residents and students may assist in my care.

Procedure: **Any necessary dental treatment under general anesthesia, including x-rays, prophylaxis, periodontal therapy, restorations, extractions, pulpal therapy, space maintainers, fluoride treatment and photographs.**

Exceptions, if any: **None** (If none, write "none")

- I acknowledge that I have had an opportunity to discuss with _____ M.D., my condition, the planned operation or procedure, its purpose and nature, reasonable alternatives, possible consequences of remaining untreated, risks, benefits and possible consequences.
- The procedure stated above is the one planned by my physician at this time. I realize my physician is not able to anticipate or explain all the possible risks and complications which could occur. I agree that additional procedures may be performed if my physician believes they become necessary during this procedure.
- I understand there are no guarantees that this procedure will be successful and that an undesirable result does not necessarily mean that an error was made.
- During this procedure I also consent to have any anesthetics given to me that the anesthesiologist or the physician performing the procedure believe are needed. I realize that there are medical risks associated with the anesthesia.
- If I am scheduled to receive blood or blood products, my physician has advised me of the risk and alternative therapies available. If it becomes medically necessary for me to receive blood or blood products, I agree to be transfused and accept the related risks.
- I agree to the disposal of any tissue, organ or body part removed during surgery. Provided that I am not identified, I agree that any material removed during surgery may be used for research purposes.
- I give my permission for pictures to be taken for educational purposes as long as my identity is not made known.

PHYSICIAN'S NOTE: Please list the most common and serious risks, benefits, reasonable alternatives and complications of the intended procedure(s) you discussed with the patient.

Common		Uncommon	Rare
Sore throat	Pain	Aspiration	Heart and lung complications
Nausea and vomiting	Swelling	Idiosyncratic drug reaction	Cardiac depression and arrest
Bleeding	Iatrogenic trauma	Dental trauma	Life-threatening events are statistically remote
	Sleepiness		
	Separation anxiety		

ADDITIONAL NOTES (*Optional*):

Signed: _____, M.D.

WITNESS TO SIGNATURE:

_____ Patient's Name: _____

Date: _____ _____
Patient's Signature *(see reverse side)*

Time: _____ AM / PM _____
Relationship or authority if not signed by the Patient

PHYSICIAN USE ONLY/DAY OF PROCEDURE

☐ I have verified the correct surgical/procedural site prior to incision.

Signed: _____, M.D. Date: _____

Medical Record Copy	CONSENT FOR PROCEDURE							M-1
B-CLIN. NOTES	E-LAB	G-X-RAY	K-DIAGNOSTIC	M-SURGERY	Q-THERAPY	T-ORDERS	W-NURSING	Y-MISC.

[I G **15-1.** Sample of form similar to one that may be used to obtain parental consent for dental treatment of a child under general anesthesia.

Continued

SIGNATURES REQUIRED

1. If the patient is an adult (age 18 or over) — signature of patient; or, if the patient is incompetent, the guardian's signature.

2. Minor patient (under age 18) — if emancipated (providing own support and living apart from the parents) patient's signature. If married, signatures of patient and spouse are required.

   Otherwise, signature of parent or guardian is required.

3. In an emergency threatening the life or well-being of the patient, and if signatures as required above are not available, there should be an entry in the chart documenting the emergency nature of the procedure and the need for prompt action, attested by the signatures of two physicians. Also, the signature of the closest adult relative should be obtained, if available.

f I f . 15-1, cont'd.

B 0 X 15-1 Components of the Pediatric Medical History and Physical Examination for Admission to the Hospital

A. Pediatric history
1. Identification: age, sex, racial-ethnic profile
2. Informant and estimate of reliability
3. Problem leading to admittance
4. History of present illness: date of onset, chronologic description of illness, presence or absence of previous similar episodes, treatment given prior to admittance
5. Medical survey
   a. Immunization against diphtheria, pertussis, tetanus, polio, measles, mumps, rubella
   b. Previous hospitalizations, operations, major illnesses, or injuries
   c. Allergies, including allergies to food and drugs
   d. Dietary history (under 2 years of age)
   e. Current medications
6. Developmental status
   a. Infants younger than 2 years: statement regarding motor and language development
   b. Preschool children: general statement regarding development
   c. Children in school: statement regarding school performance
7. Family history

B. Physical examination
1. Vital signs: Temperature, pulse, respiration, blood pressure if older than 12 months of age
   2. Measurements: weight, height or length, head circumference if younger than 12 months of age
3. General observations: nutrition, color, distress
4. Head: description of fontanel if present
5. Eyes: pupils, extraocular movements
6. Ears: tympanic membranes
7. Nose: patency, secretions
8. Mouth: teeth, pharynx, and tonsils
9. Neck: masses
10. Lungs: auscultation
11. Cardiovascular system: heart sounds, rate, rhythm, murmurs; femoral pulses
12. Abdomen: masses, viscera
13. Genitalia
    a. Male testes
    b. Female introitus
14. Skin: eruption
15. Lymph nodes
16. Skeleton: joints, spine
17. Nervous system: state of consciousness, gait (if walking)
18. Summary list of problems on tentative diagnosis

B 0 X 15-2 Components of the Dental History and Intraoral Examination to be Completed Before Hospitalization

1. Past dental history
2. Head and neck physical examination
   a. General
   b. Head
   c. Neck
   d. Face
   e. Lateral facial profile
3. Intraoral examination
   a. Lips
   b. Tongue
   c. Floor of mouth
   d. Buccal mucosa
   e. Hard and soft palate
   f. Oropharynx
   g. Periodontium
4. Teeth
   a. Caries
   b. Eruption sequence
   c. Occlusion molar, cuspid, overbite, overjet, and midline
5. Oral habits
6. Behavior
7. Recommendations

INDIANA UNIVERSITY

July 7, 2003

SCHOOL OF DENTISTRY

Mr. and Mrs. Smith
75 Mulberry Lane
Indianapolis, Indiana

Dear Mr. and Mrs. Smith:

RE: William Smith

After evaluating William and discussing with you the extent of his dental disease, it has been decided to accomplish all necessary dental care in the hospital. A medical history and physical examination must be completed by your physician, Dr. Charles Brown, prior to William's admission to the hospital. It has been scheduled for him at 9:00 a.m. on August 9, 2003, at Dr. Brown's office. You should also bring your child to the hospital at 10:00 a.m. on August 10, 2003, for admission.

You are encouraged to remain with your child overnight if possible. Your child may bring his favorite toy or book. If any cold symptoms (runny nose or congestion) should develop before the scheduled admission, please contact our office immediately. It is better to postpone the general anesthesia procedure if your child has a cold.

If you have any further questions concerning this procedure, please contact our office.

Sincerely,

John J. Doe, D.D.S.

John J. Doe, D.D.S.

JJD/db

DEPARTMENT OF
ORAL FACIAL DEVELOPMENT

UNIVERSITY PEDIATRIC
DENTISTRY ASSOCIATES

702 Barnhill Drive
Indianapolis, Indiana
46202-5200

317-274-9604
Fax: 317-278-0760

Located on the campus of
Indiana University
Purdue University
Indianapolis

[I G. **15-2.** Sample letter sent to parents 2 weeks before their child's admission to the hospital.

ADMISSION TO THE HOSPITAL

If the child is to be admitted as an inpatient, the child and parents should report to the hospital the day before the surgery. If the medical history is less complex, however, the patient may be admitted for "23-hour observation." In this instance the child may come to the hospital the same day as the operative procedure and stay postoperatively until the next morning. The parents must complete the necessary forms for admission to the hospital. The dentist must write the child's admission orders, which give the nursing staff the preliminary information needed and outline the basic care procedures for the child (Fig. 15-3). The nursing staff will explain standard hospital procedures to the parents and make any recommendations needed to foster a comfortable experience for the patient.

During this time the child will be visited by the anesthesiologist involved in the anticipated procedure. The anesthesiologist will assess the child's present state of health and review the past and present hospital records, focusing on prior exposures to general anesthetics and any complications that may have occurred. The anesthesiologist will explain the procedures involved during his or her part of the procedure and answer any questions that the child or parent might have. The decision regarding how long to keep the child off solid foods and liquids before the procedure is also determined by the anesthesiologist and may vary for younger patients to prevent hypoglycemia (Fig. 15-4).

The evening before surgery the dentist should visit the child in the ward. At this time the dentist can answer any questions the parents or the child might have. The dentist should also evaluate the preoperative laboratory data so that appropriate consultations can be initiated if any abnormal values are found. The dentist should record an admitting note in the medical chart to provide the supporting staff with a concise record of the child's medical history, current medical and oral status, diagnosis, and proposed treatment (Box 15-3). Abbreviations can be used when information is recorded in the medical chart (Box 15-4).

On the morning of the procedure the dentist and the staff should be in the operating room area 30 minutes before the start of the procedure. The dentist should evaluate how the child spent the previous evening, check that preanesthetic medications have been administered, and determine that the child has been given nothing by mouth *(nil per os* INFO]) for the recommended time. An appropriate note should be made in the medical chart. If all information is found acceptable, the child is ready to be taken to the surgical suite.

1. Please admit (patient's name) to ward for comprehensive dental care under general anesthesia.
2. CBC and differential, platelet, bleeding time, PT, PTT, (if indicated)
3. UA (if indicated)
4. Lateral and PA chest films (if indicated)
5. History and physical examination by medical house officer (if not already completed)
6. Routine diet per age (special diet if indicated)
7. Weight of patient
8. Bedside privileges for parents
9. Restraints (if indicated)
10. Activity for patient (up ad lib, BRP with assistance, and so forth)
11. Continue present medications (list all normally taken, dosages, and times given), for example:

phenytoin	50 mg	po	bid
phenobarbital	60 mg	po	bid

12. Notify anesthesiology of admission for preoperative evaluation and medication
13. Medication for pain, infection, or sleep (if indicated)
14. Consultations (if indicated)
15. Arrange for transport to the operating room on (date) at (time)
16. Contact me if problems develop

Answering service phone number: 555-5000
Home phone number: 555-7886
Signature: John J. Doe, D.D.S.

f I G **15-3.** Dentist's admission orders for a patient.

OPERATING ROOM PROTOCOL

All persons involved in the care of patients in the operating room must follow Occupational Safety and Health Administration (OSHA) guidelines. They must wear appropriate attire designed to prevent contamination of the surgical suite, hallways, and recovery room. This generally consists of a shirt, pants or skirt, and coverings for the face, head, and feet. A hood is used to cover all unshaven facial hair. Eyeglasses, goggles, or a face shield must be used to protect the surgeon's eyes, and a mask must cover the mouth and nose.

The dentist and staff should be familiar with the standard scrub technique for sterile procedures. Neither the medical nor the dental literature documents that a sterile technique is more advantageous than a modified sterile, or clean, technique for restorative dental procedures. Therefore intraoral dental procedures are generally considered clean procedures rather than sterile procedures. However, the dentist should wear sterile gloves. A sterile gown is worn at the discretion of the dentist. It is important to remember that barrier technique should be followed to prevent cross-contamination between patients in the hospital.

PROPERTIES OF INHALATION GENERAL ANESTHETICS

All inhalation anesthetic agents produce anesthesia by depressing specific areas of the brain. The magnitude of

CLARIAN HEALTH
Preoperative Care Center
Riley Day Surgery

NPO Guidelines Policy

All patients will receive specific instructions regarding preanesthetic dietary limitations during the preoperative phone call. These instructions will be appropriate and specific for the age of the patient and the time for which the procedure is scheduled. Patients with a history of neurological impairment, gastroesophageal reflux or delayed gastric emptying time may require a longer NPO period. Before giving instructions to these patients an anesthesiologist must be consulted.

Guidelines

1. The following time frames will be used in setting the NPO instructions for a particular patient. (See attachment for age-specific table.)

Clear liquids	Up to 2-3 hours before surgery
Breast milk	Up to 4 hours before surgery
Infant formula and nonhuman milk	Up to 6 hours before surgery
Light meal	Up to 6-8 hours before surgery

2. If procedure is scheduled for 0815, use the times for 0830; if scheduled for 0845, use the times for 0900.
3. Clear liquids are those you can see through such as water, Gatorade, Kool-Aid, apple or white grape juice, carbonated drinks, etc. They **do not** include milk, gelatin, ALL other fruit juices, formula, or cereal.
4. Light meals include items that can be easily digested such as soup, ice cream, toast, cereal, and crackers. They **do not** include steak, potatoes, pizza, eggs, bacon, sausage, or other heavy breakfast food.
5. Meals that include fried or Fatty foods or meat may prolong gastric emptying time. Both the amount and type of foods ingested must be considered when determining an appropriate fasting period. Following the guidelines does not guarantee a complete gastric emptying has occurred.

Revised 4/8/02 SL

Gopal Krishna, MD
Director of Pediatric Anesthesia
Riley Hospital for Children

f **I [15-4.** Nothing by mouth *(nil per os [NPO]),* times according to age.

B0X **15-3** Components of Dentist's Admitting Note on the Medical History-Physical-Progress Note Form

1. Name, age, sex, race, chief complaint, and rationale for admission
2. History of the present illness
3. Past medical history
4. Present medications (list all with dosages and times given)
5. Results of current laboratory tests
6. Documentation of informed consent and physical examination
7. Impression of case (intraoral examination, diagnosis, and prognosis)
8. Plan for treatment
9. Dentist's signature

depression is proportional to the partial pressure of the inhalation agents reaching specific sites in the central nervous system (CNS) after entering through the lungs and being distributed by the circulation to the tissues. The resulting physiologic signs of CNS depression produced by general anesthetic agents have been described by Guedel and modified by Roberts as stages of anesthesia with ether (Fig. 15-5).[1] " Guedel's classification of the stages of anesthesia is largely of historical interest, however. The modern inhaled anesthetics are extremely potent. Induction of anesthesia occurs quickly, and passage through the stages of anesthesia is quite rapid.

Techniques of inhalation anesthesia vary with the type of equipment used (i.e., reservoir bag, directional valves), chemical absorption of expired carbon dioxide, and rebreathing of expired gases. The techniques range

BOOK 15-4 Abbreviations Commonly Used in the Hospital

ac	Before meals *(ante cibos)*
ad lib	At liberty or at pleasure
anom	Anomalies
AP	Anteroposterior
aq	Aqueous, water
B/F	Black female
bid	Twice a day
B/M	Black male
BP	Blood pressure
BRP	Bathroom privileges
BSS	Black silk sutures
BUN	Blood urea nitrogen
bx	Biopsy
c	With (cum)
C	Celsius (formerly centigrade)
cap	Capsule
CBC	Complete blood count
cc	Cubic centimeter (liquid: milliliter)
CC	Chief complaint
CHD	Congenital heart disease
CNS	Central nervous system
congen	Congenital
CP	Cerebral palsy
CR	Cardiorespiratory
CV	Cardiovascular
d/c	Discontinue
Dent	Dental
diff	Differential blood count
disch	Discharge
D5W	5% dextrose in water
Dx	Diagnosis
ECG	Electrocardiogram
El	Elixir
ER	Emergency department
FH	Family history
FUO	Fever of unknown origin
Fx	Fracture
GA	General anesthesia
ging	Gingiva
gtt	Drops *(guttae)*
h	Hour
Hct	Hematocrit
HEENT	Head, eyes, ears, nose, and throat
Hg	Mercury
hist	History
HPI	History of present illness
hs	Just before sleep *(hora somni)*
I & D	Incision and drainage
IM	Intramuscular
I & 0	Intake and output
IV	Intravenous
kg	Kilogram
L	Left
mand	Mandible
max	Maxilla
M	Molar, in moles
MCH	Mean corpuscular hemoglobin
MCHC	Mean corpuscular hemoglobin concentration
MCV	Mean corpuscular volume
meds	Medication
N	Normal
NC	Noncontributory
neg	Negative
N_20	Nitrous oxide
NPO	Nothing by mouth *(nil per os)*
NSA	No significant abnormality
n/v	Nausea and vomiting
OOB	Out of bed
op	Operation
OPD	Outpatient department
OR	Operating room
ox	Mouth (os)
PA	Posteroanterior
pc	After meals *(post cibos)*
PE (or Px)	Physical examination
ped	Pediatric
PH	Past history
PI	Present illness
PMH	Past medical history
po	By mouth *(per os)*
postop	Postoperative
preop	Preoperative
prep	Prepare
prn	As required *(pro re nata)*
pro time	Prothrombin time
Pt	Patient
PT	Physical therapy
PTT	Partial thromboplastin time
Px	Physical examination
q	Every *(qua quo)*
qd	Every day
qh	Every hour
qid	Four times a day *(quater in* die)
qn	Every night
qod	Every other day
qs	Sufficient quantity *(quantum sufficiat)*
RHD	Rheumatic heart disease
RO (or R/O)	Rule out
ROS	Review of symptoms
RR	Respiratory rate
RR	Recovery room
RSR	Regular sinus rhythm
Rx	Take (or prescription) *(recipe)*
s	Without *(sine)*
SBE	Subacute bacterial endocarditis
SC	Subcutaneous
SH	Social history
SH	Serum hepatitis
S/P	Status post
Stat	At once *(statim)*
r	Rectal
R	Right

BOX 15-4 Abbreviations Commonly Used in the Hospital—cont'd

surg	Surgery	WBC	White blood count
Sx	Signs and symptoms	WD	Well developed
tbsp	Tablespoon	W/F	White female
tid	Three times a day	W/M	White male
TPR	Temperature, pulse, and respiration	WN	Well nourished
Tx	Treatment	WNL	Within normal limits
UA	Urinalysis	w/o	Without

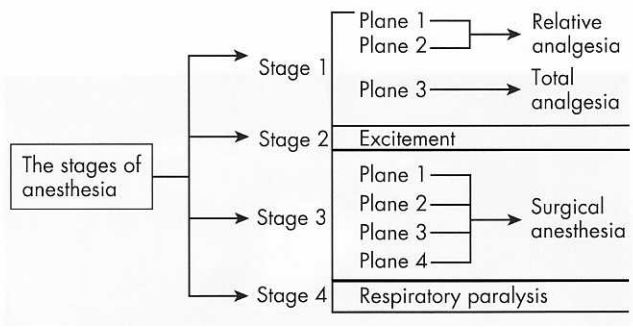


FIG . **15-5.** Stages of anesthesia observed with ether. *(Adapted from Roberts GJ: Relative analgesia in clinical practice. In Coplans MD, Green RA, editors:* Anaesthesia and sedation in dentistry, *vol 12, Amsterdam, 1983, Elsevier Science.)*

from insufflation to open or nonbreathing systems, semiopen systems, semiclosed systems, and closed systems. The semiclosed system is most often used in modern anesthesia. Exhaled gases mingle with fresh gas and are rebreathed after all the carbon dioxide is removed by a chemical absorber. Inhaled gases are humidified and a reservoir bag or ventilator allows assisted respiration. Reduced loss of body heat and water vapor, increased economy of flow, and decreased environmental contamination are advantages of the semiclosed system.

An anesthetic's potency is defined as the concentration of the agent required to inhibit response to a standard surgical stimulus. The potency is expressed in terms of the minimal alveolar concentration (MAC) value for the agent. The MAC of a given agent will abolish the response to stimulus in 50% of patients. Expressed as a number, MAC values are additive when different agents are used in combination. MAC is very useful, since it provides an estimate of the anesthetic requirement for each patient. Fine adjustment of anesthetic administration can then be made by monitoring the patient's physiologic responses (such as heart rate, blood pressure, and respiratory rate). MAC values and some other properties of inhalation anesthetics are listed in Table 15-1.

Available inhalation anesthetics include nitrous oxide, halothane, enflurane, isoflurane, desflurane, and sevoflurane. In children, because they dislike needles, anesthesia is commonly induced by inhalation of a halogenated volatile anesthetic via a face mask. For decades, halothane enjoyed widespread popularity as an induction agent in children. More recently, sevoflurane became available and in comparison to halothane has a lower blood/gas partition coefficient (producing more rapid induction and emergence) and is associated with less myocardial depression and fewer and less significant respiratory problems when used for inhalational induction. As a result, sevoflurane has become the agent of choice for inhalational induction. For maintenance of anesthesia, halothane, sevoflurane, isoflurane, and desflurane are all acceptable.

ANESTHETIC PREPARATION OF THE CHILD

After donning operating room attire, the dentist should report to the surgical suite and inform the anesthesiologist of any special requests concerning the procedure prior to induction of anesthesia. Nasotracheal intubation is preferred to ensure good access to the oral cavity. Oral tracheal intubation is not contraindicated, however, and can be used in a dental case with minimal restorative needs. The anesthesiologist should be careful to avoid complications during induction (i.e., laryngospasm, tooth avulsion or aspiration, traumatic intubation, compromised airway, malignant hyperthermia).

The anesthesiologist is responsible for starting intravenous fluids (IV), securing the necessary monitoring equipment, performing the intubation, and stabilizing the endotracheal tube. The anesthesiologist will select the type of IV fluid and calculate the estimated fluid replacement and fluid deficit volumes, and perform a physical assessment of dehydration. Since the patient is NPO, 5% dextrose lactated Ringer solution (D5LR) is initially used. The monitoring equipment should include (1) a precordial stethoscope, (2) an automatic sphygmomanometer, (3) electrocardiographic leads, (4) a temperature-monitoring device, (5) a pulse oximeter, and (6) a capnography device. The anesthesiologist must confirm that the child is in a stable condition for anesthesia and that the equipment is functioning properly (Fig. 15-6).

TABff **15-1** Some Physical and Chemical Properties of Commonly Used Inhalation Anesthetics

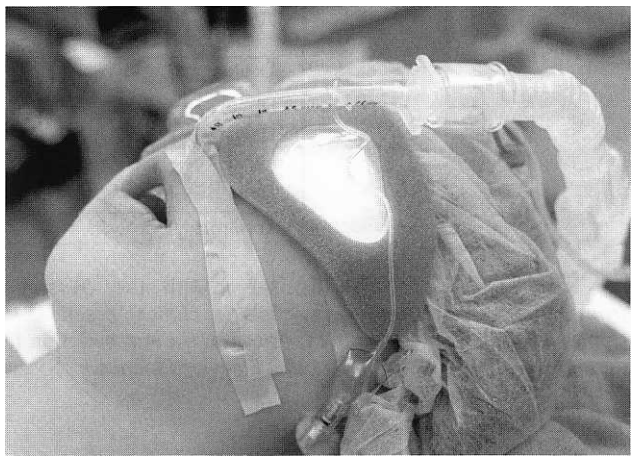
	VAPOR PRESSURE (MM HG) AT 20° C	BLOOD GAS PARTITION COEFFICIENT AT 37° C	MINIMUM ALVEOLAR CONCENTRATION IN ADULTS
Nitrous oxide		0.47	104
Halothane	243	2.5	0.76
Enflurane	175	1.91	1.7
Isoflurane	238	1.43	1.15
Sevoflurane	157	0.63	2.1
Desflurane	664	0.42	6.0

Courtesy Dr. Gopal Krishna.



f **I i 15-6.** Patient is in a stable anesthetic condition and ready for the dental procedure. Notice the position of the precordial stethoscope, blood pressure cuff, and nasotracheal tube.

Special care is taken to protect the child's eyes (Fig. 15-7). In addition, a shoulder roll is placed, padding is added to the patient's pressure points, the endotracheal tube and head are stabilized, heating or cooling blankets are used as needed, and the safety belt is secured. The dentist has the table positioned to conduct



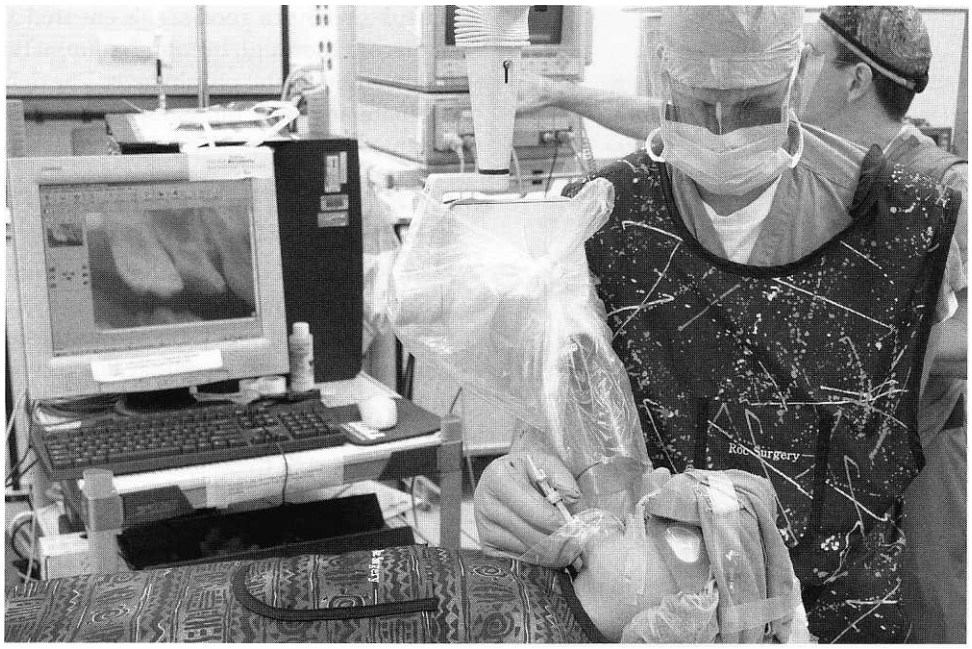
f I G. **15-7.** Special eye guard protects the patient's eyes during the procedure.

dental procedures, and the anesthesiologist administers any preoperative IV medications requested.

Before scrubbing, the dentist should obtain any necessary preoperative radiographic studies. All persons involved in the radiologic procedure should wear protective lead apparel. Radiographs of excellent quality can be made while a patient is under general anesthesia without exposing the patient or staff to unnecessary radiation (Fig. 15-8). Digital radiographs are advantageous due to decreased radiation exposure and immediate image feedback.

PERIORAL CLEANING, DRAPING, AND PLACEMENT OF PHARYNGEAL THROAT PACK

Before the dental procedure is begun, the perioral area is cleansed with three sterile 4 x 4-inch gauze pads. The first gauze pad is saturated with a bacteriostatic cleansing agent, the second with sterile water, and the third with alcohol (Fig. 15-9). This procedure is not intended to sterilize the area but only to remove gross debris (Fig. 15-10). A surgical sheet is then positioned over the remainder of the child's body. This helps to maintain



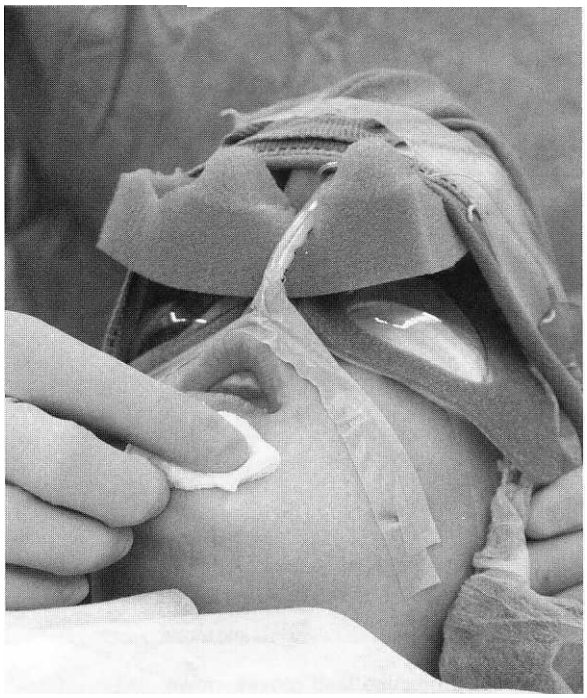
[I G. 15-8. Obtaining diagnostic radiographs. Notice the use of protective lead gloves, gown, and apron.



FIG 15-9. Materials required for perioral cleaning. *From upper left:* pharyngeal throat pack, towel clamps, patient drapes, bacteriostatic cleaning agent, sterile water, and alcohol.

body temperature and provides a clean field during the procedure. The head is draped with three towels arranged to form a triangular access space for the mouth. The towels are secured in place with towel clamps or hemostats. The mouth should be fully exposed (Fig. 15-11). The anesthesiologist may request that part of the nasotracheal tube remain exposed so that all connections can be easily monitored. The assistants then place all supporting carts and stands around the table in positions that the dentist finds comfortable and efficient (Fig. 15-12).

The patient's mouth is opened with the aid of a mouth prop. Care should be taken not to impinge on the lips or tongue with the prop (Fig. 15-13). The mouth is



f 11. **15-10.** Special care must be taken during perioral cleaning to prevent materials from entering the oral cavity.

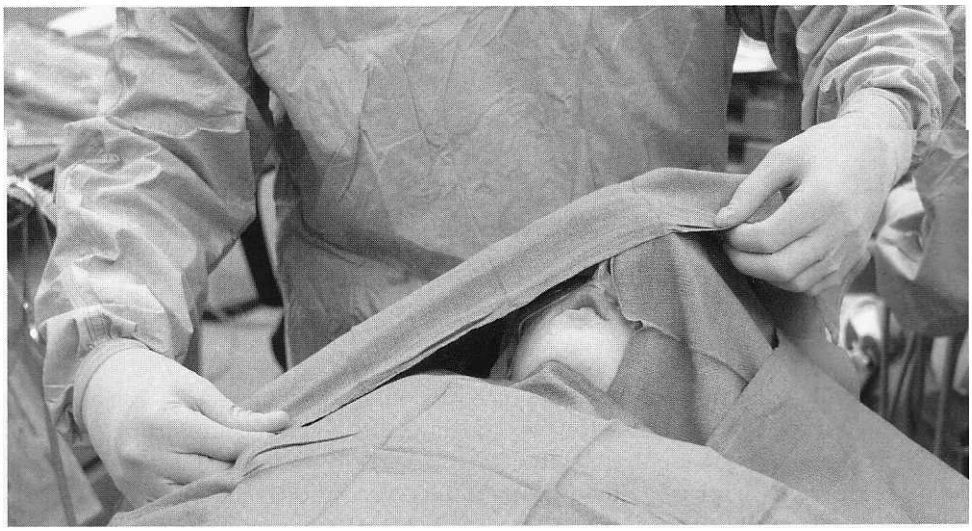
thoroughly aspirated. The pharyngopalatine area is sealed off with a strip of moist 3-inch sterile gauze approximately 12 to 18 inches long (Fig. 15-14). Written documentation of throat pack placement and removal is required on the physical history form of the medical chart. This packing reduces the escape of anesthetic agents and prevents any material from entering the pharynx. The gauze should be tightly packed around the tube, so that a good seal is ensured. Once the pack is in place, a thorough intraoral examination is performed, followed by dental prophylaxis. The dentist should then evaluate any new radiographic studies that have been obtained and formulate a final treatment plan.

RESTORATIVE DENTISTRY IN THE OPERATING ROOM

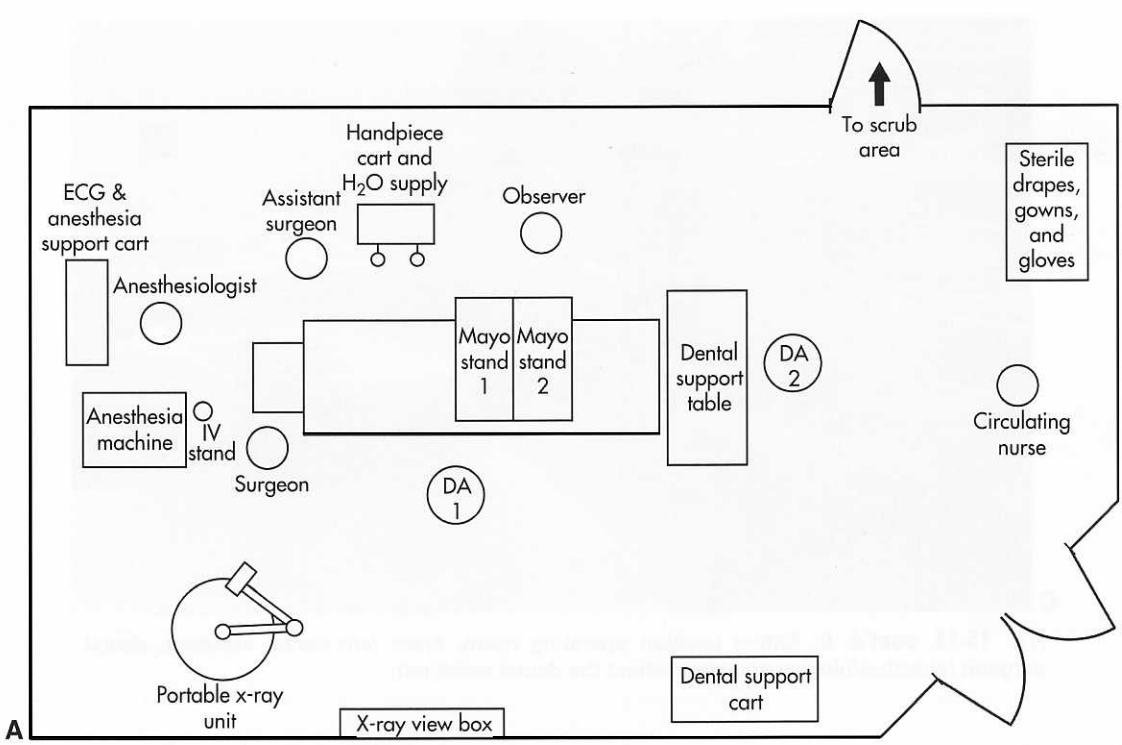
Instruments used for restorative dental procedures in the operating room are the same as those for procedures in the dental operatory. Local anesthesia may be used to minimize pain and bleeding. The use of quadrant isolation with a rubber dam is preferred (Fig. 15-15). After the completion of the procedures for each quadrant, a topical fluoride treatment should be applied before the removal of the rubber dam. Written documentation of blood loss and hydration in the medical chart is required.

Restorative dental care under general anesthesia allows excellent patient compliance and easy achievement of a well-lighted field, and therefore increases the quality and quantity of dental care while decreasing the anxiety level for the clinician and patient during dental treatment. Spiro and Burns found that they were able to treat seven teeth per hour in children under general anesthesia compared with only three teeth per hour in children of similar age in a clinic setting.[Z] Eidelman, Faibis, and Peretz reported that the quality of restorative treatment performed was better under general anesthesia than under conscious sedation."

The dentist should place restorations that will pro-vide the greatest longevity with the least amount of maintenance, for example, full-coverage stainless steel crowns rather than large amalgam restorations on



fl G 15-11. Placement of the surgical sheet and triangular draping of the oral cavity area. The nasotracheal tube is exposed to allow easy monitoring of its connections.



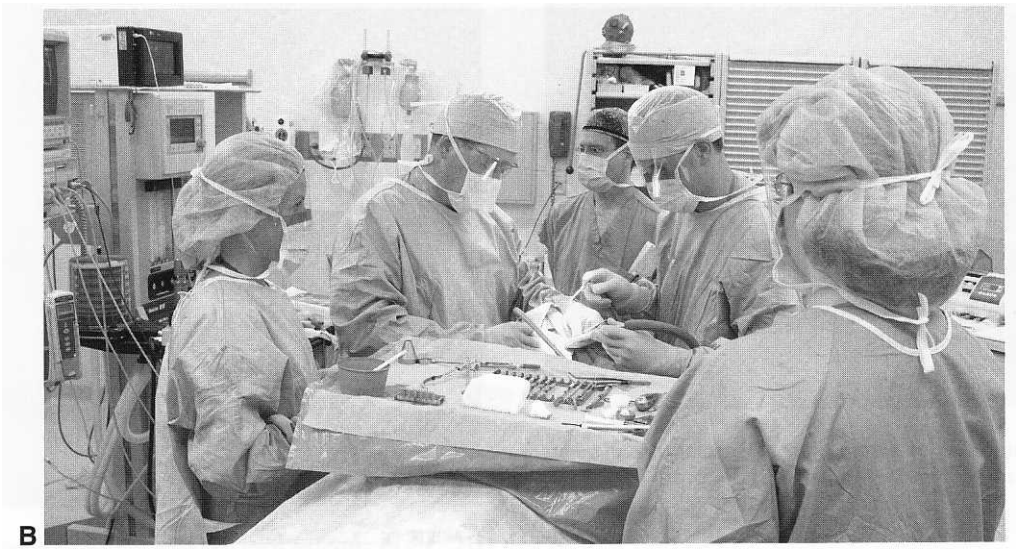
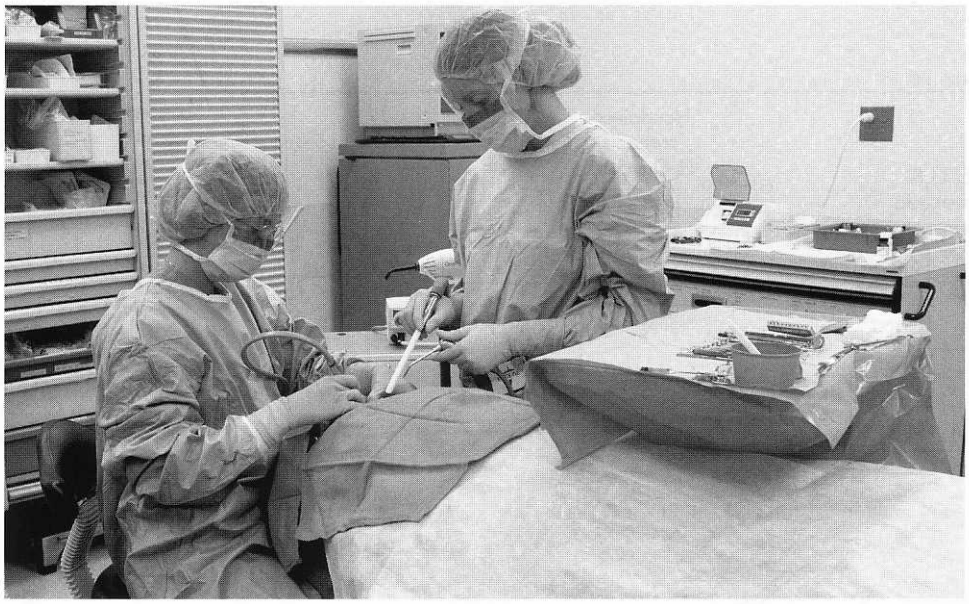
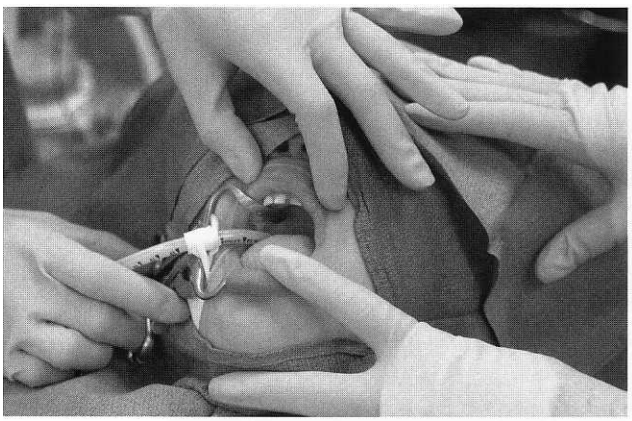


FIG 15-12. **A,** Schematic drawing of one example of the positioning of personnel and equipment in the operating room. DA, Dental assistant; *ECG,* electrocardiograph; *IV,* intravenous. **B,** Operating room positions of the staff while performing the necessary dental procedures. *From left:* dental surgeon, anesthesiologist, assistant dental surgeon, and dental assistant.



C

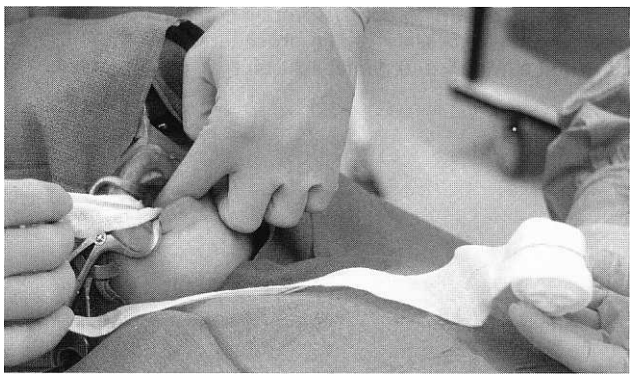
f I G 15-12, Contd. C, Sitting position operating room. *From left:* dental assistant, dental surgeon (anesthesiologist is hidden behind the dental assistant).



f **I G 15-13.** Positioning of a mouth prop. Special care is taken not to impinge on the lips or tongue with the prop.



f **I G 15-15.** Rubber dam isolation of the maxillary left quadrant.



f **G 15-14.** Placement of the pharyngeal throat pack.

posterior primary teeth. In a 3-year study of comprehensive dental cases treated under general anesthesia, O' Sullivan and Curzon found stainless steel crowns to be significantly more successful (3% failure rate) than an amalgam or composite restoration (29% failure rate).[14] A 6-month retrospective study by Tate et al to assess the failure rates of dental restorative procedures performed under general anesthesia by pediatric dental residents found stainless steel crowns to be best (8%), followed by amalgam (21%) and composite (30%); composite strip

crowns (51%) had the lowest successful restorative treatment rate."

Intraoperative anesthesia complications (e.g., dislodged or obstructed endotracheal tube, IV infiltrates or disconnects, nasal bleeding or edema of the tongue and lips, and arrhythmias) rarely occur. Care should be taken by the dentist not to displace the endotracheal tube. Priority is given by the dentist to the anesthesiologist to treat any adverse event quickly, even to the point of stopping treatment and removing rubber dams to allow access.

COMPLETION OF THE PROCEDURE

The anesthesiologist should be notified 10 minutes before the completion of the procedure so that the child can begin to be aroused and preparations can be made for extubation. The recovery room personnel are notified that the child will soon be arriving so that they can begin preparations. On completion of the dental procedure, the oral cavity is thoroughly debrided and the throat pack is removed carefully to prevent aspiration of any materials that might be lodged against it. The dentist verbalizes a needle and sponge count and removal of the throat pack to the nurse. At this time the anesthesiologist assumes responsibility for the child. The dentist should remain in the operating room during the extubation process to assist the anesthesiologist if necessary. When the child is transported to the recovery room, the dentist should accompany the anesthesiologist and provide assistance during the transportation.

POSTANESTHESIA CARE UNIT

When the child arrives in the postanesthesia care unit or recovery room, the dentist should inform the nursing staff of the procedures accomplished and of any special requests or instructions. If teeth have been removed, the nurse should be specifically instructed how and where to apply gauze packs for hemostasis. The nurses and other medical staff are available to deal with immediately postoperative complications if they should occur (i.e., fever, nausea, vomiting, croup, hypoxia, bleeding, and laryngospasm). After the dentist has confirmed that the airway is patent and the vital signs are stable, and after the anesthesiologist is confident the child is recovering well, the dentist should meet with the parents or guardian to provide a brief report of the child's condition and a review of the treatment. The parents or guardian of *inpatients* should be informed of the approximate time the child will be transported to the ward. The parents or guardian of *outpatients* should be informed of the time to meet the child in the recovery area. Prescriptions may be written for pain control agents (i.e., acetaminophen with codeine, or ibuprofen suspension) antibiotics (i.e., amoxicillin, clindamycin), or antiemetics for nausea (i.e., prochlorperazine, ondansetron [Zofran]).

POSTOPERATIVE CARE

Postoperative orders and the operative note for the staff should be completed by the dentist and recorded in the medical chart while the child is in the postanesthesia care unit (Fig. 15-16 and Box 15-5). The operative report should be dictated as soon after the completion of the procedure as possible (Box 15-6).

If the child was treated as an *outpatient,* once the child is awake and alert, displays appropriate behavior, maintains his or her own airway, has stable vital signs, has no uncontrolled bleeding or pain, is voiding, and has no retention of liquids, a decision is made to release the child rather than keep the child overnight for further evaluation. If the child is to be kept for 23-hour observation *(inpatient),* an appropriate note is recorded in the medical chart and a discharge summary is dictated (Box 15-7) after the child is released. Postoperative instructions and necessary prescriptions are given to the parents or guardian, and an observation appointment for the child is arranged before the discharge. The dentist must be available that evening (a contact number must be given) in the event the parents or guardian need assistance in caring for the child after returning home. These instructions and prescriptions help the patient deal with common complications after discharge, such as fever, nausea, vomiting, pain, and bleeding.

Effective communication is essential at this time to reaffirm the parents' or guardian's cooperation in performing oral health care and in keeping follow-up observation appointments and recall appointments. Enger and Mourino reported that only 57% of dental patients undergoing a treatment in the operating room returned for 6-month recall visits.' They suggest that procedures requiring monitoring (i.e., space maintenance) be postponed if the patient's return is unlikely.

A study by Almeida et al of future caries susceptibility in children with early childhood caries following dental treatment under general anesthesia found that, despite implementation of increased preventative measures for these children, they were still *highly predisposed* to greater caries incidence in later years.' The researchers concluded that more aggressive preventative therapies may be required in children who experience early childhood caries.

There are several categories of dental problems in children that cannot be handled well in the office setting and are best managed in the hospital or outpatient surgery center. The ability to treat children in the hospital environment and to provide comprehensive dental care using a general anesthetic for such children is a valuable part of the dentist's treatment regimen. Granting hospital staff privileges to qualified dentists has become

A

Outpatient Orders

1. Vital signs per recovery room routine
2. D/C IV when released from recovery
3. Begin clear liquids when indicated in Day Surgery
4. Call surgery house officer at:
5. Return appointment:
6. Tylenol per standing order: 1 grain per year of age q4h prn
7. Release from Day Surgery
8. Call if problems arise: Pageboy number: 2001

   Office phone number: 274-8492

   Signature: John J. Doe, DDS.

B

Inpatient Orders

1. Maintain present IV solution (i.e., 5% dextrose with 1½ normal saline) at a specified rate (i.e., 40 mL/hr) and discontinue when empty or if it infiltrates (longer if indicated)
2. Vital signs q15 minutes until stable, then q2h per recovery room routine
3. Elevate head 30 degrees
4. For swelling: apply ice packs to area for 30 minutes
5. For hemostasis: check oral hemorrhage q15 minutes for 1 hour; if needed, apply 4 × 4 inch sterile gauze to area with pressure
6. Restraints prn (if needed)
7. Force fluids and encourage to void
8. Continue present antibiotic (if indicated)
9. Maintain present medications (list all normally taken, dosages, and times given)
10. Medication for nausea (if indicated)
11. Pain medication (if indicated) Tylenol per standby order: 1 grain per year of age q4h prn
12. Liquid diet per age, as patient tolerates (diet as indicated)
13. Moist air with mist tent if patient suffers from croup
14. Apnea monitor (if indicated)
15. Call if needed: Office phone number: 274-3865

    Answering service phone number: 274-5000

    Signature: John J. Doe, DDS.

FIG. 15-16. Components of the dentist's postoperative orders for a patient. A, As an outpatient. **B,** As an inpatient.

BOX 15-5 Components of the Dentist's Operative Note

1. Title of dental procedure
2. Type of intubation and anused
3. Teeth restored
4. Teeth extracted
5. Other procedures completed
6. Dental prophylaxis and topical fluoride used
7. Summary (length of procedure, how the child tolerated the procedure, blood loss, complications, and so forth)
8. Prognosis
9. Dentist's signature

BOX 15-6 Components of the Dentist's Operative Report

1. Doctor's name and assistants' names
2. Patient's name and hospital number
3. Preoperative diagnosis
4. Postoperative diagnosis
5. Title of the operative procedure
6. Preparation for anesthesia (preoperative medications, type of intubation, and anesthetic agents used)
7. Surgical procedure
   a. Radiographs taken
   b. Description of scrub, draping procedure, and throat pack
   c. Number of teeth restored and type of restorations
   d. Number of teeth receiving pulp therapy
   e. Teeth extracted (name each)
   f. Gingival therapy procedures
   g. Band(s) for appliance(s) and impression(s)
   h. Dental prophylaxis and fluoride applied
   i. Type and amount of intraoperative fluids used
   j. Other information (if indicated)
8. Estimated blood loss and hemostasis
9. Condition of the patient at the conclusion of the surgical procedure (complications incurred if indicated)
10. Condition of the patient on arrival in the recovery room
11. Prognosis

BOX 15-7 Components of the Dentist's Discharge Summary Statement

1. Patient's name and hospital number
2. Date of admission
3. Date of discharge
4. Date of dictation
5. Preoperative diagnosis
6. Postoperative diagnosis
7. Age, race, and sex of patient
8. Reason for admission and treatment using general anesthetic
9. Results of preoperative history and physical examination (medical and dental) and present medications
10. Name of the physician completing the history and physical examination
11. Complete description of the surgical procedure (see Box 15-6)
12. Patient's tolerance in the recovery room and ward
13. Condition of patient on discharge
14. Individual to whom patient is discharged
15. Home care instructions given to parents and medications prescribed (dosage and times to be given)
16. Patient's next appointment
17. Copies of discharge summary sent to patient's physician and referring dentist or physician

routine at many hospitals seeking to provide comprehensive health care for the community. The dentist who uses the hospital or outpatient surgery center in the care of patients often finds it to he a rewarding component of practice.

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CHAPTER

16

Dental Materials

B. KEITH MOORE

DAVID R. AVERY

انتشارات
شایان نمودار

CHAPTER OUTLINE

MICROLEAKAGE AND BIOLOGIC CONSIDERATIONS
CAVITY VARNISHES
CEMENT BASES
AMALGAM
- Selection of the Alloy
- High-Copper Alloys
- Mercury/Alloy Ratio
- Trituration
- Mechanical Amalgamators
- Condensation
- Moisture
- Marginal Breakdown and Bulk Fracture
- Bonded Amalgam Restorations
- Mercury Toxicity

CEMENTS
LUTING CEMENTS
- Zinc Phosphate Cement
- Polycarboxylate Cement
- Glass Ionomer Cement

RESIN-MODIFIED GLASS IONOMER CEMENTS
ZINC OXIDE—EUGENOL
RESIN CEMENTS
TEMPORARY AND PERMANENT RESTORATIONS
RESTORATIVE RESINS
- Conventional Composites
- Microfilled Composites
- Small-Particle and Hybrid Composites
- Light-Cured Composites
- Posterior Composite Restoration
- Resin Inlays
- Acid-Etching Technique
- Bonding Agents

Although the past 30 years have seen a marked reduction in the dental caries rate in children, restorative dentistry remains an important part of pediatric dentistry. Development continues on improved restorative materials and manipulation techniques for restorative dentistry for the general population. As these become available to the pediatric dentist they may displace materials that have been used for many years. Much current effort addresses the growing awareness, expectations, and concerns of the dental consumer. Aesthetics has become a major consideration in choice of materials and procedures. Concern is also increasing about the biocompatibility of materials from the standpoint of the patient and the environment. Research has resulted in an ever-increasing body of knowledge related to the behavior of dental materials and an avalanche of new products. These developments place a continuing responsibility on the dentist, who must critically analyze the literature and the claims of manufacturers to determine which materials and techniques will provide optimal service to the patient. For intelligent and best choices to be made, an appreciation of the clinical significance of the chemical, physical, and biologic properties of dental materials is essential. This provides the justification for the content of the present chapter.

The oral cavity is a formidable obstacle to maintenance of the integrity of tooth structure and the materials used in its restoration or replacement. Biting stress on the cusp of a molar tooth may be as high as 207 MPa (30,000 psi). The pH of saliva, dental plaque, foods, and beverages fluctuates daily from very acid to alkaline. The temperature of a meal may vary as much as 150° F (66° O. The warm, moist oral cavity contains a variety of enzymes and debris, providing optimum conditions for the accumulation of surface deposits that can degrade restorations and tooth structure. For these and other reasons, restorative materials are readily subject to fracture, solubility, dimensional change, and discoloration. If these problems are to be minimized, a dental material must possess certain minimum chemical and physical properties. Furthermore, those properties must be maintained during the manipulation and placement of the restoration and for its projected lifetime in service.

The American Dental Association (ADA)/American National Standards Institute Specification Program has contributed greatly to providing the dentist with high-quality dental materials that have been carefully developed to resist the rigors of the oral cavity. Many commonly used restorative materials are encompassed by this certification program. Because the specifications are based on in vitro tests that are believed to correlate with clinical performance, the ADA also maintains an acceptance program that is based on usage tests for safety and efficacy. Newly developed classes of materials often are covered by this program in the interim until a specification can be developed. As an example, an acceptance program exists for dental adhesive systems. If a certain type of material is covered by a specification or acceptance program, those materials on the list of certified or accepted products that is available on the web site of the ADA (http://www.ADA.org) should be considered first. From that list the dentist should be able to select a brand that provides the desired manipulative characteristics.

In a like manner to the ADA, the Federation Dentaire Internationale has been instrumental in the development of international specifications under the auspices of the International Standards Organization (ISO). The dental materials market has become international, and developments such as the creation of the European Union suggest that international specifications for dental materials will play the major role in the twenty-first century in selection of dental materials.

The Medical Devices Amendments of 1976 to the Food and Drug Act gave the U.S. Food and Drug Administration (FDA) regulatory authority to protect the public from hazardous or ineffective medical and dental devices. Some dental products with claims for therapeutic effects (such as fluoride products) are considered drugs, but most dental materials used professionally are considered devices and are subject to regulation by the FDA Bureau of Medical Devices. Also included are over-the-counter dental products sold to the public, such as floss and denture adhesives.

In addition to the issue of the safety of dental materials and devices for the patient, other safety issues have been raised in recent years that have dramatically influenced the practice of dentistry. Concern about the transmission of infectious disease during dental procedures has led state and federal agencies to develop rigorous standards for infection control. These standards have placed new demands on dental materials, devices, and equipment. Today, most of these must be capable of being subjected to disinfection procedures and, in some cases, sterilization without loss of properties. Delivery systems for materials have been significantly influenced. Most materials are now packaged for unit-dose delivery at chairside, which makes infection control much easier than when bulk containers of materials are used.

The second safety issue to come to the forefront is the safety of individuals working in the dental office. Federal and state occupational safety and health administrations (OSHAs) are responsible for developing and enforcing standards to ensure safety in the workplace. Dental offices that employ any person besides the dentist fall within the regulatory scope of these agencies. Many of the dental materials commonly used in restorative dentistry are considered to present an occupational

hazard. One of the federal OSHA regulations requires the dentist to have on file a materials safety data sheet for every material—from impression materials to the cleaning fluids used in the office. All employees must be informed about the nature of any hazardous materials to which they may be exposed and must receive training in the safe handling of these materials. Information to assist the dentist/employer is available from sources such as the *ADA Regulatory Compliance Manual,* first published in 1989 and continuously updated as new regulations are developed.

Environmental concerns also have affected the practice of dentistry. State and federal environmental protection agencies are giving scrutiny to all waste discharged from the dental office, whether solid, liquid, or gas. Disposal of hazardous materials, such as toxins or biohazards, is regulated and the level of such regulation can be expected to grow. In most cases the generator of waste material remains legally responsible even if another party has been paid to dispose of it.

All of the concerns mentioned can seriously affect the practice of dentistry and require on the part of the dentist an increasing body of knowledge about dental materials and a constant vigilance to stay current with developing regulations.

MICROLEAKAGE AND BIOLOGIC CONSIDERATIONS

Possibly the greatest deterrent to the development of an "ideal" restorative material is the leakage that occurs along the restoration-tooth interface. There is as yet no truly adhesive dental material. No restorative dental material exactly duplicates the physical properties of tooth structure. Changes in temperature and mechanical stress can result in the development of gaps at the tooth-material junction. Moreover, no matter how proficient the dentist may be, no restoration exactly fits the prepared space in the tooth. Overwhelming evidence shows that all restorative materials permit ingress of deleterious agents, such as acid, food debris, and microorganisms, between the walls of the prepared cavity and the restoration. A certain incidence of the clinical failure of materials can be associated with this phenomenon. Microleakage may be the precursor of secondary caries, marginal deterioration, postoperative sensitivity, and pulp pathology. With restorations, microleakage often results in unsightly marginal discoloration and necessitates replacement of the restoration. Microleakage poses a particular problem for teeth in the pediatric patient because the floor of the cavity preparation may be close to the pulp. The added insult to the pulp caused by the seepage of irritants that penetrate around the restoration and through the thin layer of dentin, or a microscopic pulpal exposure, may produce irreversible pulp damage.

One method of bonding substances together is entirely mechanical. A liquid adhesive is used that will flow into irregularities in the surfaces being bonded and then solidify. In dentistry, acid etching is commonly used to accomplish bonding of a restorative resin to enamel by the formation of resin tags into the etched enamel, as discussed in this chapter and elsewhere in this text.

For true adhesion to occur, bonding must take place at a molecular level and must involve a chemical interaction between the molecules of the adhesive and the adherend. The only dental materials currently in use that have the potential for true adhesion to tooth structure and an established clinical record for success are based on polyacrylic and other polyalkenoic acids. These materials are the polycarboxylate and glass ionomer cements.

With the advancements that have been made in surface chemistry and the development of adhesives for all types of unusual applications, the dentist may ask why other adhesive dental materials have not been developed. Availability of a truly adhesive restorative material would greatly alter many phases of dental practice. The need for the typical cavity preparation would no longer be a prime consideration because adhesion would eliminate the need for mechanical retention by the cavity preparation. It would no longer be necessary to use auxiliary aids, such as cavity varnishes and etching techniques, to minimize the microleakage around direct filling restorations. The compromised tooth could actually be reinforced by the restorative material, which would reduce the need for full occlusal coverage to protect the tooth.

Unfortunately, tooth structure possesses numerous undesirable characteristics as a substrate for bonding of an adhesive. It is rough, inhomogeneous in composition, covered with a tenacious layer of surface debris, and wet. These factors discourage adhesion. Furthermore, the reactivity (surface energy) of enamel is low, and therefore the surface does not easily attract other molecules to it.

It has been shown that topical fluoride applications reduce even further the surface energy of enamel. Although this may be beneficial because it can reduce plaque accumulation and hence caries, it also interferes with the wetting of the tooth surface by a liquid adhesive.

On the other hand, the surface energy of most restorative materials, particularly metallic ones, is higher than that of normal intact tooth structure. Therefore, debris accumulates on the surface of restorations more than on the adjoining enamel. This could, in part, account for the surprisingly high incidence of secondary caries associated with most restorative materials, except for those that release fluoride ion. Debris accumulation can promote marginal deterioration by the loss of tooth

structure or restorative material at the interface. Such deterioration would normally increase microleakage.

CAVITY VARNISHES

Cavity varnishes have been used empirically for many years as a liner for cavity preparations. When varnish is painted onto the cavity preparation, the solvent evaporates and leaves a thin resin film. The varnish may reduce microleakage when it is used with certain restorative materials. Cavity varnish may reduce initial microleakage around amalgam. Since varnish films are very thin and do not adhere to tooth structure, some clinicians have replaced cavity varnish with one of the dental adhesives currently marketed. In theory, if such a material adheres to dentin and enamel, any leakage should occur between the amalgam and the adhesive. The results of laboratory studies provide limited support for this theory, although a controlled clinical study by Mahler et al has shown no reduction in postoperative sensitivity with the use of dentin adhesives as liners for amalgam restorations.

CEMENT BASES

The function of the cement base is to promote recovery of the injured pulp and to protect it against further insult. The base serves as a thermal insulator and replaces missing dentin when it is used under the metallic restoration. The base must be of sufficient thickness to provide effective insulation. A minimum of approximately 0.5 mm is required for this purpose.

A base must be able to support the condensation of the restorative material placed over it. If the strength of the base is inadequate, it may fracture during condensation and permit amalgam to penetrate and come into contact with the dentin floor, which thereby compromises the thermal protection afforded by the base. Zinc phosphate, hard-setting calcium hydroxide, zinc oxide—eugenol, and glass ionomer cements have sufficient strength to serve effectively. In certain cases, such as a class II preparation that involves the restoration of an angle or of a deep depression, it may be necessary to cover a calcium hydroxide base with a layer of stronger zinc phosphate or glass ionomer cement.

AMALGAM

Controversy regarding the safety of the dental amalgam restoration has existed since the material was introduced to the profession more than 150 years ago. Periodically this controversy surfaces in the press and other news media and becomes a matter for public, as well as professional, debate. As a result, the dentist who uses dental amalgam can expect questions to be raised by patients and their guardians and also can expect requests for replacement of intact amalgam restorations with other materials.

Amalgam is no longer the most commonly employed material for restoring posterior carious lesions. Tooth-colored restorative materials, such as composite resins and resin-modified glass ionomers, are seeing increased usage based primarily on considerations. The popularity of dental amalgam likely will continue to decline as the longevity of these other materials and their suitability as general amalgam replacements in the permanent dentition is demonstrated.

The unique clinical success of amalgam during 150 years of use has been associated with many characteristics. It is likely that its excellent clinical service, even under adverse conditions, is attributable to the tendency for its microleakage to decrease as the restoration ages in the oral cavity. Although amalgam does not bond to tooth structure and the margins of an amalgam restoration may appear open, the restoration-tooth interface immediately below the exposed margin becomes filled with relatively insoluble corrosion products that inhibit leakage. Amalgam is unique from this standpoint. The microleakage around other restorative materials usually increases with time. Amalgam is the least technique sensitive of all current direct restorative materials. One of the factors slowing the acceptance of posterior composite resin restorations has been the very exacting clinical technique and time required for placement. Another unique property of amalgam as a direct filling material is its lack of dimensional change during hardening. The ADA specification for dental amalgam limits maximum acceptable dimensional change to ±0.2%. If this is compared with a typical value of *2.0%* or higher for the polymerization shrinkage of a resin matrix composite material, the potential impact on microleakage is obvious.

Nevertheless, failures of amalgam restorations are observed. These may occur in the form of recurrent caries, fracture (either gross or severe marginal breakdown), dimensional change, or involvement of the pulp or periodontal membrane. More significant than the type of failure is its cause. Two factors that lead to such clinical failures are improper design of the prepared cavity and faulty manipulation. In other words, the deterioration of amalgam restorations often can be associated with neglect in observing the fundamental principles of cavity design or abuse in preparing and inserting the material.

One other factor also is involved, and that is the choice of the alloy used.

SELECTION OF THE ALLOY

Several criteria are involved in the selection of an amalgam alloy. The first criterion is that the alloy should meet the requirements of the ADA Specification No. 1 or the corresponding ISO specification for dental amalgam alloys.

The manipulative characteristics of dental amalgam are extremely important and a matter of subjective preference. Rate of hardening, smoothness of the mix, and ease of condensation and finishing vary with the alloy. For example, the resistance felt with lathe-cut amalgams during condensation is entirely different from that with spherical amalgams. The alloy selected must be one with which the dentist feels comfortable, because the operator variable is a major factor influencing the clinical lifetime of the restoration. Use of alloys and techniques that encourage standardization in the manipulation and placement of the amalgam enhances the quality of the service rendered. Coincident with this is the delivery system provided by the manufacturer—its convenience, expediency, and ability to reduce human variables.

Obviously the physical properties should be reviewed in the light of claims made for the superiority of one alloy over competing products. Ideally such a list of properties should be accompanied by documented clinical performance in the form of well-controlled clinical studies. Although the cost of the alloy is a factor, this criterion should not be overemphasized when balanced against the alloy's ability to render maximum clinical service. The dentist should always weigh the fraction that the material cost contributes to the total charges for a dental procedure when making price comparisons between brands, particularly when comparing a brand with documented clinical performance against a generic brand of material.

Dental amalgam alloys generally are available as either small filings called *lathe-cut* alloys or spherical particles called *spherical alloys.* Spherical alloys tend to amalgamate very readily. Therefore amalgamation can be accomplished with smaller amounts of mercury than required for lathe-cut alloys, and the material gains strength more rapidly. Also, the condensation pressure and technique employed by the dentist in placing the restoration are somewhat less critical in achieving the same properties of the amalgam. This is an advantage in difficult clinical situations in which optimal access for condensation is limited. Spherical amalgam alloys have a somewhat different feel during condensation and require less condensation pressure than lathe-cut alloys. The dentist and auxiliary should familiarize themselves with the handling characteristics of a new alloy before clinical restorations are placed.

HIGH-COPPER ALLOYS

The original dental amalgam alloys were alloys of silver and tin with a maximum of 6% copper. When significantly more copper is available, improved laboratory properties and clinical performance has been demonstrated. This improvement has been attributed to the displacement of the tin-mercury reaction product with a copper-tin phase during the amalgamation reaction. Alloys that contain enough copper to eliminate the formation of the tin-mercury phase (11% to 30%) are called high-copper amalgam alloys. The first such alloy of this type was an *admixed* system. Small spherical particles of a silver-copper alloy were added to filings of a conventional silver-tin alloy. High-copper alloys also can be made using *single composition* particles. Each of these alloy particles has the same chemical composition, usually silver, copper, and tin. Amalgams made from high-copper alloys have low creep. *Creep is* the tendency of a material to deform continuously under a constant applied stress. This property has been associated with the marginal breakdown (ditching) commonly noted with amalgam restoration. Although the ADA specification for dental amalgam permits a maxi-mum of 3% creep, a modern high-copper amalgam alloy should not exceed 1% creep.

Choice of amalgam alloy today should be limited to high-copper alloy systems.

Regardless of the alloy used, manipulation plays a vital role in controlling the properties and the clinical performance of the restoration.

MERCURY/ALLOY RATIO

Most of the properties of amalgam restorations have been shown to depend on the relative amount of mercury contained in the finished restoration (the residual mercury). One of the variables that control the final mercury content is the amount of mercury used to mix the amalgam.

Although dental amalgam alloy still may be available in the form of powder or preweighed compressed pellets and bulk mercury can be dispensed by volume, most of the amalgam alloy sold today is in the form of prefilled, disposable mixing capsules containing the proper amounts of alloy and mercury. This delivery system should be used for several reasons. The alloy/mercury ratio is accurately preproportioned. The need for disinfection procedures is minimized because the capsule system is discarded after use. Most importantly, exposure of dental personnel and environmental contamination by mercury vapor is minimized. These prefilled capsules are usually available for different size mixes, often called *single* or *double spill capsules.*

TRITURATION

The second manipulative variable that controls the residual mercury content is trituration. Trituration time can significantly influence both consistency and working time of the mixed amalgam. These in turn relate to the ability to bring excess mercury to the surface during condensation. The correct trituration time varies depending on the composition of the alloy, the mercury/alloy ratio, the size of mix, and other factors.

The best practice is to acquire an appreciation for the appearance of a proper mix and then to adjust the trituration time accordingly. The most serious error in amalgamation generally is undertrituration. An undertriturated mix appears dry and sandy and does not cohere into a single mass. Such an amalgam will set too rapidly, which results in a high residual mercury content, reduced strength, and the increased likelihood of fracture or marginal breakdown. Properly mixed amalgam is a shiny, coherent mass that can be readily removed from the capsule.

MECHANICAL AMALGAMATORS

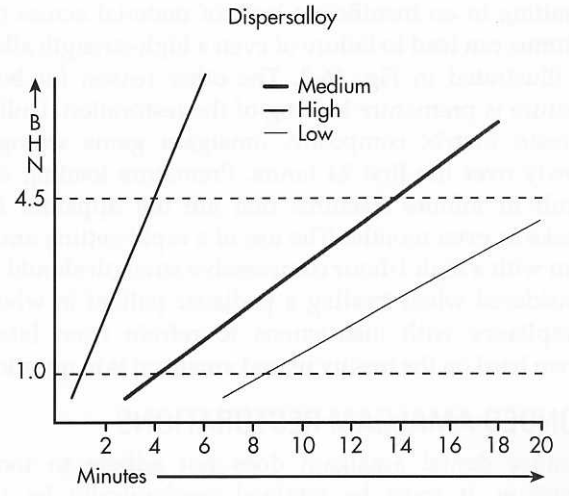
When first introduced, mechanical amalgamators for dental amalgam operated at a single speed that was usually below 3000 cpm. High-copper alloys in prefilled, self-activating capsules are designed for shorter trituration times at higher trituration speeds. Failure to activate these capsules reliably results in undertrituration and is a common problem with the use of older single-speed amalgamators. Because amalgamators also deteriorate with time, replacement of an older unit with a new high-speed amalgamator is desirable. A unit that allows multiple speeds of operation should be selected, because numerous other products such as dental cements are now marketed in capsules to be mixed in a dental amalgamator. The trituration times suggested by the amalgam alloy supplier are starting points. Amalgamators may vary in operating speed even within the same brand, and a unit's performance may vary with line voltage or the number of times it is used in rapid succession. Trituration speed, as well as time, significantly influences the rate at which some amalgams harden, as seen in Fig. 16-1.

CONDENSATION

The purpose of condensation is to adapt the amalgam to the walls of the cavity preparation as closely as possible, to minimize the formation of internal voids, and to express excess mercury from the amalgam. Within reasonable limits, the greater the condensation pressure, the lower the amount of residual mercury left in the restoration and the greater the strength of the restoration. The selection of the condenser and the technique of "building" the amalgam should be designed to achieve those objectives, as described in detail in textbooks of operative dentistry, and should be tailored to the handling characteristics of the type of amalgam alloy chosen, as previously discussed.

MOISTURE

Moisture contamination of an amalgam restoration can promote failure. If zinc is present in the alloy, it will react with water, and hydrogen gas will be formed. As this gas builds up within the amalgam, a significant



f **I G 16-1.** The influence of amalgamator speed (low-medium-high) on the hardening rate of a high-copper amalgam alloy as measured by the Brinell Hardness (BHN). BHN = 1.0 indicates the working time and BHN = 4.5 indicates the carving time. *(Redrawn from Brackett W: Master's thesis, Indianapolis, 1986, Indiana University School of Dentistry.)*

delayed expansion can occur and may cause protrusion of the amalgam from the cavity preparation, which enhances the possibility of fracture at the margins.

Such moisture contamination can result from failure to maintain a dry field during the placement of the restoration. Exposure to saliva *after* the amalgam has been completely condensed is not harmful. It is only moisture incorporated within the amalgam as it is being prepared or inserted that must be avoided.

Zinc-free alloys are available, and their physical properties are generally comparable to those of their counterparts that contain zinc. A zinc-free, high-copper alloy should be used when the dentist operates in a field
where moisture control is difficult.

MARGINAL BREAKDOWN AND BULK FRACTURE

Because dental amalgam is a brittle material, a commonly observed type of amalgam failure is the restoration in which the marginal areas have become severely chipped. The exact mechanisms that produce this breakdown of the amalgam or the adjoining tooth structure are not established, but it is likely that the deterioration is precipitated by manipulation and technique of finishing rather than by dimensional changes during setting.

If the restoration is improperly finished by the dentist, a thin ledge of amalgam may be left that extends slightly over the enamel at the margins. These thin edges of such a brittle material cannot support the forces of mastication. In time they fracture, leaving an opening at the margins.

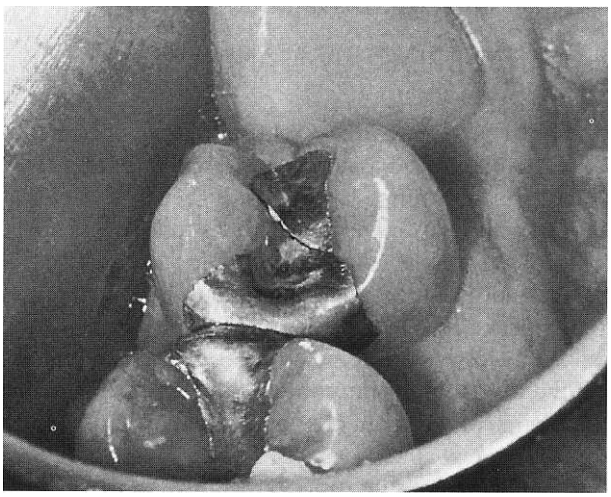
Bulk fracture of amalgam is much less common with high-copper amalgam alloys. Those cases that do occur likely have one of two causes. Poor cavity design

resulting in an insufficient bulk of material across the isthmus can lead to failure of even a high-strength alloy, as illustrated in Fig. 16-2. The other reason for bulk fracture is premature loading of the restoration. Unlike a resin matrix composite, amalgam gains strength slowly over the first 24 hours. Premature loading can result in minute fractures that are not apparent for weeks or even months. The use of a rapid-setting amalgam with a high 1-hour compressive strength should be considered when treating a pediatric patient in whom compliance with instructions to refrain from biting down hard on the freshly placed amalgam is in question.

BONDED AMALGAM RESTORATIONS

Because dental amalgam does not adhere to tooth structure, it must be retained mechanically by the design of the cavity preparation and/or mechanical devices such as pins. The placement of an amalgam does not strengthen the compromised remaining tooth structure and subsequent fracture may occur, particularly in molar teeth with relatively large mesiodistocclusal amalgam restorations. The use of dental adhesive systems, as described in detail in the section related to resin composites, as lining materials for amalgam has been suggested in an attempt to create a "bonded amalgam restoration." Several products are marketed specifically for this purpose. In general, they are chemically activated dentin-bonding systems over which the amalgam is condensed *before the resin adhesive has hardened.* This results in an intermixing of the unset resin and the plastic amalgam at the interface and forms a mechanical bond as both materials harden. It is important to distinguish this application from the use of a dental adhesive to seal the dentin surface and reduce early microleakage as previously discussed. When dental adhesives are used to seal the dentin surface, the adhesive should be polymerized before the amalgam is placed. Although bond strengths reported in laboratory studies between amalgam and dentin are lower than the maximum reported for resin composite bonded to dentin, they are in the same range. In vitro studies also show that teeth restored with bonded amalgams are more resistant to fracture than those in which amalgam is placed without a bonding adhesive.

A caution must be added that these are relatively short-term laboratory studies. Some longer-term clinical data are available; however, little is known about the potential influence of embedding the resin into the bulk of the amalgam on the long-term properties of the restoration. At the present time, amalgam bonding should be considered only as an adjunct for conventional, accepted practices of cavity preparation and mechanical retention of amalgam.



f**IG 16-2.** Bulk fracture of an amalgam restoration. Such failure may occur from improper cavity design or premature occlusal loading. *(From Anusavice KJ:* Phillips' *science of dental materials, ed 11, St Louis, 2003, WB Saunders.)*

MERCURY TOXICITY

The amalgam restoration is possible only because of the unique characteristics of mercury. Mixing this liquid metal with the alloy powder provides a plastic mass that can be inserted into the tooth and then hardens rapidly to a structure that resists the rigors of the oral environment. As the restoration hardens, mercury reacts with silver and tin to form stable, intermetallic compounds. Most of the public controversy about the safety of dental amalgam has focused on the hazards associated with elemental mercury and some of its organic compounds. Many substances commonly regarded as quite safe contain extremely dangerous elemental ingredients. No one would ever consider human ingestion of elemental sodium or chlorine, but ordinary table salt, which is the compound sodium chloride, is an important dietary substance. From the time of the earliest use of amalgam, it has been asked whether mercury in a dental restoration can produce local or systemic toxic effects in humans. It is periodically conjectured that mercury toxicity from dental restorations is the cause for numerous illnesses of unknown etiology.

The possibility of toxic reactions by the patient to traces of mercury penetrating the tooth or sensitization from mercury dissolving from the surface of the amalgam is remote, however. The danger has been evaluated in numerous studies. The patient's encounter with mercury vapor during insertion of the restoration is brief, and the total amount of mercury vapor is too small to be injurious. Furthermore, the amount of mercury released from the amalgam in service is small compared with other sources of mercury from air, water, and food. Metallic mercury in the human digestive track is apparently not converted to lethal organo-mercury compounds and is excreted by the body.

Both the National Institutes of Health and the FDA have examined the evidence for risk of dental restorative materials to the patient. The conclusion was that, except for the very small fraction of the population with a true allergic reaction to mercury or other constituents of amalgam, the dental amalgam restoration remains a safe and effective treatment. No evidence was found that related the presence of amalgam restorations to disorders such as arthritis, multiple sclerosis, or other diseases in which amalgam has been implicated. It should be noted that no currently available restorative material is completely risk free and that patients should be informed of the relative risks associated with all dental treatment alternatives.

The question about the replacement of existing serviceable amalgams with other materials remains one of professional judgment. Both the ADA and some state dental licensing boards have found that a dentist who recommends replacement of amalgam restorations with other materials on the claim that this will improve the physical health of the patient may be acting unethically and may be subject to sanctions by licensing bodies and to suits for civil damages. Patients who feel that they have medical problems related to the presence of any dental restorative material should be referred to a physician for diagnosis and treatment recommendations.

What about dental office personnel? Restorative dentists and their office personnel potentially are exposed daily to mercury, even in offices in which amalgam restorations are not being placed. Although metallic mercury can be absorbed through the skin or by ingestion, the primary risk to dental personnel is from inhalation. A potential hazard exists for dentist and staff from chronic inhalation of mercury vapor in the dental clinic, although the few actual incidents reported have been related to poor technique in handling mercury. The maximum level considered safe for occupational exposure is 50 p.g of mercury per cubic meter of air averaged over a standard 8-hour workday. Mercury at room temperature has a vapor pressure almost 400 times the maximum level considered safe. This vapor has no color, odor, or taste and cannot be readily detected by simple means at the level of maximum safe exposure. Because liquid mercury is almost 14 times more dense than water, a small spill can be significant. Eliminating the use of bulk mercury by employing prefilled, disposable capsules should significantly reduce exposure to mercury vapor.

The dental operatory should be well ventilated. All mercury waste and amalgam scrap removed during placement or removal of amalgam restorations should be collected and stored in well-sealed containers. When amalgam is cut, water spray and high-speed evacuation should be used. More detailed recommendations can be obtained from the *Regulatory Compliance Manual* published by the ADA. The risk to dental personnel from mercury exposure cannot be ignored. However, adherence to simple hygienic procedures will ensure a safe working environment.

Waste materials containing mercury or amalgam scrap should be disposed of responsibly in accordance with the regulations of the environmental protection agency in the area in which the dentist works. These materials should not be incinerated or subjected to heat sterilization. Biologically contaminated wastes containing mercury, including extracted teeth, should be cold-sterilized with a chemical agent before disposal. The most significant threat to the continued use of dental amalgam will likely be from government regulations on environmental waste discharge. In Japan, use of amalgam has been discontinued because it is not feasible for a dental office using amalgam to meet restrictions on mer cury discharge into sewers. Amalgam-mercury separators on dental clinic wastewater discharge lines are now required in several countries in Europe. Local and state authorities should be consulted about limitations on discharge of mercury into wastewater from a dental practice.

CEMENTS

Dental cements have several functions in restorative dentistry. One is to serve as a luting agent to fill the space between a restoration fabricated outside the mouth and the tooth structure. By flowing into irregularities in both materials and then hardening, the cement provides mechanical retention as previously discussed. A second function is to serve as a filling material for either permanent or temporary restorations. Cements are also used as bases for other restorative materials as previously described.

Silicate cement was an early tooth-colored filling material. Although it is no longer used, its ability to reduce development of secondary caries has made it a model for the development of caries-resisting dental materials. Recurrent or secondary caries was seldom encountered around silicate cement restorations even when gross disintegration had occurred. Most other restorative materials have not shown such an ability to resist recurrent caries, which is today the most common cause for replacement of restorations.

This beneficial characteristic is attributed to the presence of fluoride in silicate cement powder, which typically contains approximately 15% fluoride. After placement of the silicate restoration, fluoride ion is released and reacts with the adjoining tooth structure in much the same manner as does topically applied fluoride. The enamel solubility is reduced, which builds up its resistance to acid attack and caries. Because there is evidence that the fluoride ions are released slowly throughout the life of the restoration, the protective mechanism is undoubtedly a continuous one.

LUTING CEMENTS

Several types of cement may be used as luting agents. Each has inherent advantages and disadvantages. Thus the selection of a particular type of cement is governed by the individual situation presented by the patient.

ZINC PHOSPHATE CEMENT

Formerly, zinc phosphate cement was the most widely used luting agent. Composed essentially of phosphoric acid liquid that is mixed with zinc oxide powder, the cement has excellent handling characteristics such as setting time, fluidity, and film thickness. Furthermore, this type of cement has a long history of successful application for permanent cementation. It does not have an anticariogenic effect, does not adhere to tooth structure, and demonstrates a moderate degree of intraoral solubility.

Because of the phosphoric acid liquid, zinc phosphate cement is an irritant, and proper pulp protection is recommended. In those situations in which experience indicates that sensitivity and pulp response are likely to be problems, use of a cement that is more biologically compatible, such as a polycarboxylate cement, is recommended.

POLYCARBOXYLATE CEMENT

Polycarboxylate cement is one of the few dental materials that demonstrate true adhesion to tooth structure. The powder is primarily zinc oxide, and the liquid is polyacrylic acid or a copolymer of that acid. Although the final pH of the set cement is comparable to that of zinc phosphate cement, its biologic properties are excel-lent. For this reason, polycarboxylate cement is useful as a base or as a luting agent, particularly when the cavity preparation is close to the pulp. In addition, as the cement sets against the tooth structure, a chemical bond is formed between the cement liquid and the calcium in the hydroxyapatite in enamel and dentin.

When the cement is used as a luting agent, several manipulative factors influence the wetting of the tooth by the cement and thereby retention of the restoration. After cavity preparation the enamel and dentin surfaces are covered with a thin layer of tenacious debris, referred to as the *smear layer*. Also the preparation may be covered by a thin film of material, such as zinc oxide—eugenol, if a provisional restoration was placed. Unless this contamination is removed, it may inhibit adhesive bonding of the setting cement to the tooth. One means of cleaning the surface is a 10- to 15-second swabbing with 10% polyacrylic acid.

As with all types of cement, the liquid should not be dispensed until just before the mix is to be made. To slow down the setting reaction and provide longer working time, a chilled mixing slab may be used. The powder and liquid should be mixed rapidly, and the mix should be completed within 30 seconds.

The recommended powder/liquid ratio should be used. If the mix is too thick, insufficient acid is present to produce bonding to the tooth. If excess liquid is used, the intraoral solubility increases significantly. When properly prepared, the mix has a glossy appearance and can be extruded into a thin film. It is important that minimal time elapse between completion of the mix and placement of the cement; the mix must not have lost its glossy appearance.

When polycarboxylate cement is used with cast restorations, the inside surface of the casting must be cleaned thoroughly. After the casting is cleaned in a pickling bath, the interior should be treated with an air abrasive or a fine stone. Polycarboxylate cement will not wet a chemically dirty surface. In time, leakage and loss of retention may occur along the cement restoration interface.

Although polycarboxylate cement demonstrates adhesion to tooth structure, it has a relatively low tensile strength, no significant fluoride release, and modest intraoral solubility. Good practices of tooth preparation should be used to insure retention of the restoration.

GLASS IONOMER CEMENT

Another type of cement that is based on polyacrylic acid is glass ionomer cement (GIC). Because of its biologic kindness, fluoride release, and potential for adherence to the calcium in the tooth (as with the polycarboxylate system), glass ionomer cement is used as a restorative material (type II) for treatment of the eroded area, as a luting agent (type I), and as a base and liner material (type III).

Like zinc polycarboxylate cement, the glass ionomer liquid is polyacrylic acid or another alkenoic acid, such as itaconic or maleic, with tartaric acid added to improve handling properties. The acid has the potential for bonding to calcium in the manner described for polycarboxylate. This chemical bond provides retention of the cement to the tooth.

The powder is a fluoro-aluminosilicate glass similar to silicate cement powder and displays fluoride release patterns similar to that of silicate cement. Data from glass ionomer restoration of class V erosion lesions for periods of more than 7 years indicates that GIC shows resistance to secondary caries. One can immediately see the attraction of the GIC system: it has a potential for adherence to tooth structure and possesses anticariogenic potential.

The material is supplied as a powder and liquid and is commonly preproportioned in a disposable capsule to be mixed in an amalgamator. With type I GIC, the liquid acid may be freeze-dried and combined in the powder. When this powder is mixed with water, the acid reconstitutes, which results in the same setting reaction. The freeze-dried products have better shelf life and

somewhat lower viscosity, which are important characteristics for luting cements.

The mix can be made either on a disposable, moisture-resistant paper pad or on a glass slab. A plastic spatula is preferred to a metal one to minimize contamination of the mix from abraded metal. As with polycarboxylate cement, the polyacrylic acid–based liquid is not dispensed until just before the start of the mix. The glass ionomer cements are mixed in a manner like that used for polycarboxylate cements: large increments of the powder are rapidly incorporated into the liquid, and the mix should be completed within 40 seconds. The working time is short, usually no more than 3 minutes from the start of the mix. In no instance should the material be used if the mix has lost its gloss or a skin has formed on the surface.

After setting, the material is more brittle than a polycarboxylate cement. It can be trimmed and finished in much the same manner as zinc phosphate cement. Before the patient is dismissed, all the accessible margins should be covered with the varnish or protective resin supplied by the manufacturer. This protects the cement from oral fluids and dehydration during the next few hours as the setting reaction continues.

Instances of postoperative sensitivity have been reported when GIC is used as a luting agent, particularly in deep preparations with minimal remaining dentin. This is possibly attributable to the low initial pH of the cement and its relatively slow set. To guard against potential irritation, in very deep areas calcium hydroxide should be placed. The cut dentin surface can be cleaned mechanically with pumice, but the smear layer should not be removed. After cleaning, the dentin should be rinsed and dried but not desiccated. A slightly damp surface appears to help minimize sensitivity and does not interfere with the setting reaction.

Glass ionomer luting cements have mechanical properties similar to those of zinc phosphate cements and lower intraoral solubility. Because of their potential for fluoride release and adhesion to tooth structure, they are becoming the most commonly employed luting cements for metallic restorations.

In addition to its use as a luting agent for cast restorations, GIC has been employed for bonding orthodontic brackets to acid-etched enamel. GIC has lower cohesive strength than do the resin orthodontic adhesives, but the fluoride release from the GIC should minimize the white spotting and decalcification sometimes seen around orthodontic brackets or bands. If orthodontic bands are employed on posterior teeth, the GIC is the luting agent of choice.

RESIN-MODIFIED GLASS IONOMER CEMENTS

The most recent addition to the cement field is the resin-modified glass ionomer cement. These cements are also sometimes referred to as *hybrid glass ionomers* or in the case of type II and III cements as *light-cured glass ionomers*. Disadvantages of conventional glass ionomers include short working time, slow development of ultimate properties, sensitivity to both moisture exposure and dehydration during setting, and lower cohesive strength compared with resin cements. These problems have been addressed by the development of resin-modified GIC. Resin monomers or a comonomer of acrylic acid and a methacrylate such as hydroxyethyl methacrylate in the same manner as light-activated restorative resin composites. The resin component hardens immediately on exposure to the light, which results in an initial set of the cement. The material then continues to undergo the acid-base GIC setting reaction that occurs more slowly than that of a conventional GIC; this results in a much longer working time for the light-cured glass ionomer. The rapid set after light exposure yields a material that is much less sensitive to dehydration or moisture. Type I resin-modified GIC luting cements are also available; in this case the resin component is either chemically activated or dual (chemical and light) activated. Resin-modified GIC type II restorative materials appear to exhibit the advantages of conventional GIC and have received rapid acceptance. The use of resin-modified GIC luting cements is less well established. Fracture of all-ceramic crowns cemented with resin-modified GIC has been reported, which prompts concern about their use with ceramic restorations.

ZINC OXIDE—EUGENOL

The acid-base reaction between zinc oxide and eugenol results in a cement that can be used as both a luting and restorative material. Because of its low strength and high oral solubility, zinc oxide–eugenol is not recommended as a permanent luting cement. However, because of its exceptionally kind biologic behavior, it is often used as a base material, as a temporary luting cement, and as a temporary restorative material. Eugenol is an inhibitor for additional polymerizing resins and can interfere with subsequent use of resin cements, restorative materials, and even impression materials.

RESIN CEMENTS

Resin luting cements are derived from the composite resin systems used for restorative materials. They may be viewed as lightly filled composites. The resin matrix systems used are the same as those employed for restorative resins. Although these materials are not new to dentistry, they have only recently become used extensively. Their first major clinical application was in direct bonding of orthodontic attachments to acid-etched enamel, for which they quickly became the materials of choice. Similar formulations were developed into pit and fissure sealants, which are discussed in Chapter 17.

The resin-bonded bridge such as the "Maryland" bridge is another application in which resin cements came to the forefront. The demand for dentistry has resulted in extensive use of both resin and ceramic veneers. Here, too, resin cements are the cements of choice. Finally, new technology for fabricating all-ceramic crowns and inlays has greatly increased the use of these restorations, which are normally cemented with resin cements. Resin cements have high strength, low film thickness, and very low oral solubility, and can be bonded to etched enamel, ceramics, resins, and etched or treated metal surfaces. With the advent of dentin adhesives, resin cements provide the possibility of bonded, indirect restorations.

Resin cements are usually available in different shades for color matching beneath translucent restorations, and opaque cements are made for masking metal substructure or discolored tooth structure. The first resin cements were two-component, chemically activated curing systems. Visible light-activated, single-component systems are now available and are popular when used with translucent restorative materials. Dual-activated materials, which are both chemical and light activated, are recommended for use beneath thick restorations and in locations where geometry may limit access to the curing light.

TEMPORARY AND PERMANENT RESTORATIONS

The temporary restoration should possess good biologic characteristics, have minimal solubility, and be rigid, strong, and resistant to abrasion. The relative importance of each of these properties depends on the degree of permanence desired. For example, in the carious mouth it is often desirable to remove some or all of the caries immediately and place temporary restorations. These restorations subsequently are replaced with more permanent restorative materials. In such situations it may be necessary for the temporary restoration to serve for several months or longer. Strength and abrasion resistance are of paramount importance in these cases. Usually, temporary restorations need to remain in place only for days. In the latter instance more emphasis may be placed on the biologic properties when a material is selected.

Because of its excellent tissue tolerance and ability to minimize initial microleakage, zinc oxide-eugenol cement has been commonly used for holding or inter-mediate restorations. The strength, rigidity, and resistance to abrasion of the conventional zinc oxide-eugenol mixture have been improved by the addition of polymers and by the surface treatment of the zinc oxide powder.

Type II glass ionomer cements or the newer resin-modified GICs also are useful as long-term, temporary restoratives, for example, in restoring eroded areas in patients when exposed areas of cementum and dentin are present. Because of its desirable biologic and adhesive characteristics, the GIC can be used to restore these lesions without the need for a retentive cavity preparation. If conventional GICs are employed as restorative materials, they must be protected from exposure to moisture in the early stages of setting and from dehydration for a very long time, likely the entire time the restoration will serve. In general, a resin-modified glass ionomer is a better choice for reasons given previously.

GIC formulations that include fillers to improve their mechanical properties are marketed. In one product particles of amalgam alloy are mixed with the GIC powder; another uses a cermet—silver sintered onto the GIC glass powder. Although use of these materials for core buildup and for very conservative class I and class II restorations in primary dentition has been advocated, the improvements in mechanical properties of these " reinforced" GICs are very modest. Whether their mechanical properties, such as fracture toughness, are adequate to resist masticatory stress is questionable. Again, the resin-modified glass ionomer is probably a better choice.

RESTORATIVE RESINS

CONVENTIONAL COMPOSITES

The term *composite material* refers to a combination of at least two chemically different materials with a distinct interface separating the components. When properly constructed, such a combination of materials provides properties that could not be obtained with any of the components alone. (Examples of natural composites are bone, tooth enamel, and wood.) In a resin composite dental restorative material, an inorganic filler has been added to a resin matrix in such a way that the properties of the matrix have been improved.

A number of parameters have a pronounced influence on the properties that are obtained by the addition of inorganic fillers to a resin matrix. The characteristics of the dispersed phase in terms of its shape, size, orientation, concentration, and distribution are very important. Likewise, the composition of the continuous phase, the resin matrix, is equally significant.

The resin matrix of many currently available composite materials is bisphenol A-glycidyldimethacrylate (bis-GMA) or urethane dimethacrylate resin. Triethylene glycol dimethacrylate, a lower-viscosity resin, is usually added as a diluent. Among the materials used for macro fillers are ground particles of fused silica, crystalline quartz, and soft glasses such as barium, strontium, and zirconium silicate glass. These particles, which make up 70% to 80% of the material by weight, resist deformation of the soft resin matrix. The high filler content and the chemistry of the resin matrix substantially reduce the

coefficient of thermal expansion compared with an unfilled acrylic resin. The filler also reduces the polymerization shrinkage and increases the hardness.

The filler and the resin matrix must be chemically bonded together with a coupling agent on the surface of the filler. If this is not done, the particles may be easily dislodged, water sorption at the filler-matrix interface may take place, and stress transfer between matrix and filler may not occur. The filler particles are coated with a reactive silane product. Despite use of this coupling system, the filler particles do become dislodged during cutting and finishing and under abrasive action such as tooth brushing or occlusal contact. This abrasive action likely affects the softer resin matrix, which becomes worn away and exposes the filler particles. When enough of the filler particle is exposed, it will break free of the resin. This process leaves a continual rough surface as seen in Fig. 16-3. Because the composites are 70% to 80% filled, this surface roughness is clinically noticeable.

Based on clinical experience, there has been a definite preference for use of smaller filler particles. In the early resin composites it was common for the particle size to approach 100 μm; now the coarsest particles would not exceed 30 μm. The average mean particle size of the fillers in conventional composites is in the 8- to 12-μm range, and the trend is to reduce the filler size even further.

MICROFILLED COMPOSITES

Efforts to improve the surface smoothness and polishability of composite resins led to the development of the microfilled composite. These composites are based on the use of an extremely small silica filler particle, whose size is 0.02 to 0.04 μm, and hence are called *microfine, microfilled,* or *polishable* resins. The particles may be dispensed directly into the paste, but the amount that can be added in this manner is very limited. Addition of amounts in excess of 20% results in a paste too viscous for the dentist to use. Matrix resin monomer can be heavily filled with microfine silica and polymerized in the manufacturing process. The resulting composite is ground to filler particle sizes comparable to those of the inorganic filler in conventional composite. This "organic" filler with additional colloidal silica is then added to the resin monomer to form the composite resin paste. The structure of such a resin is illustrated in Fig. 16-4.

The appealing characteristic of these microfilled resins is their ability to be finished to an extremely smooth surface, which was a major problem with the conventional composites. When microfilled resins are finished, the polymerized resin filler particles cut at the same rate as the matrix and a much smoother surface results, as shown in Fig. 16-4. Even if some of the very small silica particles are dislodged, the surface irregularities cannot be detected by the eye.

Because of the small silica particle size, the filler has a very large surface area, and the total amount of filler that can be incorporated is reduced to about 50% by weight compared with a filler loading of 70% to 80% for conventional composites. Thus, the microfilled composite has a higher resin matrix content. As a result, such resins are softer and have a slightly higher coefficient of thermal expansion, a higher water absorption, more polymerization shrinkage, and somewhat lower mechanical properties. Because of this tradeoff in properties, microfilled resins should be used where esthetics is the principal consideration and where undue stress will not be placed on the restoration, such as in class III or class V restorations. In situations in which the restoration is subject to stress, such as the incisal margin of a class IV

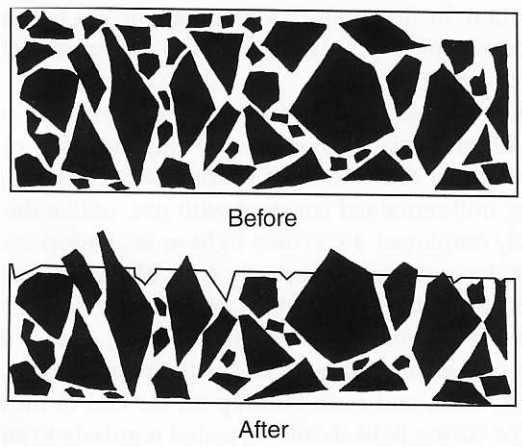


FIG. 16-3. Schematic drawing of a conventional composite resin with macrofiller *(black areas)* before and after finishing or wear. *(Redrawn from Phillips RW:* Science of dental materials, *ed 9, Philadelphia, 1991, WB Saunders.)*

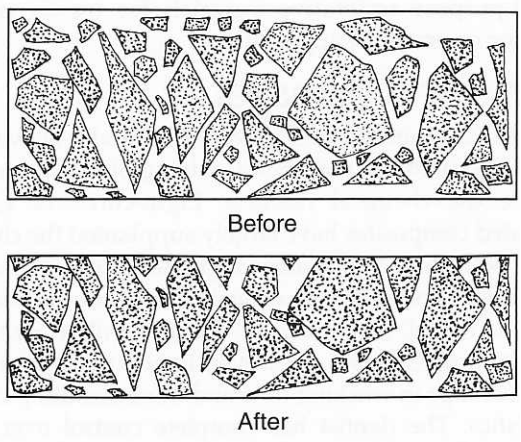


FIG. 16-4. Structure of a microfilled resin showing microfiller *(dots)* and prepolymerized macrofiller particles before and after finishing. *(Redrawn from Phillips RW* Science of dental materials, *ed 9, Philadelphia, 1991, WB Saunders.)*

restoration, a composite having better physical properties is preferred. The development of hybrid and small-particle composites has significantly reduced the use of microfilled composites.

SMALL-PARTICLE AND HYBRID COMPOSITES

The conventional macrofilled composite is no longer in common use. Filler sizes have been continually reduced in an effort to approach the surface smoothness of the microfilled resin but retain the filler levels and physical properties of the conventional composite. Small-particle composites have an average filler size of 1 to 5 μm, with a broad distribution of sizes. This permits higher filler loading than in the conventional composites and results in the best combination of physical properties of all the currently available composites. Small-particle composites are recommended for stress-bearing applications such as class IV and class II restorations. Their surface finish is inferior to that of a microfill resin but much better than that of a conventional composite.

Hybrid composites are the most recent step toward smaller particle size. They contain radiopaque glass particles with an average size of 0.6 to 1.0 pm in addition to 10% to 20% colloidal silica. The total filler level, 70% to 80%, is lower than in a small-particle composite but higher than in a microfill resin. Because these combine two types of fillers, the result is called a *hybrid composite.*

Although the surface of hybrid resins is not as smooth as that of microfilled resins, these resins find extensive anterior use if they are carefully polished. Also, one of the primary motivations in the development of these hybrid materials was to find a material that could compare favorably with dental amalgam in wear resistance in class I and II restorations. The use of composites in such situations is discussed in the section on posterior composite restorations. The most recent trend in resin composites has been the marketing of so-called universal or all-purpose restorative materials for use in either anterior or posterior applications.

LIGHT-CURED COMPOSITES

Originally, composite resins were chemically activated, which required the mechanical mixing of two pastes to initiate the chemical reaction. Light-cured or light-activated composites have largely supplanted the chemically activated composites. Light-activated resins do not differ significantly in composition from the chemically activated resins except for the polymerization activation mechanism. However, light curing provides an advantage in working time and other handling characteristics. The dentist has complete control over the working time and is not confined to the rather short working time of the chemically activated systems. This is particularly beneficial when large restorations such as class IV restorations are placed.

Most currently available *visible* light–cured resins contain the photosensitive initiator camphoroquinone, which absorbs visible light at wavelengths between 450 and 500 nm (blue light) and forms free radicals that activate an amine accelerator.

Visible light activation units are simple and relatively inexpensive, and their output remains fairly constant throughout the life of the bulb. Visible light is capable of polymerizing a reasonable thickness of resin (2 mm). It also will cure the resin through a layer of enamel, a particular advantage in class III restorations. Although protective glasses are recommended to shield the operator's eyes from the glare of the intense blue light, visible-spectrum curing lights do not pose a significant safety risk.

One major disadvantage of light-cured composites must be emphasized. Polymerization will only occur if the resin is exposed to light of sufficient intensity for an adequate length of time. The top surface of a restoration that is nearly in direct contact with the light source will always be cured if the light and resin are serviceable. However, the curing of the portion of the restoration farthest removed from the light is less certain. Normally this portion of the restoration is not accessible for any kind of probing to test its hardness. If the cure is incomplete on the bottom side of the resin compared with the top surface, the physical properties will be reduced and a color shift may occur in time. Likewise, unpolymerized monomer may increase the potential for pulpal irritation. Microleakage is another likely scenario. To ensure maximum polymerization, the end of the light source should be within 1.0 mm of the surface of the resin. The curing time should be at least 40 seconds, and the depth of resin to be cured should not exceed 2.5 mm. Larger restorations and dark shades of resin require an incremental placement technique. Dual-activated resins are available that combine both light and chemical activation. In situations in which light access to parts of the restoration is problematic, a dual-activated material may be preferred.

When visible light curing systems first were introduced into dentistry, considerable emphasis was placed on the claim that the light output of the visible light curing unit remained constant with use, unlike the previously employed ultraviolet light units. Unfortunately, this statement is only partially true. Numerous factors do influence the light output of a visible light curing unit such as power line variations, aging of the filters, aging of the lamp, damage to the light-conducting pipe or optic fiber, and resin buildup on the end of the light tip. The curing light should be tested regularly to ensure adequate light intensity. Inexpensive meters are avail-able for this purpose and should be used regularly. Many of the newer visible light activation units have built-in meters to verify adequate light intensity. If such

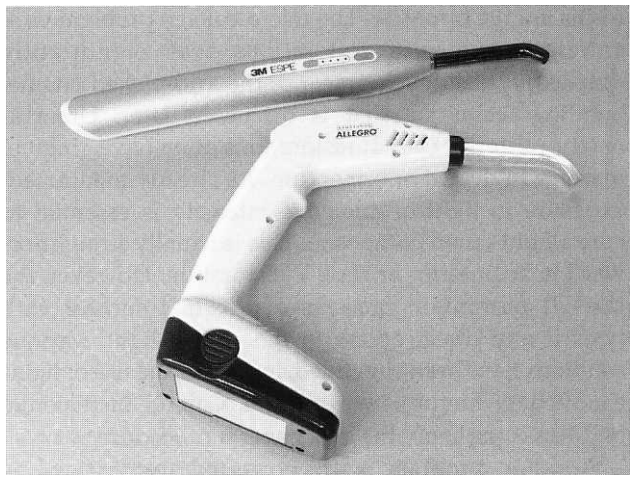
a device is not available, a simple usage test should be performed. Place a mass of light-curing resin that is 2 mm thick on a Mylar matrix strip over a sheet of white paper. Cover this mass with a second matrix strip. Holding the curing light as close to the top surface as possible, cure the resin for the length of time normally used. Remove the matrix strips and probe both the top and bottom resin surfaces with an explorer. There should be no noticeable difference in hardness or scratch resistance. Dark shades of composite resin may require longer curing times or curing of a thinner layer according to the manufacturer's instructions. If a comparable cure on both top and bottom cannot be achieved, the combination of resin and curing light is unsatisfactory for clinical use.

The most commonly used visible light activation units employ a quartz-tungsten-halogen lamp (QTH) as a light source. This light is filtered to retain the wave-lengths between 400 and 500 nm (blue light). In an effort to accomplish more rapid polymerization of a greater thickness of resin, other light sources have been marketed. These include plasma arc lamps and lasers. These are significantly more expensive than the QTH units, and there is evidence that more rapid polymerization may increase polymerization shrinkage stresses at the tooth-resin interface. A more recent addition to activation units uses a blue light-emitting diode (LED). More than 95% of the electrical energy delivered to a QTH light becomes heat and light of wavelengths longer than 500 nm. LEDs are nearly 100% efficient at generating light over a relatively narrow wavelength band (460 to 480 nm). This makes it possible to construct self-contained, rechargeable, battery-powered activation lights. These lights generate much less heat than QTH units. Unlike QTH lamps, an LED should maintain a constant output throughout its lifetime.

The total power delivered in the 400- to 500-nm band from LED lights was initially much lower than that of QTH units. Recently marketed LED lights have significantly improved power output. The dentist who decides to use an LED activating light should be aware that some evidence exists that, compared with QTH lights, the LED light does not result in as great a depth of cure with the same exposure time for all resin composites. In addition, light-activated resin composites that use photoinitiators other than camphoroquinone may not be effectively activated by the narrow band-width of light emitted by LED units. The main advantage of the LED lights appears to be convenience, because they do not require a fixed power cord to a base unit, as can be seen in Fig. 16-5.

POSTERIOR COMPOSITE RESTORATION

Composite resins were originally used for anterior or non-stress-bearing locations such as class III, class V, and class IV restorations. Since the introduction of resin composites, however, attempts have been made to use them as alternatives to dental amalgam for class I and II restorations. These attempts with conventional macro-filled composite failed because of unacceptable wear on the occlusal surface. Only in the case of conservative restorations in primary dentition was any success observed. A major goal of composite resin research has been to develop properties adequate to allow their use as an alternative to dental amalgam.



f I G **16-5.** Rechargeable battery-powered light-emitting diode-type visible light activation units.

The improved strength, hardness, and modulus of elasticity of some of the newer composite resins, with their low thermal conductivity and superior esthetics, indicate that they may serve as alternatives for amalgam in the restoration of occlusal and proximal surfaces in posterior teeth (class I and class II restorations). Extensive clinical testing has been done to compare the performance of these new resins with that of amalgam. There now exist resin composites, mostly of the small-particle type, with documented clinical wear of less than 20 μm per year over a 5-year period. These materials qualify for full acceptance under the ADA acceptance program for posterior composite resins. In a patient who has a known sensitivity to dental amalgam, use of a composite chosen from the ADA list of accepted materials should be considered. The patient and guardian should be cautioned that the profession does not have long-term data to suggest that such restorations will perform as well as amalgam over long periods (5 to 10 years or longer), and the wear and occlusal relation-ships of the restorations should be monitored.

The dentist needs to be aware of an additional factor when choosing resin composites for posterior occlusal service. Whereas dental amalgam is one of the least technique-sensitive direct restorative materials and one whose microleakage decreases with time, composite

resins are the opposite. The microleakage problem with anterior composite restorations was significantly reduced by the development of the acid-etched enamel bonding technique that is discussed later. Unfortunately, posterior class II restorations often have gingival margins in dentin or cementum. As already mentioned, exposure to light of adequate intensity is essential to cure a light-cured composite. This is usually straightforward in an anterior or class V restoration. However, the class II restoration presents a proximal surface with poor if any direct access to the curing light. Various solutions have been suggested, including the use of light-conducting interproximal wedges. Careful incremental buildup is probably the best general procedure.

Another problem with posterior restorations is related to the curing shrinkage pattern. Most composite resins exhibit linear shrinkage of 2% or more during curing. The light-cured composite hardens first on the surface immediately adjacent to the curing light tip. As a consequence, the shrinkage is directed toward the curing light and away from the floor of the preparation or the gingival margin. This places the largest stresses from curing shrinkage on the sections of resin that are least well cured and whose bond to tooth structure may be the poorest. Increased microleakage may be the result. Resin composites are also compromised by moisture contamination during placement, and effective isolation of the operative field is essential.

Operative techniques exist to reduce these effects, as described in Chapter 18, but the dentist must understand the basic phenomena to appreciate the importance of the rather detailed and meticulous techniques recommended for placement of posterior composite restorations. Unless the practitioner is able and willing to take the time to place posterior composites properly, the restorations are likely to fail prematurely. Obviously the dentist must be compensated for this service in keeping with the time required for placement and should carefully explain to the patient the advantages and disadvantages of the treatment alternatives along with their costs. Placement of bonded resin restorations in posterior permanent teeth of young patients is problematic if those restorations have an expected clinical lifetime of 5 years or less. Each time a bonded restoration is replaced additional tooth structure will be lost. Repeated replacement of resin composites over the lifetime of a patient is not only costly but may eventually compromise the tooth.

Since the dentist may frequently be faced with a request for placement of resin composites rather than amalgam restorations, knowledge about the safety of these materials is important. A resin monomer often employed in restorative resin composites is bis-GMA. This monomer is the reaction product of bisphenol A and a dimethacrylate. Commercial bisphenol A often contains impurities that are chemically similar to synthetic estrogen. Concerns have been raised about the potential for promoting certain types of malignancies. Components used in the light activation of polymerization initiation are very reactive. Traces of these components remain after the resin has hardened. The resin monomers employed as the matrix of composites are not completely converted to polymers. Conversion of 65% or less of the reactive double bonds in the monomer is typical with these resins. Unreacted monomer may result in pulpal irritation.

RESIN INLAYS

The resin inlay restoration addresses some of the shortcomings of composite resins, particularly the difficulties with placement and light curing discussed previously. The indirect inlay restoration is fabricated in the laboratory on a die poured from an impression of the prepared tooth like a wax pattern for a cast restoration. An alternative for the resin inlay is a direct technique. A separating medium is applied to the prepared tooth, and the resin is condensed and light-cured. It is then removed from the mouth and subjected to additional curing procedures. Either technique allows better access for light-curing the composite, and the finished restoration can be subjected to additional curing under intense light, heat, and pressure or some combination of these. In theory, the properties of the resulting composite are maximized, and the presence of unreacted monomer is minimized. More importantly, the polymerization shrinkage occurs outside the mouth. The finished inlay is cemented in the mouth using a resin cement. The stresses at the tooth-restoration interface should be much lower than in a direct-placement resin restoration.

Although laboratory data support some of the claims made for these inlays, long-term clinical data are not yet available. The resin inlay systems have not achieved wide popularity in the United States, although they are commonly used in Europe. Resin inlay restorations are more time consuming and expensive than direct resin restorations. Their appropriateness as alternatives to dental amalgam or cast restorations needs long-term clinical investigation.

ACID-ETCHING TECHNIQUE

Marginal leakage is probably a more acute problem with resins than it is with any other restorative material. As has been noted, the amalgam restoration tends to counteract the microleakage phenomenon by the formation of corrosion products along the tooth-restoration interface. Other restorative materials may provide a mechanism for resisting secondary caries attributable to microleakage, such as the fluoride released from GIC. With the presently available direct restorative resins, however, there is no inherent resistance to the dangers

of marginal penetration of deleterious agents. Thus the resin restoration presents a challenge to the dentist to attain good initial adaptation of the material to the tooth surface and maintain it under oral conditions.

One of the most satisfactory methods for mechanical bonding of resin to enamel is the use of the acid-etch technique. The enamel is etched with a solution of phosphoric acid (usually about 35%) for approximately 15 to 20 seconds. Both liquid and gel etching agents are available; placement is more readily controlled with the gel.

The next critical step in the technique is the use of a water rinse to remove the debris produced during etching. If this debris is not flushed off the enamel surface, the resin will not wet the etched surface. A minimum wash time of 30 seconds is usually recommended. Before the resin is placed, the etched surface must then be dried for at least 15 seconds.

Any film of moisture on this cleaned surface will inhibit resin penetration into the etched enamel. If the surface is accidentally contaminated by saliva, the saliva film cannot be completely removed by washing. Rather, the surface should be re-etched for 10 seconds, washed, and dried.

An etched enamel surface can be seen in Fig. 16-6, A. The acid cleans the enamel to provide better wetting of the resin and creates pores into which the resin flows to produce "tags" that greatly increase retention, as shown in Fig. 16-6, B. The resulting bond should reduce the possibility of marginal stain, which is the invariable result of microleakage. In addition to providing a seal between the resin and tooth, the acid-etch technique results in mechanical retention. Resin bond strengths to etched enamel range from 16 to 22 MPa.

BONDING AGENTS

As an adjunct to the acid-etch technique, manufacturers formerly supplied enamel-bonding agents. The bonding agent consisted of bis-GMA resin matrix material diluted with a low-viscosity methacrylate monomer. After acid etching of the enamel, the bonding agent was applied. The composite resin was then immediately inserted, and it in turn bonded to the intermediate layer of resin-bonding agent. It should be emphasized that the resulting bond to tooth structure was strictly mechanical.

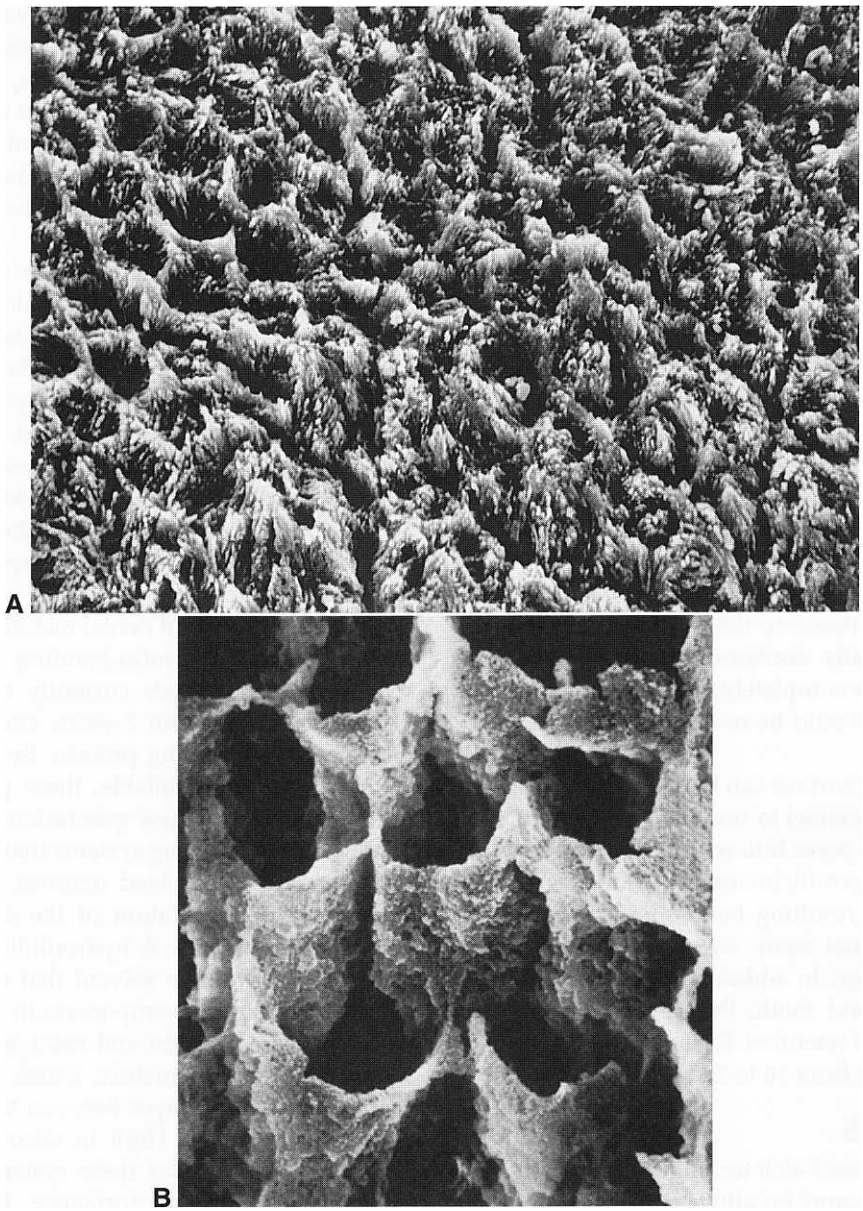
The success of the acid-etch, enamel-bond technique led to attempts to do the same for bonding to dentin. Early efforts to etch dentin and apply enamel-bonding agents were not successful. The structure of dentin is far more complex, and it has a lower concentration of inorganic material; because of its microstructural organization, the regular etch patterns seen with enamel are not produced. Due to the organic component of dentin and to the permeability that results from the dentinal tubules, the dentin surface has low surface energy and is constantly wet. This presents the ultimate challenge for adhesion. For the past 20 years, research has focused on the development of agents that will bond adhesively to dentin. This has led to the introduction of *dentin-bonding* agents, which are either chemically activated or light cured. They are applied to the dentin before placement of the composite.

The chemistry of the different products is complex and varied. Some rely on mechanical retention whereas the claim is that others form chemical bonds to the organic or inorganic portions of the dentin. Such claims are not generally supported by direct evidence of chemical bonding. Instead, bond strengths to dentin are measured and reported. These vary from a few megapascals to values comparable to those for etched enamel. Often the bond strengths measured in vitro decrease markedly with time, exposure to water, and thermocycling.

Probably no area of dental materials has seen as much activity as that of dentin bonding in the past 15 years. Many of the products currently marketed have been available for less than 2 years, and their development has been a continuing process. By the time short-term clinical data are available, these products often have been replaced by a new generation of bonding systems.

The dentin-bonding systems that first exhibited good bond strengths involved removal of the dentin smear layer and decalcification of the outer layer of intact dentin with an acid. A hydrophilic primer component carried by a volatile solvent that displaces water was then applied. Resin components in the primer penetrate the decalcified dentin and react with and modify the remaining dentin structure, which results in creation of a so-called hybrid layer between the intact dentin and the resin adhesive. High in vitro bond strengths are generally reported for these systems along with good short-term clinical performance. Unlike with enamel bonding, it is important that the etched dentin surface not be desiccated before application of the primer when systems with hydrophilic primers are used. Current advances have focused on the development of delivery systems that simplify the steps involved in using dentin-bonding systems. Acidic primers that are self-etching have been introduced. These simultaneously demineralize and penetrate the smear layer and underlying dentin. Other systems combine the primer and the resin adhesive into one component.

The most recently introduced systems mix together the acidic primer and resin adhesive before they are placed on the tooth surface. Even though these newer systems use fewer separate components, their application still requires that several steps be preformed with great attention to detail. Use of these materials remains extremely technique sensitive.



f I i. **16-6.** Effects of acid etching. **A,** Scanning electron micrograph of enamel surface etched with phosphoric acid, **B,** Scanning electron micrograph of resin tags formed by penetration of resin into etched enamel surface (x5000). (A *and* **B** *from Anusavice KJ:* Phillips' science of dental materials, ed 99, *St Louis, 2003, WB Saunders, courtesy K-J Sdderholm.)*

Little if any convincing evidence exists that any of the dentin-bonding agents currently marketed forms significant chemical bonds to dentin. More likely the bonding mechanism is micromechanical. The status of these agents remains controversial, particularly with regard to their long-term stability in the oral cavity. Until these matters are resolved, one should not deviate too far from accepted restorative procedures. One should assume that the dentin adhesive will not eliminate the need for the use of traditional methods for retention of the resin restoration, such as acid etching of the enamel and retentive cavity preparations in dentin or cementum.

Recent laboratory data have shown incompatibility between some of the newer dental bonding systems and restorative resins or resin cements that are activated by chemical or dual means. Many of the resins used for crown buildup as core materials fall into these categories. It appears that the use of acidic primers may interfere with the chemical activation of these resin materials unless a separate resin adhesive is placed over the primer and light activated before the core material or cement is placed.

Most of the bonding agents currently marketed are sold as universal bonding agents to be used with both dentin and enamel. Bond strengths reported for etched enamel usually equal those for the original enamel-bonding agents that have largely disappeared. Regardless of whether an auxiliary resin-bonding agent is used, adequate etching of the enamel is an important step in securing mechanical bonding of any restorative resin to enamel. Some of the systems that use self-etching primers or acids other than phosphoric acid do not routinely yield enamel-bond strengths as high as expected with conventional acid etching. Questions exist about the effectiveness of dental bonding systems employing self-etching primers when these are used in place of phosphoric acid treatment for bonding orthodontic appliances and pit and fissure sealing. Conventional phosphoric acid etchants can be used to ensure etching of the enamel margins of a cavity preparation.

In the context of dental bonding, one should not ignore the polyacrylic acid systems. GIC has been recommended for use as a dentin-bonding agent in the so-called sandwich technique. Fast-setting GICs are available for use as cavity-lining materials (type III GIC and light-cured GIC). The enamel is not covered with the GIC but rather acid etched in the conventional manner. Then a resin-bonding agent is applied and a composite resin placed. The ionomer bonds adhesively to the tooth whereas the bonding agent bonds mechanically to the ionomer and the enamel. This adds yet another dimension to dentin-bonding technology, particularly for the class II restoration.

Dental resins are no more or less irritating to the pulp than are several other commonly used restorative materials. Whenever the cavity preparation is deep, the same precautions should be taken with resins as with other restorative materials.

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CHAPTER 11

Pit and Fissure Sealants and Preventive Resin Restorations

BRIAN J. SANDERS
ROBERT J. FEIGAL
DAVID R. AVERY

انتشارات
شایان نمودار

CHAPTER OUTLINE

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PIT AND FISSURE SEALANTS

In 1955 Buonocore described the technique of acid etching as a simple method of increasing the adhesion of self-curing methyl methacrylate resin materials to dental enamel.' He used 85% phosphoric acid to etch enamel for 30 seconds. This produces a roughened surface at a microscopic level, which allows mechanical bonding of low-viscosity resin materials.

The first materials used experimentally as sealants were based on cyanoacrylates but were not marketed. By 1965 Bowen et al had developed the bis-GMA resin, which is the chemical reaction product of bisphenol A and glycidyl methacrylate.[2] This is the base resin to most of the current commercial sealants. Urethane dimethacrylate and other dimethacrylates are alternative resins used in sealant materials.

For the chemically cured sealants, a tertiary amine (activator) in one component is mixed with another component containing benzoyl peroxide, and their reaction produces free radicals, which initiates polymerization of the sealant material.

The other sealant materials are activated by an external energy source. The early light-activated sealants were polymerized by the action of ultraviolet rays (which are no longer used) on a benzoin methyl ether or higher-alkyl benzoin ethers to activate the peroxide curing system. The visible light—curing sealants have diketones and aromatic ketones, which are sensitive to visible light in the wavelength region of 470 run (blue region). Some sealants contain filler, usually silicon dioxide microfill or even quartz.

Sealant materials may be transparent or opaque. Opaque materials are available in tooth color or white. Transparent sealants are clear, pink, or amber. The clear and tooth-colored sealants are esthetic but are difficult to detect at recall examinations. Recent advances in sealant technology include light-activated coloring agents that allow for color change during and/or after polymerization. These compositional changes do not affect the sealant, but only offer some arguable benefit in the recognition of sealed surfaces.

The cariostatic properties of sealants are attributed to the physical obstruction of the pits and grooves. This prevents colonization of the pits and fissures with new bacteria and also prevents the penetration of fermentable carbohydrates to any bacteria remaining in the pits and fissures, so that the remaining bacteria cannot produce acid in cariogenic concentration.

CLINICAL TRIALS

Many clinical studies have reported on the success of pit and fissure sealants with respect to caries reduction. As the longevity of the sealant increases, the retention rate becomes a determinant of its effectiveness as a caries-preventive measure.

In 1983 a National Institutes of Health Consensus Panel considered the available information on pit and fissure sealants and concluded that "the placement of sealants is a highly effective means of preventing pit and fissure caries.... Expanding the use of sealants would substantially reduce the occurrence of dental caries in the population beyond that already achieved by fluorides and other preventive resources."'

In 1991 Simonsen reported on a random sample of participants in a sealant study recalled after 15 years." He reported that, in the group with sealant, 69% of the surfaces were sound 15 years after a single sealant application, whereas 31% were carious or restored. In the group without sealant, matched by age, gender, and residence, 17% of the surfaces were sound, whereas 83% were carious or restored. He also estimated that a pit and fissure surface on a permanent first molar is 7.5 times more likely to be carious or restored after 15 years if it is not sealed with a single application of pit and fissure sealant.

The use of glass ionomer as a sealant material has the advantage of continuous fluoride release, and its preventive effect may continue with the visible loss of the material. Glass ionomer may be useful as a sealant material in deeply fissured primary molars that are difficult to isolate due to the child's precooperative behavior and in partially erupted permanent molars that the clinician believes are at risk for developing decay. In such cases, glass ionomer materials must be considered a provisional sealant to be reevaluated and probably replaced with resin-based sealants when better isolation is possible. Because questions exist regarding the strength and retention of glass ionomer, further long-term research is necessary before it is recommended as a routine pit and fissure sealant material.

A 1996 survey of Indiana dentists' found that 91% of them were placing sealants on permanent teeth, whereas in 1985 a similar study[£0'] had found that only 73.5% were placing sealants on permanent teeth. This increased use of sealants may be related to increased practitioner comfort with the materials, because a direct correlation was found between sealant usage and year of graduation from dental school. The increase may also be related to a decreased concern over the possibility of caries developing under the sealant.

Several studies have reported decreased viable bacterial counts in occlusal fissures that have been sealed. Handleman, Washburn, and Wopperer placed an ultraviolet-radiation—polymerized sealant on pits and fissures of teeth with incipient caries.' They reported a 2000-fold decrease in the number of cultivable microorganisms in the carious dentin

samples of the sealed teeth compared with unsealed control teeth at the end of 2 years.

Going et al obtained bacteriologic samples from teeth that had been sealed with an ultraviolet-radiation--polymerized sealant for 5 years.[s] They found an apparent 89% reversal from a caries-active to a caries-free state in the sealed teeth.

Jeronimus, Till, and Sveen placed three different pit and fissure sealants on molars with incipient, moderate, and deep carious lesions.' Samples of carious dentin were removed immediately after and 2, 3, and 4 weeks after placement of the sealants and bacteriologic cultures were made. They reported usually positive culture results in teeth where the sealant was lost. Although their short-term study indicated that incipient carious lesions may not be of prime concern when sealants are applied, they cautioned against the use of sealants over deeper lesions because of the potential for advancement of caries when sealants over these lesions are lost. One must keep in mind that their deep-lesion group consisted of teeth with caries that had advanced pulpally greater than half the distance from the dentinoenamel junction.

Studies have shown definitively that deficient sealants are not effective in caries prevention and that loss of sealants leads to immediate risk of caries attack from undercover surfaces. Sealants require regular maintenance and repair or replacement to assure success in caries prevention over the long term.

Going declared that, given the results of many well-documented studies, practitioners' fear of sealing pits and fissures with incipient caries is not warranted.[10] He pointed out that sufficient studies of scientific merit reported negative or low bacterial concentrations after sealant had been in place for several years.

Wendt and Koch annually followed 758 sealed occlusal surfaces in first permanent molars for 1 to 10 years." At the end of their study, evaluation of the surfaces that had been sealed 10 years previously revealed that only 6% showed caries or restorations. Romcke et al annually monitored 8340 sealants placed on high-risk (for caries) first permanent molars during a 10-year period." Maintenance resealing was performed as indicated during the annual evaluations. One year after the sealants were placed, 6% required resealing; thereafter 2% to 4% required resealing annually. After 8 to 10 years, 85% of the sealed surfaces remained caries free.

Retrospective studies based on billing data from large third-party databases reveal that sealant use is still surprisingly low, even in populations for which sealants are a covered benefit."•'[4] In addition, these studies show that the effectiveness of sealants in preventing the need for future restorative care on the sealed surfaces declines after the first 3 years following sealant treatment. These data argue again for the importance of vigilant recall and upkeep of sealants after placement.

Another concern is the placement of sealants immediately after topical fluoride application. Clinical and in vitro studies have shown that topical fluoride does not interfere with the bonding between sealant and enamel.[' 5r'8]

RATIONALE FOR USE OF SEALANTS

In 1997 the ADA Councils on Access, Prevention, and Interprofessional Relations and on Scientific Affairs reconfirmed the safety and effectiveness of pit and fissure sealants as a caries-preventive measure and added that "dental sealants have proved to be highly effective in preventing pit and fissure dental caries.... Many individuals and groups stand to benefit from sealant placement. The key element is the professional judgment of the dentist, for a specific patient, at a specific time.""

The American Academy of Pediatric Dentistry's Pediatric Restorative Dentistry Consensus Conference's confirmed support for sealant use and published these recommendations:

1. Bonded resin sealants, placed by appropriately trained dental personnel, are safe, effective, and underused in preventing pit and fissure caries on at-risk surfaces. Effectiveness is increased with good technique and appropriate follow up and resealing as necessary.
2. Sealant benefit is increased by placement on surfaces judged to be at high risk or surfaces that already exhibit incipient carious lesions. Placing sealant over minimal-enamel caries has been shown to be effective at inhibiting lesion progression. Appropriate follow-up care is recommended, as with all dental treatment.
3. Presently, the best evaluation of risk is made by an experienced clinician using indicators of tooth morphology, clinical diagnostics, past caries history, past fluoride history, and present oral hygiene.
4. Caries risk, and therefore potential sealant benefit, may exist in any tooth with a pit or fissure, at any age, including primary teeth of children and permanent teeth of children and adults.
5. Sealant placement methods should include careful cleaning of the pits and fissures without removal of any appreciable enamel. Some circumstances may indicate use of a minimal-enameloplasty technique.
6. Placement of a low-viscosity, hydrophilic material-bonding layer as part of or under the actual sealant has been shown to enhance the long-term retention and effectiveness.

7. Glass ionomer materials have been shown to be ineffective as pit and fissure sealants but can be used as transitional sealants.
8. The profession must be alert to new preventive methods effective against pit and fissure caries. These may include changes in dental materials or technology.

SELECTION OF TEETH FOR SEALING

To gain the greatest benefit, the clinician should determine the caries risk; thus, the term *risk-based sealant treatment* has come into use. In risk-based sealant treatment, the practitioner takes into account prior caries experience, fluoride history, oral hygiene, and fissure anatomy in determining when sealant should be applied.

Good professional judgment should be used in the selection of teeth and patients. The use of pit and fissure sealants is contraindicated when rampant caries or interproximal lesions are present. Occlusal surfaces that are already carious with involvement of dentin require restoration.

All caries-susceptible surfaces should be carefully evaluated, because caries is unlikely in well-coalesced pits and fissures. In this case, sealants might be unnecessary or, at least, not cost effective. Finally, although sealant application is relatively simple, the meticulous technique requires patient cooperation and should be postponed for uncooperative patients until the procedures can be properly executed.

SEALANT TECHNIQUE

After selection, the tooth is washed and dried and the deep pits and fissures are reevaluated (Fig. 17-1, A). If caries is present, restoration or a combination of restoration and sealing may be indicated, as explained later in this chapter.

Marking centric stops with articulating paper will provide information so that excess sealant does not interfere with the occlusion. This is not necessary when the tooth has just erupted but is helpful in a well-established occlusion.

CLEANING

Adequate retention of the sealant requires that the pit and fissures be clean and free of excess moisture (Fig. 17-1, B and O. Acid etching completely removes the enamel pellicle, and a dental prophylaxis (even with a dental explorer) does not increase the retention of sealants. From a practical standpoint, in cases of poor oral hygiene, fissure cleansing with a rotating dry bristle brush may be beneficial.

Pope et al found that the use of a quarter round bur produced the greatest penetration of the sealant into etched enamel in laboratory studies.[17] The use of an aluminum oxide air abrasion system allows sealant penetration greater than that achievable by use of pumice or a dry bristle brush alone. It is not known if the increased depth of sealant penetration will result in greater sealant retention. When pumice or aluminum oxide is used, particulate matter is left in the deep recesses of the pits, the impact of which has not been determined.

Hatibovic-Kofman, Wright, and Braverman measured the microleakage of sealants placed in three groups of extracted teeth.[20] The teeth received conventional (etch), quarter round bur, or air abrasion surface preparation. Teeth prepared with the bur exhibited the least microleakage. The amount of microleakage in the conventional and air abrasion groups was about equal. No clinical studies exist to substantiate the value of using a bur to clean fissure surfaces prior to sealant placement.

The routine procedure of fissure eradication is probably not necessary. In fact, inappropriate or aggressive use of fissure opening or enameloplasty often removes the last of the enamel overlying dentin at the bottoms of fissures, which leaves the tooth more susceptible to future caries in the event of sealant loss. Good sealant methodology and proper sealant volume is probably more beneficial than enameloplasty.

ISOLATION

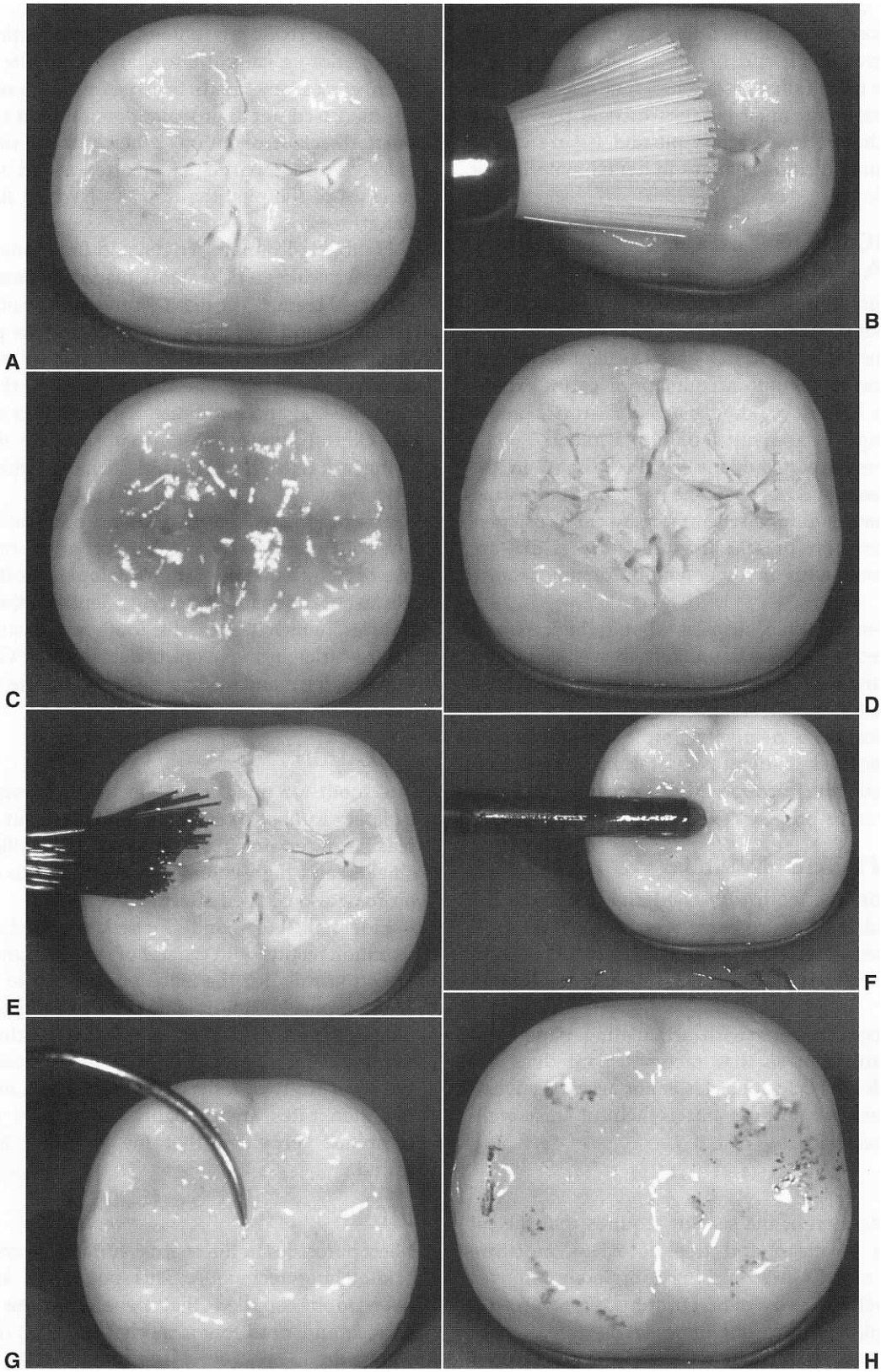
The tooth (or quadrant of teeth) to be sealed is first isolated. Rubber dam isolation is ideal but may not be feasible in certain circumstances. Cotton rolls, absorbent shields, and high-volume evacuation with compressed air may also be used effectively.

Eidelman, Fuks, and Chosack reported comparable retention results with the use of rubber dam and the use of cotton rolls for the isolation of teeth to be sealed.[1-3] Matis reported 96% retention of sealants with rubber dam isolation and 91% retention with cotton roll isolation at 12 months in young adults.[12] These values are not statistically different, however, which indicates that retention rates are probably not related to isolation technique, provided that the insertion technique is sound.

ETCHING

Microporosities in the enamel surface are created by the acid-etching technique. This permits a low-viscosity resin to be applied that penetrates the roughened surface and produces a mechanical lock of resin tags when cured.

Various phosphoric acid solutions have been evaluated for the etching procedure. Zidan and Hill tested the amount of surface loss of enamel after 60 seconds of etching with different phosphoric acid concentrations ranging from 0.5% to 80%.[23] They reported that the


A
B
C
D
E
F
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H

maximum loss of enamel was produced by the 35% concentrations, whereas the bond strengths were not significantly different after etching with 2%, 5%, and 35% concentrations. Generally, 30% to 50% acid solutions or gels are now recommended.

The etchant in solution should be placed on the enamel with either a bnish, small sponge, cotton pellet, or applicator provided by the manufacturer. The etchant should be placed widely across the surface to be sealed so that there is no chance that resin placement and polymerization will occur over a unetched enamel area. If a solution is used, one should gently agitate and replenish it, making an effort to avoid rubbing and breaking the enamel rods.

Occasionally a very viscous gel etchant may show a " skipping" effect, which occurs when the etchant does not completely and uniformly wet the entire enamel surface, and unetched areas are evident after washing and drying. If this occurs, reetching is necessary.

Generally, a 20-second etching time is recommended. Enamel rich with fluorhydroxyapatite may be resistant to etching and may need to be exposed for longer periods. Primary teeth may also sometimes be resistant to etching and may require a longer etching time. Redford, Clarkson, and Jensen reported no increase in bond strength with 120-second etching on primary teeth compared with 15-, 30-, or 60-second etching times.[24] Their in vitro study showed that the etch depth increased between 60 and 120 seconds, but there was no corresponding increase in bond strengths.

Some advocate preparing enamel for sealant application with an aluminum oxide air abrasion system or a laser system approved for hard-tissue procedures. To date, studies indicate that additional acid etching is needed after each of these techniques to allow adequate resin bonding to enamel.

WASHING

Most manufacturers' instructions advocate a thorough washing and drying of the etched tooth surface but do not specify a time interval. Phillips advocated a 40-second washing time.[25] Norling has advocated 20 seconds.[26]

The etched enamel is dried using a compressed air stream that is free of oil contaminants. The dry etched enamel should exhibit the characteristic frosty appearance (Fig. 17-1, D).

Feigal, Hitt, and Splieth found that the use of a dentin-bonding agent increased sealant retention in teeth even when salivary contamination occurred.[27] Choi et al have reported corresponding findings in vitro on moisture-contaminated bovine enamel.[2] In a later extensive review article, Feigal recommended routine placement of bonding agents before all sealant applications.[29]

Although the recommendation is still to avoid moisture contamination whenever possible during sealant application, the use of a dentin-bonding agent as part of the technique appears to be warranted (Fig. 17-1, E). Furthermore, the use of a dentin-bonding agent is definitely recommended in clinical situations that do not lend themselves to strict isolation, for example, when newly erupted teeth are sealed or when patient cooperation is not ideal. The use of a dentin-bonding agent is also advantageous on the buccal surfaces of molars, which traditionally have shown a lower retention rate than the occlusal surfaces of teeth.[3"] When used, the bonding agent must be thoroughly air-dried across the surface to be sealed to avoid a thick layer of adhesive residue.

APPLICATION OF SEALANT

Chemically Cured Sealant. The manufacturer's instructions should be followed. Precise mixing without vigorous agitation can help to prevent the formation of air bubbles.

The addition of the catalyst to the base immediately begins the polymerization of the material, and this should be kept in mind so that no time is lost in carrying the material to the etched and dried tooth. Working time is limited with a chemically cured sealant.

Visible Light—Cured Sealant. The curing of a light-polymerized sealant is not completed without the exposure of the material to the curing light, but the operating light and ambient light can also affect the material over a period of time, and so material should be dispensed only when it is time to place it on the tooth. The working time is longer than with chemically cured sealant. The method of placement varies with the different applicators provided by the manufacturers. The sealant is applied to the prepared surface in moderation and then gently teased with a brush or probe into the pits and grooves (Fig. 17-1, F, G). Careful application will avoid incorporation of air bubbles. Care should also be taken to avoid applying large amounts of the sealant material.

If a light-curing material is used, the intensity of the light should be considered. If a large surface area requires polymerization, place the light directly over each area of the occlusal surface for the recommended time.

With light-cured sealants there is less chance of incorporating air bubbles, because no mixing of materials is required. After the material has been cured and while the treated teeth are still isolated, the unpolymerized surface layer should be removed by washing and drying the surface to avoid an unpleasant taste.

CHECK OF OCCLUSAL INTERFERENCES

Articulating paper should be used to check for occlusal interferences and the occlusion adjusted if necessary (Fig. 17-1, H). All centric stops should be on enamel.

If a filled sealant has been used, it is essential to adjust the occlusion before the patient is dismissed.

Other excess sealant that may have flowed over the marginal ridge or toward the cervical area should also be removed. If the tooth is isolated with a rubber dam, the excess should be removed before detaching the rubber dam. A small round bur at slow speed will remove the excess effectively. If etchant has been well localized, excess sealant may be removed with a sharp instrument from unetched tooth enamel without removing sealant from the etched groove areas.

REEVALUATION

It is important to recognize that sealed teeth should be observed clinically at periodic recall visits to determine the effectiveness of the sealant. Periodic recall and reapplication of sealants is necessary, because it is estimated that between 5% and 10% of sealants need to be repaired or replaced yearly. If a sealant is partially or completely lost, any discolored or defective old sealant should be removed and the tooth reevaluated. A new sealant can be applied using the method previously described.

PREVENTIVE RESIN RESTORATION (SEALED COMPOSITE RESIN RESTORATION)

The preventive resin restoration is an alternative procedure for restoring young permanent teeth that require only minimal tooth preparation for caries removal but also have adjacent susceptible fissures.

Simonsen and Stallard described the technique of removing only the carious tooth structure in small class I cavi.ties.[31] A resin restoration was then placed, and the adjacent pits and fissures were sealed at the same time.

Henderson and Setcos described the sequence of the preventive resin restoration that is particularly applicable for young patients with recently erupted teeth and minimally carious pits and fissures."- They pointed out that this preparation requires a meticulous technique that involves more time than the traditional occlusal amalgam restoration. This type of restoration was advocated for carefully selected non—stress-bearing areas to minimize anatomic wear.

Occlusal surfaces often have small carious pits. For minimal caries, restorations are not likely to be subjected to substantial stresses that might lead to wear of resin materials. Fig. 17-2 shows diagrams illustrating the principles of the sealant-composite combination. In this case a small carious lesion has penetrated to the dentin. In general, bite-wing radiographs should indicate no interproximal caries.

A clinical series showing the sequence for this conservative preparation and restoration is portrayed in Fig. 17-3. Caries is identified by careful visual examination of a dry occlusal tooth surface using a sharp explorer, a mirror, and a light (Fig. 17-3, A). Articulating

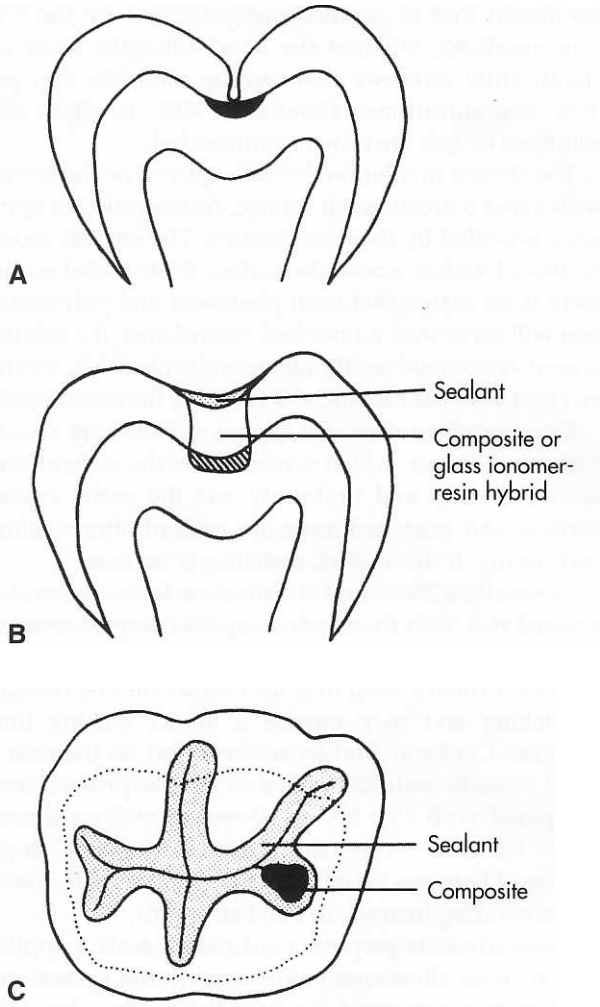


FIG **17-2** Diagrams illustrating the sealed composite resin restoration. A, A cross section showing caries extending to the dentin. **B,** A cross section through a preparation with a glass ionomer or composite restoration and a sealant. C, An occlusal view of the outline of a small restoration where a pit and fissure sealant provides the extension-for-prevention principle of cavity preparation.

paper marks on the tooth would indicate the points of occlusal contact.

The tooth is anesthetized if deemed necessary, isolated, and reexamined to determine the extent of the caries process. A No. 329 bur, aluminum oxide air abrasion, or a laser system approved for hard tissue can be used to gain access to the depth of the lesion and to complete caries removal (Fig. 17-3, M. The preparation, which should not extend to the occlusal contact marks, is washed, dried, and examined.

The cavity and the enamel beside the susceptible grooves are etched (Fig. 17-3, C). A gel or liquid form of 37% phosphoric acid is commonly used for 20 seconds. Surface preparation with aluminum oxide air abrasion

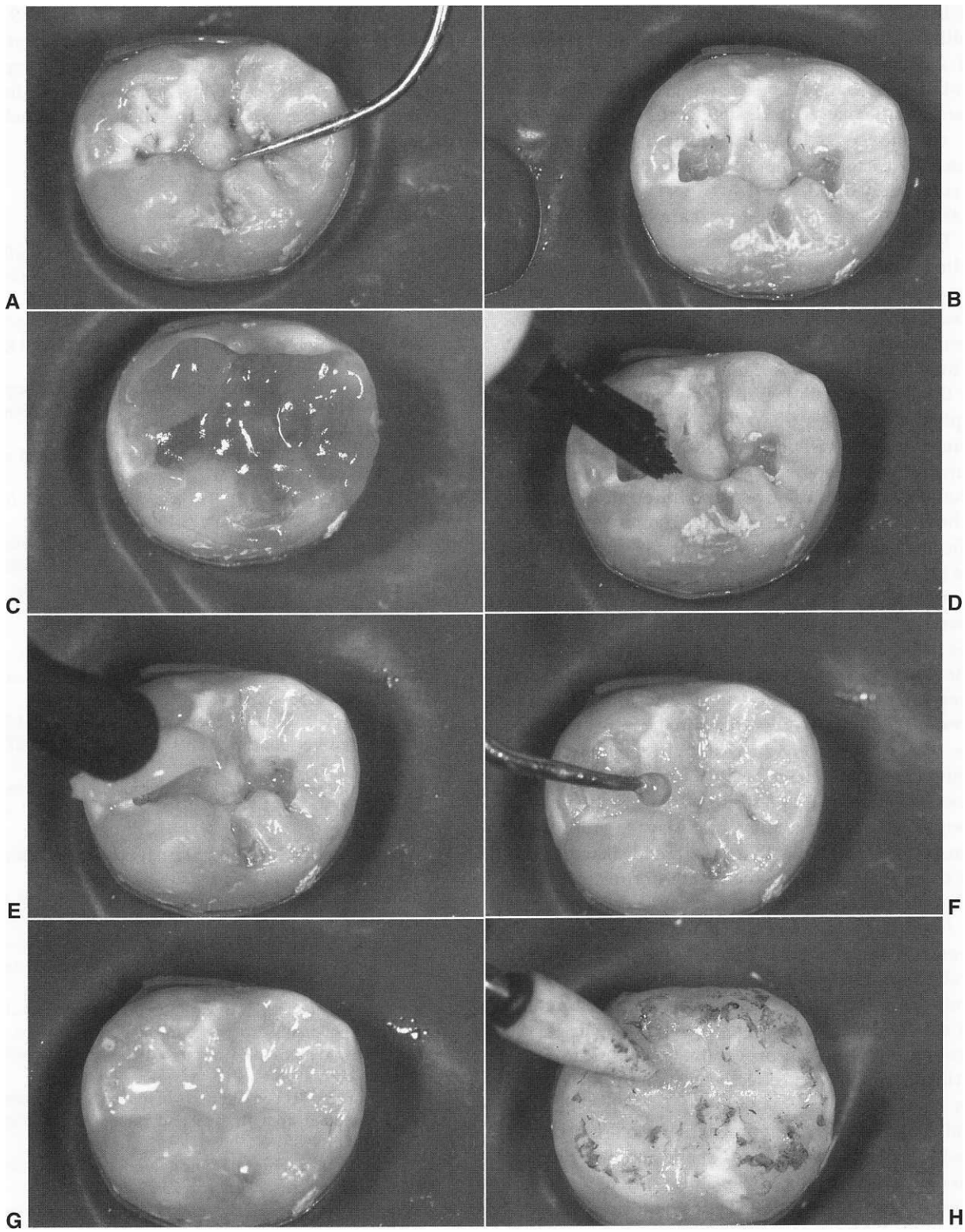


FIG 17-3 **A,** An occlusal caries identified with susceptible pits and grooves. **B,** Caries is removed into dentin. C, The tooth is etched. D, The bonding agent is placed. **E,** Composite resin is placed. **F,** Sealant is placed over resin. **G,** Polymerized preventive resin restoration. **H,** The occlusion is adjusted.

or a laser system approved for hard tissue may *not* substitute for acid etching. If these cleaning methods are used, etchant must still be applied to provide adequate resin-bonding enamel. The lingual groove of maxillary molars and the buccal groove of mandibular molars are also commonly etched and sealed. The tooth is thoroughly washed for approximately 30 to 40 seconds and completely dried.

A thin layer of bonding agent is applied to the cavity (Fig. 17-3, D). A stream of air must be used to thin the bonding agent and to prevent pooling of bonding agent in the cavity.

The cavities are filled with a light-curing composite or resin-modified glass ionomer, which may be cured at this time (Fig. 17-3, E). A light-curing sealant is placed over the remaining susceptible areas and brushed into the pits and grooves (Fig. 17-3, F). The materials are polymerized with visible light in accord with the manufacturer's instructions.

The rubber dam is removed, and the occlusal contacts are checked. A small-particle diamond rotary instrument can be used to remove excess sealant and ensure centric stops on enamel (Fig. 17-3, G and FT).

A meticulous technique is used in the selection, preparation, and restoration of small pit and fissure caries using the preventive resin restoration.

The bonded restoration with sealant overlay has proven long-term effectiveness. The restorations have equivalent or better success than amalgam restorations. Once again, however, success is dependent upon keeping the sealant intact.

The use of flowable composite systems is also gaining in popularity because they are easy to apply and because evidence shows that less microleakage occurs with these systems than when teeth are restored with condensable composite resins, such as sealant materials that have slightly more filler than filled sealants. Therefore, the practical results of sealing with a flowable or a filled sealant should not differ.

There is no single perfect conservative restoration. Each dentist must decide, on an individual basis, the appropriate type of procedure. The restoration described can be very effective in carefully selected cases.

Walker et al reported on preventive resin restorations placed in patients 6 to 18 years of age and observed for up to 6.5 years.[33] Of the 5185 restorations, 83% did not require further intervention. Those requiring intervention included 37% that needed sealant alone and 21% that required treatment because of the development of an interproximal lesion. Houpt, Fuks, and Eidelman reported complete retention of 54% of their preventive resin restorations, partial loss of sealant in 25%, and complete loss of sealant in 20% after 9 years.[3"] Caries occurred in 25% of the teeth that had sealant loss, and 88% of the restored surfaces remained caries free 9 years after treatment. These researchers concluded that preventive restorations produce excellent long-term results. Conservative cavity preparation with sealing for prevention is a successful approach for treating selected decayed teeth.

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CHAPTER 18

Restorative Dentistry

RALPH E. McDONALD

DAVID R. AVERY

انتشارات
شایان نمودار

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Advances in preventive dentistry and their application in the private dental office, the widespread acceptance of communal fluoridation, and greater emphasis on dental health education have dramatically changed the nature of dental practice. Today the dentist devotes more time to preventive procedures and less time to the routine restoration of carious teeth.

Nevertheless, the restoration of carious lesions in primary and young permanent teeth continues to be among the important services that pediatric dentists and general practitioners provide for the children in their practices. Patients and fellow practitioners often judge dentists on the effectiveness of their preventive programs and the skill with which they perform routine operative procedures.

The *Reference Manual* of the American Academy of Pediatric Dentistry (AAPD) includes a Guideline on Pediatric Restorative Dentistry (revised in 1998 and 2001) that states in part:

> Restorative treatment shall be based upon the results of an appropriate clinical examination and ideally be part of a comprehensive treatment plan. The treatment plan shall take into consideration:
>
> 1. the developmental status of the dentition;
> 2. a caries-risk assessment based upon the caries history of the patient;
> 3. the patient's oral hygiene;
> 4. anticipated parental compliance and likelihood of timely recall;
> 5. the patient's ability to cooperate for treatment.
>
> The restorative treatment plan must be prepared in conjunction with an individually tailored preventive program.
>
> Restoration of primary teeth differs significantly from restoration of permanent teeth, due in part to the differences in tooth morphology.'

The AAPD, with financial assistance from the American Society of Dentistry for Children, held a pediatric restorative dentistry consensus conference on April 15-16, 2002, in San Antonio, Texas. Sixteen literature review and position papers were presented at the conference and numerous consensus statements about appropriate pediatric restorative materials and procedures were developed. All of the papers and the consensus statements may be found in *Pediatric Dentistry 24(5),* Sept/Oct 2002.

STATUS OF COMMON RESTORATIVE MATERIALS

Recent advances in the development of improved biomaterials for dental restorations have been rapid, and they continue to occur at a fast pace. This fact creates a significant challenge for dentists striving to remain at the cutting edge of dental technology. At this writing the more common restorative materials used in pediatric dentistry are composite and other resin systems, glass ionomers, silver amalgam alloys, and stainless steel alloys. Porcelain and cast metal alloy materials are also used in pediatric restorative dentistry but less frequently than those listed in the previous sentence.

Composite resins, glass ionomers, or some combination of the two are being used progressively more and silver amalgam progressively less in pediatric restorative dentistry. Many pediatric dentistry practices do not use silver amalgam at all; instead, some form of composite resin and/or glass ionomer is used. These materials have bonding capability. Glass ionomers may be considered pharmacologically therapeutic because they release fluoride over time; they also have minimal shrinkage during setting. Composite resins possess durability and superior esthetic qualities. When managed properly, both materials are capable of providing superior marginal sealing at the tooth—restorative material interface. The manufacturers of these materials have also combined them in an effort to join the primary advantages of each type of material. Berg has suggested that we think of these materials and their combinations on a continuum, with glass ionomer on the left, composite resin on the right, and the combined materials somewhere in between depending on the relative amounts of each material in the mix. Two major categories on the continuum are described as "resin-modified glass ionomer" (or "hybrid ionomer" or "light-cured glass ionomer") and "compomers" (or "polyacid-modified composite resin" or "glass ionomer—modified composite resin"). A fifth formula-tion has been added on the right side of the continuum in the form of " flowable composite resin." Berg points out that knowing the particular strengths and weaknesses of each type of material on the continuum will enhance the clinician's ability to make the best choices for each individual restorative situation.' Use of any of these restorative materials generally requires more effort and time than corresponding conventional amalgam restorations.

Despite its declining use, silver amalgam remains one of the most durable and cost-effective restorative materials. Success in using this filling material depends on adherence to certain principles of cavity preparation that do not always apply when materials on the glass ionomer—composite resin continuum are used. Some renewed interest in silver amalgam has occurred because of the development of "bonded amalgams." Bonded amalgams are silver amalgam restorations that have been condensed into etched cavity preparations lined with a dentin-bonding agent and some material on the glass ionomer—composite resin continuum. Bonded amalgams require considerable extra effort and

expense to place compared with conventional amalgam restorations. The improvements in tooth support and marginal integrity gained with these restorations have been demonstrated in many studies. Some longer-term studies, however, suggest that the advantages of bonded amalgams may be transient and relatively short-lived, possibly 1 year or less.[3,4] In general, the use of bonded amalgams seems difficult to justify for the routine restoration of primary teeth because traditional silver amalgam should provide comparable quality more efficiently and cost effectively in most situations.

Stainless steel alloy is another commonly used pediatric restorative material. It is used extensively for full coronal coverage restorations of primary teeth. Stainless steel crowns have undoubtedly preserved the function of many primary teeth that otherwise would have been unrestorable. In addition, stainless steel crowns are often used to restore all posterior teeth in young patients with high caries risk who exhibit multiple proximal lesions that could otherwise be restored with silver amalgam or esthetic materials. Crowns are used instead simply because they better protect all posterior tooth surfaces from developing additional caries in the near future and because the posterior crown restoration has proven to be the most durable and cost effective in the primary dentition. Anterior stainless steel crowns may have labial resin or porcelain veneers to enhance esthetics.

MAINTENANCE OF A CLEAN FIELD

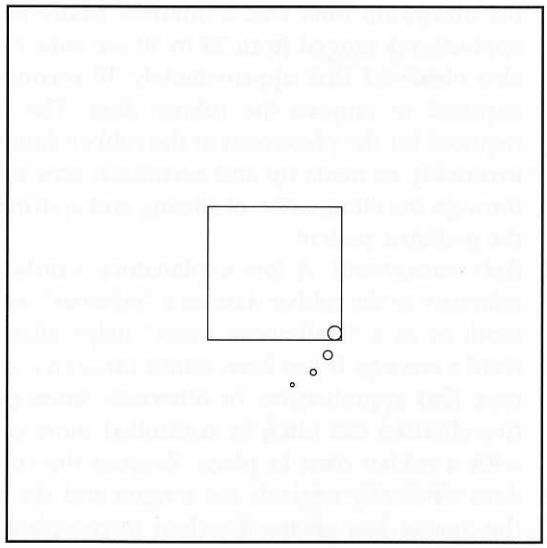
The maintenance of a clean operating field during cavity preparation and placement of the restorative material helps ensure efficient operation and development of a serviceable restoration that will maintain the tooth and the integrity of the developing occlusion.

The rubber dam aids in the maintenance of a clean field. It is generally agreed that the use of the rubber dam offers the following advantages:

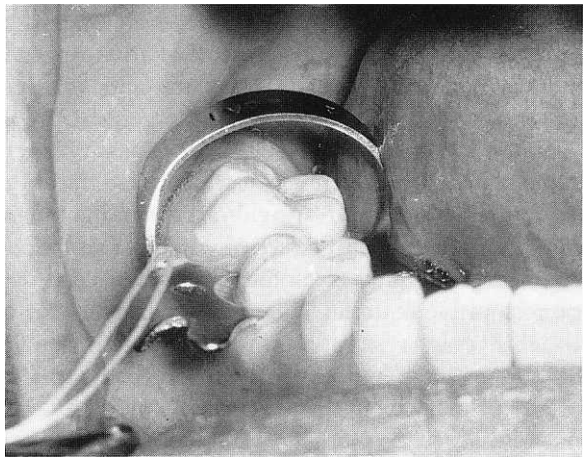
1. *Saves time.* The dentist who has not routinely used the rubber dam needs only to follow the routine presented later in this chapter or a modification of it for a reasonable period to be convinced that operating time can be appreciably reduced. The time spent in placing the rubber dam is negligible, provided that the dentist works out a definite routine and uses a chairside assistant. Heise reported an average time of 1 minute, 48 seconds to isolate an average of 2.8 teeth with the rubber dam in 302 cases.[5] These applications of the rubber dam, placed with the aid of a capable dental assistant, were for routine operative dentistry procedures. The minimum time recorded for placing a rubber dam was 15 seconds (single-tooth isolation), and the maximum time was 6 minutes. Many of the applications ranged from 25 to 50 seconds. Heise also observed that approximately 10 seconds is required to remove the rubber dam. The time required for the placement of the rubber dam will invariably be made up and additional time saved through the elimination of rinsing and spitting by the pediatric patient.
2. *Aids management. A* few explanatory words and reference to the rubber dam as a "raincoat" for the tooth or as a "Halloween mask" helps allay the child's anxiety. It has been found through experience that apprehensive or otherwise uncooperative children can often be controlled more easily with a rubber darn in place. Because the rubber darn efficiently controls the tongue and the lips, the dentist has greater freedom to complete the operative procedures.
3. *Controls saliva.* Control of saliva is an extremely important consideration when one is completing an ideal cavity preparation for primary teeth. The margin of error is appreciably reduced when a cavity is prepared in a primary tooth that has a large pulp and extensive carious involvement. Small pulp exposures may be more easily detected when the tooth is well isolated. It is equally important to observe the true extent of the exposure and the degree and type of hemorrhage from the pulp tissue. Thus the rubber dam aids the dentist in evaluating teeth that are being considered for vital pulp therapy.
4. *Provides protection.* The use of the rubber dam prevents foreign objects from coming into contact with oral structures. When filling material, debris, or medicaments are dropped into the mouth, salivary flow is stimulated and interferes with the operative or restorative procedure. *A* rubber dam also prevents the small child in a reclining position from swallowing or aspirating foreign objects and materials.
5. *Helps the dentist to educate parents.* Parents are always interested in the treatment that has been accomplished for their child. While the rubber dam is in place, the dentist can conveniently show parents the completed work after an operative procedure. The rubber dam creates the feeling that the dentist has complete control of the situation and that a conscientious effort has been made to provide the highest type of service.

ARMAMENTARIUM FOR RUBBER DAM PLACEMENT

The armamentarium consists of 5 x 5 inch sheets of medium latex, a rubber dam punch, clamp forceps, a selection of clamps, a flat-blade instrument, dental



f **I C 18-1.** The corners of the square represent points where punch holes should be made for the clamp-bearing tooth.



1 **1 1 1 18-2.** An Ivory No. 3 clamp has been trial-fitted to the second primary molar. The clamp will be removed and placed in the rubber dam.

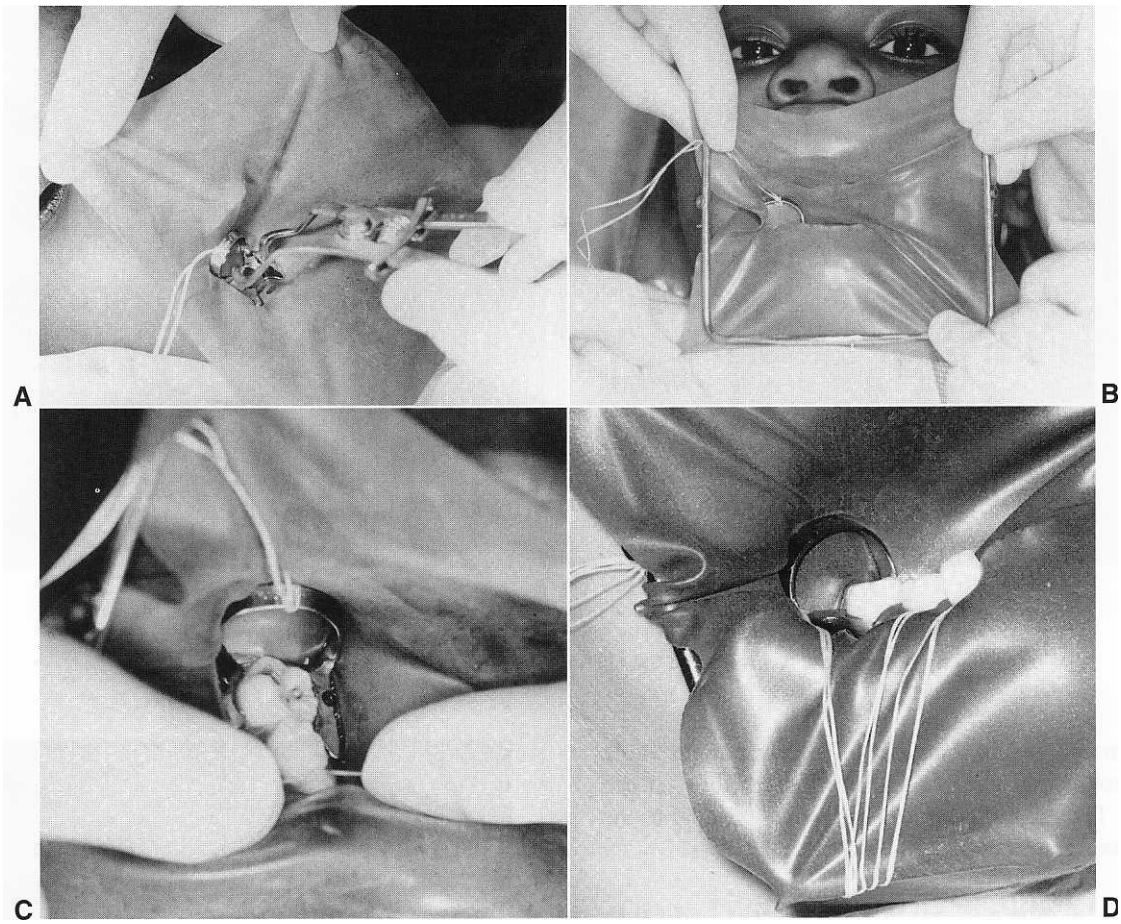
floss, and a rubber dam frame. If one visualizes an approximately $1^1/4$-inch square in the center of a sheet of rubber darn, each corner of the square indicates where the punch holes for the clamp-bearing tooth in each of the four quadrants of the mouth are to be made (Fig. 18-1). As experience is gained in applying the dam, the dentist and assistant will soon learn the proper location for punching the holes. If the holes are punched too far apart, the dam will not readily fit between the contact areas. In addition, the greater bulk of material between the teeth will greatly increase the possibility that the rubber will become a barrier to proximal surface preparation. Conversely, if the holes are punched too close together, salivary leakage will contaminate the operating field. In general, the holes should be punched the same distance apart as the holes on the cutting table of the rubber darn punch.

The large punch hole is used for the clamp-bearing tooth and for most permanent molars. The medium-sized punch hole generally is used for the premolars and primary molars. The second smallest hole is used for maxillary permanent incisors, whereas the smallest hole is adequate for the primary incisors and lower permanent incisors.

SELECTION OF A CLAMP

The operator will soon develop a personal preference for which clamps to use to secure the darn in isolating different areas in the mouth. Unless the clamp is firmly anchored to the tooth, the tension of the stretched rubber will easily dislodge it. Therefore the proper selection of a clamp is of utmost importance. It is recommended that the clamp be tried on the tooth before the rubber darn is placed to ascertain that the clamp can be securely seated and will not be easily dislodged by the probing tongue, lip, or cheek musculature. An 18-inch length of dental floss should be doubled and securely fastened to the bow of the clamp. The floss will facilitate retrieval in the unlikely event that the clamp should slip and fall toward the pharynx (Fig. 18-2).

The following procedure is recommended for rubber dam application (Fig. 18-3). The previously selected and ligated clamp is placed in the rubber darn. The dentist grasps the clamp forceps with the clamp engaged. The assistant, seated to the left of the patient (the dentist is right-handed in this example), grasps the upper corners of the dam with the right hand and the lower left corner between the left thumb and index finger. The dam is moved toward the patient's face as the dentist carries the clamp to the tooth while holding the lower right portion of the dam. After securing the clamp on the tooth, the dentist transfers the clamp forceps to the assistant, who receives it while continuing to hold the upper corners of the dam with the right hand. The dentist then places the frame over the rubber darn. Together the assistant and dentist attach the corners of the dam to the frame. The flat blade of a plastic instrument or a right-angle explorer may be used to remove the rubber darn material from the wings of the clamp and to complete the seal around the clamped tooth. If necessary, light finger pressure may seat the clamp securely by moving it cervically on the tooth. If additional teeth are to be isolated, the rubber is stretched over them, and the excess rubber between the punched holes is placed between the contact areas with the aid of dental floss. The most anterior tooth and others if necessary are ligated to aid in the retention of the darn



[**I C 18-3. A,** The dental assistant holds the top and lower right corners of the rubber dam as the dentist holds the lower left corner and carries the clamp to the tooth. **B,** The assistant and dentist attach the corners of the rubber dam to the frame. C, Dental floss is used to carry the rubber dam between the teeth. D, The teeth are isolated and ready for the operative procedure. *(A,* **B,** *and C courtesy Dr. Richard Troyer.)*

and the prevention of cervical leakage. The free ends of the floss are allowed to remain, because they may aid in the further retraction of the gingival tissue or the patient' s lip during the operative procedure. At the end of the operative procedure, the length of floss will also aid in removing the ligature.

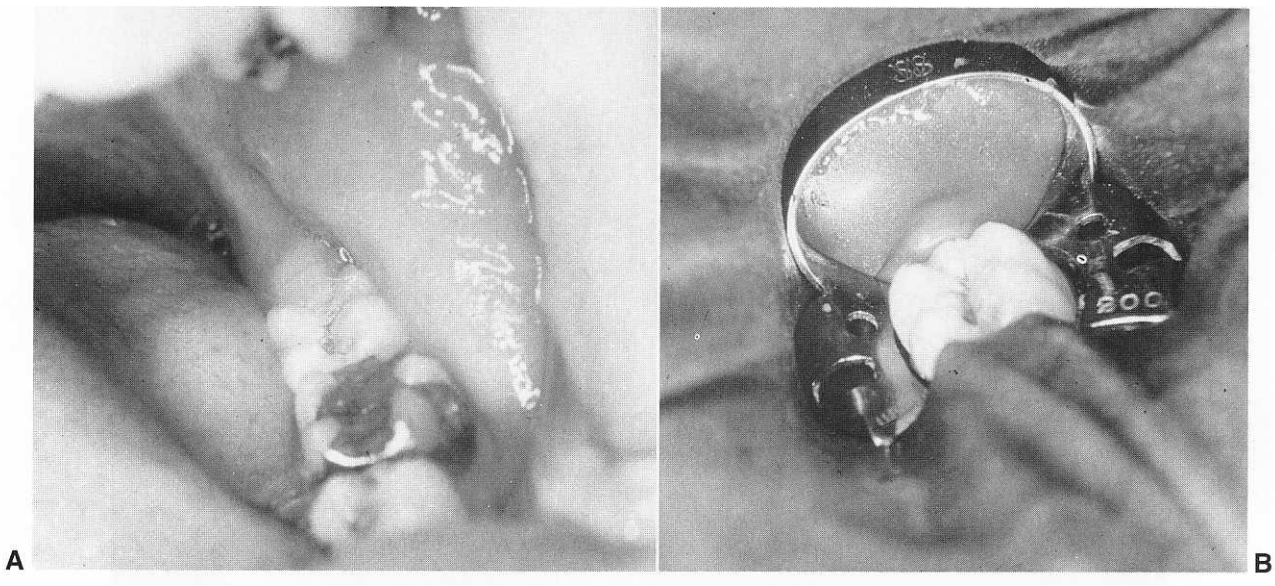
When a quadrant of restorations in the primary dentition is planned and no pulp therapy is anticipated, Croll recommends the "slit-dam method."' One long opening is made in the dam, and the entire quadrant is isolated without interseptal dam material between the teeth.

It is unwise to include more teeth in the rubber dam than are necessary to isolate the working area adequately. If the first or second permanent molar is the only tooth in the quadrant that is carious and if it requires only an occlusal preparation, it is often desirable to punch only one hole in the dam and to isolate the single tooth (Fig. 18-4). This procedure will require only seconds and will save many minutes.

MORPHOLOGIC CONSIDERATIONS

The crowns of the primary teeth are smaller but more bulbous than the corresponding permanent teeth, and the molars are bell-shaped, with a definite constriction in the cervical region. The characteristic sharp lingual inclination occlusally of the facial surfaces results in the formation of a distinct faciogingival ridge that ends abruptly at the cementoenamel junction. The sharp constriction at the neck of the primary molar necessitates special care in the formation of the gingival floor during class II cavity preparation. The buccal and lingual surfaces of the molars converging sharply occlusally form a narrow occlusal surface or food table; this is especially true of the first primary molar.

The pulpal outline of the primary teeth follows the dentoenamel junction more closely than that of the permanent teeth. The pulpal horns are longer and more pointed than the cusps would indicate. The dentin also has less bulk or thickness, and so the pulp is



f **I G 18-4. A,** The second permanent molar requires an occlusal restoration. It is not necessary to isolate more than a single tooth. **B,** A No. 200 clamp has been selected to hold the rubber dam in place. The rubber dam has retracted the tissue that extended over the distal marginal ridge.

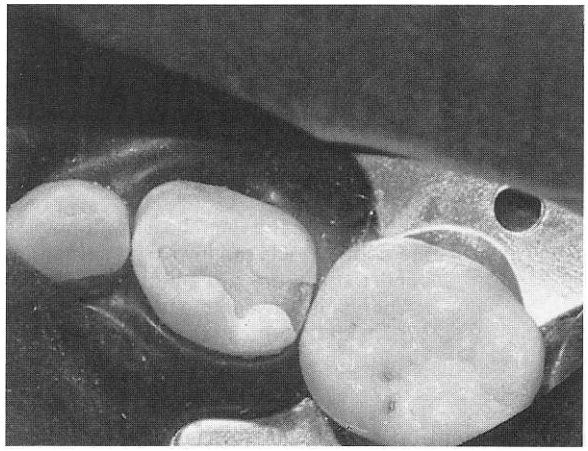
proportionately larger than that of the permanent teeth. The enamel of the primary teeth is thin but of uniform thickness. The enamel surface tends to be parallel to the dentinoenamel junction.

BASIC PRINCIPLES IN THE PREPARATION OF CAVITIES IN PRIMARY TEETH

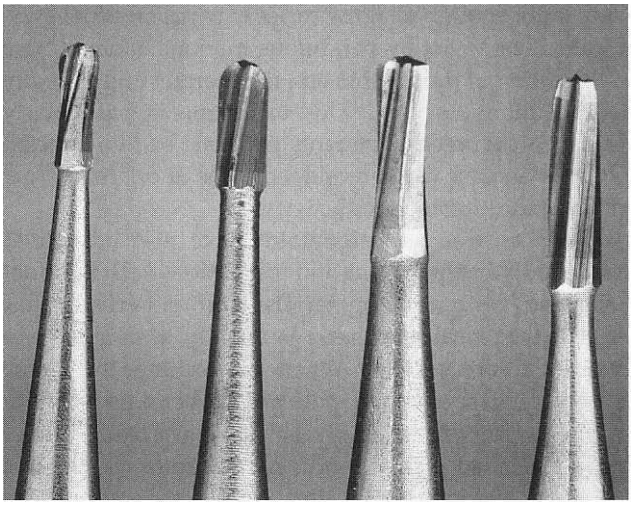
Traditional cavity preparations for class I and class II lesions include areas that have carious involvement and, in addition, those areas that retain food and plaque material and may be considered areas of potential carious involvement. A flat pulpal floor is generally advocated. However, a sharp angle between the pulpal floor and the axial wall of a two-surface preparation should be avoided. Rounded angles throughout the preparation will result in less concentration of stresses and will permit better adaptation of the restorative material into the extremities of the preparation.

Although the traditional class I cavity preparation and restoration may occasionally be the most practical treatment for a tooth in certain circumstances, such treatment is currently obsolete for most class I lesions. The traditional treatment has been replaced, for the most part, by conservative caries excavation and restoration using a combination of bonding restorative and sealant materials (see Chapter 17).

Likewise, the traditional class II cavity preparation and restoration, though not yet considered obsolete, is currently used less frequently as steadily improving restorative materials with therapeutic and bonding capability are developed. In the traditional class II cavity preparation for amalgam, the buccal and lingual extensions should be carried to self-cleansing areas. The cavity design should have greater buccal and lingual extension at the cervical area of the preparation to clear contact with the adjacent tooth. This divergent pattern is necessary because of the broad, flat contact areas of the primary molars and because of the distinct buccal bulge in the gingival third. Ideally, the width of the preparation at the isthmus should be approximately one third the intercuspal dimension (Fig. 18-5). The axiopulpal line angle should be beveled or grooved to reduce the concentration of stresses and to provide greater bulk of material in this area, which is vulnerable to fracture.



[I 1 **18-5.** Conservative but adequate extension of a traditional class II cavity preparation in a mandibular first primary molar.



f **I L 18-6.** Rounded-end, high-speed carbide burs No. 329, No. 330, No. 245, and No. 256, which may be used for cutting cavity preparations.

Because many occlusal fractures of amalgam restorations are caused by sharp opposing cusps, it is advisable to identify these potentially damaging cusps with articulating paper before cavity preparation. The slight reduction and rounding of a sharp opposing cusp will reduce the number of such fractures.

CAVITY PREPARATION IN PRIMARY TEETH

The steps in the preparation of a cavity in a primary tooth are not difficult but do require precise operator control. Many authorities advocate the use of small, rounded-end carbide burs in the high-speed handpiece to establish the cavity outline and perform the gross preparation. For efficiency and convenience, all necessary high-speed instrumentation for a given preparation may be completed with a single bur in most situations. Therefore the dentist should select the bur that is best designed to accomplish all the high-speed cutting required for the procedure being planned. Fig. 18-6 illustrates four high-speed carbide burs designed to cut efficiently and yet allow conservative cavity preparations with rounded line angles and point angles. Alternatively, cavity preparations may be made with aluminum oxide air abrasion systems or with laser systems approved for hard-tissue procedures.

INCIPIENT CLASS I CAVITY IN A VERY YOUNG CHILD

During the routine examination of a child younger than 2 years of age, the dentist may occasionally discover a small but definite carious lesion in the central fossa of one or two first primary molars, with all other teeth being sound. Thus restorative needs are present but minimal. Because of the child's psychologic immaturity and because it is usually impossible to establish effective communication with the child, the parent should hold the child on his or her lap in the dental chair. This will help the child feel more secure and provide a better opportunity to restrain the child's movement during the operative procedure. The small-cavity preparation may be made without the aid of a rubber dam or local anesthetic. A No. 329 or No. 330 bur is used to open the decayed area and extend the cavo-surface margin only to the extent of the carious lesion. If the patient is resistant (usually), completing the preparation with an air abrasion or laser system would be inconvenient. The preparation can be completed in just a few seconds. Restoring the tooth with amalgam or a resin-modified glass ionomer will arrest the decay and at least temporarily prevent further tooth destruction without a lengthy or involved dental appointment for the child. If the child is cooperative, a preventive resin restoration, preceded by application of a dentin-bonding agent, may be used.

PIT OR FISSURE CLASS I CAVITY

The preparation and restoration of a pit or fissure class I cavity are discussed in chapter 17 (see the section on preventive resin restoration).

DEEP-SEATED CLASS I CAVITY

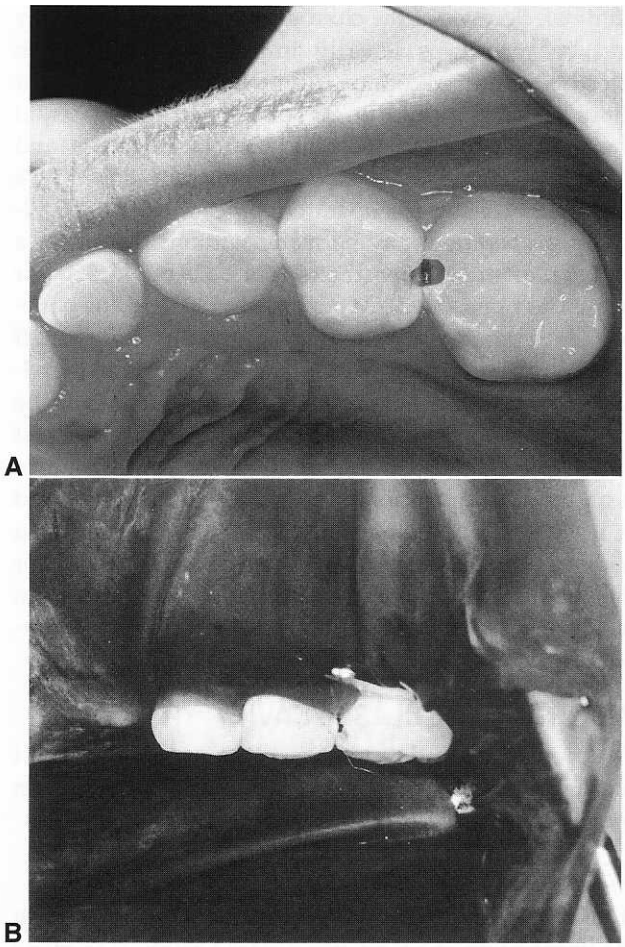
If an amalgam restoration is planned, the first step in the preparation of an extensive class I cavity is to plane back the enamel that overhangs the extensive carious lesion. Then the cavity preparation should be extended throughout the remaining grooves and anatomic occlusal defects. The carious dentin should next be removed with large, round burs or spoon excavators. If a carious exposure is not encountered, the cavity walls should be paralleled and finished as previously described. With deep carious lesions and near pulp exposures, the depth of the cavity should be covered with a biocompatible base material to provide adequate thermal protection for the pulp.

If a composite resin and/or glass ionomer restoration is planned, any disease-free pits and grooves may be sealed as part of the bonded restoration. The restorative material will also provide thermal insulation to the pulp.

CLASS II CAVITY

Proximal lesions in a preschool child indicate excessive caries activity; a preventive and restorative program should be undertaken immediately.

Small Lesions. Very small incipient proximal lesions may be chemically restored with topical fluoride therapy provided by the dentist, along with the judicious use of fluoride products designed for topical



F I G . 18-7. Approximating conservative preparations to remove small class II carious lesions in primary molars. A, Marginal ridge access. **B**, Facial surface access.

application at home. If this treatment regimen is accompanied by improved diet and improved oral hygiene, some incipient proximal lesions may remineralize or remain in an arrested state indefinitely. However, the parents should be informed of the incipient lesions and emphasis should be placed on the need to continue practicing the recommended procedures and to bring the child back for periodic examinations. If the parents and the patient do not follow the instructions properly, subsequent bite-wing radiographs will reveal growth of the lesion, and restorative procedures should be initiated before the defects become extensive carious lesions.

As bonded restorations have improved, especially those restorations capable of fluoride release, more conservative cavity preparation designs have also been advocated. In otherwise sound teeth free of susceptible pits and fissures, accessing small class II carious lesions via small openings in the marginal ridges or in the facial surfaces of the teeth is becoming a popular technique (Fig. 18-7). Gaining access to the lesion with openings only large enough to allow caries excavation is the goal. Caries is removed by pendulous motions of small burs or by tilting of the air abrasion tip laterally and pulpally at the initial opening. This technique is particularly useful in cooperative patients with one or two affected primary molars who are judged to be at relatively low risk for additional caries activity.

Croll' as well as Vaikuntam[$] have also advocated conservative preparations and restorations with fluoride-releasing restorative materials. Our experience has shown that local anesthesia is usually unnecessary to make the preparation. When performing this short procedure in cooperative patients, rubber dam isolation is often optional, especially on maxillary teeth. Use of resin-modified glass ionomer materials results in excellent restorations for this conservative procedure (Fig. 18-8).

Marks et al" and also Welbury[10] et al (who also restored class I preparations) have reported *very* satisfactory results using conservative class II preparations and compomers to restore primary molars in studies of 36 and 42 months' duration, respectively. In a 3-year study, Hubei and Mejare reported the successful performance of conservative class II resin-modified glass ionomer restorations in primary molars."

Lesions with Greater Dentin Involvement. The first step in the traditional preparation of a class II cavity in a primary tooth for an amalgam or an esthetic restoration involves opening the marginal ridge area. Extreme care must be taken when breaking through the marginal ridge to prevent damage to the adjacent proximal surface.

Amalgam. The gingival seat and proximal walls should break contact with the adjacent tooth. The angle formed by the axial wall and the buccal and lingual walls of the proximal box should approach a right angle. The buccal and lingual walls necessarily diverge toward the cervical region, following the general con-tour of the tooth (Fig. 18-9). The occlusal extension of the preparation should include any caries-susceptible pits and fissures. If the occlusal surface is sound and not caries susceptible, then a minimal occlusal dovetail is still often needed to enhance the cavity retention form. If carious material remains after the preparation outline is established, it should next be removed. The appropriate liner or intermediate base, if indicated, and a snug-fitting matrix should be placed before the insertion of the amalgam.

Esthetic Materials. Because of the improvements in the properties of composite resins, many dentists use them routinely for posterior restorations. More recently the use of glass ionomer restoratives (or other materials on the glass ionomer—composite resin continuum) has also been advocated. The preparation and restoration may be similar to that described earlier for amalgam

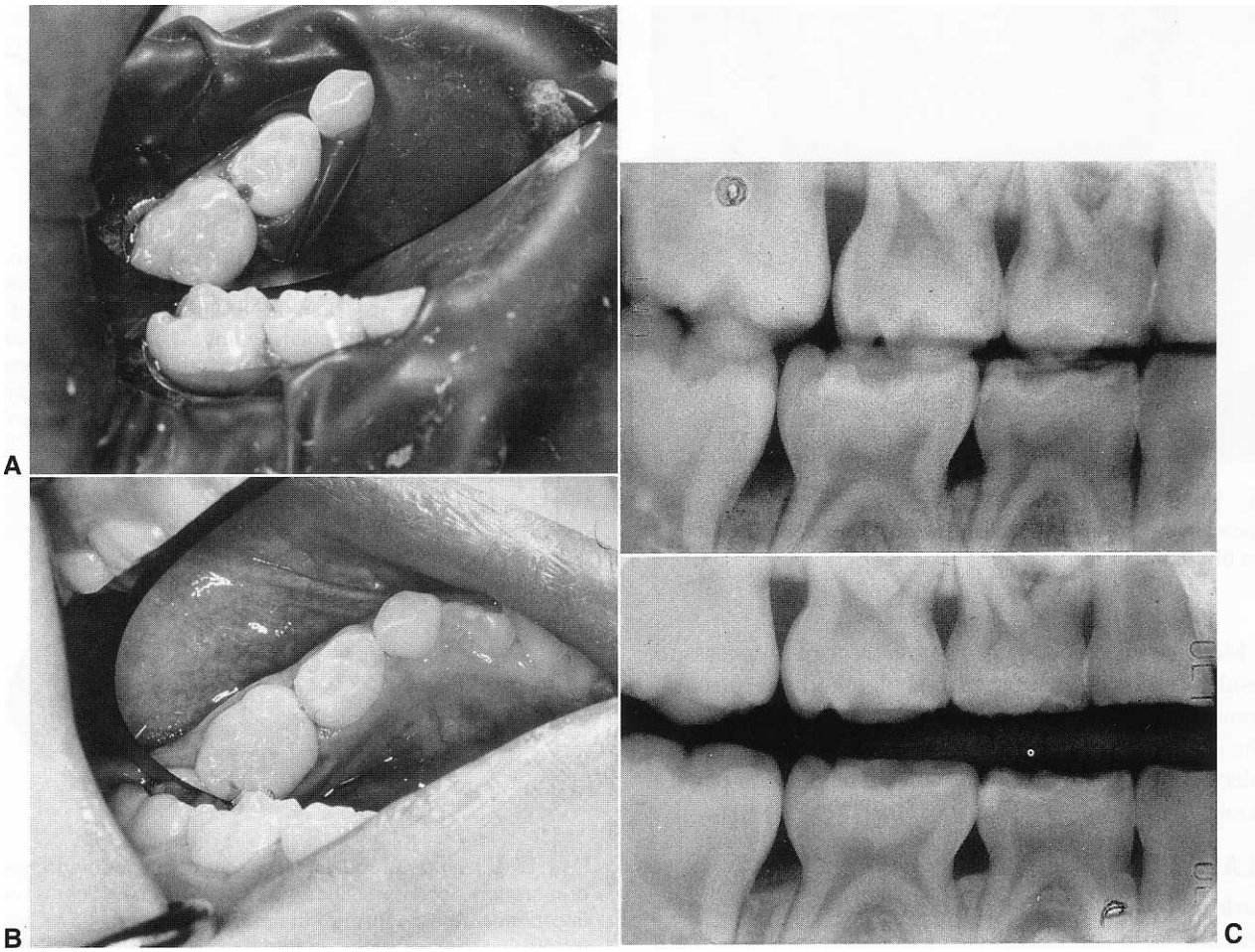


FIG 18-8. **A,** Conservative class II preparation. **B,** Resin-modified glass ionomer restoration. C, Preoperative radiograph *(top)* and 17-month postoperative film.

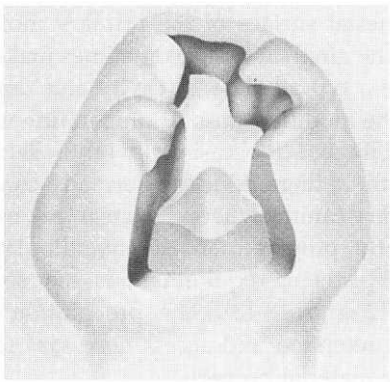
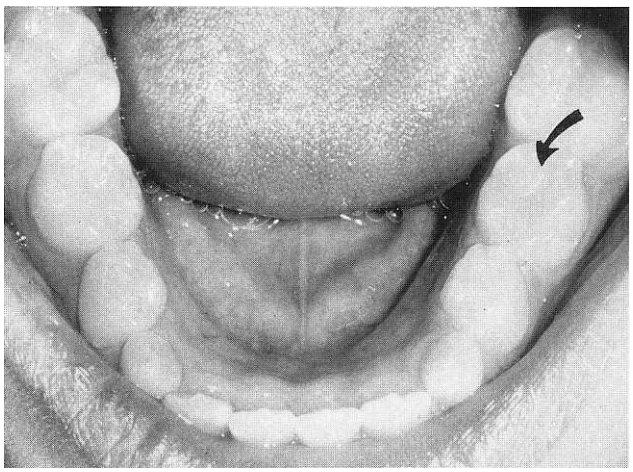


FIG 18-9. Traditional class II cavity preparation for a primary molar. The preparation includes diverging proximal walls and a beveled and grooved axiopulpal line angle.

when significant caries exists on both the occlusal and proximal surfaces (Fig. 18-10). However, little or no occlusal preparation may be required when the occlusal pits and fissures are caries susceptible but sound or incipient. Then the proximal restoration may be combined with application of an occlusal sealant (with or without enameloplasty). Whenever composite restorative materials are employed, enamel beveling, etching, and application of bonding agents are recommended.

Clinical trials of restoration of primary molars reported by Paquette et al[12] and by Oldenburg, Vann, and Dilley [13] revealed that traditional preparations modified only by beveling of enamel margins and restored with bonded composite resins yielded highly successful results during 12- and 24-month observation periods. Tonn and Ryge have also reported acceptable 2-year results for primary molars restored with bonded composite resins in traditional cavity preparations modified only by beveling of enamel margins.[14]

Dilley et al have demonstrated that the placement and finishing of posterior composite restorations are significantly more time consuming than those for comparable amalgam restorations.' In addition to increasing the cost of care, the extra time required for treatment may complicate patient management for some young patients.



(I G **18-10.** A distal occlusal composite resin restoration was placed in the lower left second primary molar 6 months before the photograph.

More recently Donly et al have reported successful results for class II resin-modified glass ionomer restorations in primary molars after 3 years of observation." The dentist's sound professional judgment is the key to selecting the restoration that will best serve the patient in each situation.

CLASS III CAVITY

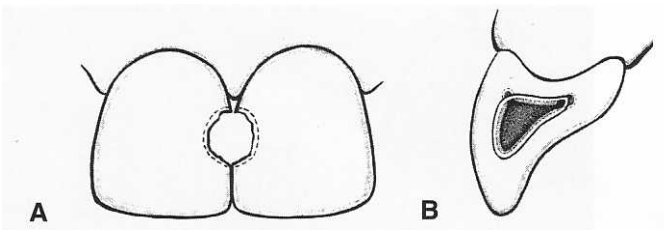
Carious lesions on the proximal surfaces of anterior primary teeth sometimes occur in children whose teeth are in contact and in children who have evidence of arch inadequacy or crowding. Carious involvement of the anterior primary teeth, however, may be interpreted as evidence of excessive caries activity requiring a comprehensive preventive program.

If the carious lesion has not advanced appreciably into the dentin and removal of the caries will not involve or weaken the incisal angle, a small conventional class III cavity may be prepared and the tooth may be restored with the dentist's choice of bonding materials (Fig. 18-11).

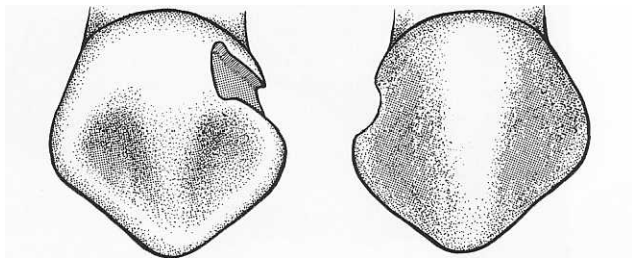
Mandibular primary incisors with small proximal carious lesions may not require conventional restorations at all. Enameloplasty of the affected proximal surface (usually described as "disking") to open the proximal contact and to remove most, if not all, of the cavitation, followed by topical treatments with fluoride varnish, will often suffice until the teeth exfoliate naturally. Extraction is usually indicated when mandibular primary incisors have extensive caries.

MODIFIED CLASS III CAVITY PREPARATION

The distal surface of the primary canine is a frequent site of caries attack in patients at high risk for caries if the canine is in proximal contact with the first molar. The position of the tooth in the arch, the characteristically



f **I G 18-11.** A, Schematic drawing of carious lesions on the mesial surfaces of maxillary primary central incisors that do not undermine the mesial angles of the teeth. The dotted line indicates the proposed labial outline of the class III cavity preparation. **B,** Proximal view illustrates that the class III preparation is limited to the cervical two thirds of the primary incisor. *(From Roche JR: Restorative dentistry. In Goldman HM et al, editors:* Current therapy in dentistry, *vol4, St Louis, 1970, Mosby.)*



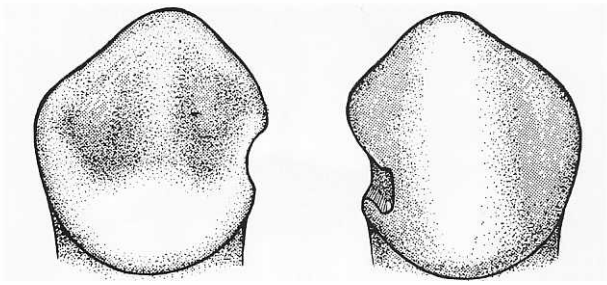
f **I G 18-12.** Lingual and labial views of a modified class III preparation for a maxillary primary canine. The dovetail improves retention form of the preparation and allows access for placing the restorative material to ensure adequate contact with the adjacent tooth.

broad contact between the distal surface of the canine and the mesial surface of the primary molar, and the height of the gingival tissue sometimes make it difficult to prepare a typical class III cavity and restore it adequately. The modified class III preparation uses a dovetail on the lingual or occasionally on the labial surfaces of the tooth. A lingual lock is normally considered for the maxillary canine, whereas a labial lock may be more conveniently prepared on the mandibular teeth for which the esthetic requirement is not so important (Figs. 18-12 and 18-13). The preparation allows for additional retention and access necessary to insert the restorative material properly.

RESTORATION OF PROXIMAL INCISAL CARIES IN PRIMARY ANTERIOR TEETH

PREFORMED STAINLESS STEEL BANDS

The use of preformed stainless steel bands has been advocated by McConville and Tonn for the restoration of anterior primary teeth with deep mesial or distal



[I G **18-13.** Lingual and labial views of a modified class III preparation for a mandibular primary canine.

caries involving the incisal angle.' The band is fitted before caries removal. After caries removal, the cavity and the band are filled with a creamy mix of glass ionomer cement, and the band is seated to place. After the cement has hardened, the excess cement is removed.

Although this cemented-band technique was recommended before the development of good esthetic restorative materials, it may still be the restoration of choice in the very young child with early childhood caries when the parents decide against the more time-consuming and expensive esthetic restorative procedures. If the pulps of the teeth are healthy, cemented-band restorations are preferable to extractions. With the use of accepted methods of restraining the child's movements, this procedure can be carried out quickly and yet provide a satisfactory long-term restoration. After the child has gained more maturity and has become a cooperative patient, the bands may be removed and more esthetic restorations provided, if desired.

If the carious involvement of an anterior tooth endangers the pulp, the cemented band is excellent for supporting the intermediate dressing in indirect pulp therapy. Only the gross caries is removed at the initial appointment, and the previously fitted band is cemented on the tooth to seal the cavity and arrest the caries process. A waiting period of at least 6 weeks is indicated to allow adequate reparative dentin formation before the band is taken off the tooth and the remaining caries removed. If there is no evidence of a pulp exposure, the final restorative procedure is carried out.

ESTHETIC RESIN RESTORATION

One type of preparation used for the esthetic restoration of primary incisors in which dental caries approximates or involves the incisal edge of the teeth is illustrated in Fig. 18-14. As with other operative procedures for the pediatric patient, the use of the rubber dam aids in maintenance of a dry field, provides better vision, and facilitates control of the lips and tongue.

The preparation includes a proximal reduction through the incisal angle and the carious lesion, and ends at the established cervical seat. Labial and lingual locks are then prepared in the cervical third of the tooth. The remaining caries is removed, the tooth is etched, and a bonding agent is applied.

A properly placed matrix tightly wedged at the cervical seat aids the operator in placing, shaping, and holding the composite resin during the curing process. A good matrix also simplifies the finishing procedures.

McEvoy has described a similar preparation and restoration for primary incisors, except that the retentive locking component is placed on the labial surface only in the gingival one third of the tooth.'' The lock extends minimally across two thirds of the labial surface and may extend even farther to include decalcified enamel in the cervical area. We would also recommend slightly beveling the enamel margins before etching to further improve the marginal bonding of the restoration.

Initial shaping of the restoration may be accomplished with a flame-shaped finishing bur. The excess resin is removed, and the contour of the restoration is established. The gingival margins may be finished with a sharp scalpel blade. Final polishing may be accomplished with the rubber cup and a fine, moist abrasive material or one of the composite polishing systems (Fig. 18-15).

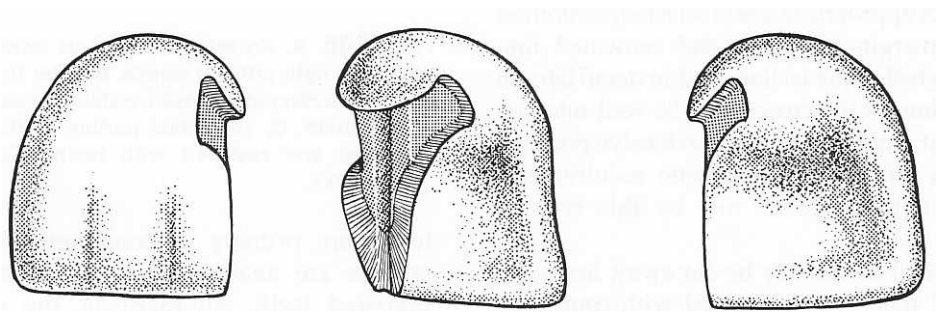
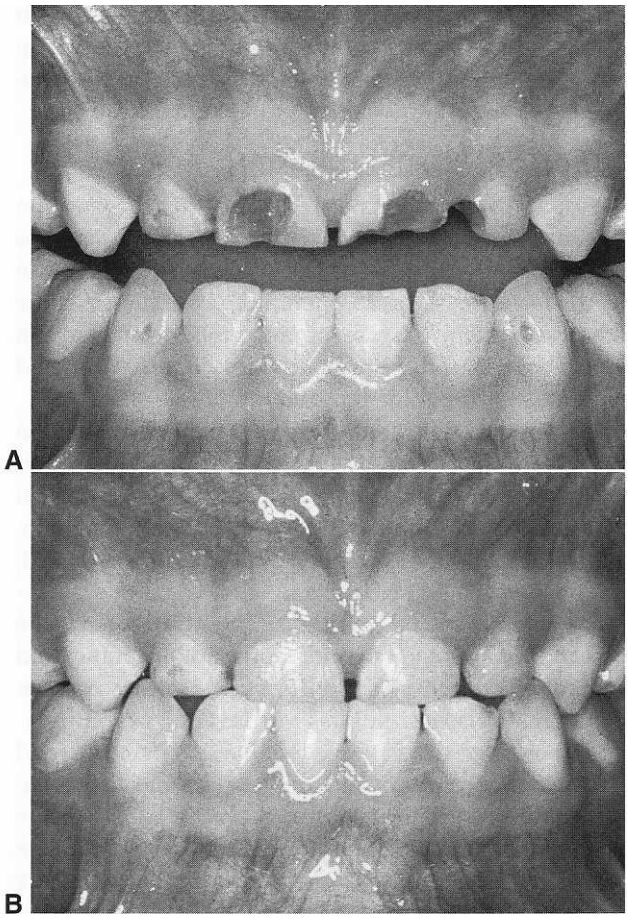


FIG **18-14.** Labial, proximolingual, and lingual views of a preparation for an esthetic resin restoration in a primary incisor. The preparation includes a proximal reduction and the establishment of a definitive cervical seat that extends to labial and lingual locks in the cervical third of the tooth.



[I fi **18-15. A,** Extensive carious lesions of the maxillary right central, left central, and lateral incisors of a $3^1/2$-year-old patient. **B,** Postoperative view of the restored teeth. The restorations are retained with labial and lingual locks incorpo rated in the preparations. The maxillary lateral preparation was designed as illustrated in Fig. 18-14.

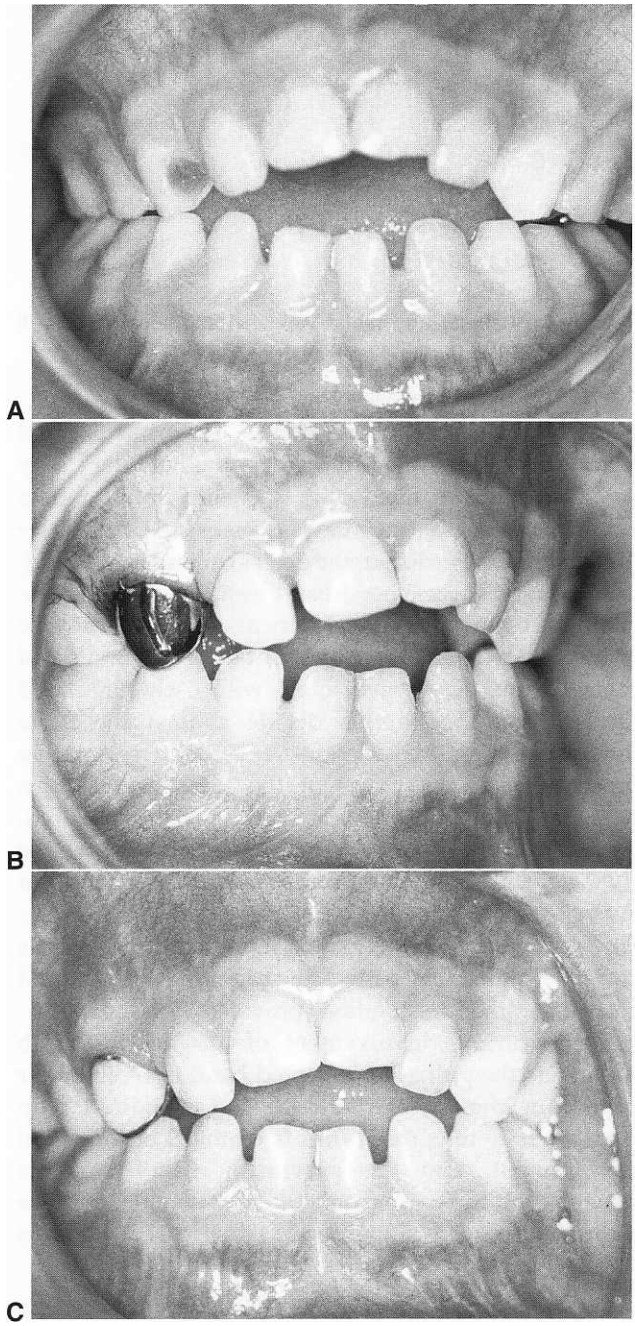
STAINLESS STEEL CROWNS

Primary incisors or canines that have extensive proximal lesions involving the incisal portion of the tooth may be restored with stainless steel crowns.

A steel crown of appropriate size is selected, contoured at the cervical margin, polished, and cemented into place. The crown technique is discussed in detail later in this chapter. Although the crown will be well retained even on teeth that require removal of extensive portions of carious tooth structure, the esthetic requirements of some children may not be met by this type of restoration.

Most of the labial metal may be cut *away,* leaving a labial "window" that is then restored with composite resin (Fig. 18-16). This restoration is called an *open-face stainless steel crown.*

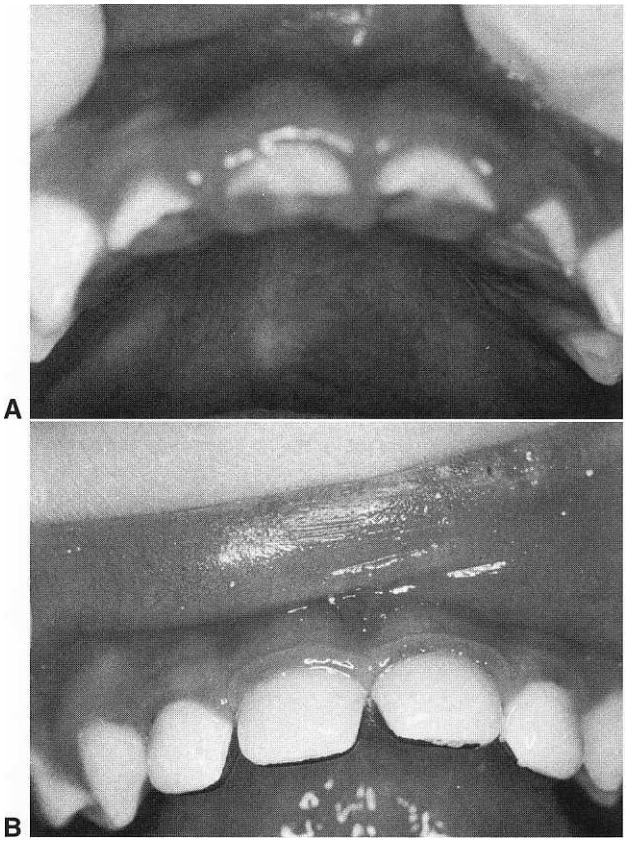
Several brands of stainless steel crowns with esthetic facings veneered to the labial surfaces are also available



[I 1 **18-16. A,** An extensive carious lesion is evident in the maxillary right primary canine. **B,** After the removal of caries and preparation of the tooth, a stainless steel crown was fitted to the tooth. C, The labial portion of the steel crown was removed and restored with resin. *(Courtesy Dr. Lionel Traubman.)*

to restore primary anterior teeth (Fig. 18-17). Such crowns are available for direct adaptation to the prepared teeth. Alternatively, the restorations may

be completed in two appointments, with the labial veneers added in the laboratory after the bare steel crowns are adapted to the teeth but before final cementation.



f 11. **18-17. A,** Extensive caries and coronal destruction of the maxillary incisors in a child 21 months of age. **B,** After pulp therapy, the teeth were restored with veneered stainless steel crowns. *(Courtesy Dr. Gary J. Hinz.)*

Croll recommends that an anterior alginate impression be made before the restorative appointment.[19] The crown preparations can then be simulated on a stone model, and most of the crown adaptation can be achieved in advance. This procedure enables the clinician to cement the crowns at the same appointment at which the preparations are made (rather than waiting for laboratory veneering of the adapted bare crowns). Croll's technique also gives the clinician a better opportunity to focus on fitting the crowns so that optimal tooth alignment will result, which further enhances the esthetic outcome.

DIRECT RESIN CROWNS

Doyle introduced a preparation design for primary incisor jacket crowns that uses an undercut area around the gingival shoulder, retains as much enamel as possible for etching, and preserves the midportion of the natural incisal edge whenever possible to improve retention.[-0] After excavating the caries, protecting the exposed dentin, and etching the enamel, the dentist restores the prepared incisor with a preformed acrylic jacket crown lined with self-curing restorative resin.

Webber et a1[21] have described a resin crown technique very similar to Doyle's except that the tooth is restored with composite resin using a celluloid crown form as a matrix. They did not advocate preserving a portion of the incisal edge. They point out that very little finishing of the restoration is required when the celluloid crown has been properly fitted.

Doyle's idea of preserving a portion of the tooth's natural incisal edge whenever possible seems valid.[20] Preservation of the incisal area of the tooth provides a greater surface area of etched enamel and greater length in the preparation, both of which will improve the retentive quality of the restoration.

The jacket crown technique illustrated in Fig. 18-18 incorporates the use of a celluloid crown form and composite resin as advocated by Webber et a1[21] and also includes the preservation of the tooth's natural incisal edge as advocated by Doyle.[20]

Celluloid crown forms are also available for primary posterior teeth. These crown forms are useful matrices for some posterior bonded restorations. A good example of an indication for using such a crown form is to provide a bonded crown buildup to temporarily reestablish arch integrity and occlusion of an ankylosed (submerged) primary molar.

PREPARATION OF CAVITIES IN YOUNG PERMANENT TEETH

Many of the caries management procedures presented in this textbook also often apply to young permanent teeth. Entire textbooks are devoted to operative dentistry procedures, and the primary focus of these books is restoration of permanent teeth. Repeating all that information (or portions thereof) in this chapter is impractical and realistically impossible. For detailed information about the various cavity preparation designs for permanent teeth and the matrix systems to facilitate the placement and contour of restorations, please consult a standard textbook of operative dentistry listed in the references, such as those by Roberson, Heymann, and Swift[22] and by Baum, Phillips, and Lund.[-']

INTERIM RESTORATION FOR HYPOPLASTIC PERMANENT MOLARS

The dentist who routinely treats children occasionally faces a difficult restorative problem when severely hypoplastic first permanent molars erupt. Often the teeth are so defective that they require restoration at a very early stage of eruption. Many of these teeth have been saved by early restoration with stainless steel crowns as an interim procedure. However, this procedure may require sacrificing sound tooth surfaces to provide adequate space for the crown. Such full-coverage restorations are sometimes difficult to fit.

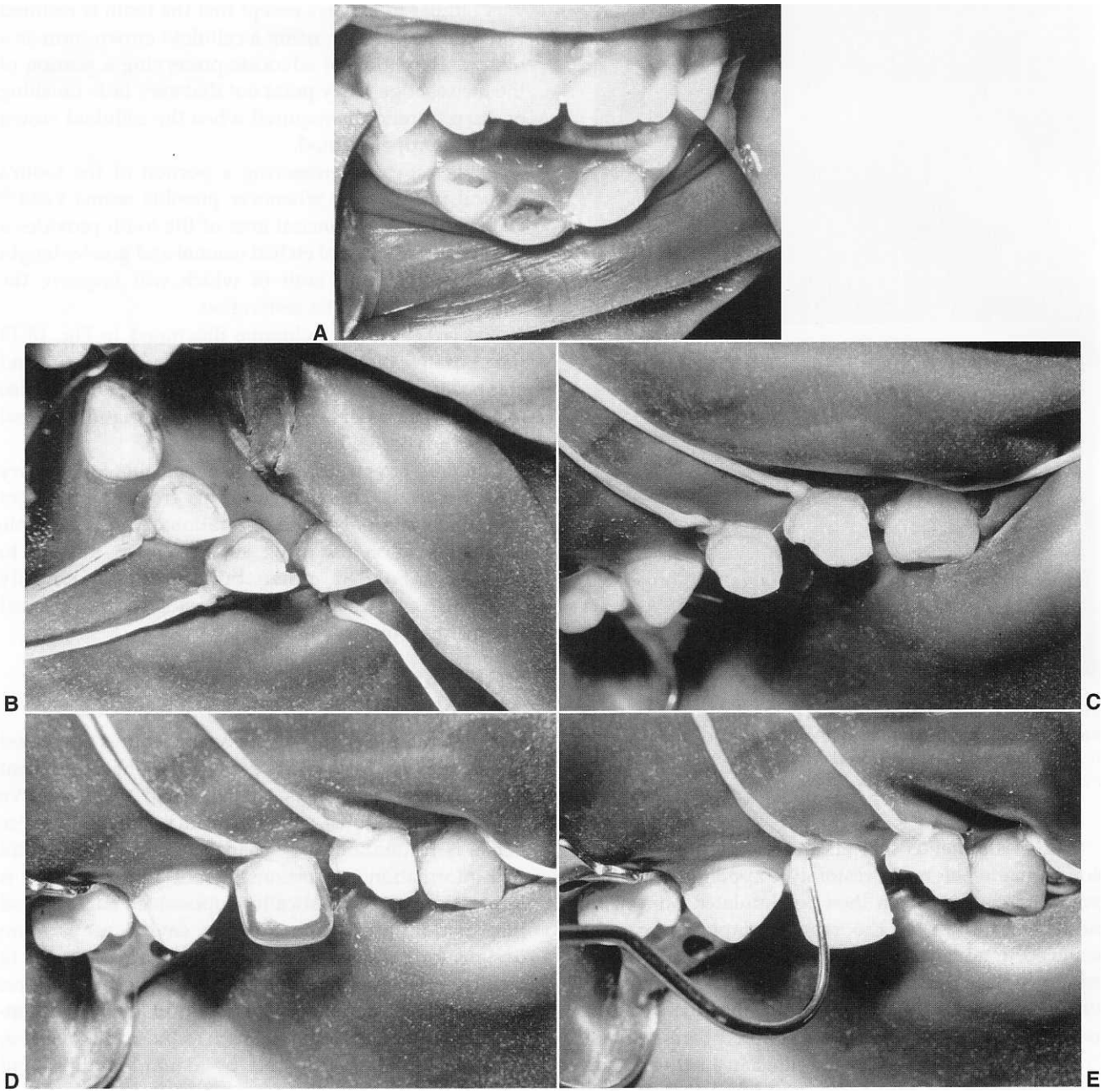
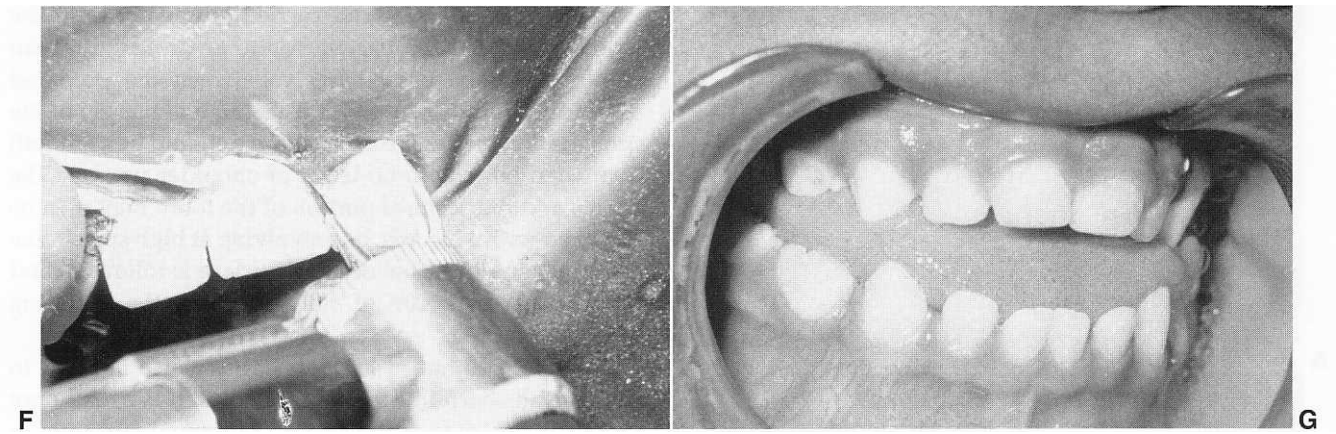


FIG 18-18. **A,** Extensive caries on the lingual surfaces of the maxillary right primary central and lateral incisors in a 2½-year-old patient. The caries has also severely undermined the proximal and incisal surfaces of the teeth. The maxillary left central incisor had been previously restored. **B,** The carious lesions have been excavated, and the exposed dentin has been covered with calcium hydroxide. C, Completed jacket preparations with cervical shoulders, slightly undercut walls at the cervical areas, and preservation of as much enamel and incisal tooth structure as possible. The enamel has been etched. **D,** Fitted celluloid crown form on the lateral incisor. The crown form should be trimmed to fit snugly and to just cover all cervical margins of the preparation. A snug fit at the cervical margin is very desirable, even if the incisal is too long, to minimize cervical finishing of the restoration. **E,** The crown form filled with composite resin has been seated, and the excess material is being carefully removed at the cervical margins with an explorer. Note excess material exuding from the vent hole placed on the mesial incisal of the crown form.



f I C 18-18, contd. **F,** The central incisor was restored in a similar fashion. The incisal edges of the restorations have been trimmed back to the natural tooth structure with a No. 7901 finishing bur, which is also being used to trim the cervical margins and embrasure areas before polishing. **G,** The resin jacket crown restorations 2 months postoperatively. (Courtesy Dr. Robert Rust.)

The composite materials have proved to be a more satisfactory interim restoration for many of these teeth. Such a bonded composite buildup restoration allows preservation of all sound tooth structure and depends on the presence of some enamel surfaces to provide bonded retention for the restorative material. Any soft defective areas are excavated, but little or no additional tooth preparation is done. Usually even undermined enamel surfaces are preserved for additional retention and support of the restorative material. In some cases gingivoplasty around the erupting tooth may first be necessary to allow adequate access to and isolation of the defective areas. Even if the restoration requires occasional repair, it still often provides a more satisfactory interim result than the stainless steel crown. Some of the newer restorative materials on the glass ionomer–composite resin continuum may also be useful interim restorations for hypoplastic teeth.

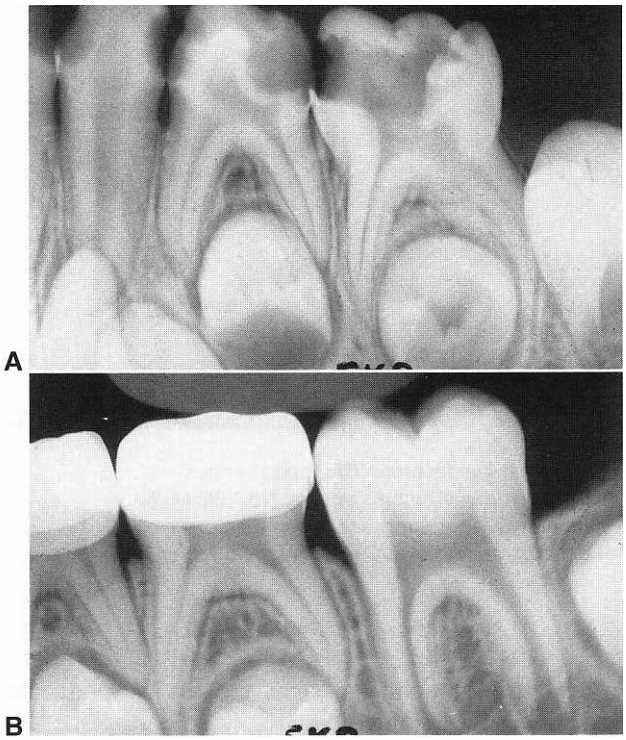
In situations in which a stainless steel crown is required to restore a young permanent molar, Radcliffe and Cullen have noted the importance of conservative tooth preparation to preserve better options for future restoration of the same tooth.' They advocate a preparation similar to that described in the following section.

STAINLESS STEEL CROWNS FOR POSTERIOR TEETH

Chrome steel crowns, as introduced by Humphrey in 1950, have proved to be serviceable restorations for children and adolescents and are now commonly called *stainless steel* crouws.[25] There are a number of indications for the use of stainless steel crowns in pediatric dentistry, including the following:

1. Restorations for primary or young permanent teeth with extensive and/or multiple carious lesions (Fig. 18-19)
2. Restorations for hypoplastic primary or permanent teeth that cannot be adequately restored with bonded restorations
3. Restorations for teeth with hereditary anomalies, such as dentinogenesis imperfecta or amelogenesis imperfecta
4. Restorations for pulpotomized or pulpectomized primary or young permanent teeth when there is increased danger of fracture of the remaining coronal tooth structure
5. Restorations for fractured teeth
6. Restorations for primary teeth to be used as abutments for appliances
7. Attachments for habit-breaking and orthodontic appliances

Randall published an extensive review of the literature that reports on the use of preformed metal crowns for primary and permanent molars.-[s] She found five clinical studies that have compared the performance of crown restorations with that of multisurface amalgam restorations. The five studies included a total of 1210 crowns and 2201 amalgams that were followed from a minimum of 2 years to a maximum of 10 years. The findings in all five studies were in agreement that the crown restorations were superior to the amalgam restorations in the treatment of multisurface cavities in



F **I C 18-19** A, Primary molars with extensive carious lesions. **B**, Adequately contoured steel crowns have maintained function and the relationship of the primary teeth in the arch.

primary molars. Randall's review was followed by a position paper prepared by Seale that included additional scientific evidence favoring the use of stainless steel crown restorations, especially in children at high risk for caries.[27] Seale's published abstract states in part:

The stainless steel crown (SSC) is an extremely durable restoration.... Children with extensive decay, large lesions or multiple surface lesions in primary molars should be treated with stainless steel crowns. Because of the protection from future decay provided by their feature of full coverage and their increased durability and longevity, strong consideration should be given to the use of SSCs in children who require general anesthesia. Finally, a strong argument for the use of the SSC restoration is its cost effectiveness based on its durability and longevity.

PREPARATION OF THE TOOTH

A local anesthetic should be administered and a rubber dam placed as for other restorative procedures. The proximal surfaces are reduced using a No. 69L bur at high speed (Fig. 18-20). Care must be taken not to damage adjacent tooth surfaces during the proximal reductions. A wooden wedge may be placed tightly between the surface being reduced and the adjacent surface to provide a slight separation between the teeth for better access. Near-vertical reductions are made on the proximal surfaces and carried gingivally until the contact with the adjacent tooth is broken and an explorer can be passed freely between the prepared tooth and the adjacent tooth. The gingival margin of the preparation on the proximal surface should be a smooth feathered edge with no ledge or shoulder present. The cusps and the occlusal portion of the tooth may then be reduced with a No. 69L bur revolving at high speed. The general contour of the occlusal surface is followed, and approximately 1 mm of clearance with the opposing teeth is required.

The No. 69L bur at high speed may also be used to remove all sharp line and point angles. It is usually not necessary to reduce the buccal or lingual surfaces; in fact, it is desirable to have an undercut on these surfaces to aid in the retention of the contoured crown. In some cases, however, it may be necessary to reduce the distinct buccal bulge, particularly on the first primary molar.

If any carious dentin remains after these steps in crown preparation are completed, it is excavated next. In the event that a vital pulp exposure is encountered, a pulpotomy procedure is usually carried out.

SELECTION OF CROWN SIZE

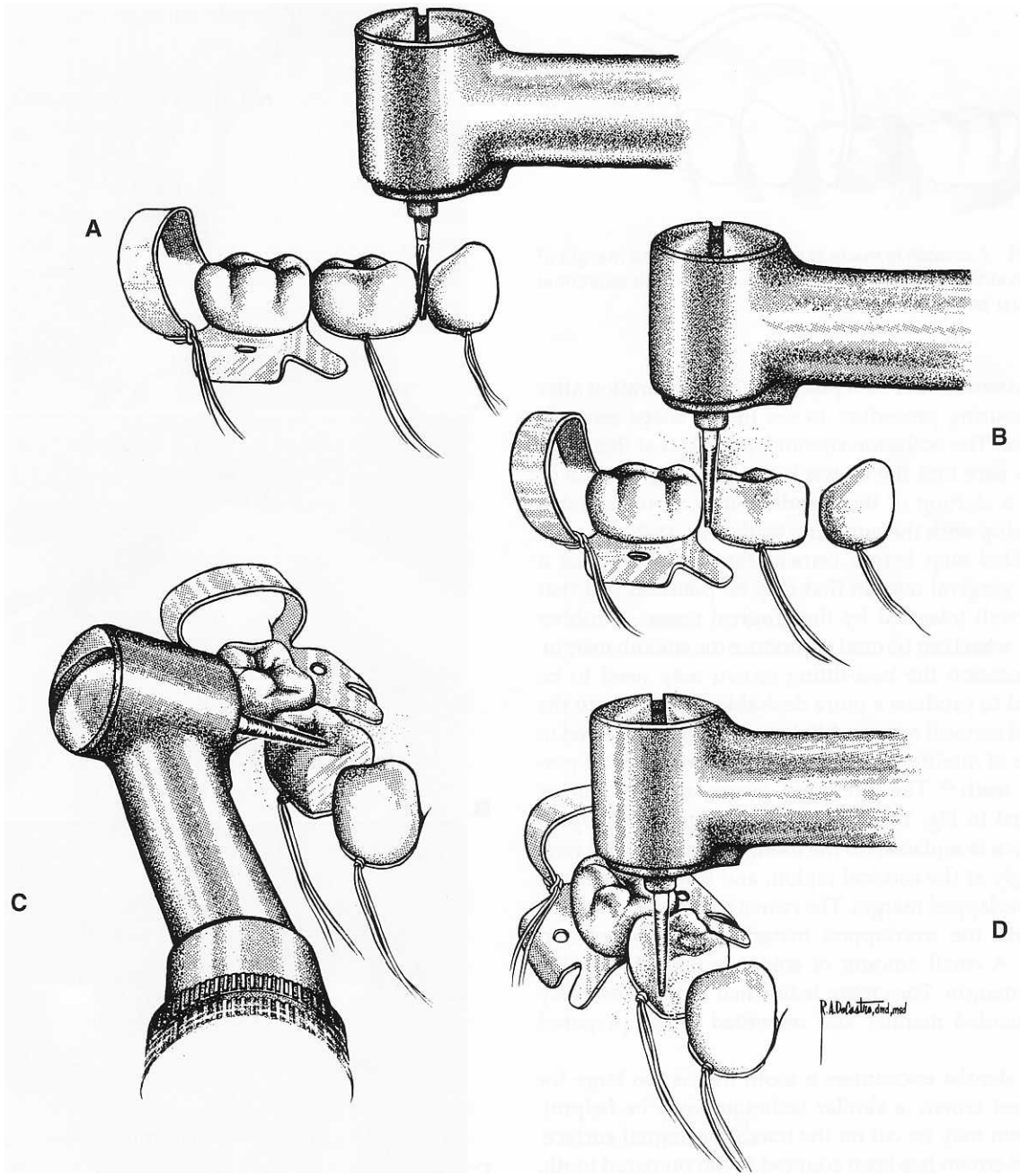
The smallest crown that completely covers the preparation should be chosen. Spedding has advocated adhering to two important principles that will help consistently to produce well-adapted stainless steel crowns."[s] First, the operator must establish the correct occlusogingival crown length; and second, the crown margins should be shaped circumferentially to follow the natural contours of the tooth's marginal gingivae. The crown should be reduced in height, if necessary, until it clears the occlusion and is approximately 0.5 to 1 mm beneath the free margin of the gingival tissue. The patient can force the crown over the preparation by biting an orangewood stick or a tongue depressor. After making a scratch mark on the crown at the level of the free margin of the gingival tissue, the dentist can remove the crown and determine where additional metal must be cut away with a No. IIB curved shears or a rotating stone (Fig. 18-21).

With a curved-beak pliers, the cut edges of the crown are redirected cervically and the crown is replaced on the preparation. The child is again directed to bite on an orangewood stick to forcibly seat the crown so that the gingival margins may be checked for proper extension.

The precontoured and festooned crowns currently available often require very little, if any, modification before cementation.

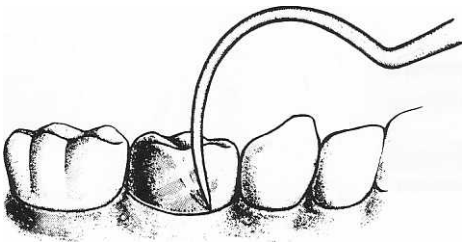
CONTOURING OF THE CROWN (WHEN NECESSARY)

A crown-contouring pliers with a ball-and-socket design is used at the cervical third (if loosely fitting,



l.. **18-20.** Steps in the preparation of a primary molar for a steel crown restoration using a No. 69L bur in the high-speed handpiece. A, Mesial reduction. **B,** Distal reduction. C, Occlusal reduction. D, Rounding of the line angles.

start at the middle third) of the buccal and lingual surfaces to help adapt the margins of the crown to the cervical portion of the tooth. The handles of the pliers are tipped toward the center of the crown, so that the metal is stretched and curled inward as the crown is moved toward the pliers from the opposite side. A curved-beak pliers is used to further improve the con-tour on the buccal and lingual surfaces (Fig. 18-22). The curved-beak pliers may also be used to contour the proximal areas of the crown and develop desirable contact with adjacent teeth. Many clinicians prefer to complete the crown contouring procedures with a crown-crimping pliers (Fig. 18-23). If necessary, solder may be added to the proximal surfaces of the crown to improve the proximal contacts and contour. Trimming and contouring are continued until the crown fits the preparation snugly and extends under the free margin of the gingival tissue.



f **I G 18-21.** A scratch is made at the level of the free margin of the gingival tissue as an aid in determining where additional metal must be removed.

The crown should be replaced on the preparation after the contouring procedure to see that it snaps securely into place. The occlusion should be checked at this stage to make sure that the crown is not opening the bite or causing a shifting of the mandible into an undesirable relationship with the opposing teeth (Fig. 18-24).

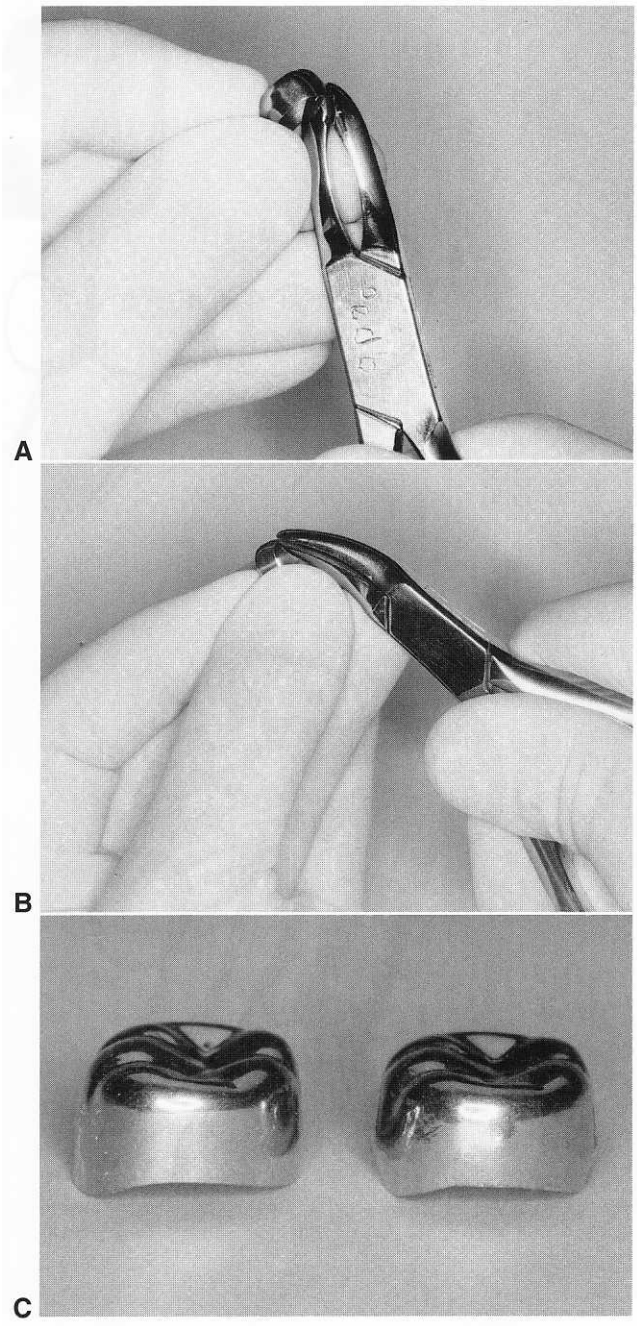
The final step before cementation is to produce a beveled gingival margin that may be polished and that will be well tolerated by the gingival tissue. A rubber abrasive wheel can be used to produce the smooth margin.

On occasion the best-fitting crown may need to be modified to produce a more desirable adaptation to the prepared cervical margin. Mink and Hill have referred to methods of modifying steel crowns for primary and permanent teeth.[-] The oversized crown may be cut as illustrated in Fig. 18-25 and the cut edges overlapped. The crown is replaced on the tooth to ensure that it now fits snugly at the cervical region, and a scratch is made at the overlapped margin. The crown is removed from the tooth and the overlapped material repositioned and welded. A small amount of solder is flowed over the outside margin. The crown is finished in the previously recommended manner and cemented to the prepared tooth.

If the dentist encounters a tooth that is too large for the largest crown, a similar technique may be helpful. The crown may be cut on the buccal or lingual surface. After the crown has been adapted to the prepared tooth, an additional piece of 0.004-inch stainless steel band material may be welded into place. A small amount of solder should be added to the outer surface of the margins. The crown may then be contoured in the usual manner, polished, and cemented into place.

ALTERNATIVE RESTORATIVE TREATMENT

Alternative restorative treatment, or *ART,* has become a popular descriptive term to describe a conservative method of managing both small and large carious lesions in situations in which treating the disease by more traditional restorative procedures is impossible or impractical. This method may prevent pain and



F I 18-22. **A,** A crown-contouring pliers is used to contour the buccal and lingual surfaces of the crown. The crown is held firmly with the pliers, and pressure is exerted with the finger from the opposite side of the crown to bend the surface inward. **B,** The curved-beak pliers is "walked" com- pletely around the cervical margins of the crown to direct all margins inward with smooth, flowing contour. C, The crown on the right was the same size and shape as the crown on the left before it was contoured. This illustrates the effectiveness of the contouring procedures with the pliers as described.

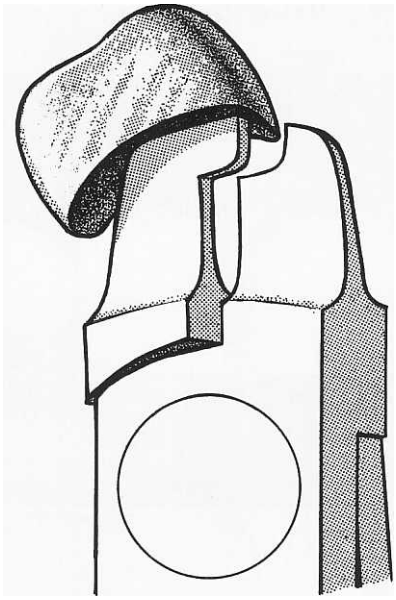


FIG 18-23. A crown-crimping pliers may also be used for crown contouring.

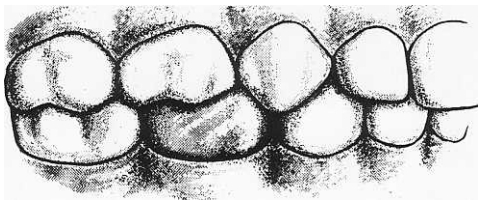


FIG. 18-24. Final adaptation of the crown should result in good occlusion before cementation.

preserve teeth in individuals who do not have access to regular and conventional oral health care. ART may be performed with only hand instruments when no other dental equipment is available, but it may be useful some-times in the conventional dental setting as well. ART does not require the complete excavation of dentinal caries before placement of the restorative material. This is not a totally new concept in dentistry, but it has enjoyed renewed recognition as a viable restorative approach because of the development of the more durable fluoride-releasing glass ionomer and resin-modified glass ionomer restorative materials. The principles validating this technique are discussed further in Chapter 19 (in the section Treatment of the Deep Carious Lesion).

The AAPD adopted a policy on ART in 2001. The discussion and the policy statement as they appear in the AAPD *Reference Manual* (excluding references) are as follows:

Purpose

Removal of dental caries and restoration of teeth can often present unique challenges to the practitioner. Not all

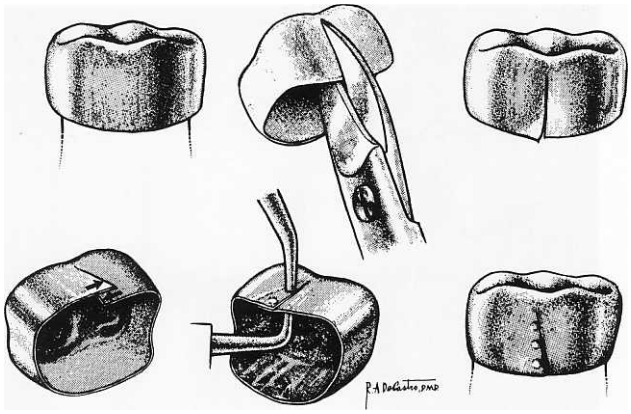


FIG 18-25. Technique for adapting an oversized crown to a prepared tooth.

dental disease can be treated by "traditional" restorative techniques. Young patients, uncooperative patients, patients with special needs and situations where traditional cavity preparation and placement of traditional dental restorations is not possible, may require the use of an alternative restorative treatment.

Background

Alternative restorative treatment (ART), formerly known as atraumatic restorative treatment, is a technique used to restore defective or carious teeth with minimal cavity preparation followed by placement of a fluoride-releasing material such as glass ionomer. This technique is promoted and endorsed by the World Health Organization with the goals of preserving tooth structure, reducing infection and avoiding discomfort. The International Association for Dental Research held a symposium on ART in June 1995 recognizing the technique as a means of restoring and preventing dental caries.

The procedure does not require a traditional dental setting. Preventive measures to control the bacterial infection and the causative agents of the disease should also be utilized for optimal results following treatment.

Policy statement

The American Academy of Pediatric Dentistry recognizes ART as a useful and beneficial technique in the treatment and management of dental caries where traditional cavity prepara tion and placement of traditional dental restorations are not possible.[30]

COSMETIC RESTORATIVE PROCEDURES FOR YOUNG PERMANENT ANTERIOR TEETH

A common problem confronting dentists who treat children is the esthetic management of anterior teeth that are discolored, developmentally undersized or malformed, malposed, or fractured. Dentists recognize that esthetic impairments of the teeth often adversely



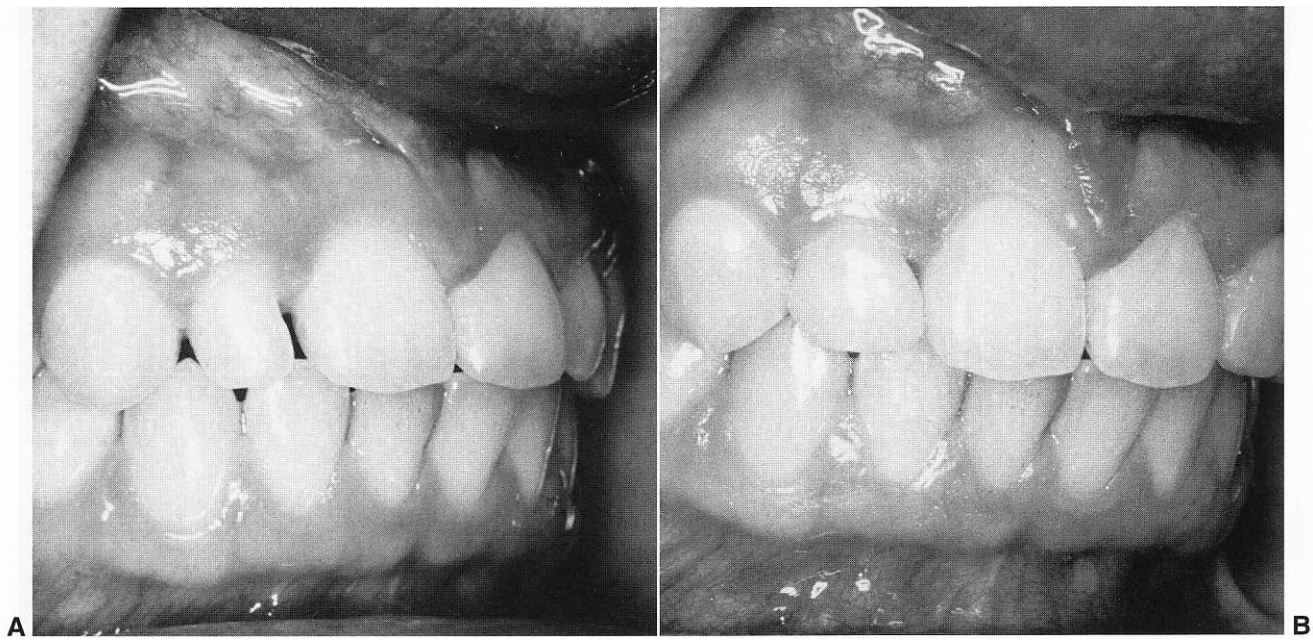
f **I G 18-26.** Composite resin bonding. **A,** Preoperative appearance of a 15-year-old girl who said, "I hate the spaces between my teeth." **B,** Maxillary anterior teeth isolated and etched. C, Composite restorative material bonded to teeth 7, 8, 9, and 10 before finishing. D, Postoperative appearance of finished restorations. *(Courtesy Dr. Wayne A. Moldenhauer.)*

affect the social and psychologic development of the growing child. Esthetic restorative systems and bonding techniques are usually employed when restorations are indicated in these situations. Although bonding procedures are also applicable to primary tooth restorations (as described earlier in this chapter), the following discussion applies primarily to permanent anterior teeth simply because few indications are encountered in the primary dentition. However, Aron has reported the successful use of porcelain laminate veneer restorations on maxillary primary incisors in one unusual case."

The following discussion assumes that one understands dental bonding principles and has a working knowledge of the process. These principles and procedures are similar for sealants, restorative resins, and resin luting agents, and they are presented in Chapter 17. Some tooth preparation confined to enamel (as much as possible) is often indicated, although not always required, before cosmetic bonding procedures are performed.

BONDED COMPOSITE VENEER RESTORATIONS (COMPOSITE RESIN BONDING)

Composite restorative resins (and bonding agents) are frequently applied directly to etched enamel. The restorative resin simply becomes a veneer to improve tooth color or contour. Restorative resin-bonding techniques are particularly useful for restoring anterior crown fractures (see Chapter 21) and for cosmetically increasing the mesial-distal widths of young permanent anterior teeth (Fig. 18-26). Bonded composite veneers are also useful for restoring small hypoplastic or discolored areas on visible tooth surfaces. Many dentists also use this type of restoration to mask intrinsic



f I C 18-27. A, Undersized maxillary right lateral incisor in a young patient. B, Improved appearance of tooth after restoration with a bonded laminate veneer restoration.

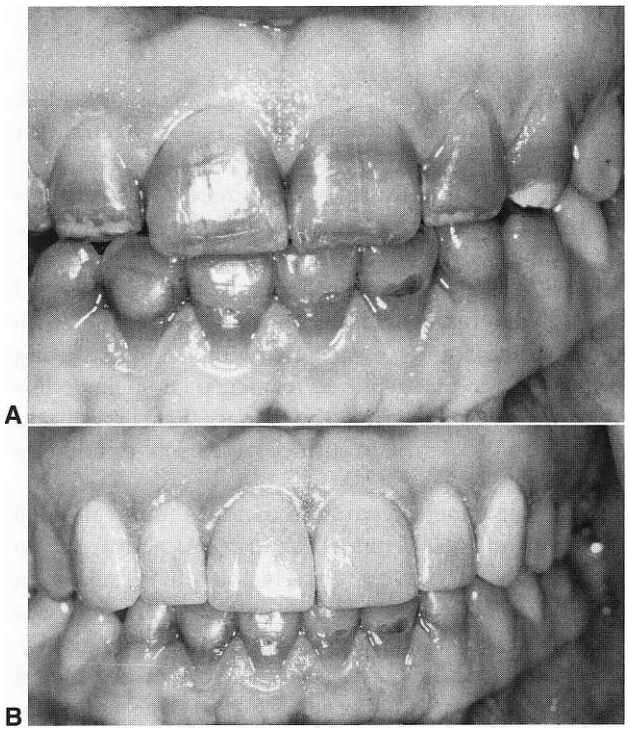
discolorations by veneering the entire labial surfaces of the discolored anterior teeth. This approach may provide satisfactory cosmetic restorations for teeth with mild to moderate discolors tions that will not respond to the bleaching or microabrasion procedures discussed in Chapter 7.

BONDED LAMINATE VENEER RESTORATIONS (DENTAL LAMINATES OR LAMINATE VENEERS)

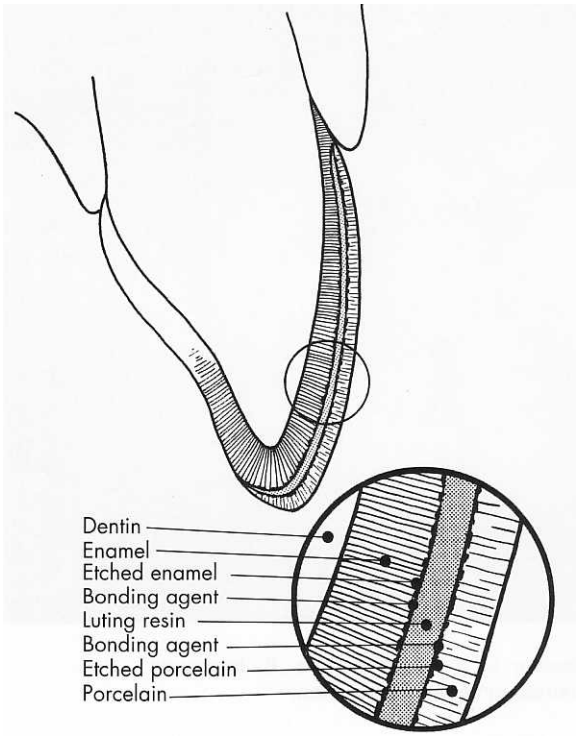
The use of thin, prefitted porcelain facings (laminate veneers) that are bonded to enamel surfaces has become commonplace in cosmetic dentistry. Interest in laminate veneer restorations has grown steadily since their introduction by Faunce and Faunce.'-' Such restorations for maxillary anterior teeth are recognized as conservative, esthetically satisfactory restorations, especially in children and young adults. Laminate veneer restorations have also been used successfully on mandibular anterior teeth.

The laminate veneer technique offers esthetic improvement because the restored teeth simulate the natural hue and appearance of normal, healthy tooth structure. When properly finished, the laminate restorations are well tolerated by the gingival tissues even though their contour may be slightly excessive. Immaculate oral hygiene is essential, but experience has shown that the maintenance of gingival health around the restorations is certainly possible in cooperative patients (Fig. 18-27).

The luting materials are tooth-colored resin systems designed for use in bonding techniques. If the teeth being treated are severely discolored, tinting or opaquing agents also may be required (Fig. 18-28).



f I C 18-28. A, Severe intrinsic tooth discoloration in a teenager. B, Porcelain-bonded laminate veneer restorations have been placed onto the maxillary anterior teeth for a more natural appearance. The laminates were intentionally lengthened incisally to provide more coverage of the discolored lower incisors and to improve the patient's smile line.



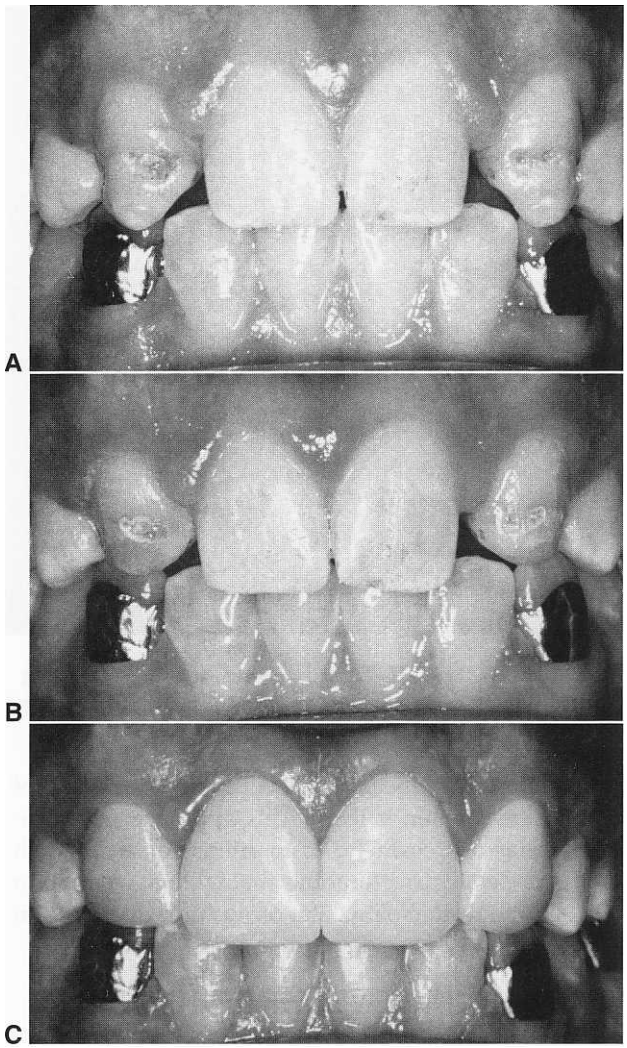
f **I G . 18-29.** Cross-sectional sketch of the intraenamel preparation and the precision-fitted porcelain laminate veneer that had been fabricated in the laboratory to restore the natural tooth contours when bonded in place.

The laminate veneer procedure is not complicated, but it requires meticulous attention to detail for success.

The bonding procedure for a laminate veneer restoration requires proper preparation of the inside laminate surface and proper etching of the outer enamel surface. The inside of the porcelain laminate surface is etched with a hydrofluoric acid etchant and then coated with silane, which results in a bond with the resin luting agent similar to that achieved on etched enamel but also enhanced chemically by the silane. Excellent bond strengths to the porcelain surface have been reported by Lee et al."

The intraenamel preparation includes removal of 0.5 to 1.0 mm of facial enamel, tapering to about 0.25 to 0.5 mm at the cervical margin. This margin is finished in a well-defined chamfer level with the crest of the gingival margin or not more than 0.5 mm subgingivally. The incisal margin may end just short of the incisal edge, or it may include the entire incisal edge ending on the lingual surface (Fig. 18-29). It is better not to place incisal margins where direct incising forces occur. Bonded porcelain techniques have significant value in cosmetic dental procedures (Fig. 18-30).

It is beyond the scope of this chapter to discuss in detail the many varieties of materials and techniques



F **I** G **18-30. A,** Anterior teeth of a teenaged patient immediately after the removal of bonded orthodontic appliances. Maxillary canines have been orthodontically positioned forward because of the congenital absence of the lateral incisors. Notice the interdental spaces distal to the central incisors and also the enlarged gingival tissues secondary (at least in part) to many months of orthodontic appliance therapy. The hypoplastic defects are obvious on the canines, and the central incisors are mildly affected as well. **B,** Photograph of same patient after completion of the intraenamel preparation of the canines. The central incisors received only minimal preparation so that the final restorations would provide slightly more support to the upper lip. No anesthesia was required during the tooth preparations, and no temporary restorations were necessary during the time the porcelain laminates were being fabricated in the laboratory. C, Bonded porcelain laminate restorations were placed several days later. The restored canines now resemble lateral incisors, and the interdental spaces are closed. Although the gingival tissues are somewhat irritated because of manipulation during the bonding procedures, some spontaneous reduction of the gingival enlargements are noticed now that better brushing and flossing have been instituted.

available for dental cosmetic procedures. Additional useful information has been published by Nixon for those interested.[34]

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CHAPTER

19

Treatment of Deep Caries, Vital Pulp Exposure, and Pulpless Teeth

RALPH E. McDONALD
DAVID R. AVERY
JEFFREY A. DEAN

انتشارات
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CHAPTER OUTLINE

DIAGNOSTIC AIDS IN THE SELECTION OF TEETH FOR VITAL PULP THERAPY
- History of Pain
- Clinical Signs and Symptoms
- Radiographic Interpretation
- Pulp Testing
- Physical Condition of the Patient

EVALUATION OF TREATMENT PROGNOSIS BEFORE PULP THERAPY

TREATMENT OF THE DEEP CARIOUS LESION
- Indirect Pulp Treatment (Gross Caries Removal or Indirect Pulp Therapy)

VITAL PULP EXPOSURE
- Size of the Exposure and Pulpal Hemorrhage
- Dental Hemogram

VITAL PULP THERAPY TECHNIQUES
- Direct Pulp Capping
- Pulpotomy
  - *Pulpotomy technique for permanent teeth*
  - *Pulpotomy technique for primary teeth*
- Partial Pulpectomy

NONVITAL PULP THERAPY TECHNIQUE
- Complete Pulpectomy

SUMMARY OF PULP THERAPY

RESTORATION OF THE PULPALLY INVOLVED TOOTH

REACTION OF THE PULP TO VARIOUS CAPPING MATERIALS
- Zinc Oxide-Eugenol
- Calcium Hydroxide
- Preparations Containing Formalin
- Glutaraldehyde
- Ferric Sulfate
- Other Experimental Capping Materials (Mineral Trioxide Aggregate, Bone Morphogenetic Protein, and Others)
- Other Experimental Capping Methods

FAILURES AFTER VITAL PULP THERAPY

Internal Resorption
- Alveolar Abscess

EARLY EXFOLIATION OR OVERRETENTION OF PRIMARY TEETH WITH PULP TREATMENTS

The treatment of the dental pulp exposed by the caries process, by accident during cavity preparation, or even as a result of injury and fracture of the tooth has long presented a challenge in treatment. As early as 1756, Pfaff reported placing a small piece of gold over a vital exposure in an attempt to promote healing.

Although it has been established that the pulp is capable of healing, there is still much to learn regarding the control of infection and inflammation in the vital pulp. Current methods of diagnosing the extent of pulpal injury are inadequate. More effective methods of pulp therapy are still needed, and more research is necessary.

DIAGNOSTIC AIDS IN THE SELECTION OF TEETH FOR VITAL PULP THERAPY

HISTORY OF PAIN

The history of either presence or absence of pain may not be as reliable in the differential diagnosis of the condition of the exposed primary pulp as it is in permanent teeth. Degeneration of primary pulp even to the point of abscess formation without the child's recalling pain or discomfort is not uncommon. Nevertheless, the history of a toothache should be the first consideration in the selection of teeth for vital pulp therapy. A toothache coincident with or immediately after a meal may not indicate extensive pulpal inflammation. The pain may be caused by an accumulation of food within a carious lesion, by pressure, or by a chemical irritation to vital pulp protected by only a thin layer of intact dentin.

A severe toothache at night usually signals extensive degeneration of the pulp and calls for more than a conservative type of pulp therapy. A spontaneous toothache of more than momentary duration occurring at any time usually means that pulpal disease has progressed too far for treatment with even a pulpotomy.

CLINICAL SIGNS AND SYMPTOMS

A gingival abscess or a draining fistula associated with a tooth with a deep carious lesion is an obvious clinical sign of an irreversibly diseased pulp. Such infections can be resolved only by successful endodontic therapy or extraction of the tooth.

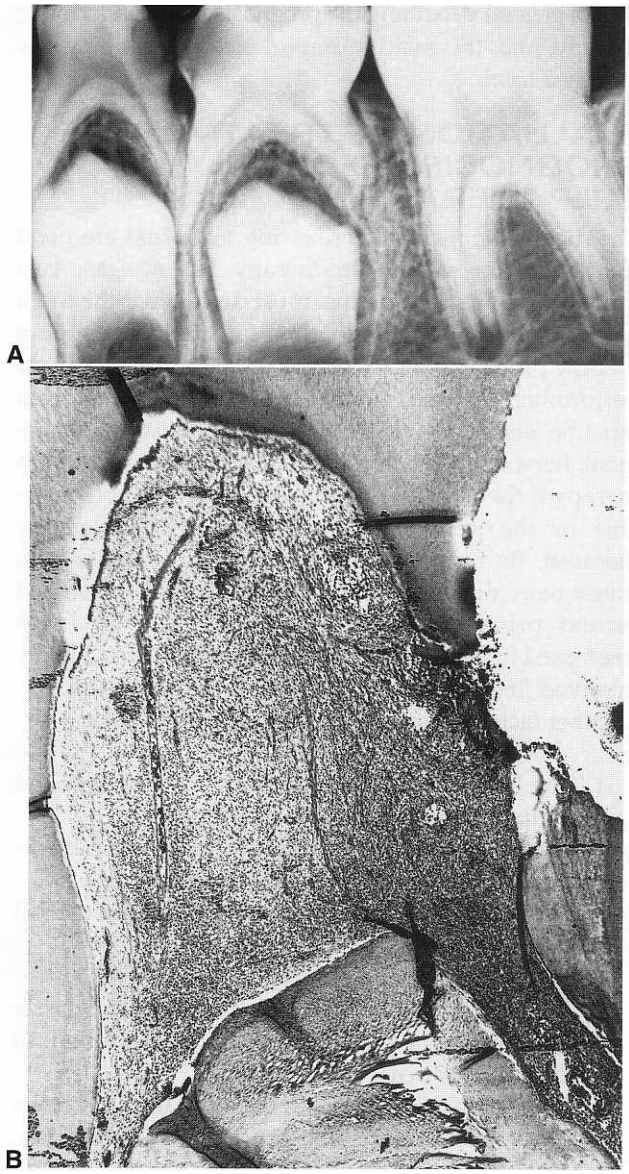
Abnormal tooth mobility is another clinical sign that may indicate a severely diseased pulp. When such a tooth is evaluated for mobility, the manipulation may elicit localized pain in the area, but this is not always the case. If pain is absent or minimal during manipulation of the diseased mobile tooth, the pulp is probably in a more advanced and chronic degenerative condition. Pathologic mobility must be distinguished from normal mobility in primary teeth near exfoliation.

Sensitivity to percussion or pressure is a clinical symptom suggestive of at least some degree of pulpal disease, but the degenerative stage of the pulp is probably of the acute inflammatory type. Tooth mobility or sensitivity to percussion or pressure may be a clinical signal of other dental problems as well, such as a high restoration or advanced periodontal disease. However, when this clinical information is identified in a child and is associated with a tooth having a deep carious lesion, the problem is most likely to be caused by pulpal disease and possibly by inflammatory involvement of the periodontal ligament.

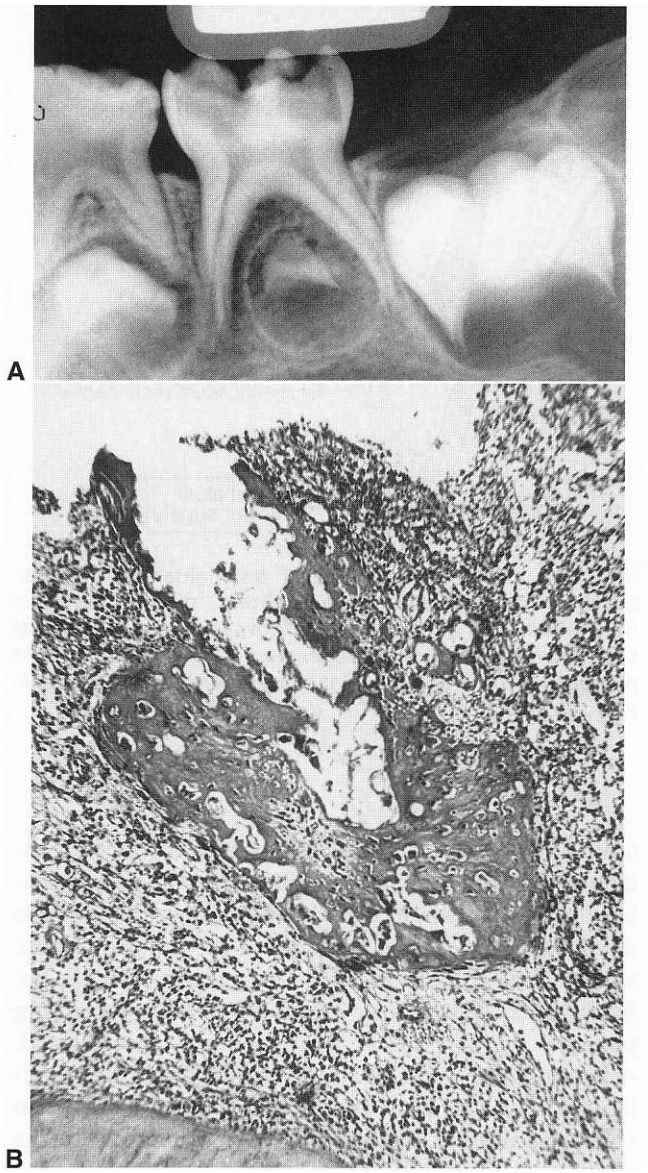
RADIOGRAPHIC INTERPRETATION

A recent x-ray film must be available to examine for evidence of periradicular or periapical changes, such as thickening of the periodontal ligament or rarefaction of the supporting bone. These conditions almost always rule out treatment other than an endodontic procedure or extraction of the tooth. Radiographic interpretation is more difficult in children than in adults. The permanent teeth may have incompletely formed root ends, giving an impression of periapical radiolucency, and the roots of the primary teeth undergoing even normal physio-logic resorption often present a misleading picture or one suggestive of pathologic change.

The proximity of carious lesions to the pulp cannot always be determined accurately in the x-ray film. What often appears to be an intact barrier of secondary dentin protecting the pulp may actually be a perforated mass of irregularly calcified and carious material. The pulp beneath this material may have extensive inflammation (Fig. 19-1). Radiographic evidence of calcified masses within the pulp chamber is diagnostically important. If the irritation to the pulp is relatively mild and chronic, the pulp will respond with inflammation and will attempt to eliminate the irritation by blocking with irregular dentin the tubules through which the irritating factors are transmitted. If the irritation is intense and acute and if the carious lesion is developing rapidly, the defense mechanism may not have a chance to lay down the reparative dentin barrier, and the disease process may reach the pulp. In this instance the pulp may attempt to form a barrier at some distance from the exposure site. These calcified masses are sometimes evident in the pulp horn or even in the region of the pulp canal entrance. A histologic examination of these teeth shows irregular, amorphous masses of calcified material that are not like pulp stones (Fig. 19-2). The masses bear no resemblance to dentin or to a dentinal barrier. In every instance they are associated with advanced degenerative changes of the coronal pulp and inflammation of the tissue in the canal.



f **G 19-1. A,** First primary molar appears to have an intact dentinal barrier beneath the carious lesion. **B,** Histologic section shows a perforation of the barrier with necrotic material at the exposure site. There is advanced inflammation of the pulp tissue, which is likely to evoke a spontaneous pain response.

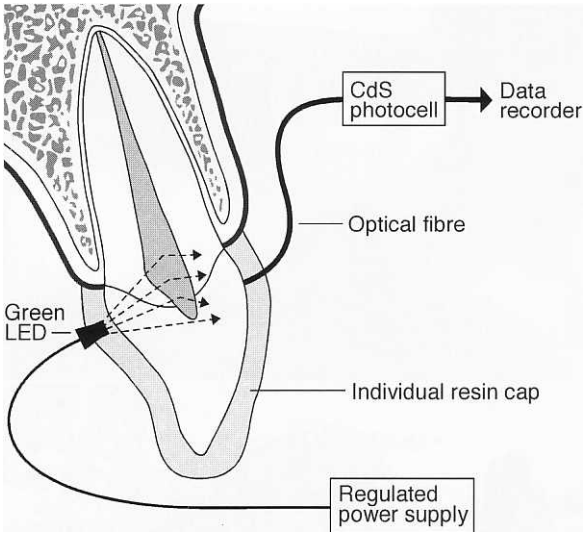


H G. **19-2. A,** Calcified mass in the pulp chamber beneath the exposure site is associated with extensive inflammation of the pulp in the coronal area and in the pulp canals. **B,** The amorphous mass is surrounded by pulp tissue with advanced inflammation.

PULP TESTING

The value of the electric pulp test in determining the condition of the pulp of primary teeth is questionable, although it will give an indication of whether the pulp is vital. The test does not provide reliable evidence of the degree of inflammation of the pulp. A complicating factor is the occasional positive response to the test in a tooth with a necrotic pulp if the content of the canals is liquid. The reliability of the pulp test for the young child can also be questioned sometimes because of the child's apprehension associated with the test itself. Thermal tests have reliability problems in the primary dentition, too. The lack of reliability is possibly related to the young child's inability to understand the tests.

Several methods have been developed and advocated as noninvasive techniques for recording the blood flow in human dental pulp. Two of these methods include the use of a laser Doppler flowmeter and transmitted-light photoplethysmography. As shown in the schematic in Fig. 19 these methods essentially work by transmitting a laser or light beam through the crown of the tooth; the signal is picked up on the other side of the



f **I C 19-3.** Schematic drawing of transmitted-light photoplethysmography. *LED,* light-emitting diode. *(Adapted from Miwa Z et al: Pulpal blood flow in vital and nonvital young permanent teeth measured by transmitted-light photoplethysmography: a pilot study,* Pediatr Dent *24(6):594-598, 2002.)*

tooth by an optical fiber and photocell. A distinct advantage of this technique is its noninvasive nature, particularly in comparison to electric pulp testing. Not only is there inaccuracy in the response of the pulp to electric stimuli, but the electric pulp tester may elicit pain. Because the testing may be uncomfortable for young patients, further dental treatment may be affected. A study by Miwa et al suggests that the transmitted-light technique can detect pulpal blood flow in young permanent teeth and is thus applicable to the assessment of pulp vitality.'

PHYSICAL CONDITION OF THE PATIENT

Although the local observations are of extreme importance in the selection of cases for vital pulp therapy, the dentist must also consider the physical condition of the patient. In seriously ill children, extraction of the involved tooth after proper premedication with antibiotics, rather than pulp therapy, should be the treatment of choice. Children with conditions that render them susceptible to subacute bacterial endocarditis or those with nephritis, leukemia, solid tumors, idiopathic cyclic neutropenia, or any condition that causes cyclic or chronic depression of granulocyte and polymorphonuclear leukocyte counts should not be subjected to the possibility of an acute infection resulting from failed pulp therapy. Occasionally, pulp therapy for a tooth of a chronically ill child may be justified, but only after careful consideration is given to the prognosis of the child's general condition, the prognosis of the endodontic therapy, and the relative importance of retaining the involved tooth.

EVALUATION OF TREATMENT PROGNOSIS BEFORE PULP THERAPY

The diagnostic process of selecting teeth that are good candidates for vital pulp therapy has at least two dimensions. First, the dentist must decide that the tooth has a good chance of responding favorably to the pulp therapy procedure indicated. Second, the advisability of performing the pulp therapy and restoring the tooth must be weighed against extraction and space management. For example, nothing is gained by successful pulp therapy if the crown of the involved tooth is nonrestorable or the periodontal structures are irreversibly diseased. By the same rationale, a dentist is likely to invest more time and effort to save a pulpally involved second primary molar in a 4-year-old child with unerupted first permanent molars than to save a pulpally involved first primary molar in an 8-year-old child.

Other factors to consider include the following:

1. The level of patient and parent cooperation and motivation in receiving the treatment
2. The level of patient and parent desire and motivation in maintaining oral health and hygiene
3. The caries activity of the patient and the overall prognosis of oral rehabilitation
4. The stage of dental development of the patient
5. The degree of difficulty anticipated in adequately performing the pulp therapy (instrumentation) in the particular case
6. Space management issues resulting from previous extractions, preexisting malocclusion, ankylosis, congenitally missing teeth, and space loss caused by the extensive carious destruction of teeth and subsequent drifting
7. Excessive extrusion of the pulpally involved tooth resulting from the absence of opposing teeth

These examples, in any combination, illustrate the almost infinite number of treatment considerations that could be important in an individual patient with pulpal pathosis.

TREATMENT OF THE DEEP CARIOUS LESION

Children and young adults who have not received early and adequate dental care and optimal systemic fluoride and do not have adequate oral hygiene often develop deep carious lesions in the primary and permanent teeth. Many of the lesions appear radiographically to be dangerously close to the pulp or to actually involve the

dental pulp. Approximately 75% of the teeth with deep caries have been found from clinical observations to have pulpal exposures. Work by Dimaggio and Hawes supports this observation.[2,3] They also showed that well over 90% of the asymptomatic teeth with deep carious lesions could be successfully treated without pulp exposure using indirect pulp therapy techniques. This procedure is described in the following pages.

If a carious exposure discovered at the time of the initial caries excavation could be routinely treated with consistently good results, a major problem in dentistry would be solved. Unfortunately, the treatment of vital exposures, especially in primary teeth, has not been entirely successful. For this reason, clinicians prefer to avoid pulp exposure during the removal of deep caries whenever possible.

INDIRECT PULP TREATMENT (GROSS CARIES REMOVAL OR INDIRECT PULP THERAPY)

The procedure in which only the gross caries is removed from the lesion and the cavity is sealed for a time with a biocompatible material is referred to as *indirect pulp treatment* (Fig. 19-4). Indirect pulp treatment is not a new procedure but has attracted renewed interest. Laboratory studies and favorable clinical evidence justify its routine use. Teeth with deep caries that are free of symptoms of painful pulpitis are candidates for this procedure.

The clinical procedure involves removing the gross caries but allowing sufficient caries to remain over the pulp horn to avoid exposure of the pulp. The walls of the cavity are extended to sound tooth structure because the presence of carious enamel and dentin at the margins of the cavity will prevent the establishment of an adequate seal (extremely important) during the period of repair. The remaining thin layer of caries in the base of the cavity is covered with a radiopaque biocompatible base material and sealed with a durable interim restoration (Fig. 19-5). Some interim restorative materials may also serve as the base material. It is often helpful to adapt and cement a preformed stainless steel band to the tooth to support the interim restoration during the observation period (Fig. 19-6).

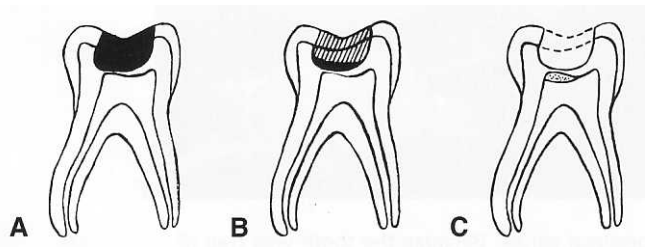


FIG. 19-4. Indirect pulp therapy. **A,** A primary or permanent tooth with deep caries. **B,** The gross caries has been removed and the cavity sealed with durable biocompatible cement or restorative material. C, Six to 8 weeks later the cavity is reopened and the remaining caries excavated. A sound dentin barrier protects the pulp, and the tooth is ready for final restoration. *(Courtesy Dr. Paul E. Starkey.)*

Other operative procedures can be performed at subsequent visits. However, the treated teeth should not be reentered to complete the removal of caries for at least 6 to 8 weeks. During this time the caries process in the deeper layer is arrested.

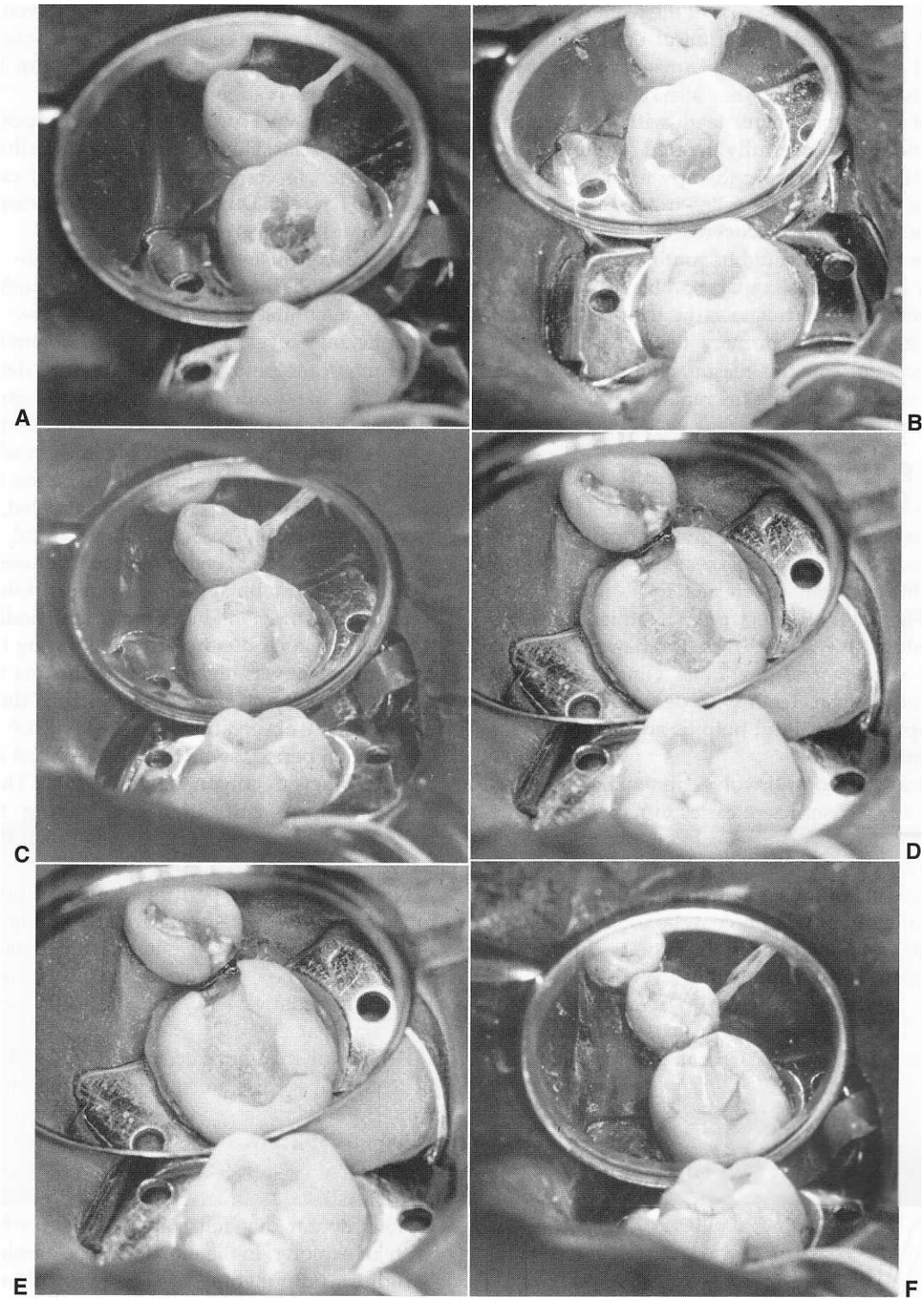
At the conclusion of the minimum 6- to 8-week waiting period, the tooth is reentered. Careful removal of the remaining carious material, now somewhat sclerotic, may reveal a sound base of dentin without an exposure of the pulp. If a sound layer of dentin covers the pulp, the tooth is restored in the conventional manner (Fig. 19-7). Al-Zayer et al report that the use of a base over the calcium hydroxide liner, in addition to a stainless steel crown, dramatically increases the success rate.` If a small pulp exposure is encountered, a different type of treatment, based on the clinical signs and symptoms and local conditions, must be used.

Studies by Traubman, who used television linear and density measurement instrumentation, indicated that the rate of regular dentin formation during the indirect pulp treatment was highest during the first month, but dentin formation continued during the year of experimental observation.' At the end of the 1-year observation period, some teeth had formed as much as 390 μm of new dentin on the pulpal floor. This observation provides justification for leaving the sealed interim restoration in place for longer than the minimal 6 weeks.

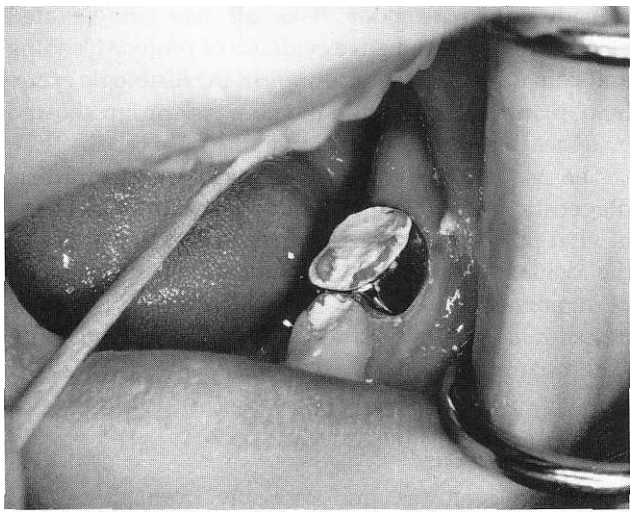
Nirschl and Avery performed indirect pulp therapy on 38 carefully selected primary and young permanent teeth.° Gross caries removal under rubber dam isolation was accomplished, calcium hydroxide was used in each tooth as a sedative base, and the teeth were restored with amalgam.

Successful treatment occurred in 32 (94.1%) of the 34 teeth that were available for the 6-month evaluation procedure. In all cases of successful treatment the base material and the residual carious dentin were observed to be dry on reentry and clinical examination. Of the successfully treated teeth, only four had residual carious dentin that felt somewhat soft when probed with an explorer; in the remainder the dentin felt hard.

Indirect pulp therapy has been proved to be a valuable therapeutic procedure in treating asymptomatic teeth with deep carious lesions. The procedure reduces the risk of direct pulp exposure and preserves pulp vitality. One may question the need to reenter the tooth if it has been properly selected and monitored, if a durable restoration is placed initially, and if no adverse signs or symptoms develop. Most clinicians are successfully practicing indirect pulp treatment without reentry



HI; G **19-5. A,** Second primary molar with deep occlusal caries. Because the tooth was free of symptoms of painful pulpitis, indirect pulp therapy was completed. **B,** The gross caries has been removed. A small amount of soft carious dentin remains at the base of the cavity. C, Calcium hydroxide has been placed over the remaining caries. The cavity may be sealed with a durable intermediate restorative material. **D,** After 6 to 8 weeks, the intermediate restorative material is removed. The caries in the base of the cavity appears arrested and dry. **E,** The remaining caries has been removed. F, After placement of a biocompatible base, the primary second molar has been restored with amalgam.



f If] **19-6.** Preformed steel band has been cemented to the tooth to support the indirect pulp treatment material.

after the initial caries excavation. The inexperienced dentist, however, should perform the treatment in two appointments until confidence in proper case selection has been achieved.

VITAL PULP EXPOSURE

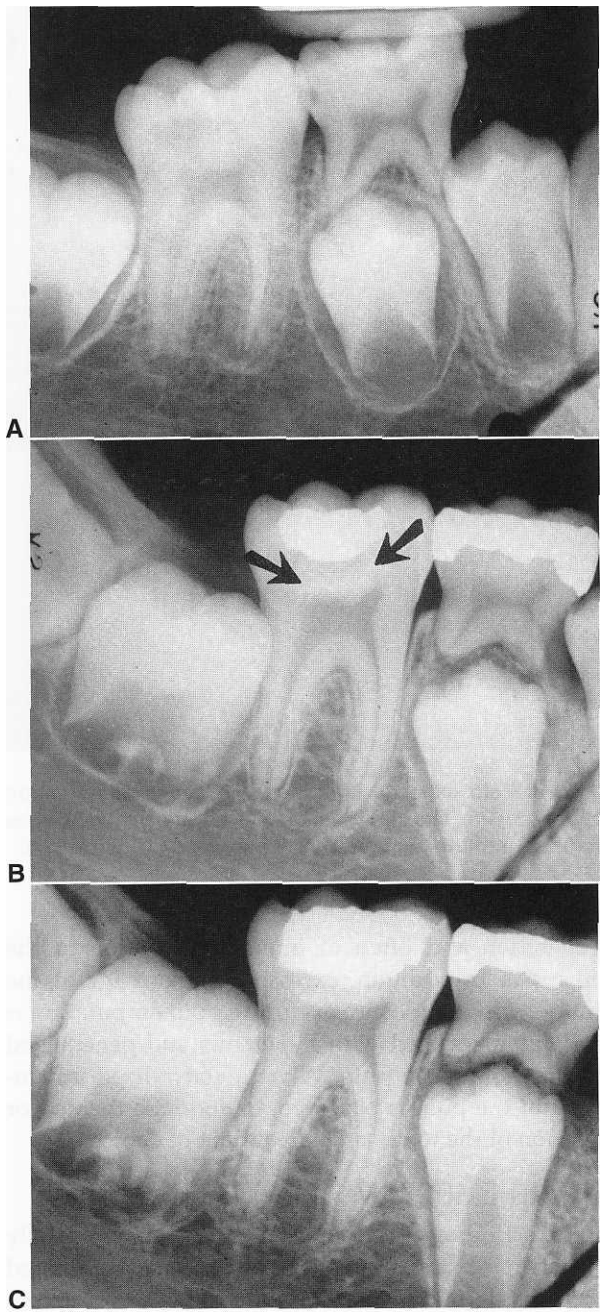
Although the routine practice of indirect pulp therapy in properly selected teeth will significantly reduce the number of direct pulp exposures encountered, all dentists who treat severe caries in children will be faced with treatment decisions related to the management of vital pulp exposures.

The appropriate procedure should be selected only after a careful evaluation of the patient's symptoms, results of diagnostic tests, and conditions at the exposure site. The health of the exposed dental pulp is sometimes difficult to determine, especially in children, and there is often lack of conformity between clinical symptoms and histopathologic condition.

SIZE OF THE EXPOSURE AND PULPAL HEMORRHAGE

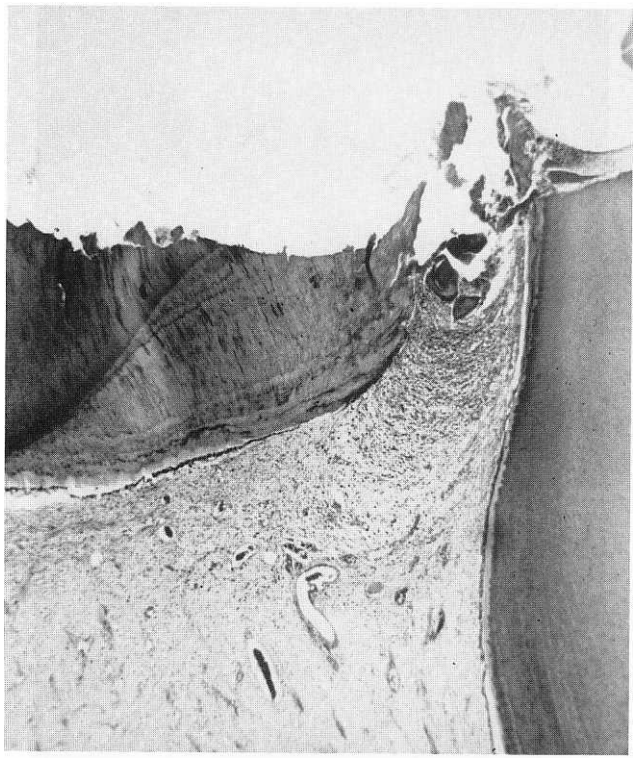
The size of the exposure, the appearance of the pulp, and the amount of bleeding are valuable observations in diagnosing the condition of the primary pulp. For this reason the use of a rubber dam to isolate the tooth is extremely important; in addition, with the rubber dam the area can be kept clean and the work can be done more efficiently.

The most favorable condition for vital pulp therapy is the small pinpoint exposure surrounded by sound dentin. However, a true carious exposure, even of pinpoint size, will be accompanied by inflammation of the pulp, the degree of which is usually directly related to the size of the exposure (Fig. 19-8).



II **19-7. A,** Radiograph of the first permanent molar revealed a deep carious lesion. Gross caries was removed, and calcium hydroxide was placed over the remaining caries. The tooth was restored with amalgam and was not reentered for complete caries removal for 3 months. **B,** Sclerotic dentin can be seen beneath the remaining caries and the covering of calcium hydroxide *(arrows).* C, The tooth was reentered, and the remaining caries was removed. A sound dentin barrier was observed at the base of the cavity. A new amalgam restoration was placed after complete caries removal.

A large exposure—the type that is encountered when a mass of leathery dentin is removed—is often associated with a watery exudate or pus at the exposure site. These conditions are indicative of advanced pulp



GIG G. **19-8.** Pulp exposed by caries will show inflammation at the exposure site. Fragments of necrotic dentin will be introduced into the pulp during the excavation of the caries.

degeneration and often of internal resorption in the pulp canal. In addition, excessive hemorrhage at the point of carious exposure or during pulp amputation is invariably associated with hyperemia and generalized inflammation of the pulp. When a generalized inflammation of the pulp is observed, endodontic therapy or extraction of the tooth is the treatment of choice.

DENTAL HEMOGRAM

Guthrie's findings have substantiated the previously mentioned observations.[7] His study was designed to investigate the value of a white blood cell differential count (hemogram) of the dental pulp as a diagnostic aid in determining pathologic or degenerative changes in the pulp. The first drop of blood from an exposed pulp was used for making the hemogram. The teeth were subsequently extracted. Based on a histologic examination it was decided whether they would have been good candidates for a pulpotomy procedure. Those teeth in which the inflammatory process was localized to the coronal pulp area were classified as good candidates for a pulpotomy. If the inflammation extended into the pulp canal beyond the area of convenient amputation, the tooth was considered a poor candidate. Although there was no consistent blood picture throughout the group, the teeth considered to be poor risks all had an elevated neutrophil count and gave evidence of profuse bleeding and pain other than at mealtime. In the histologic examination, numerous teeth in the poor risk group showed evidence of internal resorption in the pulp canal.

The use of the dental hemogram is not a practical diagnostic method in the routine clinical management of vital pulp exposures. However, experimental use of the dental hemogram has confirmed that a history of spontaneous pain and clinical evidence of profuse pulpal hemorrhage tend to correlate well with significant inflammation of pulpal tissue.

VITAL PULP THERAPY TECHNIQUES

For many centuries, and probably from almost the beginning of time for human beings, there has been a search for the best (safe and effective) method(s) of managing pulpal disease and traumatic pulpal exposure. During the twentieth century a significant share of the total dental research effort was devoted to finding better treatments and prevention methods for pulpal problems. These efforts have generated considerable controversy and debate as proponents of specific materials and methods attempt to justify their chosen technique(s). These controversies are unsettled even now in the twenty-first century, despite many impressive scientific advancements. Identifying the best formulation of ingredients and techniques to predictably produce pulpal healing remains elusive. To further complicate this issue, the predominant belief is that pulp therapies appropriate for permanent teeth may not always be equally effective in treating similar pulpal conditions in primary teeth.

It is generally agreed that the prognosis after any type of pulp therapy improves in the absence of contamination by pathogenic microorganisms. Thus biocompatible neutralization of any existing pulpal contamination and prevention of future contamination (e.g., microleakage) are worthy goals in vital pulp therapy. If the treatment material in direct contact with the pulp also has some inherent quality that promotes, stimulates, or accelerates a true tissue-healing response, so much the better; however, it is recognized that vital pulp tissue can recover from a variety of insults spontaneously in a favorable environment.

The techniques and procedures discussed in the following pages represent the standards as we perceive them at this writing. Some go back to the time when treatment decisions were made empirically. Their effectiveness has been proved over time, if not by science, and they represent the benchmarks with which newer techniques are compared. We look forward to having more effective, biologically compatible, and scientifically sound methods in the future.

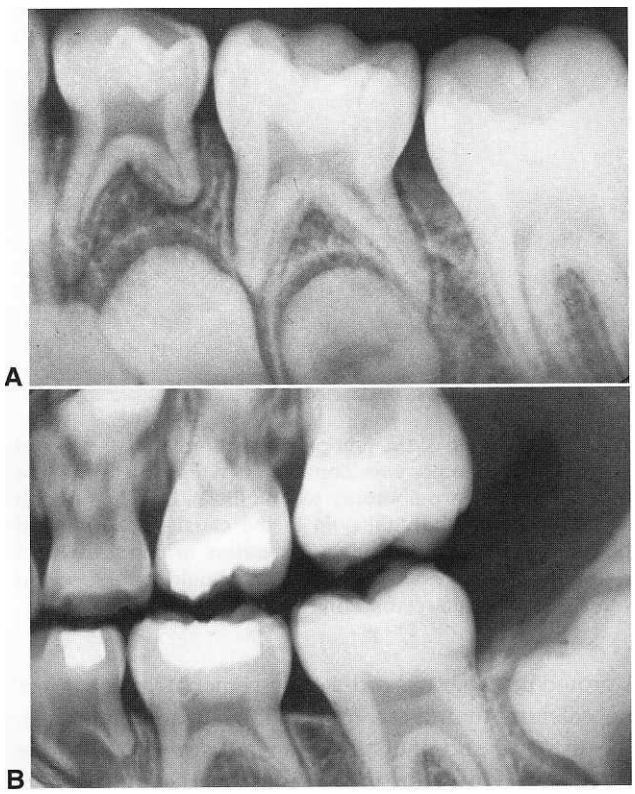
DIRECT PULP CAPPING

The pulp-capping procedure has been widely practiced for years and is still the favorite method of many dentists for treating vital pulp exposures. Although pulp capping has been condemned by some, others report that, if the teeth are carefully selected, excellent results are obtained.

It is generally agreed that pulp-capping procedures should be limited to small exposures that have been produced accidentally by trauma or during cavity preparation or to true pinpoint carious exposures that are surrounded by sound dentin (Fig. 19-9). Pulp capping should be considered only for teeth in which there is an absence of pain, with the possible exception of discomfort caused by the intake of food. In addition, there should be either no bleeding at the exposure site, as is often the case in a mechanical exposure, or bleeding in an amount that would be considered normal in the absence of a hyperemic or inflamed pulp.

All pulp treatment procedures should be carried out under clean conditions using sterile instruments. Use of the rubber dam will help keep the pulp free of external contamination. All peripheral carious tissue should be excavated before excavation is begun on the portion of the carious dentin most likely to result in pulp exposure. Thus most of the bacterially infected tissue will have been removed before actual pulp exposure occurs. The work of Kakehashi, Stanley, and Fitzgerald[11] and of Walshe,[9] which is described later in this chapter, supports the desirability of using a surgically clean technique to minimize bacterial contamination of the pulpal tissue.

Calcium hydroxide remains the standard material for pulp capping normal vital pulp tissue. The possibility of its stimulating the repair reaction is good. A hard-setting calcium hydroxide capping material should be used. If the tooth is small (such as a first primary molar), the hard-setting calcium hydroxide may also be used as the base for the restoration. According to Fuks, some recent studies have shown successful results with direct capping of exposed pulps with adhesive bonding agents, whereas others have reported pulp inflammation and unacceptable results using this technique.[10] She therefore suggests that the traditional practice of using calcium hydroxide be maintained.



G **19-9.** A, Mesial pulp horn of the mandibular second primary molar accidentally exposed during cavity preparation was covered with calcium hydroxide. **B,** Dentinal bridge across the mesial pulp horn is evidence of pulp healing.

PULPOTOMY

The removal of the coronal portion of the pulp is an accepted procedure for treating both primary and permanent teeth with carious pulp exposures. The justification for this procedure is that the coronal pulp tissue, which is adjacent to the carious exposure, usually contains microorganisms and shows evidence of inflammation and degenerative change. The abnormal tissue can be removed, and the healing can be allowed to take place at the entrance of the pulp canal in an area of essentially normal pulp. Even the pulpotomy procedure, however, is likely to result in a high percentage of failures unless the teeth are carefully selected.

In the pulpotomy procedure the tooth should first be anesthetized and isolated with the rubber dam. A surgically clean technique should be used throughout the procedure. All remaining dental caries should be removed, as well as the overhanging enamel, to provide good access to coronal pulp. Pain during caries removal and instrumentation may be an indication of faulty anesthetic technique. More often, however, it indicates pulpal hyperemia and inflammation, which makes the tooth a poor risk for vital pulpotomy. If the pulp at the exposure site bleeds excessively after complete removal of caries, the tooth is also a poor risk for vital pulpotomy.

The entire roof of the pulp chamber should be removed. No overhanging dentin from the roof of the pulp chamber or pulp horns should remain. No attempt is made to control the hemorrhage until the coronal pulp has been amputated. A funnel-shaped access to the entrance of the root canals should be produced. A sharp discoid spoon excavator, large enough to extend across

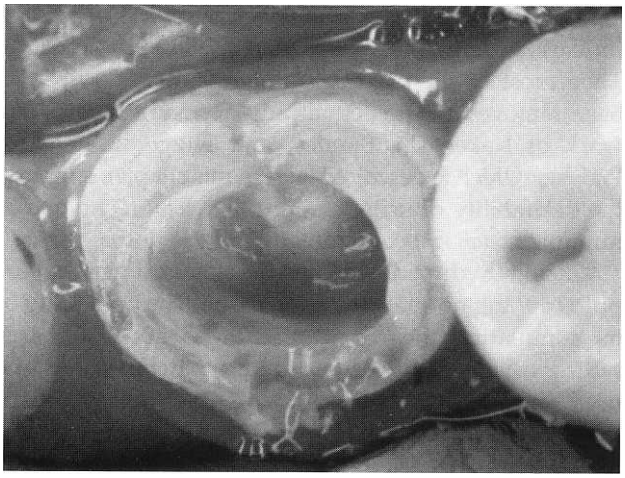
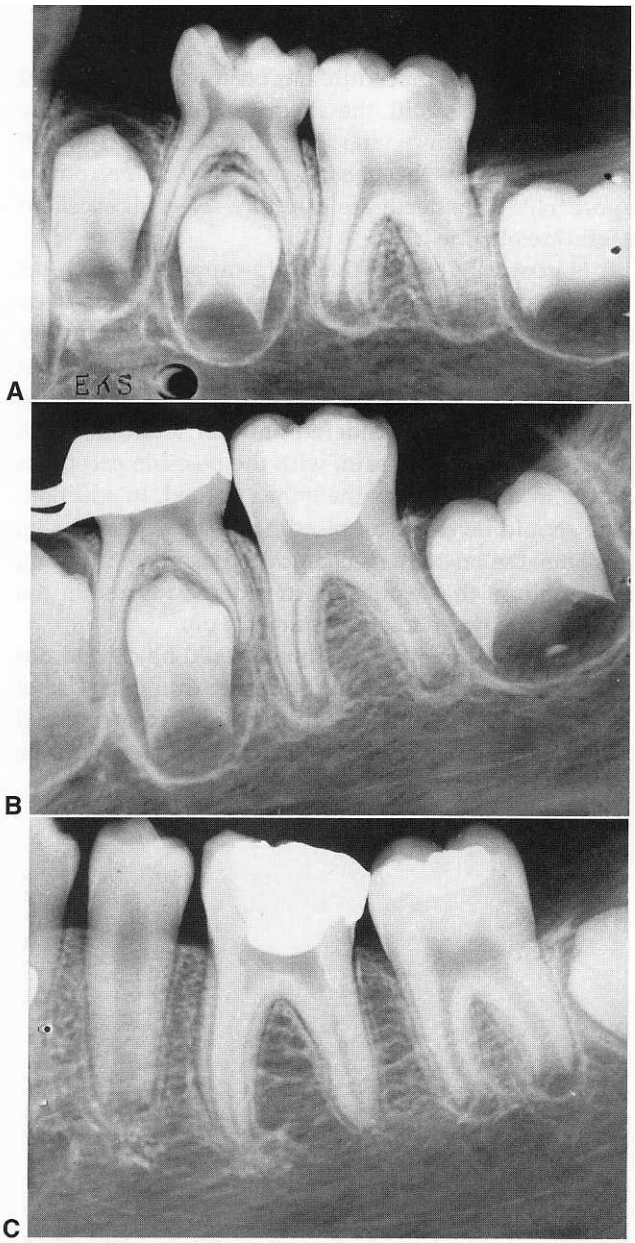


FIG **19-10.** Cleanly excised pulpal stumps with no tags of tissue across the floor or along the walls of the chamber. The hemorrhage has been controlled. Notice also that the roof of the pulp chamber has been completely removed to provide total access to the pulp canals.

the entrance of the individual root canals, may be used to amputate the coronal pulp at its entrance into the canals. The pulp stumps should be cleanly excised with no tags of tissue extending across the floor of the pulp chamber. The pulp chamber should then be irrigated with a light flow of water from the wa ter syringe and evacuated. Cotton pellets moistened with water should be placed in the pulp chamber and allowed to remain over the pulp stumps until a clot forms (Fig. 19-10).

Laboratory and clinical observations indicate that a different technique and capping material are necessary in the treatment of primary teeth than in treatment of permanent teeth. As a result of these observations, two specific pulpotomy techniques have evolved and are in general use today.

Pulpotomy Technique for Permanent Teeth. The calcium hydroxide pulpotomy technique is recommended in the treatment of permanent teeth with carious pulp exposures when there is a pathologic change in the pulp at the exposure site. This procedure is particularly indicated for permanent teeth with immature root development but with healthy pulp tissue in the root canals. It is also indicated for a permanent tooth with a pulp exposure resulting from crown fracture when the trauma has also produced a root fracture of the same tooth. The procedure is completed during a single appointment. Only teeth free of symptoms of painful pulpitis are considered for treatment. The procedure involves the amputation of the coronal portion of the pulp as described, the control of hemorrhage, and the placement of a calcium hydroxide capping material over the pulp tissue remaining in the canals (Fig. 19-11).



[I G **19-11.** A, Pulp of the first permanent molar was exposed by caries. The tooth was considered a candidate for the calcium hydroxide pulpotomy technique. **B,** Calcified bridge has formed over the vital pulp in the canals. C, Continued root development and pulpal recession are indicative of continuing pulpal vitality. The crown should be supported with a full-coverage restoration.

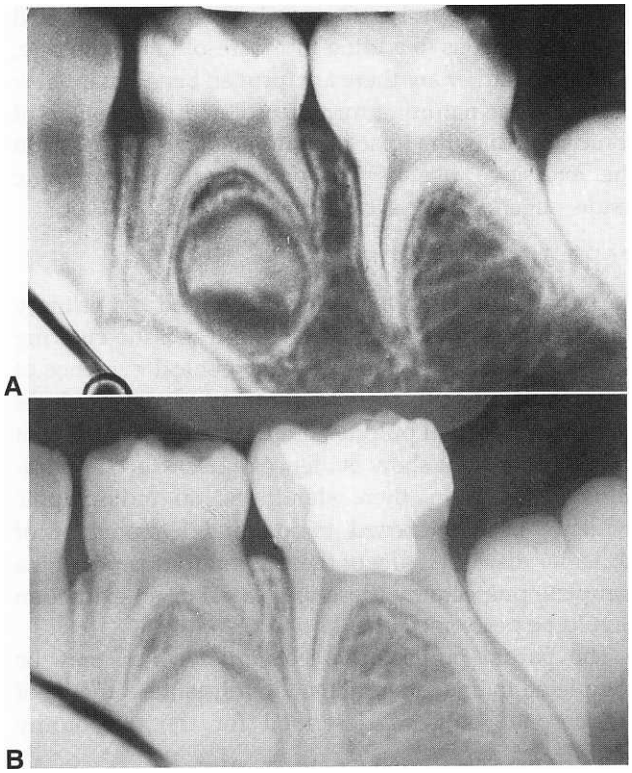
A protective layer of hard-setting cement is placed over the calcium hydroxide to provide an adequate seal. The tooth is subsequently prepared for full-coverage restoration. However, if the tissue in the pulp canals appears hyperemic after the amputation of the coronal tissue, a pulpotomy should no longer be considered. Endodontic treatment is indicated if the tooth is to be saved.

After 1 year, a tooth that has been treated successfully with a pulpotomy should have a normal periodontal ligament and lamina dura, radiographic evidence of a calcified bridge if calcium hydroxide was used as the capping material, and no radiographic evidence of internal resorption or pathologic resorption. The treatment of permanent teeth by the calcium hydroxide method has resulted in a higher rate of success when the teeth are selected carefully based on existing knowledge of diagnostic techniques.

Pulpotomy Technique for Primary Teeth. The same diagnostic criteria recommended for the selection of permanent teeth for the pulpotomy procedure should be used in the selection of primary teeth for the pulpotomy procedure. The treatment is also completed during a single appointment. A surgically clean technique should be used. The coronal portion of the pulp should be amputated as described previously, the debris should be removed from the chamber, and the hemorrhage should be controlled. If there is evidence of hyperemia after the removal of the coronal pulp, which indicates that inflammation is present in the tissue beyond the coronal portion of the pulp, the technique should be abandoned in favor of the partial pulpectomy or the removal of the tooth. If the hemorrhage is controlled readily and the pulp stumps appear normal, it may be assumed that the pulp tissue in the canals is normal, and it is possible to proceed with the pulpotomy.

Although the formocresol pulpotomy technique has been recommended for many years as the principal method for treating primary teeth with carious exposures, a substantial shift away from use of this medicament has occurred because of concerns about its toxic effects. Many alternatives have been investigated to replace formocresol as the medicament of choice for a pulpotomy technique. Despite this, formocresol continues to be a very commonly used pulpotomy medicament. The pulp chamber is dried with sterile cotton pellets. Next, a pellet of cotton moistened with a 1:5 concentration of Buckley's formocresol and blotted on sterile gauze to remove the excess is placed in contact with the pulp stumps and is allowed to remain for 5 minutes. Because formocresol is caustic, care must be taken to avoid contact with the gingival tissues. The pellets are then removed, and the pulp chamber is dried with new pellets. A thick paste of hard-setting zinc oxide—eugenol is prepared and placed over the pulp stumps. The tooth is then restored with a stainless steel crown (Fig. 19-12).

Although the recommendation is that the blotted cotton pellet moistened with a 1:5 concentration of formocresol be applied to the pulp stumps for 5 minutes, it should be acknowledged that the 5-minute application time has been determined somewhat arbitrarily. Few data are available to verify the optimal application time. Garcia-Godoy, Novakovic, and



F G 19-12. A, Formocresol pulpotomy technique was completed. **B,** Normal appearance of the supporting tissues is indicative of a successful treatment. The tooth should now be restored with a stainless steel crown.

Carvajal have suggested that a 1-minute application time may be adequate and perhaps superior to the recommended 5 minutes based on their limited work with pulpotomies in dogs." These authors agree, however, that further studies are needed for verification. A series of research studies by Loos and Han,[12] Loos, Straffon, and Han," and Straffon and Han[le] have led to the conclusion that a dilute (1:5 concentration) of Buckley's formocresol applied to tissue achieves the desired cellular response as effectively as the full-strength formocresol agent, yet allows a faster recovery of the affected cells. The researchers suggested that the 1:5 concentration is a safer medicament that would produce equally good results with fewer postoperative problems in pulpotomy procedures. The original Buckley's formula for formocresol calls for equal parts of formaldehyde and cresol.* The 1:5 concentration of this formula is prepared by first thoroughly mixing three parts of glycerin with one part of distilled water, then adding four parts of this diluent to one part of Buckley's formocresol, and thoroughly mixing again.

Some dentists prefer to make the pulp-capping material by mixing the zinc oxide powder with equal

*Sultan Chemists, Inc., Englewood, N.J.

parts of eugenol and formocresol. There are no proved contraindications to adding formocresol to the mixture; however, neither are there any proven benefits. In view of the caustic nature of formocresol and the concern of some regarding the toxic and mutagenic potential from the excessive use of formocresol, its use in zinc oxide—eugenol paste is discouraged.

PARTIAL PULPECTOMY

A partial pulpectomy may be performed on primary teeth when coronal pulp tissue and the tissue entering the pulp canals are vital but show clinical evidence of hyperemia (Fig. 19-13). The tooth may or may not have a history of painful pulpitis, but the contents of the root canals should not show evidence of necrosis (suppuration). In addition, there should be no radiographic evidence of a thickened periodontal ligament or of radicular disease. If any of these conditions is present, a complete pulpectomy (described later) or an extraction should be performed.

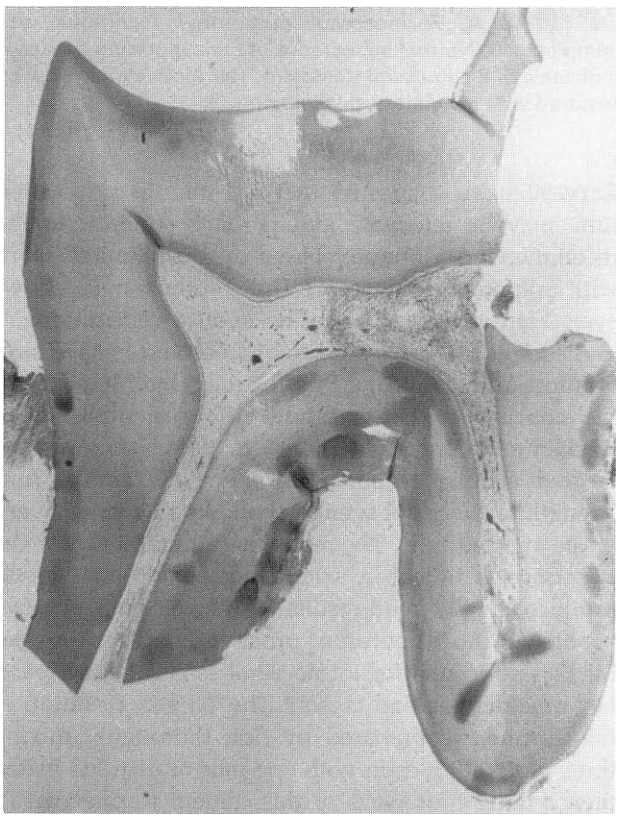
The partial pulpectomy technique, which may be completed in one appointment, involves the removal of the coronal pulp as described for the pulpotomy technique. The pulp filaments from the root canals are removed with a fine barbed broach; considerable hemorrhage will occur at this point. A Hedstrom file will be helpful in the removal of remnants of the pulp tissue. The file removes tissue only as it is withdrawn and penetrates readily with a minimum of resistance. Care should be taken to avoid penetrating the apex of the tooth.

Many dentists prefer to use root canal instruments placed in a special handpiece for root canal debridement. Root canal instrumentation may be facilitated with the judicious use of this mechanical technique, especially in canals that are difficult to negotiate with hand instruments. Cautious manipulation is important, however, to prevent breaking the file or overinstrumenting the canal and apical tissues.

After the pulp tissue has been removed from the canals, a syringe is used to irrigate them with 3% hydrogen peroxide followed by sodium hypochlorite. The canals should then be dried with sterile paper points. When hemorrhaging is controlled and the canals remain dry, a thin mix of unreinforced zinc oxide--eugenol paste may be prepared (without setting accelerators), and paper points covered with the material are used to coat the root canal walls. Small Kerr files may be used to file the paste into the walls. The excess thin paste may be removed with paper points and Hedstrom files. A thick mix of the treatment paste should then be prepared, rolled into a point, and carried into the canal. Root canal pluggers may be used to condense the filling material into the canals. An x-ray film may be necessary to allow evaluation of the success in filling the canals (Fig. 19-14). Further condensation may be carried out if required. The tooth should be restored with full coverage.

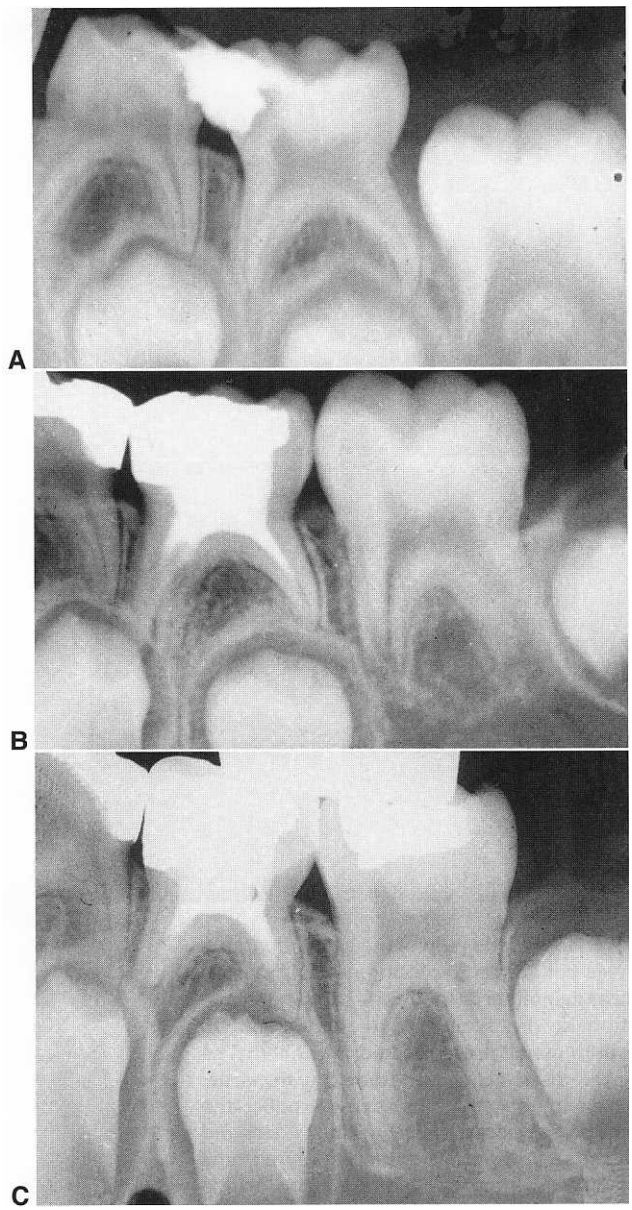
Although zinc oxide—eugenol paste is viewed as the traditional root canal filling material for primary teeth, work reported by Holan and Fuks,[75] Ranly and Garcia-Godoy,[16"] Garcia-Godoy," and Rifkin[9.2o] suggest that KRI paste* may be preferable. Excellent results have been observed in many cases. The primary components of KRI paste are zinc oxide and iodoform. The main advantages of KRI paste compared with zinc oxide—eugenol paste are that KRI paste resorbs in synchrony with primary roots and is less irritating to surrounding tissues if a root is inadvertently overfilled.

Another popular root canal filling material for primary teeth is Vitapex,[t] a product that has received many favorable anecdotal reports about its successful use in infected primary teeth. The primary components of Vitapex are calcium hydroxide and iodoform. Considerable laboratory and animal research has been reported. Vitapex may be at least as effective as KRI



f **I C 19-13.** Histologic section of a second primary molar with a carious pulp exposure. There was clinical evidence of hyperemia and inflammation of the pulp. Inflammation is evident in half the coronal pulp and into the pulp canal. This condition may be treated using the partial pulpectomy technique.

*Pharmachemie AG, Drusberstr. 125, CH-8053 Zurich, Switzerland. ' Dia Dent Group International, Inc., Vancouver, British Columbia, Canada.



[I G **19-14. A**, Pulp in the second primary molar showed evidence of inflammation. The tooth was treated with the partial pulpectomy technique. **B,** Thirteen months after treatment, the second primary molar was asymptomatic and the supporting tissues appeared normal. C, Three years after the initial treatment, the radiograph of the second primary molar appears normal. The first permanent molar has erupted into a good position.

paste, and Nurko and Garcia-Godoy[L1] and Garcfa-Godoy[u] ' have published some human reports.

NONVITAL PULP THERAPY TECHNIQUE

COMPLETE PULPECTOMY

It is unwise to maintain untreated infected primary teeth in the mouth. They may be opened for drainage and often remain asymptomatic for an indefinite period. However, they are a source of infection and should be treated or removed. The morphology of the root canals in primary teeth makes end odontic treatment difficult and often impractical. Mature first primary molar canals are often so small that they are inaccessible even to the smallest barbed broach. If the canal cannot be properly cleansed of necrotic material, sterilized, and adequately filled, endodontic therapy is more likely to fail.

Hibbard and Ireland studied the primary root canal morphology by removing the pulp from extracted teeth, forcing acrylic resin into the pulp canals, and dissolving the covering of tooth structure in 10% nitric acid.[2] It was apparent that initially only one root canal was present in each of the mandibular and maxillary molar roots. The subsequent deposition of secondary dentin throughout the life of the teeth caused a change in the morphologic pattern of the root canal, producing variations and eventual alterations in the number and size of the canals. The variations included lateral branching, connecting fibrils, apical ramifications, and partial fusion of the canals. These findings explain the complications often encountered in root canal therapy.

Endodontic procedures for the treatment of primary teeth with necrotic pulps are indicated if the canals are accessible and if there is evidence of essentially normal supporting bone. If the supporting bone is also compromised, the likelihood of successful endodontic therapy is lower. If the second primary molar is lost before the eruption of the first permanent molar, the dentist is confronted with the difficult problem of preventing the first permanent molar from drifting mesially during its eruption. Special effort should be made to treat and retain the second primary molar even if it has a necrotic pulp. Similarly, longer than normal retention of a second primary molar may be desired when the succedaneous second premolar is congenitally missing (Fig. 19-15).

Starkey developed the following complete pulpectomy technique for primary molars.' The rubber dam should be applied, and the roof of the pulp chamber should be removed to gain access to the root canals as described previously in the pulpotomy technique. The contents of the pulp chamber and all debris from the occlusal third of the canals should be removed, with care taken to avoid forcing any of the infected contents through the apical foramen. A pellet moistened with camphorated monochlorophenol (CMCP) or a 1:5 concentration of Buckley's formocresol, with excess moisture blotted, should be placed in the pulp chamber. The chamber may be sealed with zinc oxide–eugenol. At the second appointment, several days later, the tooth should be isolated with a rubber dam and the treatment



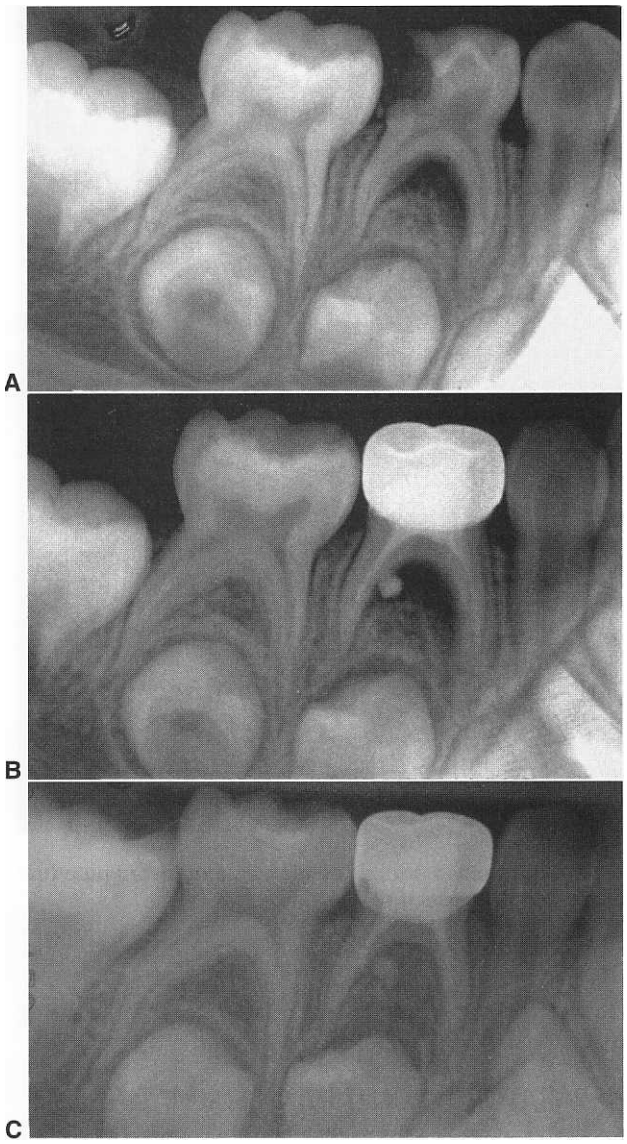
FIG 19-15. **A,** Necrotic tooth resulting from a carious exposure of the pulp of the second primary molar. Because the succedaneous second premolar was congenitally missing, a decision was made to attempt to save the tooth as a functional space maintainer through the growing years, if possible. Note the evidence of internal resorption at the floor of the pulp chamber. **B,** Radiograph made 1 year and 7 months after the pulp canals were treated and filled. The mesial canal was treated with complete pulpectomy; the distal canal was treated with partial pulpectomy. C, Six years and 7 months after treatment, the tooth is asymptomatic; the supporting tissues appear normal but some root resorption has occurred. **D,** Fourteen years and 6 months postoperatively, the tooth was extracted because of the development of symptoms and loss of bone support. At this time, the patient was a young adult and a fixed bridge was made.

pellet removed. If the tooth has remained asymptomatic during the interval, the remaining contents of the canals should be removed using the technique described for the partial pulpectomy. The apex of each root should be penetrated slightly with the smallest file. (The dentist should experiment with dissociated primary molars to develop a feel for the instrument as it just penetrates the apex.) A treatment pellet should again be placed in the pulp chamber and the seal completed with zinc oxide—eugenol. After another few days the treatment pellet should be removed. If the tooth has remained asymptomatic, the canals may be prepared and filled as described for the partial pulpectomy. However, if the tooth has been painful and there is evidence of moisture in the canals when the treatment pellet is removed, the canals should again be mechanically cleansed and the treatment repeated.

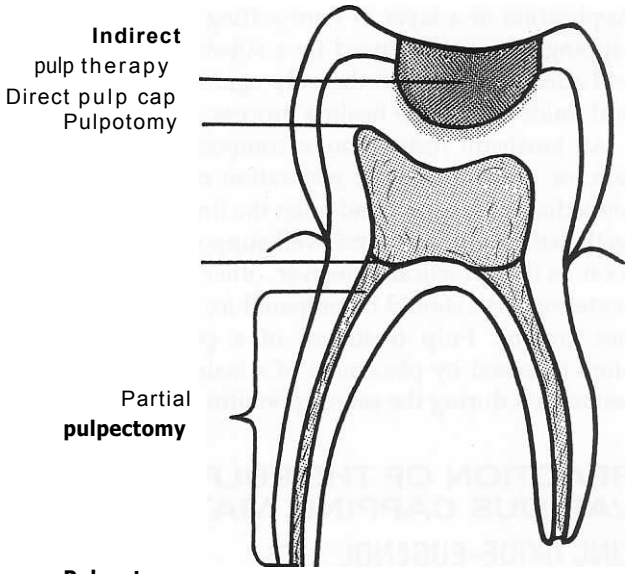
Currently, pulpectomies in primary teeth are commonly completed in a single appointment (Fig. 19-16). If the tooth has painful necrosis with purulence in the canals, however, completing the pulpectomy procedure over two or three visits should improve the likelihood of success.

SUMMARY OF PULP THERAPY

The preceding discussion of various pulp therapies conforms, in principle, to the Guidelines for Pulp Therapy for Primary and Young Permanent Teeth as



[H G **19-16.** Successful single-appointment complete pulpectomy. Note extrusion of zinc oxide–eugenol into furcal area from distal root accessory canal, but adequate subsequent healing. A, Pretreatment. **B,** Immediately after treatment. C, 10 months after treatment.



f I G **19-17.** Pulp therapy progression.

reaffirmed by the American Academy of Pediatric Dentistry in 2002.[2"]

When one encounters clinical problems that will likely require pulp therapy to return the patient to satisfactory oral health, treatment decisions are not always clear-cut. Proper diagnosis of the pulpal problem is important to allow the dentist to select the most conservative treatment procedure that offers the best chance of long-term success with the least chance of subsequent complications. The dentist should think of the possible treatment options in a progressive manner that takes into account both treatment conservatism (e. g., a pulpotomy is more conservative than a partial pulpectomy) and posttreatment problems (Fig. 19-17). The most conservative treatment possible may not always be the indicated procedure after the dentist also weighs the risks of posttreatment failure in a particular case.

RESTORATION OF THE PULPALLY INVOLVED TOOTH

It has been a common practice for some dentists to delay for weeks or months the permanent restoration of a tooth that has undergone vital pulp therapy. The purpose has been to allow time to determine whether the treatment procedure will be successful. However, failures in pulp therapy are usually not evident for many months. Rarely does a failure in pulp therapy or an endodontic procedure on a primary tooth cause the child to experience acute symptoms. Failures are usually evidenced by pathologic root resorption or rarefied areas in the bone and are discovered during regular recall appointments.

Primary and permanent molars that have been treated by the pulpotomy or pulpectomy technique have a weak, unsupported crown that is liable to fracture. Often a failure of the buccal or lingual plate occurs below the gingival attachment or even below the crest of the alveolar bone. This type of fracture makes subsequent restoration of the tooth impractical. Also, a delay in restoring the tooth with a material that will adequately seal the tooth and prevent an ingress of oral fluids is one cause for failure of pulp therapy.

Application of a layer of hard-setting cement over the capping material followed by a substantial restoration will adequately protect the pulp against contaminating oral fluids during the healing process.

An amalgam restoration, a composite resin restoration, or a glass ionomer restoration may serve as the immediate restoration and often the final restoration for teeth with pulp caps and well-supported crowns. As soon as it is practical, however, other pulpally treated posterior teeth should be prepared for stainless steel or cast crowns. Pulp treatment of a primary molar is often followed by placement of a stainless steel crown restoration during the same appointment.

REACTION OF THE PULP TO VARIOUS CAPPING MATERIALS

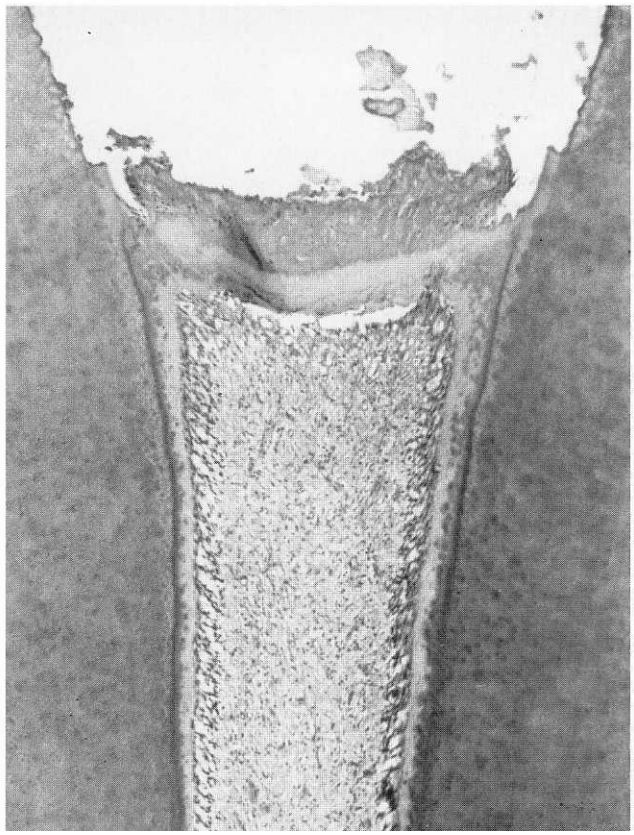
ZINC OXIDE-EUGENOL

Before calcium hydroxide came into common use, zinc oxide—eugenol was used more often than any other pulp-capping material. Many dentists have apparently had good clinical results with the use of zinc oxide—eugenol, but it is no longer recommended as a direct pulp-capping material.

CALCIUM HYDROXIDE

Herman first introduced calcium hydroxide as a biologic dressing.[25] Because of its alkalinity (pH of 12), it is so caustic that when it is placed in contact with vital pulp tissue the reaction produces a superficial necrosis of the pulp. The irritant qualities seem to be related to its ability to stimulate development of a calcified barrier. The superficial necrotic area in the pulp that develops beneath the calcium hydroxide is demarcated from the healthy pulp tissue below by a new, deeply staining zone comprising basophilic elements of the calcium hydroxide dressing. The original proteinate zone is still present. However, against this zone is a new area of coarse fibrous tissue likened to a primitive type of bone. On the periphery of the new fibrous tissue, cells resembling odontoblasts appear to be lining up. One month after the capping procedure, a calcified bridge is evident radiographically. This bridge continues to increase in thickness during the next 12 months (Fig. 19-18). The pulp tissue beneath the calcified bridge remains vital and is essentially free of inflammatory cells.

Many research studies can be cited regarding the use of calcium hydroxide as a pulp-capping material, and a few are included in the references for this chapter. Investigators who evaluate experimental pulp-capping agents commonly compare their results with the agent being tested to the results they can obtain with calcium hydroxide under similar conditions. Thus calcium hydroxide currently serves as the standard or control material for experimentation related to pulp-capping agents.

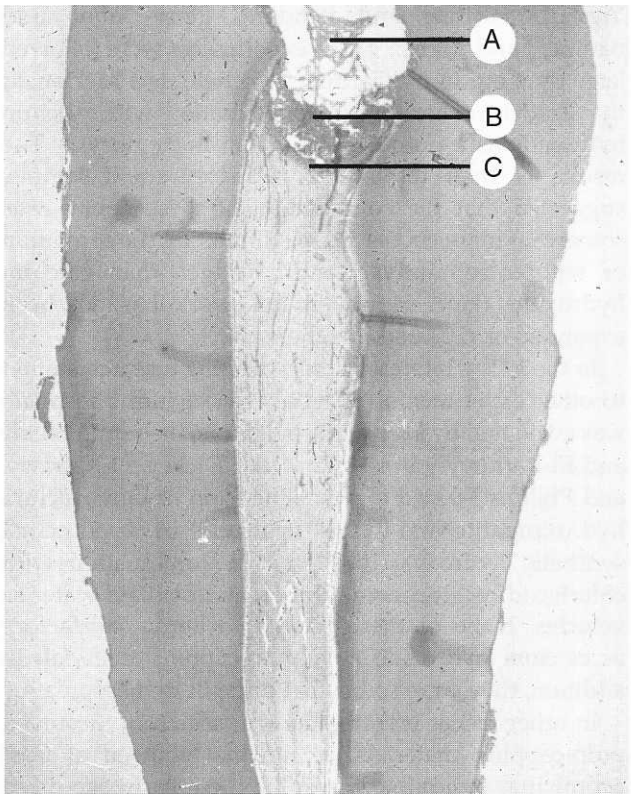


f **I G 19-18.** Calcified bridge covering an amputated pulp that was capped with calcium hydroxide.

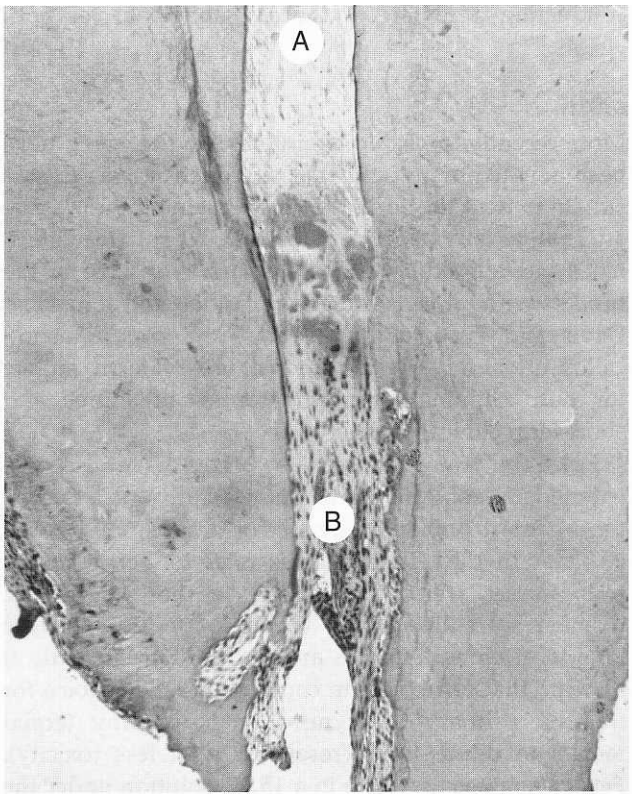
PREPARATIONS CONTAINING FORMALIN

The belief that exposing the pulp to formocresol or capping it with materials that contain formocresol will promote pulp healing or even maintain the pulp in a healthy state has not been adequately substantiated. Some studies have indicated that the formocresol pulpotomy technique may be applied to permanent teeth, but its use in permanent teeth remains an interim procedure to be followed by conventional endodontic therapy. The clinical success experienced in the treatment of primary pulps with these materials is possibly related to the drug's germicidal action and fixation qualities rather than to its ability to promote healing.

Doyle, McDonald, and Mitchell compared the success of the full-strength formocresol pulpotomy technique with the success of the calcium hydroxide pulpotomy technique.[2"] Experimental pulpotomies were performed on 65 normal human primary teeth, many of which could later be extracted for histologic examination. The formocresol technique was used on 33 teeth, and the calcium hydroxide technique was used in the treatment



[I G **19-19.** Histologic section of a primary pulp exposed to formocresol for 4 days. The medicament contacted the pulp at A, the debris and blood clot are evident at B, and a noticeably eosinophilic, compressed line is evident at C. The underlying pulp was a pale, homogeneously stained tissue with a loss of basophilic nuclei. *(Courtesy Dr. Walter A. Doyle.)*



f **I G 19-20.** Histologic section of a primary pulp exposed to formocresol for 41 days. The pulp, A, appeared pale and pink, and there was a loss of cellular definition. Vital tissue can be seen in the apical portion, B. *(Courtesy Dr. Walter A. Doyle.)*

of the other 32. Under the conditions of this study the formocresol pulpotomy technique yielded outcomes superior to those of the calcium hydroxide technique for at least the first 18 months after treatment. The results of the combined methods of evaluation indicated that the calcium hydroxide pulpotomy technique for primary teeth was successful in 61% of cases. The formocresol pulpotomy resulted in success in 95% of cases at the end of 1 year.

Formocresol did not stimulate the healing response of the remaining pulp tissue but rather tended to fix essentially all the remaining tissue (Figs. 19-19 and 19-20). Use of calcium hydroxide was associated with the formation of a dentin bridge and the complete healing of the amputated primary pulp in 50% of the cases that were available for histologic study.

GLUTARALDEHYDE

Glutaraldehyde has received attention as a potential pulp-capping agent for pulpotomy techniques in primary teeth. It is an excellent bactericidal agent and seems to offer some advantages compared with formocresol.

Berson and Good have reported that glutaraldehyde appears to be superior to formaldehyde preparations for pulp therapy in the following ways":

1. Formaldehyde reactions are reversible, but glutaraldehyde reactions are not.
2. Formaldehyde is a small molecule that penetrates the apical foramen, whereas glutaraldehyde is a larger molecule that does not.
3. Formaldehyde requires a long reaction time and an excess of solution to fix tissue, whereas glutaraldehyde fixes tissue instantly and an excess of solution is unnecessary.

Although glutaraldehyde seems to compare favorably with formocresol as a pulp-capping agent, it has not consistently demonstrated significant superior results in clinical trials. One clinical study by Fuks et al found an 18% failure rate after 25 months in pulpotomized primary molars.- They concluded that their results did not justify substituting glutaraldehyde for formocresol treatment in primary tooth pulpotomies. Feigal and Messer have questioned the rationale for using glutaraldehyde as an alternative to formocresol after

conducting a review of the available data for both agents.'

FERRIC SULFATE

More recently, considerable interest and research have been devoted to investigating the effectiveness of ferric sulfate to treat the surface of the remaining pulp tissue after pulpotomy of primary teeth. Ferric sulfate agglutinates blood proteins and controls hemorrhage in the process without clot formation. Landau and Johnsen,[30] Davis and Furtado,[31] and Fei, Udin, and Johnson[32] called attention to the potential use of ferric sulfate for pulp capping after pulpotomies in animal and short-term clinical studies.

Fuks and two groups of coworkers have also contributed favorable data in an animal study and a longer-term clinical human study (mean observation period, 20.5 months).[33,34] Their success rates for ferric sulfate pulpotomies were very similar to those for dilute formocresol pulpotomies (control condition). More long-term clinical studies are needed, but currently it appears that ferric sulfate could be a better choice for treating primary teeth needing pulpotomy (equal results to dilute formocresol but with less toxicity). Ferric sulfate is available in a 15.5% solution under the trade name of Astringedent.*

An interesting study by Casas et al compared the outcome of ferric sulfate pulpotomy with that of primary tooth root canal therapy (pulpectomy) on cariously exposed vital pulps of primary molars.[3S] Although their study showed that root canal therapy had produced more acceptable treatment outcomes than ferric sulfate pulpotomy in vital pulp treatment of primary molars at a 2-year follow-up visit, the survival rates for the two techniques were not statistically different. There was no clinical evidence of pathosis in 96% of the ferric sulfate pulpotomies and 98% of the molars undergoing root canal therapy. They suggest that, for clinicians who wish to avoid aldehydes in vital molar pulp therapy for children, either one of these two alternatives is feasible. Of course, the main advantage of the ferric sulfate pulpotomy over a pulpectomy when working with children is the considerably faster speed with which a pulpotomy can be performed.

OTHER EXPERIMENTAL CAPPING MATERIALS (MINERAL TRIOXIDE AGGREGATE, BONE MORPHOGENETIC PROTEIN, AND OTHERS)

Pulp-capping experiments in animals have tested a variety of antibiotics and corticosteroids, alone or in combination with calcium hydroxide. Some of the earlier experiments were reported by Kutscher and Yigdall,[3f] Seltzer and Bender," Fiore-Donno and Baume,[38] and Baker." These experiments were followed later by a study by Gardner, Mitchell, and McDonald that tested vancomycin in combination with calcium hydroxide as a pulp-capping agent in monkeys. [10] The results of their tests, in a relatively small sample, suggested that the combination of these agents was somewhat more successful in stimulating the formation of regular reparative dentin bridges than calcium hydroxide alone. However, this work has not been expanded or repeated by others.

In the 1970s, interest in pulp-capping research shifted to other experimental materials. Tricalcium phosphate was evaluated by several investigators, including Boone and El-Kafrawy[41] and Heys et al.'[2] Dickey, El-Kafrawy, and Phillips' tested a crystalline form of pure calcium hydroxyapatite, and Ibarra`[n] evaluated an experimental synthetic hydroxyapatite used in combination with chlorhexidine gluconate solution and distilled water as vehicles. None of these proved to be as satisfactory as calcium hydroxide as a pulp-capping material. In addition, they were somewhat difficult to manipulate.

In other recent investigations in search of improved pulp-capping materials, agents that showed at least promising preliminary results include freeze-dried bone, chlorhexidine, feracrylum, calcium phosphate ceramics, tetracalcium phosphate cement, dentin-bonding agents in combination with bonded resin or glass ionomer materials, mineral trioxide aggregate, and bone morphogenetic proteins.[45-"]

Pulp-capping with dentin-bonding agents combined with bonding restorative materials has created considerable debate and controversy among dental investigators. Perhaps the most exciting and promising areas of pulp-capping research are the investigations under way with mineral trioxide aggregate and bone morphogenetic proteins. Both pulp treatment approaches seem to stimulate natural dentin repair at pulpal exposure sites. In recent unpublished research by Agamy et al, gray mineral trioxide aggregate, white mineral trioxide aggregate, and formocresol were compared as pulp dressings in pulpotomized primary teeth." Sixty pulpotomized teeth in 20 patients were studied. In both the clinical and histologic portions of the study, the gray mineral trioxide aggregate appeared to be superior to the white mineral trioxide aggregate and to formocresol as a pulp dressing for pulpotomized primary teeth.

In an excellent review on pulpotomies in primary teeth, Ranly suggested that pulpotomy modalities in primary teeth can be classified by treatment objective into three categories: devitalization, preservation, and regeneration.[58] He noted that the treatment objective of an ideal pulpotomy agent is to leave the radicular pulp vital and healthy and completely enclosed within an odontoblast-lined dentin chamber. The regeneration

*Ultradent Products, Inc., South Jordan, Utah.

modality most closely resembles this ideal. Through the use of a family of bone morphogenetic proteins, it may be possible to induce reparative dentin formation with recombinant dentinogenic proteins similar to the native proteins of the body. Fuks suggests that, because the specificity of growth factors such as trans-forming growth factor 13 and bone morphogenetic protein in inducing reparative processes is not clear, further studies are required to fully understand the kinetics of growth factor release and the sequence of growth factor–induced reparative dentinogenesis.[10] Commercially available recombinant human bone morphogenetic proteins for pulp therapy are now available for experimentation and clinical trials.

OTHER EXPERIMENTAL CAPPING METHODS

The pulp response to formocresol has been compared with electrosurgical coagulation after pulpotomies in the teeth of monkeys by Ruemping, Morton, and Anderson.'" The sample size was not large, and the observation periods were relatively short (maximum was 2 months after the operation), but the results of their histologic study showed the electrosurgical technique to be as favorable as the full-strength formocresol technique. Shaw et al have also demonstrated favorable results lasting up to 6 months with electrosurgical pulpotomies in monkeys.[60]

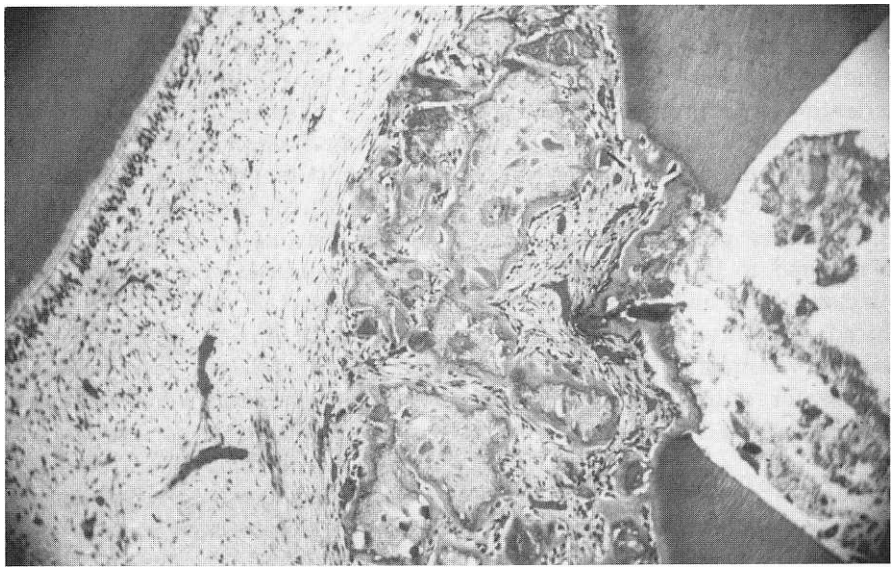
Mack and Dean reported the results of a retrospective human study of electrosurgical pulpotomies performed on primary molars." The mean postoperative observation time for the 164 teeth studied was 2 years, 3 months. They reported a 99.4% success rate (one failure) for this pulpotomy technique. In addition, Dean et al demonstrated no statistically significant difference between the electrosurgical and formocresol pulpotomy techniques in a prospective clinical study involving 50 children requiring at least one pulpotomy.[6]- The children were randomly divided into two groups, with 25 undergoing the electrosurgical technique and 25 undergoing the formocresol technique. The mean age at treatment was 63.6 months and the mean postoperative observation time was 10.9 months. The clinically and radiographically determined success rates were 96% and 84%, respectively, for the electrosurgical group and 100% and 92%, respectively, for the formocresol group. There was no statistically significant difference between results for the two techniques, although the electrosurgical group did have four failures whereas two failures occurred in the formocresol group. These researchers concluded that the results of their study support the use of electrosurgical pulpotomy as a viable alternative to formocresol pulpotomy. Rivera et al[63] obtained results similar to those of Dean et al; however, Fishman et al[b]" found considerably lower success rates with the use of electrosurgical pulpotomy.

Shoji, Nakamura, and Horiuchi reported the results of some preliminary studies on the treatment of amputated pulps (pulpotomies) in dogs by CO, laser radiation.[65] Wilkerson, Hill, and Arcoria reported favorable pulpal responses of healing and repair in swine following pulpotomies using an argon laser.`' Moritz et al applied 200 direct pulp caps in adult patients after mechanical pulp exposures." Half of the teeth (control group) received a conventional calcium hydroxide pulp cap. The other half (experimental group) received a calcium hydroxide cap after first undergoing CO_7 laser radiation until the "exposed pulps were completely sealed." The teeth were monitored monthly. One year after treatment, the success rate for teeth in the experimental group was 89%, whereas the success rate in the control group was 68%. Both the electrosurgical and the laser techniques seem to be favorable areas for further research in pulp therapy.

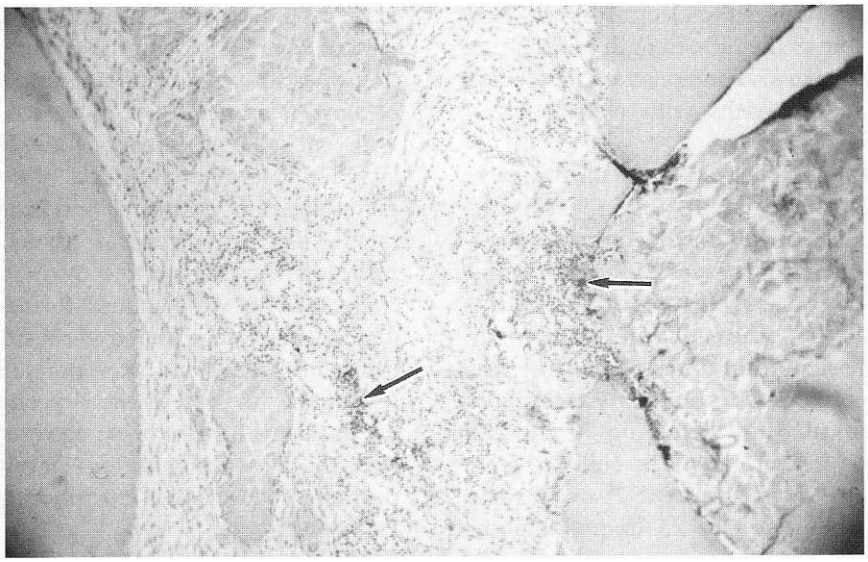
FAILURES AFTER VITAL PULP THERAPY

Failure in the formation of a calcified bridge across the vital pulp has often been related to the age of the patient, degree of surgical trauma, sealing pressure, improper choice of capping material, low threshold of host resistance, and presence of microorganisms with subsequent infection. Kakehashi, Stanley, and Fitzgerald studied the effect of surgical exposures of dental pulps in germ-free and conventional laboratory rats." The injured pulpal tissue contaminated with microorganisms failed to show evidence of repair; especially lacking were matrix formation and attempted dentinal bridging. In the germ-free animals, bridging began in 14 days and was complete in 28 days regardless of the severity of the exposure. The major determinant in the healing of exposed rodent pulps appeared to be the presence or absence of microorganisms. These findings were later corroborated by Watts and Paterson.[65]

Walshe provided further evidence that the success of vital pulp therapy depends on adherence to a surgically aseptic technique. In his experiment the teeth of monkeys were capped with bovine dentin mixed with methylcellulose, and histologic observations were made 42 days postoperatively.`" Approximately half the teeth capped with the experimental material were successfully repaired with atubular dentin (Fig. 19-21). The remaining teeth showed varying degrees of inflammation and repair. The Brown and Bren staining technique demonstrated the presence of microorganisms in the pulp of the teeth that failed to repair (Figs. 19-22 and 19-23). The stain also revealed microorganisms between the dentin walls and the filling material. The microorganisms were apparently introduced at the time of the pulp-capping procedure, or



[IG. 19-21. Surgical exposure of the pulp of a monkey was capped with powdered bovine dentin. Atubular dentin bridging the exposure site was evident 42 days postoperatively.



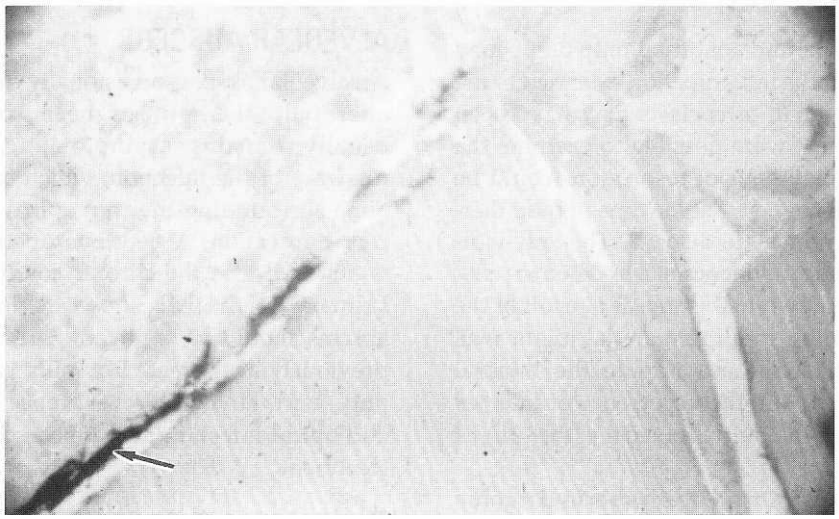
f IG 19-22. Pulps capped with bovine dentin failed to undergo calcific repair and demonstrated microorganisms (arrows) in the pulp. (Courtesy *Dr. Martin Walshe.)*

leakage of the restoration allowed them to gain entrance to the pulp chamber. This study likewise supports the need for a good surgical technique and the placement of a restoration that will provide the best possible seal.

INTERNAL RESORPTION

Radiographic evidence of internal resorption occurring within the pulp canal several months after the pulpotomy procedure is the most frequently seen evidence of an abnormal response in primary teeth (Fig. 19-24). Internal resorption is a destructive process generally believed to be caused by osteoclastic activity, and it may progress slowly or rapidly. Occasionally, secondary repair of the resorbed dentinal area occurs.

No satisfactory explanation for the postpulpotomy type of internal resorption has been given. It has been demonstrated, however, that with a true carious exposure of the pulp, an inflammatory process of some degree will be present. The inflammation may be limited to the exposure site, or it may be diffuse throughout the coronal portion of the pulp. Amputation of all the pulp that shows the inflammatory change may



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[IG 19-23. Microorganisms (arrow) may be seen between the dentin walls and the filling material. (Courtesy Dr. Martin W.9/she.)



fIG 19-24. A, Preoperative radiograph of a second primary molar treated with the calcium hydroxide pulpotomy technique. **B,** Two years after treatment, internal resorption and bone rarefaction are evident. C, There may have been inflammation of the pulp apical to the amputation site and beginning internal resorption at the time of the initial treatment.

be difficult or impossible, and abnormal pulp tissue may be allowed to remain. If the inflammation extended to the entrance of the pulp canal, osteoclasts may have been attracted to the area; if it were possible to examine the tooth histologically, small bays of resorption would be evident. This condition may exist at the time of pulp therapy, although there is no way to detect it. The only indication would be the clinical evidence of a hyperemic pulp.

Inflammatory cells drawn to the area as a result of the placement of an irritating capping material might well attract the osteoclastic cells and initiate the internal resorption. This may explain the occurrence of internal resorption even though the pulp is normal at the time of treatment.

Because the roots of primary teeth are undergoing normal physiologic resorption, vascularity of the apical region is increased. Osteoclastic activity is present in the area. This may predispose the tooth to internal resorption when an irritant in the form of a pulp-capping material is placed on the pulp.

ALVEOLAR ABSCESS

An alveolar abscess occasionally develops some months after pulp therapy has been completed. The tooth usually remains asymptomatic, and the child is unaware of the infection, which may be present in the bone surrounding the root apices or in the area of the root bifurcation. A fistulous opening may be present, which indicates the chronic condition of the infection. Primary teeth that show evidence of an alveolar abscess should be removed. Permanent teeth that have previously been treated by pulp capping or by pulpotomy and later show evidence of pulpal necrosis and apical infection may be considered for endodontic treatment.

EARLY EXFOLIATION OR OVERRETENTION OF PRIMARY TEETH WITH PULP TREATMENTS

Occasionally a pulpally treated tooth previously believed to be successfully managed will loosen and



F **I G 19-25. A,** A pulpally involved second primary molar was successfully treated with a formocresol pulpotomy and restored with a steel crown. **B,** This 4-year posttreatment radiograph reveals long-term successful management. C and D, Bilateral bite-wing radiographs for the same patient 7 years after the pulpotomy reveal that the resorption of the pulpotomized molar is falling behind compared with its antimere. The eruption of the permanent second premolar is also being delayed. The pulpally treated tooth should be extracted at this time. *(Courtesy Dr. Wayne A. Moldenhauer.)*

exfoliate (or require extraction) prematurely for no apparent reason. It is believed that such a condition results from low-grade, chronic, asymptomatic, localized infection. Usually, abnormal and incomplete root resorption patterns of the affected teeth are also observed. When this occurs, space management must be considered.

Another sequela requiring close observation is the tendency for primary teeth undergoing successful pulpotomies or pulpectomies to be overretained. This situation may have the untoward result of interfering with the normal eruption of permanent teeth and adversely affecting the developing occlusion. Close periodic observation of pulpally treated teeth is necessary to intercept such a developing problem. Extraction of the primary tooth is usually sufficient. Starkey believes that this phenomenon occurs when normal physiologic exfoliation is delayed by the bulky amount of cement contained in the pulp chamber.[23] Even though the material is resorbable, its resorption is slowed significantly when large quantities are present (Fig. 19-25).

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CHAPTER 20

Gingivitis and Periodontal Disease

RALPH E. McDONALD
DAVID R. AVERY
JAMES A. WEDDELL

انتشارات
شایان نمودار

CHAPTER OUTLINE

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- Gingivitis Associated with Poor Oral Hygiene
- Allergy and Gingival Inflammation

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EXTRINSIC STAINS AND DEPOSITS ON TEETH

Green Stain
- Orange Stain
- Black Stain
- Removal of Extrinsic Stains
- Pigmentation Caused by Stannous Fluoride Application

CALCULUS

The gingiva is the part of the oral mucous membrane that covers the alveolar processes and the cervical portions of the teeth. It has been divided traditionally into the free and the attached gingiva.' The free gingiva is the tissue coronal to the bottom of the gingival sulcus. The attached gingiva extends apically from the free gingival groove to the mucogingival junction.

The gingival tissues are normally light pink, although the color may be related to the complexion of the person, the thickness of the tissue, and the degree of keratinization. The gingival color of the young child may be more reddish due to increased vascularity and thinner epithelium. The surface of the gingiva of a child appears less stippled or smoother than that of an adult. In the healthy adult the marginal gingiva has a sharp, knifelike edge. During the period of tooth eruption in the child, however, the gingivae are thicker and have rounded margins due to the migration and cervical constriction of the primary teeth.

Delaney reports probing depths around primary teeth to be approximately 2 mm, with the facial and lingual probe sites shallower than the proximal sites.'-Children have a wider periodontal ligament than the adult. The width of the attached gingiva is narrower in the mandible than in the maxilla, and both widths increase with the transition from the primary to permanent dentition in the child. The alveolar bone surrounding in the primary dentition demonstrates fewer trabeculae, less calcification, and larger marrow spaces.

Recent recognition that periodontal disease may have its origins in childhood has led dentists to be more aggressive in treatment. Studies confirm a high prevalence of gingival inflammation in children. Periodontal conditions that progress rapidly and result in the loss of primary and permanent teeth have been noted with increased frequency. Therefore, the American Academy of Pediatric Dentistry's Dental Health Objectives for Children for the Year 2000 includes placing greater emphasis on the prevention, early diagnosis, and treatment of gingival and periodontal disease in children.' By establishing excellent oral hygiene habits in children, which will carry over to adulthood, the risk of periodontal disease is lowered.

Gingivitis is an inflammation involving only the gingival tissues next to the tooth. Microscopically, it is characterized by the presence of an inflammatory exudate and edema, some destruction of collagenous gingival fibers, and ulceration and proliferation of the epithelium facing the tooth and attaching the gingiva to it. Numerous studies indicate that marginal gingivitis is the most common form of periodontal disease and starts in early childhood.

Severe gingivitis is relatively uncommon in children, although numerous surveys have shown that a large portion of the pediatric population has a mild, reversible type of gingivitis. The major etiologic factors associated with gingivitis and more significant periodontal disease are uncalcified and calcified bacterial plaque. However, gingivitis rarely progresses to periodontitis in the preschool and grade school child.

Bacterial plaque, which is composed of soft bacterial deposits that adhere firmly to the teeth, is considered to be a complex, metabolically interconnected, highly organized bacterial system consisting of dense masses of microorganisms embedded in an intermicrobial matrix. In sufficient concentration it can disturb the host-parasite relationship and cause dental caries and periodontal disease.

Eastcott and Stallard have observed that plaque begins to form within 2 hours after the teeth are brushed.' Coccal forms of bacteria form first on a thin fenestrated pellicle (organic bacteria-free film deposited on the tooth surface). The surface is completely covered with a smooth material 3 hours after brushing. Within 5 hours, plaque microcolonies develop, apparently by cell division. Between 6 and 12 hours, the covering material becomes thinner and is reduced to discontinuous small scattered areas. About 30% of the coccus bacteria are in various stages of division by 24 hours. Rod-shaped bacteria appear for the first time in 24-hour-old plaque. Within 48 hours the surface of the plaque is covered with a mass of rods and filaments.

Dental calculus, which is considered to be calcified dental plaque, is discussed again later in the chapter. It is classified as supragingival or subgingival, depending on its location on the tooth. Supragingival calculus occurs as hard, firmly adherent masses on the enamel of teeth. Subgingival calculus is found as a concretion on the tooth in the confines of the periodontal pocket. The surface of dental calculus is always covered by uncalcified plaque. Calculus is an important factor in the development of gingival and periodontal disease.

In a study of approximately 1700 children 9 to 14 years of age by Suomi et al, it was found that a relatively high percentage of children of all racial-ethnic groups had calculus (both supragingival and subgingival).[5] From 56% to 85% of the children in the various age, sex, and racial-ethnic groups had supragingival calculus. The findings of this study indicate that most children 9 to 14 years of age who are of low socioeconomic status would benefit from inclusion in a preventive periodontal disease program based on improvement of oral hygiene.

SIMPLE GINGIVITIS

ERUPTION GINGIVITIS

A temporary type of gingivitis is often observed in young children when the primary teeth are erupting. This gingivitis, often associated with difficult eruption, subsides after the teeth emerge into the oral cavity.

Weddell and Klein conducted a study to determine the prevalence of gingivitis in a group of children between 6 and 36 months of age.' The children, patients of pediatricians in the Indianapolis area, had been born in the area, which has a fluoridated water supply. Among 299 white children, gingivitis was present in 13% of those 6 to 17 months of age, 34% of those in the 18- to 23-month age group, and 39% of those in the 24- to 36-month age group. Black children were not included in the study because of the inconsistency of their gingival colors. The gingivitis observed by Weddell and Klein was for the most part eruption gingivitis.' Nevertheless, their findings support the view that an oral hygiene program should be initiated by parents when the child is very young.

The greatest increase in the incidence of gingivitis in children is often seen in the 6- to 7-year age group when the permanent teeth begin to erupt. This increase in gingivitis apparently occurs because the gingival margin receives no protection from the coronal contour of the tooth during the early stage of active eruption, and the continual impingement of food on the gingivae causes the inflammatory process.

Food debris, materia alba, and bacterial plaque often collect around and beneath the free tissue, partially cover the crown of the erupting tooth, and cause the development of an inflammatory process (Fig. 20-1). This inflammation is most commonly associated with the eruption of the first and second permanent molars,

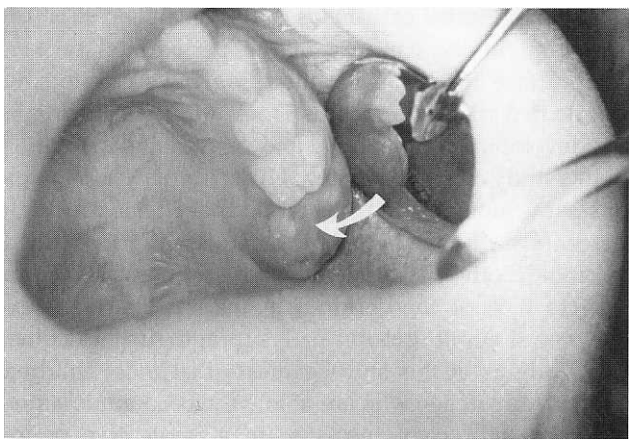


FIG **20-1.** Mild inflammation (arrow) is evident in the tissue partially covering the crown of the erupting first permanent molar.

and the condition can be painful and can develop into a pericoronitis or a pericoronal abscess. Mild eruption gingivitis requires no treatment other than improved oral hygiene. Painful pericoronitis may be helped when the area is irrigated with a counterirritant, such as Peroxyl.* Pericoronitis accompanied by swelling and lymph node involvement should be treated with antibiotic therapy.

GINGIVITIS ASSOCIATED WITH **POOR** ORAL HYGIENE

The degree of dental cleanliness and the condition of the gingival tissues in children are definitely related. Horowitz et al observed significant improvements in the gingivitis scores of schoolchildren after the initiation of a supervised daily plaque removal program.' Children in grades 5 to 8 participated in the program, and successful results were maintained during three school years. The mean gingivitis scores were reduced 40% among girls and 17% among boys during the program period, whereas the children in the control group maintained essentially the same gingivitis scores for the period of the study. Adequate mouth hygiene and cleanliness of the teeth are related to frequency of brushing and the thoroughness with which bacterial plaque is removed from the teeth. Favorable occlusion and the chewing of coarse, detergent-type foods, such as raw carrots, celery, and apples, have a beneficial effect on oral cleanliness.

In a study of 2876 children residing in a naturally fluoridated area, Murray confirmed the high prevalence of gingivitis in the young population.' He observed that inflammation of one or more papillae or margins associated with the incisor and canine teeth occurred in 90% of children 8 to 18 years of age. He, too, pointed to the importance of a good standard of oral cleanliness in reducing gingivitis and, ideally, preventing the progression of the disease in later life.

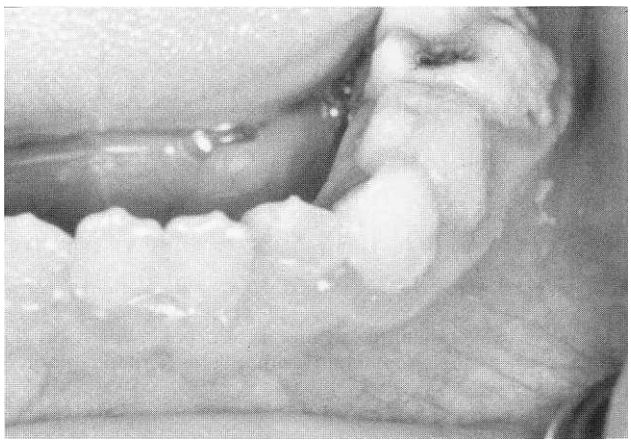
Gingivitis associated with poor oral hygiene is usually classified as early (slight), moderate, or advanced. Early gingivitis is quickly reversible and can be treated with a good oral prophylactic treatment and instruction in good toothbrushing and flossing techniques to keep the teeth free of bacterial plaque (Figs. 20-2 and 20-3). Gingivitis is generally less severe in children than in adults with similar plaque levels.

ALLERGY AND GINGIVAL INFLAMMATION

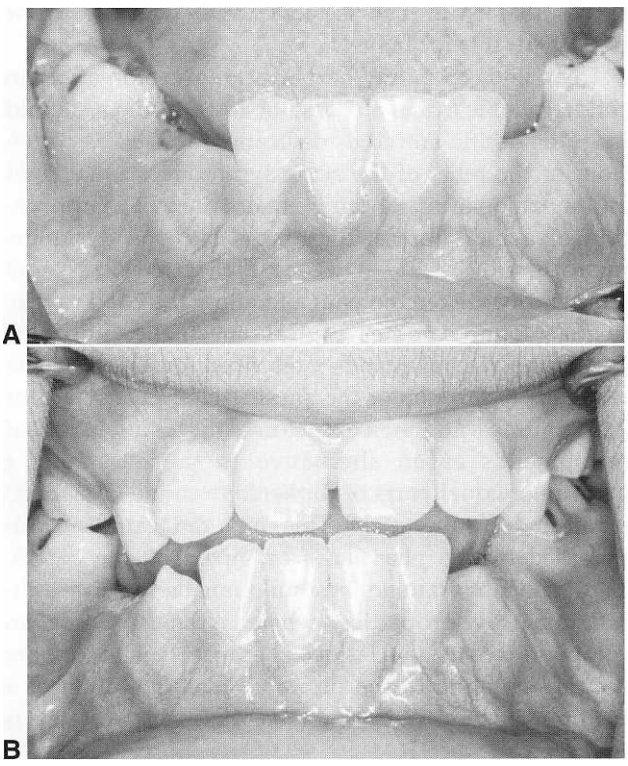
Matsson and Moller studied the degree of seasonal variation of gingival inflammation in children with allergies to birch pollen.`' Thirty-four allergic children were examined during two successive spring seasons and the one intervening fall. Age- and sex-matched

'Colgate-Palmolive Co., New York, NY.

controls were also examined in the fall. Gingival inflammation and the presence or absence of plaque were recorded, and a bleeding/plaque ratio was calculated for each subject. The results indicated an enhanced gingival inflammatory reaction in the allergic children during the pollen seasons. Although the authors acknowledge that the significance of gingival reaction during short allergic seasons is difficult to assess, they speculate that patients with complex allergies who have symptoms for longer periods may be at higher risk for more significant adverse periodontal changes.



[I fi **20-2.** Gingivitis resulting from poor oral hygiene and reduced function in the area. A painful second primary molar has interfered with normal function on this side of the mouth.



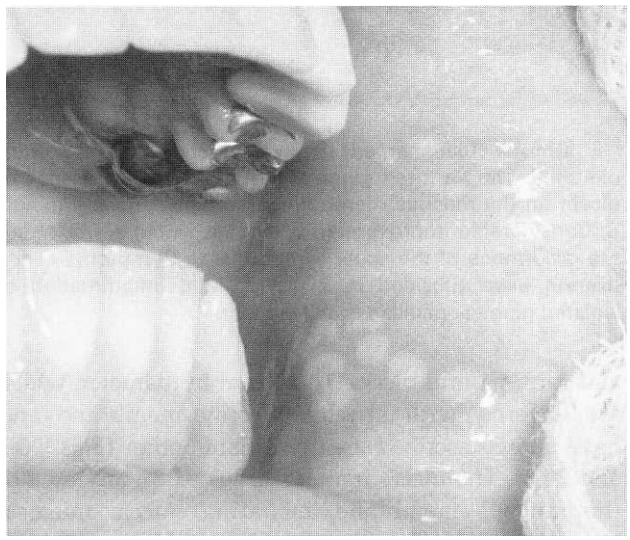
[I G **20-3.** A, Localized gingival inflammation and recession associated with minimal plaque accumulation on mandibular right central incisor. **B,** Gingival health was greatly improved after a thorough plaque removal regimen was initiated at home.

ACUTE GINGIVAL DISEASE

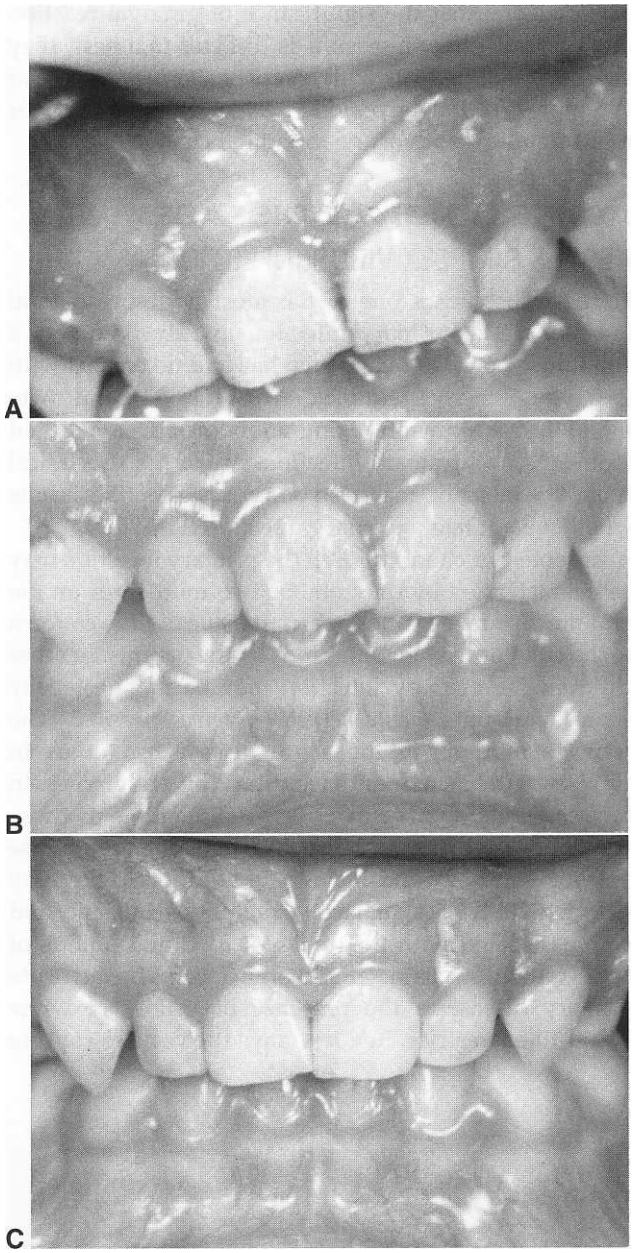
HERPES SIMPLEX VIRUS INFECTION

Herpesvirus causes one of the most widespread viral infections. The primary infection usually occurs in a child under 6 years of age who has had no contact with the type 1 herpes simplex virus (HSV-1) and who therefore has no neutralizing antibodies. It is believed that 99% of all primary infections are of the subclinical type. The infection may also occur in susceptible adults who have not had a primary infection (Fig. 20-4).

In some preschool children the primary infection may be characterized by only one or two mild sores on the oral mucous membranes, which may be of little concern to the child or may go unnoticed by the parents. h1 other children the primary infection may be manifested by acute symptoms *(acute herpetic gingivostomatitis)*. The active symptoms of the acute disease can occur in children with clean mouths and healthy oral tissues. hl fact, these children seem to be as susceptible as those with poor oral hygiene. The symptoms of the disease develop suddenly and include, in addition to the fiery red gingival tissues, malaise, irritability, headache, and pain associated with the intake of food and liquids of acid content. A characteristic oral finding in the acute primary disease is the presence of yellow or white liquid-filled vesicles. In a few days the vesicles rupture

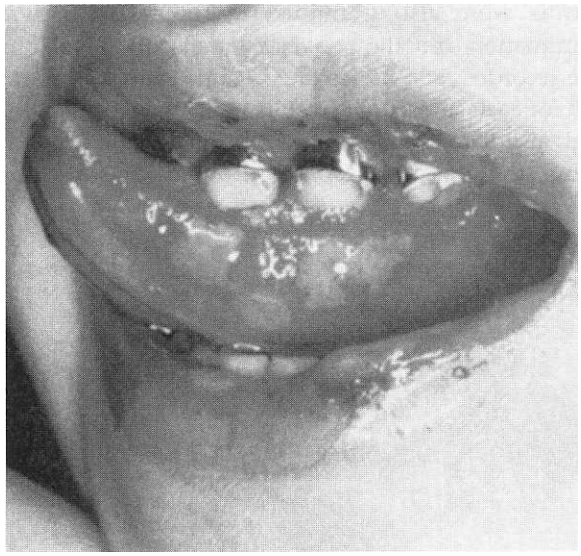


f1 **20-4.** Ulcerated stage of primary herpes in a young adult. Notice the circumscribed confluent areas of inflammation.



f **I 20-5. A**, Acute herpetic gingivostomatitis in an 18-month-old child. The fiery red gingival tissues and the presence of ulcers on the mucous membrane are characteristic findings. **B**, Considerable improvement is evident within a week after the occurrence of the acute symptoms. C, Symptoms of the disease have subsided in 2 weeks. Mild inflammation of isolated gingival papillae is still evident.

and form painful ulcers, 1 to 3 mm in diameter, which are covered with a whitish gray membrane and have a circumscribed area of inflammation (Figs. 20-5 and 20-6). The ulcers may be observed on any area of the mucous membrane, including buccal mucosa, tongue, lips, hard and soft palate, and the tonsillar areas. Large ulcerated lesions may occasionally be observed on the palate or gingival tissues or in the



f **G 20-6.** Several large, painful ulcers are evident on the tongue of a preschool child with acute herpetic gingivostomatitis.

region of the mucobuccal fold. This distribution makes the differential diagnosis more difficult. An additional diagnostic criterion is a fourfold rise of serum antibodies to HSV-1. The lesion culture will also show positive results for HSV-1.

Treatment of acute herpetic gingivostomatitis in children, which runs a course of 10 to 14 days, should include specific antiviral medication as well as provision for the relief of the acute symptoms so that fluid and nutritional intake can be maintained. The application of a mild topical anesthetic, such as dyclonine hydrochloride *(0.5%)* (Dyclone) before mealtime will temporarily relieve the pain and allow the child to take in soft food. Another topical anesthetic, lidocaine (Xylocaine Viscous), can be prescribed for the child who can hold 1 teaspoon of the anesthetic in the mouth for 2 to 3 minutes and then expectorate the solution. Schaaf recommends as an alternative to the anesthetic a mixture of equal parts of diphenhydramine (Benadryl) elixir and Kaopectate.[f0] This material can be compounded by the pharmacist or mixed by the parent. The diphenhydramine has mild analgesic and anti-inflammatory properties, whereas the kaolin-pectin compound coats the lesions. Because fruit juices are usually irritating to the ulcerated area, ingestion of a vitamin supplement during the course of the disease is indicated.

Although the treatments just described may prove useful at certain times, they are only palliative. The mainstay of definitive therapy is regular doses of specific systemic antiviral medication combined with systemic analgesics (acetaminophen or ibuprofen)

during the course of the disease." The antiviral medications currently available are acyclovir, famciclovir, and valacyclovir. These medications inhibit viral replication in cells infected with the virus. Acyclovir (Zovirax*) should be administered in 5 daily doses to equal 1000 mg per day for 10 days. Acyclovir is available in capsules or suspension. Acyclovir therapy has been successfully used in infants and children. Famciclovir (Famvir†) and valacyclovir (Valtrex§) are newer and possibly more effective antiviral agents, but their use in pediatric populations has not yet been studied. If the newer antivirals are prescribed for teenagers, the course of treatment is also 10 days but daily doses are less frequent and usually fewer milligrams daily than acyclovir. Valacyclovir should not be prescribed for immunocompromised patients.

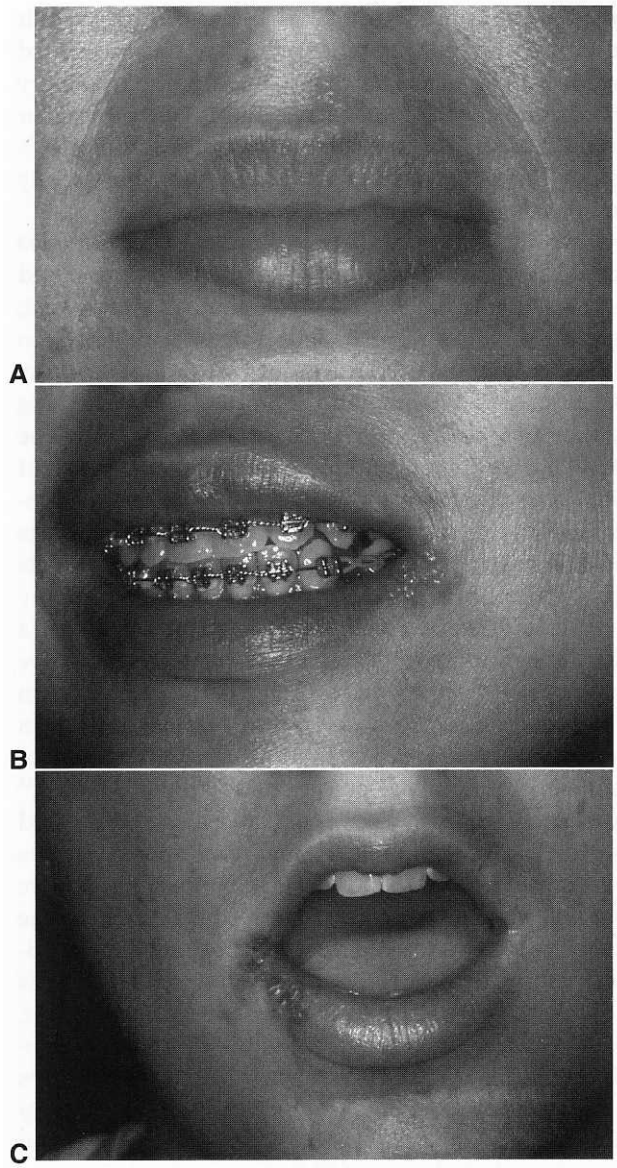
Bed rest and isolation from other children in the family are also recommended. Hale et al reported an outbreak of herpes simplex infection in a group of 13 children occupying one floor in an orphanage.[12] The children ranged in age from 11 to 35 months. In three of the children a mild fever of brief duration and small oral lesions were the only signs of infection, and these might easily have been unobserved in a situation different from that of an institutional study. The remaining children had symptoms of acute infection.

After the initial primary attack during early childhood, the herpes simplex virus becomes inactive and resides in sensory nerve ganglia. The virus will often reappear later as the familiar cold sore or fever blister, usually on the outside of the lips (Fig. 20-7). Thus the disease has been commonly referred to as *recurrent herpes labialis* (RHL). However, approximately 5% of recurrences are intraoral. With the recurring attacks, the sores develop in essentially the same area. Kleinman, Swango, and Pindborg published the results of a national survey of 39,206 schoolchildren aged 5 to 17 years.[13] A history of RHL was reported by 33% of the children.

The recurrence of the disease has often been related to conditions of emotional stress and lowered tissue resistance resulting from various types of trauma. Excessive exposure to sunlight may be responsible for the appearance of the recurrent herpetic lesions on the lip. Use of sunscreen can prevent sun-induced recurrences. Lesions on the lip may also appear after dental treatment and may be related to irritation from rubber dam material or even routine daily procedures.

The most effective treatment for these recurrences is the use of the specific systemic antiviral medications already discussed in connection with the treatment of the primary herpetic infection (acyclovir, famciclovir, and valacyclovir)." The medication should be taken immediately after the prodromal symptom of recurrence. The daily dosages are the same as those for the primary infection, but the course of treatment is usually 5 days instead of 10. One-day therapy for recurrent herpes labialis is a total of 4 g valacyclovir given in a divided dose; 2 g initially with the prodrome, followed 12 hours later with another 2 g. This regimen has been approved for children 12 years of age and older.

Another topical antiviral agent, penciclovir cream (Denavir*), may be applied to perioral lesions but



f **I G 20-7.** Recurrent herpes labialis. A, Early vesicular lesions. **B,** Mature vesicular lesion. C, Appearance of herpes labialis after rupture of vesicles and crusting of the lesion. **(A, B,** and C Courtesy Dr. Susan L. Zunt.)

*GlaxoSmithKline, Inc., Research Triangle Park, NC.
†Novartis Pharmaceuticals Corporation, East Hanover, NJ.
§GlaxoSmithKline, Inc., Research Triangle Park, NC.

*Novartis Consumer Health, Inc., Parsippany, NJ.

should not be applied to intraoral lesions. The penciclovir cream and systemic antivirals should not be prescribed for concurrent use. The cream can be applied every 2 hours while awake for 4 days, and it is approved for use in children 12 years of age and older. Topical 5% acyclovir cream may be prescribed for use 5 times daily for 4 days in children 12 years of age and older.

Other remedies for herpes simplex infection also include the amino acid lysine. The oral therapy is based on lysine's antagonistic effect on another amino acid, arginine. Griffith, Norins, and Kagan conducted an initial study in which 250 patients were given daily lysine doses of 1000 mg and were told to avoid eating arginine-rich foods, such as chocolate and nuts.[13] The lysine therapy was continued until the patients had been lesion free for 6 months. L-Lysine monohydrochloride is available commercially in capsule form or tablets containing 100 or 312 mg of L-Lysine.* The patients reported that pain disappeared overnight in virtually every instance. New vesicles failed to appear, and a majority considered the resolution of the lesions to be more rapid than in the past. There was also a reduction in frequency of occurrences. Griffith, Norins, and Kagan conclude that improper food selection may make adequate lysine intake precarious for some persons.' Ingestion of cereals, seeds, nuts, and chocolate would produce a high arginine/lysine ratio and favor the development of herpetic lesions. Similar results are obtained when arginine is added to the medium in the laboratory to induce herpes proliferation. The avoidance of these foods, coupled with the selection of foods with adequate lysine, such as dairy products and yeast, should discourage herpes infection. These authors postulate that this may explain the low incidence of herpes in infants before they are weaned from a predominantly milk diet. Prophylactic lysine is apparently useful in managing selected cases of RI IL if serum lysine is maintained at adequate concentrations.

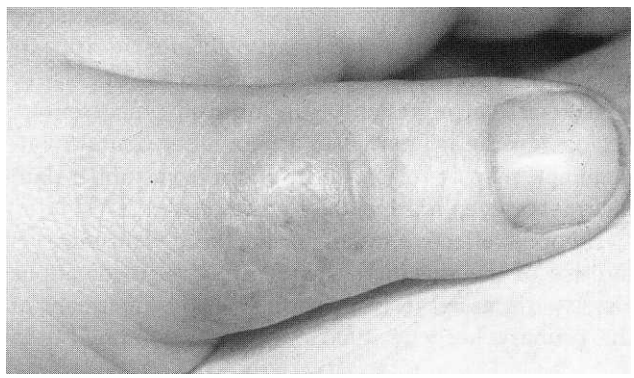
Brooks et al have reported that dentists are frequently exposed to HSV-1.[15] They evaluated the risk of infection with the virus by assessing disease experience, comparing the individual's history with the results of a complement fixation or antibody titration test or both. Their study group consisted of 525 dental students, 94 dental faculty members, and 23 staff members. Although almost all of those with a history of herpetic infection showed antibodies to HSV-1, only 57% of those lacking such a history had neutralizing antibody titers of 1:10 or higher. This finding suggests that a significant portion of practicing dentists risk primary herpetic infection. Consequently, dentists and dental auxiliaries without a history of herpetic lesions might benefit from serologic testing. Considering the occupational disability that often accompanies HSV-1 infection of the finger or eye, effective barrier protection for health professionals is important.

Primary herpetic infection has been observed on the dorsal surface of the thumb of a pediatric patient (Fig. 20-8). The child was a thumb sucker, and the acute primary infection was present in the mouth. The dorsal surface of the thumb, which rested on the lower incisor teeth, apparently became irritated, and an inoculation of the virus took place. The oral condition and the lesions on the thumb subsided in 2 weeks.

RECURRENT APHTHOUS ULCER (CANKER SORE)

The *recurrent aphthous ulcer* (RAU)—also referred to as *recurrent aphthous stomatitis* (RAS)—is a painful ulceration on the unattached mucous membrane that occurs in school-aged children and adults. The peak age for RAU is between 10 and 19 years of age. It has been reported to be the most common mucosal disorder in people of all ages and races in the world. This disorder, according to definitions adopted in the epidemiologic literature, is characterized by recurrent ulcerations on the moist mucous membranes of the mouth, in which both discrete and confluent lesions form rapidly in certain sites and feature a round to oval crateriform base, raised reddened margins, and pain. They may appear as attacks of minor or single, major or multiple, or herpetiform lesions. They may or may not be associated with ulcerative lesions elsewhere.

In the national survey reported by Kleinman, Swango, and Pindborg mentioned earlier, a history of RAU was reported by 37% of the schoolchildren." Ship et al reported the prevalence estimates of RAU to range between 2% to 50% with most estimates between 5% and 25% (among medical and dental students, estimated prevalence is between 50% and 60%).[6] Lesions persist for 4 to 12 days and heal uneventfully, leaving scars only rarely and only in cases of unusually large

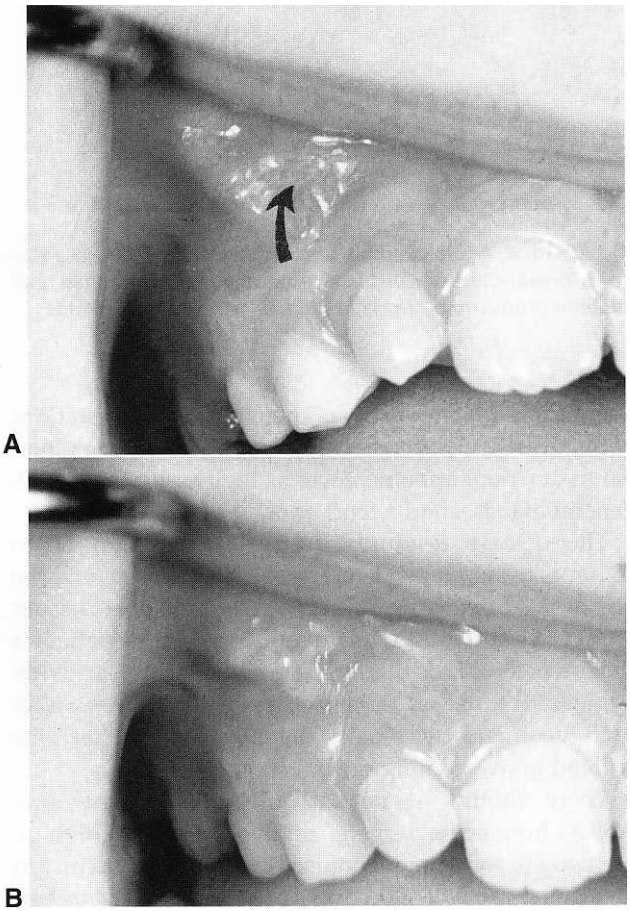


II G **20-8.** Primary herpetic infection involving the dorsal surface of the thumb of a 3-year-old child. An acute primary infection was present in the mouth.

*General Nutrition Corp., Pittsburgh, Pa.

lesions. The description of RAU frequently includes the term *canker sores* (Fig. 20-9). The major form (RAS) is less common and has been referred to as periadenitis mucosa necrotica recurrens and Sutton disease. RAS has been associated with other systemic diseases: PFAPA (periodic fever, aphthous stomatitis, pharyngitis, adenitis), Behcet disease, Crohn disease, ulcerative colitis, celiac disease, neutropenia, immunodeficiency syndromes, Reiter syndrome, systemic lupus erythematosus, and MAGIC (mouth and genital ulcers with inflamed cartilage) syndrome.

The cause of RAU is unknown. Local and systemic conditions and genetic, immunologic, and infectious microbial factors have been identified as potential causes. The condition may be caused by a delayed hypersensitivity to the L form of *Streptococcus sanguis,* which is a common constituent of the normal oral microbiota of humans. It is also possible that the lesions are caused by an autoimmune reaction of the oral epithelium. Epidemiologic studies by Ship et al have provided evidence for this hypothesis.'" These data indicate that both RHL and RAU may be produced by the same mechanism, despite the known infectious agent of RHL and the absence of any known virus for RAU. Graykowski, Barile, and Stanley have isolated the L form of bacteria from patients with aphthous lesions." Cultures of blood from patients with lesions in the acute stage tested positive for the organisms, whereas blood examined during quiescence was sterile.



[I G **20-9. A,** Evidence of the development of a recurrent aphthous ulcer in the mucobuccal fold above the primary canine. An area of inflammation and vesicle formation is apparent *(arrow).* **B,** Five days later the lesion is a well-developed ulcer with a circumscribed area of inflammation.

Local factors include trauma, allergy to toothpaste constituents (sodium lauryl sulfate), and salivary gland dysfunction. In a review of the clinical problem, Antoon and Miller suggested that minor trauma is a common precipitating factor accounting for as many as 75% of the episodes." Injuries caused by cheek biting and minor facial irritations are probably the most common precipitating factors. Nutritional deficiencies are found in 20% of persons with aphthous ulcers. The clinically detectable deficiencies include deficiencies of iron, vitamin B_{72}, and folic acid. While screening patients with aphthous ulcers, Wray et al observed a history of unusually high incidence of gastrointestinal disorders.[19] Stress may prove to be an important precipitating factor, particularly in stress-prone groups, such as students in professional schools and military personnel.

According to Greenspan et al, either nonspecific factors (trauma, food allergy) or specific factors (bacterial or viral infection) may trigger a temporary imbalance in various cell subpopulations.'-0 This imbalance could then upset immune regulation and result in local destruction of the oral epithelium and thus ulceration. Ship et al also suggested herpes simplex virus, human herpesvirus type 6, cytomegalovirus, Epstein-Barr virus, and varicella-zoster virus as possible causes of RAS."'

Current treatment is focused on promoting ulcer healing, reducing ulcer duration and patient pain, maintaining the patient's nutritional intake, and preventing or reducing the frequency of recurrence of the disease.

A variety of treatments have been recommended for RAU, but a completely successful therapy has not been found. Topical antiinflammatory and analgesic medicines and/or systemic immunomodulating and immunosuppression agents have been used for RAU. The primary line of treatment uses topical gels, creams, and ointments as antiinflammatory agents. Currently, a topical corticosteroid (e.g., 0.5% fluocinonide, 0.025% triamcinolone, 0.5% clobetasol) is applied to the area with a mucosal adherent (e.g., isobutyl cyanoacrylate, Orabase). For example, the application of triamcinolone acetonide (Kenalog in Orabase) to the surface of the lesions before meals and before sleeping may also be helpful. Binnie et al report that an antiinflammatory and antiallergic medication in the form of a topical paste is effective in reducing pain and accelerating healing of RAU ulcers.`'

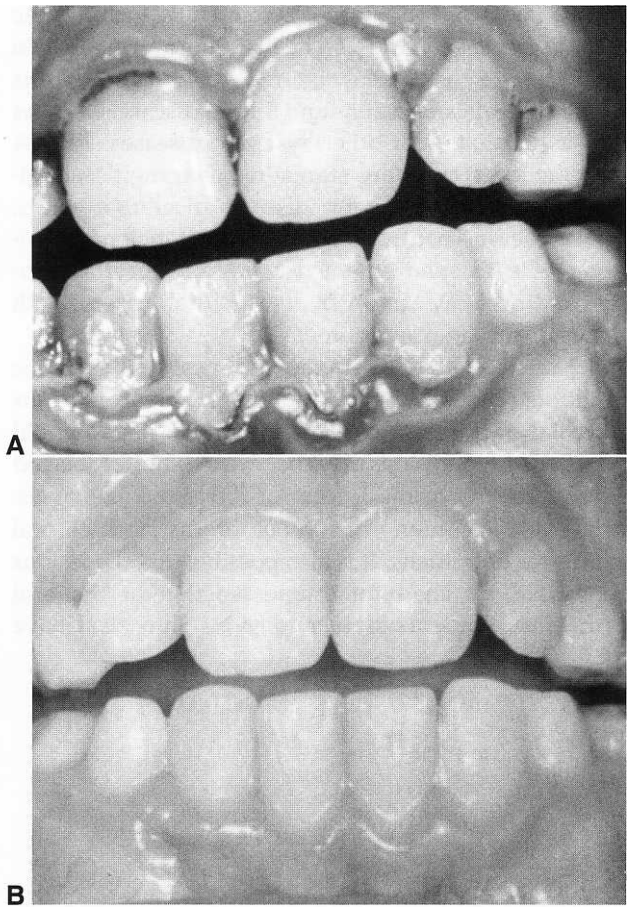
The active ingredient in the paste is 5% amlexanox and it is available under the trade name Aphthasol.* The paste is applied to the ulcer four times daily, after meals and at bedtime, until the ulcer heals. Zilactin,' a topical paste with hydroxypropyl cellulose film, has also been used to adhere to the mucosa and cover the ulcer while providing pain relief for an extended period of time. Occlusive topical 2-otylcyanoacrylate adheres for 6 hours. Aloe vera freeze-dried gel extract adheres and forms an occlusive protective patch. In severe cases, oral prednisone has been prescribed.

Topical rinses have also been helpful for relief of RAU. Sucralfate has proved useful by coating the area. The topical application of tetracyclines to the ulcers is often helpful in reducing the pain and in shortening the course of the disease. A mouthwash containing suspension of one of the tetracyclines has been helpful to some, but the mouthwash should not be swallowed. Chlorhexidine mouthwash has also been known to alleviate the symptoms of RAU. Swished dexamethasone elixir is useful to treat ulcerations in areas of the mouth that are difficult to access. Other findings that may be of significance include those of the two following studies: Meiller et al have reported that the duration and severity of RAU lesions can be significantly reduced by vigorous twice-daily rinsing with an antimicrobial mouthwash.[t22] Pedersen reported that some of her recent work suggests that varicella-zoster virus may be of etiologic importance in RAU.'- Furthermore, she found that six of eight patients with chronic severe RAU who were treated with acyclovir responded favorably within 2 days.

ACUTE NECROTIZING ULCERATIVE GINGIVITIS (VINCENT INFECTION)

The infectious disease commonly referred to as *acute necrotizing ulcerative gingivitis (ANUG) is* rare among preschool children in the United States, occurs occasionally in children 6 to 12 years old, and is common in young adults. Reade has reported one instance of the typical picture of ulcerative gingivitis in a 15-month-old infant.[24] *Treponema vincentii* and gram-negative fusiform bacilli were demonstrated in a smear. Recovery occurred within 36 hours after initiation of penicillin therapy and the application of hydrogen peroxide.

ANUG can be easily diagnosed because of the involvement of the interproximal papillae and the presence of a pseudomembranous necrotic covering of the marginal tissue (Fig. 20-10). Two microorganisms, *Borrelia vincentii* and fusiform bacilli, referred to as *spirochetal organisms,* are generally believed to be responsible for the disease. The clinical manifestations of the disease include inflamed, painful, bleeding gingival tissue, poor appetite, fever as high as 40° C (104° F), general malaise, and a fetid odor.

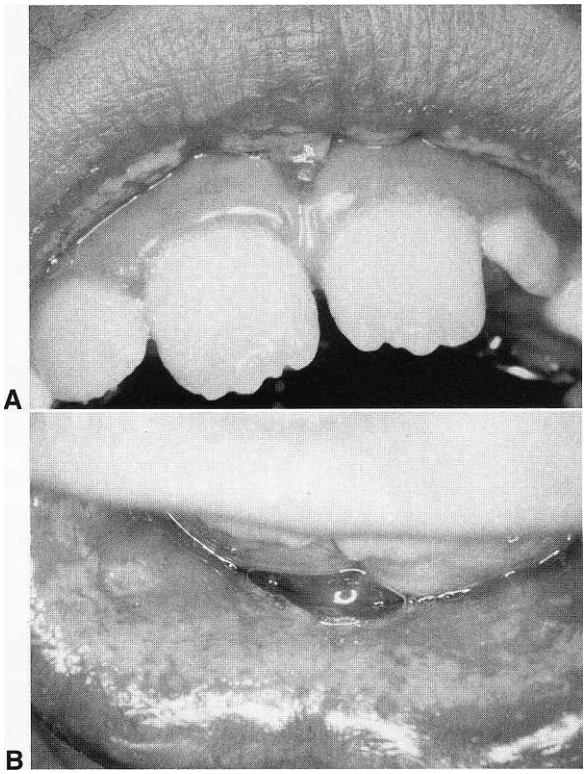


f **I G 20-10. A,** A rare example of necrotizing ulcerative gingivitis in an 8-year-old boy. **B,** Local treatment and improved oral hygiene produced a dramatic recovery from the infection.

The disease responds dramatically within 24 to 48 hours to subgingival curettage, debridement, and the use of mild oxidizing solutions. If the gingival tissues are acutely and extensively inflamed when the patient is first seen, antibiotic therapy is indicated. Improved oral hygiene, the use of mild oxidizing mouthrinses after each meal, and twice-daily rinsing with chlorhexidine will aid in overcoming the infection.

There should be no difficulty in distinguishing ANUG from acute herpetic gingivostomatitis, although the two are sometimes confused. Round ulcers with red areolae on the lips and cheeks are characteristic of herpetic gingivostomatitis. Therapeutic prophylaxis and debridement will bring about a favorable response in cases of ANUG but not in acute herpetic gingivostomatitis. A therapeutic trial of antibiotics will reduce the acute symptoms in ANUG but not in the viral infection.

*Access Pharmaceuticals, Dallas, Texas.
'Zila Pharmaceuticals, Phoenix, Ariz.
[tListerine Antiseptic, Pfizer Warner Lambert Division, Morris Plains, NJ.



F **I G 20-11.** The characteristic appearance of acute candidiasis on the upper and lower lips of a young patient. The hard and soft palates were also severely affected. The infection was controlled with a 7-day regimen of nystatin.

Acute herpetic gingivostomatitis is most frequently seen in preschool children, and its onset is rapid. As stated earlier, ANUG rarely occurs in the preschool-aged group and develops over a longer period, usually in a mouth in which irritants and poor oral hygiene are present. On the other hand, acute oral infections initially diagnosed as ANUG have frequently been found later to be an oral manifestation of one of the xanthomatoses. The early stages of conditions such as Hand-Schiiller-Christian disease and Letterer-Siwe disease are associated with many of the symptoms of ANUG.

ACUTE CANDIDIASIS (THRUSH, CANDIDOSIS, MONILIASIS)

Candida (Monilia) albicans is a common inhabitant of the oral cavity but may multiply rapidly and cause a pathogenic state when tissue resistance is lowered. Young children sometimes develop thrush after local antibiotic therapy, which allows the fungus to proliferate. The lesions of the oral disease appear as raised, furry, white patches, which can be removed easily to produce a bleeding underlying surface (Fig. 20-11). Neonatal candidiasis, contracted during passage through the vagina and erupting clinically during the first 2 weeks of life, is a common occurrence. This infection is also common in immunosuppressed patients and is discussed in Chapter 24.

Antifungal antibiotics are available to control thrush. For infants and very young children, a suspension of 1 ml (100,000 U) of nystatin (Mycostatin) may be dropped into the mouth for local action four times a day. The drug is nonirritating and nontoxic. Clotrimazole sus-pension (10 mg/ml), 1 to 2 ml applied to affected areas four times daily, is an effective antifungal medication. Systemic fluconazole suspension (10 mg/ml) is safe to use in infants at a total dosage of 6 mg/kg or less per day. For children old enough to manage solid medication allowed to dissolve in the mouth, clotrimazole troches or nystatin pastilles are recommended, because the therapeutic agent remains in the saliva longer than with the liquid medication. For children old enough to swallow, systemic fluconazole (100-mg tablets) in a 14-day course may be prescribed for patients whose infection has not responded to topical antifungal agents.

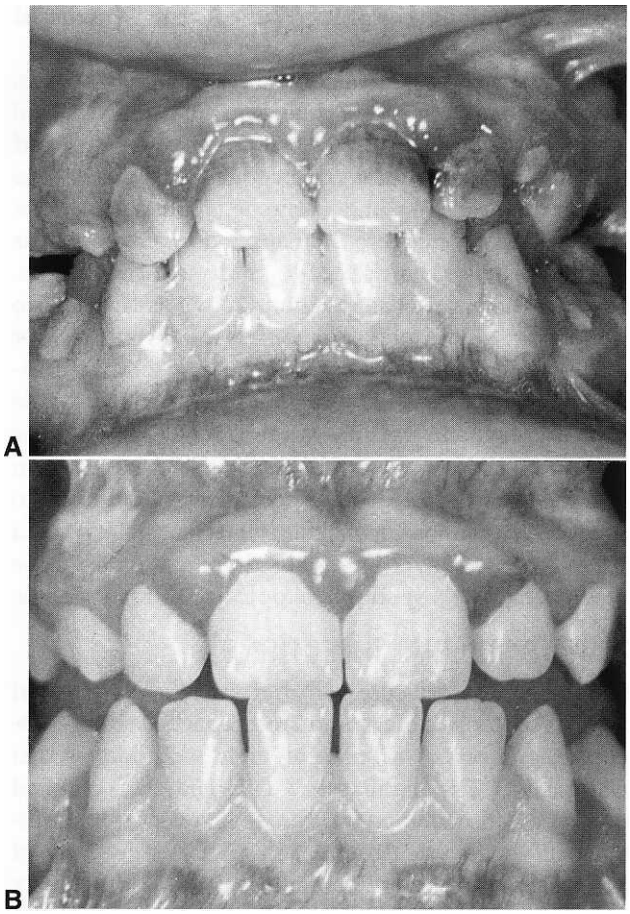
ACUTE BACTERIAL INFECTIONS

The prevalence of acute bacterial infection in the oral cavity is unknown. Blake and Trott reported acute streptococcal gingivitis with painful, vivid red gingivae that bled easily2[5] The papillae had enlarged, and gingival abscesses had developed. Cultures showed a predominance of hemolytic streptococci. Acute infections of this type may be more common than was previously realized. Littner et al reported five cases, all in adults between 20 and 27 years of age.- The diagnosis is difficult to make, however, without extensive laboratory tests. Broad-spectrum antibiotics are recommended if the infection is believed to be bacterial in origin. Improved oral hygiene is important in treating the infection. As with any acute microbial oral infection, chlorhexidine mouthrinses are also appropriate. The placement of dental restorations to restore adequate function and contour after the reduction of acute symptoms is equally important.

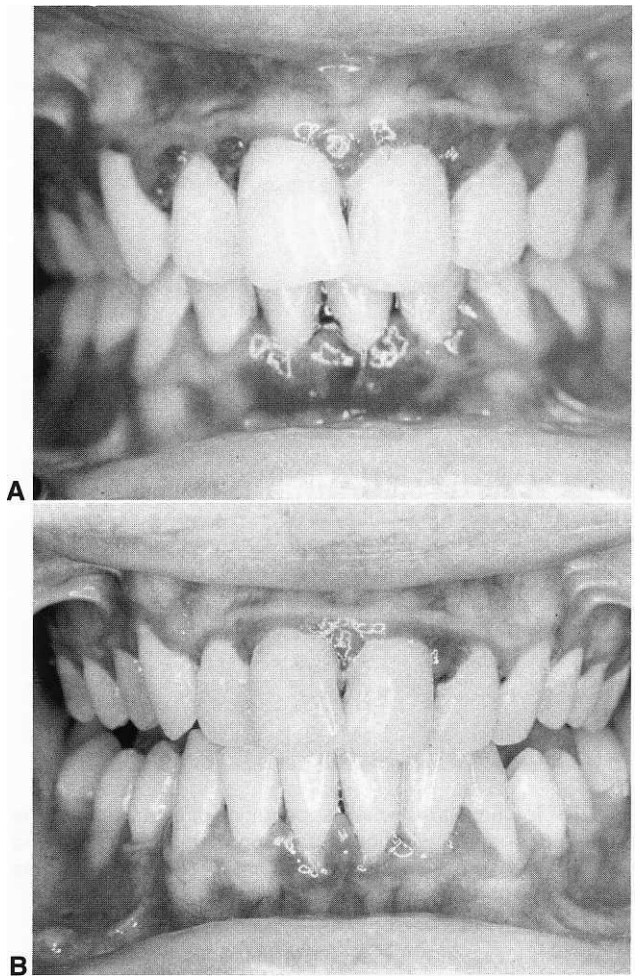
CHRONIC NONSPECIFIC GINGIVITIS

A type of gingivitis commonly seen during the preteenage and teenage years is often referred to as *chronic nonspecific gingivitis.* The chronic gingival inflammation may be localized to the anterior region, or it may be more generalized. Although the condition is rarely painful, it may persist for long periods without much improvement (Fig. 20-12).

Glauser et al have observed an unusual gingivitis in Navajo Indians between 12 and 18 years of age, similar to that seen in Fig. 20-13, in which the fiery red gingival lesion is not accompanied by enlarged interdental labial papillae or closely associated with local irritants." The gingivitis showed little improvement after a



II G **20-12. A,** Chronic nonspecific gingivitis. The cause of this type of gingivitis is complex, and it often persists for prolonged periods without significant improvement. **B,** After 9 months of treatment, hyperplastic gingival tissue is still evident in the anterior maxillary region.



f **I G 20-13. A,** Fiery red gingival lesions essentially limited to the anterior labial tissues. Only minimum local deposits were evident. The gingivitis was classified as the chronic nonspecific type. **B,** Limited improvement was evident after 6 months of local treatment. A hormonal imbalance and vitamin deficiency were suspected as contributing etiologic factors.

prophylactic treatment. The age of the patients involved and the prevalence of the disease in girls suggested a hormonal imbalance as a possible factor. Histologic examination of tissue sections and the use of special stains ruled out a bacterial infection. Inadequate oral hygiene, which allows food impaction and the accumulation of materia alba and bacterial plaque, is undoubtedly the major cause of this chronic type of gingivitis.

Kaslick, West, and Chasens studied 238 young adults and looked for a correlation between their periodontal status and their ABO blood type." They discovered that the chronic gingivitis group had a larger percentage of AB blood types and a smaller percentage of 0 blood type than the control group.

The cause of gingivitis is complex and is considered to be based on a multitude of local and systemic factors. Because dietary inadequacies are often found in the preteenage and teenage groups, the 7-day diet survey described in Chapter 10 is an important diagnostic aid. Insufficient quantities of fruits and vegetables in the diet, leading to a subclinical vitamin deficiency, may be an important predisposing factor. An improved dietary intake of vitamins and the use of multiple-vitamin supplements will improve the gingival condition in many children.

Malocclusion, which prevents adequate function, and crowded teeth, which make oral hygiene and plaque removal more difficult, are also important predisposing factors in gingivitis. Carious lesions with irritating sharp margins, as well as faulty restorations with overhanging margins (both of which cause food accumulation), also favor the development of the chronic type of gingivitis.

A wide variety of local irritants can produce a hyperplastic type of gingivitis in children and young adults. The irritation to the gingival tissue produced by mouth breathing is often responsible for the development of

the chronic hyperplastic form of gingivitis, particularly in the maxillary arch. All these factors should be considered contributory to chronic nonspecific gingivitis and should be corrected in the treatment of the condition. The importance of thorough daily oral hygiene must be emphasized repeatedly to the patients.

CHLORHEXIDINE AS A THERAPEUTIC PLAQUE CONTROL AGENT

Chlorhexidine (CH) is a chlorophenyl biguanide with broad antimicrobial activity. It has been used commonly as an antiseptic skin and wound cleanser for presurgical preparation of the patient and as a hand wash and surgical scrub for health care personnel. It has also been added as a preservative to ophthalmic products and has been used internally in very dilute concentrations in the peritoneal cavity and urinary bladder.

In dentistry, CH has been studied for control of smooth surface caries, for use as a denture disinfectant, and as a plaque control agent. Its use in controlling dental plaque accumulations has received the most attention in dental research. Mouthrinses containing CH have been very popular as therapeutic agents in several countries for some time, and in 1986 CH was approved for use in the United States. As of this writing, two products under the trade names Peridex* and PerioGard[t] have received Food and Drug Administration approval as prescription agents. This mouthrinse contains 0.12% CH gluconate as the active ingredient.

Widespread use of CH mouthrinses over many years, especially in Europe, has had an excellent safety record. Few adverse side effects have been reported with CH mouthrinses, but their use has been linked to mouth dryness and burning sensations in some persons. Poorly defined desquamative lesions have been observed in others after the mouthrinse was used. Allergic reactions to CH are rare. If the rinse is inadvertently swallowed, it has essentially little systemic effect due to poor absorption in the gastrointestinal system.

Loe and Schiott reported highly significant inhibition of plaque formation and the prevention of gingivitis with use of an aqueous solution of 0.2% CH digluconate as a mouthrinse twice daily with swishing for 1 minute.[29] Studies by Grossman et al compared the 0.12% CH gluconate mouthrinse with a placebo over 3 and 6 months." The study included 430 subjects who rinsed twice daily for 30 seconds with 15 ml of mouthrinse solution. At both the 3-month and 6-month evaluations, the CH group had significantly less gingivitis, gingival bleeding, and plaque accumulation. However, the accumulation of dental calculus and extrinsic dental stain increased in the CH group. Yankell et al have shown that dental stain from CH mouthrinse can be significantly reduced with regular use of a tartar-control dentifrice."

It is important to recognize that the beneficial use of CH as a therapeutic mouthrinse should be considered adjunctive to the practice of sound conventional plaque control measures as presented in Chapter 11 and elsewhere in this text. Its adjunctive use would also seem most appropriate for therapy in cases in which attaining adequate plaque control is more difficult, such as during illness or convalescence after serious injuries.

*Zila Pharmaceuticals, Phoenix, Ariz. '
Colgate-Palmolive Co., New York, NY.

GINGIVAL DISEASES MODIFIED BY SYSTEMIC FACTORS

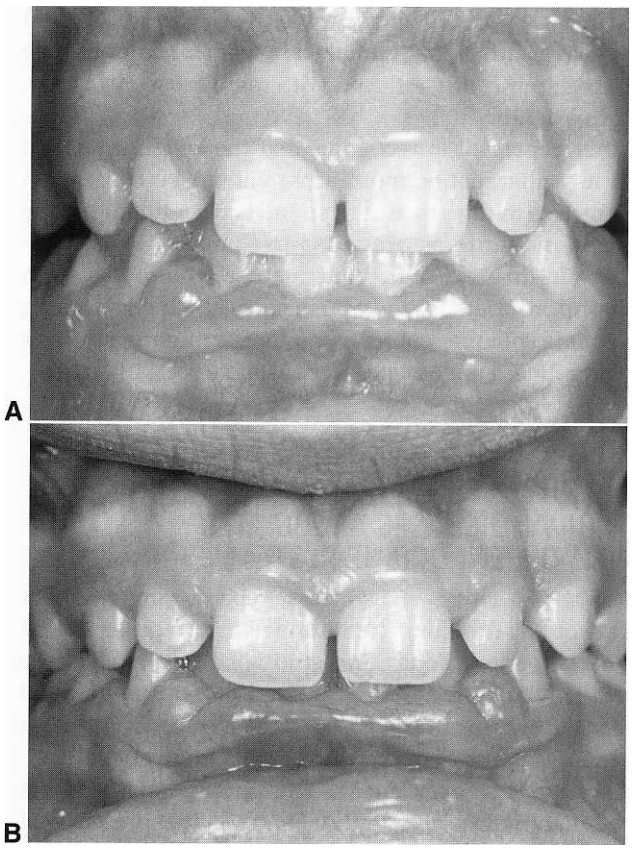
GINGIVAL DISEASES ASSOCIATED WITH THE ENDOCRINE SYSTEM

Puberty *gingivitis* is a distinctive type of gingivitis that occasionally develops in children in the prepubertal and pubertal period. Cohen, in a study of 270 boys and girls in the 11- to 14-year age group, observed that gingival enlargement in the anterior segment occurred with regularity in the prepubertal and premenarcheal period, as well as in pubescence.[32] The gingival enlargement was marginal in distribution and, in the presence of local irritants, was characterized by prominent bulbous interproximal papillae far greater than gingival enlargements associated with local factors.

Sutcliffe's survey of a group of children between 11 and 17 years of age revealed an initial high prevalence of gingivitis that tended to decline with age.[33] In both sexes the prevalence of gingivitis tended to decrease with age. Initially, 89% of 11-year-olds and 92% of 12-yearolds were affected. It should be recalled, however, that with increasing age there is increased evidence of more adequate brushing. Girls tended to reach their maximum gingivitis experience earlier than boys did.

The enlargement of the gingival tissues in puberty gingivitis is confined to the anterior segment and may be present in only one arch. The lingual gingival tissue generally remains unaffected (Fig. 20-14). Treatment of puberty gingivitis should be directed toward improved oral hygiene, removal of all local irritants, restoration of carious teeth, and dietary changes necessary to ensure an adequate nutritional status. Cohen observed a sharp improvement in gingival inflammation and enlargement after the oral administration of 500 mg of ascorbic acid.[3] " However, the improvement did not occur until the vitamin had been taken for approximately 4 weeks.

Severe cases of hyperplastic gingivitis that do not respond to local or systemic therapy should be treated by gingivoplasty. Surgical removal of the thickened fibrotic marginal and interproximal tissue has been

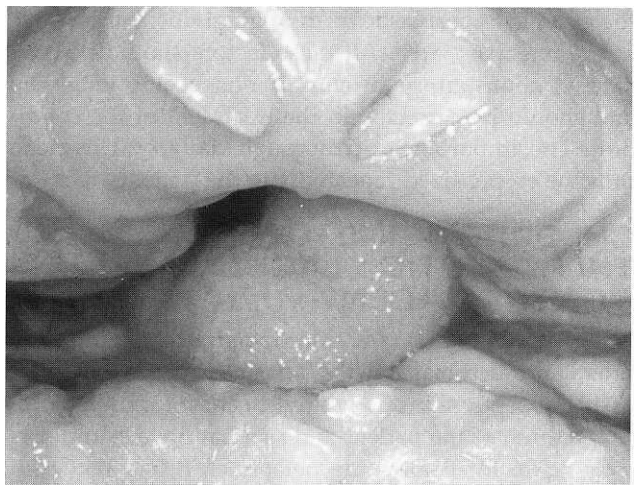


Ff G· **20-14. A,** Puberty gingivitis. The enlargement of the gingival tissues was limited to the mandibular anterior region. **B,** Local treatment resulted in only a slight improvement of the condition. A persistence of the hyperplastic enlargement would indicate the need for gingivoplasty.

found effective. Recurrence of any hyperplastic tissue will be minimal if adequate oral hygiene is maintained.

GINGIVAL LESIONS OF GENETIC ORIGIN

Hereditary gingival fibromatosis (HGF) is characterized by a slow, progressive, benign enlargement of the gingivae. Genetic and pharmacologically induced forms of gingival enlargement are known. The most common genetic form, HGF, usually has an autosomal dominant mode of inheritance. Hart et al have identified the first polymorphic marker for HGF phenotype on chromosome band 2p21." This rare type of gingivitis has been referred to as *elephantiasis gingivae* or *hereditary hyperplasia of the gums.* The gingival tissues appear normal at birth but begin to enlarge with the eruption of the primary teeth. Although mild cases are observed, the gingival tissues usually continue to enlarge with eruption of the permanent teeth until the tissues essentially cover the clinical crowns of the teeth (Fig. 20-15). The dense fibrous tissue often causes displacement of the teeth and malocclusion. The condition is not painful until the tissue enlarges to the extent that it partially covers the occlusal



I **i i 20-15.** Fibromatosis in a 14-year-old girl. The gingival tissues have enlarged to the extent that they almost cover the crowns of the teeth.

surface of the molars and becomes traumatized during mastication.

Zackin and Weisberger described fibromatosis histologically as a moderate hyperplasia of the epithelium, with hyperkeratosis and elongation of the rete pegs.[3"] The increase in tissue mass is primarily the result of an increase and thickening of the collagenous bundles in the connective tissue stroma. The tissue shows a high degree of differentiation, and a few young fibroblasts are present.

Surgical removal of the hyperplastic tissue achieves a more favorable oral and facial appearance. However, hyperplasia can recur within a few months after the surgical procedure and can return to the original condition within a few years. Although the tissue usually appears pale and firm, the surgical procedure is accompanied by excessive hemorrhage. Therefore quadrant surgery is usually recommended. Brown et al have reported a case in which apically positioned flap surgery and CO_2 laser evaporation were used to reduce the gingival tissue." The importance of excellent plaque control should be stressed to the patient because this delays the recurrence of the gingival overgrowth.

PHENYTOIN-INDUCED GINGIVAL OVERGROWTH

Phenytoin (Dilantin, or diphenylhydantoin), a major anticonvulsant agent used in the treatment of epilepsy, was first introduced in 1938 by Merrit and Putnam.[3b] Varying degrees of gingival hyperplasia, one of the most common side effects of phenytoin therapy, were first described by Kimball in 1939.[39] He reported that 57% of 119 patients taking phenytoin for the control of seizure activity experienced some degree of gingival overgrowth.

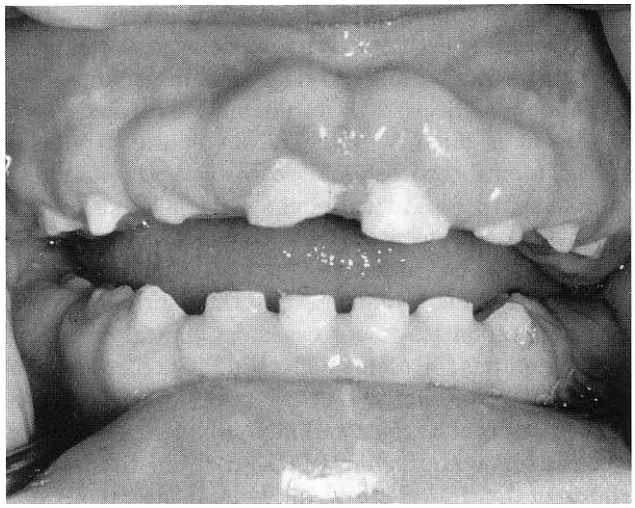
Today an estimated 2 million persons use this medication because of its comparative safety and broad therapeutic range. The incidence of phenytoin-induced gingival overgrowth (PIGO) in patients undergoing long-term phenytoin therapy has been reported as ranging between 0% and 95%, with numerous investigators reporting figures at the 40% to 50% level.

Early research showed an increase in the number of fibroblasts in patients receiving Dilantin; thus the condition was termed *Dilantin hyperplasia.* Now the term *phenytoin-induced gingival overgrowth is* preferred because Hassell et al found true hyperplasia not to exist." There was no excessive collagen accumulation per unit of tissue, nor did the fibroblasts appear abnormal in number or size.

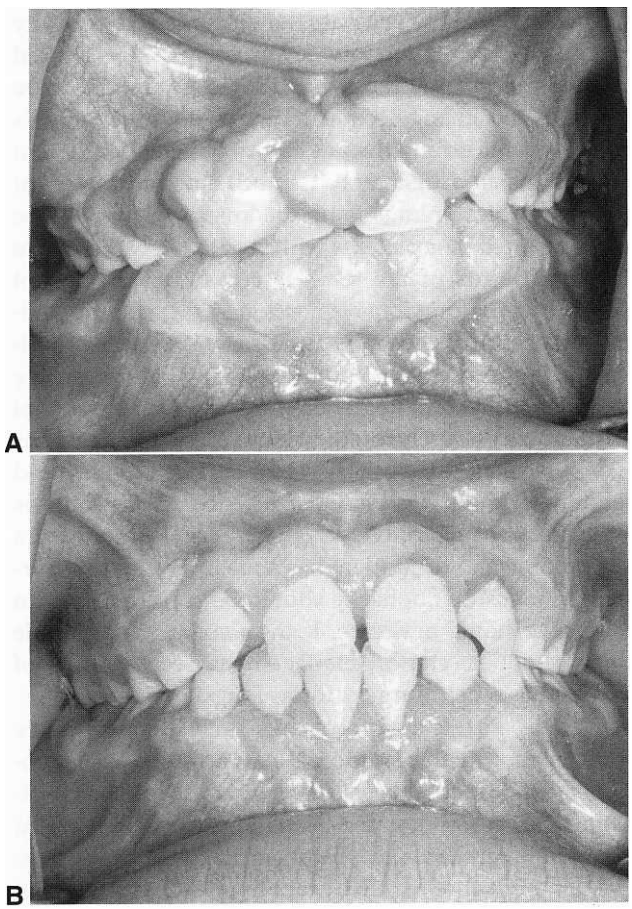
The influence of serum and salivary levels of phenytoin on the development of PIGO has also been investigated. Some authors report a positive relationship between the level of phenytoin in serum and saliva and the severity of PILO in some cases.[4143] Other investigators have reported that no such correlation exists.` ' I t is generally agreed that a relationship exists between dosage and PIGO when the level of phenytoin per unit body weight or actual serum level is considered. Sasaki and Maita reported a significant correlation between the degree of gingival overgrowth and a high level of basic fibroblast growth factor in serum.[4H]No such correlations were observed for patient age, daily or total phenytoin dose, duration of therapy, or serum phenytoin level.

Most investigators agree on the existence of a close relationship between oral hygiene and PIGO. PIGO can be decreased or prevented by scrupulous oral hygiene and dental prophylaxis. The relationship between plaque, local irritants, and PIGO is also supported by the observation that patients without teeth almost never develop PIGO.

PIGO, when it does develop, begins to appear as early as 2 to 3 weeks after initiation of phenytoin therapy and peaks at 18 to 24 months. The initial clinical appearance is painless enlargement of the interproximal gingiva. The buccal and anterior segments are more often affected than the lingual and posterior segments (Figs. 20-16 and 20-17). The affected areas are isolated at first but can become more generalized later. Unless secondary infection or inflammation is present, the gingiva appears pink and firm and does not bleed easily on probing. As the interdental lobulations grow, clefting becomes apparent at the midline of the tooth (see Fig. 20-17). With time the lobulations coalesce at the midline, forming pseudopockets and covering more of the crown of the tooth. The epithelial attachment level usually remains constant. In some cases, the entire occlusal surface of the teeth becomes covered. These lesions may remain purely fibrotic in nature or may be combined with a noticeable inflammatory component.



GIG **20-16.** Phenytoin gingivitis in a preschool child. The enlarged gingival tissue covers two thirds of the maxillary primary lateral incisors and canines.



GIG. **20-17. A,** Phenytoin gingivitis of the severe generalized type. **B,** Surgical removal of the overgrowth of hyperplastic gingival tissue results in temporary improvement of the condition. Excellent oral hygiene is essential in controlling the gingival enlargement.

PIGO may impose problems of esthetics, difficulty in mastication, speech impairment, delayed tooth eruption, tissue trauma, and secondary inflammation leading to periodontal disease. Unfortunately, no cure exists and treatment is often symptomatic in nature. Antihistamines, topical corticosteroids, ascorbic acid (vitamin C) supplements, topical antibiotics, and alkaline mouthwashes have been used with limited success and are considered to be ineffective.

Steinberg and Steinberg describe current recommended dental treatment based on clinical oral signs and symptoms." Patients with mild PIGO (i.e., less than one third of the clinical crown is covered) require daily meticulous oral hygiene and more frequent dental care. For patients with moderate PIGO (i.e., one third to two thirds of the clinical crown is covered) meticulous oral home care and the judicious use of an irrigating device may be needed. Use of an antiplaque mouthrinse (0.12% chlorhexidine gluconate) in the device will further help control bacterial growth. Initially, a series of four consecutive weekly office visits for prophylaxis and topical stannous fluoride application is recommended. The fifth week is used to evaluate the gingivae and note any change in size. Phenytoin levels should be checked (normal therapeutic range is 10 to 15 gg/ml). If there has been no change, consultation with the patient's physician concerning the possibility of using a different anticonvulsant drug may be helpful. If no improvement occurs, surgical removal of the overgrowth may be recommended. For patients with severe PIGO (i.e., more than two thirds of the tooth is covered) who do not respond to the previously mentioned therapeutic regimens, surgical removal is necessary. As in any periodontal surgery, scaling and root planing before surgery and meticulous oral hygiene after surgery are essential to minimize the overgrowth, which can occur as early as 3 to 4 weeks after surgery. Donnenfeld, Stanley, and Bagdonoff found no PIGO recurrence for as long as 9 months postoperatively.[\$] If surgery is required a second time and the patient has a history of rapid recurrence, a pressure appliance should be considered as an adjunct to home oral care. If the patient has multiple gingivectomies, clinical management becomes one of postponing repeat surgery for as long as possible.

Specific surgical approaches for PIGO include gingivectomy with periodontal knives, laser, or electrosurgery, and internal bevel flap surgery. The use of periodontal knives allows the tissue to heal more quickly, but more operative and postoperative bleeding occurs, and the procedure requires more time and patient cooperation. Electrosurgery is less time consuming, decreases blood loss, improves visibility, allows superior control for areas of limited access, is self-sterilizing, and does not always require the use of periodontal packs. Disadvantages include its contraindication in patients with cardiac pacemakers, unpleasant odor, delayed healing, and the potential for error in application that results in undesired bone or tissue loss. Advantages of the surgical laser include lack of hemorrhage, which yields a dry field; noncontact during surgery; sterilization of the surgical area; prompt healing; minimal postoperative discomfort; and minimal time spent in performing the procedures. Disadvantages include cost and size of equipment, necessity for hospitalization, potential for delayed healing of some tissues, greater degree of expertise required, loss of tactile feedback, requirement for eye protection, ability of the laser to ignite a plastic or rubber endo tracheal tube, and a need to cover the nonsurgical field with moist sponge shields to prevent penetration of the laser beam. Internal bevel flap surgery can also be used for PIGO patients and has some advantages over the previously mentioned treatment modalities. The internal bevel flap promotes faster healing (stimulates primary healing), controls postoperative bleeding, minimizes postoperative pain, and allows the optional use of a periodontal pack. The choice of surgical approach must be left to the operator and is based on patient cooperation and compliance, the degree of gingival overgrowth, and opera-tor expertise. Healing is usually rapid with all these modalities.

The surgical removal of severely overgrown tissue in PIGO and good oral hygiene after surgery are generally considered to be the most effective treatment. However, even these procedures have often been followed by a gradual recurrence of the fibrous tissue.

A preliminary study of use of a pressure appliance for PIGO hyperplasia has been reported by Davis, Baer, and Palmer." Immediately after the surgical removal of hyperplastic tissue, an impression was taken and a positive-pressure splint was constructed. Periodontal dressings were removed at the end of 1 week, and the positive-pressure appliance was inserted. Seven of the nine members of the experimental group had no recurrence of gingival hyperplasia, one had a slight recurrence, and one had a moderate recurrence. The natural rubber, mouth-protector type of appliance and the type with a cast chromium-cobalt framework lined with soft plastic were equally effective. The appliance is generally used only at night but may be worn night and day if such a schedule is required. Babcock[a0] and Sheridan and Reeve'' have also reported success in controlling the gingival overgrowth with positive-pressure appliances.

Steinberg suggested that use of a series of pressure appliances may help reduce the size of the gingival overgrowth without surgery.[52] He reported one case in which existing systemic conditions contraindicated surgical removal of the gingival tissue. After oral hygiene was improved, pressure appliances were made on stone

casts of the patient's upper and lower arches after trimming away 2 mm of the stone in the gingival overgrowth areas. The patient wore the appliance about 12 hours each day for 4 weeks; then a new pressure device was made. Steinberg observed a pronounced decrease in the size of the gingival lesions after 8 weeks of therapy. He suggested that a series of such appliances could succeed in gradually reducing gingival over-growth to clinically tolerable limits. He also pointed out that this therapeutic approach is not practical for the average patient but that it may prove valuable for patients with contraindications to oral surgery.

Drew et a1[53] and Backman et a1[5'] have reported that patients with PIGO or patients at risk for developing it may benefit from receiving folate therapy. Their work demonstrated a reduction in the severity of PIGO in patients who received systemic folic acid supplementation. The work of Drew et al further demonstrated that twice-daily oral rinsing with a topical folic acid solution resulted in significantly better tissue responses than systemic folic acid therapy.[53] Their studies suggest that folic acid therapy may inhibit PIGO and that additional related studies are indicated.

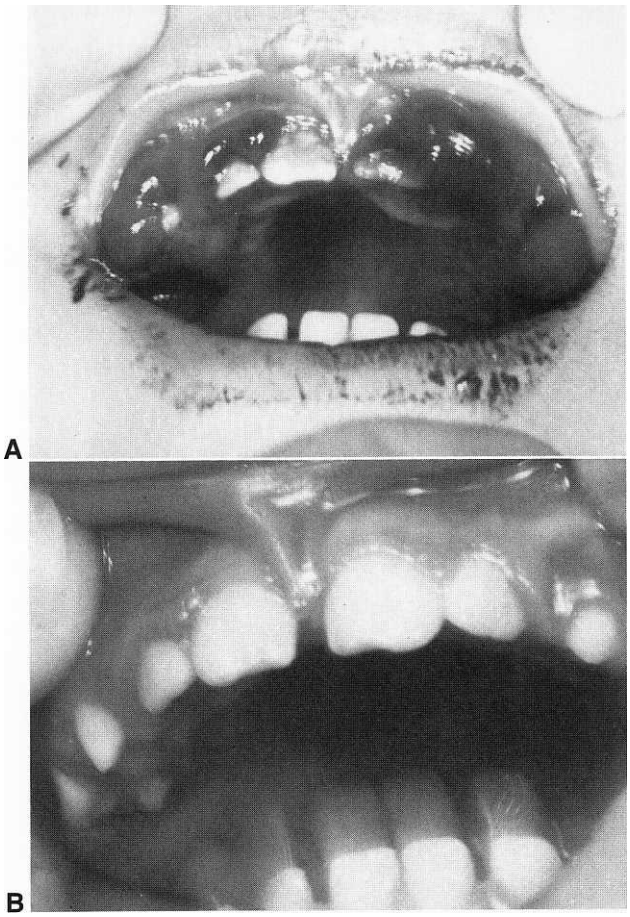
Other drugs that have been reported to induce gingival overgrowth in some patients include cyclosporin, calcium channel blockers, valproic acid, and phenobarbital. As with all disorders affecting periodontal tissues, maintaining excellent oral hygiene is the primary key to successful therapy.

ASCORBIC ACID DEFICIENCY GINGIVITIS

Scorbutic gingivitis is associated with vitamin C deficiency and differs from the type of gingivitis related to poor oral hygiene. The involvement is usually limited to the marginal tissues and papillae. The child with scorbutic gingivitis may complain of severe pain, and spontaneous hemorrhage will be evident. Fain, Mathieu, and Thomas believe that bleeding and gingivitis occur in some adult cancer patients because of scurvy rather than because of their malignant disease or chemotherapy.[55]

Severe clinical scorbutic gingivitis is rare in children. However, it may occur in children allergic to fruit juices when provision of an adequate dietary supplement of vitamin C is neglected (Fig. 20-18). When blood studies indicate a vitamin C deficiency and exclude other possible systemic conditions, the gingivitis will respond dramatically to the daily administration of 250 to 500 mg of ascorbic acid. Older children and adults may require 1 g of vitamin C for 2 weeks to speed recovery.

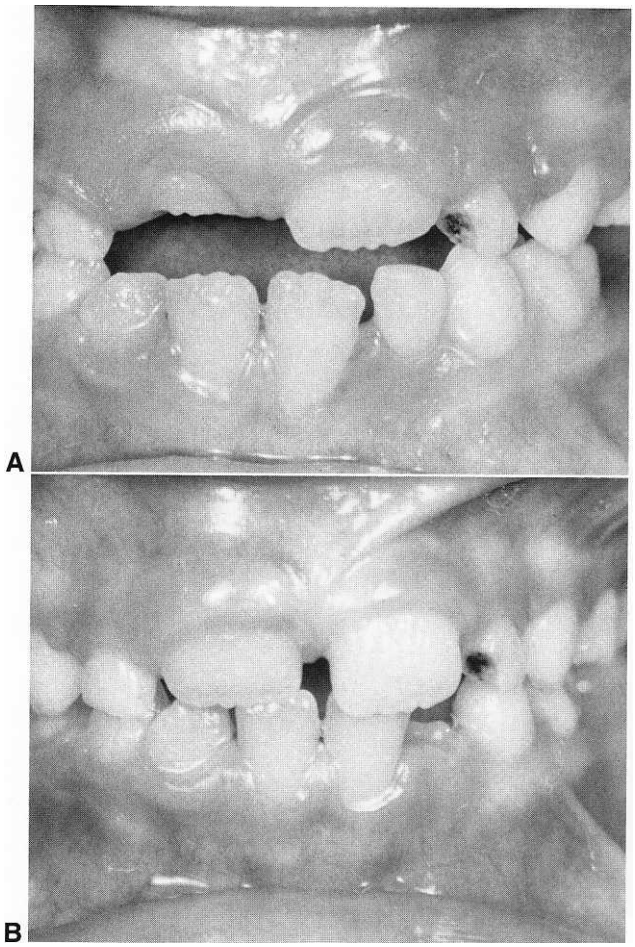
A less severe type of gingivitis resulting from vitamin C deficiency is probably much more common than most dentists realize. Inflammation and enlargement of the marginal gingival tissue and papillae in the absence of local predisposing factors are possible evidence of scorbutic gingivitis (Figs. 20-19 and 20-20). Questioning the child and parents regarding eating habits and using the 7-day diet survey will frequently reveal that the child is receiving inadequate amounts of foods containing vitamin C. Complete dental care, improved oral hygiene, and supplementation with vitamin C and other water-soluble vitamins will greatly improve the gingival condition.



f **I G 20-18. A,** Severe scorbutic gingivitis in a 16-month-old child. Large hematomas were evident in the maxillary arch. The condition was initially incorrectly diagnosed as Vincent infection. B, Daily administration of 400 mg of ascorbic acid resulted in a dramatic recovery.

PERIODONTAL DISEASES IN CHILDREN

Periodontitis, an inflammatory disease of the gingiva and deeper tissues of the periodontium, is characterized by pocket formation and destruction of the supporting alveolar bone. Bone loss in children can be detected in bite-wing radiographs by comparing the height of the alveolar bone to the cementoenamel junction.

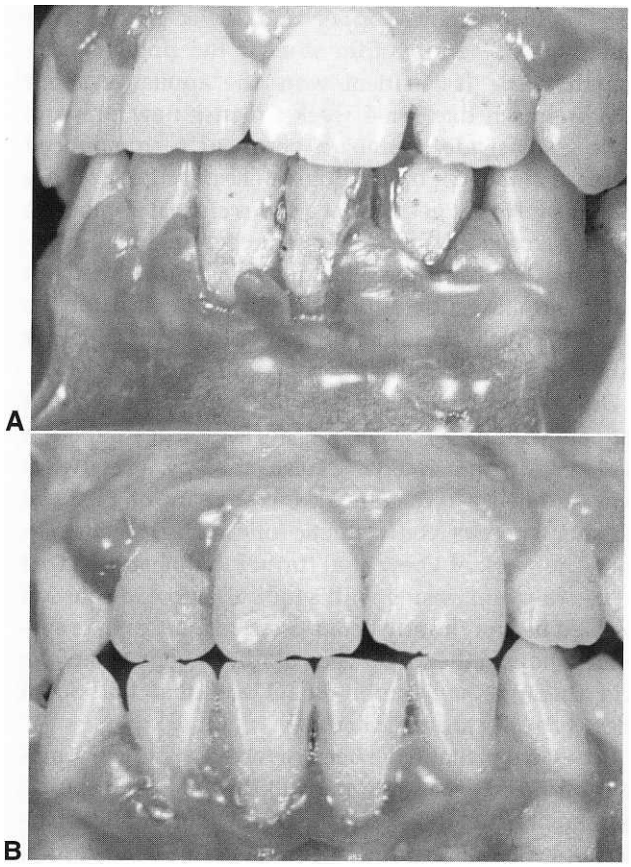


f **I 20-19. A,** Mild gingivitis caused by a vitamin C deficiency. The marginal tissue and papillae were painfully enlarged. A dietary history revealed that the child's diet was grossly deficient in fruits and vegetables. **B,** Improvement in diet and greater emphasis on oral hygiene resulted in a great improvement in the oral health.

Distances between 2 and 3 mm can be defined as questionable bone loss and distances greater than 3 mm indicate definite bone loss.

According to Delaney, in preschool children with periodontitis, recession, gingival erythema, and edema are not usually found unless the child is neutropenic.[2] Periodontal probing for attachment loss and bite-wing radiography are often used to clinically confirm the diagnosis. Bone loss is usually between the primary first and second molars. Bimstein, Delaney, and Sweeney demonstrated abnormal alveolar bone resorption in 7.6% of 4-year-old children and in 5.9% of 5-year-old children of primarily Hispanic origin with high caries.-

In its classification of periodontitis, the American Academy of Periodontology has categorized the early-onset form under Aggressive Periodontitis.[5i,5b]



[I G **20-20. A,** Scorbutic gingivitis in a 13-year-old girl. The diet was almost entirely lacking in foods containing vitamin C. **B,** An improved diet, supplemental amounts of fresh fruit juices, and toothbrushing instruction resulted in an improved gingival condition in 2 weeks.

EARLY-ONSET PERIODONTITIS

Albandar et al proposed the term *early-onset periodontitis* (EOP).[5u] EOP is used as a generic term to describe a heterogeneous group of periodontal disease occurring in young individuals who are otherwise healthy. EOP can be viewed as three categories of periodontitis that may have overlapping etiologies and clinical presentations: (1) a localized form (localized juvenile periodontitis [LJP]), (2) a generalized form (generalized juvenile periodontitis [GJPI), and (3) a prepubertal category that is thought to have both localized and generalized forms (localized and generalized prepubertal periodontitis). Albandar, Brown, and Loe, using data from a 1986-87 survey, estimated the prevalence of EOP in adolescent schoolchildren in the United States to be 10% in blacks, 5% in Hispanics, and 1.3% in whites.[uo]

Loe and Brown have reported observations from a periodontal assessment of 1107 adolescents aged 14 to 17 years.[61] Approximately 0.53% were estimated to have LJP, 0.13% to have GJP, and 1.61% to have incidental loss

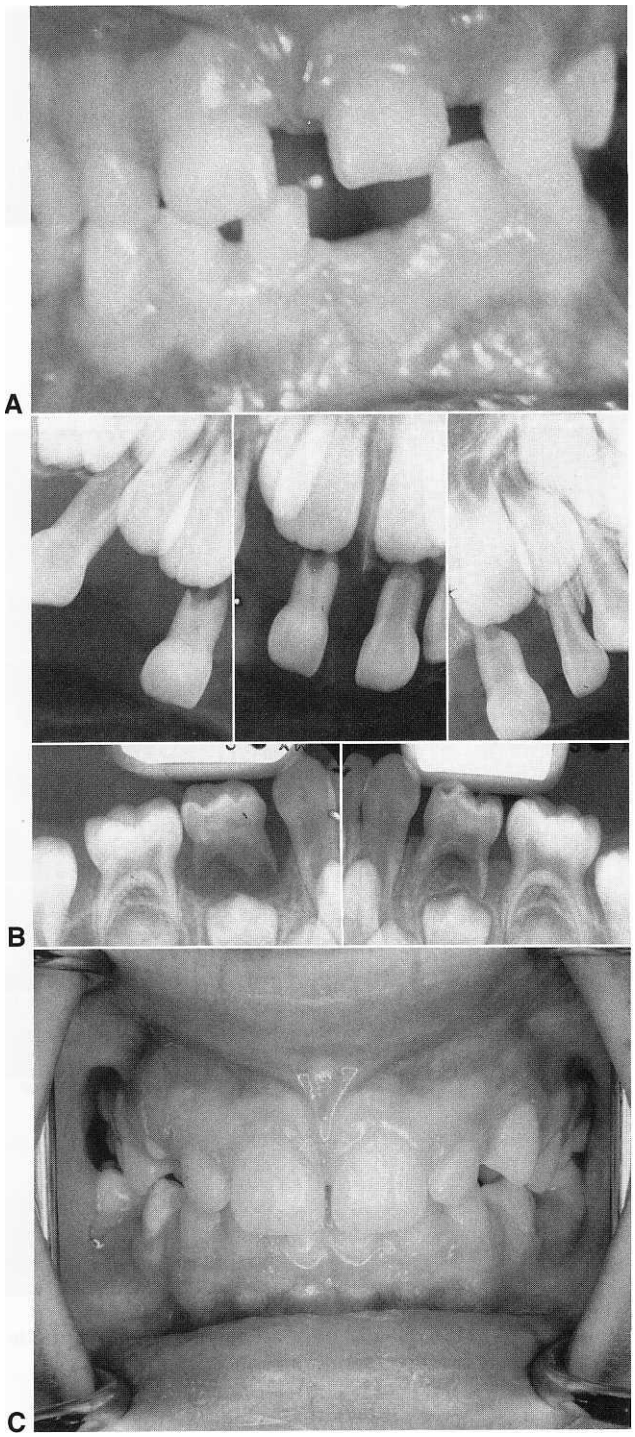
of attachment. Boys were more likely to have GJP than girls (ratio of 4.3:1).

Page et al believe that there are four different forms of periodontitis: prepubertal, juvenile, rapidly progressing, and adult.[tiz] As mentioned earlier, a new classification of periodontal disease has been published which recommends that EOP be recategorized as aggressive periodontitis and that its subclassifications be discarded. The old categorization has been retained because the new classification is not as widely used.

PREPUBERTAL PERIODONTITIS

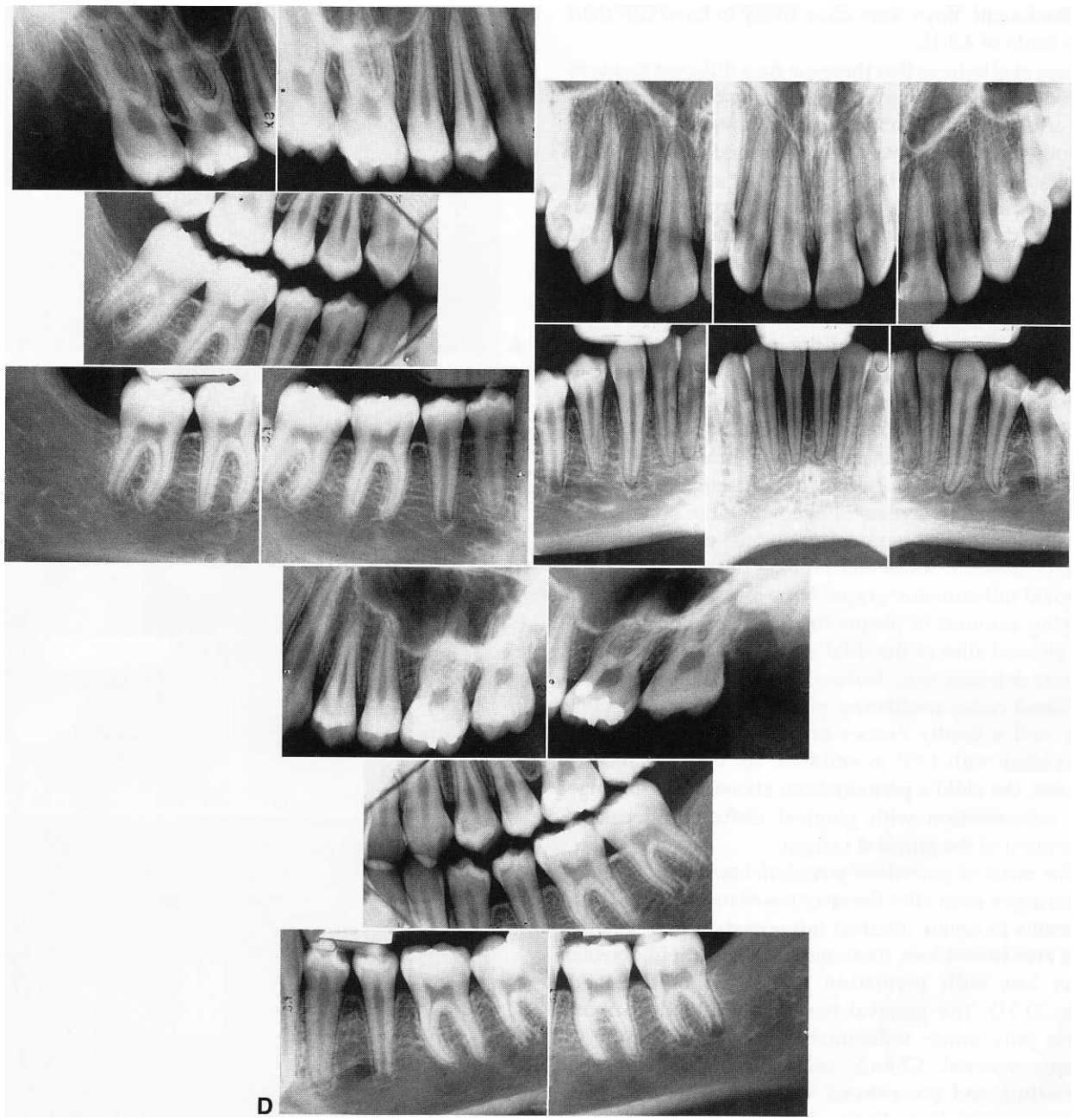
Prepubertal periodontitis of the primary dentition can occur in a localized form but usually is seen in the generalized form. *Localized prepubertal periodontitis (LPP) is* localized attachment loss and alveolar bone loss only in the primary dentition in an otherwise healthy child. The exact time of onset is unknown, but it appears to arise around or before 4 years of age, when the bone loss is usually seen on radiographs around the primary molars and/or incisors. Abnormal probing depths with minor gingival inflammation, rapid bone loss, and minimal to varying amounts of plaque have been demonstrated at the affected sites of the child's dentition. Abnormalities in host defenses (e.g., leukocyte chemotaxis), extensive proximal caries facilitating plaque retention and bone loss, and a family history of periodontitis have been associated with LPP in children. As the disease progresses, the child's periodontium shows signs of gingival inflammation with gingival clefts and localized ulceration of the gingival margin.

The onset of *generalized prepubertal periodontitis (GPP)* is during or soon after the eruption of the primary teeth. It results in severe gingival inflammation and generalized attachment loss, tooth mobility, and rapid alveolar bone loss with premature exfoliation of the teeth (Fig. 20-21). The gingival tissue may initially demonstrate only minor inflammation with a minimum of plaque material. Chronic cases display the presence of clefting and pronounced recession with associated acute inflammation. Testing may reveal a high prevalence of leukocyte adherence abnormalities and an impaired host response against bacterial infections. Alveolar bone destruction proceeds rapidly, and the primary teeth may be lost by 3 years of age. Micro-organisms predominating in the gingival pockets include *Actinobacillus actinomycetemcomitans (Aa), Porphyromonas (Bacteroides) gingivalis, Bacteroides melaninogenicus, Prevotella intermedia, Capnocytophaga sputigena,* and *Fusobacterium nucleatum*. Recent findings of Asikainen et al suggest that the major periodontal pathogens are transmitted among family members." Often the past medical history of the child reveals a history of recurrent infections. (e.g., otitis media, skin infections, upper respiratory tract infections) .



f **I G 20-21. A,** Prepubertal periodontitis in a 4 1/2-year-old girl. Loosening, migration, and spontaneous loss of the primary teeth occurred. **B,** A generalized loss of alveolar bone can be seen in the radiographs. C, Eight years after the initial observation of an involvement of the supporting tissues, there is evidence of normal gingival tissues. It is believed that dietary counseling and excellent oral hygiene contributed to the success of the treatment.

Continued



[l g **20-21, contd. D**, Radiographs made after the eruption of the permanent teeth demonstrate normal alveolar structures.

Consultation with a pediatrician is needed to rule out systemic diseases. Treatment of LPP or GPP depends on early diagnosis, dental curettage, root planing, prophylaxis, oral hygiene instruction, restoration of decayed teeth, removal of the primary teeth that have lost bony support, and more frequent recalls. Use of antimicrobial rinses (chlorhexidine) and therapy with broad-spectrum antibiotics are effective in eliminating the periodontal pathogens. Amoxicillin (Augmentin) has been used in children (250-mg liquid three times a day for 10 days). Because tetracycline (250-mg elixir twice a day for 10 days), or a derivative, is an effective antibiotic against Aa and other gram-negative anaerobic microorganisms, its use should be considered in the treatment of LPP or GPP. The child's parents should be made aware of the potential for pigmentation change in the developing permanent teeth and an increased susceptibility to oral candidiasis as a result of tetracycline therapy. Treatment of GPP is less successful overall and sometimes requires extraction of all primary teeth. Delaney reported that

children affected with LPP or GPP may experience severe periodontitis of the permanent teeth.'

See the later section Premature Bone Loss in the Primary Dentition.

LOCALIZED EARLY-ONSET PERIODONTITIS (LOCALIZED JUVENILE PERIODONTITIS)

LIP occurs in otherwise healthy children and adolescents without clinical evidence of systemic disease. It is characterized by the rapid and severe loss of alveolar bone around more than one permanent tooth, usually the first molars and incisors (Fig. 20-22). It appears self-limiting, and retrospective data obtained from LJP patients suggest that bone loss around the primary teeth can be an early finding in this disease. Reported estimates of the prevalence of LIP range from 0.1% to 1.5% with a bilaterally symmetric pattern of bone loss in a geographically diverse adolescent population. The prevalence in the black population is greater, at 2.5%. Clinically, LIP patients have little or no tissue inflammation and very little supragingival dental plaque or calculus. Progression of bone loss is three to four times faster than in adult periodontitis.

LIP is not thought to be a single disease entity. The probable causative microbial species are Aa or Aa in combination with Bacteroides-like species. A variety of neutrophil defects have been reported in patients with LIP. According to Page et al, most patients with LIP manifest abnormalities in peripheral blood neutrophil (polymorphonuclear leukocyte) chemotaxis and in some cases in monocyte chemotaxis.[6] Also, anomalies of phagocytosis, bacterial activity, leukotriene B_4 generation, and other defects have been reported. Page et al suspect a hereditary basis for LIP; some believe the mode of transmission is autosomal recessive, but others have provided evidence that the pattern is typical of an X-linked dominant mode.

GENERALIZED EARLY-ONSET PERIODONTITIS (GENERALIZED JUVENILE PERIODONTITIS)

The generalized form of EOP occurs at or around puberty in older juveniles and young adults. It often affects the entire periodontium of the dentition. Because of its wide distribution and rapid rate of alveolar bone destruction, the generalized form of EOP also is known by the terms *generalized juvenile periodontitis (GJP), severe periodontitis,* and *rapidly progressive periodontitis.* Affected teeth harbor more nonmotile, facultative, anaerobic, gram-negative rods (especially *Porphyromonas gingivalis)* in GJP than in UP. The localized and generalized forms of EOP are distinctly different radiographically and clinically. Neutrophils in GJP patients have suppressed chemotaxis. Individuals with GJP exhibit marked periodontal inflammation and have heavy accumulations of plaque and calculus. Loe and Brown reported a 0.13% prevalence of GJP among adolescents in the United States."

TREATMENT OF EARLY-ONSET PERIODONTITIS

Successful treatment of EOP depends on early diagnosis, use of antibiotics against the infecting microorganisms, and provision of an infection-free environment for healing. Treatment of EOP, both the localized and generalized types (LIP and GJP), includes surgery (Fig. 20-23) and the use of tetracyclines (sometimes in combination with metronidazole). In patients with LIP, Aa organisms penetrate into the crevicular epithelium. Treatment with antibiotics alone, such as a 2-week course of doxycycline (a synthetic tetracycline), has been shown to reduce the Aa population. Surgical removal of infected crevicular epithelium and debridement of root surfaces during surgery while the patient is on a 14-day course of doxycycline hyclate (1 g per day) is considered the best effective treatment modality. In a study of deep periodontal lesions, Christersson et al demonstrated that scaling and root planing alone were ineffective for the elimination of Aa.[65]

Microdentex manufactures the DMDx* test, a DNA test kit for periodontal pathogens. The test aids in establishing the risk of EOP and confirms that the child has responded favorably to the use of adjunctive antibiotics. Retesting in 4 to 6 weeks after the completion of antibiotic therapy will determine the patient's response to the treatment.

The test involves collecting a plaque specimen by inserting a paper point provided in the kit into a periodontal pocket for 10 seconds. The paper point is placed into a test vial and returned for the microbial test. Although the Pedo Probe test provides a detailed analysis only for Aa, the laboratory will also perform a more detailed microbial analysis.

Rams, Keyes, and Wright described the Keyes technique as effective in treating LJP.[66] The treatment involves meticulous scaling and root planing of all teeth, with concomitant irrigation to probing depth of saturated inorganic salt solutions and 1% chloramine T. In addition, they recommended administration of systemic tetracycline (1 g per day) for 14 days. Patient home care treatment included daily application of a paste of sodium bicarbonate and 3% hydrogen peroxide and inorganic salt irrigations.

Treatment of GJP is often less predictable. Alternative antibiotics directed at the specific pathogenic flora may be required when there is no response to traditional therapies. The multidisciplinary approach combines clinical laboratory evaluation with conventional periodontal therapeutic methods for diagnosis and treatment of severe GJP cases.

*Microdentex, Fort Myers, Fla.

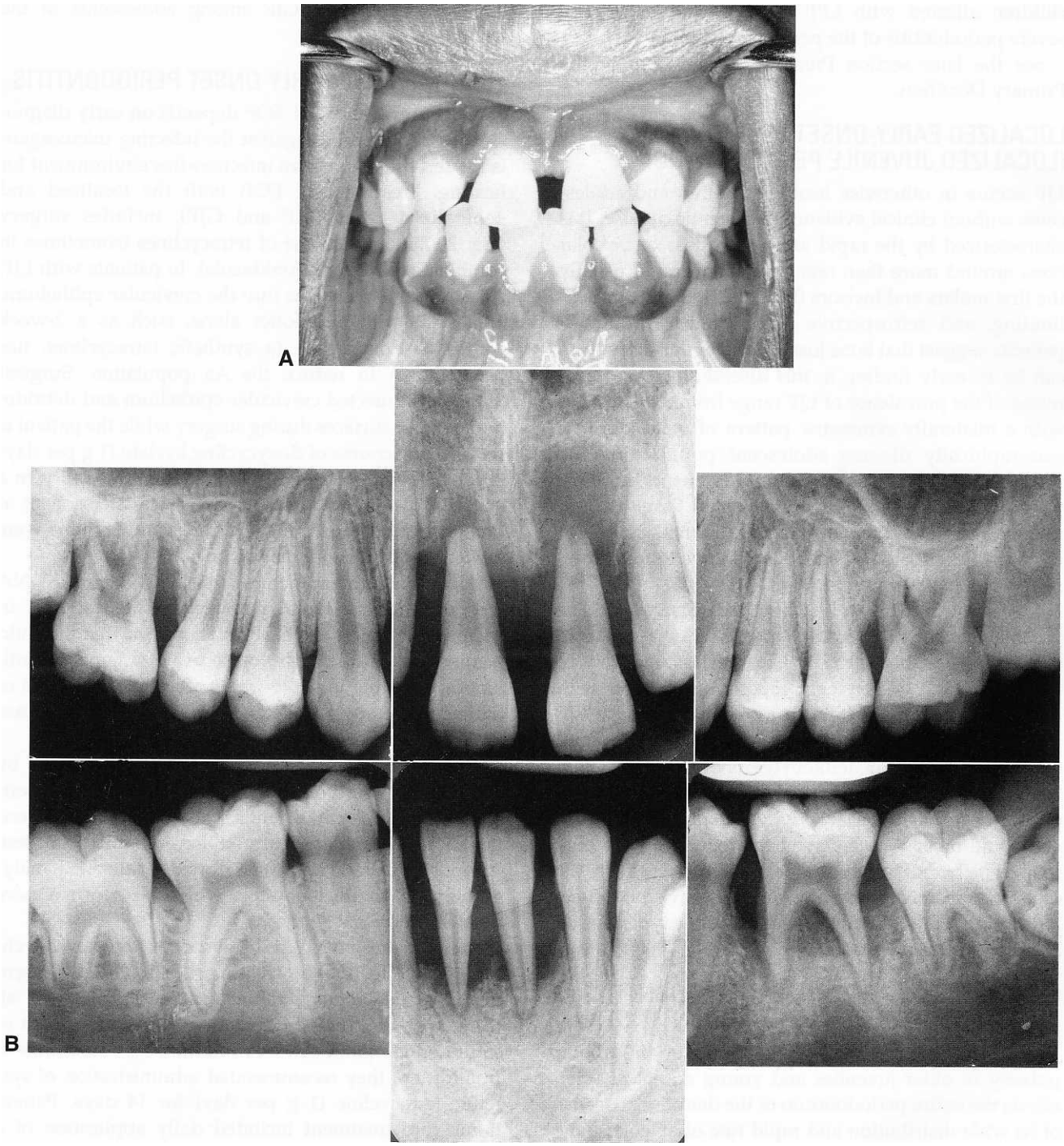
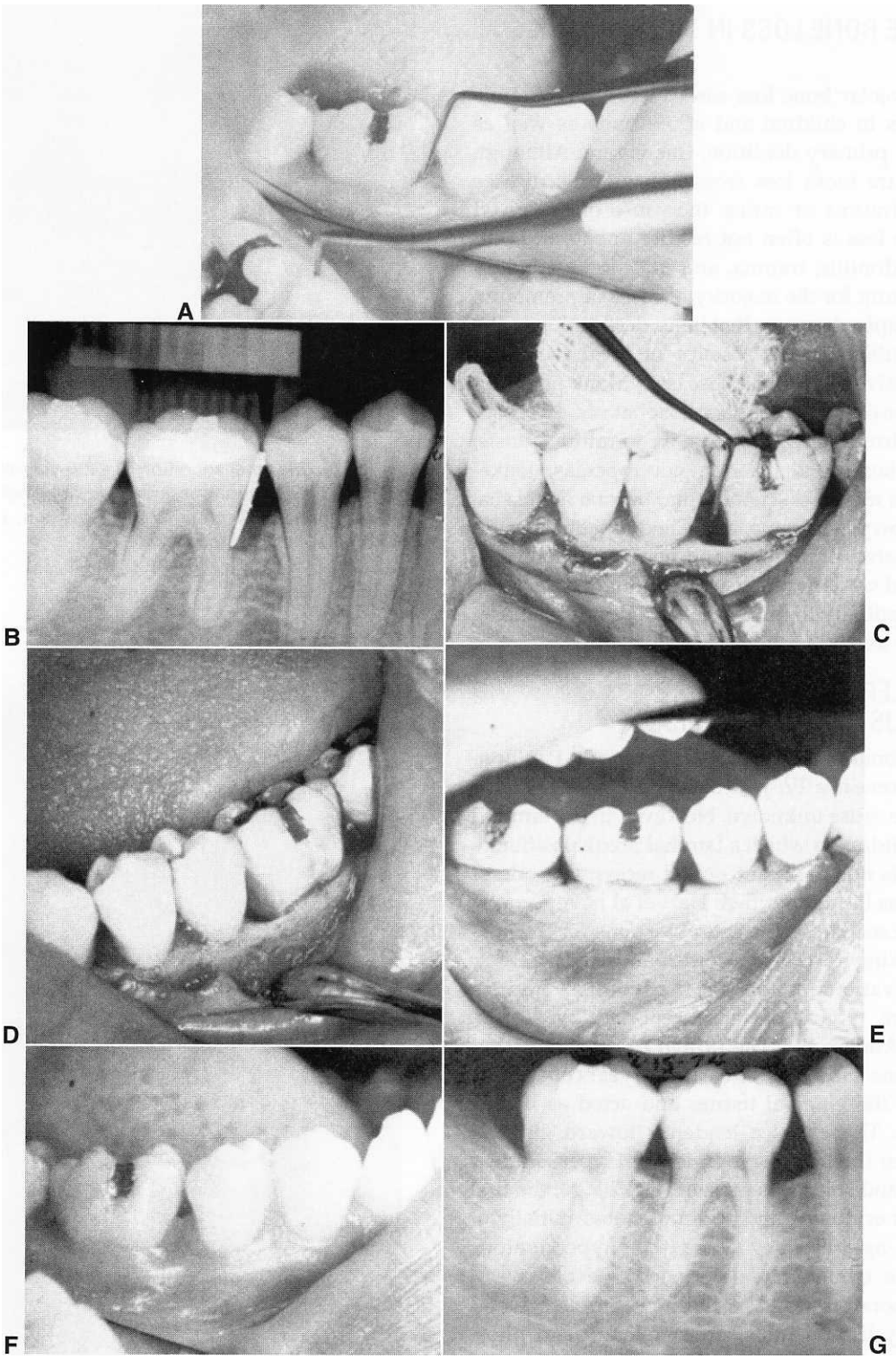


FIG. 20-22. **A,** Evidence of mild gingivitis in a 12-year-old boy with the localized form of early-onset periodontitis. Clinical examination revealed extensive mobility of the anterior teeth. **B,** Radiographs revealed loss of support in the incisor and first permanent molar areas. The maxillary central incisors, mandibular incisors, and first permanent molars were removed. Partial dentures were constructed.



f G. **20-23. A,** Probe in a mesial defect of the mandibular left first molar of a 16-year-old boy (mirror view). **B,** Radiograph showing depth of osseous defect. C, Periodontal probe showing depth of osseous defect. D, Defect completely debrided. **E,** Area sutured (mirror view). F, Area 6 months after surgery (mirror view). **G,** Radiograph 15 months after surgery. (Courtesy Dr. Timothy J. O'Leary.)

PREMATURE BONE LOSS IN THE PRIMARY DENTITION

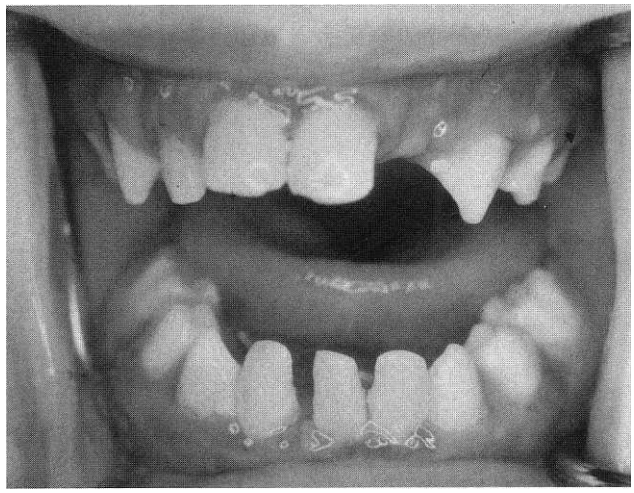
Advanced alveolar bone loss associated with systemic disease occurs in children and adolescents as well as adults. In the primary dentition, this is rare. Although most premature tooth loss from nonsystemic disease results from trauma or caries, the cause of advanced alveolar bone loss is often not readily apparent. Local factors (periodontitis, trauma, and infection secondary to caries) account for the majority of cases of premature bone loss. Goepferd reports that bony destruction in the primary dentition in the absence of local factors is highly suggestive of systemic disease.[57] Many possibilities exist; some of them are hypophosphatasia, Papillon-Lefevre syndrome, histiocytosis X, agranulocytosis, leukocyte adherence deficiency, neutropenias, leukemias, diabetes mellitus, scleroderma, fibrous dysplasia, acrodynia, Down syndrome, and Chediak-Higashi syndrome. The defect in immune and neutrophil cell function associated with these diseases is thought to increase patient susceptibility to infectious periodontitis causing alveolar bone loss and to other infections.

PAPILLON-LEFEVRE SYNDROME (PRECOCIOUS PERIODONTOSIS)

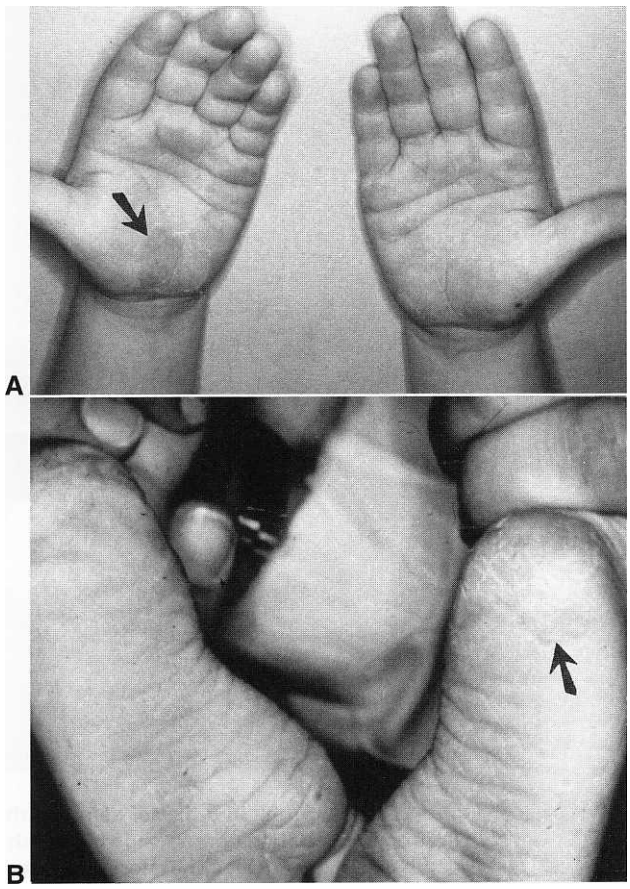
Coccia, McDonald, and Mitchell observed Papillon-Lefevre syndrome in a 2¹/2-year-old child.[68] The syndrome is rare and the cause unknown. I-Iowever, in the families of affected children in which a familial predisposition to the disorder is noted, an autosomal recessive mode of inheritance has been identified. Hart et al have recently narrowed the search for the causative gene for the syndrome by linking it to chromosome band 11g14-g2l.[6"]

The observations made in the young child in Fig. 20-24 are typical of those reported by Gorlin, Sedans, and Anderson.[70] The primary teeth erupted at the normal time. However, as early as 2 years of age, the child rubbed the gingival tissues and acted as if they were painful. There was a tendency toward gingival bleeding when the teeth were brushed. Hyperkeratosis of the palms and soles was present (Fig. 20-25); the first evidence was erythema and scaliness noted initially at 8 months of age. Delaney noted that hyperkeratotic lesions of the elbows and knees may be observed.[2] Repeated laboratory tests, including complete blood count, urinalysis, and microserum calcium and phosphorus determinations, yielded essentially normal results.

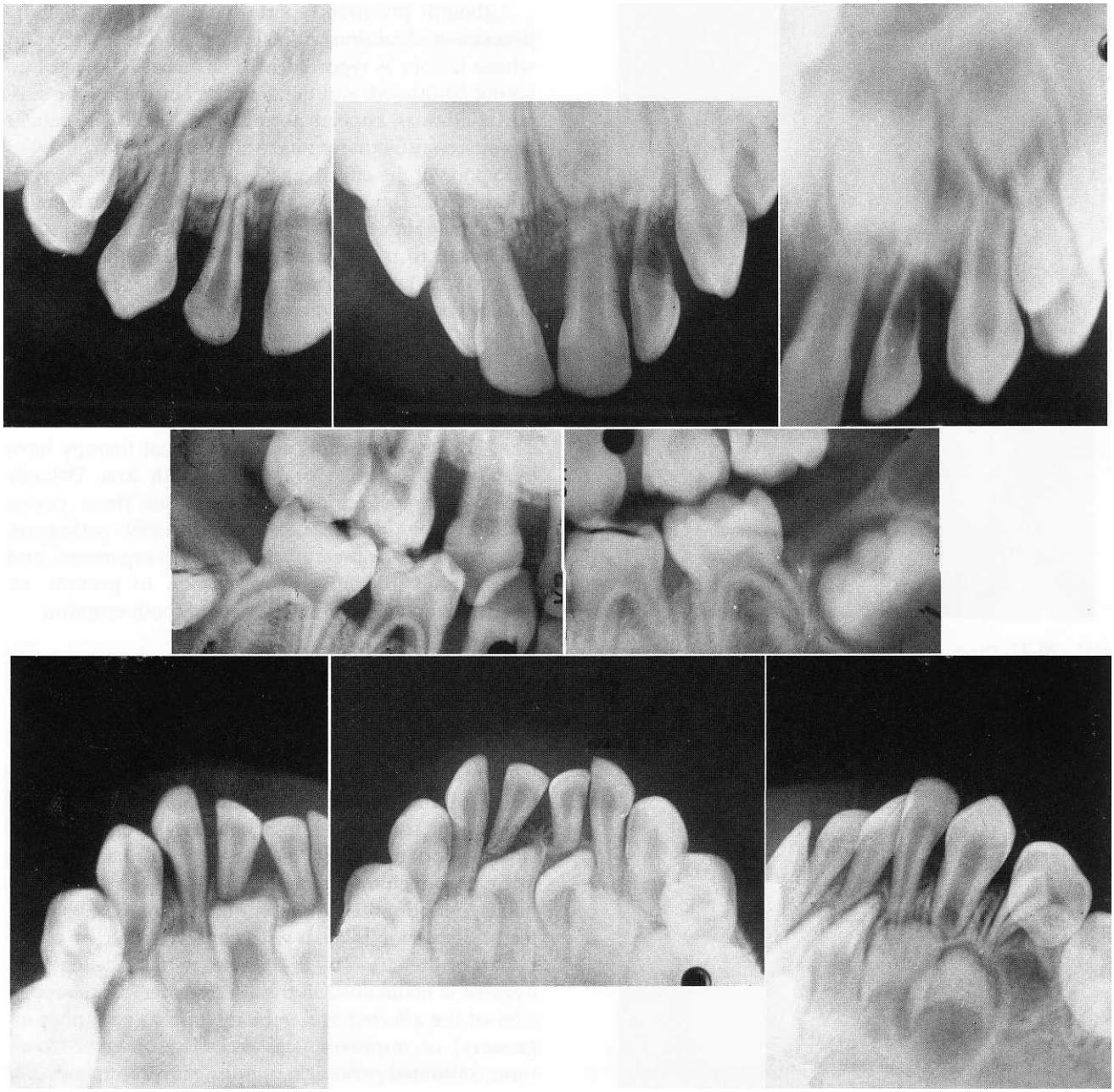
At 2¹/2 years of age, all the primary teeth showed looseness, and full-mouth radiographs revealed severe horizontal bone resorption (Fig. 20-26). Because of gingival inflammation, patient discomfort, and the presence of infected periodontal pockets, all the primary teeth were removed by 3 years of age. Histologic



f **I G 20-24.** Intraoral condition of a 2¹/2-year-old child who had Papillon-Lefevre syndrome. Inflammatory gingival engorgement and accumulated accretions were present, especially in the mandibular incisor area.



[I G . **20-25. A**, Hyperkeratosis of the palmar surfaces of the hands (arrow). Erythema and scaliness were noted initially at 8 months of age. **B,** Hyperkeratosis of the plantar surfaces of the feet (arrow).



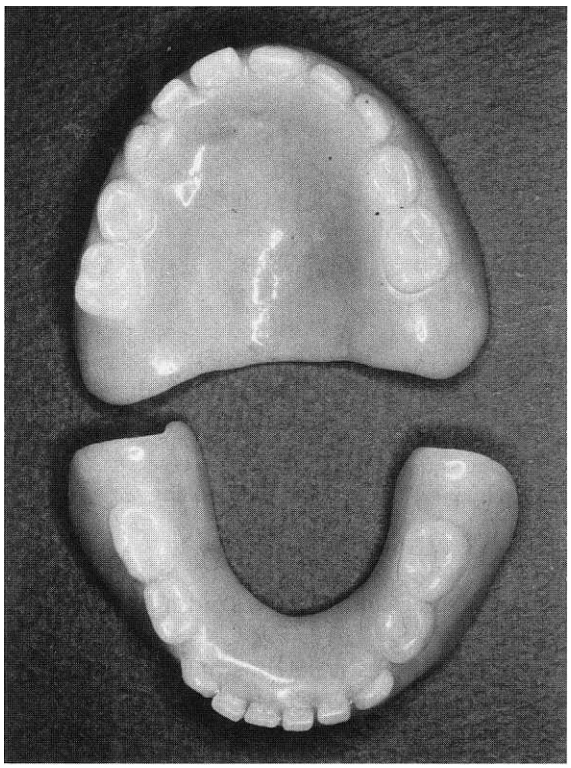
[I G. **20-26.** Full-mouth and bite-wing radiographs of the child with Papillon-Lefevre syndrome. Severe horizontal bone resorption is readily detected in all four quadrants. The incisor areas display extreme alveolar bone loss to the extent that only the apical third of the root remains supported.

sections of the teeth displayed a premature resorption pattern with essentially normal pulp tissue. Cementum was apparently normal and covered the root structure. An accumulation of adherent basophilic plaque, made up of a mass of filamentous microorganisms, was noted on almost the entire length of the root surface. Periodontal pathogens *(Actinobacillus actinomycetemcomitans, F. nucleatum, Capnocytophaga* species, and *Eikenella corrodens)* have been isolated in the dental plaque of patients with Papillon-Lefevre syndrome according to Tinamoff et al."""-

Complete dentures were constructed 3 months after the removal of the primary teeth. The child tolerated the dentures well, both functionally and psychologically (Figs. 20-27 and 20-28). The first permanent molars and mandibular central incisors erupted at the expected time, and the denture base was adjusted to allow for the emergence of the teeth.



[IG. 20-27. Three-year-old child with complete dentures.



f IG 20-28. Constructed complete dentures, which were later modified to allow the eruption of the mandibular incisors and the first permanent molars.

Although previous reports have indicated that the permanent dentition will also be affected, the child whose history is reported here has been followed into young adulthood, and the dentition, including the supporting tissues, appears normal (Fig. 20-29). The patient has successfully undergone orthodontic treatment.

Reports of the effectiveness of tetracycline therapy as an adjunct to meticulous subgingival debridement in the management of periodontal disease prompted McDonald to reinvestigate the history in the reported case of Papillon-Lefevre syndrome. The father, a physician, reported that tetracyclines were given to the child repeatedly for ear infections between 3 and 6 years of age. This regimen may have been responsible for eliminating pathogens and preventing the destructive process from being carried into the permanent dentition. However, attempts at conventional therapy have been unsuccessful in preventing tooth loss. Delaney reports that periodontal treatment for these young children includes identification of specific pathogens, specific antibiotic therapy against these organisms, and full-mouth extractions early enough to provide an edentulous period before permanent tooth eruption.'

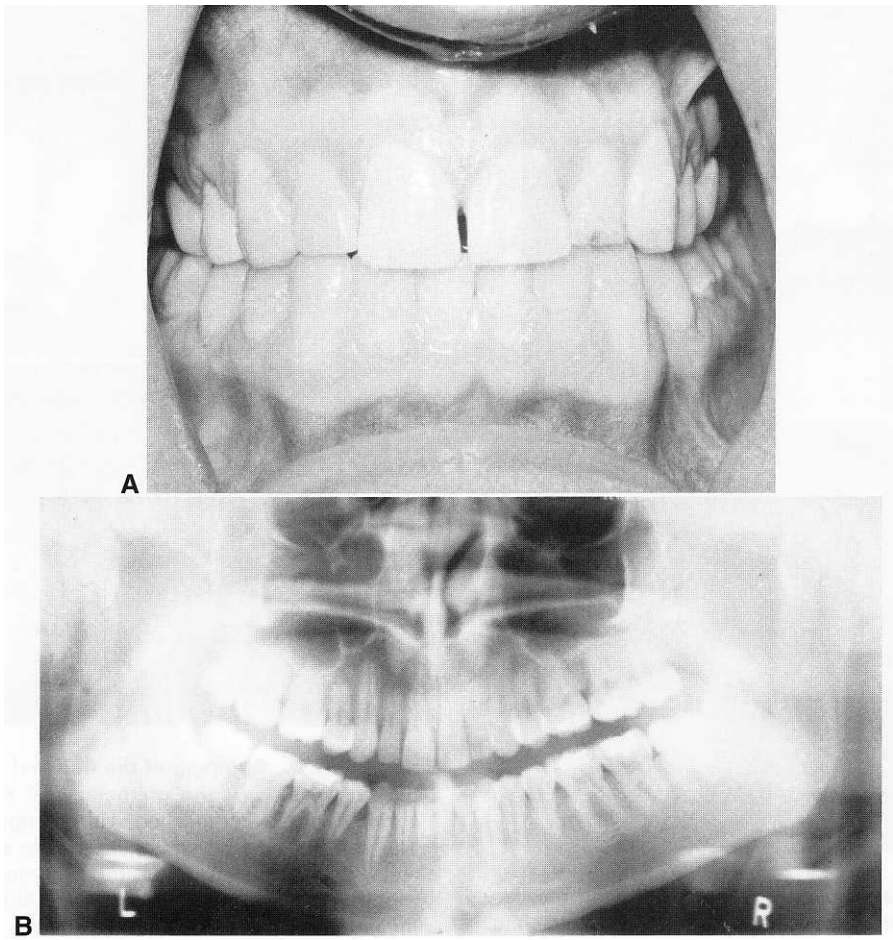
GINGIVAL RECESSION

Gingival recession is often observed in children. Several factors predispose patients to gingival recession. These factors include presence of a narrow band of attached or keratinized gingiva, alveolar bony dehiscence, toothbrush trauma, tooth prominence, impinging frenum attachment, soft tissue impingement by opposing occlusion, orthodontic tooth movement, use of impression techniques including subgingival tissue retraction, oral habits, periodontitis, and pseudorecession (extrusion of teeth). Recession is dealt with conservatively by elimination of the stimulus if possible, while excellent oral hygiene is maintained in the affected areas. If the recession of the affected area remains unchanged (nonprogressive) or improves (less recession observed) over time, continued periodic monitoring is recommended. If the recession has progressed after a 4- to 8-week period of observation, other periodontal procedures may be required based on the identified predisposing factor.

SELF-MUTILATION

Although there has been infrequent reference to self-mutilation in the dental literature, occasionally children purposely traumatize their oral structures. Plesset reported observing a 9-year-old girl of apparently normal intelligence who worked her maxillary primary canine and mandibular permanent incisors loose from their supporting tissues and removed them.[73]

Self-mutilation probably occurs more frequently than is realized because relatively few children will admit to the act unless they are observed practicing it. Therefore



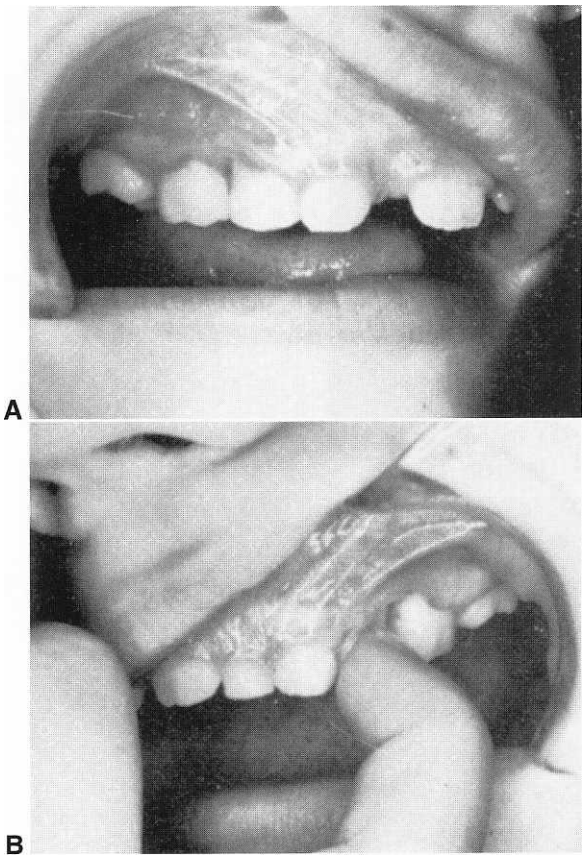
f **I G 20-29.** Photographs 15 years after initial diagnosis of Papillon-Lefevre syndrome at 2 1/2 years of age. A, Functional occlusion is noted after 18 months of orthodontic treatment. The clinical examination revealed good oral hygiene and normal gingival tissues. **B,** Panoramic radiographic surgery revealed normal alveolar bone. Some apical root resorption related to orthodontic treatment can be seen in the mandibular incisors.

the self-inflicted lesions may be incorrectly diagnosed. Dentists should be aware of the possibility of this condition and should approach the problem in the same manner as they do thumb sucking. An attempt should be made to determine the cause. If it is found to be the result of local dental factors, it can be corrected. However, in the majority of children an emotional problem is involved, and the family must be directed to competent counseling services.

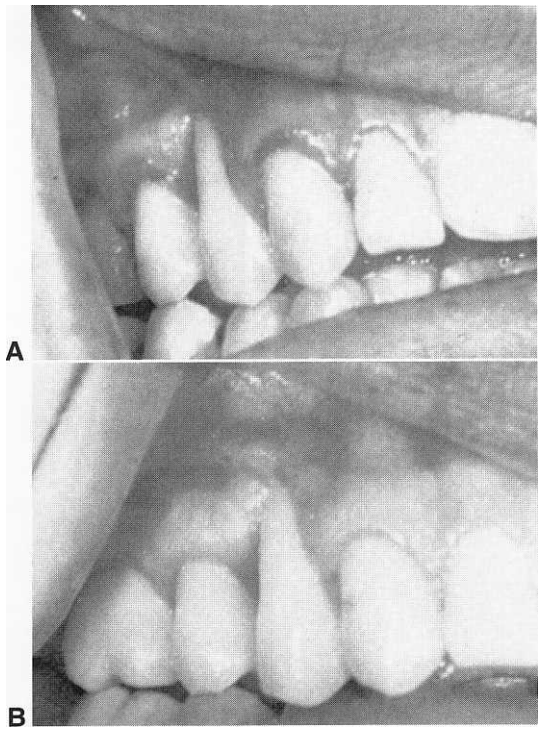
Children as young as 4 years of age have been observed who have traumatized the free and attached gingival tissues with a fingernail, occasionally to the extent that the supporting alveolar bone has been destroyed (Fig. 20-30). A 14-year-old girl produced bilateral stripping of the buccal tissue in the maxillary premolar areas (Fig. 20-31) with her fingernail and a bobby pin. In addition, she bit the inner surface of her cheek and produced large necrotic areas. The parents were unaware of the habit and of the cause of the ulcerated areas in her mouth, because the daughter did not reveal that they were self-inflicted. The history revealed an unhappy child who was poorly adjusted to the home and school environments. Referral to a child guidance clinic was accepted by the child and the parents and resulted in a solution to the problem. The self-mutilation was apparently an escape from reality.

Tension and conflicts in the home can cause self-mutilation in young patients. Fisher reported that unhappiness and conflict in the home can be hidden more easily from a 15-year-old child than from a 15-week-old child."

Traumatic gingival recession in infants resulting from a dummy (pacifier) sucking habit has been observed by Stewart and Kernohan." In the unconventional sucking habit a segment of the plastic shield is embraced by the infant's lower lip so that the inner surface of the shield bears against the labial aspect of the incisors and the gingival tissues (Fig. 20-32). If the shield is held in this position, the edge of the shield moves with an abrasive



f **I G 20-30.** Traumatization of the gingiva between the maxillary primary molars with the index fingernail in a 6-year-old child.



f **I C 20-31. A,** Stripping of the free and attached tissue from the buccal side of the premolar and exposure of the root surface were accomplished with a fingernail and a hairpin. The patient had a psychologic problem and was referred to a child guidance clinic. **B,** After treatment of the emotional problem the habit was discontinued. Although the soft tissue inflammation was reduced, there was no regeneration of tissue over the root surface.

action during sucking, which leads to gingival injury, recession, and loss of alveolar bone.

ABNORMAL FRENUM ATTACHMENT

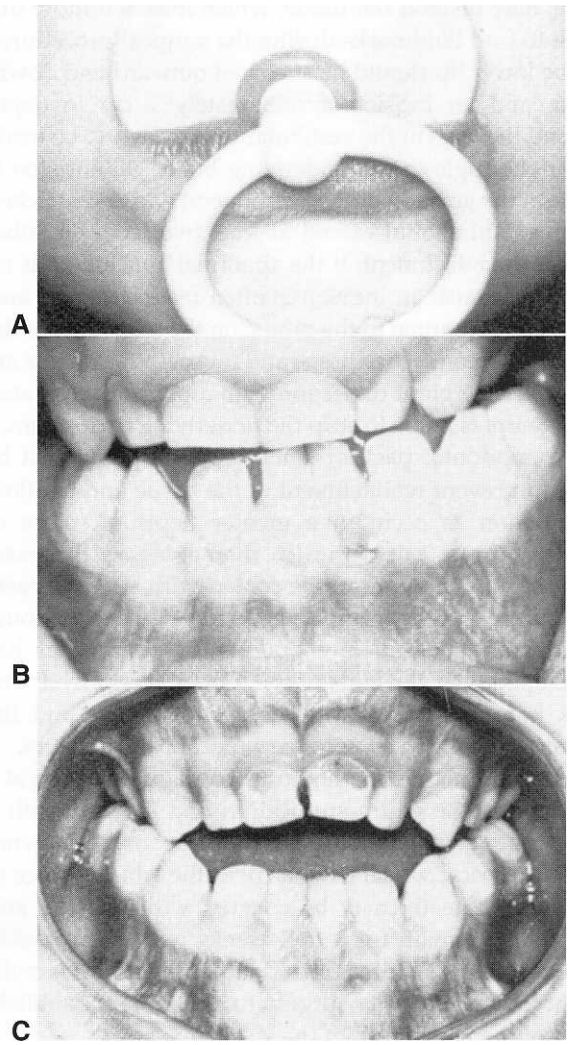
A frenum is a membranous fold that joins two parts and restricts the individual movement of each. Henry, Levin, and Tsaknis describe a frenum as a mucous membrane fold containing epithelium and connective tissue fibers but no muscle." A normal frenum attaches apically to the free gingival margin so as not to exert a pull on the zone of the attached gingiva, usually terminating at the mucogingival junction. Although a wide variety of aberrant positions occur, commonly observed locations of frenums in the child are on the facial gingival surface of the anterior midline of the maxilla, on the facial and lingual gingival surfaces of the anterior midline of the mandible, and on the mandibular and maxillary premolar facial area. Some frenums have bifid and trifid attachments to the alveolar process.

An abnormal or high frenum is present when there is inadequate attached gingiva in the terminal insertion area. A frenum attached too closely to the gingival margin may interfere with proper toothbrush placement, may cause opening of the gingival crevice during function, or may interfere with speech. High frenum attachments may also be associated with isolated gingival recessions and diastemas, although a cause-and-effect relationship may or may not exist.

A mandibular anterior frenum occasionally inserts into the free or marginal gingival tissue and causes subsequent recession and pocket formation. The abnormal frenum attachment is most often observed in the central incisor area (Fig. 20-33), although it may involve the labial tissue in the canine areas. The abnormal attachment is frequently associated with a vestibular trough throughout the anterior region that is more shallow than normal.

An abnormally short lingual frenum and the inability to extend the tongue is a congenital condition known as ankyloglossia (tongue-tie). Normal function can occur with this mild form, and the frenum may lengthen with normal growth and maturation of the child.

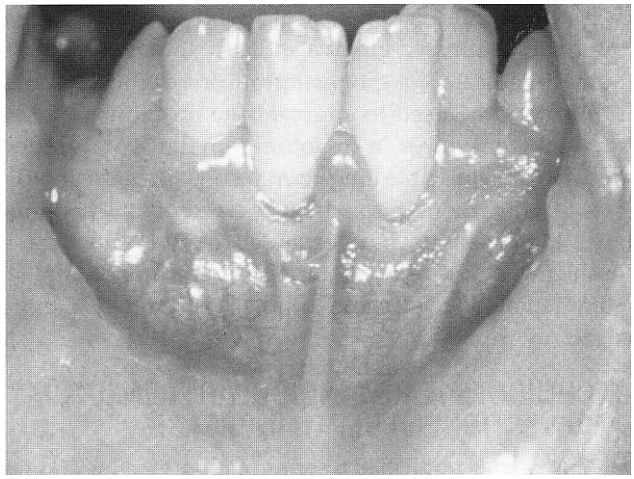
Movements of the lip cause the abnormal frenum to pull on the fibers inserting into the free marginal tissue. Food accumulates and causes inflammation and eventually the development of a pocket between the labial surface of the tooth and the vestibular mucosa. Early treatment of the abnormal frenum attachment is



I **I G . 20-32. A,** An unconventional manner of pacifier sucking may lead to gingival trauma and recession. **B** and C, Gingival recession in the lower incisor region in two children. Note anterior open bite in C. *(From Stewart DJ, Kernohan* DC: Br Dent J *135:157-158, 7973.)*

indicated to prevent continued stripping of the labial tissue, subsequent loss of alveolar bone, and possible eventual loss of the tooth. Although traumatic occlusion and poor oral hygiene are occasionally associated with the gingival stripping condition, the abnormal frenum attachment is more often the offender.

The maxillary anterior frenum connects the upper lip to the suture line areas between the central incisors. The importance of a frenum that prevents esthetic contact of the central incisors has been the subject of debate. The literature indicates that a diastema may be considered normal as the upper central incisors erupt and can be expected to close as the other permanent front teeth erupt. Seldom has any correlation been found between a maxillary frenum problem and recession.



I C 20-33. Abnormal attachment of the frenum. The fibers can be seen extending to the papilla between the central incisors with branching auxiliary fibers inserting into the marginal tissue.

FRENECTOMY AND FRENOTOMY

A frenectomy involves complete excision of the frenum and its periosteal attachment. *A frenotomy* involves incision of the periosteal fiber attachment and possibly suturing of the frenum to the periosteum at the base of the vestibule. It is associated with less postoperative discomfort than a frenectomy and will usually suffice. A frenectomy is indicated when large, fleshy frenums are involved. The need for a frenectomy or frenotomy should be based on the ability to maintain gingival health. The surgical management of the abnormal maxillary labial frenum is presented in Chapter 7 (see Figs. 7-49 to 7-51) .

Indications for treating a high frenum include the following:

1. A high frenum attachment associated with an area of persistent gingival inflammation that has not responded to root planing and good oral hygiene.
2. A frenum associated with an area of recession that is progressive.
3. A high maxillary frenum and an associated mid-line diastema that persists after complete eruption of the permanent canines.
4. A mandibular lingual frenum that inhibits the tongue from touching the maxillary central incisors. This would interfere with the child's ability to make /t/, /d/, and /1/ sounds. As long as the child has enough range of motion to raise the tongue to the roof of the mouth, no surgery would be indicated. Most children cannot normally make these sounds until after 6 or 7 years of age. Speech therapy may be indicated (see Fig. 7-45).

If a high frenum is associated with an area of no or minimal keratinized gingiva and a frenotomy or frenectomy is indicated, a gingival graft or vestibular extension should be used to augment the procedure. Under these circumstances, a frenotomy or frenectomy often does not create stable long-term results. Bohannan indicated that, if there is an adequate band of attached gingiva, high frenums and vestibular depth do not pose a problem."-" Use of the latter procedures to accomplish elimination of the frenum pull is considered a more standard approach.

TECHNIQUES FOR MANDIBULAR FRENECTOMY AND VESTIBULAR DEPTH INCREASE

Bohannan has published a series of reports of his studies in the alteration of vestibular depth and frenectomy.[77-79] Three different surgical procedures, which he refers to as *complete denudation, periosteum retention,* and *vestibular incision,* were studied as methods to produce increased vestibular depth and frenum alteration.

Complete Denudation. The complete denudation procedure is preceded by a routine gingivectomy extending laterally to the first premolars. By blunt dissection, the periosteum and adherent fibrous tissue are detached apically, and the labial plate is exposed to a depth of approximately 12 mm. The resulting soft tissue flap is removed by excision. A rapid-setting dressing is placed directly over the osseous tissue and is changed at 7-day intervals for 4 weeks.

Periosteum Retention. The periosteum retention procedure, as carried out by Bohannan, does not always result in maintenance of the desirable amount of vestibular depth." The procedure is essentially the same as that described previously, except that the periosteum is allowed to remain.

Vestibular Incision. The vestibular incision method (mandibular frenectomy) is a surgical procedure that we have used with success. The elimination of the abnormal frenum should remain the objective of the procedure, although it is often desirable also to alter the vestibular depth. However, the frenum is the primary etiologic factor in the stripping of the gingival tissue and labial pocket formation.

A preliminary prophylactic procedure should be performed to remove hard deposits, debris, and plaque material from the teeth. The surgical procedure should be more extensive than a conservative incision of the frenum. Such a procedure would allow the muscle fibers to reattach, with resultant scar tissue formation, and would perhaps make the condition more severe (Fig. 20-34).

A local anesthetic is given before the surgical procedure. A right and left inferior alveolar injection is the method of choice. Some dentists prefer to inject the local anesthetic solution throughout the operative field. However, caution should be exercised because the anesthetic may distend the tissue, which makes it more difficult to find landmarks during the surgical procedure.

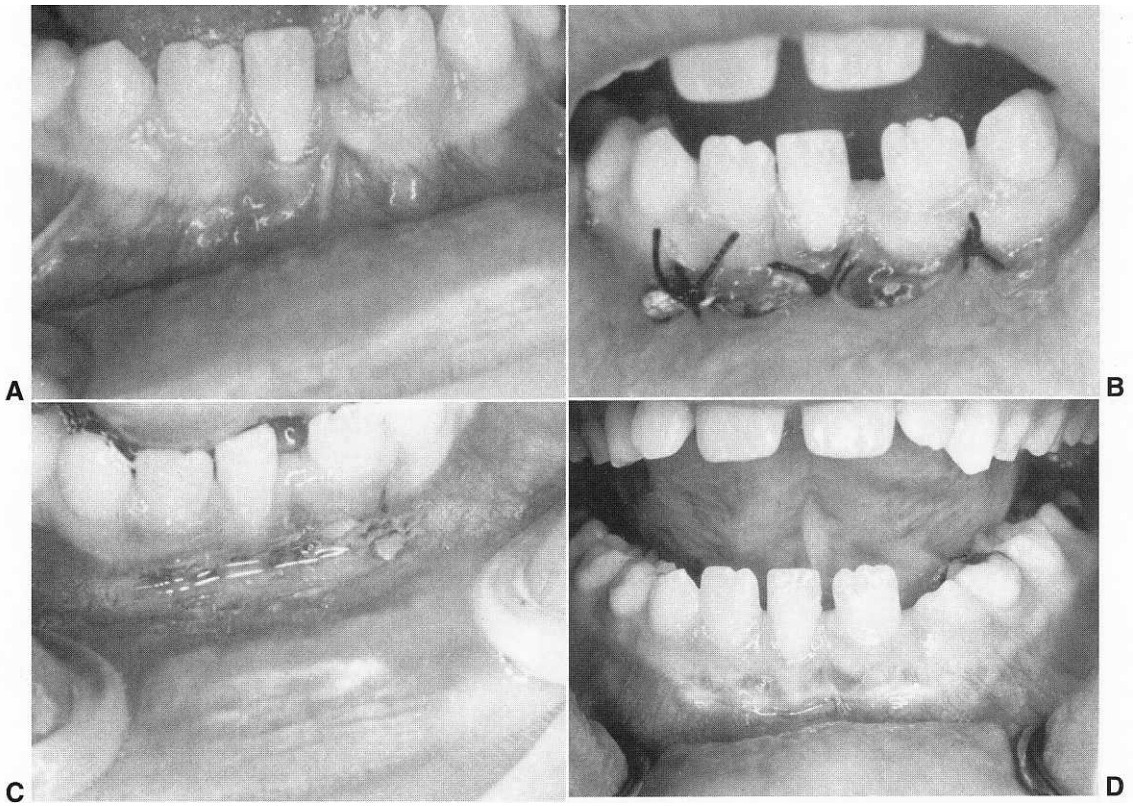
The lower lip should be stretched outward and downward, and an incision approximately 1 cm in depth beyond the level of the vestibular trough should be made at a right angle to the underlying bone. The incision is made at the junction of the mucobuccal fold and attached gingiva, and should extend at least two teeth on either side of the attachment. If the abnormal attachment is on the incisor area, an incision is often made from an area opposite one canine to the canine on the other side of the mouth. The connective tissue and muscle attachments are then freed by blunt dissection with a periosteal elevator. No attempt is made to strip the underlying periosteum.

A periodontal pack or some type of splint must be used to prevent reattachment of the tissue and to allow granulation to occur at a greater depth. A piece of rubber tubing 2 to 3 mm in diameter and the exact length of the incision can be coated with surgical paste and sutured in the trough. The patient is seen 24 hours postoperatively, and any granulation tissue that has developed over the ends of the tubing is removed. The pack is normally removed after 4 to 5 days, and the wound is irrigated as necessary until healing occurs.

As an alternative method of encouraging healing at a new depth, the entire surgical wound is filled with a stiff periodontal dressing of the zinc oxide—eugenol type. The pack, which extends over the labial surface of the anterior teeth, may be covered with dry foil and allowed to remain for 3 to 4 weeks with only weekly changes (Fig. 20-35). Some dentists ligate an acrylic splint to the teeth after surgery to aid in the reestablishment of the new depth of the sulcus.

FREE GINGIVAL AUTOGRAFT PROCEDURE

The free gingival autograft procedure (FCC) may be considered for children and young adults when there is a prominent tooth position that is complicated by absence of attached gingiva, a shallow vestibular fornix, and a high midline frenum attachment (Fig. 20-36). FGG is also indicated when there are areas of root exposure that are an esthetic concern to the patient, when the labial gingivae of some teeth require subgingival crown margins, when teeth are clasped by a removable partial denture having a narrow zone of gingiva, and when teeth that have no labial keratinized gingiva need orthodontic movement, which makes them more prominent in the arch. When these indications are kept in mind, it will be apparent that FGG is not always the best procedure. There may be an esthetic concern with the surrounding gingiva because the autograft will remain the same color and texture as the palatal donor site (Fig. 20-37). FGG will not cover the area of recession but rather will prevent its progression. If root coverage is a



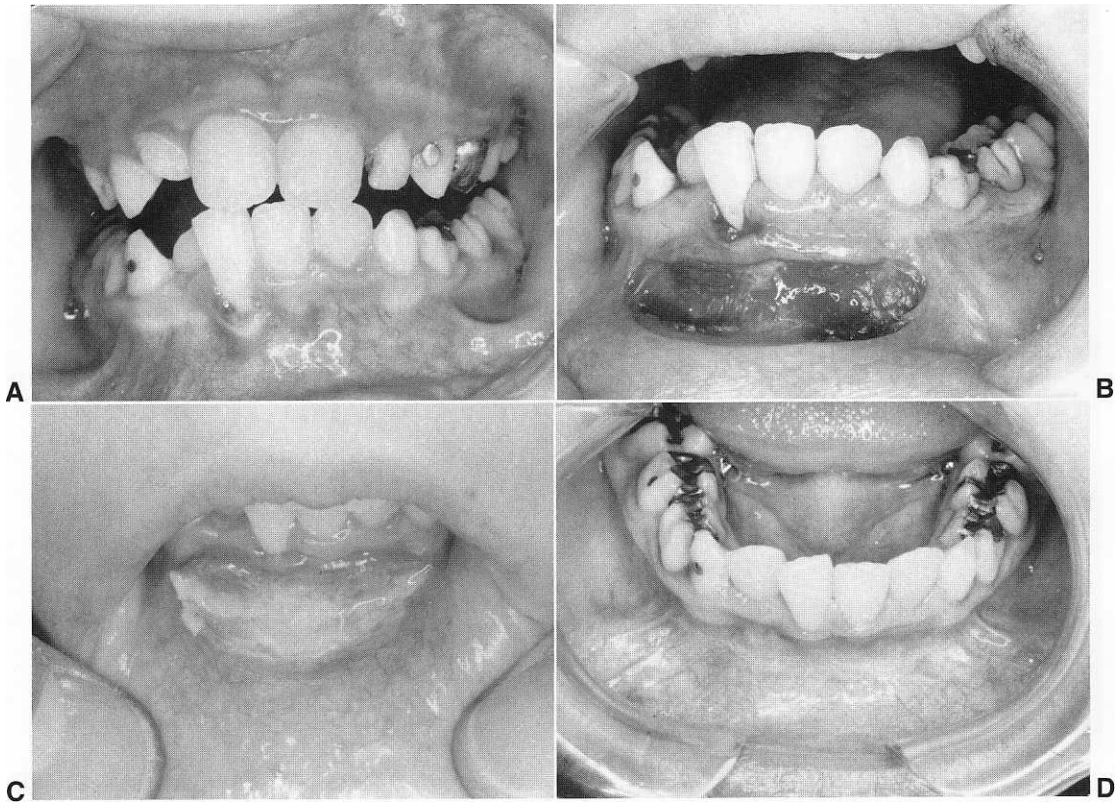
f I C 20-34. **A,** Tissue has been stripped and a pocket formed on the labial surface of the right central incisor. Frenectomy and procedure to increase the depth of the vestibular trough are indicated. **B,** An incision has been made and a pack has been placed and secured with three sutures. C, Five days after the operation the pack was removed. Granulation tissue has formed, and normal healing is taking place. D, Three months after the operation, improvement in the health of the gingival tissue in the mandibular anterior region is evident.

goal, the laterally positioned pedicle graft and coronally positioned flap procedures are more predictable. When the amount of attached gingiva is adequate, FGG is not indicated. If pocket elimination is a concern, an apically repositioned flap should be a choice.

In the FGG procedure the receptor site is first prepared. When a high frenum attachment has contributed to the mucogingival problem, tension is placed on the lower lip to activate the frenum and trace its insertion into the marginal tissue. A horizontal incision is made at the mucogingival junction, including the attachment of the frenum into the gingiva. The mucosa is displaced apically by blunt dissection, and a nonmovable receptor site is left consisting of periosteum and a thin covering of firm connective tissue. Any gingival epithelium coronal to the primary incision is trimmed away, which leaves a receptor bed of connective tissue with an adequate blood supply.

The next step is to fenestrate the periosteum to help prevent mobility of the graft after healing. To do this, two horizontal incisions, 1 mm apart, are made through the periosteum at the apical boundary of the displaced mucosa. The isolated periosteum between the two horizontal incisions is peeled away, so that the facial cortical plate of bone is exposed. A template of adhesive foil slightly larger than the mandibular receptor site is made, and the palatal donor site is anesthetized. The template is placed on the anesthetized tissue and is outlined with a shallow incision. The graft is freed from the underlying submucosa by sharp dissection. The operator should strive to secure an intermediate-thickness graft (approximately 1 mm thick).

After the graft is removed from the palate, the connective tissue side is inspected to ensure that no fatty tissue remains on it (adherent fatty tissue would prevent revascularization of the graft from the connective tissue of the receptor site). If present, the fatty tissue is cut away. The graft is carried to the receptor site, and any final trimming is done to secure an approximation of the donor tissue to the receptor site. The graft is sutured into position with a 5-0 Dacron suture and an atraumatic needle. Normally a suture at the coronal aspect of each end of the graft is enough to secure it. The graft is covered with adhesive foil, which in turn is covered with a hard-setting



f **I G . 20-35. A,** Stripping of the tissue and loss of alveolar bone from the labial aspect of the lower right central incisor are related to an abnormal frenum attachment and shallow vestibule. **B,** An incision has been made and the connective tissue and muscle attachments have been freed by blunt dissection. C, One week after the operation there is evidence of granulation tissue. D, An improvement is evident in the gingival contour of the tissue surrounding the right central incisor.

periodontal dressing. When the patient returns 7 to 10 days after surgery, the dressing is removed, the area is debrided and irrigated, and the sutures are removed. A second dressing is usually placed for another 5 to 7 days.

When the second dressing is removed, the area is again debrided, and the teeth are polished. At this time the graft should be firmly fixed to the underlying receptor site, and the epithelial covering should be continuous with that of the contiguous gingiva and oral mucosa. The patient is instructed to gently clean away any debris from the area with a cotton ball soaked in warm water and to continue the interdental flossing routine. Normal brushing can usually be reintroduced between 21 and 25 days after the graft is placed.

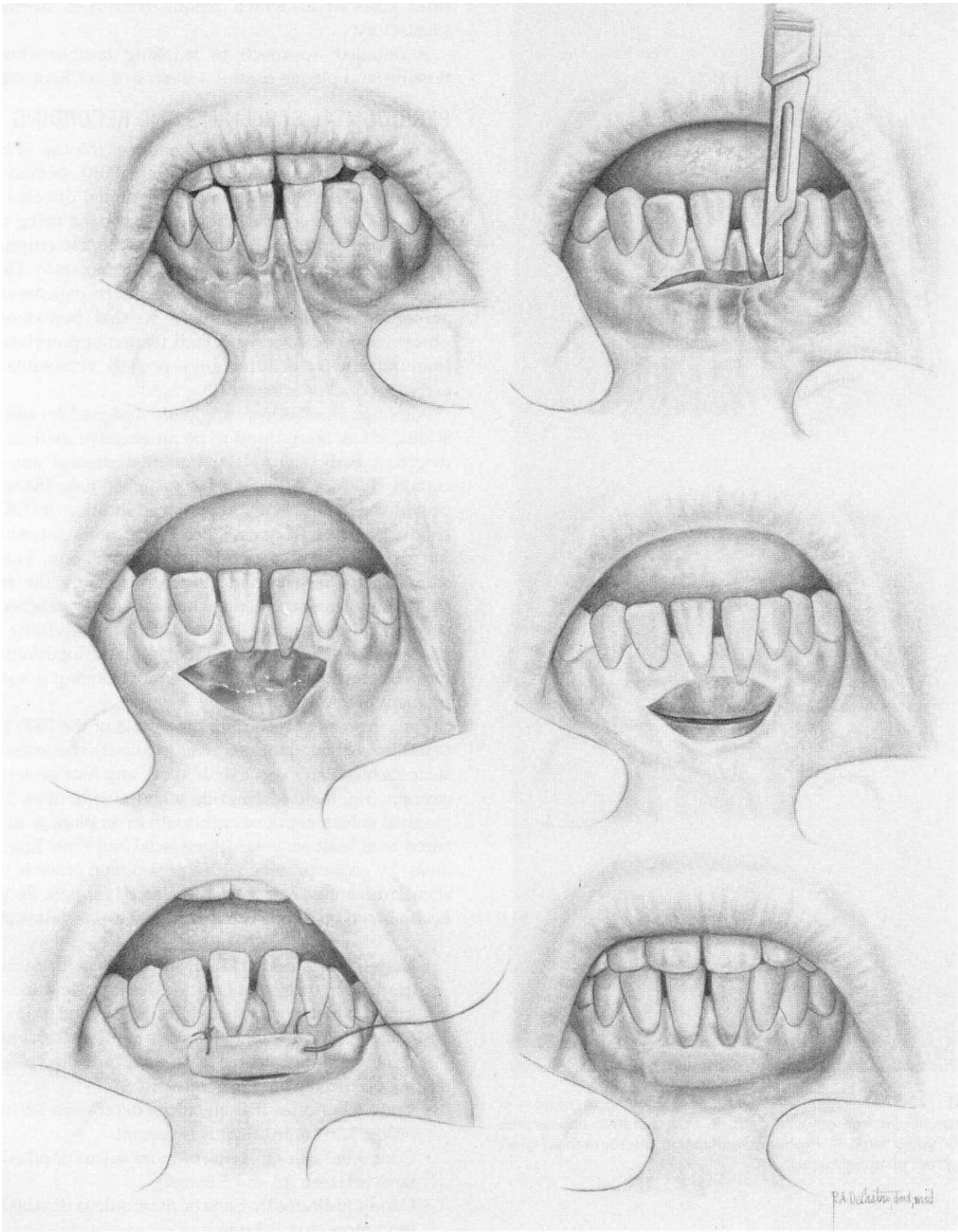
CLINICAL ASSESSMENT OF ORAL CLEANLINESS AND PERIODONTAL DISEASE

PLAQUE CONTROL RECORD

O'Leary, Drake, and Naylor developed the plaque control record to give the dentist, hygienist, and dental educator a simple method of recording the presence of plaque on individual tooth surfaces (mesial, distal, facial, and lingual).[80]

At the initial appointment a suitable disclosing solution, such as Bismarck brown, is painted on all exposed tooth surfaces. After the patient has rinsed, the operator, using an explorer, examines each stained surface for soft accumulations at the dentogingival junction. When found, these accumulations are recorded by making a dash in the appropriate spaces on the record form. Fig. 20-38, A, shows a form completed at the patient's first appointment for learning plaque control. No attempt is made to differentiate between varying amounts of plaque on tooth surfaces.

After all teeth are examined and scored, an index can be derived when the number of plaque-containing surfaces is divided by the total number of available surfaces. The same procedure is carried out at subsequent appointments to determine the patient's progress in learning and carrying out the prescribed oral hygiene procedures. Fig. 20-38, B, shows a patient's progress from the initial assessment to the point



f **I C 20-36.** The free gingival graft procedure may be considered for children and adults when there is a shallow vestibular fornix and a high midline frenum attachment.

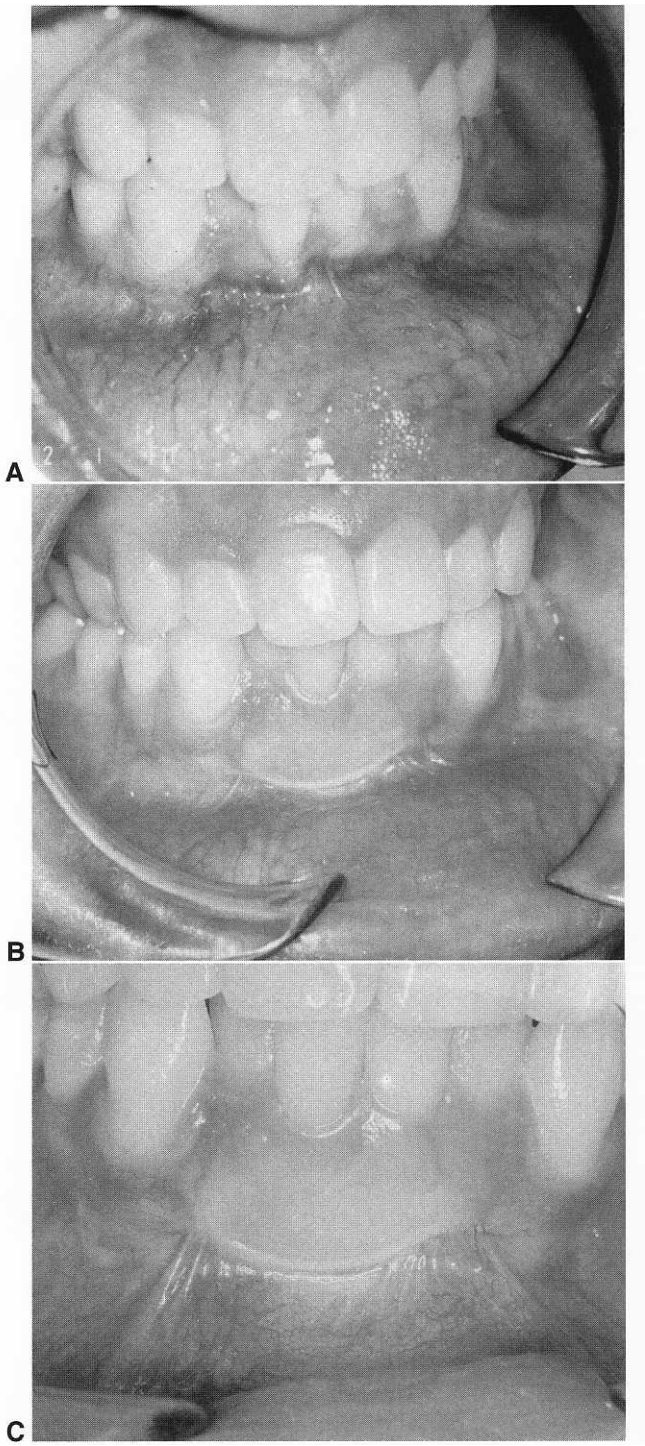


FIG **20-37.** A, Initial presentation of patient to receive a mandibular free gingival graft. **B,** Postoperative appearance $3\frac{1}{2}$ years later. C, Higher-magnification photograph of graft 5 years postoperatively.

(fifth session) at which plaque control is deemed satisfactory.

A detailed approach to teaching toothbrushing, flossing, and plaque control is described in Chapter 11.

PERIODONTAL SCREENING AND RECORDING

As we noted in Chapter 1, we endorse the use of the periodontal screening and recording (PSR) method to facilitate the early detection of periodontal diseases in children. Clerehugh and Tugnait recommend using the PSR for pediatric dental patients following the eruption of the permanent incisors and first molars.[8'] They suggest routine screening for new pediatric patients and at regular recare appointments so that periodontal problems are detected early and treated appropriately. Immunodeficient children are especially vulnerable to early loss of bone support.

Although the PSR was originally designed for use in adults, it has been found to be an effective method of detecting early signs of periodontal disease among certain children who may be at higher risk. Piazzini provided a favorable report on the application of PSR in children and adolescents.[82] The PSR is not a substitute for a comprehensive periodontal examination, but it should help the clinician efficiently identify the few child or adolescent patients who need a comprehensive periodontal evaluation. In addition, the possibility of false readings caused by gingival pseudopocketing must be considered, because pseudopocketing is more frequent in young patients.

For purposes of recording the results of the PSR, the dentition is divided into six areas (sextants): two anterior sextants (maxillary and mandibular), and four posterior sextants (right and left maxillary and mandibular). The gingival sulcus depth of each tooth or implant is measured in at least six areas (three facial and three lingual areas) by gentle probing with a periodontal probe in the standard manner. Each sextant is scored for periodontal health based on a code number from 0 to 4 as follows:

- Code 0 indicates that all sulcus depths in the sextant are 3.5 mm or less, and no calculus is present and no gingival bleeding occurred from the gentle probing.
- Code 1 indicates that all sulcus depths are 3.5 mm or less and no calculus is present, but some bleeding occurred from the gentle probing.
- Code 2 indicates that all sulcus depths are 3.5 mm or less and some calculus is present.
- Code 3 indicates that one or more sulcus depth(s) is (are) between 3.5 and 5.5 mm.
- Code 4 indicates that one or more sulcus depth(s) is (are) more than 5.5 mm.

In addition to the code number, an asterisk (*) may accompany the code score to indicate furcation

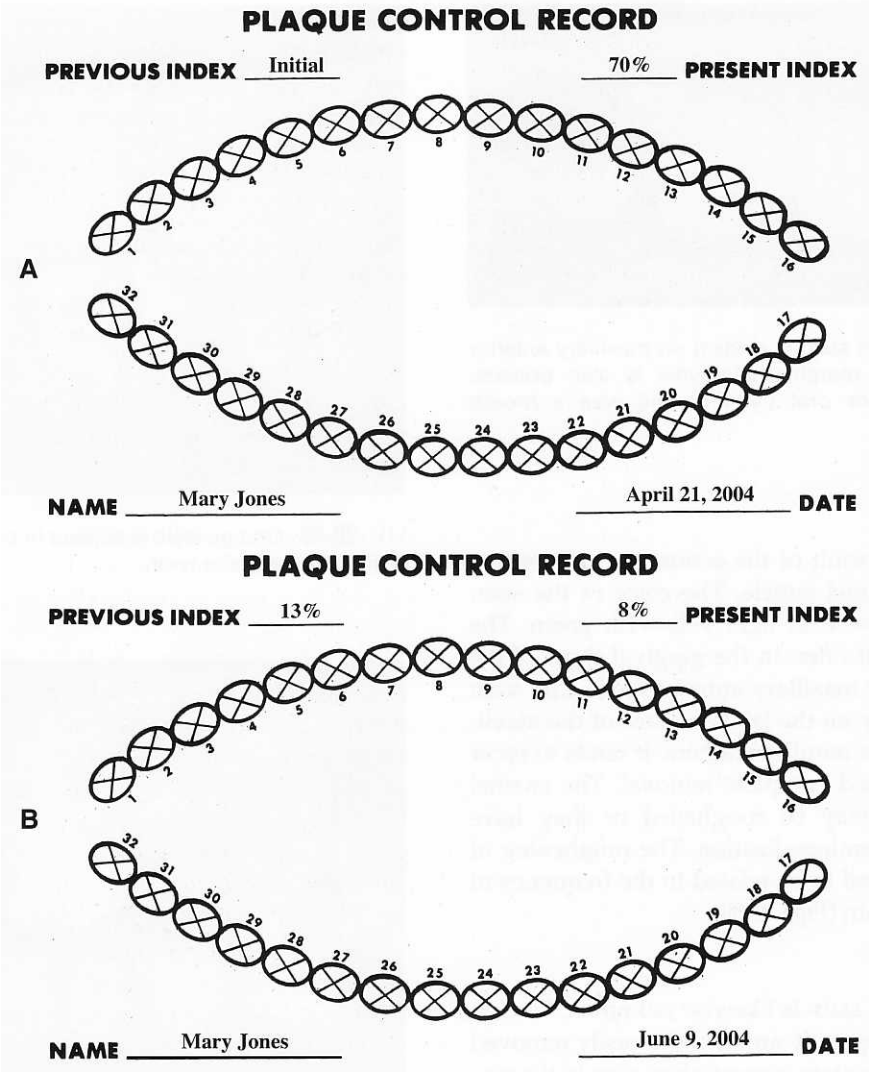


FIG. 20-38. **A,** Plaque accumulations recorded at the initial control appointment. **B,** Plaque accumulations recorded at the fifth session.

involvement, abnormal tooth mobility, mucogingival problems, or significant gingival recession.

Appropriate preventive care, oral hygiene instruction, and therapy (plaque and calculus removal) are indicated, as required, for patients who receive PSR code scores of 2, 1, or O. Patients receiving code scores of 3 or 4 require more comprehensive periodontal evaluation and treatment.

EXTRINSIC STAINS AND DEPOSITS ON TEETH

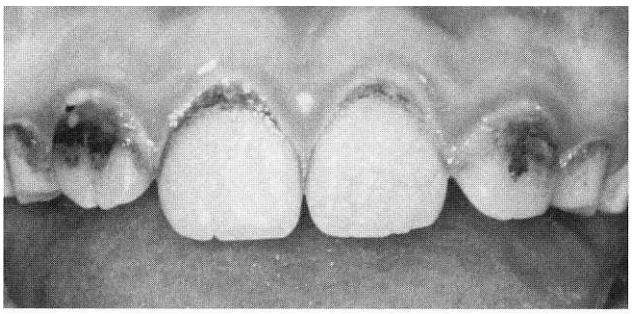
Previous work regarding the staining of children's teeth has been related primarily to studies of orange and green stain of the extrinsic type. It has been generally accepted that the stain has a microbial origin, although some reports have indicated that oral iron preparations or other medications may be responsible for an additional type of extrinsic staining.

Staining is generally believed to be caused by extrinsic agents, which can be readily removed from the surface of the teeth with an abrasive material. The agents that are responsible for staining are deposited in enamel defects or become attached to the enamel without bringing about a change in its surface.

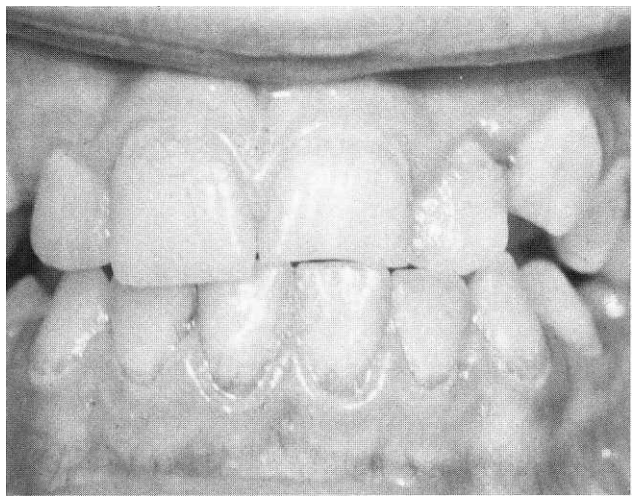
Pigmentation, in contrast to extrinsic staining, is associated with an active chemical change in the tooth structure, and the resultant pigment cannot be removed without alteration of the tooth structure.

GREEN STAIN

The cause of green stain, which is most often seen on the teeth of young persons, is unknown, although it is



f **I G 20-39.** Dark green stain is evident on maxillary anterior teeth. Papillary and marginal gingivitis is also present. The patient had poor oral hygiene and was a mouth breather.



F **I G 20-40.** Orange stain is evident in the gingival third of the mandibular anterior teeth.

believed to be the result of the action of chromogenic bacteria on the enamel cuticle. The color of the stain varies from dark green to light yellowish green. The deposit is seen most often in the gingival third of the labial surface of the maxillary anterior teeth. The stain collects more readily on the labial surface of the maxillary anterior teeth in mouth breathers. It tends to recur even after careful and complete removal. The enamel beneath the stain may be roughened or may have undergone initial demineralization. The roughening of the surface is believed to be related to the frequency of recurrence of the stain (Fig. 20-39).

ORANGE STAIN

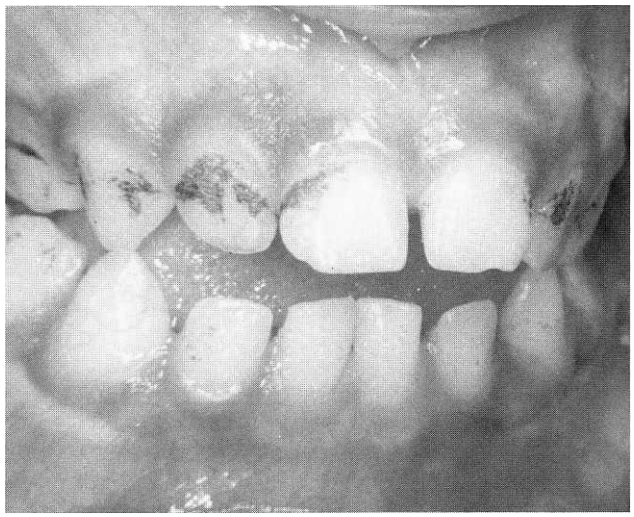
The cause of orange stain is likewise unknown. Orange stain occurs less frequently and is more easily removed than green stain. The stain is most often seen in the gingival third of the tooth and is associated with poor oral hygiene (Fig. 20-40).

BLACK STAIN

A black stain occasionally develops on the primary or permanent teeth of children, but it is much less common than the orange or green type (Fig. 20-41). The stain may be seen as a line following the gingival contour or it may be apparent in a more generalized pattern on the clinical crown, particularly if there are roughened or pitted areas. The black type of stain is difficult to remove, especially if it collects in pitted areas. Many children who have black stain are relatively free from dental caries.

REMOVAL OF EXTRINSIC STAINS

Extrinsic stains can be removed by polishing with a rubber cup and flour pumice. If the stain is resistant and difficult to remove, the excess water should be blotted from the pumice and the teeth should be dried before the polishing procedure is performed. Because stains



f **I G 20-41.** Black stain is evident on the primary teeth. The stain is difficult to remove, particularly when it collects in roughened areas of the tooth.

are most often seen in a mouth in which there is poor oral hygiene, improving the oral hygiene will minimize the recurrence of the stain.

PIGMENTATION CAUSED BY STANNOUS FLUORIDE APPLICATION

During the first clinical trials involving the topical application of an 8% stannous fluoride solution, certain areas of the tooth became discolored. A characteristic pigmentation of both carious and precarious lesions has been found to be associated with exposure to stannous fluoride (Fig. 20-42).

CALCULUS

Calculus is not often seen in preschool children, and even in children of grade school age it occurs with much lower frequency than in adult patients. A low caries incidence is related to high calculus incidence.

Bhat has reported findings in 14- to 17-year-old children who participated in the 1986-1987 National Survey of Oral Health.[83] Supragingival calculus was observed in nearly 34% of the children and subgingival calculus in approximately 23%. Both types show a predilection for molars in the maxilla and incisors and canines in the mandible.

Children with mental retardation often have accumulations of calculus on their teeth. This accumulation may be related to abnormal muscular function, a soft diet, poor oral hygiene, and stagnation of saliva.

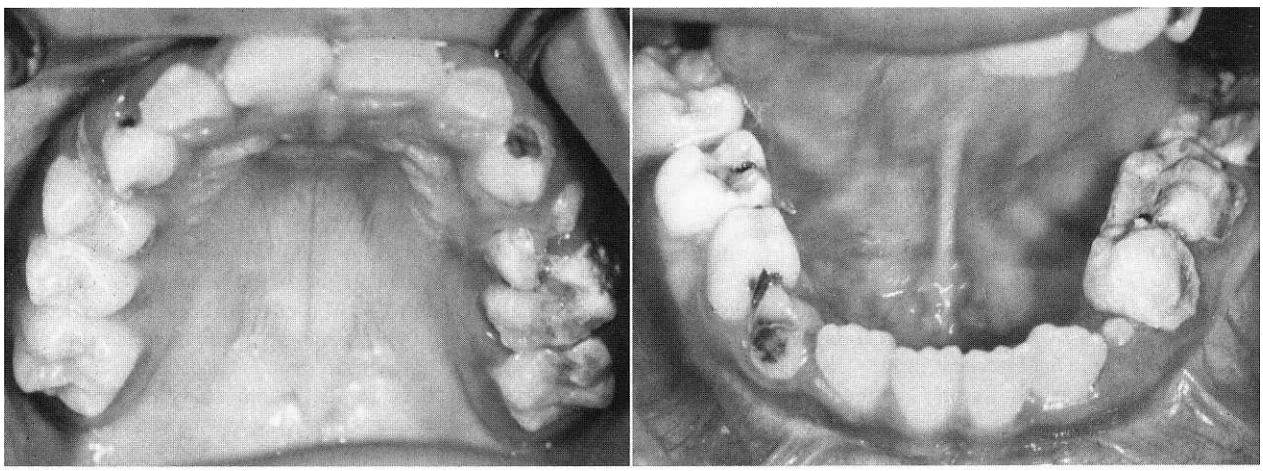
The observations of Turesky, Renstrup, and Glickman regarding early calculus formation in children and adults substantiate those of others and indicate that calculus begins as a soft, adherent, bacteria-laden plaque that undergoes progressive calcification.[84] They observed calculus formation on cellulose acetate strips that were fixed in children's mouths. Plaque material that accumulated on the strips underwent progressive hardening. A soft plaque material consisted for the most part of bacteria appearing as a dense meshwork of diffusely distributed gram-negative cocci with occasional rod forms. Filamentous or thread-shaped organisms were scarce. Leukocytes and epithelial cells were also scattered within the amorphous matrix.

Gross accumulations of calculus are occasionally seen in teenaged and preteenaged children (Figs. 20-43 and 20-44). Traiger reported on a 14-year-old girl who had excessive deposits of supragingival and subgingival calculus.[SS] The calculus covered the labial surfaces of the anterior teeth, extended into the mucobuccal fold, and covered the attached gingiva.

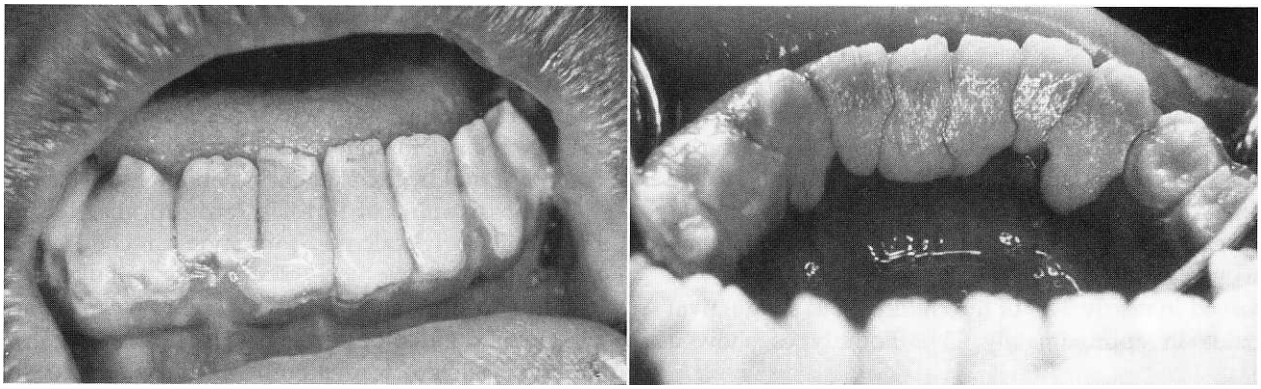
Supragingival deposits of calculus occur most frequently and in greater quantity on the buccal surfaces of the maxillary molars and the lingual surfaces of the mandibular anterior teeth. These areas are near the openings of the major salivary glands. Local factors are unquestionably important in the initiation of calculus formation.



[I G **20-42.** Pigmentation of carious and precarious enamel. The color ranges from light brown to black, the darker pigmentation being evident in the area of greatest caries involvement. The pigmentation is evidence of caries arrest following stannous fluoride application.



[I G 20-43. Gross accumulations of calculus are seen on the clinical crowns of the posterior teeth on the left side of the mouth. The fact that the child chewed mostly on his right side accounts partially for the greater cleanliness on that side.



f IG. 20-44. Young adult with calculus covering the labial and lingual surfaces of the mandibular anterior teeth. After the calculus was removed, amelogenesis imperfecta was apparent. The rough surface of the crown favorably influenced the deposition of calculus.

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CHAPTER

21

Management of Trauma to the Teeth and Supporting Tissues

RALPH E. McDONALD
DAVID R. AVERY
JEFFREY A. DEAN

انتشارات
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CHAPTER OUTLINE

HISTORY AND EXAMINATION
- History of the Injury
- Clinical Examination
- Radiographic Examination

EMERGENCY TREATMENT OF SOFT TISSUE INJURY

EMERGENCY TREATMENT AND TEMPORARY RESTORATION OF FRACTURED TEETH WITHOUT PULP EXPOSURE
- Fragment Restoration (Reattachment of Tooth Fragment)
- Temporary Bonded Resin Restoration
- Orthodontic Band
- Stainless Steel Crown

TREATMENT OF VITAL PULP EXPOSURES
- Direct Pulp Cap
- Pulpotomy
- Pulpectomy with Endodontic Treatment
- Therapy to Stimulate Root Growth and Apical Repair Subsequent to Pulpal Necrosis in Anterior Permanent Teeth (Apexification)

REACTION OF THE TOOTH TO TRAUMA
- Pulpal Hyperemia
- Internal Hemorrhage
- Calcific Metamorphosis of the Dental Pulp (Progressive Canal Calcification or Dystrophic Calcification)
- Internal Resorption
- Peripheral (External) Root Resorption
- Pulpal Necrosis
- Ankylosis

RESTORATION OF FRACTURED TEETH
- Esthetic Bonded Composite Resin Restoration

REACTION OF PERMANENT TOOTH BUDS TO INJURY
- Hypocalcification and Hypoplasia
- Reparative Dentin Production
- Dilaceration

DISPLACEMENT OF PRIMARY AND PERMANENT ANTERIOR TEETH (LUXATION)
- Intrusion and Extrusion of Teeth
  - *Primary teeth*
  - *Permanent teeth*
- Avulsion and Replantation
  - *Stabilization of replanted teeth*
  - *Endodontic management of replanted teeth*
- Management of Root Fractures
- Other Displacement Injuries of Teeth Requiring Stabilization

MANAGEMENT OF ORAL BURNS
- Nature of the Injury
- Treatment

TRAUMA PREVENTION

trauma with accompanying fracture of a permanent incisor is a tragic experience for the young patient and is a problem whose management requires experience, judgment, and skill perhaps unequaled by any other portion of the dentist's practice. The dentist whose counsel and treatment are sought after a trauma is obligated either to treat the patient with all possible means or to immediately refer the patient to a specialist. The oral and emotional health of the young patient is involved, and the child's appearance, marred by an unsightly oral injury, must be restored to normal as soon as possible to relieve the consciousness of being different from other children. Slack and Jones observed that the progress of children in school and their behavior elsewhere, as well as their psychologic well-being, can be adversely influenced by an injury to the teeth that causes an unsightly fracture.[1] In addition, the short- and long-term costs associated with managing trauma to the oral and perioral structures can be large. Borum and Andreasen estimate that the yearly cost from traumatic dental injuries in Denmark ranges from US $2 million to US $5 million per 1 million inhabitants per year.[2]

Injuries to the teeth of children or adults present unique problems in diagnosis and treatment. The diagnosis of the extent of the injury after a blow to a tooth, regardless of loss of tooth structure, is difficult and often inconclusive. Trauma to a tooth is invariably followed by pulpal hyperemia, the extent of which cannot always be determined by available diagnostic methods. Congestion and alteration in the blood flow in the pulp may be sufficient to initiate irreversible degenerative changes, which over time can cause pulpal necrosis. In addition, the apical vessels may have been severed or damaged enough to interfere with the normal reparative process. Treatment of injuries causing pulp exposure or tooth displacement are particularly challenging, because the prognosis of the involved tooth is often uncertain.

The treatment of fractured teeth, particularly in young patients, is further complicated by the often difficult but extremely important restorative procedure. Although the dentist may prefer to delay the restoration because of a questionable prognosis for the pulp, often a malocclusion can develop within a matter of days as a result of a break in the normal proximal contact with adjacent teeth. Adjacent teeth may tip into the area created by the loss of tooth structure (Fig. 21-1). This loss of space will create a problem when the final restoration is contemplated. There must often be a compromise of an ideal esthetic appearance, at least in the initial restoration, because the prognosis is questionable or because the tooth is young and has a large pulp or is still in the stage of active eruption.



FIG 21-1. Loss of the incisal third of the maxillary left central incisor has allowed the right central incisor to tip into the area. Satisfactory restoration of the fractured tooth cannot be accomplished until the space is regained. Orthodontic treatment will be required.

Often the likelihood of success depends on the rapidity with which the tooth is treated after the injury, regardless of whether the procedure involves protecting a large area of exposed dentin or treating a vital pulp exposure. Several factors can be considered common to all types of injury to the anterior teeth. These important considerations should become a checklist invariably used by the dentist in the diagnosis of and treatment planning for traumatic injuries.

The International Association of Dental Traumatology reports that one out of every two children sustains a dental injury, most often between the ages of 8 and 12. They suggest that in most cases of dental trauma a rapid and appropriate treatment can lessen its impact from both an oral and an esthetic standpoint. To that end the association has developed guidelines for the evaluation and management of traumatic dental injuries, which is available at the following web site: http://www.iadtdentaltrauma.org/Trauma/dental_trauma.htm.

HISTORY AND EXAMINATION

The routine use of a clinical evaluation sheet for injured anterior teeth is helpful during the initial examination and subsequent examinations of an injured tooth (Fig. 21-2). The form, which becomes a part of the patient's record, serves as a checklist of important questions that must be asked and observations that must be made by the dentist and the auxiliary personnel during the examination of the child.

HISTORY OF THE INJURY

The time of the injury should first be established. Unfortunately, many patients do not seek professional advice and treatment immediately after an injury. Occasionally the accident is so severe that dental

ASSESSMENT OF ACUTE TRAUMATIC INJURIES	PATIENT NAME: DATE OF BIRTH:
DATE: TIME:	REFERRED BY:

HISTORY

MEDICAL HISTORY:	
ALLERGIES:	DATE OF LAST TETANUS INNOCULATON:
DATE AND TIME OF INJURY:	TIME LAPSED SINCE INJURY:
WHERE INJURY OCCURRED:	
HOW INJURY OCCURRED:	

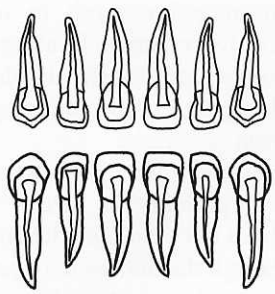
Check if present and describe	MANAGEMENT PRIOR TO EXAM
☐ Nondental injuries	By whom:
☐ Loss of consciousness	Describe:
☐ Altered orientation/mental status	
☐ Hemorrhage from nose/ears	
☐ Headache/nausea/vomiting	
☐ Neck pain	
☐ Spontaneous dental pain	
☐ Pain on mastication	
☐ Reaction to thermal changes	
☐ Previous dental trauma	
☐ Other complaints	

EXTRAORAL EXAM

Check if present and describe	OTHER FINDINGS/COMMENTS:
☐ Facial fractures	
☐ Lacerations	
☐ Contusions	
☐ Swelling	
☐ Abrasions	
☐ Hemorrhage/drainage	
☐ Foreign bodies	
☐ TMJ deviation/asymmetry	

INTRAORAL EXAMINATION

Check if injured and describe	DIAGRAM OF INJURIES
☐ Lips	
☐ Frenae	
☐ Buccal mucosa	
☐ Gingivae	
☐ Palate	
☐ Tongue	
☐ Floor of mouth	
Occlusion	
☐ Molar classification R___ L___	
☐ Canine classification R___ L___	
☐ Overbite (%)_____	
☐ Overjet (mm)_____	
☐ Crossbite Y N	
☐ Midline deviation Y N	
☐ Interferences Y N	



f **I G 21-2.** Clinical evaluation sheet for injured anterior teeth. *(Adapted from American Academy of Pediatric Dentistry:* Pediatr Dent *24(7 suppl):95-96, 2002.)*

DENTAL INJURIES	TOOTH NUMBER					
	AVULSION — Extraoral time					
	Storage medium					
	INFRACTION					
	CROWN FRACTURE					
	PULP EXPOSURE — Size					
	Appearance					
	COLOR					
	MOBILITY (mm)					
	PERCUSSION					
	LUXATION — Direction					
	Extent					
	PULP TESTING — Electric					
	Thermal					
	CARIES/PREVIOUS RESTORATIONS					
RADIOGRAPHS	PULP SIZE					
	ROOT DEVELOPMENT					
	ROOT FRACTURE					
	PERIODONTAL LIGAMENT SPACE					
	PERIAPICAL PATHOLOGY					
	ALVEOLAR FRACTURE					
	FOREIGN BODY					
	DEVELOPMENTAL ANOMALY					
	OTHER					

TREATMENT	Check if performed and describe
☐	Soft tissue management
☐	Medication
☐	Pulp therapy
☐	Repositioning
☐	Stabilization
☐	Restoration
☐	Extraction
☐	Prescription
☐	Referral
☐	Other

INSTRUCTIONS AND DISPOSITION	Check if discussed
☐	Diet
☐	Hygiene
☐	Pain
☐	Swelling
☐	Infection
☐	Prescription
☐	Complications:
☐	Damage to developing teeth
☐	Abnormal position/ankylosis
☐	Tooth loss
☐	Pulp damage to injured teeth
☐	Other:
☐	Follow-up:
☐	Other

SUMMARY

Subsequent visit No. 1 Date_____

1. Pulpal response (7 8 / 9 10) ☐☐ / ☐☐
2. Radiographic exam (26 25 / 24 23) ☐☐ / ☐☐
3. Treatment and comments: _____

Subsequent visit No. 2 Date_____

1. Pulpal response (7 8 / 9 10) ☐☐ / ☐☐
2. Radiographic exam (26 25 / 24 23) ☐☐ / ☐☐
3. Treatment and comments: _____

Subsequent visit No. 3 Date_____

1. Pulpal response (7 8 / 9 10) ☐☐ / ☐☐
2. Radiographic exam (26 25 / 24 23) ☐☐ / ☐☐
3. Treatment and comments: _____

Subsequent visit No. 4 Date_____

1. Pulpal response (7 8 / 9 10) ☐☐ / ☐☐
2. Radiographic exam (26 25 / 24 23) ☐☐ / ☐☐
3. Treatment and comments: _____

FIG 21-2, contd. See legend on opposite page.

treatment cannot be started immediately because other injuries have higher priority. Davis and Vogel emphasized that a force strong enough to fracture, intrude, or avulse a tooth is also strong enough to result in cervical spine or intracranial injury. The dentist must be particularly alert to such potential problems, be prepared ahead of time to make a neurologic assessment, and make appropriate medical referral when indicated without delay.[1] The patient should be assessed for nausea, vomiting, drowsiness, or possible cerebral spinal fluid leakage from the nose and ears, which would be indicative of a skull fracture. In addition, the patient should be evaluated for lacerations and facial bone fractures, and even such things as baseline temperature, pulse, blood pressure, and respiratory rate could be considered as information to be gathered prior to addressing the dental needs of the patient. Finally, Davis[1] recommends a quick cranial nerve evaluation involving the following four areas:

- Extraocular muscles are intact and functioning appropriately; that is, the patient can track a finger moving vertically and horizontally through the visual field with the eyes remaining in tandem.
- Pupils are equal, round, and reactive to light with accommodation.
- Sensory function is normal as measured through light contact to various areas of the face.
- Symmetry of motor function is present, as assessed by having the patient frown, smile, move the tongue, and perform several voluntary muscular movements.

The prognosis of an injured tooth depends logically, often to a great extent, on the time that has elapsed between the occurrence of the accident and the initiation of emergency treatment. This is particularly true in cases of pulp exposure, for which pulp capping or pulpotomy would be the procedure of choice. Rusmah treated 123 traumatized permanent incisors and monitored them over a 24-month period. His findings suggest that the interval between trauma and emergency treatment is directly related to the severity of the injury and the dental awareness of the patients.[1] Furthermore, the prognosis of the injured teeth maintaining pulpal vitality diminished when treatment was delayed. The loss of vitality of some injured teeth occurred as early as 3 months and as late as 24 months after the injury, which justifies a long follow-up period after injury.

For practical and especially economic reasons, Andreasen et al have attempted to classify pulpal and periodontal healing of traumatic dental injuries based on the effect of treatment delay.[1] They developed three major categories of treatment timing: acute treatment (i.e., within a few hours), subacute treatment (i.e., within the first 24 hours), and delayed treatment (i.e., after the first 24 hours). Unfortunately, they lament that we presently have a rather limited knowledge of the effect of treatment delay on wound healing, with very little data available in the literature.

Taking a complete dental history can help the dentist learn of previous injuries to the teeth in the area. Repeated injuries to the teeth are not uncommon in children with protruding anterior teeth and in those who are active in athletics. In these patients the prognosis may be less favorable. The dentist must rule out the possibility of a degenerative pulp or adverse reaction of the supporting tissues as a result of previous trauma.

The patient's complaints and experiences after the injury are often valuable in determining the extent of the injury and in estimating the ability of the injured pulp and supporting tissues to overcome the effects of the injury. Pain caused by thermal change is indicative of significant pulpal inflammation. Pain occurring when the teeth are brought into normal occlusion may indicate that the tooth has been displaced. Such pain could likewise indicate an injury to the periodontal and supporting tissues. The likelihood of eventual pulpal necrosis increases if the tooth is mobile at the time of the first examination; the greater the mobility, the greater the chance of pulp death.

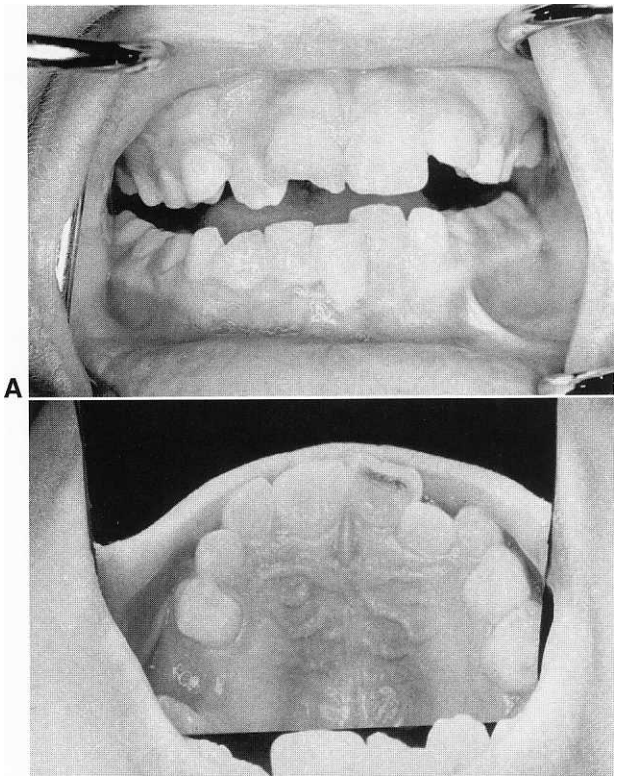
Trauma to the supporting tissues may cause sufficient inflammation to initiate external root resorption. In instances of severe injury, teeth can be lost as a result of pathologic root resorption and pulpal degeneration.

CLINICAL EXAMINATION

The clinical examination should be conducted after the teeth in the area of injury have been carefully cleaned of debris. When the injury has resulted in a fracture of the crown, the dentist should observe the amount of tooth structure that has been lost and should look for evidence of a pulp exposure. With the aid of a good light, the clinical crown should be examined carefully for cracks and craze lines, the presence of which could influence the type of permanent restoration used for the tooth. With light transmitted through the teeth in the area, the color of the injured tooth should be carefully compared with that of adjacent uninjured teeth. Severely traumatized teeth often appear darker and reddish, although not actually discolored, which indicates pulpal hyperemia (Fig. 21-3). This appearance suggests that at some later time the pulp may undergo degenerative change terminating in pulpal necrosis.

The Ellis and Davey classification of crown fractures is useful in recording the extent of damage to the crown.[1] The following is a modification of their classification (Fig. 21-4):

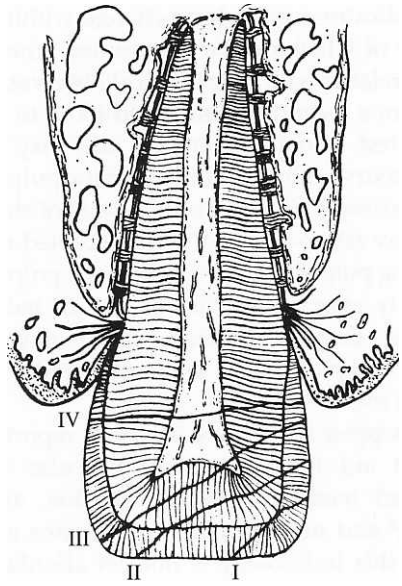
- Class I—Simple fracture of the crown involving little or no dentin



FIC **21-3. A,** Fractured central incisor appeared darker than the adjacent teeth 3 days after the injury. The pulp was not exposed, but it was in a state of shock and did not respond to pulp tests. **B,** The reddish appearance of the dentin is evidence of severe hyperemia within the pulp tissue. The prognosis for retaining vitality of the pulp is poor.

Class II—Extensive fracture of the crown involving considerable dentin but not the dental pulp
Class III—Extensive fracture of the crown with an exposure of the dental pulp
Class IV—Loss of the entire crown

A vitality test of the injured tooth should be performed, and the teeth in the immediate area, as well as those in the opposing arch, should be tested. The best prediction of continued vitality of the pulp of a damaged or traumatized tooth is the vital response to electric pulp testing at the time of the initial examination. A negative response, however, is not reliable evidence of pulp death because some teeth that give such a response soon after the injury may recover vitality after a time. When the electric pulp tester is used, the dentist should first determine the normal reading by testing an uninjured tooth on the opposite side of the mouth and recording the lowest number at which the tooth responds. If the injured tooth requires more current than does a normal tooth, the pulp may be undergoing degenerative change. If less current is



FIC. **21-4.** Classification of crown fractures of anterior teeth.

needed to elicit a response from a traumatized tooth, pulpal inflammation is usually indicated.

Many practitioners question the need for the electric vitality test immediately after the injury. Because the electrical stimulus has been shown to produce negligible additional pulpal irritation, its use is not contraindicated on this basis. However, the patient's measured responses to the test may be almost meaningless. The reliability of the electric pulp test depends on eliciting valid responses from the patient. The mere presence of this new, unknown instrument may create anxiety in children that hampers their ability to respond accurately to the test. Because an unscheduled emergency appointment for treatment of an injury is a new experience, it seems reasonable to introduce the child to the instrument during the first emergency visit when the child does not know what to expect. This gives the dentist an opportunity to allay the child's anxiety about the instrument during a time when the responses are not as important as they will be on subsequent visits. It should also be remembered that the electric pulp test is frequently unreliable even on normal teeth when apices are incompletely formed.

The thermal test is also somewhat helpful in determining the degree of pulpal damage after trauma. Although there are difficulties with the thermal test, it is probably more reliable in testing primary incisors in young children than the electric pulp test. Failure of a tooth to respond to heat is indicative of pulpal necrosis. The response of a tooth to a lower degree of heat than is necessary to elicit a response in adjacent teeth is an indication of inflammation. Pain occurring when ice is applied to a normal tooth will subside when the ice is removed. A more painful and often lingering reaction

to cold indicates a pathologic change within the pulp, the nature of which can be determined when the reaction is correlated with other clinical observations.

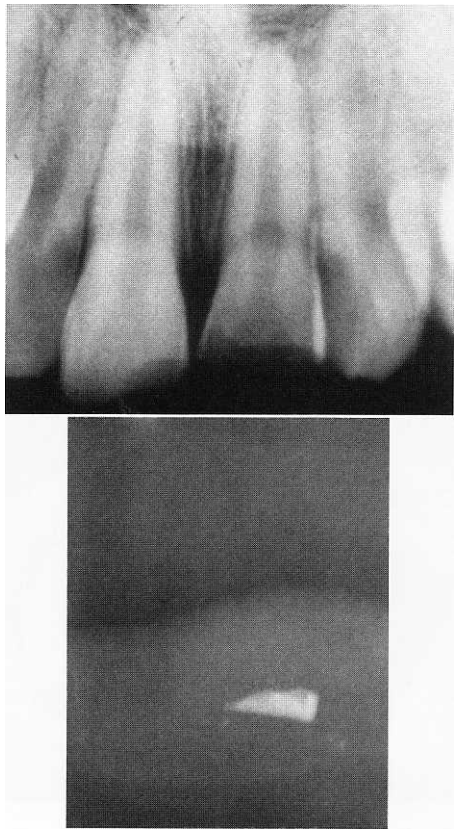
Failure of a recently traumatized tooth to respond to the pulp test is not uncommon and may indicate a previous injury with a resulting necrotic pulp. However, the traumatized tooth may be in a state of shock and as a result may fail to respond to the accepted methods of determining pulp vitality. The failure of a pulp to respond immediately after an accident is not an indication for endodontic therapy. Instead, emergency treatment should be completed, and the tooth should be retested at the next follow-up visit.

Laser Doppler flowmetry has been reported to be a significant aid in determining vascular vitality of traumatized teeth by Olgart, Gazelius, and Lindh-Stromberg[s] and more recently by Mesaros and Trope.' Although this technology is not yet affordably priced for dental offices, it may be in the future.

RADIOGRAPHIC EXAMINATION

The examination of traumatized teeth cannot be considered complete without a radiograph of the injured tooth, the adjacent teeth, and sometimes the teeth in the opposing arch. It may even be necessary to obtain a radiograph of the soft tissue surrounding the injury site in search of a fractured tooth fragment (Fig. 21-5). The relative size of the pulp chamber and canal should be carefully examined. Irregularities or an inconsistency in the size of the chamber or canal compared with that of adjacent teeth may be evidence of a previous injury. This observation is important in determining the immediate course of treatment. In young patients the stage of apical development often indicates the type of treatment, just as the size of the coronal pulp and its proximity to the area of fracture influence the type of restoration that can be used. A root fracture as a result of the injury or one previously sustained can be detected by a careful examination of the radiograph. However, the presence of a root fracture may not influence the course of treatment, particularly if the fracture line is in the region of the apical third. Teeth with root fractures in this area rarely need stabilization, and a fibrous or calcified union will usually result. If teeth have been discernibly dislocated, with or without root fracture, two or three radiographs of the area at different angles may be needed to clearly define the defect and aid the dentist in deciding on a course of treatment.

Another value of the radiograph is that it provides a record of the tooth immediately after the injury. Frequent, periodic radiographs reveal evidence of continued pulp vitality or adverse changes that take place within the pulp or the supporting tissues. In young teeth in which the pulp recovers from the initial trauma, the pulp chamber and canal will decrease in size coincident with the normal formation of secondary dentin. After a period of time, an inconsistency in the true size or contour of the pulp chamber or canal compared with that of adjacent teeth may indicate a developing pathologic condition.



I **I I 21-5.** A radiograph with a reduced exposure time (25% of the usual time) was useful in detecting where this fractured tooth fragment was located within the patient's lip.

In cases in which more complex facial injuries have occurred or jaw fractures are suspected, extraoral films may also be necessary to help identify the extent and location of all injury sequelae. Oblique lateral jaw radiographs and panoramic films are often useful adjuncts to this diagnostic process.

EMERGENCY TREATMENT OF SOFT TISSUE INJURY

Injury to the teeth of children is often accompanied by open wounds of the oral tissues, abrasion of the facial tissues, or even puncture wounds. The dentist must recognize the possibility of the development of tetanus after the injury and must carry out adequate first-aid measures.

Children with up-to-date active immunization are protected by the level of antibodies in their circulation produced by a series of injections of tetanus toxoid.

Primary immunization is usually a part of medical care during the first 2 years of life. However, primary immunization cannot be assumed but must be confirmed by examining the child's medical record.

When the child who has had the primary immunization receives an injury from an object that is likely to have been contaminated, the antibody-forming mechanism may be activated with a booster injection of toxoid. An unimmunized child can be protected through passive immunization or serotherapy with tetanus antitoxin (tetanus immune globulin, or TIG).

The dentist examining the child after an injury should determine the child's immunization status, carry out ade quate debridement of the wound, and, when indicated, refer the child to the family physician. Tetanus is often fatal, and preventive measures must be taken if there is a possibility that an injured child is not adequately immunized.

Debridement, suturing, and/or hemorrhage control of open soft tissue wounds should be carried out as indicated. Working with an oral and maxillofacial surgeon or a plastic surgeon may also be indicated.

EMERGENCY TREATMENT AND TEMPORARY RESTORATION OF FRACTURED TEETH WITHOUT PULP EXPOSURE

A trauma to a tooth that causes a loss of only a small portion of enamel should be treated as carefully as one in which greater tooth structure is lost. The emergency treatment of minor injuries in which only the enamel is fractured may consist of no more than smoothing the rough, jagged tooth structure. However, without exception a thorough examination should be conducted as previously described. The patient should be reexamined at 2 weeks and again at 1 month after the injury. If the tooth appears to have recovered at that time, continued observation at the patient's regular recall appointments should be the rule.

Sudden injuries with a resultant extensive loss of tooth structure and exposed dentin require an immediate temporary restoration or protective covering, in addition to the complete diagnostic procedure. In this type of injury, initial pulpal hyperemia and the possibility of further trauma to the pulp by pressure or by thermal or chemical irritants must be reduced. In addition, if normal contact with adjacent or opposing teeth has been lost, the temporary restoration or protective covering can be designed to maintain the integrity of the arch. Because providing an adequate permanent restoration may depend on maintaining the normal alignment and position of teeth in the area, this part of the treatment is as important as maintaining the vitality of the teeth. Several restorations that will satisfy these requirements can easily be fabricated.

FRAGMENT RESTORATION (REATTACHMENT OF TOOTH FRAGMENT)

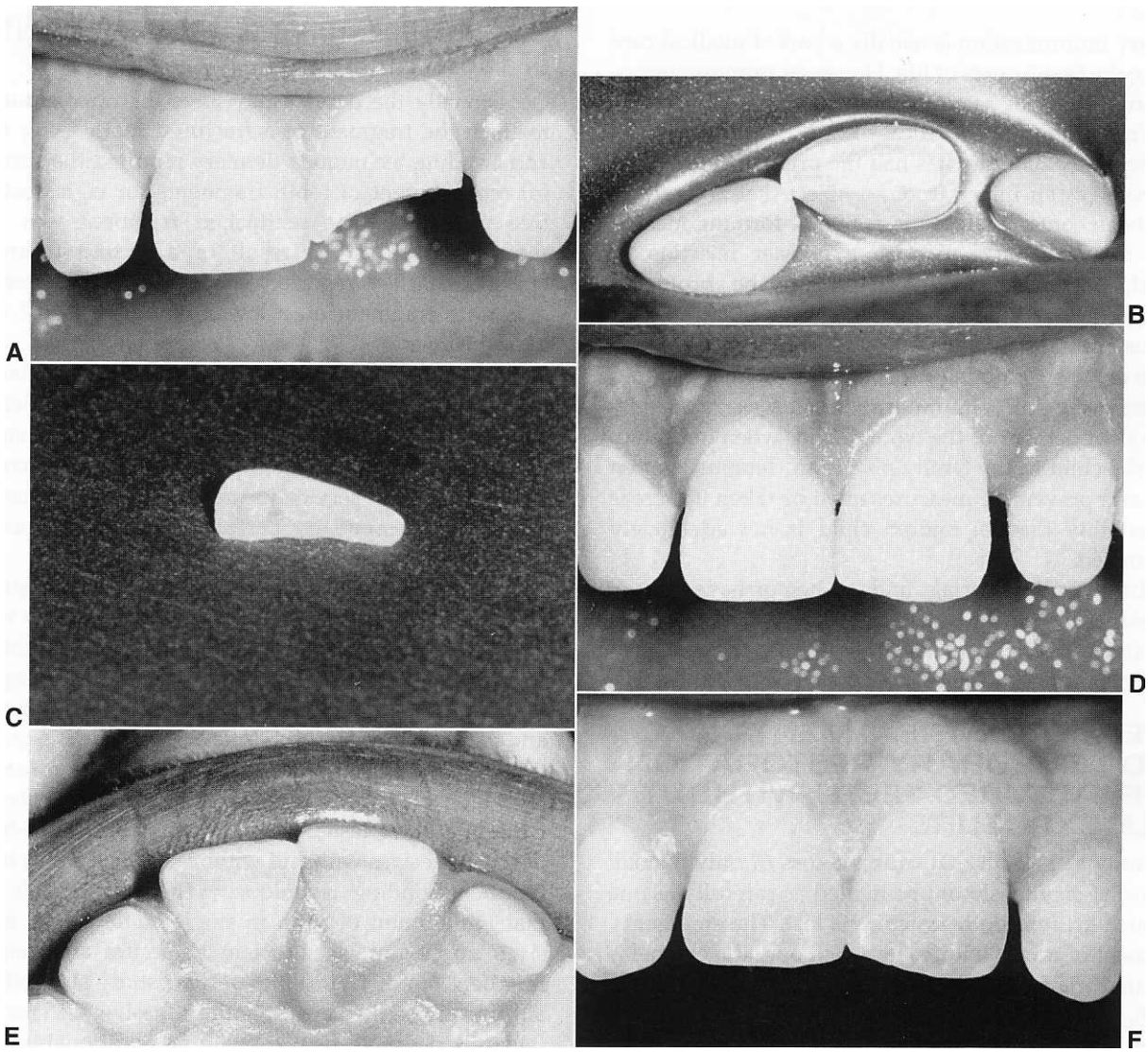
Occasionally the dentist may have the opportunity to reattach the fragment of a fractured tooth using resin and bonding techniques. Tennery reported the successful reattachment of tooth fragments for eight teeth in five patients.[10] One reattached fragment was subsequently lost as the result of a second traumatic episode. Starkey reported successful reattachment of one tooth fragment on a lower central incisor 2 days after the injury."

This procedure is atraumatic and seems to be the ideal method of restoring the fractured crown. Sealing the injured tooth and esthetically restoring its natural contour and color are accomplished simply and constitute an excellent service to the patient. The procedure provides an essentially perfect temporary restoration that may be retained a long time in some cases.

It is not often that the fractured tooth fragment remains intact and is recovered after an injury, but when this happens, the dentist may consider the reattachment procedure. The tooth requires no mechanical preparation because retention is provided by enamel etching and bonding techniques. If little or no dentin is exposed, the fragment and the fractured tooth enamel are etched and reattached with bonding agents and materials. Farik et al have tested the use of the new single-bottle dentin adhesives with and without unfilled resins in the fragment-bonding technique.[12] Their hypothesis was that the amount of resin in single-bottle dentin adhesives might not be sufficient to secure an adequate fragment bond. The results of their study showed that all but one of the seven dentin adhesive systems that they tested should be used with an additional unfilled resin when fractured teeth are restored by reattachment.

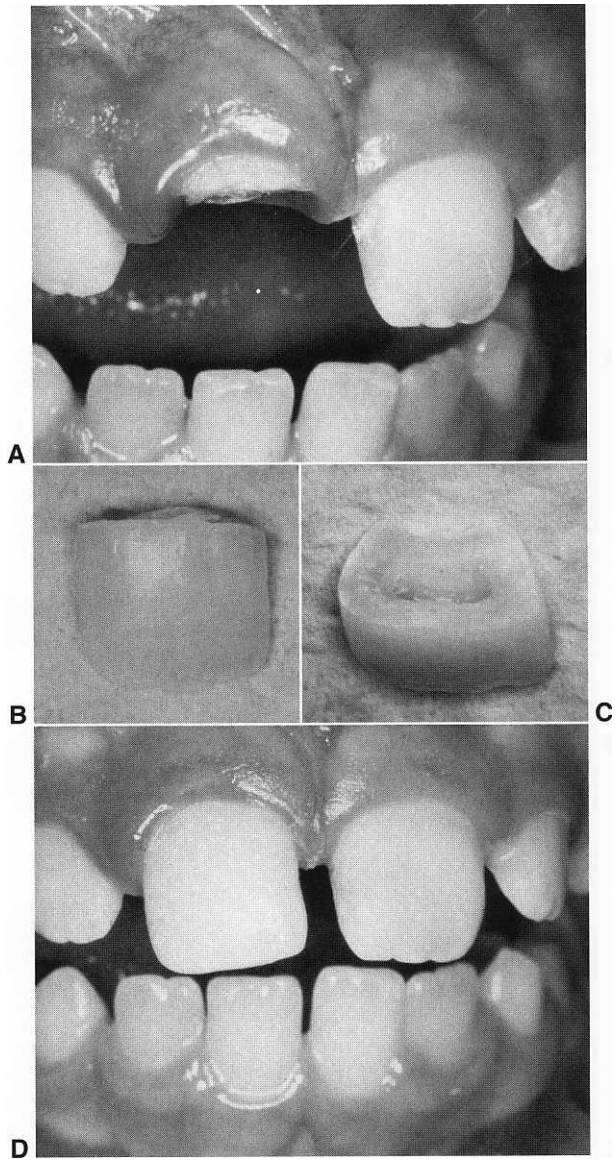
For cases in which considerable dentin is exposed or a direct pulp cap is indicated, some controversy currently exists about the best treatment to enhance the likelihood of maintaining pulpal vitality. Some believe that the meticulous use of bonding agents and materials to directly cap the exposed dentin and the pulp (if exposed) is best, whereas others believe that calcium hydroxide should be applied to the exposed dentin and pulp before completing the bonding procedure. The former method has been called the total-etch technique.

Fig. 21-6 illustrates the successful management of a class II fracture of the maxillary left central incisor in a 15-year-old boy who was treated approximately 2 hours after the injury. This reattached fragment has been a successful restoration for more than 7 years. The fragment has been reattached twice during this time, and each failure resulted from an additional direct blow to the tooth. The natural labial position of the tooth made it more susceptible to injury, especially in this very



FII **21-6.** A, Class II fracture of the crown of a maxillary left central incisor. **B,** The exposed dentin of the tooth is covered with hard-setting calcium hydroxide; all fractured enamel remains exposed. C, Part of the dentin is removed from the tooth fragment; the enamel is not disturbed. **D,** Fragment restoration immediately after removal of the rubber dam. **E,** Incisal view of the restored tooth illustrates the tooth's natural susceptibility to fracture because of its labial position. **F,** Restored tooth 22 months after injury.

athletically active patient. After the fragment was trial-seated to confirm a precise fit, the exposed dentin of the fractured tooth was covered with a thin layer of hard-setting calcium hydroxide that was allowed to remain as a sedative dressing between the tooth and the restored fragment. A portion of the dentin in the fragment was removed to provide space for the calcium hydroxide dressing. The fragment was then soaked in etchant, and the fractured area of the tooth was also etched well beyond the fracture site. After thorough rinsing and drying of all etched enamel, the fragment and the etched portion of the tooth were painted with a light-curing sealant material. Although no bonding agent was used here, its use is currently recommended. The selected shade of composite resin was used to fill the prepared void in the fragment, and it was then carefully seated into its correct position and held firmly while the material was cured with the light. A small amount of external enamel had been lost, which left a defect about 1 mm in diameter on the labial surface. This defect was also filled with the composite resin, and a second layer of sealant was painted over all margins and cured. Subsequent radiographs and vitality tests indicated that the tooth had probably responded favorably. Croll has reported successfully using light-hardened glass ionomer material for reattaching



H G. **21-7. A,** Clinical appearance of the anterior teeth of a young boy after a sledding accident that resulted in a mesial-incisal class II crown fracture of the maxillary left central incisor and a class IV fracture of the maxillary right central incisor. The right incisor has been treated by calcium hydroxide pulpotomy. **B** and C, Labial and cervical views, respectively, of the coronal fragment of the right central incisor; C also illustrates the cavity preparation of the crown made by enlargement of the pulp chamber. D, Clinical appearance of the right central incisor after the fragment restoration procedure, which used the mechanical retention of composite resin placed in the pulp canal and pulp chamber preparations along with enamel bonding and cure into a single unit. This fragment restoration served for 7 years 2 months before the tooth was finally restored with a root canal filling, post and core, and crown. During the time the fragment restoration was in place, the patient underwent 2 years of comprehensive orthodontic therapy in which a bonded bracket was attached to the fragment. The fragment broke off and was reattached, but in each instance, failure occurred as a result of new traumatic episodes (once while a protective mouth guard was being worn). Despite the occasional need to reattach the crown, we believe that overall it was the best and most convenient restoration for the patient during his young and accident-prone growing years.

fragments.' Kanca reported reattachment of a fragment that successfully capped the pulp using the total-etch technique." The restoration was more than 5 years old (replaced once) and was still in place at the time of the report.

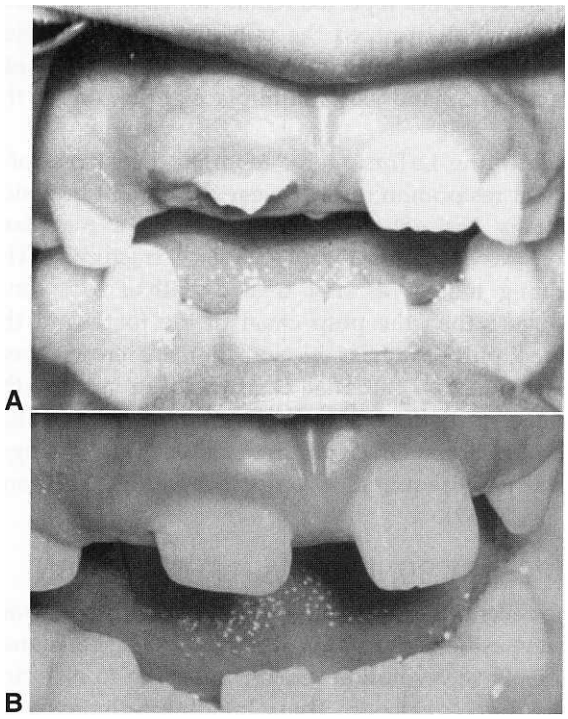
Ludlow and LaTurno have reported the success of a fragment restoration for a 13-year-old patient in which essentially the entire clinical crown of a maxillary incisor was fractured away (Class IV fracture).[15] The remaining tooth was first treated with a root canal filling, and then the pulp canal of the tooth and the enlarged pulp chamber in the fragment crown were used as retentive internal cavities to strengthen the reattached crown. One of the authors (Avery) has performed a similar crown restoration for a younger patient who required a pulpotomy (Fig. 21-7 and also Fig. 21-12).

TEMPORARY BONDED RESIN RESTORATION

The excellent marginal seal and retention derived from applying esthetic restorative materials to etched enamel surfaces have revolutionized the approach to restoring fractured anterior teeth. These bonding techniques are highly successful and versatile in many situations involving anterior trauma.

It may not be advisable to restore an extensive crown fracture with a finished esthetic resin restoration on the day of the injury, because it is usually best not to manipulate the tooth more than is absolutely necessary to make a diagnosis and provide emergency treatment. Also, such emergencies are usually treated at unscheduled appointments, and this treatment should be carried out as efficiently as possible to prevent significant disruption of the dentist's scheduled appointments. A temporary restorative resin restoration can be placed in an efficient manner and is often the treatment of choice.

The restorative resin material is applied as a protective covering at the fracture site using conventional bonding procedures. As a short-term temporary restoration, it requires little or no finishing and does not need to restore the tooth to normal contour. However, the restoration should cover the fractured surfaces and maintain any natural proximal contacts the patient may have had before the injury (Fig. 21-8). After an adequate recovery period, an esthetic resin restoration may be completed, often without removing all the temporary resin material. However, the outer surfaces of the temporary restoration should be removed superficially



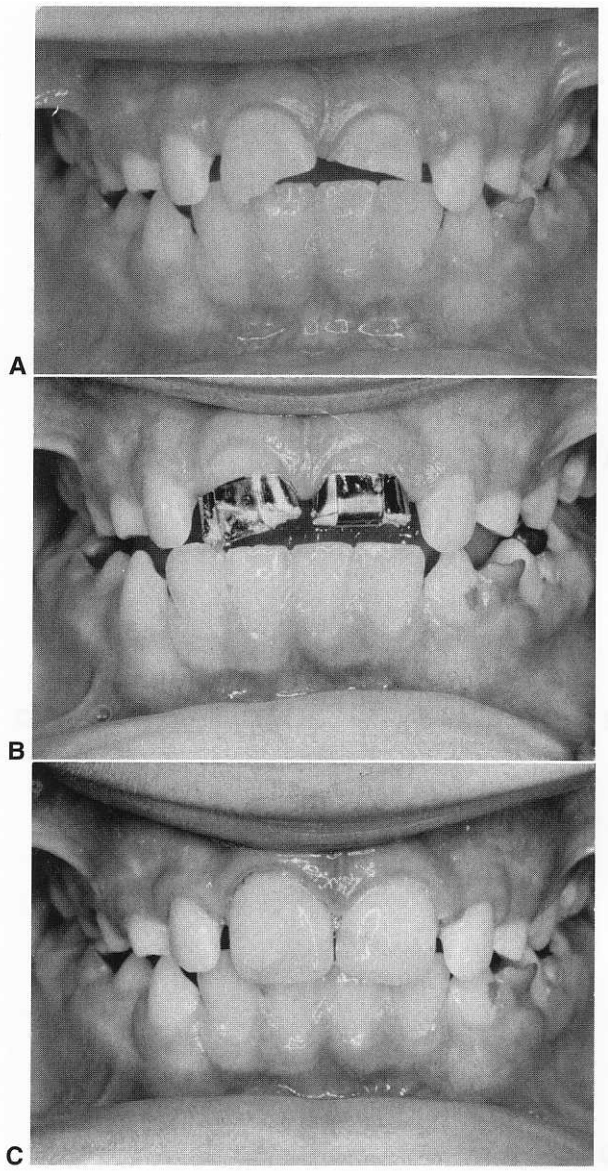
F I **G 21-8. A,** Extensive crown fracture of the partially erupted maxillary right central incisor. The area was isolated with cotton rolls, and a temporary bonded resin restoration was placed. **B,** A few months later the temporary restoration is still satisfactory and the tooth has recovered from the injury. Further eruption now makes it convenient to restore the tooth with an esthetic bonded resin restoration.

before the new material is applied. The margins of the new restoration should extend beyond the margins of the temporary restoration and onto newly etched enamel. The esthetic bonded resin restoration is discussed and illustrated later in this chapter.

ORTHODONTIC BAND

The orthodontic band will serve adequately as a retainer for a therapeutic dressing on the exposed dentin and will maintain contact with the adjacent teeth (Fig. 21-9). However, it does not meet the esthetic requirements of an anterior restoration and should be replaced as soon as possible after an adequate recovery period. It may be the temporary restoration of choice immediately after an injury if the fractured margins of the tooth cannot be easily isolated for a bonding procedure.

A preformed orthodontic band can be adapted directly to the injured tooth. If extensive loss of tooth structure or tooth mobility makes this procedure difficult, however, the band can be adapted to a corresponding intact tooth. After the tooth has been cleaned, the protective band may then be cemented in place. This type of protective covering has the advantage of being easily and quickly prepared for the fractured



F **I** G **21-9. A,** Class II fractures of the maxillary central incisors. The exposed dentin must be covered to protect the pulps from thermal and chemical irritation. **B,** A modified band has been cemented to each fractured tooth. C, Two months after the injury the teeth have been restored with resin restorations.

tooth. In addition, the clinical crown is usually sufficiently exposed to enable the dentist to make periodic pulp tests during the initial period of observation. The band is generally allowed to remain undisturbed for 4 to 6 weeks or until the initial recovery of the dental pulp is reasonably evident.

STAINLESS STEEL CROWN

The stainless steel crown is one of the most stable restorations for the temporary protection of a fractured tooth. If the coronal fracture is extensive with a vital

pulp exposure (especially class IV), this crown may be the temporary restoration of choice to provide the protection and space maintenance required during the tooth's initial recovery.

The stainless steel crown may also be indicated when emergency dental services are required away from the dental operatory. For example, the dentist may be called to a hospital or a private home to alleviate pain associated with a fractured tooth in a patient recovering from other multiple injuries sustained in an accident. The patient may be confined to a bed and completely immobilized. In such a situation the dentist may need to provide stable coverage of the fractured tooth under compromised conditions. A steel crown restoration may be the most practical choice until the patient is in a better position to receive more complete dental care.

The principal disadvantages of this restoration include its unsatisfactory esthetic appearance and the inability to accurately evaluate pulpal responses during the recovery period without removing and usually ruining the crown. If skillfully placed, however, this restoration will protect the fractured tooth in harmony with the adjacent and opposing teeth.

A crown that is essentially the same mesiodistal width as the natural tooth crown is adjusted with contouring scissors to extend approximately 0.5 mm beneath the free gingival margin.

When the crown has been fully seated on the tooth, the occlusion should be checked to make certain there will be no premature contact. In some instances a slight reduction of the remaining incisal tooth structure may be necessary. The gingival margin can be contoured with No. 114 pliers to help ensure a snug fit at the cervical area of the tooth and thus prevent excessive gingival irritation after cementation.

TREATMENT OF VITAL PULP EXPOSURES

Injury resulting in an exposure of the pulp in young patients often presents a challenge in diagnosis and treatment even greater than that of a pulp exposed by caries. In addition to treating the pulp at the exposure site, the dentist must keep in mind that, as a result of the blow, conditions may be present for many unpredictable reactions in the pulp or supporting tissues. The immediate objective in treatment, however, should be the selection of a procedure designed to maintain the vitality of the pulp whenever possible. In the management of vital pulp exposure at least three choices of treatment are available: direct pulp cap, pulpotomy, and pulpectomy with endodontic therapy.

DIRECT PULP CAP

If the patient is seen within an hour or two after the injury, if the vital exposure is small, and if sufficient crown remains to retain a temporary restoration to support the capping material and prevent the ingress of oral fluids, the treatment of choice is direct pulp capping (Fig. 21-10). If the final restoration of the tooth will require the use of the pulp chamber or the pulp canal for retention, a pulpotomy or a pulpectomy is the treatment of choice.

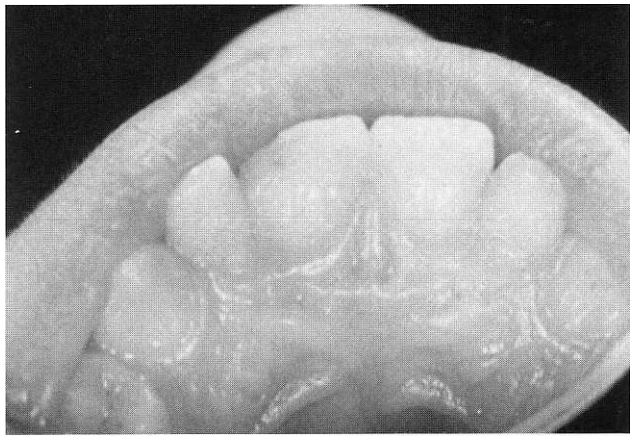


FIG. 21-10. Class III injury to a permanent central incisor. A small pulp exposure is evident that should be capped and protected with an orthodontic band or bonded restoration.

Even though the pulp at the exposure site has been exposed to oral fluids for a period of time, the tooth should be isolated with a rubber dam, and the treatment procedure should be completed in a surgically clean environment. The healthy pulp may survive and repair small injuries even in the presence of a few bacteria, the same as any other connective tissue. The crown and the area of the actual exposure should be washed free of debris, and the pulp should be kept moist before the placement of the pulp-capping material.

As mentioned in Chapter 19, numerous pulp-capping materials have been studied. Pulp capping with conventional bonding materials is now accepted by many, although the procedure is also considered inadvisable by others. Early reports of the use of both mineral trioxide aggregate and bone morphogenetic proteins appear promising, not only for pulp capping but also for general use in endodontic therapy for vital and nonvital teeth. However, as of this writing, calcium hydroxide remains the standard material against which the others are measured.

The prime requisite of pulpal healing is an adequate seal against oral fluids. Therefore a restoration should be placed immediately that will protect the pulp-capping material until the healing process is well advanced (Fig. 21-11). A thin layer of dentinlike material should cover the vital pulp tissue within 2 months.

If the injured tooth presents a good indication for direct pulp capping, there is a definite advantage in



f**I G . 21-11. A,** The pulp of a permanent central incisor has been exposed as a result of trauma. The pulp was capped with calcium hydroxide. **B,** A successful pulp capping has been accomplished. Continued root end development indicates pulp vitality. The tooth was restored with an esthetic resin-faced steel crown, but a bonded resin restoration is currently recommended.

providing this treatment. The pulp will remain functional and reparative, and dentin will develop and allow the tooth to be restored without loss of normal pulp vitality.

PULPOTOMY

If the pulp exposure in a traumatized, immature permanent (open apex) tooth is large, if even a small pulp exposure exists and the patient did not seek treatment until several hours or days after the injury, or if there is insufficient crown remaining to hold a temporary restoration, the immediate treatment of choice is a shallow pulpotomy or a conventional pulpotomy (Fig. 21-12). A shallow or partial pulpotomy is prefer-able if coronal pulp inflammation is not widespread and if a deeper access opening is not needed to help retain the coronal restoration.'" This treatment is also indicated for immature permanent teeth if necrotic pulp tissue is evident at the exposure site with inflammation of the underlying coronal tissue, but a conventional or cervical pulpotomy would be required. Yet another indication is trauma to a mature permanent (closed apex) tooth that has caused both a pulp exposure and a root fracture.

The exposure site should be conservatively enlarged, and 1 to 2 mm of coronal pulp tissue should be removed for the shallow pulpotomy or all pulp tissue in the pulp chamber removed for the conventional pulpotomy. When pulp amputation has been completed to the desired level, the pulp chamber should be thoroughly cleaned with copious irrigation. No visible dentin chips or pulp tissue tags should remain. If the remaining pulp is healthy, hemorrhage will be easy to control with a pledget of moist cotton lightly compressed against the tissue. The pulp should also have a bright reddish pink color and a concave contour (meniscus). A deeper amputation may be necessary if the health of the pulp is questionable. A dressing of calcium hydroxide is gently applied to the vital pulp tissue so that it is in passive contact with the pulp. The remaining access opening is filled with a hard-setting, biocompatible material with excellent marginal sealing capability. Then the crown may be restored with a separate bonding procedure.

Some experts on pulp therapy recommend conventional pulpectomy and root canal fillings for all teeth treated with calcium hydroxide pulpotomies soon after the root apices close. They view the calcium hydroxide pulpotomy as an interim procedure performed solely to achieve normal root development and apical closure. They justify the pulpectomy and root canal filling after apical closure as necessary to prevent an exaggerated calcific response that may result in total obliteration of the root canal (calcific metamorphosis or calcific degeneration).

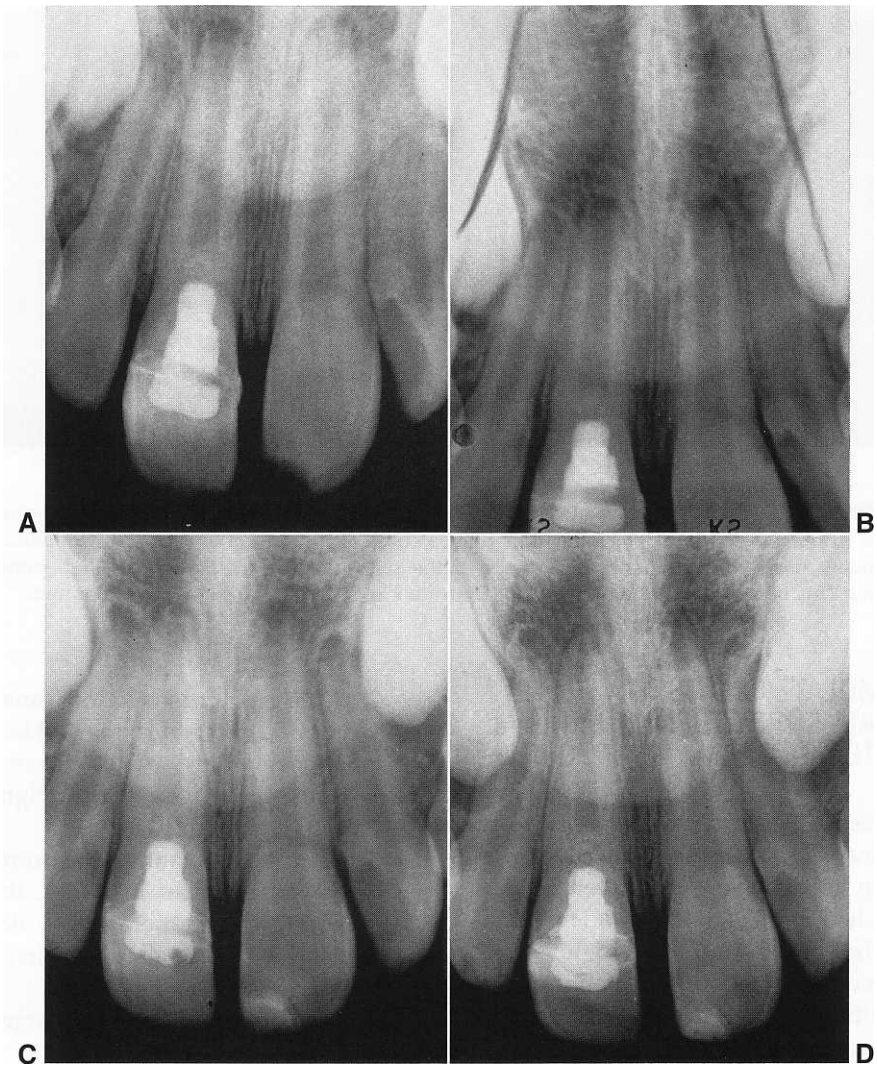
We have observed this calcific degenerative response and agree that it should be intercepted with root canal therapy if possible after apical closure (Fig. 21-13). However, long-term successes after calcium hydroxide pulpotomy in which no calcific metamorphosis has been observed can be documented. We have followed such successful cases for more than 10 years without seeing any adverse results. McCormick has reported one case of a tooth successfully treated with a calcium hydroxide pulpotomy that was observed for more than 19 years and never required further pulp therapy."

If healthy pulp tissue remains in the root canal, if the coronal pulp tissue is cleanly excised without excessive tissue laceration and tearing, if the calcium hydroxide is placed gently on the pulp tissue at the amputation site without undue pressure, and if the tooth is adequately sealed, there is a high probability that long-term success can be achieved without follow-up root canal therapy.

PULPECTOMY WITH ENDODONTIC TREATMENT

One of the most challenging endodontic procedures is the treatment and subsequent filling of the root canal of a tooth with an open or funnel-shaped apex. The lumen of the root canal of such an immature tooth is largest at the apex and smallest in the cervical area and is often referred to as a blunderbuss canal. Hermetic sealing of the apex with conventional endodontic techniques is usually impossible without apical surgery. This surgical procedure is traumatic for the young child and should be avoided if possible.

In instances of class III or class IV fractures of young permanent teeth with incomplete root growth and a vital pulp, the pulpotomy technique (as just described) is the procedure of choice. The successful pulpotomy allows the pulp in the root canal to maintain its vitality and also allows the apical portion to continue to



f I C 21-12. A, Radiographic appearance of the restored maxillary right central incisor 10 days after the class IV crown fracture occurred and the calcium hydroxide pulpotomy was performed. A fragment restoration was also used to restore the class II fracture of the maxillary left central incisor at this same appointment, but after this radiograph was obtained. **B,** This radiograph made 77, weeks after the pulpotomy shows evidence of a calcified bridge developing at the level of the pulpal amputation. C, Eight months after initial treatment, it appears that root development is progressing normally for both maxillary central incisors. D, Root completion has been achieved on both maxillary central incisors 20 months after the original injury. The root canals exhibit normal anatomic configuration. Photographs of the clinical appearance of these same teeth are seen in Figure 21-7.

develop (apexogenesis). For class IV fractures the eventual restoration may require a post in the root canal. Before this type of restoration is completed, the dentinal bridge that has formed after the pulpotomy can be perforated and routine endodontic procedures can be undertaken in a now completely developed root canal.

Occasionally a patient has an acute periapical abscess associated with a traumatized tooth. The trauma may have caused a very small pulp exposure that was overlooked, or the pulp may have been devitalized as a result of injury or actual severing of the apical vessels. A loss of pulp vitality may have caused interrupted growth of the root canal, and the dentist is faced with the task of treating a canal with an open apex.

If an abscess is present, it must be treated first. If there is acute pain and evidence of swelling of the soft tissues, drainage through the pulp canal will give the child almost immediate relief. A conventional endodontic access opening should be made into the pulp chamber. If pain is caused by the pressure required to make the opening into the pulp, the tooth should be supported by the dentist's fingers. Antibiotic therapy is also generally indicated.

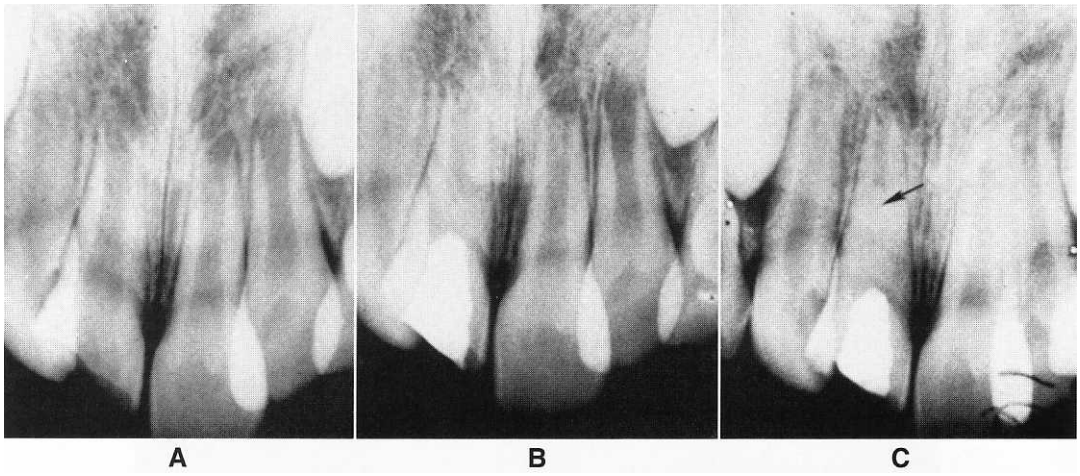


FIG 21-13. **A,** Pulp exposure was treated by the pulpotomy technique. **B,** A wider-than-normal dentin bridge has formed, an indication of possible calcific metamorphosis. C, Evidence of continued apical development. Two thirds of the pulp canal is filled with calcified material (arrow). This barrier would be difficult to penetrate if root canal therapy were attempted.

THERAPY TO STIMULATE ROOT GROWTH AND APICAL REPAIR SUBSEQUENT TO PULPAL NECROSIS IN ANTERIOR PERMANENT TEETH (APEXIFICATION)

The conventional treatment of pulpless anterior teeth usually requires apical surgery if the teeth have open apices. Many young teeth have been saved in this manner. However, a less traumatic endodontic therapy called apexification has been found to be highly effective in the management of immature, necrotic permanent teeth. The apexification procedure should precede conventional root canal therapy in the management of teeth with irreversibly diseased pulps and open apices.

Frank has described a technique based on the normal physiologic pattern of root development that brings about the resumption of apical development so that the root canal can be obliterated by conventional canal-filling techniques. [Is] The procedure described by Frank and demonstrated to be successful in repeated clinical trials stimulates the process of root end development, which was interrupted by pulpal necrosis, so that it continues to the point of apical closure (Fig. 21-14).'" Often a calcific bridge develops just coronal to the apex. When the closure occurs, or when the calcific "plug" is observed in the apical portion, routine endodontic procedures may be completed; the possibility of recurrent periapical pathosis is thus prevented.

The following steps are included in the technique:

1. The affected tooth is isolated with a rubber dam, and an access opening is made into the pulp chamber.
2. A file is placed in the root canal, and a radiograph is made to establish the root length accurately. It is important to avoid placing the instrument through the apex, which might injure the epithelial diaphragm.
3. After the remnants of the pulp are removed using barbed broaches and files, the canal is flooded with hydrogen peroxide to aid in the removal of debris. The canal is then irrigated with sodium hypochlorite and saline.
4. The canal is dried with large paper points and loose cotton.
5. A thick paste of calcium hydroxide and camphorated mono-parachlorophenol (CMCP) or calcium hydroxide in a methylcellulose paste* is transferred to the canal with the aid of an amalgam carrier. An endodontic plugger may be used to push the material to the apical end, but an excess of material should not be forced beyond the apex of the tooth.
6. A cotton pledget is placed over the calcium hydroxide, and the seal is completed with a layer of reinforced zinc oxide–eugenol cement.

Weine recommends that the apexification procedure be completed in two appointments.'" After instrumentation, irrigation, and drying of the canal during the first appointment, he advises sealing a sterile, dry cotton pellet in the pulp chamber for 1 to 2 weeks. Placing a calcium hydroxide dressing in the canal is optional

*Pulpdent Corp., Watertown, MA.

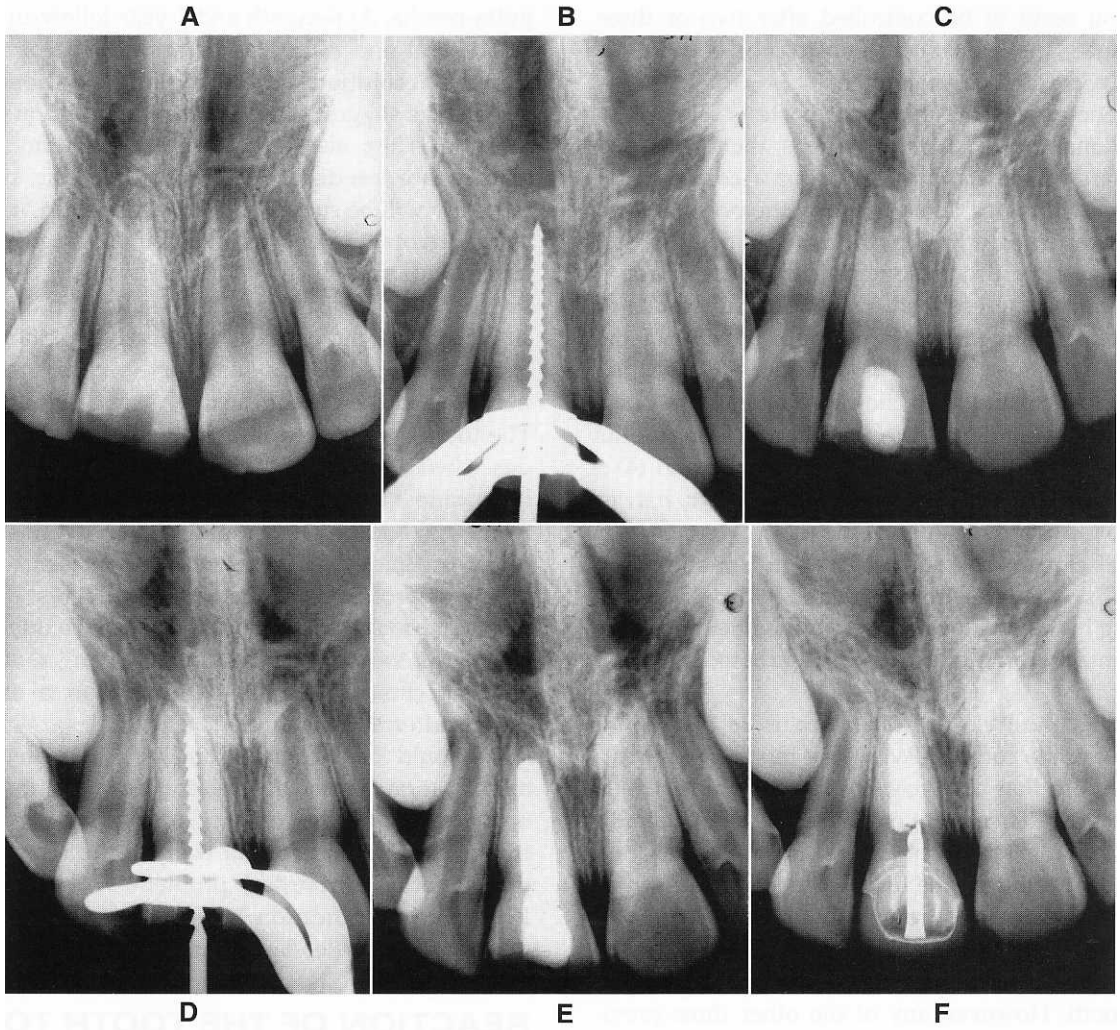


FIG 21-14. Series of radiographs demonstrating treatment to stimulate root end development of a pulpless young anterior permanent central incisor. A, An injury several months before had resulted in a pulp exposure. An acute abscess was present at the beginning of treatment. An opening to the pulp chamber was made to allow drainage. **B,** Four days after the initial treatment the canal length was established. Files were used to clean the canal. After cleansing and irrigation of the canal, calcium hydroxide and camphorated mono-parachlorophenol were used to fill the canal. C, One month after initial treatment. **D,** Six months after initial treatment a definite calcified stop is encountered when the file is introduced. The canal was cleansed thoroughly, and gutta-percha was used to fill the canal. **E,** Five months after the placement of the gutta-percha canal filling. F, A 6-month postoperative radiograph. A tube and resin core was placed and the tooth was restored with a jacket crown. Currently, an esthetic bonded composite resin restoration (or a fragment restoration if possible) would probably be the preferred interim restoration until the patient has attained adulthood. *(Courtesy Drs. Paul E. Starkey and Joe Camp.)*

at the first appointment. During the second appointment the debridement procedures are repeated before filling the canal with a thick paste of calcium hydroxide and CMCP or calcium hydroxide in a methylcellulose paste.

Whether the tooth is filled in one or two appointments (or more) should be determined to a large extent by the clinical signs and symptoms present and to a lesser extent by operator convenience. The signs and symptoms of active infection should be eliminated before the canal is filled with the treatment paste. Absence of tenderness to percussion is an especially good sign before filling the canal. Because of the wide-open communication to periapical tissues, it is not always possible to maintain complete dryness in the root canal. If the canal continues to weep but other signs

of infection seem to be controlled after two or three appointments, the dentist may elect to proceed with the calcium hydroxide paste treatment.

As a general rule the treatment paste is allowed to remain 6 months. The root canal is then reopened to determine if the tooth is ready for a conventional gutta-percha filling as determined by the presence of a " positive stop" when the apical area is probed with a file. Often there is also radiographic evidence of apical closure. Frank has described four successful results of apexification treatment's: (1) continued closure of the canal and apex to a normal appearance, (2) a dome-shaped apical closure with the canal retaining a blunderbuss appearance, (3) no apparent radiographic change but a positive stop in the apical area, and (4) a positive stop and radiographic evidence of a barrier coronal to the anatomic apex of the tooth.

If apical closure has not occurred in 6 months, the root canal is retreated with the calcium hydroxide paste. If weeping in the canal was not controlled before filling, retreatment is recommended 2 or 3 months after the first treatment.

Although apical closure often occurs in a 6-month period, one of the authors (Avery) has monitored retreatment for more than $2^{1}/2$ years before favorable results were achieved. Retreatment of the canal at 3- to 6-month intervals for an extended period seems justified as long as the patient remains free of adverse signs and symptoms, because apical closure is likely to occur eventually.

Ideally the postoperative radiographs should demonstrate continued apical growth and closure as in a normal tooth. However, any of the other three previously described results are considered successful. When closure has been achieved, the canal is obliterated in the conventional manner with gutta-percha.

Currently there seems to be a trend away from incorporating antibacterial agents, such as CMCP, in the calcium hydroxide treatment paste. It is generally agreed that calcium hydroxide is the major ingredient responsible for stimulating the desired calcific closure of the apical area. Calcium hydroxide is also an antibacterial agent. It may be that CMCP does not enhance the repair; on the other hand, its use as described here has not been shown to be detrimental. Certainly more than one treatment paste has been employed with success. Giulinani et al have demonstrated the use of mineral trioxide aggregate (MTA) to form an apical plug for apexification in three clinical cases.'[0] The root canals of central incisors that had suffered premature interruption of root development as a consequence of trauma were rinsed with 5% sodium hypochloride. Calcium hydroxide was then placed in the canals for 1 week. Following this, the apical portion of the canal (4 mm) was filled with MTA, and the remaining portion of the root canals were closed with thermoplastic gutta-percha. At 6-month and 1-year follow-up periods the clinical and radiographic appearance of the teeth showed resolution of the periapical lesions. These researchers suggest that MTA is a valid option for apexification. They also suggest that long-term outcome studies are needed to test whether this treatment modality will be successful in a large group of teeth.

Trairatvorakul reported on a most interesting and unusual case in which a maxillary primary central incisor successfully responded to apexification therapy." A 14-month-old child experienced early childhood caries to the extent that one maxillary central incisor was necrotic and the root of the tooth was not fully formed. The tooth was treated in a manner similar to that just described. The tooth was retreated with calcium hydroxide 3 months later and restored with composite resin. An additional 3 months later (6 months after initial treatment) an apical stop was present when the area was probed from the root canal access opening. The root canal was then filled with zinc oxide—eugenol paste. The tooth remained functional and radiographically and clinically successful until its normal exfoliation 6 years after initial treatment.

It should be recognized that teeth treated by the apexification method are susceptible to fracture because of the brittleness that results from nonvitality and from the relatively thin dentinal walls of the roots. In addition, another important problem with the calcium hydroxide apexification technique is the duration of therapy, which often lasts many months.

REACTION OF THE TOOTH TO TRAUMA

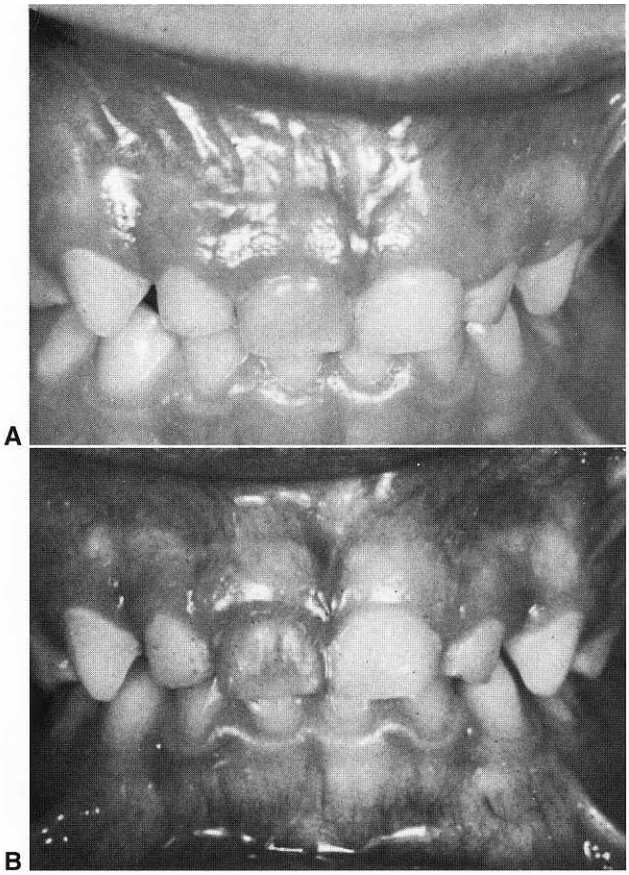
PULPAL HYPEREMIA

The dentist must be cognizant of the inadequacies of present methods of determining the initial pulpal reaction to an injury and of the difficulty in predicting the long-range reaction of the pulp and supporting tissues to the insult. A trauma of even a so-called minor nature is immediately followed by a condition of pulpal hyperemia.

Congestion of blood within the pulp chamber a short time after the injury can often be detected in the clinical examination. If a strong light is directed to the labial surface of the injured tooth and the lingual surface is viewed in a mirror, the coronal portion of the tooth will often appear reddish compared with the adjacent teeth. The color change may be evident for several weeks after the accident and is often indicative of a poor prognosis.

INTERNAL HEMORRHAGE

The dentist will occasionally observe temporary discoloration of a tooth after injury. Hyperemia and increased pressure may cause the rupture of capillaries and the



f l **G 21-15. A,** Maxillary right central incisor became discolored within 2 weeks after trauma. A pulp test indicated that the pulp was vital. **B,** Six months after the injury, the tooth responded to the vitality test and there was less discoloration of the crown.

escape of red blood cells, with subsequent breakdown and pigment formation. The extravasated blood may be reabsorbed before gaining access to the dentinal tubules, in which case little if any color change will be noticeable and what appears will be temporary (Fig. 21-15). In more severe cases there is pigment formation in the dentinal tubules. The change in color is evident within 2 to 3 weeks after the injury, and although the reaction is reversible to a degree, the crown of the injured tooth retains some of the discoloration for an indefinite period. In cases of this type there is some chance that the pulp will retain its vitality, although the likelihood of vitality is apparently low in primary teeth with dark gray discoloration. Croll, Pascon, and Langeland found that 33 of 51 traumatized teeth (65%) with gray-black discoloration were necrotic.[22] Holan and Fuks conducted a retrospective study of 88 pulpectomized primary incisors, of which 48 met their 9 clinical and radiographic criteria for further investigation.[23] Briefly, their criteria included dark-gray coronal discoloration as the primary diagnostic sign before pulpectomy. The remaining criteria were indicative of normal conditions or conditions only somewhat suggestive of a pulpal problem. They found that 47 (98%) of the teeth included in the study were either necrotic (37, or 77%) or partially necrotic (10, or 21%). Because all of these teeth were previously determined to need pulpectomy, the 98% confirmation is not so surprising. However, the fact remains that all the teeth exhibited dark-gray discoloration and few, if any, other minor signs or symptoms of a problem.

Discoloration that becomes evident for the first time months or years after an accident, however, is evidence of a necrotic pulp.

CALCIFIC METAMORPHOSIS OF THE DENTAL PULP (PROGRESSIVE CANAL CALCIFICATION OR DYSTROPHIC CALCIFICATION)

A frequently observed reaction to trauma is the partial or complete obliteration of the pulp chamber and canal (Fig. 21-16). Although the radiograph may give the illusion of complete obliteration, an extremely fine root canal and remnants of the pulp will persist.

The crowns of teeth that have undergone this reaction may have a yellowish, opaque color. Primary teeth demonstrating calcific metamorphosis will usually undergo normal root resorption, although Peterson, Taylor, and Marley have reported observing one patient who exhibited calcific metamorphosis of a maxillary primary central incisor that subsequently showed evidence of significant internal resorption in the root." They emphasize the need for careful monitoring of traumatized teeth that have undergone calcific metamorphosis.

Permanent teeth will often be retained indefinitely. However, a permanent tooth showing signs of calcific changes as a result of trauma should be regarded as a potential focus of infection. A small percentage demonstrate pathologic change many years after the injury (Fig. 21-17).

INTERNAL RESORPTION

Internal resorption is a destructive process generally believed to be caused by odontoclastic action. It may be observed radiographically in the pulp chamber or canal within a few weeks or months after an injury. The destructive process may progress slowly or rapidly. If progression is rapid, it may cause a perforation of the crown or root within a few weeks (Fig. 21-18). Mummery described this condition as "pink spot" because when the crown is affected, the vascular tissue of the pulp shines through the remaining thin shell of the tooth.[25] He referred to the occurrence of a perforation as " perforating hyperplasia of the pulp."

If evidence of internal resorption is detected early, before it becomes extensive with resulting perforation,



f **I G 21-16.** A, Maxillary primary central incisors have a yellowish, opaque appearance, indicative of a previous injury. **B,** The radiograph demonstrates almost complete obliteration of the pulp chambers and canals. C, Normal root resorption of the primary central incisors has occurred. D and **E,** The permanent incisors have erupted. White opaque areas appear on the labial surface of the maxillary central incisors, indicative of the initial injury.

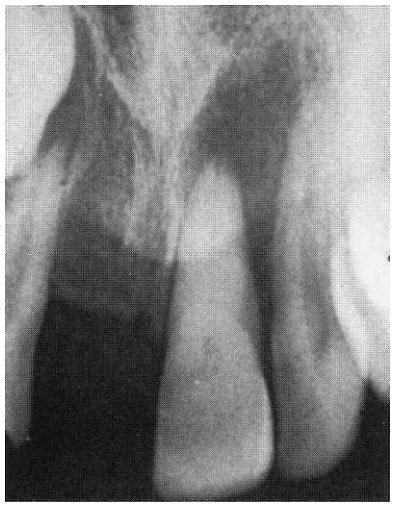
the tooth may possibly be retained when endodontic procedures are instituted.

PERIPHERAL (EXTERNAL) ROOT RESORPTION

Trauma with damage to the periodontal structures may cause peripheral root resorption (Fig. 21-19). This reaction starts from without, and the pulp may not become involved. Usually the resorption continues unabated until gross areas of the root have been destroyed. In exceptional cases the resorption may become arrested, and the tooth may be retained. Peripheral root resorption is most often observed in cases of severe trauma in which there has been some degree of displacement of the tooth.

PULPAL NECROSIS

Little relationship exists between the type of injury to the tooth and the reaction of the pulp and supporting tissues. A severe blow to a tooth causing displacement often results in pulpal necrosis. The blow may cause a severance of the apical vessels, in which case the pulp undergoes autolysis and necrosis. In a less severe type of injury the hyperemia and slowing of blood flow through the pulpal tissue may cause eventual necrosis



Hi. **21-17.** Trauma occurred 10 years before acute symptoms developed in the left central incisor. An apical lesion area may be seen. The right central incisor was lost at the time of the injury.

of the pulp. In some cases the necrosis may not occur until several months after the injury.

A tooth receiving an injury that causes coronal fracture may have a better pulpal prognosis than a tooth that sustains a severe blow without fracturing the crown. Part of the energy of the blow dissipates as the crown fractures, rather than all of the energy's being absorbed by the tooth's supporting tissues. Thus the periodontium and the pulp of the injured tooth sustain less trauma when the crown fractures. The prognosis for long-term retention of the tooth and for maintenance of pulp vitality may then improve. However, because some teeth do not recover from traumatic blows that seem relatively minor, all injured teeth should be closely monitored.

Injured teeth with subsequent pulpal necrosis are commonly asymptomatic, and the radiograph is essentially normal. It should be realized, however, that these teeth are probably infected and that acute symptoms and clinical evidence of infection will inevitably develop at a later date. The tooth with a necrotic pulp should therefore be extracted or treated with endodontic procedures, whichever is indicated.

A necrotic pulp in an anterior primary tooth may be successfully treated if no extensive root resorption or bone loss has occurred (Fig. 21-20). The treatment technique is essentially the same as that for permanent teeth. However, trauma to the periapical tissues during canal instrumentation must be carefully avoided. After the canal has been properly prepared, it is filled with slow-setting zinc oxide—eugenol. The canal walls are first lined with a thin mix of the canal-filling material. A thicker mix should then be placed in the pulp chamber. Over this is placed a pledget of cotton, and the material is forced into the canal with a small amalgam plugger.

AN KYLOSIS

Another reaction observed after trauma to anterior primary or permanent teeth is ankylosis. The condition is caused by injury to the periodontal ligament and subsequent inflammation, which is associated with invasion by osteoclastic cells. The result is irregularly resorbed areas on the peripheral root surface. In histologic sections repair can be seen that may cause a mechanical lock or fusion between alveolar bone and root surface. Clinical evidence of ankylosis is a difference in the incisal plane of the ankylosed tooth and adjacent teeth. The adjacent teeth continue to erupt, whereas the ankylosed tooth remains fixed in relation to surrounding structures (Figs. 21-21 and 21-22). The radiograph shows an interruption in the periodontal membrane of the ankylosed tooth, and the continuous dentin and alveolar bone can often be seen.

The ankylosed anterior primary tooth should be removed if there is evidence of its causing delayed or ectopic eruption of the permanent successor. If ankylosis of a permanent tooth occurs during active eruption, eventually a discrepancy between the position of this tooth and its adjacent ones will be obvious. The uninjured teeth will continue to erupt and may drift mesially with a loss of arch length. Therefore the removal of a permanent tooth that becomes ankylosed is often necessary, especially if the ankylosis occurs during the preteenage or early teenage years.

RESTORATION OF FRACTURED TEETH

The restoration of a fractured tooth is as important as the emergency treatment designed to aid in the recovery of the pulp after the trauma. Several restorations have been advocated, and although the dentist has a wide choice of techniques and types of restorations, the circumstances surrounding the case often dictate the type of restoration for a given patient. The prognosis of pulp healing, the amount of tooth structure remaining, the stage of eruption of the tooth and adjacent teeth, the size of the dental pulp and degree of root closure, the normalcy of the occlusion, and the wishes of the patient must all be considered in the selection of a temporary restoration, an intermediate restoration, or the permanent restoration. In the young patient, although it is often desirable to wait for continued eruption of the tooth or to determine the outcome of a vital pulp procedure, a delay of even a few weeks is often sufficient to allow the tipping of adjacent teeth, overeruption of opposing teeth, or other undesirable changes in the occlusion.



FIG 21-18. Internal resorption in a traumatized maxillary left primary incisor. **A,** The tooth 6 months after the injury. The crown had a slightly pinkish color. **B,** The radiograph shows internal resorption in the pulp chamber and canal and some evidence of attempted repair. C, There was subsequent deep discoloration of the crown as the destructive process continued, accompanied by proliferation of the pulp tissue. **D,** A perforation can be seen on the lingual surface of the primary incisor. **E,** The radiograph shows the degree of the resorption. The tooth was extracted.

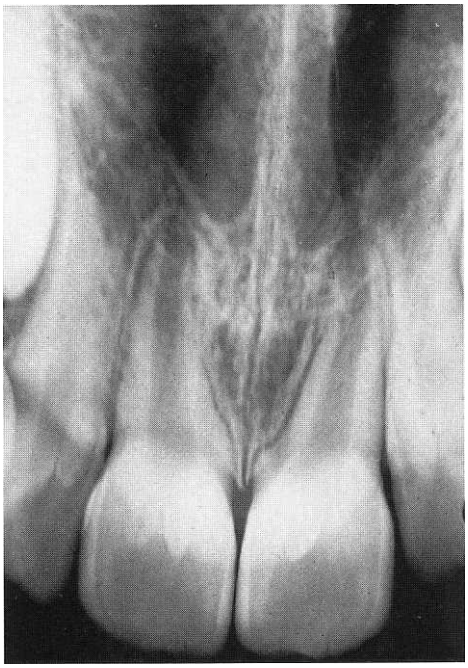


FIG **21-19.** Radiographic evidence of peripheral root resorption. In these teeth the pulpal vitality was retained, and root resorption did not continue.

ESTHETIC BONDED COMPOSITE RESIN RESTORATION

The feathered-edge restorative technique without mechanical tooth preparation is appropriate in some situations, but it requires excessive contour in the restoration. It offers the advantage of creating less irritation to the pulp, because little or no enamel modification is required. In some cases the excess contour is relatively insignificant, but it may be more significant if the restoration is large *(50%* or more of the crown) or the fracture extends near or below gingival tissue. Excessive contour on the lingual surface of a maxillary anterior tooth may interfere with normal occlusion.

A beveled preparation affords the dentist the opportunity to reduce the amount of overcontouring of larger restorations and also reduces contour in areas where the occlusion prohibits overcontouring. The dentist may elect the feathered-edge technique (no preparation) on the labial tooth surface and the beveled preparation on the proximal or lingual surfaces or both. Both techniques are described in the following discussion.

When a beveled preparation is desired, the bevel is made in the enamel around the entire circumference or the selected part of the fracture (Figs. 21-23 and 21-24). The bevel should be about 1 to 2 mm incisocervically and about halfway (or more) through the thickness of the enamel at the fracture margins. The labial enamel margin should be irregular to provide a better esthetic blending of the resin with the tooth structure. It should be mentioned that this sequence may be altered if the practitioner uses some of the latest-generation bonding adhesive systems.

The exposed dentin from a recent injury may be covered with a calcium hydroxide liner. Dilute phosphoric acid (etchant or tooth conditioner) is applied to the enamel surface of the preparation for about 20 seconds (longer for primary teeth). The tooth is then thoroughly flushed with water, and excess moisture is removed with air. The etched area should appear frosty and opaque. Alternatively, the total-etch technique may be employed. In either case a bonding agent is applied to all etched tooth structure. Again, this sequence may be altered with the use of some of the latest bonding adhesive systems.

A celluloid matrix strip may be placed interproximally and wedged for close adaptation at the gingival margin. In large restorations a custom-cut celluloid crown form matrix may be used to help contour the restoration.

Often, little or no mechanical preparation of the fractured incisor is necessary with the feathered-edge technique. Instead, the resin margins are allowed to overlap the fractured edges to become feathered-edge margins on the etched sound enamel cervical to the fracture (Figs. 21-25 to 21-29). As already mentioned, this procedure requires a slightly overcontoured restoration and therefore has limitations. The dentist should be alert to resultant potential undesirable changes in gingival health or the creation of traumatic occlusion. Slight beveling of the fractured enamel margins is usually recommended with the feathered-edge technique to remove loose enamel rods and ensure a fresh surface for etching. The exposed dentin may be protected with a layer of hard-setting calcium hydroxide, and etching should extend 2 or 3 mm beyond the fracture to allow an adequate surface for feather-edging the resin restorative materials. Most manufacturers supply a kit, which usually includes an etchant, a bonding agent, the restorative materials, and a shade guide. The bonding agent is applied to the etched surfaces.

The light-polymerized materials offer the advantage of allowing the clinician to build or sculpt the restoration in small increments and minimize finishing time. Clinical studies have confirmed that excellent and durable restorations may be obtained. Finishing disks and large, round finishing burs or diamonds may be used to contour the labial and lingual surfaces. A sharp, curved scalpel blade (No. 12) may be used to remove flash from the proximal margins of the restoration.

REACTION OF PERMANENT TOOTH BUDS TO INJURY

The dentist who provides emergency care for a child after an injury to the anterior primary teeth must be

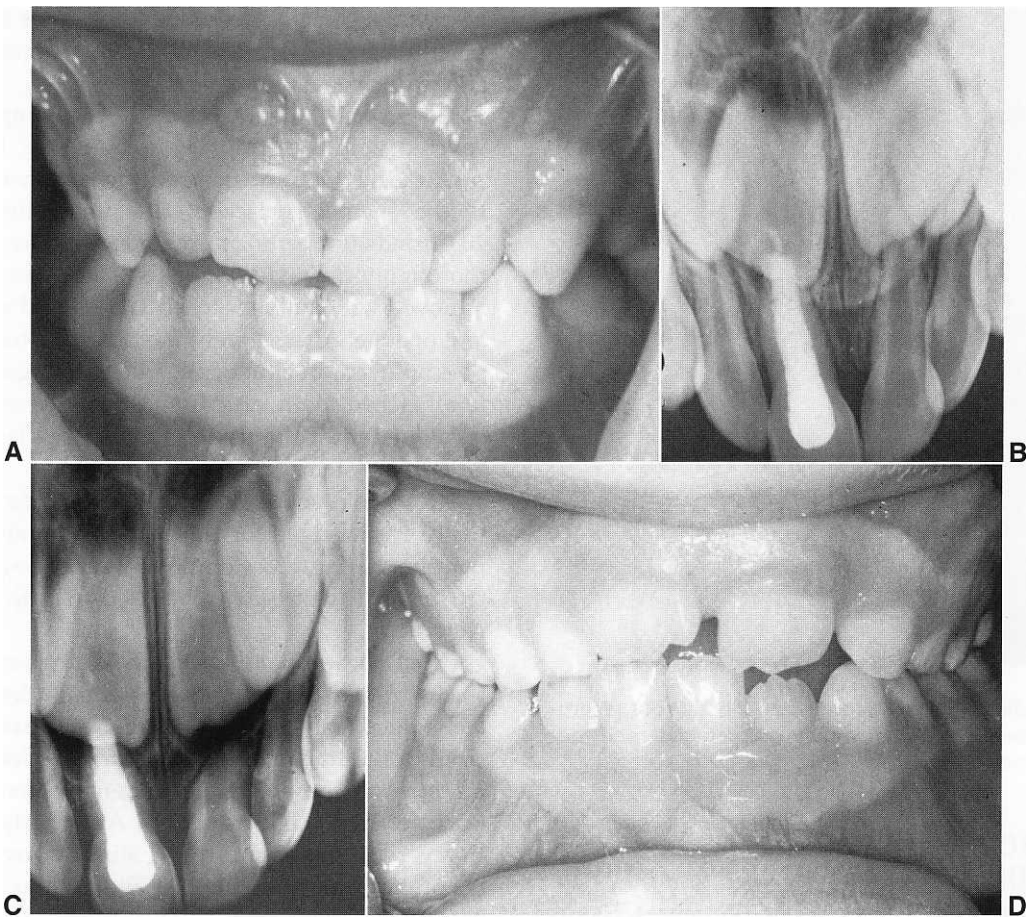


FIG 21-20. A, Evidence of a chronic alveolar abscess associated with the maxillary primary right central incisor in a preschool child. The tooth was considered suitable for endodontic treatment. **B,** Radiograph of the tooth after endodontic treatment and the filling of the canal with zinc oxide—eugenol. C, The treated tooth is undergoing normal root resorption. The root canal filling material is also being resorbed. **D,** The primary incisor was retained for an adequate period to allow the normal eruption of the permanent successor.



[IG 21-21. Left primary central incisor received a blow 1 year before this photograph was taken. The tooth was not depressed at the time of the injury. However, it became ankylosed and failed to continue to erupt.

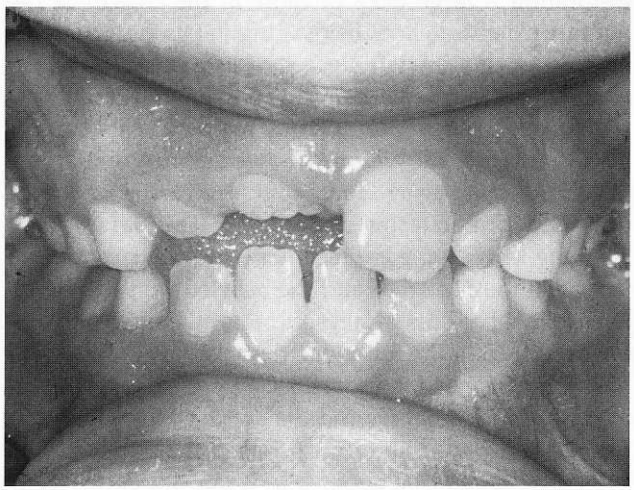
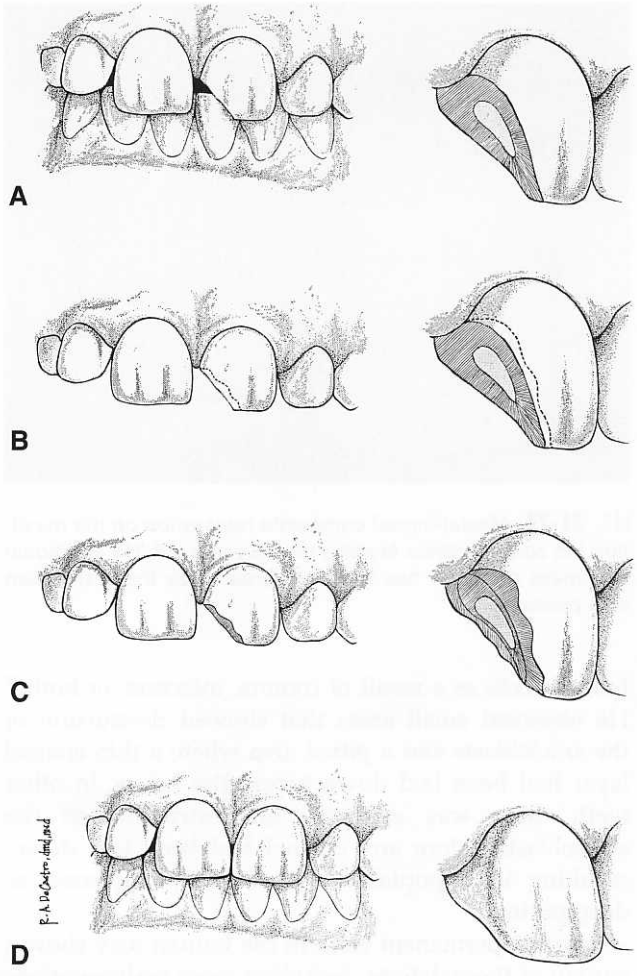


FIG 21-22. Permanent central incisor that became ankylosed after an injury. The incisor failed to erupt and had to be removed surgically.

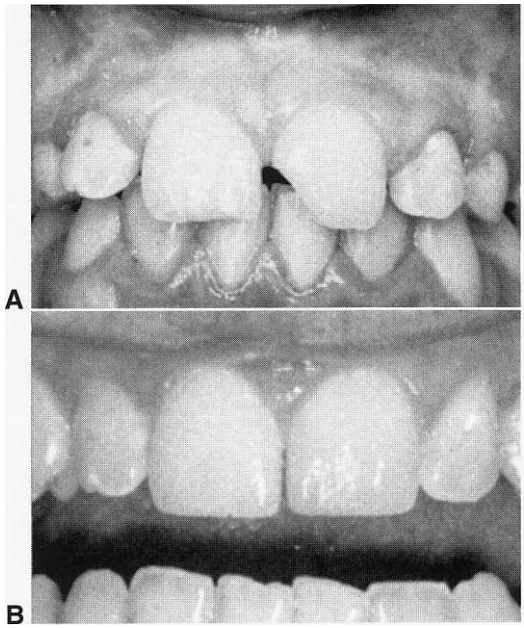


f **1 G 21-23. A,** Example of a typical class II fracture of a permanent incisor. **B,** Dotted line represents the approximate location of the irregular enamel margin to be placed on the labial surface in the bevel preparation. C, Bevel preparation complete and ready for etching. **D,** Bonded composite material restores natural contour and color.

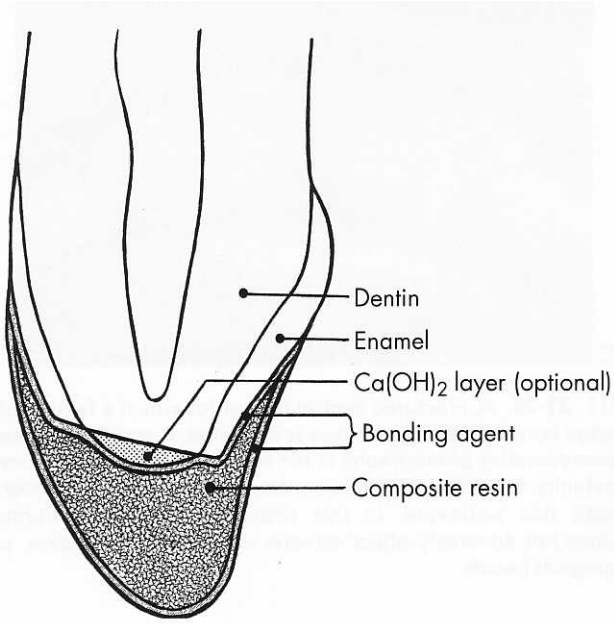
aware of the possibility of damage to the underlying developing permanent teeth.

Andreasen, Sundstrom, and Ravn have reported on the effect of injuries to primary teeth on their permanent successors.[26] In a clinical and radiographic study of 213 teeth, these investigators demonstrated that more than 40% of their young patients had changes in the permanent teeth that could be traced to injury to the primary dentition. The close anatomic relationship between the apices of primary teeth and their developing permanent successors explains why injuries to primary teeth may involve the permanent dentition (Fig. 21-30).

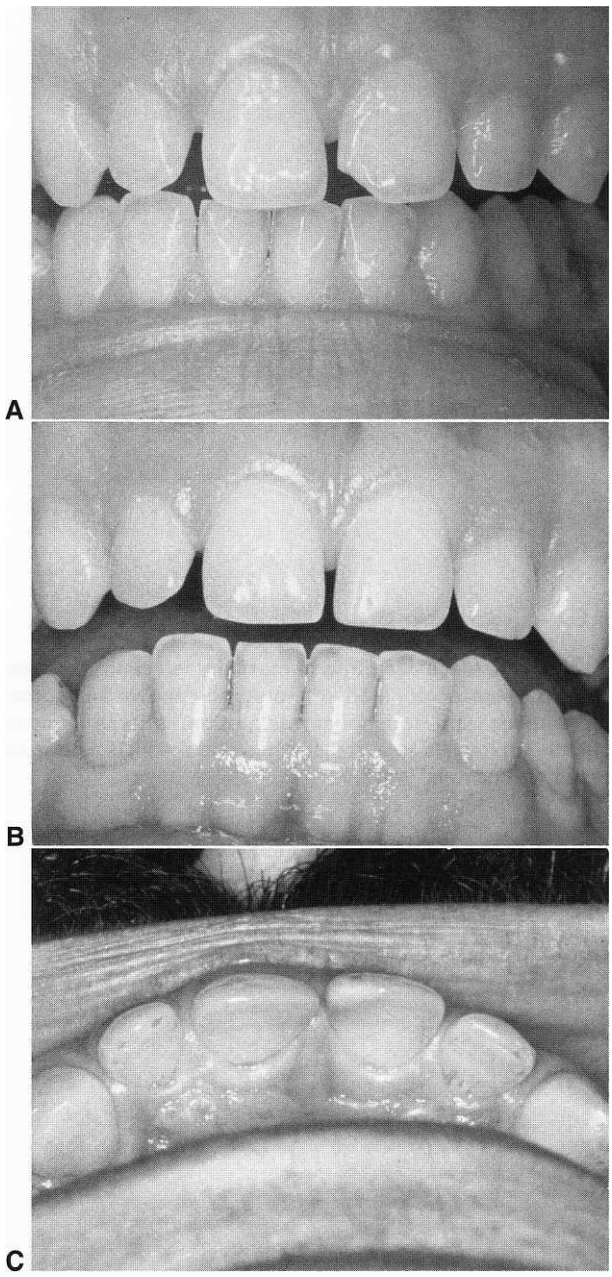
The dentist and the physician should also be aware of the possibility of trauma to permanent tooth buds from other unusual injuries so that the parents may be informed of the possibility of defective permanent tooth development. Some injuries to the face and jaws may



El G 21-24. **A,** Class II fracture of maxillary left central incisor. **B,** This 7/2-year postoperative photograph shows an acceptable esthetic and functional restoration, although close examination reveals loss of material along the incisal edge as a result of functional wear.



f G 21-25. This sagittal drawing illustrates the application of a feathered-edge restoration on a fractured incisor. Little or no tooth preparation is required before calcium hydroxide (optional) is placed on the exposed dentin and the tooth is etched. Slight beveling of the fractured enamel margins is suggested. The drawing also illustrates the need to overcontour the labial and lingual surfaces with the feathered-edge restoration.



f I G **21-26. A,** Fractured central incisor for which a feathered-edge bonded resin restoration is indicated. **B** and C, One-year postoperative photographs of the restoration. The incisal view exhibits the amount of overcontouring that naturally occurs with this technique. In this situation the overcontouring does not adversely affect esthetic appearance, occlusion, or gingival health.

not appear to have caused any dental injuries initially, but the problem maybe noticed several months or years later (Fig. 21-31).

HYPOCALCIFICATION AND HYPOPLASIA

Cutright's experiments with miniature pigs have shown many lesions similar to those seen in permanent



6 **1 6 21-27.** Mesial-incisal composite restoration on the maxillary left central incisor is more than 6 years old. No additional treatment or repair has been required since the restoration was placed.

human teeth as a result of trauma, infection, or both.' He observed small areas that showed destruction of the ameloblasts and a pitted area where a thin enamel layer had been laid down before the injury. In other teeth there was evidence of destruction of the ameloblasts before any enamel had been laid down, resulting in hypoplasia that clinically appeared as deep pitting.

Erupted permanent teeth in the human may show a variety of these defects, including gross malformations of the crown (Figs. 21-32 and 21-33). The presence of a small, pigmented hypoplastic area has been referred to as Turner tooth. Small hypoplastic defects may be restored by the resin-bonding technique.

REPARATIVE DENTIN PRODUCTION

In cases in which the injury to the developing permanent tooth is severe enough to remove the thin covering of developing enamel or cause destruction of the ameloblasts, the subjacent odontoblasts have been observed to produce a reparative type of dentin. The irregular dentin bridges the gap where there is no enamel covering to aid in protecting the pulp from further injury.

DILACERATION

The condition referred to as dilaceration occasionally occurs after the intrusion or displacement of an anterior primary tooth. The developed portion of the tooth is twisted or bent on itself, and in this new position growth of the tooth progresses. Cases have been observed in which the crown of a permanent tooth or a portion of it develops at an acute angle to the remainder to the tooth (Fig. 21-34). Kilpatrick, Hardman, and Welbury reported a dilacerated root of a primary central incisor

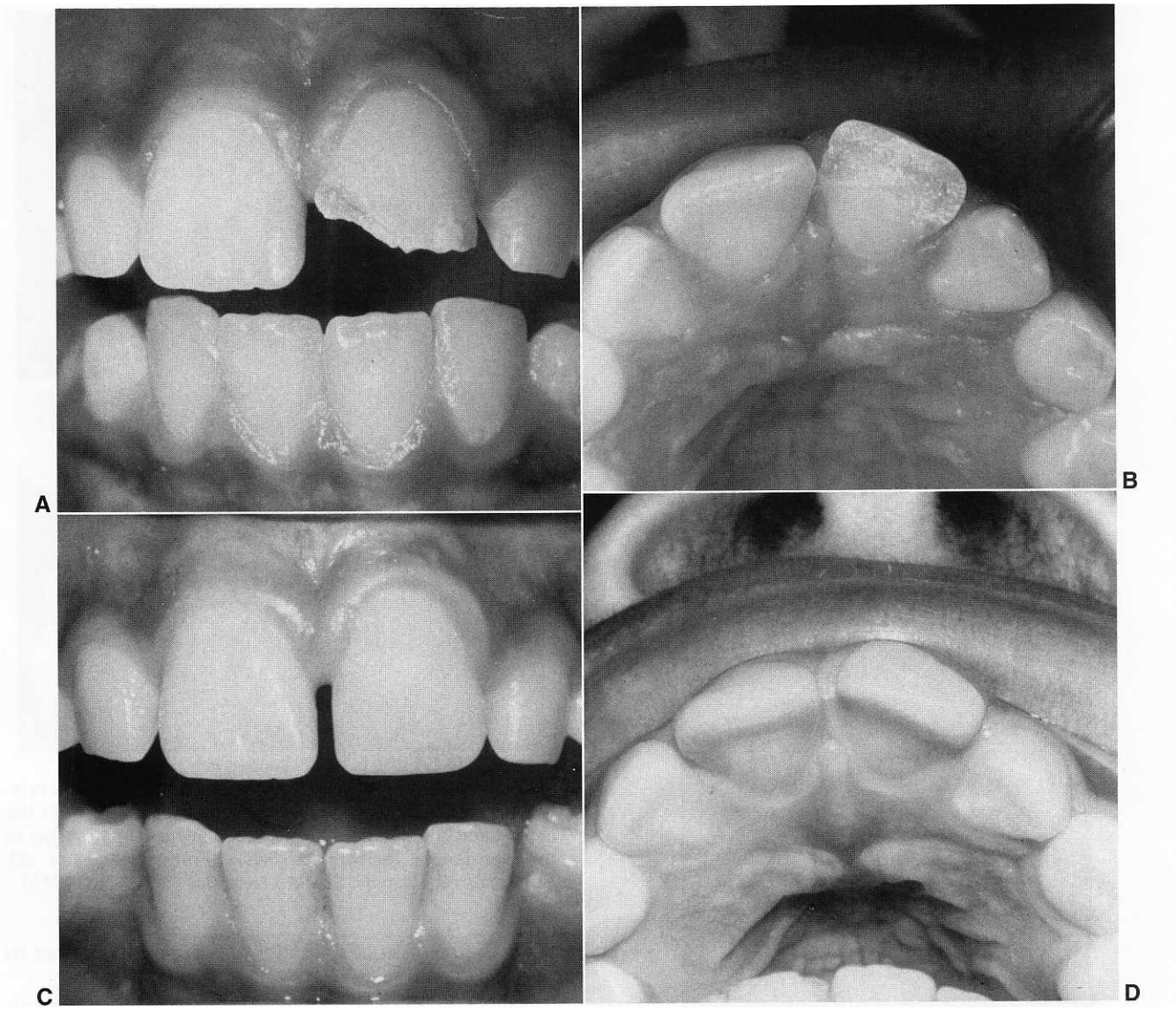


FIG 21-28. **A,** Class II crown fracture involving the entire incisal edge of the maxillary left central incisor. **B,** Incisal view illustrates rotation and labial position of the tooth, which makes it more susceptible to trauma. C and D, Six-month postoperative views of the tooth restored with an esthetic bonded composite resin restoration using the feathered-edge technique. Incisal view illustrates how an illusion of better tooth alignment was achieved when the mesial half of the labial surface was deliberately undercontoured during finishing.

in a 6-year-old boy.[28] The tooth was necrotic, the root had not resorbed, and the apex of the root was exposed in the labial sulcus and was associated with a draining sinus. No specific history of trauma could be confirmed, but the child was prone to accidents. The authors speculate that this unusual dilaceration may have been due to injury soon after initial eruption of the tooth.

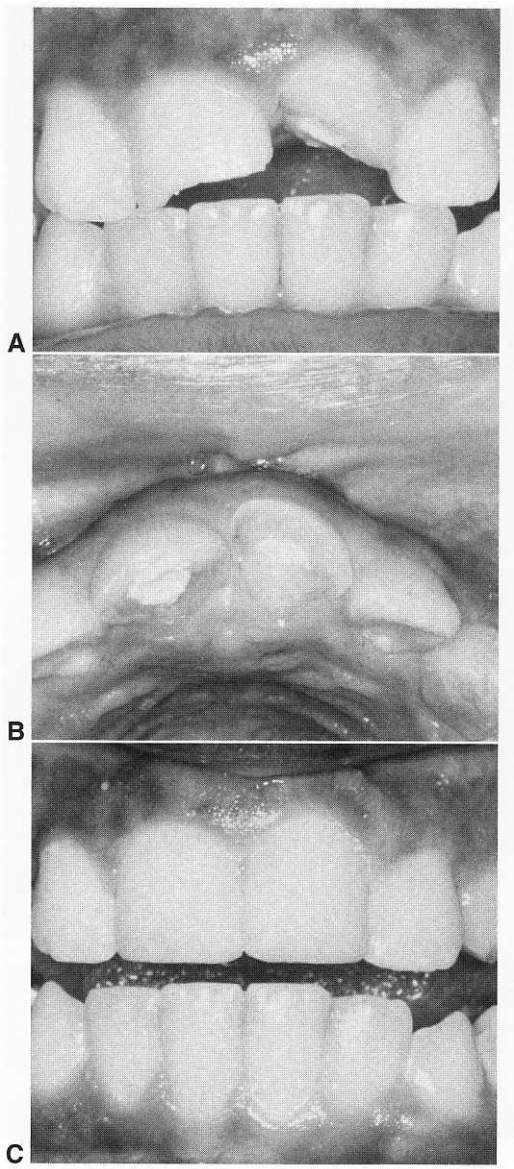
Rushton reported that an injury during development may cause the subsequent appearance of an additional cusp, crown, or denticle.[29] Partial duplication of the affected teeth may occur, with the appearance of gemination in the part of the tooth formed after the injury.

DISPLACEMENT OF PRIMARY AND PERMANENT ANTERIOR TEETH (LUXATION)

INTRUSION AND **EXTRUSION** OF TEETH

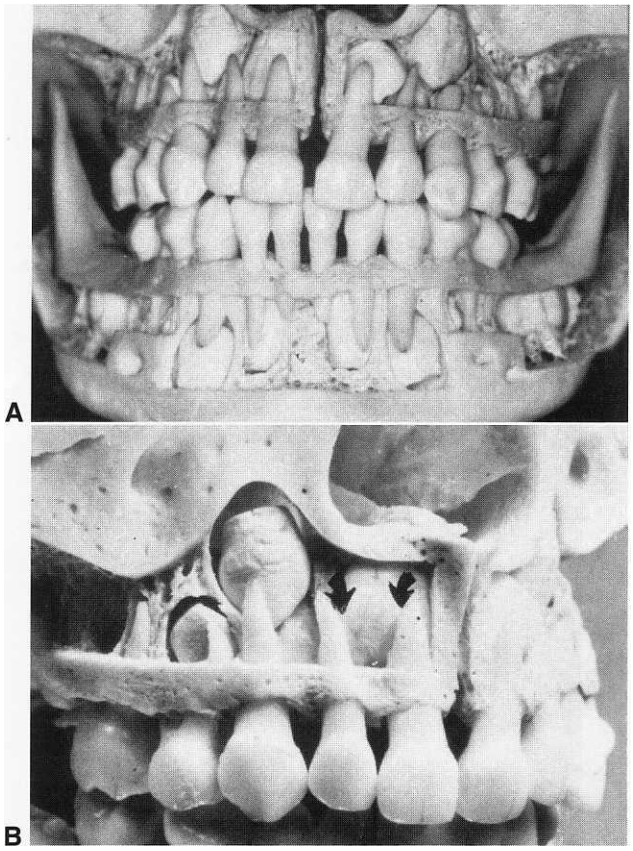
The displacement of anterior primary and permanent teeth presents a challenge in diagnosis and treatment for the dentist (Figs. 21-35 to 21-37). Relatively few studies have been reported that can be used as a guide in treating injuries of this type.

Primary Teeth. The intrusion by forceful impaction of maxillary anterior primary teeth is a common occurrence in children during the first 3 years of life. Frequent



f I C. **21-29.** A and **B,** Extensive crown fractures caused pulp exposures of both maxillary central incisors. The entire lingual plate of enamel was lost on the right central incisor. Pulpotomies have been completed. C, The occlusion permitted restorations using the feathered-edge technique. The teeth were restored with esthetic bonded composite resin restorations. Additional retention was gained by placement of restorative material in the pulp chambers; no pins or posts were used.

falls and striking of the teeth on hard objects may force the teeth into the alveolar process to the extent that the entire clinical crown becomes buried in bone and soft tissue. Although there is a difference of opinion regarding treatment of injuries of this type, it is generally agreed that immediate attention should be given to soft tissue damage. Intruded primary teeth should be observed; with few exceptions, no attempt should be made to reposition them after the accident. Most injuries of this type occur at an age when it would be



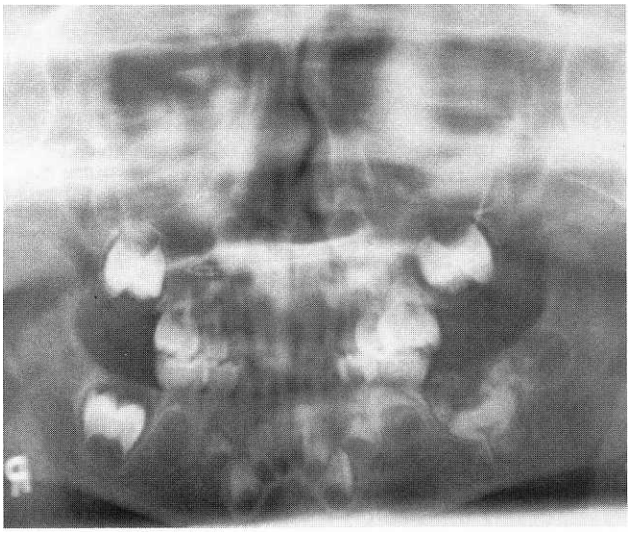
f **I G 21-30. A,** Skull of a 5-year-old child, revealing the relationship between primary and permanent teeth. **B,** Both the central and lateral primary incisors are in close relationship to the permanent incisors *(arrows). (From Andreasen JO, Sundstrom B, Ravn JJ:* Scand J Dent *Res 79:219-283, 1971.)*

difficult to construct a splint or a retaining appliance to stabilize the repositioned teeth.

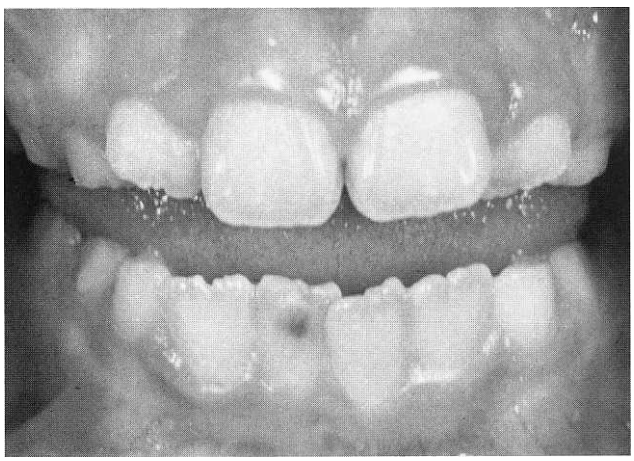
Normally the developing permanent incisor tooth buds lie lingual to the roots of the primary central incisors. Therefore, when an intrusive displacement occurs, the primary tooth usually remains labial to the developing permanent tooth (Fig. 21-38). If the intruded primary tooth is found to be in a lingual or encroaching relationship to the developing permanent tooth, it should be removed. Such a relationship may be confirmed from a lateral radiograph of the anterior segment.

The examination should be carried out as previously described, and radiographs should be made to detect evidence of root fracture, fracture of the alveolar bone, and damage to permanent teeth. However, predicting whether the permanent successors will show evidence of interrupted growth and development is impossible unless actual encroachment of their space can be seen radiographically.

Primary anterior teeth intruded as a result of a blow may often reerupt within 3 to 4 weeks after the injury



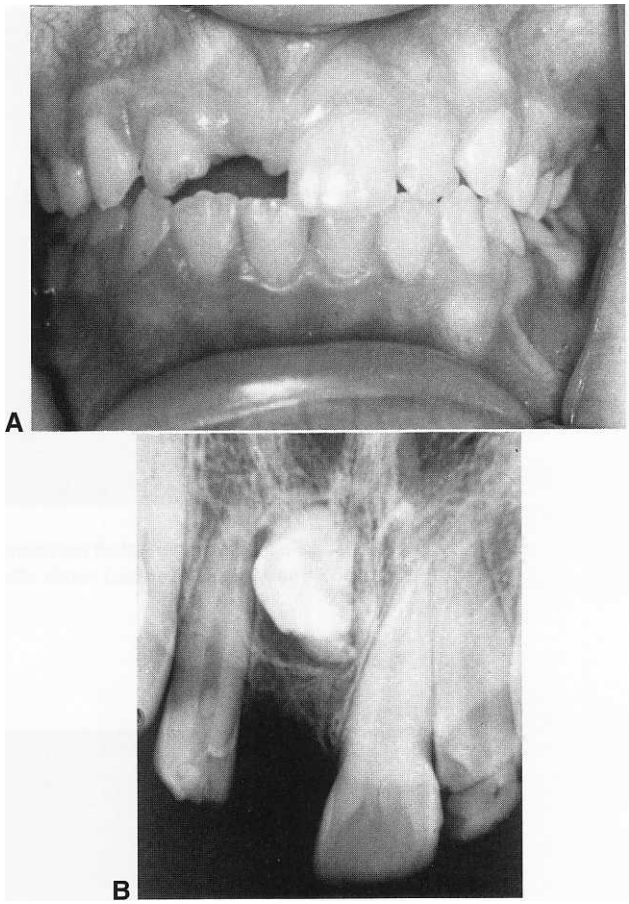
f **I G 21-31.** This radiograph of a 4-year-old child reveals improper formation of the mandibular left first permanent molar. At 18 months, the child had been viciously attacked by a dog. There had been a severe puncture wound of the lower left jaw from the dog's canines, although it was not known then that the injury involved the permanent molar tooth bud. The calcified lesion was removed and microscopically diagnosed as a developing tooth with displaced enamel matrix into follicular tissue. Early removal enhances the potential of the normally developing second permanent molar to eventually acquire an acceptable first permanent molar position.



II G **21-32.** Isolated hypoplastic area on the labial surface of the mandibular right permanent central incisor. There was a history of a trauma to the primary tooth when the child was younger than 2 years old.

In a study of the results of 248 traumatic episodes to primary incisors, Ravn reported 88 cases of intrusion.[10]

Of these teeth, four were extracted within 2 weeks because of infection and four did not reerupt and were extracted several months later, but the remaining 80 teeth fully reerupted within 6 months. Incipient



F **I G 21-33. A,** Hypoplastic areas on the permanent lateral incisors. The right central incisor has not erupted. **B,** Malformed central incisor. This condition can be traced to a severe trauma to the primary teeth.

reeruption was observed 14 days after the injury in a few instances. These teeth may even retain their vitality and later undergo normal resorption and be replaced on schedule by their permanent successors. During the first 6 months after the injury, however, the dentist often observes one or more of the reactions of the pulp and supporting tissues that have been mentioned previously in this chapter, the most common of which is pulpal necrosis. Even after reeruption a necrotic pulp can be treated if the tooth is sound in the alveolus and no pathologic root resorption is evident.

Primary teeth that are displaced but not intruded should be repositioned by the dentist or parent as soon as possible after the accident to prevent interference with occlusion. The prognosis for severely loosened primary teeth is poor. Frequently the teeth remain mobile and undergo rapid root resorption.

Skieller observed 60 children treated for looseness of one or more young teeth.' Loosened teeth were divided into three groups: simple looseness, dislocation with impaction, and dislocation with extrusion. He concluded

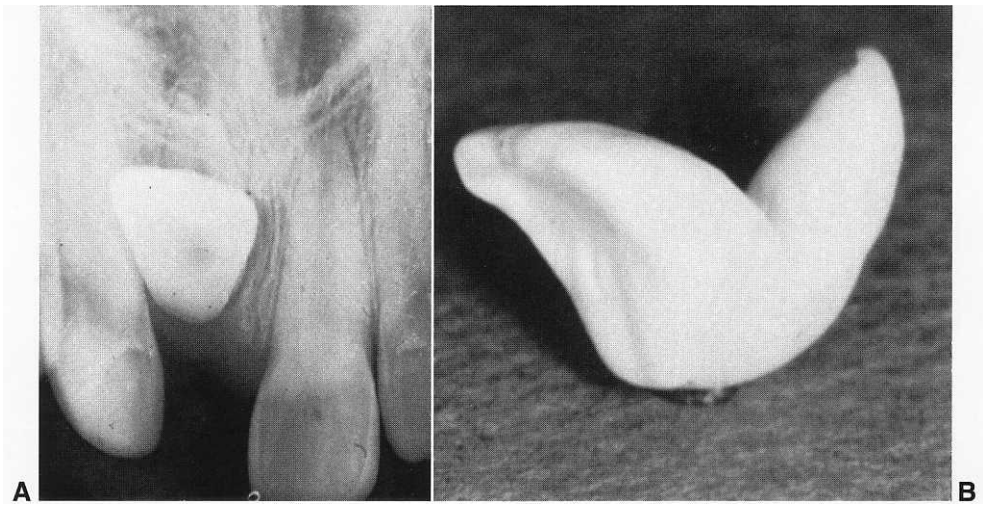


FIG 21-34. **A,** Dilacerated unerupted permanent central incisor. There was a history of a trauma at the age of 3 years. **B,** Dilacerated tooth after its surgical removal.

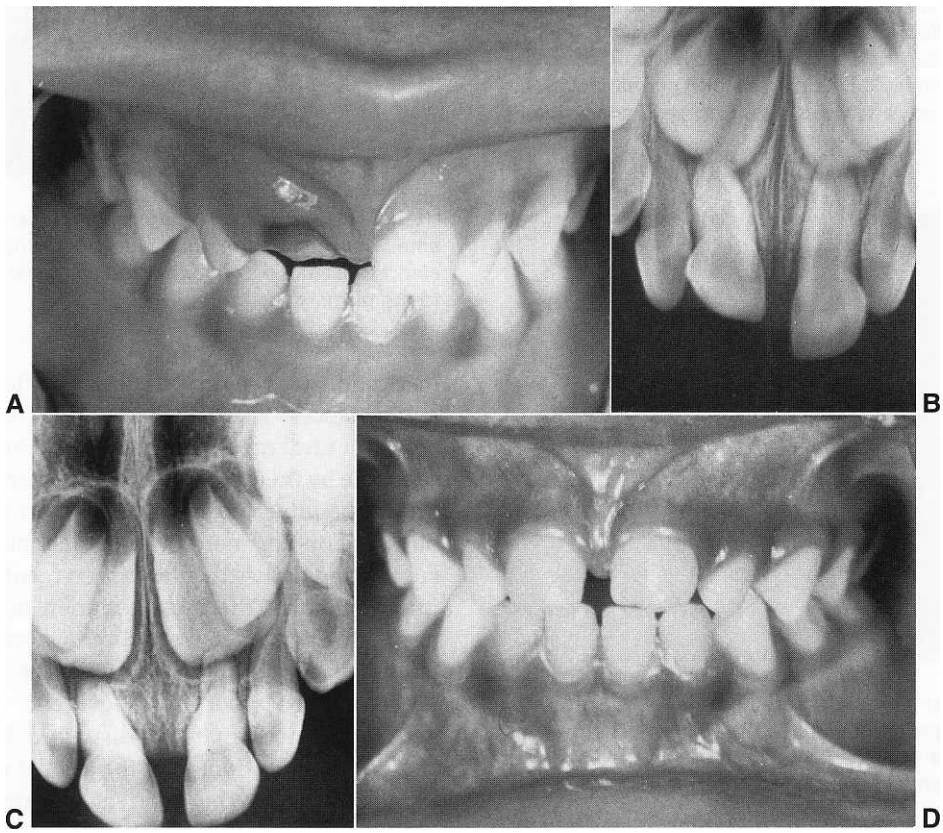
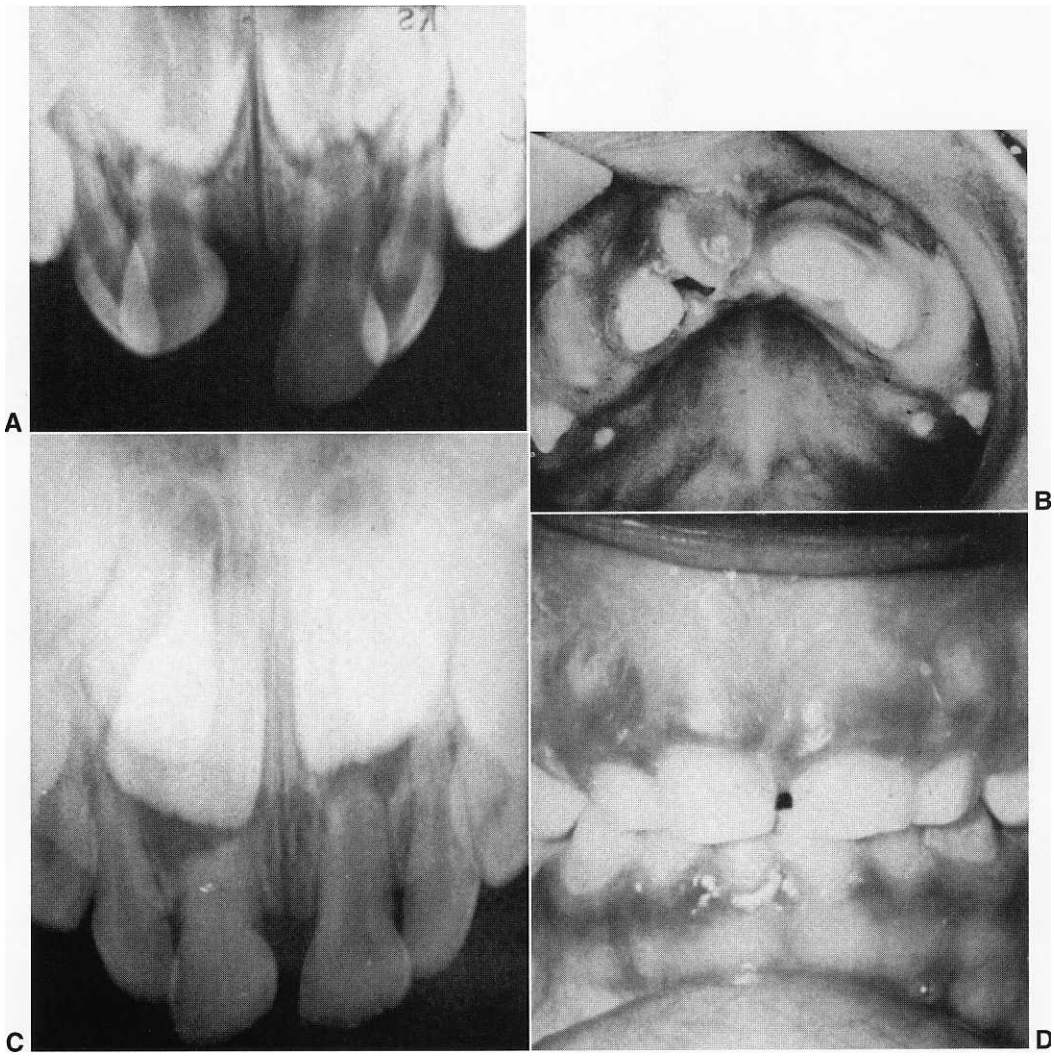


FIG 21-35. **A,** Intrusion of the maxillary right primary central incisor. **B,** The degree of intrusion of the primary tooth. Damage to the permanent teeth often results from an injury of this type. C, A radiograph taken 8 months after the injury shows that the injured tooth has reerupted. The pulp has retained its vitality, although there is evidence of partial obliteration of the pulp canal. External root resorption has occurred on the adjacent central incisor. **D,** The injured central incisor has assumed its normal place in the arch and probably will be retained until its normal exfoliation time.



F **I G 21-36. A,** Radiograph made shortly after the total intrusion of the maxillary right primary central incisor of a child 22 months of age. The tooth was rotated approximately 90 degrees. **B,** Appearance of tooth and healing soft tissues 2 weeks after the initial injury. No definitive treatment of the tooth was rendered. C and D, The same patient at 6 years, 5 months of age (4 years, 7 months after injury). The intruded tooth has undergone advanced root resorption and calcific metamorphosis, but the area is asymptomatic and the soft tissues appear healthy. The improved position of the tooth occurred spontaneously as the tooth reerupted. One year after this photograph was made, the tooth exfoliated normally.

that the immediate and future prognosis for the pulp was more favorable if root formation was still incomplete at the time of the accident. Root resorption, which was observed in all three groups of loosened teeth, was most common in impaction cases. Teeth with complete root formation seemed to undergo resorption more frequently than those with incomplete root formation. However, when resorption did occur, it was more extensive and progressed more rapidly in teeth with incomplete root development.

Permanent Teeth. Intruded permanent teeth apparently have a poorer prognosis than similarly injured primary teeth. The tendency for the injury to be followed by rapid root resorption, pulpal necrosis, or ankylosis is greater. The treatment for a permanent tooth with a closed root end consists in gradually repositioning the tooth orthodontically over a 2- or 3-week period and then continuing to stabilize the tooth for 2 to 4 weeks. The pulp should be extirpated 2 weeks after the injury, and calcium hydroxide should be placed in the root canal as an interim dressing. This treatment approach is recommended by Malmgren, Malmgren, and Goldson[32] and also by Trope, Chivian, and Sigurdsson.[33]

These same authors recommend that intruded immature permanent teeth be left to reerupt spontaneously, unless the intrusion is severe. If the immature tooth does



[I c **21-37. A,** A partially intruded maxillary right primary lateral incisor, a totally intruded right central incisor, an avulsed left central incisor, and a loosened left lateral incisor in a 14-month-old child. **B,** Radiograph of injured area made the same day as the trauma. C, Area 2 weeks after injury; the right central incisor shows evidence of reeruption. Space loss was a special concern, since the canines were actively erupting. D, Cemented space maintainer in position. **E,** Eruption of right central incisor is continuing 2 months after the injury. **F,** Four months after the injury the response is favorable. The space maintainer should be recemented and should remain in place at least until the canines and the central incisor are fully erupted. A more esthetically pleasing space maintainer may be considered later.



I **I I 21-38.** Radiograph of intruded primary central incisor with its root apex lying labial to the permanent central incisor.

not show early evidence of spontaneous reeruption (after 1 to 2 weeks) or if the intrusion is severe, orthodontic repositioning should be initiated. The clinician may elect to reposition the tooth orthodontically but delay endodontic therapy in the absence of unfavorable signs or symptoms. Endodontic therapy is often required, however, and the tooth should be monitored closely while a decision on endodontic therapy is pending.

Tronstad et al have reported on the management of severely intruded mature maxillary central incisors in an 11-year-old patient in whom spontaneous reeruption occurred." Rather than repositioning the teeth to gain endodontic access, they performed a palatal gingivectomy and endodontic treatment 10 days after the injury while the teeth remained in their intruded position. Eight weeks after the injury the teeth had reerupted naturally and were judged to be near their original position. Similar management of severely intruded immature central incisors has been reported by Shapira, Regev, and Liebfeld.[35] In these cases the palatal gingivectomies and endodontic treatment were per-formed 8 to 10 weeks after injury when periapical rarefactions and root resorption were noted radiographically. They noted accelerated spontaneous reeruption of all treated teeth soon after the gingivectomies and calcium hydroxide endodontic treatments were per-formed. Complete reeruption of the teeth and apparent periapical and periodontal healing had occurred 2 to 3 months after the surgical intervention.

It seems that both treatment approaches to treatment of severely intruded permanent teeth (early repositioning or waiting for spontaneous reeruption) have demonstrated reasonably successful results. However, the affected teeth seem to benefit by early calcium hydrox ide endodontic therapy with either treatment approach. The decision to reposition mechanically or hope for spontaneous reeruption of intruded permanent teeth remains a matter of clinical judgment that may be based on several conditions associated with the particular case.

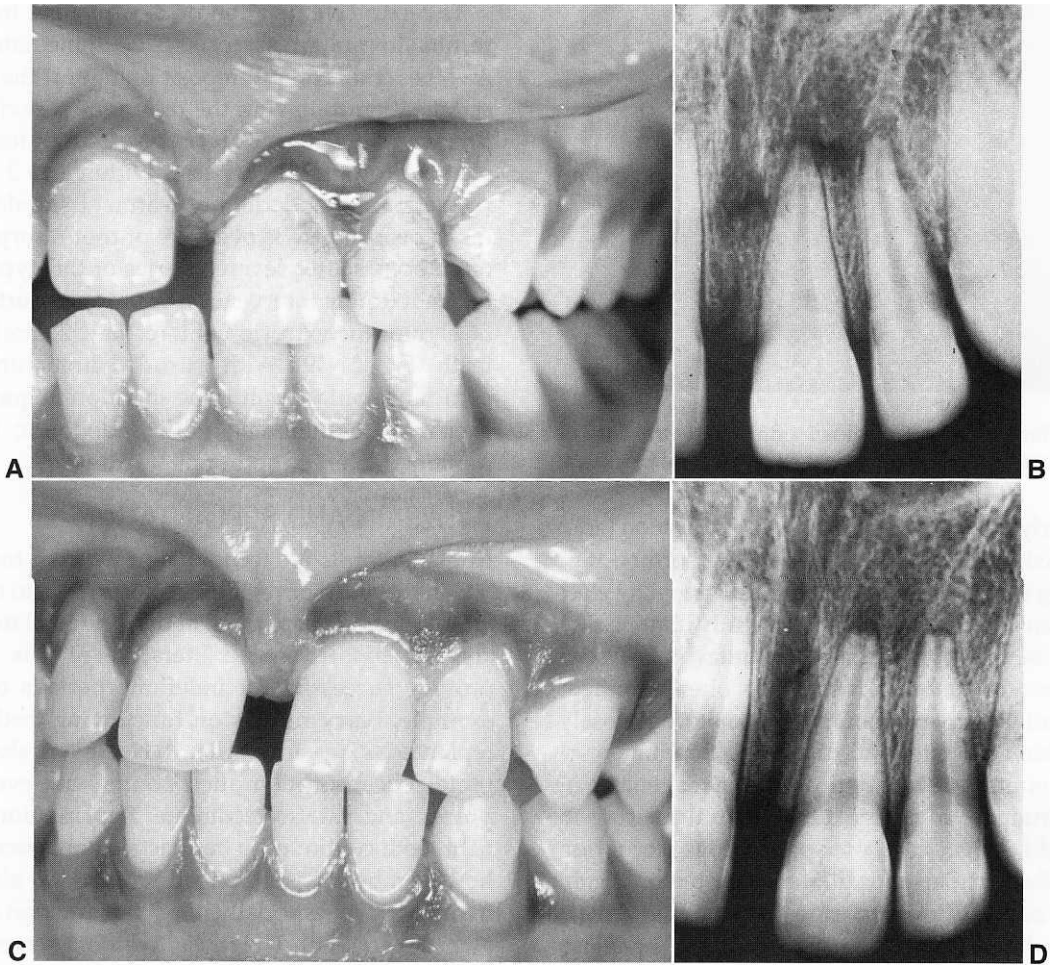
The extrusive luxation of a permanent tooth usually results in pulpal necrosis. The immediate treatment involves the careful repositioning of the tooth and stabilization following the technique described later in this chapter. If mature repositioned teeth do not respond to pulp vitality tests within 2 to 3 weeks after repositioning, endodontic treatment should be undertaken before there is evidence of root resorption, which often occurs after severe injuries of this type. The need for endodontic intervention is virtually certain in cases of significant extrusion (more than 2 mm) of mature teeth (Fig. 21-39). With extruded immature teeth, the clinician should monitor the situation frequently and be prepared to intervene with endodontic therapy, as described later, if conditions warrant.

AVULSION AND REPLANTATION

Replantation is the technique in which a tooth, usually one in the anterior region, is reinserted into the alveolus after its loss or displacement by accidental means. There are few reports in the literature of this technique's proving successful for indefinite periods of time. For example, Barry reports on functioning teeth that were replanted 42 years earlier.[3t] However, slow or even rapid root resorption often occurs with even the most precise and careful technique. Replantation of permanent teeth continues to be practiced and recommended, however, because prolonged retention is also achieved in many cases, especially when replantation occurs soon after the accident. The replanted tooth serves as a space maintainer and often guides adjacent teeth into their proper position in the arch, a function that is important during the transitional dentition period. The replantation procedure also has psychologic value. It gives the unfortunate child and parents hope for success; even though they are told of the possibility of eventual loss of the tooth, the early result often appears favorable and softens the emotional blow of the accident (Fig. 21-40).

The success of the replantation procedure is undoubtedly related to the length of time that elapses between the loss of the tooth and its replacement in the socket. The condition of the tooth, and particularly the condition of the periodontal ligament tissue remaining on the root surface, are also important factors that influence the success of replantation. There have been reports that immediate replacement of a permanent tooth occasionally results in maintenance of vitality and indefinite retention. However, replantation should generally be viewed as a temporary measure. Under favorable conditions, many replanted teeth are retained for 5 or 10 years and a few for a lifetime. Others, however, fail soon after replantation.

Camp reports that the tooth most commonly avulsed in both the primary and the permanent dentition is a maxillary central incisor.[37] Most often an avulsion injury involves only a single tooth. Avulsion injuries are three

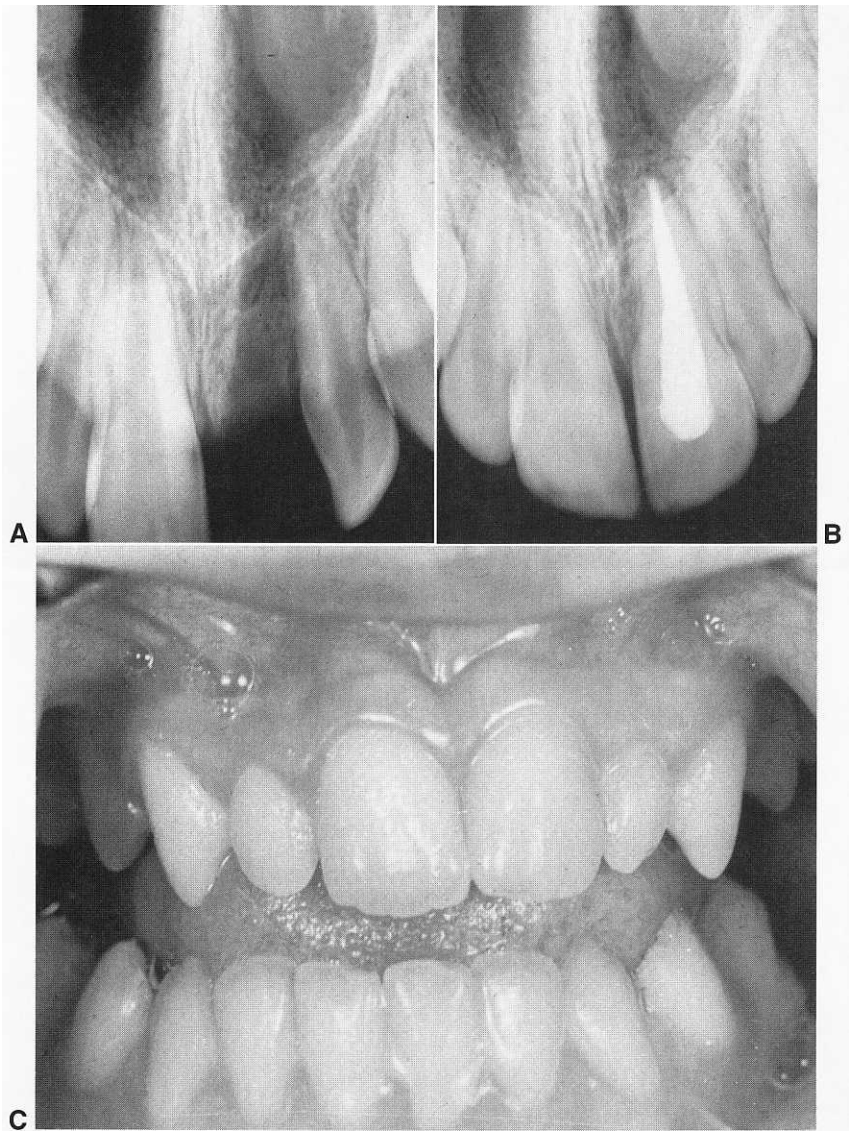


f G. 21-39. A, A severe blow to the maxillary anterior teeth resulted in the extrusion of the left central and lateral incisors. **B,** A radiograph demonstrates a fracture of alveolar bone. C, One month after the repositioning and stabilization of the teeth, they are firmly attached and the gingival tissues appear normal. D, A radiograph demonstrates healing of the fractured alveolar bone, but there is evidence of apical root resorption. Endodontic treatment was recommended.

times more frequent in boys than in girls and occur most commonly in children 7 to 9 years of age when permanent incisors are erupting. Andreasen suggests that the loosely structured periodontal ligament surrounding the erupting teeth favors complete avulsion."

The sooner a tooth can be replanted in its socket after avulsion, the better the prognosis will be for retention without root resorption (Figs. 21-41 and 21-42). Andreasen and Hjorting-Hansen reported a follow-up study of 110 replanted teeth. Of those replanted within 30 minutes, 90% showed no discernible evidence of resorption 2 or more years later.'" However, 95% of the teeth replanted more than 2 hours after the injury showed root resorption. If the tooth has been out of the mouth for less than 30 minutes, the prognosis is therefore more favorable. Also, if the apical end of the tooth is incompletely developed at the time of the injury, there is a greater chance of regaining pulp vitality after replantation. If the apex is closed, the dentist should proceed with a pulpectomy a few days after the replantation, even if the extraoral time for the tooth was brief.

If a parent calls to report that a tooth has been avulsed and it can be determined that the injury is without other oral, neurologic, or higher-priority physical complications, the dentist may instruct the parent to replace it in the socket immediately and to hold it in place with light finger pressure while the patient is brought to the dental office. If the avulsion occurred in a clean environment, nothing should be done to the tooth before the parent replants it. If the tooth is dirty, an attempt should be made to clean the root surface, but it is very important to preserve any remnants of the periodontal ligament that are still attached to the root. Therefore the parent would then be instructed to keep the tooth immersed in a suitable storage medium and bring the child and the tooth for immediate care.

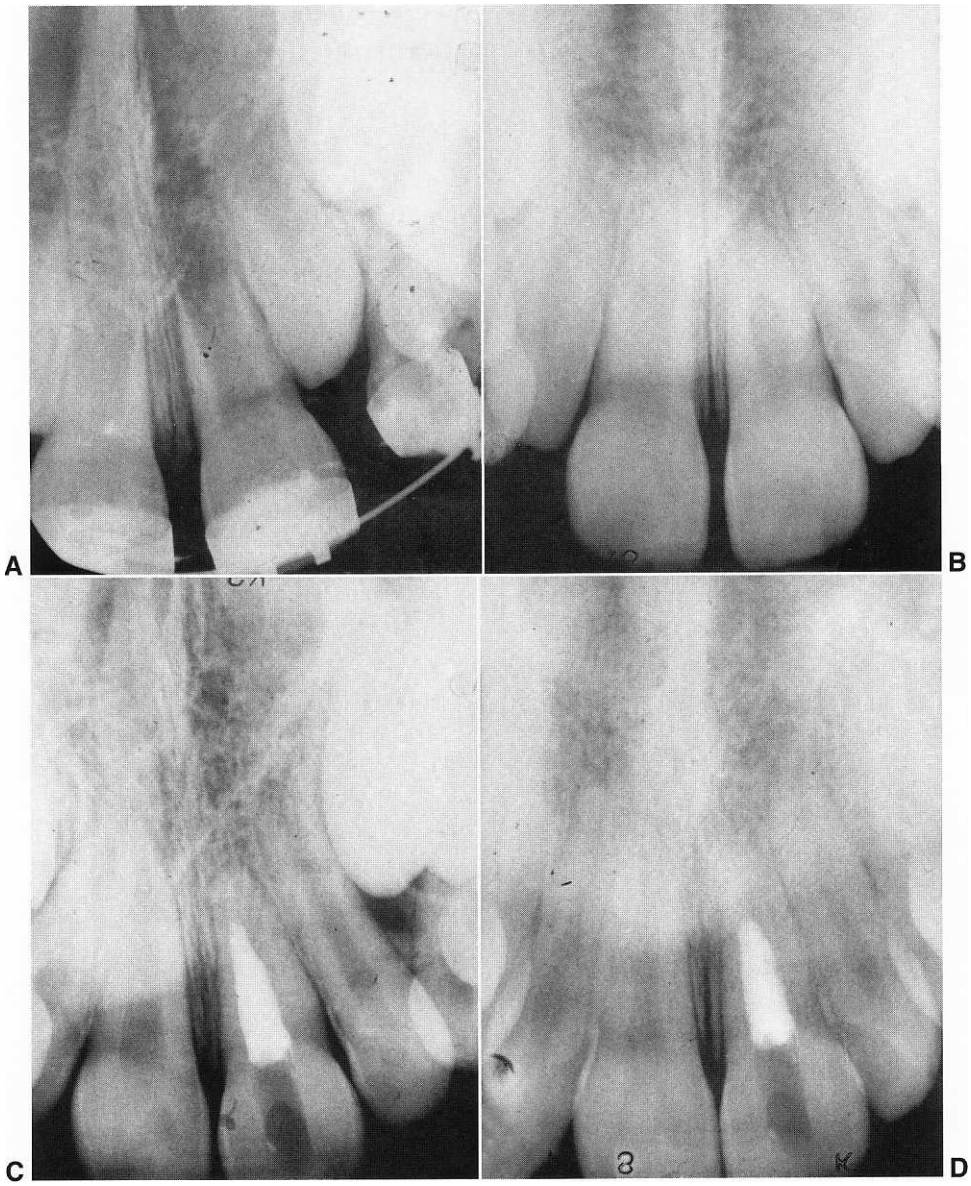


f I i. **21-40. A,** Radiograph of a patient who lost a central incisor as the result of trauma. Replantation was performed. **B,** After the replantation procedure, peripheral root resorption is evident along the mesial surface. C, The relationship of the replanted tooth in the arch; the gingival tissue is normal.

Milk has been shown to be a suitable storage medium that is also often readily available (skim or low-fat milk, if available, is preferred). Isotonic saline is another excellent solution to use for this purpose if it is available. A commercial product designed specifically for storing avulsed teeth is the Emergency Medical Treatment Toothsaver (EMT Toothsaver).* The system includes an appropriate container for storage and transport of the tooth while immersed in a pH-balanced cell culture fluid (similar to Hanks balanced salt solution). This product has a 2-year shelf-life without refrigeration.

*SmartPractice, Phoenix, Ariz.

The tooth must be kept moist during the trip to the dental office if the parent cannot or will not replant it. Allowing the avulsed tooth to dehydrate before replantation is damaging to a favorable prognosis. Many studies, including that by Blomlof, Otteskog, and Hammarstrom, have compared various storage solutions for avulsed teeth.'"A compilation of this information indicates that Hanks buffered saline, isotonic saline, and pasteurized bovine milk may be the most favorable known storage media. If none of these solutions is readily available, human saliva is an acceptable short-term substitute storage liquid. Presumably, the patient's saliva (and perhaps blood) would be readily available to collect in a small container (or a



f 1. **21-41. A,** An immature permanent left central incisor soon after replantation and stabilization. The tooth was replanted by the mother within 15 minutes after avulsion. The dentist instructed her by phone before the child was brought to the dental office. The tooth had erupted only about 3 mm when it was avulsed. **B,** Replanted tooth 11 months after injury shows continued but blunted root development and thickened apical calcification. The tooth was then treated with a paste of calcium hydroxide and camphorated mono-parachlorophenol for several months. C, Two years after the injury, soon after the root canal was obliterated with gutta-percha. **D,** A favorable response can be seen 11 h years after the injury. The crown was restored with a full-coverage restoration approximately 25 years after the avulsion. *(Courtesy* Dr. Guthrie Carr.)

cupped hand) in which to keep the tooth moist while transporting it to the dental office. Although tap water has been a commonly recommended storage solution (and its use would be preferable to allowing dehydration of the tooth), saliva is a better storage medium. Neither water nor saliva is as good as milk or saline, if the tooth must be stored for a long period (more than 30 minutes before replantation). Because water is hypotonic, its use leads to rapid cell lysis and increased inflammation on replantation.

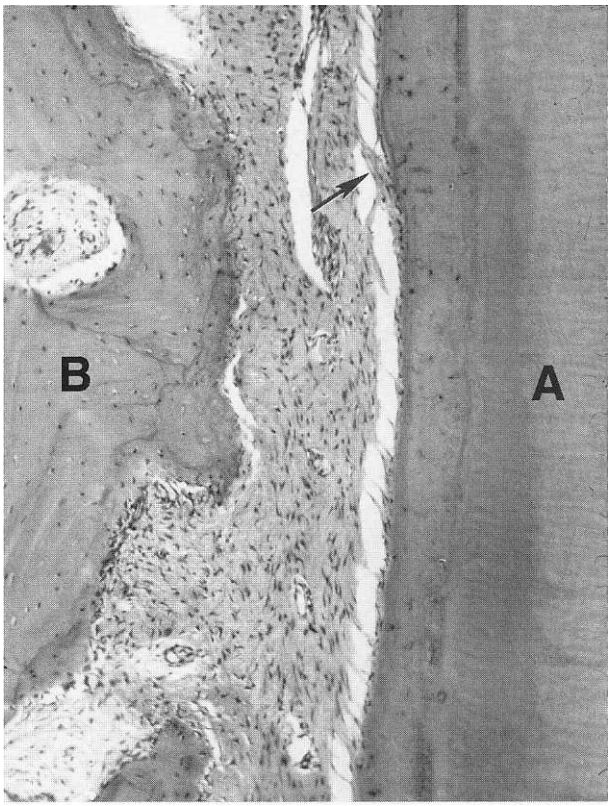
The patient should receive immediate attention after arriving at the dental office. If the tooth has not already been replanted, the dentist should make every effort to minimize the additional time that the tooth is out of the



[I G **21-42. A,** Maxillary left central incisor immediately after replantation and root canal filling with calcium hydroxide and camphorated mono-parachlorophenol. The tooth had been avulsed 2'/2 days earlier and had been stored in a dresser drawer and allowed to dry since that time. **B,** Radiograph 15 months after the injury shows evidence of external root resorption along the middistal portion of the root and to a lesser degree along the mesial root surface. C, Radiograph made 4 2/3 years after the injury shows the resorptive process continuing. D, Although the tooth was asymptomatic when this radiograph was made 5'/, years postoperatively, the progressive resorption will soon render the tooth nonfunctional. The response of the replanted tooth has been more favorable than might have been expected in view of the long, dry storage period before replantation. The treatment has enabled the patient to become psychologically prepared for the tooth loss, and it has delayed the need for prosthetic replacement. (Courtesy Dr. Guthrie E. Carr.)

socket. The patient's general status should be quickly assessed to confirm that there are no higher-priority injuries.

If an evaluation of the socket area shows no evidence of alveolar fracture or severe soft tissue injury, the tooth is intact, and only a few minutes have elapsed since the injury, the dentist should replant the tooth immediately. Under the conditions just described, every effort should be directed toward preserving a viable periodontal ligament. Trope correctly asserts that treatment is directed at avoiding or minimizing the resultant inflammation, which occurs as a direct result of the two main consequences of tooth avulsion: attachment damage and pulpal infection' If the tooth was cleanly avulsed, it can probably be replanted without local anesthetic, and obtaining the initial radiograph can also be delayed until the tooth is replaced in the socket and held with finger pressure. The minutes saved may contribute to a more successful replantation. If a clot is present in the socket, it will be displaced as the tooth is repositioned; the socket walls should not be scraped with an instrument. If the tooth does not slip back into position with



FIC **21-43.** Microscopic section of a tooth replanted with the periodontal ligament intact. The reattachment of the periodontal fibers is illustrated by their crossing the tear in the periodontal ligament. *A,* Tooth; B, alveolar bone. (Courtesy Dr. Philip Sherman, Jr.)

relative ease when finger pressure is used, local anesthesia and a radiographic evaluation are indicated. Local anesthetic should also be administered when fractured and displaced alveolar bone must be repositioned before the tooth is replanted. Soft tissue suturing may be delayed until the tooth has been replaced in the socket; however, the suturing should be performed to control hemorrhage before the tooth is stabilized with a bonded splint. Splinting techniques are discussed in the next section of this chapter.

Sherman studied the mechanism by which the replanted tooth becomes secured in the alveolus.` Intentional replantation was performed on 25 incisors in dogs and monkeys. The root canals were hermetically sealed with gutta-percha, and the teeth were splinted for 1 month. Subsequent microscopic examination under fluorescent and incandescent light revealed deposition of secondary cementum and new alveolar bone, which entrapped the periodontal fibers (Fig. 21-43).

The preservation of an intact and viable periodontal ligament is the most important factor in achieving healing without root resorption. Delicate handling of the tooth, storage in an appropriate moist environment, quick replantation, and appropriate stabilization are all important in preserving the periodontal ligament. Undesirable periodontal ligament reactions may result in replacement resorption (ankylosis) or inflammatory resorption of the root. Either reaction may cause eventual loss of the tooth unless the resorption can be controlled. Use of an enamel matrix derivative (Emdogain*) has been shown to increase the incidence of healed periodontal ligament when this gel is applied to the root surface of the avulsed tooth and/or inserted directly into the alveolar socket prior to implantation. It appears to aid in preventing or retarding resorption and ankylosis.

In the past, few attempts were made to replant avulsed primary teeth; however, there have been a few reports of success with this procedure. Andreasen and Andreasen have stated that replantation of primary teeth is contraindicated because of the poor prognosis for success and the additional risk of further injury to the succedaneous tooth.}[3] We agree that, even under the most ideal conditions, replanting a primary tooth has higher risk and poorer prognosis despite the remote chance for a favorable outcome.

Stabilization of Replanted Teeth. After replantation of a tooth that has been avulsed, a splint is required to stabilize it during at least the first week of healing. Camp" has stated that an acceptable splint should meet the following criteria:

1. It should be easy to fabricate directly in the mouth without lengthy laboratory procedures.
2. It should be able to be placed passively without causing forces on the teeth.
3. It should not touch the gingival tissues, causing gingival irritation.
4. It should not interfere with normal occlusion.
5. It should be easily cleaned and allow for proper oral hygiene.
6. It should not traumatize the teeth or gingiva during application.
7. It should allow an approach for endodontic therapy.
8. It should be easily removed.

The splint should also allow mobility of the replanted tooth that is comparable with the normal mobility of a tooth. Rigid stabilization seems to stimulate replacement resorption of the root. Hurst has demonstrated that rigid stabilization of a replanted tooth is detrimental to proper healing of the periodontal ligament."

The bonded resin and wire splint satisfies all the criteria just described. It can be used in most situations requiring the stabilization of one or more teeth if

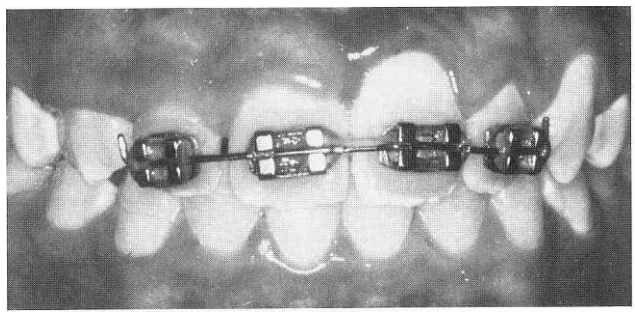
*Biora, Chicago, Ill., and Malmh Sweden.

sufficient sound teeth remain for anchorage (see Fig. 21-48). Rectangular or round orthodontic wire is bent to approximate the arch configuration along the midportion of the labial surfaces of the teeth to be incorporated in the splint. At least one sound tooth on each side of the tooth to be stabilized is included. The size of the wire is not too critical, but rectangular wire should be at least 0.016 x 0.022 inch and round wire at least 0.018 inch. If three or four teeth must be stabilized, a stiffer wire (such as 0.028-inch round wire) is required. If round wire is used, a right-angle bend should be made near each end of the wire to prevent rotation of the wire in the resin. A 20- to 30-pound-test monofilament nylon line is an acceptable substitute for wire in the splint.

If the labial enamel surfaces to be etched are not plaque free, they should be cleaned with a pumice slurry, rinsed, thoroughly dried, and isolated with cotton rolls. The enamel surfaces are etched with a phosphoric acid etchant; the gel form is convenient. The enamel surfaces are thoroughly washed and dried again. The wire is then attached to the abutment teeth by placing increments of the resin material over the wire and onto the etched enamel. The resin should completely surround a segment of the wire, but it should not encroach on the proximal contacts or embrasures. The replanted tooth is then held in position while resin is used to bond it to the wire. The resin may be lightly finished if necessary after polymerization. The splint is easily removed (usually 7 to 14 days later) by cutting through the resin with a bur to uncover the wire. The remaining resin may then be removed with conventional finishing instruments. If the splint is used to stabilize lower teeth, it may be necessary to affix the wire to the lingual surfaces if placing it on the labial surfaces will interfere with natural occlusion. Because lingual surfaces are more likely to be contaminated with saliva during the procedure, however, labial placement is preferred whenever possible.

Direct-bonded orthodontic brackets may also be placed on the teeth, and a light labial archwire bent to accurately conform to the natural curvature of the arch is then ligated to the brackets. The brackets are properly aligned on the archwire and bonded to the abutment teeth first. Then the avulsed tooth is ideally positioned and additional bonding material is placed, if necessary, to fill any remaining small space between the tooth and the bracket before bonding it to the splint. If performed properly, this technique results in an excellent splint (Fig. 21-44). However, it requires much more accurate and precise wire bending than the bonded resin and wire technique (without brackets) to achieve a passive appliance.

If the patient is mentally disabled or has immature behavior and does not tolerate foreign objects in the mouth well, or if there are insufficient abutment teeth

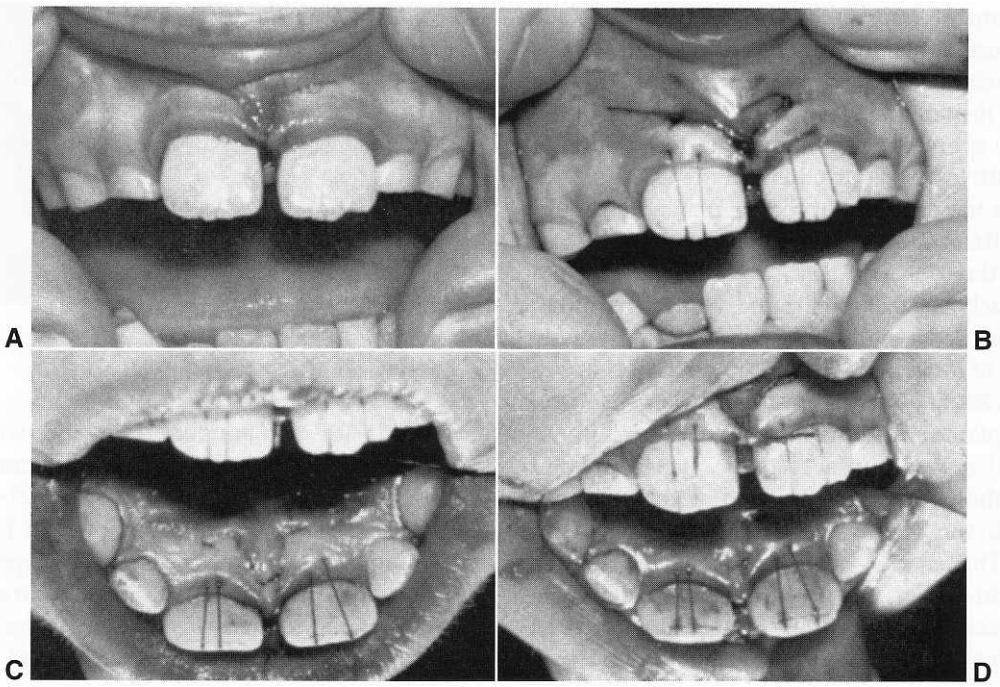


[I G **21-44.** Bonded brackets and archwire splint.

available for the bonded resin and wire splint, the suture and bonded resin splint advocated by Camp[37] may be an acceptable alternative (Fig. 21-45). Examples of other types of splint are shown in Fig. 21-46. The titanium trauma splint has been developed by von Arx, Filippi, and Buser to ease the application and removal of the splint and to increase comfort for the patient.}[5]

In general, stabilization for replanted teeth without other complications is required for 7 to 14 days. The periodontal ligament fibers should have healed sufficiently after the first week to remove the splint. However, the patient should be advised not to bite directly on the replanted tooth for 3 to 4 weeks after the injury and then gradually to begin to return to normal use of the tooth. During this time, food may be cut into bite-size pieces and chewed carefully with unaffected teeth. The patient should maintain good oral hygiene by brushing and flossing normally and using chlorhexidine mouthrinses.

We recommend that systemic antibiotic therapy begin immediately and continue for at least a week following replantation. If the apex is closed, extending the antibiotic therapy until the pulp is extirpated seems to be a good way to determine the duration of antibiotic coverage. Research by Sae-Lim, Wang, and Trope has shown that antibiotic therapy is effective in preventing the development of external inflammatory root resorption of replanted teeth in which the pulps were not extirpated.[4] This finding suggests that antibiotic therapy may also be helpful in those cases in which the pulps of immature replanted teeth are allowed to remain while hoping for revitalization. Additional studies in this area are indicated. In fact, research in this area is ongoing to find better storage and soaking media. Various antibiotics, fluoride liquids and gels, glucocorticoids, and a liver transport medium are being investigated. Krasner and Rankow have published detailed recommendations for replanting a tooth based on its status as judged by the clinician's determination of the physiologic condition of the root periodontal ligament cells, the development of the root apex, and the length of extraoral time." Their recommendations recognize 10 different



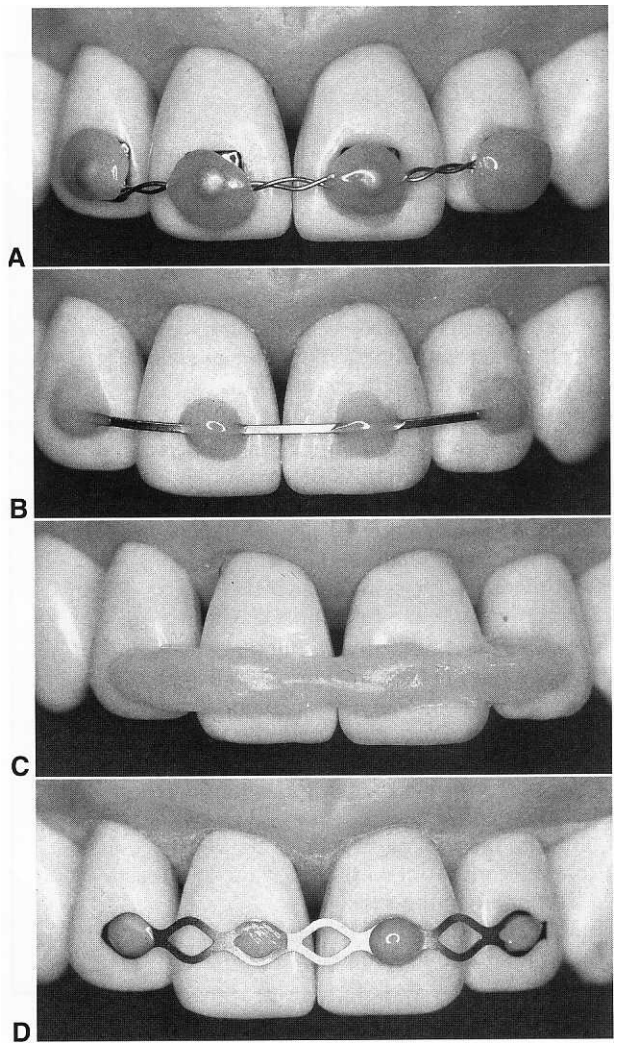
F **I G 21-45.** Suture and a bonded resin splint. A, The maxillary central incisors have been replanted. Proper stability of a bonded resin and wire splint may be difficult to obtain, because two permanent teeth need to be supported and only smaller primary teeth are readily available as abutments. The labial surfaces of the replanted teeth are etched. **B** and C, A suture in place over each tooth. Starting in the labial tissue, each suture crosses the incisal edge, enters and exits the lingual tissue, recrosses the incisal edge, and reenters the labial tissue. The ends then are tied. D, Resin is placed over a small portion of the suture and the etched enamel to retain the suture in proper position. *(From Camp JH: Replantation of teeth following trauma. In McDonald RE et al, editors:* Current therapy in dentistry, *vol 7, St Louis, 1980, Mosby.)*

categories of avulsed teeth and detail a specific treatment regimen for each category. A modified version of Krasner's (2003) Table of Categories for Treatment of Avulsed Teeth is shown in Box 21-1. Of course, the dentist should confirm at the time of replantation that the patient is adequately immunized against tetanus.

Endodontic Management of Replanted Teeth. All replanted permanent teeth with complete apical root development should undergo a pulpectomy soon after replantation regardless of the length of time the tooth was out of the mouth. Even though a few reports of revitalization exist, the chances for revitalization are remote at best. Moreover, adverse reactions are virtually certain if degenerating pulp tissue is allowed to remain in the canals for more than a few days. The risk/benefit ratio for the patient favors pulpal extirpation.

Because replantation should be done as soon as possible after the injury, the dentist should not take time to extirpate the pulp before replantation. The pulp should be extirpated before the splint is removed, how-ever, and preferably within 1 week after the injury. A sterile, dry cotton pellet or one dampened with CMCP and blotted on sterile gauze may be sealed in the pulp chamber after debridement and irrigation. The canal should be filled approximately 2 weeks after the injury. When the canal is filled, calcium hydroxide paste is the material of choice. However, Trope suggests that the pulp contents be removed at the emergency visit and a tetracycline-corticosteroid combination (Ledermix) be placed in the root canal.' He feels that this combination decreases the inflammatory response after replantation to allow for more favorable healing than in those teeth that do not receive the medicament.

Research by Andreasen indicates that early extirpation of the pulp may help to control the early onset of inflammatory root resorption.[3s] Filling the root canal with calcium hydroxide also controls and may even arrest external inflammatory root resorption. If the calcium hydroxide is placed in the canal too soon (before adequate healing of the periodontal ligament), however, it may stimulate replacement root resorption. Andreasen suggested that 2 weeks after the replantation is the ideal time to fill the canal with calcium hydroxide. The use of calcium hydroxide as a root canal—filling material was described previously in the discussion of apexification.



f **I G 21-46.** Examples of bonded splints. A, Button with stainless steel ligature and acrylic caps. **B,** Orthodontic archwire. C, Fiber-filled acrylic. **D,** Titanium trauma splint. *(Courtesy* Dr. Adreas Filippi.)

If the avulsed permanent tooth has immature root formation with an open apex, the chances of pulpal revitalization after replantation improve considerably, especially if replantation occurs less than 30 minutes after avulsion. If the avulsed tooth has been cared for properly, there is a small chance for revitalization even if the tooth is replanted 2 hours after the injury. However, many do not revitalize. Those that do respond favorably may still require root canal treatment several months later. During the time beyond 1 week that the pulp tissue is allowed to remain, evaluation of the tooth is recommended at weekly intervals until favorable signs of healing without pulp pathosis are conclusive (vitality tests are unreliable) or until a decision is made to extirpate the pulp. The pulp should be extirpated when the first signs of degeneration appear.

Rubber darn isolation is always desirable when performing pulp therapy. It can usually be employed even during the pulp extirpation procedure while several teeth are splinted together. Instead of separate holes in the rubber dam for each tooth, a slit is made so that the rubber can be placed over all teeth in the splinted segment. This does not afford ideal isolation, but it is generally better than the use of cotton rolls. In addition, the rubber dam helps prevent the swallowing or aspiration of foreign objects during treatment. If small endodontic instruments are used without rubber dam protection, they should be secured with a length of dental floss to facilitate retrieval in the unlikely event that they are dropped in the patient's mouth.

The calcium hydroxide material used to fill the root canal should be replaced every 3 to 6 months until a decision is made to fill the canal with gutta-percha. The optimum duration of the calcium hydroxide treatment is unknown, but generally calcium hydroxide should be kept in the canal for at least 6 months or until root end closure (apical plug) occurs beyond 1 year. hz cases in which an adjacent tooth is still unerupted, Fountain and Camp recommend continuing the calcium hydroxide treatment until after the eruption of the adjacent tooth.'s It is believed that eruption may stimulate or accelerate the resorptive process in a nearby replanted root.

MANAGEMENT OF ROOT FRACTURES

Root fracture of primary teeth is relatively uncommon because the more pliable alveolar bone allows displacement of the tooth. When root fracture does occur, it should be treated in the same manner as was recommended for permanent teeth; however, the prognosis is less favorable. The pulp in a permanent tooth with a fractured root has a better chance to recover.

Root fractures that occur in the apical half of the tooth are more likely to undergo repair (Fig. 21-47). Fractures in the apical third are often repaired without treatment. In fact, many apparently are undetected until evidence of a calcified repair is seen radiographically some time after the injury.

Andreasen[33] has described four tissue reactions after root fracture: (1) healing with calcified tissue, which is characterized by a uniting callus of hard tissue that may consist of dentin, osteodentin, or cementum; (2) healing with interposition of connective tissue, in which the fractured root surfaces are covered by cementum with connective tissue fibers joining the two fragments; (3) healing with interposition of bone and connective tissue, in which a bony bridge and connective tissue are positioned between the fragments; and (4) interposition of granulation tissue.

The last is the least favorable form of attempted repair, and the fracture will not heal spontaneously.

21-1 Categories for Treatment of Avulsed Teeth

MATURE APEX

Less than 15 minutes out of mouth:
Rinse the tooth with physiologic solution to remove debris from the root surface.
Flush the socket with sterile water or saline.
Reimplant the tooth in the socket.

15 minutes to 6 hours in physiologic solution:
Place the tooth in Hank's Balanced Salt Solution for 30 minutes.
Flush the socket with sterile water or saline.
Reimplant the tooth in the socket.
Splint the tooth in a functional position.

15 minutes to 1 hour in nonphysiologic storage:
Place the tooth in Hank's Balanced Salt Solution for 30 minutes.
Flush the socket with sterile water or saline.
Reimplant the tooth in socket.
Splint the tooth in a functional position.

Greater than 1 hour dry storage:
Remove remnants of the periodontal ligament by soaking in sodium hypochlorite for 10 to 15 minutes.
Instrument the root canal with the tooth out of the mouth.
Soak the tooth in 2% stannous fluoride solution for 5 minutes.
Obturate the root canal with gutta-percha.
Coat the tooth root with Emdogain, and place Emdogain in the socket.
Reimplant the tooth in the socket.
Splint the tooth in a functional position.

IMMATURE APEX

Less than 15 minutes out of mouth:
Soak the tooth in a doxycycline solution for 5 minutes.
Reimplant the tooth in the socket.
Splint the tooth in a functional position.
Check the tooth for vitality and apex closure every month.

15 minutes to 6 hours in physiologic solution:
Soak the tooth in a doxycycline solution for 5 minutes.
Flush the socket with sterile water or saline.
Reimplant the tooth in the socket.
Splint the tooth in a functional position.

15 minutes to 1 hour in nonphysiologic solution:
Place the tooth in Hank's Balanced Salt Solution for 30 minutes.
Soak the tooth in a doxycycline solution for 5 minutes.
Flush the socket with sterile water or saline.
Reimplant the tooth in the socket.
Splint the tooth in a functional position.

Greater than 1 hour dry storage:
Remove remnants of the periodontal ligament by soaking in sodium hypochlorite for 10 to 15 minutes.
Instrument the root canal with the tooth out of the mouth.
Soak the tooth in a 2% stannous fluoride solution for 5 minutes.
Obturate the root canal with gutta-percha.
Coat the tooth root with Emdogain, and place Emdogain in the socket.
Reimplant the tooth in the socket.
Splint the tooth in a functional position.

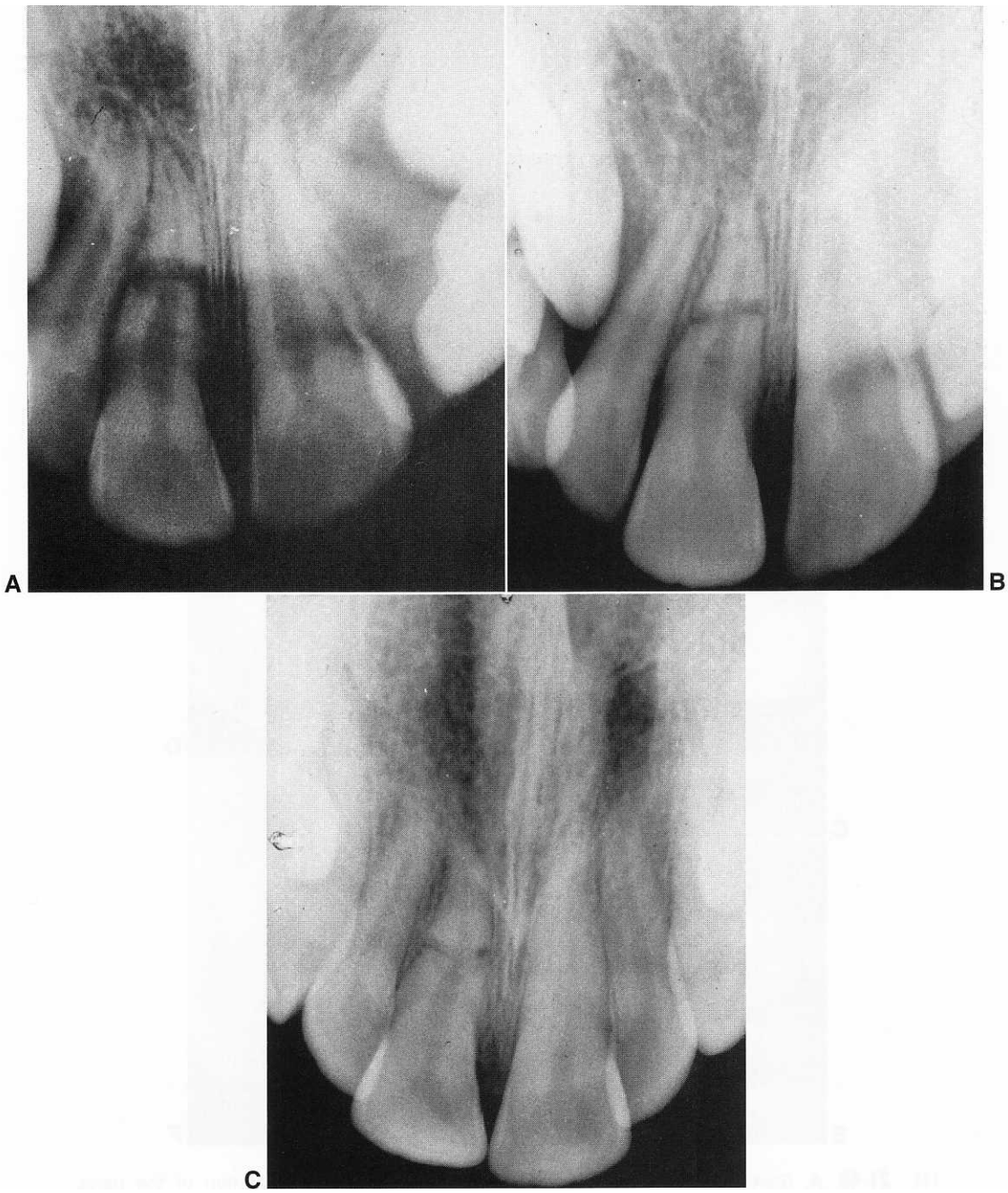
*Adapted from Krasner P: Advances in the treatment of avulsed teeth, Data *Today* 22:84-87, 2003.

The teeth usually present unfavorable symptoms that may be accompanied by fistulas resulting from necrosis of the coronal portion and also sometimes the apical portion of the pulp. These teeth require follow-up endodon tic treatment or extraction.

Gross separation of the root fragments invariably causes inflammation in the area and subsequent resorption of the approximating fractured surfaces (Fig. 21-48). For repair to take place, the fragments must be maintained in apposition. Therefore splinting is usually necessary, particularly if the coronal fragment is mobile.

Although a relatively long stabilization period (3 months) has been previously recommended for teeth with fractured roots, Cvek et al have cast doubts on the efficacy of long-term splinting and the types of splints used for root fracture healing."[9,50] Previously a longer stabilization period seemed necessary to encourage a more favorable type of healing of calcified tissue; however, their study showed no significant difference in the frequency of healing when short periods (less than 60 days) and long periods (60 to 90 days) of splinting were compared. They found that hard tissue healing also took place in teeth that were not even splinted. A comparison between nonsplinted and splinted teeth showed no difference in frequency of healing. They suggest that optimal positioning of dislocated fragments significantly increases the frequency of healing, particularly in mature teeth. Their study showed that, in immature teeth, healing took place even after suboptimal repositioning of dislocated coronal fragments and persistent diastases between the fragments after splinting. They conclude by suggesting that teeth with no or slight loosening of the fragment may not require splinting.

In addition, there is general agreement that splints for root fractures should be more rigid than the splints used for stabilization after other types of displacement injuries. Application of a more rigid splint is also believed to enhance the opportunity for a calcified tissue repair. Therefore use of heavier wires is recommended (0.032 to 0.036 inch) when one is stabilizing teeth with fractured roots.

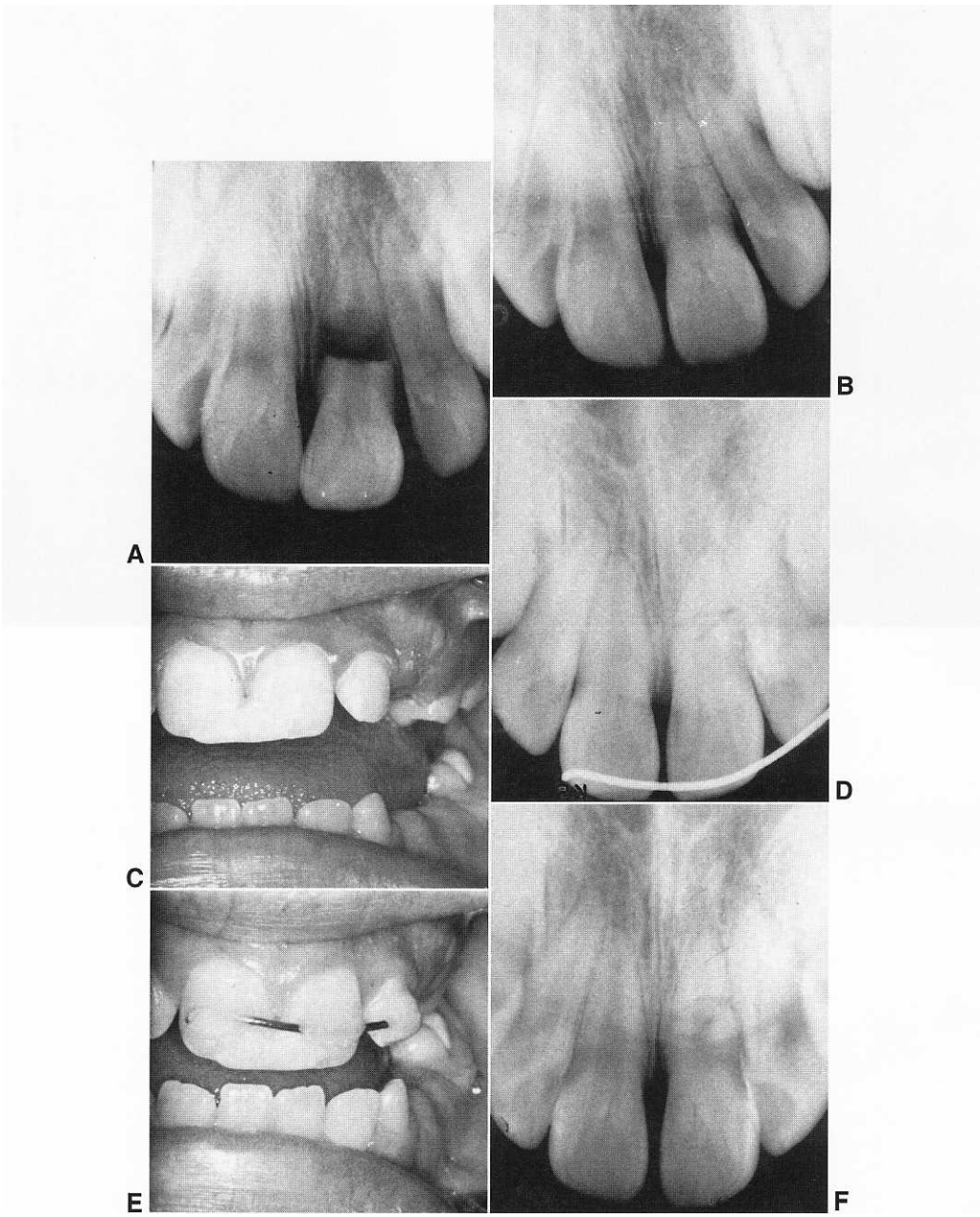


[I G **21-47. A,** Root fracture is evident in the apical half of the central incisor, but parent did not seek treatment until 2 weeks after the injury. The tooth was stabilized with a splint for 1 month. **B,** The tooth responded favorably to pulp testing and had this radiographic appearance 1 month later. C, More than 2 years had elapsed when this radiograph revealed a normal periapical appearance. The tooth responded normally to vitality tests, and there was slight mobility but no sensitivity to percussion.

The occlusion should be adjusted so that the injured tooth is not further traumatized during normal masticatory function. Follow-up radiographs should be made and pulp tests performed at frequent intervals during the first 6 months after the injury.

OTHER DISPLACEMENT INJURIES OF TEETH REQUIRING STABILIZATION

Teeth subjected to less severe luxation injuries may also benefit from stabilization with a bonded resin and wire splint during the recovery period. The severity of



f **I G 21-48. A,** Root fracture in middle third of root with a 5-mm separation of the parts. B, Approximately 1 hour after the injury the coronal portion of the tooth had been repositioned under local anesthesia with finger pressure. The mesial-incisal-labial areas of both central incisors were etched and self-curing resin was applied to hold the tooth in position while this radiograph was made to confirm proper reapproximation of the fractured root surfaces. C, Appearance of stabilized left central incisor at the time radiograph was made to confirm good reapproximation. After satisfactory repositioning was confirmed, the tooth was further stabilized using the bonded resin and wire technique. This injury did not result in significant soft tissue trauma, and hemorrhage was controlled easily. D and **E,** Radiograph and photograph reveal satisfactory progress 2 weeks after the injury. **F,** Nine months after the injury the tooth was vital, sound in the alveolus, and asymptomatic.

the injury will help determine the length of time the splint should remain in place. Splinting times may vary from 1 to 2 weeks for teeth that have been discernibly loosened (subluxation) to 4 to 6 weeks for teeth that have been laterally displaced, fracturing the alveolar process. As with all tooth injuries, frequent periodic evaluation is required for at least the first 6 months to afford the dentist the opportunity for early intervention if adverse sequelae develop; after this, evaluation at regular recall appointments should continue.

Nearly all significantly displaced teeth with closed apices and many with open apices will require follow-up endodontic therapy. As with many of the other injuries already discussed, calcium hydroxide paste is the currently recommended canal-filling material initially, and the canal should be recleaned and refilled with calcium hydroxide periodically if signs or symptoms warrant retreatment. This current recommendation of using calcium hydroxide may change in the relatively near future. Placement of a permanent gutta-percha filling should be delayed for at least 1 year (arbitrarily determined), and the calcium hydroxide should be replaced at least once (again arbitrarily determined) during this time. If the injured tooth had an open apex when endodontic therapy was initiated, the calcium hydroxide filling material should be used until the apexification process is complete or at least 1 year has elapsed, whichever is longer.

MANAGEMENT OF ORAL BURNS

As a result of secondary wound healing and scar contracture, burns involving the perioral and intraoral tissues can cause varying degrees of microstomia. A common cause of oral burns is electrical trauma. The most frequently encountered electrical injury to children is a burn about the mouth. These burns occur most often in children between 6 months and 3 years of age and are equally common among boys and girls.

Oral electrical burns occur when (1) the child places the female end of a "live" extension cord into the mouth, (2) the child places the female end of a "live" appliance cord (such as that for a hot plate, shaver, or portable radio) into the mouth, or (3) the child sucks or chews on exposed or poorly insulated electrical wires.

How does the burn occur? One plausible theory is that an electric arc is produced between a source of the current, such as the female end of an extension cord, and oral tissues. The electrolyte-rich saliva provides a short circuit between the cord terminals and the mouth, which results in the arc phenomenon. This type of burn characteristically involves intense heat that causes coagulation tissue necrosis.

NATURE OF THE INJURY

The clinical appearance of electrical burns is variable and depends on several factors: (1) the degree and duration of contact, (2) the source and magnitude of electric current, (3) the state of grounding, and (4) the relative degree of resistance at the point of contact. The wound may be superficial, involving only the vermilion border of one or both lips, or it may be a very destructive, full-thickness, third-degree burn. The more serious burns generally involve portions of the upper and lower lips and the commissure. Damage with more serious burns may extend intraorally to the tongue, the



[I G **21-49.** Appearance of injury to the upper lip and corn-missure 5 days after electrical burn. *(Courtesy Dr. Theodore R. Lynch.)*

labial vestibule, the floor of the mouth, or the buccal mucosa. There have also been reports of damage to hard tissue, such as the mandible and the primary and permanent teeth.

With third-degree burns, subcutaneous tissues may be damaged. The tissue destruction may be much more extensive than is initially evident. Because nerves are frequently damaged, the patient will probably have paresthesia or anesthesia. Therefore pain is generally not a significant problem. Hemorrhage is usually inconsequential because blood vessels are cauterized when the injury is sustained. However, spontaneous arterial bleeding may occur anytime during the first 3 weeks of healing. The hemorrhage may be caused by the rupture of blood vessel walls weakened by the passage of current. Bleeding can also occur with sloughing of necrotic tissue that overlies regenerating granulation tissue.

The clinical appearance of an electrical burn involving the lips and commissures is characteristic of a wound caused by intense, localized heat, perhaps as high as 3000° C. Necrosis, in which there has been heat-induced coagulation of protein, liquefaction of fats, and vaporization of tissue fluids, is evident.

During the first few days after the accident, the center of the lesion is generally composed of grayish or yellowish tissue that may be depressed relative to a slightly elevated, narrow, erythematous margin of tissue that surrounds it (Fig. 21-49).

Within a few hours after the injury there may be a great increase in edema. The margins of the wound may become ill defined and the lips protuberant. The patient may drool uncontrollably because of the loss of sensation. In 7 to 10 days the edema begins to subside. The delineation between the central nonviable tissue

and the surrounding viable tissue becomes more apparent. The necrotic tissue, known as *eschar,* becomes charred or crusty in appearance and begins to separate from the surrounding viable tissue (Fig. 21-50). The eschar sloughs off 1 to 3 weeks after the burn incident. Healing occurs by secondary intention as granulation tissue proliferates and matures. Two or 3 months after the accident the wound becomes indurated as a result of fibrous tissue formation. For an additional 6 months the immature scar tissue may bind the lips, alveolar ridges, and other involved structures. If it is not treated, contraction of the fibrotic scar tissue results in unesthetic and functionally debilitating microstomia. The scar tissue softens as it matures, and by 9 months to 1 year after the injury the potential for tissue contraction is greatly decreased. The duration of the healing process and the selected course of treatment depend on the extent and severity of tissue destruction. Because of the variable nature of burn injuries, surgery or appliance therapy may be used or no treatment may be needed.

TREATMENT

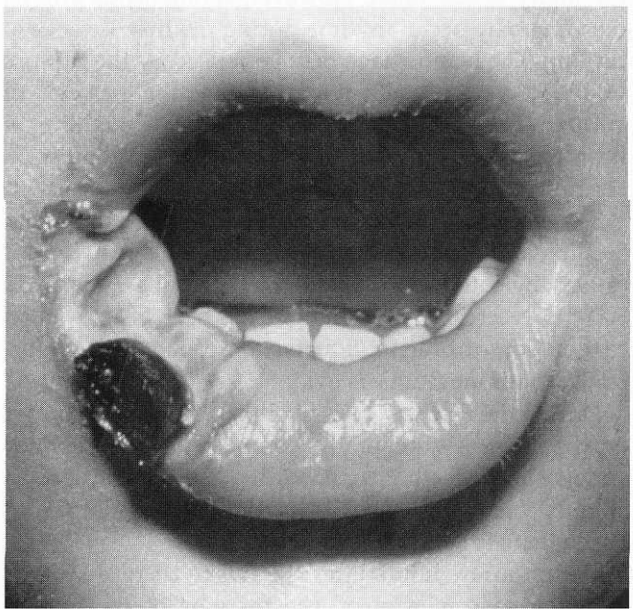
Assessing the general physical status of a patient who has sustained an electrical burn to the mouth is the first priority. Subsequently, the extent of the burn is carefully evaluated and local measures are initiated, such as control of minor hemorrhage or conservative debridement of nonviable tissue.

The immunization status of the patient must be ascertained, and tetanus toxoid or depot triple antigen (diphtheria-pertussis-tetanus vaccine) administered when appropriate. Many physicians prescribe a broad-spectrum antibiotic as prophylaxis. However, it may not be necessary or prudent to prescribe antibiotics in the absence of infection.

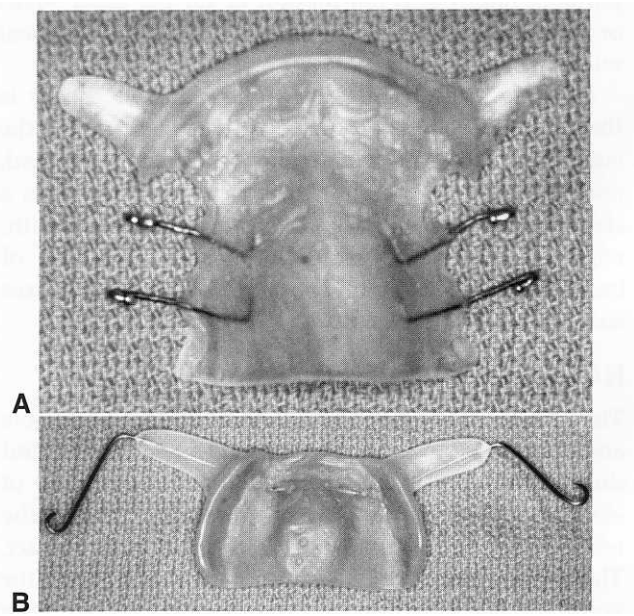
The parents should be informed of the possibility of spontaneous arterial hemorrhage during the first 3 weeks. They should be instructed to place firm pressure, with gauze, to the bleeding area for 10 minutes. If bleeding persists, they should take the child to the emergency department. Usually, hemorrhage is not a significant problem and does not warrant prophylactic hospitalization except for the most severe and extensive injuries.

The surgical management of burn injuries to the mouth, especially with regard to the time when such surgery should be performed, is controversial. Initially, no surgical intervention is generally warranted. Instead, the treatment of choice is the use of a prosthetic appliance. The primary functions of such an appliance are to prevent contracture of healing tissue and to serve as a framework on which a more normal-appearing commissure may be created and preserved after completion of the healing process. Many patients at James Whitcomb Riley Hospital for Children have been successfully treated with these appliances. Moreover, surgical procedures have not been needed in cases with good patient compliance in appliance use.

The major components of the burn appliance are illustrated in Fig. 21-51. The appliance is removed when the patient eats, when the teeth and appliance are cleaned, or when modifications of the wings are



El E. **21-50.** Appearance of injury to the lower lip and commissure 10 days after electrical burn. The dark lesion on the lower lip is an eschar. *(Courtesy Dr. Theodore R. Lynch.)*



H I. **21-51. A,** Example of a burn appliance designed for intraoral retention. **B,** Example of a burn appliance designed for intraoral and extraoral retention in combination with headgear. *(A courtesy Dr. David K. Hennon;* **B** *courtesy Dr. John D. Hiester.)*

necessary. The appliance is a static base with wings extending laterally to provide contact with both commissures. If symmetry relative to the midline is to be maintained during the healing process, the wings must contact the commissures equidistant from the midline and must exert essentially equal pressure at these points.

The shape and location of the wings are important, not only in preventing contracture or cohesion of the lips during healing but also in shaping the affected commissure to duplicate the unaffected side. The wing is contoured so that it is thickest in its occlusocervical dimension on the labial aspect. It is tapered as thinly as possible at the point of contact with the commissure (Fig. 21-52). The wings should be large enough just to maintain the correct shape of the commissure. Wings of the proper size will look more pleasing during wear and will enhance acceptance and compliance by the child and parent.

If compliance is a problem with an acrylic appliance, a modified fixed appliance can be constructed and ligated in the mouth. Bands are adapted on the upper second primary molars, and an impression is made. Headgear tubes are welded on the bands, and a Nance appliance is constructed from 0.036- or 0.040-inch wire. If the Nance button is not used, the wire is contoured along the gingival portion of the teeth (Fig. 21-53, A). For even more rigidity, an additional wire can be contoured and soldered transpalatally from molar to molar.

After the stabilizing framework is made, the outer portion of the appliance is formed. Using 0.045-inch wire, the anterior arch form is established. Horizontal loops are placed in the area approximating the location of the commissures, as determined by clinical measurements. The wire is continued posteriorly, and omega loops are placed mesial to the headgear tubes. Fig. 21-53, B, illustrates the completed outer portion of the appliance. Adjustments are made in the acrylic in one or both of the omega loops to achieve the correct fit. With the omega loops used as tiebacks, the appliance is ligated in the mouth. The patient should be evaluated as often as needed during the first month to make any necessary adjustments. Then the patient should be seen at least once a month.

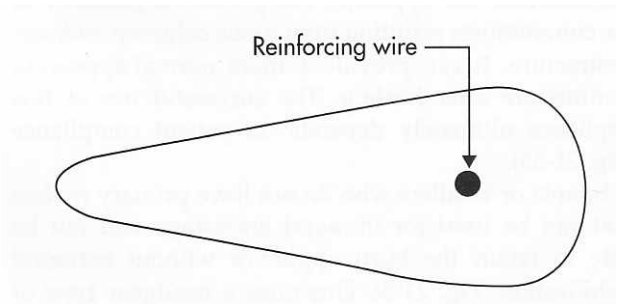


FIG **21-52.** Cross-sectional view of the commissure wing of the burn appliance.

Ideally the patient with an electrical burn to the mouth should be seen by the dentist between the fifth and tenth days after the accident. The initial appointment is probably the most crucial to the success or failure of treatment with the burn appliance. Parental apprehension and feelings of guilt are often high, and the trust and confidence of both the parents and the child must be acquired as soon as possible. They should be told in detail what they can expect from the dental services offered and what is expected of them. The parents and the child should be shown pictures from previous cases. This not only demonstrates the appearance and purpose of the appliance but also emphasizes that they are not the only ones who have experienced the physical and psychologic trauma associated with such an accident. Pictures of patients who did not have an appliance or who did not wear their appliance as instructed are also shown. The impact of such illustrative materials is dramatic.

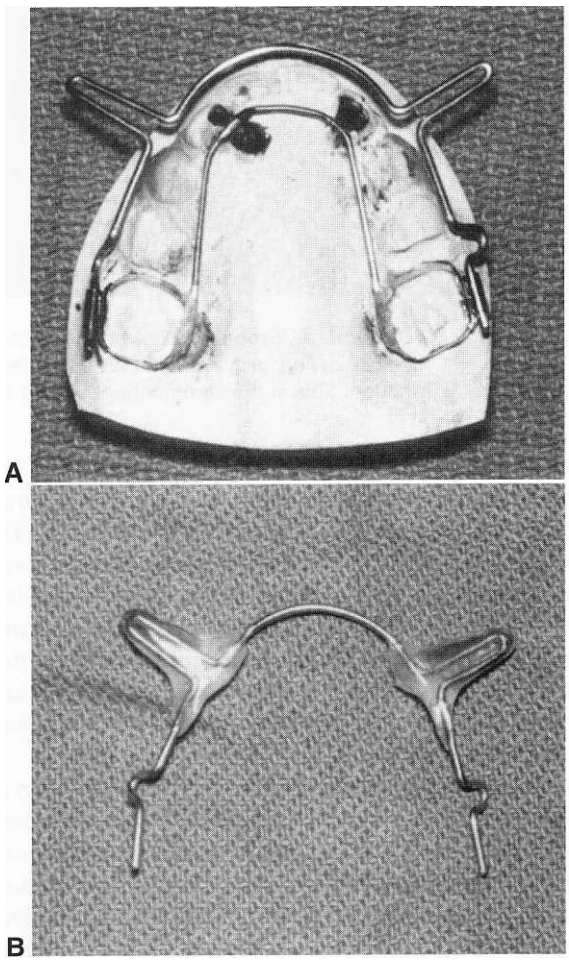
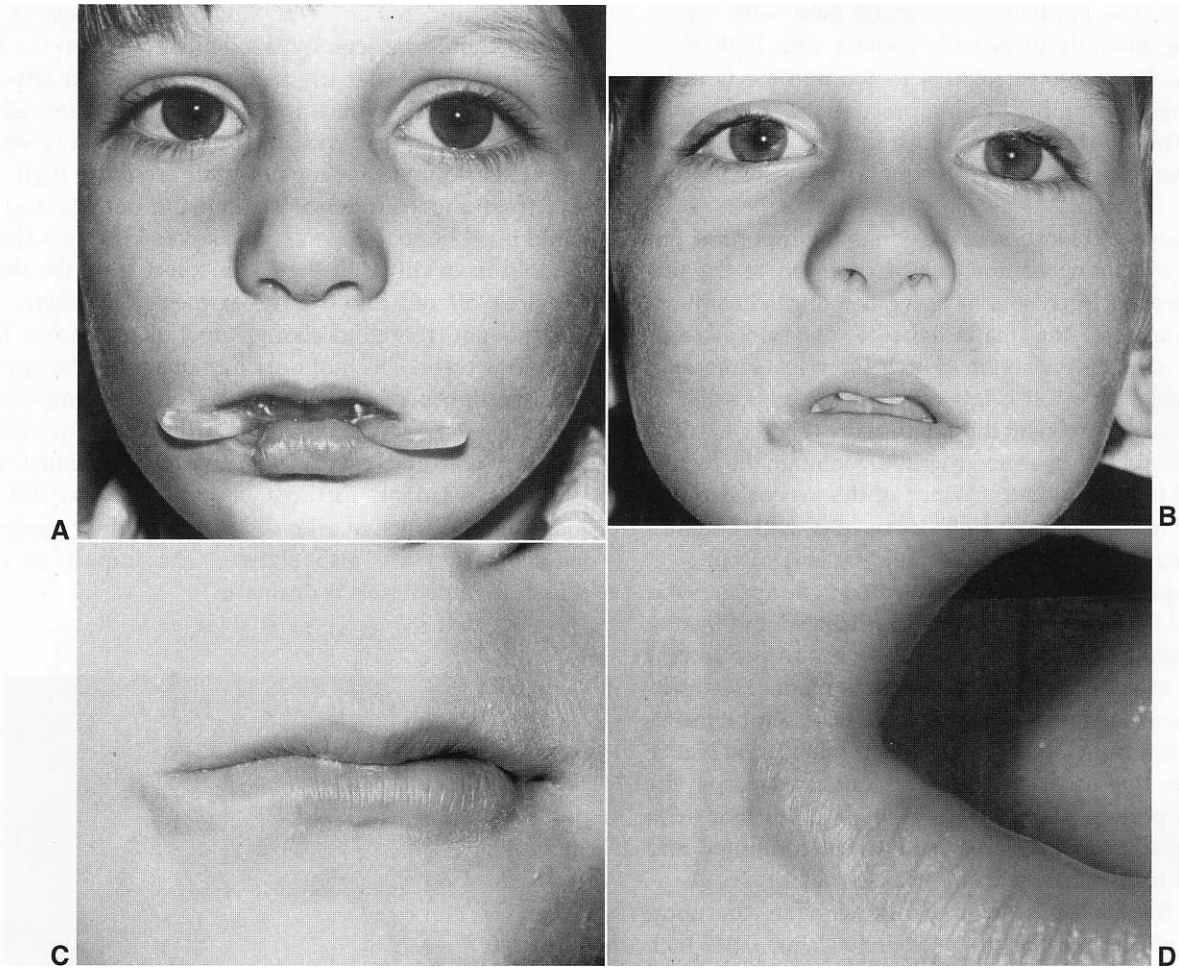


FIG **21-53. A,** Metal components of a modified fixed burn appliance on the working model. B, Finished outer portion of the appliance ready to insert and ligate to the cemented lingual arch. *(Courtesy Dr. Stephanie M. Litz.)*



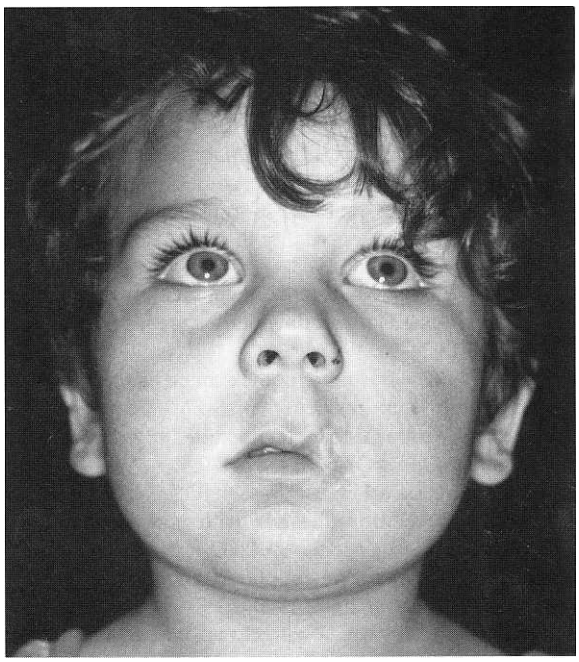
f **I G 21-54. A,** Properly fitted burn appliance in the mouth. **B,** Nine-month result. C, One-year result showing properly shaped commissure and symmetry of the lips. D, Patient has good function. This is the same patient shown in Fig. 21-50. *(Courtesy Dr. Theodore R. Lynch)*

After the consultation session, the initial data are recorded, photographs are taken, and alginate impressions are obtained for fabrication of the appliance. The appliance is generally delivered between the tenth and fourteenth days after the injury. At the delivery appointment and at each subsequent appointment, the information and instructions given in the initial consultation session are reinforced. Constant encouragement and positive reinforcement are important psychologic aids in enhancing compliance.

After delivery of the appliance the patient is seen as often as needed during the first week, but at least once during that time. The patient is checked again 4 and 8 weeks after appliance delivery. During this period, most of the major modifications to the wings and other components of the appliance are made, and the patient's compliance is closely monitored. Once the appliance is properly modified and the patient is wearing the appliance as instructed, the appointments can be spaced out over 4- to 6-week intervals. The appliance should be worn 24 hours a day for 9 to 12 months except for eating and cleaning (Fig. 21-54).

The burn appliance may not eliminate the need for minor surgical revisions of the lips or surrounding cutaneous tissues. Its purpose is to obviate the necessity for more difficult surgical procedures, because good results are hard to achieve and maintain when one is surgically restoring the shape and location of the oral commissure. The appliance can prevent asymmetry of the commissures resulting from tissue cohesion and scar contracture. It can provide a more normal-appearing commissure after healing. The successful use of this appliance ultimately depends on patient compliance (Fig. 21-55).

Infants or toddlers who do not have primary molars that can be used for intraoral anchorage will not be able to retain the burn appliance without extraoral stabilization. Fig. 21-56 illustrates a headgear type of extraoral anchorage apparatus. It is made of durable cloth, such as denim lined with gingham, and provides



f **I I 21-55.** Burn patient who did not wear the appliance as recommended, shown 10 months after the injury. Surgical correction will be considerably more difficult in this patient. *(Courtesy Drs. Roger Wood, Richard Quinn, and Joe Forgey.)*

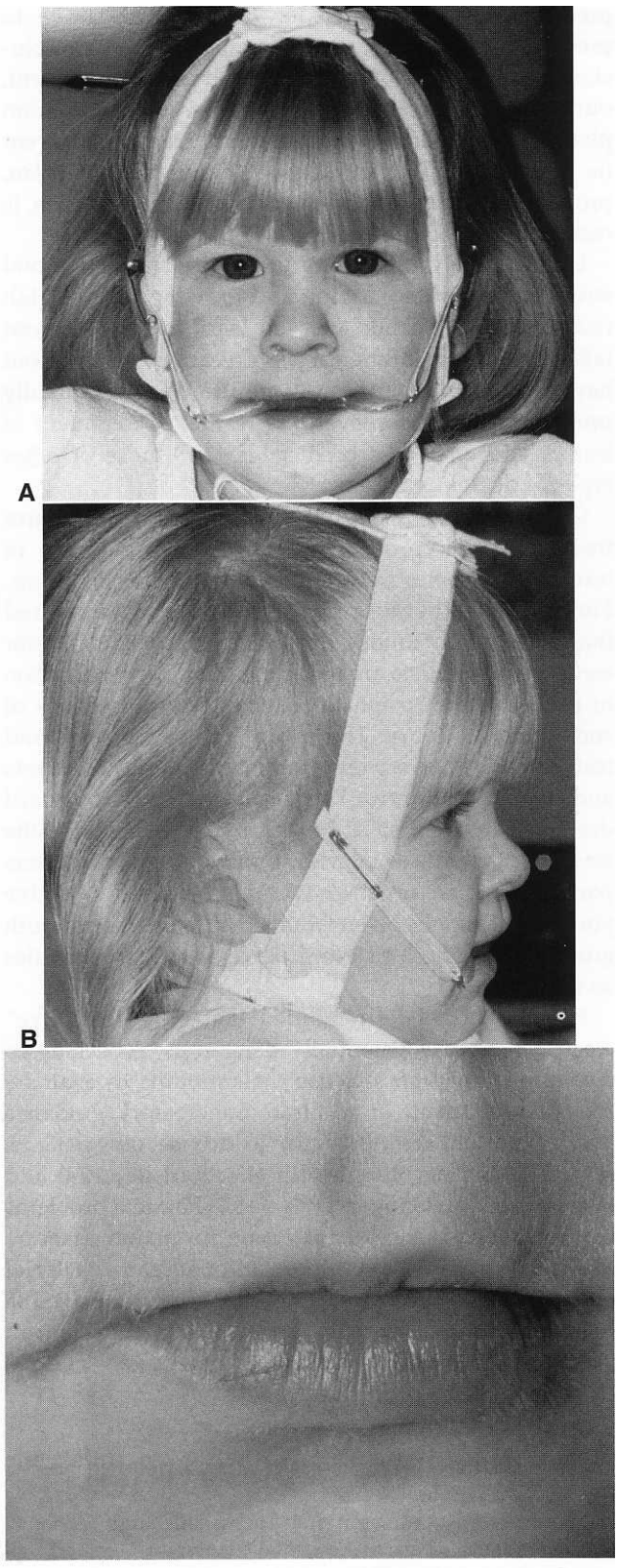
a static base from which elastic material extends to the wings of the burn appliance. A well-fitted bonnet may also be used.

Patients with burns to the mouth who did not have access to appliance therapy or were noncompliant in wearing the burn appliance may have tissue cohesion, contraction, and deformation as a result of healing. Such patients may require a commissurotomy to reestablish the original dimensions and symmetry of the mouth. Unfortunately, with healing of the commissurotomy there is again a tendency for wound contraction and distortion. Therefore it may be necessary for the patient to undergo more than one such surgical procedure unless surgery is followed by appliance therapy.

Clinical management of the burn appliance after a commissurotomy differs from management of the appliance after a burn injury. First, the appliance must be delivered by the time the sutures are removed. By the second week after surgery there may already be a decrease in the lateral extension of the primary incision as a result of wound healing. Second, the total time the patient needs to wear the appliance may be less than 1 year, depending on the clinical course of the healing process. The patient still wears the appliance 24 hours a day except for eating and cleaning.

TRAUMA PREVENTION

Dental practitioners should be proud that we are a prevention-oriented profession. Prevention is especially



[**1 6 21-56. A** and **B,** Properly fitted burn appliance with headgear to enhance stability and retention of the appliance. C, Nine-month result with appliance therapy. This is the same patient shown in Fig. 21-49. *(Courtesy Dr. Theodore R. Lynch.)*

predominant in dentistry for children. We strive to prevent dental caries, periodontal disease, malocclusion, and anxiety about dental care. If disease is present, our treatment becomes part of an overall prevention plan designed to halt the progress of disease and prevent its recurrence. The success of the prevention plan, provided that there is parent and patient cooperation, is reasonably predictable.

Unfortunately, our ability to prevent injuries to oral structures is limited. Living and growing carry a high risk of trauma. A child will not learn to walk without falling, and few children reach 4 years of age without having received a blow to the mouth. We cannot totally prevent trauma. Moreover, the results of treatment of trauma are often less predictable than those of other types of dental treatment.

On the brighter side, there are preventive measures that have been proved to reduce the prevalence of traumatic episodes in certain environmental situations. For example, because the prevalence of fractured incisors is higher among those with protrusive anterior teeth, many dentists are recommending early reduction of excessive protrusion to reduce the susceptibility of such teeth to injury. The use of car safety seats and restraining belts has prevented many injuries to infants and young children. The protective mouth guard described in Chapter 22 has prevented or reduced the severity of countless injuries to the teeth of youngsters participating in organized athletic activities; active youngsters should be encouraged to wear their mouth guards during high-risk unsupervised athletic activities as well.

Parents should be reminded that accessible "live" electric cords are potentially dangerous, especially to small children who still use their mouths to evaluate their environment. In addition, anticipatory guidance and education are necessary to advise our patients regarding the negative health effects of intraoral and perioral body piercing (see Fig. 7-48). Possible problems that can occur include scar tissue formation, fracture of the dentition, allergic reactions, and bacterial endocarditis in susceptible patients. When we have the opportunity to save a child from pain and suffering, an ounce of prevention is worth a pound of cure.

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CHAPTER

22

Prosthodontic Treatment of the Adolescent Patient

CHARLES J. GOODACRE
ROBERT J. CRONIN, JR.
DAVID T. BROWN

انتشارات
شایان نمودار

CHAPTER OUTLINE

RESTORATION OF SINGLE MALFORMED, DISCOLORED, OR FRACTURED TEETH All-Ceramic and Metal-Ceramic Crowns Teeth with Pulpal Involvement
FIXED PARTIAL DENTURES
Resin-Bonded Retainers
Complete Crown Retainers
Fixed Partial Denture Pontics
REMOVABLE PARTIAL DENTURES
OVERDENTURES
IMPLANT PROSTHESES
Implant Usage Before Growth Completion
Implants for Orthodontic Anchorage RECARE PROGRAM
PROTECTIVE MOUTH GUARDS

Scientific advancements in the areas of preventive dentistry, access to and use of dental services, water fluoridation, topical application of fluorides, and new commercial preventive dentistry products have led to substantial reductions in dental disease in developed countries. However, Caplan and Weintraub determined that adolescents are still affected by caries, particularly those who are minorities, rural inhabitants, have minimal fluoride exposure, and are from less educated and less affluent families) Results of a recent health and nutrition survey reported by Vargas, Crall, and Schneider also supports a higher caries prevalence among lower-income children and minorities.' Assessments of periodontal health by Barmes and Leous[3] and by Bader et al' show a decrease in severity but indicate that some adolescents are still affected.

Dental trauma continues to be a significant problem among adolescents as supported by Gift and Bhat's assessment of orofacial injury[5]; Bader, Martin, and Shugars' estimates of the incidence and consequences of tooth fracture'; and Haug, Prather, and Indresano's epidemiologic study of facial fractures and concomitant injuries.[7] Pilo et al indicate that congenital anomalies continue to result in missing or malformed teeth.' In addition, bulimia, anorexia, and dietary habits have led to an increase in the erosion of tooth structure among teenagers, particularly in girls.

Some of the esthetic treatment needs resulting from these conditions can be managed with resin-bonding procedures and porcelain laminate veneers, and whenever possible these should be considered as the treatments of first choice. When these procedures have not been able to provide a satisfactory result or when teeth are missing, then prosthodontics such as single crowns, fixed partial dentures, implant prostheses, or removable prostheses are indicated.

Because adolescents are often affected psychologically by the unacceptable appearance of diseased, damaged, or missing teeth, one should not allow chronologic age to preclude performance of whatever treatment is necessary to provide proper function and esthetics. If the teeth involved are fully erupted, have achieved complete root formation, and may be prepared without causing irreversible damage to the pulp, successful prosthodontic treatment can often be provided for patients as young as 12 to 14 years of age. Patient cooperation, however, is mandatory during and after treatment. Adolescent patients must be able to tolerate long appointments and remain still for extended periods while teeth are being prepared and impression materials are setting. Also, they must be able to achieve and maintain good oral hygiene around both the provisional and definitive restorations, as well as in the rest of the mouth. All these conditions make it highly desirable to perform the necessary treatment as expeditiously as possible. Finally, it must be understood that an adolescent is more likely to sustain trauma to the oral structures than is an adult and thus there is greater risk of damage to restorations and prostheses than in an adult patient.

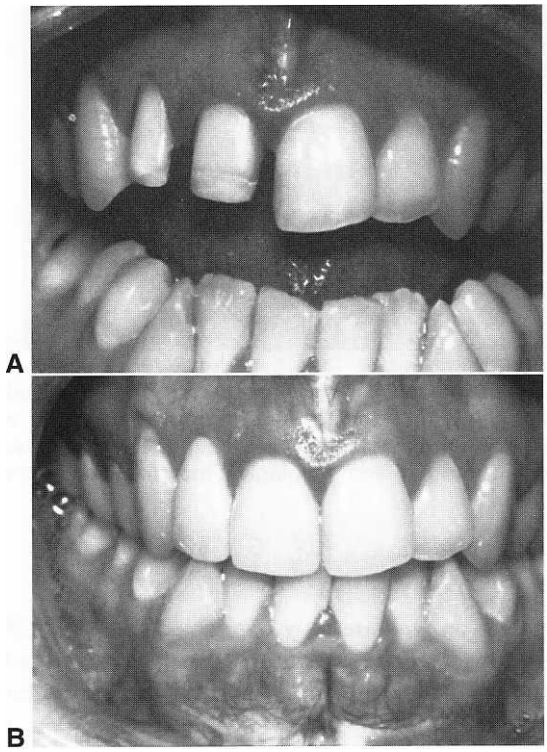
Prosthodontic treatment of the adolescent patient often requires highly intricate procedures that go beyond the scope of this chapter. The goal of this chapter is to offer the reader an opportunity to develop a better appreciation of the achievable and available solutions for the young patient with a prosthodontic need. The interested reader should consult the prosthodontic literature and current textbooks on fixed, removable, and implant prosthodontics for more detailed information in this area of dentistry.

RESTORATION OF SINGLE MALFORMED, DISCOLORED, OR FRACTURED TEETH

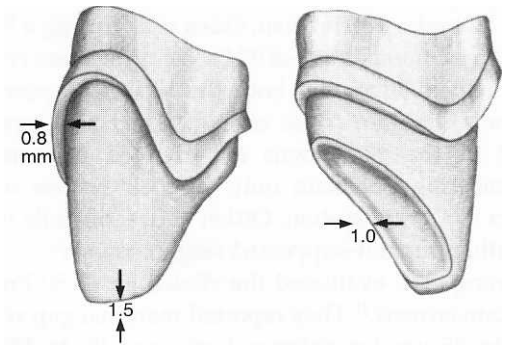
ALL-CERAMIC AND METAL-CERAMIC CROWNS

All-ceramic crowns are the most esthetic complete-coverage restorations currently available in dentistry. The achievement of optimal longevity with all-ceramic crowns requires normal tooth preparation form because the prepared tooth must provide support for the restoration. Therefore, if a large portion of tooth structure is missing because of trauma or caries, or if previous restorations become dislodged during tooth reduction, then a separate restoration that is well retained in remaining tooth structure should be placed to establish an ideal preparation form (Fig. 22-1). Also, the fracture resistance of all-ceramic crowns is enhanced when other characteristics are present. Occlusal forces should be average or below-average. The centric occlusal contacts should ideally be located over the concave lingual portion of the prepared tooth and not cervical to the cingulum where fracture of the crown is more likely to occur. The prepared tooth should possess average or greater incisocervical length and should not be short, round, or overtapered in form.

The tooth preparation for an all-ceramic crown (Fig. 22-2) should possess a well-defined, smooth finish line that is 0.8 mm deep around the entire tooth, with the axial surfaces reduced to a depth of 0.8 mm. The lingual reduction for occlusal clearance should be 1.0 mm. An incisal edge reduction of 1.5 to 2.0 mm is required and is biologically acceptable even in the presence of large pulps. The use of resin cement and associated dentin bonding is recommended because crown strength is significantly improved. Both chamfer and shoulder finish lines can be used in conjunction with resin cement without compromising restoration



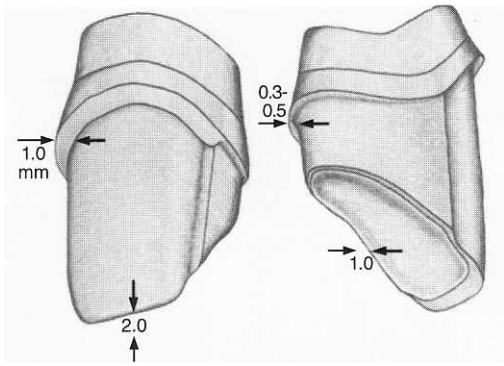
F I G 22-1. **A,** Traumatically injured central and lateral incisors that have been restored with bonded resins and then prepared to receive all-ceramic crowns. **B,** All-ceramic crowns have been cemented.



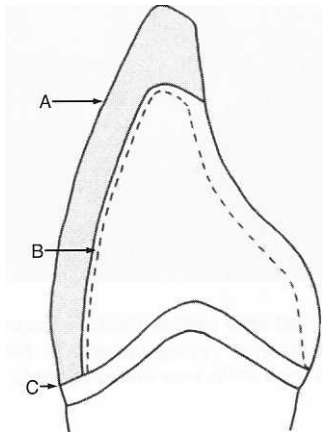
f G. **22-2.** Two views of all-ceramic (porcelain jacket crown) preparation showing recommended reduction depths and shoulder finish line.

strength. If zinc phosphate or glass ionomer cement is used, however, then a shoulder finish line should be used to optimize crown strength.

When the ideal tooth preparation form is seriously compromised or the magnitude of occlusal forces contraindicates restoration with an all-ceramic crown, use of the stronger metal-ceramic crown is indicated. The tooth preparation design and reduction depths for a metal-ceramic crown are shown in Fig. 22-3. When



f I G 22-3. Two views of metal-ceramic crown preparation showing minimal facial reduction and shoulder finish line, minimal incisal reduction, lingual axial reduction depth and chamfer finish line, and lingual reduction for occlusal clearance.

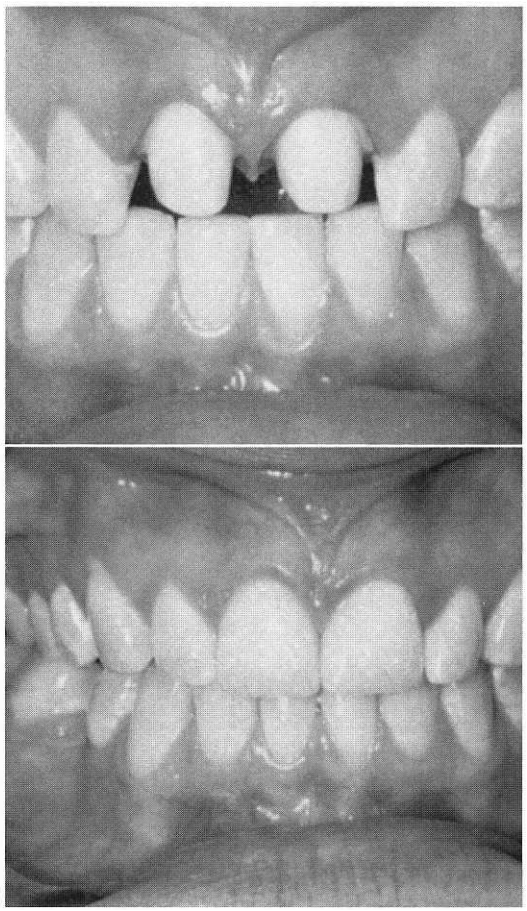


f **I G 22-4.** Collarless metal-ceramic framework design that eliminates visible cervical metal. A indicates porcelain; B indicates underlying metal framework that does not cover shoulder finish line; Cis margin where porcelain contacts only prepared tooth.

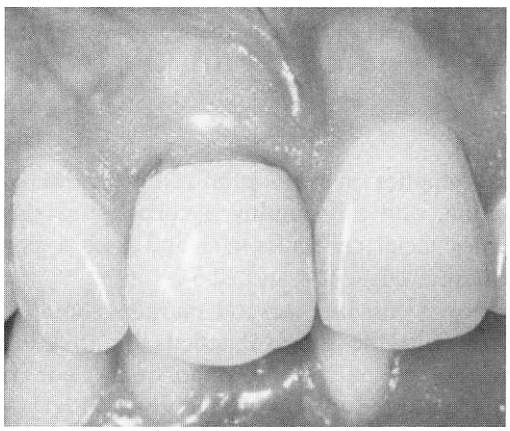
cervical esthetics must be optimized in a metal-ceramic restoration, one can employ a collarless design that eliminates facial cervical metal and uses a porcelain facial margin (Figs. 22-4 and 22-5).

Whenever possible, cervical margins should not be extended into the gingival sulcus of an adolescent patient. If oral hygiene is inadequate, subgingival margins may produce accelerated gingival recession or interfere with the normal cervical relocation of the gingival tissues as the patient matures. Both occurrences produce an esthetic liability (Figs. 22-6 and 22-7).

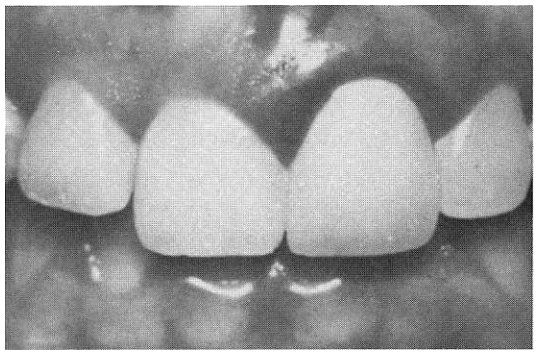
In addition to the conventional all-ceramic crowns, several computer-aided design and computer-aided manufacturing (CAD-CAM) systems for fabricating



F **I G 22-5. A,** Injured vital central incisors prepared for metal-ceramic restoration in a young patient. **B,** Maxillary central incisors restored with collarless metal-ceramic crowns.



f **I** G . **22-6.** Gingival contour in 25-year-old patient resulting from placement of subgingival crown on maxillary right central incisor at 8 years of age. The gingival crest is not positioned as far apically on the restored central incisor, and its form is rounded and thick rather than the normal form of the gingival margin, which is thinner and sharper.



[I G **22-7.** Accelerated gingival recession around maxillary left central incisor resulting from metal-ceramic crown with subgingival margins placed at a young age. The gingiva is edematous and red, and the gingival margin is rounded and thick.

all-ceramic crowns are now available. The CAD-CAM systems are generally designed to help streamline the fabrication and placement of esthetic crowns. Some of these systems are intended to allow preparation and placement at the same appointment. The AllCeram and CEREC systems are among the popular CAD-CAM systems.

The Procera AllCeram system (Nobel Biocare AB, Goteburg, Sweden) uses CAD-CAM technology to fabricate a densely sintered aluminum oxide coping. The coping is then veneered with dental porcelain by a dental laboratory technician. Oden et al, during a 5-year clinical trial, noted that 3 of 97 Procera AllCeram crowns had fractures involving both the veneering porcelain and the aluminum oxide coping.[9] They also reported that 2 of the 97 crowns experienced fractures in the veneering porcelain only. In their review of the Procera AllCeram system, Ottl et al described its use in fabricating implant-supported single crowns)" Boening et al evaluated the clinical fit of 80 Procera AllCeram crowns." They reported marginal gap widths of 80 to 95 µm for anterior teeth, and 90 to 145 µm for posterior teeth. May et al found mean marginal openings of less than 63 µm in an in vitro study.[1z] Boening's study concluded that the accuracy of fit of the Procera AllCeram system was within the range of clinical acceptance."

The CEREC CAD-CAM system (Sirona, Bensheim, Germany) is designed to fabricate all-ceramic restorations (crown, inlays, and onlays) at the chairside. This one-appointment approach, without the need for a provisional restoration, is its principal potential advantage. The CEREC CAD-CAM system incorporates an optical scanning of the prepared tooth and adjacent teeth. The CAD-CAM instrumentation is then used to mill internal and external surfaces of the restoration.

Mou et al noted criticisms of the marginal fit and adaptation of these restorations. Their in vitro study found average internal gaps ranging from 100 to 200 μm.[75] They found little differences in internal fit when the tooth preparations had a convergence angle of 12 to 20 degrees and a total tooth preparation height of 6 mm or less, occlusal cervically.

Reiss and Walther, using the Kaplan-Meier method of analysis, reported the probability of survival of CEREC restorations to be 90% at 10 years.' Restorations of premolars were rated better than those of molars, and restorations of vital teeth better than those of nonvital teeth. The most frequent reasons for failure were ceramic and tooth fractures.

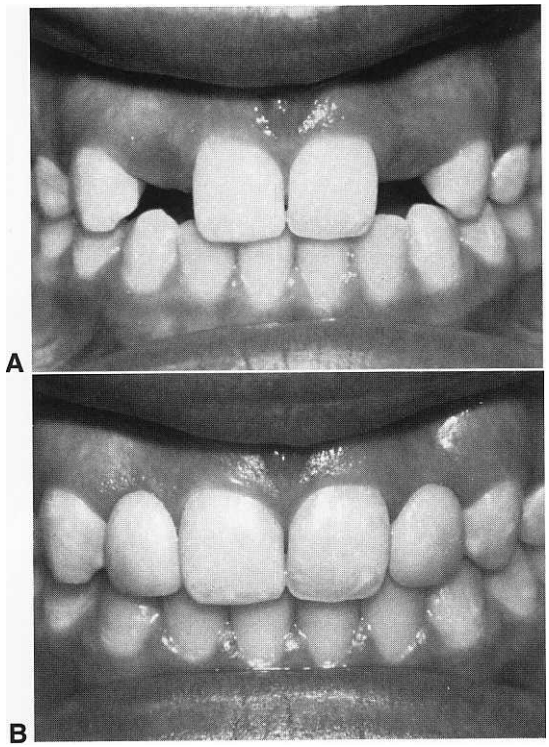
TEETH WITH PULPAL INVOLVEMENT

When tooth fracture or caries involves the pulp and root development is complete, a routine pulpectomy and gutta-percha root canal filling should be completed. Because posts and cores do not strengthen endodontically treated teeth, their use is indicated only when remaining coronal tooth structure does not provide adequate retention for the definitive restoration.[15] Restorations that do not employ a post should be used whenever possible to replace missing tooth structure and serve as a retentive foundation. It is particularly important that teeth in the mouths of accident-prone adolescents or those in whom athletic trauma has previously occurred be restored without using a post, if possible. This practice helps to avoid irreparable damage in the form of root fracture should the restored tooth once again be subjected to trauma. Even though trauma may result in restoration dislodgement or perhaps even fracture of the tooth, the tooth will have survived at least one more traumatic experience.

In the case of pulpal involvement when the root is incompletely formed, a pulpotomy followed by placement of an appropriate restoration is indicated. Subsequently, when root formation is completed, a pulpectomy is performed, followed by placement of the definitive restoration or crown, if needed.

FIXED PARTIAL DENTURES

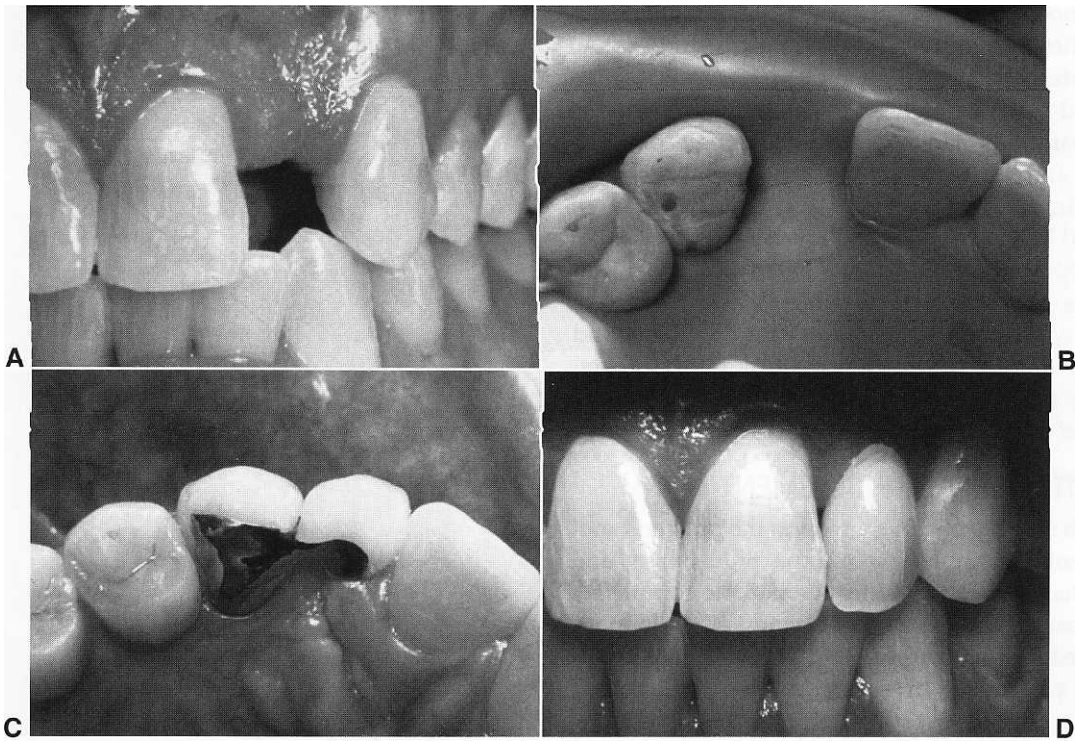
Although preventive procedures have reduced the number of teeth lost, fixed partial dentures continue to be needed in adolescent patients because of trauma or congenital lack of tooth formation, and occasionally for reasons such as extensive caries and periodontal disease. When a tooth is lost, space maintenance should be provided immediately after extraction to prevent tipping, tilting, or rotation of the abutment teeth or eruption of the opposing teeth. Space maintenance should be continued until the fixed prosthesis is completed. If the abutment teeth are malaligned and pulp size does not permit the amount of tooth reduction necessary to align the preparations, orthodontic repositioning of the abutment teeth should be initiated.



f **I G 22-8. A**, Congenitally missing maxillary lateral incisors. **B,** Resin-bonded prostheses were used instead of conventionally cemented prostheses to preserve as much tooth structure as possible on the central incisor and canine abutments.

RESIN-BONDED RETAINERS

For reasons of pulpal and periodontal health and conservation of tooth structure, resin-bonded retainers should be the first choice whenever possible (Fig. 22-8). Retention and resistance form is achieved through tooth preparations, terminating in enamel, coupled with acid etching of the enamel and fixation with resin cement. The conservative approach of using resin-bonded retainers does, however, require that the abutment teeth be intact or minimally restored, with substantial enamel present for bonding procedures. To produce an adequate area for resin bonding, the existing crown should be of average or greater length. A maximal amount of the nonvisible portions of the lingual and proximal surfaces should be covered by the retainers, because Pegoraro and Barrack have determined that bond strength increases with the area of enamel covered.'[b] The existing crown form, color, and axial alignment must be satisfactory, because this prosthesis



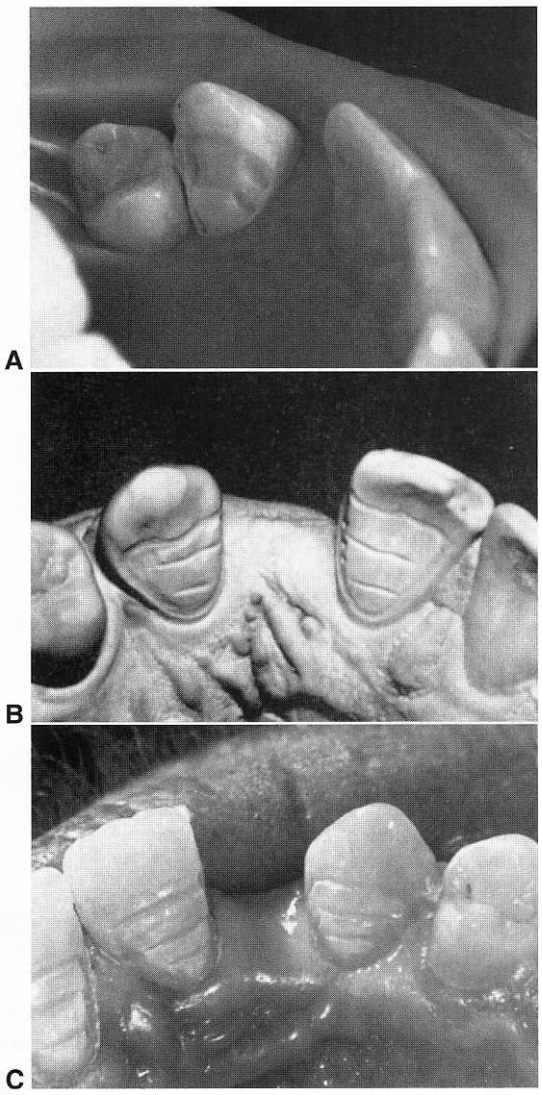
[l **G 22-9. A,** Congenitally missing maxillary lateral incisor. **B,** Mirror view of the canine has been prepared as a single abutment for a two-unit cantilevered resin-bonded prosthesis. Note the two mesial proximal grooves, the lingual groove, and the distal pinhole, which provide retention and resistance form along with acid etching and use of an adhesive resin cement. C, Mirror view of bonded two-unit cantilevered prosthesis. D, Facial view of completed prosthesis.

design does not permit the incorporation of changes in the facial enamel of abutment teeth. When abutment crown contours or color require esthetic changes, then complete-coverage retainers may produce a superior result.

These prostheses can be successful for many years, but suitable attention must be paid to four factors: appropriate diagnosis and treatment planning, correct tooth preparation, good-fitting castings, and meticulous adherence to the required resin-bonding procedures.

Several factors important to diagnosis and treatment planning have been identified in clinical studies. Because Dunne and Millar determined that prostheses with only one pontic have much higher success rates, the use of long-span prostheses should be avoided." The use of multiple splinted retainers was associated with higher failure rates according to Marinello et al.'s As with any prosthesis, the presence of heavy occlusal forces reduces longevity." Recently the use of resin-bonded cantilevered prostheses (using only one retainer) has been reported, and data of Hussey, Pagni, and Linden[20] and of Leempoel et al[27] indicate that this type of design can be used successfully in certain situations such as a missing maxillary lateral incisor (Fig. 22-9).

Barrack and Bretz determined that the successful use of resin-bonded prostheses requires establishment of retention and resistance form through tooth preparation (Figs. 22-9, B, and 22-10).2[22] Because the tooth preparation is limited principally to enamel, these retainers can be used without pulpal damage and the teeth can often be prepared without anesthesia. Besimo states that the proximal surfaces adjacent to the edentulous area should be reduced to remove interproximal undercuts and to provide parallel surfaces that aid retention.[23] One or two proximal grooves must be placed to enhance the resistance and retention form (Fig. 22-10, A). Proximal grooves have been identified as a key factor in the resistance to debonding in several studies.[222-26] The tooth preparation should include a small peripheral chamfer finish line (Fig. 22-10, B) formed with the tip of a rounded-end diamond instrument. The lingual surfaces of anterior teeth are reduced to create occlusal clearance with the opposing teeth. The minimal occlusal clearance space for short-span (three-unit) prostheses with normal occlusal forces



f I G 22-10. A, The maxillary canine has been isolated for prosthesis cementation using a rubber dam, which affords a good view of the well-defined proximal grooves. **B,** Cast of prepared maxillary central incisor and canine. Note the small peripheral chamfer, the lingual ledges, and the small proximal grooves. C, Another patient in whom the maxillary central incisors and canine have been prepared using multiple lingual ledges and multiple proximal grooves.

is 0.5 mm. It may not be necessary to reduce the abutment teeth lingually when there is existing occlusal clearance, whereas reduction of opposing teeth may be necessary when occlusal contact occurs over broad areas of the lingual surfaces. Multiple ledges, prepared across the reduced lingual surface (Fig. 22-10, C), increase the casting rigidity and, along with the proximal grooves, aid in retention and resistance form and in orientation of the casting during cementation.

The preparation of posterior abutment teeth should include reduction of the proximal surfaces to eliminate undercuts and to produce minimal occlusal convergence for retention and resistance form. The reduction should also include lingual surfaces and at least 180 degrees of circumferential reduction as determined by Creugers et a1[27] and by Crispin.' One or two proximal grooves are placed, one or two occlusal rest seats are prepared," and a small chamfer finish line is formed. The use of an inlaylike occlusal rest has been advocated to increase resistance and retention form. The occlusal aspect of the lingual cusp can also be slightly reduced and covered with metal to increase the bonding area and prosthesis retention (Fig. 22-11).

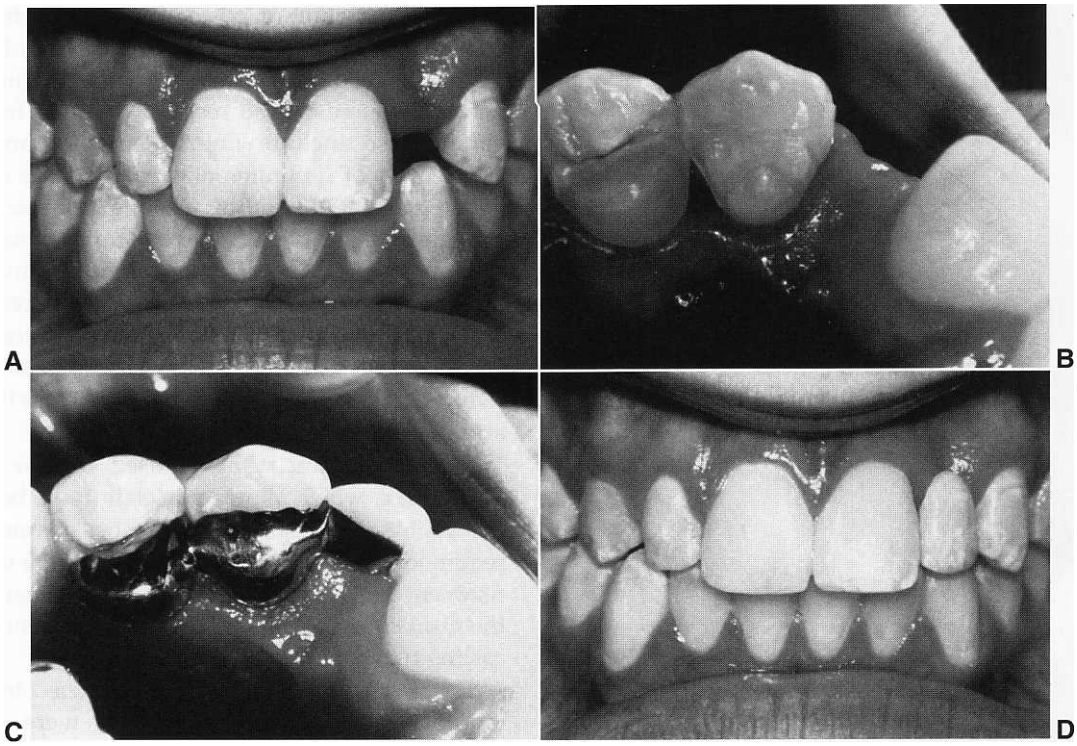
A metal-ceramic alloy is used for the prosthesis framework, which allows porcelain to be bonded over the visible facial surface of pontics to meet esthetic requirements. Several design variations are used for the retainer castings, differing in the manner in which the resin mechanically interlocks with the surface of the casting that contacts the prepared teeth.

The first prosthesis design, introduced by Rochette, employed retainer castings that were perforated lingually, which allowed resin to encompass the casting. `' Subsequently, Livaditis and Thompson introduced a technique that uses a base metal alloy and thereby takes advantage of the ability of the alloy to be acid-etched to provide microscopic areas of retention (Fig. 22-12).[30] This technique is not suitable for use with gold-containing alloys because they cannot be etched in this manner.

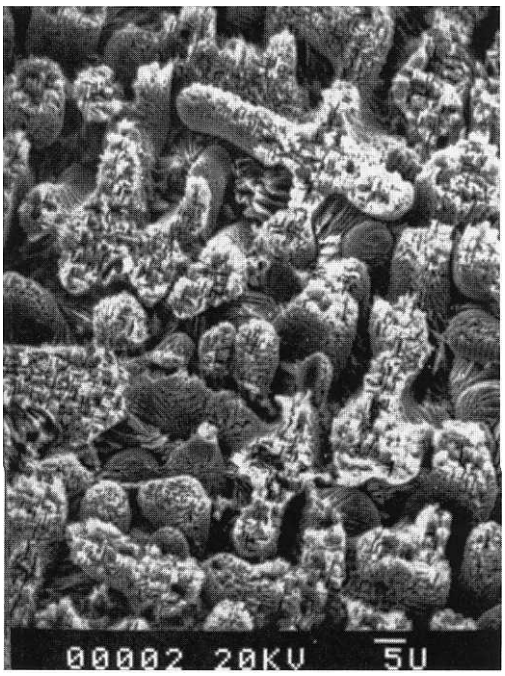
An alternate method of treating the metal surface to provide retention for the resin is to subject the bonding surface to airborne-particle abrasion using 50-m aluminum oxide. This method has been shown to provide retention comparable to that of chemically etched metal and can be used with any casting alloy.[17,'s,3'.32]

The completed prosthesis is first trial-seated so that any required adjustments of form, color, occlusion, and glaze can be completed. The prosthesis then should be cleaned ultrasonically to remove debris that may be present in the retentive areas and dried thoroughly with clean, dry, compressed air.

Cementation of the prosthesis with the acid-etching and resin procedure requires optimal moisture control, which is best obtained with use of a rubber dam. Composite resins specially designed for cementation of this type of prosthesis are employed. After isolation of the prepared teeth (Fig. 22-13, A), the enamel is acid-etched in the usual manner, rinsed, and dried. The resin is applied to the casting and the prosthesis seated (Fig. 22-13, B). Excess cement is quickly removed, and the prosthesis is held motionless until polymerization is complete. Resin cement left in interproximal undercut areas until hard can be very difficult to remove.



[**I C 22-11. A**, Congenitally missing maxillary lateral incisor. **B,** Mirror view, the canine and first premolar have been prepared for a resin-bonded prosthesis. The lingual cusp of the premolar has been reduced so the retainer can be bonded over the cusp. C, Mirror view of bonded prosthesis showing coverage of the premolar lingual cusp. D, Facial view of completed prosthesis.



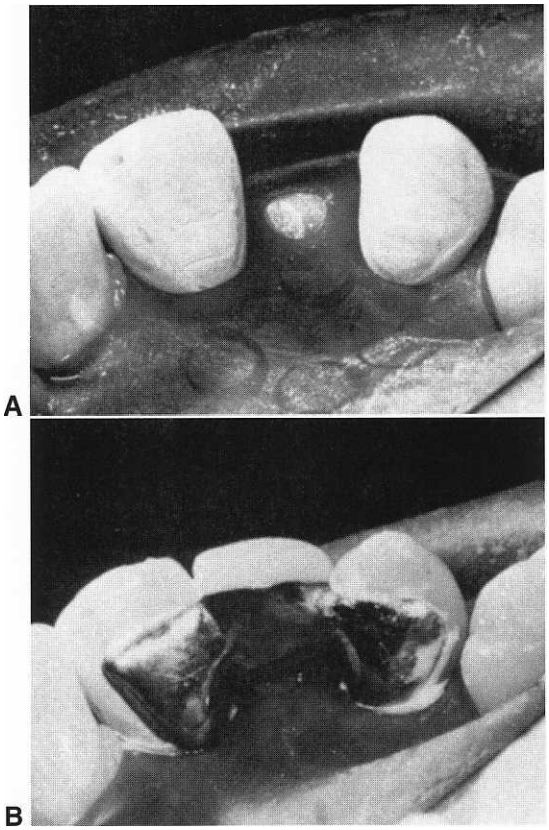
FSC. **22-12.** Scanning electron micrograph of etched base metal (x1000). *(Courtesy B.K. Moore.)*

Opaque resins can be used to help mask the metal when abutment teeth are very translucent. This procedure helps reduce the darkening effect of lingual metal's showing through the incisal aspect of a tooth. Also, when adequate bonding area is present lingually, it may be possible to reduce the area of retainer coverage by cutting away lingual metal before cementation so that none is located behind thin translucent areas of the tooth.

COMPLETE CROWN RETAINERS

When a resin-bonded retainer cannot be used because of the tooth condition, crown form, crown length, axial alignment, occlusal forces, or conditions requiring maximal retention and resistance form, use of complete-coverage retainers is indicated.

Cast metal retainers should be used posteriorly wherever esthetically possible, since they require less tooth reduction than a metal-ceramic retainer. When short clinical crowns are encountered, it may be necessary to use auxiliary grooves or boxes in the axial surfaces to achieve the required degree of retention and resistance form. It may also be necessary to



E **I** G **22-13. A,** Teeth isolated with rubber dam in preparation for resin-bonding procedures. **B,** Prosthesis bonded with resin.

perform a gingivoplasty so that more tooth structure is exposed.

When esthetic requirements demand and pulp size permits, metal-ceramic restorations are indicated. An example of ectodermal dysplasia in which crown form and esthetics dictated full-coverage metal-ceramic restorations is shown in Fig. 22-14.

When complete-coverage retainers are indicated but pulp size allows only minimal reduction, an all-resin fixed prosthesis may be the best choice. The all-resin restoration offers good esthetics initially and requires only minimal axial tooth reduction (0.5 mm), but because it has less wear resistance, strength, and color stability than porcelain, it must be replaced periodically. All-resin prostheses rarely last more than a few years without significant wear, color change, or fracture. If the casts and records are retained, however, additional replacement prostheses can be made and several additional years of service obtained. Subsequent treatment may permit the use of metal-ceramic retainers because enough pulpal recession may have occurred so that additional tooth reduction can be safely accomplished. The development of new resins and fiber reinforcement may extend the longevity of such prostheses.

Complete crown retainers can also be used in conjunction with cantilevered prostheses, so that tooth preparation is limited to one tooth. Schwartz et al" and Foster[34] have shown use of the cantilever design to be a viable treatment alternative. The absence of a maxillary lateral incisor, which is encountered in adolescents because of congenital abnormality, is a situation well suited to this design. A two-unit fixed prosthesis can be fabricated with a lateral incisor pontic cantilevered from the canine retainer (Fig. 22-15). The positional stability of the canine is important to the success of this design. The canine should not be mobile, and the arch form should be stable (i.e., teeth have not and are not shifting position). The canine should not have recently undergone orthodontic repositioning that involved significant rotation.

FIXED PARTIAL DENTURE PONTICS

Pontics are usually of an all-metal or metal-ceramic design, depending on esthetic requirements. The metal-ceramic pontic is a highly versatile replacement, combining the esthetic benefits of porcelain with the strength of metal, and is widely adaptable to edentulous spaces of varying sizes.

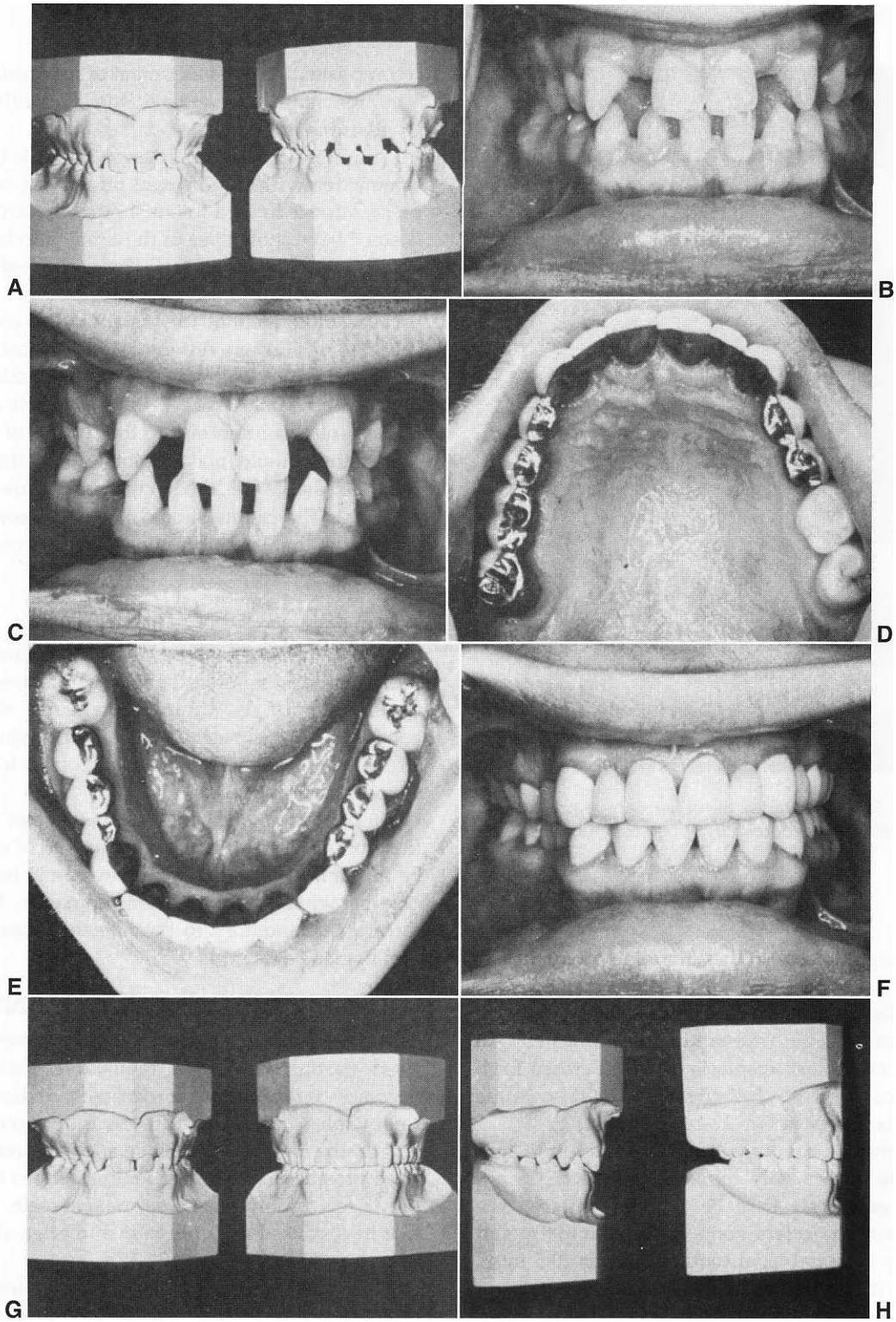
Pontic design must include meticulous attention to the amount of ridge coverage; the area of contact must be minimized and the embrasures must be as large as possible while meeting esthetic demands. These procedures are mandatory to provide soft tissue access for oral hygiene aids.

REMOVABLE PARTIAL DENTURES

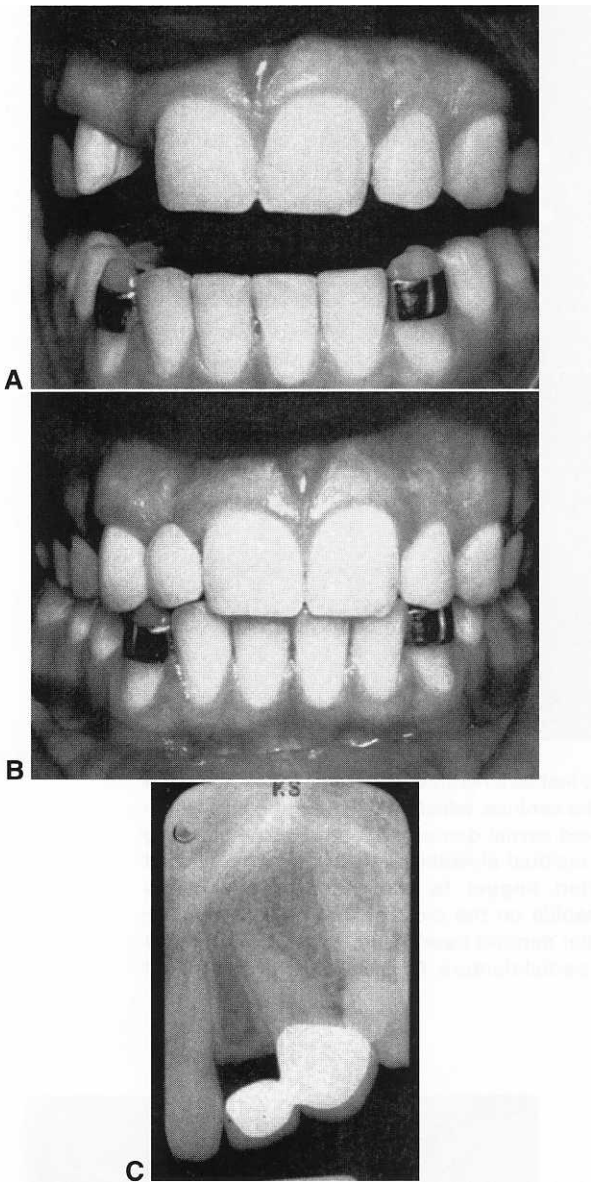
When the number of missing teeth prevents use of a fixed partial denture, a removable partial denture becomes a restoration of necessity. Indications for a removable partial denture include excessive span length, the inability to achieve adequate retention for a fixed prosthesis, congenital malformations that result in only a few widely spaced permanent teeth, and injuries that have caused multiple teeth and often alveolar bone to be lost (Figs. 22-16 and 22-17).

When treatment is planned for an adolescent patient who needs a removable partial denture, there are three major objectives: (1) the restoration of the functions of mastication and speech, (2) the restoration of dental and facial esthetics, and (3) the preservation of the remaining teeth and their supportive tissues.

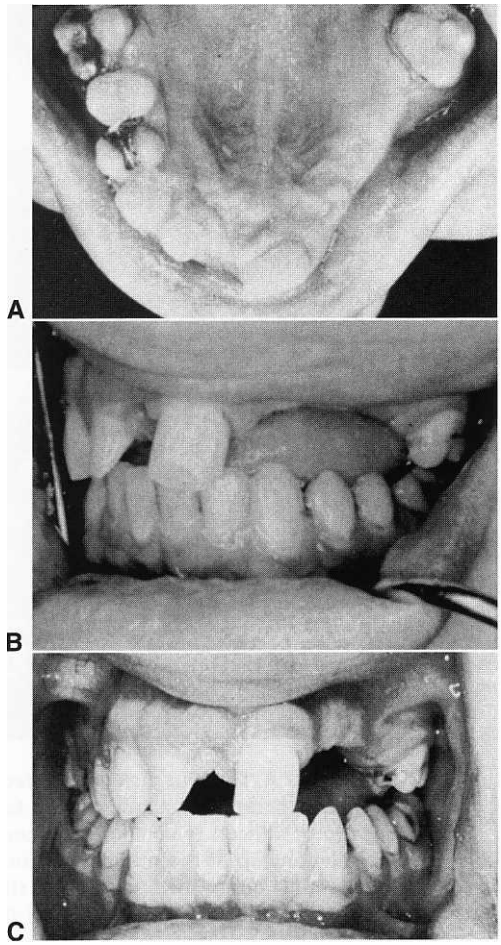
The function of mastication can be restored when correct, harmonious, and nondestructive occlusal relationships are provided between the supplied teeth and the opposing remaining natural dentition.



[I G . 22-14. **A,** Casts of patient exhibiting incomplete dental manifestation of hereditary ectodermal dysplasia. Left, Casts before orthodontic treatment. *Right,* Progress casts near completion of orthodontic treatment. **B,** Dentition of the patient shown in A after orthodontic treatment. Notice vertical overbite correction and space adjustment to facilitate prosthetic treatment. Additional illustration of the treatment may be seen in C through **H. C,** After periodontal treatment, gingivoplasty was performed to gain crown length and improve tissue contours. D, Mirror view of maxillary arch after prosthetic treatment. **E,** Mirror view of mandibular arch after prosthetic treatment. F, Anterior view in occlusion after prosthetic treatment. Metal-ceramic fixed partial dentures were employed to correct abnormal tooth form, replace congenitally missing teeth, and restore occlusion. **G** and **H,** Anterior and right lateral views of casts before and after treatment.



G I G 22-15. A, Maxillary canine has been prepared for metal-ceramic cantilevered prosthesis. Notice discrepancy in path of insertion with central incisor, which necessitated cantilever design. **B,** Facial view of cemented prosthesis. C, Postinsertion periapical radiograph showing malalignment of canine and central incisor roots.



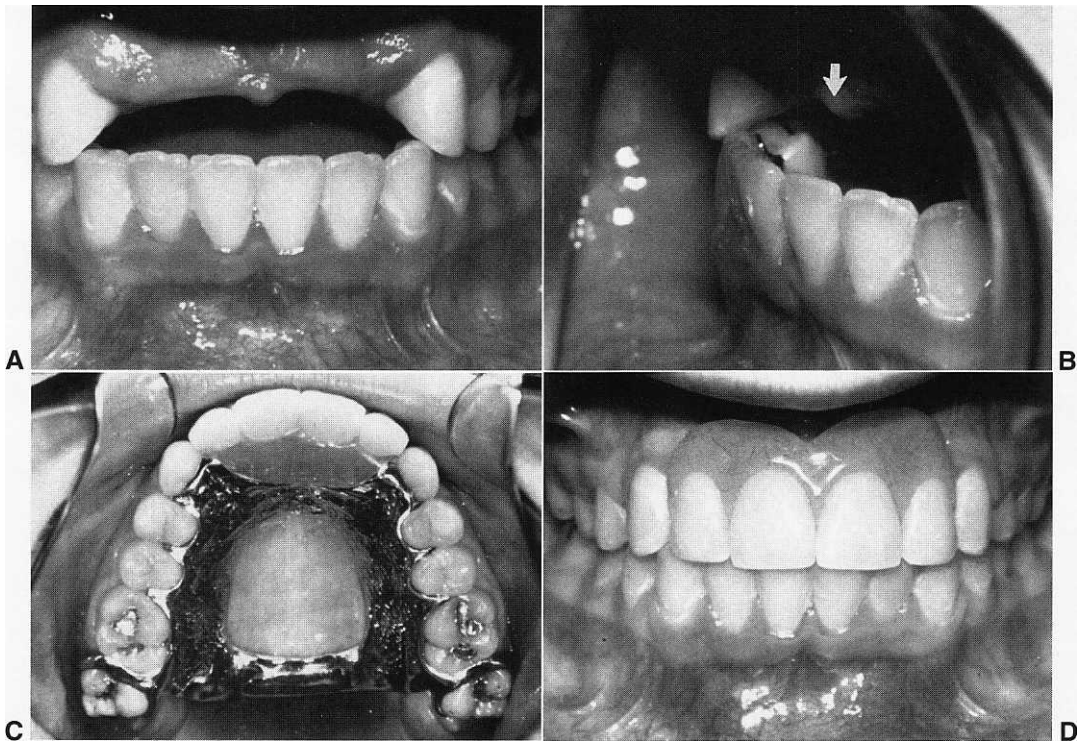
G IG **22-16. A,** Mirror view of maxillary arch. Multiple teeth have been lost in this young adult as a result of a gunshot accident. **B,** Left lateral view in centric occlusion. C, Anterior view in centric occlusion.

The development of proper speech can be ensured if the parts of the partial denture are given correct form, dimension, and position in their relationships to the tongue, cheek, and lips.

The restoration of esthetics is often the most important personal consideration for adolescent patients. Artificial teeth of compatible color, size, and form, naturally arranged and positioned, enhance dental esthetics. In addition, the form and size of the base of a partial denture must be correct to ensure the restoration of normal facial contours.

The preservation of the remaining teeth and their supportive tissues is the most important objective of all but will not be achieved without adequate mouth preparation, correct partial denture design, accurate fabrication of that design, periodic professional follow-up care, and continued proper home care by the patient.

Additional dental procedures may be required to create an oral environment that will furnish proper support and retention for the removable partial denture and that will prevent the development of forces or processes that are harmful to the remaining teeth and their supportive tissues. These preparatory procedures may involve all phases or branches of dentistry. Adolescents may require periodontal procedures, particularly to increase crown length for crown or fixed



f G. **22-17. A,** The four maxillary incisors have been lost as a result of traumatic injury. Note the long span, short clinical crowns, and facial flare to the canines, which make retention and resistance form difficult to achieve for a conventional fixed partial denture. **B,** Lateral view showing the relationship of the mandibular incisors to the residual alveolar ridge. The trauma caused substantial bone loss, and the ridge is located lingual to the mandibular incisors (the *arrow* indicates the position of the incisive papilla on the crest of the residual alveolar ridge), which necessitates use of a removable partial denture base for lip support and proper esthetics. C, Occlusal view of maxillary removable partial denture. D, Facial view of completed prosthesis.

partial denture retention or to improve tissue contours so that more ideal results can be achieved.

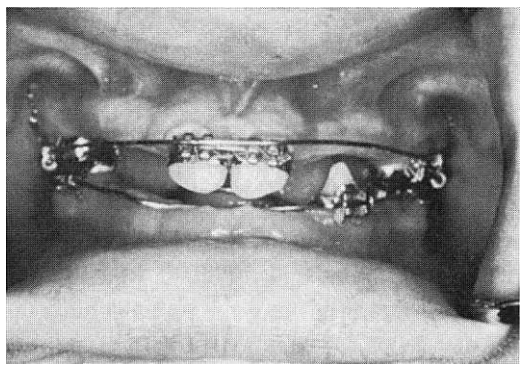
Orthodontic procedures can be used to reposition severely malpositioned teeth that would otherwise require extraction. Orthodontic procedures are particularly indicated for malpositioned teeth that are vital to an adequate plan of treatment (Fig. 22-18).

OVERDENTURES

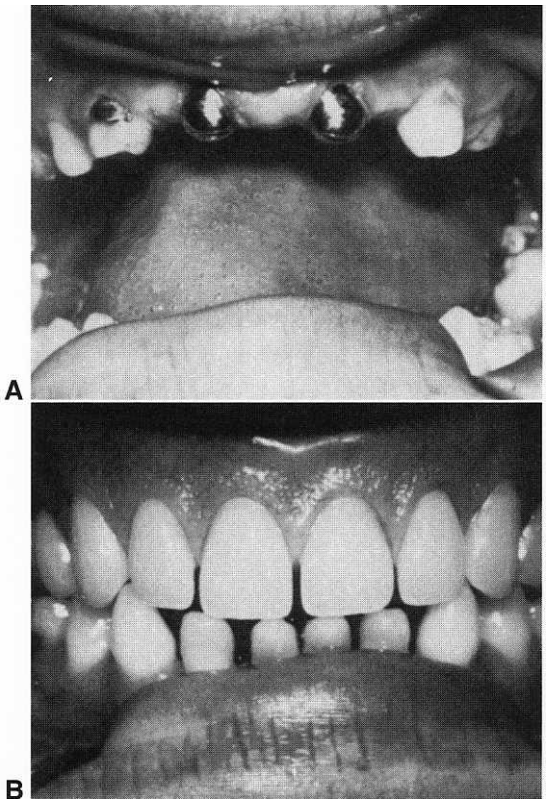
Occasionally, congenital abnormalities or trauma result in the loss of multiple teeth and the resulting interarch relationship does not allow a conventional removable partial denture to reestablish proper occlusion with opposing teeth. This situation may necessitate fabrication of a prosthesis that overlays all or part of the remaining teeth so that proper function and facial esthetics are established (Fig. 22-19).[35]

IMPLANT PROSTHESES

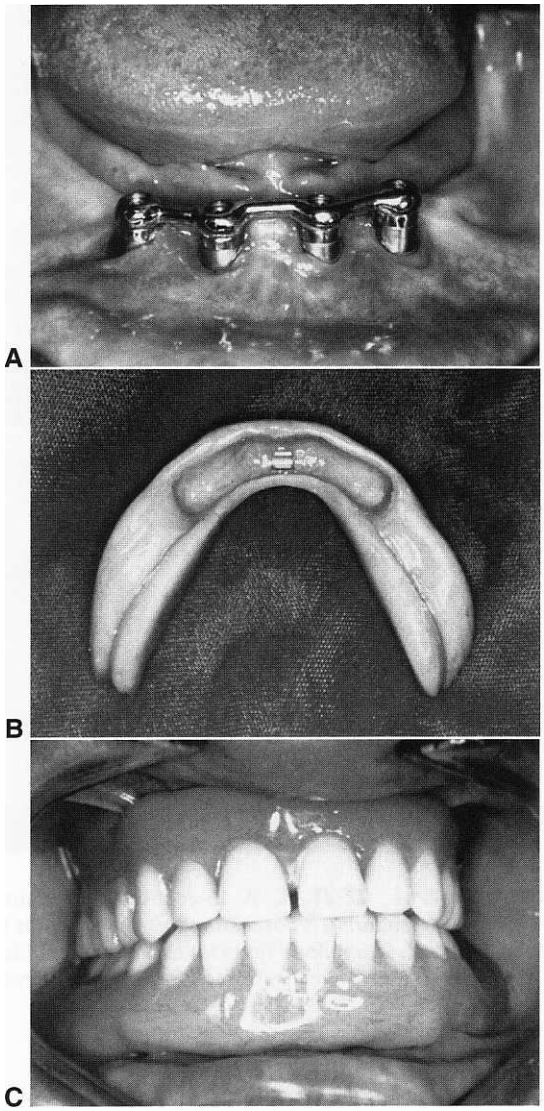
The routine successful use of osseointegrated dental implants in total or partial support of prostheses in adults has heightened interest in their use in younger patients. Adolescents who have congenital anomalies, have undergone ablative surgical procedures, or have experienced traumatic tooth loss are frequently seen, and the use of dental implants would greatly assist



f I G. **22-18.** Orthodontic treatment is in progress to erupt the molars, depress the mandibular left canine, and close the maxillary central incisor diastema.



I **I G 22-19. A,** Facial view of maxillary arch where remaining primary and permanent teeth were improperly spaced and lacked adequate bone support for a fixed prosthesis. Lip support, facial esthetics, and proper occlusal interdigitation with the opposing mandibular teeth could be achieved only by use of an overdenture. Central incisors required endodontic treatment and have been restored with cast posts and cores. **B,** Facial view of overdenture.



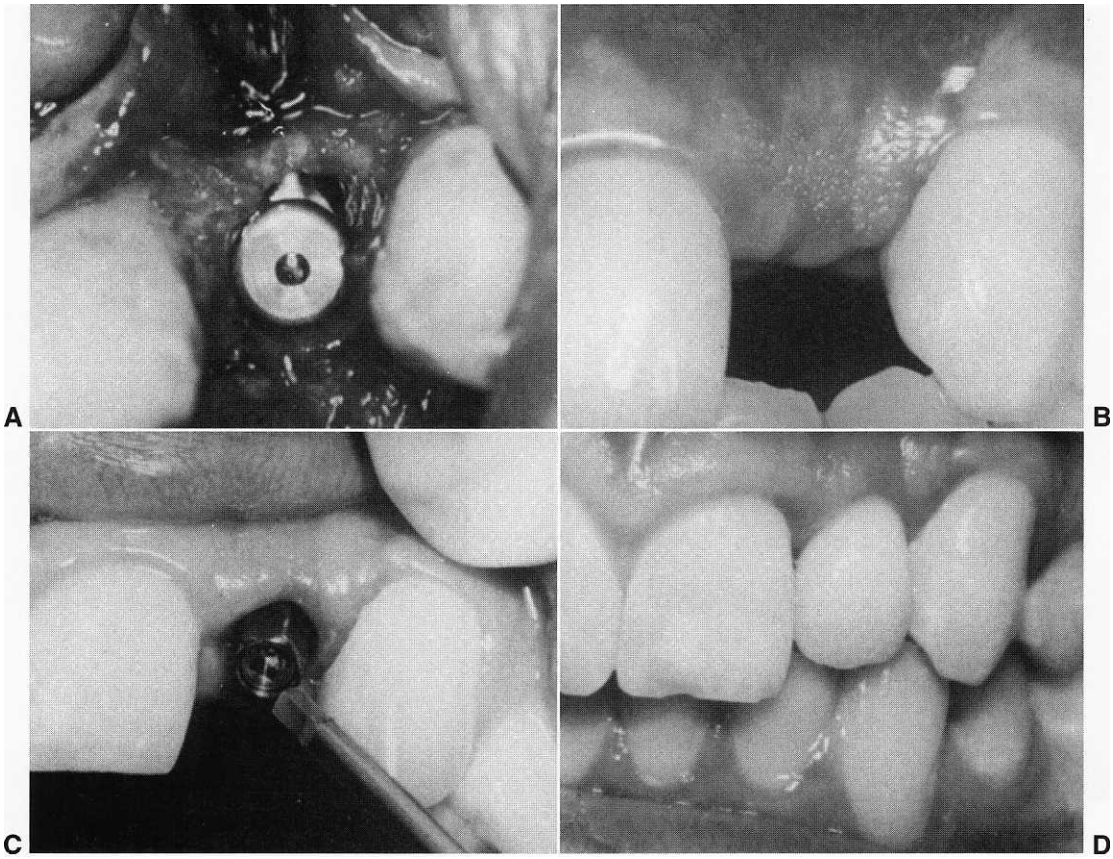
[I G **22-20. A,** Prepubertal periodontitis resulted in extensive and aggressive bone changes and loss of the teeth. Four implants were placed at age 12. This treatment greatly enhances prosthesis stability and will help preserve the remaining mandibular anterior bone. Because the mandibular symphysis fuses at birth or shortly thereafter, early implant placement in this area is less affected by future growth. **B,** Mandibular overdenture prosthesis that will be supported by the four implants and bar. C, Facial view of the completed prostheses.

prosthesis support (Figs. 22-20 and 22-21). Replacement of congenitally missing teeth with implants can often be accomplished in an esthetic manner that preserves the integrity of adjacent teeth (Fig. 22-22). However, dental and skeletal growth is a major confounding variable related to the use of dental implants in adolescent patients.

There are two primary concerns related to the placement of implants before growth is completed: (1) the effect of growth on the long-term relative position of the dental implant, and (2) the effect of the implant-supported prosthesis on future dental and skeletal growth.

An understanding of dental development and craniofacial growth are certainly a prerequisite for anyone anticipating the use of dental implants in growing patients.[36] Growth and development in the maxilla and mandible are essentially quite different, as are growth and development in the specific areas of each arch.[37 40]

In the maxilla, growth is intimately associated with the growth of the cranial base in early childhood, whereas later growth primarily occurs by enlargement of the maxilla. This growth is extremely variable and can be observed as vertical growth, transverse growth, and anteroposterior growth. Transverse growth occurs primarily at the midpalatal suture of the maxilla. The sutural growth site is extremely important and poses a



F I **G 22-21. A,** A 16-year-old boy traumatically fractured the maxillary left lateral incisor, rendering it nonrestorable. An implant is being placed in the socket immediately after extraction. **B,** Edentulous ridge following implant placement. C, Abutment being connected to implant with screwdriver. D, Completed metal-ceramic restoration.

risk to the placement of an implant-supported prosthesis that crosses this suture and could limit its growth potential. The maxilla also grows vertically by passive displacement as well as by alveolar appositional growth. It is the vertical component of maxillary growth that causes the most concern in the long-term positional stability of the individual implant and its effect on restorative function and esthetics.

Mandibular growth differs greatly from the complex growth in the maxilla. Not closely associated with major cranial passive growth, mandibular growth is primarily downward and forward, mediated by appositional condylar growth. This growth is not purely linear but can be rotational secondary to the precise direction of condylar growth patterns. The appositional growth is also refined by certain areas of resorption, primarily seen on the anterior aspect of the ramus. As the mandible increases in length, it also increases in width, secondary to the flaring of posterior growth direction. Anterior stabilization is accomplished by the early closure of the mandibular synthesis. Although less dynamic than maxillary growth, mandibular growth can create many complexities that could place dental implants at positional risk, especially in the posterior mandible, secondary to vertical changes and resorptive processes.

In addition to understanding growth and development, the clinician must also comprehend the dynamics of the positional relationship between the dental implant and its biologic environment in the growing patient. A wealth of dental literature using in vivo evidence-based studies attests to the long-term success of dental implants and their associated prostheses in adults. Such a wealth of knowledge does not exist, however, for the growing patient. The behavior of an osseointegrated dental implant essentially resembles that of an ankylosed tooth and the latter therefore provides an accurate model of the behavior of an implant in a growing patient. Two facets of the relationship of ankylotic teeth to their actively growing environment must be understood. *First,* the ankylotic tooth, lacking the adaptive mechanisms of a healthy

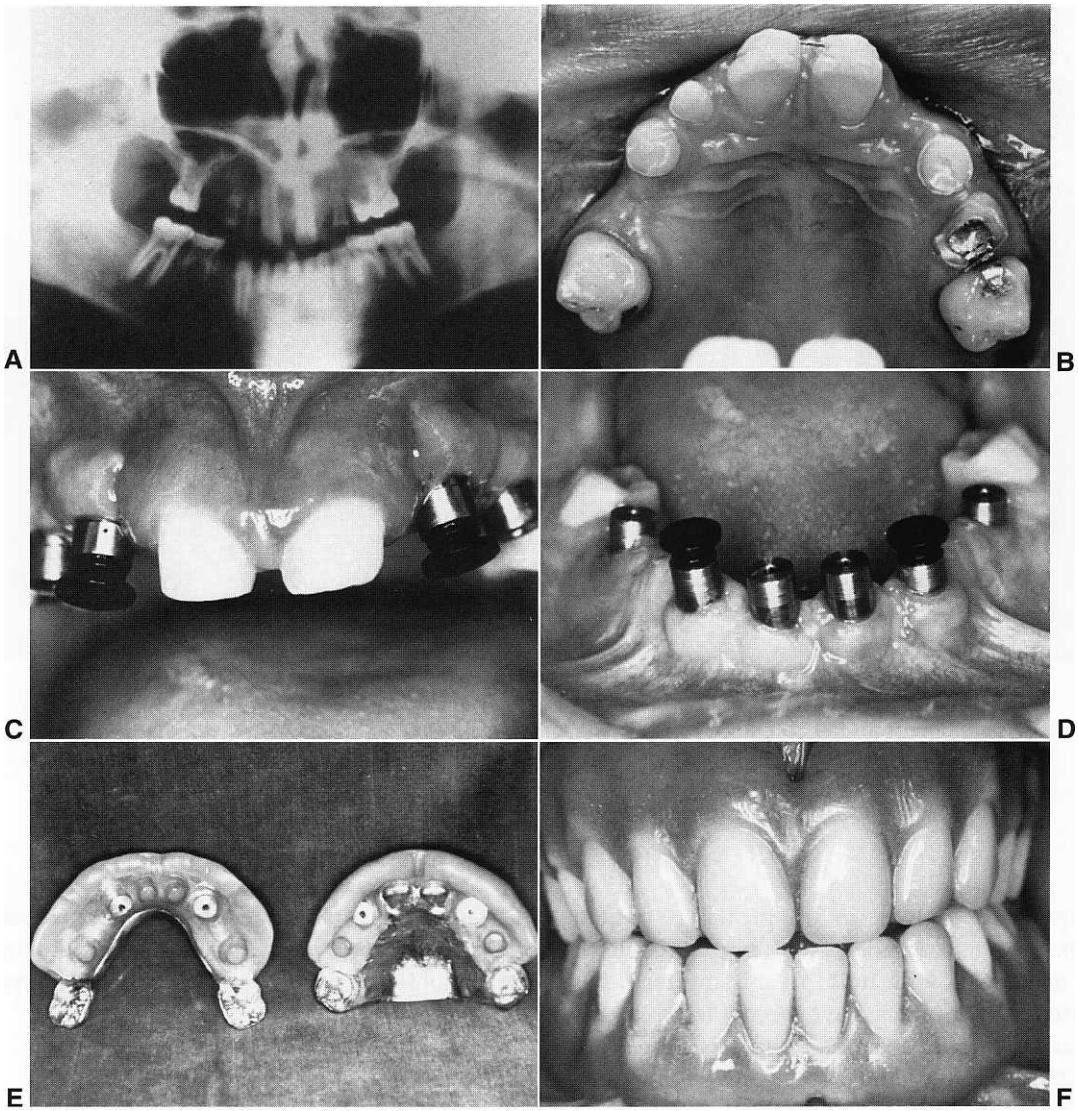


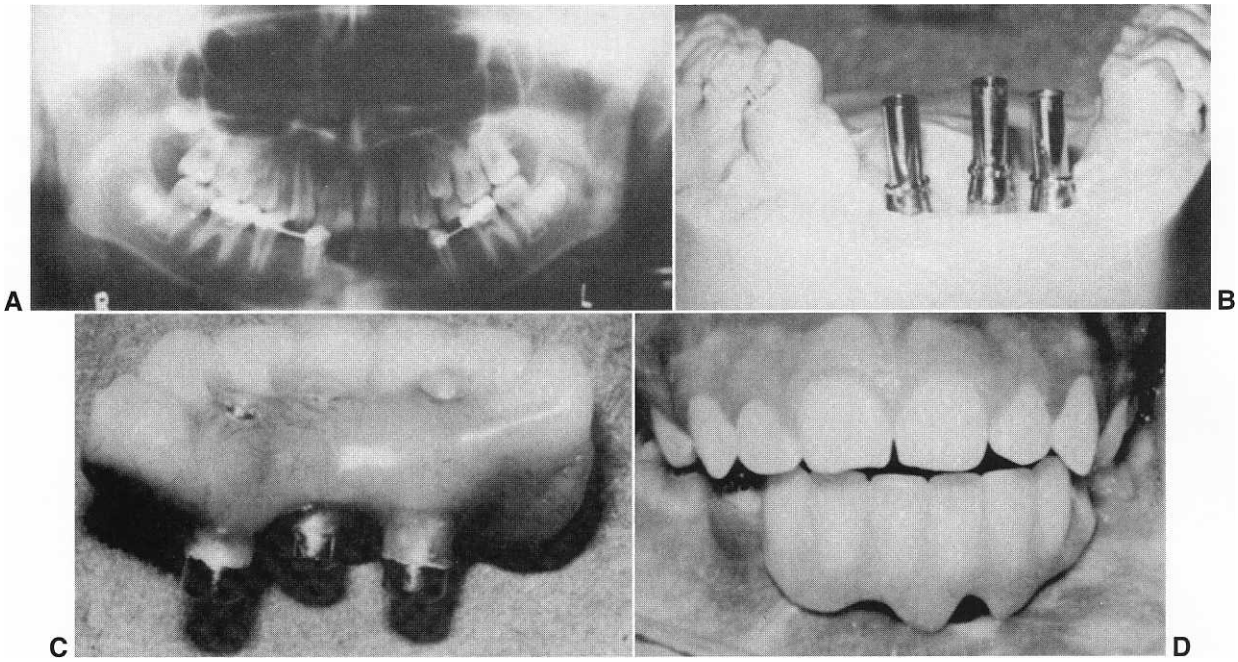
FIG 22-22. A, Panoramic radiograph of a 12-year-old with partial anodontia of unknown origin. **B,** Occlusal view of maxillary arch showing primary teeth that will be extracted. C, Four maxillary implants have been placed and the mouth prepared for an impression. D, Six mandibular implants have been placed to provide prosthesis stability and preserve bone. E, Maxillary and mandibular overdentures using magnets to augment stability and retention. F, Facial view of completed overdentures.

tooth, does not erupt normally and becomes buried. *Second,* failing to participate in vertical growth, it often creates severe malocclusions secondary to tipping and associated growth changes in normal teeth adjacent to the affected ankylotic tooth. It seems logical that an osseointegrated implant placed prematurely could elicit the same negative growth effects.

An understanding of growth and development and its variability in the male and female adolescent population raises serious concern about the premature placement of implants. The reader is referred to Chapter 25 for a more in-depth discussion of this topic.

IMPLANT USAGE BEFORE GROWTH COMPLETION

Although it is best to wait until maxillary and mandibular growth is completed before placing osseointegrated implants, dentists are constantly faced with apparent indications for implant treatment. The clinician must understand the disadvantages of early placement and weigh those factors against the functional and esthetic advantages afforded by implants. If a determination is made that implants are needed before growth completion, it is more predictable to restore larger edentulous areas with implants than to place a single



F **I G 22-23.** **A**, Panoramic radiograph of a 13-year-old patient after removal of a giant cell granuloma from the anterior mandible. **B**, Working cast showing ridge defect and the location of the three implants. C, Fixed prosthesis that will be attached to the three implants. **D,** Prosthesis in place.

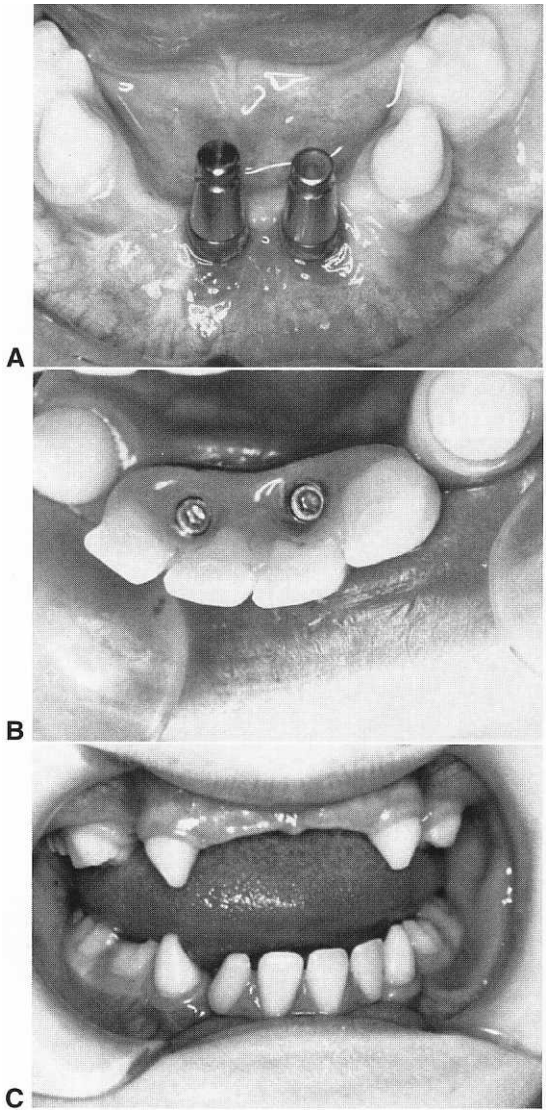
implant-supported crown in a growing patient (Fig. 22-23). The successful use of implant-assisted prostheses in patients with multiple missing teeth has been reported.[4]'-[43] Implants have been placed in the anterior mandible in patients as young as 5 years of age (Fig. 22-24). Often, the placement of implants in this area will diminish the residual alveolar re-sorption anticipated from many years of removable prosthesis wear. Unfortunately, one of the most frequently encountered implant indications is treatment of the traumatic loss or congenital absence of a single anterior maxillary tooth. The placement of dental implants in this area should not be attempted until the accelerated phase of peripubertal growth is close to complete.

IMPLANTS FOR ORTHODONTIC ANCHORAGE

Osseointegrated implants have been used as valuable adjuncts to orthodontic treatment when there is a need for anchorage but no conventional tooth anchorage is available." Because the implants act as ankylosed teeth and are incapable of being moved by orthodontic or orthopedic forces, they serve as ideal anchorage units.' Their use has produced tooth movements that otherwise would not be possible, particularly in cases in which a large number of teeth are missing as a result of trauma or congenital anomalies. Implants are especially valuable when their postorthodontic positioning permits them to be used to support a future prosthesis.

RECARE PROGRAM

The prosthodontic treatment of an adolescent does not end with the placement of the prosthesis. Periodic recare appointments for inspection, maintenance, repair, or replacement are a necessity. For patients who have removable partial dentures, relining or rebasing should be performed when indicated. When all-ceramic crowns or metal-ceramic restorations are used in an adolescent patient, replacement may be needed periodically as the gingival tissue assumes its adult position. Patients with fixed partial dentures should be examined periodically for soft tissue health, evidence of occlusal wear, and response of the supportive tissues to the added stress loads. Patients with implant-assisted restorations should be evaluated frequently to ensure the health of the osseointegrated implant and its surrounding tissue, as well as to assess the effect of the implant-supported prosthesis on the overall growth and development of the patient. The use of dental implants to assist in the support of a prosthesis necessitates an extremely vigilant recare program.



f **I G . 22-24. A**, Two implants placed at age 5 in a young patient with partial anodontia resulting from ectodermal dysplasia. Impression copings have been attached for the impression procedure. Because of the early fusion of the mandibular symphysis, future bone growth will have minimal impact. **B,** Incisal view of the retrievable prosthesis showing two lingually located screws. A retrievable design permits the prosthetic teeth to be replaced when the surrounding teeth change positions from growth. C, Facial view of completed prosthesis.

Every adolescent patient must be taught proper oral hygiene and home care for his or her prosthodontic restorations and must be motivated until adequate performance is routinely achieved. Each patient with fixed or implant-supported prostheses should be taught the use of aids such as the floss threader and interproximal brush to enhance oral hygiene efforts. Only with regular recare programs can maximum longevity of service can be realized.

PROTECTIVE MOUTH GUARDS

Although this chapter is concerned with the prosthodontic treatment of adolescents, emphasis should always be placed on the prevention of oral disease and injury. The number and severity of injuries to the teeth and jaws can be significantly reduced through the faithful use of protective mouth guards by athletes who are engaged in contact sports.'

Effective and relatively inexpensive prefabricated mouth guards are available at sporting goods stores. A custom-made guard may be fabricated, however, by vacuum-molding a sheet of thick, clear material over a stone cast of the maxillary arch. Seals and Dorrough have reviewed the advantages of custom-made mouth protectors.'

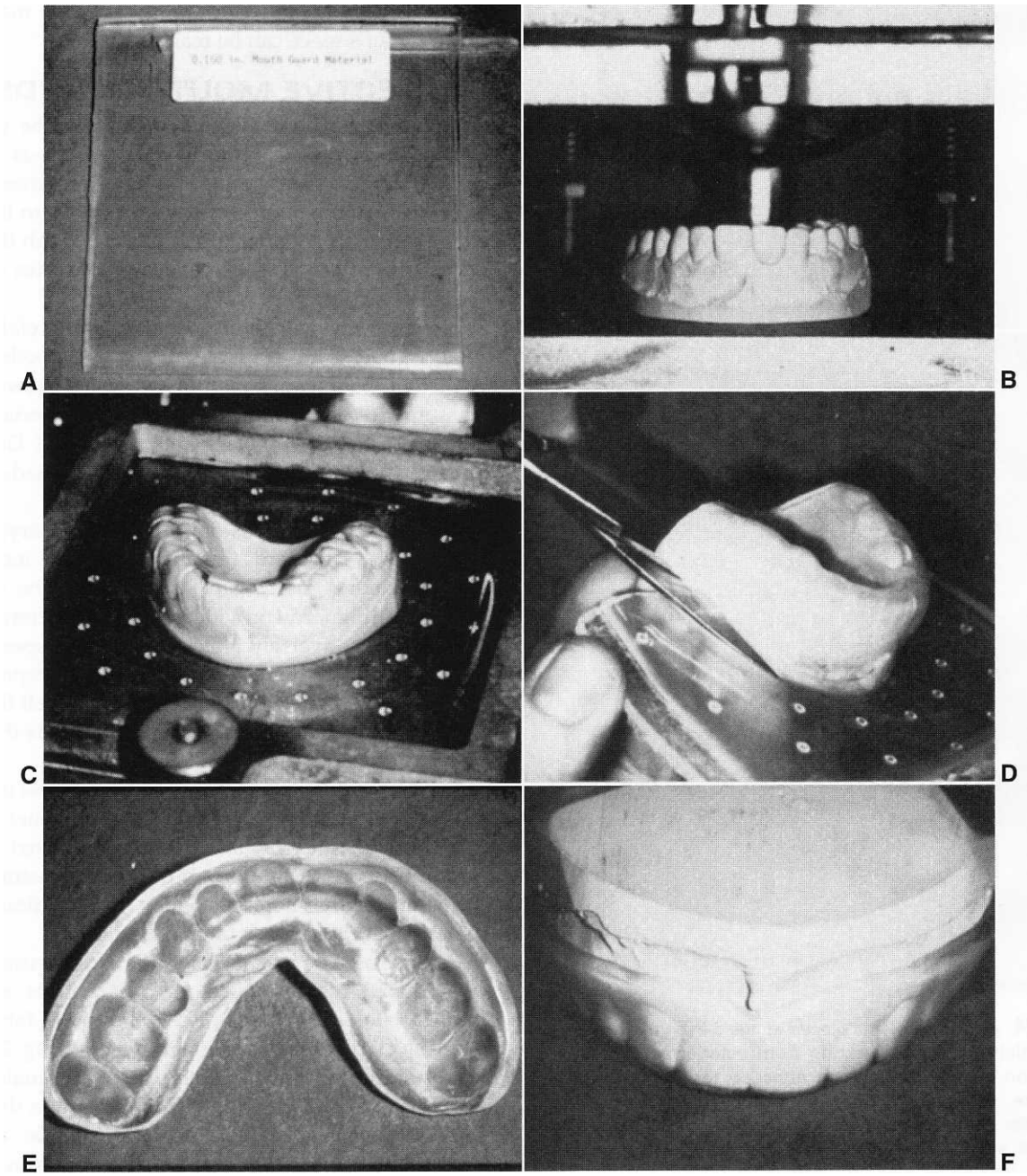
Studies comparing custom-made (laboratory) mouth guards with standard (manufactured) or intraorally formed mouth guards have shown that the custom-made mouth guards provide better fit and comfort, are less likely to adversely affect the player's speech, and are less likely to come loose." In a study reported by Bass and Williams, the majority of athletes felt that they were more likely to wear their mouth guards if they fit better and were comfortable."

Several materials have been suggested for use in mouth guards, including poly (vinyl acetate-ethylene) copolymer thermoplastic, polyurethane, and laminated thermoplastic. Chaconas, Caputo, and Bakke demonstrated that the laminated thermoplastic underwent significantly less dimensional change than other materials.[50]

Because a custom-made mouth guard accurately fits individual tooth and arch form, it affords maximal resistance to dislodgement. The technique of fabrication involves placing the mouth guard material (Fig. 22-25, A) in a molding machine, which softens the material by heat (Fig. 22-25, B) and closely adapts it to a dry stone cast by vacuum (Fig. 22-25, O. After the adapted material has cooled, the guard is removed from the cast and the excess peripheral material is trimmed off with scissors (Fig. 22-25, D). The borders are rounded by trimming the material with a resin-trimming bur and flaming with a torch or polishing with wet pumice on a rag wheel (Fig. 22-25, E).

Maximum retention is obtained when the entire hard palate is covered. If the guard interferes with speech, however, a portion of the palatal area of the guard can be removed.

The successful use of mouth guards by many young athletes has proven that they can be worn with comfort and serve as effective safeguards against injuries to the teeth.



F **I G 22-25. A,** Clear material (0.150 inch) used to fabricate a mouth guard. **B,** Material clamped in machine and heated until it softens and droops. C, Material vacuum-adapted to dry stone cast. D, Guard being trimmed with scissors. **E,** Appearance of borders after being trimmed with acrylic bur and polished with pumice. **F,** Completed mouth guard on cast.

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CHAPTER

23

Dental Problems of Children with Disabilities

JAMES A. WEDDELL
BRIAN J. SANDERS
JAMES E. JONES

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CHAPTER OUTLINE

DENTAL ACCESS
FIRST DENTAL VISIT
RADIOGRAPHIC EXAMINATION
PREVENTIVE DENTISTRY
- Home Dental Care Diet
- and Nutrition Fluoride
- Exposure Preventive
- Restorations
- Regular Professional Supervision

MANAGEMENT OF A CHILD WITH DISABILITIES DURING DENTAL TREATMENT
- Treatment Immobilization

MENTAL DISABILITY
- Dental Treatment of a Person with Mental Disability

DOWN SYNDROME (TRISOMY 21 SYNDROME)
LEARNING DISABILITIES FRAGILE X SYNDROME FETAL ALCOHOL SYNDROME
AUTISM
CEREBRAL PALSY
SPINA BIFIDA AND LATEX ALLERGY
RESPIRATORY DISEASES
- Asthma (Reactive Airway Disease)
- Bronchopulmonary Dysplasia
- Cystic Fibrosis

HEARING LOSS
VISUAL IMPAIRMENT
HEART DISEASE
- Congenital Heart Disease
  - *Acyanotic congenital heart disease*
  - *Cyanotic congenital heart disease*
- Acquired Heart Disease
  - *Rheumatic fever*
  - *Infective bacterial endocarditis*
  - *Endocarditis prophylaxis*
- Dental Management
- Cardiac Surgery Patients

Approximately 11 million children and adults in the United States have a disabling condition. The term *disability* includes all handicapping conditions or combinations a health professional might encounter. Children with disabilities may have a mental, physical, medical, or social condition that interferes with normal functioning.

Many children with disabilities are best managed initially by a multidisciplinary team in which a dentist is available to evaluate extraoral and intraoral findings of the child. A diagnosis is then established, and an impression is gained of the child's strengths and weaknesses and the team's recommendations for future care. The child may then be referred to a dental practitioner whom the family chooses, if a preference is indicated. The team is available to help the family practitioner or pediatric dentist treat the child and prepare the family for the child's future treatment needs.

Children with disabilities may present challenges that require special preparation before the dentist and office staff can provide acceptable care. In addition, parental anxiety concerning the problems associated with a child's disabilities frequently delays dental care until significant oral disease has developed. Also, some dentists feel uncomfortable providing treatment for children with disabilities, which results in a loss of greatly needed services. Giddon, Rude, and Belton' indicate that it is not accessibility, availability, and acceptability that determine the extent to which a patient with disabilities will be treated by the dentist. Rather, it is the perception of the situation by the dentist and patient and their willingness to interact.

This chapter discusses some of the disabling conditions of children that a dentist may encounter in practice. If a dentist becomes familiar with the special needs of a child with disabilities and with the parents' concerns, the dental management of the child can be quite gratifying.

DENTAL ACCESS

Improving access to dental and oral health care for those now deprived of needed services should be of great concern to our profession. Large segments of the population do not have access to dental care. Children with special health needs, such as those who are chronically ill, homebound, developmentally disabled, and emotionally impaired, fall into this group. In addition, these groups have been identified as a significant portion of the 20% of the U.S. population who exhibit *80%* of all caries.

The rapid expansion in the elderly population, the presence of children with disabilities, and the emergence of legislative guidelines for people of all ages with disabilities are three important factors that should prompt dentists to address cost-efficient ways to make their office facilities and operatory areas accessible for persons with disabilities. In August 1984, another federal-level cornerstone was laid for barrier-free facility access with the passage of the Uniform Federal Accessibility Standards.' As additional legislation is passed and existing legislation is applied, people with disabilities will be in a better position to seek services, career opportunities, education, and social growth. Dental consumer populations are becoming more sensitive to the service needs and desires of people with disabilities by improving family conditions and public education. The dental practitioner is beginning to meet these needs by establishing barrier-free facilities to accom modate people with any kind of disability. Table 23-1 lists common minimum requirements needed to gain access.

In the dental operatory, doorways should be 4 inch (10 cm) wider than normal. In the dental suite where floor circulation space is at a premium, aisle passage in the operatory area should be planned with the dimensions shown in Fig. 23-1. The required wheelchair turning space and top space under furniture and fixtures may be more readily accommodated if one operatory is specifically designed with a movable dental chair, instrument control unit, and suction system. Movable equipment should enhance the opportunity to back the patient's wheelchair into the operatory and thus reduce the need for more wheelchair turning space. If possible, a wider radius for turning space is desirable to accommodate wheelchair extensions and adaptations that are required for some persons. Dental chairs should be adjustable for height to match different wheelchair designs.

FIRST DENTAL VISIT

Many people avoid dental treatment for themselves or their children because of their fear that dental visits are routinely painful. Dental professionals should use every opportunity to help patients or parents overcome this barrier into the mainstream of effective dental care.

The initial dental examination for a child with disabilities is like the initial examination described in Chapter 1. Special attention should be given to obtaining a thorough medical and dental history. The names and addresses of medical or dental personnel who have previously treated the patient are necessary for consultation purposes. Consultation with these specialists is common and helps provide insight in case management and planning.

The first dental appointment is very important and can set the stage for subsequent appointments. As previously noted, parental anxiety about a child's dental treatment may be a significant factor. By scheduling the patient at a designated time (early in the day) and

TABLE 23-1 Disabled Accessibility Guidelines

EXTERNAL/INTERNAL BUILDING FEATURES	GRADIENT	LENGTH	WIDTH	SURFACE, OTHER SPECIFICS
Parking space	1:50 max slope	Standard	Auto: 96 inches Van: 144 inches	Nonskid, paved, sign-posted, adjacent walkway
Walkway	1:12 max slope	Not applicable	36 inches	Nonskid, no obstructions, overhangs, smooth
Passenger loading zone	Flat	20 feet	60 inches	Same as above
Curb ramps	1:12 max slope		36 inches	Nonskid, side flair <1:10 slope
Door	5-foot entrance and exit platform area	Standard	32-inch min with preferably 36 inches	Away from prevailing winds, lever with 10-lb pull, auto-assisted door available, kick plate
Interior ramp	1:20 max slope	72-inch min length if rise >6	36 inches	Nonskid, handrails
Wheelchair lift	Bilevel	8-foot max drop	36 x 48 inches	Nonskid, dependent on specific chair
Corridor		Standard	48 inches/64 inches	New facility, no obstacles
Flooring	Flat, firm carpet	Not applicable	1/2-inch max thickness	No doormats, level thresholds
Signs	Braille, raised letters	Above 5 feet	Readable	Near latch of office door
Waiting room	Flat	Standard	36-inch aisle One cleared area: 36 x 52 inches	No carpet pad, well insulated, min low-frequency background noise
Restrooms	Flat		32-inch stall min, preferably 36 inches	Nonskid, magnetic catch door
Public telephone	No higher than 4 feet	3 feet above floor	26-inch clearance	Phone directory near phone, adjustable volume control
Elevator	Flat		**54** x 68 inches	Nonskid, call and control box 48 inches high, include braille or incised letters
Operatory	Flat 8 x 10 feet	Standard	32- to 36-inch door	Nonskid, rotating or movable chair, drill, and suction

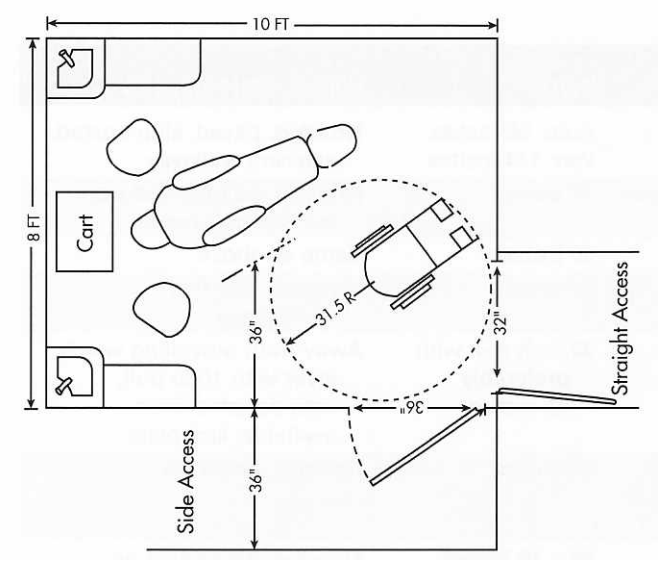
Adapted from Bill D, Weddell JA: *Spec Care* Dentist 7:246-252, 1987.

allowing sufficient time to talk with the parents (or the guardian) and the patient before initiating any dental care, a practitioner can establish an excellent relationship with them. Sending a letter before the appointment explaining the first visit to the family and sending another letter afterward letting them know how helpful they were is beneficial. This initial demonstration of sincere interest in the child often proves advantageous and saves time throughout the entire treatment process.

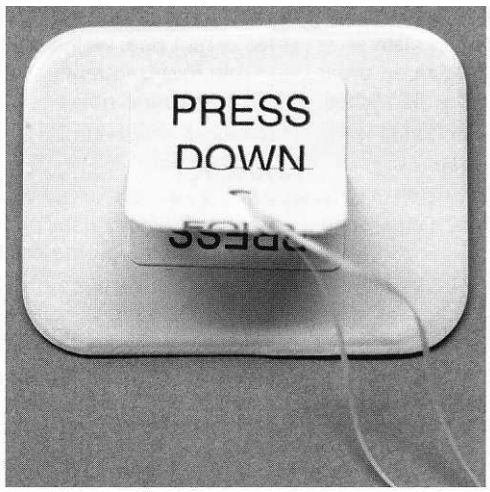
RADIOGRAPHIC EXAMINATION

Adequate radiographic records are often necessary in planning dental treatment for the child with disabilities. Through appropriate behavior management of the child, a dentist can usually perform a complete radio-graphic examination of the teeth when indicated. Occasionally, assistance from the parent and dental auxiliaries and the use of immobilization devices may be necessary to obtain the films. Better cooperation may be elicited from some children by delaying radiography until the second visit, when they are familiar with the dental office and have found it a friendly place.

For patients with limited ability to control film position, intraoral films with bite-wing tabs are used for all bite-wing and periapical radiographs. An 18-inch (46-cm) length of floss is attached through a hole made in the tab, as shown in Fig. 23-2, to facilitate retrieval of the film if it falls toward the pharynx. Regardless of the types of radiographs to be made, the patient should wear a lead apron with a thyroid shield, and anyone who helps hold the patient and the film or sensor steady should wear a lead-lined apron and gloves (Fig. 23-3).



f **G 23-1.** An accessible dental operatory floor plan designed for either a straight or side access doorway. *(From Bill D, Weddell* JA: Spec Care Dentist 7:246-252, 1987.)



f **G . 23-2.** Bite-wing radiographic film secured with floss.

PREVENTIVE DENTISTRY

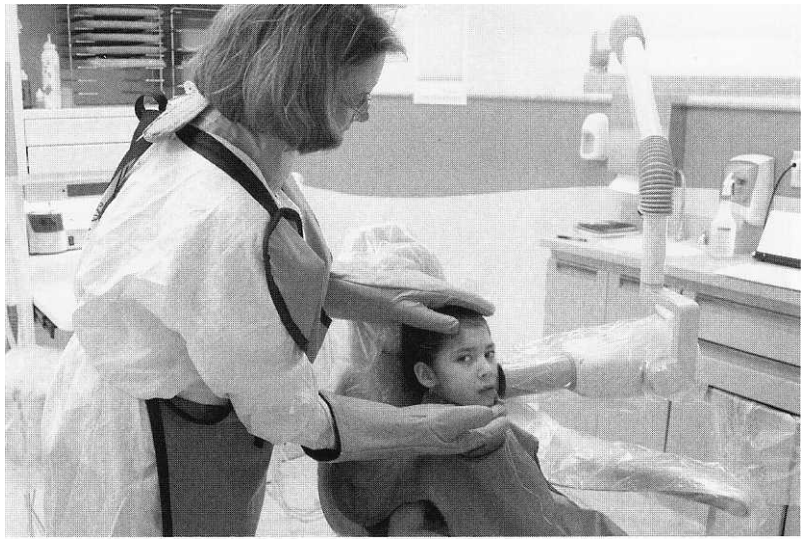
Preventing oral disease before it starts is the most desir able way of ensuring good dental health for any dental patient. An effective preventive dentistry program is important for a child with disabilities because of the predisposing factors that make restorative dental care harder to obtain when it is necessary. After the diagnosis the dentist should determine the patient's needs, assume the responsibility for formulating an individual program for the child, and adequately communicate to the parents and patient how such a program can be effected. A clear perception of the situation by everyone involved is essential for a successful preventive program; adequate communication is vital.

HOME DENTAL CARE

The parents (or the guardian) are initially responsible for establishing good oral hygiene in the home. Reinforcement of good home dental care is provided through mass media (e.g., newspapers, radio, and television), communication with other people, and school activities (e.g., health classes, parent-teacher association meetings, and observation of National Dental Health Month). This supplementary support relieves the dentist having sole responsibility for explaining the need for home dental care and reinforces the receptivity of the parent and child to such a program. The dentist or the hygienist is responsible for consulting with the caregiver of the child with disabilities (i.e., parent, guardian, or nursing home attendant) in those cases in which continued oral hygiene problems appear. Follow-up observation is essential for effective implementation of the preventive dental treatment plan.

Home dental care should begin in infancy; the dentist should teach the parents to gently cleanse the incisors daily with a soft cloth or an infant toothbrush. For older children who are unwilling or physically unable to cooperate, the dentist should teach the parent or guardian correct toothbrushing techniques that safely restrain the child when necessary. Fig. 23-4 shows several positions for toothbrushing that permit firm control and support of the child, adequate visibility, and convenient positioning of the adult, with reasonable comfort for both adult and child (also see Fig. 11-16). Some of the positions most commonly used for children requiring oral care assistance are as follows:

- The standing or sitting child is placed in front of the adult so that the adult can cradle the child's head with one hand while using the other hand to brush the teeth.
- The child reclines on a sofa or bed with the head angled backward on the parent's lap. Again, the child's head is stabilized with one hand while the teeth are brushed with the other hand.
- The parents face each other with their knees touching. The child's buttocks are placed on one parent's lap, with the child facing that parent, while the child's head and shoulders lie on the other parent's knees; this allows the first parent to brush the teeth.
- The extremely difficult patient is isolated in an open area and reclined in the brusher's lap. The patient is then immobilized by an extra attendant while the brusher institutes proper oral care. If a child cannot be adequately immobilized by one person, then both parents and perhaps siblings may be required to complete the home dental care procedures.
- The standing and resistive child is placed in front of the caregiver so that the adult can wrap his or her



f I G 23-3. Extra assistance in holding the patient's head steady to prevent movement while a radiograph is being made.

legs around the child to support the torso while using the hands to support the head and brush the teeth.

If a child is institutionalized, the staff should be instructed in the proper dental care regimen for the child. Wrapped tongue blades may be of benefit in helping to keep a child's mouth open while plaque is being removed. Stabilization of the child's head will prevent the occurrence of any unnecessary trauma from sudden movements. Follow-up observation is carried out by the dentist or the hygienist, and it is appropriate to offer in-service training sessions and to check with the staff periodically to identify and solve the problems associated with an oral hygiene program in the institution.

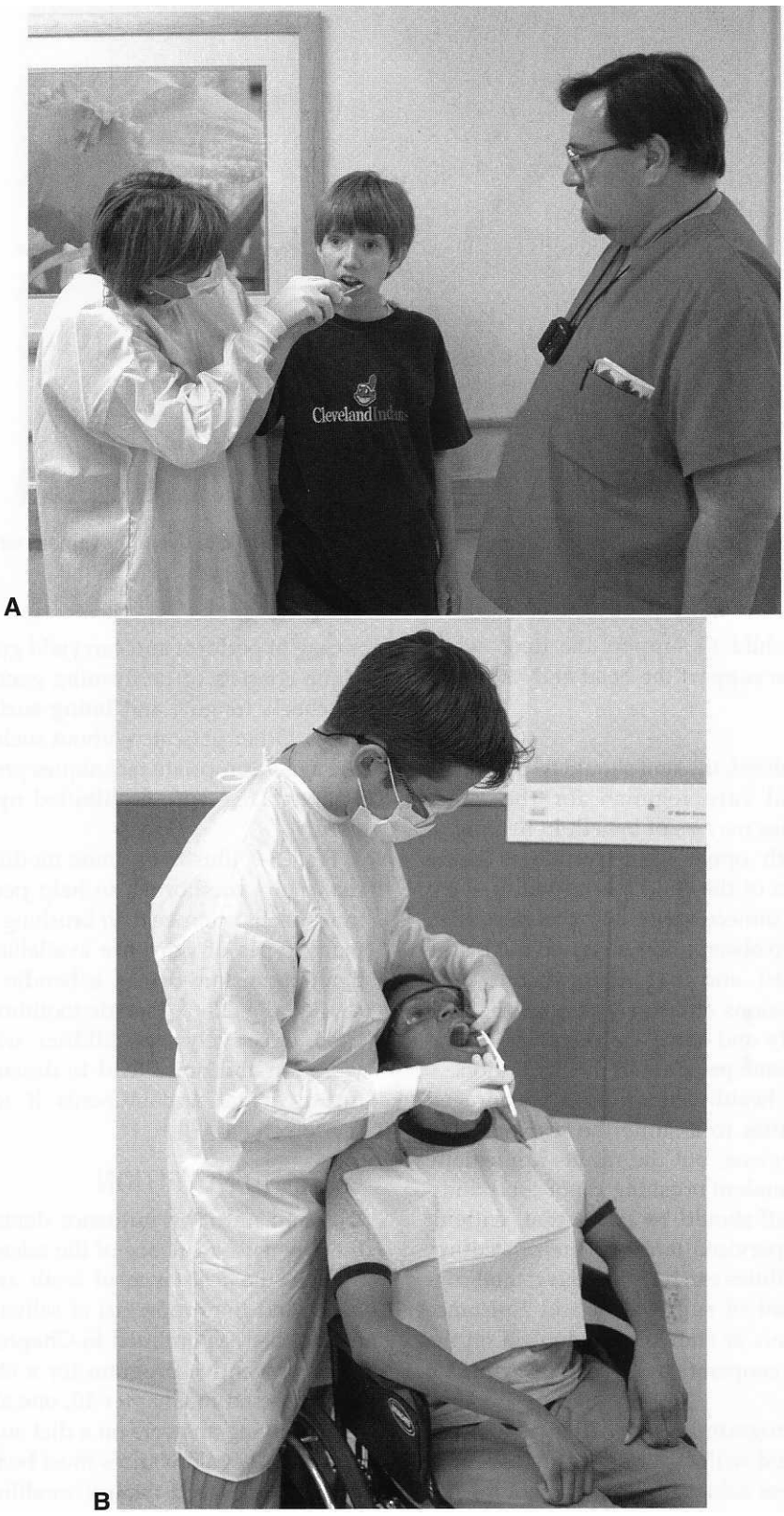
Some parents and health centers have encouraged children with disabilities to assume the responsibility for their own oral hygiene, but the results are usually poor. Although independent brushing is not contraindicated, parents and staff should be aware that, without their follow-up, unsupervised oral hygiene procedures in children with disabilities can have serious dental consequences. The amount of supervision and assistance provided by the parents or staff should depend on the child's willingness to cooperate and ability to maintain good oral hygiene.

A plaque control program is essential in monitoring oral hygiene in the child with disabilities and determining the level of success achieved by each patient. The brushing technique for patients with disabilities who have fine or gross motor deficiencies limiting their ability to brush should be effective and yet simple for the person performing the brushing. One technique often recommended is the horizontal scrub method because it is easy to perform and can yield good results. This technique consists of performing gentle horizontal strokes on cheek, tongue, and biting surfaces of all teeth and gums. Other patients without such motor problems can use age-appropriate techniques previously discussed in Chapter 11. A soft, multitufted nylon brush should be used.

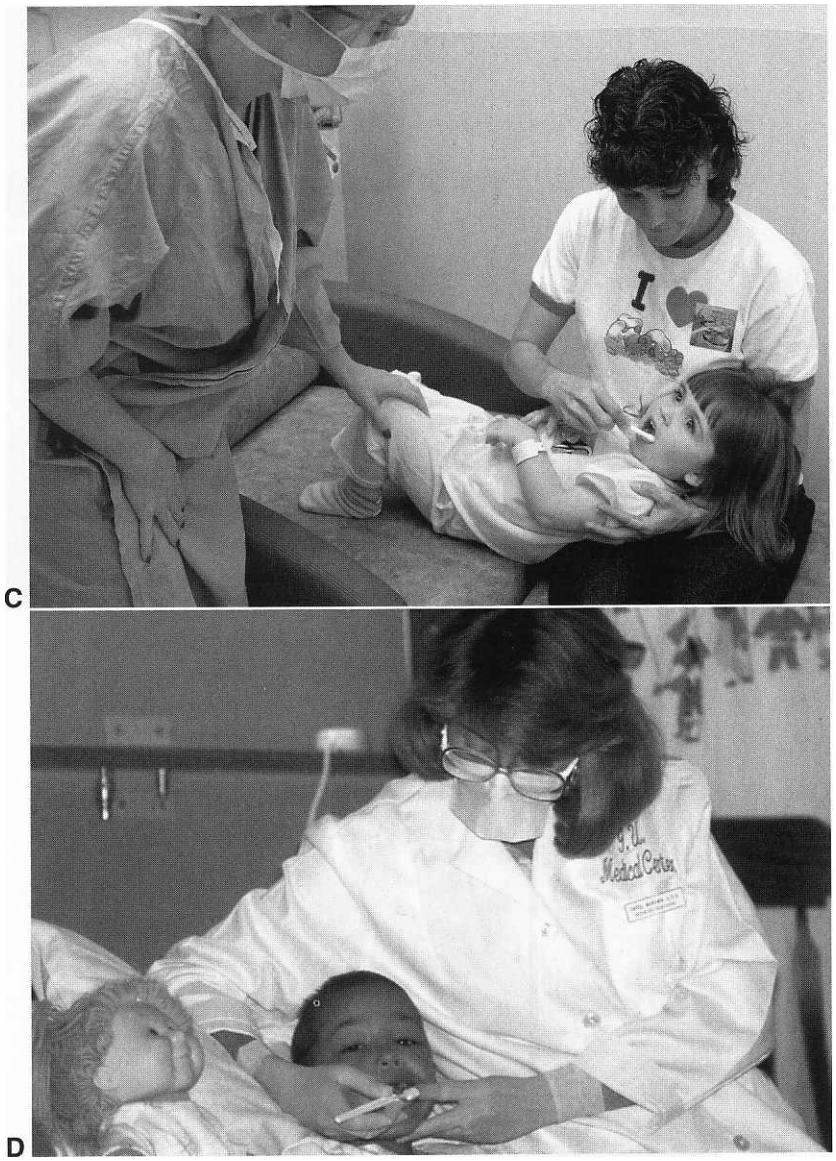
Fig. 23-5 illustrates some modifications that may be made to a toothbrush to help persons with poor fine motor skills improve their brushing techniques. Although many types of grips are available, using the patient's hand to custom-design a handle has often had good results (Fig. 23-6). Electric toothbrushes have also been used effectively by children with disabilities. The vibration and noise tend to desensitize the patient for future dental appointments if followed by positive reinforcement.

DIET AND NUTRITION

Diet and nutrition influence dental caries by affecting the type and virulence of the microorganisms in dental plaque, the resistance of teeth and supporting structures, and the properties of saliva in the oral cavity. A proper diet, as outlined in Chapter 12, is essential to a good preventive program for a child with disabilities. As discussed in Chapter 10, one should assess the diet by reviewing answers on a diet survey with the parent, realizing that allowances must be made for certain conditions for which dietary modifications are required. For example, conditions associated with difficulty in swallowing, such as severe cerebral palsy, may require that the patient be on a pureed diet. Patients with certain metabolic disturbances or syndromes, such as phenylketonuria, diabetes, or Prader-Willi syndrome,



fi G 23-4. Common positions for plaque removal. A, Standing. **B,** Upright wheelchair.



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FIG 23-4, contd. C, Reclining on couch. D, Reclining in bed. *Continued*

have diets that restrict specific foods or total caloric consumption. Whatever the special circumstances, any dietary recommendations should be made individually after proper consultation with the patient's primary physician or dietitian. Particular emphasis should be placed on discontinuation of the nursing bottle by 12 months of age and cessation of at-will breast-feeding after teeth begin to erupt to decrease the likelihood of nursing caries.

FLUORIDE EXPOSURE

The judicious use of systemic fluoride is important in the comprehensive management of any dental patient. Special emphasis should be placed on ensuring adequate systemic fluoride for patients with disabilities.

The dentist should first determine the concentration of fluoride in the patient's daily water supply. If the level of fluoride is between 0.7 and 1 ppm, no supplementation is normally required. If the dentist is not sure of the fluoride level of the patient's drinking water or other sources, an analysis to determine the level is indicated. Once the level has been documented, a determination of the need for fluoride supplementation can be made. The amount of systemic fluoride supplementation necessary, along with the various forms available (i.e., drops, tablets, and rinses), is outlined in Chapter 10.

Whether the patient lives in an area with a fluoridated or nonfluoridated water supply, a topical fluoride should be applied after a regularly scheduled professional prophylaxis. An American Dental

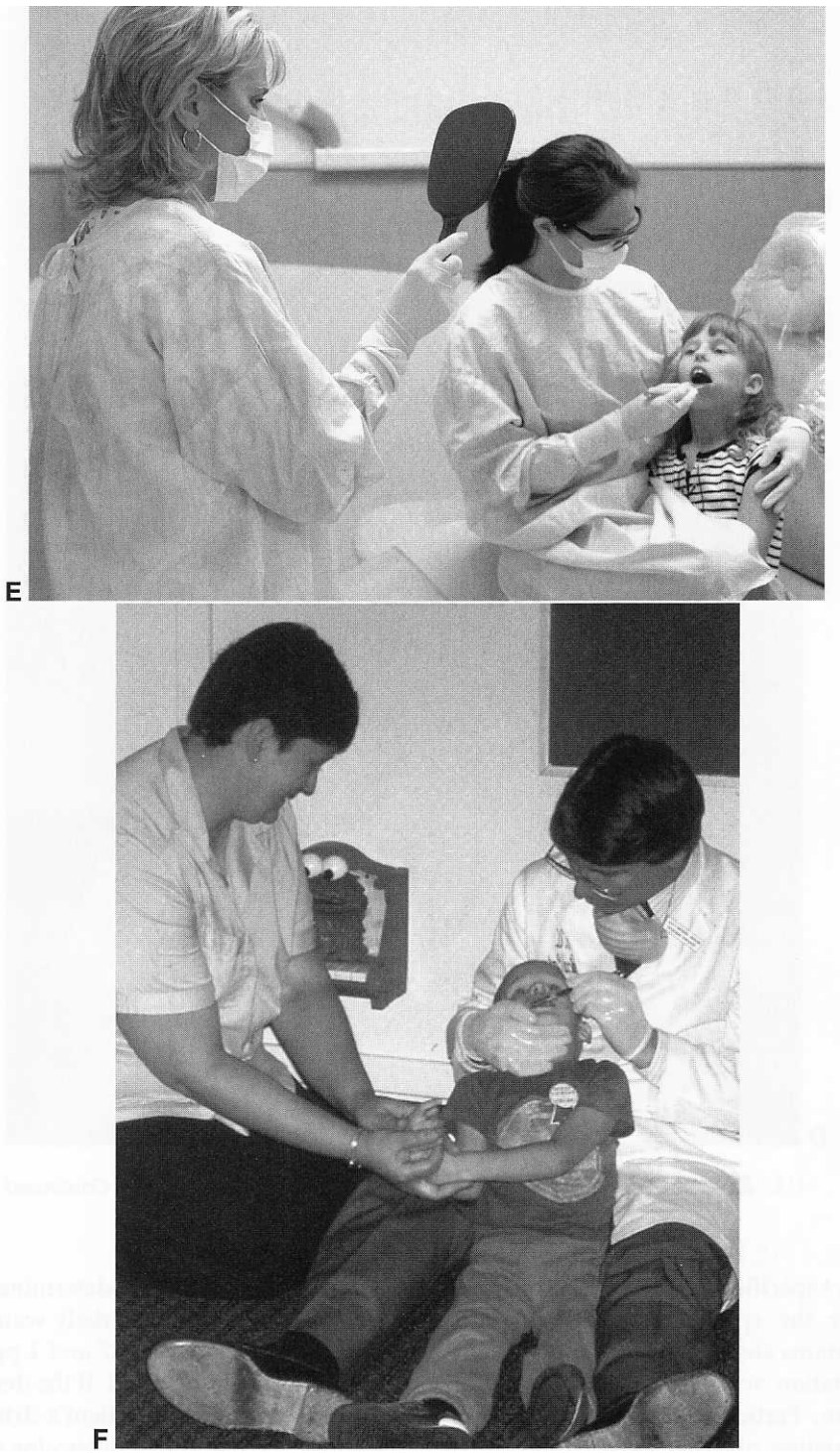
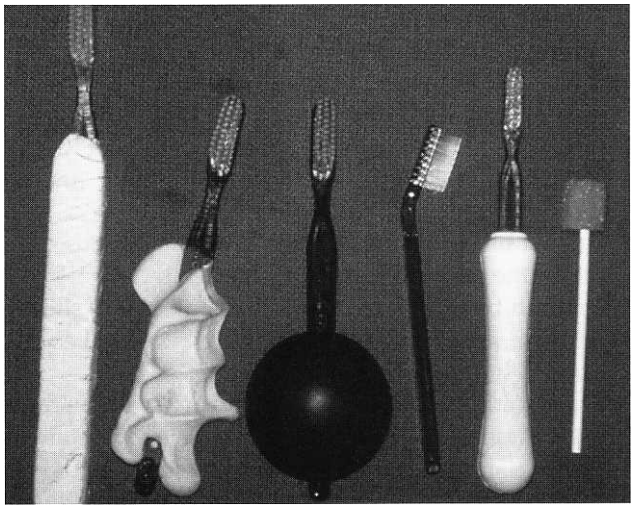


FIG. 23-4, contd. **E,** "Leg-lock" position. F, Reclining on floor.

Association–accepted dentifrice containing a therapeutic fluoride compound should also be used daily. Some clinicians treating patients with disabilities who have chronically poor oral hygiene and high decay rates suggest a daily regimen of rinsing with 0.05% sodium fluoride solution. Nightly application of a 0.4% stannous fluoride brush-on gel has also been successfully used to decrease caries in children.

PREVENTIVE RESTORATIONS

Pit and fissure sealants have been shown to reduce occlusal caries effectively. Sealants are appropriate in



i I G. **23-5.** Various toothbrush handle modifications.

patients with disabilities. For a patient who requires dental work under general anesthesia, deep occlusal pits and fissures should be restored with amalgam or long-wearing composites to prevent further breakdown and decay. Patients with severe bruxism and interproximal decay may need their teeth restored with stainless steel crowns to increase the longevity of the restorations.

REGULAR PROFESSIONAL SUPERVISION

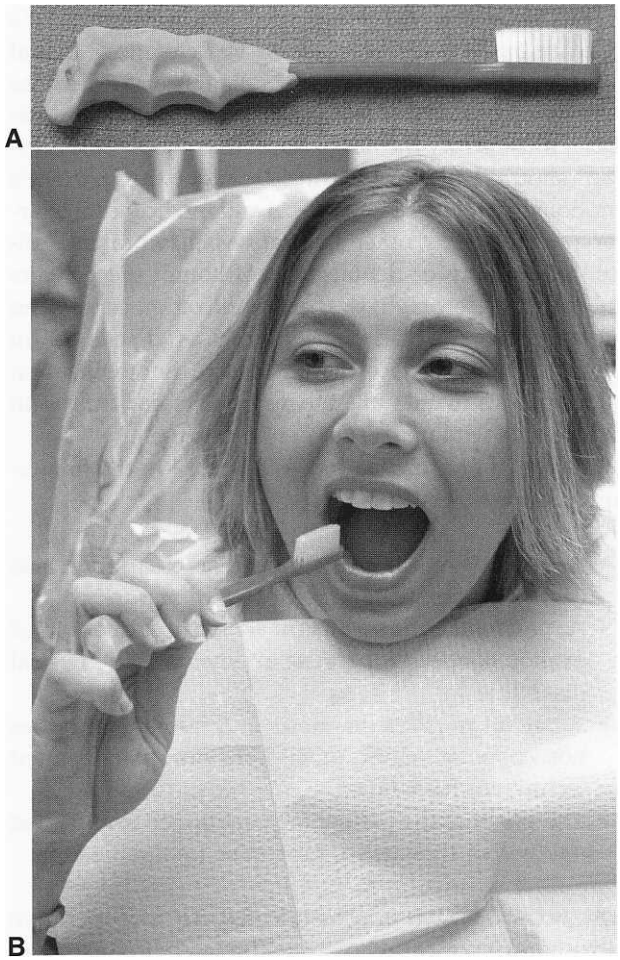
Close observation of caries-susceptible patients and regular dental examinations are important in the treatment of patients with disabilities. Although most patients are seen semiannually for professional prophylaxis, examination, and topical fluoride application, certain patients can benefit from recall examinations every 2, 3, or 4 months. This is particularly true of patients who are confined to institutions in which dental health pro-grams are inadequate.

MANAGEMENT OF A CHILD WITH DISABILITIES DURING DENTAL TREATMENT

The principles of behavior management discussed in Chapter 3 are even more important in treating a child with disabilities. Because hospital visits or previous appointments with a physician frequently result in the development of apprehension in the patient, additional time must be spent with the parent and the child to establish rapport and dispel the child's anxiety. If patient cooperation cannot be obtained, the dentist must consider alternatives such as treatment immobilization and conscious sedation to allow performance of the necessary dental procedures.

TREATMENT IMMOBILIZATION

Partial or complete immobilization of the patient is sometimes a necessary and effective way to diagnose and



H E **23-6. A,** Custom-designed acrylic handle. **B,** Patient using the custom-handle toothbrush.

deliver dental care to patients who need help controlling their extremities, such as infants or patients with certain neuromuscular disorders. Immobilization is also useful for managing combative, resistant patients, so that the patient, practitioner, and/or dental staff may be protected from injury while care is being provided. Treatment immobilization can be performed by the dentist, staff, or parent, with or without the aid of an immobilization device.

The parents, guardian, or patient (if an adult) must be informed and must give consent, and the consent must be documented, before immobilization is used. These individuals should have a clear understanding of the type of immobilization to be used, the rationale, and the duration of use. In many cases this information should be included in the explanation of the overall management approach for the child during the initial examination and conference with the parents.

In October 1990 the Omnibus Budget Reconciliation Act of 1987 became effective. It provided recommended guidelines to reduce the risk of injury and death from the use of patient restraints.

The American Academy of Pediatric Dentistry's *Guideline on Behavior Management* in Reference Manual 2002-03 indicates that the need to diagnose and treat, as well as to protect the safety of the patient and practitioner, must justify the use of immobilization .[3] This deci sion should take into consideration the patient's emotional development, physical and medical characteristics, dental needs, other alternative behavioral modalities, and quality of dental care. Although the benefits and importance of physical immobilization have been documented, the use of sedation, as discussed in Chapter 14, can reduce the amount of immobilization required. This is only one means of behavior control to achieve an adequate level of dental treatment.

The use of immobilization is indicated in the following situations:

- A patient requires diagnosis or treatment and cannot cooperate because of lack of maturity.
- A patient requires diagnosis or treatment and cannot cooperate because of mental or physical disabilities.
- A patient requires diagnosis or treatment and does not cooperate after other behavior management techniques have failed.
- The safety of the patient or practitioner would be at risk without the use of protective immobilization.

Immobilization is contraindicated for a cooperative patient and a patient who cannot be safely immobilized because of underlying medical or systemic conditions. Immobilization should not be used as punishment and should not be used solely for the convenience of the staff. The patient's record should display an informed consent, the indications for use, the type of immobilization used, and the duration of application.

Common mechanical aids for maintaining the mouth in an open position are shown in Fig. 23-7. Padded and wrapped tongue blades are easy to use, disposable, and very inexpensive. Frequently, parents of a child with disabilities are given wrapped tongue blades or Open Wide* disposable mouth props to aid with home dental care. The Open Wide mouth prop has a durable foam core on the end of a tongue depressor. It is also easy to use, disposable, and available in two sizes, but it is slightly more expensive than wrapped tongue blades. The Molt Mouth Prop' can be very helpful in the management of a difficult patient for a prolonged period. It is made in both adult and child sizes, allows accessibility to the opposite side of the mouth, and operates on a reverse scissor action. Its disadvantages include the possibility of lip and palatal lacerations and luxation of teeth if it is not used correctly. Caution must be exercised to prevent injury to the patient, and the prop should not be allowed to rest on anterior teeth. The patient's mouth should not be forced beyond its natural limits because patient discomfort and panic will result, which will cause further resistance and perhaps airway compromise.

Rubber bite blocks can be purchased in various sizes to fit on the occlusal surfaces of the teeth and stabilize the mouth in an open position. The bite blocks should have floss attached for easy retrieval if they become dislodged in the mouth.

Body control is gained through a variety of methods and techniques. For children who are severely retarded or very young, parents and dental assistants can assist in the control of movements during dental procedures, as shown in Fig. 23-8. Usually, however, for a child who has severe mental retardation, better working conditions and a more predictable patient response are obtained through the combined use of psychologic management techniques, parental assistance, pharmacologic aids, and immobilization.

The following are commonly used for immobilization:

Body

Papoose Board
Triangular sheet
Pedi-Wrap
Beanbag dental chair insert
Safety belt
Extra assistant

Extremities

Posey straps
Velcro straps
Towel and tape
Extra assistant

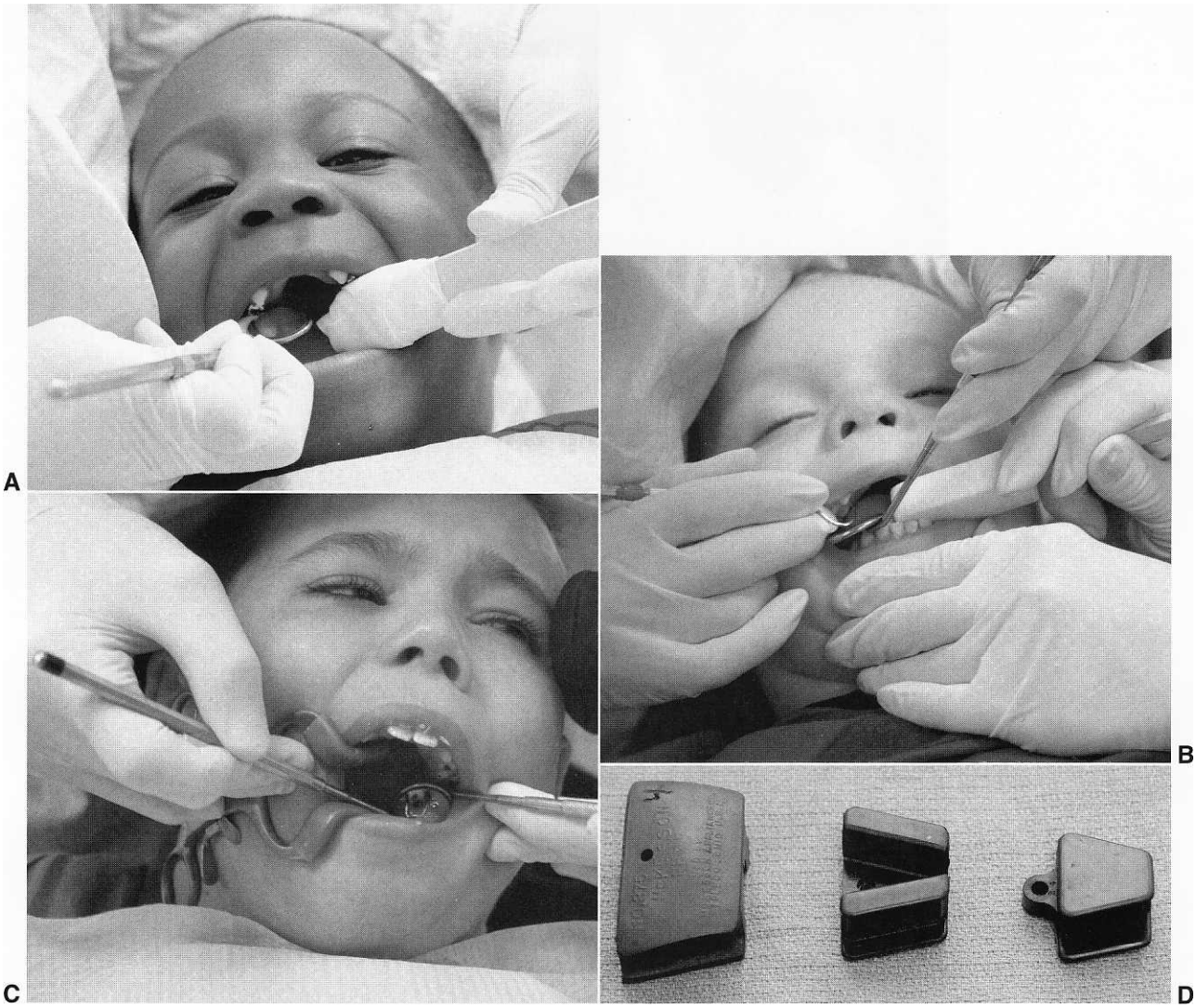
Head

Forearm-body support
Head positioner
Plastic bowl
Extra assistant

The Papoose Board* (Fig. 23-9) has several advantages. Simple to store and use, it is available in sizes to hold both large and small children. It has attached head stabilizers and is reusable. It does not always fit the contours of a dental chair, however, and sometimes a supporting pillow is needed. Because it covers the patient's diaphragm, a pretracheal stethoscope is necessary to monitor respiration if it is used in combination

'Specialized Care Co., Hampton, N.H. '
Hu-Friedy, Chicago, Ill.

*Olympic Medical Corp., Seattle, Wash.



f I G **23-7.** Physical aids to keep the patient's mouth open. **A,** Wrapped tongue blades. **B,** Open Wide disposable mouth prop in proper position. C, Molt Mouth Prop in proper position. D, McKesson bite blocks.

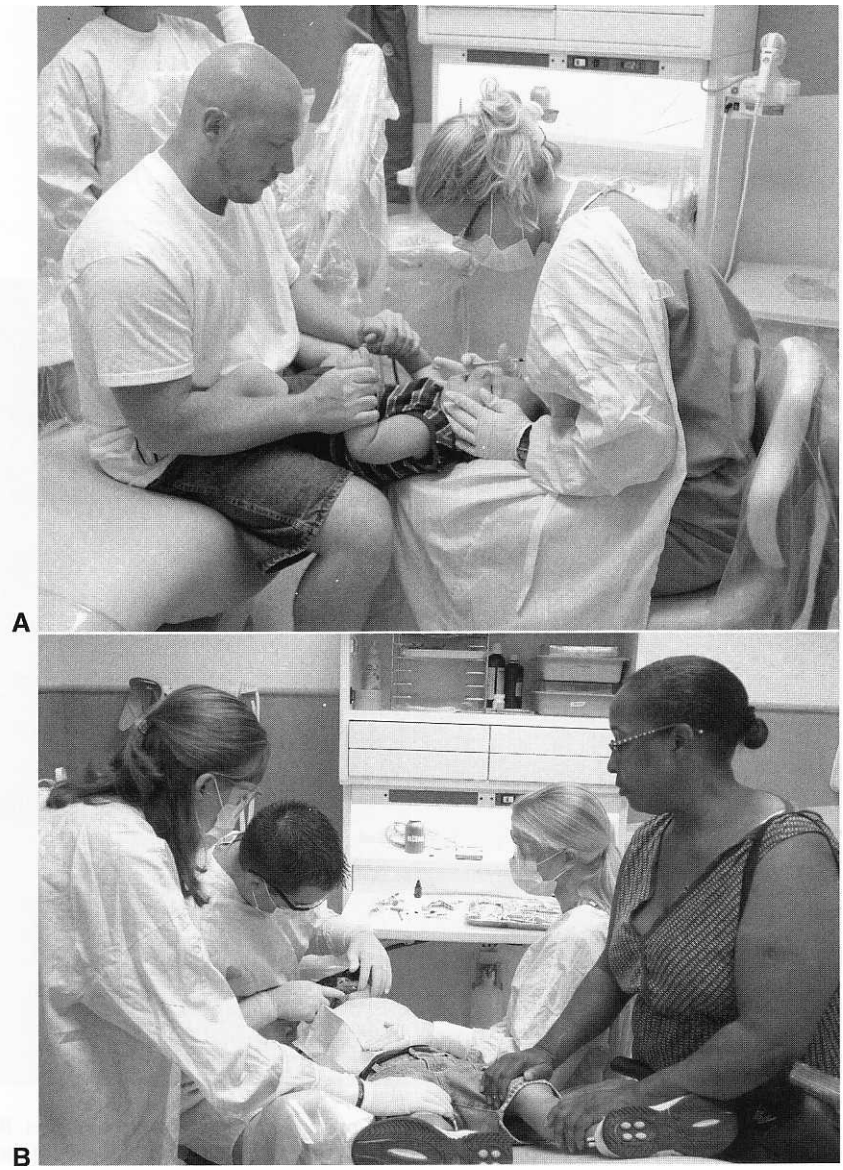
with sedation. An extremely resistant patient may develop hyperthermia if immobilized too long, and, of course, any restrained patient requires constant attendance and supervision.

Mink' describes the bedsheet (triangular sheet) technique and its use in controlling an extremely resistant child (Fig. 23-10, A). This method allows the patient to sit upright during radiographic examinations. Its disadvantages include the frequent need for straps to maintain the patient's position in the chair, the difficulty of its use with small patients, and the possibility of airway impingement should the patient slip downward unnoticed. Hyperthermia may be another problem during long periods of immobilization. Again, the need for constant supervision is emphasized so that these problems may be avoided.

The Rainbow Stabilizing System* (Fig. 23-10, B), which is available with or without the backboard, also comes in various sizes and allows some movement while still confining the patient. Its mesh fabric permits better ventilation, lessening the chances of the patient developing hyperthermia. It, too, requires straps to maintain body position in the dental chair and constant supervision to prevent the patient from rolling out of the chair.

The beanbag dental chair insert was developed to help comfortably accommodate hypotonic and severely spastic persons who need more support and less immobilization in a dental environment (Fig. 23-10, C). It is reusable and washable, and one size fits most people. Many patients with physical disabilities relax more in this setting.

*Specialized Care Co., Hampton, NH.



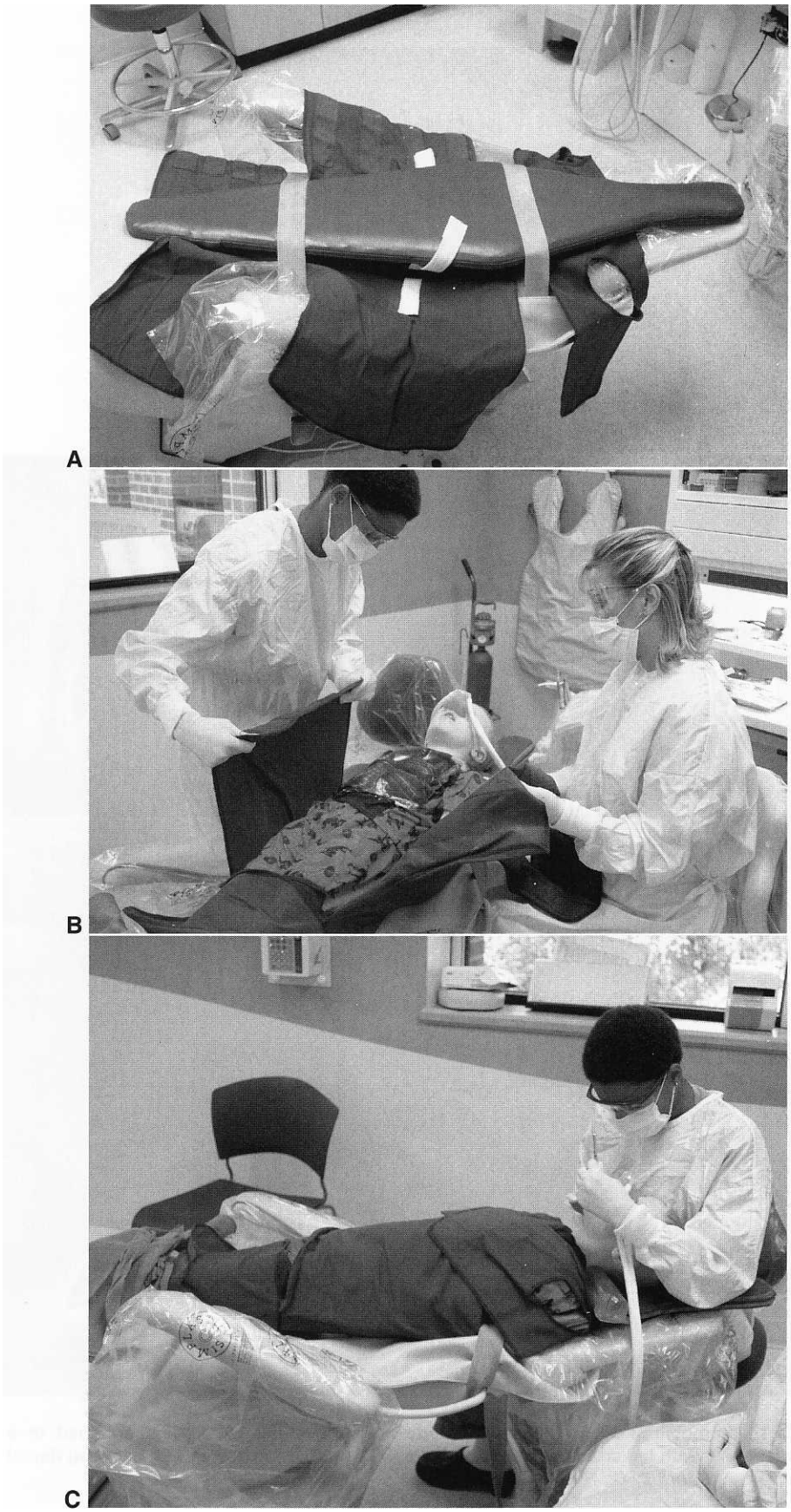
f **I C 23-8.** Assistance for immobilization. **A,** Parental aid during an examination. **B,** Additional assistance during the dental procedure.

The child's arms and legs can be immobilized with help from the parent or the dental assistant, with Posey straps,* or with a towel and adhesive tape (Fig. 23-11). If movement of the extremities is the only problem, having a dental assistant restrain the child is very helpful. Posey straps fasten to the arms of the dental chair and allow limited movement of the patient's forearm and hand. This limited movement frequently prevents overaction by resistant or combative patients. Wrapping a towel wrapped around the patient's forearms and fastening it with adhesive tape (without impeding circulation) is often helpful for an athetoid-spastic cerebral palsy patient who tries desperately, but without success, to control body movements. Immobilization actually encourages relaxation and prevents undesired reflexes by keeping the patient's arms in the midline of the body.

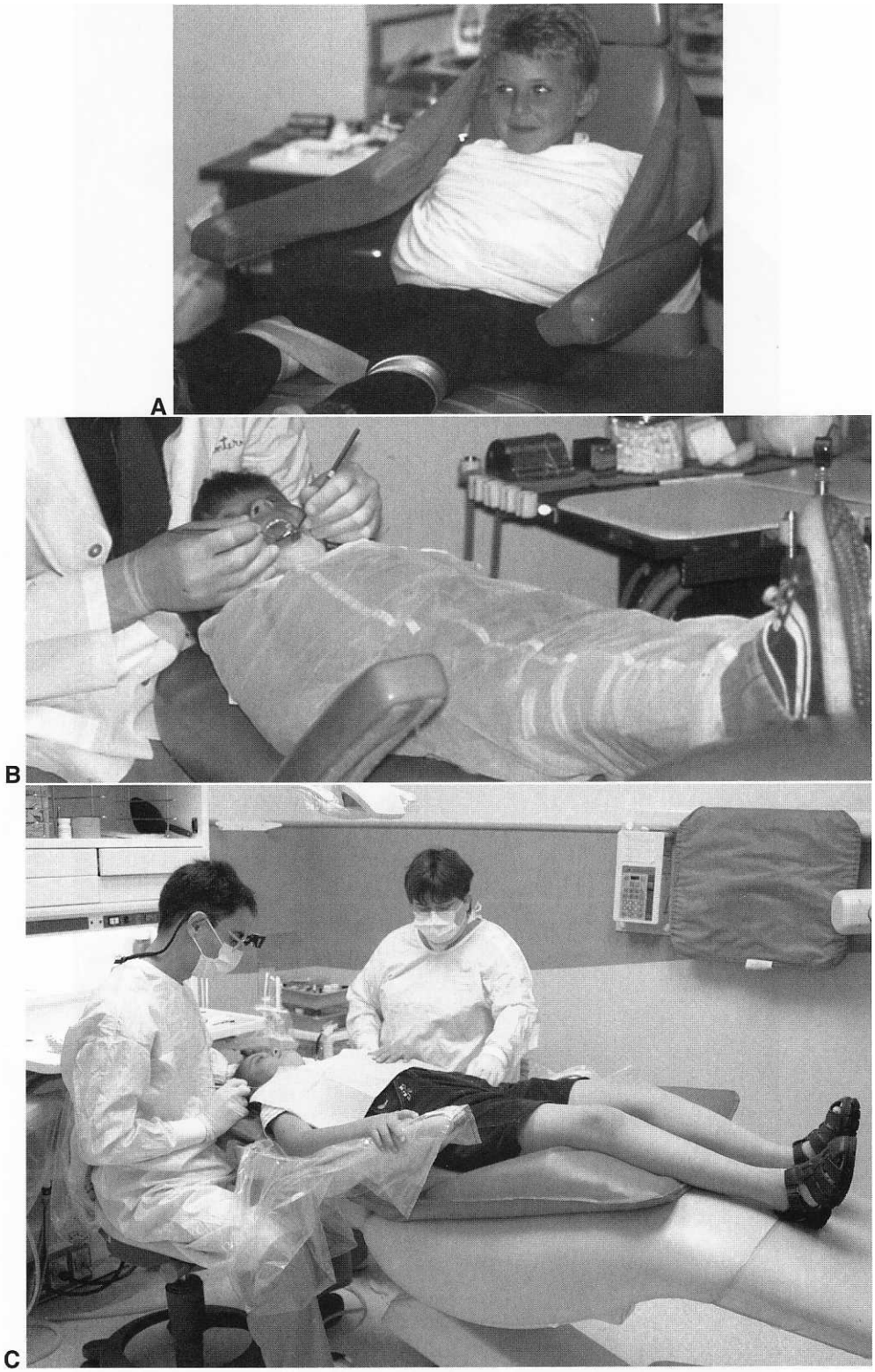
A patient's head position can usually be successfully maintained through the use of forearm-body pressure by the dentist. Other options include presence of an additional assistant to stabilize the child's head or use of a Papoose Board head positioner or a plastic bowl (doggie bowl) to provide position guidance (Fig. 23-12).

An explanation of the benefits of this management should be presented by the dentist before use if communication with the patient is possible. The mouth prop

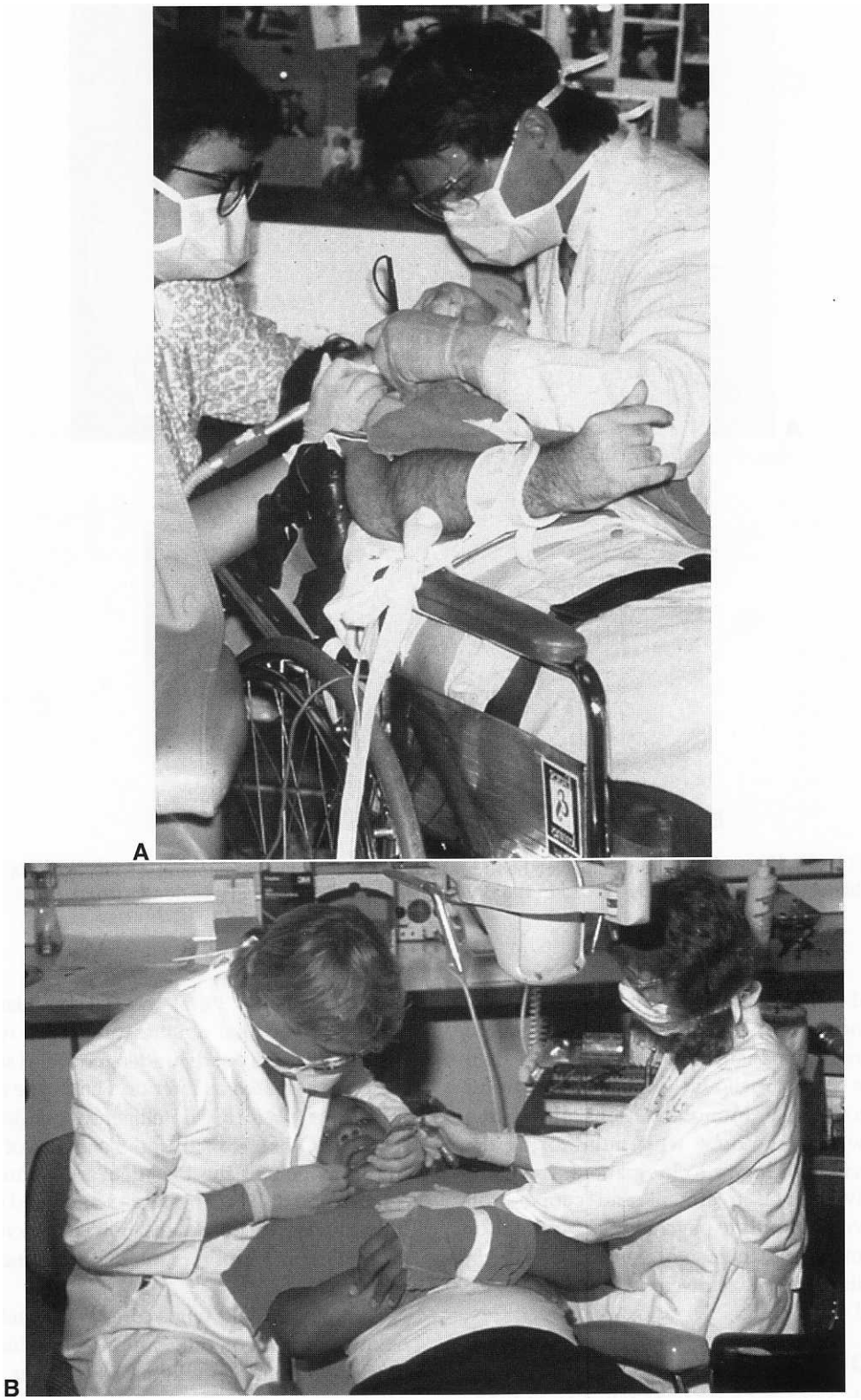
ST. Posey Co., Arcadia, Calif.



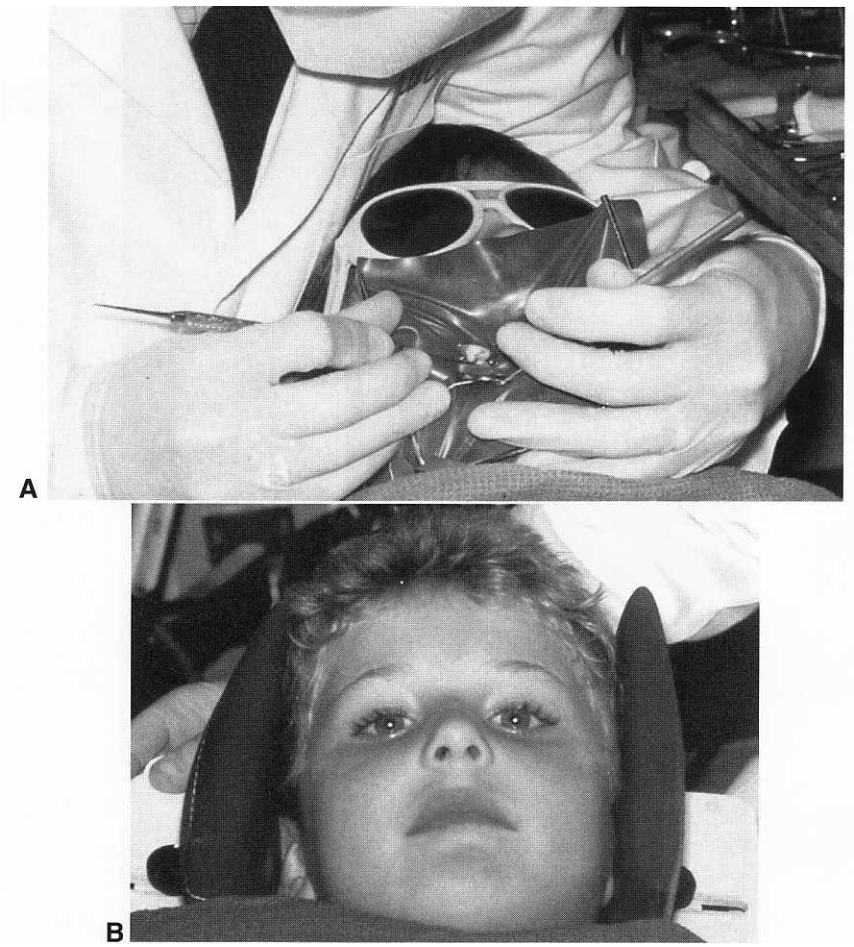
[I G **23-9.** A, The Olympic Papoose Board secured to a dental chair. **B,** Patient being placed in Papoose Board. C, Papoose Board in use.



FIC **23-10**. Immobilization for control of body and extremities. A, Patient confined in a triangular sheet with leg straps. **B,** Patient in a Pedi-Wrap. C, Patient lying in a beanbag dental chair insert. *(C Courtesy Dr. Priscilla John Bond.)*



GIG. **23-11.** Immobilization for the extremities. A, Posey strap on wrist. **B,** Towel and tape on forearm.



[I G **23-12.** Immobilization aids for head stabilization. **A,** Proper positioning of the dentist's hands, forearm, and body. **B,** Use of the Olympic Papoose Board head positioner.

can be identified as a "tooth chair," the Pedi-Wrap as a " safety robe," and an immobilization strap as a "safety belt," which allows the patient to feel secure rather than threatened. The parents should be given a careful explanation about how immobilization allows the needed dental work to be done while minimizing the possibility of accidental injury to the patient. If a child requires extensive dental treatment and cooperation cannot be achieved by routine psychologic, physical, or pharmacologic measures, the use of general anesthesia in a controlled atmosphere, as discussed in Chapter 15, is recommended.

MENTAL DISABILITY

Mental disability is a general term used when an individual's intellectual development is significantly lower than average and his or her ability to adapt to the environment is consequently limited. The condition varies in severity and cause. A classification of mental disability is presented in Table 23-2. Mental disability has been identified in approximately 3% of the U.S. population. For many years the potential abilities of people with intellectual disabilities were poorly understood, and such individuals were often treated as inferior. They were described using the terms *idiot* (IQ [intelligence quotient] below 25), *imbecile* (IQ of 25 to 50), and *moron* (IQ of 50 to 70). With the formation of the President's Committee on Mental Retardation in 1968, emphasis was placed on education of individuals with mental disabilities to increase their social and civic responsibilities, motor skills, and independence within society.

Although a child who scores two standard deviations below the mean on the Stanford-Binet Intelligence Scale or the Wechsler Intelligence Scale for Children may have some degree of retardation, a diagnosis of mental disability is not made based on IQ alone. Both inadequate adaptive functioning and intellectual deficiency are required to fulfill a diagnosis of mental disability.

A child with mild mental retardation is one who, because of low intelligence, requires special supports in

TABLE 23-2 Classification of Mental Retardation

DEGREE OF MENTAL DISABILITY	SB-IV*	WISC-III†	COMMUNICATION	SPECIAL REQUIREMENTS FOR DENTAL CARE
Mild	67-52	69-55	Should be able to speak well enough for most communication needs	Treat as normal child; mild sedation or nitrous oxide oxygen analgesia may be beneficial
Moderate	51-36	54-40	Has vocabulary and language skills such that the child can communicate at a basic level with others	Mild to moderate sedation may be beneficial; use restraints and positive reinforcement; general anesthesia may be indicated in cases of severe, generalized, dental decay
Severe or profound	35 and below	39 and below	Mute or communicates in grunts; little or no communication skills	Same as for moderately retarded

*Stanford-Binet Intelligence Scale (ed 4). '
Wechsler Intelligence Scale for Children (ed 3).

the school environment. In the academic environment these children may be eligible for special education services for students with mild mental handicap or disability. Educational programs for such children are generally simplified versions of regular school programs and usually lead to literacy and attainment of skills necessary for employment. Most children in this group, which accounts for approximately 80% of all persons with mental disability, will function acceptably as adults.

Children who are capable of some education and partial independence but who are not expected to experience full independence as adults may be eligible for special education services for students with moderate mental handicap or disability. Classroom activities may focus on attainment of daily living skills. Classrooms are often designed and furnished like a home, and the curriculum includes dressing, grooming, cooking, table setting, feeding, and cleaning. Individuals with moderate mental handicap or disability can be expected to master many vocational, leisure, and self-help skills within a supportive environment with trained personnel who help them with problems with which they may not be able to cope on their own.

A child with severe or profound mental disability may present a significant challenge. These individuals may participate in special education programs as students with severe to profound mental handicap or disability. A student with severe to profound mental handicap or disability can achieve success with self-help, leisure, and some vocational skills given sufficient training and support. Group home placement can allow individuals with severe to profound mental handicap or disability a measure of independence while providing sufficient support for safety and continuing reinforcement.

DENTAL TREATMENT OF A PERSON WITH MENTAL DISABILITY

Providing dental treatment for a person with mental disability requires adjusting to social, intellectual, and emotional delays. A short attention span, restlessness, hyperactivity, and erratic emotional behavior may characterize patients with mental disability undergoing dental care. The dentist should assess the degree of mental disability by consulting the patient's physician or other caregiver if the patient does not live with the parents.

The following procedures have proved beneficial in establishing dentist-patient rapport and reducing the patient's anxiety about dental care:

1. Give the family a brief tour of the office before attempting treatment. Introduce the patient and family to the office staff. This will familiarize the patient with the personnel and facility and reduce the patient's fear of the unknown. Allow the patient to bring a favorite item (stuffed animal, blanket, or toy) to hold for the visit.
2. Be repetitive; speak slowly and in simple terms. Make sure explanations are understood by asking the patient if there are any questions. If the individual has an alternative communication system, such as a picture board or electronic device, be sure it is available to assist with dental explanations and instructions.
3. Give only one instruction at a time. Reward the patient with compliments after the successful completion of each procedure.

4. Actively listen to the patient. People with mental disability often have trouble with communication, and the dentist should be particularly sensitive to gestures and verbal requests.
5. Invite the parent into the operatory for assistance and to aid in communication with the patient.
6. Keep appointments short. Gradually progress to more difficult procedures (e.g., anesthesia and restorative dentistry) after the patient has become accustomed to the dental environment.
7. Schedule the patient's visit early in the day, when the dentist, the staff, and the patient will be less fatigued.

With adequate preparation the dentist and the staff can provide a valuable service. By thoroughly understanding the patient's degree of mental disability and abilities and by exercising patience and understanding, the dentist should have no significant problems in delivering dental care.

DOWN SYNDROME (TRISOMY 21 SYNDROME)

Down syndrome is the best-known chromosomal disorder and is caused by the presence of three copies of chromosome 21. Medical conditions that occur more frequently in infants and children with Down syndrome and increase the mortality of these individuals include cardiac defects, leukemia, and upper respiratory infections. The incidence of congenital cardiac defects is about 40%, and because of these patients' high susceptibility to periodontal disease, knowledge of a heart condition is essential for dental treatment. Children with Down syndrome have a ten- to twentyfold greater incidence of leukemia during infancy compared with the general population. This increased incidence of leukemia is not maintained later in life.

Skeletal findings are an underdeveloped midface creating a prognathic occlusal relationship. Oral findings include mouth breathing, open bite, appearance of macroglossia, fissured lips and tongue, angular cheilitis, delayed eruption times, missing and malformed teeth, oligodontia, small roots, microdontia, crowding, and low level of caries. Children with Down syndrome experience a high incidence of rapid, destructive periodontal disease, which may be related to local factors such as tooth morphology, bruxism, malocclusion, and poor oral hygiene. Certain systemic factors are also believed to contribute to periodontal disease, including poor circulation, decreased humoral response, general physical deterioration at an early age, and genetic influences. Bell, Kaidonis, and Townsend[5] reported that severity of tooth wear (both attrition and erosion) was significantly greater in children with Down syndrome than in children without the syndrome.

Many children with Down syndrome are affectionate and cooperative, and dental procedures can be provided without compromise if the dentist works at a slightly slower pace. Emphasis should be placed on preventive dental care with frequent follow-up visits to monitor oral hygiene. Comprehensive dental care is an overall goal with alteration based on the individual's level of functioning. Light sedation and immobilization may be indicated in those children who are moderately apprehensive. Severely resistive patients may require general anesthesia.

Delayed tooth eruption frequently occurs in children with Down syndrome. For this reason, additional discussion of this disorder is presented in Chapter 9.

LEARNING DISABILITIES

For years, students who consistently had difficulty achieving a level of academic performance concomitant with their intellectual capacity were unfortunately labeled as *retarded.* Today the term *learning disabled is* applied to children who exhibit a disorder in one or more of the basic psychologic processes involved in understanding or using spoken or written language. Learning disabilities affect between 3% and 15% of the population. They occur four times more frequently among boys than among girls.

In 1968 the National Advisory Committee on Handicapped Children of the U.S. Department of Health, Education, and Welfare stated that learning disabilities " may be manifested in disorders of listening, thinking, talking, reading, writing, spelling or arithmetic. They include conditions that have been referred to as perceptual handicaps, brain injury, minimal brain dysfunction, dyslexia, and developmental aphasia."[5] It should be emphasized that learning disabilities do not include learning problems caused mainly by hearing, visual, or motor handicaps, mental disability, emotional disturbance, or environmental conditions.

The cause of learning disabilities remains unclear. Physiologic factors, such as minimal brain injury or damage to the central nervous system, have been implicated. Because learning disabilities appear more frequently in some families than in others, a genetic factor has also been suggested. The possibility exists that severe emotional disturbances can develop as a result of learning disabilities. This potential has prompted early diagnosis and treatment of affected persons.

Most children with learning disabilities accept dental care and cause no unusual management problems for the dentist. If a child is resistant, behavioral management and conscious sedation techniques can be used with success.

FRAGILE X SYNDROME

Fragile X syndrome is a common inherited form of mental disability and autism. It accounts for 30% to 50% of cases of X-linked mental disability. The defect is an

abnormal gene on the terminal portion of the long arm of an X chromosome. Males are more vulnerable because they have only one X chromosome and are more significantly affected than females. Numerous studies have investigated fragile X syndrome in males, but fragile X syndrome in females has not been investigated as extensively because the physical and cognitive deficits in females are usually less severe.

A history of developmental delay and hyperactivity, and physical features such as prominent ears, long face, prominent jaw, high arched palate, flattened nasal bridge, hyperextensible joints, flat feet, cardiac murmur, simian creases of the palms, and postadolescent macroorchidism in males should be considered potential indicators for fragile X syndrome. Behavioral features such as hand slapping, hand biting, and poor eye contact are frequently seen. Fragile X syndrome may be diagnosed in individuals with another diagnosis such as Down syndrome or cerebral palsy.

Treatment of children with fragile X syndrome is multidisciplinary, and speech, language, and occupational therapy are required to address the cognitive, language, and sensory integration problems. Medical intervention can be useful in decreasing the hyperactivity and improving the attention span. Females with fragile X syndrome have a more favorable outcome with appropriate intervention than males with fragile X syndrome.

Mode of dental treatment depends on the level of developmental delay, cognitive ability, and degree of hyperactivity. Those with mild cases may be treated by scheduling short appointments and using immobilization and/or conscious sedation. Severely affected individuals must be treated in the operating room under general anesthesia.

FETAL ALCOHOL SYNDROME

More than 5000 babies are born annually with fetal alcohol syndrome. The consumption of one to three drinks a day during the first 2 months of pregnancy can result in significant damage to the developing baby. Alcohol consumption can cause multiple problems, including neurologic abnormalities, cardiac defects, growth retardation, and cognitive defects. Physical findings include microcephaly; bilateral ptosis; a short, depressed midface; a flat nasal bridge; a short philtrum; and a thin upper lip. Cleft palate, micrognathic mandible, or a relative prognathism of both jaws may be seen as well.

Most of the dental problems associated with fetal alcohol syndrome in children are related to the high incidence of dental and skeletal malocclusions. As young children (younger than age 5), these individuals tend to be hyperactive. As they become older, these children are usually cooperative and relatively easy to treat.

AUTISM

Autism is an incapacitating disturbance of mental and emotional development that causes problems in learning, communicating, and relating to others. This lifelong developmental disability manifests itself during the first 3 years of life, is difficult to diagnose, and has no cure. In the past, autism was believed to be an emotional disability. The *Diagnostic and Statistical Manual of Mental Disorders,* published by the American Psychiatric Association, has listed autism as a neurologic disorder. It is now believed to be caused by a physical disorder of the brain.

Autism occurs in approximately 5 of every 10,000 births and is four times more common in boys than in girls. Children with autism look like unaffected children and have normal life spans. However, they have a limited capacity to communicate, socialize, and learn. Approximately 60% of persons diagnosed with autism have IQ scores below 50, 20% have scores between 50 and 70, and only 20% have scores greater than 70. Special educational programs that use behavior modification techniques designed for the specific individual have proved helpful in training children with autism. Counseling the family on effective management techniques in the home is essential in the overall training of persons with autism.

Children with autism have multiple medical and behavioral problems that make dental treatment difficult. These children often have poor muscle tone, poor coordination, drooling, a hyperactive knee jerk, and strabismus, and 30% eventually develop epilepsy. Children with autism may have strict routines and prefer soft foods and sweetened foods. Because of poor tongue coordination, children with autism tend to pouch food instead of swallowing. This habit, combined with the desire for sweetened foods, leads to increased susceptibility to caries.

Because of their tendency to adhere to routines, children with autism may require several dental visits to acclimate to the dental environment. The use of a Papoose Board or Pedi-Wrap and preappointment conscious sedation may be necessary and in some instances has a calming effect on the child.

CEREBRAL PALSY

Cerebral palsy is one of the primary handicapping conditions of childhood. The incidence of cerebral palsy in the United States, for all ages, is 1.5 to 3 cases per 1000 individuals. One newborn in approximately 200 live births will be affected with this condition. Cerebral palsy is not a specific disease entity but rather a collection of disabling disorders caused by insult and permanent damage to the brain in the prenatal and peri-natal periods, during which time the central nervous system is still maturing. This disability might involve

muscle weakness, stiffness, or paralysis; poor balance or irregular gait; and uncoordinated or involuntary movements.

Although many recognized conditions result in damage to the motor centers of the brain, in at least one third of the cases of cerebral palsy no discernible cause is found. It has been well established that any factor contributing to decreased oxygenation of the developing brain can be responsible for brain damage. In addition, causal relationships have been established between cerebral palsy and complications of labor or delivery; infections of the brain, such as meningitis and encephalitis; toxemias of pregnancy; congenital defects of the brain; kernicterus; poisoning with certain drugs and heavy metals; and accidents resulting in trauma to the head. There is a high correlation between premature birth and cerebral palsy (Approximately one third of all infants born prematurely have a demonstrable nervous system abnormality.)

There are various types of cerebral palsy, which are distinguished according to the neuromuscular dysfunctions observed and the extent of anatomic involvement. Some persons may have almost imperceptible symptoms. Others are severely disabled, with no appreciable use of the muscles of their limbs and other voluntary muscles. It is imperative to keep in mind that two patients with the same type of cerebral palsy may show very disparate symptoms. The following terms are commonly used to designate involved areas of the body:

1. Monoplegia—involvement of one limb only
2. Hemiplegia—involvement of one side of the body
3. Paraplegia—involvement of both legs only
4. Diplegia—involvement of both legs with minimum involvement of both arms
5. Quadriplegia—involvement of all four limbs

The following outline provides a classification of cerebral palsy according to the type of neuromuscular dysfunction and lists a few of the basic characteristics of each type:

I. *Spastic* (approximately 70% of cases)
   A. Hyperirritability of involved muscles, resulting in exaggerated contraction when stimulated
   B. Tense, contracted muscles (e.g., in spastic hemiplegia, which affects one third of all children with cerebral palsy, the hand and arm are flexed and held in against the trunk. The foot and leg may be flexed and rotated internally, which results in a limping gait with circumduction of the affected leg.)
   C. Limited control of neck muscles, which results in head roll
   D. Lack of control of the muscles supporting the trunk, which results in difficulty in maintaining upright posture
   E. Lack of coordination of intraoral, perioral, and masticatory musculature; possibility of impaired chewing and swallowing, excessive drooling, persistent spastic tongue thrust, and speech impairments

II. *Dyskinetic* (athetosis and choreoathetosis) (approximately 15% of cases)
   A. Constant and uncontrolled motion of involved muscles
   B. Succession of slow, twisting, or writhing involuntary movements (athetosis) or quick, jerky movements (choreoathetosis)
   C. Frequent involvement of neck musculature, which results in excessive movement of the head (Hypertonicity of these muscles may cause the head to be held back, with the mouth constantly open and the tongue positioned anteriorly or protruded.)
   D. Possibility of frequent, uncontrolled jaw movements, causing abrupt closure of the jaws or severe bruxism
   E. Frequent hypotonicity of perioral musculature with mouth breathing, tongue protrusion, and excessive drooling
   F. Facial grimacing
   G. Chewing and swallowing difficulties
   H. Speech problems

III. *Ataxic* (approximately 5% of cases)
   A. Inability of involved muscles to contract completely, so that voluntary movements can be only partially performed
   B. Poor sense of balance and uncoordinated voluntary movements (e.g., stumbling or staggering gait or difficulty in grasping objects)
   C. Possibility of tremors and an uncontrollable trembling or quivering on attempting voluntary tasks

IV. *Mixed* (approximately 10% of cases)
   A. Combination of characteristics of more than one type of cerebral palsy (e.g., mixed spastic-athetoid quadriplegia)

Two additional forms of cerebral palsy have been described but occur very infrequently. In hypotonia the muscles are flaccid (i.e., there is an inability to elicit muscle activity on volitional stimulation). In rigidity the muscles are in a constant state of contraction. The condition is characterized by prolonged periods in which the muscles of the extremities or trunk remain rigid, resisting any effort to move them.

In many patients with cerebral palsy, certain neonatal reflexes may persist long after the age at which they

normally disappear. These primitive reflexes are usually modified or are progressively replaced as the subcortical dominance of the infant's behavior is suppressed by higher centers of the maturing central nervous system. Three of the most common reactions, which a dentist should recognize, are the following:

1. *Asymmetric tonic neck reflex*. If the patient's head is suddenly turned to one side, the arm and leg on the side to which the face is turned will extend and stiffen. The limbs on the opposite side will flex.
2. *Tonic labyrinthine reflex*. If the patient's head suddenly falls backward while the patient is supine, the back may assume the position known as postural extension; the legs and arms will straighten out, and the neck and back will arch.
3. *Startle reflex*. This reflex, which is frequently observed in persons with cerebral palsy, consists of sudden, involuntary, often forceful bodily movements. This reaction is produced when the patient is surprised by stimuli, such as sudden noises or unexpected movements by other people.

Because the motor involvement in cerebral palsy results from irreversible damage to the developing brain, other symptoms of organic brain damage may also be present. The fact that these other symptoms are frequently seen underscores the premise that cerebral palsy does not denote one specific disease entity. Rather, it is a complex of disabling conditions, the clinical manifestations of which depend on the extent and location of damage to the brain. The following are some common manifestations:

1. *Mental retardation.* Approximately 60% of persons with cerebral palsy demonstrate some degree of mental retardation.
2. *Seizure disorders.* Seizures are an accompanying condition in 30% to 50% of cases; they occur primarily during infancy and early childhood. Most seizures can be controlled with anticonvulsant medications.
3. *Sensory deficits or dysfunctions.* Impairment of hearing is more common than in the normal population, and eye disorders affect approximately 35% of persons with cerebral palsy. The most common visual defect is strabismus.
4. *Speech disorders.* More than half of patients with cerebral palsy have some speech problem—usually dysarthria, an inability to articulate well because of lack of control of the speech muscles.
5. *Joint contractures.* Persons with spasticity and rigidity demonstrate abnormal limb postures and contractures during growth and at maturity, primarily because of disuse of muscle groups.

No intraoral anomalies are unique to persons with cerebral palsy. However, several conditions are more common or more severe than in the general population. These conditions are as follows:

1. *Periodontal disease*. Periodontal disease occurs with great frequency in persons with cerebral palsy. Often the patient will not be physically able to brush or floss adequately. When oral hygiene measures must be provided for the person by another individual, they may be performed infrequently and inadequately. Diet may also be significant; children who have difficulty chewing and swallowing tend to eat soft foods, which are easily swallowed and are high in carbohydrates. Patients with cerebral palsy who take phenytoin to control seizure activity will generally have a degree of gingival hyperplasia.
2. *Dental caries.* The data are conflicting regarding the incidence of dental caries in patients with cerebral palsy compared with the incidence in the general population. Except among institutionalized patients, the incidence of caries does not seem to be significantly greater among persons with cerebral palsy.
3. *Malocclusions.* The prevalence of malocclusions in patients with cerebral palsy is approximately twice that in the general population. Commonly observed conditions include noticeable protrusion of the maxillary anterior teeth, excessive overbite and overjet, open bites, and unilateral crossbites. A primary cause may be a disharmonious relationship between intraoral and perioral muscles. Uncoordinated and uncontrolled movements of jaws, lips, and tongue are observed with great frequency in patients with cerebral palsy.
4. *Bruxism*. Bruxism is commonly observed in patients with athetoid cerebral palsy. Severe occlusal attrition of the primary and permanent dentition may be noted, with the resulting loss of vertical interarch dimension. Temporomandibular joint disorders may be sequelae of this condition in adult patients.
5. *Trauma.* Persons with cerebral palsy are more susceptible to trauma, particularly to the maxillary anterior teeth. This situation is related to the increased tendency to fall, along with a diminished extensor reflex to cushion such falls, and the frequent increased flaring of the maxillary anterior teeth. Susceptibilities also include aspiration and ingestion of a foreign body.

To an uninformed dentist, a person with cerebral palsy who has involuntary movements of the limbs and head might be perceived as an uncooperative and

unmanageable patient. In addition, patients who have unintelligible speech, uncontrollable jaw movements, and spastic tongue are often erroneously assumed to be intellectually delayed. A clinician who is not knowledgeable about cerebral palsy and other physically and mentally disabling conditions may feel uncomfortable about treating such patients and may refuse to do so.

In providing treatment for children with cerebral palsy, it is imperative that a dentist evaluate each patient thoroughly in terms of personal characteristics, symptoms, and behavior and then proceed as conditions and needs dictate.

The dentist should never make assumptions about the degree of a child's physical or mental impairments without first acquiring the facts. Taking a thorough medical and dental history is very important, and the parent or guardian should be interviewed before the initiation of any treatment. It may also be beneficial to consult the patient's physician regarding the patient's medical status.

A patient with cerebral palsy who has involuntary head movements may be cognizant of the need to minimize these movements while receiving dental care. Paradoxically, the patient's own endeavors to control these movements may only exacerbate the problem. Therefore it is imperative that all dental personnel be empathic about the fears and frustrations that such a person experiences. The importance of maintaining a calm, friendly, and professional atmosphere cannot be overemphasized.

The following suggestions are offered to the clinician as being of practical significance in treating a patient with cerebral palsy:

1. Consider treating a patient who uses a wheelchair in the wheelchair. Many patients express such a preference, and it is frequently more practical for the dentist. For a young patient, the wheelchair may be tipped back into the dentist's lap.
2. If a patient is to be transferred to the dental chair, ask about a preference for the mode of transfer. If the patient has no preference, the two-person lift is recommended.
3. Make an effort to stabilize the patient's head throughout all phases of dental treatment.
4. Try to place and maintain the patient in the midline of the dental chair, with arms and legs as close to the body as feasible.
5. Keep the patient's back slightly elevated to minimize difficulties in swallowing. (It is advisable not to have the patient in a completely supine position.)
6. On placing the patient in the dental chair, determine the patient's degree of comfort and assess the position of the extremities. Do not force the limbs into unnatural positions. Consider the use of pillows, towels, and other measures for trunk and limb support.
7. Use immobilization judiciously to control flailing movements of the extremities.
8. For control of involuntary jaw movements, choose from a variety of mouth props and finger splints. Patient preference should weigh heavily, since a patient with cerebral palsy may he very apprehensive about the ability to control swallowing. Such appliances may also trigger the strong gag reflex that many of these patients possess.
9. To minimize startle reflex reactions, avoid presenting stimuli such as abrupt movements, noises, and lights without forewarning the patient.
10. Introduce intraoral stimuli slowly to avoid eliciting a gag reflex or to make it less severe.
11. Consider the use of the rubber dam, a highly recommended technique, for restorative procedures.
12. Work efficiently and minimize patient time in the chair to decrease fatigue of the involved muscles.

SPINA BIFIDA AND LATEX ALLERGY

Although the etiology of spina bifida is unknown, it is thought to be the result of a genetic predisposition whose manifestation is triggered by the environment. There are two common forms of this neural tube defect, spina bifida occulta and myelomeningocele. Spina bifida occulta (i.e., closed) presents with skin covering an area where tissue protrudes through a bony cleft in the vertebral column. These children may develop foot weakness or bowel and bladder sphincter disturbances. Myelomeningocele (spina bifida aperta, i.e., open) is the most severe because the spinal cord, spinal fluid, and membranes protrude in a sac through the defect. These children can suffer from hydrocephalus, paralysis, orthopedic deformities, and genitourinary abnormalities. Taking folic acid during the first 6 weeks of pregnancy can prevent over 50% of neural tube defects.

Children with neural tube defects are at higher risk for caries secondary to poor hygiene, poor nutritional intake, and long-term drug therapy. They are also at higher risk for latex allergy because they are frequently exposed to latex as a result of undergoing procedures in which latex products are used. Therefore, Nettis et al' recommend that all patients be screened for conditions such as spina bifida and exposure to recurrent surgical procedures, and for a history of atopy, cross-reactive food allergies (i.e., allergies to banana, avocado, kiwi, and chestnuts, which may sensitize allergic patients to latex exposure), and previous reactions to natural rubber latex.

For all patients with a latex allergy or latex allergy risk factors, all equipment that comes in intimate contact with the patient should be made of nonlatex

substitutes. Nettis et al' suggest avoiding handling of nonlatex products while wearing latex gloves or with unwashed hands to prevent the transfer of latex allergens to nonlatex products. They suggest that the ideal time to schedule dental appointments for such individuals is at the beginning of a working session, such as in the morning or after a vacation when the office has been closed. This will allow for settling of airborne latex particles. Another good scheduling time is after the office has been professionally vacuumed and cleaned to remove latex-tainted cornstarch. Mild irritant reactions to latex can be managed with immediate removal of the rubber object and administration of an antihistamine. However, systemic reactions may require immediate treatment with drugs such as adrenaline and may necessitate emergency resuscitation.

RESPIRATORY DISEASES

ASTHMA (REACTIVE AIRWAY DISEASE)

Asthma is a very common childhood disease, affecting 1 in 10 children. Although often thought of as acute respiratory distress brought on by environmental factors, asthma is a chronic airway disease characterized by inflammation and bronchial constriction.

Asthma is a diffuse obstructive disease of the airway caused by edema of the mucous membranes, increased mucous secretions, and spasm of smooth muscle. It is twice as common in prepubertal boys but affects both sexes equally during adolescence and adulthood. The etiology includes biochemical, immunologic, infectious, endocrine, and psychologic factors. The typical symptoms of asthma are coughing, wheezing, chest tightness, and dyspnea. The clinical onset of an episode may occur over minutes (acute) or hours and days. An acute attack is associated with exposure to irritants such as cold air, fumes, or dust, and it may develop in minutes. An attack developing over days is usually precipitated by a viral respiratory infection. Severe bronchial obstruction results in labored breathing, wheezing, tachypnea, profuse perspiration, cyanosis, hyperventilation, tachycardia, and sometimes chest pain. A dental procedure constitutes an acute irritant to the airways of the asthmatic child and may precipitate an attack.

Fortunately, three fourths of childhood asthma is mild, with minimal daily symptoms and short-lived exacerbations. Before initiating dental treatment, the dentist should know what are the frequency and severity of the attacks, what are the triggering agents, when the patient was hospitalized and/or in the emergency department, when the last attack occurred, what medications the patient takes, and what limitations on activity the patient may have. Patients taking systemic corticosteroids and those who were hospitalized or in the emergency department in the past year should be treated with caution because they are at higher risk of morbidity and mortality. Sometimes, deferring the dental visit until the patient's asthma is controlled is the best approach.

Patients who use bronchodilators should take a dose before their appointment, and they should bring their inhalers or nebulizers into the dental office in case trouble arises. Acute symptoms may be prevented by the use of the child's bronchodilator (inhaled $_{12}$ receptor agonist such as albuterol or terbutaline sulfate). Behavioral methods are employed to reduce anxiety, and the use of nitrous oxide–oxygen analgesia may be helpful. Hydroxyzine hydrochloride (Vistaril) and diazepam (Valium) have been successful in alleviating anxiety. Barbiturates and narcotics are not indicated because of their potential for histamine release leading to a bronchospasm. Aspirin compounds and nonsteroidal antiinflammatory agents are contraindicated because about 4% of patients experience wheezing after taking these drugs. Acetaminophen is recommended. Positioning a child with mild asthmatic symptoms in an upright or semiupright position for the dental procedure may be beneficial.

Oral findings of children with moderate to severe asthma include higher caries rate, decreased salivary rate, increased prevalence of oral mucosal change charac teristic of chronic mouth breathers, and increased levels of gingivitis. Increased incidence of orofacial abnormalities such as high palatal vault, more posterior crossbites, greater overjets, and increased facial height is also seen.

Dental goals are similar to those for other patients, with care taken to avoid the potential for dental materials and products to exacerbate the asthma. The patient's pulmonary function, propensity for developing an attack, immune status, and adrenal status should be evaluated prior to dental treatment. Emergency treatment for a person with asthma who is in respiratory distress requires discontinuing the dental procedure, reassuring the patient, and opening the airway. Administer 100% oxygen while placing the patient in an upright or comfortable position. Keeping the airway open, administer the patient's [3_2 agonist with an inhaler or nebulizer. If there is no improvement, administer subcutaneous epinephrine (0.01 mg/kg of 1:1000 solution) and obtain medical assistance immediately.

BRONCHOPULMONARY DYSPLASIA

Bronchopulmonary dysplasia is a chronic lung disease usually resulting from the occurrence during infancy of respiratory distress syndrome that requires prolonged ventilation with a high concentration of inspired oxygen. Chronic lung changes are more likely to occur in the premature infant. The incidence is 80% to 90% in infants weighing less than 1000 g at birth. With the increased survival of low-birth-weight infants, the prevalence of

bronchopulmonary dysplasia has increased. The lung pathology of children with bronchopulmonary dysplasia shows evidence of bronchial ulceration, necrosis with plugging of bronchiolar lumina, and inflammatory cells. This bronchiolar injury compromises further lung development. Inflammatory changes and bronchiolar fibrosis lead to increased airway resistance and contribute to the hypoxemia seen in infants with bronchopulmonary dysplasia. Some children with bronchopulmonary dysplasia develop right ventricular hypertrophy (cor pulmonale). Other significant pulmonary complications include tracheal stenosis, upper airway obstruction secondary to subglottic cysts, and hoarseness because of partial or complete vocal cord paralysis. About 20% of infants with bronchopulmonary dysplasia die within the first year of life. The major causes of death are cor pulmonale, respiratory infections, and sudden death.

An increased oxygen supply must be provided to prevent hypoxic pulmonary vasoconstriction and to decrease the work of breathing. The nasal cannula provides continuous oxygen delivery, which results in fewer fluctuations in oxygen tension. Weaning off oxygen is possible with improved lung function and lung size. Children who develop cor pulmonale may require diuretic therapy to prevent congestive heart failure.

Dental care for children with bronchopulmonary dysplasia requires more chair time than usual. These children often spend a significant part of their early lives in the hospital and exhibit significant oral defensiveness.

After the initial dental evaluation, consultation with a pulmonologist is beneficial to plan safe dental treatment for the patient. If the dental patient is taking oxygen continuously via a nasal cannula, short appointments with frequent breaks are necessary to prevent the development of pulmonary vasoconstriction. Parents of children with bronchopulmonary dysplasia may need to provide additional oral hygiene for their children when these children are required to eat frequent small meals to maintain the proper caloric intake. Any non-emergent dental care should be avoided when the patient is not doing well medically.

CYSTIC FIBROSIS

Cystic fibrosis is an autosomal recessive disorder occurring in 1 of every 2000 births. It is the most common lethal genetic disorder affecting whites. The genetically altered protein affects exocrine gland function. The defective exocrine gland function leads to micro-obstruction of the pancreas, which results in cystic degeneration of the pancreas and ultimately a digestive enzyme deficiency producing malabsorption of nutrients.

The defective gene products cause abnormal water and electrolyte transport across epithelial cells, which results in a chronic disease of the respiratory and gastrointestinal system, elevated levels of electrolytes in sweat, and impaired reproductive function.

In the lungs, retention of mucus occurs, which causes obstructive lung disease and increased frequency of infections. As the progressive lung disease develops, there is an increase in chest diameter, clubbing of the fingers and toes, decreased exercise tolerance, and a chronic productive cough. Before advances in antibiotic therapy, physical therapy, and nutritional supplementation, these individuals rarely survived childhood. The median life expectancy has been increased to 31 years. Death is most frequently the result of pneumonia and anoxia after a long period of respiratory insufficiency. Cystic fibrosis–related diabetes is becoming more common as patients live longer.

Children with cystic fibrosis have a high incidence of tooth discoloration when systemic tetracyclines are taken during tooth formation. With the advent of alternative antibiotics the incidence of tooth discoloration is decreasing. The incidence of dental caries in children with cystic fibrosis is low secondary to long-term antibiotic therapy, buffering capacity of excess calcium in the saliva, and pancreatic enzyme replacement therapy. There is a high incidence of mouth breathing and open-bite malocclusion associated with chronic nasal and sinus obstruction. Patients with cystic fibrosis may prefer to be treated in a more upright position to allow them to clear secretions more easily. The use of sedative agents that interfere with pulmonary function should be avoided, and the patient's physician should be contacted before using nitrous oxide—oxygen sedation in a patient exhibiting evidence of severe emphysema.

HEARING LOSS

Hearing loss (deafness) is a disability that is often overlooked because it is not obvious. Total hearing loss affects 1.8 million people, and there are 14 million hearing-impaired individuals in the United States. About 1 in 600 neonates has a congenital hearing loss. During the neonatal period, many more acquire hearing loss from other associated conditions. Almost inevitably, speech is affected. If an impairment is severe enough that dentist and child cannot communicate verbally, the dentist must use sight, taste, and touch to communicate and to allow the child to learn about dental experiences. Table 23-3 shows how speech and psychologic problems relate to various degrees of hearing loss. Many times, mild hearing losses are not diagnosed, which leads to management problems because of the child's misunderstanding of instructions; children with more severe hearing losses already have psychologic and social disturbances that make dental behavior management more complex. Parents may suspect profound hearing loss if their infant does not respond to ordinary sounds or voices. Early identification and correction of hearing loss is essential for normal

TABLE 23-3 Implications of Auditory Disability Relative to International Standards Organization (ISO) Reference Levels*

ISO (DB)	DISABILITY	SPEECH COMPREHENSION	PSYCHOLOGIC PROBLEMS IN CHILDREN
0	Insignificant	Little or no difficulty	None.
>25	Slight	Difficulty with faint speech; language and speech development within normal limits	Child may show a slight verbal deficit.
>40	Mild-moderate	Frequent difficulty with normal speech at 3 feet (91.4 cm); language skills are mildly affected	Psychologic problems can be recognized.
>55	Marked	Frequent difficulty with loud speech at 3 feet (91.4 cm); difficulty understanding with hearing aid in school situation	Child is likely to be educationally retarded, with more pronounced emotional and social problems than in children with normal hearing.
>70	Severe	May understand only shouts or amplified speech at 1 foot (30.5 cm) from ear	The prelingually deaf show pronounced educational retardation and evident emotional and social problems.
>90	Extreme	Usually no understanding of speech even when amplified; child does not rely on hearing for communication	The prelingually deaf usually show severe educational retardation and emotional underdevelopment.

Adapted from Goetzinger CP: The psychology of hearing impairment. In Katz J, editor: *Handbook of clinical audiology,* ed 2, Baltimore, 1978, Williams & Wilkins.

*Reference levels are in decibels relative to threshold in young patients with normal hearing.

development of communication skills. No abnormal dental findings are associated with hearing loss. The following are known causes of hearing loss:

Prenatal factors

Viral infections, such as rubella and influenza
Ototoxic drugs, such as aspirin, streptomycin, neomycin, kanamycin
Congenital syphilis
Heredity disorders (e.g., Alport, Arnold-Chiari, Crouzon, Hunter, Klippel-Feil, Stickler, Treacher Collins, and Waardenburg syndromes)

Perinatal factors
Toxemia late in pregnancy
Prematurity
Birth injury
Anoxia
Erythroblastosis fetalis

Postnatal factors

Viral infections, such as mumps, measles, chickenpox, influenza, poliomyelitis, meningitis
Injuries
Ototoxic drugs, such as aspirin, streptomycin, neomycin, kanamycin

The following should be considered when treating a hearing-impaired patient:

1. Prepare the patient and parent before the first visit with a welcome letter that states what is to be done and include a medical history form.
2. Let the patient and parent determine during the initial appointment how the patient desires to communicate (i.e., interpreter, lipreading, sign language, note writing, or a combination of these). Look for ways to improve communication. It is useful to learn some basic sign language. Face the patient and speak slowly at a natural pace and directly to the patient without shouting. Exaggeration of facial expressions and the use of slang make lipreading difficult. Even the best lip-readers comprehend only 30% to 40% of what is said.
3. Assess speech, language ability, and degree of hearing impairment when taking the patient's complete medical history. Identify the age of onset, type, degree, and cause of hearing loss, and determine whether any other family members are affected.
4. Enhance visibility for communication. Watch the patient's expression. Make sure the patient understands what the dental equipment is, what is going to happen, and how it will feel. Have the patient use hand gestures if a problem arises. Write out and display information.
5. Reassure the patient with physical contact; hold the patient's hand initially, or place a hand reassuringly on the patient's shoulder while the patient maintains visual contact. Without visual contact the child may be startled. Explain to the patient if you must leave the room.
6. Employ the tell-show-feel-do approach. Use visual aids and allow the patient to see the instruments,

and demonstrate how they work. Hearing-impaired children may be very sensitive to vibration.

7. Display confidence; use smiles and reassuring gestures to build up confidence and reduce anxiety. Allow extra time for all appointments.
8. Avoid blocking the patient's visual field, especially with a rubber dam.
9. Adjust the hearing aid (if the patient has one) before the handpiece is in operation, because a hearing aid amplifies all sounds. Many times the patient will prefer to have it turned off.
10. Make sure the parent or patient understands explanations of diagnosis, treatment, and payment. Deaf persons have different levels of skill with English. Use of an interpreter is extremely helpful.

VISUAL IMPAIRMENT

Total visual impairment (blindness) affects more than 30 million people. The list that follows gives some of the known causes of visual impairment; however, in more than 35% of those affected the cause is either unknown or unreported. Blindness is not an all-or-nothing phenomenon; a person is considered to be affected by blindness if the visual acuity does not exceed 20/200 in the better eye with corrective lenses or if the acuity is greater than 20/200 but is accompanied by a visual field of no greater than 20 degrees.

The following are known causes of visual impairment:

Prenatal causes

Optic atrophy
Microphthalmos
Cataracts
Colobomas
Dermoid and other tumors
Toxoplasmosis
Cytomegalic inclusion disease
Syphilis
Rubella
Tuberculous meningitis
Developmental abnormalities of the orbit

Postnatal causes

Trauma
Retrolental fibroplasia
Hypertension
Premature birth
Polycythemia vera
Hemorrhagic disorders
Leukemia
Diabetes mellitus
Glaucoma

Visual impairment may be only one aspect of a child's disability. For example, a patient with congenital rubella may have deafness, mental retardation, congenital heart disease, and dental defects, as well as blindness resulting from congenital cataracts. Total visual impairment is one disorder that may result in frequent hospitalizations, separation from family, and slow social development. Because the capabilities of a child with blindness are difficult to assess, the child may be considered developmentally delayed.

Consideration must be given to every developmental aspect of a child with blindness. Early in development the parents may experience guilt and either overprotect or reject the child; this can result in a lack of development of self-help skills and delayed development in general, which is often misinterpreted as mental retardation. Assessment of parental attitudes is of primary importance in behavioral management. In addition, children with blindness may exhibit self-stimulating activities, such as eye pressing, finger flicking, rocking, and head banging. Therefore assessment of the child's socialization is useful in the management of dental behavior.

A distinction should be made between children who at one time had sight and those who have not and thus do not form visual concepts. More explanation is needed for children in the latter category to help them perceive the dental environment. Dentists should realize that congenitally visually impaired children need a greater display of affection and love early in life and that they differ intellectually from children who are not congenitally visually impaired. Although explanation is accomplished through touching and hearing, reinforcement takes place through smelling and tasting. The modalities of listening, touching, tasting, and smelling are extremely important for these children in helping to learn coping behavior. Reports indicate that, once speech is developed, the other senses assume heightened importance and other development can occur that is comparable to that in children with sight.

Reports also reveal that motor activity affects the development of language and perception. Visually impaired children tend to have more accidents than other children during the early years while they are acquiring motor skills.

Hypoplastic teeth and trauma to the anterior teeth have been reported to occur with greater than average frequency in visually impaired children. Such children are also more likely to have gingival inflammation because of their inability to see and remove plaque. Other dental abnormalities occur with the same frequency as in the general population.

Before initiating dental treatment for a visually impaired child, the dentist should keep the following points in mind:

1. Determine the degree of visual impairment (e.g., can the patient tell light from dark?).

2. If the patient is accompanied by a companion, find out if the companion is an interpreter. If he or she is not, address the patient.
3. Establish rapport; afterward offer verbal and physical reassurance. Avoid expressions of pity or references to visual impairment as an affliction.
4. In guiding the patient to the operatory, ask if the patient desires assistance. Do not grab, move, or stop the patient without verbal warning. Encourage the parent to accompany the child.
5. Paint a picture in the mind of the visually impaired child by describing the office setting and treatment. Always give the patient adequate descriptions before performing treatment procedures. It is important to use the same office setting for each dental visit to allay the patient's anxiety.
6. Introduce other office personnel very informally.
7. When making physical contact, do so reassuringly. Holding the patient's hand often promotes relaxation.
8. Allow the patient to ask questions about the course of treatment and answer them, keeping in mind that the patient is highly individual, sensitive, and responsive.
9. Allow a patient who wears eyeglasses to keep them on for protection and security.
10. Rather than using the tell-show-feel-do approach, invite the patient to touch, taste, or smell, recognizing that these senses are acute. Avoid sight references.
11. Describe in detail instruments and objects to be placed in the patient's mouth. Demonstrate a rubber cup on the patient's fingernail.
12. Because strong tastes may be rejected, use smaller quantities of dental materials with such characteristics.
13. Some patients may be photophobic. Ask parents about light sensitivity and allow these patients to wear sunglasses.
14. Explain the procedures of oral hygiene and then place the patient's hand over yours as you slowly but deliberately guide the toothbrush.
15. Use audiocassette tapes and braille dental pamphlets explaining specific dental procedures to supplement information and decrease chair time.
16. Announce exits from and entrances to the dental operatory cheerfully. Keep distractions to a minimum, and avoid unexpected loud noises.
17. Limit providers of the patient's dental care to one dentist whenever possible.
18. Maintain a relaxed atmosphere. Remember that your patient cannot see your smile.

The provision of dental care to a visually impaired child is facilitated by an in-depth understanding of the patient's background. A team approach by all health professionals involved in the care of the child is ideal. Disease prevention and continuity of care are of utmost importance.

HEART DISEASE

Heart disease can be divided into two general types: congenital and acquired. Because individuals with heart disease require special precautions during dental treatment (such as antibiotic coverage for prevention of bacterial endocarditis), a dentist should closely evaluate the medical histories of all patients to ascertain their cardiovascular status.

CONGENITAL HEART DISEASE

The incidence of congenital heart disease is approximately 9 in 1000 births. The following is the relative incidence of congenital heart defects (Toronto Heart Registry):

Defect	Percent
Ventricular septal defect	22
Patent ductus arteriosus	17
Tetralogy of Fallot	11
Transposition of the great vessels	8
Atrial septal defect	7
Pulmonary stenosis	7
Coarctation of the aorta	6
Aortic stenosis	5
Tricuspid atresia	3
All others	14

The cause of a congenital heart defect is obscure. Generally it is a result of aberrant embryonic development of a normal structure or the failure of a structure to progress beyond an early stage of embryonic development. Only rarely can a causal factor be identified in congenital heart disease. Maternal rubella and chronic maternal alcohol abuse are known to interfere with normal cardiogenesis. If a parent or a sibling has a congenital heart defect, the chances that a child will be born with a heart defect are about 5 to 10 times greater than average. Congenital heart disease can be classified into two groups: acyanotic and cyanotic.

Acyanotic Congenital Heart Disease. Acyanotic congenital heart disease is characterized by minimal or no cyanosis and is commonly divided into two major groups. The first group consists of defects that cause left-to-right shunting of blood within the heart. This group includes ventricular septal defect and atrial septal defect. Clinical manifestations of these defects can include congestive heart failure, pulmonary congestion, heart murmur, labored breathing, and cardio-megaly.

The second major group consists of defects that cause obstruction (e.g., aortic stenosis and coarctation of the aorta). The clinical manifestations can include labored breathing and congestive heart failure.

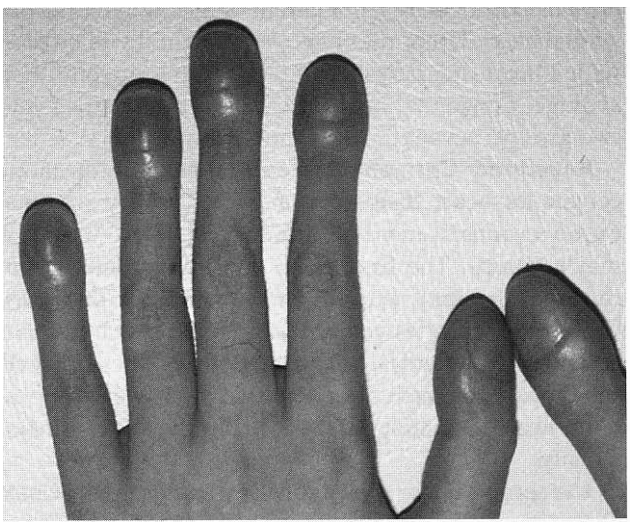
Cyanotic Congenital Heart Disease. Cyanotic congenital heart disease is characterized by right-to-left shunting of blood within the heart. Cyanosis is often observed even during minor exertion. Examples of such defects are tetralogy of Fallot, transposition of the great vessels, pulmonary stenosis, and tricuspid atresia. Clinical manifestations can include cyanosis, hypoxic spells, poor physical development, heart murmurs, and clubbing of the terminal phalanges of the fingers (Fig. 23-13).

ACQUIRED HEART DISEASE

Rheumatic Fever. Rheumatic fever is a serious inflammatory disease that occurs as a delayed sequela to pharyngeal infection with group A streptococci. Rheumatic fever is a commonly diagnosed cause of acquired heart disease in patients under 40 years of age. The mechanism by which the group *A Streptococcus* strains initiate the disease is unknown. The infection can involve the heart, joints, skin, central nervous system, and subcutaneous tissue. In general the incidence of rheumatic fever is decreasing. However, the incidence after exudative pharyngitis in epidemics is approximately 3%. (The incidence is much lower when the streptococcal pharyngitis is less severe.)

Although rheumatic fever can occur at any age, it is rare in infancy. It appears most commonly between the ages of 6 and 15 years. Rheumatic fever is most prevalent in temperate zones and at high altitudes and is more common and severe in children who live in substandard conditions. The clinical symptoms of rheumatic fever vary.

Cardiac involvement is the most significant pathologic sequela of rheumatic fever; carditis develops in approximately 50% of patients. Cardiac involvement can be fatal during the acute phase or can lead to chronic rheumatic heart disease as a result of scarring and deformity of heart valves.



f **I G 23-13.** Hand of a 9-year-old boy with tetralogy of Fallot. Clubbing of the terminal phalanges is apparent.

Infective Bacterial Endocarditis. Infective bacterial endocarditis is one of the most serious infections of humans. It is characterized by microbial infection of the heart valves or endocardium in proximity to congenital or acquired cardiac defects. Infective bacterial endocarditis has been classically divided into *acute* and *subacute* forms. The acute form is a fulminating disease that usually occurs when microorganisms of high pathogenicity attack a normal heart, causing erosive destruction of the valves. Microorganisms associated with the acute form include *Staphylococcus,* group A *Streptococcus,* and *Pneumonococcus.* In contrast, subacute bacterial endocarditis (SBE) usually develops in persons with preexisting congenital cardiac disease or rheumatic valvular lesions. Surgical placement of prosthetic heart valves can also predispose a patient to SBE; heart valve infections occur in 1% to 2% of such patients. The subacute form is commonly caused by viridans streptococci, microorganisms common to the flora of the oral cavity.

Embolization is a characteristic feature of infective endocarditis. Microorganisms introduced into the bloodstream may colonize the endocardium at or near congenital valvular defects, valves damaged by rheumatic fever, or prosthetic heart valves. These vegetations, composed of microorganisms and fibrous exudate, may separate and, depending on whether the endocarditis involves the left or right side of the heart, be propelled into the systemic or pulmonary circulation.

The clinical symptoms of SBE include low, irregular fever (afternoon or evening peaks) with sweating, malaise, anorexia, weight loss, and arthralgia. Inflammation of the endocardium increases cardiac destruction, and murmurs subsequently develop. Painful fingers and toes and skin lesions are also important symptoms. Laboratory findings can include leukocytosis and neutrophilia and normocytic, normochromic anemia. The erythrocyte sedimentation rate is rapid.

Endocarditis Prophylaxis. Transient bacteremia is an important initiating factor in SBE. Procedures known to precipitate transient bacteremias in dentistry and for which endocarditis prophylaxis is or is not recommended are included in Box 23-1.

Certain heart conditions are known to be more susceptible to the development of SBE (Box 23-2). Any dental patient who has a history of congenital heart disease or rheumatic heart disease or who has a prosthetic heart valve should be considered susceptible. The American Heart Association's antibiotic recommendations for prevention of bacterial endocarditis are presented in Fig. 23-14.

Epstein[$] reports that fewer than 25% of cases of bacterial endocarditis are associated with oral streptococci

BOX 23-1 Endocarditis Prophylaxis for Dental Procedures

PROPHYLAXIS RECOMMENDED*	PROPHYLAXIS NOT RECOMMENDED
Dental extractions	Restorative dentistry' (operative and prosthodontic) with or without retraction cord[s]
Periodontal procedures including surgery, scaling and root planing, probing, and recall maintenance	Local anesthetic injections (nonintraligamentary)
Dental implant placement and reimplantation of avulsed teeth	Intracanal endodontic treatment; post placement and buildup
Endodontic (root canal) instrumentation or surgery only beyond the apex	Placement of rubber dams
Subgingival placement of antibiotic fibers or strips	Postoperative suture removal
Initial placement of orthodontic bands but not brackets	Placement of removable prosthodontic or orthodontic appliances
Intraligamentary local anesthetic injections	Creation of oral impressions
Prophylactic cleaning of teeth or implants where bleeding is anticipated	Fluoride treatments
	Orthodontic appliance adjustment
	Shedding of primary teeth

Adapted from American Academy of Pediatric Dentistry: *Reference manual 2002-03,* Chicago, 2002, The Academy.
*Prophylaxis is recommended for patients with high- and moderate-risk cardiac conditions.
'This includes restoration of decayed teeth (cavity filling) and replacement of missing teeth.
[t]Clinical judgment may indicate use in selected circumstances that may cause significant bleeding.

BOX 23-2 Cardiac Conditions Associated with Endocarditis

PROPHYLAXIS RECOMMENDED	PROPHYLAXIS NOT RECOMMENDED
High-risk category	Negligible-risk category (no greater than in the general population)
Prosthetic cardiac valves, including bioprosthetic and homograft valves	Isolated secundum atrial septal defect
Previous bacterial endocarditis	Surgically repaired atrial septal defect, ventricular septal defect, or patent ductus arteriosus (without residua beyond 6 months)
Complex cyanotic congenital heart disease (e.g., single-ventricle states, transposition of the great arteries, tetralogy of Fallot)	Previous coronary artery bypass graft surgery
Surgically constructed systemic pulmonary shunts or conduits	Mitral valve prolapse without valvar regurgitation
Moderate-risk category	Physiologic, functional, or innocent heart murmurs
Most other congenital cardiac malformations (other than above and below)	Previous Kawasaki disease without valvar dysfunction
Acquired valvar dysfunction (e.g., rheumatic heart disease)	Previous rheumatic fever without valvar dysfunction
Hypertrophic cardiomyopathy	Cardiac pacemaker (intravascular and epicardial) or implanted defibrillator
Mitral valve prolapse with valvar regurgitation and/or thickened leaflets	

Adapted from American Academy of Pediatric Dentistry: *Reference manual* 2002-03, Chicago, 2002, The Academy.

and fewer than 15% of patients who develop infective endocarditis had dental or medical treatment in the 3 months before their diagnosis. Fewer than 4% of cases of infective endocarditis are associated with dental treatment. With only one large dose of antibiotics, routine restorative and surgical dental procedures can be safely completed.

DENTAL MANAGEMENT

Parents of patients with cardiac risks typically lack knowledge about endocarditis even after being informed during routine cardiology visits. Hayes and Fasules[9] report a deficiency of knowledge among dentists regarding the indications for prophylaxis and the antibiotic regimen required to prevent endocarditis. Before initiating care, the dentist should obtain a thorough medical and dental history, perform a physical examination, formulate a complete treatment plan, and discuss the treatment with the child's physician or cardiologist. Behavior management techniques are useful, and conscious sedation and nitrous oxide-oxygen analgesia have also proved beneficial in reducing anxiety in such

This wallet card is to be given to patients by their physician. Healthcare professionals: Please see back of card for reference to the complete statement.

Name:_____
needs protection from
BACTERIAL ENDOCARDITIS because of an existing **HEART CONDITION**
Diagnosis: _____
Prescribed by: _____
Date: _____

Dental procedures for which endocarditis prophylaxis is recommended[1]

- Dental extractions
- Periodontal procedures including surgery, scaling, and root planing, probing, and recall maintenance
- Endodontic (root canal) instrumentation or surgery only beyond the apex
- Subgingival placement of antibiotic fibers or strips
- Initial placement of orthodontic bands but not brackets
- Intraligamentary local anesthetic injections
- Prophylactic cleaning of teeth or implants where bleeding is anticipated

[1]Prophylaxis is recommended for patients with high- and moderate-risk cardiac conditions

Other procedures for which endocarditis prophylaxis is recommended

- Respiratory tract
  - Tonsillectomy and/or adenoidectomy
  - Surgical operations that involve respiratory mucosa
  - Bronchoscopy with a rigid bronchoscpe
- Gastrointestinal tract[2]
  - Sclerotherapy for esophageal varices
  - Esophageal stricture dilation
  - Endoscopic retrograde cholangiography with biliary obstruction
  - Biliary tract surgery
  - Surgical operations that involve intestinal mucosa
- Genitourinary tract
  - Prostatic surgery
  - Cystoscopy
  - Urethral dilation

[2]Prophylaxis is recommended for high-risk patients; it is optional for medium-risk patients.

Prophylactic Regimens for Dental, Oral, Respiratory Tract, or Esophageal Procedures. (Follow-up dose no longer recommended.) Total children's dose should not exceed adult dose.

I. Standard general prophylaxis for patients at risk:
Amoxicillin: Adults, 2.0 g (children, 50 mg/kg) given orally one hour before procedure.

II. Unable to take oral medications:
Ampicillin: Adults, 2.0 g (children 50 mg/kg) given IM or IV within 30 minutes before procedure.

III. Amoxicillin/ampicillin/pencillin allergic patients:
Clindamycin: Adults, 600 mg (children 20 mg/kg) given orally one hour before procedure. *-OR-*
Cephalexin* or Cefadroxil*: Adults, 2.0 g (children 50 mg/kg) orally one hour before procedure *-OR-*
Azithromycin or Clarithromycin: Adults, 500 mg (children15 mg/kg) orally one hour before procedure.

*Cephalosporins should not be used in patients with immediate-type hypersensitivity reaction to pencillins.

IV. Amoxicillin/ampicillin/penicillin allergic patients unable to take oral medications:
Clindamycin: Adults, 600 mg (children 20 mg/kg) IV within 30 minutes before procedure. *-OR-*
Cefazolin*: Adults, 1.0 g (children 25 mg/kg) IM or IV within 30 minutes before procedure.

Cardiac Conditions Associated with Endocarditis:

High-risk category:
- Prosthetic cardiac valves, including bioprosthetic and homograft valves
- Previous bacterial endocarditis
- Complex cynotic congenital heart disease (e.g., single ventricle states, transposition of the great arteries, tetralogy of Fallot)
- Surgically constructed systemic pulmonary shunts or conduits

Moderate-risk category
- Most other congenital cardiac malformations (other than above)
- Acquired valvar dysfunction (e.g., rheumatic heart disease)
- Hypertrophic cardiomyopathy
- Mitral valve prolapse with valvar regurgitation and/or thickened leaflets

Prophylactic Regimens for Genitourinary/Gastrointestinal Procedures:

I. High-risk patients:
Ampicillin plus gentamicin: Ampicillin (adults, 2.0 g: children 50 mg/kg) plus gentamicin 1.5 mg/kg (for both adults and children, not to exceed 120 mg) IM or IV within 30 minutes before starting procedure. 6 hours later, ampicillin (adults, 1.0 g: children, 25 mg/kg) IM or IV, or amoxicillin (adults, 1.0 g: children, 25 mg/kg) orally.

II. High-risk patients allergic to ampicillin/amoxicillin:
Vancomycin plus gentamicin: Vancomycin (adults, 1.0 g; children, 20 mg/kg) IV over 1–2 hours plus gentamicin 1.5 mg/kg (for both adults and children, not to exceed 120 mg) IM or IV. Complete injection/infusion within 30 minutes before starting procedure.

III. Moderate-risk patients:
Amoxicillin: Adults, 2.0 g (children 50 mg/kg) orally one hour before procedure. *-OR-*
Ampicillin: Adults, 2.0 g (children 50 mg/kg) IM or IV within 30 minutes before starting procedure.

IV. Moderate-risk patients allergic to ampicillin/amoxicillin:
Vancomycin: Adults, 1.0 g (children 20 mg/kg) IV over 1–2 hours. Complete infusion within 30 minutes of starting the procedure.

NOTE: For patients already taking an antibiotic, or for other special situations, please refer to the full statement referenced below.

Health Care Professionals—Please refer to these recommendations (endorsed by the American Dental Association and American Society for Gastrointestinal Endoscopy) for more complete information as to which patients and which procedures need prophylaxis.

ADA ACCEPTED American Dental Association

The Council on Dental Therapeutics of the American Dental Association has approved this statement as it relates to dentistry.

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[I G. 23-14. Patient education card supplied to all patients with congenital and rheumatic heart disease at James Whitcomb Riley Hospital for Children, Indianapolis, Ind. *(From American Heart Association: Prevention of bacterial endocarditis: recommendations by the Committee on Rheumatic Fever, Endocarditis, and Kawasaki Disease,* JAMA *277:1794-1801. Circulation. July 1, 1977. ©1997, American Medical Association. Used with permission.)*

patients. Cardiopulmonary resuscitation equipment should be readily available during the appointment. If general anesthesia is indicated, the dental procedures should be completed in a hospital setting, where adequate supportive care is available if needed.

Other considerations are especially important in treating patients who are susceptible to bacterial endocarditis:

- Pulp therapy is not recommended for primary teeth with a poor prognosis because of the high incidence of associated chronic infection. Extraction of such teeth with appropriate fixed-space maintenance is preferred.
- Endodontic therapy in the permanent dentition can usually be accomplished successfully if the teeth to be treated are carefully selected and the endodontic therapy is adequately performed.
- A dentist who feels uncomfortable in treating patients who are susceptible to infective endocarditis has a responsibility to refer them to someone who will care for them.

CARDIAC SURGERY PATIENTS

Patients who are to undergo cardiac surgery should first have a careful dental evaluation so that any needed dental treatment can be completed beforehand. This practice, along with an implemented preventive dental program, will decrease the incidence of postoperative endocarditis from oral sources.

After dental radiographs are taken and an evaluation is performed, a consultation with the patient's cardiologist is in order to plan required dental treatment before surgery. The cardiologist will indicate the specific desired antibiotic prophylaxis needed before the dental treatment. The dental examination and preventive dental program should be implemented before 6 months of age when possible. Ideally, dental treatment should be completed within 3 or 4 weeks of the planned surgery to allow for healing and the return of normal flora.

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CHAPTER

24

Management of the Medically Compromised Patient: Hematologic Disorders, Cancer, Hepatitis, and AIDS

BRIAN J. SANDERS
AMY D. SHAPIRO
RANDY A. HOCK
JAMES A. WEDDELL
CHRISTOPHER EDWARD BELCHER

انتشارات
شایان نمودار

CHAPTER OUTLINE

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- Disorders of Hemostasis
- Procoagulant Classification
- Treatment
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  - *Hemophilia B*
  - *von Willebrand disease*
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- Risks to Dental Staff Development of a Treatment Plan Use of Antifibrinolytics
  - *Dosages*
  - *Side effects*
- Pain Control
  - *Analgesia*
  - *Local anesthesia*
- Dental Management *Prevention of dental disease Periodontal therapy Restorative procedures Pulpal therapy Oral surgery Surgical complications Antibiotic prophylaxis Orthodontic treatment Dental emergencies*

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SICKLE CELL ANEMIA

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  - *Viral Infection*
  - *Bacterial Infection*
  - *Neoplasms*
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  - *HIV-Associated Gingivitis and HIV-Associated Periodontitis*

LEUKEMIA
- Oral Manifestations of Leukemia
- Dental Management of Patients with Leukemia

BONE MARROW/STEM CELL TRANSPLANTATION
- Oral Complications of Bone Marrow Transplantation
- Graft-Versus-Host Disease
- Pretransplantation Preparation
- Admission and Nursing Interventions
- Remission Phase

SOLID TUMORS

To achieve optimal oral health for the medically compromised patient, the dentist and physician must establish a close working relationship. Because of the complexity of many of these medical conditions additional treatment time may be needed to provide these services. To minimize the risk of possible complications that may affect the physical health of medically compromised patients, an aggressive prevention-oriented program is needed for such individuals. Each patient presents a unique set of challenges to the dentist, but achieving a successful outcome can be a rewarding experience. This chapter discusses major medical conditions and their dental management.

HEMOPHILIA

DISORDERS OF HEMOSTASIS

The hemophilias are disorders of hemostasis resulting from a deficiency of a procoagulant. Hemophilia is an inherited bleeding disorder affecting approximately 1 in 7500 males.

Hemophilia A, or classic hemophilia, is a deficiency of factor VIII, also known as *antihemophilic factor.* Factor VIII deficiency is the most common of the hemophilias and is inherited as an X-linked recessive trait. Therefore males are affected, females are carriers, and there is no male-to-male transmission. If a normal male has children with a carrier of hemophilia, there is a 50% chance that hemophilia will occur in each male offspring and a 50% chance that each female offspring will be a carrier. If a male hemophilic has children with a normal female, all male offspring will be normal, and all female offspring will be carriers.

Hemophilia B, or Christmas disease, is caused by a deficiency of factor IX (plasma thromboplastin component) and is also inherited as an X-linked recessive trait. Factor IX deficiency is one fourth as prevalent as factor VIII deficiency.

Factor XI (plasma thromboplastin antecedent) deficiency is inherited as an autosomal recessive trait, with male and female offspring equally affected. This disorder is most frequently observed in those of Ashkenazi Jewish descent.

Other factor deficiencies, such as those of factors II, V, and XIII (one case per 1 million) and factor VII (one case per 500,000) are rare and are also inherited as autosomal recessive traits.

Von Willebrand disease is a hereditary bleeding disorder resulting from an abnormality of the von Willebrand factor (VWF) that is found in the plasma, platelets, megakaryocytes, and endothelial cells. VWF circulates in conjunction with factor VIII and is important in platelet adhesion to the subendothelium via collagen and therefore in the formation of the primary platelet plug. In von Willebrand disease, the VWF may have a quantitative or qualitative abnormality. The VWF is composed of subunits called *multimers.* Von Willebrand disease is divided into subtypes based on the platelet and plasma multimeric structure of the VWF. Treatment of this disorder is dependent on the subtype.

Impaired formation of the platelet plug may result in bleeding from the skin and mucosa, bruising, epistaxis, prolonged bleeding after surgical procedures, and menorrhagia. This is in contrast to hemophilia involving deficiencies of factors VIII and IX, in which bleeding tends to involve muscles and joints.

PROCOAGULANT CLASSIFICATION

Hemophilia A (factor VIII deficiency) and hemophilia B (factor IX deficiency) are classified into the following three groups based on the level of the procoagulant present (normal levels range from 55% to 100%):

- Severe deficiency: levels less than 1%
- Moderate deficiency: levels between 1% and 5%
- Mild deficiency: levels greater than or equal to 5%

Patients with severe deficiency may experience frequent bleeding episodes, occurring two to four times per month. Bleeding episodes are often spontaneous, without a specific history of trauma. The common sites of bleeding include joints, muscles, and skin. Hemarthroses (joint hemorrhages) are common, and symptoms include pain, stiffness, and limited motion. Repeated episodes of hemarthroses or muscle hemorrhage result in chronic musculoskeletal problems. Individuals with hemophilia may develop debilitating painful arthritis. Commonly affected joints include knees, elbows, ankles, hips, and shoulders.

Pseudotumors (hemorrhagic pseudocysts) may occur in several locations including the jaw, in which case curettage is indicated.

Patients with moderate deficiency experience less frequent bleeding episodes (approximately four to six times per year). However, if a patient with moderate deficiency develops a target joint (a joint with repeated episodes of bleeding), spontaneous bleeding may occur.

Patients with mild deficiency bleed infrequently and only in association with surgery or injury. The diagnosis of a mild deficiency may be when an abnormality is found during presurgical evaluation or when bleeding occurs in association with surgery or trauma.

Mouth lacerations are a common cause of bleeding in children with all severities of hemophilia. Sonis and Musselman evaluated 132 patients with factor VIII–deficient hemophilia and noted that "persistent oral bleeding resulted in the diagnosis of 13.6% of all

cases of hemophilia."' About 29% of the cases of mild hemophilia observed were discovered as a result of bleeding from the oral cavity. Of the cases diagnosed secondary to oral bleeding, 78% were the result of bleeding from the maxillary frenum, and 22% resulted from tongue bleeds. Thus initial diagnosis of hemophilia, especially in moderate or mild disease, may directly involve the dentist.

TREATMENT

The mainstay of therapy for hemophilia is replacement of the deficient coagulation factor, by the use of either purified concentrate made from pooled plasma or factor produced through recombinant technology. In the past, whole blood, plasma, or cryoprecipitate was used for replacement therapy. Factor concentrates are advantageous because they are generally accessible, easy to handle and store, and virally inactivated, and lead to more consistent hemostatic results. The dosage, frequency of administration, and duration of therapy depend on the activity level needed, the half-life of the procoagulant, and the location and severity of the bleeding episode. The half-life of factor VIII is approximately 12 hours, whereas that for factor IX is approximately 24 hours.

Hemophilia A. Factor VIII concentrate is used for treatment of hemophilia A. Vials of factor concentrate are labeled with the number of units of activity contained, where 1 U is the amount of activity of the procoagulant present in 1 ml of normal plasma. For routine hemorrhagic episodes, such as early joint, soft tissue, and oral bleeds, a one-time correction of the procoagulant to approximately a level of 40% will achieve hemostasis and resolution of the bleeding episode. For mild factor VIII—deficient hemophilia, DDAVP* (1-deamino-8-D-arginine vasopressin) may be used for minor hemorrhagic episodes to achieve hemostasis. DDAVP is a synthetic analogue of vasopressin (antidiuretic hormone). This drug, when given intravenously, subcutaneously, or intranasally Stimate (desmopressin)+ causes a rise in factor VIII activity and in VWF, often to the hemostatic range. An appropriate rise in factor VIII activity to hemostatic levels should be document for a given patient before the therapeutic use of DDAVP, since response may vary across individuals. Peak levels are obtained approximately 1 hour after administration. Repeated administration of DDAVP may result in tachyphylaxis. Use of DDAVP to treat hemorrhagic disorders may also be associated with water retention, hyponatremia, and, rarely, seizures.

Hemophilia B. Factor IX—deficient hemophilia is treated with purified coagulation factor IX concentrate (monoclonal and recombinant). In the past less pure products in the class of prothrombin complex concentrate (PCC) were used. PCCs contained several other coagulation factors in addition to factor IX. Individuals who require high doses or repeated infusions of PCC are at risk for development of disseminated intravascular coagulation and thrombosis. The level necessary for hemostasis is the same for factor IX as for factor VIII (40%). However, the number of units needed to achieve that level is different because the volume of distribution of plasma-derived factor IX (1.0) is greater than that for factor VIII (0.5). The volume of distribution of recombinant factor IX is greater than that of plasma-derived factor IX (estimated minimum volume of 1.2 compared to 1, respectively).

von Willebrand Disease. Patients with von Willebrand disease should undergo subtyping to determine optimal therapy. DDAVP may be used to achieve hemostasis in type I von Willebrand disease. Type I von Willebrand disease is a quantitative deficiency of VWF with all multimers present. When DDAVP is used, a test dose should be administered to document adequate response. For patients who have less common subtypes of von Willebrand disease, patients who do not respond to DDAVP or for whom it is inappropriate, or bleeding events for which DDAVP should not be used, other therapeutic modalities may be needed, and interventions should be discussed with a hemophilia comprehensive center.

COMPLICATIONS

Inhibitors are antibodies that may develop in approximately 28% of patients with severe factor VIII deficiency and in 3% to 5% of patients with severe factor IX deficiency. The key to successful treatment of patients with inhibitors is accurate knowledge of the classification and level of the inhibitor. Patients with inhibitors are divided into two general groups, high responders and low responders, based on the past peak anamnestic response.

Patients in the low-response group may continue to be treated with factor concentrate, whereas those in the high-response group must be given bypassing products (either PCC or activated PCC and recombinant factor VIIa). Hemophilic patients with inhibitors pose a challenging treatment problem and should be managed only in conjunction with a hemophilia comprehensive center, since hemostasis may be difficult to achieve.

Other complications of hemophilia include arthritis and degenerative joint disease secondary to recurrent bleeding, and bloodborne viral infections that may have been transmitted via the necessary blood or blood products used for therapy. Therefore hepatitis (either B or C) and resultant liver disease have been a significant source of morbidity and mortality in this patient population. The human immunodeficiency virus (HIV)

*Aventis Pharmaceuticals Products Inc., Bridgewater, N.J. + Aventis Behring, King of Prussia, Penn.

has also been a major source of morbidity and mortality since approximately 1979. Before 1985, there was no antibody test for HIV and no consistent method of inactivation of this virus in the manufacture of factor concentrates. Therefore between 1979 and 1985, factor concentrates and blood products may have been contaminated with HIV. Approximately 90% of hemophilic patients with severe factor VIII deficiency and 30% of those with severe factor IX deficiency who received factor concentrate during these risk years may have become infected with HIV. HIV infection is a sensitive issue to these individuals, who may now bear the burden of two chronic conditions. Present-day treatment of factor concentrate and recombinant technology have effectively eliminated new transmission of HIV by this mode. Nevertheless, universal precautions should be followed in treating all hemophilic patients with a history of receiving either factor VIII or factor IX concentrate.

RISKS TO DENTAL STAFF

The risk of acquiring hepatitis B virus infection following an accidental stick with a needle used by a hepatitis B virus carrier ranges from 6% to 30%, far higher than the risk of HIV infection (less than 1 ‰) following a stick with a needle used by a patient infected with HIV. Moreover, although HIV antibodies have been isolated in saliva and other body fluids, there is no evidence to suggest that the disease is easily transmitted through saliva alone.

A study by Klein et al demonstrated a less than 0.5% occupational risk of HIV infection among dental professionals despite their infrequent compliance with recommended infection control precautions, frequent occupational exposure to persons at increased risk for HIV infection, and frequent accidental parenteral inoculations with sharp instruments.'

DEVELOPMENT OF A TREATMENT PLAN

With recent advances in treatment, most hemophilic patients can receive outpatient dental care routinely. With a thorough understanding of the patient's coagulopathy, the dentist, in conjunction with the hematologist, can make appropriate decisions regarding treatment.

The dentist must be fully aware of the procedures that can be safely performed and those in which complications may arise. The dentist should confer with the patient's physician and hematologist to formulate an appropriate treatment plan. The dentist should know the specific type of bleeding disorder, the severity of the disorder, the frequency and treatment of bleeding episodes, and the patient's inhibitor status. Many individuals with hemophilia self-administer the infusion products at home. The dentist should be prepared to discuss with the hematologist the type of anesthetic anticipated to be administered, the invasiveness of the dental procedure, the amount of bleeding anticipated, and the time involved in oral wound healing to help establish an appropriate treatment plan.

USE OFANTIFIBRINOLYTICS

Antifibrinolytics are adjunctive therapeutic agents for dental management of patients with bleeding disorders to help control oral bleeding. These agents include E-aminocaproic acid (Amicar*) and tranexamic acid (Cyklokapron}). Hemophilic patients form loose, friable clots that may be readily dislodged or quickly dissolved. Antifibrinolytics prevent lysis of clots within the oral cavity. They are used as an adjunct to factor concentrate replacement to prevent or control oral bleeding. For some dental procedures in which minimal bleeding is anticipated, they may be used alone without factor replacement.

Dosages. In children, e-aminocaproic acid is given immediately before dental treatment in an initial loading dose of 100 to 200 mg/kg by mouth up to a total dose of 10 g. Subsequently, 50 to 100 mg/kg per dose up to a total dose of 5 g of e-aminocaproic acid is administered orally every 6 hours for 5 to 7 days. Alternatively, for patients of approximately adult size, a regimen of 3 g by mouth four times daily without a loading dose may also be used. The advantage of E-aminocaproic acid for children is that it is available in both tablet and liquid form.

Adults require a 10 g initial loading dose and 5 g every 6 hours for 5 to 7 days.

The adult and pediatric dosage of tranexamic acid is 25 mg/kg given immediately before dental treatment. The same dose is continued every 8 hours for 5 to 7 days.

Side Effects. The common side effects associated with the use of antifibrinolytics include headache, nausea, and dry mouth. These side effects are usually tolerable and, unless severe, do not require discontinuation of the medication. To avoid thrombosis, antifibrinolytics should not be used when renal or urinary tract bleeding is present or when there is any evidence of disseminated intravascular coagulation. Repeated use of PCC or bypassing products (activated PCC) in patients with inhibitors should also be avoided during a course of antifibrinolytic therapy because they may predispose the patient to thrombotic episodes.

PAIN CONTROL

Analgesia. If patient apprehension is significant, sedation or nitrous oxide–oxygen inhalation analgesia may be considered. Hypnosis has also proved beneficial for some individuals. Intramuscular injections of

*Lederle Laboratories, Pearl River, N.Y. '
Upjohn Co., Kalamazoo, Mich.

hypnotic, tranquilizing, or analgesic agents are contraindicated because of the risk of hematoma formation. Analgesics containing aspirin or antiinflammatory agents (e.g., ibuprofen) alter platelet function and should not be used. Acute pain of moderate intensity can frequently be managed using acetaminophen (Tylenol,* Tempra[t]). Propoxyphene hydrochloride (Darvon[$]) is another analgesic acceptable for use in patients with hemophilia. (Darvon Compound-65 contains aspirin and should be avoided.) For severe pain, narcotic analgesics are required and are not contraindicated in the hemophilic patient.

Local Anesthesia. In the absence of factor replacement, periodontal ligament (PDL) injections may be used. The anesthetic is administered along the four axial surfaces of the tooth by placement of the needle into the gingival sulcus and the periodontal ligament space. Infiltration anesthesia can generally be administered without pretreatment with e-aminocaproic acid or replacement therapy. However, if the infiltration injection is into loose connective tissue or a highly vascularized area, then factor concentrate replacement to achieve a level of approximately 30% to 40% activity is recommended.

One must proceed with caution when considering block anesthesia. The loose, connective, nonfibrous, and highly vascularized tissue at the sites of inferior alveolar nerve injection and posterior superior alveolar injections are predisposed to the development of a dissecting hematoma, which has the potential of obstructing the airway and creating a life-threatening bleeding episode. A minimum of a 40% factor correction is mandatory. The dentist must carefully aspirate to make certain that the needle has not entered a blood vessel. In the event of a bloody aspirate, further factor replacement may be required, and the attending hematologist should be notified immediately following the operative procedure. All patients should be observed for the development of a hematoma and immediately referred for treatment in the event of hematoma formation following the administration of local anesthesia.

DENTAL MANAGEMENT

Most hemophilic patients can receive outpatient dental care routinely. Appointments should be arranged so that maximum treatment is accomplished per visit to minimize factor infusions and hence cost. Patients with inhibitors are best treated at a center with experience in dealing with this problem.

The dental procedures used in treating a patient with hemophilia do not differ significantly from those used for unaffected individuals.

Prevention of Dental Disease. A program that includes toothbrushing, flossing, appropriate topical fluoride exposure, and adequate systemic fluoride administration, as well as consumption of a proper diet and professional examination at regular intervals are effective measures that prevent dental problems. Rubber cup prophylaxis and supragingival scaling may be safely performed without prior factor replacement therapy. Minor bleeding can be readily controlled with local measures, such as direct pressure with a moistened gauze square. If bleeding persists for several minutes, the topical application of bovine thrombin,* microfibril-lar collagen (Avitene[t]), and local fibrin glue (Tisseel) may be of value.

Periodontal Therapy. Patients who require deep scaling because of gross calculus should initially undergo supragingival scaling. The tissue should be allowed to heal for 7 to 14 days, during which time the gingiva recedes as edema and hyperemia diminish. Subsequent treatments to remove calculus and irritants will therefore incur less risk of bleeding from the tissue. If subgingival scaling is planned, replacement therapy may be considered, depending on the amount of bleeding anticipated and the severity of the factor deficiency. It is imperative that periodontal patients be placed on a maintenance schedule for proper management.

An abnormal frenum attachment may cause gingival recession and pocket formation. Early treatment is indicated to prevent continued recession of gingiva and loss of alveolar bone. Any appropriate frenectomy technique is surgically acceptable for the patient with hemophilia. Both factor concentrate replacement and antifibrinolytic therapy are required before frenum or other periodontal surgery. If a large amount of bleeding is anticipated, these procedures should be done in a hospital environment, with the requisite preparation. The hematologist or attending physician must be contacted to help determine the factor correction required and the possible need for hospital management.

Restorative Procedures. The patient with hemophilia should be allowed to consider the prospect of all restorative procedures. Most restorative procedures on primary teeth can be successfully completed without factor concentrate replacement using PDL injections of local anesthesia or local infiltration. Small lesions may be restored using nitrous oxide-oxygen inhalation analgesia alone. The use of acetaminophen with codeine may also help to decrease discomfort in the child.

Most operative procedures for adults may also be completed using local infiltration of anesthetic, which usually does not require factor concentrate replacement.

*McNeil Consumer Products Co., Fort Washington, Penn. ' Mead Johnson Nutritionals, Evansville, Ind. +Eli Lilly and Company, Indianapolis, Ind.

*Although not reported in the dental literature, bovine thrombin-induced factor V deficiency, a rare acquired coagulopathy, has been reported.

[t]Davol Inc., Cranston, RI.

If a mandibular block or a posterior superior alveolar injection is anticipated, factor concentrate replacement to a level of 40% and antifibrinolytic therapy are required before injection. If factor concentrate replacement is required, all possible restorative treatment should be completed in one visit to minimize the number of infusions needed to complete the restorative treatment plan.

A rubber dam should be used to isolate the operating field and to retract and protect the cheeks, lips, and tongue. These soft tissues are highly vascular, and their accidental laceration could present a difficult management problem. A thin rubber dam is preferred because there is less tendency to torque the rubber dam retainer and cause abrasion of gingival tissues. The retainer should be placed carefully so that it is stable. If a retainer slips, it may lacerate the gingival papilla. Retainers with subgingival extensions, such as the 8A or 14A, should be avoided.

Wedges and matrices can be used conventionally. During proximal preparation, the wedge retracts the papilla, thus protecting it. A properly placed matrix should not cause bleeding.

High-speed vacuum and saliva ejectors must be used with caution so that sublingual hematomas do not occur. Care must also be used in the placement of intraoral radiographic films, particularly in highly vascular sublingual tissues.

The preparation of a tooth for a cast crown requires caution in the gingival preparation, as does the placement of retraction cord and impression material. Periphery wax is used on the impression tray to prevent possible intraoral laceration during tray placement. No undue trauma should be created in cementing or finishing a crown.

Pulpal Therapy. Sometimes, pulp exposures in primary and permanent teeth may be avoided if carious *dentin* is not entirely removed in one procedure (indirect pulp therapy). A pulpotomy or pulpectomy is preferable to extraction. The extraction of a tooth in an individual with hemophilia implies more complicated treatment and expense to the patient. Most vital pulp-otomy and pulpectomy procedures can be successfully completed using local infiltration anesthesia. Nitrous oxide-oxygen inhalation analgesia may also help alleviate any discomfort. If the pulp of a vital tooth is exposed, an intrapulpal injection may be safely used to control pain. Bleeding from the pulp chamber does not present a significant problem because it is readily con-trolled with pressure from cotton pledgets. If pulp tissue is necrotic, local anesthetic is usually unnecessary.

Oral Surgery. Preoperative evaluation and postoperative management of the patient with hemophilia undergoing extractions must be coordinated with the hematologist. The dentist should discuss with the hematologist the surgical procedure, including the anesthetic technique, the degree of surgical trauma anticipated, and the duration of the healing process. The hematologist can then determine the amount and duration of factor concentrate replacement required for surgery and postoperative management. Today it is possible to perform oral surgery in the hemophilic patient on an outpatient basis. Requirements include an experienced dentist and hematologist, a facility available for the patient to receive infusions if home infusion is not feasible, and a coagulation laboratory capable of performing any needed laboratory evaluations. Patients with inhibitors should be treated in a hospital setting by those experienced in their management. For simple extractions of erupted permanent teeth and multirooted primary teeth, a 30% to 40% factor correction is administered within 1 hour before dental treatment. Antifibrinolytic therapy should be started immediately before or after the procedure and should be continued for 5 to 10 days. The patient should be placed on a clear liquid diet for the first 72 hours. For the next week, a soft, pureed diet is recommended. During this time, the patient should not use straws or metal utensils, pacifiers, or bottles.

After 10 days, the patient may begin to consume a more normal diet. Specific postoperative instructions should be given to the patient and the parent. Because of the cost of factor concentrate, all extractions should be completed in one appointment if possible.

After extractions are completed, the direct topical application of hemostatic agents, such as bovine thrombin or microfibrillar collagen hemostat (Avitene), may help with local hemostasis. The socket should be packed with an absorbable gelatin sponge (e.g., Gelfoam*). Microfibrillar collagen or topical thrombin or fibrin glue may then be sprinkled over the wound. Direct pressure with gauze should then be applied to the area.

Stomahesive† may be placed over the wound for further protection from the oral environment. In general, the use of sutures should be avoided unless suturing would markedly enhance healing. Resorbent sutures are recommended. The patient must be given specific and thorough postoperative instructions.

For surgical extractions of impacted, partially erupted, or unerupted teeth, a higher factor activity level may be needed before surgery. This should be discussed with the hematologist, because there would be a greater degree of surgical trauma and a longer healing period. The hematologist may also elect to administer factor replacement to the patient postoperatively. Antifibrinolytic therapy should be started immediately before or after the procedure and continued for 7 to 10 days.

*The Upjohn Co., Kalamazoo, Mich. '
Bristol-Myers Squibb Co., Princeton, NJ.

For simple extractions of single-rooted primary teeth (i.e., incisors and canines), one must evaluate the amount of root development present to determine whether factor replacement therapy is required. If there is complete root development, factor replacement therapy may be necessary. If there is only partial root formation, antifibrinolytic therapy along with local hemostatic agents may be all that is required.

The normal exfoliation of primary teeth does not usually require factor replacement, and bleeding can usually be controlled with direct finger and gauze pressure maintained for several minutes. The direct topical application of hemostatic agents may also help with local hemostasis. If there is continuous slow bleeding, antifibrinolytic therapy may be started.

Surgical Complications. Despite all precautions, bleeding may occur 3 to 4 days postoperatively when the clot begins to break down. Both systemic and local treatment must be used for control of hemorrhage. Sufficient replacement factor should be administered to control any recurrent bleeding.

It is not prudent to protect a loose clot. The typical clot in this situation is characterized as a "liver clot." It is dark red and usually protrudes from the surgical site, often covering the surfaces of several teeth. Following adequate replacement with factor concentrate, usually to achieve a 30% to 40% activity level, the clot should be removed and the area cleansed to help isolate the source of bleeding. The socket should then be repacked.

Antibiotic Prophylaxis. Total joint replacement, usually of the hip or knee, may be performed in patients with hemophilia to restore function and alleviate the pain associated with degenerative arthritis that has developed after multiple hemarthroses. Antibiotic prophylaxis is required for patients with artificial joints before invasive dental procedures. The American Heart Association recommendations for subacute bacterial endocarditis prophylaxis, as revised in 1997, are usually followed. If the patient is immunocompromised because of HIV infection, intravenous antibiotic prophylaxis may be considered.

Orthodontic Treatment. Early recognition of an orthodontic problem is important, because selective guidance can diminish or eliminate complex orthodontic problems. Both interceptive and full-banded orthodontics may be performed. Care must be taken in the adaptation and placement of bands to avoid lacerating the oral mucosa and to avoid leaving protruding sharp edges and wires. Bleeding caused by an accidental scratch or minor laceration of the gingiva usually responds to applied pressure for 5 minutes. The use of preformed orthodontic bands and brackets, which can be bonded directly to the teeth, almost totally eliminates contact of orthodontic appliances with the gingiva during placement. Longer-acting wires and springs require less frequent adjustment of orthodontic appliances. Oral hygiene is particularly important to avoid inflamed, edematous, and hemorrhagic gingival tissues. A water-irrigating device may be helpful for good home dental care.

Dental Emergencies. Oral trauma is a common occurrence during childhood. Management of bleeding injuries, including hematomas, in the oral cavity of the patient with hemophilia may require a combination of factor replacement and antifibrinolytic therapy, and treatment with local hemostatic agents.

VIRAL HEPATITIS

Viral hepatitis is an infection that produces inflammation of liver cells, which may lead to necrosis or cirrhosis of the liver. Acute hepatitis classically presents with lethargy, loss of appetite, nausea, vomiting, abdominal pain, and ultimately jaundice.

Acute viral hepatitis may be caused by any of the following: hepatitis A virus (HAV), hepatitis B virus (HBV), hepatitis delta virus (HDV), and two forms of non-A, non-B hepatitis virus (NANB)—parenterally transmitted NANB or hepatitis C virus (HCV), and enterically transmitted NANB or hepatitis E virus.

Infection with hepatitis A virus results in an acute febrile illness with jaundice, anorexia, nausea, and malaise. Most HAV infections in infants and children cause mild, nonspecific symptoms without jaundice. HAV is spread by the fecal-oral route and is endemic in developing areas. Spread occurs readily in households and day-care centers, where symptomatic illness occurs primarily among adult contacts of children. No HAV carrier state exists, and the presence of immunoglobulin G-anti-HAV indicates past infection and lifelong immunity to HAV.

Hepatitis B transmission is of major concern to the dentist. Members of the dental profession assume a risk of acquiring HBV that is at least three times higher than that in the general population. An additional concern, beyond that of acquiring I-IBV, is the potential of becoming an asymptomatic yet infectious carrier of HBV and of having the capability of transmitting the disease to patients and dental staff members and family.

HBV is transmitted from person to person by parenteral, percutaneous, or mucous membrane inoculation. It can be transmitted by the percutaneous introduction of blood, administration of certain blood products, or direct contact with secretions contaminated with blood containing HBV. Infection may also result from inoculation of mucous membranes, including sexual transmission. Wound exudate contains HBV, and open-wound-to-open-wound contact can transmit infection. There can also be vertical transmission from an infected mother to her baby, and this almost always leads to chronic infection.

A medical history is unreliable in identifying patients who have actually had HBV infection, because

approximately 80% of all HBV infections are undiagnosed. However, the medical history is useful in indicating groups of patients who are at higher risk of being undiagnosed carriers. Among populations at high risk for HBV infection are patients undergoing hemodialysis, patients requiring frequent large-volume blood transfusions or administration of clotting factor concentrates, residents of institutions for the mentally disabled, and users of illicit injectable drugs.

In 2001, an estimated 78,000 people in the United States became infected with HBV, and an estimated 1.25 million chronically infected people live in America. Overall, chronic liver disease from hepatitis B claims 5000 lives a year in the United States.

Chronic active hepatitis develops in more than 25% of carriers and often progresses to cirrhosis. Furthermore, HBV carriers have a risk of developing primary liver cancer that is between 12 and 300 times higher than that of uninfected individuals.

For detection of acute or chronic HBV infection, the serologic test for hepatitis B surface antigen (HepBsAg) is most commonly used. The antibody to surface antigen (HepBsAb or anti-HepBsAg) is protective and indicates a resolved natural infection or successful vaccination. Antibody to the core antigen (HepBsAb or anti-HepBc) indicates exposure to natural hepatitis B virus but can be present in either resolved or chronic infection. The hepatitis Be antigen (HepBeAg) is a useful marker for infectivity. Patients who test positive for HepBeAb and HepBsAg are most likely to transmit the disease. If the patient still shows a positive test result for HepBsAg 6 months after an acute HBV infection, the atient is considered to be chronically infected.

The availability of a safe, effective hepatitis B vaccine affords the dentist and staff additional protection against acquiring HBV infection. HBV vaccine is recommended for all health care personnel. The vaccine is derived from recombinant DNA and therefore does not have the potential to transmit the disease. When administered in a three-dose injection regimen (0, 1, 6 months), the recombinant DNA vaccine has induced protective antibody production (anti-HepBs) in 95% to 100% of adults.

A fulminant type of hepatitis occurs with infection by HDV but only with coexisting or simultaneous infection with HBV. HDV is defective in that it requires HBV for outer coat proteins (HepBsAg), as well as requiring HBV for replication. Transmission is similar to that of HBV, by parenteral, percutaneous, or mucous membrane inoculation.

According to Cottone, HDV infection occurs in two primary modes:

The first is simultaneous infection with HBV and I-IDV. When simultaneous infection occurs, the acute clinical course of hepatitis often is limited with resolution of both hepatitis B and HDV infections. The second mode of transmission, acute delta super-infection, involves those with chronic Hepatitis B infection. In this situation, the patient already has a high titer of circulating HepBsAg; thus HDV can rapidly replicate. These patients are more likely to have a serious and possibly acute fulminant form of hepatitis that more often leads to chronic HDV .[3]

Hepatitis C (formally known as parentally transmitted NANB hepatitis) is of great concern to the dentist, because it can be transmitted by needle stick (risk of about 1.8% with a range of 0% to 10%) and no immunization is available. Patients at risk of hepatitis C include those who received blood transfusions or organ transplants before 1992, those who received clotting factors before 1987, those who have experimented with illicit intravenous drugs regardless of how many times or how long ago, and those on chronic dialysis. Of those infected, 70% to 85% develop chronic infection, and about 70% develop chronic liver disease. Although only 3% of infected people die from liver failure, hepatitis C is the leading reason for liver transplantation in adults in the United States.

In 1989, parenterally transmitted NANB was identified as HCV. Subsequently, three genotypes have been identified. Diagnosis is made by detection of antibody to hepatitis C virus (anti-HCV) in the serum and can be confirmed by radiommunoblot assay. Polymerase chain reaction testing (PCR) can be done in a qualitative fashion to confirm diagnosis or in a quantitative fashion to assess response to treatment. Treatment, when indicated, includes administration of interferon or pegylated interferon with or without ribavirin. Response rates vary from 50% for type 1 infection to 80% for types 2 and 3.

In 2001, the Centers for Disease Control and Prevention estimated that there were 25,000 new cases of infection with hepatitis C in the United States.' There are 2.7 million carriers of hepatitis C in America, and chronic liver disease in these patients results in 8000 to 10,000 deaths per year. The number of new cases per year in the United States dropped from a high of 291,000 in 1989 to 25,000 in 2001. Infection associated with drug use accounts for 60% of the new cases in the United States.

As noted earlier, of concern to dentists and staff is the present unavailability of immunization against HCV. Antibodies formed in patients with HCV are not protective antibodies. However, the use of universal precautions should provide adequate protection.

Enterically transmitted NANB has now been identified as the hepatitis E virus. Transmission is by the fecal-oral route. Large, well-documented outbreaks have been seen in India, the former Soviet Union, North America, Mexico, and Southeast Asia. There appears to be a 6- to 8-week incubation period and a low incidence of carrier

state after infection. However, a high fatality rate (10% to 20%) is seen in women in their third trimester of pregnancy who contract this virus. There is no immunization presently available against this pathogen.

SICKLE CELL ANEMIA

Patients with sickle cell anemia have an autosomal recessive hemolytic disorder that occurs predominantly in persons of African descent but is also found among Italian, Arabian, Greek, and Indian people.

Patients with sickle cell anemia produce hemoglobin S instead of the normal hemoglobin A. Hemoglobin S has a decreased oxygen-carrying capacity. Decreased oxygen tension causes the sickling of cells. These patients are susceptible to recurrent acute infections, which result in an "aplastic crisis" caused by decreased red blood cell production and in subsequent joint and abdominal pain with fever. Over time there is a progressive deterioration of cardiac, pulmonary, and renal function.

Many factors can precipitate a sickle cell crisis, including acidosis, hypoxia, hypothermia, hypotension, stress, hypovolemia, dehydration, fever, and infection.

Radiographic changes are associated with sickle cell anemia. There is a generalized radiolucency and loss of trabeculae with prominent lamina dura, caused by increased erythropoietic demands that result in expansion of the marrow spaces. Bone growth may be decreased in the mandible, resulting in retrusion, and the teeth may be hypomineralized. Occasionally, patients with sickle cell anemia have infarcts in the jaw, which may be mistaken for a toothache or osteomyelitis. The patients experience dental pain with absence of pathology.

Dental appointments should be short to reduce potential stress on the patient. The importance of an aggressive preventive program cannot be understated, and such a program should have the goal of maintaining excellent oral health and decreasing the possibility of oral infection. Dental treatment should not be initiated during a sickle cell crisis. If emergency treatment is necessary during a crisis, only treatment that will make the patient more comfortable should be provided. Patients with sickle cell anemia may have skeletal changes that make orthodontic treatment beneficial. Special care must be taken to avoid tissue irritation, which may induce bacteremias, and the disease process may compromise the proposed treatment. Careful monitoring is a necessity when proposing elective orthodontic treatment in patients with sickle cell anemia.

Many patients with sickle cell anemia have defective spleen function or undergo a splenectomy, which leaves them more vulnerable to infection because immunoglobulin production is decreased and phagocytosis of foreign antigens is thus impaired. Most patients with sickle cell anemia are taking low-dose daily prophylactic antibiotics, and the need for additional antibiotics for dental procedures is debatable. Some authors have recommended the use of antibiotics for all dental procedures, whereas others recommend the administration of additional antibiotics when there is obvious dental or periodontal infection. The selection of an antibiotic is usually similar to that in cases of heart defect.

The use of local anesthetics with a vasoconstrictor is not contraindicated in patients with sickle cell anemia. Some textbooks do recommend against the use of vasoconstrictors, although there is no evidence to support this practice. Similarly, the use of nitrous oxide is not contraindicated in these patients. Care must be taken in treating patients with sickle cell anemia to avoid diffusion hypoxia at the completion of the dental procedure.

The restoration of teeth, including pulpotomies, is preferable to extraction. Pulpectomy in a nonvital tooth is reasonable if the practitioner is fairly confident that the tooth can remain noninfected. If the tooth is likely to persist as a focus of infection, then extraction is indicated.

The use of general anesthesia for dental procedures must be approached cautiously in consultation with the hematologist and anesthesiologist. Previously, the standard protocol was to perform a direct transfusion (immediate introduction of whole blood or blood components) or an exchange transfusion (repetitive withdrawal of small amounts of blood and replacement with donor blood until a large portion of the patient blood has been exchanged) before general anesthesia. The goal of the transfusion is to increase the patient's hemoglobin level to higher than 10 g/dl and to decrease the hemoglobin S level below 40%. Transfusions do not provide complete protection against venous complications, but they may temporarily improve the patient's condition and reduce the hazards of surgery.

The current thinking is to weigh the risks associated with transfusion prior to anesthesia induction. Suggested guidelines for performing a prophylactic trans-fusion before general anesthesia have been proposed. Patients with a hemoglobin level of less than 7 g/dl and a hematocrit of less than 20% may require a transfusion. Pediatric patients are usually less likely to have post-transfusion complications than are adults. A high frequency of hospitalizations is indicative of a more severe anemia, and such patients may require transfusion prior to surgery. Minor surgeries may not require a transfusion.

ACQUIRED IMMUNODEFICIENCY SYNDROME

Acquired immunodeficiency syndrome (AIDS) is a clinically defined condition caused by infection with HIV type 1 or, much less commonly, type 2. Estimates

are that, in the United States, about 900,000 people are infected with HIV and one quarter of them do not know they are infected. Worldwide in 2002, 38.6 million adults and 3.2 million children were HIV infected.

The incubation period from the time of infection to the appearance of symptoms of AIDS is approximately 11 years in adults. Therefore HIV-infected individuals can unknowingly spread the virus to sexual or needle-sharing partners or, in the case of infected mothers, to their children. AIDS was the fifth leading cause of death in the United States in 2001 among people from 25 to 44 years of age.

HIV infects cells of the immune system, specifically lymphocytes and macrophages. These white blood cells contain the greatest number of CD4 cell surface receptors (glycoproteins), which permit attachment with viral surface proteins (GP120) and enhance host-cell invasion and infection. Under the control of the HIV "POL" gene, the virus produces the enzyme reverse transcriptase, which is essential for incorporating viral RNA into host nuclear DNA. The viral genome is integrated into the host-cell genome and leads to progressive and eventually irreversible immunosuppression by producing more virus and further killing the CD4 (T_4) helper-inducer lymphocytes that are important modulators of the immune system. The subsequent immunodeficiency results in a variety of opportunistic infections, malignancies (such as Kaposi sarcoma and lymphoma), and autoimmune diseases. Diagnosis is made by screening the serum for antibodies to HIV and is confirmed by Western blot analysis. Ongoing management is guided by the patient's CD4' cell count and viral load as measured by PCR. The former is an indication of the patient's immune status, whereas a higher viral load is associated with a more accelerated disease. The current antiretroviral drugs target the virus at several steps: (1) the fusion of the virus to the host cell (fusion inhibitors), (2) the transcription of DNA from viral RNA by reverse transcriptase (nucleoside, nonnucleoside, and nucleotide reverse transcriptase inhibitors), and (3) the cleavage of viral proteins by the viral protease enzyme (protease inhibitors). The most effective treatment strategies use a combination of several drugs to inhibit the virus at several steps.

In the United States, 60% of newly infected men acquired HIV through homosexual contact, infection from illicit drug use accounted for 25% of new cases, and 15% of infections were acquired through heterosexual contact. Among infected women in the United States, 75% acquired HIV through heterosexual contact and 25% through intravenous drug use. Almost 30% of newborns of untreated HIV-infected mothers can acquire the HIV virus through vertical transmission. However, treatment of pregnant women with antiretroviral medications, including azidothymidine (AZT), has decreased the rate of transmission by 70%. The onset of symptoms is shortened in children who have acquired their infection prenatally and go untreated. Only 75% survive to age 5 years, and by that age 50% have severe symptoms.

Infants and children with AIDS have clinical findings similar to those in adults. Early manifestations of HIV infection include *Pneumocystis carinii* pneumonia, interstitial pneumonitis, weight loss and failure to thrive, hepatomegaly or splenomegaly, generalized lymphadenopathy, and chronic diarrhea. Unlike in adults, recurrent and severe bacterial infections are common in pediatric patients with HIV infection.

ORAL MANIFESTATIONS OF HIV INFECTION

The types of oral lesions seen in HIV infection may be caused by fungal, viral, or bacterial infections, as well as neoplastic and idiopathic processes.

Fungal Infection. Pindborg stated that the most common HIV-associated infection of the mouth is caused by the fungus *Candida albicans*.[5] Oral candidiasis is frequently present and may lead to esophageal or disseminated candidiasis. There are four major types of oral candidiasis: (1) pseudomembranous, (2) hyperplastic, (3) erythematous (atrophic), and (4) angular cheilotic.

The pseudomembranous lesion is characterized by the presence of creamy white or yellow plaques that can easily be removed from mucosa, leaving a red, bleeding surface. The most common locations for these lesions are the palate, buccal and labial mucosa, and dorsum of the tongue.

The hyperplastic lesion is characterized by white plaques that cannot easily be removed. The most common location is the buccal mucosa.

The erythematous (atrophic) lesion is characterized by a red appearance. Common locations are the palate and the dorsum of the tongue. The lesion may also appear as spotty areas on the buccal mucosa.

Angular cheilitis is characterized by fissures radiating from the commissures of the mouth, often associated with small, white plaques.

The treatment of C. *albicans* infection can be either systemic or topical. Topical therapy involves the use of nystatin (Mycostatin) rinses (100,000 U, three to five times daily) or clotrimazole (Mycelex) troches. Treatment for 1 to 2 weeks is usually effective. Systemic therapy calls for ketoconazole (Nizoral) 200 or 400 mg daily with food, or fluconazole (Diflucan), 100 mg daily. Amphotericin B or fluconazole (administered intravenously) is used when candidal infection has become systemic.

Candidal infections frequently recur. Therefore patients may remain on antifungal medication indefinitely. As an adjunctive measure, mouthrinses with

Peridex* (0.12% chlorhexidine digluconate) may be used. Chronic oral candidiasis may be a poor prognostic sign indicating a phase of more rapid decline of immune function to the terminal phase of AIDS.

Viral Infection. In the same way that fungi can cause oral disease because of the immune dysfunction induced by HIV infection, several viruses can produce lesions in the mouth following colonization or reactivation. These include herpes group viruses and papillomaviruses according to Greenspan.'

Oral warts may be seen in the HIV-infected patient, with human papillomavirus as the etiologic agent. Some warts have a raised, cauliflower-like appearance, whereas others are well circumscribed, have a flat surface, and almost disappear when the mucosa is stretched.

Herpes simplex virus (HSV) can produce recurrent episodes of painful ulceration. Intraorally the lesions appear most commonly on the palate. Typically these lesions present as vesicles that break open to form ulcers. However, they may also have an atypical appearance as slitlike lesions on the tongue or may mimic other diseases. Diagnosis can be made from culture or fluorescent antibody testing.

Herpetic lesions may be treated with oral acyclovir (Zovirax[t]). Acyclovir may also be administered intravenously (750 mg/m^2 in divided doses three times a day until lesions clear) in individuals with more severe oropharyngeal lesions or in those unable to swallow.

Herpes zoster (shingles) is caused by varicella-zoster virus (VZV), the chickenpox virus. VZV can produce oral ulcerations, which are usually accompanied by skin lesions generally restricted to one side of the face. These lesions are also treated with acyclovir.

Oral hairy leukoplakia (HL) is a white lesion that does not rub off, located on the lateral margins of the tongue. The surface may be smooth, corrugated, or markedly folded. HL is seen only in patients who are HIV infected. HL is a virally induced lesion caused by the Epstein-Barr virus. Treatment may include the use of high-dose acyclovir. However, the lesions usually recur.

Bacterial Infection. Bacteria causing oral lesions may include *Mycobacterium avian-intracellulare* and *Klebsiella pneumoniae.* Many of the oral lesions seen in association with HIV infection are not new entities; rather, they are known diseases that either follow an atypical course or that show an unusual response to treatment. This is frequently the case with neoplasms as well.

Neoplasms. Kaposi sarcoma is the most common malignancy seen in AIDS and occurs in 15% to 20% of AIDS patients according to Silverman.' Intraoral lesions may occur alone or along with skin, visceral, and lymph node lesions. Often the first lesions of Kaposi sarcoma appear in the mouth. They may be red, blue, or purple, flat or raised, and solitary or multiple. The most common oral site is the hard palate, although lesions may be found on any part of the oral mucosa. Treatment for aggressive lesions involves radiation, laser surgery, or chemotherapy. Conventional surgery may be appropriate for small lesions.

The group of malignancies whose incidence is growing the fastest among patients with AIDS is the lymphomas, most commonly the non-Hodgkin's lymphomas. The first manifestation may be a firm, painless swelling in the mouth. Biopsies of these growths are indicated to establish a diagnosis. Treatment includes multidrug chemotherapy and radiation. Less than 20% of patients survive 2 years; the mean survival time is approximately 6 months from diagnosis.

Oral squamous cell carcinomas also occur more frequently in the HIV-infected population.

Idiopathic Lesions. According to Greenspan oral ulcers of unknown etiology are being reported with increasing frequency in people with HIV infection.' The ulcers resemble aphthous lesions, appearing as well-circumscribed ulcers with an erythematous margin. Patients sometimes exhibit extremely large and painful necrotic ulcers that may persist for several weeks.

Salivary gland swelling has been seen in both adults and children with HIV infection. The cause of the swelling is unknown. It usually involves the parotid glands and is also accompanied by xerostomia.

HIV-infected patients may develop autoimmune disorders, including immune thrombocytopenic purpura. Oral lesions appear as small, blood-filled purpuric lesions or petechiae. Spontaneous gingival bleeding may also occur.

HIV-Associated Gingivitis and HIV-Associated Periodontitis. Progressive and premature periodontal disease is seen relatively frequently in HIV-infected individuals and may even be the first sign of HIV infection. Unlike conventional periodontal disease, these lesions do not respond effectively to standard periodontal therapy. There may be a rapid progression from mild gingivitis to advanced, painful, spontaneously bleeding periodontal disease in a few months. Treatment includes aggressive curettage, Peridex (0.12% chlorhexidine digluconate) rinses three times daily, and possibly antibiotic treatment.

LEUKEMIA

Malignancy is second only to accidents as the leading cause of death in children. Leukemias are hematopoietic malignancies in which there is a proliferation of abnormal leukocytes in the bone marrow and dissemination of these cells into the peripheral blood. The abnormal leukocytes (blast cells) replace normal cells in bone

*Zila Pharmaceuticals, Phoenix, Ariz. [t]
GlaxoSmithKline, Inc., Research Triangle Park, N.C.

marrow and accumulate in other tissues and organs of the body.

Leukemia is classified according to the morphology of the predominant abnormal white blood cells in the bone marrow (Table 24-1). These types are further categorized as acute or chronic, depending on the clinical course and the degree of differentiation, or maturation, of the predominant abnormal cells.

In the United States, about 6550 new cases of cancer are diagnosed each year in children under the age of 15. Acute leukemia is the most common malignancy in children, with about 2500 new cases diagnosed annually in the United States. Thus acute leukemia accounts for about one third of all childhood malignancies; of these, approximately 80% are lymphocytic (acute lymphocytic leukemia, or ALL). Chronic leukemia in children is rare, accounting for less than 2% of all cases.

Leukemia affects about 5 in 100,000 children in the United States. The peak incidence is between 2 and 5 years of age. Although the cause of leukemia is unknown, ionizing radiation, certain chemical agents, and genetic factors have been implicated. For example, children with chromosomal abnormalities (Down syndrome and Bloom syndrome), children with an identical twin who has leukemia, and children with immunologic disorders have an increased risk of leukemia.

The clinical manifestations of acute leukemia are caused by the infiltration of leukemia cells into tissues and organs. Infiltration and proliferation of leukemia cells in the bone marrow lead to anemia, thrombocytopenia, and granulocytopenia. Because these cytopenias develop gradually, the onset of the disease is frequently insidious. The history at presentation may reveal increased irritability, lethargy, persistent fever, vague bone pain, and easy bruising. Some of the more common findings on initial physical examination are pallor, fever, tachycardia, adenopathy, hepatosplenomegaly, petechiae, cutaneous bruises, gingival bleeding, and evidence of infection.

In approximately 90% of the cases of acute leukemia a peripheral blood smear reveals anemia and thrombocytopenia. In about 65% of cases the white blood cell count is low or normal, but it may be greater than 50,000 cells/mm^3.

When a new case of leukemia is diagnosed, the patient is hospitalized and therapy is directed toward stabilizing the patient physiologically, controlling hemorrhage, identifying and eliminating infection, evaluating renal and hepatic functions, and preparing the patient for chemotherapy.

These interventions proceed while the definitive studies to determine the exact type of leukemia are undertaken. These include obtaining bone marrow for microscopic analysis, special cytochemical staining, immunophenotyping by flow cytometry, and cytogenetic analysis.

The goal of treatment is to induce and maintain a complete remission, which is defined as resolution of the physical findings of leukemia (e.g., adenopathy, hepatosplenomegaly, petechiae) and normalization of peripheral blood counts and bone marrow (less than 5% blasts).

The basic principle of treatment of ALL is substantially different from that of acute myelogenous or nonlymphocytic leukemia (ANLL). In general, the treatment of ANLL is very intense and results in profound bone marrow hypoplasia, but the treatment duration is usually short (less than 1 year). For ALL, the treatment is less intense but more prolonged ($2^1/2$ to $3^1/2$ years).

In all, the treatment regimens vary considerably depending on prognostic factors and the parameters being evaluated by the strategists' cooperative group (e. g., children's oncology group). The initial phase of treatment, induction, incorporates the use of a combination of antileukemic drugs at staggered intervals during a 4-week regimen (Table 24-2). This combination of drugs should rapidly destroy the leukemic cells, yet maintain the regenerative potential of the nonmalignant hematopoietic cells within the bone marrow. About 95% of patients with ALL will be in complete remission at day 28 of therapy.

The second phase of ALL treatment, consolidation, attempts to consolidate remission and intensify prophylactic central nervous system (CNS) treatment. Prevention of CNS relapse uses intrathecally administered chemotherapy (methotrexate with or without cytosine arabinoside and hydrocortisone) to destroy leukemic cells within the CNS. This chemotherapeutic agent is instilled directly into the lumbar spinal fluid because antileukemic drugs do not readily cross the blood-brain barrier. Intensive intrathecal chemotherapy to prevent CNS relapse has replaced cranial irradiation for patients in the good and intermediate-risk groups of ALL patients. However, both cranial irradiation and intrathecal chemotherapy are still used to prevent CNS relapse in high-risk ALL patients.

The third phase of treatment, interim maintenance, uses a combination of agents that are relatively nontoxic and require only monthly visits to the outpatient clinic. In most cases, another phase, delayed intensification, follows interim maintenance. This serves to intensify antileukemic therapy again after a short period of less intensive therapy. The addition of a late phase of intensive therapy substantially improves survival in patients with ALL. Following delayed intensification, therapy continues for 2 years for girls and 3 years for boys (maintenance phase), with chemotherapeutic agents given as in interim maintenance.

The prognosis for a child with acute leukemia has improved dramatically over the past 30 years.

TABLE 24-1 Childhood Leukemias

TYPE	AGE AND WHITEBLOOD CELL COUNT (WBC) AT ONSET	PROGNOSIS	TREATMENT
ACUTE LYMPHOCYTIC (ALL)			
	Peak age 3-6 years	>94% remission induction, varies with age and presenting WBC count	Vincristine, L-asparaginase, prednisone, 6-mercaptopurine, methotrexate, and others (including daunomycin, doxorubicin, cyclophosphamide, cytosine arabinoside [araC], dexamethasone)
Standard risk	1-9 years with WBC 50,000/mm^3	80%-90% EFS at 60 months	
Poor risk	>10 years or any age with WBC >50,000/mm^3	50%-60% EFS at 60 months	
Infants	<1 year, any WBC	20%-30% EFS at 60 months	
ACUTE NONLYMPHOCYTIC			
		80% remission induction; 40% EFS at 36 months; >95% EFS in children with Down syndrome	
Myelocytic (AML)	Older children and adolescents		Cytosine arabinoside, daunomycin, VP-16
Monocytic or myelomonocytic (AMoL; AMML)	Infants and young children more common		6-Thioguanine, idarubicin, dexamethasone; bone marrow transplantation*
Promyelocytic (APL)	Uncommon; seen in older children		Responds to transretinoic acid
Erythroleukemia	Very rare	Generally very poor	
Megakaryocytic leukemia	Very rare	Generally very poor	
CHRONIC MYELOCYTIC (CML)			
Juvenile, Philadelphia chromosome negative	Infants and toddlers very rare	Median survival <9 months	Bone marrow transplantation[s]
Adult, Philadelphia chromosome positive	Older children and adolescents	Chronic phase about 2-4 years; then death in blast crisis, usually of AML, which is very resistant to therapy	Imatinab (Gleevec), hydroxyurea; interferon alpha; busulfan, bone marrow transplantation'
CHRONIC LYMPHOCYTIC			
	Not seen in children		

Courtesy Dr. Randy Hock, Dr. Alexandra Cheerua.

EFS, Event-free survival.

Bone marrow transplantation for most subtypes of acute nonlymphocytic leukemia is considered the treatment of choice. Each sibling has one chance in four of being a perfect match. The patient is put into remission using standard chemotherapy before bone marrow transplantation. Of AML patients undergoing transplantation in first remission, 60% show no evidence of disease after 2 years.

'Bone marrow transplantation has been the treatment of choice for adult and juvenile chronic myelocytic leukemia. Response to standard chemotherapy is generally poor. In adults, imatinab, a selective inhibitor of tyrosine kinase at the breakpoint cluster region on the ABL gene (causative abnormality in CML), is highly effective and well tolerated in all phases of CML when taken orally. Clinical trials of imatinab in children are ongoing to determine its role in the treatment of childhood Philadelphia chromosome—positive CML. Transplantation for adult-type CML in first chronic phase using a matched sibling donor gives 60% disease-free survival at 2 years.

TABLE **24-2** Major Chemotherapeutic Agents and Their Side Effects

AGENT	NAUSEA*	BONE MARROW SUPPRESSION	HAIR LOSS	STOMATITIS/ ULCERS	RENAL	NEUROLOGIC
ALKYLATING AGENTS						
Carboplatin	Yes	Yes	Yes	No	No	No
Cisplatin	Yes (severe)	Yes	Yes	No	No	Peripheral neuropathy
Cyclophosphamide	Yes	Yes	Yes	No	No	Seizures (rare)
Ifosfamide	Yes	Yes	Yes	No	No	Somnolence
ANTIBIOTICS[t]						
Actinomycin D	Yes	Yes	Yes	Yes	No	No
Daunomycin[$]	Yes	Yes	Yes	Yes	No	No
Doxorubicin[t]	Yes	Yes	Yes	Yes	No	No
Idarubicin[t]	Yes	**Yes**	Yes	Yes	No	No
ANTIMETABOLITES						
Cytosine arabinoside (araC)	Yes	Yes	Yes	Yes	No	Cerebellar dysfunction
Methotrexate	Yes	Yes	Yes	Yes	Yes	Leukoencephalopathy
6-Mercaptopurine	Yes	Yes	Mild	Rare	No	No
PLANT ALKALOIDS						
Etoposide	Yes	Yes	Yes	No	No	No
Vincristine*	No	No	Yes	No; jaw pain	No	Peripheral neuropathy, seizures (rare)
MISCELLANEOUS						
Dexamethasone	No	No	No	No	No	Pseudotumor, psychoses
Prednisone	No	No	No	No	No	Pseudotumor, psychoses
L-AsparaginaseH	No	No	No	No	No	Seizures (rare), thrombosis

*Nausea is substantially alleviated by judicious use of antiemetics.
'These agents cause severe skin burns if they extravasate from the vein.
=High cumulative doses may result in cardiac toxicity.
Leukoencephalopathy is usually seen with methotrexate in conjunction with whole-brain radiation.
i[.-Asparaginase may cause pancreatitis and diabetes.

Thirty-five years ago, there would have been little need to discuss dental treatment for a child with leukemia because the disease was invariably fatal, in most cases within 6 months of diagnosis. Today, with the development of new and better antileukemia drugs, the use of intensive combination drug therapy, the incorporation of radiation therapy, and improvements in diagnostic techniques and general supportive care, complete cures are being achieved in the majority of patients.

Pretreatment prognostic factors identify patients who are likely to benefit from either standard or more intensive therapy. Factors that identify patients who are likely to benefit from the type of therapy just outlined, which causes relatively minimal toxicity, are patient age between 1 and 10 years, a white blood cell count of less than 50,000/mm^3, lymphoblast morphology that is not of the Burkitt type, and absence of certain cytogenetic abnormalities [t(4;11); t(9;22); or hypoploidyl in the lymphoblast.

Children with ALL who are at particularly high risk are those who are younger than 1 year of age at diagnosis, have a high white blood cell count, or have the cytogenetic abnormalities just mentioned. More intrusive treatment regimens are used for these patients.

The prognosis for children with ANLL has improved significantly over the past several years, with event-free survivals of approximately 50% (at least 3 years) for children receiving chemotherapy and somewhat higher percentages for patients undergoing allogeneic bone marrow transplantation after achieving remission with chemotherapy. The treatment regimens are intrusive and result in profound bone marrow suppression. These patients have severe prolonged neutropenia and often have severe mucositis.

ORAL MANIFESTATIONS OF LEUKEMIA

Pathologic changes in the oral cavity as a result of leukemia occur frequently. Oral signs or symptoms suggestive of leukemia have been reported in as many as 75% of adults and 29% of children with leukemia. The lower incidence of oral manifestations in children can be attributed in part to the early age at diagnosis and the high percentage of ALL in the pediatric age group. The incidence of ALL peaks at 3 years of age, when

preexisting inflammatory and degenerative changes are comparatively less frequent.

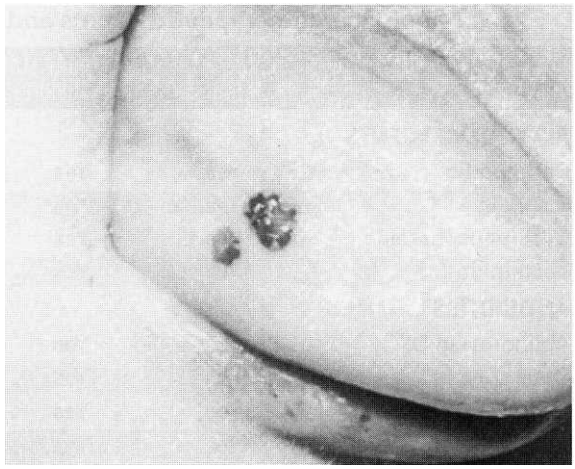
Abnormalities in or around the oral cavity occur in all types of leukemia, and in all age groups. However, oral pathoses are more commonly observed in acute leukemias than in chronic forms of the disease. Oral findings suggestive of leukemia are also more common in nonlymphocytic leukemias.

The most frequently reported oral abnormalities attributed to the leukemic process include regional lymphadenopathy, mucous membrane petechiae and ecchymoses, gingival bleeding, gingival hypertrophy, pallor, and nonspecific ulcerations. Manifestations seen occasionally are cranial nerve palsies, chin and lip paresthesias, odontalgia, jaw pain, loose teeth, extruded teeth, and gangrenous stomatitis. Each of these findings has been reported in all types of leukemia. Regional lymphadenopathy is the most frequently reported finding. Gingival abnormalities, including hypertrophy and bleeding, are more common in patients with nonlymphocytic leukemia, whereas petechiae and ecchymoses are more common in those with ALL.

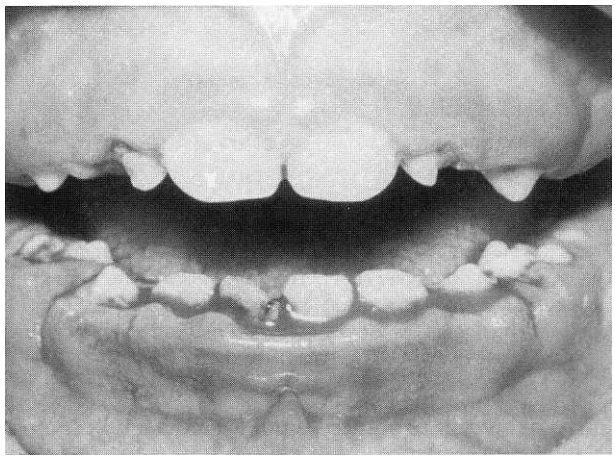
Like the systemic manifestations of leukemia, oral changes can be attributed to anemia, granulocytopenia, and thrombocytopenia, all of which result from the replacement of normal bone marrow elements by undifferentiated blast cells, or to direct invasion of tissue by these leukemic cells. Very high circulating white blood cell numbers in the peripheral blood can lead to stasis in small vascular channels. The subsequent tissue anoxia results in areas of necrosis and ulceration that can readily become infected by opportunistic oral microorganisms in patients with neutropenia. A person with thrombocytopenia, having lost the capacity to maintain vascular integrity, is likely to bleed spontaneously. Clinical manifestations of this are petechiae or ecchymoses of the oral mucosa or frank bleeding from the gingival sulcus (Figs. 24-1 and 24-2). The propensity for gingival bleeding is greatly increased in persons with deficient oral hygiene, because accumulated plaque and debris are significant local irritants.

Direct invasion of tissue by an infiltrate of leukemic cells can produce gingival hypertrophy (Fig. 24-3). Such gingival changes can occur despite excellent oral hygiene. Infiltration of leukemic cells along vascular channels can result in strangulation of pulpal tissue and spontaneous abscess formation as a result of infection or focal areas of liquefaction necrosis in the dental pulp of clinically and radiographically sound teeth. In a similar fashion, the teeth may rapidly loosen as a result of necrosis of the periodontal ligament.

Skeletal lesions caused by leukemic infiltration of bone are common in childhood leukemia. The most common finding is a generalized osteoporosis caused by enlargement of the haversian canals and Volkmann canals. Osteolytic lesions resulting from focal areas of hemorrhage and necrosis and leading to loss of trabecular bone are also common.



f **I G 24-1.** Two small ecchymoses on the lateral border of the tongue in a 10-year-old girl with acute lymphocytic leukemia. The platelet count was 22,000 cells/mm^3. *(Courtesy* Dr. *Bruce W. Vash.)*



f **G 24-2.** Spontaneous gingival hemorrhaging in a 2-year-old girl with acute myelocytic leukemia. The platelet count was less than 10,000 cells/mm^3. *(Courtesy* Dr. *Bruce W. Vash.)*

Evidence of skeletal lesions is visible on dental radiographs in up to 63% of children with acute leukemia. Manifestations in the jaws include generalized loss of trabeculation, destruction of the crypts of developing teeth, loss of lamina dura, widening of the periodontal ligament space, and displacement of teeth and tooth buds (Fig. 24-4). Because none of the oral changes is a pathognomonic sign of leukemia and all can be associated with numerous local or systemic disease processes, a diagnosis of leukemia cannot be based on oral findings alone. Such changes should, however, alert the clinician to the possibility of malignancy as the underlying cause.

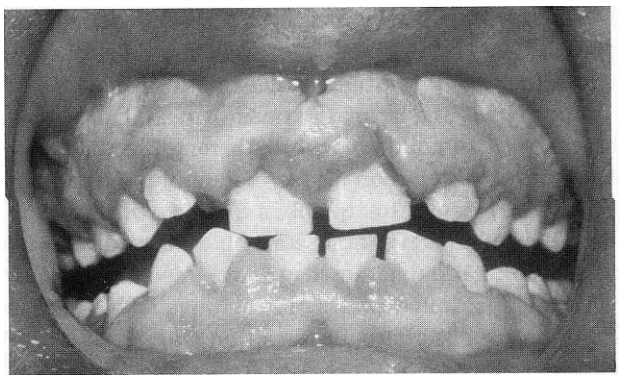


FIG. 24-3. Massive gingival hypertrophy, which biopsy results showed to be leukemic infiltrate, in a 16-year-old boy with acute monocytic leukemia. *(Courtesy Dr. Bruce W. Vash.)*



FIG. 24-4. Right lateral jaw radiograph of a 14-year-old boy with acute myelocytic leukemia. Notice generalized loss of trabeculation in the mandible, thinning and loss of lamina dura about the mandibular molar and premolar teeth, and destruction of the cortical lining of the mandibular and maxillary third molar tooth crypts. *(Courtesy Dr. Bruce W. Vash.)*

DENTAL MANAGEMENT OF PATIENTS WITH LEUKEMIA

Before any dental treatment is administered to a child with leukemia, the child's hematologist and oncologist or primary care physician should be consulted. The following information should be ascertained:

1. Primary medical diagnosis
2. Anticipated clinical course and prognosis
3. Present and future therapeutic modalities
4. Present general state of health
5. Present hematologic status

It is also important to establish, by consultation with the patient's physician, when dental treatment may be most propitious, and to schedule the patient's treatment accordingly. The proposed procedures should be discussed to determine if they are appropriate.

For a child whose first remission has not yet been obtained, or one who is in relapse, all elective dental procedures should be deferred. However, it is essential that potential sources of systemic infection within the oral cavity be controlled or eradicated whenever they are recognized (e.g., immediate extraction of carious primary teeth with pulpal involvement).

Routine preventive, restorative, and surgical procedures can usually be provided for a patient who is in complete remission yet is undergoing chemotherapy. The time when such procedures may be completed without complications will depend on the specific agents administered and the time of administration. Before the appointment—preferably the same day—a blood cell profile (complete blood count) and platelet count should be taken to confirm that the patient is not unexpectedly at undue risk of hemorrhage or infection. A patient who has been in complete remission for at least 2 years and no longer requires chemotherapy may be treated in an essentially normal manner. A preappointment blood workup is not necessary.

Pulp therapy on primary teeth is contraindicated in any patient with a history of leukemia. Endodontic treatment for permanent teeth is not recommended for any patient with leukemia who may have a chronic, intermittent suppression of granulocytes. Even with the most exacting technique, an area of chronic inflammatory tissue may remain in the periapical region of endodontically treated teeth. An area of low-grade, chronic inflammation in a healthy patient is generally well tolerated, but in an immunosuppressed, neutropenic patient the same area can act as an anachoretic focus with devastating sequelae. The decision to perform an endodontic procedure on a patient who has been in prolonged complete remission and who is not undergoing chemotherapy must be made by the dentist.

A platelet level of 100,000/mm' is adequate for most dental procedures (Table 24-3). Routine preventive and restorative treatment, including nonblock injections, may be considered when the platelet count is at least 50,000/mm'. With inadequate oral hygiene, unhealthy periodontal tissues, and the presence of local irritants, hemorrhage from the gingival sulcus may be observed when platelet counts are between 20,000 and 50,000/mm^3. Such hemorrhaging is usually noted only after manipulation of the tissues, such as during toothbrushing. If the platelet count is lower than 20,000/mm^3, all the intraoral mucosal tissues may show clinical evidence of spontaneous hemorrhaging (e.g., petechiae, ecchymoses, or frank hemorrhage). No dental treatment should be performed at such a time without a preceding prophylactic platelet transfusion. Good oral hygiene must be maintained while the

TABLE 24-3 Clinical Importance of Platelet Count*

COUNT (CELLS/mm³)	SIGNIFICANCE
150,000-400,000	Normal
50,000-100,000	Bleeding time is prolonged, but patient would tolerate most routine procedures
20,000-50,000	At moderate risk of bleeding; defer elective surgical procedures
<20,000	At significant risk for bleeding; defer elective dental procedures

Courtesy Dr. Thomas D. Coates.

*An absolute indication for platelet transfusion is significant *bleeding.* If the count is <20,000 to 30,000 cellslmm³, the patient should probably be given prophylactic platelet transfusion before dental procedures. Prophylactic platelet transfusions are given for platelet levels below 10,000 cells/mm³. Indiscriminate use of platelet therapy may lead to the development of antiplatelet antibodies.

TABLE 24-4 Clinical Importance of White Blood Cell Count

ANC*	SIGNIFICANCE
>1500	Normal
500-1000	Patient at some risk for infection; defer elective procedures that could induce significant transient bacteremia
200-500	Patient must be admitted to hospital if febrile and given broad-spectrum antibiotics; at moderate risk for sepsis; defer all elective dental procedures
<200	At significant risk for sepsis

Courtesy Dr. Thomas D. Coates.

*All values refer to absolute neutrophil count (ANC) computed as follows:

ANC = (% of polymorphonuclear leukocytes + % of bands) x total white count + 100

platelet count is at this level, but it may be necessary to discontinue the use of a toothbrush and to substitute cleaning with moist gauze wipes, supplemented by frequent saline rinses.

The absolute neutrophil count (ANC) is an indicator of the host's ability to suppress or eliminate infection. It is calculated using the following formula:

ANC = (% of neutrophils + % of bands) x total white count - 100

The clinical significance of the ANC is presented in Table 24-4. If the ANC is less than 1000/mm', elective dental treatment should be deferred. A leukemic patient with a low ANC may require prophylactic broad-spectrum antibiotic therapy before certain dental procedures. The patient's physician should be consulted regarding the appropriate drugs and dosages.

Infection is the primary cause of death in approximately 80% of children with leukemia. Bleeding is the second most common cause. Therefore the primary objective of dental treatment in a child with leukemia should be the prevention, control, and eradication of oral inflammation, hemorrhage, and infection.

Frequently, initial oral manifestations of bleeding or infection are observed in association with an unhealthy periodontium. In patients with leukemia who are neutropenic or who are being treated with corticosteroids, the true degree of periodontal inflammation or infection may be masked, because the cardinal signs of inflammation may not be apparent. There is a much greater propensity for gingival bleeding when the periodontium is unhealthy. When oral hygiene is neglected and local irritants are present, spontaneous hemorrhaging from the gingival sulcus may be observed if the patient is thrombocytopenic.

Needless to say, it is imperative that a patient who is diagnosed as having leukemia be enrolled in a good preventive dental care program in which special emphasis is placed on the initiation and maintenance of a comprehensive oral hygiene regimen. The use of a soft nylon toothbrush for the removal of plaque is recommended, even if the patient is thrombocytopenic. As long as the gingiva remains in a healthy state and its manipulation by brushing does not induce significant hemorrhage, it is not appropriate to discontinue the use of a toothbrush because of the platelet level alone. The practicality of flossing must be assessed on an individual basis.

It is important that significant local irritants, including orthodontic appliances, be removed. Scaling and subgingival curettage should not necessarily be perceived as elective dental treatment in all patients. This is especially true if the anticipated clinical course may place the patient at high risk of hemorrhage and infection. Patients with classic leukemic gingivitis experience varying degrees of discomfort. The use of warm saline rinses several times each day may assist in the relief of symptoms.

Erosive or ulcerative lesions are common in children with leukemia. These lesions are often associated with the use of certain chemotherapeutic agents (see Table 24-2), especially methotrexate and the anthracycline antibiotics daunomycin and doxorubicin. The lesions may be an early indicator of drug toxicity. After administration of the drug is discontinued, these lesions usually disappear within a few days. Treatment is directed toward the relief of discomfort (Table 24-5).

In a patient who is granulocytopenic, trauma may result in the occurrence of ulcerative lesions, especially along the lateral border of the tongue and buccal mucosa.

TABLE 24-5 Topical Obtundents for Oral Pain (Frequently Used at James Whitcomb Riley Hospital for Children and St. Vincent Children's Hospital)

COMBINATION	ADMINISTRATION	INDICATIONS
1 PART KAOPECTATE AND 1 PART BENADRYL ELIXIR	Swish 5-10 ml for 1 minute; then expectorate	Generalized stomatitis and mild discomfort
BENACORT-TETRASTAT (MARY'S MAGIC POTION) Hydrocortisone poser 60 mg Tetracycline 1.5 g Nystatin suspension 100,000 U/ml 60 ml Benadryl elixir qs 240 ml	Swish 5-10 ml for 1 minute; then expectorate	Generalized stomatitis and mild discomfort
PHILADELPHIA MOUTHWASH* Benadryl elixir 90 ml Maalox suspension 90 ml Viscous lidocaine 2% 90 ml Distilled water 180 ml Cherry or vanilla flavoring to taste	Swish 5-10 ml for 1 minute; then expectorate	Generalized stomatitis and mild discomfort
ORABASE WITH BENZOCAINE, OR VISCOUS LIDOCAINE 2%,* OR DYCLONINE HCl 0.5%	Apply locally to lesions with cotton swab	Discrete, painful lesions

*Use should be supervised to prevent lidocaine toxicity.

The clinical course of these ulcerations is generally benign, although the time necessary for healing may be prolonged. Use of topical obtundents for pain may be the only treatment indicated.

Infrequently, deep lesions will bleed spontaneously or as a result of trauma. Local measures, such as the topical application of either bovine thrombin or Avitene, and the placement of an oral adhesive for protection, may be beneficial.

In a patient who is physically debilitated or who is in relapse, septic, and severely granulocytopenic, ulcerative lesions require close observation. Such lesions may serve as a nidus for the proliferation of microorganisms, which can lead to potentially fatal viral, fungal, or bacterial infection. Therefore specimens from these ulcerative lesions should be cultured and subsequent sensitivity testing performed, and antibiotic therapy should be initiated or modified accordingly.

Candidiasis is common in children with leukemia. They are especially susceptible to this fungal infection because of (1) general physical debilitation, (2) immunosuppression, (3) prolonged antibiotic therapy, (4) chemotherapy, and (5) poor oral hygiene. The following topical use of nystatin can be particularly beneficial:

- Nystatin oral suspension, 100,000 U/ml.
- Swish 5 ml for 5 minutes and then swallow.
- Repeat every 6 hours. Continue for 48 hours after lesions disappear.

In more resistant cases of oral candidiasis, fluconazole may be useful, administered once daily either orally or intravenously (in young children 16 mg/kg on day 1, then 3 mg/kg to a maximum of 12 mg per day; in older children and adults 200 mg on day 1, then 100 mg).

For patients who are thrombocytopenic or at risk of intermittent episodes of thrombocytopenia because of chemotherapy or active disease, the dentist should avoid prescribing drugs that may alter platelet function, such as salicylates (aspirin).

BONE MARROW/STEM CELL TRANSPLANTATION

The transplantation of hemopoietic stem cells can be curative for a variety of disorders, including aplastic anemia, thalassemia, and severe combined immunodeficiency. Stem cell transplantation has also been shown to be effective as rescue therapy for patients undergoing extremely aggressive treatment for leukemia in which both lethal doses of chemotherapy and total-body irradiation are used to destroy all bone marrow elements, including normal marrow cells. Aggressive chemotherapy, total-body irradiation, and stem cell rescue are being used in experimental treatment protocols for children with very aggressive malignancies (e.g., advanced neuroblastoma and Ewing sarcoma).

As with any organ transplantation, hemopoietic stem cell transplantation candidates must have a compatible donor. The ideal donor, in most cases, is a healthy identical twin, which would result in a syngeneic recipient and donor. Next best is an HLA-compatible sibling yielding an allogeneic transplant. In the absence of a related HLA-compatible donor, stem cells from matched unrelated donors or from placental cord

blood can sometimes be used. In certain cases, autologous transplantation is performed in which the patient's own bone marrow or peripheral blood stem cells are harvested and stored and then returned to the patient when remission occurs and after intensive treatment.

In almost all organ transplantation procedures, host rejection of the transplanted organ and infection are prime reasons for treatment failure. An additional complication in allogeneic hemopoietic stem cell transplantation is graft-versus-host disease (GVHD) in which immunocompetent donor lymphocytes attack the recipient host cells.

During and after hemopoietic stem cell transplantation, the most common cause of serious morbidity and mortality is infection. The risk of infection from exogenous sources is reduced by good hand washing. Despite multiple-agent antibacterial, antifungal, and antiviral therapy, endogenous opportunistic organisms are usually the cause of life-threatening infection in hemopoietic stem cell transplantation patients, who, because of ablated bone marrow function, are unable to mount protective inflammatory and immune responses before engraftment of donor bone marrow.

ORAL COMPLICATIONS OF BONE MARROW TRANSPLANTATION

The oral complications of bone marrow transplantation differ from those seen during conventional therapy for malignant disease only in degree and duration. Oral ulceration, mucositis, and transient salivary gland dysfunction are frequent consequences of stomatotoxic chemotherapy and total-body irradiation. Minor trauma to atrophic mucous membranes often results in self-induced ulceration of the buccal mucosa, lips, and tongue. Thrombocytopenic gingival bleeding and bleeding from oral ulcerations are also frequently encountered.

Oral bleeding responds well to platelet transfusions and intravenous aminocaproic acid (Amicar). Topical application of Avitene (bovine corium collagen hydrochloride), powdered thrombin, and pressure are often effective for control of localized bleeding and often must be relied on in those cases in which the patient fails to respond to transfused platelets.

Oral ulceration and mucositis resolve with the return of bone marrow function and rising absolute neutrophil counts. Interim treatment is directed toward palliation and prevention of secondary infection. Any of the topically applied obtundent solutions or topical local anesthetics mentioned previously may be used. The efficacy of each will vary among patients, and in some cases only potent systemic analgesics will provide relief. In all cases, however, infection must be suspected, and specimens from all lesions should be cultured and growing organisms tested for antibiotic susceptibility.

GRAFT-VERSUS-HOST DISEASE

A major problem of allogeneic bone marrow transplantation is the development of GVHD. This disorder appears to result from an interaction between donor immunocompetent cells and recipient cells that display disparate antigens. The acute form of the disease involves the lymphoid system, skin, liver, and gastrointestinal tract. Cutaneous and oral mucosal involvement in chronic GVHD is common and is manifested as painful overt mucosal erythema, reticular or lichenoid eruptions, or desquamation and ulceration. Treatment with dexamethasone oral rinses may be helpful in cases of severe mucosal involvement in chronic GVHD. Mucosal involvement may be accompanied by salivary gland dysfunction with xerostomia, dysphagia, and dysgeusia.

Treatment of both acute and chronic GVHD is directed at correcting, masking, or preventing disordered immune regulation. Therapies with low- and high-dose steroids (prednisone, dexamethasone), antithymocyte globulin, cyclosporin, azathioprine, or methotrexate have been attempted with mixed results. Although acute GVHD can be rapidly fatal, chronic GVHD is usually transient and controllable with systemic therapy. The goals of therapy for mucosal and salivary gland manifestations, which relies on abundant rinses, are palliation and prevention of secondary infection.

Of note, the positive side of GVHD is an associated graft-versus-leukemia effect. In both acute and chronic GVHD, the immune response associated with GVHD appears to affect leukemia cells in that there is an inverse relationship between the severity of GVHD and the incidence of relapse.

PRETRANSPLANTATION PREPARATION

The bone marrow transplantation program at Indiana University Hospitals places a high priority on pretransplantation dental care and daily mouth care in the transplant unit. The primary goal of this oral health care protocol is to prevent, reduce in severity, or palliatively relieve oral problems involved in the oral care of patients receiving oncologic therapy. The integrity of the oral cavity is maintained by keeping the oral structures, both hard and soft tissues, clean, moist, and free of infection. The resultant decrease in septic episodes in the oral cavity should diminish the patient's morbidity and mortality because the mouth is the most common source of infection in these patients.

Four weeks before bone marrow transplantation all candidates for this procedure receive a thorough clinical and radiographic oral examination during the workup

phase initiated from the oncology physician's consult. The resultant dental treatment plan, along with the primary goals of oral health care, are discussed with the patient's oncologist, the patient, and the parents. Each patient is also informed of all potential oral complications of bone marrow transplantation and GVHD.

Along with professional preventive dental care, each patient receives instruction in daily mouth care, including brushing and flossing. The daily use of self-applied topical fluoride gel and Peridex rinse (every 12 hours) is recommended. Dental appointments are scheduled for any necessary periodontal, restorative, or oral surgery treatment, if indicated, to eliminate and control oral infections or conditions that may lead to episodic problems. Potential foci of infection or bleeding, such as pulpally involved teeth, periodontally involved teeth, partially erupted teeth, exfoliating primary teeth and carious teeth, or teeth with defective restorations, must be treated before admission to the transplant unit. Recommended treatment should ideally be completed at least 2 weeks before admission to allow for healing of violated tissues and the reestablishment of optimal oral hygiene.

Unfortunately, there are times when the patient's medical status necessitates admission for transplantation before all dental treatment can be completed. Therefore the minimum preadmission dental requirement is a mouth free of foci of acute infection and free of physical irritants. Each patient must receive thorough supragingival and subgingival scaling and prophylaxis; open carious lesions and carious lesions approaching the pulp must be restored, and abscessed teeth and teeth with severe periodontal involvement, as well as exfoliating primary teeth causing gingival irritation, must be removed.

ADMISSION AND NURSING INTERVENTIONS

Peridex has been shown to be effective in minimizing the septic episodes in the oral cavity when used with other chemotherapeutic agents. On admission, the patient is to use Peridex every 6 hours by brushing the teeth three times a day with a Peridex-soaked ultrasoft toothbrush (e.g., Sensodyne Gentle) and then rinsing the mouth with 10 to 15 ml of Peridex for 30 seconds three additional times a day (1 time during night hours). When the platelet count is less than 20,000/mm^3 and gum bleeding is apparent, a toothette should be used in place of a toothbrush. The patient may not eat or drink for 1 hour following the use of Peridex. Surgilube may be applied to the lips following each rinse session and every 4 hours for dryness.

Allogeneic and autologous transplant patients are started on antifungal prophylaxis with fluconazole (5 mg/kg per day by mouth, maximum dose 200 mg per day). The condition of the oral cavity should be monitored and charted on each shift. Doctors should be notified of any change in the oral cavity from the previous day or shift.

The treatment of mucositis is described in Box 24-1. Mild mucositis is defined as mild erythema, no mucosal ulcerations, and no pain; the patient is able to swallow, and no local analgesia is required. Moderate mucositis is defined as moderate erythema, multiple mucosal ulcerations with pain that may or may not require analgesics; the patient can swallow but it maybe painful. Severe mucositis is defined as erythema, multiple

O 0 S 24-1 Treatment of Mucositis and Candidiasis (Guidelines for Nursing Staff)

MILD MUCOSITIS

Continue same oral regimen and add 2% sodium bicarbonate.
Chart condition of oral cavity each shift; notify doctor of any change from the previous day or shift.

MODERATE OR SEVERE MUCOSITIS

Obtain order for pain medication to be administered either before oral care or as a continuous drip (i.e., morphine sulphate); may require bolus administration in addition to continuous drip.
Follow same regimen as with mild mucositis using both 2% sodium bicarbonate and Peridex with toothette.
Use Peridex every 4 hours; if patient is having difficulty rinsing with full-strength Peridex, dilute Peridex 1:1 with sterile water.

CANDIDA INFECTION

Contact dentist to examine and obtain a culture specimen. Hold one dose of Peridex and nystatin until culture is obtained.
Patient must be started on additional antifungal therapy once culture results shows evidence of yeast infection.
If oral candidiasis is documented by culture, nystatin should be applied with a toothette, swished for 30 to 60 seconds, then swallowed.
Chart condition of oral cavity; each shift should notify doctor of any change from the previous day shift.

ulcerations, severe pain requiring analgesia, and difficulty in or inability to swallow. Although the regimens shown are not always successful in cases of moderate *or* severe mucositis, consistent follow-through will minimize many signs and symptoms.

The presence of white patches (i.e., *Candida* infection) may occur. Care must be taken in obtaining culture specimens and providing specific antifungal therapy.

REMISSION PHASE

During the remission phase of post—bone marrow transplantation therapy, consistent oral hygiene should be provided by the patient or parent. For the first 90 days the dental staff should provide maintenance oral evaluations and preventive oral care reinforcement for the bone marrow transplantation patient when he or she returns for periodic hospital oncologic evaluations.

Afterward, the patient should be referred to his or her private dentist for continuity of oral health care. If the patient has no dentist, referral should be made to a dental diagnostic clinic at the patient's medical center for dental health status evaluation and dental care.

SOLID TUMORS

Solid tumors account for approximately half of the cases of childhood malignancy. The most common tumors include brain tumors, lymphoma, neuroblastoma, Wilms tumor, osteosarcoma, and rhabdomyosarcoma. Because many of the malignancies can involve bone marrow and their treatment with chemotherapy and radiation can suppress marrow function, many of the complications seen in acute leukemia are also seen with these patients. Bleeding diatheses and the propensity to infection are the most notable medical complications seen. In general, the dental management of patients with solid tumors is similar to that of patients with acute leukemia.

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CHAPTER

25

Growth of the Face and Dental Arches

DONALD J. FERGUSON

انتشارات
شایان نمودار

CHAPTER OUTLINE

THE NATURE OF GROWTH
- Basic Concepts of Human Growth
- Craniofacial Growth Principles
- Basic Concepts of Craniofacial Growth

CRANIOFACIAL PATTERN
- Ideal Paradigms for Dentofacial Pattern

GROWTH AND FACIAL PATTERN
- Consistency in Pattern Maturation
- Ideal Frontal Facial Pattern
- Ideal Facial Profile Pattern
- Maintenance of Overall Pattern
- Facial Growth Emulates General Somatic Growth

GROWTH AND PATTERN OF OCCLUSION

Consistency in Pattern Development
- Primary Dentition Terminus
- Opposing First Molars at Initial Contact
- Ideal Static Occlusion Pattern
- Maintenance of Overall Pattern

GROWTH AND DENTAL ARCH PATTERN
- Similar Stage Sequencing Ideal
- Dental Arch Pattern
- Tooth Size/Arch Size Ratio as Pattern Determinant
- Computation of Tooth Size/Arch Size Balance
- Compensations in Dental Arch Development
- Maintenance of Overall Pattern
- Effect of Environmental Factors on Dental Arch Pattern

SUMMARY

Historically, patient care in medicine and dentistry has been oriented toward the elimination of disease and the resolution of debilitating conditions. Competent care in dentistry today includes issues related not only to disease and functional disability but also to the patient's well-being. The appearance of the face and dentition is recognized with increasing frequency as a major factor in human psychosocial health.'

This chapter is about dental and facial malocclusion—the recognition and anticipation of malocclusion during the growing years. The dentofacial pattern can be easily and accurately assessed at chairside. In clinical terms pertinent growth issues are discussed in relation to how growth changes the pattern of the face, occlusion, and dental arches. Knowledge of pattern appraisal and growth can be integrated in coming to efficacious clinical decisions about a young patient. This chapter will enhance the reader's diagnostic and treatment planning skills with reference to malocclusion in the pediatric patient.

The clinician treating malocclusion is primarily interested in the growth and development of craniofacial tissues as they result in facial and dentoalveolar pattern. Our understanding of how genes express their influence on dentofacial pattern and how environment influences gene expression has advanced at a remarkable pace. How molecular mechanisms are implicated at a clinically relevant level, however, has yet to be elucidated. Mao has pointed out that what we understand about induced treatment effects at the macroscopic phenotype level has been described in moderate detail at the cellular level but is only beginning to be described at the level of protein and peptide production.[2] For this reason, this chapter discusses dentofacial growth and development at a macroscopic level, from the perspective of the practicing clinician.

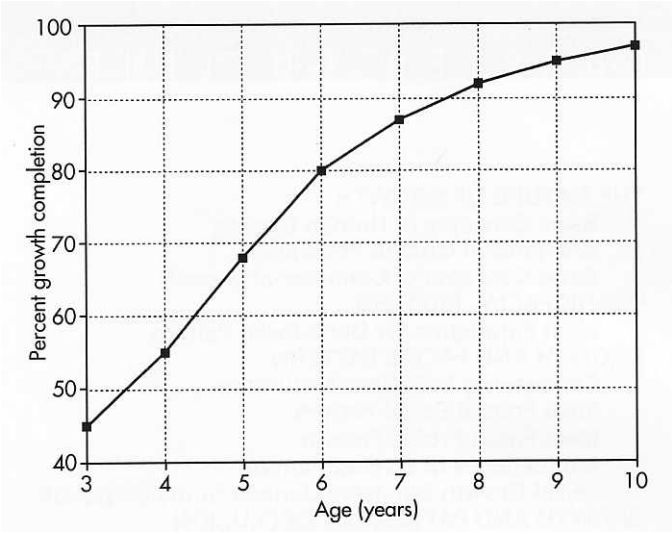


FIG. 25-1. Cumulative (distance) growth curve.

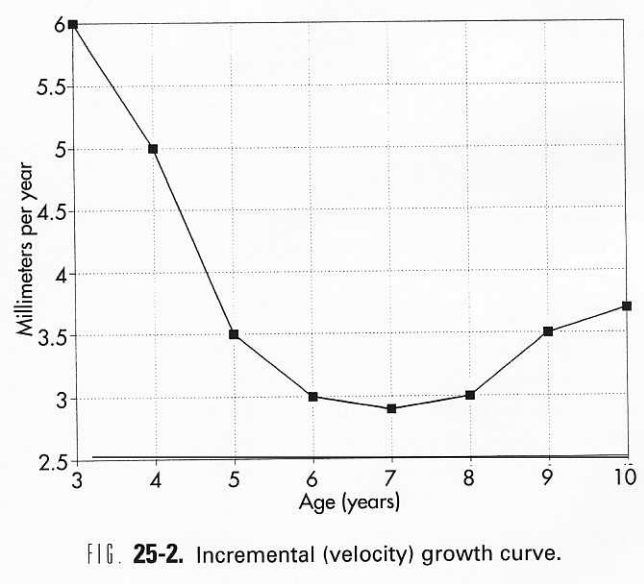


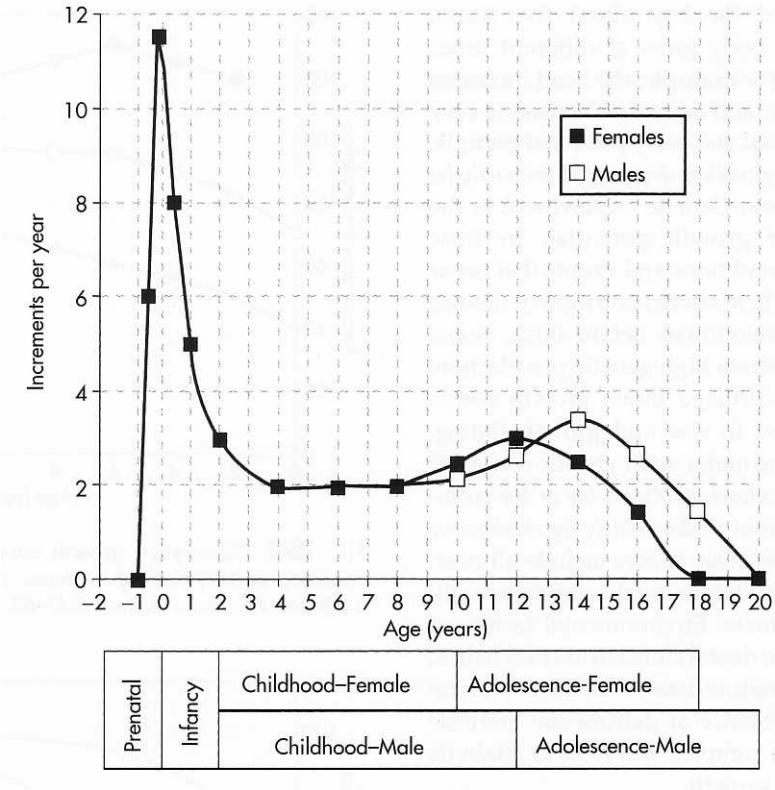
FIG. **25-2.** Incremental (velocity) growth curve.

THE NATURE OF GROWTH

Growth refers to an increase in anatomic size. Three parameters commonly used in growth literature to assess craniofacial size increase are magnitude, velocity, and direction. *Magnitude* refers to the linear dimension overall or the dimension of a part. *Direction* means the vector of size increase as might be described on a three-dimensional coordinate system. *Velocity* is defined as the amount of change per unit of time.

Size increase is typically illustrated in one of two ways. When growth is measured periodically and measurements are plotted as percentages of total growth, the result is a cumulative or distance curve (Fig. 25-1). A human postnatal cumulative curve is characterized by two plateaus and one period of accelerated growth. A second method of graphically demonstrating growth change is by use of an incremental or velocity growth curve (Fig. 25-2). A velocity curve plots growth increments (i.e., centimeters per year) as a function of time. Characteristic of an incremental human growth curve is rapid accelerating prenatal growth, rapid decelerating postnatal growth for the first 2 or 3 years, and a period of relatively slow incremental growth during childhood followed by growth acceleration for 2 or 3 years during pubertal adolescence.

Three observations are central to a clinically relevant understanding of growth. First, growth implies change, a transition from one condition to another. This broader meaning of growth helps define growth as a concept.



G 25-3. Incremental growth curve illustrating growth stages.

Conceptual growth refers to a passage from one anatomic form (i.e., size and. shape) to another. Transitions in functional stage or activity refer to development. Development, in biologic literature, usually means increased specialization or a higher order of organization and also connotes an interaction of functioning parts. Development means increased organization or specialization of functioning (physiologic) parts.

Growth is more readily understood when a physical pattern is used to describe the effects of growth change. Growth, by nature, is a relational concept. Without reference to a structural model, growth has little clinical utility or meaning. An effort is made in this chapter to discuss growth with reference to "ideal" facial, occlusion, and dental arch paradigms.

Growth is a complex phenomenon. The volume of information available on craniofacial growth is astonishing. Moreover, there is little consensus in the literature as to which data or combination of data are most useful to the practitioner committed to making competent decisions about direct patient care. In light of these prevailing circumstances, the following concepts and principles about craniofacial growth are presented in a way that should be clinically useful and difficult to refute. These postulates are derived and adapted in part from widely held tenets about general human growth and development presented by Valadian and Porter.' The discussion of general craniofacial principles is followed by application of the principles to three areas of craniofacial growth: the face, occlusion, and dental arches. Integrating growth principles in patient appraisal to enhance diagnostic and treatment planning efficacy is the goal of this chapter.

BASIC CONCEPTS OF HUMAN GROWTH

1. Growth disposition is similar for all healthy individuals.

Healthy individuals go through growth stages that are the same for everyone, according to Valadian and Porter.' The prenatal period, from conception to birth, averages 40 weeks in length. Infancy includes the first 2 years of life after birth, and childhood ranges from 2 to 10 years for girls and 2 to 12 years for boys. The length of adolescence is the same for both sexes but comprises different years, 10 to 18 years for females and 12 to 20 years for males (Fig. 25-3). Each growth stage is unique. Rate of size increase is most remarkable during the prenatal period and declines substantially during infancy. Generally, growth velocity plateaus during childhood and increases again during adolescence. All healthy individuals experience these growth cycles, although the various basic tissues and body parts are affected differently.

2. Different body parts increase in length at different rates.

From birth to adulthood, the head increases about twice in length, the trunk about three times, the arms

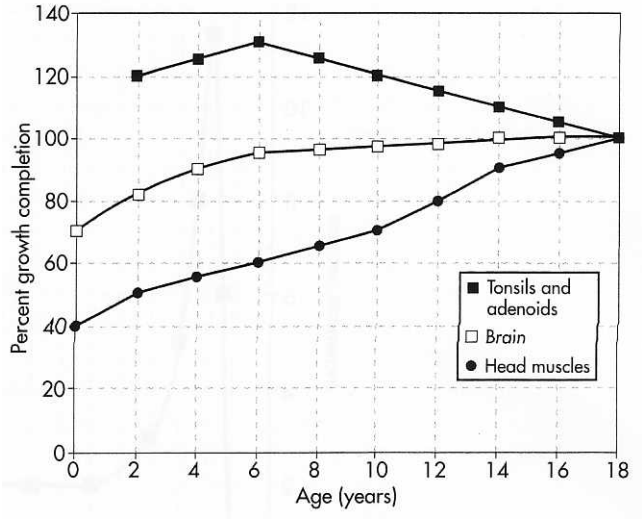
about four times, and the legs about five times. Different parts of the body grow at different times and at different rates. For example, the head increases in size very early in life, and its rate of increase is very rapid during the prenatal and early postnatal periods.

3. *The overall potential for growth is determined primarily by intrinsic or genetic factors.* Genetic endowment is the main determinant of growth potential. Intrinsic factors are also those conditions and events that occur from conception to birth. Maternal nutrition or disease can modify child development before birth. Some tissues tend to demonstrate high genetic predilection. Neural and primary cartilage tissue growth seems genetically predisposed in size and growth timing. Tooth size appears to be under strict genetic control.[4,5]
4. *The extent to which an individual attains his or her potential for growth is determined predominately by extrinsic or environmental factors.* Extrinsic factors include all postnatal environmental conditions, such as nutrition, illness, exercise, and climate. Environmental factors of particular interest to the dental clinician are oral habits, pathology, caries, premature loss of teeth, and metabolic disease. In the absence of detrimental extrinsic factors, the dentofacial complex will tend to attain its maximum potential in growth.

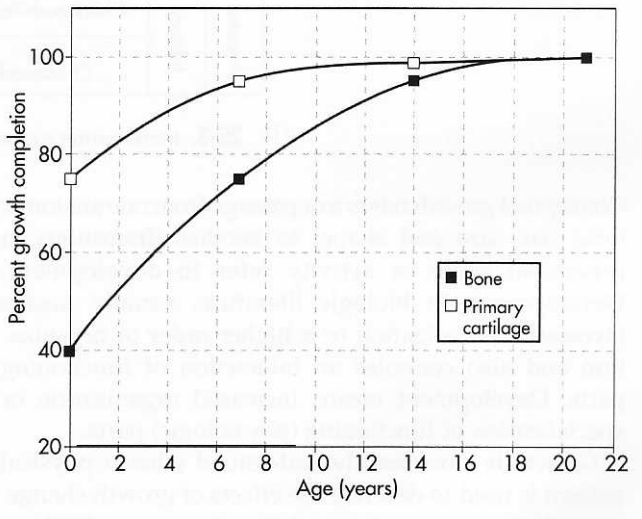
CRANIOFACIAL GROWTH PRINCIPLES

1. *The basic tissue types and functioning spaces that comprise the head and face are subject to growth timing differences.* The human head is composed of a variety of basic tissue types; the relative percentage of these types, at any given age, depends on timing of their growth. Neural tissue completes its growth at an early age. By contrast, general somatic tissues, such as muscle, bone, and connective tissue, mature at a slower rate. Neural tissue has attained about 60% to 70% of adult size by birth and its growth is about 95% completed by middle childhood. This is in sharp contrast to growth of other craniofacial soft tissues (Fig. 25-4). Muscle tissue is only 40% to 45% of its adult size by birth, and its growth is approximately 70% completed by 7 years of age. The size of cranio-facial lymphoid tissue (tonsils and adenoids) is about 125% of adult size at 5 years of age and decreases gradually to adulthood. Linder-Aronson and Leighton have shown that functional pharyngeal space increases in relation to decreased tonsillar-adenoid mass.'

   Growth timing of skeletal tissues also demonstrates variation. Craniofacial bone growth is about 45% completed by birth and 70% completed by 7 years of age. In contrast, primary cartilage of the head and face has achieved approximately 75% of adult size by birth and 95% by 7 years of age (Fig. 25-5). The small amount of primary cartilage remaining in the head and face after middle childhood, however, continues to grow through puberty.



f I c 25-4. Cumulative growth curve for craniofacial neural, muscle, and lymphoid tissues. (From Linder-Aronson S, Leighton BC: Eur J Orthod *5:47-58, 1983.)*



f I C. **25-5.** Cumulative growth curve for craniofacial bone and primary cartilage.

2. *Growth of primary cartilage and functioning spaces has a directing influence on cranio facial pattern change.* Primary cartilage is a tissue of particular interest to craniofacial growth theorists. According to Enlow and Hans, it is singular in form; has the capacity to grow from within (interstitial growth); is pressure tolerant, noncalcified, flexible, and nonvascular; and does not require a covering nutrient membrane for survival.' Primary cartilage found in the head and face is identical to the growth-plate cartilage of long bones. Scott contends that primary cartilage is

genetically predisposed, acts during growth as an autonomous tissue, and is able to directly influence the craniofacial pattern.[8]

Sperber documents that primary cartilage first appears in the head during the fifth week prenatally.`' By the eighth prenatal week, a cartilaginous mass called the *chondrocranium* is present and is the precursor to the adult cranial base and nasal and otic structures. By middle childhood, most primary cartilage is replaced by bone in a process called *endochondral bone formation.*

The overall growth-directing influence of primary cartilage on craniofacial pattern change is most profound in early life. By birth, cartilage comprises a substantial portion of the nasal septum and cranial base. Interstitial expansion of primary cartilage probably has a direct influence on the position of the maxilla by way of the septopremaxillary suspensory ligament, as suggested by Latham" and later contended by Gange and Johnston." The maxilla is most likely thrust downward and forward during infancy and early childhood. The contributions to midface growth of primary cartilage is greatly diminished after middle childhood.

The development of functioning spaces has also received considerable attention as a key concept among craniofacial growth theories."- The head carries out numerous functions. Some functions are more essential than others, but all require the development and maintenance of spaces. Neural integration is a critical function, and space is required for the brain and central and peripheral nervous system expansion. Respiration and deglutition are also essential to life and require development of nasal, pharyngeal, and oral spaces. Sight, olfaction, hearing, and speech are important but less critical craniofacial functions that also require development of functioning spaces for operation.

According to Moss, a likely craniofacial growth scenario of functioning space development in head and facial patterns includes the following sequence of events.[13] Rapid size increase of the brain during prenatal and early postnatal life thrusts the calvarial bony plates outward and the midface forward. Birth invokes a set of functional processes previously not essential for life (i.e., breathing and swallowing). Repositioning of the mandible and tongue takes place to ensure patency of nasal-oral-pharyngeal spaces. The mandible is depressed and thrust forward for these functions to be supported and maintained.

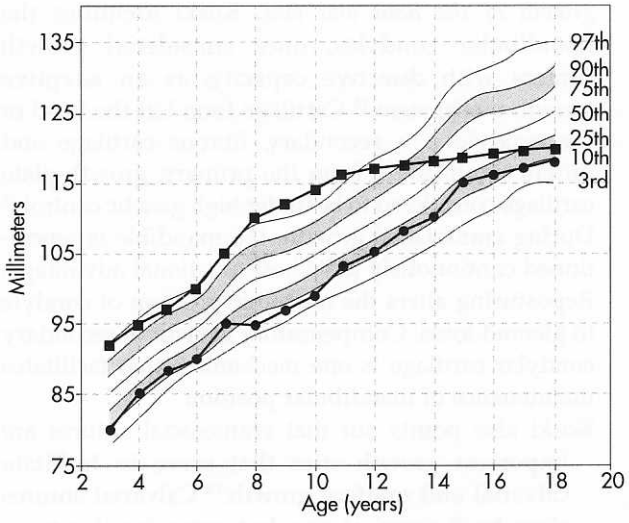
3. Mandibular condylar cartilage, craniofacial sutures, and appositional-resorptive bone change facilitate pattern growth of the head and face. Koski identifies the mandibular condyles, once considered growth centers with directive capacity, as an adaptive growth mechanism.[14] Cartilage found at the head of the condyle is a secondary, fibrous cartilage and differs significantly from the primary, growth-plate cartilage considered to be under high genetic control." During craniofacial growth, the mandible is repositioned continuously to its best functional advantage. Reposturing alters the anatomic position of condyle to glenoid fossa. Compensatory growth of secondary condylar cartilage is one mechanism that facilitates maintenance of mandibular position.

Koski also points out that craniofacial sutures are important growth sites that serve to facilitate calvarial and midface growth.[14] Calvarial sutures close by 5 years of age, but some facial sutures remain patent through puberty. Craniofacial bones are thrust apart by primary cartilage and functioning space increases. Sutures enable osseous deposition at bone edges, which allows bones of the face and skull to adapt.

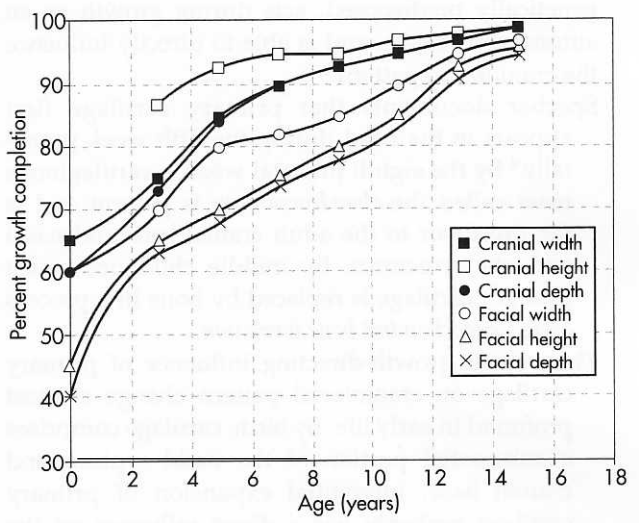
Enlow has shown that bone, unlike primary cartilage, is subject to environmental controls.'[b] Bone may assume many forms during growth; it is pressure sensitive, calcified, vascular, and relatively inflexible, and requires a covering membrane for survival. The craniofacial skeleton increases in size by way of surface addition only and increases in shape through differential appositional-resorptive bone growth. This differential growth process accounts for a considerable amount of size increase after middle childhood.

One growth theorist, Dr. Melvin Moss,[12] conjectures that the general somatic tissues (i.e., bone, muscle, and connective tissue) demonstrate growth change as a consequence of supporting the functioning operations of the head. Indeed, the research evidence of Linder-Aronson" and of Harvold et al[18] are convincing in that bone and muscle, as basic tissues, are adaptive and compensatory in nature. Understanding bone and muscle growth may come through understanding the temporal development of functioning spaces and the effects of interstitial cartilage expansion on surrounding tissues.

4. *Growth of the head and face tends to demonstrate relative equivalency*. Humans tend to grow with relative consistency. A percentile growth chart is a valuable instrument for assessing growth consistency over a time period (Fig. 25-6). Percentile charts are customarily divided into the following seven percentile levels: 97th, 90th, 75th, 50th, 25th, 10th, and 3rd. Healthy children tend to maintain a similar percentile level through successive stages of development.



[**I G 25-6.** Cumulative growth chart for male face height (hard tissue nasion to menton), illustrating seven percentile levels. • Relatively normal growth; ■ deviation of several percentile levels during growth, suggestive of abnormalcy. *(From Broadbent BH Sr, Broadbent BH Jr, Golden WH:* Bolton standards of dentofacial developmental growth, *St Louis, 1975, Mosby).*



f 1 G 25-7. Cumulative growth curve for calvaria and face in width, height, and depth. *(From Scott JH: Proc R Soc Med* 47: 5, 1954; *Meredith HV: Growth* 24:215-264, 1960; *and Ranley DM: A* synopsis of craniofacial growth, *New York,* 1980, *Appleton & Lange.)*

Deviations during growth of more than two percentile levels may indicate developmental problems, such as illness or disease.

Attributes (craniofacial parts) that are structurally related will also maintain a consistent relationship throughout successive stages of growth after infancy. Enlow[16] identifies the dental arches of the maxilla and mandible as an example of a structural part-counterpart relationship. An Angle class II skeletal pattern at 3 years of age will be maintained into adulthood without corrective therapy. Both dental arches in healthy individuals tend to increase in size at about the same rate. Hence, balanced or equivalent growth tends to maintain architecturally related structures of any craniofacial pattern that is present after 2 years of age. BASIC

CONCEPTS OF CRANIOFACIAL GROWTH 1.

Different parts of the craniofacial complex grow at different times. The head takes on appearance characteristics unique to each particular growth stage. Different parts of the face experience differences in growth timing as well. The infant has a disproportionately large calvaria and forehead compared with the adult because growth of the neural tissue takes place earlier in life than facial growth.

Size increase of the face and calvaria in the three spatial planes is a differential growth process. Scott,'`' Meredith,° and Ranly' have contributed to an understanding of this process. By birth, the cranial height dimension has attained about 70% of its adult status; cranial width, 65%; and cranial length or depth, 60% (Fig. 25-7). In contrast, only 40% of facial height and 45% of facial length (depth) has been achieved by birth. Face width (i.e., bizygomatic and bigonial), on the other hand, has attained about 60% of adult stature. Growth in face width actually falls between the classic neural and general somatic growth curves.

After birth, a pattern in facial growth timing emerges. The anterior cranial base completes most of its growth during infancy and early childhood, but frontal and nasal bones continue outward expansion through appositional-resorptive bone growth.'-' Growth magnitude and duration are greater for the anterior maxilla than for the forehead but less than for the anterior mandible. The posterior face demonstrates the greatest incremental growth during late puberty.

2. Differences in growth size, direction, velocity, and timing are observed among individuals. Bergersen has also noted large variations in growth patterns among individuals and has shown that any measured attribute will demonstrate a range of expression about a central tendency.[23] Incremental growth curves for healthy males and females will demonstrate the same general disposition but may show marked differences in maturation timing (Fig. 25-8). Generally, females mature 2 years earlier than males, but Valadian and Porter have indicated that variations are so great that an early-maturing boy may mature earlier than a late-maturing girl.' Males tend to grow larger in size than females.

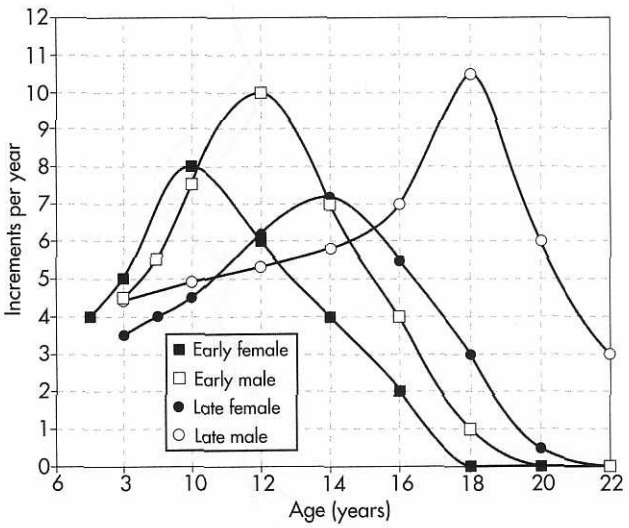


FIG. **25-8.** Incremental growth curves for early- and late-maturing males and females.

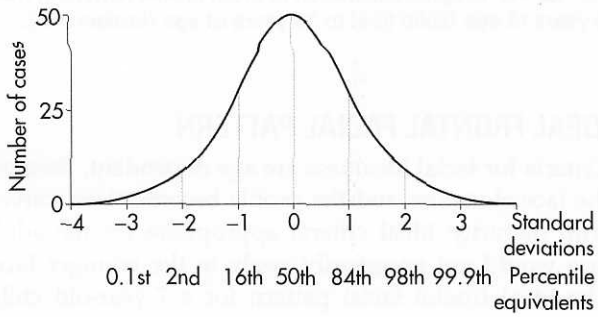


FIG. **25-9.** Normal distribution curve illustrating standard deviations and percentile equivalents.

3. The heads and faces of no two humans are exactly the same. Brodie pointed out that no two humans are exactly the same.[21] This fact is no more clearly evident than when one compares, at any given age, a measured attribute shared by healthy individuals. Most attributes have a range of expression that can be graphically illustrated by a normal distribution curve (Fig. 25-9).

If the same attribute were measured in a population of individuals, the most frequently occurring value (mode), middle value in the series (median), or arithmetic average of all the measured values combined (mean) would represent the central tendency of the population. Central tendency is often referred to as *normalcy*. Another way to describe attribute distribution is by using percentile equivalents. The 50th percentile indicates the center of the distribution, the 25th percentile the lower one fourth, and so on.

A third statistical parameter often used in growth literature to indicate distribution is the standard deviation. A standard deviation (SD) of ± 1 includes about 68% of the entire population; ± 2 SD and ± 3 SD are equivalent to approximately 95% and 99% of the distribution, respectively. The mean values and SDs for a normative population are invaluable aids in describing a patient's condition. By comparing a patient's value to a population value for the same trait, the clinician can make statements about relative largeness or smallness. Generally, measurements beyond ± 2 SD are considered clinically important because those values fall outside 95% of the population on which the normative value is based.

References will be made in the rest of this chapter to craniofacial growth principles and concepts in discussing growth of the face, occlusion, and dental arches.

CRANIOFACIAL PATTERN

In clinical assessment and treatment planning for the young patient, information about growth is often not considered to the degree that it should be. Craniofacial growth issues can be made more central to patient care concerns when a physical model is used to help visualize growth effects. For this reason, a particularly strong effort is made here to define physical craniofacial pattern.

There are two methods commonly used in dentistry to gather information about craniofacial pattern. One method is to examine the patient physically at chairside. Information collected in this fashion is based on criteria contrived and established in the practitioner's mind. The second method is to analyze dental records. Historically, cephalometric analysis has been a particularly useful tool for collecting objective information about craniofacial patterns. Generally, the patient's radiographic values measured on the cephalogram are compared with normative values derived from a population database. In this way, degrees of normalcy can be estimated by the clinician. One database is unique in its composition in that only individuals presenting with optimal or ideal craniofacial pattern were included in the study." This unique conceptual approach to defining craniofacial pattern enables the practitioner to make assessments about patient optimality. Patient-measured values are compared with values from cephalograms that have relatively ideal patterns. Cephalometric analysis is discussed in Chapter 26. Darwis, Messer, and Thomas suggest that using a combination of methods, such as three-dimensional facial morphometry and Fourier analysis, can provide a more comprehensive knowledge of growth and development of craniofacial structures and thus may allow improved prediction of clinical outcomes." Fourier analysis is a mathematical curve-fitting procedure that can represent boundaries so that the outlines of objects can be addressed.

IDEAL PARADIGMS FOR DENTOFACIAL PATTERN

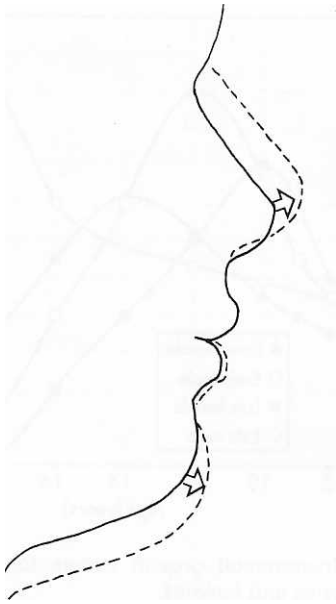
Standards for chairside facial appraisal have been offered by Ackerman and Proffit,[27] Angle," Bell, Proffit, and White,'" Cox and van der Linden,[30] Lucker, Ribbens, and McNamara,' and Patterson and Powell[31] Most of these physical appraisal models refer to the adult face. Horowitz and Hixon[32] describe idealized facial pattern as "the way things ought to be." Models available for examining the face espouse an assessment of proportion, balance, and harmony—concepts that help define overall facial attractiveness. The concept of an ideal face can be a useful clinical tool if it is used properly and its limitations are acknowledged. The first limitation is the fact that an ideal has little or no biologic basis. Biologic data can neither refute nor support the contention that the face should be ideal. Second, faces do not need to be ideal to work properly; ideal pattern, for the most part, has little connection with physiologic function. Third, an ideal model is simply a mental construct, a fiction. The words *ideal paradigm* mean "perfect example."

A perfect example can, on the other hand, be a powerful diagnostic and treatment-planning tool. The patient's facial pattern can be compared with criteria for idealness, the differences noted, and hence a problem list constructed. Criteria for an ideal face can help organize a vast array of information that is readily available to the clinician through physical observation. An ideal facial paradigm can serve as a treatment-planning tool as well. Although the concept of an ideal face is fictitious and biologically unsupported, it can serve as a guide by providing an example toward which treatment may be directed. Ideal paradigms for dental occlusion and dental arch pattern are also represented in dental literature; good examples may be found in the works of Angle,[z"] Andrews,[33] and Roth.[31] The purposes served by these paradigms are the same as for ideal facial models; they are powerful diagnostic and treatment-planning aids.

GROWTH AND FACIAL PATTERN

CONSISTENCY IN PATTERN MATURATION

Following birth, the face increases in size to a greater extent than does the calvaria. Bell, Proffit, and White propose that, by adulthood, the ideal face should be equally proportioned in forehead, midface, and lower face heights.[2"] Enlow has demonstrated that the facial profile flattens as the face ages. Nose and chin become more prominent and lips less pronounced.[3"] (Fig. 25-10) Every healthy individual, regardless of the overall craniofacial pattern, experiences profile flattening and face height increases relative to cranium.



HI; **25-10.** Graphic illustration of facial profile flattening from 6 years of age *(solid line)* to 18 years of age *(broken line).*

IDEAL FRONTAL FACIAL PATTERN

Criteria for facial idealness are age dependent. Because the face elongates and the profile becomes less convex with maturity, ideal criteria appropriate for the adult face would not necessarily apply to the younger face. The ideal frontal facial pattern for a 7-year-old child might include the following criteria (Fig. 25-11):

1. Right and left face halves are symmetrical.
2. Glabella (midpoint between eyebrows) to subnasale (point where columella merges with upper lip) equals subnasale to menton (inferior aspect of chin).
3. Subnasale to lower border of upper lip represents one third the distance from subnasale to menton.
4. Upper central incisor edge is 2 mm inferior to lower border of upper lip.
5. Alar base width equals inner canthal width.

IDEAL FACIAL PROFILE PATTERN

Use of a reference plane is very helpful for evaluation of the facial profile at chairside. The Frankfort horizontal plane is an anthropometric reference line frequently used for analysis of the lateral face. It is defined by Farkas as the superior limit of the external auditory meatus and the palpated border of the infraorbital bony rim.[36] A second reference line constructed perpendicular to the Frankfort horizontal plane and through the glabella (FHP) has been used in lateral profile assessment by Legan and Burstone.[37]

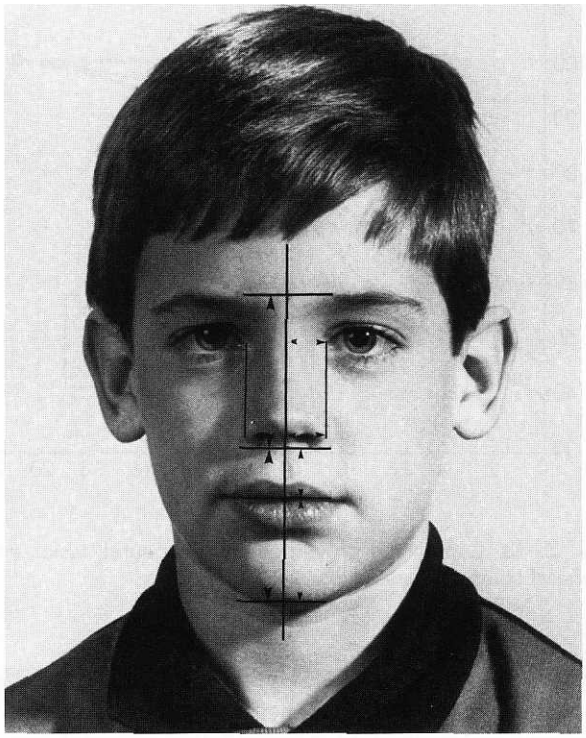


FIG. **25-11.** Ideal frontal facial pattern for a 7-year-old child.

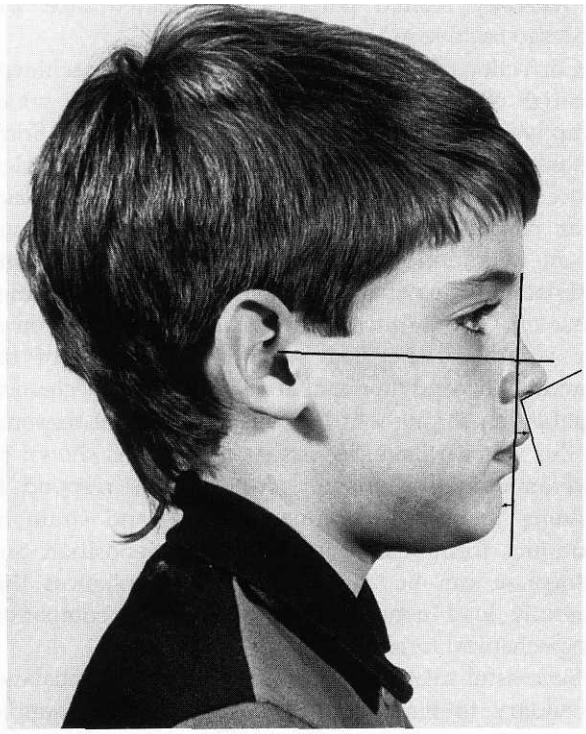


FIG. **25-12.** Ideal profile facial pattern for a 7-year-old child.

The ideal profile pattern for a 7-year-old child might include the following criteria (Fig. 25-12):

1. Chin 5 mm behind FHP
2. Most anterior aspect of lower lip on FHP
3. *Most anterior aspect of upper lip* 5 mm ahead of FHP
4. Nasolabial angle of 100 degrees
5. No more than 2 mm lip separation when relaxed

MAINTENANCE OF OVERALL PATTERN

The overall pattern presented by the individual at an early age will be maintained into adulthood. Although every individual experiences profile flattening and facial elongation as the face matures, Enlow et al demonstrated that the magnitude of these changes is not great enough to offset disharmonies in overall facial structure.[3"] Discrepancies between the position of the maxilla and mandible persist throughout life unless clinical therapy is employed to rectify the disharmonies.

At chairside, disharmony between the maxilla and the mandible can be simply and readily identified. A list of differences can be formulated by comparing the patient's facial measurements with the criteria of an ideal face. The differences serve as a patient problem list. Adding average growth change (i.e., magnitude, direction, and velocity) to the pattern presented by the individual will give an estimate of how facial patterns will look at a later age. This growth scheme is known as *a mean-change-expansion scheme.*[32] Balbach demonstrated it to be the most useful to predict the effects of growth on facial pattern.[39] The mean-change-expansion scheme is useful f or evaluation of almost all patie»ts zot~tinely seen in the dental office. Balanced or average growth affecting all aspects of the head and face relatively equally, however, cannot be assumed for all patients. The heads and faces of individuals who have some craniofacial congenital anomalies, hypoplastic defects, or acquired deformities that alter primary or compensatory cranio-facial growth mechanisms do not grow in a typical manner. A discussion of growth in these individuals is beyond the scope of this chapter.

Because growth change in healthy children affects the face in a relatively consistent and predictable way, the key to facial diagnosis and treatment planning is the clinician's ability to identify and diagnostically describe facial pattern. Identification of balanced, proportional facial pattern, as well as recognition of facial imbalance, should be routine during patient assessment. The use of criteria related to ideal facial pattern can be helpful.

The goal in treating facial imbalance in children is to establish architectural balance in the facial pattern. If corrective measures include compensation for the effects from treatment rebound or relapse, the facial pattern established by therapy will be maintained. As the face continues to grow and increase in size, all

structurally related parts of the treated face will undergo relative growth equality.

Correction of facial imbalance in the child is achieved through clinical manipulation of the means by which adaptive, compensatory facial growth occurs. Some sutures of the upper face remain patent into adolescence. Application of forces through orthopedic headgear, controlled in direction and amount, can result in an alteration of maxillary growth direction and ultimately of maxillary position. Also maxillary transverse size can be increased by judicious expansion of the palatal suture. The secondary cartilage of the mandibular condyle remains responsive to mechanical stimulation throughout life, but appositional response of this fibrocartilage decreases with age, as shown by McNamara and Carlson."[0] Facial bones respond to changes in microenvironmental stress and strain by changing form. Patterns of osseous deposition and resorption can be altered by using appliances that carefully load bone with physiologically compatible biomechanical forces.

Successful treatment of a child with facial imbalance secondary to mandibular retrognathia, for example, involves manipulation of several growth mechanisms. Mandibular anterior repositioning with a functional appliance probably affects many sites. Graber and Swain" believe that modification of the dentofacial complex occurs by the following means:

1. Condylar growth (secondary cartilage growth)
2. Glenoid fossa adaptation (apposition-resorption bone growth)
3. Elimination of functional retrusion
4. More favorable mandibular growth direction
5. Withholding of downward and forward maxillary arch movement (apposition-resorption bone growth)
6. Differential upward and forward eruption of lower buccal segment (apposition-resorption bone growth)
7. Orthopedic movement of maxilla and upper dentition (maxillary suture system growth)

FACIAL GROWTH EMULATES GENERAL SOMATIC GROWTH

The degree to which the facial pattern can be altered through biomechanical therapy depends on the amount of growth potential remaining. In general, the magnitude of facial pattern alteration possible is inversely proportional to age; the older the individual, the less the facial pattern can be therapeutically modified. The opportunity to alter compensatory, adaptive growth mechanisms is also greater in a rapidly growing individual. The adolescent growth spurt is characterized by increased growth velocity at about 10 to 12 years of age for girls and 12 to 14 years of age for boys. The maximum velocity or peak height velocity of growth is attained approximately 2 years after pubertal onset. Cumulative facial growth closely parallels general somatic growth (Fig. 25-13). Analysis of skeletal hand development can be helpful in estimating general skeletal maturation and, hence, facial skeletal maturation. It is relevant to evaluate a child's maturity in direct relation to the child's own pubertal growth spurt to assess whether maximum pubertal growth is imminent, has been reached, or has been passed.

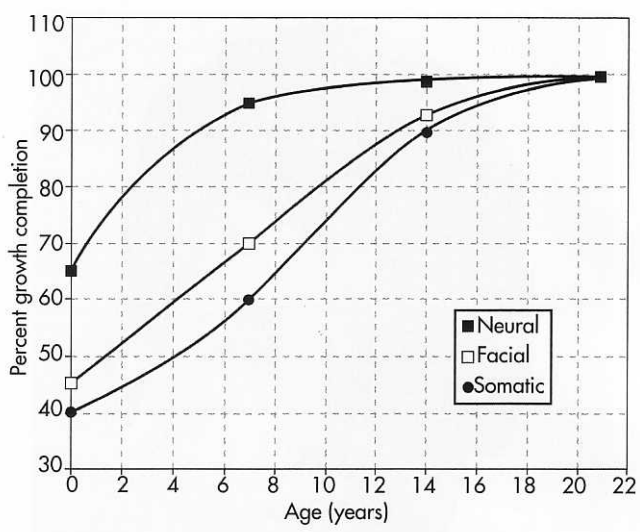


FIG. **25-13.** Cumulative growth curves for neural, facial, and general somatic tissues.

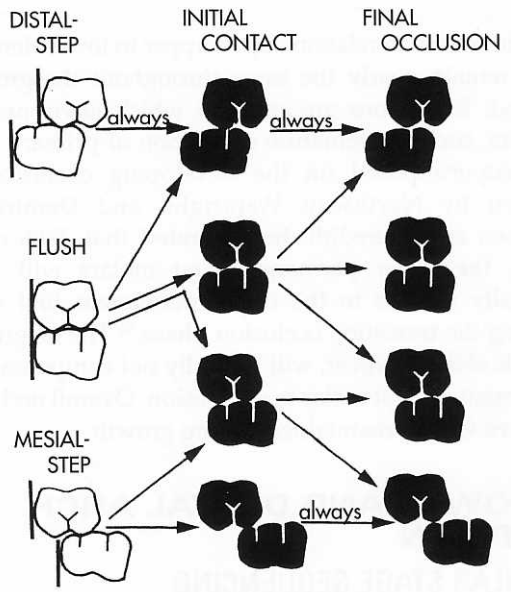
GROWTH AND PATTERN OF OCCLUSION

CONSISTENCY IN PATTERN DEVELOPMENT

Usually, no teeth are clinically visible at birth. Leighton has shown that the upper anterior gum pad (intercuspid width) is typically wider than the lower anterior pad, and the upper anterior gum pad protrudes (overjet) about 5 mm relative to the lower anterior gum pad." The upper anterior gum pad usually overlaps (overbite) the lower anterior pad by about 0.5 mm. In the first 6 months of postnatal life, there is marked palatal width increase, and the overjet decreases rapidly.

PRIMARY DENTITION TERMINUS

By 3 years of age, the occlusion of 20 primary teeth is usually established. The relationship of the distal terminal planes of opposing second primary molar teeth can be classified into one of three categories (Fig. 25-14). A flush terminal plane (flush terminus) means that the anterior-posterior positions of the distal surfaces of opposing primary second molars are in the same vertical



f **I G 25-14.** Graphic illustration of permanent first molar occlusion development. Outlined crown images represent three terminal plane relationships of primary second molars at about 5 years of age. Darkened images represent various permanent first molar relationships at initial occluding contact (about 6½ years of age) and at full occlusion contact (about 12 years of age). *(From Arya BS, Savara BS, Thomas DR:* Am J Orthod *63:610-621, 1973; Carlsen D8, Meredith HV:* Angle Orthod *30:162-173, 1960; and Moyers RA:* Handbook of orthodontics, *ed 3, Chicago, 1973, Mosby.)*

plane. A mesial-step terminus is defined as a lower second primary molar terminal plane that is mesial to the maxillary primary terminus. Distal-step terminal plane is descriptive of the situation in which the mandibular second primary molar terminus is distal to the upper second primary molar terminus.

Statistical studies of primary terminal plane status report that 49% of the time, the terminal plane of the lower primary second molar is mesial to the upper terminus (mesial step); the lower terminus is flush with the upper terminus 37% of the time; and the distal-step primary terminus is seen in approximately 14% of cases. These data are derived from studies reported by Arya, Savara, and Thomas" and by Carlsen and Meredith."

OPPOSING FIRST MOLARS AT INITIAL CONTACT

The permanent first molars are clinically visible at about 6 years of age and are the first permanent teeth to emerge. The relationship of permanent first molars when initial occluding contact occurs during eruption may be represented by one of four categories (see Fig. 25-14). A class I relationship means that the mesial-buccal (m-b) cusp of the upper permanent molar contacts at or very near the buccal groove of the lower permanent first molar. This occurs approximately

TABLE 25-1 Incidence of Terminal Molar Relationships at Three Stages of Occlusion Development

PRIMARY TERMINAL PLANE AT AGE 5 YEARS	INITIAL PERMANENT FIRST MOLAR OCCLUSION AT AGE 6½ YEARS	FINAL OCCLUSION AT ABOUT AGE 12 YEARS
	1% Class III	3% Class III
49% Class I (ms)	27% Class I	59% Class I
37% Flush	49% End-on	
14% Class II ids)	23% Class II	39% Class II

From Arya BS, Savara BS, Thomas DR: *Am J Orthod* 63:610-621, 1973; Carlsen DB, Meredith HV: *Angle Orthod* 30:162-173, 1960; HEW reports: *JClin Orthod* 12:849, *1978.*
ms, Medial step; *ds,* distal step.

55% of the time. An end-on relationship means that m-b cusps of both molars oppose one another. The incidence of this situation is about 25%. A class II relationship, occurring 19% of the time, is one in which an upper m-b cusp is anterior to the lower m-b cusp. Class III represents the situation in which an upper m-b cusp is distal to the lower buccal groove. This occurs in only 1% of the population.`' Table 25-1 shows the incidence of medial-step, flush, and distal-step primary terminus and end-on, class I, class II, and class III permanent first molar occlusions during three stages of occlusion development."'

IDEAL STATIC OCCLUSION PATTERN

The concept of ideal occlusion development has been described by Friel'[6] and by Lewis and Lehman.[47] Sanin and Savara have also shown that, to a considerable extent, ideal occlusion at a young age predisposes to an ideal adult occlusion.[45] The most desirable occlusion in the permanent dentition is a class I interdigitation, and certain features in the primary and mixed dentitions, if observed accurately, can provide clinical clues as to whether a class I relationship of the dentition will eventually develop.

The major difference between ideal adult and child occlusions is the teeth present. By 7 years of age, the primary central and lateral incisors have been or are in the process of being replaced by their permanent successors, and the permanent first molars have already erupted. The primary dentition remaining usually includes the canine and first and second molars of both arches. Criteria for ideal dental occlusion for a 7-year-old child might include the following:

Class I molar and canine interdigitation
2-mm anterior and posterior overjet 2-mm anterior overbite
Coincident dental midlines

MAINTENANCE OF OVERALL PATTERN

Gum pad relationships at birth cannot be used as reliable diagnostic criteria for predicting subsequent arch relationship. The primacy of life-supporting functions (i.e., respiration and swallowing) is so great at birth that major unpredictable adjustments in maxillary and mandibular positions take place in the first few years of life. By 3 years of age, however, the relationship of maxilla to mandible is well established, and overall maxillomandibular pattern does not change significantly thereafter.

One key diagnostic feature regarding future occlusion status is the relationships of the primary terminal planes. The likelihood of developing a class I relationship in the permanent dentition is greatest when a mild mesial-step terminus exists during the primary dentition stage (see Fig. 25-14). If an exaggerated mesial step exists, a class III permanent molar relationship will develop. The possibility that a class I relationship will develop from a distal-step primary terminus is virtually nonexistent. Hence, the presence of a distal step is highly predictive of a developing class II permanent molar relationship.

Another important diagnostic feature that is predictive of later occlusion status is the relationships of the first permanent molars during initial occluding contact. The first permanent molars erupt between 5 and 7 years of age. The chance that a class I interdigitation of the dentition will evolve is best when a class I relationship is represented at initial permanent first molar occluding contact. A class II first permanent molar occlusion at initial occluding contact will predictably remain a class II occlusion into the complete adult dentition. Also indicative of a developing malocclusion are some initially occluding end-to-end relationships. Three quarters of initial contacting end-on first molar occlusions will shift toward a class I during the transition dentition phase. However, 25%, of these end-on relationships will shift into a class II relationship. A class III occlusion at initial contact will predictably lead to a future class III molar relationship. This discussion regarding diagnostic and predictive information is based on the work of Arya, Savara, and Thomas,"[3] Carlsen and Meredith,'} and Moyers.[49]

The development of the transitional phase occlusion and malocclusion is graphically illustrated in Fig. 24-14. Note that distal-step terminus always leads to class II initial contact and final permanent first molar occlusions. The probability that a class III final first permanent molar relationship will develop from a class III initial contact occlusion is also very high. Development of the occlusion from flush terminus, end-on, and class I initial contact molar relationships is highly variable.

The occlusion relationship of upper to lower dentition will remain nearly the same throughout the growing period. Exceptions are cases in which environmental factors, such as premature exfoliation of primary teeth, are superimposed on the developing occlusion, as shown by Northway, Wainright, and Demirjian.[50] Carlsen and Meredith demonstrated that, 70% of the time, the lower permanent first molars will move mesially relative to the upper permanent first molar during the transition occlusion phase." The magnitude of this shift, however, will typically not compensate for a permanent first molar malocclusion. Overall occlusion pattern will be maintained during growth.

GROWTH AND DENTAL ARCH PATTERN

SIMILAR STAGE SEQUENCING

The stage sequence of dental arch development is the same for everyone. According to Nery and Oka, the crowns of primary teeth begin calcification between 3 and 4 months prenatally.[5'] The calcification of mandibular teeth usually precedes that of the maxillary dentition; the central incisors typically show first evidence of calcification and the second molars last. Boys typically begin calcification before girls.

The first primary tooth to erupt is the central incisor at about 7[1]/2 months, and the last to erupt is the second primary molar at about 2'/2 years. Closure of root apex occurs at 3 years for the second primary molar. The usual sequence of primary dentition eruption is the central incisor (in Palmer notation designated by the letter A), the lateral incisor (B), the first primary molar (C), and the canine (D), followed by the second primary molar (E). Hence, the typical eruption sequence is A-B-D-C-E.

Calcification of the permanent teeth does not begin until after birth.[S1] The first permanent molar is the first to show evidence of calcification, which takes place during the second postnatal month. The third molar is the last to begin calcification, which occurs at about 9 years.

The typical eruption sequence for the mandibular arch is as follows: first molar (in Palmer notation, designated by the number 6), central incisor (1), lateral incisor (2), canine (3), first premolar (4), second premolar (5), and second molar (7), followed by the third molar (8). For the maxillary arch, the usual sequence of eruption for the permanent teeth is as follows: 6-1-2-4-5-3-7-8. Eruption timing in girls generally precedes that in boys by an average of 5 months.

Eruption times for permanent teeth can vary considerably depending on the specific tooth. According to Garn, eruption time for the lower incisor varies the least; 90% of lower permanent incisors will erupt within

a span of 3 years. In contrast, eruption time varies the most for the lower second permanent premolar, which shows a 02-year span.'

Dimensional changes for dental arch length, circumference, and intermolar and intercanine widths during childhood and adolescence have been compiled by Moorrees."," Average dimensional dental arch changes from age 6 to 18 years for maxillary and mandibular arches are as follows:

Lower **Arch**

Arch width:	Bicanine: 3-mm increase Bimolar: 2-mm increase
Arch length:	1-mm decrease because of up -righting of incisors
Arch circumference:	Decrease of 4 mm

Upper Arch

Arch width:	Bicanine: 5-mm increase Bimolar: 4-mm increase
Arch length:	Slight decrease because of up-righting of incisors
Arch circumference:	Increase of 1 mm

IDEAL DENTAL ARCH PATTERN

Development of dental arch malocclusion is predictable. Development of a clinically acceptable dental arch likewise can be predicted. The status of the dental arch at mid-adolescence is contingent on clinical features that can be easily recognized during the transition phase dentition. The simplest method of evaluating the dental arch for factors predisposing to malocclusion is to compare the patient's mixed dentition dental arch with an ideal dental arch pattern.

For the dental arch, the ideal pattern for a 7-year-old child might meet the following criteria:

1. Tight proximal contacts
2. No rotations
3. Specific buccal-lingual axial inclinations
4. Specific mesial-distal axial inclinations
5. Even marginal ridges vertically
6. Flat occlusal plane
7. Excess (positive) leeway space

TOOTH SIZE/ARCH SIZE RATIO AS PATTERN DETERMINANT

Tooth size and alveolar size are the primary factors that determine the status of the permanent dental arch. If tooth size and arch size are not balanced, the effect on the permanent dental arch is crowding or spacing. Crowding is the most common feature of dental arch malocclusion. Only when the combined size of the permanent teeth is balanced with the size of the alveolar apical area is an ideal dental arch possible.

Van der Linden refers to the alveolar bone surrounding the dental apex regions as the *apical area."* Ten Cate reports evidence that the alveolus probably forms as a result of inductive action from cells of the dental follicle.[55] The size of alveolar bone is influenced by the many environmental factors that affect intramembranous bone growth. It is possible to clinically increase or decrease the size of the alveolar apical area during growth. Frankel has demonstrated that alveolar arch size can be increased dramatically during childhood and that the increases are stable into adulthood.[56]

Tooth size, for the purpose of discussing dental arch development, refers to the mesial-distal dimensions of each tooth. According to Garn} and Potter et al,[3] mesial-distal tooth size is determined primarily by genetic factors. Four chromosomal gene loci appear responsible for mesial-distal maxillary tooth size, and the mandibular dentition seems to be under the genetic control of six loci. Tooth size is polygenically determined and continuously variable (i.e., a wide range of individuality exists in terms of the width of any single tooth). Dental size is expressed through X-linked inheritance, and racial differences are known. The upper lateral incisor shows the most variability in tooth size.

Tooth size and alveolar apical area size are the most pertinent factors in the determination of the intraarch component of malocclusion. Therefore, it is relevant to consider these factors at length. The alveolar apical area will respond to biomechanical stimulation from orthodontic appliances, because intramembranous bone is adaptive and compensatory in nature. Crown size, on the other hand, cannot be predictably influenced during growth by clinical therapy.

The clinical crowns of all permanent teeth, except for the third molar, are completely formed by middle childhood. Mesial-distal crown widths will not change after crown formation unless affected by factors such as caries. Hence, mesial-distal crown dimension is a stable factor in the tooth size/arch size ratio. In an attempt to exploit the clinical usefulness of crown dimension stability, tooth size relationships are examined.

Comparison of primary to permanent mesial-distal tooth sizes is one such consideration. Studies by Moorrees reveal that there is little about primary dentition size that will predict permanent dentition size.[52] Correlation coefficient *(r)* values ranging from $r = 0.2$ to $r = 0.6$ are indicative of the poor predictive relationship between primary mesial-distal tooth size and the size of their permanent successors. Correlation coefficients of $r = 0.8$ or higher are required to make predictions for the individual patient at chairside.[3]- The combined mesial-distal sizes of all primary teeth and the combined sizes for the permanent teeth show a correlation of $r = 0.5$. Hence, Moorrees concluded that the size of the primary teeth is of little predictive value in estimating the size of their permanent successors.[52]

The strength of the size relationships among the permanent teeth, however, is clinically important for some comparisons. Potter and Nance demonstrated that the size of an individual tooth is highly correlated with the size of the contralateral tooth in the same arch, as reflected in an r value of around Q9.[57] The combined mesial-distal dimensions of contralateral quadrants of teeth show a slightly higher correlation of r = 0.95. Intraarch comparisons of tooth groupings, such as mesial-distal size of the lower incisors versus mesial-distal sizes of the lower canine and premolars combined, show only moderate correlation (r = 0.6) and therefore are not useful clinically.[58]

COMPUTATION OF TOOTH SIZE/ARCH SIZE BALANCE

The primary reason for dental arch malocclusion is imbalance between tooth size and alveolar apical size. In the transition (mixed) dentition, it is possible to accurately determine if combined mesial-distal tooth size will be balanced with alveolar arch size in later life. This process of determination is called *mixed dentition space analysis.* Many methods of mixed dentition space analysis are available. Common to all of these methods is the attempt to determine the combined mesial-distal size of the unerupted permanent canine and first and second premolars. According to Horowitz and Hixon, the lower dental arch is the focus for space analysis and the basis of orthodontic diagnosis and treatment planning." The mandibular alveolar base can be modified less therapeutically than can the upper alveolus and therefore restricts treatment possibilities. The mandibular arch also undergoes less growth change than does the upper arch.

Efficacy studies by Gardner,[5`'] Kaplan, Smith, and Kanarek,[6i0] and Staley and colleagues"[I-63] reveal one method to be the most accurate in predicting the combined size of the unerupted canine and premolars during the mixed dentition. This method, originally devised by Hixon and Oldfather,[58] has recently been refined by Bishara and Staley." In summary, the analysis involves the following steps:

1. Measure the combined width of the lower lateral and central incisors on one side.
2. Measure directly from the radiograph the crown sizes of the unerupted 4-5 on the same side.
3. Add together the incisor and the premolar sizes.
4. Refer to the prediction chart to determine the sizes of the unerupted 3-4-5.

Techniques of mixed dentition space analysis allow estimation of the sizes of the unerupted canine and premolars on the lower arch. This size estimate must then be compared with a measurement of the arch space available between the mesial aspect of the lower molar and the distal aspect of the lateral incisor in the same quadrant. The difference between the combined width of the three unerupted permanent teeth and this arch space has been called *leeway space.*

The most favorable dental arch pattern is one in which leeway space is excessive (i.e., combined size of unerupted canine and premolars is smaller than arch space available). If leeway space is deficient, dental arch crowding will predictably result. Average growth changes in the dental arch will not be great enough to compensate for leeway deficiencies.

COMPENSATIONS IN DENTAL ARCH DEVELOPMENT

Tooth size/arch size imbalances result in dental arch conditions that are less than ideal. When combined mesial-distal tooth size exceeds alveolar arch size, compensatory adjustments occur, which results in dental arch crowding, excessive curve of Spee, or deviant axial tooth inclinations. Dental spacing results when alveolar arch size exceeds the combined mesial-distal size of the teeth.

Competent treatment planning during the mixed dentition must account not only for differences between the size of unerupted canine and premolars and the space available for them, but also for compensating dental factors. Ideal dental arch status provides a model for such planning. Each compensating factor (i.e., crowding, spacing, excess occlusal curve, or deviant axial tooth position) can be appraised relative to an ideal dental arch. Alteration of a crowded arch to an ideally aligned arch is not possible without creating extra space to resolve the crowding. Consequently, a competent dental arch treatment plan must specify the manner in which space will be clinically created. Several means are available for creating dental arch space. They include the following:

1. Move molars distally.
2. Decrease the mesial-distal dimension of the teeth present in the arch.
3. Increase the buccal-lingual axial inclination of the incisors.
4. Reduce the number of teeth in the arch by extraction.

Resolution of excessive occlusal curve also requires more space. Merrifield has indicated that generally, for each millimeter of excessive occlusal curve, 1 mm of arch length space is required."[s] To upright labially inclined incisors, arch length space is also required. In contrast, more arch length is created when retroclined incisors are proclined through therapy; the length of the arch is increased by repositioning the incisal edges from a lingual to a more labial position.

MAINTENANCE OF OVERALL PATTERN

Space analysis combined with evaluation of the impact of compensating factors on dental arch status is the means by which overall space requirements for the lower arch can be determined during the mixed dentition phase. Overall space appraisal during the mixed dentition is highly indicative of future arch status. The condition presented during the mixed dentition will, to a high degree, be maintained in the permanent dental arch. For this reason, a nonideal adult arch status can be anticipated early, and many undesirable conditions can be resolved during the transition from the primary to the permanent dental arch.

Overall space appraisal is typically expressed as millimeters of arch length space excess or deficiency. Dental arch space excess (1 to 2 mm) is a relatively ideal situation. Clinically, little intervention is usually required because mesial drifting of the permanent teeth often results in little or no crowding or residual spacing. Space excess exceeding 3 to 4 mm, however, can lead to dental arch problems. For example, congenital absence of one or more teeth can leave so much arch space that mesial drifting cannot compensate. Decisions favoring retention of primary teeth as long as possible, extraction of primary teeth and retention of space for later restorative prosthesis, or extraction followed by space closure must be made as long-term planning decisions.

Space deficiencies less than -2 mm can usually be managed with a lower lingual holding arch. Arch space deficiencies of -3 to -6 mm should be scrutinized carefully. Typically, a space-regaining lower lingual arch or arch length expansion treatment measure is indicated. Arches with deficiencies in excess of -6 mm are candidates for aggressive space-regaining techniques, dental arch expansion treatment, or one of a number of serial extraction sequences. Clinical approach to various conditions of space excess and deficiency is based on overall space appraisal (space analysis plus compensating factors) as shown in Table 25-2.

TABLE 25-2 Clinical Disposition Guidelines for Various Dental Arch Space Conditions Resulting from Overall Mixed Dentition Space Appraisal

OVERALL APPRAISAL	mm	CLINICAL DISPOSITION
Large space excess	Greater than +3	Long-term planning
Space excess	Less than +3 to 0	No action; observation
Equivalency	0	Careful observation
Deficiency	Less than -3 to 0	Lower lingual holding arch
Moderate deficiency	-3 to -6	Space regaining or arch expansion
Large deficiency	Greater than -6	Space regaining, arch expansion, or extraction

EFFECT OF ENVIRONMENTAL FACTORS ON DENTAL ARCH PATTERN

The primary determinant of dental arch malocclusion is mesial-distal tooth size/arch size imbalance. Nevertheless, secondary factors can dramatically influence the disposition of the dental arch during childhood. Dental arch status is subject to the ravaging effects of environmental factors that include early loss of primary teeth, interproximal caries, pathology, ankylosis of primary teeth, oral habits, trauma, and early eruption of permanent second molars.

The environmental factors most commonly affecting dental arch status are probably caries and premature loss of primary teeth. Early primary tooth loss and caries can have a profound effect on dental arch status. Caries and early loss of the primary first molars (D), second molars (E), or both (D + E) result in a decrease in dental arch length. A study by Northway, Wainright, and Demirjian[50] showed the following specific details:

1. E loss had the most deleterious effect on dental arch length.
2. Early posterior primary loss resulted in 2- to 4-mm space closure per quadrant in both arches.
3. Space loss was age related in the upper but not in the lower arch.
4. Upper D loss typically resulted in blocked-out cuspids; upper E loss usually led to an impacted second permanent premolar.
5. The greatest space loss was caused by mesial molar movement.
6. More space was lost in the first year after premature tooth loss than in successive years.
7. No recovery of space was demonstrated during growth in the upper arch, and little was found in the lower arch.

SUMMARY

The goal of this chapter was to integrate basic growth principles with patient appraisal to enhance diagnostic and treatment-planning efficacy. Merging growth principles with dentofacial pattern brings to light specific growth features pertinent to clinical patient-care decision making. This chapter focused on growth events germane to a better understanding of malocclusion as it affects the face, occlusion, and dental arches. Two themes were consistent throughout the chapter.

First, overall pattern is maintained from early childhood until growth completion. Growth change affects architecturally equivalent structures in a balanced way. For this reason, craniofacial pattern can be predicted to a great extent. The best estimation of future status is obtained by taking the pattern present at an early age and adding the average growth change. Second, dentofacial pattern changes regionally as an individual matures, and these maturation changes are common in all healthy individuals. Regional variation introduced by the maturing process, however, is not great enough to alter overall dentofacial pattern.

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CHAPTER

26

Cephalometrics and Facial Esthetics: the Key to Complete Treatment Planning

JOHN T. KRULL
GEORGE E. KRULL
THOMAS H. LAPP
DAVID A. BUSSARD

انتشارات
شایان نمودار

CHAPTER OUTLINE

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- Frontal (Posteroanterior) Film

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- Mandibular Dental
- Vertical
- Soft Tissue

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- Class II, Division II
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- Brachyfacial Pattern

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- Deep Bite

ANGLE CLASSIFICATION OF OCCLUSION
Descriptive Skeletal and Dental Evaluation
EVALUATION OF FACIAL ESTHETICS
- Frontal View
- Profile View

In studying a case of malocclusion, give no thought to the methods of treatment or appliances until the case shall have been classified and all peculiarities and variations from the normal in type, occlusion, and facial lines have been thoroughly comprehended. Then the requirements and proper plan of treatment become apparent.

—Edward H. Angle

Cephalometrics, the assessment of craniofacial dimensions, particularly the ethnographic determination of cranial morphology, is an ancient skill practiced by anthropologists for centuries.

Beauty and harmony are the traditional guiding principles used to assess facial proportions, although the definition of beauty may change as civilizations change. Greek sculpture during the golden age of art (fourth century BC) shows facial proportions very similar to those found desirable today. Basic facial features of Greek male and female figures appear to be depicted identically, with most sculpture angles within 5 degrees of contemporary standards; the exceptions are a more acute mentolabial sulcus and nasofacial angle for the ancient Greek ideal.

With the advent of the twentieth century, dentistry began to include the concepts of facial harmony and balance in the theory and practice of cephalometrics. In 1922, Simon introduced this modern era with the development of gnathostatics, a photographic technique that related the teeth and their respective bony bases to each other, as well as to specific craniofacial structures. Although Racini and Carrera obtained the first x-ray films of the skull by the standard lateral view in 1926, it was not until the introduction of the cephalometer by Broadbent in 1931 that the science of cephalometrics became standardized. This sophisticated form of radiography enabled the practitioner to identify specific problem areas of craniofacial disproportion and devise detailed therapeutic interventions. Through the contributions of investigators such as Brodie, Downs, Reidel, Steiner, Tweed, and Ricketts, the clinical application of cephalometrics has developed the techniques that permit the observation of discrepancies observed in the mandible, maxilla, dental units, and soft tissue profile.

The primary aim of cephalometric analysis is to localize malocclusion within a tracing of facial bone and soft tissue structures. The analysis is performed by using standardized cephalometric landmarks to construct lines, angles, and imaginary planes, which permits linear and angular assessments of dental and facial relationships as seen on radiographic films of the head and face. These findings are compared to established normal values, and an individualized treatment protocol is developed for orthopedic, orthodontic, and orthognathic therapies.

The science of cephalometrics has often been referred to as a "numbers game" and has the reputation of being difficult to master. There appears to be a universal search for a reliable group of numbers that will ultimately lead one to an accurate diagnosis. Obviously this search is futile, because all cephalometric measurements may at times lead one to an erroneous conclusion. However, an accurate, in-depth analysis provides one with an assessment of dentofacial and craniofacial morphology. A cephalometric radiograph furnishes one with a static analysis, whereas subsequent films allow the clinician to follow the growth patterns of the adolescent patient on a longitudinal basis. In addition, comparison of serial cephalograms of the same patient may allow some developmental predictions to be made.

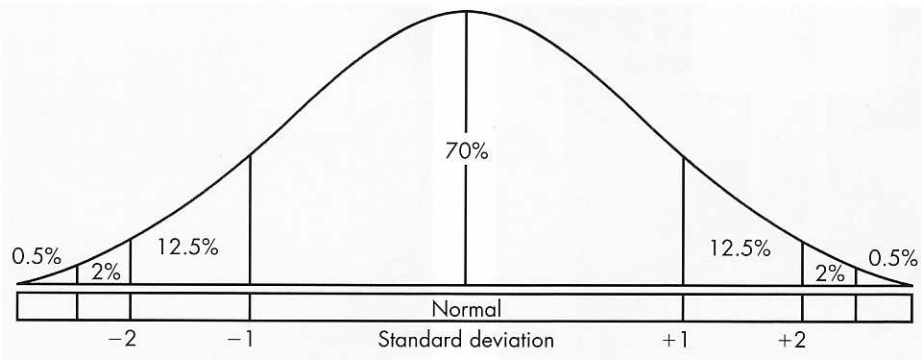
The use of cephalometrics serves to confirm the diagnosis and makes it possible to include the morphology of the cranium when alternative treatment modalities are considered. In patient care, cephalometrics can provide valuable data when treatment is first initiated and can serve a monitoring function during the course of orthodontic care. On completion of treatment, cephalometric radiology allows assessment of the relative degree of posttreatment stability and evaluation of treatment results produced by various mechanical and appliance selections.

Cephalometric numbers or central tendencies have been developed to serve as guidelines in evaluation of the patient. Dentists must keep in mind that they are treating individuals, not averages, and that the numbers merely help or guide in the formulation of an accurate diagnosis and treatment plan. Because of individual anatomic, biologic, and environmental variations, it is imperative that the clinician consider several factors to achieve a comprehensive case analysis. Any attempt to simplify the analysis is likely to lead to an erroneous conclusion.

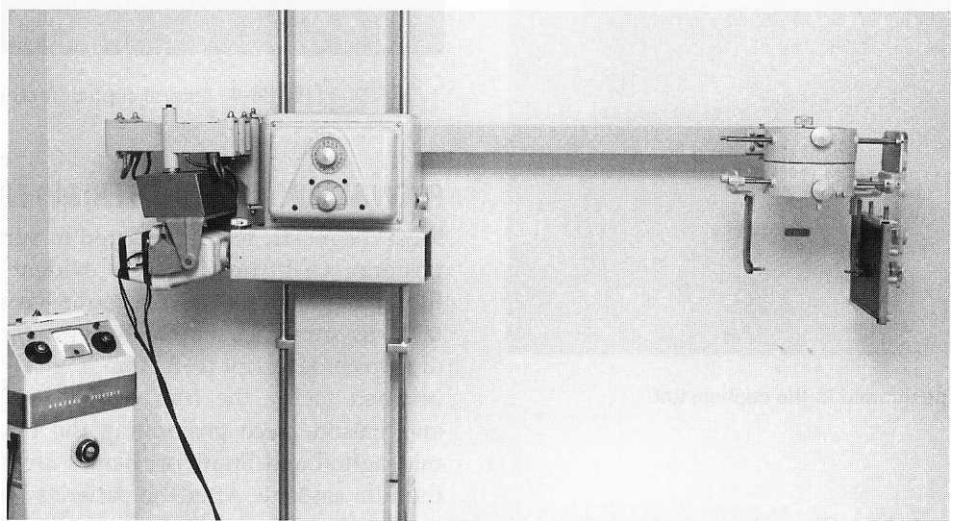
The norm is commonly referred to as the mean or average. On the contrary, however, the norm, as it is applied in cephalometrics, is not a set of averages. The average patient in any given population will generally deviate from the norm, because the norm is derived from samples demonstrating ideal dental occlusions of the class I variety.

Most biologic variables are randomly distributed in the population and can be graphically illustrated by a bell-shaped curve (Fig. 26-1). Within this curve, approximately 70% of any given population lies within 1 standard deviation of the mean, whereas 95% of the group falls within 2 standard deviations. Throughout this chapter, the statistical concept of standard deviation is referred to as *clinical deviation (CD).*

As a general rule, the goal in treatment planning is to treat in the direction of cephalometric norms.



f **I G 26-1.** Bell-shaped curve illustrating the approximate distribution of biologic variables in the general population.



f 1. **26-2.** Wall-mounted, counterbalanced cephalometer. (Courtesy Dr. William W. Merow.)

The clinical advantages include the following:

1. A more favorable and predictable esthetic result
2. Greater posttreatment stability
3. Improved function and periodontal health

RADIOGRAPHIC TECHNIQUE

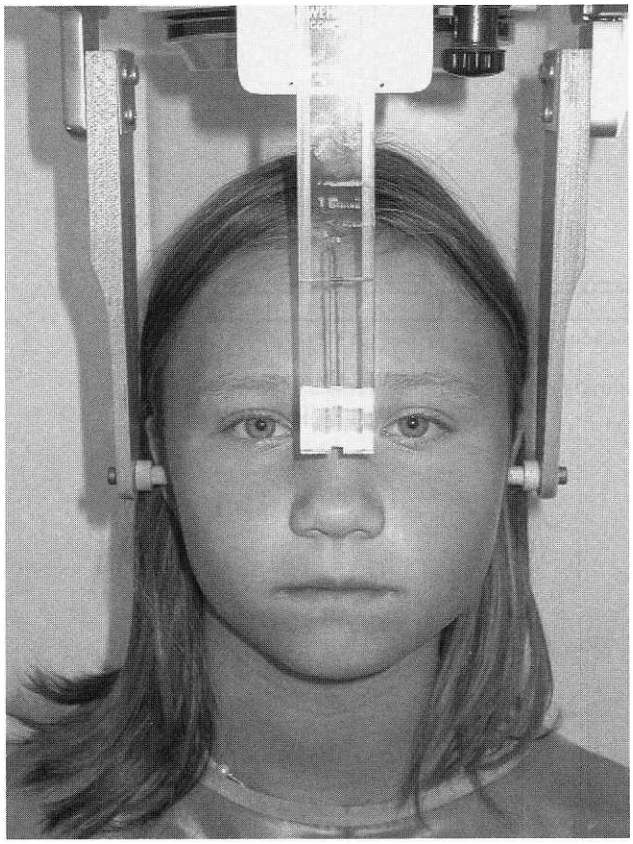
The technique employed in cephalometric radiology has been standardized to permit the comparison of initial and subsequent films for the same patient so that growth can be assessed and treatment progress monitored.

This standardization requires that the equipment include a headholder (cephalostat) and an x-ray tube positioned at a distance of 60 inches from the mid-sagittal plane of the subject and that the distance from the midsagittal plane of the patient to the film be approximately 7.5 inches (Fig. 26-2). The cephalostat maintains a reproducible spatial relationship with respect to the position of the patient's head, the film, and the x-ray source. The most common device uses a counterbalanced beam with the radiographic tube on one end and the cephalostat on the other. This entire unit can be adjusted vertically to compensate for variations in patient height.

The patient is positioned in the cephalostat by means of laterally adjusted ear rods and a vertically adjusted nasal piece (Fig. 26-3). The nasal piece allows the clinician to orient the patient's head so that the Frankfort horizontal plane (a plane extending from the tragus of the ear to the inferior border of the orbital rim) is parallel to the floor. The ear posts should be centrally aligned to the source of radiation so that a transporionic axis is established.

LATERAL HEAD FILM

For a lateral head radiograph, the patient is first positioned so that the left side of the face is tangent to an 8- by 10-inch film cassette, which permits less



I **I I 26-3.** Patient positioned in the cephalostat.

magnification and less distortion of the left-sided structures (Fig. 26-4).

The film cassette should be positioned as close as possible to the patient to minimize the effects of magnification, maximize resolution, and standardize the technique. The distance from the film cassette to the patient's midsagittal plane should be recorded to allow for comparison of serial films. Generally, the film is obtained with the mandible in its most retruded position and the lips in repose. Use of additional positions may be indicated. Once the patient has been positioned, the x-ray beam should enter through the ear rods perpendicular to the film.

Grids and intensifying screens are accessories used to improve the quality of the radiographic image. Rare-earth intensifying screens allow for a reduction of radiographic exposure while increasing the clarity of the radiographic image. Because the film range does not provide for sharp skeletal and soft tissue contrast, a movable aluminum screen attached to the cassette must be used over the soft tissue profile area to reduce the radiation and provide a better differential contrast between the two tissue types.

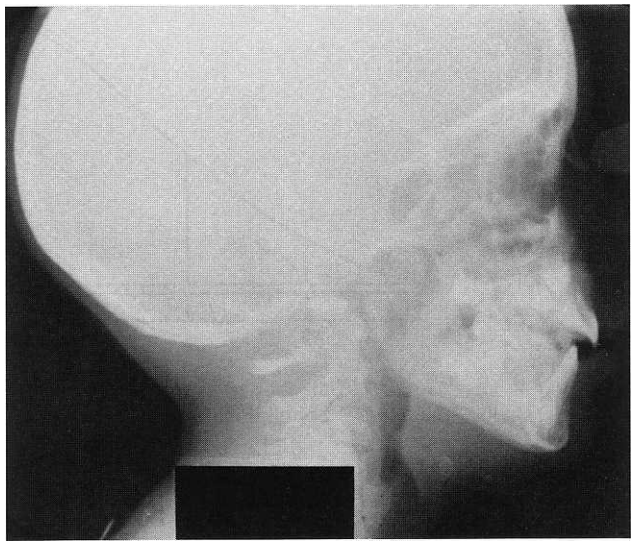


FIG. **26-4.** Lateral cephalometric film.

FRONTAL (POSTEROANTERIOR) FILM

Most diagnostic features related to vertical and anteroposterior (A-P) problems are evident from the lateral film, though severe maxillary transverse deficiencies or facial asymmetries may be better diagnosed by the use of a posteroanterior (P-A) film (Fig. 26-5). The patient is oriented facing the film cassette, with the ear rods and nasion piece positioning the patient so that the midsagittal and Frankfort planes are at right angles to the film cassette. After the patient's head is positioned so that the central x-ray beam passes through the head at the level of the transporionic axis and at its midpoint, the film cassette is moved into contact with the patient's nose. Because more radiation is required for this view, the milliamperage must be increased over that used in the lateral film technique.

CEPHALOMETRIC TRACING TECHNIQUE

Precise localization of the anatomic landmarks used in cephalometric analysis requires adequate knowledge of the radiographic and anatomic appearance of the facial bones and their relationships to adjacent structures. Various features are discernible: lines, shadows, the projections of bony structures, and contours of varying density. All of these make it difficult for the clinician to interpret and identify the anatomic relationships. A clear understanding of craniofacial structures and their relative spatial relationships is imperative before a lateral head film is traced.

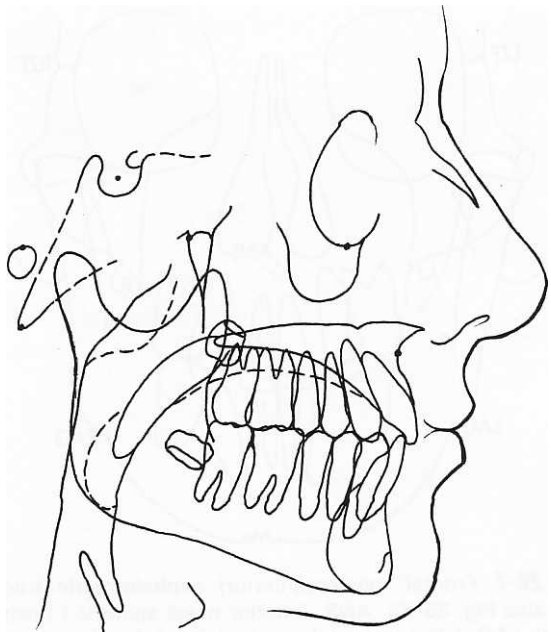
Fig. 26-6 depicts a lateral cephalometric tracing. The lateral tracing should include the soft tissue outline, bony profile, outline of the mandible, posterior and anterior cranial base, odontoid process of the axis,



f I G 26-5. Frontal (posteroanterior) cephalometric film. (Courtesy Dr. William W. Merow.)

anterior lip of the foramen magnum, clivus, planum orbitale, sella turcica, orbit, pterygomaxillary fissure, floor of the nose, roof of the palate, and body of the hyoid bone. In addition to the bony tissues, at least the first permanent molars as well as the most anterior maxillary and mandibular incisors are commonly included. In certain situations it may be desirable to trace other teeth or the complete dentition as shown in Fig. 26-6.

To make the tracing, the radiograph is placed on a view box with the facial profile to the right side. Acetate tracing paper (0.003 matte) is then placed over the radiograph with the matte side up. With a sharp No. 2 or 3H drawing pencil, all the necessary structures are traced. Because all x-rays become divergent once they emanate from the collimator, magnification of the subject will result, and a double-image effect will occur along the inferior border of the mandible and the area of the posterior teeth. All paired structures will produce double images on the head films. Because left-sided structures are magnified less by the radiographic beam and are considered more accurately rendered, the outline of these structures can be traced, although some prefer to make the tracing lines bisect bilateral images.



f I G. 26-6. Lateral cephalometric tracing.

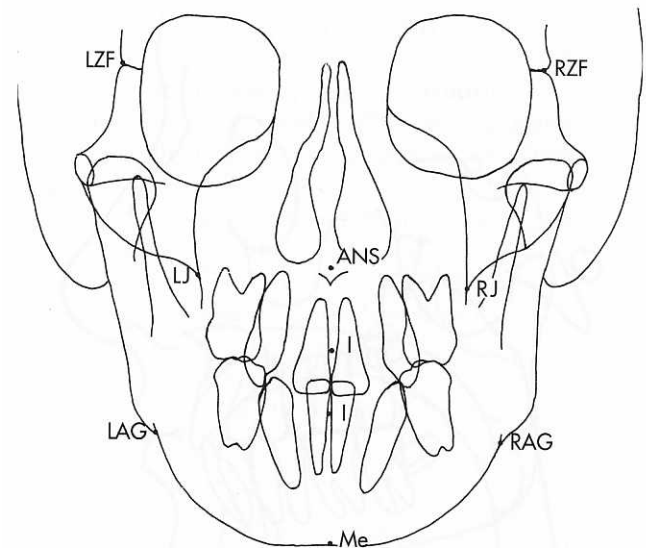
A P-A cephalometric radiograph, as illustrated in Fig. 26-5, can be of significant diagnostic value in cases demonstrating mandibular displacement, facial asymmetry, severe posterior crossbite, or other types of bony dysplasia. Cephalometric analysis and a thorough and systematic clinical examination of these patients often reveal malocclusions accompanied by mandibular shifts when the patient is in maximum occlusion.

The P-A radiograph is traced in the same manner as the lateral film. Fig. 26-7 illustrates the important skeletal and dental structures that must be traced for an accurate and complete analysis.

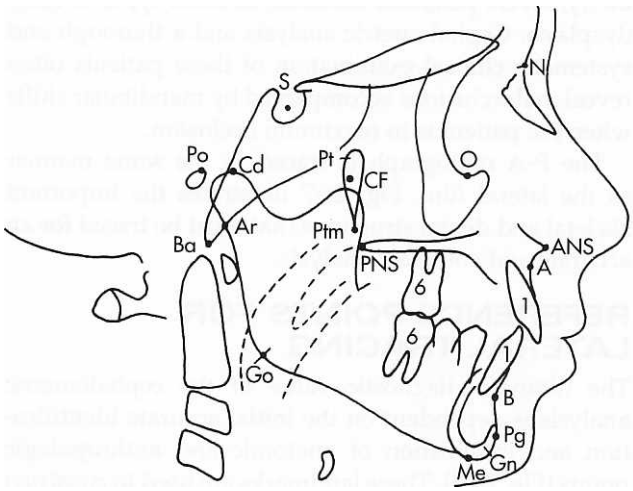
REFERENCE POINTS FOR LATERAL TRACING

The ultimate diagnostic value of the cephalometric analysis is dependent on the initial accurate identification and localization of anatomic and anthropologic points (Fig. 26-8). These landmarks are used to construct the lines, angles, and planes used to make a two-dimensional assessment of the patient's craniofacial and dental relationships. Although each analysis is completed in two dimensions, when the lateral analysis and the P-A analysis for the same patient are considered together, a three-dimensional simulation emerges to contribute to the overall diagnosis and treatment plan. The following reference points are used in this chapter (see Fig. 26-8):

Sella turcica (S, **or sella).** The midpoint of the hypophyseal fossa. This is the ovoid area of the spheroid bone that contains the pituitary gland.



f **I C 26-7.** Frontal (posteroanterior) cephalometric tracing (see also Fig. 26-10). ANS, Anterior nasal spine; 1, I (incisor) point; LAG, left antegonial notch; LJ, left jugal process of maxillary tuberosity; LZF, left zygomaticofrontal suture; Me, menton; RAG, right antegonial notch; RJ, right jugal process of maxillary tuberosity; RZF, right zygomaticofrontal suture.



f G. **26-8.** Lateral tracing with cephalometric reference points. (Adapted from Dr. William W. Merow.)

Nasion (N). The external junction of the nasofrontal suture in the median plane. If the suture is not visible, this point is located at the deepest concavity of the two bones.

Orbitale (0). The most inferior point on the external border of the orbit.

Condylion (Cd). The most superior point on the articular head of the condyle.

Anterior nasal spine (ANS). The most anterior projection of the anterior nasal spine of the maxilla in the median plane.

A point **(subspinale, or A).** The deepest point of the curvature of the anterior maxilla between the ANS and the alveolar crest. Although the A point may change with treatment, it represents the most forward point of the maxilla.

B point (supramentale, or B). The most posterior point on the outer curve of the mandibular alveolar process between the alveolar crest and the bony chin. The B point delineates the most anterior point of the mandible in the median plane.

Pogonion (Pg). The most anterior point on the midsagittal mandibular symphysis.

Menton (Me). The most inferior point of the mandibular symphysis.

Gnathion (Gn). A constructed point that is formed by the intersection of the facial and mandibular planes.

Gonion (Go). Another constructed point that is represented by the intersection of the lines tangent to the posterior margin of the ascending ramus and the mandibular plane.

Articulare (Ar). The point of intersection of the posterior margin of the ascending ramus and the outer margin of the cranial base.

Porion (Po). A point located at the most superior point of the external auditory meatus or the superior aspect of the metal ring that is a component of the left ear rod of the cephalostat.

Basion (Ba). The most inferior posterior point on the occipital bone that corresponds to the anterior margin of the foramen magnum.

Pterygomaxillary fissure (Ptm). A teardrop-shaped fissure of which the posterior wall is created by the anterior borders of the pterygoid plates of the sphenoid bone and the anterior wall represents the posterior border of the maxilla (maxillary tuberosity). The tip of this fissure denotes the posterior extent of the maxilla.

Posterior nasal spine (PNS). The tip of the posterior spine of the palatine bone. This landmark is usually not visible even on well-exposed lateral head films; therefore it is a constructed point that is represented by the intersection of a continuation of the anterior wall of the pterygopalatine fossa and the floor of the nose. It also denotes the posterior limit of the maxilla.

Pt point (Pt). The intersection of the inferior border of the foramen rotundum with the posterior wall of the Ptm.

CF point (center of face). The cephalometric landmark formed by the intersection of the Frankfort horizontal plane and a perpendicular line through Pt.

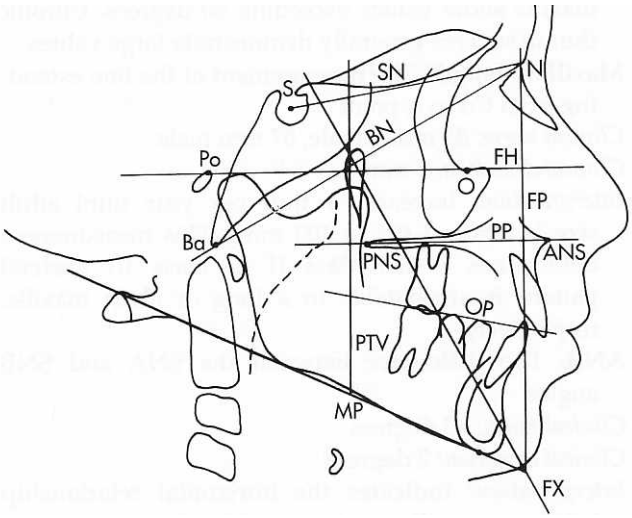


FIG **26-9.** Cephalometric reference lines and planes. (Adapted from Dr. William W. Merow.)

REFERENCE LINES, ANGLES, AND PLANES

Linear assessment is derived when two reference points are connected. Angular measurements are possible when three points are used. Planes (and some lines) are actually imaginary when the cephalometric tracing is viewed because the planes are at right angles to the tracing and can be seen only as a line on the two-dimensional tracing (Fig. 26-9). In cephalometric analysis the dentist must become accustomed to thinking in three dimensions while viewing a two-dimensional representation. Therefore a point on the tracing may not only be a point but also may represent a line (or axis). A line on the tracing may actually be a line (or axis) or it may represent a plane.

Several lines or planes are used in the different cephalometric analyses, although one line or plane generally serves as the major reference on which the entire analysis is based. Two common references are the sella-nasion plane (anterior cranial base) and the Frankfort horizontal plane.

The basic units of cephalometric analysis are angles and distances (lines). Measurements may be treated as absolute values, or they may be related to one another and expressed as relative proportions. These measurements and interrelationships provide the basic framework for describing craniofacial abnormalities. The following definitions help explain the planes of reference used in this chapter (see Fig. 26-9).

Frankfort horizontal plane (FH). This plane is constructed from porion (Po) to orbitale (0) and represents the basic horizontal plane of the head.

Sella-nasion plane (SN). This plane is represented by a line connecting the sella (S) and the nasion (N). It denotes the A-P extent of the anterior cranial base. This reference plane is of questionable diagnostic value in true mandibular prognathism.

Occlusal plane (OP). This plane separates the maxillary and mandibular permanent molars (or in younger patients the primary second molars) and passes through the contact between the most anterior maxillary and mandibular incisors. If the incisors do not contact, the line passes midway between the incisal edges. Ideally, OP is nearly parallel to both the palatal plane (PP) and the FH.

Facial plane (FP). A line constructed through the nasion (N) perpendicular to the FH represents this plane.

Mandibular plane (MP). The mandibular plane is constructed as a tangent to the inferior border of the mandible.

Pterygoid vertical plane (PTV). This plane is represented by a line perpendicular to the FH through the Pt point. Studies have shown that the intersection of FH and PTV is extremely stable, because growth has little effect on this point. An overall view of patient growth may be gained by evaluation of serial cephalometric films on which FH and PTV are superimposed. PTV represents a basic vertical reference plane.

Basion-nasion plane (BN). This plane passes through the basion (Ba) and nasion (N). The plane represents cranial base and is the dividing plane between the cranium and the face.

Facial axis (FX). This line is constructed from the Pt point through the gnathion. FX ideally crosses BN at a right angle.

Palatal plane (PP). This plane extends through the anterior nasal spine (ANS) and posterior nasal spine (PNS). The relationship of this plane to FH is useful in evaluating treatment changes occurring in the maxilla.

INTERPRETATION OF MEASUREMENTS

The objectives of cephalometric interpretation are summarized as follows:

1. To define both the skeletal and facial types
2. To evaluate the relationship between the maxillary and mandibular basal bones
3. To assess the dental relationships (the spatial relationship between the teeth, maxilla, mandible, and cranial base)
4. To locate the malocclusion within the dentofacial complex and analyze its origin (skeletal or dentoalveolar)
5. To study the facial soft tissue contours with respect to the cause of the malocclusion

6. To consider the impact of the various options for correcting the malocclusion on the facial contours as well as on the skeletal and dental components
7. To facilitate selection of a treatment plan
8. To evaluate the results of various soft tissue surgical procedures

LATERAL CEPHALOMETRIC ASSESSMENT

MAXILLARY SKELETAL

SNA: The angle between SN and N—A point

Clinical norm: 82 degrees

Clinical deviation: 2 degrees

Interpretation: Establishes horizontal location of the maxilla. Deviation in cranial base (SN, angulation, or length) or vertical maxillary excess proves that this measurement is unreliable. Therefore reduced emphasis should be given in these instances.

Maxillary depth: The angle formed by the intersection of the FH and N—A point planes

Clinical norm: 90 degrees

Clinical deviation: 3 degrees

Interpretation: Indicates horizontal position of maxilla. Class II skeletal patterns caused by a prognathic maxilla show values exceeding 90 degrees. Chronic thumb suckers generally demonstrate large values.

Maxillary length: The measurement of the line extending from Cd to A point

Clinical norm: 85 mm female, 87 mm male

Clinical deviation: 6 mm

Interpretation: Increases 1 mm per year until adult size is attained (95 to 100 mm). This measurement determines if the class II or class III skeletal pattern is attributable to a long or short maxilla, respectively.

ANB: The difference between the SNA and SNB angles

Clinical norm: +2 degrees

Clinical deviation: 2 degrees

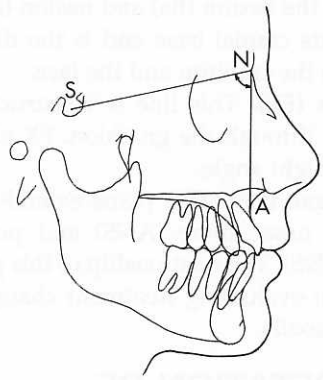
Interpretation: Indicates the horizontal relationship between maxilla and mandible. Positive values indicate that the maxilla is forward of the mandible, whereas negative values indicate a class III skeletal relationship.

MAXILLARY DENTAL

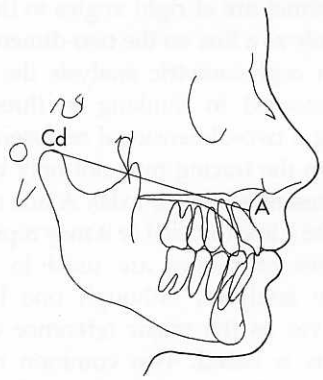
Maxillary incisor angulation: The angle formed by SN and the incisor long axis

Clinical norm: 102 degrees

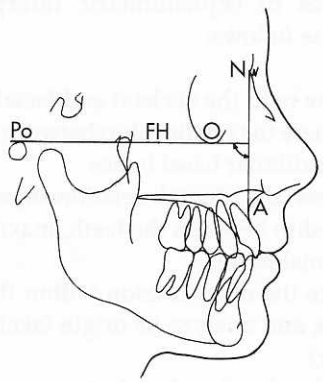
Clinical deviation: 3 degrees



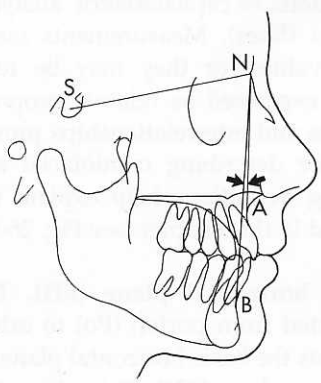
SNA



MAXILLARY LENGTH



MAXILLARY DEPTH



ANB

Interpretation: Relates the upper incisor angulation to the upper and middle face. Values well above 102 degrees indicate angular incisor protrusion, whereas values significantly below demonstrate angular retrusion.

Maxillary incisor A-P position: The horizontal distance from the facial surface of the maxillary central incisors to the N–A point line

Clinical norm: 4 mm

Clinical deviation: 2 mm

Interpretation: Indicates horizontal position of the maxillary incisors. Values in excess of 6 mm indicate anterior dental protrusion, whereas values 1 mm or less show dental retrusion.

Upper molar position: The horizontal distance from PTV to the distal surface of the maxillary first molar

Clinical norm: Chronologic age of the patient + 3 mm (e.g., a 10-year-old has a clinical norm of 10 + 3 = 13 **mm).** The growth change is approximately 1 mm per year through the years of active growth.

Clinical deviation: 3 mm

Interpretation: Determines if the dental malocclusion is caused by the A-P position of the maxillary molar. It is important in treatment planning considerations involving distal movement of the maxillary molars.

Maxillary incisor to upper lip: The vertical distance between the inferior border of the upper lip and the incisal edge of the maxillary incisor

Clinical norm: 3 mm

Clinical deviation: 1 mm

Interpretation: Gives an evaluation of the amount of upper incisor in repose. Values of 5 mm or more may be associated with vertical maxillary excess. This value must be compared with upper lip length. Obviously, patients with short upper lips will show more incisor at rest.

MANDIBULAR SKELETAL

SNB: The angle formed between the SN and N–B point planes

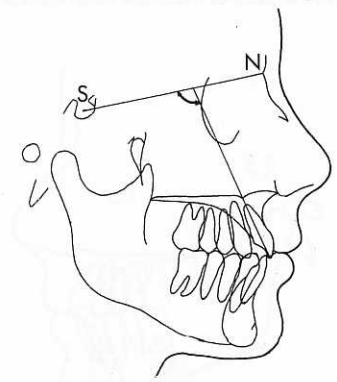
Clinical norm: 80 degrees

Clinical deviation: 2 degrees

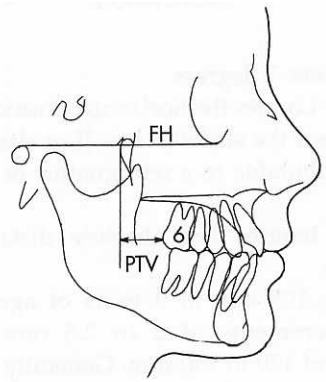
Interpretation: Indicates horizontal location of the mandible. Abnormal cranial base angulation and vertical facial excess will adversely affect the reliability of this measurement.

Facial angle (depth): The angle formed between the N-Pg and FH planes

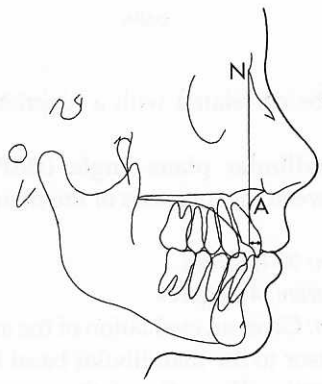
Clinical norm: 87 degrees at 9 years of age. Increases 0. 33 degree per year.



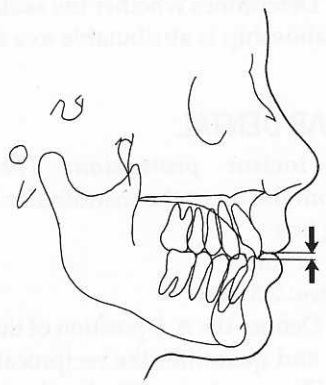
MAXILLARY INCISOR ANGULATION



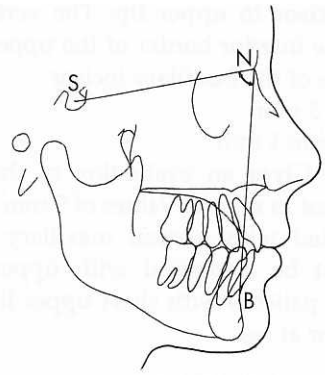
UPPER MOLAR POSITION



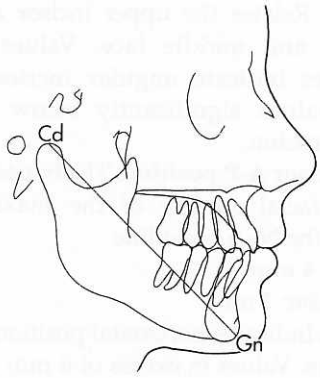
MAXILLARY INCISOR (A-P) POSITION



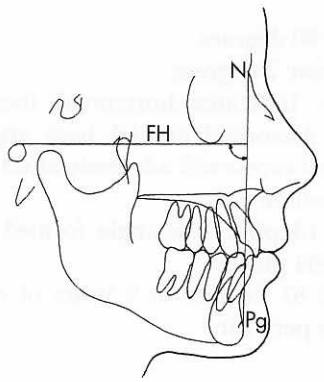
MAXILLARY INCISOR TO UPPER LIP



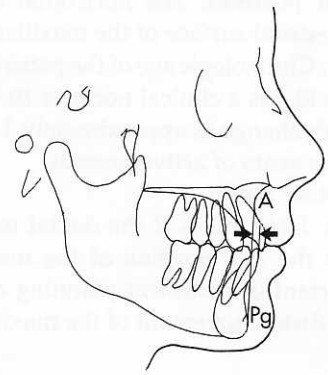
SNB



MANDIBULAR LENGTH



FACIAL ANGLE



MANDIBULAR INCISOR PROTRUSION

Clinical deviation: 3 degrees

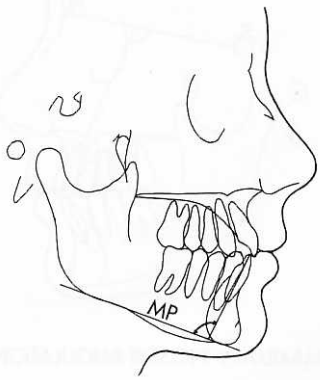
Interpretation: Locates the horizontal position of the chin. Determines if the skeletal class II or class III relationship is attributable to a retrognathic or a prognathic mandible.

Mandibular length: The absolute distance between Cd and Gn

Clinical norm: 105 mm at 9 years of age with yearly growth increments of 2 to 2.5 mm, reaching a maximum of 120 to 130 mm. Generally 2 mm less in females than in males at 9 years of age.

Clinical deviation: 6 mm

Interpretation: Determines whether the skeletal class II or class III relationship is attributable to a small or large mandible.



IMPA

MANDIBULAR DENTAL

Mandibular incisor protrusion: The horizontal distance from the tip of the mandibular incisor to the A point—Pg line

Clinical norm: +2 mm

Clinical deviation: 2.3 mm

Interpretation: Defines the A-P position of the mandibular dental unit and quantifies the reciprocal relationship of the maxillary and mandibular dental units. Not only is this a key esthetic relationship, but it also needs to be correlated with a functional arch length analysis.

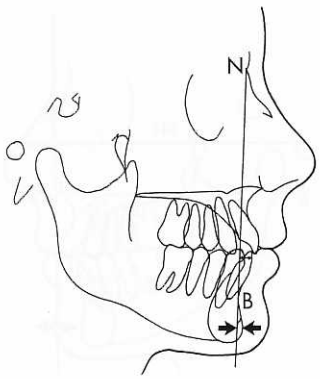
Incisor mandibular plane angle (IMPA): The inner angle between the long axis of the mandibular incisor and MP

Clinical norm: 90 degrees

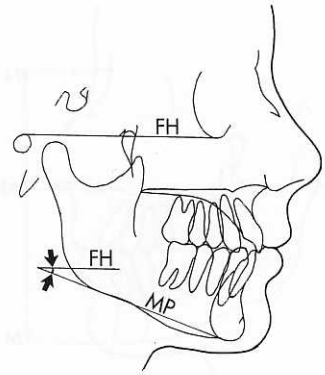
Clinical deviation: 4 degrees

Interpretation: Gives an evaluation of the angular position of the incisor to the mandibular basal bone.

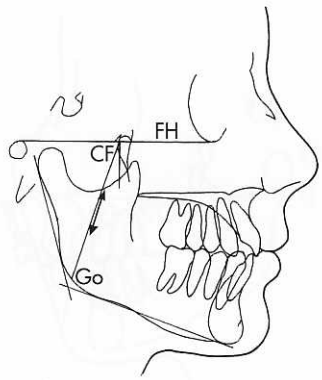
Holdaway ratio: The ratio of the mandibular incisor and Pg to the N—B point line



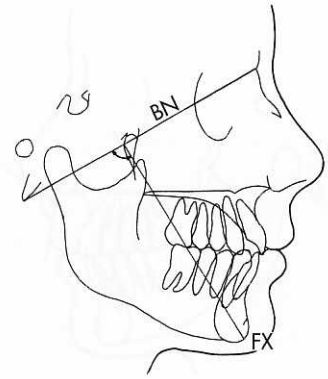
HOLDAWAY RATIO



FMA



POSTERIOR FACIAL HEIGHT



FACIAL AXIS ANGLE

Clinical norm: 1:1
Clinical deviation: 2 mm
Interpretation: The A-P position of the mandibular incisor and Pg should project equally from the N—B point line for good facial balance.

VERTICAL

Posterior facial height: The linear distance between Go and the CF point
Clinical norm: 55 mm for the average-sized patient at *8.5* years of age. Increases 1 mm per year.
Clinical deviation: 3.3 mm
Interpretation: Measures vertical growth of the ramus and can therefore be of value in predicting clockwise or counterclockwise growth patterns. Values below 51 mm represent a leaning toward dolichofacial patterns, whereas values in excess of 59 mm may indicate brachyfacial or counterclockwise growth trends.

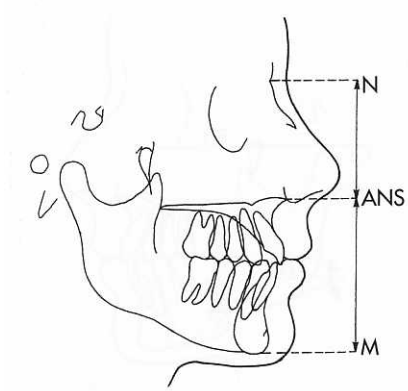
Mandibular plane angle (FMA): The angle formed by the intersection of FH and MP
Clinical norm: 26 degrees. Decreases 1 degree every 4 years during normal growth.
Clinical deviation: 4 degrees
Interpretation: Values in excess of 31 degrees may indicate clockwise growth with dolichofacial growth trends, whereas values less than 21 degrees imply vertical deficiency as often seen in brachyfacial growth patterns.

Facial axis angle: The angle between FX and BN
Clinical norm: 90 degrees
Clinical deviation: 3.5 degrees. Changes 1 degree every 3 years in the average patient.
Interpretation: Expresses the ratio of facial height to depth and thus indicates the direction of growth of the chin. Values in excess of 94 degrees may indicate counterclockwise growth, whereas those less than 85 degrees may imply clockwise growth in brachyfacial and dolichofacial facial types, respectively.

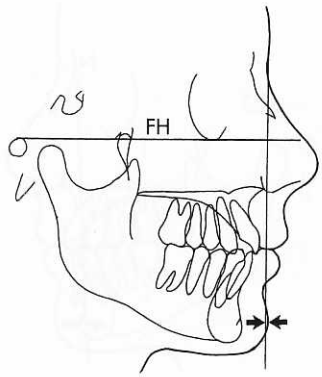
Facial height: The vertical relationship between upper and lower facial height (N-ANS:ANS-M)
Clinical norm: Upper 53 mm; lower 65 mm
Interpretation: More important than the absolute value is the ratio between upper and lower facial height, which should be approximately *5:6* for a well-balanced face.

SOFT TISSUE

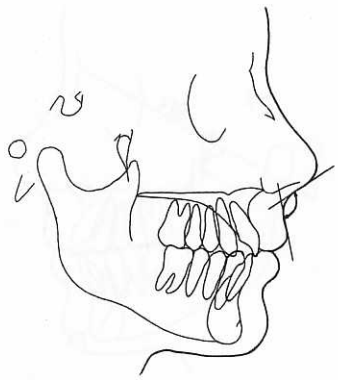
Nasolabial angle: The angle formed by the intersection of the lines tangent to the columella of the nose and the upper lip



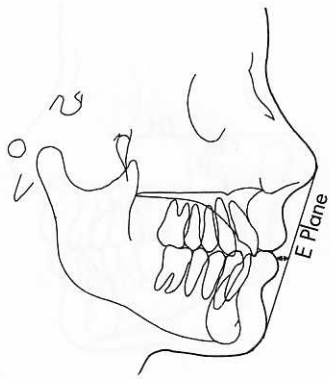
FACIAL HEIGHT



ZERO MERIDIAN



NASOLABIAL ANGLE



LIP PROTRUSION

Clinical norm: 90 to 110 degrees
Interpretation: Provides an assessment of the nose- to-upper-lip relationship. Values in excess of 114 degrees may indicate upper lip retrusion, whereas values of 96 degrees or less may be associated with dental protrusion.

Zero meridian: The horizontal distance from the chin to a line perpendicular to FH and tangent to the soft tissue nasion
Clinical norm: 0 mm
Clinical deviation: 2 mm
Interpretation: May be helpful in assessment of the projection of the chin relative to FH.

Interlabial distance: The vertical distance between the inferior aspect of the upper lip and the superior surface of the lower lip with the patient in repose
Clinical norm: 1.9 mm
Clinical deviation: 1.2 mm
Interpretation: High values indicate lip incompetence and are often associated with hyperactivity of the mentalis muscle. Low values may be associated with overclosure.

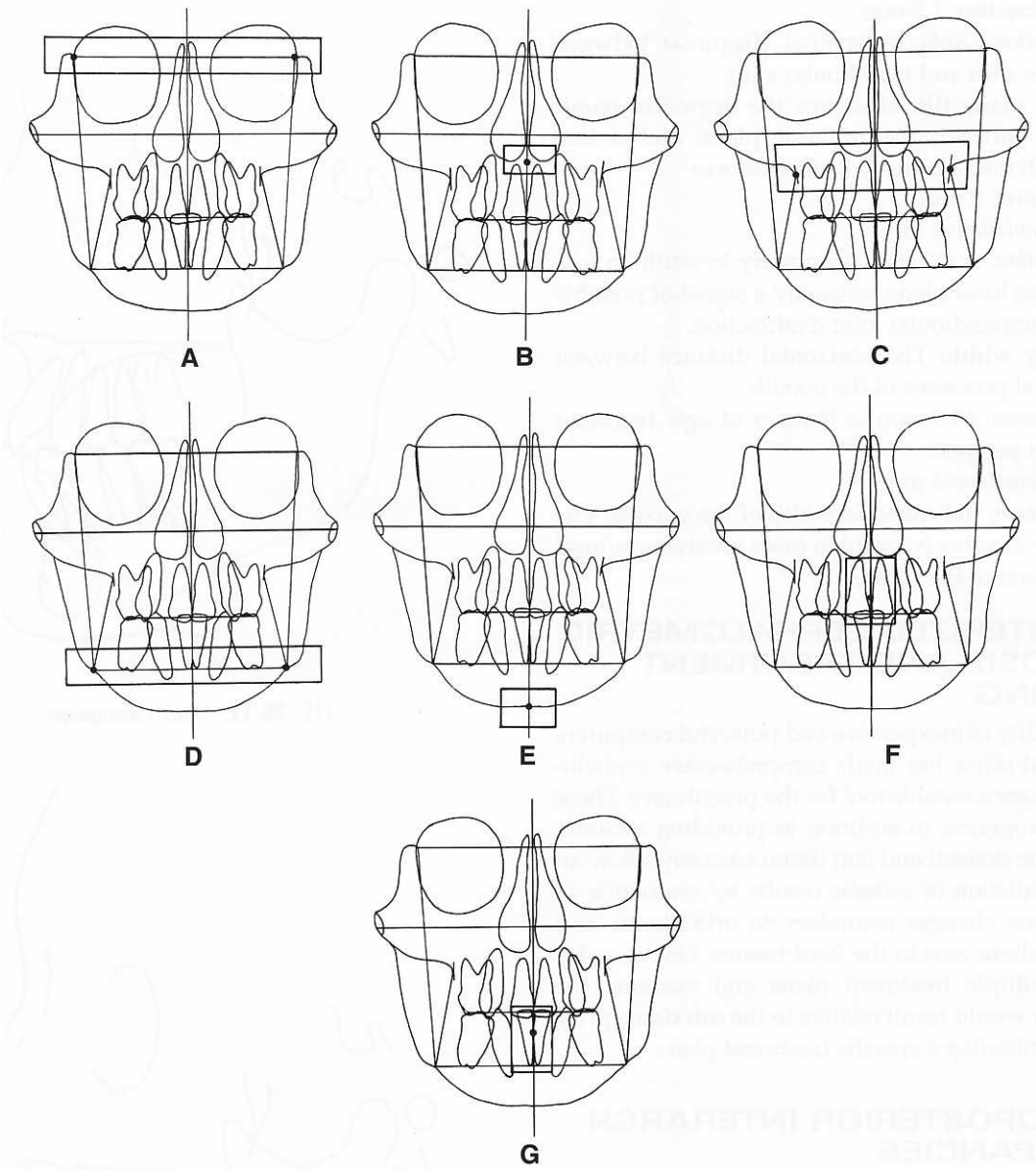
Lip protrusion: The horizontal distance between the lower lip and the esthetic plane (E plane). The esthetic plane is a line connecting the tip of the nose and the most anterior point on the soft tissue chin.
Clinical norm: -2 mm at 8.5 years of age; decreases 0.2 mm per year. The values tend to decrease with age until adult values of -5 mm are reached.
Clinical deviation: 2 mm
Interpretation: Indicates soft tissue balance between lips and profile (nose-chin).

FRONTAL (POSTEROANTERIOR) CEPHALOMETRIC ASSESSMENT

Frontal cephalometric points and planes are used to evaluate the overall relationships of the cranium, maxilla, mandible, and denture from a frontal view. Fig. 26-10 is a graphic representation of the points, lines, and planes used in frontal cephalometric analysis.

Dental midline: The horizontal distance between the maxillary and mandibular incisor midlines *Clinical norm: 0* mm
Clinical deviation: 1.5 mm
Interpretation: Determines dental midline asymmetry.

Maxillomandibular width: The horizontal distance between the jugal process of the maxilla and the frontal facial plane
Clinical norm: 10 mm for patient of average size at $8^1/_2$ years of age. Needs to be corrected for size.



f I **G 26-10.** Frontal reference points (see also Fig. 26-7). A, LZF/RZF, bilateral points on the medial aspect of the zygomaticofrontal sutures at the intersections of the orbits. **B,** ANS, tip of anterior nasal spine. C, LJ/RJ, bilateral points on the jugal processes and the intersection of the maxillary tuberosities and the zygomatic buttresses. D, LAG/RAG, points at the lateral inferior margin of the antegonial protuberances of the mandible. **E,** Me, menton, point of the inferior border of the mandibular symphysis directly inferior to the mental protuberance. F, / point, a point selected at the interdental papilla of the upper incisors at the junction of the crowns and gingiva. G, t point, a point selected at the interdental papilla of the lower incisors at the junction of the crowns and gingiva.

Interpretation: Determines if a crossbite is skeletal in nature. Large values are associated with skeletal lingual crossbites, whereas lesser values indicate skeletal buccal crossbites.

Maxillomandibular midline: The angle formed by the ANS-Me plane through ANS and perpendicular to the zygomatic frontal suture plane

Clinical norm: 0 mm

Clinical deviation: 2 mm

Interpretation: Determines whether facial asymmetry is attributable to total size discrepancy or a functional shift of the mandible.

Denture to jaw midlines: The horizontal distance between the midlines of the mandibular incisors and maxilla and mandible

Clinical norm: 0 mm

Clinical deviation: 1.5 mm

Interpretation: Aids differential diagnosis between denture shift and mandibular shift.

Occlusal plane **tilt:** Measures the degree of parallelism between the occlusal plane and a line through the zygomatic frontal sutures

Clinical norm: 0 mm

Clinical deviation: 2 mm

Interpretation: A skeletal asymmetry in addition to a tilt in occlusal plane is usually a signal of possible temporomandibular joint dysfunction.

Maxillary width: The horizontal distance between the jugal processes of the maxilla

Clinical norm: 61.9 mm at 9 years of age. Increases 0.6 mm per year.

Clinical deviation: 3 mm

Interpretation: Indicates the width of the maxilla. The change in value is useful in cases involving sutural expansion of the palate.

COMPUTERIZED CEPHALOMETRIC DIAGNOSIS AND TREATMENT PLANNING

The availability of inexpensive and powerful computers in the dental office has made comprehensive cephalometric software a reliable tool for the practitioner. These computer programs, in addition to providing accurate models of the skeletal and soft tissue anatomy, allow an accurate prediction of esthetic results by evaluation of the soft tissue changes secondary to orthodontic and orthopedic alterations in the hard tissues. One can also evaluate multiple treatment plans and examine the changes that would result relative to the soft tissue prior to actually initiating a specific treatment plan.

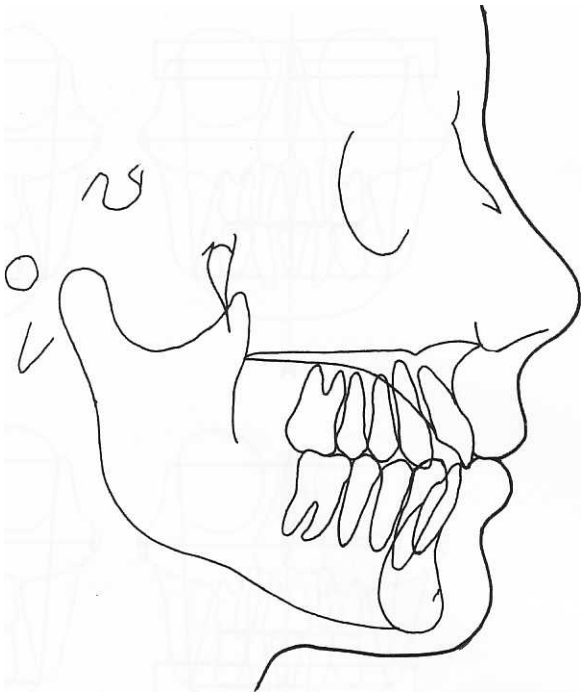
ANTEROPOSTERIOR INTERARCH DISCREPANCIES

CLASS I

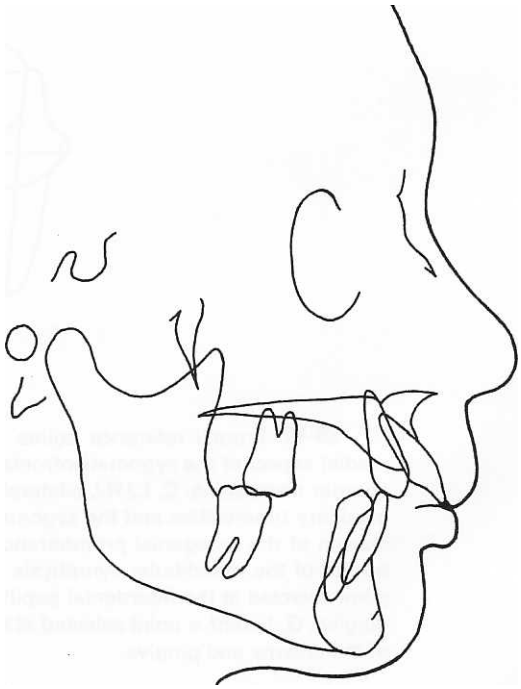
A class I occlusion is one in which the mesiobuccal cusp tip of the maxillary first molar aligns with the buccal groove of the mandibular first molar (Fig. 26-11). Because of this sagittal relationship, most class I occlusions demonstrate reasonably normal skeletal and soft tissue profiles.

CLASS II, DIVISION I

In class II, division I malocclusion, the mesiobuccal cusp tip of the maxillary first molar is positioned anterior to the buccal groove of the mandibular first molar (Fig. 26-12). The sagittal molar relationship of these patients is referred to as a disto-occlusion as opposed to a neutro-occlusion in patients with class I occlusion. The exact reason for this relationship may be skeletal, dental, or a combination of the two. The nature of the problem can be more accurately determined by the use of cephalometric analysis. This type of malocclusion is often characterized by excessive overjet in the anterior region. Unlike the patient with class I occlusion, these patients often exhibit more downward growth, abnormal muscle pressure, and a convex soft and hard tissue profile.



f **I G 26-11.** Class I occlusion.



E I Fi **26-12.** Class II, division I occlusion.

In vertical growth patterns in which the upper molars are erupting along the facial axis and the upper incisors are erupting in a protruded direction, space between the upper molars and incisors is increased, which results in the typical dental characteristics of the class II, division I malocclusion.

CLASS II, DIVISION II

The molar position observed in patients with class II, division II malocclusion is similar to that of those with class II, division I malocclusion, although the excessive overjet associated with the latter is not seen (Fig. 26-13). The anterior relationship of a class II, division II malocclusion is characterized by lingual tipping of the central incisors and labial flaring of the lateral incisors. Whereas patients with class II, division I malocclusion show a weak chin, patients with division II malocclusion tend to have a square jaw, skeletal deep bite, and a short lower facial height. Class II, division II malocclusions demonstrate strong growth patterns in which the upper molar grows down the facial axis while the upper incisor moves down with a retroclination. In this case, there is a diminution of space between the molar and incisor. This results in a pinching or closing of the arch, which gives the characteristic flaring of the upper lateral incisors and linguoversion of the centrals. In severe cases, an hourglass-shaped upper arch form may result.

CLASS III

In class III malocclusion the mesiobuccal cusp tip of the maxillary first permanent molar is posterior to the buccal groove of the mandibular first permanent molar (Fig. 26-14). The most common cause of class III maloc clusions is excessive growth of the mandible. The molar position of these patients is referred to as mesio-occlusion, whereas the anterior relationship shows a negative overjet. Many cases demonstrate dental compensations in which the maxillary incisors are excessively flared and the mandibular incisors are severely tipped to the lingual. These patients typically show concave facial profiles and steep mandibular plane angles attributable, in part, to an obtuse gonial angle.

FACIAL TYPES

Classification of the face by one of the three general facial types is imperative even though no definitive correlation between malocclusion and facial types has been demonstrated. It should be obvious that the prognosis for a pleasing facial result in treatment of a class II malocclusion associated with a retrognathic mandible would be more uncertain than that in treatment of a class II malocclusion occurring with an orthognathic mandible.

The three basic facial types or patterns are dolichofacial (vertical), mesofacial (ideal), and brachyfacial (horizontal). The determination of the patient's facial pattern is important in growth prediction as well as in treatment planning. Therefore one of the first assessments necessary for an accurate craniofacial diagnosis is classification of the patient's facial type.

Although all facial types may be observed in association with different malocclusions, a statistically significant higher incidence of specific types does occur with

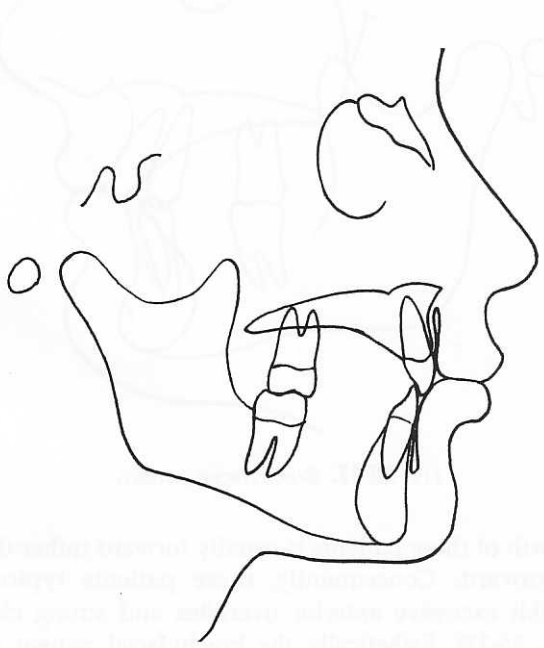


FIG. **26-13.** Class II, division II occlusion.

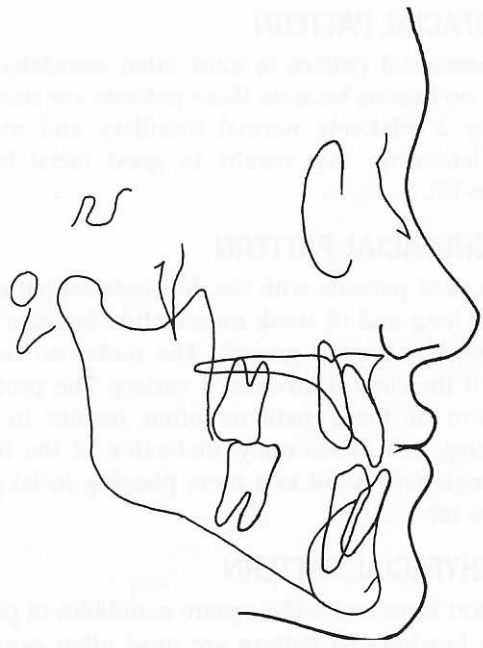


FIG. **26-14.** Class III occlusion.

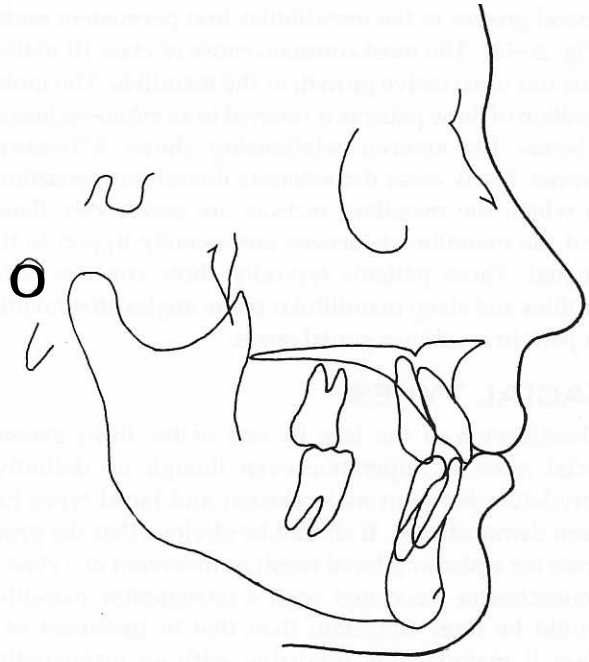


FIG. **26-15.** Mesofacial pattern.

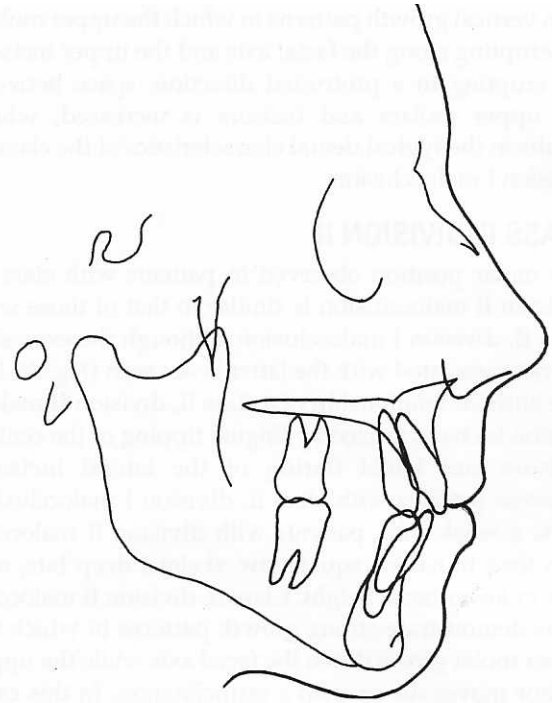


FIG. **26-16.** Dolichofacial pattern.

certain types of malocclusion, such as the association of class II malocclusions with retrognathic mandibles and of class III malocclusions with prognathic mandibles. On the other hand, an orthognathic facial type is not always associated with an ideal class I occlusal relationship. As the clinician becomes more familiar with the different types of malocclusion, it will become obvious that certain facial patterns are commonly associated with each classification of malocclusion.

MESOFACIAL PATTERN

The mesofacial pattern is most often associated with class I occlusions because these patients are characterized by a relatively normal maxillary and mandibular relationship that results in good facial balance (Fig. 26-15).

DOLICHOFACIAL PATTERN

The faces of patients with the dolichofacial pattern are usually long and of weak musculature because of the tendency for vertical growth. The molar occlusion is often of the class II, division I variety. The protruded dentition of these patients often results in facial grimacing and disharmony. Reduction of the interincisal angle will result in a more pleasing facial profile (Fig. 26-16).

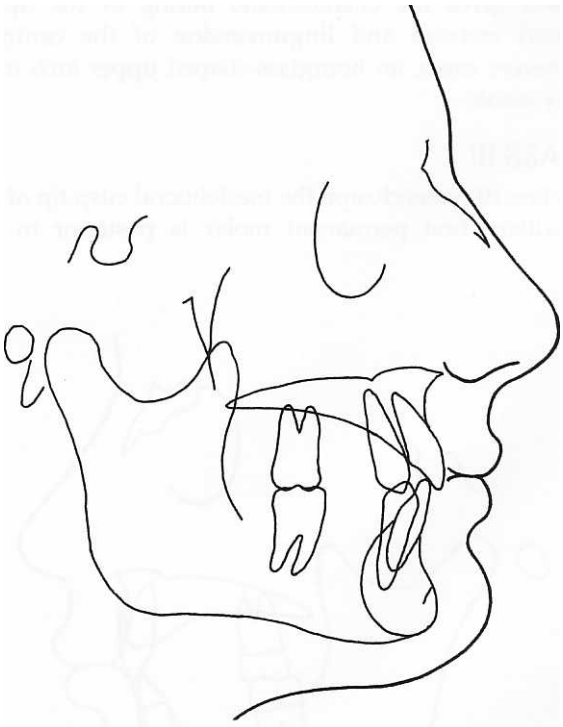


FIG. **26-17.** Brachyfacial pattern.

BRACHYFACIAL PATTERN

The short faces and wide, square mandibles of patients with a brachyfacial pattern are most often associated with class II, division II malocclusions. The mandibular growth of these patients is usually forward rather than downward. Consequently, these patients typically exhibit excessive anterior overbites and strong chins (Fig. 26-17). Esthetically the brachyfacial patient can generally accommodate a fuller dentition with a more

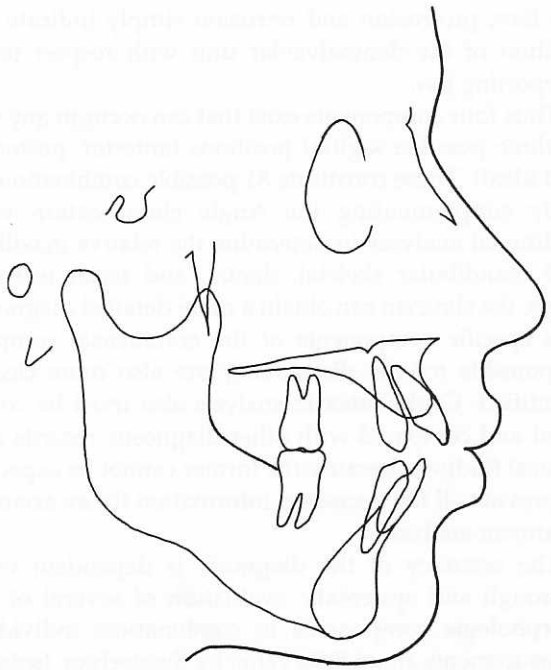


FIG. **26-18.** Open-bite pattern.

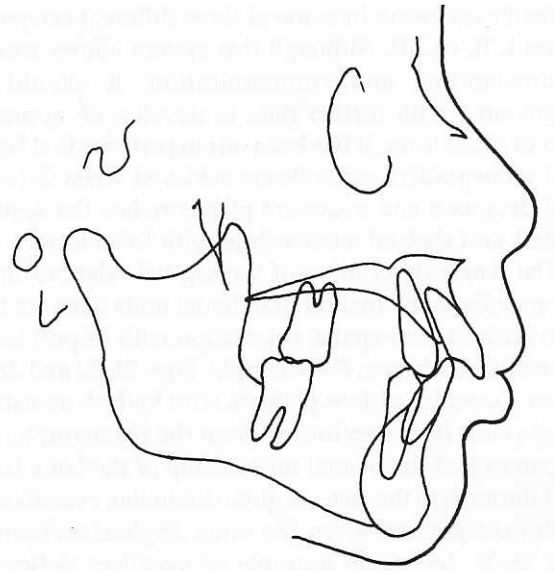


FIG. **26-19.** Deep-bite pattern.

acute interincisal angle. The fuller dentition helps balance the strong chin and the shorter lower facial height by giving more forward projection to the midfacial region.

VERTICAL ARCH DISCREPANCIES

OPEN BITE

Open-bite relationships are characterized by failure of the teeth in both arches to meet properly (Fig. 26-18). Open bites may be observed in the anterior or posterior region and may be attributable to supraeruption of the adjacent teeth or infraeruption of the teeth in the area of question. Open bites may be caused by abnormal habits, deviant growth patterns, or an abnormal tongue position.

DEEP BITE

Deep bites are most often observed in class II, division I malocclusion in which, because of the excessive overjet, the mandibular incisors supraerupt until they come into contact with the hard palate (Fig. 26-19). The class II, division II malocclusion is also associated with a deep bite, although in these patients the cause may be infraeruption of the posterior teeth or supraeruption of the maxillary anterior teeth. In many deep-bite cases, the condition results in overclosure of the mandible leading to labial movement of the upper incisors and, in some cases, generalized spacing of the maxillary anterior teeth. The correction leading to opening of the bite is determined by the type of malocclusion, the esthetic goals, and the philosophical approach of the clinician. A comprehensive case analysis is therefore necessary to define the etiologic factors.

ANGLE CLASSIFICATION OF OCCLUSION

Orthodontists are concerned with facial form, function, oral health, and beauty. The primary criteria for classifying the occlusion were developed in 1899 by Edward Angle, the father of modern orthodontics, to evaluate the sagittal relationship of the canines and molars. Angle was a nonextractionist who considered the bust of Apollo Belvedere to be the epitome of facial balance, and consequently it served as a guide to his treatment objectives. Angle contended that dental arch expansion was necessary for proper orthodontic treatment. With this method of treatment, Dr. Angle rarely removed teeth. Insightful orthodontists such as Charles Tweed, Hays Nance, and P.R. Begg refuted Angle's approach to treatment, which often resulted in poor facial esthetics, instability, and periodontal problems. These latter orthodontists often recommended the removal of teeth to improve facial esthetics and avoid double protrusion. Over the years the pendulum has swung back and forth between extraction and nonextraction. The fear of creating a "dished-in" profile has been reported. The statement has been made that the removal of premolars in orthodontic therapy will result in flat facial profiles and temporomandibular joint dysfunction. A study carried out at Washington University evaluated 160 extraction cases. The findings of the study indicate that, if proper diagnostic criteria are used in the treatment evaluation, the removal of teeth is not detrimental to good facial balance. The Angle system of classification (described earlier) is a simple analysis that allows one to classify a

patient's occlusion into one of three different categories (class I, II, or III). Although this system allows ease of understanding and communication, it should be augmented with further data to develop an appreciation of facial form. It has been our experience that better and more gratifying results are achieved when the original diagnosis and treatment plan correlate the sagittal, dental, and skeletal relationships with facial form.

The Angle description of the sagittal relationship of the maxillary and mandibular dental units does not take into account their spatial orientation with respect to the patient's facial type. For example, Figs. 26-20 and 26-21 show examples of two patients who both demonstrate Angle class III malocclusion. From the photographs it is apparent that the overall relationship of the bony bases and the teeth to the face are quite dissimilar, even though both examples are given the same Angle classification. Fig. 26-20 depicts an example of maxillary deficiency (retrognathism), whereas Fig. 26-21 is representative of a case demonstrating mandibular prognathism. Even though the Angle relationship is the same, the comprehensive diagnosis and treatment plan should be quite different if the most favorable result for each patient is to be achieved. Therefore, further supplementation of the Angle system of classification is in order.

DESCRIPTIVE SKELETAL AND DENTAL EVALUATION

Before the basic steps involved in comprehensive analysis are outlined, five descriptive terms must be defined and clearly understood. They are as follows:

Orthognathism. A skeletal term indicating the ideal balance between the cranial base, the maxilla, and the mandible from a sagittal view.

Prognathism. The anterior positioning of either one or both bony jaws relative to the facial skeleton and soft tissues. Hence the following entities may exist: maxillary prognathism, mandibular prognathism, or bimaxillary (maxillary and mandibular) prognathism.

Retrognathism. The posterior placement of either one or both jaws relative to the face. Similar entities can be demonstrated as were mentioned in the previous category.

Protrusion. A forward positioning of the dental units (teeth) relative to their bony base. Again, protrusion may occur with maxillary or mandibular teeth or both.

Retrusion. A posterior placement of either one or both dentoalveolar units relative to their respective bony bases.

Although the terms *prognathism* and *retrognathism* describe improper skeletal relationships of the jaws and the face, protrusion and retrusion simply indicate the relation of the dentoalveolar unit with respect to its supporting jaw.

Thus four components exist that can occur in any one of three possible sagittal positions (anterior, posterior, and ideal). These constitute 81 possible combinations.

By supplementing the Angle classification with additional analyses to determine the relative maxillary and mandibular skeletal, dental, and facial relation-ships, the clinician can obtain a more detailed diagnosis. The specific components of the craniofacial complex responsible for the discrepancy are also more clearly identified. Cephalometric analysis also must be correlated and compared with other diagnostic records and clinical findings, because the former cannot be expected to provide all the necessary information for an accurate treatment analysis.

The accuracy of the diagnosis is dependent on a thorough and systematic evaluation of several of the morphologic components in combination; individual measurements are of little value by themselves. Isolated measurements may demonstrate clinical deviation from the norm, but when these dimensions are combined with others, they may show collective compensation yielding a normal occlusal relationship. In contrast, a malocclusion may also be the result of individual dimensions that are considered within normal limits yet in combination result in an abnormal arrangement.

Any cephalometric measurement may be misleading at one time or another. Unfortunately there are no specific groups of factors that provide 100% accuracy. It is important to realize that the fewer the factors one uses in the analysis, the greater the risk of misdiagnosis.

It is the borderline cases that often require a more detailed analysis. Obvious cases involving severe skeletal dysplasia often can be diagnosed using relatively few factors.

Remember that, to properly analyze a cephalometric tracing, one must evaluate the interarch as well as the facial characteristics of the patient in question.

EVALUATION OF FACIAL ESTHETICS

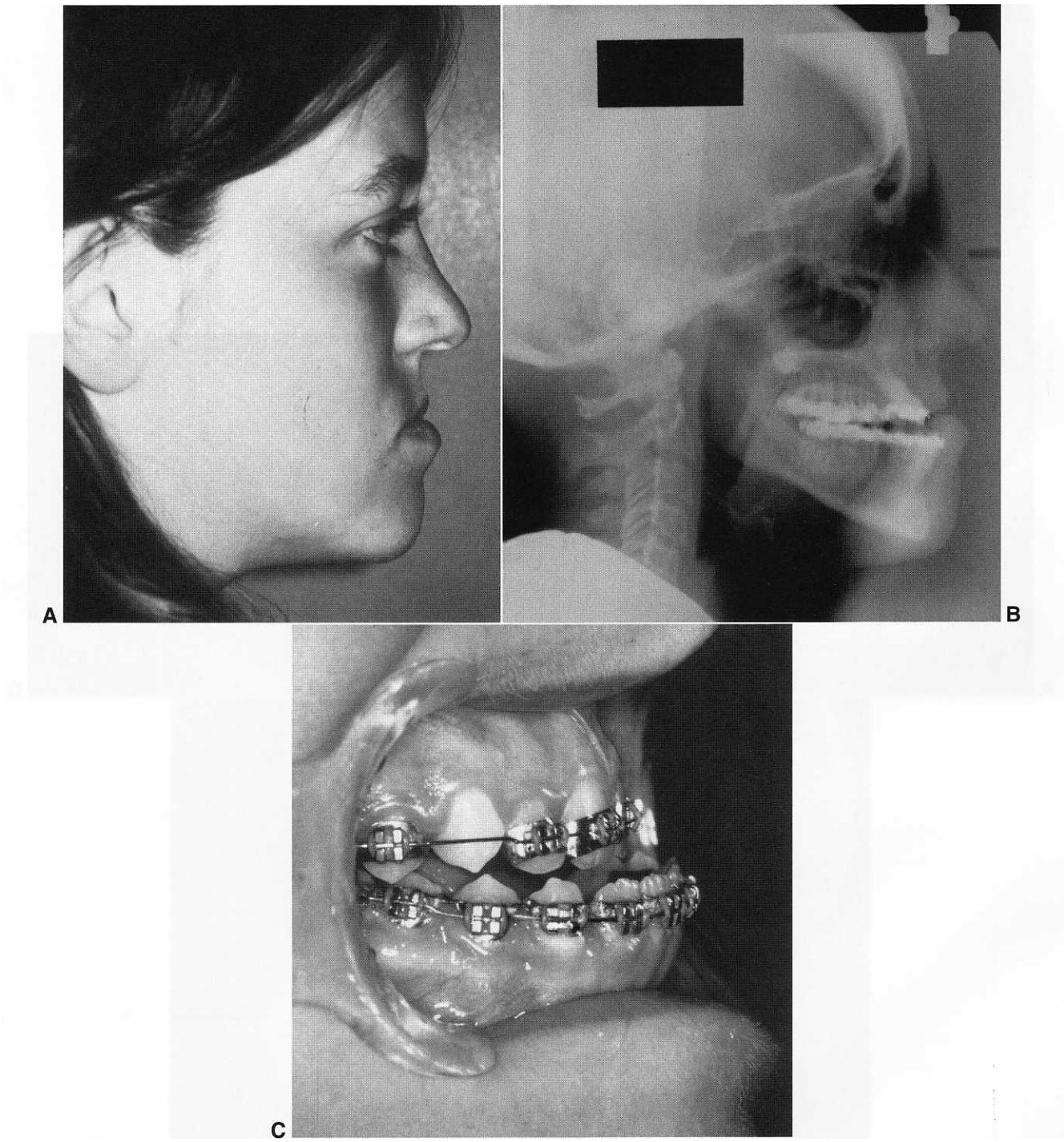
A thorough and systematic evaluation of a patient's facial structures forms the basis for an accurate diagnosis and subsequent treatment. Too often the lateral cephalometric radiograph and diagnostic casts are used as substitutes for a complete clinical examination of the patient's facial characteristics. Lines, angles, and numbers should not be used as the only diagnostic tools, but rather should be viewed as an adjunct to what our eyes reveal about the patient's facial form. To develop the clinical ability to observe variations from the normal facial structure, the practitioner must have a firm grasp of what constitutes an ideal face



[I G **26-20. A,** Maxillary retrognathism (clinical appearance). **B,** Maxillary retrognathism (cephalometric radiograph). C, Class III occlusion.

(complete with ideal variations). This section deals with the facial examination of the ideal face. The guidelines are directed at the adolescent and teenage patient and do not have complete application to the patient 5 to 10 years of age, because facial proportions generally change with the approach of puberty.

To properly evaluate the patient, the clinician should have the patient stand in a relaxed position. The patient's



li **26-21. A,** Mandibular prognathism (clinical appearance). **B,** Mandibular prognathism (cephalometric radiograph). C, Class III occlusion.

head should be positioned with the Frankfort horizontal plane parallel to the floor (Fig. 26-22). Do not ask the patient simply to "look straight ahead," because the patient will tend to place his or her head in the position the patient habitually prefers. It is also important to position the patient's occlusion in centric relation rather than centric occlusion. The patient's lips should be in repose during the examination. Patients frequently mask lip incompetence by forcing their lips together.

FRONTAL VIEW

The evaluation begins with the frontal view. This is the view people most often see of themselves. The balance

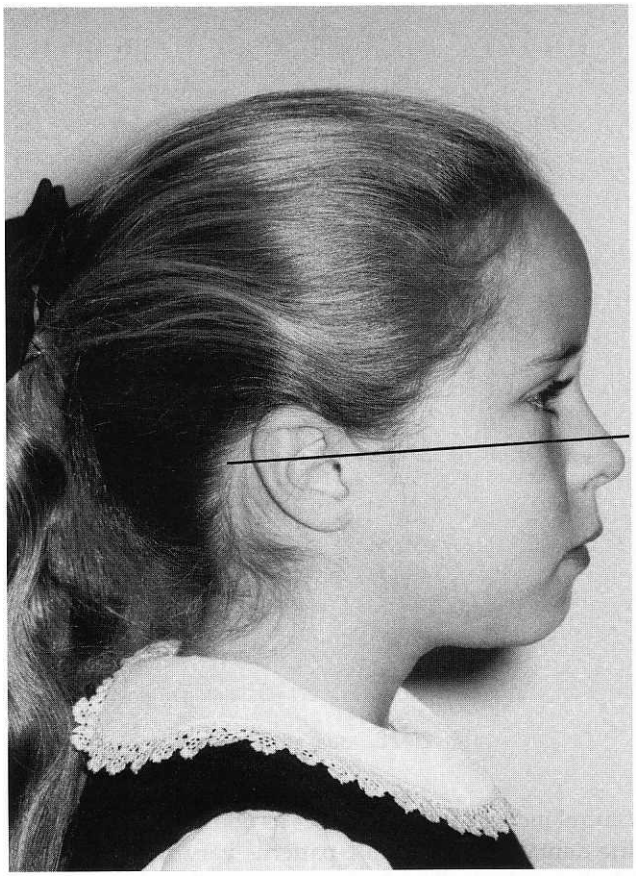


FIG **26-22.** Patient's head positioned with the Frankfort horizontal parallel to the floor.



FIG. **26-23.** Frontal facial thirds.

between the upper, middle, and lower thirds of the face is analyzed (Fig. 26-23). The upper third is bounded by the hairline (when combed back) and glabellar area. This area is least informative and is not the area to which corrections would normally be directed. More emphasis is placed on the proportions and symmetry of the middle third (from the glabellar region to subnasale) and the lower third (subnasale to menton).

In the middle third of the face, when the patient is looking straight ahead, the sclera of the eye is not seen superior or inferior to the pupil. Normal intercanthal distance is 30 to 32 mm (CD, ± 2 mm). Normal interpupillary distance is 60 to 65 mm. The inner and outer canthal tendons should fall close to a straight horizontal axis through the palpebral fissures (the fissures created when the eyelids are closed). The distance between the semilunar folds in the intercanthal area should approximate the alar base width (Fig. 26-24). Deviations from these general guidelines could indicate some deformity of the middle facial third.

Evaluation of the lower facial third is then carried out. The ratio of the middle and lower facial thirds in

vertical height should be approximately 5:6. The upper width.

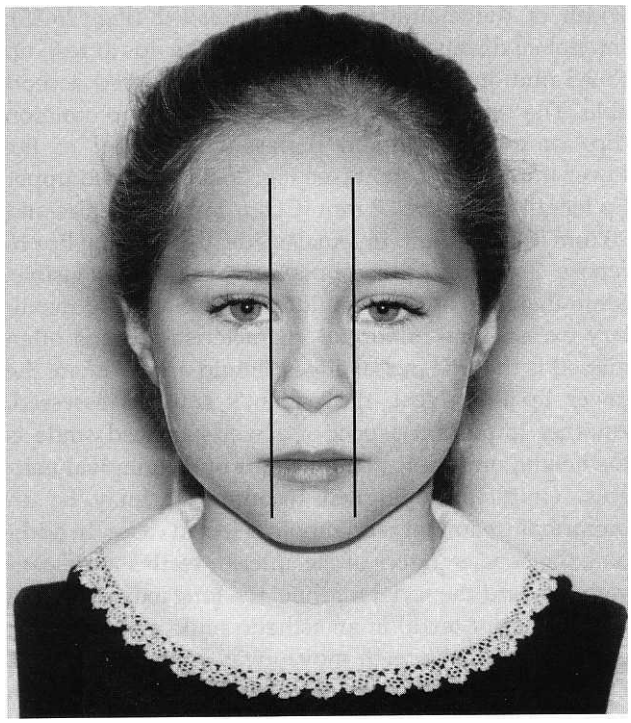


FIG . **26-24.** Comparison of the intercanthal distance and alar base

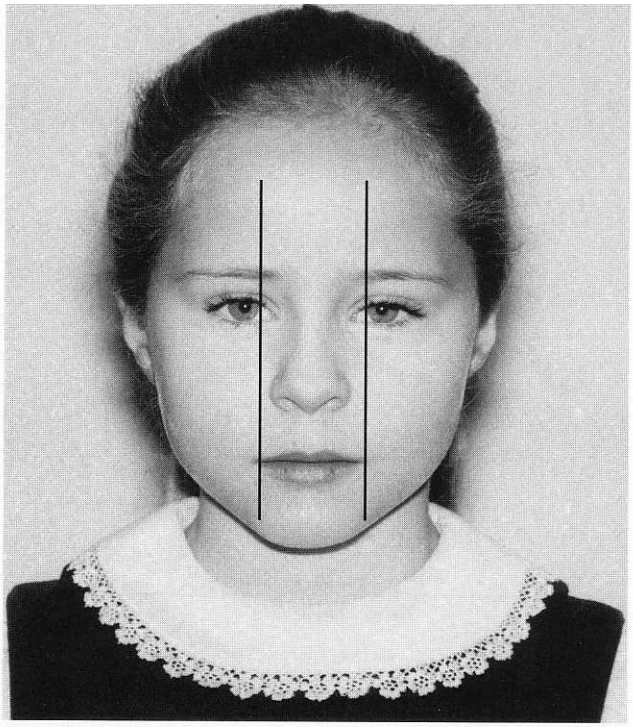


FIG. **26-25.** Comparison of the medial limbus width and the width of the mouth.



FIG. **26-26.** Ideal smile pattern.

lip and its relationship to the teeth are noted with the lips in repose and also during smiling. The distance between the medial limbus of the eyes should equal the width of the mouth when it is relaxed (Fig. 26-25). Interlabial distance is measured with the lips at rest; up to 3.5 mm of interlabial distance is considered acceptable. The upper lip length from subnasale to stomion (lip commissure) should represent one third of the lower third facial height (see Fig. 26-23). Normal upper lip length should be 22 mm (CD, ± 2 mm) in males and 20 mm (CD, ± 2 mm) in females. Ideally, with the lips in repose, 2 to 4 mm of the upper incisor should be visible. More than this amount could indicate a vertical maxillary excess. One important characteristic that is often missed is that the incisal edges should approximate the lower lip line. Next, the patient's smile is assessed. Another important aspect of a well-balanced smile is the height, uniformity, and contour of the gingival margins. Grafts and gingival recontouring may be an important treatment modality in selected cases. In addition, the facial surfaces of the anterior teeth should converge toward the facial midline; the long axis (direction of the anterior teeth) in an esthetic smile also follows a progression as the teeth move away from the midline. The separations between the maxillary anterior teeth help to define an attractive smile. The spaces between the edges of the teeth are known as embrasure spaces. These spaces follow a pattern that begins between the central incisors and progresses as one moves away from the dental midline. Smile patterns vary with individuals, but esthetically, when a person smiles, the upper lip vermilion should rest on or near the cervicogingival margin of the incisors (Fig. 26-26). The position of the lower lip is also noted. Excessive eversion of the lower lip is seen in patients with mandibular retrognathism (Fig. 26-27). The mentalis muscle can be hyperactive during forced closure of the lower lip (Fig. 26-28); such muscle hyperactivity is frequently seen in patients with mandibular retrognathia, vertical maxillary excess, apertognathia (open-bite deformity), and lip incompetence.

The last evaluation of the full face is that of facial symmetry. The face is divided in half by a line that bisects the glabella, nasal tip, upper lip, and chin (Fig. 26-29). The face is also divided vertically into equal fifths (Fig. 26-30).

PROFILE VIEW

The patient is next evaluated from the profile view. The examination considers many of the same features noted in the frontal examination. The face is divided into thirds (Fig. 26-31). A comparison of the vertical facial heights and the A-P relationship of the facial thirds constitutes the initial assessment. The vertical relationships are the same in the profile view as they are in frontal views.

The upper facial third profile establishes the relationship between the forehead and superior orbital rim.

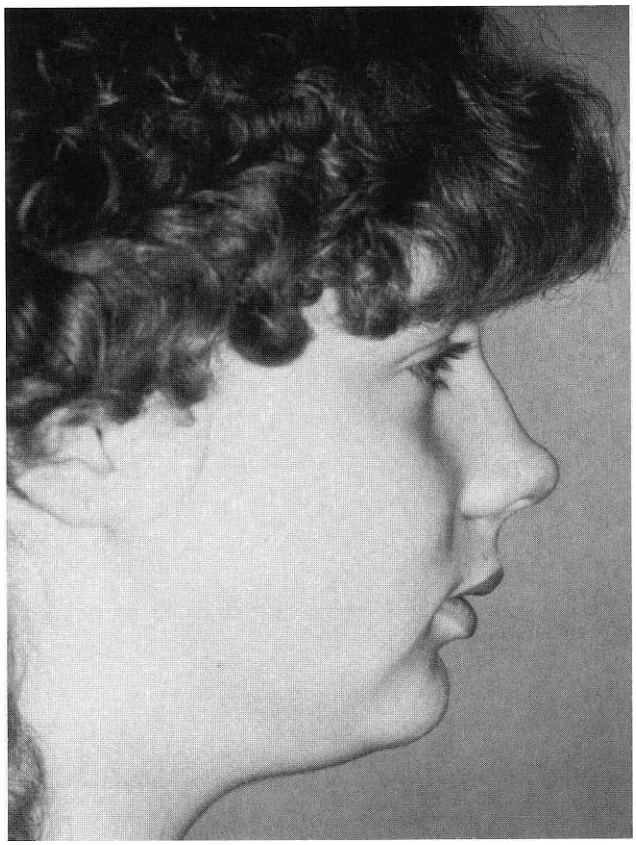


FIG **26-27.** Excessive eversion of the lower lip.

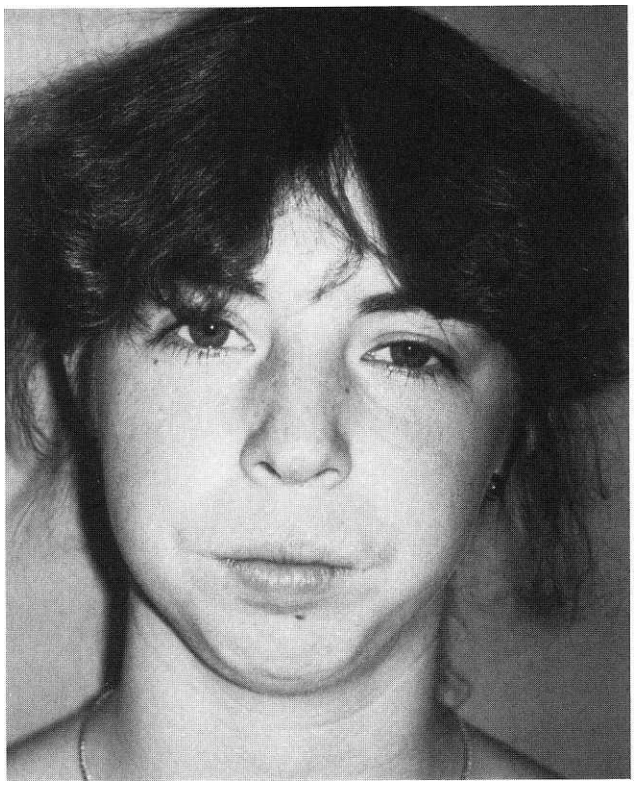


FIG. **26-28.** Hyperactive mentalis muscle during forced lip closure. Patient has lip incompetence.

The further the forehead protrudes beyond the superior orbital rim, the less esthetically acceptable it is. The globe of the eye and its relationship to the superior orbital rim is assessed. The superior orbital rim is normally 8 to 16 mm anterior of the globe.

The evaluation of the middle facial third involves an assessment of the relationship between the globes, inferior orbital rims, cheekbones, nose, and upper lip. The nasal bridge should project anterior to the globe by 5 to 15 mm. A reference line dropped from the most anterior projection of the globe and perpendicular to the Frankfort horizontal plane should fall on, or slightly behind, the soft tissue of the cheek.

In evaluation of the lower facial third, the vertical dimensions as described in the frontal view need to be considered. In addition, A-P assessments are done. Projection of the upper lip is clinically evaluated by measuring from the nasolabial fold to the subnasale and comparing that numeric value to the distance from the subnasale to the tip of the nose. Ideally this ratio should be 1:1 (Fig. 26-32). The relationship of the nose and upper lip is determined by measuring the nasolabial angle (Fig. 26-33). This value can range from 90 to 110 degrees.

The lower facial third is compared with the middle and upper thirds. The zero meridian is a straight line

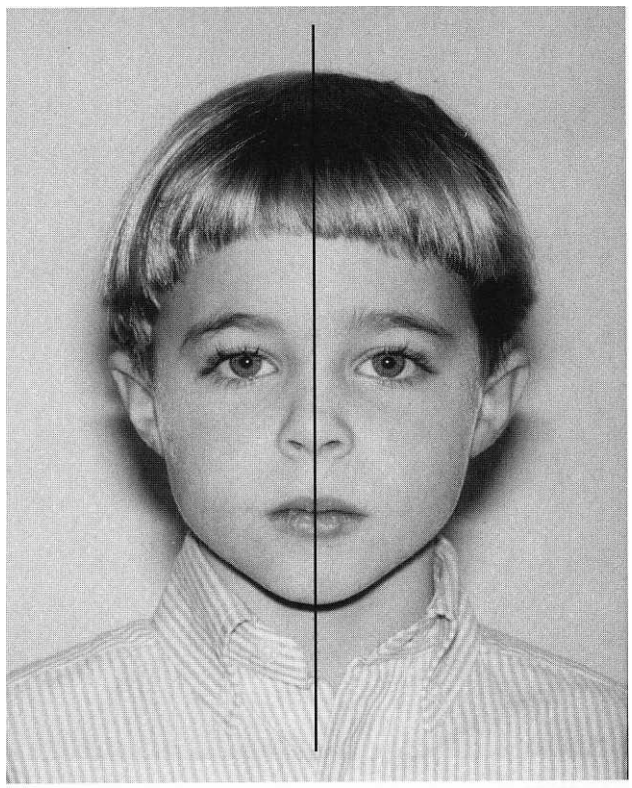


FIG. **26-29.** Facial symmetry.

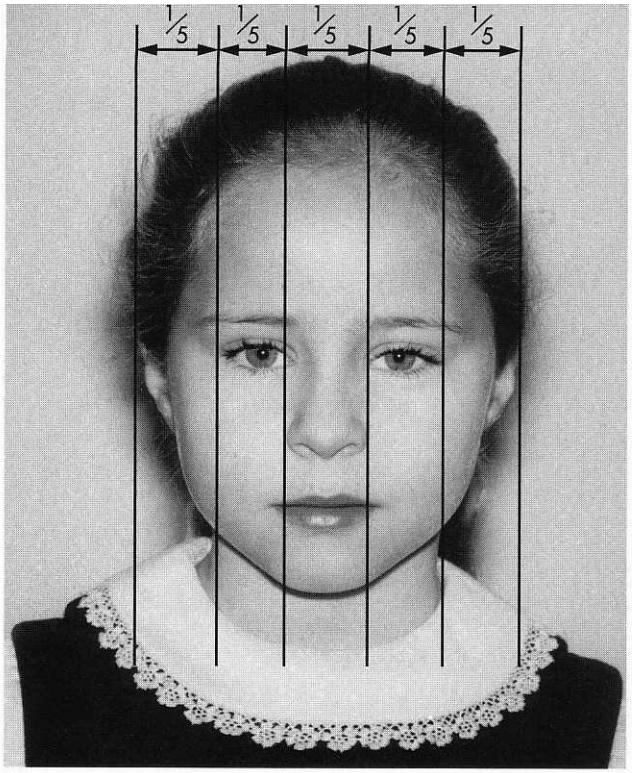


FIG. **26-30.** Vertical facial fifths.

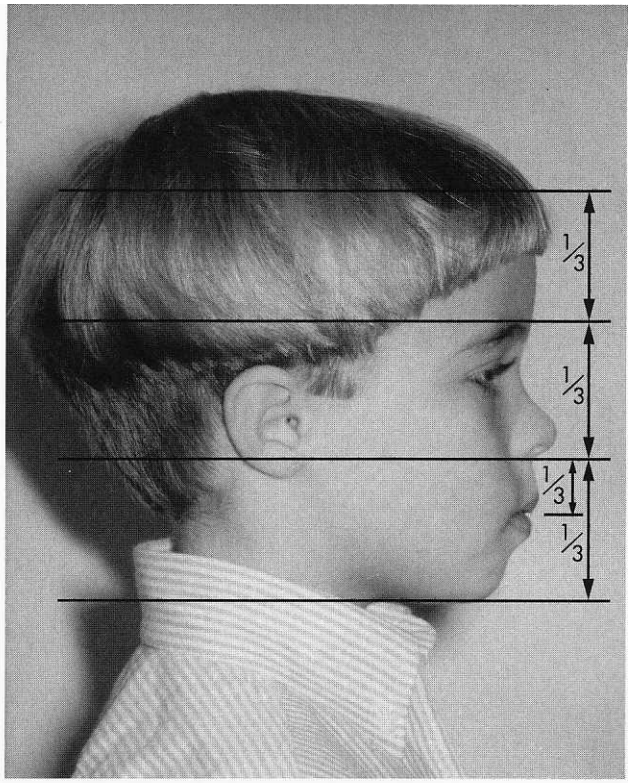


FIG. **26-31.** Profile facial thirds.

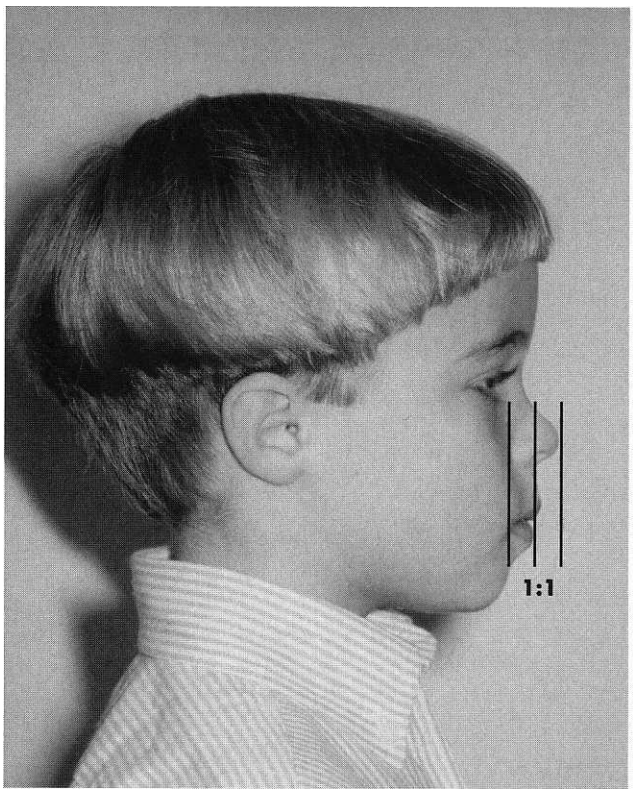


FIG. **26-32.** Ratio shows 1:1 relationship between nasolabial fold to subnasale, and subnasale to tip of the nose.

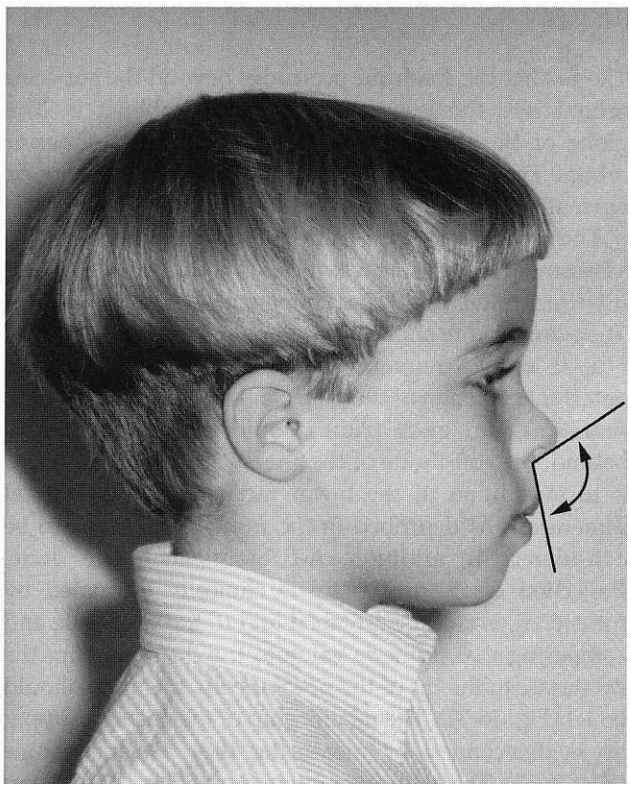


FIG **26-33.** Nasolabial angle.

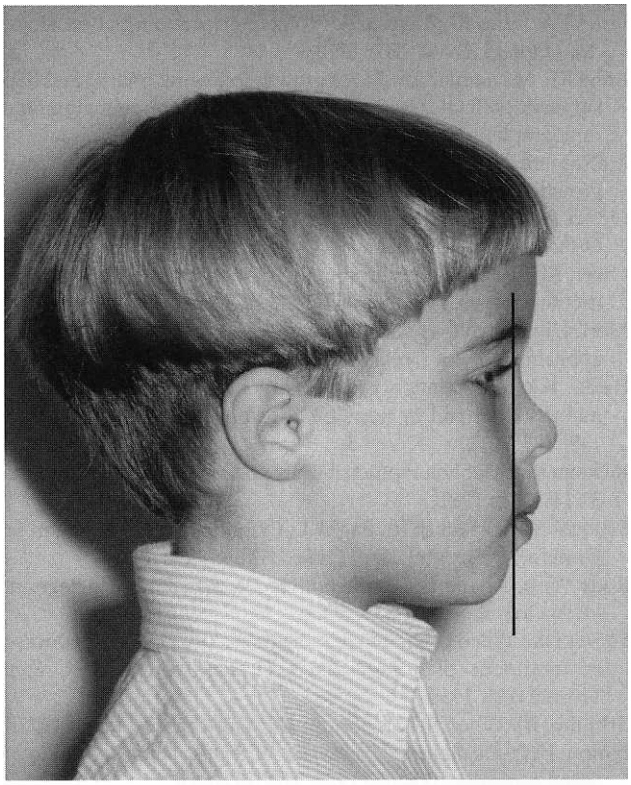


FIG. **26-34.** Zero meridian.

constructed by placing a line through the soft tissue nasion, perpendicular to the Frankfort horizontal plane. The lips and chin should fall near this line. (Fig. 26-34). During the profile evaluation of the lower facial third, any A-P discrepancies between the maxilla and mandible (i.e., prognathism and retrognathism) are noted. The upper and lower lip positions are also assessed. The shape and size of the chin button are appraised. In some cases, apparent mandibular retrognathism is in reality a flat or deficient chin button (microgenia). Conversely, too prominent a chin may be visualized as a pseudoprognathism and may be esthetically unacceptable.

The evaluation of the patient's face constitutes an important portion of the initial diagnosis and treatment plan phase of orthodontics. A systematic approach to examining the face is essential (Fig. 26-35). Basic guidelines of facial form have been reviewed. The values discussed are normal values for whites, but these must be used as relative guidelines only. The clinician must look at the patient's face and assess the overall harmony that exists. When deviations from normal facial form are detected, variations in treatment modalities must be considered to achieve better facial harmony. The main treatment objective is always to provide the patient with the best functional and esthetic result possible.

Lateral View

1. Frankfort Horizontal Plane: (FH)
   This plane is constructed from superior porion: (Po) to inferior orbitale (O) and represents the basic horizontal plane of the head.
2. Zero Meridian:
   The horizontal distance from the chin to a line perpendicular to (FH) and tangent to soft tissue nasion.
3. Maxillary Depth:
   The angle formed by the intersection of (FH) and N-A point planes.
4. Nasolabial Angle:
   The angle formed by the intersection of the lines tangent to the columella of the nose and the upper lip.

Lateral View Lip Measurements

1. Interlabial Gap:
   0-3.5 mm with lips in repose.
2. Upper Lip Length:
   Measured from (SN) to the inferior border of the upper lip.
3. Upper Incisor relative to the Upper Lip:
   The amount of upper incisor relative to the inferior border of the upper lip in repose.

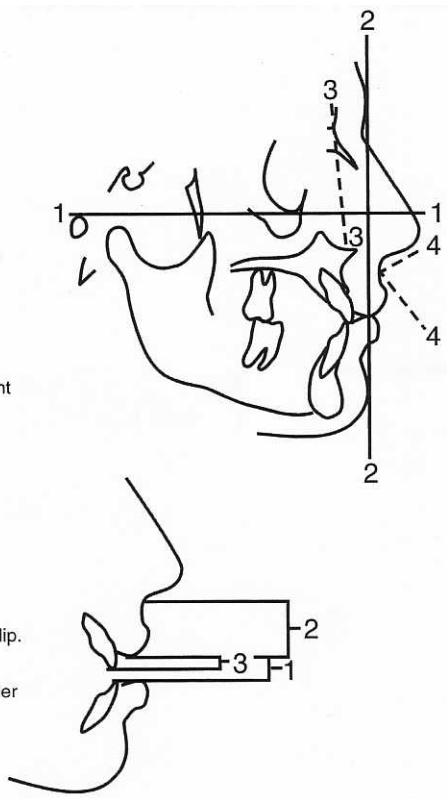


FIG **26-35.** Summary of lateral dentofacial measurements.

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CHAPTER

27

Management of the Developing Occlusion

JEFFREY A. DEAN
RALPH E. McDONALD
DAVID R. AVERY

انتشارات
شایان نمودار

CHAPTER OUTLINE

DEVELOPMENT OF THE OCCLUSION
- Diagnostic Records and Analysis
- Arch-Length Analysis
  - Nance analysis
  - Moyers mixed dentition analysis
  - Tanaka and Johnston analysis

PREVENTIVE MANAGEMENT OF THE DEVELOPING OCCLUSION
- Planning for Space Maintenance
- Factors Related to Arch-Length Adequacy
- Space Maintenance
  - Space maintenance for the first and second primary molar and the primary canine area
  - Loss of the second primary molar before eruption of the first permanent molar
  - Space maintenance for the primary and permanent incisor area
  - Space maintenance for areas of multiple tooth loss
  - Loss of the first permanent molar
  - Loss of the first permanent molar before the eruption of the second permanent molar
  - Loss of the first permanent molar after the eruption of the second permanent molar
- Cause and Correction of Deleterious Oral Habits
- Bruxism
- Digit and nonnutritive sucking behaviors
- Tongue and swallowing habits

INTERCEPTIVE ORTHODONTICS
- Anterior Crossbite in Primary and Mixed Dentition
  - Tongue blade therapy
  - Lower cemented bite plane
  - Palatal appliances
- Posterior Crossbite in Primary and Mixed Dentition
  - Soldered W arch
  - Cross-elastic technique
- Problems Related to the Eruption of Teeth
  - Ectopic eruption of the first permanent molars
  - Impacted second permanent molars
  - Ectopic eruption of the permanent lateral incisors
  - Impaction and delayed eruption of permanent canines
  - Supernumerary teeth and accompanying malocclusion
  - Anterior diastemas
  - Congenitally missing teeth
- Appliances to Regain Space
- Serial Extraction for the Pediatric Patient

COMPREHENSIVE ORTHODONTICS FOR THE DEVELOPING OCCLUSION
- Primary Dentition
- Mixed Dentition—Phase I
  - Treatment approaches
- Early Mixed Dentition—Phase II

t h e goal of every practitioner who provides dental care for children and adolescents should be to properly assess and manage the developing dental occlusion of their patients. Indeed, the following position statement is made by the American Academy of Pediatric Dentistry in the Guideline on Management of the Developing Dentition in Pediatric Dentistry':

Guidance of the eruption and development of the primary and permanent dentitions is an integral part of the specialty of pediatric dentistry. Such guidance should contribute to the development of a permanent dentition that is in a harmonious, functional, and esthetically acceptable occlusion. Pediatric dentists have the responsibility to recognize, differentiate, and either appropriately manage or refer abnormalities in the developing dentition as dictated by the complexity of the problem and the individual clinician's training, knowledge, and experience. Early diagnosis and successful treatment of developing malocclusions can have both short-term and long-term benefits while achieving the goal of occlusal harmony, function, and dental facial esthetics.

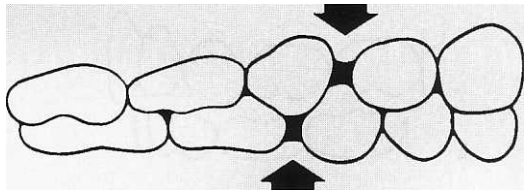
The following statement was made by Ngan, Wei, and Yen'-:

Pediatric dentistry has increasingly shifted from a conservative-restorative approach toward a concept of total pediatric patient care. Thus, all aspects of oral health care including diagnosis, prevention, oral medicine, restoration, and correction of malocclusion are increasingly the responsibility of the pediatric dentist.

With this background, it is the goal of this chapter to address management of the developing occlusion in the child and adolescent. The chapter progresses from a short review of the development of the normal occlusion to a discussion of current concepts in space maintenance for the pediatric patient. It also includes an overview of interceptive and comprehensive orthodontic care for the primary, mixed, and early permanent dentition.

DEVELOPMENT OF THE OCCLUSION

The supervision of the developing dentition and the initiation of preventive procedures, including space maintenance, require an understanding of the biogenetic course of the primary and permanent dentition. A review of the clinical studies by Baume provides this information.[3] Plaster study models of the primary dentitions of 30 children were made at various developmental stages. Two consistent morphologic arch forms of the primary dentition were found: either spaces between the teeth were present at all stages (type I) or the teeth were in proximal contact at all stages (type II). Spacing in the primary dentition is apparently congenital rather than developmental. Spaced arches frequently exhibit two



f I G **27-1.** Primate spaces between the maxillary primary lateral incisor and primary canine and between the mandibular primary canine and mandibular first molar. *(Adapted from Baume LJ: J* Dent Res *29:129, 1950.)*

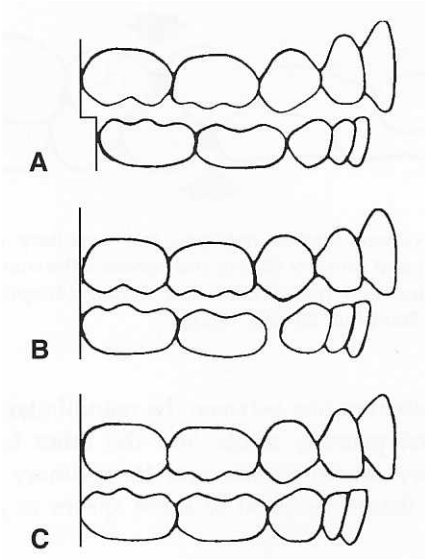
distinct diastemas: one between the mandibular canine and the first primary molar and the other between the maxillary lateral incisor and the primary canine (Fig. 27-1) . Baume referred to these spaces as *primate spaces.*

Baume observed that from about 4 years of age until the eruption of the permanent molars the sagittal dimensions of the dental arches remained essentially unchanged. A slight decrease in this dimension can occur either as a result of mesial migration of the primary second molar just after eruption or after the development of dental caries on the proximal surfaces of the molar teeth. Only minor changes in the transverse dimension of the maxillary and mandibular primary arches occurred during the period from $3^1/_2$ to 6 years of age.

A comparative study of models of the dentitions of 60 children before and after the eruption of the permanent molars revealed three distinct kinds of normal molar adjustment (Fig. 27-2). Any mesial shift of molars will use the leeway space. If this space is needed for the eruption of the premolars and canines, the molar shift should be prevented by a holding lingual arch.

Moyers believed that the pattern of transition involving the straight terminal plane is normal but that the occlusion forming a mesial step (distal surface of the lower second primary molar is mesial to the same surface of the maxillary molar) is more ideal.[4] Proper permanent molar occlusion was achieved by a late mesial shift of the mandibular permanent molars. A distal step (distal surface of the lower second primary molar is distal to the same surface of the maxillary molar) is abnormal and is indicative of a developing class II malocclusion.

Further evaluation of serial casts of the dentitions of 60 children were made by Baume at the time of eruption of the permanent incisors. A transverse widening of the mandibular arches occurred, representing a physiologic process to provide space for the erupting permanent incisors with their greater mesiodistal widths. This widening was brought about by lateral and frontal alveolar growth during the time of the eruption of the permanent incisors. The mean increase in intercanine



f G **27-2. A,** Occurrence of terminal plane forming a mesial step that allows the first permanent molar to erupt into proper occlusion. **B,** Straight terminal plane with primate space; an early shift of mandibular molars into the primate space allows proper first permanent molar occlusion (early shift). C, Straight terminal plane without primate space; proper first permanent molar occlusion is not attained until the mandibular second primary molar exfoliates, which then allows the desirable mesial shift of the mandibular first permanent molar (late shift).

width was greater in the maxillary arch than in the mandibular arch. The increase was also greater in previously closed upper or lower primary arches than in previously spaced arches. In the mandibular arch the greatest tendency to lateral growth was noticed during the eruption of the lateral incisors, whereas in the maxillary arch it occurred during the eruption of the central incisors. A secondary spacing of the maxillary primary incisors occasionally occurred when the still undeveloped maxillary arch was widened somewhat by the eruption of the mandibular permanent central incisors. Spaced primary arches generally produced favorable alignment of the permanent incisors, whereas about 40% of the arches without spacing produced crowded anterior segments.

Bishara et al noted similar arch development changes in their study of patients from 6 weeks to 45 years of age as follows':

> Significant increases in maxillary and mandibular arch width occurred between 6 weeks and 2 years of age.
> Although arch width increased significantly between 3 and 13 years of age, after complete eruption of the permanent teeth there was a slight decrease in the width, more in the intercanine than in the intermolar area.
> 9. The mandibular intercanine width was established by 8 years of age (i.e., after the eruption of the four incisors).

Therefore Bishara et al suggest that in the average patient there is no scientific basis for expanding the arches beyond the dimensions established at the time of the complete eruption of the canines and molars.

DIAGNOSTIC RECORDS AND ANALYSIS

The clinical examination should assess the patient's overall health status as well as the patient's occlusion (from both an aesthetic and a functional standpoint) and temporomandibular joint function. As for the diagnostic records that are necessary, the records obtained may be as simple as study models only, such as when treating a bilateral posterior crossbite, or as detailed as complete orthodontic records, such as when treating a 4-year-old patient with a class III skeletal malocclusion. These diagnostic records may include an eight-film series of extraoral and intraoral photographs, appropriately trimmed orthodontic study models, a full-mouth series or panoramic radiograph, lateral and anteroposterior cephalograms, and, when indicated, appropriate temporomandibular diagnostic views such as corrected axis tomograms or magnetic resonance imaging. The diagnostic records can be analyzed using many different methods. A form similar to the one used at the Indiana University School of Dentistry Pediatric Dentistry section is very helpful in composing the clinical examination and formulating the patient's diagnosis, problem list, and treatment plan summary (Fig. 27-3). Cephalometric analysis is reviewed in Chapter 26. Finally, the patient's neuromuscular growth and nasopharyngeal airway must be assessed. Patients who are obligate mouth breathers secondary to hypertrophic adenoid tissue or allergic conditions can have correspondingly poor postural relationships that can influence the developing skeletal face. Appropriate referral to the pediatrician or otolaryngologist for further assessment is warranted in these situations.

ARCH-LENGTH ANALYSIS*

Nance Analysis. Nance concluded, as a result of comprehensive studies, that the length of the dental arch from the mesial surface of one mandibular first permanent molar to the mesial surface of the corresponding tooth on the opposite side is always shortened during the transition from the mixed to the permanent dentition' Nance further observed that in the average patient's mandibular arch a leeway of 1.7 mm per side exists between the combined mesiodistal widths of the

*The revised Hixon and Oldfather method for analyzing arch length is presented in Chapter 25.

ORTHODONTIC DIAGNOSIS, TREATMENT, AND MECHANICS PLAN

Name _____ Race ____ Sex ____ Birthdate _____ Age ____ Chart No. _____

Resident's Name: _____ Records Date: _____

1. Patient History

A. Significant Medical History: _____

B. Patient's and/or Parents' Chief Complaint: _____

C. Attitude Toward Treatment: _____

2. Clinical Examination

A. Soft Tissue

Profile _____ Lip Competence _____ Lip/Incisor at Rest _____ Smiling _____

Oral Hygiene _____ Periodontal Status _____ Other _____

B. Occlusion Class: I II III Division: I II

Overjet _____ mm Overbite _____ mm Midline _____ mm

Crossbite _____

Molar Relation: Left _____ Right _____

Cuspid Relation: Left _____ Right _____

C. Dental Development Stage and Eruption Sequence:

D. Habits and/or Other Significant Clinical Findings:

E. TMJ and Function:

Opening Path: Normal Deviated: _____

Closing Path: Normal Deviated: _____

Range of Motion: Vertical ____ mm Protrusion: _____ mm

Left Deviation: _____ mm Right Deviation: _____ mm

Joint Sounds	None	Left	Right
	Opening	_____	_____
	Closing	_____	_____
	Crepitus	_____	_____

Muscle Tenderness: None _____

Tongue Function: Normal _____

3. Model Analysis **Static Tanaka and Johnston Analysis (JADA 1974)**

Total M-D Width of Upper Incisors ____ mm "A"

Maxillary Arch Length Discrepancy (From $\underline{6}|$ to $|\underline{6}$)

Total Predicted Tooth Mass:

[("B" ÷ 2) + 11 mm] × 2 + "A" = ____ mm

Total Measured Arch Length = ____ mm

Difference ____ mm

Total M-D Width of Lower Incisors ____ mm "B"

Mandibular Arch Length Discrepancy (From $\overline{6}|$ to $|\overline{6}$)

Total Predicted Tooth Mass:

[("B" ÷ 2) + 10.5 mm] × 2 + "B" = ____ mm

Total Measured Arch Length = ____ mm

Difference ____ mm

[I G **27-3.** Indiana University School of Dentistry Pediatric Dentistry section "Orthodontic Diagnosis, Treatment, and Mechanics Plan" form. Continued

4. Attach computerized cephalometric tracing and appropriate analysis.

5. Diagnostic and Arch Length Analysis Summary and Problem List

6. Treatment Plan or Objective Sequence

7. Mechanics Plan–Appliance Selection—Retention

8. Projected Treatment Time (With Good Compliance), Treatment Fees

9. Faculty Authorization to Start Treatment:

Signature

Date

f I G 27-3, cont'd. Indiana University School of Dentistry **Pediatric** Dentistry section " Orthodontic Diagnosis, Treatment, and Mechanics Plan" form.

primary mandibular canine and first and second primary molars and the mesiodistal widths of the corresponding permanent teeth, with the primary teeth being larger. This difference in the total mesiodistal width of the corresponding three primary teeth in the maxillary arch compared with the width of the three permanent teeth that succeed them is only 0.9 mm per side.

Currently the Nance arch-length analysis is seldom used, partly because the involved procedures for this analysis require a complete set of periapical radiographs. The clinical reliability of other analyses that do not use radiographs is sufficient for determining major arch-length inadequacies.

Moyers Mixed Dentition Analysis. The analysis advocated by Moyers has numerous advantages. It can be completed in the mouth as well as on casts, and it may be used for both arches.' The analysis is based on a correlation of tooth size; one may measure a tooth or a group of teeth and predict accurately the size of the other teeth in the same mouth. The mandibular incisors, because they erupt early in the mixed dentition and may be measured accurately, have been chosen for measurement to predict the size of the upper, as well as the lower, posterior teeth.

Tanaka and Johnston Analysis. The Tanaka and Johnston method of arch-length analysis is a variation of Moyers' analysis except that a prediction table is not needed.' The estimated widths in millimeters of the unerupted canines and premolars correspond to the 75% level of probability in Moyers' prediction table. The sum of the widths of the mandibular permanent incisors is measured and divided by 2. For the lower arch, 10.5 mm is added to the result and, for the upper arch, 11 mm is added to the result to obtain the total estimated widths of the canines and premolars. For example, if the width of the lower incisors is 23 mm, divide by 2 and add 10.5 mm for the lower arch. The result is 22 mm compared with 22.2 mm obtained from Moyers' table. The corresponding values for the maxillary arch are 22. 5 mm according to the Tanaka and Johnston analysis and 22.6 in Moyers' table. One can then take these tooth mass predictions and compare them with the total measured arch length and obtain any redundancies or inadequacies in the arch length.

Irwin, Herold, and Richardson reviewed the various methods of mixed dentition analysis.' They concluded that the Hixon and Oldfather method is more accurate and any error involves a consistent underprediction of tooth size, which is of less clinical significance than the overprediction of tooth size, as seen in all the other methods. Its disadvantage is that it is more cumbersome. Moyers claimed that, for the measure of mandibular incisors alone, 95% of the dentitions of patients with a combined width of canine and premolar teeth within 1 mm of the predicted value should be clinically acceptable.' Therefore the Tanaka and Johnston analysis provides significant clinical acceptability with a minimal amount of time and effort.'

Another particularly valuable study model analysis is the Bolton analysis." This analysis addresses tooth mass discrepancies between the maxillary and mandibular arches. It can be used to compare the sum of the mesiodistal widths of the 12 maxillary teeth with that of the 12 mandibular teeth, first molar to first molar, and to compare the 6 maxillary teeth with the 6 mandibular teeth, canine to canine. The Bolton analysis ratio is as follows:

(Sum mandibular)/(Sum maxillary) x 100 = Tooth mass ratio

For the overall ratio (12 teeth versus 12 teeth), the mean is 91.3 (±1.91)%. For the anterior ratio (6 teeth versus 6 teeth), the mean is 77.2 (±1.65)%.

When a significant discrepancy with these ratios is noted, the clinician must assess where the tooth mass problem is located and decide on the best method to resolve it. It is not always an easy matter to detect a problem. A common example is smaller than normal maxillary lateral incisors. Depending on the size of the discrepancy and the patient's overall malocclusion, two methods that might be used to resolve this problem include slenderization of the mandibular anterior teeth or bonding to increase the mesiodistal width of the lateral incisors.

PREVENTIVE MANAGEMENT OF THE DEVELOPING OCCLUSION

PLANNING FOR SPACE MAINTENANCE

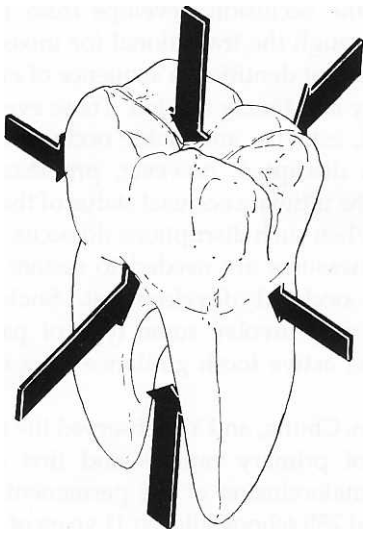
Ideally, as the occlusion develops from the primary dentition through the transitional (or mixed) dentition to the permanent dentition, a sequence of events occurs in an orderly and timely fashion. These events result in a functional, esthetic, and stable occlusion. When this sequence is disrupted, however, problems arise that may affect the ultimate occlusal status of the permanent dentition. When such disruptions do occur, appropriate corrective measures are needed to restore the normal process of occlusal development. Such corrective procedures may involve some type of passive space maintenance, active tooth guidance, or a combination of both.

Miyamoto, Chung, and Yee observed the effects of the early loss of primary canines and first and second molars on malocclusion of the permanent dentition.[1]" They studied 255 schoolchildren 11 years of age or older at the final examination of the permanent dentition. Malocclusion was evaluated by scoring malalignment and measuring crowding in the anterior teeth. Children who had a premature loss of one or more canines or

molars more commonly received orthodontic treatment for the permanent dentition. The likelihood of needing orthodontic treatment increased with the number of prematurely lost teeth. The frequency of orthodontic treatment in children who had lost one or more primary teeth through 9 years of age was more than three times higher than in the control group. In untreated children there was no detectable relationship between the premature loss of canines and the malalignment of permanent teeth. However, the premature extraction of molars significantly affected alignment and was especially associated with major malalignment of permanent teeth. No differences in effects were observed between the loss of the first and second primary molars. Crowding of the anterior teeth was directly affected by the premature loss of primary canines.

A tooth is maintained in its correct relationship in the dental arch as a result of the action of a series of forces (Fig. 27-4). If one of these forces is altered or removed, changes in the relationship of adjacent teeth will occur and will result in drifting of teeth and the development of a space problem. There is lack of agreement regarding the frequency with which space closure will occur or a malocclusion will develop after premature loss of a primary or a permanent tooth; however, the following general factors influence the development of a malocclusion:

1. *Abnormal oral musculature.* High tongue position coupled with a strong mentalis muscle may damage the occlusion after the loss of a mandibular primary molar. A collapse of the lower dental arch and distal drifting of the anterior segment will result.
2. *Oral habits.* Thumb or finger habits cause abnormal forces on the dental arch and are responsible for initiating a collapse after the untimely loss of teeth.
3. *Existing malocclusion.* Arch-length inadequacies and other forms of malocclusion, particularly class II, division 1, usually become more severe after the untimely loss of mandibular primary teeth.
4. *Stage of occlusal development.* In general, more space loss is likely to occur if teeth are actively erupting adjacent to the space left by the premature loss of a primary tooth.



[I 1 **27-4.** Forces that act on a tooth to maintain its relationship in the arch. If one of these forces were removed, as would be the case if a tooth mesial to the tooth shown were extracted, forward tipping and mesial drifting would occur.

The following factors are important when space maintenance is considered after the untimely loss of primary teeth.

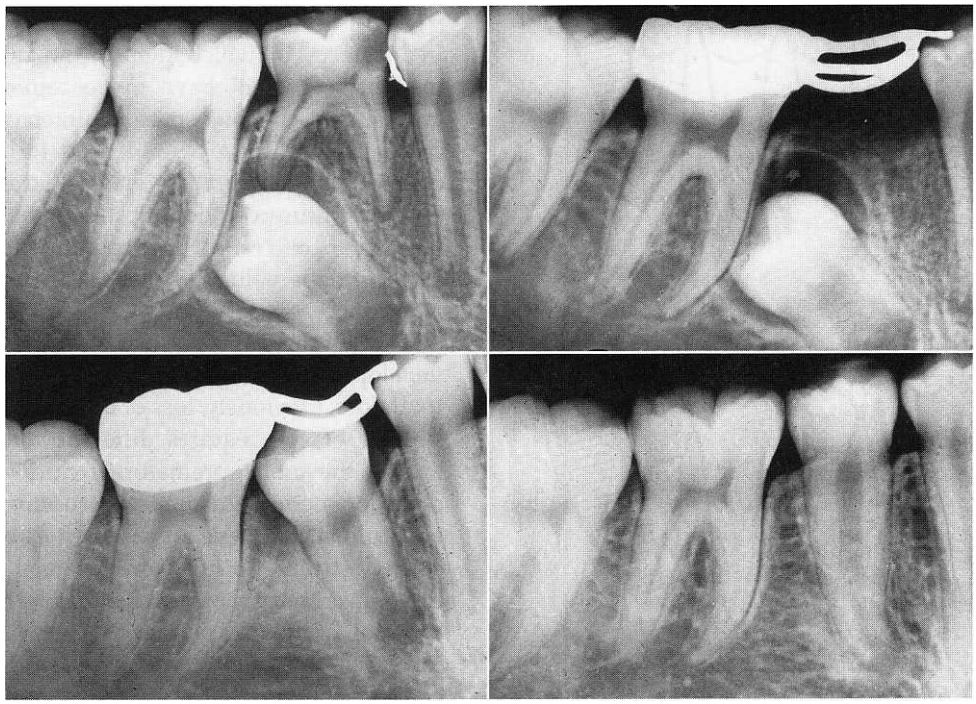
1. *Time elapsed since loss.* If space closure occurs, it usually takes place during the first 6 months after the extraction. When a primary tooth is removed and all factors indicate the need for space maintenance, it is best to insert an appliance as soon as possible after the extraction.
2. *Dental age of the patient.* The chronologic age of the patient is not as important as the developmental age. Cron studied the emergence of permanent teeth based on the amount of root development, as viewed on radiographs, at the time of emergence." She found that teeth erupt when three fourths of the root is developed, regardless of the child's chronologic age. Several studies have indicated that the loss of a primary molar before 7 years of age (chronologic) will lead to delayed emergence of the succedaneous tooth, whereas the loss after 7 years of age leads to an early emergence. The magnitude of this effect decreases with age. In other words, if a primary molar is lost at 4 years of age, the emergence of the premolar could be delayed by as much as 1 year; emergence will occur at the stage of root completion. If the same primary molar is lost at 6 years of age, a delay of about 6 months is more likely; emergence will occur at a time when root development approaches completion.
3. *Amount of bone covering the unerupted tooth.* Predictions of tooth emergence based on root development and the influence of the time of the primary tooth loss are not reliable if the bone covering the developing permanent tooth has been destroyed by infection. In such a situation the emergence of the permanent tooth is usually accelerated. If there is bone covering the crowns, it can

be readily predicted that eruption will not occur for many months; insertion of a space-maintaining appliance is indicated. A guideline for predicting emergence is that erupting premolars usually require 4 to 5 months to move through 1 mm of bone as measured on a bite-wing radiograph.

4. *Sequence of the eruption of teeth.* The dentist should observe the relationship of developing and erupting teeth adjacent to the space created by the untimely loss of a tooth. A similar situation exists if the first primary molar has been lost prematurely and the permanent lateral incisor is in an active state of eruption. The eruption of the permanent lateral incisor will often result in a distal movement of the primary canine and an encroachment on the space needed by the first premolar. This condition is frequently accompanied by a shift in the midline toward the area of the loss. In the mandibular arch a "falling in" of the anterior segment may occur and an increased overbite may result.
5. *Delayed eruption of the permanent tooth.* Individual permanent teeth are often observed to be delayed in their development and consequently in their eruption. It is not uncommon to observe partially impacted permanent teeth or a deviation in the eruption path that will result in abnormally delayed eruption. In cases of this type it is generally necessary to extract the primary tooth, construct a space maintainer, and allow the permanent tooth to erupt and assume its normal position (Fig. 27-5).-
6. *Congenital absence of the permanent tooth.* If permanent teeth are congenitally absent, the dentist must decide whether to hold the space for many years until a fixed replacement can be provided or to allow the space to close.

FACTORS RELATED TO ARCH-LENGTH ADEQUACY

Before placing space maintainers or starting tooth movement, the dentist must thoroughly evaluate arch length. This is particularly important during the primary and mixed dentition periods. Regardless of the arch-length analysis method used, several factors other than linear arch length and tooth size must be considered. First, the position of the lower incisors over basal bone must be determined. If the teeth are retroclined, one may obtain additional arch length by placing them in a more normal axial inclination. If the lower incisors are near their upper limit when measured to the mandibular plane on a cephalogram, further flaring or anterior advancement would jeopardize the periodontal support of these teeth. Also, the degree of crowding and amount of space needed to correctly align the anterior segment must be determined. Generally,

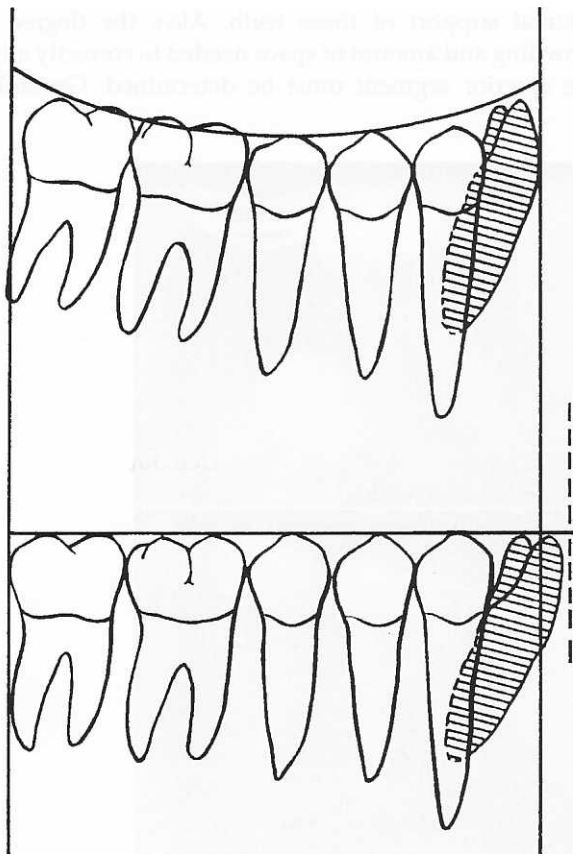


II G **27-5.** Extraction of the second primary molar and space maintenance were indicated because of prolonged retention of the primary tooth and partial impaction of the second premolar. The second premolar eventually erupted into its normal position.

every overlapped contact will require at least 1 mm or more of space for correction, depending on the severity of crowding.

Using radiographic measurements, prediction charts, or combinations of the two, one should determine the size of the unerupted premolars and canines. In some instances, unusually large permanent teeth will necessitate removal of teeth because of the significant tooth mass/arch-length discrepancy. The depth of the curve of Spee may also influence available arch length. According to Andrews, the ideal occlusion will have a nearly flat or very slight curve of Spee.' When leveled, the teeth will require more linear space than they occupied before. Generally, 1 mm of linear space is required per side for every millimeter of the depth of the curve of Spee (Fig. 27-6).

Finally, if the leeway space as described by Nance is not used, the total arch length will be decreased further as the permanent molars shift mesially. Whether this space should be maintained with holding arches depends on the space requirements as determined from an arch-length analysis. In some instances, holding leeway space may allow the permanent premolars and canines to erupt and still provide some space to alleviate anterior crowding. If the leeway space is not held, future orthodontic treatment may require premolar extractions to allow the anterior segment to be aligned.



f **I G 27-6.** When leveling the curve of Spee shown here, one needs approximately 4 mm additional length (2 mm on each side), because the average depth is 2 mm.

The available arch circumference (arch length), which is the distance from the mesial surface of the first permanent molar on one side of the arch to the mesial surface of the first permanent molar on the opposite side of the arch, is continually decreasing. Arch length decreases through the proximal wear and mesial movement of the first permanent molars at the time of the exchange of teeth. Moorrees reported that the average arch length of an individual is smaller at 18 years of age than at 3 years of age.'[3] This is the result of the decrease in maxillary and mandibular dental arch length that occurs between 10 and 14 years of age, caused by the exchange of primary molars for the first and second premolars.

Barber believes that the goal should be the prevention of arch-length loss in any degree, no matter how small." He points out that the combined mesiodistal widths of the primary teeth essentially equal the combined mesiodistal widths of their permanent successors in the same arch. Thus the leeway described by Nance between the combined mesiodistal widths of the primary canine, first and second molars, and their successors may be needed to allow the already erupted permanent incisors to unwind and alleviate anterior crowding in many persons.'

SPACE MAINTENANCE

In instances in which there is no question that permanent teeth will have to be removed to obtain a favorable occlusion, space maintenance may not be advisable; the space would need to be closed during orthodontic treatment anyway. Regardless of the possibility of future orthodontic treatment for the patient, holding space to allow the teeth to erupt and to prevent impactions is valuable. Teeth always can be removed later if necessary.

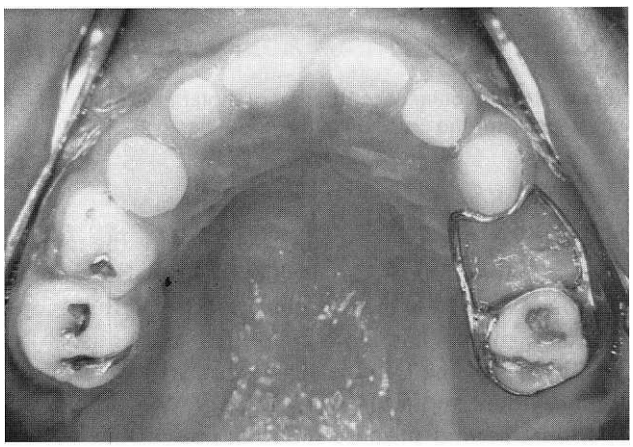
Another factor involved in space management is the developmental stage of the occlusion at the time of tooth loss. Obviously, loss of a second primary molar at 5 years of age requires different considerations than loss of the molar prematurely during the late mixed dentition period. Also, teeth erupting adjacent to the edentulous area have a greater effect on the amount of space lost than do fully erupted teeth. For example, if the first primary molar is lost during the time of active eruption of the first permanent molar, a strong forward force will be exerted on the second primary molar, causing it to tip into the space required for the eruption of the first premolar. Changes in the occlusion may extend as far as the midline after the loss of the first primary molar; a shift of the midline toward the space

is created by the untimely loss, and retrusion of the anterior segment on the affected side and an increased overbite may result.

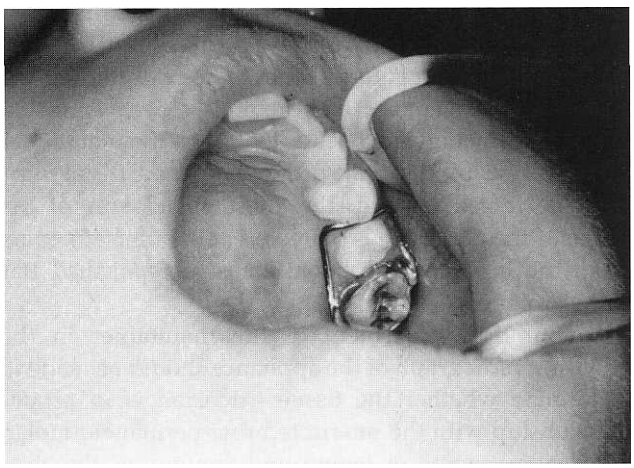
Space Maintenance for the First and Second Primary Molar and the Primary Canine Area

The Band and Loop Maintainer. The space maintainer called a *band and loop* is easy and economical to make, takes little chair time, and adjusts easily to accommodate the changing dentition (Figs. 27-7 and 27-8). However, it does not restore chewing function and will not prevent the continued eruption of the opposing teeth; these may or may not be important factors, depending on the case requirements.

The stainless steel crown and loop maintainer may be used if the posterior abutment tooth has extensive caries and requires a crown restoration or if the abutment tooth has had vital pulp therapy, in which case it is desirable to protect the crown with full coverage. The steel crown should be prepared as described in Chapter 18. I-Iowever, before cementation, a compound impression is made, the crown is removed from the tooth and seated in the impression, and the stone working model is prepared. A piece of 0.036-inch steel wire is used to prepare the loop. The advantages of the crown and loop maintainer are similar to those of the band and loop. Because it is difficult to remove the crown to make adjustments in the loop, some dentists prefer to adapt a band over a cemented crown restoration and construct a conventional band and loop appliance.



f **127-7.** Trial seating of a properly fitted and designed band and loop appliance. The appliance is ready to be cemented into place.



f IG **27-8.** Band and loop maintainer. The loop is sufficiently large to allow the eruption of the permanent tooth.

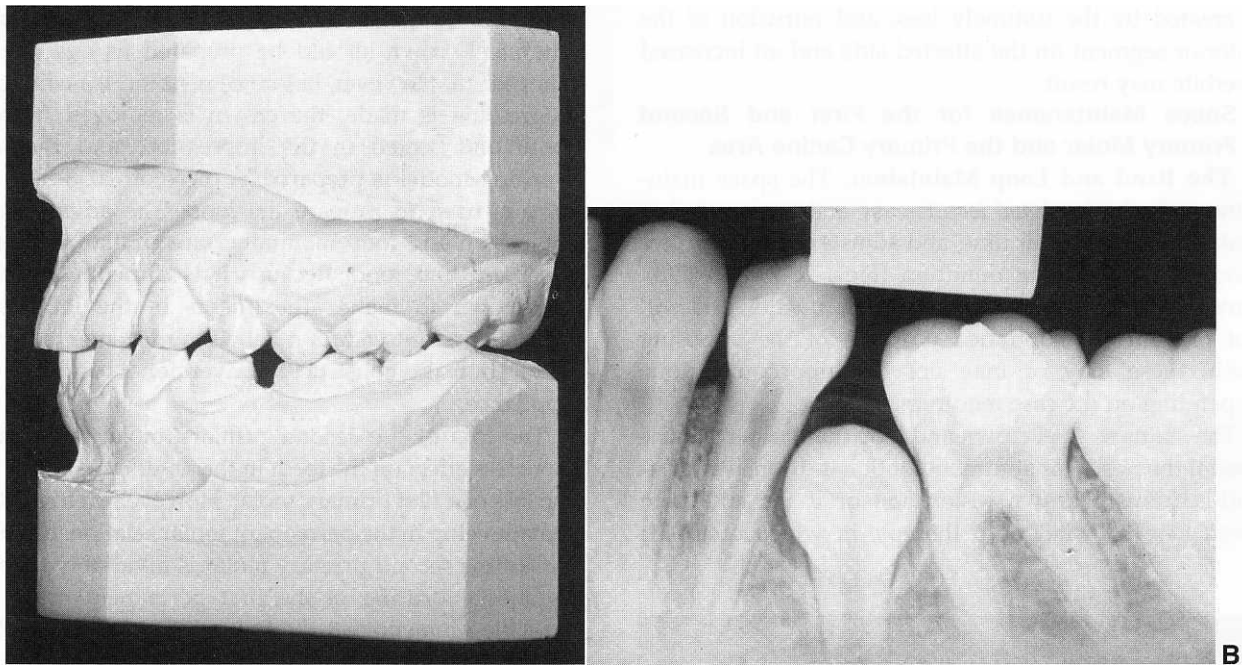
The loss of the second primary molar will usually have less effect on the teeth in the anterior segment than the loss of a first primary molar. However, an irregularity may develop in the permanent molar relationship. Early loss of the second primary molar is invariably followed by mesial drifting of the first permanent molar and possible impaction of the second premolar (Fig. 27-9). Also a maxillary molar often will rotate mesial in.

The space-maintaining appliances that are generally advocated when the second primary molar is lost are the band and loop and the passive lingual arch. The passive lingual arch is discussed later in the chapter. In relatively uncomplicated cases, the band and loop maintainer with the band placed on the first permanent molar can be used (Fig. 27-10). If the first primary molar is the abutment tooth, it may be lost before the time when the space-maintaining appliance can be discarded.

Loss of the primary canine is infrequently caused by dental caries but may occur at the time of the eruption of the permanent lateral incisor. When the loss of the primary canine occurs prematurely and there has been no shift in the midline or space closure, a band and loop or a lingual arch with a spur can be used. The first primary molar is the abutment tooth if a band and loop is made. If the permanent molars can be banded, the lingual arch is probably the preferred appliance.

A band and loop may require a vertical projection in which the loop contacts the abutment tooth (see Fig. 27-5). This occlusal rest can prevent tipping and can prevent the anterior portion of the loop from sliding below the proximal height of contour, which can lead to the wire's embedding in the gingival tissue several weeks or months later.

In the mixed dentition, a passive soldered lingual arch is almost always the appliance of choice in the mandibular arch, especially if the permanent mandibular incisors exhibit crowding. Also, the upper lingual arch (Nance appliance) or the lower lingual arch is the appliance of choice when bilateral space loss is present or when leeway space must be preserved. The use of unilateral and bilateral band and loop maintainers is



[I C **27-9. A**, Casts showing the result of the early loss of a mandibular second primary molar. **B,** Radiograph of an impacted second premolar. Space needed for the eruption of this tooth has been almost completely lost.

not indicated to manage leeway space; however, bilateral band and loop maintainers are needed before the eruption of the permanent incisors.

Loss of the Second Primary Molar Before Eruption of the First Permanent Molar

Mesial movement and migration of the first permanent molar often occurs before eruption in instances of premature loss of the second primary molar (Fig. 27-11). This is one of the most difficult problems of the developing dentition to confront the pediatric dentist. Use of a space maintainer that will guide the first permanent molar into its normal position is indicated.

The Distal **Shoe Appliance.** Roche has advocated a crown and band appliance with a distal intragingival extension.[15] This appliance or modifications of it may be used to maintain space or, in some instances, to influence the active eruption of the first permanent molar in a distal direction (Fig. 27-12).

Using the first primary molar as the abutment, the dentist first prepares the tooth for a stainless steel crown, which is carefully contoured and cemented. The stainless steel crown provides a desirable retentive contour for the placement of a stainless steel band, although a well-adapted band on a noncrowned tooth may be satisfactory.

The band is placed over the steel crown on the abutment tooth. A compound impression is made, the band is removed and placed in the impression, and a stone model is prepared. If the second primary molar has not yet been removed, it is cut off the model. A hole that simulates the position of the distal root of the tooth is made in the model using a bur. If the second primary molar has been removed previously, the positioning of the tissue extension may be determined with dividers and a bite-wing radiograph. If the corresponding second primary molar is present, the correct mesiodistal width can be measured from it.

The tissue-bearing loop is next contoured with a 0.040-inch wire extending distally and into the prepared opening on the model. The free ends of the loop are soldered to the band. Next, the band and loop appliance is removed from the model and the v of the tissue extension is filled in and soldered with pieces of 0.040-inch wire. A knife edge is formed at the apex of the V if the second primary molar has previously been extracted and the extraction site has healed. Thus the maintainer may still be used, because the sharpened distal shoe may be forced through the anesthetized area of the ridge. If the appliance is delivered at the time of extraction, the intragingival extension is just polished but not sharpened.

Before final placement of the maintainer in the mouth, a radiograph of the appliance should be made to determine whether the tissue extension is in proper relationship with the unerupted first permanent molar. Final adjustments in length and contour of the shoe may be made at that time. The soft tissue has been observed to tolerate the extension of this type of



El G. **27-10. A,** Pulpless second primary molar is indicated for extraction. Although there was considerable loss of bone covering the crown of the second premolar, there was concern about space loss before eruption of the tooth. **B,** A band and loop maintainer has been constructed immediately after the removal of the pulpless primary tooth. C, The second premolar has erupted through the loop. The maintainer may now be removed. D, Adequate space has been maintained in the arch of the second premolar.

appliance well, although a small metallic "tattoo" in the gingiva may result. A minimum of adjustment is required (Fig. 27-13). It is not necessary for the distal extension to be in direct contact with the permanent molar unless the tooth has already moved mesially. If the mesiodistal dimension of the second premolar has been duplicated in the appliance, the length of the loop will be correct. The depth of the intragingival extension should be about 1.0 to 1.5 mm below the mesial marginal ridge of the molar, or just sufficient to "capture" its mesial surface as the tooth erupts and moves forward. After the molar has erupted, the intragingival extension is removed. If the appliance is to be used as a reverse band and loop space maintainer, it may be necessary to add a supragingival extension to prevent the molar from tipping over the wire.

Brill describes the chairside-fabricated distal shoe appliance as an efficient and cost-effective appliance for guiding the unerupted permanent first molar into position after premature loss or extraction of the second primary molar (Fig. 27-14))[6] The data he presented indicate that success rates for this chairside-fabricated distal shoe are approximately equal to those in previous studies examining the longevity of other space maintainers.

There are several conditions that contraindicate the use of the distal shoe appliance. If several teeth are missing, abutments to support a cemented appliance may be absent. Poor oral hygiene or lack of patient and parental cooperation greatly reduces the possibility of a successful clinical result. Certain medical conditions, such as blood dyscrasias, immunosuppression, congenital



f **I G 27-11.** A sequence of three radiographs showing the untimely loss of the second primary molar and mesial movement of the first permanent molar before its eruption. Eventually there was complete closure of the space needed for the second premolar.

heart defects, history of rheumatic fever, diabetes, or generalized debilitation, almost without exception contraindicate the use of the distal shoe. The normal, healthy patient usually will have no problems with the appliance; the medically compromised patient will be placed at greater risk. Histologic studies show that the distal shoe implant never becomes totally lined with epithelium and is associated with a chronic inflammatory response.

In cases in which use of the distal shoe is contraindicated, two possibilities for treatment exist: (1) allow the tooth to erupt and regain space later or (2.) use a removable or fixed appliance that does not penetrate the tissue but places pressure on the ridge mesial to the unerupted permanent molar. Carroll and Jones have reported three cases in which a pressure appliance, removable or fixed, was used to guide the permanent molar as it erupted." If several teeth are missing, the removable appliance can also be designed to restore function and prevent supereruption of opposing teeth.

Space Maintenance for the Primary and Permanent Incisor Area

Some dentists believe that space closure rarely occurs in the anterior part of the mouth, but this is not true; each case must be critically evaluated. It is important to consider the occlusion and the degree of spacing, if any, between the anterior teeth. If the anterior primary teeth were in contact before the loss or there is evidence of an arch-length inadequacy in the anterior region, a collapse in the arch after the loss of one of the primary incisors is almost certain (Fig. 27-15).

Removable Partial Dentures. Even when spacing is present, it may be desirable to construct a partial denture or a fixed appliance to reproduce a desirable esthetic appearance, to reestablish function, or to prevent abnormal speech and tongue habits. Acrylic partial dentures have been used successfully to replace maxillary anterior primary teeth (Fig. 27-16). Appliances of this type can be constructed for young children who show a degree of cooperation and interest. It is unwise to place a removable partial denture, however, if an uncontrolled dental caries problem exists or if the child's mouth will not be kept clean enough to reduce the possibility of dental caries activity.

Fixed Appliances. If a fixed appliance is required, one approach is to attach the anterior replacement teeth to a 0.040- or 0.045-inch stainless steel wire framework retained with bands or crowns on the second primary molars. If the first primary molars are present, an indirect retainer may be placed on the occlusal area to prevent the wire from flexing. One can also obtain additional stabilization by using a Nance button or covering the ridge with dental acrylic resin.

The loss of anterior permanent teeth requires immediate treatment by the dentist if intraarch changes are to be prevented. Within a few days after the loss of a tooth as a result of trauma or the extraction of a severely traumatized tooth, the teeth adjacent to the space will begin to drift, and often within a few weeks several millimeters of space will be lost. Rather than allow the extraction area to heal and regain normal contour,

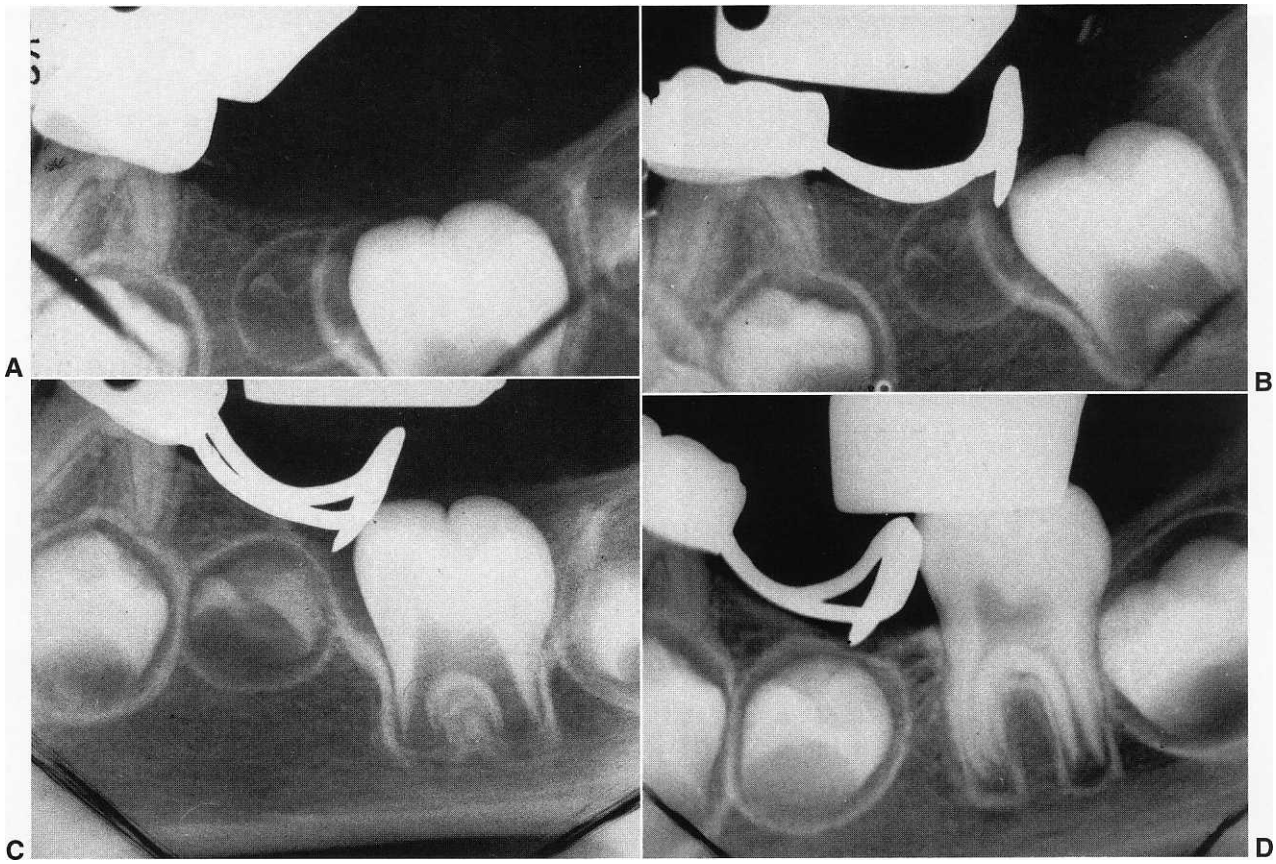


FIG 27-12. **A,** The second primary molar has been extracted. The first permanent molar has migrated mesially several millimeters after the extraction. **B,** Crown and band appliance with a distal shoe to guide an unerupted first permanent molar into its normal position. Notice the contour of the distal shoe to direct the first permanent molar distally and regain the lost space. C, Progress can be seen in the eruption of the permanent molar. D, The first permanent molar has erupted. The distal shoe tissue extension may now be removed. The appliance may be recemented as a reverse band and loop space maintainer, or a lingual arch appliance may be required.

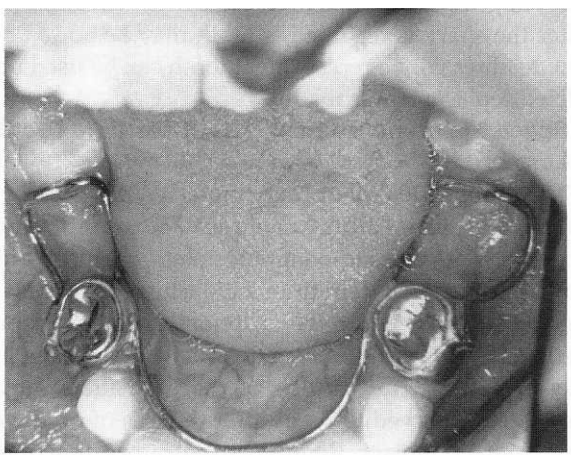


FIG 27-13. A modified Roche distal shoe appliance to provide bilateral space maintenance and eruption guidance for the first permanent molars. The permanent molars are erupting properly, and the intragingival extensions may be removed.

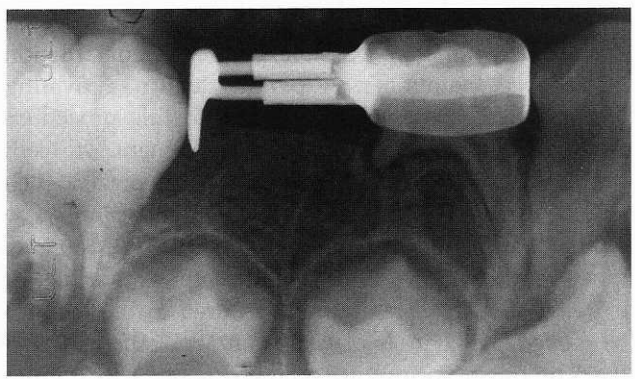


FIG 27-14. Radiograph of chairside-fabricated distal shoe space maintainer in place.

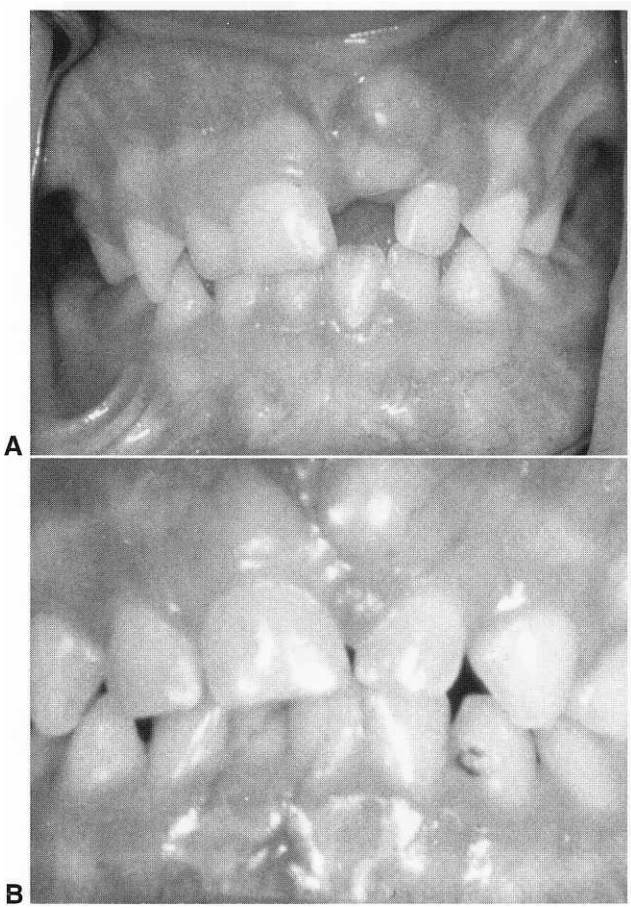


FIG 27-15. **A,** Dentition of child with arch-length inadequacy and some loss of space in the primary incisor area, which was observed at the time of the first examination. **B,** Space closure continued and was accompanied by drifting of teeth throughout the anterior area, including the canines.

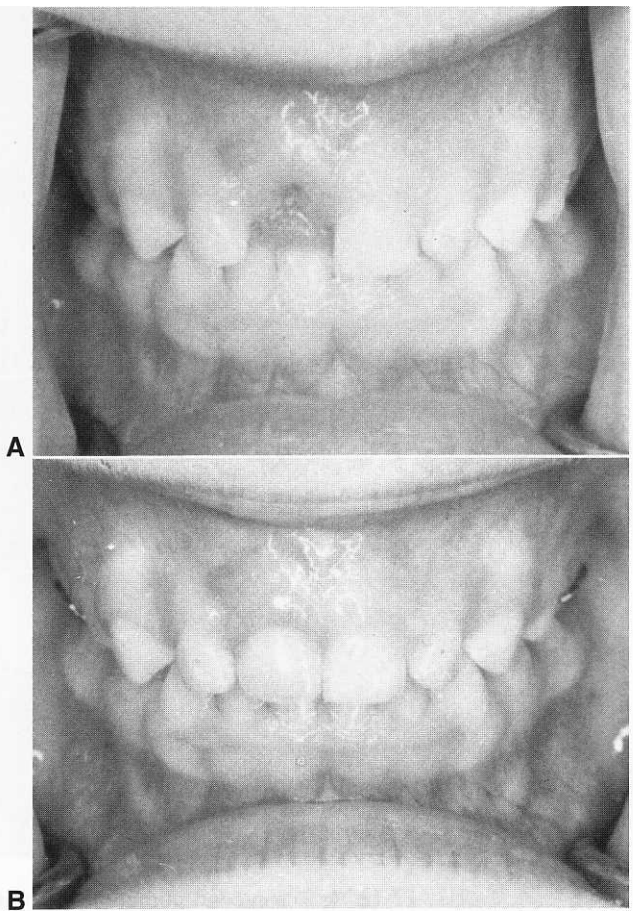


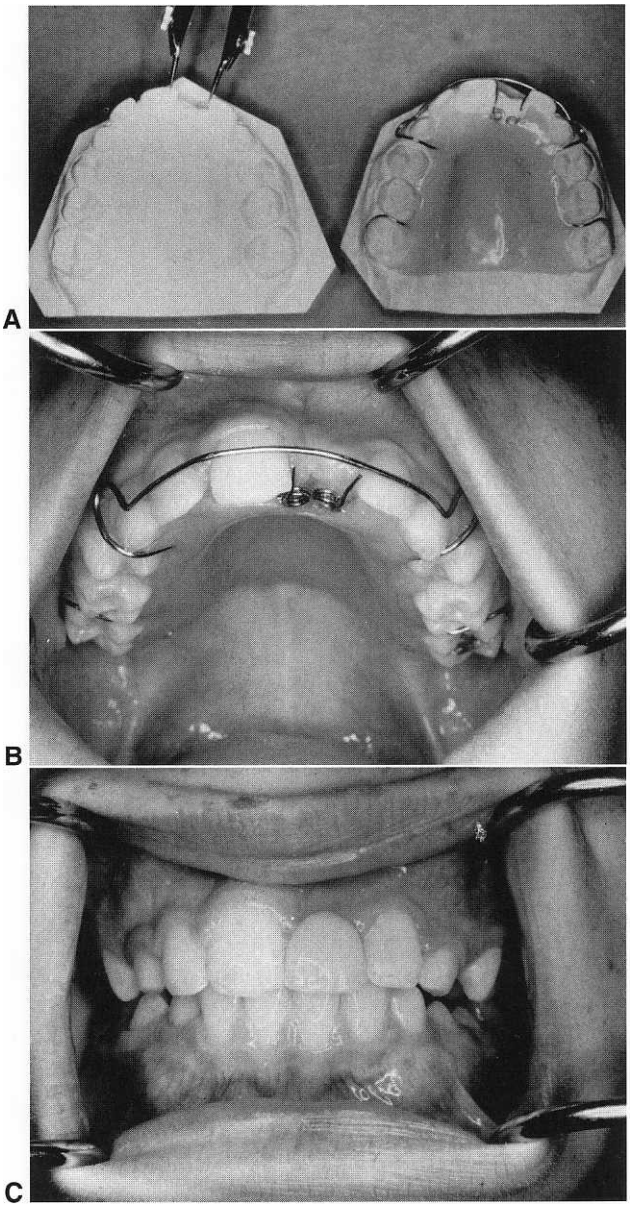
FIG. 27-16. **A,** This 3 1/2-year-old child has lost a primary incisor as the result of trauma. A space maintainer should be constructed to prevent space closure and to restore normal appearance. **B,** An acrylic palatal retainer has been constructed. The placement of 0.028-inch steel clasps around the second primary molars aids in retention of the appliance.

the dentist should take an impression at the time of the initial appointment or within a few days. The temporary appliance is constructed and inserted as soon as possible after the loss to prevent space closure.

If any degree of space closure has occurred, the space should be regained, if possible, before the construction of a space maintainer. A partial denture—activating appliance can be used successfully in this procedure if there is no need for bodily movement of teeth (Fig. 27-17). Contoured steel clasps of 0.028- or 0.030-inch steel wire are adapted to the first permanent molars to retain the appliance; Adams clasps may be used to gain more retention (Fig. 27-18). Helical finger springs of 0.018- to 0.022-inch wire are contoured to the teeth to be moved. The wire is placed as far cervically as possible. The finger springs are adjusted about 1 to 1.5 mm every 3 to 4 weeks. This procedure avoids excessive pressure on the teeth and allows their slow and regular movement. Neither the labial bow nor the palatal acrylic material should interfere with the teeth as they are being moved. Otherwise the space will not open. After the space is regained, a tooth may be added or a new retainer with a replacement tooth can be used until a fixed replacement is made.

Space Maintenance for Areas of Multiple Tooth Loss

The multiple loss of primary molars in the primary or mixed dentition will invariably lead to severe mutilation of the developing dentition unless an appliance is constructed to maintain the relationship of the remaining teeth and to guide the eruption of the developing teeth. Crossbite in the first permanent molar area and subsequent anterior drifting of the permanent molars have been observed to occur after the loss of the maxillary primary molars. Reduced masticatory function is undesirable from a nutritional standpoint. Also, the accumulation of plaque material and food debris after the loss of normal cleansing function will often result in increased dental caries activity and gingival inflammation.



F I 1 . 27-17. **A,** Drifting and tipping of the maxillary anterior teeth have taken place within 3 months after the loss of the central incisor. The space must be regained before eruption of the permanent canines. **B,** A palatal retainer with a Hawley labial wire to aid in retention and finger springs to reposition the drifted teeth. Activation of the wires 1.0 to 1.5 mm every 3 or 4 weeks resulted in desirable movement of the teeth. Repositioning of the teeth has been accomplished. A tooth can now be added to the appliance, or a new appliance can be constructed and used until a more permanent replacement can be inserted. C, A temporary replacement for the lost tooth.

Acrylic Partial **Denture.** The acrylic partial denture has been used successfully after the multiple loss of teeth in the mandible or the maxillary arch. This appliance, the use of which is indicated when there has been bilateral loss of more than a single tooth, can be readily adjusted to allow for the eruption of teeth. If artificial teeth are included on the denture, an essentially normal degree of function will be restored. However, the acrylic partial denture space maintainer is not without disadvantages. Parental and patient cooperation are imperative, and breakage of the appliance is a potential factor, since the pediatric patient may not exercise the necessary care.

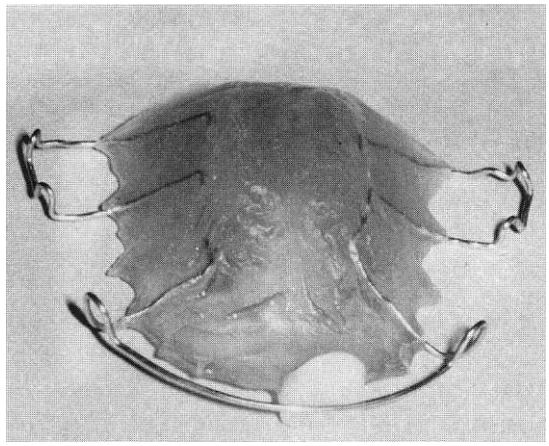
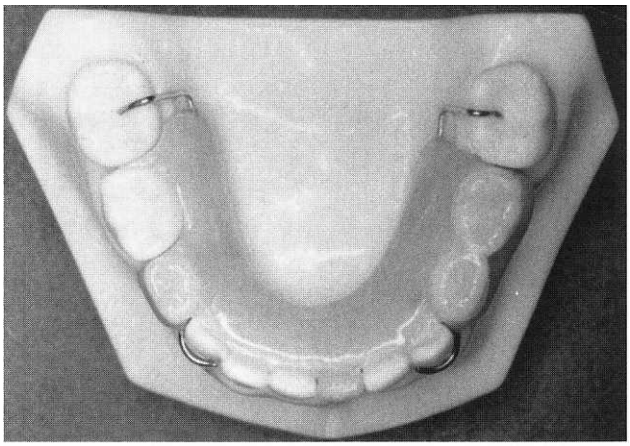


FIG **27-18.** Removable space-maintaining partial denture with Adams clasps on the first permanent molars.

A partial denture space maintainer of the contoured clasp type is acceptable from the standpoint of simplicity of construction, functional requirements, and cost to the patient. Retention is important particularly during the initial period of insertion. Stainless steel wire clasps are contoured for the primary canines, and 0.036-inch steel wire rests are contoured for the molars. If the permanent incisors are in an active state of eruption, it is desirable to remove the clasps after the child has become accustomed to wearing the appliance to allow the distal drifting and lateral movement of the primary canines and alignment of the permanent incisors. It is unlikely that there will be additional intercanine expansion in the older child. Therefore the fixation of the primary canines before the eruption of the permanent successors will have no adverse effect on the dental arch (Figs. 27-19 and 27-20).

If the loss of one or both of the second primary molars occurs a short time before the eruption of the first permanent molars, the acrylic removable appliance may be considered in preference to one of the distal shoe maintainers described previously. Immediate use of an acrylic partial denture with an acrylic distal shoe extension has been advocated by Starkey, and such a device has been employed successfully to guide first permanent molars into position (Fig. 27-21).[18] The tooth to be extracted is cut away from the stone cast, and a depression is cut into the stone model to allow the



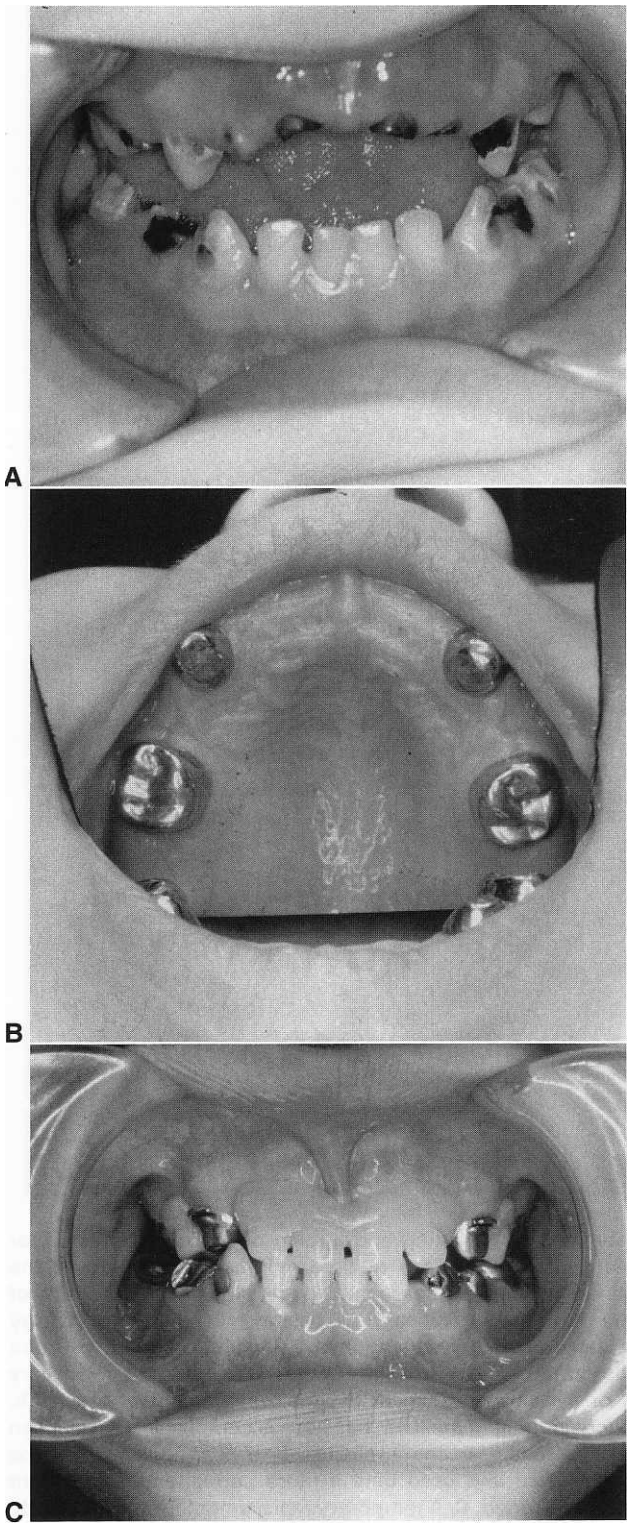
f l l; **27-19.** Acrylic partial-denture space maintainer to compensate for the untimely loss of three primary molars. The acrylic base material can be cut away to allow for the early eruption of one of the premolars before the maintainer is discarded. The acrylic may also be extended along the lingual tissues of the first permanent molars to incorporate retentive clasps for the molars if necessary.

fabrication of the acrylic extension. The acrylic will extend into the alveolus after removal of the primary tooth. The extension may be removed after the eruption of the permanent tooth. The contraindications for use of this appliance are the same as previously discussed with regard to the distal shoe appliance.

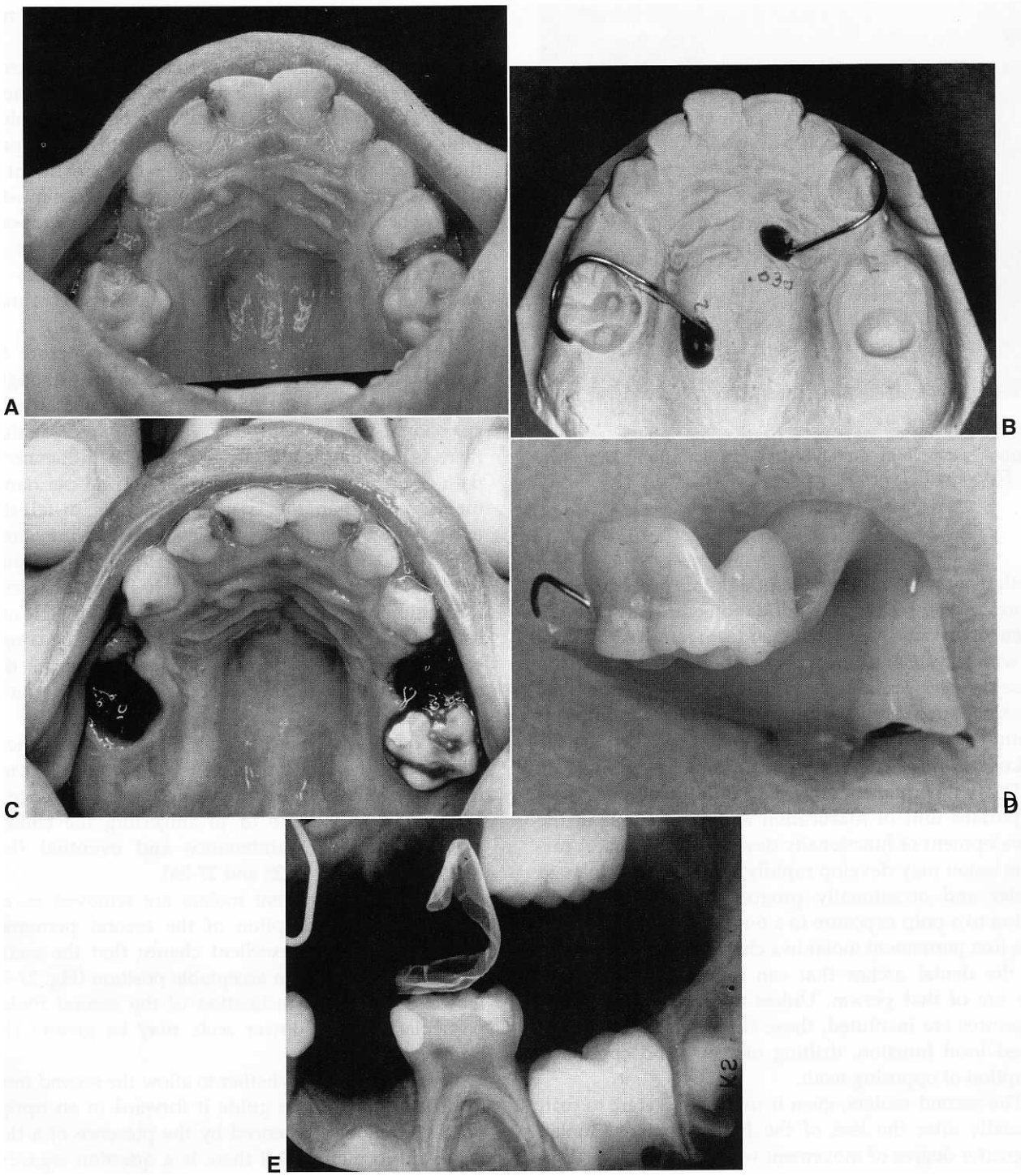
Passive Lingual Arch. The soldered lingual arch may be the space maintainer of choice after the multiple loss of primary teeth in the maxillary or in the mandibular arch (Fig. 27-22). Although it does not satisfy the requirements for restoring function, the appliance has many advantages that outweigh this fact. The use of the lingual arch essentially eliminates the problem of patient cooperation. With properly fitted bands and a well-made appliance, there should be no problems with breakage or retention and no concern about whether the child is wearing it.

On the working model a 0.036- or 0.040-inch steel wire is contoured to the arch, extending forward to make contact with the cingulum area of the incisors. In contouring the arch wire, one should allow for the path of eruption of the premolar and canines so that the arch wire will not interfere. Should this occur, the appliance will need to be remade or altered. Where possible, an ideal anterior arch form should be constructed so that the incisors have an opportunity for alignment. The arch wire should be extended posteriorly along the middle third of the lingual surface of the molar band and soldered firmly, but passively, in this position. A similarly designed lingual arch or one of the W-shaped kind can be used in the maxillary arch.

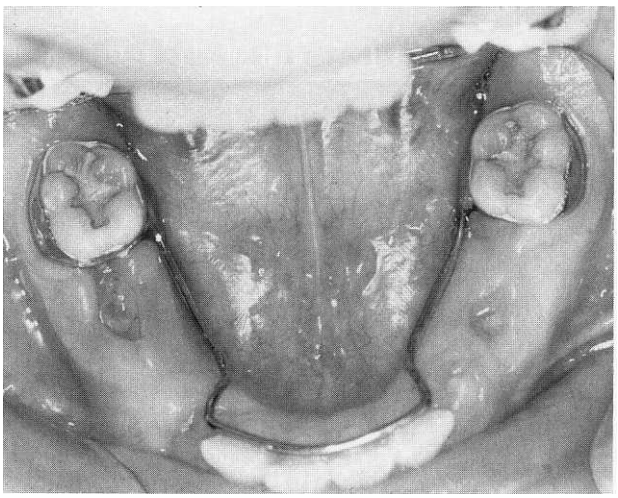
Full Dentures for Children. It is occasionally necessary to recommend the extraction of all the primary



[Ili. **27-20. A,** This child requires removal of the maxillary primary incisors and the first primary molars. **B,** Chrome steel crowns have been placed on the primary canines and second molars. C, A maxillary partial denture restores function, improves appearance, and reduces the possibility of a tongue-thrusting habit.



f **I G 27-21. A,** Clinical and radiographic examination revealed the need to extract the maxillary left first and second primary molars. The photograph is a mirror image. **B,** The teeth indicated for extraction are cut away from the stone model. A depression is made on the second molar area for the acrylic distal shoe extension. C, The primary teeth have been extracted in preparation for the placement of the partial denture. D, Acrylic distal shoe extension. **E,** Lead foil has been placed over the tissue extension to determine, with the aid of a radiograph, whether the acrylic is positioned properly to guide the eruption of the first permanent molar. (Courtesy Dr. Paul E. Starkey.)



f **G 27-22.** Mirror view of a passive mandibular soldered lingual arch immediately after cementation. (Courtesy Dr. Theodore R. Lynch.)

teeth of a preschool child. Although this procedure was more common in the prefluoridation era, some children even today must have all of their teeth removed because of widespread oral infection and because the teeth are unrestorable. Preschool children can wear complete dentures successfully before the eruption of permanent teeth (Fig. 27-23).

Loss of the First Permanent Molar

The first permanent molar is unquestionably the most important unit of mastication and is essential in the development of functionally desirable occlusion. A carious lesion may develop rapidly in the first permanent molar and occasionally progress from an incipient lesion to a pulp exposure in a 6-month period. The loss of a first permanent molar in a child can lead to changes in the dental arches that can be traced throughout the life of that person. Unless appropriate corrective measures are instituted, these changes include diminished local function, drifting of teeth, and continued eruption of opposing teeth.

The second molars, even if unerupted, start to drift mesially after the loss of the first permanent molar. A greater degree of movement will occur in children in the 8- to 10-year age group; in older children, if the loss occurs after the eruption of the second permanent molar, only tipping of this tooth can be expected. Although the premolars will undergo the greatest amount of distal drifting, all the teeth anterior to the space, including the central and lateral incisors on the side where the loss occurred, may show evidence of movement. Contacts will open and the premolars, in particular, will rotate as they fall distally (Fig. 27-24). There is a tendency for the maxillary premolars to move distally in unison, whereas those in the lower arch may move separately.

When the maxillary first permanent molar loses its opponent, it will erupt at a faster rate than the adjacent teeth. The alveolar process will also be carried along with the molars and will cause problems when prosthetic replacements are needed. The treatment of patients with the loss of first permanent molars must be approached on an individual basis. A superimposed existing malocclusion, abnormal musculature, or the presence of deleterious oral habits can affect the result, as in the case of the premature loss of primary molars.

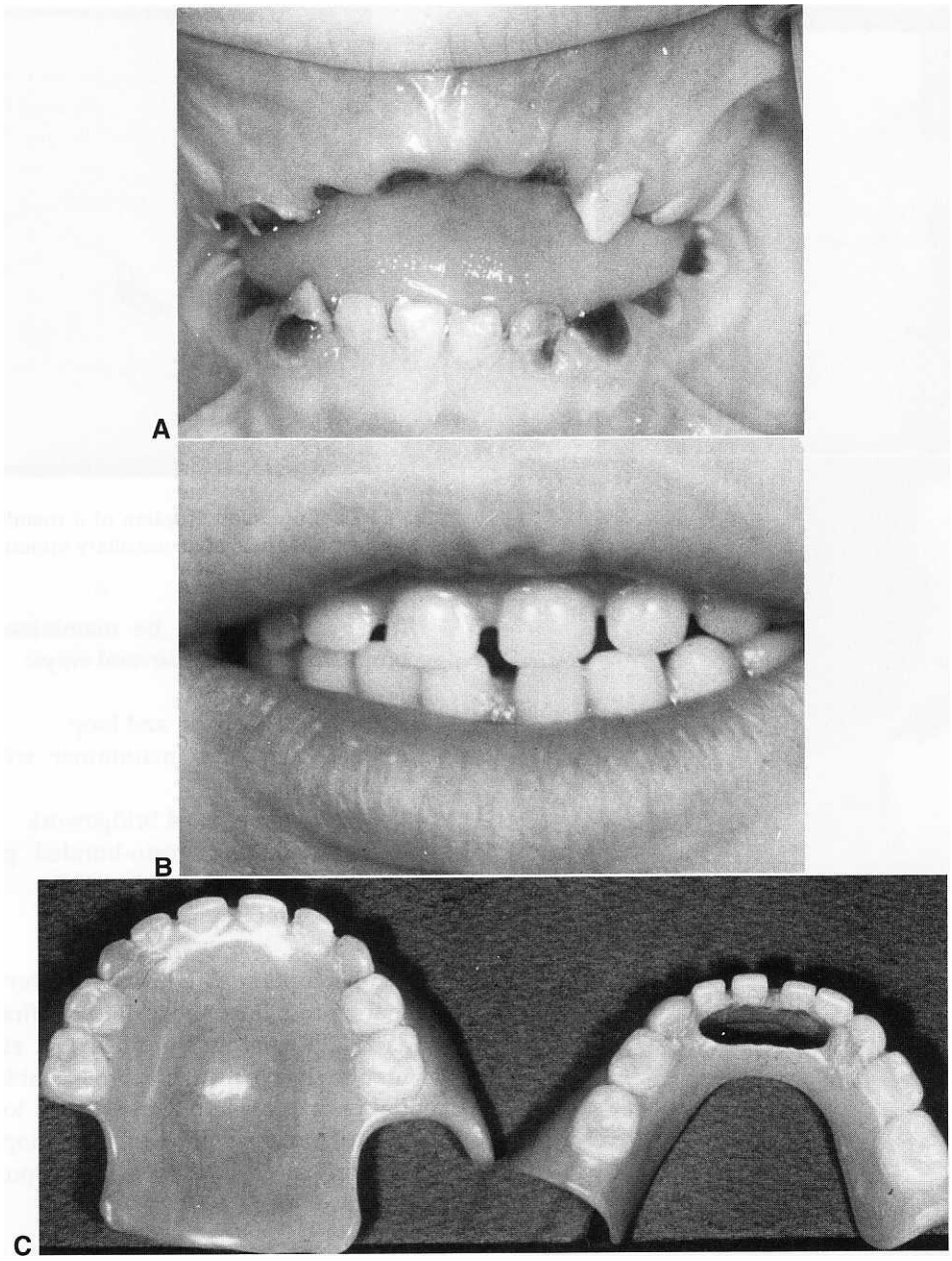
Loss of the First Permanent Molar Before the Eruption of the Second Permanent Molar. Although it is possible to prevent overeruption of a maxillary first permanent molar by placing a lower partial denture, there is no completely effective way to influence the path of eruption of the developing second permanent molar other than the use of an acrylic distal shoe extension on a partial denture as described previously. The second molar will drift mesially before eruption when the first permanent molar has been extracted. Repositioning this tooth orthodontically is possible after its eruption. However, the child must then be considered for prolonged space maintenance until the time when a more permanent tooth replacement can be inserted.

The removal of the opposing first permanent molar, even when the tooth appears to be sound and caries free, is sometimes recommended in preference to allowing it to extrude or to subjecting the child to prolonged space maintenance and eventual fixed replacement (Figs. 27-25 and 27-26).

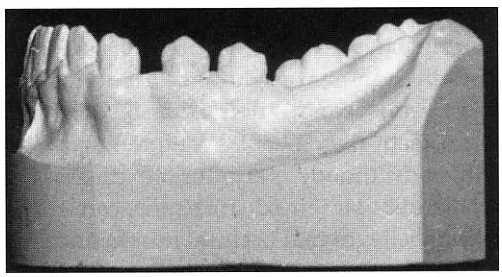
If the first permanent molars are removed several years before the eruption of the second permanent molars, there is an excellent chance that the second molars will erupt in an acceptable position (Fig. 27-27). However, the axial inclination of the second molars, particularly in the lower arch, may be greater than normal.

The decision as to whether to allow the second molar to drift mesially or to guide it forward in an upright position may be influenced by the presence of a third molar of normal size. If there is a question regarding the favorable development of a third molar on the affected side, repositioning the drifted second molar and holding space for a replacement prosthesis is usually the treatment of choice.

Loss of the First Permanent Molar After the Eruption of the Second Permanent Molar. When the first permanent molar is lost after the eruption of the second permanent molar, orthodontic evaluation is indicated, and the following points should be considered: Is the child in need of corrective treatment other

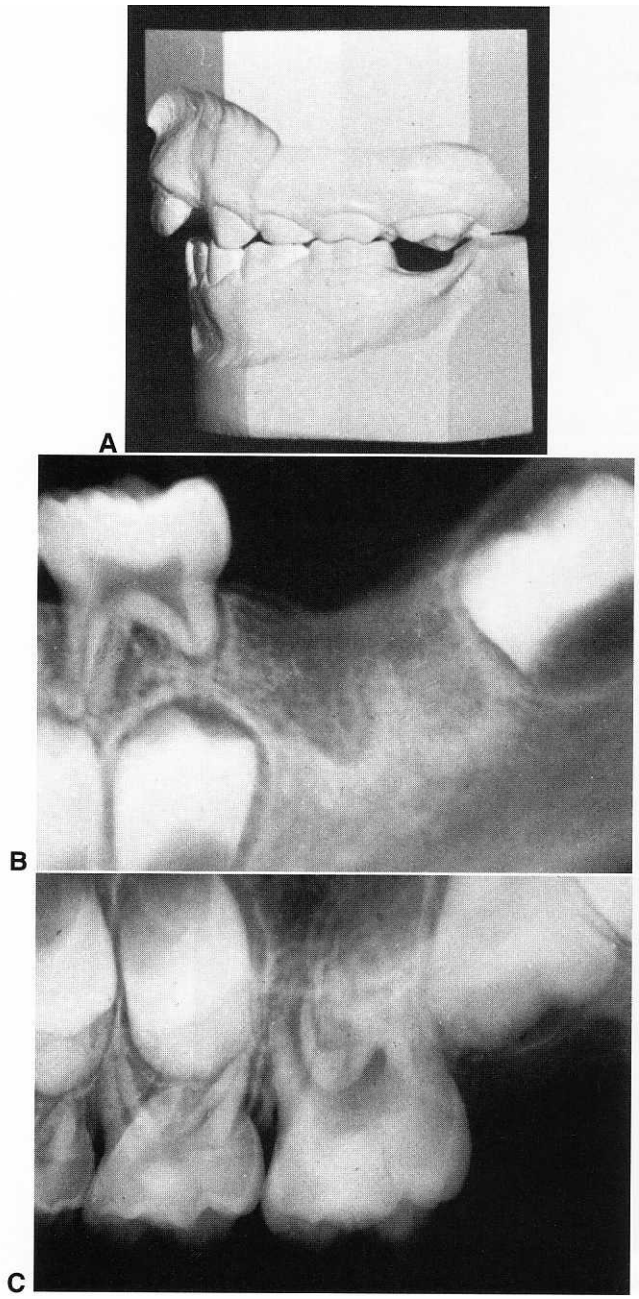


f I G 27-23. A, Primary teeth have gross caries and pulpal involvement. **B,** Complete dentures were constructed after the extraction of all primary teeth. C, The dentures were modified after the eruption of the maxillary first permanent molars and the mandibular permanent incisors.

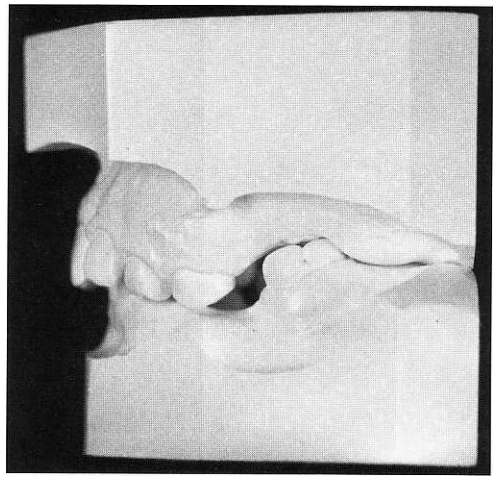


f **I G 27-24.** Lower cast showing distal drifting of the premolars and mesial tipping of the second and third molars after the extraction of the first permanent molar.

than in the first permanent molar area? Should the space be maintained for a replacement prosthesis? Should the second molar be moved forward into the area formerly occupied by the first molar? The latter choice is often the more satisfactory, even though there will be a difference in the number of molars in the opposing arch. A third molar can often be removed to compensate for the difference. Without treatment the second molar will tip forward within a matter of weeks (Fig. 27-28).



f I G. **27-25. A,** Models of the dentition of an 8-year-old child who had a mandibular first permanent molar extracted. It would be virtually impossible to guide the second permanent molar into its normal position and at the same time prevent overeruption of the opposing first permanent molar. Therefore extraction of the opposing molar is indicated. **B** and C, Position of the second permanent molars. If first permanent molars are removed at this age, reasonably good positioning of the second molars can be expected.



f G **27-26.** Continued eruption of a mandibular first permanent molar after loss of its maxillary opponent.

If the space should be maintained, this may be accomplished in one of several ways:

1. Cast overlay band and loop
2. Band and loop maintainer with occlusal bar and rest
3. Conventional fixed bridgework
4. Etched-casting, resin-bonded posterior bridge (modified fixed bridgework)
5. Single-unit implant prosthesis

Another option to consider, however, is autotransplantation of a third molar into the first molar position (Fig. 27-29). According to Bauss et al, autotransplantation has become a well-established treatment modality in cases of early tooth loss or aplasia.[19] For third molars with partly developed roots, transplantation success rates have been reported in the range of 74% to 100%.

CAUSE AND CORRECTION OF DELETERIOUS ORAL HABITS

Bruxism. Bruxism is a nonfunctional grinding or gnashing of teeth. Reding, Rubright, and Zimmerman report that 15% of the children and young adults in their study group showed some degree of bruxism.[20] The habit usually occurs at night and, if continued over a prolonged period, can result in abrasion of primary and permanent teeth (Fig. 27-30). When the habit is continued into adulthood, periodontal disease and/or temporomandibular joint disturbances can result.

Ramfjord believes that occlusal interference may trigger bruxism, particularly if it is combined with nervous tension.[21] Therefore occlusal adjustment should be the first approach to the problem if interferences are present. Sheppard recommends the construction of a palatal bite



f **I G 27-27.** Radiographs taken at 6-month intervals after a maxillary first permanent molar was lost before the eruption of the second permanent molar.



f **I G 27-28.** Radiographs taken at 6-month intervals after the loss of a mandibular first permanent molar. Notice the degree of tipping of the second molar and distal drifting of the premolars.

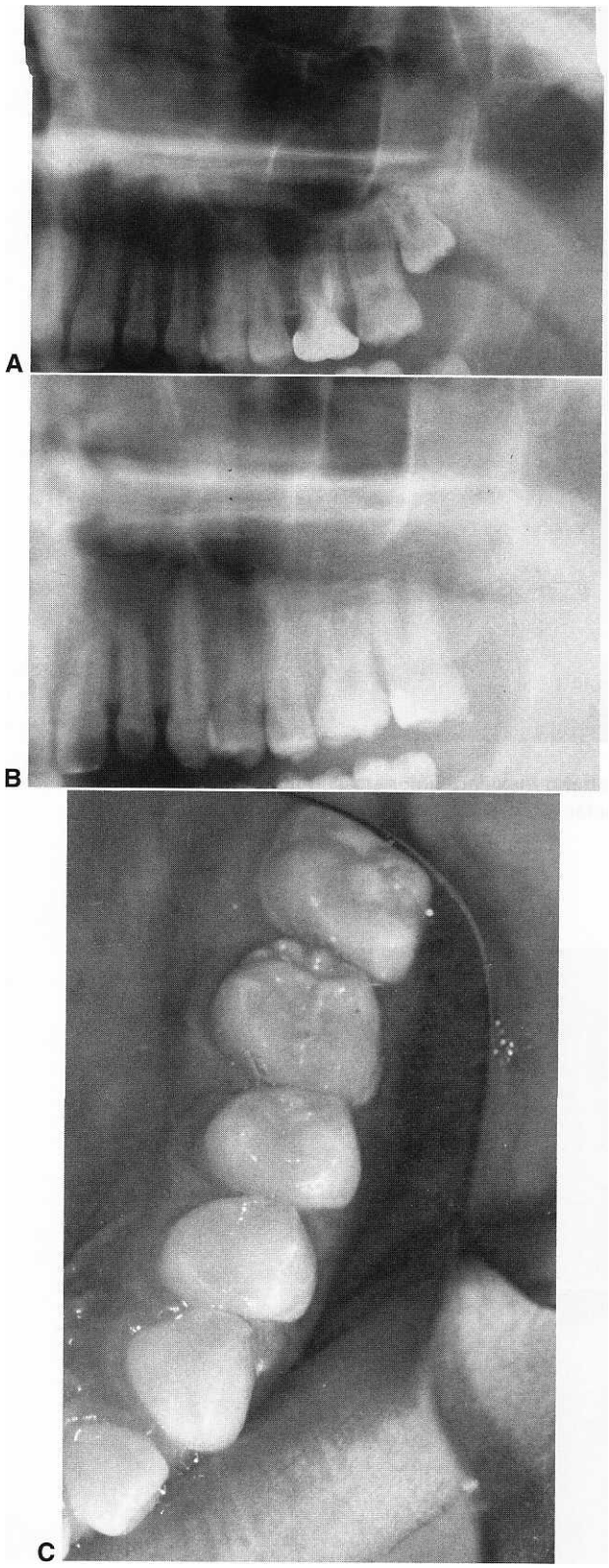


FIG . **27-29.** Third molar autotransplantation. **A,** Radiograph showing failed attempt at maintaining first permanent molar and minimal root development of third permanent molar. **B,** Radiographic and C, clinical view of third molar 15 months after autotransplantation. Note continued root development.

plate, which allows the continued eruption of the posterior teeth." This eruption is desirable if the teeth have ' been abraded by the habit. A vinyl plastic bite guard that covers the occlusal surfaces of all teeth plus 2 mm of the buccal and lingual surfaces can be worn at night to prevent continuing abrasion. The occlusal surface of the bite guard should be flat to avoid occlusal interference. A mouth guard of the type described in Chapter 22 may also help in overcoming the habit.

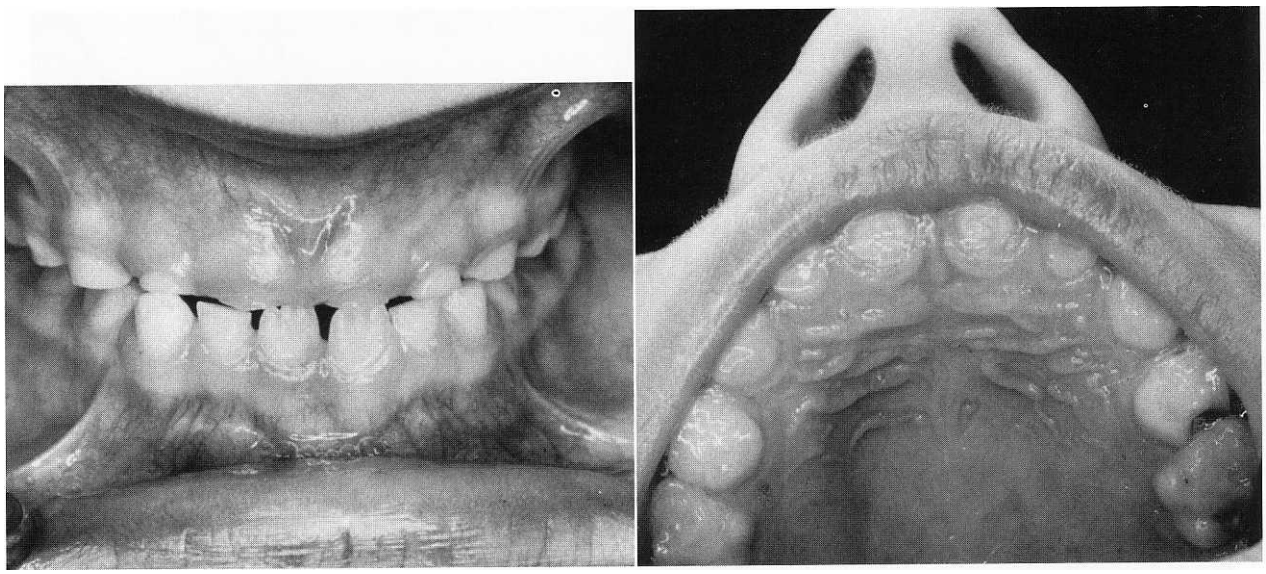
Digit and Nonnutritive Sucking Behaviors. Various and conflicting theories explain the cause of thumb sucking in children, and there are equally conflicting recommendations for its correction. Many children suck their thumbs or fingers for short periods during infancy or early childhood. Although the habit may be considered normal during the first 2 years of life, many children never develop a digit-sucking habit. Parents should be advised to observe the habit periodically. If it gradually diminishes, the child probably will stop the habit. On the other hand, if the habit persists or increases in frequency and adverse dental and skeletal changes are noted, specific corrective measures may be needed to avoid undesirable occlusion problems (Fig. 27-31).

Zardetto et al point out that more occlusal and oral-myofunctional alterations are detected among children who have pacifier habits (either conventional or physiologic pacifiers) than among those with no sucking habits.' Children who were pacifier users were significantly more likely to show open bite, posterior crossbite, increased overjet, and alteration in cheek mobility than habit-free children.

Many children stop the habit during the preschool years, but some continue into the teenage or adult years. Even if there were no ill effects on occlusion, thumb sucking is not socially acceptable; it should be discouraged when the habit is persistent and when the patient is mentally capable of understanding why it should be stopped.

Salzmann believes that the effect of the sucking habit on the maxillary and mandibular bones and on the dental arches, including the occlusion of the teeth, depends on several factors. These include the frequency with which the habit is practiced, the duration of the habit, the osteogenic development, the genetic endowment, and the child's state of health.'-'

Popovich and Thompson observed 1258 children at the Burlington Growth Centre; the group represented approximately 90% of the pediatric population of Burlington, Ontario." Many of the children were seen annually from 3 to 12 years of age, and their oral habits and occlusion status were recorded at 3, 6, 9, and 12 years of age. There was a significant association between the prevalence of class II malocclusion and persisting digit sucking in the different age groups. Class II



[I fi **27-30.** Bruxism has resulted in severe abrasion of the maxillary primary anterior teeth.

malocclusion increased from 21.5% at 3 and 4 years of age to 41.9% at 12 years of age. As the duration of the habit increased, the probability of a child's developing a class II malocclusion increased. If the habit was stopped early (before 6 years), the effects on occlusion were often transitory. In the serial sample, no child who stopped a habit after 6 years of age had a normal occlusion at 12 years of age. Another interesting observation was that children who had used a pacifier had a significantly lower rate of thumb and finger sucking.

The use of a corrective appliance to manage oral habits is indicated only when the child wants to discontinue the habit and needs only a reminder to accomplish the task. If an appliance is used, it should not be painful or interfere with occlusion; instead, it should merely act as a reminder. A removable partial retainer with a series of smooth loops placed lingual to the incisors has proved successful in helping the child overcome the habit (Fig. 27-32).

Haskell and Mink have used the Bluegrass appliance with a program of positive reinforcement to stop thumb sucking in children.[26] A modified, six-sided roller machined from Teflon* (Fig. 27-33), which permits purchase of the tongue, is constructed to skip over a 0. 045-inch stainless steel wire that is soldered to molar orthodontic bands previously fitted and in place on a poured plaster model.

An entirely different approach has been practiced by some dentists when it is evident that a child wants to discontinue the habit (Fig. 27-34). This approach requires cooperation of the parents and their consent to disregard the habit and not mention it to the child.

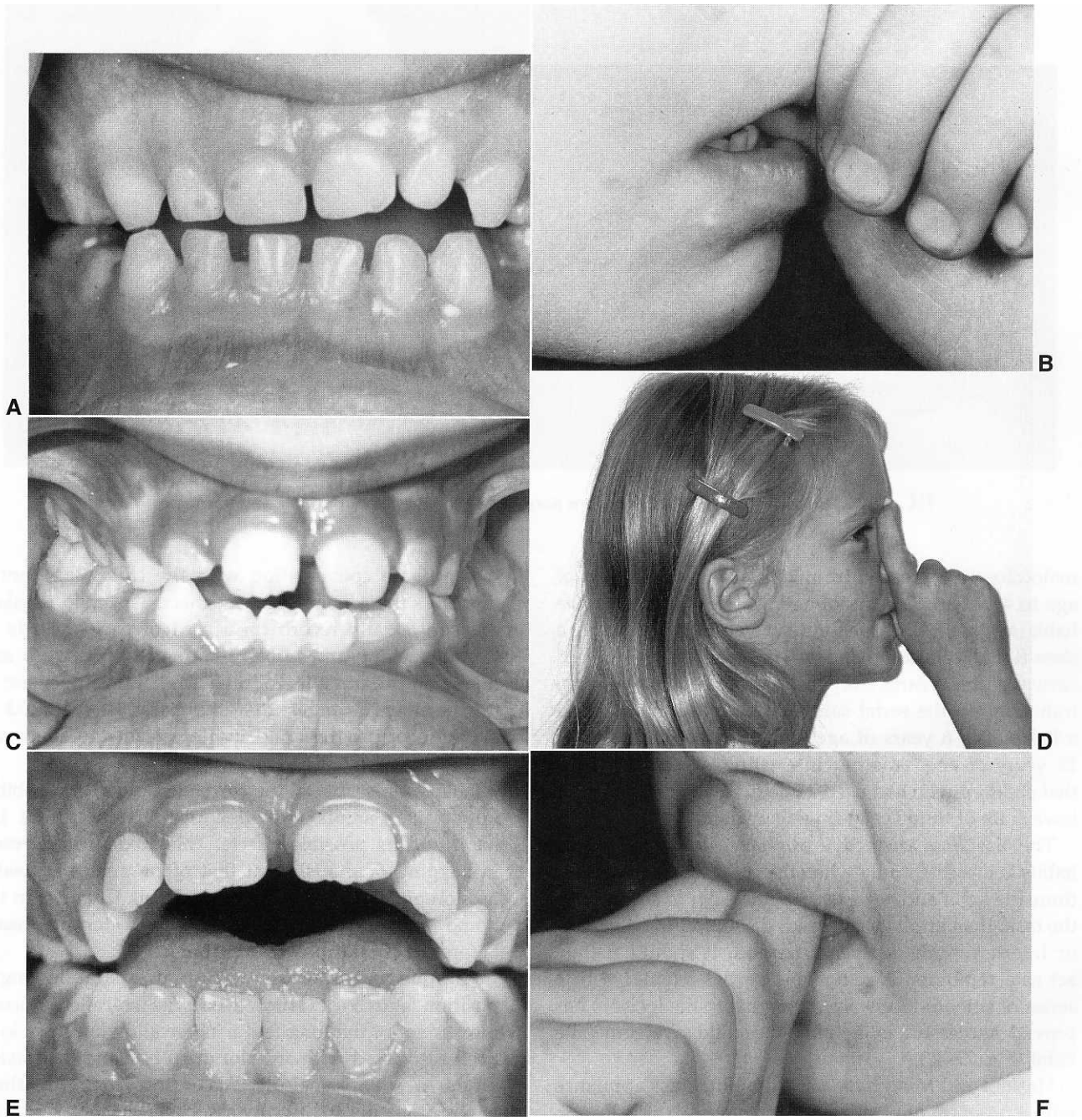
*E.I. du Pont de Nemours and Co., Wilmington, Del.

In a private conversation with the child, the dentist discusses the problem and its effect. The child is asked to keep a daily record on a card of each episode of thumb sucking and to call the dentist each week and report on progress in stopping the habit. A decrease in the number of times that the habit is practiced is evidence of progress and indicates that the child will discontinue the habit.

The parents' role in the correction of an oral habit is important. Parents are often overanxious about the habit and its possible effects. This anxiety may result in nagging or punishment that often creates a greater tension and intensification of the habit. Changes in the home environment and routine are often necessary before the child can overcome the habit.

Tongue and Swallowing Habits. Abnormal tongue position and a deviation from the so-called normal movement of the tongue during swallowing have long been associated with anterior open bite and protrusion of the maxillary incisors. According to Proffit, three major problems are usually associated with the anterior tongue position, which is also called *tongue thrust, deviate swallow, visceral swallow,* or *infantile swallow."* These problems are open bite; protrusion of the incisors, particularly the maxillary incisors; and lisping.

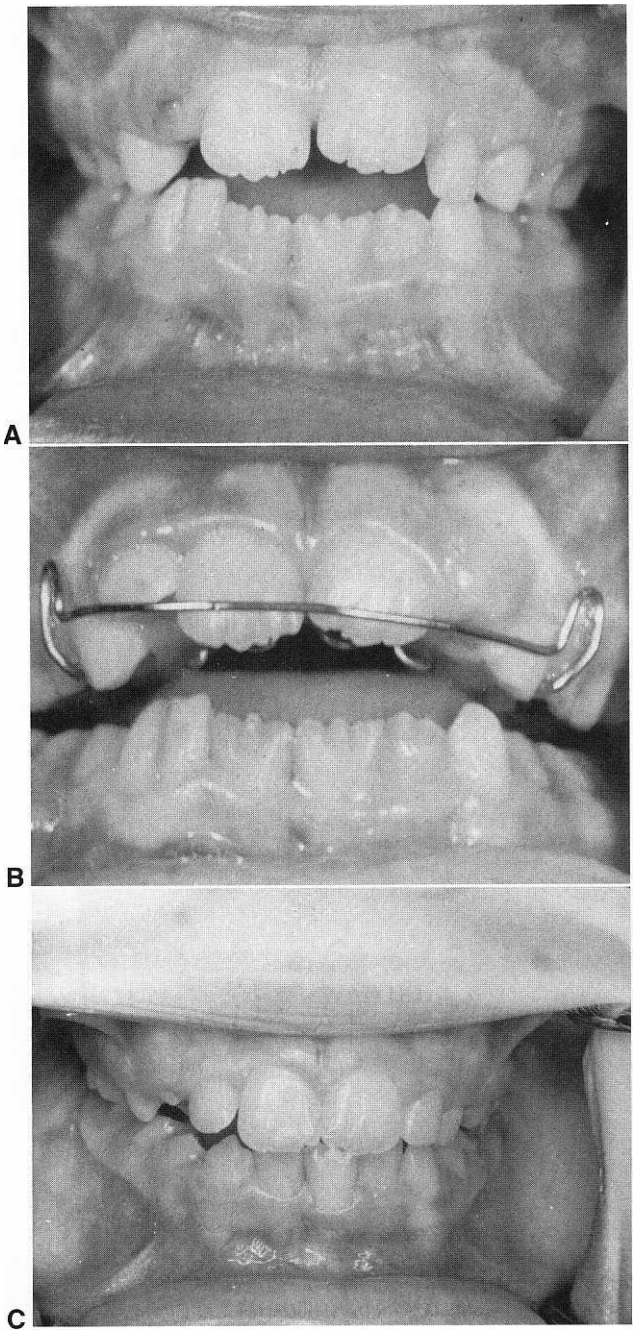
Because there is a relatively high prevalence of anterior tongue positioning in children, Proffit believes that this condition is often improperly labeled as *tongue thrust* or *deviate swallow.* He also believes that the two major reasons for it relate to the physiology of the child (maturation) and to anatomy (growth). Normal infants position the tongue anteriorly in the mouth at rest and during swallowing. The first physiologic priority at birth is establishment of an airway so that respiration



f **G 27-31.** Occlusion of three children with thumb-sucking and finger-sucking habits. **A,** An open bite in the primary dentition caused by the child's placing the thumb between the anterior teeth. **B,** A photograph showing how the thumb was held between the teeth. The child appar ently did not suck the thumb. C, An open bite and flared maxillary incisors caused by a thumb-sucking habit. D, During sucking, the index finger was placed alongside the nose. Sucking was practiced for long periods. **E,** An open bite extending into the second primary molar, a result of the child's sucking the thumb and index finger. **F,** An illustration of how the habit was practiced.

can begin. Accomplishment of this priority requires holding the tongue forward and down. The set of postural relationships of the oral and pharyngeal structures established in the first few minutes of life in response to respiratory requirements is maintained thereafter.

An infant's normal swallow is characterized by strong lip activity to seize the nipple, placement of the tongue tip against the lower lip beneath the nipple, and relaxation of the elevator muscles of the mandible so that the mouth is wide open. As oral function matures,



f G 27-32. **A,** Anterior open bite caused by a severe thumb-sucking habit. **B,** A passive type of reminder appliance was constructed after other problems in the patient's life were recognized and treated. C, The occlusion 18 months after the child had overcome the habit.

there is a gradual activation of the elevator muscles of the mandible so that the mandible is brought up toward what ultimately will be occlusal contact of the teeth. This act occurs while the tongue tip is still placed against the lower lip. Physiologic transition in swallowing begins during the first *year* of life and normally continues over the next several years. A mature swallow pattern is characterized by relaxation of the lips, placement of the tongue behind the maxillary incisors, and elevation of the mandible until posterior teeth are in contact. This pattern is not usually observed before a child is 4 or 5 years of age. Children in the normal transitional stages of swallow maturation are often considered for myofunctional therapy for tongue thrusting or deviate swallow, but this practice should be seriously questioned. Some preschool children with a forward tongue position during swallow most likely have not yet learned to follow the adult pattern.

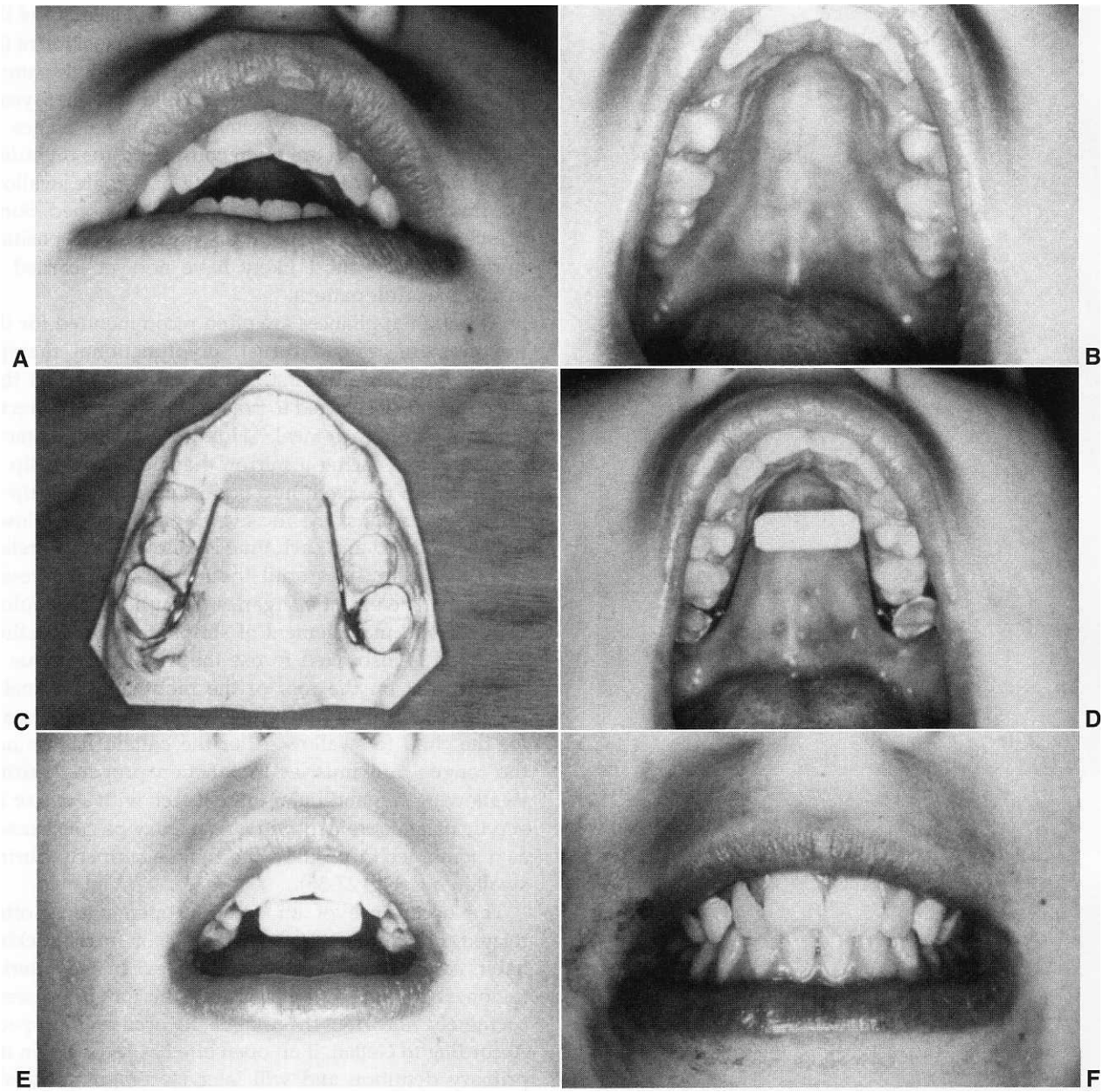
Although appliances are often recommended for the treatment of tongue thrust, myofunctional therapy should be attempted first. Andrews recommends that the patient be instructed to practice swallowing correctly 20 times before each meal.[1-8] Holding a glass of water in one hand and facing a mirror, the child takes a sip of water, closes the teeth into occlusion, places the tip of the tongue against the incisive papilla, and swallows. This is repeated and each time is followed by the relaxation of the muscles until the swallowing progresses smoothly. The use of a sugarless mint has also resulted in successful management of simple tongue thrusting. The child is instructed to use the tip of the tongue to hold a mint on the roof of the mouth until it melts. As the mint is held, saliva flows and makes it necessary for the child to swallow. After the patient has trained the tongue and muscles to function properly during swallowing, a mandibular lingual arch with a crib or an acrylic palatal retainer with a fence may be constructed as a reminder to position the tongue properly during swallowing (Fig. 27-35).

The occurrence of an anterior open bite is often related, initially at least, to a thumb- or finger-sucking habit. After the space has been created in the anterior region, it is retained by thrusting the tongue forward or merely allowing the tongue to occupy the space. According to Gellen, if an open bite has its origin in the primary dentition and will later close spontaneously, the initial closure begins by 10 years of age in 90%, of children." Thus the dentist may be justified in waiting until the child's tenth birthday before taking active steps to correct an anterior open bite.

INTERCEPTIVE ORTHODONTICS

ANTERIOR CROSSBITE IN PRIMARY AND MIXED DENTITION

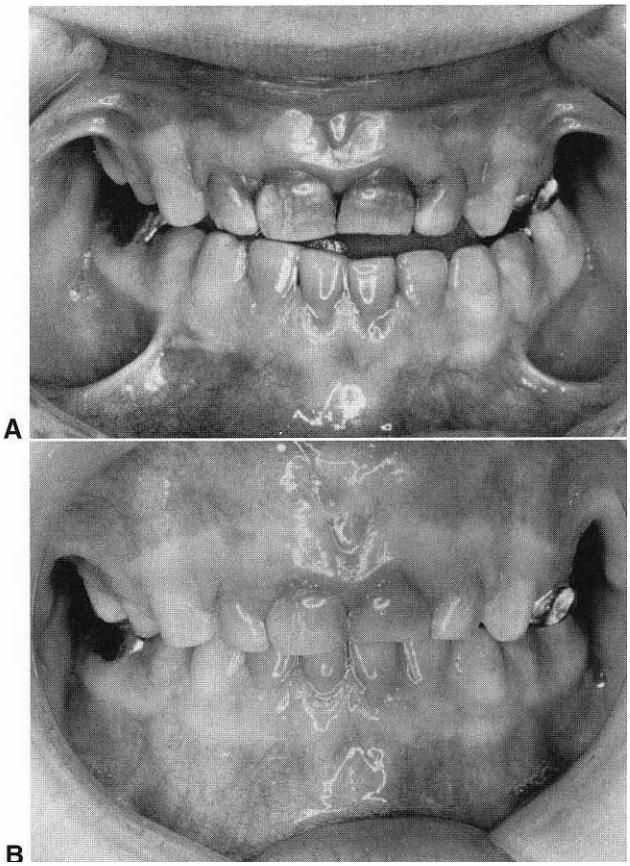
Complete anterior crossbite may indicate a skeletal growth problem and developing class III malocclusion. Anterior crossbite of one or more of the permanent incisors, however, may be evidence of a localized discrepancy, which in most cases should be treated as soon as it is discovered. Delayed treatment can lead to serious complications such as loss of arch length.



[I G 27-33. **A,** Anterior open bite in the mixed dentition because of thumb sucking. **B,** Palatal configuration of the patient. C, Appliance in place on a plaster cast. D, Occlusal view of the appliance cemented in place. **E,** Anterior view of the appliance in place. **F,** Correction of the anterior open bite. *(Courtesy Dr. John R. Mink. A,* **B, D, E,** *and* **F** *from Haskell BF Mink JR:* Pediatr Dent 13:83-85, *7997.)*

Traumatic occlusion with resultant stripping of the gingival tissue and pocket formation on the labial aspect of the lower tooth is a common result (Fig. 27-36). Unsightly wear facets may also develop on the incisal and labial surfaces of the involved maxillary incisors. Anterior crossbite is the result of a variety of conditions, including the following:

1. A labially positioned supernumerary tooth may cause torsiversion and lingual deflection of an incisor, which may erupt in a rotated or crossbite relationship.
2. Trauma to an anterior primary tooth may cause displacement of the developing permanent successor and eruption in crossbite. If a primary incisor is delayed in its exfoliation because of a necrotic pulp resulting from trauma or caries, the tooth may act as a foreign body and cause deflection of permanent teeth in the area (Fig. 27-37). Pulpless primary teeth often do not undergo



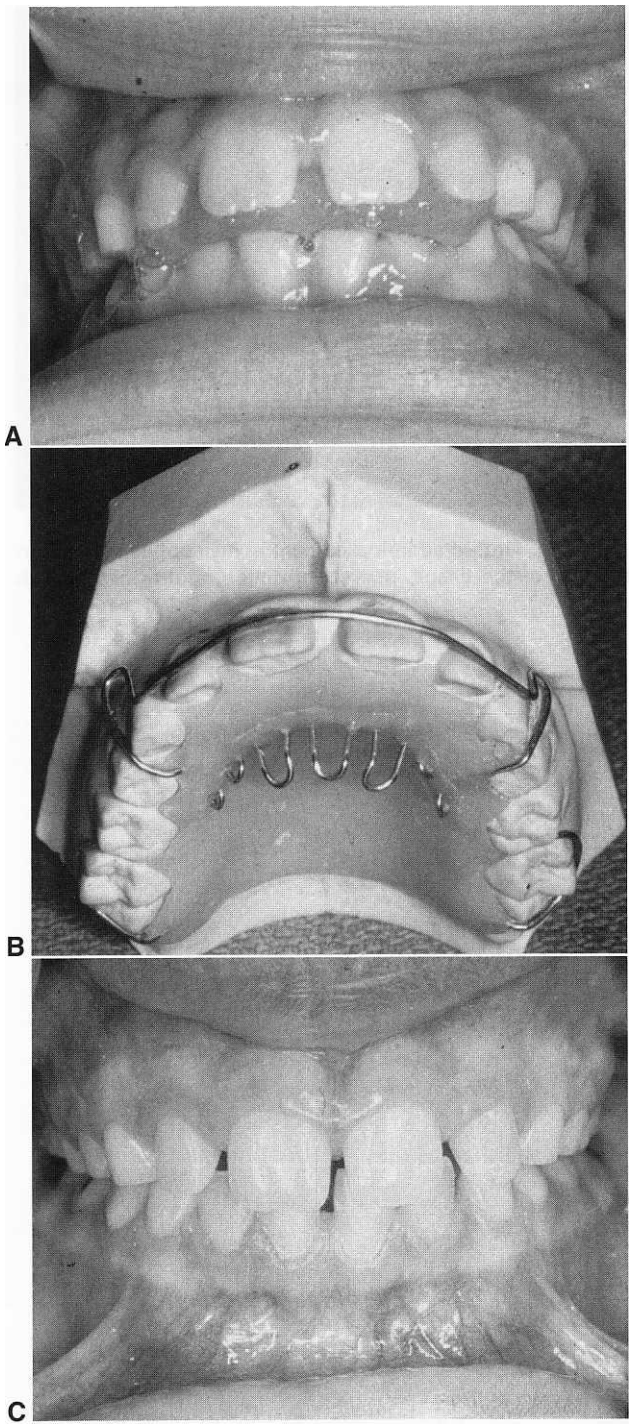
IIG **27-34. A,** Open bite is evident in the primary dentition resulting from a thumb-sucking habit. **B,** The child was encouraged by the dentist to discontinue the habit. There was self-correction of the open bite when the habit was discontinued.

normal root resorption and can cause serious complications in the developing occlusion.

3. An arch-length deficiency can cause a lingual deflection of permanent anterior teeth during eruption, which is often observed in the maxillary lateral incisor area. The premature eruption of permanent canines in instances of arch-length deficiency can cause a lateral incisor to be squeezed lingually and to erupt in crossbite.

If the following conditions are present, the problem may be considered one in which uncomplicated treatment may be undertaken:

1. Sufficient room exists mesiodistally to move the tooth into its correction position.
2. The apical portion of the in-locked tooth is in relatively the same position as a tooth in normal occlusion.
3. The patient has normal occlusion in the molar and canine areas.



[I G **27-35. A,** Anterior open bite resulting from a tongue-thrust habit. **B,** A removable palatal retainer was constructed to prevent the tongue from being thrust forward during the swallowing process. C, The tongue-thrusting habit has been overcome. The occlusion is greatly improved.



FIG 27-36. Untreated in-locked central incisor has resulted in stripping of the tissue, pocket formation, and a loss in arch length.

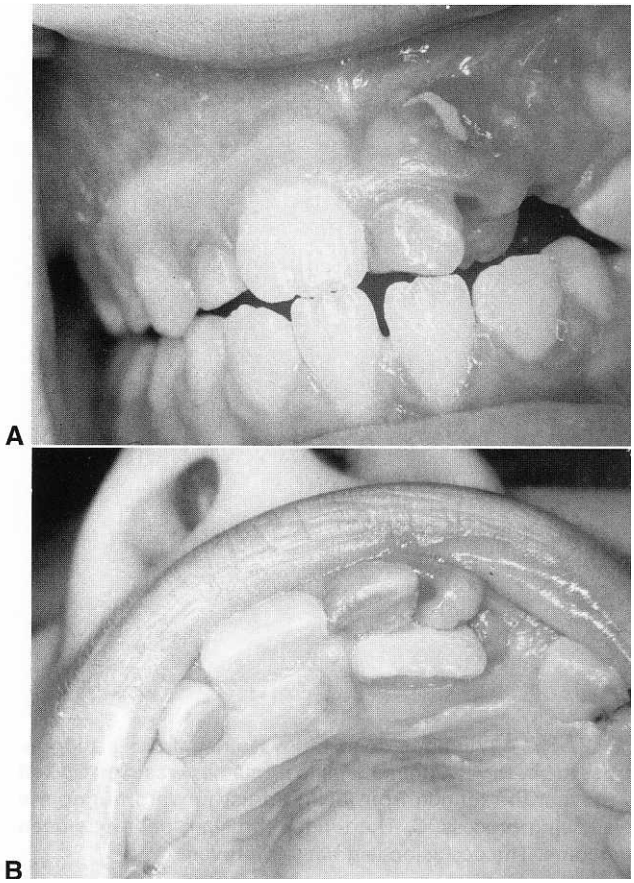


FIG 27-37. **A,** Prolonged retention of a pulpless primary incisor has resulted in lingual eruption and in-locking of permanent tooth. Notice exposed and unresorbed apex of necrotic primary incisor that has perforated the gingival tissues. **B,** Incisal view.

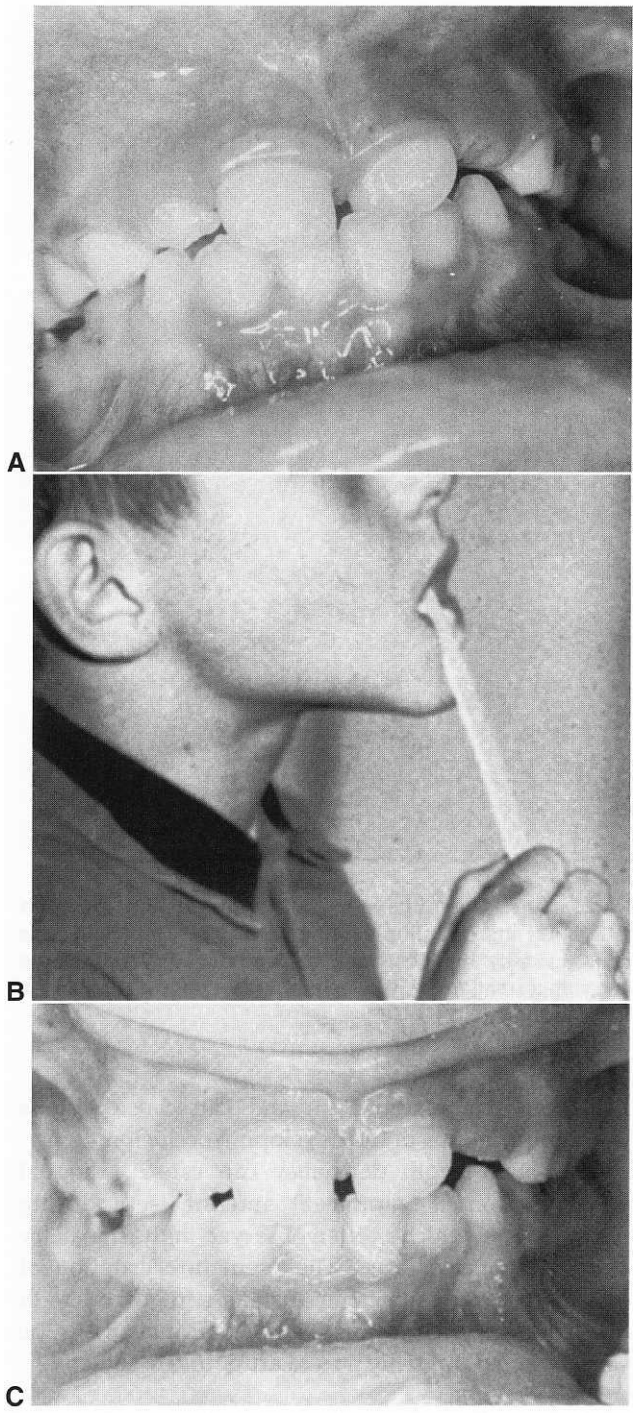


FIG 27-38. **A,** Partially erupted central incisor with a minimum degree of in-locking. A tongue blade can often be used to correct the condition successfully. **B,** Tongue blade used to exert pressure on the in-locked tooth. C, Correction of the in-locked condition accomplished with the aid of the tongue blade.

One of several treatment methods may be selected after an evaluation of factors such as patient cooperation, the degree of overbite that can be expected after correction, the state of the development of the occlusion, and the sequence of eruption.

Tongue Blade Therapy. In children who are cooperative and have proper encouragement and guidance at home, an anterior crossbite can be corrected with a narrow, wooden tongue blade (Fig. 27-38). Teeth in the initial state of eruption with a minimal degree of in-locking can often be repositioned within 24 hours.

The child is instructed to place the tongue blade behind the in-locked tooth and, using the chin as a fulcrum, to exert pressure on the tooth toward the labial side. This procedure should be practiced at least 5 minutes of each hour as often as possible during the day. The results of this type of therapy are often disappointing, however, because of poor cooperation of the child and parents. Tongue blade treatment is less effective if the tooth is almost fully erupted.

Lower Cemented Bite Plane. An acrylic inclined plane cemented to the lower anterior teeth is another way to reposition one or more in-locked anterior teeth. However, if a maxillary retainer may be needed, another appliance may be the best choice.

An acrylic bite plane is constructed on a stone model made from an accurate alginate impression (Fig. 27-39). Self-curing resin is applied to the model to cover the lower incisors and possibly the canines, depending on the degree of retention and stability required. An inclined plane approximately 1/4 inch in length is then added, extending lingually at a 45-degree angle to the long axis of the lower incisors.

Adjustment of the plane is made before cementation. Only the in-locked tooth should be in contact with it; there should be no other tooth contacting the plane, and the plane should not touch the palatal tissue. The posterior teeth should be out of occlusion by 2 to 3 mm. This limits the time the appliance can be worn. Considerable eruption of the posterior teeth may occur within 10 days, and a tendency to an open bite in the anterior region will result. The inclined plane may be removed when the in-locked tooth has passed over the incisal edge of the lower incisors. Moreover, if the crossbite is not

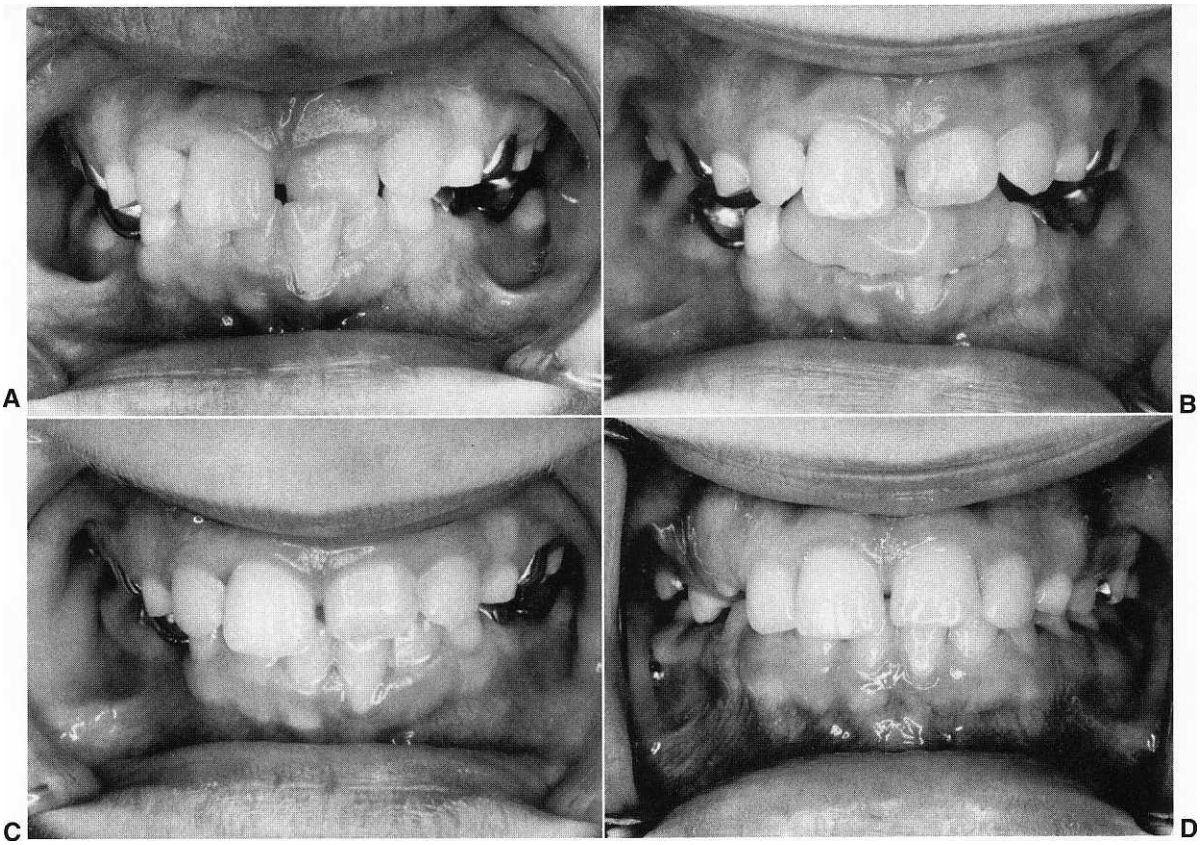


FIG 27-39. **A,** Occlusion was essentially normal except for the central incisor crossbite. **B,** A lower cemented acrylic bite plane was used to reposition the in-locked tooth. C, The in-locked tooth has been moved into correct position, and there is sufficient overbite to maintain the new relationship. **D,** Four years after the treatment of the crossbite. Notice the improvement in the appearance of the tissue on the labial surface of the lower left central incisor.

corrected within 7 to 10 days, a different appliance should be considered.

The physical activities of children wearing bite planes should be sharply restricted to minimize the possibility of injury to the teeth. Teeth that occlude on an inclined bite plane are especially vulnerable to avulsion or luxation from a blow to the chin.

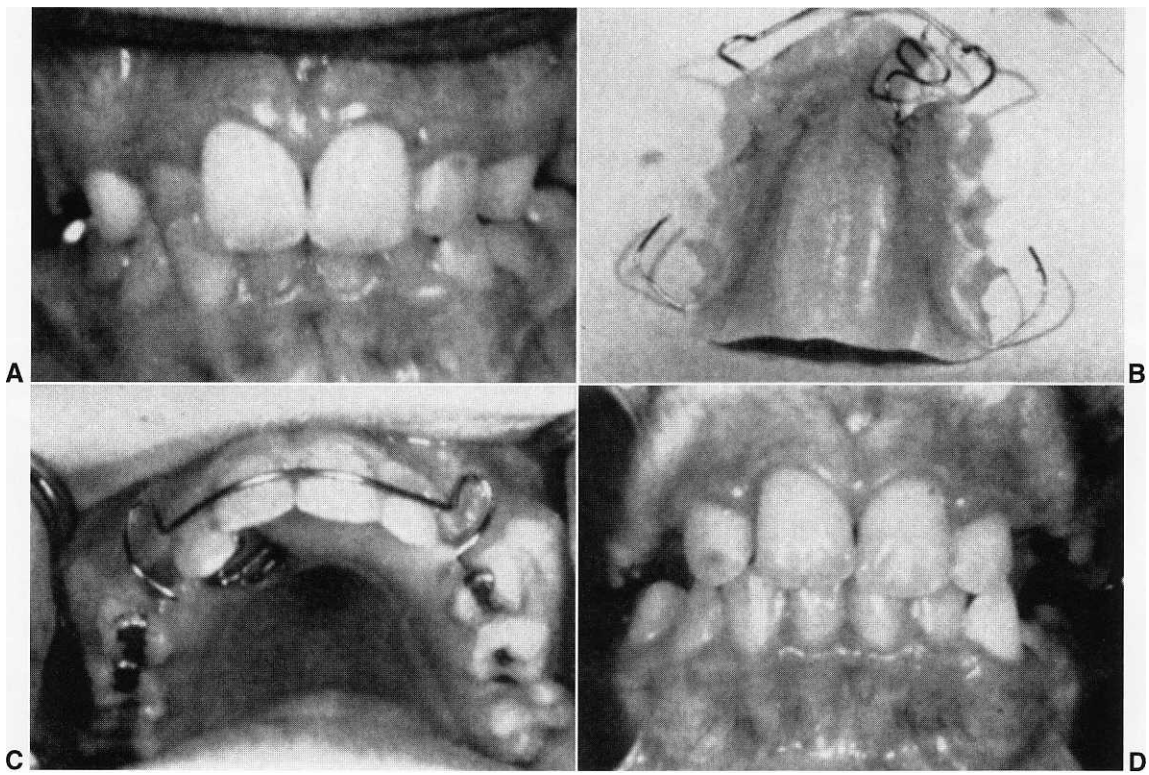
Palatal Appliances. The use of a fixed or removable palatal appliance is indicated when one or two teeth, particularly lateral incisors, are in crossbite (Fig. 27-40). Occasionally, along with providing crossbite treatment, the palatal appliance can be used to maintain space or to correct other minor irregularities in the arch. Because good retention of this type of appliance is needed, adequate clasps or other fixation must be used. It is rarely necessary to open the bite to correct the crossbite, although in cases of deep overbite an open bite may be needed until the crossbite is corrected.

POSTERIOR CROSSBITE IN PRIMARY AND MIXED DENTITION

Crossbite in the primary dentition, involving the second molar or even all of the teeth anterior to it, is common. In a study involving 515 children 3 to 9 years of age, Kutin and Hawes observed the prevalence of posterior crossbite in the primary and mixed dentition to be 1 in 13, or 7.7%.[30] Although often obscure, the cause of crossbite can be different in the three general types of the condition: skeletal, dental, and functional. The irregularity in the occlusion of the young child may not be clear-cut but may occur as a combination of all three types of buccal crossbite. A careful clinical examination, supplemented by occlusion models and an observation of the mandible at rest, are required for accurate diagnosis.

A posterior crossbite in the primary dentition usually is not self-corrected with continued development of the dentition. In fact, occlusal interference and resultant shift into a crossbite relationship can develop into a true skeletal defect if untreated. Kutin and Hawes discovered that posterior crossbite is not self-correcting and that untreated primary dentition crossbite is followed by mixed dentition crossbite. The treatment of crossbite in the primary dentition favors development of normal occlusion in the mixed dentition.

A skeletal crossbite results from a discrepancy in the structure of the mandible or maxilla. A basic discrepancy

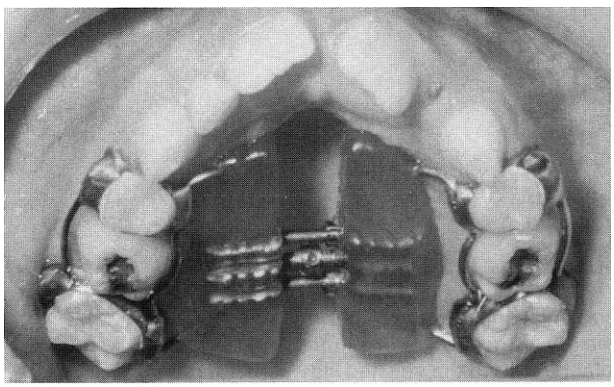


f **I G 27-40. A,** In-locked permanent lateral incisor. There was sufficient space to bring the tooth forward but a minimum amount of overbite; therefore the removable working retainer was selected. **B,** The appliance designed to correct the in-locked condition. C, Activation of the S-shaped wire at weekly intervals resulted in the labial movement of the in-locked tooth. **D,** The corrected occlusion at the time the appliance was discarded.

in the width of the arches may be noted. A narrow maxillary or wide mandibular arch is often associated with a buccal crossbite. The axial inclination of the molars, as viewed from the distal aspect, may show a flat occlusal plane relationship. In this situation, an appliance such as a W arch or quad helix, which primarily causes dental tipping, can be used. However, if the maxillary molars have a favorable axial inclination of the roots and crowns, dental expansion will only result in further lingual inclination of the roots and buccal tipping of the crowns. Thus use of an appliance that expands the palate and places less tipping force on the teeth is indicated (Fig. 27-41).

A dental crossbite results from a faulty eruption pattern; one or more of the posterior teeth erupt into a crossbite relationship. There may be no irregularity in the basal bone. After the teeth erupt, the occlusion locks them into position and drives them even further into a crossbite relationship. A low tongue position can result in the application of unequal forces to the maxillary posterior teeth and can allow these teeth to assume a crossbite relationship with the mandibular teeth. In patients who breathe through their mouths the tongue can assume a position in 'the floor of the mouth, which results in a muscle imbalance and subsequently a buccal crossbite.

Hershey, Stewart, and Warren report that breathing may improve in children with crossbites when the hard palate is widened to improve occlusion.[37] The beneficial effects of this procedure on occlusion and appearance are obvious, but the effects on breathing are often not noticed until the patient discovers that there is less nasal resistance and breathing is easier. Pressures such as those caused by thumb sucking and other aberrant sucking habits can modify the conformation of the arch. However, they can also be directly related to the development of a crossbite.



[I 1 **27-41.** An acrylic jackscrew appliance with abutments on the primary and permanent first molars. This palatal expansion appliance causes less dental tipping than a solely toothborne appliance.

A functional crossbite results from the shift of the mandible into an abnormal but often more comfortable position. The presence of a functional crossbite can be determined by observation of the relationship of the arches in the rest position. If no evidence of a discrepancy in the upper and lower midlines is seen when the mandible is at rest but a deviation of the mandible toward the side of the crossbite is noted when the teeth are brought into occlusion, the malocclusion should be considered functional. Also, when models of the upper and lower arches are examined separately, they should appear essentially symmetric. However, if there is a midline discrepancy that remains constant at the rest position and when the teeth are in occlusion, the condition is more serious and indicates a skeletal deformity requiring major orthodontic treatment.

Some functional crossbites can be corrected by reducing cuspal interference, particularly if the interference is responsible for the shift into the crossbite relationship in the canine area. Occasionally, equilibration involving a reduction of the inclined planes of the primary teeth, particularly the canines, may be all that is necessary to correct the condition. More frequently, however, the crossbite can be corrected more quickly and easily with an appliance. If examination of the occlusion reveals a developing class II or III malocclusion, the crossbite may be corrected during more comprehensive orthodontic treatment.

The time to treat a posterior crossbite is determined by several factors. If the patient is cooperative, treatment in the primary dentition is feasible. Otherwise, it is best to wait until the patient is more mature. If the permanent molars have not erupted but are no longer covered by bone, treatment should be delayed until the permanent molars can be banded. This will preclude the eruption of the molars into cross-bite if the patient is treated in the primary dentition with an appliance that mainly causes dental tipping. The absence of teeth to be used for abutments also affects treatment timing and appliance design. In some instances the early loss of primary molars may require a delay of treatment until the late mixed or early permanent dentition stage.

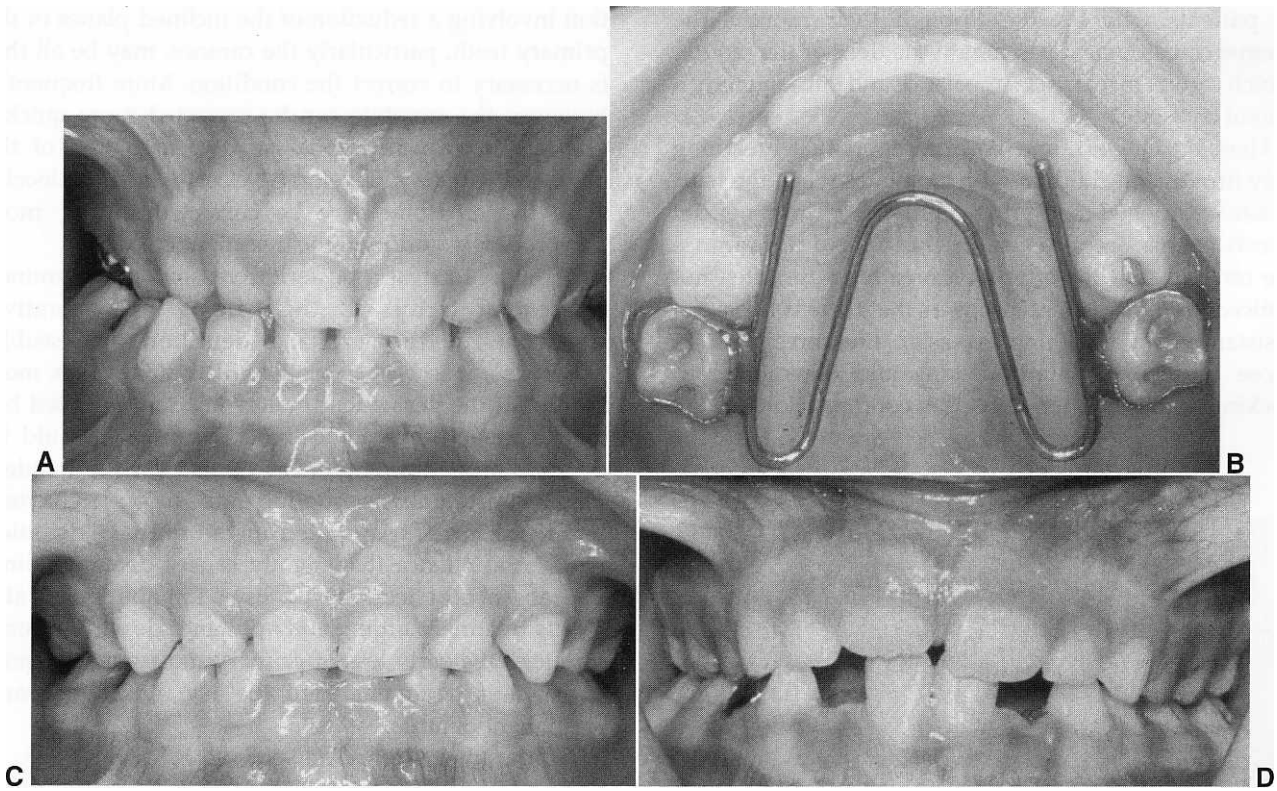
Soldered W Arch. The soldered W lingual arch, or Porter appliance, is an efficient appliance for the correction of posterior crossbites. In some instances anterior crossbites can be corrected by extension of the arm to include the canine, laterals, and centrals. The W arch may simultaneously function as a reminder appliance in some posterior crossbites associated with thumb sucking. Preformed stainless steel bands are adapted to the most distal teeth involved in the crossbite, and a

0.036- or 0.040-inch steel wire is contoured to the arch. The wire should be free of the tissue by I to 2 mm, particularly in the molar loop areas, so that no impingement on the tissue will occur during activation of the wire.

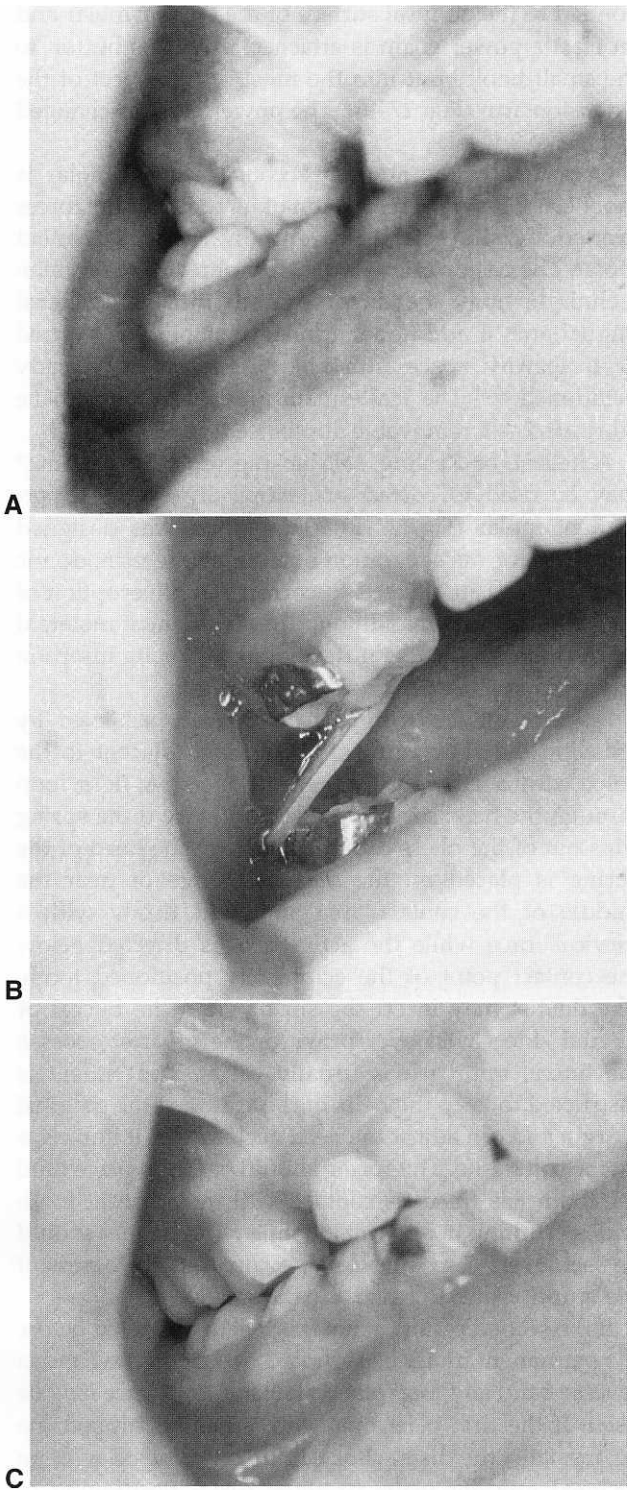
Activation is correct when the appliance must be compressed 2 to 3 mm to place it on the banded teeth. The appliance is activated approximately every 3 or 4 weeks until the crossbite has been corrected (Fig. 27-42). It is cemented during active treatment and is removed only for additional activation and adjustment. The passive appliance may be used as a retainer for 4 to 6 months after active treatment. If longer periods of retention are needed, it is best to construct a conventional Hawley type of retainer.

Although the soldered W arch is a very stable appliance, its primary value is for situations that require only buccal or labial tipping of teeth. In the younger patient (3 to 5 years of age), however, some palatal expansion occurs with the W arch. The removable W arch appliance and the quad-helix appliance allow control of root angulation of the banded permanent first molars during crossbite treatment.

Cross-Elastic Technique. If only the first permanent molar is in crossbite, the condition can often be corrected by the use of cross elastics (Fig. 27-43). A hook or button is bonded (or welded onto bands) to the lingual surface of the upper molar and the buccal surface of the lower molar and the child is shown how to place the elastics on the hooks. The elastics should be changed by the child or parent each day until the crossbite has been corrected. Normally, a crossbite involving two teeth can be corrected with cross elastics in 4 to 8 weeks. If the opposing tooth is in correct arch alignment before treatment, an anchorage appliance (lingual arch or Nance) may help prevent excessive movement of that tooth. The corrected cuspal interdigitation usually holds the teeth in their new relationship, so a retentive appliance is not needed.



f I C 27-42. **A,** Buccal crossbite in the primary dentition involving the lateral incisor and all teeth posterior to it. In the rest position the dental midlines are normal, but in maximum interdigitation, as shown here, there is a 1.5-mm mandibular midline shift to the affected side. **B,** Soldered W lingual arch ready for cementation after activation and trial seating. C, The crossbite was corrected in 6 weeks, and the appliance was removed after 3 months of retention. Notice that the dental midlines are properly aligned. D, After 3 years the first permanent molars are in proper relationship and the dental midlines remain properly aligned. There is no mandibular shift during closure.



f I G 27-43. A, Buccal crossbite is limited to the first permanent molars on the right side. Correction of the crossbite at this time will favorably influence the eruption of the second permanent molars. **B,** Molar bands with hooks and cross elastics are being used to correct the crossbite. C, The molar crossbite has been corrected in a 4-week period.

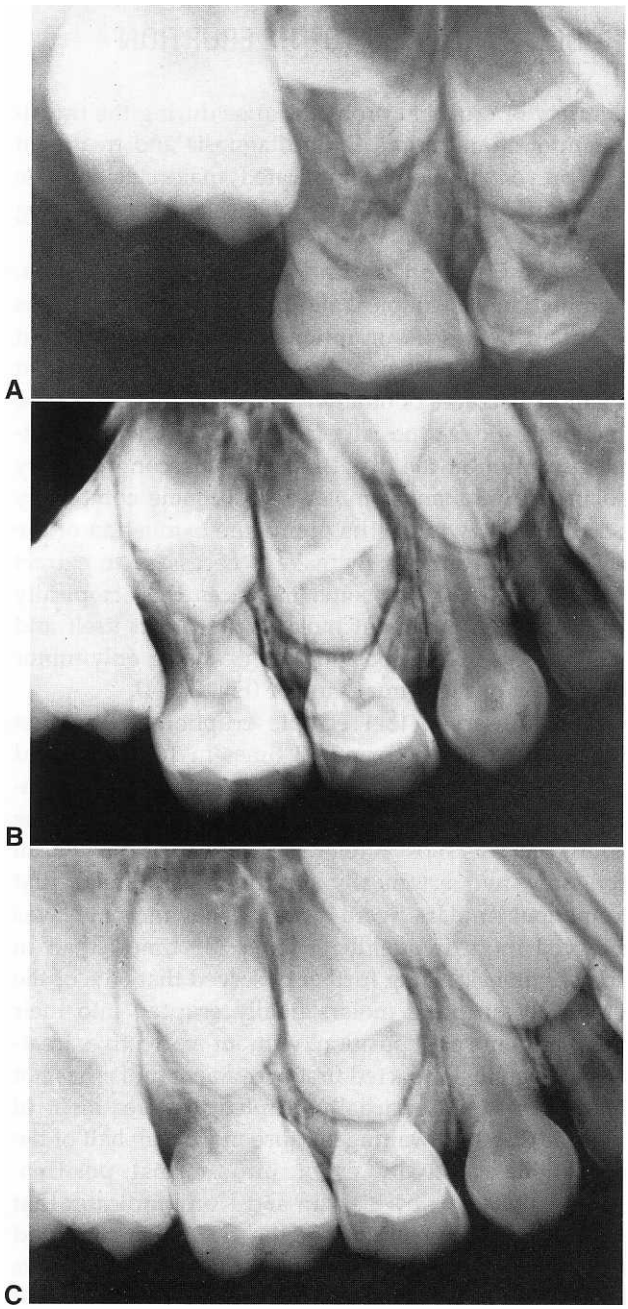
PROBLEMS RELATED TO THE ERUPTION OF TEETH

A variety of eruption problems arise during the transitional dentition period. Early diagnosis and treatment may prevent a more complicated malocclusion. The dentist must be alert to these problems and complicating sequelae.

Ectopic Eruption of the First Permanent Molars. Examination of periapical and bite-wing radiographs is important before the eruption of the first permanent molars to detect ectopic eruption. A first permanent molar, in instances of otherwise ideal occlusion, may be positioned too far mesially in its eruption, with resultant resorption of the distal root of the second primary molar. The permanent molar may become completely locked and may cause the premature exfoliation of the second primary molar or make it necessary to extract the affected tooth. In some instances the ectopically erupting first permanent molar may correct itself and erupt into its normal position after causing only minor destruction of the primary molar (Fig. 27-44).

Young observed that ectopic eruption of the first permanent molar occurred 52 times in 1619 boys and girls (3% of the time).[32] The ectopic eruption occasionally occurred in more than one quadrant in the same mouth but was most often observed in the maxilla. In fact, only two ectopically erupting mandibular first permanent molars were noted. The anomaly was observed more frequently in boys (33 times) than in girls (19 times). Young further observed that 66% of the ectopically erupting molars finally erupted into their essentially normal position without corrective treatment. When the impacted first permanent molar has not erupted or is only partially erupted, the treatment of choice is watchful waiting because more than half of the teeth will eventually erupt into normal position. However, a report by Bjerklin and Kurol indicates that ectopic permanent molars of the reversible type had freed themselves by the time the patient reached 7 years of age but that only a few molars of the locked or irreversible type had freed themselves by this age.[33] According to Bjerklin and Kurol, whether the ectopic molar is reversible or irreversible can be determined fairly accurately in patients between 7 and 8 years of age.

Humphrey described a technique for correcting ectopically erupting first permanent molars." A preformed steel band was adapted to the second primary molar on the affected side, and a wire was adapted and soldered to the band. An S-shaped loop was placed in the wire with a No. 139 pliers. The loop was opened slightly and was heat-treated with a match flame (950° F) before cementation of the loop (Fig. 27-45). The distal extension of the wire was placed in an opening in



I **G . 27-44.** A, Ectopic eruption of a maxillary first permanent molar. There is evidence of resorption of the distal buccal tooth of the second primary molar. **B** and C, Subsequent radiographs show continued resorption of the primary molar but normal positioning of the first permanent molar.

the occlusal surface of the ectopically erupting molar. It was sometimes necessary to remove the appliance in 7 to 10 days for a second activation of the loop. Another useful appliance for correcting ectopic eruptions is the Halterman appliance, which is fabricated by banding the second primary molar and soldering an extension arm that runs distal to the ectopic molar." A button is bonded to the occlusal surface of the ectopic molar and an elastic power chain is attached from this button to the small hook built into the most distal aspect of the extension arm (Fig. 27-46). The power chain is changed every 2 to 3 weeks.

Occasionally the affected second primary molar is mobile as a result of resorption of its root and the forces exerted by the ectopically erupting first permanent molar. The basic design of the Humphrey or Halterman technique may be incorporated into a bilateral lingual arch if additional appliance stability is desired or if leeway space must be preserved as already mentioned.[34, 35] The Halterman appliance may also be fabricated as a removable appliance (Fig. 27-47).

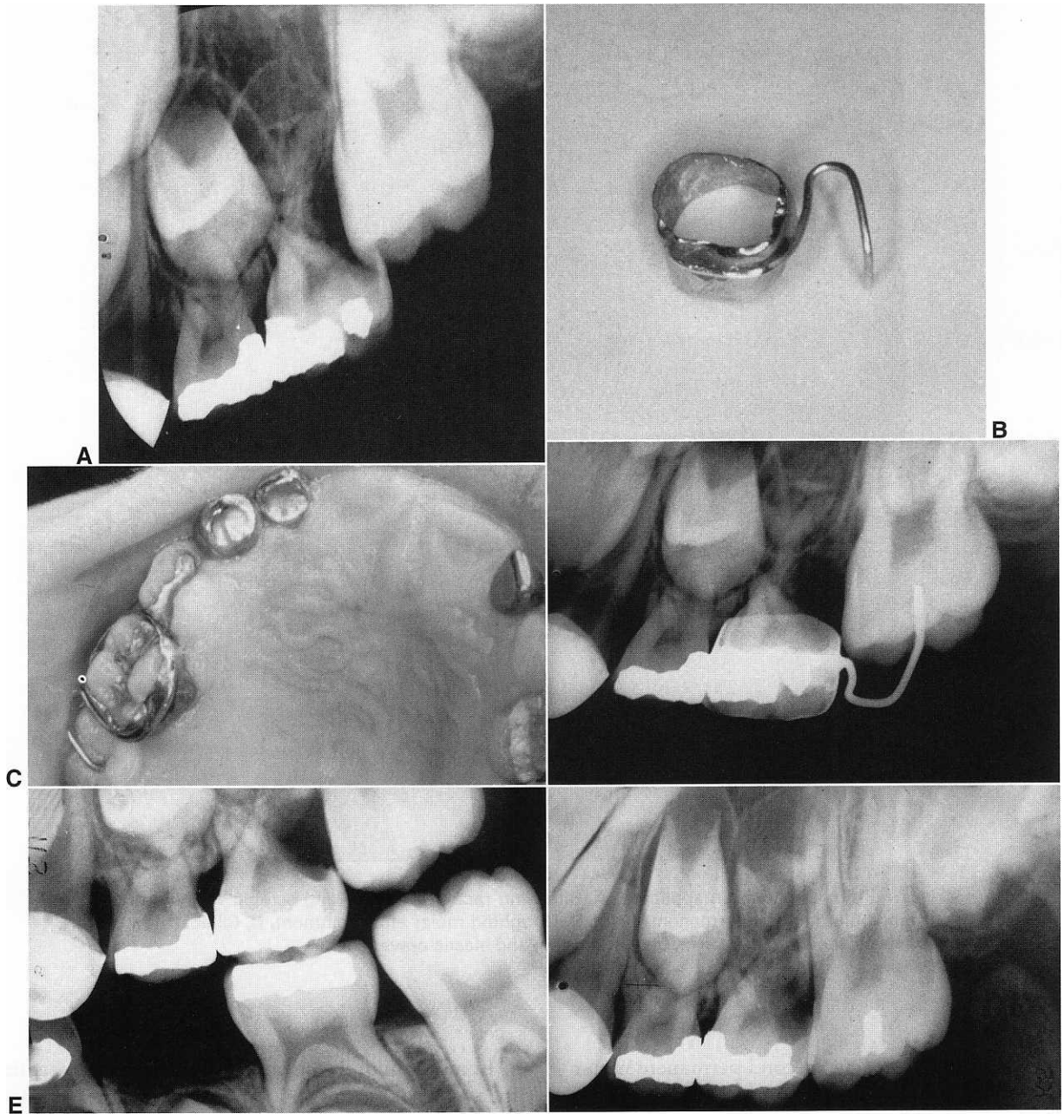
A helical, or Kesling, self-locking separating spring* may be used to correct an ectopically erupting permanent molar (Fig. 27-48). The spring was designed primarily for the separation of teeth before orthodontic banding but can be used to correct ectopic eruption of the maxillary and mandibular first permanent molars if there is sufficient dental development for its insertion and cross-arch anchorage is not required.

Insertion of the spring is most easily achieved by grasping the active arm of the spring adjacent to the helix with a How or Weingardt pliers. A floss loop through the helix serves as a safety device if the spring slips out of the pliers during insertion. The head of the spring is placed on the marginal ridge or near the middle of the contact area and held firmly with a cervical force while the active arm is directed below the contact point of the ectopically positioned tooth. The dentist may insert the spring from the buccal or lingual side (whichever provides the greatest access); the buccal approach is usually easier. The patient is instructed to keep the helix of the spring at the gingival margin and can adjust the helix occlusally if it impinges on the soft tissue. The spring should be left in place until the tooth has freed its contact with the adjacent tooth and is erupting in a normal manner. The patient should be seen every 5 to 6 weeks to evaluate the progress of the eruption and to reactivate the spring.

If the second primary molar is lost or extracted before the permanent molar has erupted sufficiently to band, a reverse band and loop or a distal shoe appliance may be used. If the first permanent molar has not tipped too far mesially, the distal shoe can be constructed with an inclined guiding plane to allow distal positioning of the tooth. If cross-arch anchorage is required, it can be incorporated into the appliance design.

Impacted Second Permanent Molars. Although impaction of second permanent molars occurs less frequently than impaction of first permanent molars,

*TP Orthodontics, Inc., LaPorte, Ind.

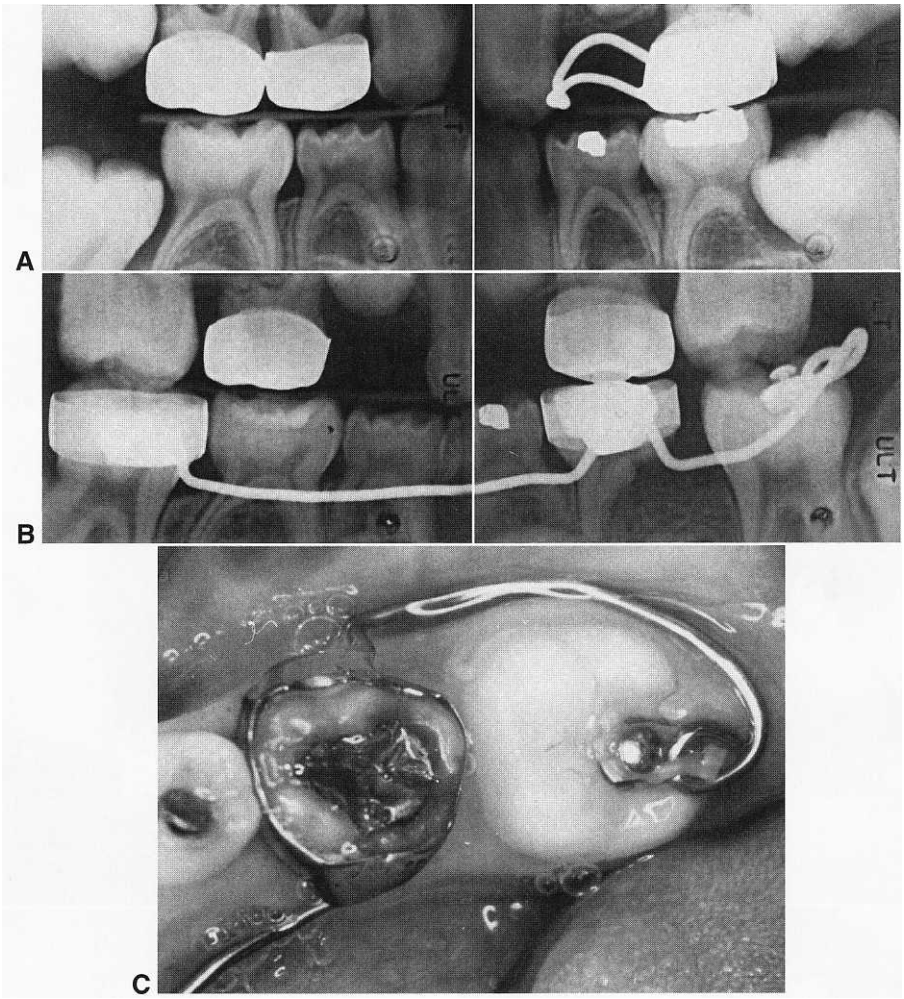


f **f . 27-45. A,** Ectopic eruption of the maxillary first permanent molar. **B** and C, A band and S-shaped wire have been fabricated to reposition the first permanent molar. D and **E,** Radiographs demonstrate the distal repositioning of the first permanent molar. F, The first permanent molar has erupted into a favorable position.

when the condition occurs, it is generally in the mandibular arch. Insufficient arch length, excessive tooth mass, or an abnormal eruption path may be responsible.

Treatment goals are to upright the tooth and achieve complete eruption (Fig. 27-49). Appliances like those recommended for the correction of an ectopically erupting first permanent molar may be used. Sometimes, simply inserting an elastic orthodontic separator in the contact area (if it is accessible) initiates uprighting. Use of a Kesling spring may also help. A spring of another design, known as the De-Impactor,* helps in uprighting many mesioangular impacted posterior teeth, particularly impacted mandibular second permanent molars.

*Arkansas Dental Products Co., West Plains, Mo.

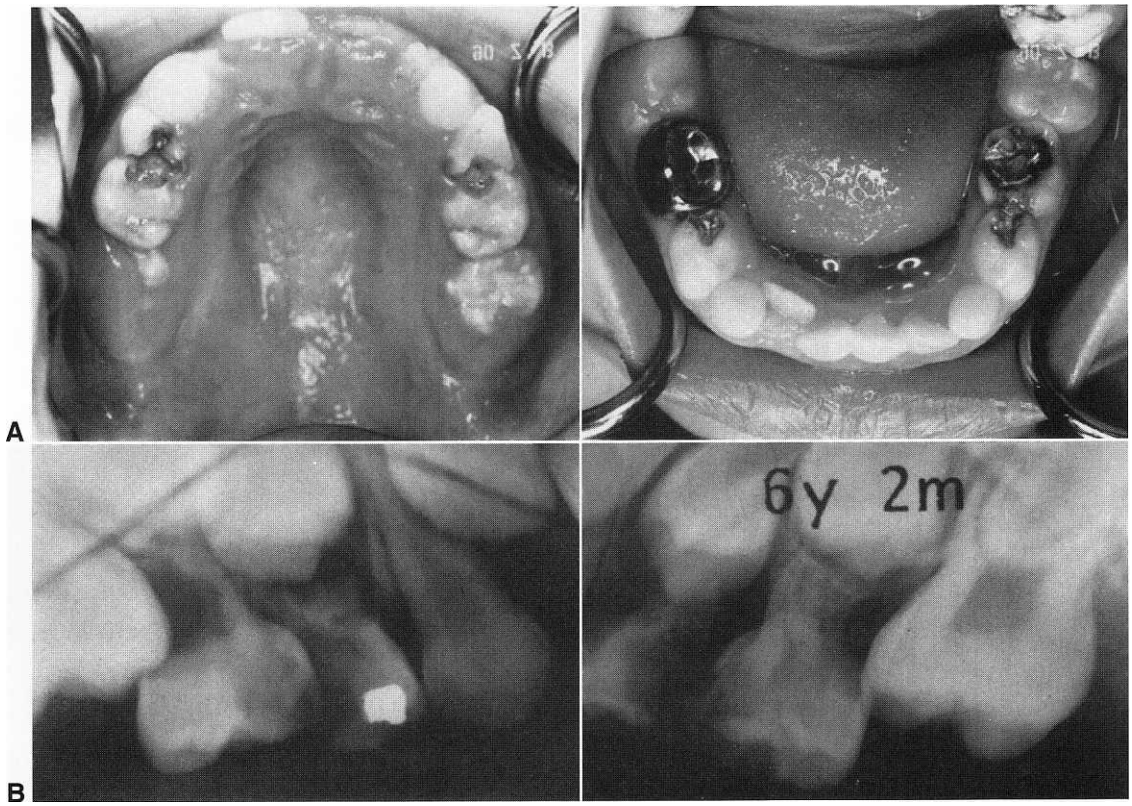


f **I f 27-46.** Fixed Halterman appliance. A, Bite-wing radiographs showing ectopic eruption of the mandibular left first permanent molar. **B,** Uprighted molar after treatment. C, Clinical view of appliance showing distal arm, bonded button, and elastic power chain.

The removal of an impacted second permanent molar to allow the third molar to move forward and take its place is impractical because the third molar usually moves into a similarly impacted position. Also, whether the third molar will have normal enamel formation and be of the correct size and shape to replace the second molar is uncertain.

Ectopic Eruption of the Permanent Lateral Incisors. The primary canine is sometimes lost prematurely when the permanent lateral incisor erupts. This condition is sometimes called *ectopic eruption* but should be distinguished from the more serious situation of the truly aberrant eruption of the lower lateral incisor in which the erupting tooth often causes loss of the primary canine and first molar. If the latter situation is not detected and treated in time, transposition of the permanent lateral and canine may occur.

In most children the lateral incisor erupts essentially in a normal manner, but because of excessive tooth mass or inadequate arch length, resorption begins on the mesial aspect of the root of the primary canine (Fig. 27-50). The process continues until the canine is prematurely lost (Fig. 27-51). Parents often fail to recognize this situation, believing that the canine is just another front tooth that should be lost at approximately 7 years of age. In reviewing the radiographs, the dentist should be alerted to the condition and should be prepared to manage the problem if an early loss of primary canines occurs. Croxton found that 32 of 400 children in the transitional dentition period had 48 ectopically erupting permanent lateral incisors.[3"] He noted 17 ectopically erupting teeth in the maxilla and 31 in the mandible. In contrast to the ectopic eruption of the first permanent molar, this condition is



[I C **27-47.** Removable Halterman appliance. **A,** Pretreatment photos. **B,** Pretreatment radiographs showing bilateral ectopic eruption. Continued

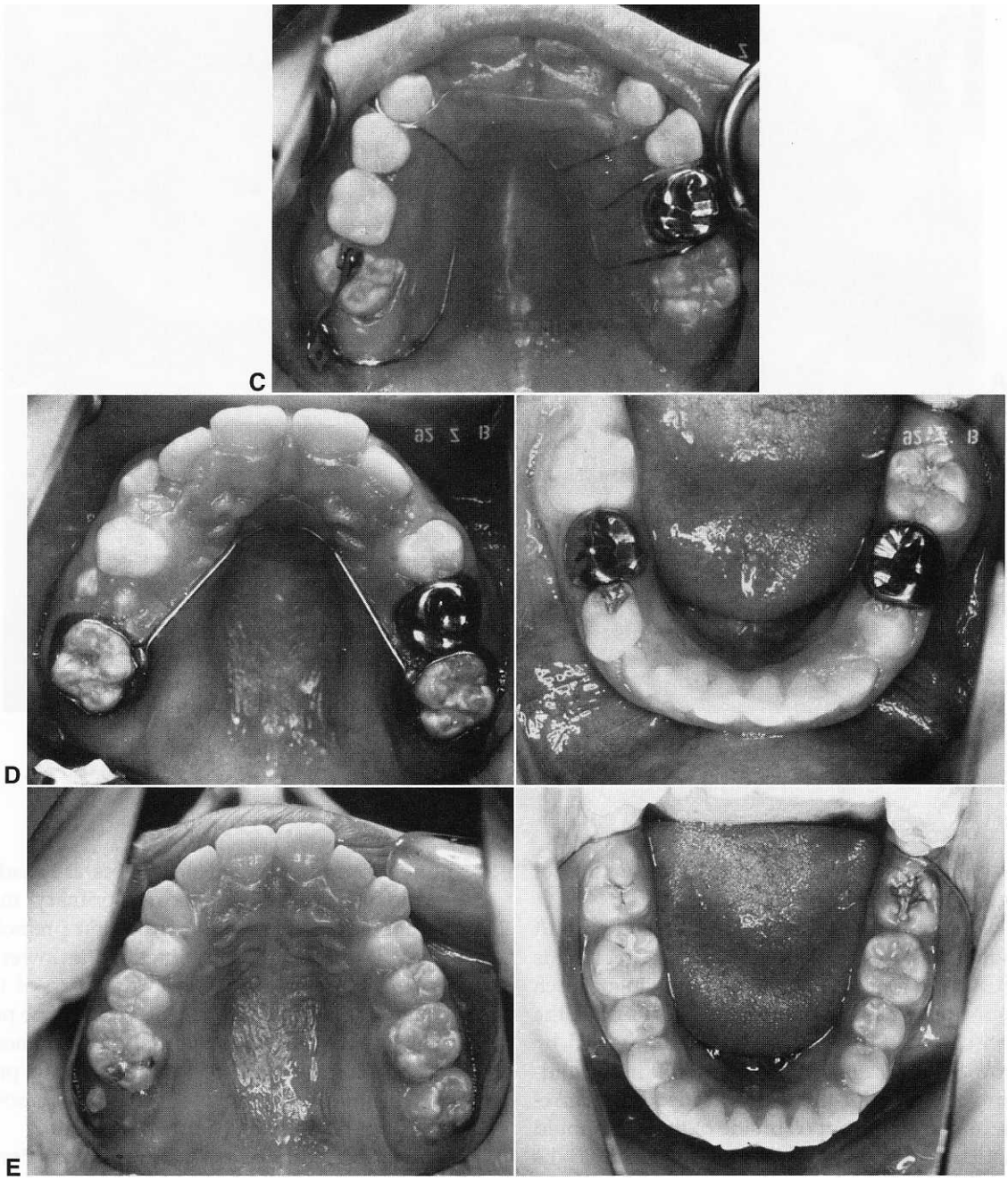
probably seen only in children with some degree of arch-length inadequacy.

If loss of the primary canine is unilateral and no shift in the midline has occurred, use of a space maintainer is indicated. A band and loop maintainer or a lingual arch with a spur can be used to prevent distal drifting of the lateral incisor. If the unilateral loss is accompanied by severe crowding of the incisors and if a shift in the mid -line toward the areas of the loss is evident, the corresponding canine on the opposite side of the arch should be extracted, and a passive lingual arch should be placed. Maintaining symmetry and preventing a shift in the midline and retrusion of mandibular incisors are important. A careful and comprehensive orthodontic analysis is indicated before further treatment is initiated.

If the problem of crowding in the lower anterior seg ment is identified before the premature loss of canines, the condition may be alleviated by a timed sequence of "slicing" procedures on the remaining primary teeth, as advocated by Wyatt." Additional space is created for favorable positioning of the lateral incisors by removal of a portion of the primary canines on the mesial surface. The slicing procedure is repeated as indicated on additional primary teeth as the permanent teeth continue to erupt in sequence. Slices are made at the appropriate time on the second primary molars to accommodate the eruption of the first premolars and canines. If the initial crowding in the lower arch is approximately equal to the leeway space of the arch, adequate space for all permanent teeth will be preserved without appliances or extraction of permanent teeth. Wyatt recommends performing the slicing procedure even when the amount of tooth structure removed may require a pulpotomy on the primary tooth.

Impaction and Delayed Eruption of Permanent Canines. Mandibular third molars are the most frequently impacted teeth. Next in frequency are the maxillary permanent canines. According to Cohen, impaction of the maxillary canine may occur because this tooth has the longest period of development, follows the most dubious course in its eruption, and occupies several developmental positions in succession.[3b] It can also be easily deflected from its normal course of eruption.

The dentist should look for evidence of asymmetric eruption of the maxillary canines in the radiographic and oral examinations. Early diagnosis of an abnormal path and sequence of eruption is advantageous. Generally, this information should have been obtained



Ilfi **27-47, contd. C,** Removable appliance in place with elastic power chain attached. **D,** Occlusal views after correction and with Nance appliance in place. **E,** Occlusal views in early permanent dentition.

from palpation of the canine crypt or from radiographs by the time the patient is 8 or 9 years of age. A delay in the eruption can allow adjacent teeth to encroach on the space needed for the canine and contributes to the impacted condition. If the maxillary permanent canine is definitely impacted, surgical intervention is indicated. However, localization of the tooth by special radiographic technique is essential. The procedure described in Chapter 5 will help in localizing the impacted tooth.

The available arch space for the canine should be compared with the size of the crown. The measurement of the opposite canine can be used, or the impacted canine can be measured directly from the radiograph. If space for the tooth is adequate and occlusion is essentially normal, the space should be maintained.

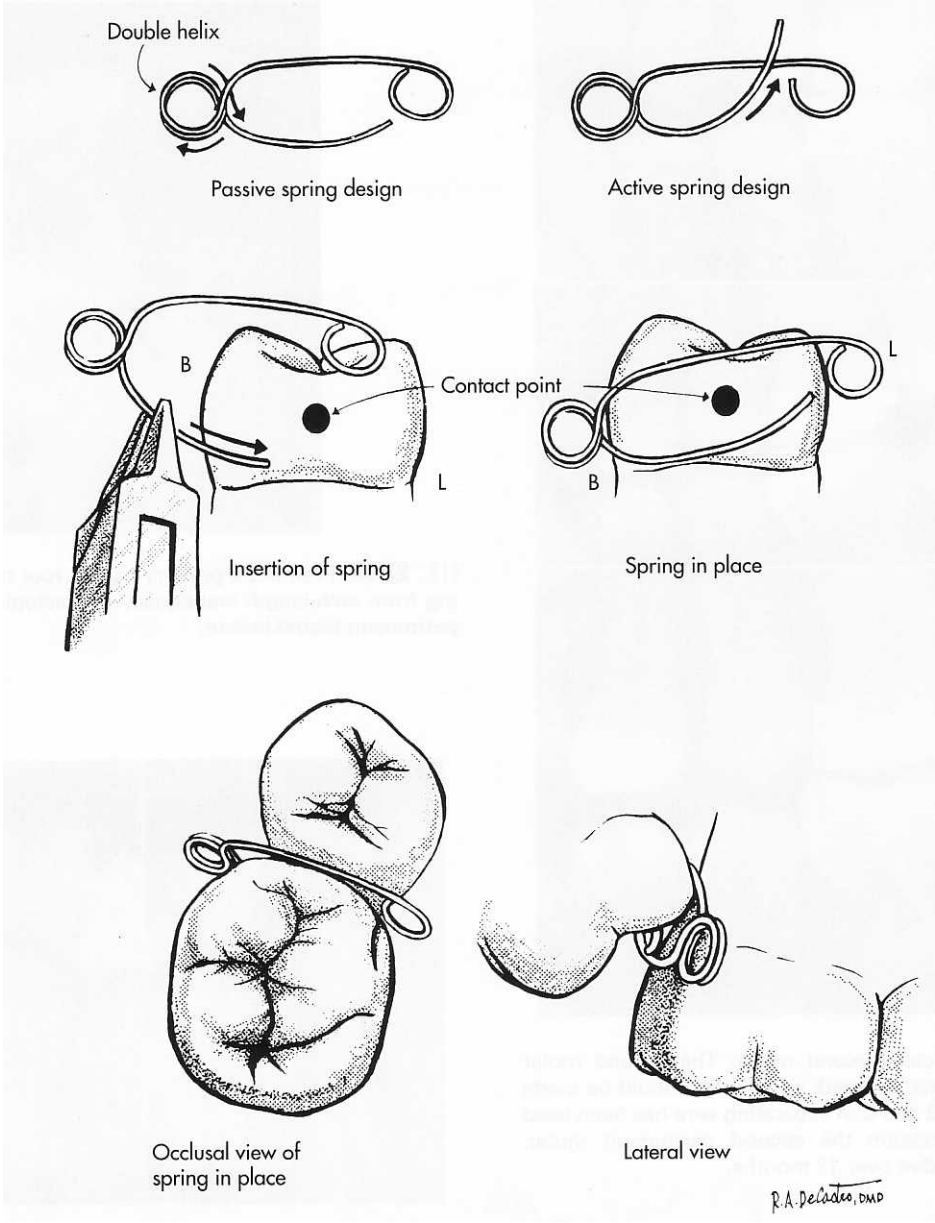


FIG. 27-48. Kesling spring of 0.022-inch stainless steel wire designed for the correction of ectopically erupting permanent molars.

Surgical exposure of the impacted canine and maintenance of the pathway are normally the treatment of choice. If the primary canine is still present, it should be extracted. Sufficient soft tissue and bone should then be removed from the crown of the impaction to maintain an opening that will stimulate the eruption of the impacted tooth. A canine in deep palatal impaction may move into an in-locked position or may require orthodontic movement to bring it into a desirable relationship with other teeth.

Supernumerary Teeth and Accompanying Malocclusion

Supernumerary teeth, which result from the continued budding of the enamel organ of the preceding tooth or from excessive proliferation of cells, can be responsible for a variety of irregularities in the primary and transitional dentition. The stage of differentiation determines whether a cyst, an odontoma, or a supernumerary tooth will result. In separate studies, Stafne[39] and Schulze' concluded that supernumerary teeth occur in approximately 1 of 110 children. The ratio

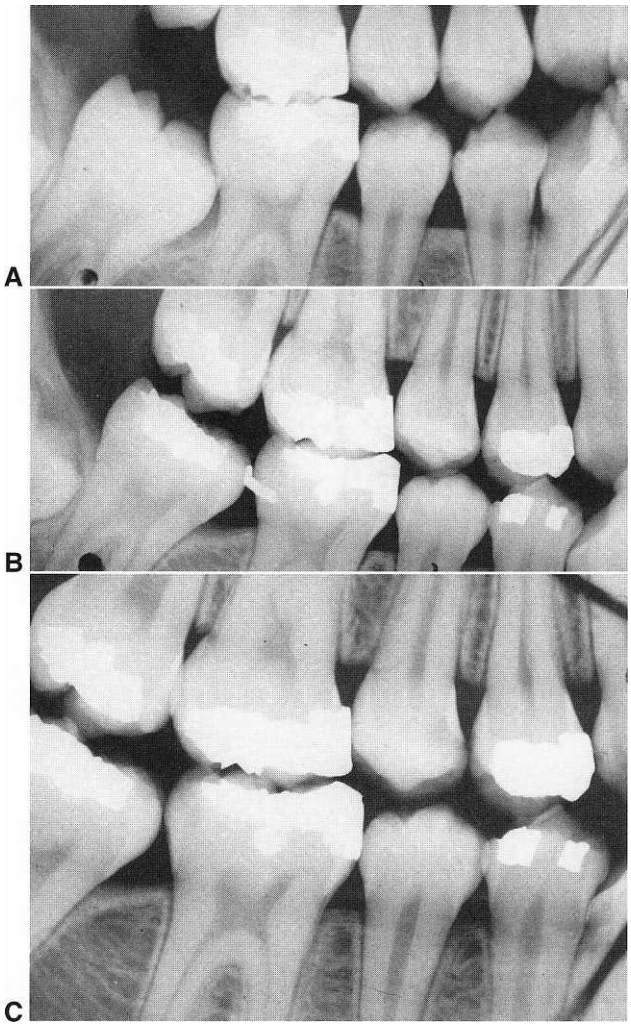


FIG 27-49. **A,** Impacted second molar. The second molar should not be extracted; instead, an attempt should be made to upright the tooth. **B** and C, A separating wire has been used successfully to reposition the second permanent molar. Treatment was extended over 12 months.

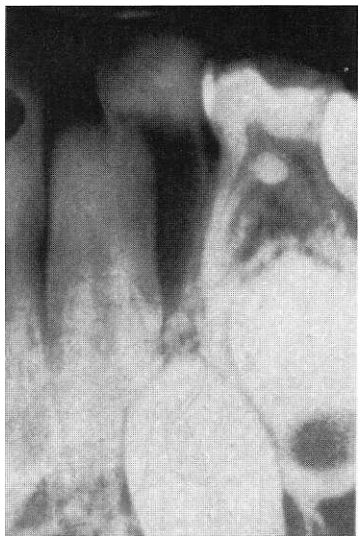


FIG. 27-50. Premature primary canine root resorption, resulting from arch-length inadequacy and ectopic eruption of the permanent lateral incisor.

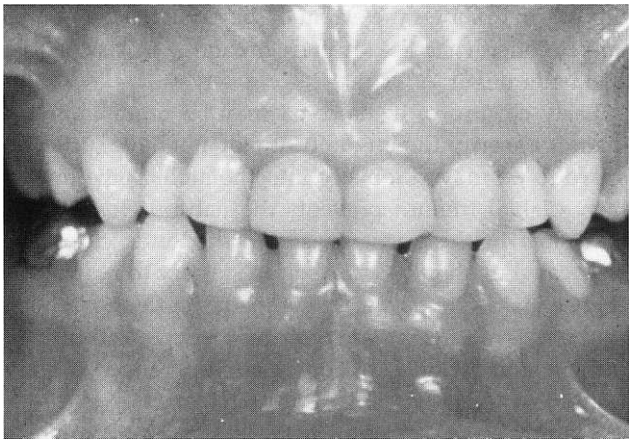


FIG 27-51. Primary canine has been lost prematurely on the left side of the arch. The permanent incisor has drifted distally into contact with the first primary molar. Extraction of the corresponding primary canine would have aided in maintaining symmetry in the arch.

of the prevalence in the maxilla and in the mandible is 8:1. The most common site for supernumerary teeth is the maxillary incisor area. Supernumerary primary teeth (Fig. 27-52) are apparently less common than supernumerary permanent teeth. The occurrence of supernumerary teeth in several members of the same family has been observed, which indicates a familial pattern.

Supernumerary teeth, particularly in the maxillary anterior region, may prevent the eruption of adjacent permanent teeth (Fig. 27-53) or cause their ectopic eruption (Fig. 27-54). Both conditions frequently result in an irregularity of the developing occlusion that requires treatment. If a supernumerary tooth is found, a special radiographic technique, as described in Chapter 5, can be used to localize the tooth. The decision whether to intervene surgically or keep the tooth under observation can then be made.

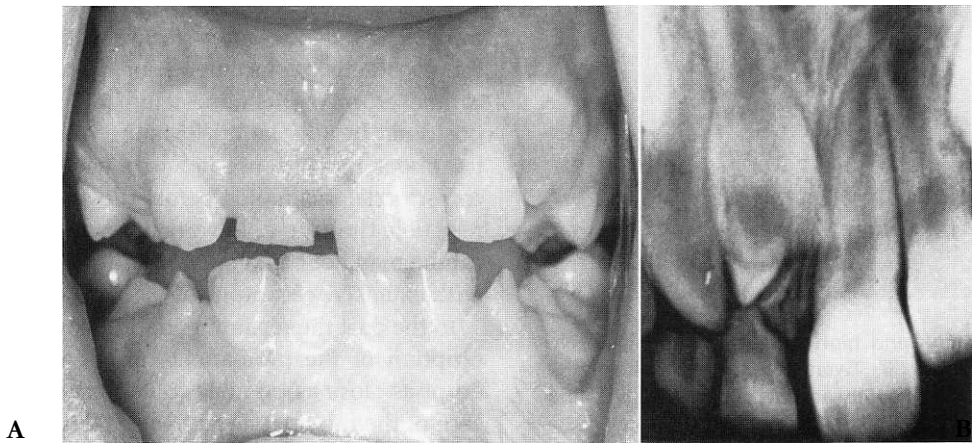
Surgical Removal of Supernumerary Teeth. The surgical removal of an unerupted supernumerary tooth is the eventual course of treatment. However, if the supernumerary tooth does not interfere with the symmetric development and eruption of adjacent teeth



f **I G 27-52.** Supernumerary maxillary primary incisors. There may be corresponding supernumerary permanent teeth. The teeth should be counted at the time of the clinical examination so that erupted supernumerary teeth will not be overlooked.

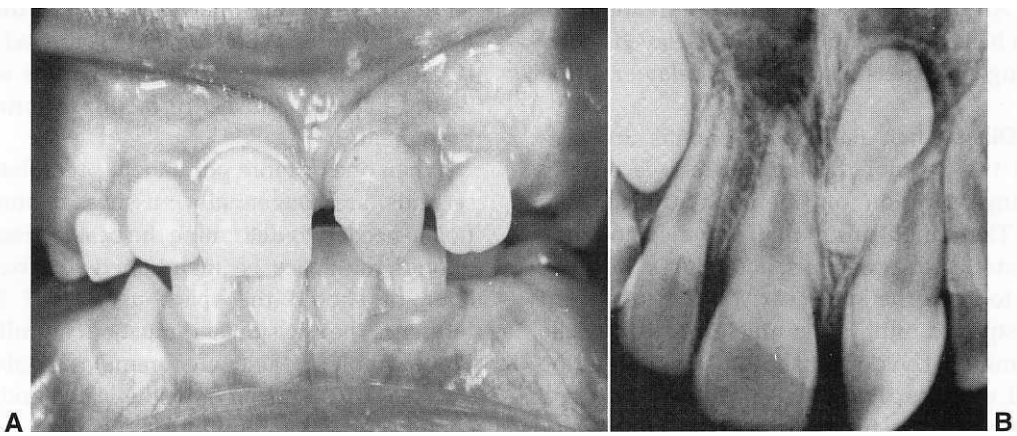
and no evidence of the formation of a cyst exists, the correct decision may be to observe the tooth until the child is old enough to tolerate the procedure better. Many pediatric dentists and surgeons prefer to delay surgery until the permanent teeth erupt and root closure is complete, provided that no evidence is seen of a developing irregularity in the occlusion.

The delayed eruption of a maxillary incisor as a result of a midline supernumerary tooth is common. In this case, immediate surgical removal of the supernumerary tooth is recommended. During surgery, the bone and soft tissue should be removed from the incisal third of the tooth or teeth that are delayed in their eruption. If the permanent teeth are positioned extremely high, however, a prolonged period of watchful waiting may be necessary until they have migrated within the bone to a position that would allow additional surgical



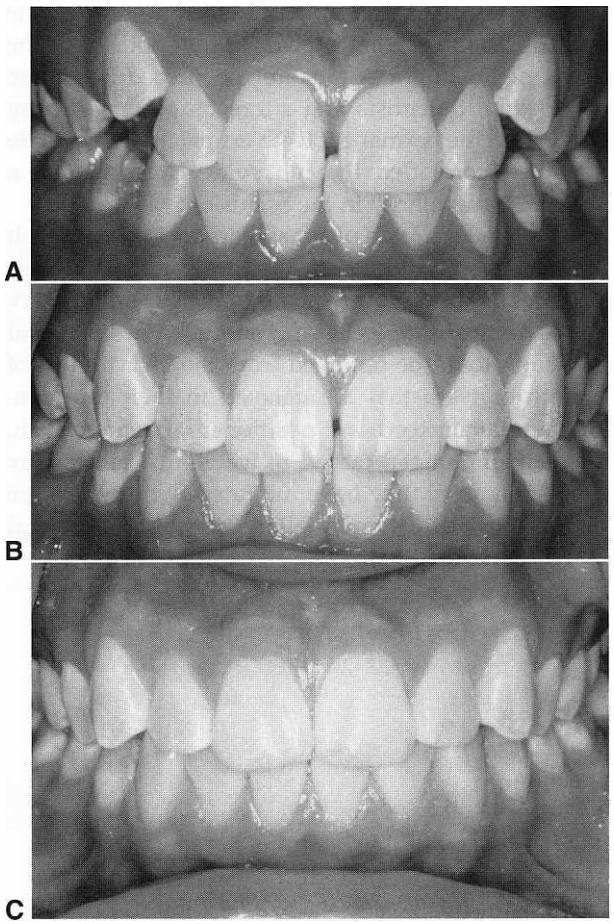
A

G **I G 27-53.** A, Dentition of a patient with an unerupted maxillary permanent incisor. Space closure is the result of delayed eruption. **B,** The radiograph shows a supernumerary tooth (mesiodens), which has delayed the eruption of the permanent incisor.



A B

[1 G **27-54.** A, Rotation and labioversion of a maxillary anterior tooth may be caused by the presence of a supernumerary tooth. The position of this tooth increases its susceptibility to fracture. **B,** Radiograph of a well-developed midline supernumerary tooth. Surgical removal of the tooth is indicated.



[I G **27-55. A,** Diastema and highly positioned canines. No treatment was instituted. **B,** Twenty-four months later, the diastema has closed significantly and the canines are erupting into a more favorable position. C, Thirty-six months later, the diastema has closed completely, and the canines have assumed a near-ideal position.

intervention. An open pathway should be maintained, if possible, to hasten the eruption of the delayed tooth. A thin covering of dense scar tissue can delay eruption indefinitely.

Anterior Diastemas. Frequently parents are concerned about the anterior diastema, which is usually present during eruption of the maxillary incisors and canines. They might insist that active orthodontic treatment be started to close the space. Unless there is a valid reason to close the space early, active treatment should be postponed until the complete eruption of the permanent canines. The dentist should explain that the diastema will close as the laterals and canines erupt. After the canines erupt, the condition can be reevaluated and appropriate treatment undertaken as needed. Fig. 27-55, A, shows a patient whose parent wanted the diastema closed and was concerned about the high position of the canines. No treatment was begun. Fig. 27-55, B, shows the extent of the diastema 24 months later. Notice that the canines are now in reasonably good alignment.

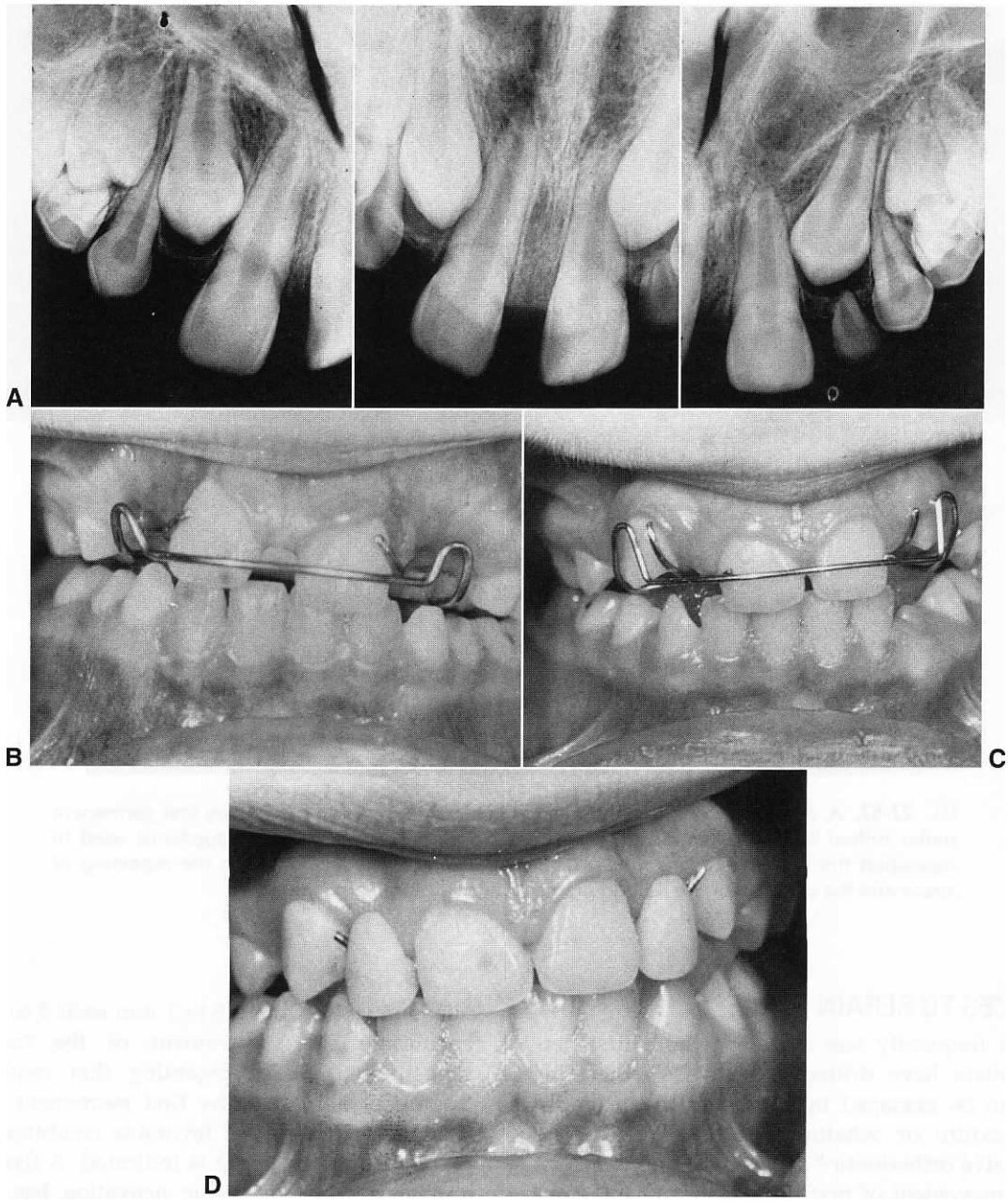
Early closure of a diastema may be required if the laterals are erupting lingually and do not have sufficient space to be moved labially into the arch. Sometimes a heavy labial frenum prevents the natural closure of a diastema. In these instances, if orthodontic closure is advocated, it should occur before surgery to reduce the chance that scar tissue will impede tooth movement. If there is sufficient arch space for the eruption of incisors and canines, it is best to delay frenum surgery until these teeth have fully erupted.

Congenitally Missing Teeth. Managing problems of congenitally missing teeth requires a complete diagnosis and thorough evaluation of arch length and occlusion. Early consultation with the orthodontist and prosthodontist may help in determining appropriate long-term care.

If one or both of the permanent maxillary laterals are missing, the dentist must decide whether to hold space for prosthetic replacements or to let the permanent canine erupt or drift mesially into the lateral incisor position. In the latter instance, limited orthodontic treatment may be needed to place the canine in the correct axial inclination before the crown is reshaped to resemble the lateral. In some instances the shape of the canine may not be favorable for its use as a lateral, even with extensive recontouring.

In the past the recommendation has been to influence eruption of the permanent canine to encourage it to erupt as close as possible to its normal position. In the case of the missing lateral incisor (Fig. 27-56), and with the advent of implants for replacement of lateral incisors, that recommendation has changed. According to Kokich, the ideal situation is to encourage the canine to erupt adjacent to the permanent central incisor.' After it has erupted, it can be moved distally into its normal position. When the tooth is moved distally, bone is laid down, forming an alveolar ridge with adequate buccal-lingual width to facilitate proper implant placement.

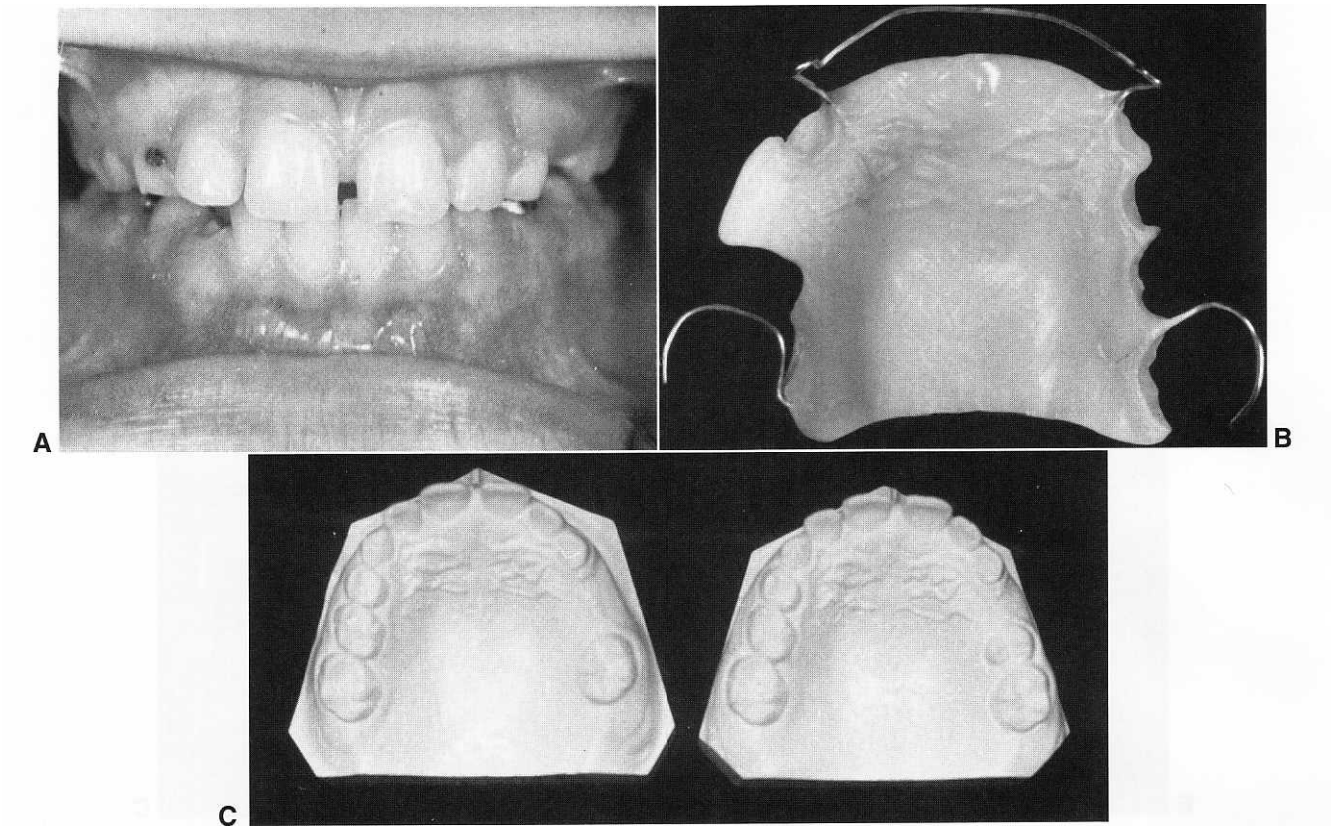
When one or more permanent premolars, usually the seconds, are congenitally missing, a thorough evaluation is needed to determine the best course of treatment. Should the space be maintained for fixed prostheses later, or should the space be closed? Many factors influence the decision and must be carefully considered. For example, if just one premolar is absent and the rest of the occlusion is esthetically and functionally sound, the treatment of choice may be prosthetics. Such treatment may maintain the most favorable occlusal relationship, even though several replacements may be required during the patient's lifetime. On the other hand, if three or four premolars are missing,



f I G. **27-56. A,** Congenital absence of permanent lateral incisors. **B,** The primary canines have been extracted, and a working appliance has been constructed to close the diastema between the central incisors. C, Auxiliary wires have been added to the appliance to guide the permanent canines into a more favorable position. **D,** Space has been regained for eventual fixed prostheses to replace the lateral incisors. Meanwhile the removable retainer will be worn.

orthodontic treatment may obviate the cost and need for continued prosthetic care and replacement.

In some instances the primary molar may be left intact, may be retained, and may function well for many years. In many cases, however, the larger mesiodistal width of the primary molar may cause incorrect occlusal relationships with the permanent teeth. Sometimes, slicing the mesial and distal surfaces of the primary molar allows the proper permanent molar interdigitation, but often the bulbous, divergent roots of the primary second molar prevent the mesial movement of the permanent molar. Also, even if the permanent successor is missing, the roots of the primary molar may be resorbed, and the tooth may be lost eventually.



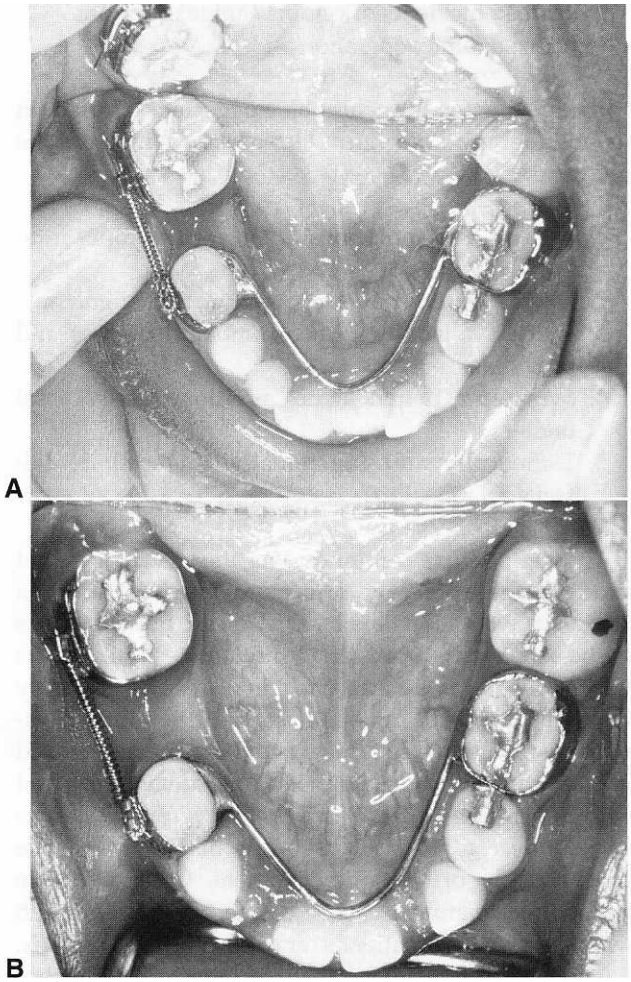
[I L **27-57. A,** An essentially normal occlusion in a child. However, a maxillary first permanent molar drifted forward after the untimely loss of primary molars. **B,** The appliance used to reposition the first permanent molar. C, Before-and-after models illustrate the regaining of space and the eruption of the second premolar into a desirable position.

APPLIANCES TO REGAIN SPACE

The dentist frequently sees children whose first permanent molars have drifted mesially. Whether this problem can be managed by means of an uncomplicated procedure or whether it will require more comprehensive orthodontics depends on many factors. The distal movement of first permanent molars, other than minimal tipping, can be obtained satisfactorily with a headgear appliance.

Several removable appliances have been recommended for regaining space (tipping), particularly when the first permanent molars have drifted mesially. When these appliances are used to reposition a molar, a reciprocal force is exerted on the teeth and supporting tissues anterior to the space, and the result may be an undesirable flaring of the anterior teeth. This is particu larly true during the mixed dentition period, when the permanent incisors are incompletely erupted and may be adversely influenced by even so-called minimal forces.

If the dentist concludes that conditions are favorable, an appliance like the type shown in Fig. 27-57 may be considered. Activation of the 0.025-inch steel wire should be from 0.5 to 1 mm each 3 to 4 weeks to encourage slow movement of the tooth to be repositioned. Space regaining that requires distal tipping of a mandibular first permanent molar is a challenge. However, if favorable conditions exist, an attempt to regain space is indicated. A fixed intraoral appliance with coil-spring activation has been used successfully for this procedure (Fig. 27-58). Bilateral stability and anchorage may be provided with a soldered lingual arch. An edgewise twin bracket is aligned and welded to the buccal surface of the abutment band adjacent to the space, before the lingual arch portion of the appliance is cemented into place. A band is also fitted to the first permanent molar to be tipped distally, and a buccal tube is properly aligned and welded to the band before it is cemented into place. A 0.016-inch round or a 0.016 x 0.016 inch rectangular wire is selected so that it will slide freely in the buccal tube but can also be securely fixed to the bracket with ligature wire. The wire is cut to the desired length and adjusted to the alignment of the teeth by making smooth, gentle bends if necessary. A section



F I i. **27-58. A,** Coil spring space-regaining appliance in place on the day it was inserted. **B,** Desired amount of space regaining accomplished in about 2 months. Although the entire amount of space was not regained, the first permanent molars on the affected side are in a class I relationship. During this period of treatment the mandibular primary lateral incisors exfoliated normally and the position of the lingual arch wire encouraged some flaring of the erupting permanent central incisors.

of open-coil spring (0.009 x 0.020 inch) approximately 2 mm longer than the space between the bracket and the tube is placed around the wire, and the entire assembly is fixed in position. As the space opens, the wire and spring are replaced with longer sections at approximately 4-week intervals until the desired position is attained.

SERIAL EXTRACTION FOR THE PEDIATRIC PATIENT

Dewel has defined the procedure referred to as *serial extraction* as the orderly removal of selected primary and permanent teeth in a predetermined sequence."- Its use is indicated only when dental arches are structurally inadequate for the developing teeth and when there is little or no hope of ever attaining a normal size and proportion. Serial extraction is indicated primarily in severe class I malocclusions in the child with mixed dentition who has insufficient arch length for the amount of tooth material. Salzmann believes that it is not always possible to predict at an early age that space will be insufficient to accommodate all the permanent teeth in normal alignment.' Dewel agrees that growth is often unpredictable; no one can be certain that it will be unfavorable to the development of the occlusion.}'- Many children with arch-length inadequacy have spectacular growth when least expected and may be treated successfully without sacrificing permanent teeth.

The primary canine is removed first, the first primary molar second, and the first premolar last in the serial extraction procedure. The interval between extractions varies from 6 to 15 months. After removal of the primary canines, there is a degree of self-correction in the position and alignment of the permanent incisors. Dewel advocates an alternative extraction sequence in the borderline malocclusion when extraction of the first premolar can be avoided; in this case, growth may yet be sufficient to accommodate all the teeth. The first primary molar is extracted 6 to 12 months before the extraction of the primary canine to encourage first premolar eruption and to retard canine eruption. If growth exceeds expectations, as it occasion-ally does, there will be no need to extract the first premolar.

The parents must be informed that, without follow-up orthodontic treatment, the serial extraction procedure may not result in a favorable occlusion. What frequently occurs is persistent spacing at the extraction sites, the development of a closed bite, lingually tipped lower incisors, distally tipped canines, and mesially tipped second premolars; none of these is desirable. Serial extraction has a limited place in dental practice and requires regular reassessment to determine whether the serial extraction plan should be continued.

COMPREHENSIVE ORTHODONTICS FOR THE DEVELOPING OCCLUSION

This section of the chapter provides a brief description of comprehensive orthodontic management of developing pediatric occlusions from birth up to the early permanent dentition. There are multiple rationales for providing orthodontic treatment during these developmental stages, and they include addressing various transverse, sagittal, and vertical discrepancies in the developing occlusion. As stated by

McNamara, there is no best time for all patients, at all times, and not all early treatment is good treatment.[44] However, there may be advantages to providing treatment during these early developmental stages in children. The American Association of Orthodontists has recommended that all children have an orthodontic screening by at least age 7. They suggest that the benefits of early treatment are as follows:

- Influences jaw growth in a positive manner
- Harmonizes width of the dental arches
- Improves eruption patterns
- Lowers risk of trauma to protruded maxillary incisors
- Corrects harmful oral habits
- Improves esthetics and self-esteem
- Simplifies and/or shortens treatment for later corrective orthodontics
- Reduces likelihood of impacted permanent teeth
- Improves speech problems
- Preserves or gains space for erupting permanent teeth

Dugoni suggests the following indications for early treatment":

- Dental and/or skeletal class II and III
- Posterior and/or anterior crossbites
- More than 6 mm of overjet
- Maxillary midface deficiency
- Moderate incisor crowding
- Crowding resulting in ectopic eruptions or periodontal compromises
- Severe anterior open bite greater than 3 mm
- Severe deep bite with palatal impingement
- Harmful oral habits
- Midline discrepancies
- Management of congenitally missing teeth or supernumerary teeth
- Ectopic maxillary canines
- Craniofacial anomalies

In addition, Dugoni suggests that the benefits of early treatment can include the following":

- Reduced incidents of premolar extraction
- Decreased extent or possible elimination of the need for a second phase of treatment
- Reduced need for surgical orthodontics
- Increased stability of transverse and anteroposterior dimension changes with phase I treatment
- Increased stability of lower incisor alignment
- Reduced incidents of root resorption
- Reduced incidents of mucogingival problems
- Reduced incidents of ectopic canine eruptions
- Better patient cooperation

Finally, treatment goals, as suggested by Bell in his monograph *Integrated Orthodontics,* include the following":

1. Super class I molar positioning with proper rotational alignment
2. Normal anterior overbite, overjet, and alignment
3. Space availability to accommodate canines and premolars
4. Symmetric dental arches positioned over basal bone
5. Esthetically and functionally balanced dental-facial relationships

Although debate continues over the advantages and disadvantages of early orthodontic treatment, in a consensus workshop held by the College of Diplomates of the American Board of Orthodontics in 1997, the participants stated that the final results of early treatment are better and that early orthodontic treatment has numerous advantages for both the patient and the practitioner.' Indeed, most of the January 1998 and June 2002 issues of the *American Journal of Orthodontics and Dentofacial Orthopedics* were dedicated to the topic of early orthodontic treatment. Comprehensive orthodontic treatment can be defined as the thorough evaluation of the patient's entire orofacial complex. It is designed to address each of the patient's orthodontic and orthopedic needs, either all at once or in specifically planned stages of treatment. It is best to look at comprehensive orthodontic care in terms of developmental stages of the patient. The three major developmental dental stages of growing patients are the primary dentition; the mixed, or transitional, dentition; and the early permanent dentition. It is also helpful to assess orthodontic problems in the three planes of space (i.e., transverse, sagittal [anteroposterior], and vertical).

Finally, if the individual dental practitioner decides to accept a patient for comprehensive orthodontic care, it is important that the practitioner recognize that he or she is accepting responsibility for complete comprehensive treatment. It is not appropriate in most circumstances for the practitioner to complete only one phase of treatment with the intention of referring the patient for any final phases of care. Comprehensive orthodontic care is just that—complete care of the patient's orofacial complex. If the practitioner recognizes the need for some interceptive orthodontic care as well as a more comprehensive need for treatment, but is unwilling or unable to provide the comprehensive care, it is only appropriate to refer the patient to a practitioner willing

to address all of the orthodontic needs. Piecemeal treatment is unacceptable, except as an orderly part of a comprehensive plan.

PRIMARY DENTITION

Abnormal dental and skeletal relationships that are perpetuated through the primary dentition can become increasingly difficult to correct as the child becomes older. Therefore, although full, fixed, edgewise appliances or orthodontic braces are rarely placed in the primary dentition, orthodontic and orthopedic types of changes can be attempted. Ngan, Wei, and Yen offer examples of orthodontic treatment in the primary dentition.[1] They suggest that functional problems which may interfere with normal growth and development can be resolved. Some of the more common types of treatment that can be performed for the primary dentition include correction of transverse discrepancies such as posterior crossbites; anteroposterior relationships of the maxilla and mandible, such as anterior crossbites (Fig. 27-59); and vertical discrepancies such as deep overbites or open bites.

Correction of anterior dental and posterior dental and skeletal crossbite was covered earlier in this chapter. Correction of class II skeletal relationships are typically well managed in the mixed dentition. However, class III skeletal relationships can and should be addressed in the primary dentition, particularly if they are the result of anterior displacement of the mandible or a retracted maxilla. A protraction headgear attached to an intraoral appliance by heavy elastics is the preferred method of treatment. The intraoral appliance is usually a fixed, bonded acrylic or cemented, banded expansion device. However, expansion is more helpful in correcting transverse width and space deficiencies than in increasing the ability to protract the maxilla. The protraction headgear is typically worn 4 to 9 months full time, until the desired overcorrection of the skeletal relationship is achieved. It is then worn only at night for an additional 6 to 12 months to retain the correction. Although correction of class III relationships caused by skeletal overgrowth of the mandible may be attempted, the results can be variable and may require retreatment as the lower jaw outgrows the correction. This is especially true if there is a strong genetic component to the skeletal conformation.

Vertical growth abnormalities can also be successfully managed in the primary dentition in selected cases. Patients with deep overbites and a horizontal growth pattern (low mandibular plane angle) may benefit from use of a removable anterior bite plane to allow supraeruption of the posterior dentition. If needed, finger springs can be added to flare upright maxillary incisors. Patients with open bites and a vertical growth pattern (high mandibular plane angle) may benefit from use of a removable posterior bite plane to intrude the posterior dentition. As stated earlier in this chapter, assessments of the patient's nasopharyngeal tissue and postural relationships are helpful.

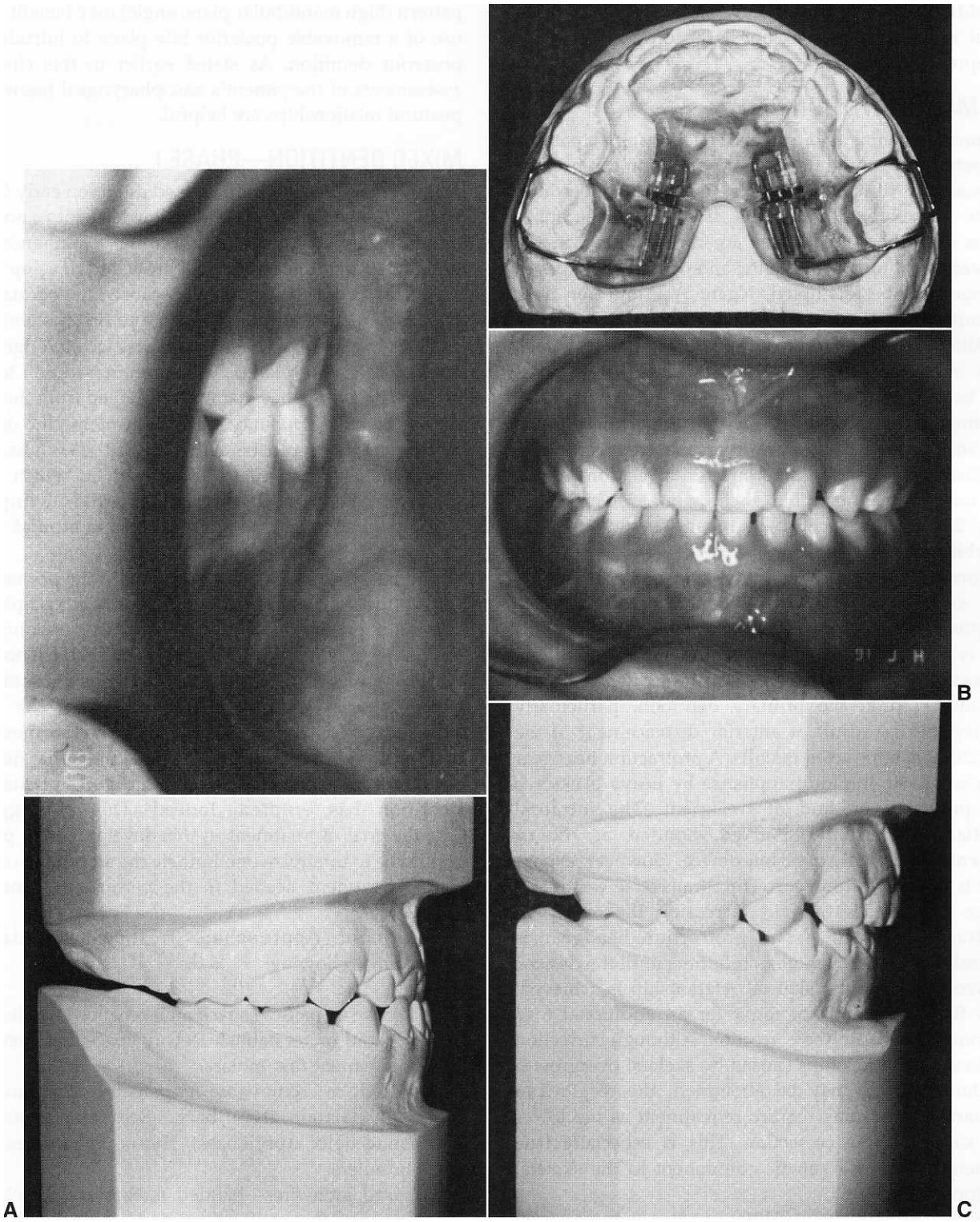
MIXED DENTITION-PHASE I

As in the primary dentition, mixed dentition early intervention is designed to put the patient back into a normal occlusal and orthopedic relationship to enhance the probability that the patient will grow and develop with a normal orofacial complex. Because the permanent maxillary and mandibular incisors and first molars have erupted in the mixed dentition phase, fixed, edgewise appliances can be placed. Therefore, in addition to the orthopedic corrections that may be gained from therapy with functional appliances or headgear, specific dental tooth corrections can be accomplished. This phase of treatment lasts from 9 to 18 months, at which time the patient is placed in a rest phase until full eruption of the permanent dentition is achieved at around 12 to 14 years of age.

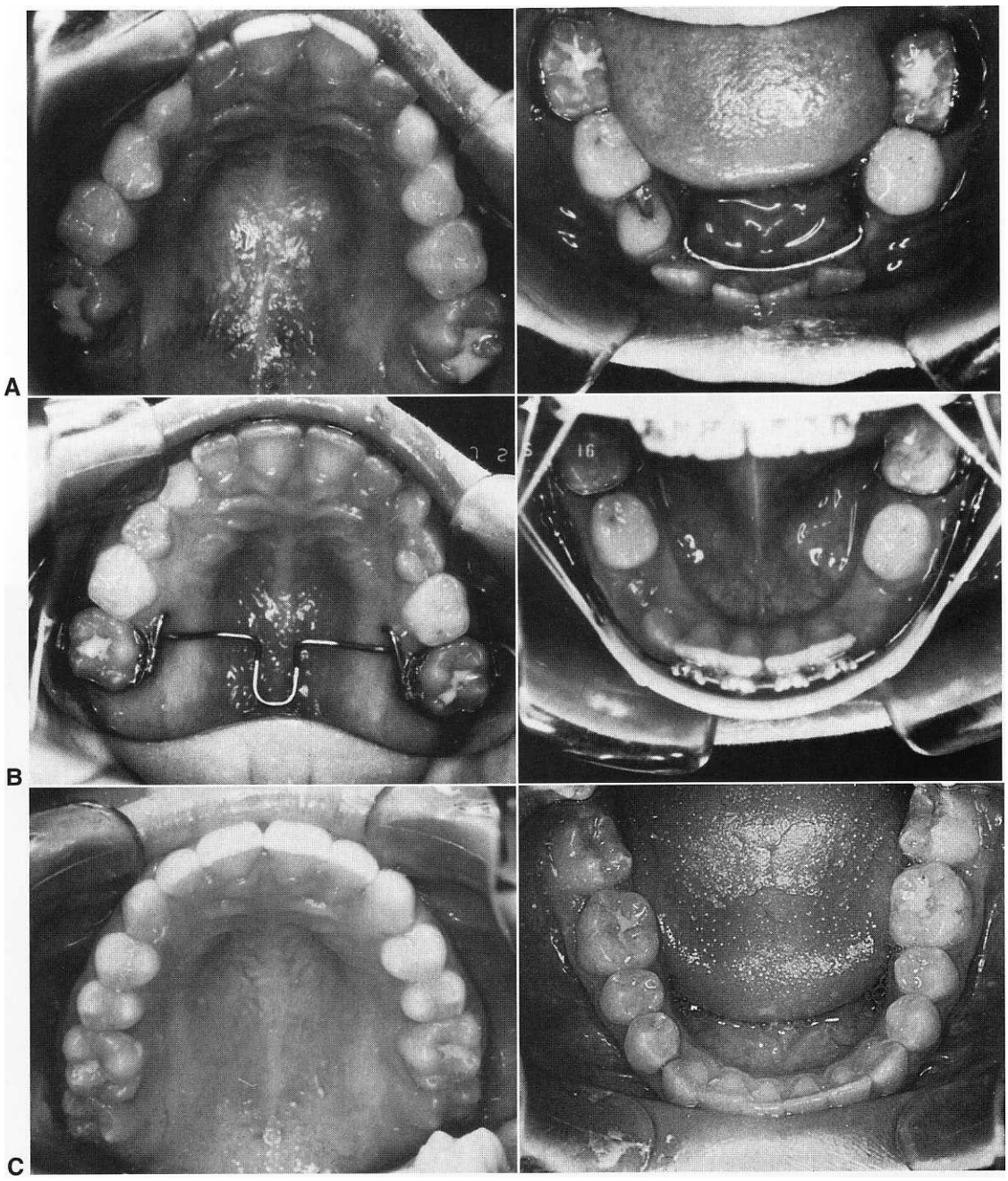
The purpose of this early treatment in the primary or mixed dentition phase is not necessarily to completely prevent the need for full, fixed, edgewise appliances in the permanent dentition. The goal is to establish normal vertical, transverse, and sagittal relationships of the dentition and the skeletal components of the face. However, it is possible to establish such a harmonious relationship of the dental and skeletal units that further treatment is unnecessary once the full, permanent dentition has erupted. Indeed, Dugoni suggests that the goal of treatment in this developmental phase should be to minimize or eliminate the amount of ortho dontic treatment needed in the permanent dentition (Fig. 27-60)[8]

Treatment Approaches. Treatment approaches include the following:

1. Active or passive space maintenance—appliances to hold or reestablish leeway space and derotate permanent first molars.
2. Fixed or removable expansion appliances—transpalatal arch bars, Schwarz retainers, quad-helix appliances, Hyrax appliances, lip bumpers.
3. Fixed archwires—banded molars and bracketed incisors (as well as primary dentition buccal segments if desired) for "2 x 4" mechanics, utility archwires, and open coil springs. These allow for molar derotation and uprighting, incisor leveling, alignment, and intrusion.
4. Headgear—cervical, high-pull, combination, or protraction (Fig. 27-61) depending on the vertical and sagittal facial growth pattern.



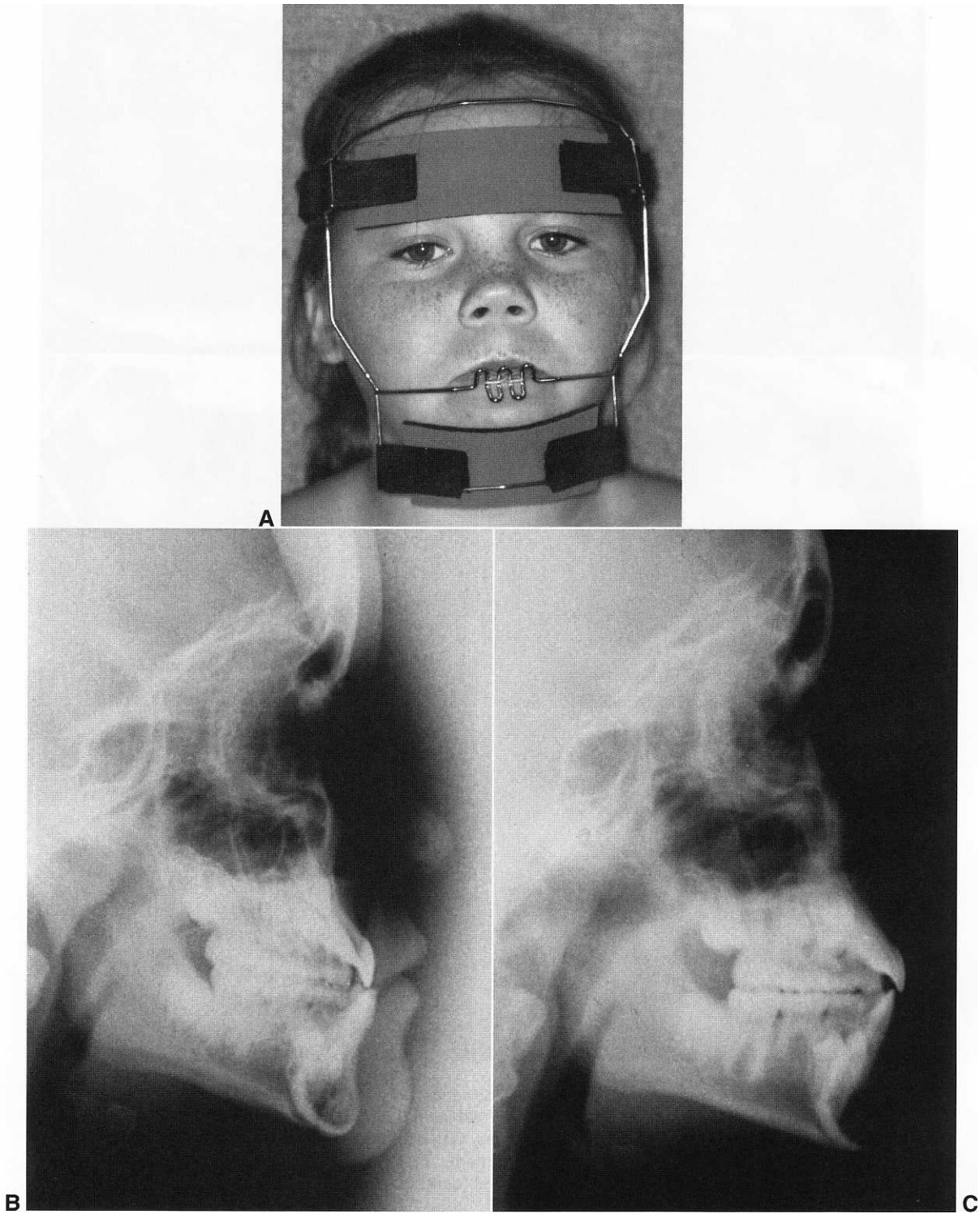
[I c 27-59. Primary dentition anterior crossbite with anterior functional shift of mandible. **A,** Pretreatment clinical (top) and model (bottom) view. **B,** Anterior push sagittal appliance designed to flare incisors. C, Posttreatment clinical (top) and model (bottom) view.



[I G **27-60.** Patient treated with phase I orthodontics only. **A,** Pretreatment occlusal views; note tapered maxillary arch and arch-length insufficiency in the mandibular arch. **B,** Phase I treatment with fixed "2 4" edgewise appliances, maxillary transpalatal arch bar, and mandibular lip bumper; note arch-length development, particularly in the mandibular arch. C, Posttreatment occlusal views in permanent dentition without additional full edgewise bracketing.

5. Functional appliances—Frankel appliances (Fig. 27-62), Bionaters, Herbst appliances, anterior and posterior bite planes. The type of functional appliance and its design vary significantly depending on the facial growth pattern.

Of course, the patient's orthodontic diagnosis will dictate which combination of the aforementioned treatment modalities will be necessary. One pervasive philosophy in early orthodontic treatment is that the simplest biomechanical approach necessary to achieve the desired treatment outcome is the best (Fig. 27-63).



fl G. **27-61. A,** Patient with protraction headgear in place. **B,** Pretreatment lateral head plate. C, Posttreatment lateral head plate.

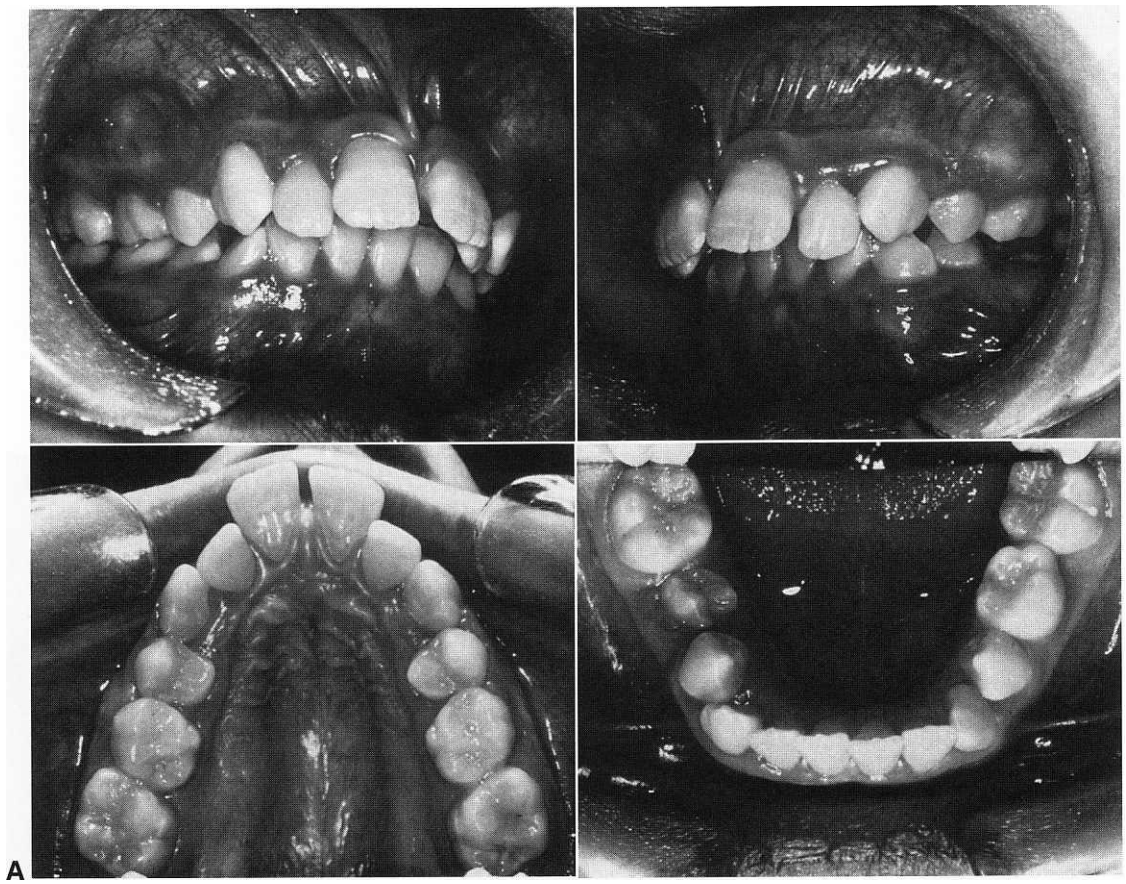


FIG 27-62. Dentition of patient treated with Frankel appliance. **A,** Pretreatment intraoral views.

Continued

In many cases of mixed dentition malocclusion, this routinely means the following:

Fixed 2 x 4 therapy to correct dental alignment and intrusion problems
Mandibular lingual arch or lip bumper treatment to facilitate arch development
Headgear therapy to address skeletal and dental vertical and sagittal problems

Often in the mixed dentition, depending on the individual patient's diagnostic assessment, significant arch development can be realized as follows:

Arch width expansion	2 mm
Leeway space maintenance or regaining	4 mm
Uprighting, tipback, or derotation of first permanent molars	2 mm
Flaring of incisors	2 mm
TOTAL	10 mm

Several different methods have been advocated for retention of the orthodontic and orthopedic effect achieved after mixed dentition treatment and until eruption of the remaining permanent teeth *(rest phase)*. They include maxillary and mandibular fixed and/or removable bilateral space maintainers (such as an upper Hawley and a lower lingual arch), custom or stock eruption guidance appliances (such as a tooth positioner), and segmented archwires with the bands and brackets remaining attached to the teeth. In patients with corrected dentofacial orthopedic problems, it is useful to continue, at night only, the use of headgear or functional appliances during this rest phase to maintain the orthopedic change.

EARLY PERMANENT DENTITION-PHASE II

Phase II of comprehensive orthodontic care again includes both orthopedic and orthodontic components. Orthopedic components might involve use of headgear or functional appliances. The orthodontic component includes the use of full, fixed, edgewise orthodontic appliances to establish as near-perfect interdental relationships as possible.

If a patient has undergone an earlier phase of comprehensive orthodontic treatment, this second phase of treatment might take as little as 12 to 18 months and

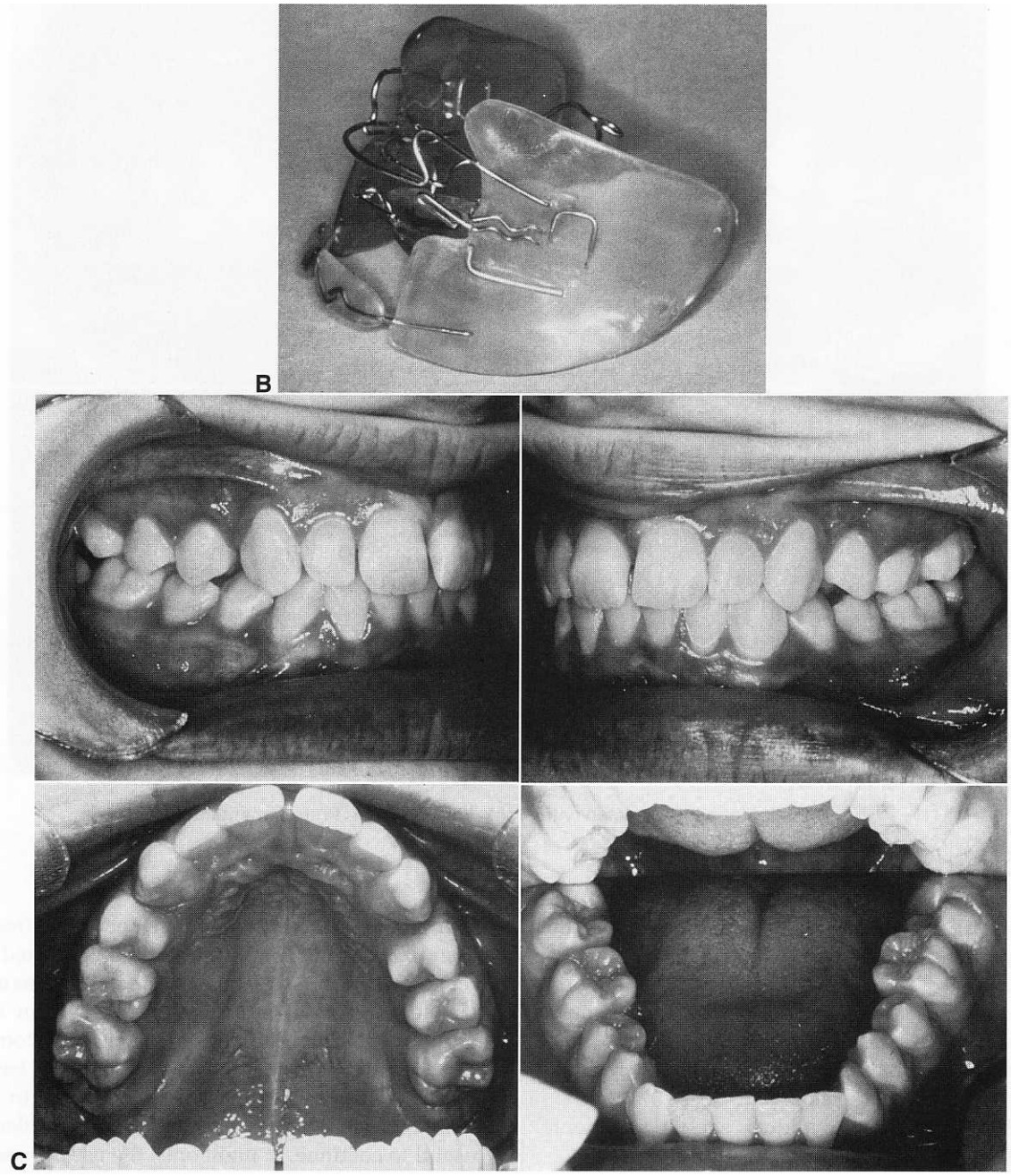
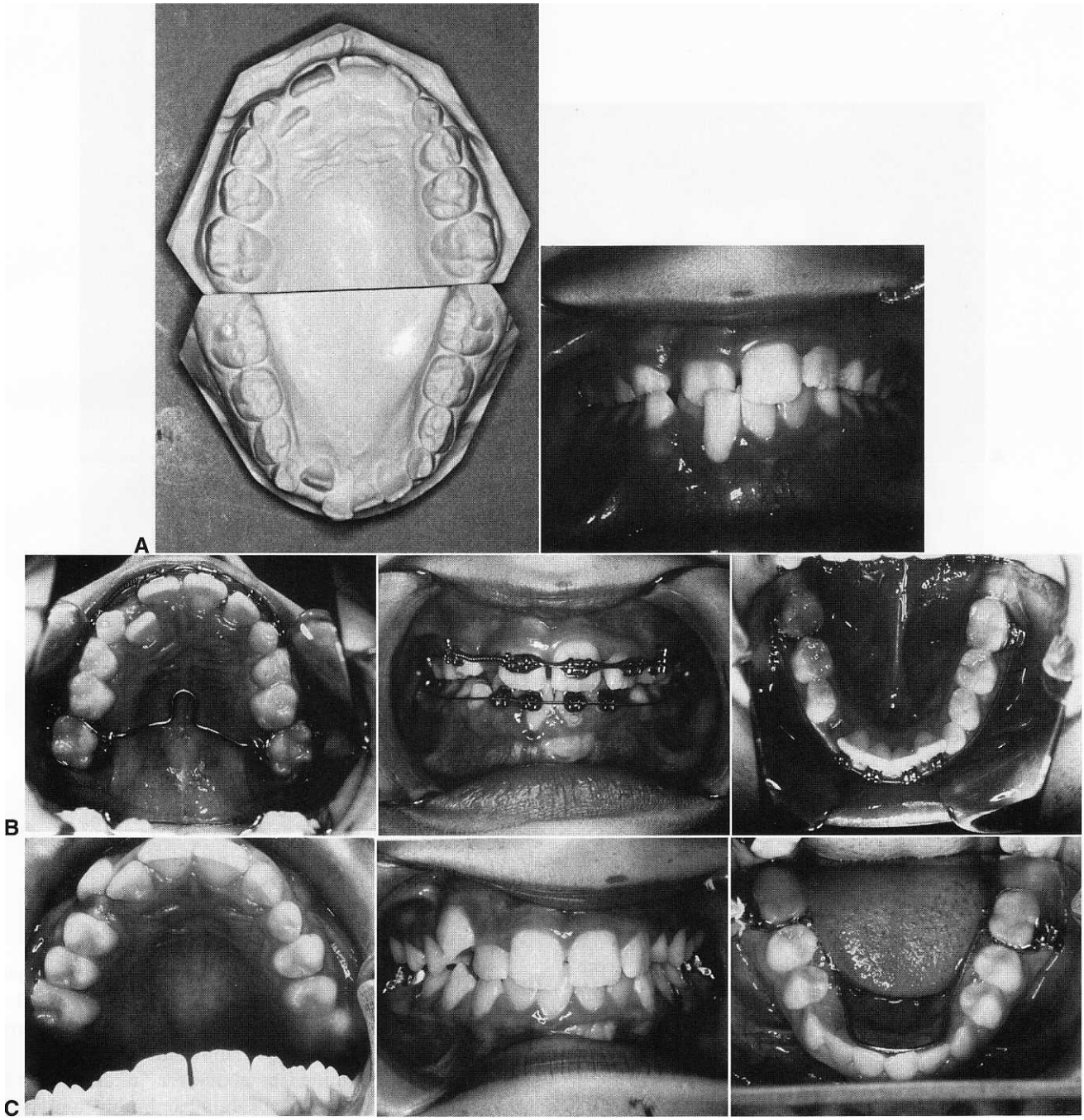


FIG. 27-62, contd. **B,** Frankel appliance. C, Rest phase retention with Frankel appliance worn only at night before phase II treatment.

hopefully will involve little, if any, orthopedic components. If this is the start of comprehensive care, however, more time will be needed (Fig. 27-64). The ultimate goal of all orthodontic treatment is to establish a stable esthetic and functional result. The specific dentition goals include the six keys to normal occlusion advocated by Andrews: proper molar relationships, proper crown angulation and inclination, no rotations, tight contacts, and a flat occlusal plane.[12]

Treatment after a mixed dentition phase includes the following:

1. Space consolidation
2. Sagittal arch coordination (class II or III elastic wear)
3. Root parallelism
4. Finishing and detailing: first-, second-, and third-order archwire bends as necessary



[I B 27-63]. Typical phase I treatment. **A,** Pretreatment models and intraoral view. **B,** Phase I fixed edgewise therapy. C, Rest phase retention with Hawley retainer and lower lingual arch before phase II treatment.

5. Buccal or anterior segment elastic wear to establish maximum interdigitation between arches
6. Orthopedics as necessary

Certainly most of this treatment is accomplished with fixed, edgewise appliances. However, in selected patients, phase II treatment will be limited in nature, determined to be unnecessary by the dental practitioner, or unwanted by the patient and/or parent. Limited treatment can involve tooth positioners or segmented arch therapy in which only the teeth or arch segments needing attention are addressed. Occasionally the practitioner may be so satisfied with the final orthodontic

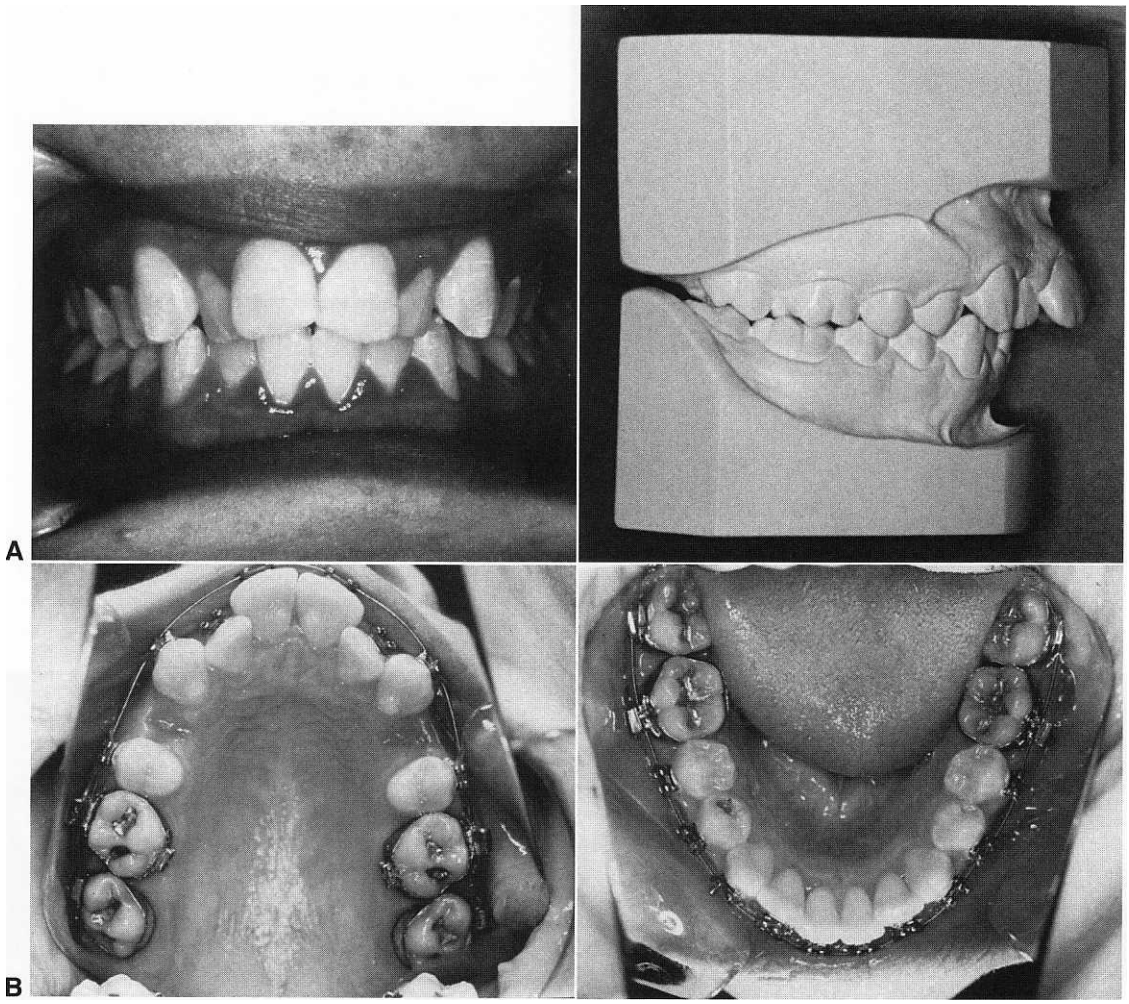


FIG **27-64.** Full edgewise orthodontic treatment in the early permanent dentition.
A, Pretreatment intraoral and model views; note overjet and full cusp class II occlusion.
B, Intraoral views of uni-arch extraction treatment.

result after phase I that no further treatment is recommended. However, this is fairly uncommon. A more likely occurrence is that the patient and/or parents decide that they are satisfied with the result, even though some finishing details are needed. To avoid this event, it is necessary to stress to parents before initiating phase I treatment that most children clearly will benefit from a final period of full orthodontic appliance use (phase II). It is particularly important to allow for final tooth positioning of the permanent second molars.

Retention following this period of treatment should involve the philosophy of "retention for a lifetime." There are many different types of retention schemes; however, the following retention schedule seems to provide for stable results:

1. Maxillary and mandibular Hawley retainers are worn 24 hours a day for 4 months, followed by 8 months of wear at night only.
2. After 1 year, a reassessment is made to see if wear can be reduced to 1 or 2 nights per week, with the patient increasing wear as needed if the retainers become tight from tooth movement.
3. Wear is continued as above for "retention for a lifetime" or, at an absolute minimum, until age 21 years, at which time the craniofacial growth rate has been reduced to that of an adult.

The provision of a backup set of thin acrylic overlay retainers is a very useful way of inexpensively giving the patient an emergency set of retainers in case the Hawley retainers are lost or damaged. As an added benefit, they can be used for home bleaching of the teeth by those patients who are interested. Fixed retainers may be necessary in special circumstances and can be fabricated from 0.0175-inch braided wire, heat-treated to be dead soft. A lower canine-to-canine fixed, bonded

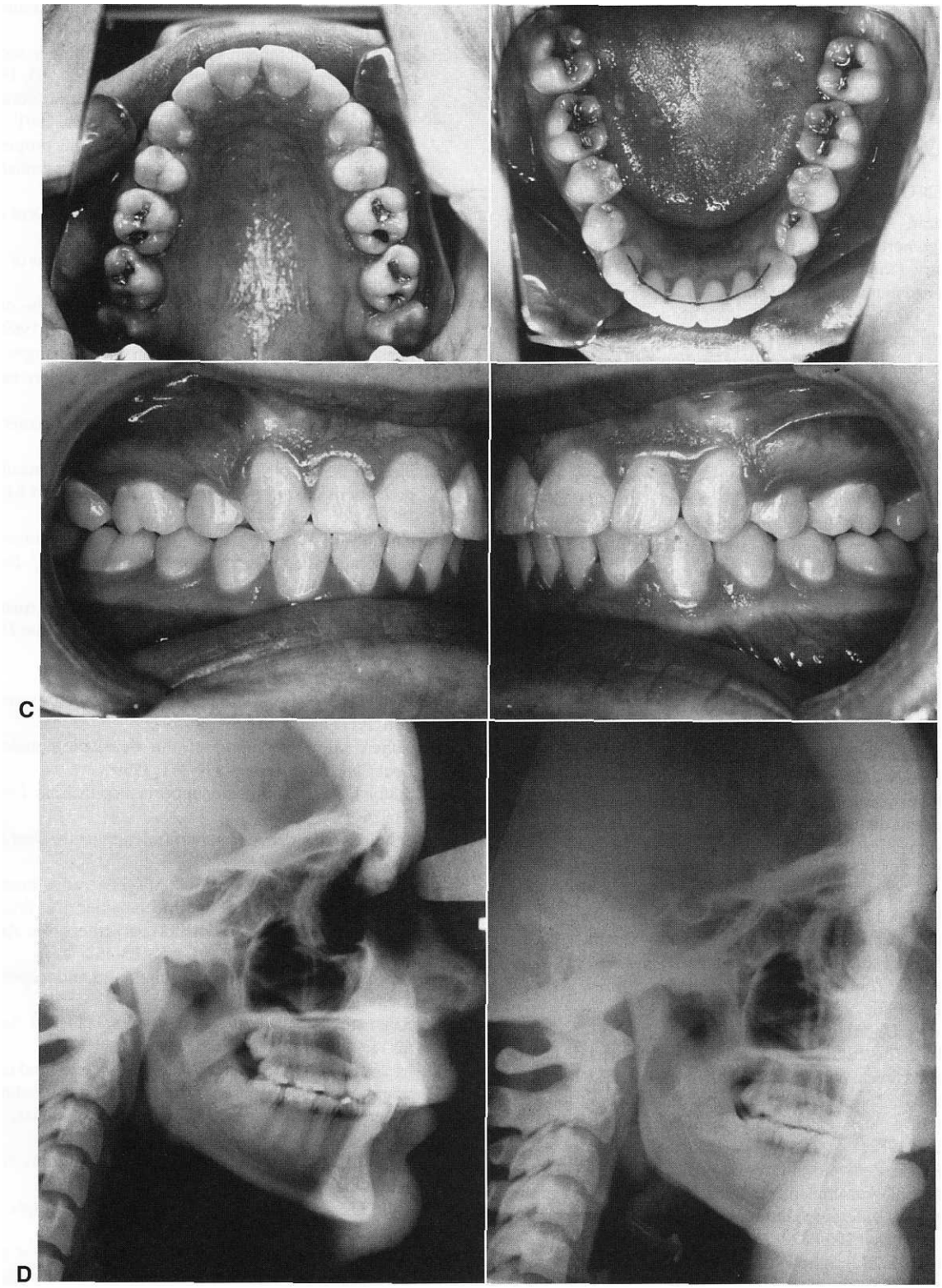


FIG **27-64, Contd. C,** Intraoral posttreatment occlusal and lateral views. D, Pretreatment and posttreatment lateral head plates; note overjet and profile improvement even though molars were left in class II position.

wire can be helpful for severe rotations and an upper lateral-to-lateral fixed, bonded wire is useful to prevent space opening in patients with initial diastemas. Unfortunately, no perfect retention scheme has been developed yet, and therefore patients must realize that periodic observation is warranted.

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CHAPTER 28

Multidisciplinary Team Approach to Cleft Lip and Palate Management

JAMES E. JONES
ALAN MICHAEL SADOVE
JEFFREY A. DEAN
DONALD V. HUEBENER

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CHAPTER OUTLINE

CLASSIFICATION OF CLEFT LIP AND PALATE
MULTIDISCIPLINARY CLEFT LIP AND PALATE TEAM
GENERAL RESPONSIBILITIES OF TEAM MEMBERS
Dental Specialties
Medical and Allied Health Specialties
MULTIDISCIPLINARY SEQUENCING OF TREATMENT IN CLEFTS
Stage I (Maxillary Orthopedic Stage: Birth to 18 Months)
Impression technique and obturator construction
Clinical management of initial obturator therapy (birth *to 3 months)*
Premaxillary orthopedics (birth to 4 or *5* months)
Appliance construction *for premaxillary retraction*
Airway obstruction
Cheiloplasty
Maxillary orthopedics *(3* to 9 months)
Bone grafting of *alveolar cleft* defects
Primary alveolar cleft bone *grafting*
Palatoplasty
Stage II (Primary Dentition Stage: 18 Months to 5 Years of Age)
Stage III (Late Primary or Mixed Dentition Stage: 6 to 10 or 11 Years of Age)
Secondary *alveolar cleft bone graft* Effect *of facial* esthetics on *self-concept* Stage IV (Permanent Dentition Stage: 12 to 18 Years of Age)
Cosmetic surgery

Cleft lip and palate, the most common of the craniofacial anomalies, are severe congenital anomalies that have an incidence of 0.28 to 3.74 per 1000 live births globally. In the United States, cleft lip and palate occur in approximately 1 in 1000 newborns. The incidence varies widely among races. Cleft lip and palate occur in about 1 in 800 white newborns, 1 in 2000 black newborns, and 1 in 500 Japanese or Navaho Indian newborns. Isolated cleft lip and palate occur in about 1 in 2000 newborns and demonstrates less racial variation. Cleft lip and palate together account for approximately 50% of all cases, whereas isolated cleft lip and isolated cleft palate each occur in about 25% of cases. Many of these congenital anomalies appear to be genetically determined, although the majority are of unknown cause or are attributable to teratogenic influences (see Chapter 6).

CLASSIFICATION OF CLEFT LIP AND PALATE

There is a tendency to conceptualize cleft lip and palate as a homogenous anomaly. If that were true, a treatment plan that would be applicable to all cases could be formulated. However, the reality is that children born with clefts vary widely in their clinical presentations (Fig. 28-1).

To standardize reporting of cleft lip and palate, the Nomenclature Committee of the American Association of Cleft Palate Rehabilitation devised a classification system that later was adopted by the Cleft Palate Association. The complexity of this system, however, has made its acceptance less than overwhelming. Veau proposed the most frequently used system.' He classified clefts of the lip as follows:

- Class I—a unilateral notching of the vermilion not extending into the lip
- Class II—a unilateral notching of the vermilion border, with the cleft extending into the lip but not including the floor of the nose
- Class III—a unilateral clefting of the vermilion border of the lip extending into the floor of the nose
- Class IV—any bilateral clefting of the lip, whether it be incomplete notching or complete clefting

Veau divided palatal clefts into four classes as follows (Fig. 28-2):

- Class I—involves only the soft palate
- Class II—involves the soft and hard palates but not the alveolar process
- Class III—involves both soft and hard palates and the alveolar process on one side of the premaxillary area
- Class IV—involves both soft and hard palates and continues through the alveolus on both sides of the premaxilla, leaving it free and often mobile

Veau did not include submucous clefts of the palate in his classification system. Submucous clefts may frequently be diagnosed by the following physical findings: bifid uvula, palpable notching at the posterior portion of the hard palate, and a "zona pellucida" (thin, translucent membrane). Submucous clefts of the palate may be associated with an incomplete velopharyngeal mechanism or eustachian tube dysfunction.

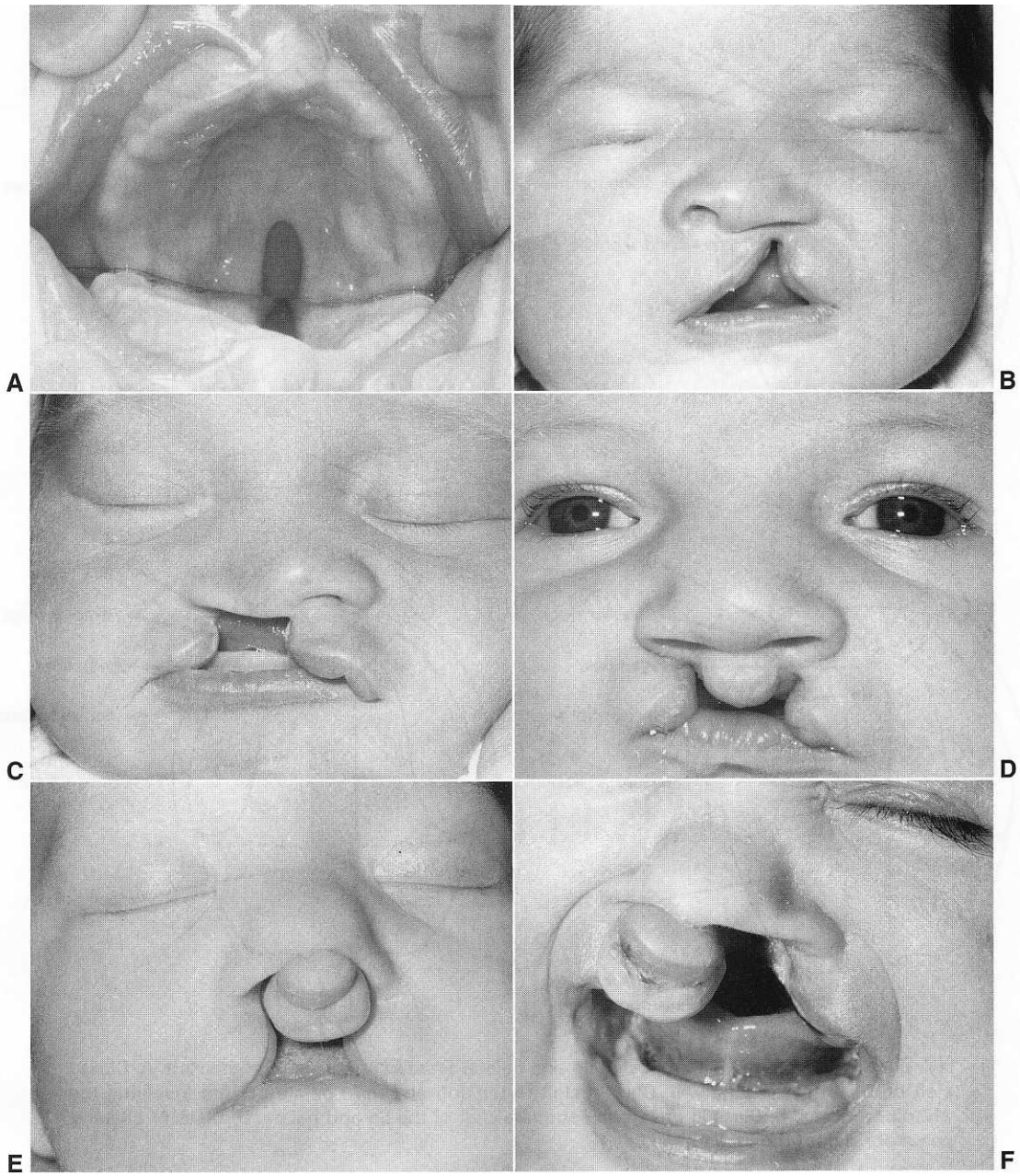
MULTIDISCIPLINARY CLEFT LIP AND PALATE TEAM

Children born with cleft lips and palates have many problems that need to be solved for successful habilita tion. The complexity of these problems requires that numerous health care practitioners cooperate in providing the specialized knowledge and skills necessary to ensure comprehensive care. The cleft palate team concept has evolved from that need.

In an effort to address the many treatment regimes and different care protocols, the American Cleft Palate–Craniofacial Association (http://www.cleftpalate-craniofacial.org) convened a consensus conference on recommended practices for the care of patients with craniofacial anomalies. This conference produced the document "Parameters for Evaluation and Treatment of Patients with Cleft Lip/Palate or other Craniofacial Anomalies."' This serves as a guide for implementing the multidisciplinary approach to cleft and craniofacial care and is used by teams in the United States and Canada.

Since optimal care is best achieved by multiple types of clinical expertise, the teams may be composed of individuals in (1) the dental specialties (orthodontics, oral surgery, pediatric dentistry, and prosthodontics), (2) the medical specialties (genetics, otolaryngology, pediatrics, plastic surgery, and psychiatry), and (3) allied health care fields (audiology, nursing, psychology, social work, and speech pathology).

These care providers assess the patient's medical status and general development, dental development, facial esthetics, psychologic well-being, hearing, and speech development (Fig. 28-3). Team members must communicate effectively among themselves, with the child and parents, and with the primary care physician and dentist. Individuals on the team must respect one another's opinions and be flexible in planning and carrying out therapy. Periodic evaluation is necessary to assess the effect of previous therapy and to determine



[I **28-1.** Various clinical presentations of cleft lip and cleft palate. (See text for descriptions of each specific type.) A, Isolated cleft palate (class II). **B,** Unilateral cleft of the lip (class II). C, Unilateral complete cleft of the lip and palate (class III). D, Bilateral incomplete cleft of the lip (class IV). **E,** Bilateral complete cleft of the lip and palate (class IV). F, Bilateral complete cleft of the lip and palate with a laterally displaced premaxillary segment (class IV).

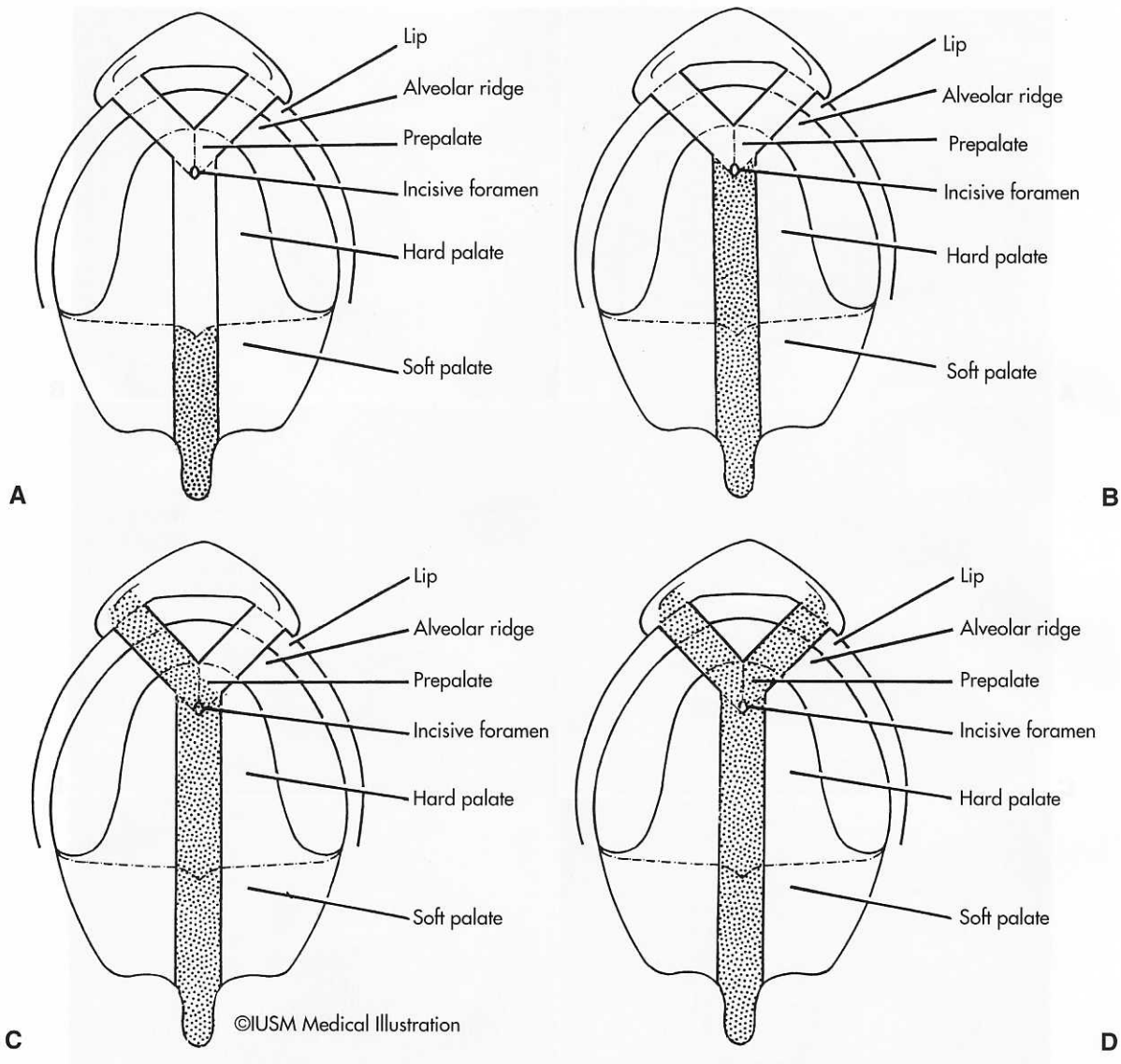
whether an alternative approach may be necessary. A team conference immediately after patient examination is a desirable way to discuss current problems and plan timely therapy.

Whitehouse describes the clinical team as a "close, cooperative, democratic, multiprofessional union devoted to a common purpose—the best treatment of the fundamental needs of the patient."[3]

GENERAL RESPONSIBILITIES OF TEAM MEMBERS

DENTAL SPECIALTIES

The pediatric dentist is responsible for the overall dental care of the patient. Numerous dental anomalies and malocclusions occur with a cleft lip or palate. These may be attributed to the congenital clefting itself or may be secondary to the surgical correction of the primary

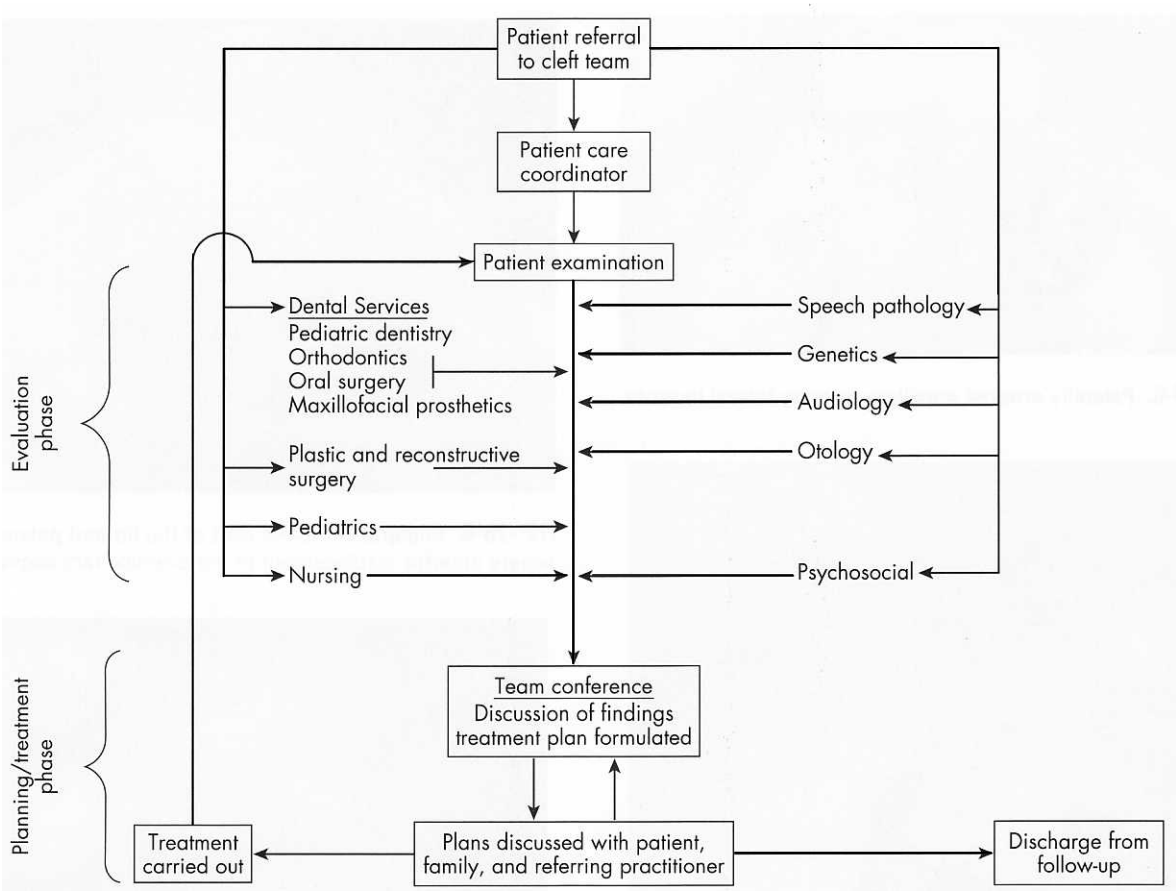


[I G **28-2.** Veau's classification of cleft lip and palate. Shaded area, Extent of the cleft. **A,** Class I cleft of the soft palate only. **B,** Class II cleft of the soft and hard palate but not involving the alveolar process. C, Class III unilateral complete cleft of the lip and palate. **D,** Class IV bilateral complete cleft of the lip and palate.

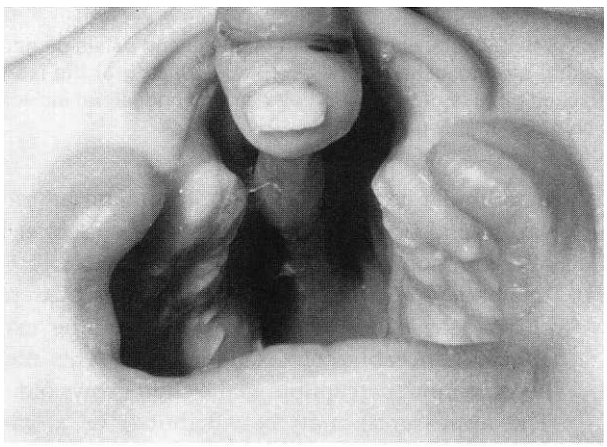
defects. A high correlation is found between the number and severity of dental problems and the type and severity of the cleft.

The pediatric dentist should discuss with the patient and parents the traditional dental problems associated with clefting. Any one, or several, of the following conditions may occur with a significantly greater frequency than in the general population:

1. Natal, or neonatal, teeth are usually maxillary central incisors observed in patients with a complete unilateral or bilateral cleft palate (Fig. 28-4).
2. There is a high incidence of congenitally absent teeth, especially the primary or permanent lateral incisor area adjacent to the alveolar cleft. There is also an increased incidence of congenitally missing premolars.
3. There is a significant increase in the frequency of supernumerary teeth (Fig. 28-5), which are often seen in those with complete unilateral or bilateral clefts. Again, the primary lateral incisors are most frequently absent. Occasionally, there can be supernumerary teeth and congenitally missing teeth in patients with cleft lip only with or without notching of the alveolus.
4. It is common to find ectopic primary lateral incisors located palatally, adjacent to, or within the cleft site (Fig. 28-6). In the permanent dentition,



fl G **28-3.** Diagram of patient interaction with members of the cleft lip and palate team.



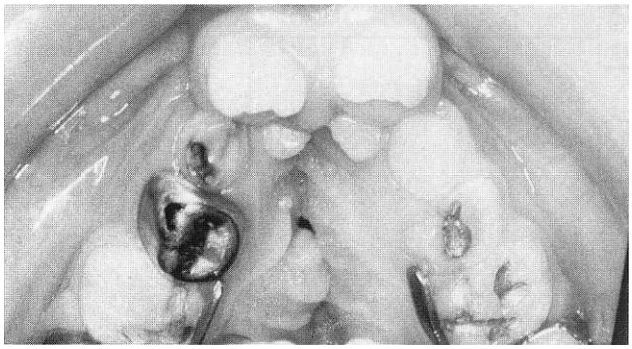
f **1 G 28-4.** Neonatal maxillary central incisor in a newborn infant with a bilateral complete cleft of the lip and palate.



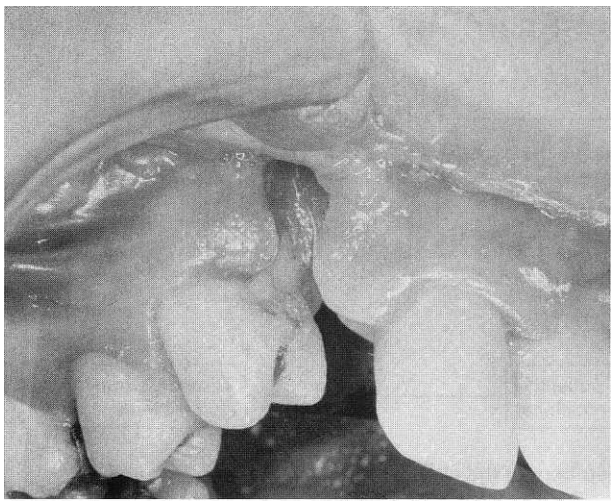
[H G **28-5.** Maxillary occlusal radiograph demonstrating supernumerary maxillary central incisors.

canines on the side of the complete alveolar cleft may erupt palatally into the cleft.

5. Various anomalies of tooth morphology are frequently seen in association with complete unilateral and bilateral clefts of the palate. These include enamel hypoplasia, microdontia or macrodontia, fused teeth, and aberrations in crown shape. The teeth most often affected are the primary and permanent maxillary incisors.
6. Permanent teeth that erupt adjacent to a cleft of the alveolar ridge usually have a deficiency of supporting alveolar bone about the root surfaces



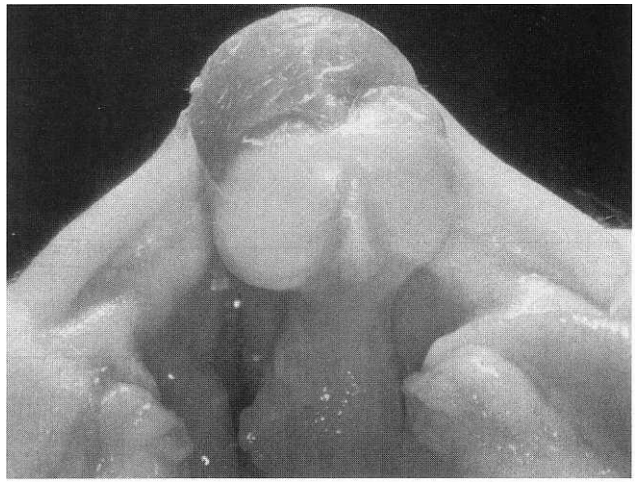
f I 1 28-6. Palatally erupted maxillary primary lateral incisors.



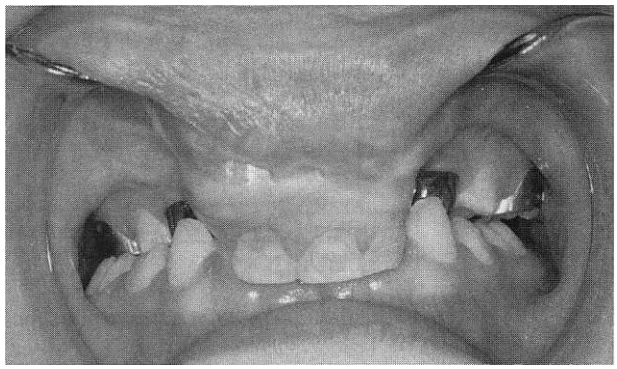
f 16 28-7. Maxillary right first permanent premolar adjacent to cleft defect. Notice the deficiency of mesial supporting alveolar bone.

(Fig. 28-7). These teeth are susceptible to premature loss. A decrease in alveolar bone support may be accentuated when periodontal disease is present or when orthodontic appliance therapy is used indiscriminately.

7. With great frequency, permanent central incisors adjacent to an alveolar cleft erupt in a rotated posi tion and with deviations of axial root inclination.
8. With a complete cleft of the palate and alveolus there is no longer a contiguous maxillary arch. External forces applied to the maxilla (e.g., by muscles of mastication or by the contraction of scar tissue after surgical repair of the cleft palate) can result in medial collapse of the posterior segments. A posterior crossbite may be observed unilaterally or bilaterally.
9. In an infant with a complete bilateral cleft of the lip and palate, the premaxilla is often protuberant and mobile (Fig. 28-8). There may be a greater than 100% overbite with subsequent stripping of the labial-attached gingiva overlying the



f I C 28-8. Bilateral complete cleft of the lip and palate with a severe anterior displacement of the premaxillary segment.



f I 1 28-9. Bilateral complete cleft of the lip and palate demonstrating a greater than 100% overbite. Stripping of the labial attached gingiva of the mandibular central and lateral incisors is common in this presentation.

mandibular incisors (Fig. 28-9). Traumatic anterior end-to-end occlusion, or an anterior crossbite, is also common.

10. In a patient with a complete unilateral or bilateral cleft of the palate, the lateral facial profile may appear noticeably convex (Fig. 28-10). This may become more perceptible as the child grows older. The appearance may be attributed to a true mandibular or pseudomandibular prognathism. In pseudomandibular prognathism, the maxilla is in spatial disharmony with the mandible. This may be caused by a retrognathic maxilla or an attenuation of the anteroposterior and vertical growth of the maxilla.

Parents are often so overwhelmed by other aspects of the cleft that they give dental care a low priority or even neglect it altogether. Preventive dental care is extremely important in these cases. The intact dental occlusion is



f I G. **28-10.** Lateral facial profile of an adolescent boy with a repaired bilateral complete cleft of the lip and palate. Maxillary hypoplasia, secondary to the cleft defect, often produces a greatly concave lateral facial profile.

the foundation around which future orthodontic therapy takes place. For this reason, optimum dental health is essential for total habilitation of the patient. Any compromise will lead to a less than optimal result. Routine prophylaxis and fluoride treatments are mandatory. Referral for preventive dental care should be made during the first year of life. Fluoride supplements, dentifrices, and rinses are indicated if the patient lives in a nonfluoridated community. The parents and patient should be instructed in proper dental hygiene techniques, especially around the defect. Close communication between the primary care dentist and the cleft team is important to ensure the continuity of care necessary during the extended treatment of such patients. Routine periodic reports from the cleft team should be forwarded to the child's primary care dentist, especially during orthodontic or surgical treatment. Pediatric dentists often are involved in the presurgical and postsurgical treatment phase of maxillary orthopedics. Both active and passive appliances are used to bring the cleft segments into a more ideal alignment and thereby promote a more favorable initial surgical outcome.

The orthodontist plays a key role in the diagnosis and treatment of a cleft condition by obtaining records necessary for diagnosis and treatment planning. These include cephalometric and panoramic radiographs, study models, and diagnostic photographs. Analysis of these records enables the orthodontist to describe and quantitate the facial skeleton and soft tissue deformities. Using expertise in the growth and development of the facial skeleton, this specialist can identify problem areas and, with some limitations, predict growth and development. Many team members depend on the orthodontist's analysis and quantitations of the cleft anomaly for treatment planning.

The orthodontist also provides comprehensive orthodontic care for patients. Most orthodontic care can be considered conventional, but for difficult dental configurations, innovation and imagination are required for treatment. If surgical treatment is indicated, the orthodontist works closely with the surgeon to plan the most appropriate procedure. Immediately postoperative function, esthetic result, and long-term stability are factors considered before surgery.

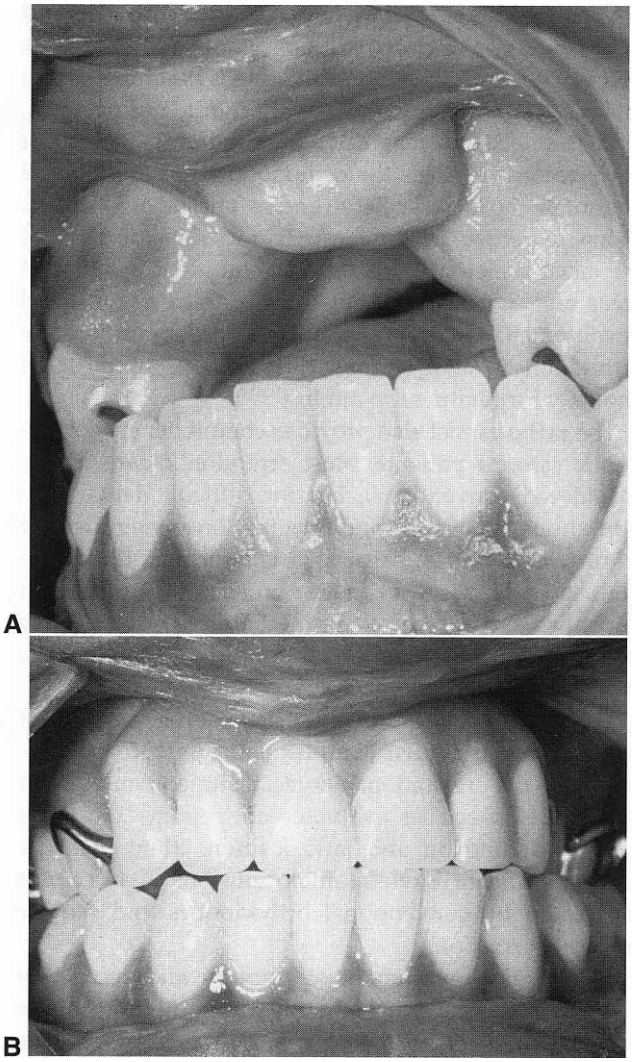
The ability to surgically alter skeletal relationships of the maxillomandibular complex is the basis for participation by the oral and maxillofacial surgeon on the cleft team. This specialist evaluates all patients for facial form and function and jaw position. Many patients have significant skeletal malocclusions that cannot be treated by conventional orthodontics and require surgical correction.

The surgical placement of primary and secondary alveolar cleft bone grafts is another important role of the oral and maxillofacial surgeon. These grafts aid in dental habilitation. The grafted bone supports the teeth adjacent to the cleft site and provides bone through which teeth may erupt. A detailed discussion of these grafts follows later in this chapter.

The maxillofacial prosthodontist replaces, restores, or rehabilitates orofacial structures that may be congenitally missing or malformed. Nonliving materials are used to restore and enhance form and anatomy. There is a special commitment to the oral cavity because this specialist fabricates prosthetic appliances to rehabilitate mastication, deglutition, speech, and oral esthetics.

Many patients with clefts have congenitally missing teeth or malformed teeth that may need to be removed. In these cases, masticatory function, speech, and orofacial esthetics are compromised, and successful habilitation dictates that these missing teeth be replaced to achieve as near normal a condition as possible (Fig. 28-11). The maxillofacial prosthodontist may do this with fixed or removable appliances or with a combination of the two.

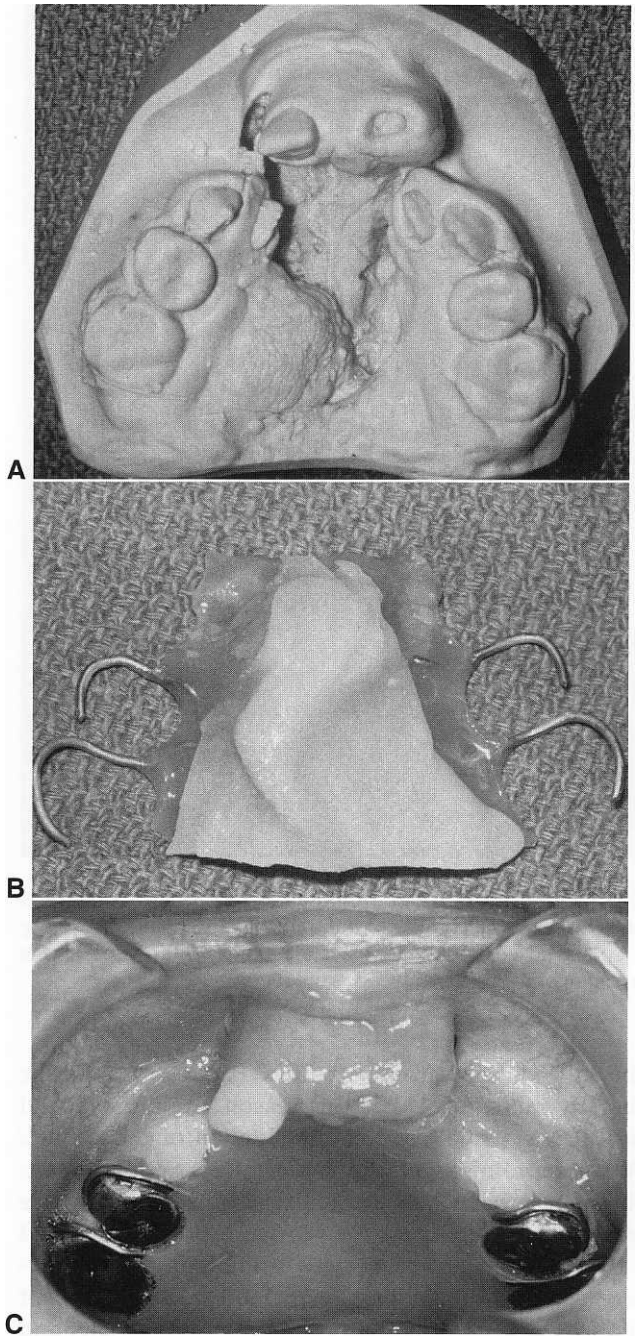
Occasionally, patients demonstrate aberrant speech patterns caused by failure of the soft palate to elevate



H **C 28-11. A,** Bilateral complete cleft of the lip and palate in which the maxillary six permanent anterior teeth have been removed. **B,** Removable prosthodontic appliance providing acceptable occlusal and esthetic results.

properly. In such cases, a palatal lift appliance is fabricated to aid the speech mechanism. In other cases the maxillofacial prosthodontist may fabricate a speech bulb prosthesis to aid or augment the velopharyngeal mechanism. In patients with considerable escape of air through persistent palatal fistulas, the fistulas can be obturated (Fig. 28-12).

MEDICAL AND ALLIED HEALTH SPECIALTIES The patient care coordinator arranges appointments, maintains patient records, and monitors the interaction of the patient and family with the various team members. The coordinator corresponds with health and school personnel near the patient's home to help provide continuity of care for the cleft patients within the community. The coordinator is the most convenient



1 **I G 28-12.** A, Diagnostic maxillary model of a bilateral complete cleft of the lip and palate. Notice the large patent oronasal fistula that resulted after several attempts to close the defect surgically. **B,** Maxillary prosthesis constructed for the patient to obturate the palatal defect. C, Palatal prosthesis in place. This closure provides a reduction in the amount of fluids and foods that enter the nasal cavity during eating. The closure also facilitates more normal speech production.

point of contact with team members for the patients, their families, and health care practitioners outside the medical center complex.

The pediatrician, often the patient's own pediatrician or family physician, is responsible for maintenance of the patient's overall health. This specialist performs complete physical evaluations and helps assess the patient's physiologic status. Close attention is also directed to growth status and other developmental milestones.

The medical geneticist examines the patient to find characteristics of syndromes associated with cleft lip and palate. Consideration is given to the genetic basis for the anomaly, and this information is related to the parents. Genetic counseling is a very important function of the geneticist. Parents are vitally interested in risk assessment relative to future offspring, and other family members who may be at risk are often counseled (see Chapter 6).

The role of the plastic and reconstructive surgeon usually begins with a determination of the timing and method of lip closure. With complete clefts, the plastic surgeon may next be responsible for obtaining bone grafts to be used in closing defects of the maxillary dental alveolus. This specialist is also involved in cleft palate repair.

An additional responsibility may be to conduct a nasopharyngoscopic examination of the speech mechanism. If a defect is identified, the plastic surgeon may perform a pharyngoplasty to improve velopharyngeal function. The final role is to correct internal or external cleft nasal deformities.

The social worker acts as the patient's advocate in many cases and aids in psychosocial assessment. This team member assists the family by making referrals to persons or agencies at the local, county, and state levels for guidance regarding financial resources for medical care. During hospitalization, the social worker provides supportive counseling and facilitates communication between the family and medical or hospital personnel. The focus is on helping the family cope with stress during and after surgery and on dealing with emotional factors involved in forming realistic expectations of surgical outcomes and in adapting to problems of body image.

The psychiatrist and psychologist evaluate the patient for strengths and weaknesses in cognitive, interpersonal, emotional, behavioral, and social development. Emphasis is placed on the patient's ability to cope with the emotional and physical stress created by the cleft defect. Consultation with the parents and school regarding educational or behavioral management occurs when indicated.

The speech pathologist functions essentially as a monitor of speech output. All speech sounds are analyzed to determine deviations from normal, and the cause of any deviation is evaluated. To the extent that anatomic variations are corrected, the speech pathologist offers therapeutic options to enhance maturation of speech or to achieve satisfactory compensation in motor production for optimal speech.

The audiologist performs tests to identify any hearing difficulties. When neither the child nor the parents recognize hearing problems, the result can be a delay in speech and language development and poor performance in school. When hearing tests or impedance measures are abnormal, a referral is made to an otolaryngologist for an ear examination. The otolaryngologist coordinates audiologic tests and any special studies that may be needed to evaluate middle ear structures. Any middle ear surgery that is to be done is performed by this member of the team. He or she also may perform a nasopharyngoscopic examination in coordination with the speech pathologist.

Nurses provide varying functions that are valuable to the cleft lip and palate team. They actively communicate with other disciplines in passing on information regarding the special needs of each child and family. Nurses emphasize total family involvement, not just treatment of the patient. They prepare patients and families for either outpatient or inpatient surgery and assist in the overall management process. Above all, they are extremely important in assessing the initial feeding issues and advising parents with ongoing nutritional concerns.

The presence of more complex craniofacial anomalies, such as Crouzon syndrome, Treacher Collins syndrome, or hemifacial microsomia, will require additional specialists on the team due to the increase in the deformity present. Some of these care providers include anesthesiologists, diagnostic medical imaging radiologists, neurologists, neurosurgeons, and ophthalmologists in addition to those cleft team providers already mentioned.

MULTIDISCIPLINARY SEQUENCING OF TREATMENT IN CLEFTS

The following discussion focuses on major treatment procedures performed by members of the cleft team. For convenience, treatment is divided into four stages, which generally correspond to stages in the child's dental development.

STAGE I (MAXILLARY ORTHOPEDIC STAGE: BIRTH TO 18 MONTHS)

Management of the patient with a cleft begins with immediate attention to the needs of the newborn. Feeding problems are often associated with cleft anomalies, which make it difficult for the infant to maintain adequate nutrition. These problems include insufficient

suction to pull milk from the nipple, excessive air intake during feeding (requiring several burpings), choking, nasal discharge, and excessive time required to take nourishment.

McNeil in the 1950s[4,5] and other authors since then have advocated various prosthetic appliances, both active and passive, for the treatment of infants born with unilateral and bilateral clefts of the lip and palate. One such prosthesis, an intraoral maxillary obturator, has proved beneficial by providing an artificial palate. The advantages of this prosthetic therapy include the following:

1. Provides a false palate against which the infant can suck, reduces the incidence of feeding difficulties in newborns, and helps maintain adequate nutrition
2. Provides maxillary cross-arch stability and prevents arch collapse after definitive cheiloplasty (surgical closure of the lip)
3. Provides maxillary orthopedic molding of the cleft segments into approximation before primary alveolar cleft bone grafting

In a recent study by Jones, maxillary obturators were constructed to facilitate feeding for 51 infants with unilateral or bilateral cleft lip and palate.[6] From birth, each infant had continuously experienced feeding difficulties before obturator therapy. After the infants had worn the obturator for at least 8 months, parents reported that they were more comfortable while feeding their infants and that nasal discharge was reduced. The time required for feeding and the difficulty experienced by the parents were also reduced. Of particular importance was the reported reduction of parental apprehension during feeding. All parents recommended the obturator for others who have infants with cleft lips and palates. It was also reported that the weights of the infants at 1, 3, and 6 months of age consistently remained at, or above, the 50th percentile compared to normative growth data. No fluctuation in weight was noted even after primary lip closure at about 3 months of age.

Not all clinicians who work with infants with cleft lips and palates advocate use of prosthetic feeding appliances. Some believe that such appliances are not effective in facilitating feeding. Pashayan and McNab recommend using a standard nipple that has been cross-cut.' This enlarged cut provides improved ejection of the milk into the infant's mouth with a minimum of effort. Although this recommendation is appropriate for infants born with isolated clefts of the palate, obturator construction is generally indicated for those born with complete clefts of the lip and palate. This is especially important in consideration of the maxillary orthopedic molding of the alveolar segments after surgical closure of the lip. In a recent survey, it was reported that approximately 32% of cleft teams in the United States and Canada use this prosthetic feeding appliance.

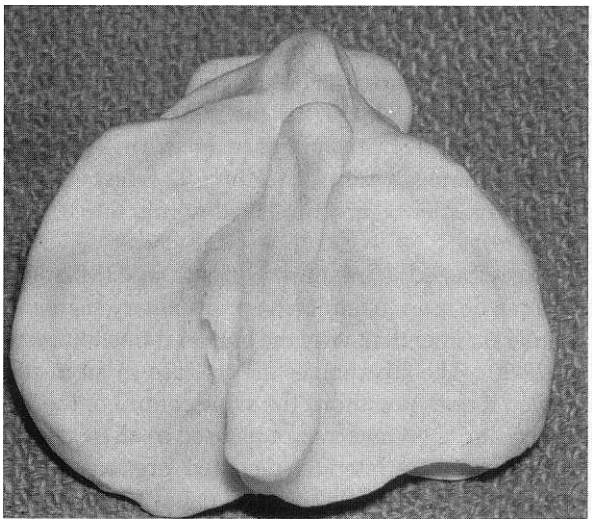
Impression Technique and Obturator **Construction.** An alginate impression of the infant's maxillary arch is made with a modified stock tray. Ideally, this is accomplished as soon after birth as possible. The infant is held upright during the impression process to prevent aspiration of excess material (Fig. 28-13). Appropriate emergency equipment, including forced oxygen, suction, and standard airway management equipment, should be available. The impression should exhibit good anatomic detail with coverage of the entire maxillary arch (Fig. 28-14). A stone model is then produced. The steps in obturator construction are as follows:

1. Block out excessive undercuts with modeling dough or wax. Modeling dough is preferred because it is easy to remove from the finished prosthesis.
2. Apply a tinfoil substitute over the entire surface of the maxillary model and let it dry.



[I fi **28-13.** Maxillary impression for obturator construction on a newborn with a cleft lip and palate. The infant is held in an upright position to prevent aspiration of excess material.

3. If necessary, place a dam of modeling dough on the back of the model to hold the resin in the palatal defect while curing.
4. Pour a mixture of soft, autopolymerizing acrylic resin into the cleft to the level of the palate. This will provide retention for the prosthesis by gently contouring into the available undercuts (Fig. 28-15).
5. Place the model in a warm, moist environment to cure for 20 minutes.

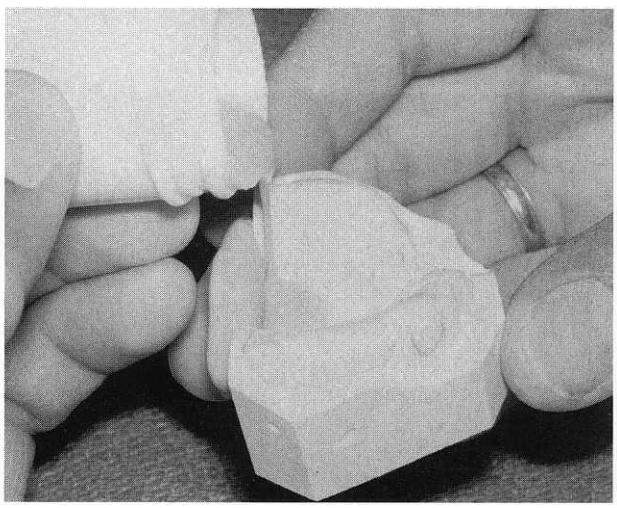


f **I G . 28-14.** Final impression of the infant's maxillary arch. Notice the extension of the material into the cleft defect, as well as the roll produced in the buccal fold. Attention to such detail ensures an excellent reproduction of the intraoral architecture for obturator construction.

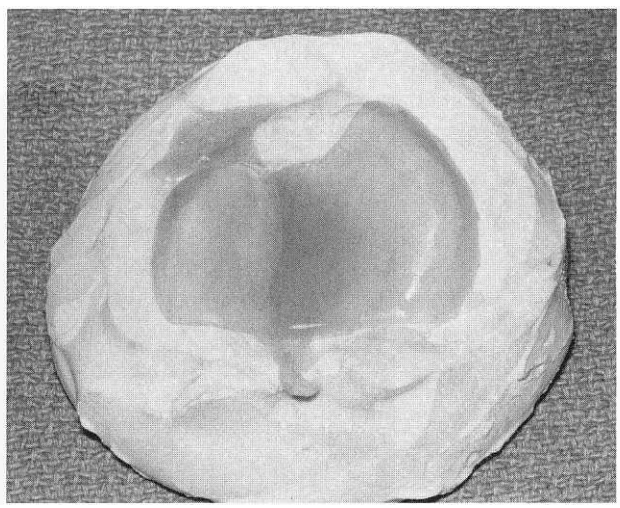
6. Add autopolymerizing acrylic resin to the palate using a "salt and pepper" method, making sure the acrylic resin extends well into the mucobuccal fold area (Fig. 28-16).
7. Remove the appliance from the model, and rinse the wax and modeling dough off with hot water. Then trim and polish the appliance.

Clinical Management of Initial Obturator Therapy (Birth to 3 Months). The appliance is positioned in the infant's mouth (Fig. 28-17). Areas of excessive pressure on any intraoral tissues by the acrylic resin are identified by observation and then reduced. Care is taken to keep the acrylic resin from impinging on muscle attachments or extending to the depth of the buccal vestibule. Parents are instructed in placement and removal of the appliance and its daily cleaning. Infants are usually seen for adjustments 2 days after appliance delivery. Monthly observations are then scheduled. In most cases, this appliance will serve until the time of initial lip closure at approximately 3 months of age. The major advantage of obturator use during this stage is to enhance the child's ability to obtain nourishment.

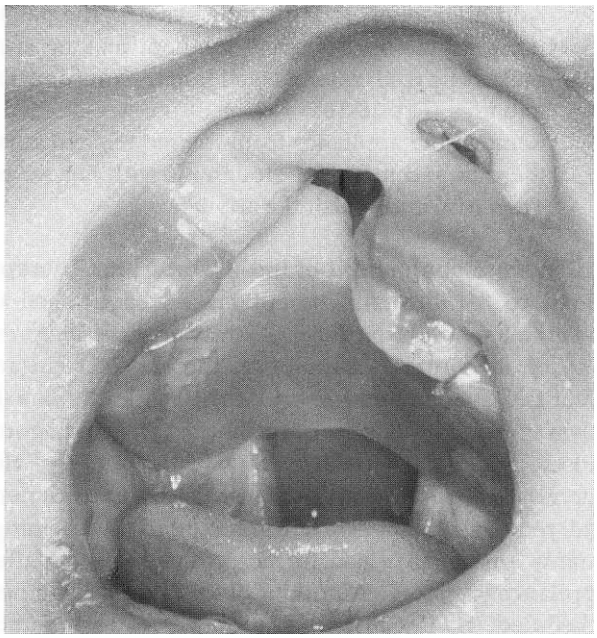
Premaxillary Orthopedics (Birth to 4 or 5 Months). In some cases of bilateral cleft lip and palate, the infant has a premaxillary segment positioned severely anterior to the maxillary arch segments or deviated laterally to one side of the cleft defect (Fig. 28-18). This presents a difficult clinical challenge for the surgeon before surgical closure of the lip. If lip surgery is undertaken with the premaxilla in such an abnormal position, the



f **G 28-15.** Application of soft, autopolymerizing resin into the cleft to the level of the palate. This material provides increased retention of the obturator by contouring into the cleft. Notice the dam of modeling dough, which aids in the placement of the resin.



f **I G 28-16.** Maxillary model at the completion of the application of autopolymerizing acrylic resin. The obturator is allowed to cure for 20 minutes and then is trimmed and polished. Notice the extension of the resin into the mucobuccal fold. This extension further increases the retention of the prosthesis.



f **I G 28-17.** Obturator in place on the infant's maxillary arch. Notice the extension of the soft resin into the cleft defect, which provides increased retention. *(From Jones JE, Kerkhof RL:* Quintessence Dent Technol 8:583-586, 1984.)

chances of lip dehiscence (lip separation caused by increased pressure at the suture lines) are increased.

As early as 1686, Hofman described the use of a head cap and premaxillary strap to reposition the premaxilla.' This type of apparatus is useful for anteroposterior and vertical repositioning. The following describes the construction and orthopedic management of the typical clinical presentations: an anteriorly positioned maxilla and a laterally displaced premaxilla.

As soon as possible (usually within 2 weeks of birth), an impression is made of the infant's maxillary arch for construction of an intraoral obturator in the manner previously described. After delivery of the obturator, the infant is allowed to become accustomed to the appliance for 1 week. At the second appointment the infant is fitted with a premaxillary retraction appliance.

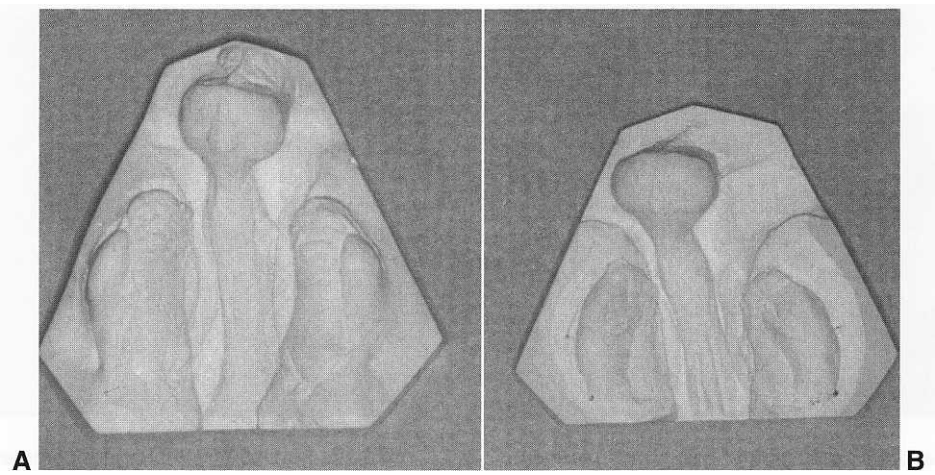
Appliance Construction for Premaxillary Retraction.

A baby bonnet is made to provide "headgear" anchorage for a premaxillary retraction strap. An elastic strap is placed over the protruding premaxilla and anchored to the infant's head using the bonnet. By the application of sequentially increasing equal forces to the premaxilla, the premaxilla is retropositioned into a more normal position relative to the maxillary segments. This bonnet-and-strap appliance is worn 24 hours a day and is removed only for feeding. The desired movement can usually be accomplished within 6 to 8 weeks (Fig. 28-19).

In many cases the use of soft, elastic tape (Microfoam Tape*) can be used to retract the premaxillary segment in a simpler manner than with the bonnet retraction system (Fig. 28-20). The advantages are its ease in fabrication; however, it does not afford the same control of the force direction and therefore cannot be used in all instances.

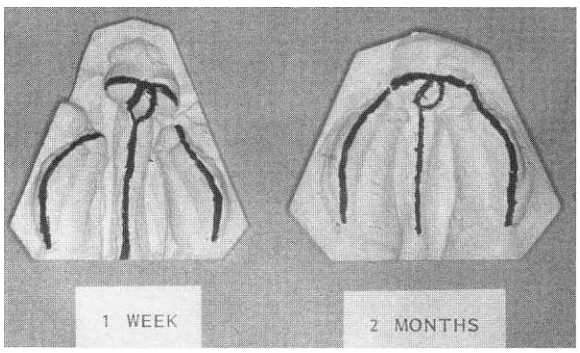
In the case of a laterally deviated premaxilla in an infant with a bilateral cleft lip and palate (Fig. 28-21, A), a straight extraoral force would not place the premaxilla in the facial midline. Therefore the premaxilla must be positioned in the midline before premaxillary retraction. In this clinical presentation, an impression is made of the infant's premaxilla for construction of an external acrylic "bulb" prosthesis. This appliance is fitted over

'3M, St. Paul, Minn.



F **I G 28-18.** Representative diagnostic models of two clinical presentations in bilateral complete cleft of the lip and palate. **A,** Severe anteroposterior protrusion of the premaxillary segment. **B,** Severe anteroposterior protrusion of the premaxillary segment with a lateral deviation.

the protruding and laterally displaced premaxilla and anchored to the infant's head with a bonnet appliance (Fig. 28-21, B). By the application of sequentially increasing differential forces to the premaxilla with elastic straps attached to the bulb prosthesis, the premaxilla is brought into the facial midline (Fig. 28-21, C). Depending on parental compliance with therapy (the appliance should be in place 24 hours a day), the laterally displaced premaxilla can be repositioned to the facial midline in 3 to 4 weeks. After the premaxilla is in the midline, the bulb appliance is replaced by a single elastic strap (Fig. 28-21, D). Over the next 1 to 2 months, equal pressure is applied on the still protruding premaxilla to retroposition it into a more normal position between the lateral segments (Fig. 28-21, E and F).



f **I G 28-19.** Diagnostic models for an infant with a complete bilateral cleft of the lip and palate at 1 week of age (before premaxillary retraction therapy is begun) and at 2 months of age (at the completion of premaxillary retraction therapy). Notice the essentially normal position of the premaxillary segment within the lateral palatal segments.



[I G . **28-20.** Premaxillary retraction using soft elastic tape (Microfoam Tape, 3M).

The rationale for use of a bulb prosthesis before elastic strap retraction includes the following considerations:

1. The bulb prosthesis affords greater control over the differential forces applied to the premaxilla.
2. Movement of the premaxilla into the facial midline before retropositioning (rather than into a laterally deviated position) decreases the risk of distorting a vomer stalk.
3. The need for a surgical premaxillary setback (a procedure known to be associated with possible growth attenuation and other complications) is eliminated.
4. Optimum premaxillary positioning may eliminate the need for a staged lip closure (adhesions before definitive lip repair) and thereby decrease total hospitalization time and cost.
5. The appearance of the nose and lip is improved because the lip can be surgically closed under less tension and the alveolar segments have an underlying symmetric alignment.

Airway Obstruction. Infants with airway obstruction secondary to Pierre Robin sequence (micrognathia, glossoptosis, and cleft palate) may require intervention to aid breathing (Fig. 28-22, A). An obturator with a posterior palatal extension should be used in an attempt to reposition the tongue downward and forward out of the cleft site (Fig. 28-22, B). If a nonsurgical approach is unsuccessful, lip-to-tongue adhesion (thus positioning the tongue anteriorly and opening the oral airway) or tracheostomy may be necessary.

Cheiloplasty. The appearance of an unrepaired wide cleft lip can be distressing. Some parents feel anxiety, depression, guilt, or rejection. Lip surgery will significantly improve the infant's appearance and may thereby relieve parental apprehensions and enhance acceptance.

Surgical closure of the cleft lip may be accomplished shortly after birth. However, most surgeons defer closure to allow the infant to be followed by the primary care physician, who assesses early growth and development and rules out the presence of any other congenital anomaly. Surgical closure is usually accomplished at 10 weeks of age. At the time of lip closure, when the infant is under general anesthesia, an impression is made of the maxillary arch for construction of a new obturator. This is necessary to accommodate craniofacial growth during the first few months of life.

Maxillary Orthopedics (3 to 9 Months). After definitive lip closure at about 3 months of age, maxillary arch collapse in unilateral or bilateral completed cleft is

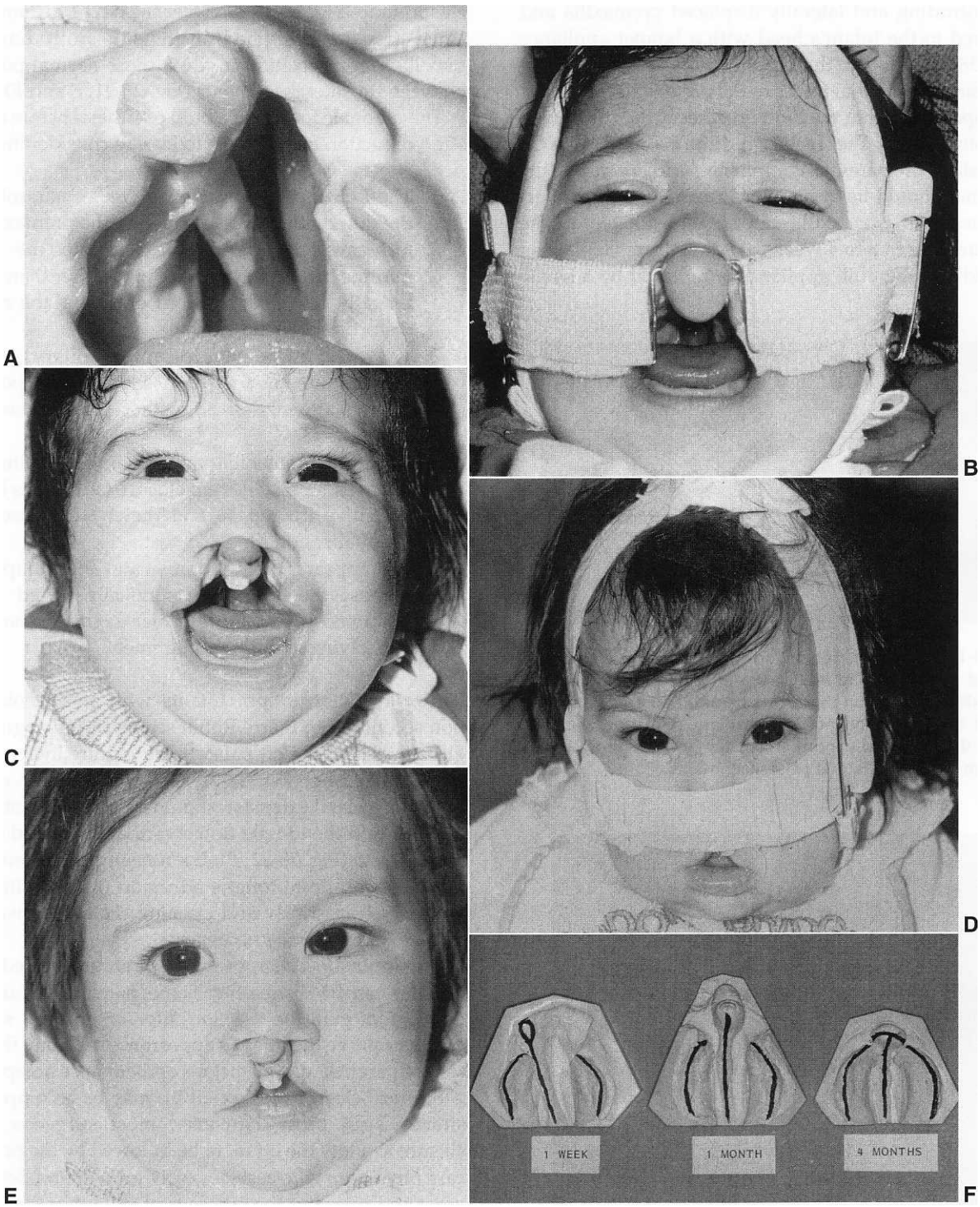
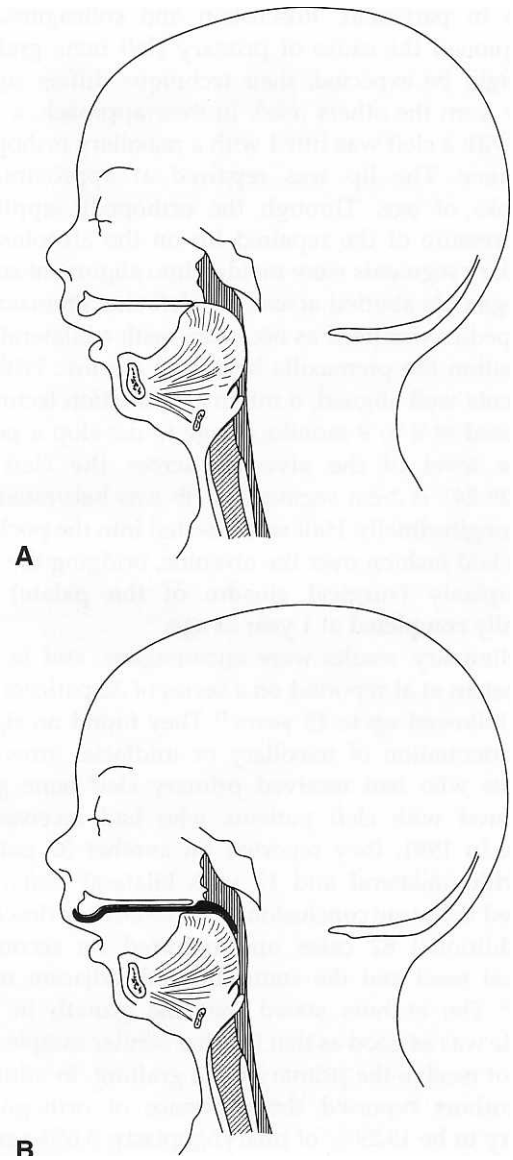
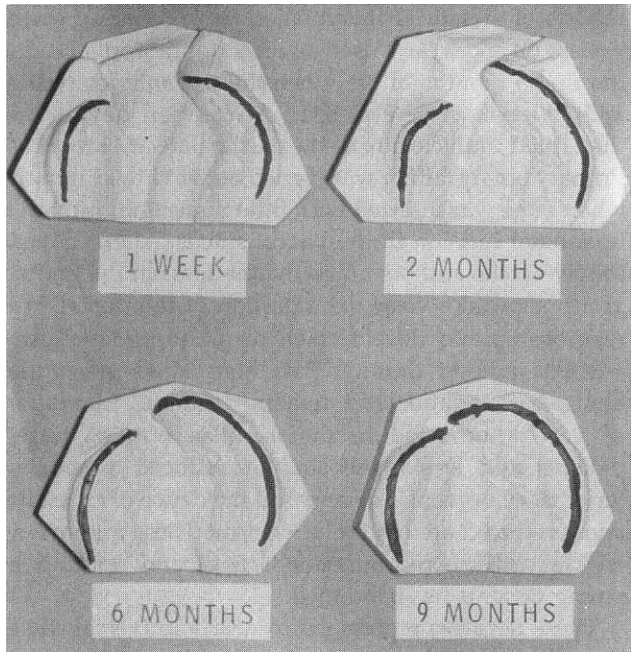


FIG 28-21. **A,** A bilateral complete cleft of the lip and palate in a newborn infant. Notice the severely anteriorly protruded and laterally deviated premaxillary segment. **B,** Placement of "bulb" prosthesis over the premaxillary segment; bulb is anchored to the bonnet. C, Patient at the end of bulb therapy to position the laterally deviated premaxillary segment to the facial midline. D, Strap therapy to improve the anteroposterior relationship of the protruding premaxillary segment before definitive lip closure. **E,** Premaxillary segment at the completion of strap therapy. Notice the improvement in position (compare with A) at this time. F, Sequential models at 1 week (initial presentation), 1 month (completion of bulb therapy), and 4 months (completion of strap therapy). Notice the improving position of the premaxillary segment at these various times. (D and F from Jones JE, Lynch TR, Sadove AM: Quintessence Int 16:229-231, 1985.)



f **G 28-22. A,** Infant with airway obstruction secondary to Pierre Robin sequence. Notice the closure of the oral airway related to the retroposition of the tongue. **B,** Infant with obturator in position. Notice the anterior placement of the tongue, which allows the oral airway to remain open.

common. It is attributed to the increased tension placed on the segments by the repaired lip. To prevent this collapse, the obturator is used to provide cross-arch stability and support. As pressure is exerted on the anterior segments of the maxilla by the repaired lip, orthopedic molding of the segments can be achieved. In unilateral cases, the force applied to the greater segment by the intact lip molds that segment around to approximate the lesser segment (Fig. 28-23). This molding is facilitated by the obturator, which provides a fulcrum around which the anterior portion of the



[I G **28-23.** Sequential maxillary arch dental models demonstrating maxillary orthopedic molding in an infant with a unilateral complete cleft of the lip and palate. Notice that, as the cleft defect closes with time, lateral arch dimension is maintained, which produces optimal maxillary arch symmetry. (From Jones JE at al: Quintessence Int 17:245-248, 1986.)

greater segment rotates. At the same time the appliance resists any tendency for the greater and lesser segments to collapse toward the midline. In bilateral cleft cases, the repaired lip provides further retraction at the premaxilla, positioning it between the two lateral maxillary segments. When the maxillary segments are in good alignment and abutted across the cleft sites, the patient is ready for the primary cleft bone graft. This generally occurs by 6 to 9 months of age.

Bone Grafting of Alveolar Cleft Defects. Bone grafting of alveolar cleft defects has been a confusing issue to many patients and practitioners. This stems in part from the lack of unanimity concerning terminology and technique. The following definitions, which have been reasonably accepted by practitioners, will be used in this discussion.

Primary bone grafting refers to bone-grafting procedures involving alveolar cleft defects in children younger than 2 years of age; this term implies nothing about technique. *Early secondary bone grafting* refers to bone-grafting procedures performed between 2 and 4 years of age. *Secondary bone grafting* is done between 4 and 15 years of age, and *late secondary bone grafting* refers to reconstruction of residual alveolar cleft defects in the adult.

Primary Alveolar Cleft Bone Grafting. Primary alveolar cleft bone grafting is controversial. The concept fell into disfavor in the early 1970s amid numerous

reports of significant attenuation in midfacial growth. Robertson and Jolleys used an orthodontic appliance for the first 3 months of life when the lip, anterior palate, and soft palate were closed.' A retention device was then fitted until closure of the hard palate at 11 months. Primary bone grafting was carried out at 12 to 15 months when bone was inserted with little dissection through a horizontal incision in the buccal sulcus. After a 5-year follow-up the authors noted that patients in the grafted group showed a clear deterioration of the dental-base relationship and development of pseudoprognathism, whereas among those in the nongrafted group the dental-base relationship remained stable. Limitation of growth occurred in the maxillae of the grafted patients and was manifested by reduced anteroposterior development, an increased incidence of crossbite, and a reduced area of the upper jaw. These authors, as well as others, recommended that bone grafting in young patients be abandoned.

These negative reports led to nearly universal cessation of primary alveolar cleft bone grafting on the basis that the procedure, performed at such an early age, significantly attenuates maxillary and midface growth. However, certain interesting facts emerge when these reports are carefully analyzed. None of the reporting groups used the same surgical technique. Although the techniques may appear similar at first glance, there are differences that make each approach unique. Therein lies a problem with the categorical statement that primary cleft bone grafting should be abandoned because it impedes maxillary and midfacial growth. What is primary cleft bone grafting? How broad is its definition? As discussed earlier, the term *primary cleft bone grafting* implies nothing about technique, only timing. The term is applied to any cleft bone grafting technique used between birth and 2 years of age. Because the techniques vary, sweeping statements that condemn or condone primary cleft bone grafting should be viewed with suspicion. Each technique should be judged on its merits, without any bias traceable to the popular view that primary cleft bone grafting retards maxillary and midfacial growth.

Many of the negative reports have used techniques involving extensive dissection in areas adjacent to and including the vomeropremaxillary suture. Friede reported that surgery in the area of the vomeropremaxillary suture may lead to impaired maxillary and midfacial growth in individuals with unilateral and bilateral clefts." He stated that techniques which disturb the vomeropremaxillary suture would never have been introduced if surgeons had realized the significant role that this suture apparently plays in the postnatal growth of the face in the cleft patient.

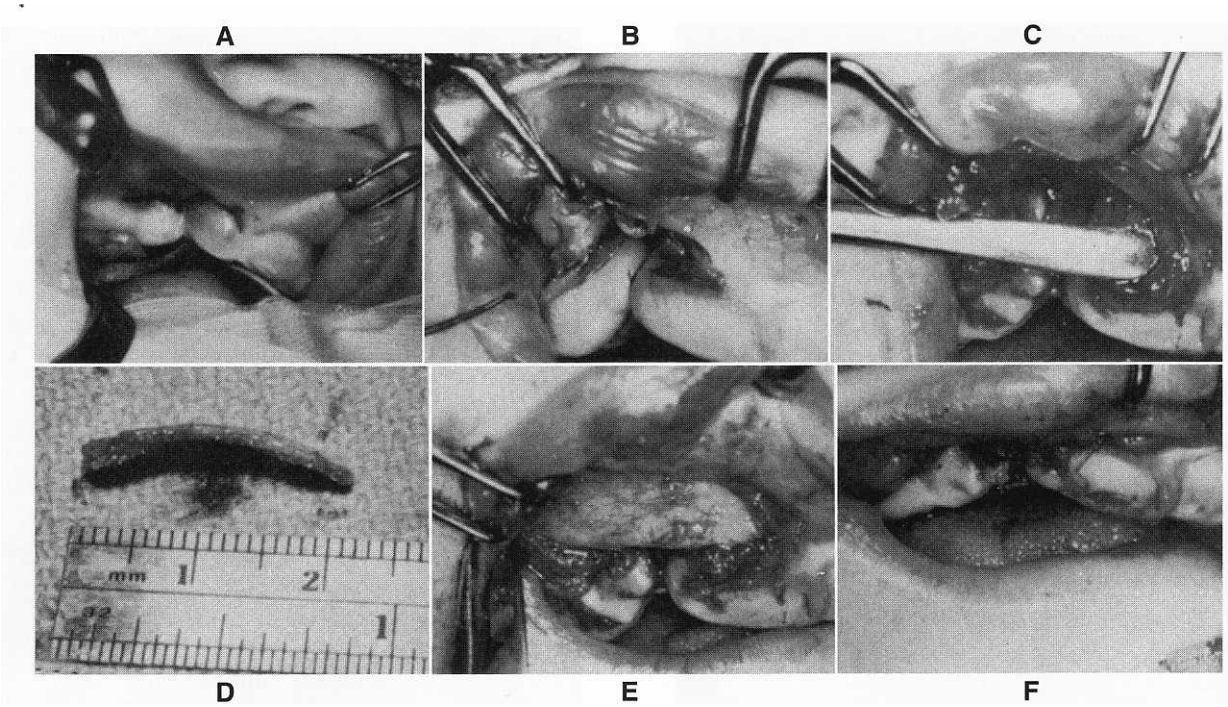
Throughout the past 35 years, although primary cleft bone grafting has been generally held in disfavor, one group in particular, Rosenstein and colleagues, has championed the cause of primary cleft bone grafting. As might be expected, their technique differs significantly from the others used. In their approach, a newborn with a cleft was fitted with a maxillary orthopedic appliance. The lip was repaired at approximately 6 weeks of age. Through the orthopedic appliance and pressure of the repaired lip on the alveolus, the maxillary segments were molded into alignment so that the segments abutted across the cleft site. Premaxillary orthopedics was used as necessary with a bilateral cleft to position the premaxilla before lip closure. With the segments well aligned, a minimal dissection technique was used at 6 to 9 months of age to develop a pocket at the level of the alveolus across the cleft site (Fig. 28-24). A 2-cm segment of rib was harvested and split longitudinally. Half was inserted into the pocket in an on-laid fashion over the alveolus, bridging the cleft. Palatoplasty (surgical closure of the palate) was typically completed at 1 year of age.

Preliminary results were encouraging, and in 1982 Rosenstein et al reported on a series of 32 patients who were followed up to 15 years.° They found no significant attenuation of maxillary or midfacial growth in patients who had received primary cleft bone grafts compared with cleft patients who had received no grafts. In 1991, they reported on another 37 patients (20 with unilateral and 17 with bilateral clefts) and reached the same conclusions.'- In 2003, they described an additional 82 cases and reported on secondary surgical need and the status of teeth adjacent to the cleft.' The authors stated that the growth in their sample was as good as that in other similar samples that did not receive the primary bone grafting. In addition, the authors reported the incidence of orthognathic surgery to be 18.29%; of pharyngoplasty, 3.65%; and of surgical closure of oronasal fistulas, *29.27%.* In cases of unilateral complete clefts, 53.13% of the lateral incisors present adjacent to the cleft area were usable, and in bilateral cases, 57.77% were usable.

The apparent success of their technique can be attributed to the following departures from previous methods:

1. Lip repair was separated from bone grafting.
2. The bone graft was inserted only when the two halves of the maxilla abutted in the alveolar region where there was no great gap to bridge.
3. The limited dissection was carried out only on the anterior surface of the maxilla and alveolus, with no dissection performed in the area of the vomeropremaxillary suture.

Experience demonstrated that in nongrafted clefts maxillary arch collapse was common after palatoplasty in unilateral and bilateral cases. When the palate was

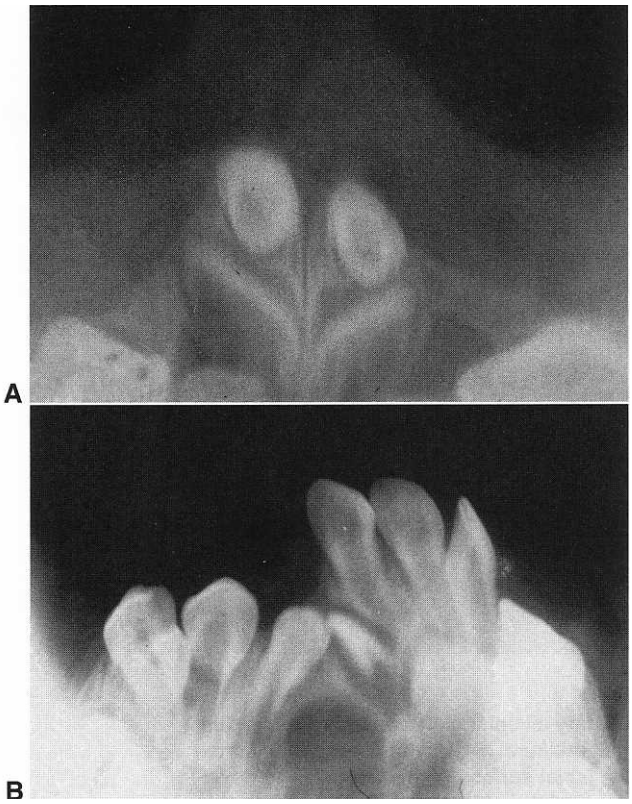


[1G **28-24.** A composite of pictures illustrating the primary alveolar cleft bone graft technique. A, Abutted maxillary segments with initial incisions outlined. **B,** Development of a small mucosal flap from the lip that will be used to cover the labial surface of the graft. C, The development of the subperiosteal pockets on the labial aspect of the alveolus. **D,** Section of rib that has been split longitudinally and contoured slightly to conform to the curvature of the maxillary anterior alveolus. **E,** Insertion of the rib segment into the labial pockets. F, Tissue closure over the graft.

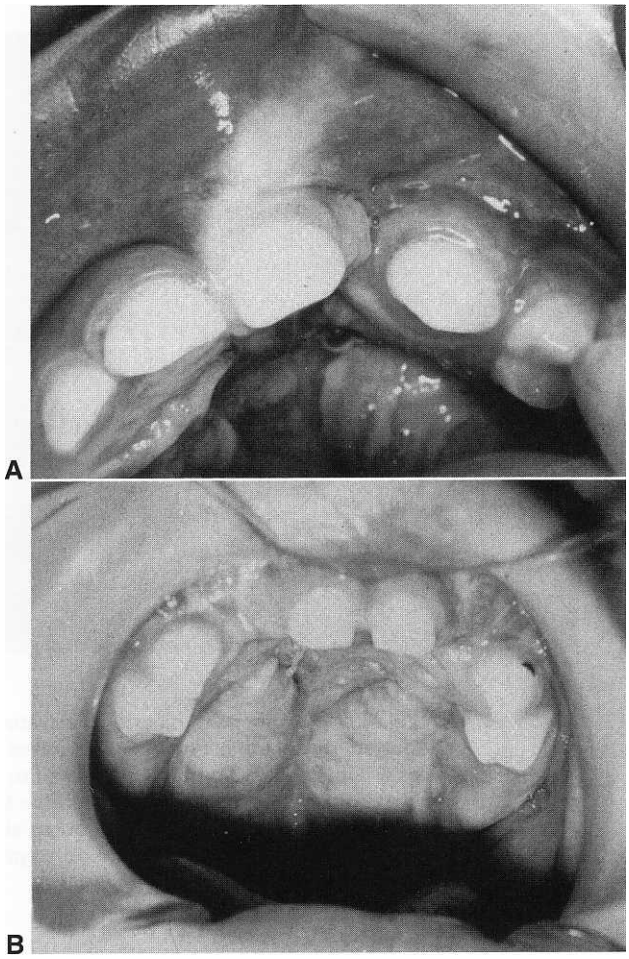
repaired, pulling the tissue together in the midline created transpalatal forces that moved the lesser segment or, in the case of bilateral clefts, the lateral maxillary segments toward the midline. This often resulted in a maxillary arch form that led to crossbite situations and compromised masticatory function. These problems were not always easily correctable. Reestablishing a reasonable arch form through orthodontic and surgical means presented a challenge because scar tissue that had formed across the palate often prevented desired segmental movement. If segmental movements could be accomplished, it was often at the expense of soft tissue form and function. Palatal fistulas frequently persisted or reopened. Periodontal compromises because of stretching of tissue were common. Primary grafting establishes maxillary arch continuity early in life by producing a one-piece maxilla rather than two or three segments (Figs. 28-25 and 28-26) and thus facilitates future treatment. Often these patients can be treated like any conventional orthodontic patient. In many instances, primary lateral incisors erupt into and through the graft.

Use of a primary alveolar cleft bone graft does not necessarily preclude the later accomplishment of a secondary alveolar cleft bone graft if it is indicated. The objectives of a secondary cleft bone graft can be somewhat different, as will be seen. Although a recent survey of cleft and craniofacial teams of the American Cleft Palate—Craniofacial Association reported that only 3% of teams perform primary alveolar cleft bone grafting, in our experience it provides excellent results. Continued critical evaluation of our results is being accomplished to document the benefits of the procedure.

A different approach to normalizing the cleft alveolar segment relationships has been advocated by Huebener and Marsh." In their treatment protocol, which utilizes the forces created by either lip adhesion or primary lip closure, a passive alveolar molding appliance similar to that used by Rosenstein is employed.[15] The passive appliance does not have an acrylic extension over the alveolar ridges and it is placed on the day of the lip surgery. The tension created by lip closure acts over time on the anterior alveolar cleft segments (in both unilateral and bilateral cleft) and shapes these cleft segments around the anterior portion of the molding appliance. The appliance is worn by the infant until palatoplasty. Usually at that time the cleft segments are abutting and the torqued maxillary frenum has returned to the midsagittal plane. In this protocol, no primary alveolar bone grafting is performed.



F **I G . 28-25.** A, Radiograph of bilateral complete cleft of the lip and palate after primary alveolar cleft bone graft. Notice the grafted bone bridging the cleft sites, which provides stabilization of the premaxillary segment. **B,** Radiograph of unilateral complete cleft of the lip and palate after primary alveolar cleft bone graft. Notice the grafted bone bridging the cleft site and the migration of the primary lateral incisor through the graft.



f **G 28-26.** A, Intraoral view of a unilateral complete cleft of the lip and palate after primary alveolar cleft bone graft. **B,** Intraoral view of a bilateral complete cleft of the lip and palate after primary alveolar cleft bone graft. Because the premaxillary segment is fixed to the lateral maxillary segments, the arch form is expected to remain in good relationship.

Grayson and Cutting have promoted the use of a molding appliance with a nasal labial extension to shape the nasal cleft cartilage."' The objective of this presurgical effort is to reduce the severity of the cleft deformity, approximate the alveolar and lip segments, decrease the nasal base width, and attempt to achieve symmetry of the nasal cartilages.

Other researchers have recommended the use of active appliances for the normalization of the infant cleft alveolar segments prior to initial lip surgery. Latham et al use a dentomaxillary advancement appliance to bring unilateral cleft segments into approximation." In bilateral clefts, they expand collapsed lateral cleft segments and actively retract the premaxilla into a more ideal arch form. They postulate that such presurgical maxillary orthopedic procedures make lip surgery easier and result in less soft tissue tension following closure.

Palatoplasty. Closure of the palate is accomplished between 12 months and 2 years of age. The primary purpose of completing palate closure by 2 years of age is to facilitate the acquisition of normal speech, because this correlates with the age at which most children begin to talk. The procedure may also improve hearing and swallowing by aligning the cleft palatal musculature.

After primary closure of the cleft palate, approximately 25% of patients demonstrate some velopharyngeal insufficiency. A persistent inability to close the nasopharynx may result in unsatisfactory speech (nasality and articulation problems), regurgitation of fluids from the nose, and facial grimacing. Of the various surgical approaches to correct velopharyngeal insufficiency, the pharyngeal flap is most commonly used at Indiana University. The procedure is generally performed when velopharyngeal insufficiency is documented in an attempt to normalize the speech of the child before he or she begins school.

STAGE II (PRIMARY DENTITION STAGE: 18 MONTHS TO 5 YEARS OF AGE)

Treatment during the primary dentition stage of dental development is initially focused on establishing and maintaining oral health. Meticulous daily oral hygiene for the child, with emphasis on direct assistance from the parents, is established to reduce the possibility of development of dental caries. Ectopic eruption of the primary maxillary anterior dentition is common around the cleft defect.

Special care should be taken to keep these teeth free from caries because food often is lodged in and around the cleft defect. An increase in the frequency of periodic recall examinations, possibly to 3- to 4-month intervals, enables the dentist to intercept areas of decalcification. This preventive regimen is continued throughout all subsequent stages in the management of the cleft.

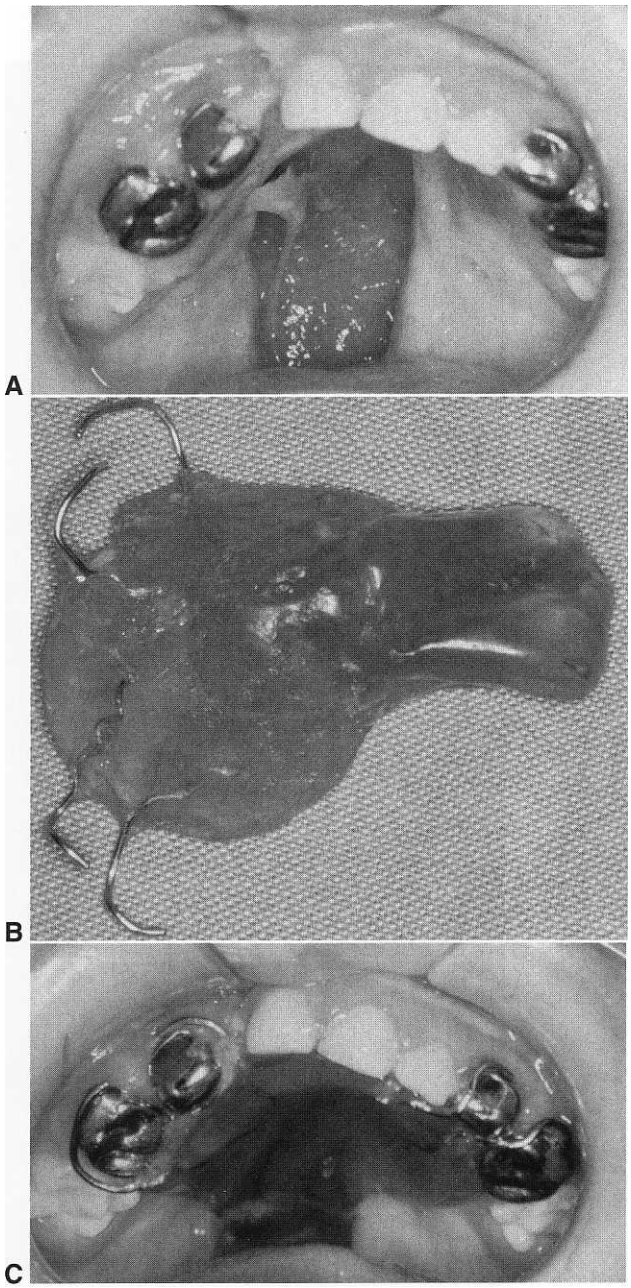
In some extensive cases of unilateral and bilateral complete clefts of the lip and palate, surgical closure is postponed beyond the usual 18 to 24 months of age. In these cases, because of the development of speech at this age, maxillary prosthetic appliances are constructed to provide normal maxillary arch integrity (Fig. 28-27). As the child grows, more tissue will become available to close the palate when doing so is surgically appropriate.

STAGE III (LATE PRIMARY OR MIXED DENTITION STAGE: 6 TO 10 OR 11 YEARS OF AGE)

Many problems encountered during the late primary and mixed dentition stage of dental development arise from ectopically erupting permanent central and lateral incisors or crossbites of the posterior dental segments. Treatment therefore concentrates on correction of a developing traumatic occlusion and posterior segmental alignment. Interceptive correction of a traumatic occlusion is essential to prevent destruction of enamel in the involved dentition (Fig. 28-28). Maxillary expansion to correct posterior segmental collapse is accomplished by routine palatal expansion (Fig. 28-29). This is especially important in patients who have not undergone primary alveolar cleft bone grafting. Once the condition is corrected, retention can be maintained by passive holding appliances.

Secondary Alveolar Cleft Bone Graft. A successful alveolar cleft bone graft satisfies several objectives. In addition to giving bony support for the teeth adjacent to the cleft and providing bone through which teeth can erupt, it offers maxillary arch continuity and aids in closure of the oronasal fistula. It also supports the alar base of the nose.

Conceptually the technique is not difficult, but technically it can be tedious. There are several approaches to developing the required soft tissue flaps, but all seem



f I G **28-27. A,** Intraoral view of a unilateral complete cleft of the lip and palate. Because of the extensive cleft of the hard and soft palate, the treatment of choice was prosthodontic obturation of the defect until growth was sufficient to allow definitive surgical closure. **B,** Removable prosthetic appliance to obturate the cleft defect. Notice the distal extension of the appliance, which approximates the posterior aspect of the patient's soft palate. This design closes the open communication between the oral and nasal cavities and facilitates normal speech production. C, Prosthesis in place on the maxillary arch. The appliance is removed daily for cleaning.

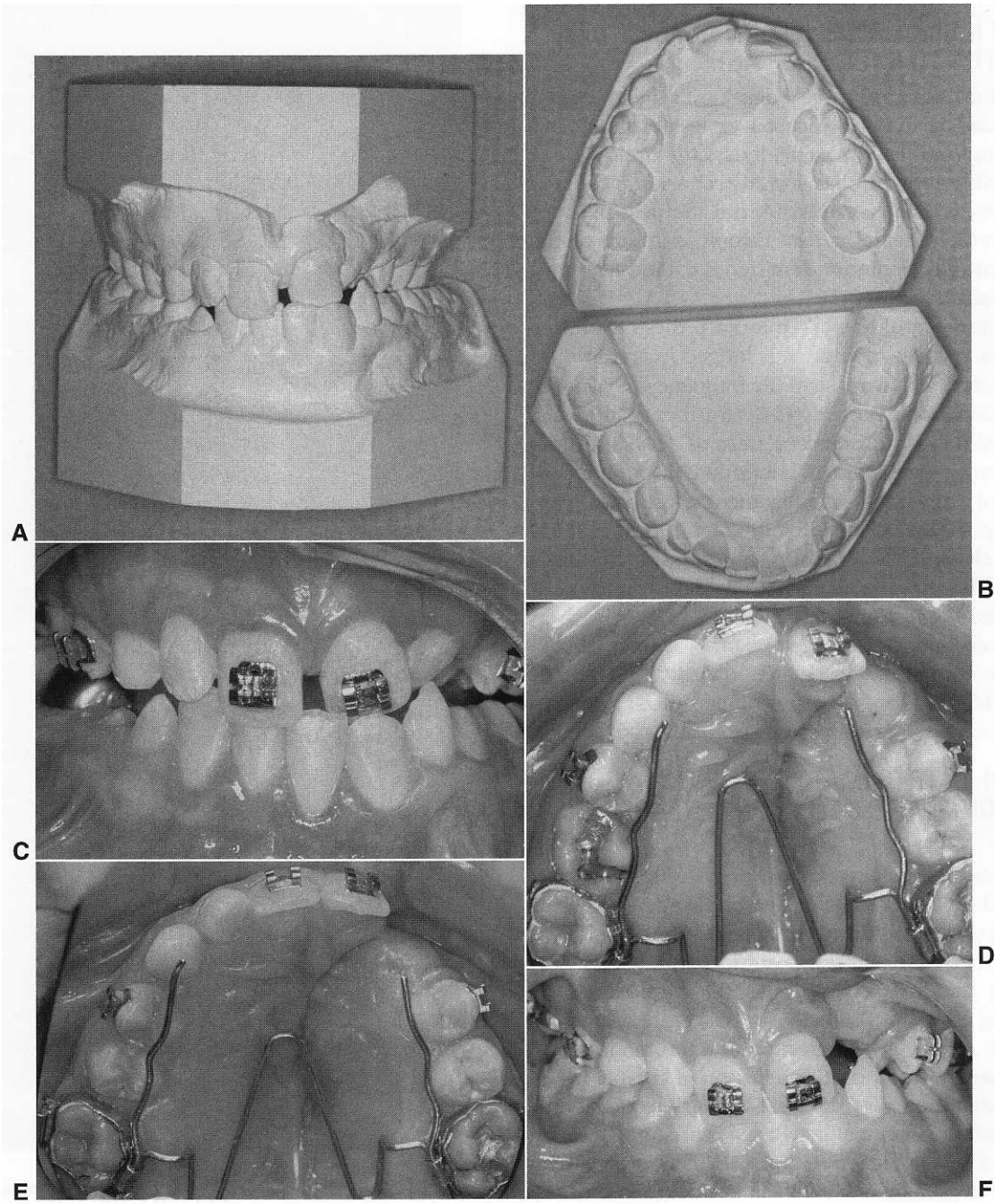
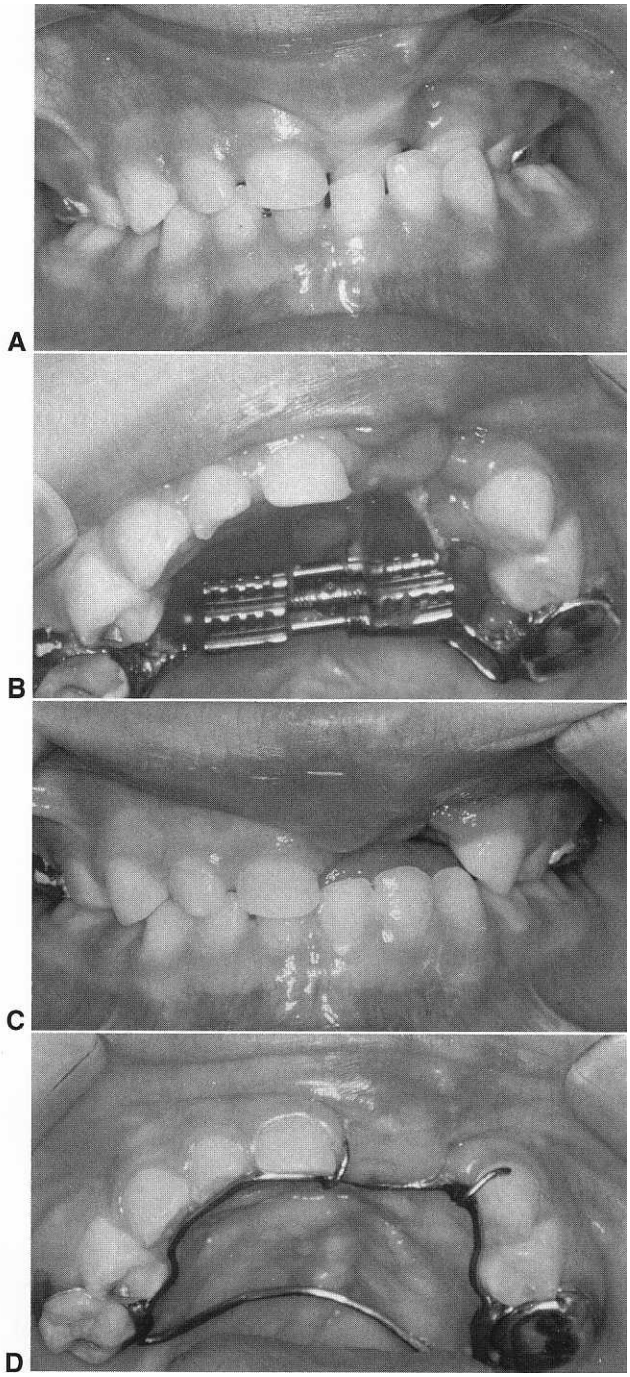


FIG. **28-28.** Interceptive correction, in the mixed dentition, of a full maxillary left crossbite in a complete unilateral cleft of the lip and palate. A, Diagnostic models for a unilateral complete cleft of the lip and palate. Notice the presence of a crossbite extending from the maxillary left central incisor to the maxillary first permanent molar. **B,** Occlusal views of maxillary and mandibular models. Notice the pronounced asymmetry of the maxillary arch. C, Occlusion with fixed appliances in place on the maxillary arch. **D,** Occlusal view demonstrating removable W arch to correct the posterior segmental crossbite. **E,** Occlusal view demonstrating correction of posterior crossbite and improved alignment of the maxillary anterior segment. Notice the improved maxillary arch symmetry. F, Occlusion at the end of interceptive therapy. Notice the correction of the anterior and posterior crossbites. At this time, the patient is ready for secondary alveolar bone grafting (see text for description of secondary bone-grafting procedure).



[**I G 28-29.** Interceptive correction, in the primary dentition, of a full maxillary left crossbite in a complete unilateral cleft of the lip and palate. **A,** Occlusion demonstrating a crossbite from the maxillary left primary incisor extending to the second primary molar. **B,** Fixed palatal expander in place on the maxillary arch at the completion of expansion. The left primary central incisor, loosened by traumatic occlusion before initiation of therapy, exfoliated during treatment. C, Occlusion at the completion of interceptive therapy. Notice the correction of the posterior crossbite. **D,** Placement of a passive maxillary arch-holding appliance to maintain optimal arch symmetry until the time of secondary alveolar bone grafting.

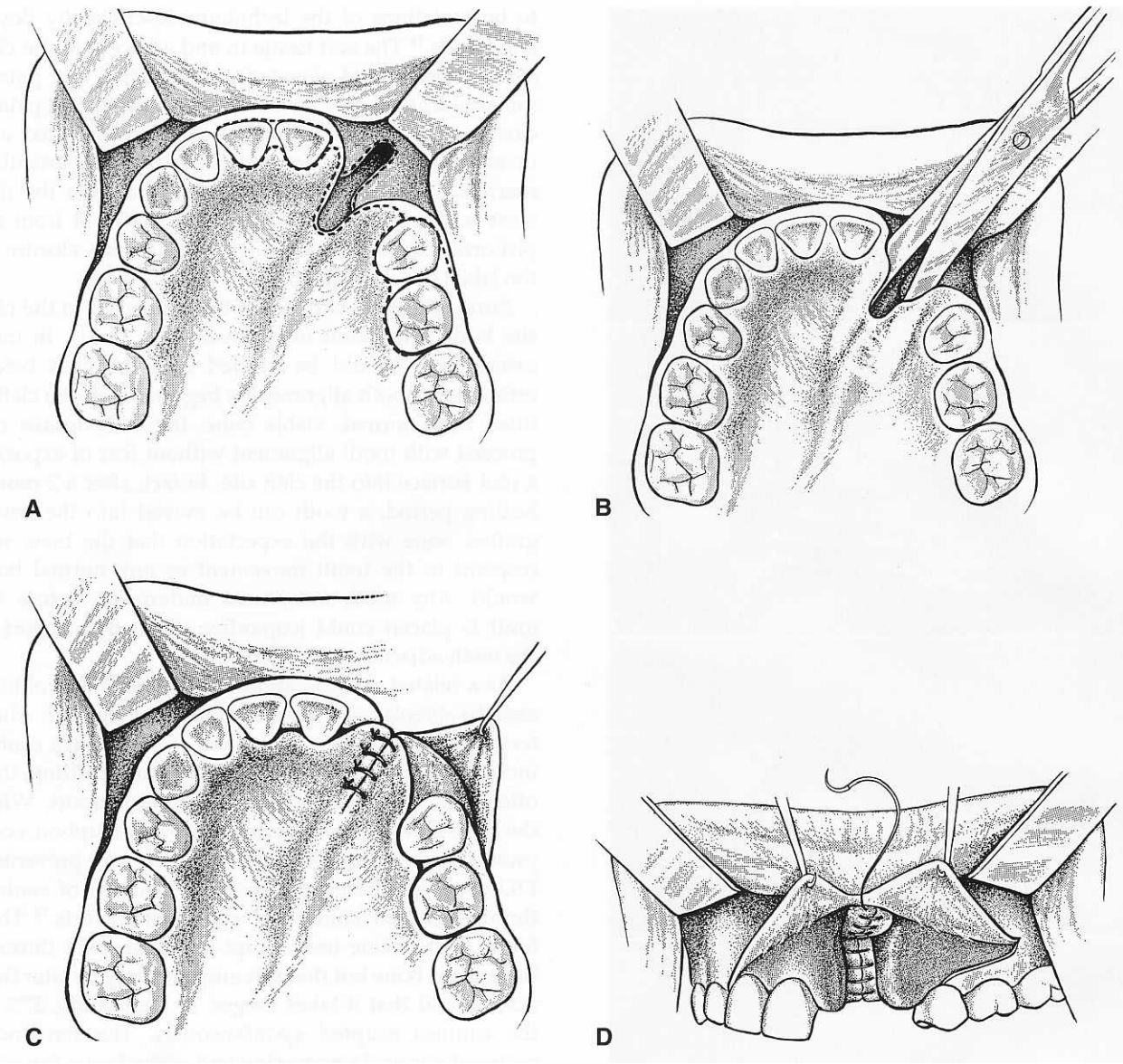
to be variations of the technique described by Boyne and Sands.' The soft tissue in and adjacent to the cleft side is incised and elevated so that labial and palatal mucosal leaflets are everted to obtain labial and palatal closure. The tissues lining the cleft are elevated and inverted into the nose for nasal floor closure. Particulate marrow and cancellous bone harvested from the iliac crest is placed into the cleft defect, filling it from the piriform rim to the alveolar crest before closure of the labial tissues (Fig. 28-30).

Providing bony support to teeth adjacent to the cleft site is of paramount importance (Fig. 28-31). In most cases, bone should be grafted into the cleft before orthodontic tooth alignment is begun. When the cleft is filled with normal, viable bone, the orthodontist can proceed with tooth alignment without fear of exposing a root surface into the cleft site. In fact, after a 2-month healing period, a tooth can be moved into the newly grafted bone with the expectation that the bone will respond to the tooth movement as any normal bone would. Any tooth movement undertaken before the graft is placed could jeopardize the bony support of the teeth adjacent to the cleft.

As a related consideration, grafted bone that obliterates the alveolar cleft also provides bone through which teeth can erupt. When canines and, in some cases, central incisors are allowed to erupt before bone grafting, they often lack adequate periodontal bone support. When the bone graft precedes permanent tooth eruption, compromised periodontal situations can often be prevented. ElDeeb et al studied the eruption patterns of canines through grafted bone in alveolar cleft defects.[19] They found that canine teeth erupt spontaneously through the grafted bone but that this eruption may be later than normal and that it takes longer. In their study, 27% of the canines erupted spontaneously. The remainder required surgical uncovering and orthodontic forces to accomplish eruption and alignment in the arch. Turvey et al found the rate of spontaneous eruption to be 95%, which represents a significant difference.`° However, the point is that canines can and do erupt through the grafts. If eruption seems greatly delayed, surgical and orthodontic intervention is appropriate.

ElDeeb et al have recommended that the graft be placed between 9 and 12 years of age when the canine root is one quarter to half formed." They reported that the canine subsequently has normal root development and that morphologic conditions will be unaffected by the surgical procedure.

Restoring maxillary arch continuity and stabilizing the maxillary segments represent other major objectives. In the bilateral cleft case, the premaxilla is stabilized as the bone grafts are incorporated between the premaxilla and the lateral maxillary segments. In the process, the alveolar ridge contour is restored so that the ability to

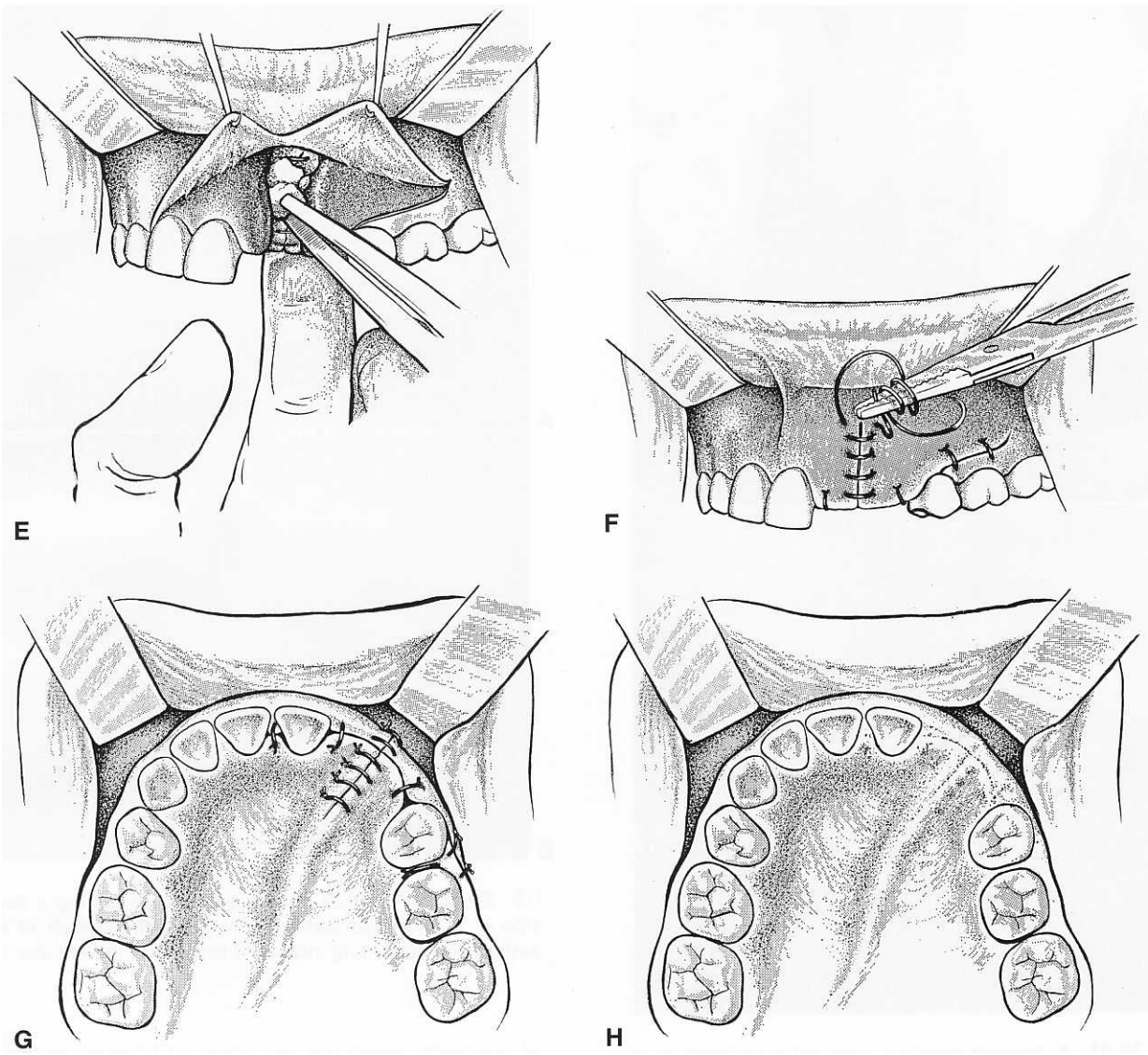


1 G 28-30. Technique for secondary alveolar cleft bone graft. **A,** Mucosal incisions outlined. **B,** Development of palatal mucoperiosteal flap. C, Closure of palatal mucosa. **D,** Closure of nasal mucosa within cleft site.

provide a stable, esthetic prosthesis is enhanced. There is often some degree of collapse of the maxillary arch form. It is possible to expand the arch after grafting, as pointed out by Boyne and Sands[1]; however, it is preferable to expand these collapsed segments to as optimal an arch form as possible before grafting. Pregraft expansion also widens the cleft site, which allows better access for nasal floor closure. After the arch expansion has occurred, the bone graft can be placed. After the graft has been incorporated, it can be expected to maintain a good arch form.

Closure of the oronasal fistula is often the most significant result of bone graft surgery, according to patients. They often have fluid regurgitation into the nose and mucus drainage from the nose into the oral cavity through the fistula. Depending on its size, the fistula can produce significant speech problems because air escapes when the patient phonates. Although closure of this fistula can be effected with only soft tissue closure, Enemark, Krantz-Simonsen, and Schramm have indicated that closure is more successful when combined with a bone graft.[1]

With a cleft maxilla the cleft extends through the piriform rim beneath the alar base of the nose. As a result, the alar base on the cleft side is often depressed because of lack of underlying bony support. Filling the cleft with bone provides underlying bony support that often elevates the alar base of the nose. Although this



[FIG 28-30, contd. **E,** Placement of fresh autogenous bone into the cleft defect. **F** and **G,** Reapproximation and closure of mucoperiosteal flaps. **H,** The reconstructed maxillary alveolus.

may not entirely correct any existing nasal deformity, it does provide good support over which nasal reconstructive and revision surgery can be accomplished (Fig. 28-32).

Secondary alveolar cleft bone grafting has been widely accepted. Boyne and Sands,[18] Bertz,[3] Hall and Posnick,[11] Kortebein, Nelson, and Sadove,[21] Kwon et al, [17] Troxell, Fonseca, and Osbon,[27] Enemark, Krantz-Simonsen, and Schramm,[26] and Turvey et al[20] have reported favorable results and emphasize the low morbidity rate. Success rates are generally in the 90% range. Morbidity has included pain in the donor site, dehiscence of mucosal flaps, and partial or complete loss of grafted bone. Infection in the donor or recipient sites has been rare. An unpublished survey by Huebener indicated that secondary alveolar bone grafting (grafting performed in the mixed dentition) is routinely performed by all teams.

Secondary alveolar cleft bone grafting is an important procedure that greatly facilitates total habilitation. Not only is speech improved but dental, esthetic, and psychosocial benefits are to be gained. It is necessary again to emphasize the different objectives of primary and secondary grafting and to reiterate that they are not mutually exclusive procedures. The primary graft, over time, may satisfy some or all the objectives of secondary grafting. However, to the extent that it does not, augmenting the primary graft with particulate marrow and cancellous bone from the iliac crest may be recommended as a secondary procedure.

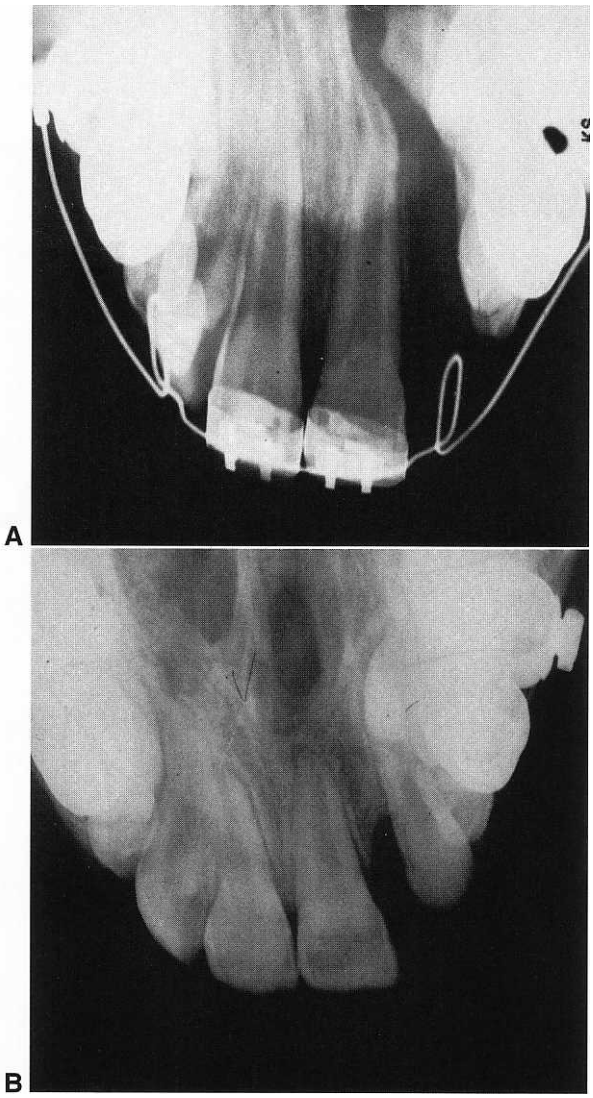


FIG **28-31. A,** Pregraft maxillary occlusal radiograph demonstrating cleft defect with thin layer of bone over the distal root surface of the maxillary left central incisor. The canine has not started to erupt at this time. **B,** Postgraft maxillary occlusal radiograph demonstrating consolidation of bone across the cleft defect. Preliminary orthodontic alignment has been completed. The canine can be expected to erupt through the grafted area. (From Nelson CL et al: J Indiana Dent Assoc 65161:9-73, 7986.)

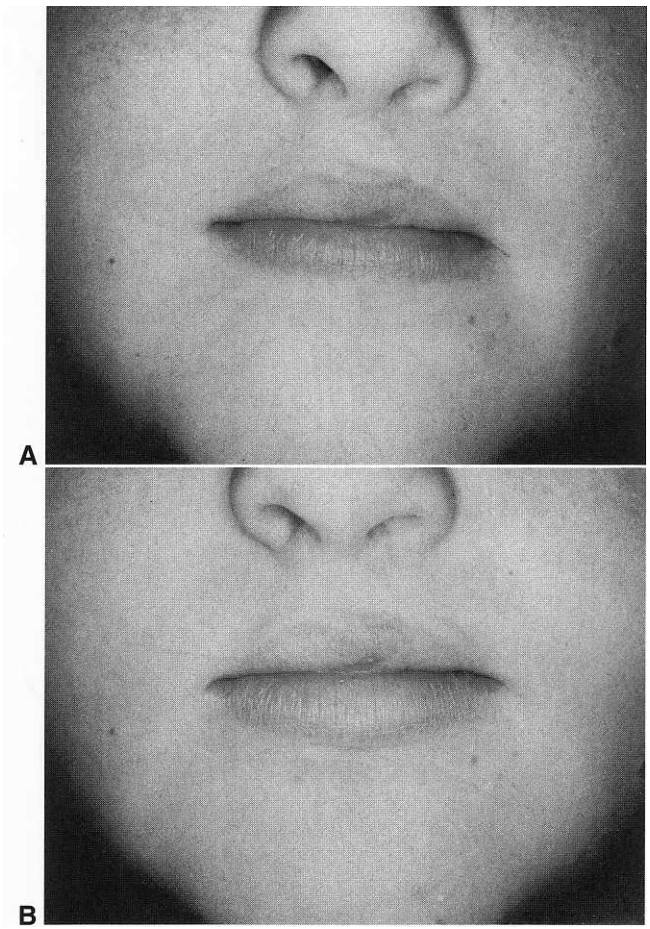


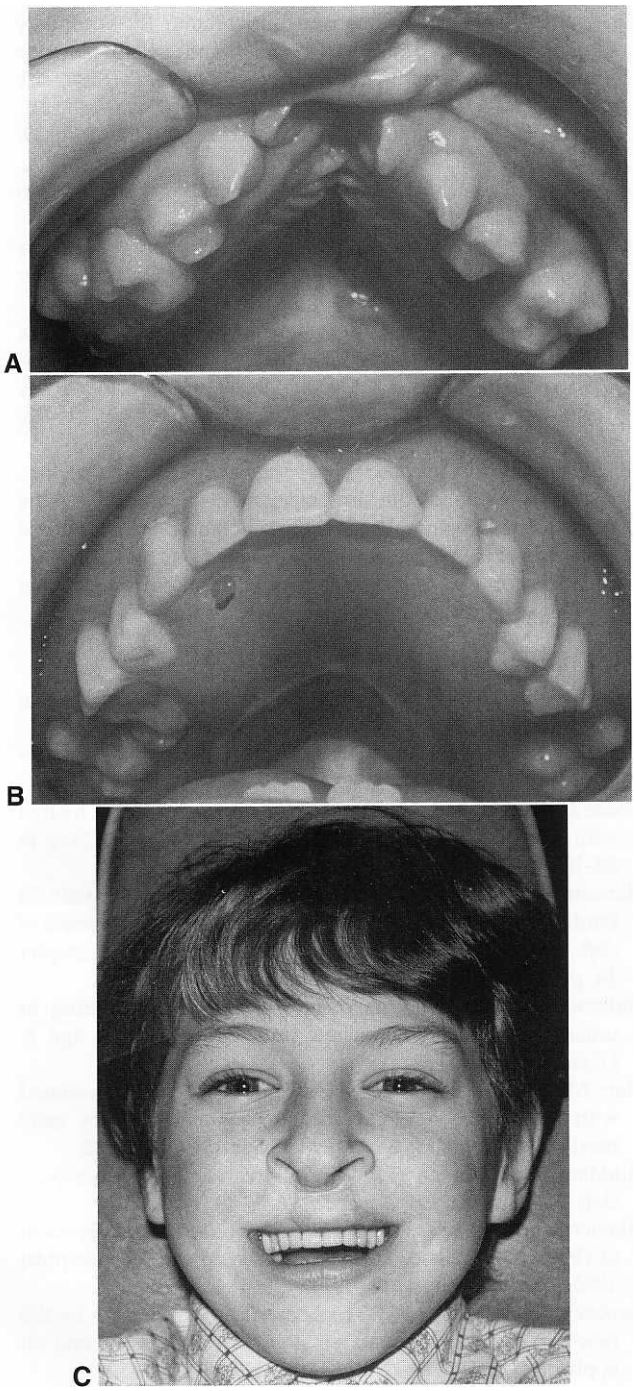
FIG **28-32. A,** Pregraft photograph demonstrating a depression of the left alar base. **B,** Postgraft photograph of same patient demonstrating increased support of the left alar base.

Effect of Facial Esthetics on Self-Concept. Appearance helps to determine how an individual inter-acts with society and, in turn, how society perceives and accepts that individual. Facial esthetics is especially important to the development of self-concept. A child born with a serious congenital facial anomaly may find adaptation difficult. For example, Striker et al have stated that the psychologic sequelae of cleft lip and palate may have as great an impact on the individual as the physical aspects." MacGregor stressed that, because of society's emphasis on physical attractiveness and conformity, the role of the face in interactions with others is such that many problems associated with cleft lip and palate involve considerations of mental health.[1-9]

Of special importance in the comprehensive dental management of cleft lip and palate is the dentist's ability to provide the young patient with interim prostheses to improve facial appearance (Fig. 28-33). These can be periodically adjusted to allow eruption of the developing dentition. Such treatment, when possible, should begin before the child starts formal education.

STAGE IV (PERMANENT DENTITION STAGE: 12 TO 18 YEARS OF AGE)

The majority of persons with cleft lips and palates require some degree of orthodontic management and can be treated in a conventional manner. Some cleft patients, however, will require a combined orthodontic-surgical approach in the permanent dentition to achieve



[**I C 28-33. A,** Severely deformed maxillary arch in young patient with a complete bilateral cleft of the lip and palate. The patient will not undergo comprehensive orthodontic therapy for several years. **B,** Transitional maxillary complete overdenture. Notice that the appliance is being adjusted to allow eruption of the developing dentition. Such appliances may be used several years without replacement. C, Transitional maxillary overdenture in place. The patient's self-esteem is greatly enhanced by his essentially normal facial esthetics.

optimal outcome. Before initiating the indicated therapy, the orthodontist completes a full diagnostic examination to determine the status of the patient's craniofacial development.

Most surgical procedures involving the maxilla and mandible are deferred until the teenage years, when maximum growth of the jaws has been attained and all permanent teeth except the third molars have erupted. In boys, surgeons usually delay osteotomies until approximately 17 to 18 years of age; in girls, because of earlier maturation, surgery sometime after 15 years of age is possible. The manner in which the maxilla and mandible relate to each other spatially after growth is frequently difficult to predict based on the patient's appearance as a child. An example is the patient with a complete bilateral cleft lip and palate who has a protuberant premaxilla at birth. In childhood, the lateral profile may appear convex, and the initial impression is that the patient will require a premaxillary segment surgical setback. This could be an erroneous assumption, and corrective surgery could be potentially delete rious if it is performed at an early age. With time, many of these persons acquire an essentially normal lateral facial profile. By the time maximum growth has been attained, a surprising number of these persons acquire a convex profile.

In some instances, children with a cleft with a severely retrusive maxilla cannot undergo orthodontic correction with conventional therapy. In these cases the surgical procedure often used is the LeFort I maxillary advancement. This procedure is not technically feasible until the patient has a full complement of permanent dentition. The horizontal cuts to free the maxilla must necessarily be made above the apexes of the permanent dentition. Unerupted cuspids or bicuspids would make this procedure impractical. Therefore surgery must be deferred until the permanent dentition has erupted.

Cosmetic Surgery. Major nasal bone surgery may be deferred until the patient is in the early teens. However, cartilaginous nasal tip asymmetries may be corrected at any time. Additional tip-cartilage revisions may be performed as needed.

Common secondary deformities of the repaired unilateral or bilateral cleft lip include an upper lip that is too long or short, a tight upper lip, a deficiency of the vermilion tissue, and residual clefts (or notching) of the lip. Accurate predictions of how such deformities will manifest themselves are impossible until growth of the tissues is complete. Consequently, final surgical revision is frequently deferred until the mid to late teens. It is also prudent to defer final lip revisions until any surgical or orthodontic treatment that will change the osseous or dental support of the upper lip is complete.

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CHAPTER

29

Practice Management

ANN PAGE GRIFFIN
LEE M. HARRISON, JR.
JASPER L. LEWIS, JR.

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CHAPTER OUTLINE

PERSONNEL SYSTEMS
- Personnel Needs
- Interviewing and Hiring
- Orientation and Training
- Wage and Benefit Administration
- Performance Appraisals
- Personnel Records
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- Communication

PATIENT SYSTEMS
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- Marketing and Practice Growth

Communications with Parents and Patients

OPERATIONAL SYSTEMS
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- Recare System
- Checking of Recare Effectiveness
- Broken Appointments and Purging of Patient Charts
- Production and Collections
- Payment Policy
- Dental Insurance and Other Third-Party Plans
- Insurance Assignment
- Insurance Reference Guide
- Predetermination
- Filing Claims
- Health Insurance Portability and Accountability Act
- Direct Reimbursement
- Managed Care
- Dental Management Companies

Fiscal Management
- Billing and Accounts Receivable
- Fees
- Accounts Payable
- Budget Setting
- Practice Monitors
- Daily Statistics
- Monthly Statistics
- Averages, Medians, and Goals in a Pediatric Dental Practice
  - Production
  - Collections
- Accounts receivable
- Appointments
  - Average work times per week, month, and year
  - Treatment and hygiene minimum goals Example of checking recare system effectiveness
- Typical Ratios in a Pediatric Dental Practice
  - Overhead
  - Hygiene department production
  - Hygiene compensation
  - Chairside assistants' compensation
  - Business staff compensation
  - Number of business staff
  - Staff compensation including wages, payroll taxes, and benefits

The practice of modern dentistry requires delivery of quality care combined with adherence to excellent business principles. Dentists, traditionally trained in the art and science of dentistry, must also become skilled in techniques of sound business management. Today's practitioner must be clinically astute and knowledgeable about consumer needs and demands, government regulations, and third-party participation—aspects of dentistry not often addressed in the clinical setting of dental school.

The transition from a role of dental student or resident to business owner and manager is an enormous challenge. That transition often begins when the new-to-practice dentist enters practice and suddenly realizes that dentistry encompasses more than clinical and technical competence. A successful dentist must be concerned with many office-management concepts, including leadership, delegation, interpersonal relationships, personal style, community image, and time management, as well as the day-to-day operation and fiscal management of a business.

The primary responsibility of a dentist is to provide quality patient care. However, private practice—and, in today's environment, many school, hospital, and government dental facilities—like any other business, must make a profit to survive. The practitioner must maintain a balance between patient care and business requirements while keeping moral, ethical, legal, and professional responsibilities in proper perspective.

While maintaining this balance a dentist must provide a place where children can feel safe, loved, and well cared for; where parents can be educated about how to help their children have a lifetime of good oral health; where the staff know that they are an integral part of the practice, that they are important and appreciated, and that their opinions count; where other practitioners and health care providers feel good about referring patients or calling the office for information; where the community as a whole knows, respects, and appreciates the practice; and where changes can be made in any area needed and new, better, innovative ideas and methods can be endorsed and implemented.

Every new dentist begins practice expecting to be successful. Those satisfied with their level of achievement after several years realize that a successful dental practice is composed of three systems: personnel, patient, and operational.

PERSONNEL SYSTEMS

PERSONNEL NEEDS

A personable, professional staff is vitally important for practice success. The dentist must invest time, effort, and money to hire, train, and retain quality individuals who can be developed into a team. A team of committed professionals, working together, focused on the patients and the practice, can make the difference between an excellent and a mediocre practice. Therefore, staff development is a sound investment in any practice.

Before becoming an employer, the dentist must be aware of state and federal regulations concerning hiring; employment policies, including Occupational Safety and Health Administration (OSHA) requirements; employee records and retention; discipline; and dismissal. All employment applications and other forms used in the hiring process and employment policies described in the office manual should be reviewed by an attorney familiar with state and federal laws.

The number of employees in an office varies accord ing to the type of practice and patient volume. Pediatric and orthodontic practices usually require more team members than other practices.

Initially, the new practitioner may need to hire only one or two staff members. If only one individual is hired at first, a second employee should be added when patient flow increases to the point that this person can no longer assist at the chair and handle the business desk (e.g., collect fees, make appointments, answer the telephone by the third ring, prepare and mail statements on time, and pursue broken appointments). The first employee, who is already knowledgeable about scheduling patients and practice flow, should move to the business desk. The second employee should serve as a full-time clinical assistant.

The third office employee may be a second clinical assistant or a dental hygienist. When the dentist treats hygiene patients more than 8 hours per week and the state practice act limits the performance of some hygiene duties by a certified dental assistant, a hygienist should be hired. This will allow the dentist to concentrate on other treatments and procedures. Depending on patient volume, the hygienist may be full time or part time. Other employees may then be added as needed.

Additional help may be gained through the use of part-time employees. These employees may *job share, a* term used to describe the splitting of a full-time position between two people, or a position may need to be filled only part time (i.e., a few hours per day or week).

The business of managing and administering a dental practice has become complex and time consuming for the dentist, even with a well-trained staff. Some larger dental practices now hire professionally educated and trained practice administrators. "The key responsibilities of a Practice Administrator could be compared to those of a Chief Executive Office (CEO) of any corporation:

1. Strategic planning and positioning—chief strategist

2. Team building—culture, positioning, performance contracts, empowerment
3. Monitoring all departments, making adjustments/changes as needed
4. Chief deal maker."'

Once a practice administrator is on staff, the doctors' position becomes similar to that of the chairman of the board of directors of a corporation or company, a major policy voice as well as owner of the right of refusal or veto authority. In some cases, several practices may share the services of a practice administrator.

INTERVIEWING AND HIRING

Mastery of the interviewing process is difficult but necessary. Hiring begins with attracting well-qualified applicants. Potential employees may come from personal recommendations made by current staff; area technical schools, colleges, and universities; or classified advertisements.

Interviewing becomes easier and more enlightening with repetition. The more opportunities an interviewer has to conduct interviews, the smoother and more productive the sessions. Use of a set of interview questions standardizes the process. These questions should explore the following traits: initiative, organization, and conscientiousness; effective communication; ability to work well with others; and technical or business training and the ability to apply previous training and experience to the job. If questions are skillfully posed, an applicant's answers will indicate strengths or weaknesses in these areas.

Following these steps should make the interview more effective:

1. Read the resume or job application to learn about the person before the interview. Chat a few moments to put the interviewee at ease as the interview begins.
2. Have a written set of questions that will elicit strengths or weaknesses in the four areas mentioned previously.
3. Tell the interviewee that some specific questions will be asked and notes will be taken.
4. Listen well. Note body language and eye contact, because they often speak louder than words.
5. The interviewer should talk less than 25% of the time. The purpose of the interview is to understand the applicant and his or her potential for the job. Excessive talking by the interviewer limits the time and opportunity that the applicant has to tell about personal abilities and experiences.
6. Do not help the interviewee answer questions. Let pauses happen. The applicant may be thinking during the silences, and the interviewer can see how well and how quickly the person responds.
7. Do not ask questions that can be answered with yes or no. If the interviewee begins answering yes or no, ask him or her to explain or expound on the answer.
8. Never argue with an applicant. Maintain poise and remember that the interviewer is in control.

Two of the most desirable traits in a dental team member are a warm, empathetic personality and cognitive ability, defined as aptitude for learning and capacity to draw from past experience in new situations. Several excellent instruments are on the market to measure cognitive ability and various aspects of personality and behavioral style.*

To maximize efficiency, a well-trained staff member may conduct the first interview. If the candidate is promising, employment tests, a tour of the office, an introduction to other staff members, and a brief conversation with the dentist should be a part of the first interview. References should be checked between the first and second interviews.

The second interview should include a longer conversation with the dentist and office observation or work time. The dentist should pay the applicant for time spent observing and/or working in the office. Inviting the prospective employee to work in the office for a few hours allows staff members to form an opinion about the person's potential. Likewise, the applicant will better understand office ambiance and patient flow before accepting the job.

Although the final hiring decision rests with the dentist, it is a good idea for certain key staff members to concur. Current employees significantly affect the success or failure of a newly hired staff member because they help train and interact closely with him or her.

An offer may be made after the working interview. If the applicant will not be hired following the first, second, or working interview, he or she may be told at that time or notified by mail within 1 week of either interview. If an applicant is rejected, maintain all applications, test forms, and other paperwork for at least 1 year in case a complaint is filed. If testing is part of the application and interviewing process, testing must be standardized. All applicants must be given the identical test. If an applicant is hired, be aware that all interview and employment records must be kept for the duration of employment plus 30 years.

*Examples include the following: cognitive ability testing instrument—Wonderlic Personnel Test, Wonderlic, Inc., Libertyville, Ill., www.wonderlic.com; personality and behavioral-style instruments—DiSC Classic (Personal Profile System), Inscape Publishing, Minneapolis, Minn., www.inscape.com.

If the applicant is to be hired, the dentist should offer the job using the term *full-time* or *part-time* employment and should avoid using the word permanent, either orally or in writing. Use of the term permanent connotes continuous work with a contractual understanding, regardless of problems that might develop later. If dismissal should become necessary, the employee might claim wrongful dismissal if promised permanent employment.

ORIENTATION AND TRAINING

In *The One Minute Manager,* Blanchard and Johnson wrote, "Most companies spend 50% to 70% of their money on people's salaries. And yet spend less than 1% of their budget to train their people. Most companies, in fact, spend more time and money on maintaining their buildings and equipment than they do on maintaining and developing people."'

The comments of Blanchard and Johnson are equally appropriate for training of auxiliaries. Adequate training, a requisite for productive staff, is often slighted in busy dental offices.

A new employee must be made to feel welcome and a part of the team. Orientation on the first day of employment and a written training schedule to be followed the first 6 to 8 weeks should be a part of this process.

The most effective way to orient new employees is to follow a checklist, which ensures that each new staff member will receive similar information. Fig. 29-1 is an example of an orientation checklist for new employees.

Orientation may begin with a review of the office manual. An up-to-date policy and procedural manual can prevent misunderstandings with current staff members and reduce training time for new auxiliaries. An office manual outlines the duties, obligations, and mutual expectations of the employee and employer, and clarifies office policies, employment procedures, benefits, training methods, and office paperwork. Preferably, the dentist writes the manual with input from experienced senior staff members. Prototypes of office manuals are available in print or in software versions to assist in the process. One example is that prepared by KnowledgePoint, Petaluma, Calif., http://www.knowledgepoint.com or telephone 707-762-0333. The attorney for the practice should review the manual before implementation.

A training period may be the first 60 to 90 days of employment, during which time the dentist determines whether there is a match between office needs and a new employee's skills and personal style. Use of the term *training period* rather than *probationary* or *trial period* connotes the importance of performance to the new employee. To an employee, a training period is considered as a time to work diligently, to strive to learn as much as possible, whereas a probationary or trial period is often seen as only a time to endure until hiring is finalized.

An employee record* set up, containing the following:
- Resume
- Application form
- Interviewing notes and test forms
- Tax and employment forms, including the often-overlooked Employment Eligibility Verification (1-9) form from the Bureau of Citizenship and Immigration Services
- Copies of licenses
- Personal data
- Salary and benefits
- OSHA-required medical and training records

Office tour provided, including review of work hours, dress code, personal item storage, parking, brief history of practice

Introduced to other staff (written list of names and brief job descriptions)

Job description reviewed

Training procedures and expectations reviewed

Beginning and end of day procedures explained

Office security, lock-up, housekeeping, etc. explained

Safe work procedures reviewed

Emergency procedures reviewed

Confidentiality emphasized concerning
- Salary
- Patient data
- Production, collection, and any other practice financial data

Hepatitis vaccine administered within 10 days or refusal statement signed

Insurance forms completed and submitted (at end of training period)

Office manual reviewed

f **I G 29-1.** Example of orientation checklist for new team members.

Much of the responsibility for training new employees can be delegated to staff. A well-trained veteran staff member may become a training coordinator, available to instruct the new employee, answer questions, and check progress. Although one person may efficiently coordinate training, all other team members and the dentist must be involved in the process.

In the most successful practices, the dentist and team members realize that training is an on-going process. To some professionals, the term *dental practice* implies that training never stops; dental professionals continue to learn and improve throughout their careers. Attendance at continuing-education courses, review of current liter ature and journals, and group study using audiotapes and videotapes are appropriate for continuous training. A portion of each staff meeting can be used for additional training and discussions of new treatment methods and materials. Also, the establishment of an office library makes books, journals, and tapes easily

available to all team members. Learning together strengthens a dental team and encourages pride in personal and professional growth.

WAGE AND BENEFIT ADMINISTRATION

Wage and benefit administration is an aspect of practice management lacking clear-cut rules or guidelines. Many dentists therefore find it a particularly difficult task. There must be a balance between managing payroll costs, sharing practice income and profits equitably with staff members, and maintaining careful control of expenses.

The most successful dentists and managers set high standards for their practice, including top-notch performance and productivity from all team members. in return, these dentists compensate team members well, sharing increased profits with staff members who helped produce the growth. As a result, auxiliaries realize that, the better the practice does financially, the better they will do. They also understand that, if the practice has an unproductive period in which production, collections, and profits decline, raises and other benefits may be delayed. Benefiting from growth and increased profits or, conversely, experiencing the pinch of fiscal decline allows auxiliaries to feel responsibility toward the practice that they might not otherwise sense.

Employees receive more than wages; net pay is only one part of a complete compensation package. Although higher take-home pay with fewer benefits may attract some employees, fair wages with expanded benefits generally help retain team members. Each employee should be given an annual total compensation statement listing gross wages, employer-paid taxes, and the dollar value of benefits such as vacation, personal leave, insurance, uniform allowance, and free or reduced-cost dental care. Fig. 29-2 is an example of an individual total compensation statement. Receiving an actual statement of wages and benefits lets employees perceive their full *value.* The dentist should communicate *value* rather than cost to staff members. Auxiliary personnel who are made to feel valued and appreciated are more productive than those told what they "cost" the practice.

Wage and benefit administration requires careful planning. A comprehensive salary plan helps retain employees by offering salaries commensurate with community wage scales. It also allows the dentist to evaluate relative pay among employees and to review wages and benefits in a timely and organized manner. If planning is neglected, a dentist may give raises that are too large or too frequent or, conversely, forget about salary reviews for an extended period. The former condition results in excessive wage costs; the latter may cause good employees to quit. A common problem expressed by many dentists in practice for 15 to 20 years is that a long-term staff member's total compensation package is excessive. The young practitioner can avoid this problem by establishing a salary plan.

To begin a salary plan, the dentist first lists the staff positions in the office, including business positions (receptionist, scheduling coordinator, patient accounts coordinator, and so on), clinical assistant, and hygienist. The relative worth of each position to the practice is determined using the following questions:

1. How long would it take to find a replacement?
2. Are there specific certification, education, or training requirements?
3. What is the value of longevity in this position?
4. How skilled is a certain individual?

The more competent, valuable, and difficult to replace the employee is, the higher the wage should be.

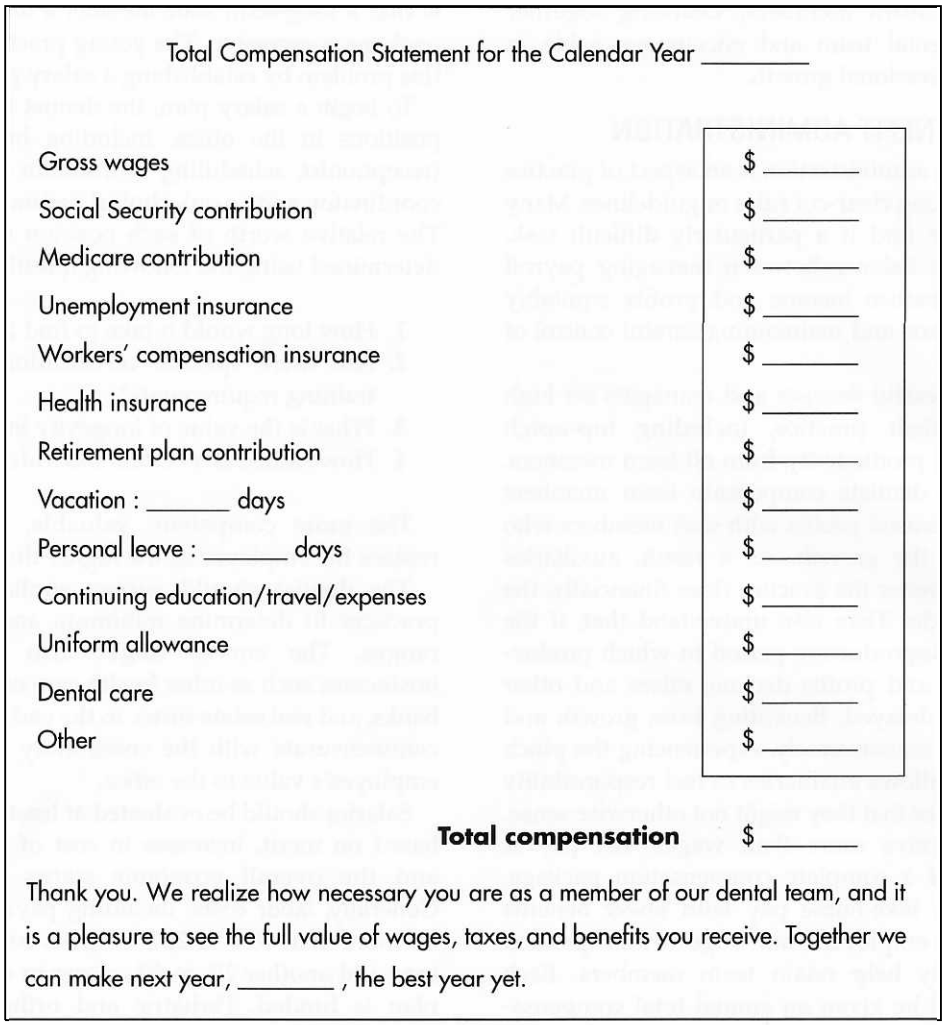
The dentist should survey similar jobs in other practices to determine minimum and maximum pay ranges. The survey might also include similar businesses such as other health care offices, law offices, banks, and real estate firms. In the end, wages should be commensurate with the community standard and an employee's value to the office.

Salaries should be evaluated at least yearly; raises are based on merit, increases in cost of living (inflation), and the overall economic status of the practice. Generally, labor costs, including payroll taxes, require no more than 18% to 22% of gross collections. Benefits may add another 2% to 4%, closer to 4% if a retirement plan is funded. Pediatric and orthodontic practices typically have wage, payroll tax, and benefit costs of 20% to 31% of gross collections because the greater patient volume requires more personnel. The pediatric dentist, in particular, may hire more people because a number of patients may be scheduled simultaneously, and many children cannot be left alone while in the treatment chair.

Fair salaries and good benefits are necessary to avoid job dissatisfaction; however, salary and benefits do not necessarily motivate an employee to perform better. Motivated staff members are most often those to whom responsibility is delegated. Their personal and professional growth is encouraged and recognized by the dentist. They are appreciated and told how valuable they are to the office, the dentist, and the patients. In short, motivated staff members are recognized for the contributions they make to the practice, and money is only one way appreciation is shown.

PERFORMANCE APPRAISALS

Potentially effective staff members are often dismissed because of poor job performance, which may result from the dentist's failure to define expectations.



Total Compensation Statement for the Calendar Year _____

Gross wages	$ _____
Social Security contribution	$ _____
Medicare contribution	$ _____
Unemployment insurance	$ _____
Workers' compensation insurance	$ _____
Health insurance	$ _____
Retirement plan contribution	$ _____
Vacation : _____ days	$ _____
Personal leave : _____ days	$ _____
Continuing education/travel/expenses	$ _____
Uniform allowance	$ _____
Dental care	$ _____
Other	$ _____
Total compensation	$ _____

Thank you. We realize how necessary you are as a member of our dental team, and it is a pleasure to see the full value of wages, taxes, and benefits you receive. Together we can make next year, _____ , the best year yet.

FIG. **29-2.** Individual total compensation statement.

Employees can be expected to perform optimally only if requirements are clear. Team members must know what they are expected to do, how to do it, and what the criteria are for an acceptable or, better yet, outstanding job performance.

The purpose of performance reviews is to recognize past growth and accomplishments while setting new goals and standards of achievement. Periodic performance appraisals allow the dentist and team members to evaluate personal attributes, job strengths, and areas to be improved, so that goals for personal and professional growth can be set. Appraisals should be positive and result in better job performance and improved efficiency.

Each staff member should be evaluated according to similar criteria, including competence in performing tasks, work habits, contributions to the team, and general attitude toward patients, coworkers, the dentist, and the office. Nebulous topics, such as attitude, poor mannerisms, or personality conflicts, should be addressed by listing specific examples of positive or negative instances involving the person. Evaluation forms that can be completed by the staff member and dentist are helpful but not absolutely necessary. Simple notes about strengths, growth opportunities, and the employee's needs as they match or conflict with the needs of the practice are sufficient for the evaluation conversation. Discussing the staff member's opinion of his or her own performance often makes the appraisal more meaningful. Performance reviews must be given regularly to improve job performance and encourage personal growth. An appraisal should be given after completion of the training period and at least annually thereafter.

Job skills and work performance should be reviewed separately from salary. A conversation that combines performance review with news of a raise interferes with the staff member's concentration. An employee may be so interested in hearing about a salary increase that

setting goals for performance improvement is relegated to a secondary level of importance.

During the review, strengths should be enumerated first. People listen better when they feel that the appraiser recognizes their strengths rather than only their deficiencies. Use of the term *development areas* rather than weaknesses when discussing areas to be improved connotes opportunity for growth.

Most people respond best when asked to concentrate on no more than three or four areas for development. Hearing about more than three or four points to be improved is overwhelming and may delay the employee's responses and improvements.

The dentist may offer to help the employee improve in certain areas through additional training, continuing-education courses, and such, and thereby give the staff member a sense of support. The dentist and staff member should agree on realistic goals to be met by a certain date, with regular discussions during the interim to evaluate progress and establish other objectives.

PERSONNEL RECORDS

Personnel records are important documents. They are a history of the employee-employer relationship, just as a patient's chart is a documentation of the patient-dentist relationship. If a problem arises with an employee (e.g., a charge of wrongful dismissal in case of termination), the record will serve as proof that the dentist correctly discharged employer responsibilities.

Records, including job application, interviewing notes, testing forms, performance appraisals, wage and benefit information, and so on, should be maintained for the duration of employment and for at least 30 years following an auxiliary's departure. Records for OSHA-mandated training must be maintained from the date the training occurred and must include dates of training, contents of the session, and the name and qualifications of the trainer. Check with your state OSHA office for information concerning the number of years that these records must be maintained. Medical and unsafe-incident records must be maintained for the duration of employment plus 30 years and include all appropriate forms with pertinent data.

Personnel records should be identical in appearance for all employees, consistently maintained, and kept confidential. A comprehensive employee record could include the following:

- Employee's name
- Address and telephone number
- Social Security number
- Completed job application and/or resume
- Employment tests given at the time of hiring
- Date of employment
- Licenses or certifications
- Completed tax forms, including federal and state income tax and Employment Eligibility Verification (I-9 form available from the Bureau of Citizenship and Immigration Services)
- Spouse's name, employer, and telephone number
- Person to contact in case of emergency
- Salary record and summary of benefits
- Training records, including OSHA training and cardiopulmonary resuscitation training
- Medical records, including injury or exposure to harmful substances; record of hepatitis inoculation or signed and dated refusal of the vaccine
- Performance appraisal forms and notes from one-on-one discussions signed and dated by the employee and the dentist
- Absentee records
- Continuing-education course records
- Record of termination, including last date of employment, the employee's letter of resignation, or notes from the dismissal conversation if the employee was fired

DISMISSAL

Dismissal of an employee is one of the most difficult tasks a dentist may face. Whereas interviewing becomes easier with experience, firing someone does not. There are, however, processes that can make a dismissal less stressful.

Dismissal usually happens because of poor attitude, incompetence in job performance, or unacceptable behavior such as patient abuse, sabotage, theft, or substance abuse on the premises. Problems arise when an employee loses interest in her or his responsibilities. Attitude and performance problems must be addressed; simply ignoring the situation will not make **it** go away. The dentist and staff member must discuss the problems and ways that they can be rectified. After an initial discussion with the employee, it should be absolutely clear to the employee that improved attitude and/or job skill is a requirement for continued employment. The employee and dentist should sign and date notes from the initial and subsequent meetings. A specific date by which attitudes and/or skills must improve should be set. The dentist and the staff member should meet frequently to assess improvements.

When coaching an employee who is trying but is incompetent, a combination of the following procedures may be used. The dentist should write a description of acceptable performance criteria, including skills needed for the job. It is more effective to name specific charac teristics and skills needed for the particular position than to enumerate only negatives (i.e., what the person is not doing). The goal is to have the employee understand the requirements of the job, the ways that he

or she fails to meet them, and ways that such expectations can be fulfilled.

The dentist then establishes a time frame for improve ments; between 1 and 3 months is usually appropriate. The employee and the dentist should discuss, sign, and date the agreement about performance improvements. The dentist should review progress with the employee each week, making notes of these discussions in the employee's record. If the employee reaches the goal of acceptable job performance within the time allowed, new goals should be set. In this way, a staff member can be coached into continuously improved productivity.

If an employee will not or cannot improve attitude or achieve required skill levels, dismissal will probably be necessary. In the dismissal conversation, failure to meet the performance improvement agreement may be discussed. The dentist must try to dismiss the person without causing anger and resentment. Although the employee's skills do not match office needs, often the person can leave without negative feelings if the dismissal conversation is conducted properly.

If an employee is behaving in an unacceptable way (e.g., consistently arriving late, abusing sick leave, or breaching confidentiality requirements), a serious warning may rectify the problem. If an employee is given serious disciplinary warnings before dismissal, the dentist should put the warnings in writing and include a statement to this effect: "Continuation of these actions by this employee may lead to disciplinary measures, up to and including dismissal." After this statement is explained, the employee and the dentist should sign and date the statement, and it should be retained in that individual's personnel file.

In most states no notice is necessary for dismissal related to unlawful behavior such as stealing, patient mistreatment, or deliberate property damage. The staff member committing such actions can be dismissed immediately if such actions can be proved indisputably. It should be clearly stated in the office manual that such conduct will not be tolerated and will result in immediate dismissal.

Preferably, a dismissed employee leaves immediately after the termination conversation. The dentist may pay severance wages, but payment is not mandatory in all states. (The dentist should check with an attorney about state law and write the policy in the office manual.) It is unwise for an employee who was fired to remain on the job. The dismissed person may sabotage the practice, even unconsciously, and bother other staff members with the details of termination. Such may be the penalty a dentist may pay for being kind-hearted enough to let a dismissed employee keep working until she or he finds another job. If a dismissed employee remains employed while searching for another job, a departure date should be set. Otherwise, the job search may be prolonged.

If the dentist is unclear about the proper way to discipline or dismiss an employee, he or she should seek advice from a lawyer versed in labor relations.

COMMUNICATION

Concern with communication is not a recent phenomenon; all human relationships depend on it. Relationships with team members, patients, other health care professionals, family, and friends depend on the skill with which one communicates. Even if the clinical dentistry is superlative, patients can perceive problems and stresses among the staff and/or with the dentist. If the dentist and team members are not functioning as a unit, patients may feel that the entire practice, including the care delivered, is below average. The following steps can be taken to strengthen intra-office communications:

1. Write a practice mission statement with input from the team. Have the finished statement engraved in calligraphy, framed, and hung in the reception room.
2. Survey team members about the state of communications among the group members. Address problem areas and set standards for improvement.
3. Use personality inventories to learn more about your own and coworkers' styles, their impact on others, and how individuals react when stressed. Hire a psychologist or other trained professional to administer and interpret personality surveys. The greater understanding and cooperation among staff is well worth the cost.
4. Schedule regular staff meetings on office time. Rotate leadership of meetings among the group so that staff members, as well as the dentist, feel responsible for achieving productive sessions. Schedule a planning retreat at least annually. A retreat is an away-from-the-office session involving the dentist and all team members, usually 1 day long, during which every aspect of the practice is assessed and improvements are planned. Goals in production, collections, new patients per month, recare system strength, and such can be set for the next year or 6 months until another retreat is scheduled and the process is repeated.
5. Attend continuing-education courses together. Use ideas and materials from the courses to enrich staff meetings and improve office processes.
6. Encourage one-on-one discussions, dentist to team member and team member to team member. Write intraoffice memos as needed. Memos are particularly helpful in a practice employing part-time people who may miss staff meetings. Post a central calendar to note staff meetings,

vacation schedules, and continuing-education course dates.
7. Post a bulletin board out of sight of patients for staff use. Pertinent professional information, personal news, and amusing items may be displayed.
8. Plan fun events involving the entire team. Groups that learn to enjoy one another are more productive, work more synergistically, and are more capable of handling stressful situations in the practice.

The five communication skills—listening, speaking, writing, reading, and using nonverbal (body) language—should be discussed and practiced among members of a dental team committed to excellence. Active listening gives proof that the listener has indeed understood. Team members can learn this skill together. Gordon's *Leader Effectiveness Training is* an excellent reference for active listening.' Team members must also learn to speak properly, using correct grammar and choice phrases when responding to others' questions, problems, or complaints. All paperwork and forms used in the office should be reviewed at least annually to make certain that they are properly written, pertinent, legal, and impeccable. All team members must be trained to read patient forms, noting all comments, pertinent facts, and diagnostic notes. The entire team should discuss nuances of body language so that they can communicate in positive ways nonverbally.

PATIENT SYSTEMS

FIRST APPOINTMENT

A good first appointment experience provides the foundation for an enjoyable, long-term relationship with patients and parents. The impression given on that visit is lasting.

Although walk-in emergencies occur occasionally, most initial contacts with a practice are by telephone. Because a caller frequently judges the entire practice by a voice on the telephone, the business staff should be trained in correct telephone etiquette and procedures. Many local telephone companies offer such training.

During the initial conversation, certain information should be gathered from the parenting adult. Use of a form can help standardize the procedure. Completed by the receptionist and used during the first visit, such a form is an invaluable aid in patient registration. It can be kept permanently in the patient chart. Fig. 29-3 is an example of such a form.

A packet mailed before the first examination is an excellent introduction to any practice. The packet might contain the following items:

1. A brochure that provides information about the practice, list of services offered, general advice about how parents can best help children prepare for the first dental appointment, and a map to the office.
2. A health history form stamped in red ink in an upper corner, "Please complete and bring with you."
3. A note of welcome to the young patient, including a reminder of the appointment date and time.

The physical layout of the reception area and business desk must allow staff members a full view of the reception room. On arrival, the patient and parents should be greeted by a team member whether or not a sign-in sheet is used.

A well-trained team member should escort new patient arrivals to a conference room or the operatory. At this time, the staff person should review the child's health history with parents, recording pertinent notes for the dentist. The patient and parents may be given a brief tour of the office, and the tour guide can reinforce information given in the practice brochure.

The dentist should then be introduced by the staff member who interviewed the new patient and parents. After a short conversation addressed mainly to the patient, the dentist should again review the health history with the parents, reading notes made by the staff member during the interview. This routine provides two opportunities to ensure that the health history has been correctly completed and that the review with parents is documented. If the initial conversation occurred in a conference room, the dentist should leave while the team member prepares to take the child to the operatory. If the child is upset, the dentist, who must gain full cooperation during the dental examination, is not involved.

The parents may remain in the conference room, accompany the child and assistant to the operatory, or be escorted back to the reception room. Typically, parents now accompany their child to the operatory. Although this often adds stress and causes the child's behavior to be worse, the presence of parents in the operatory is a reality in most pediatric dental offices in today's litigious society. The dentist can, however, wisely limit the number of accompanying adults in the operatory with each patient. Fig. 29-4 is an example of information given to parents about this limit. Note that the statement also forbids the use of cell phones in the operatory, which can be a most annoying problem.

After the examination, the dentist should present the case to the parents, who usually want answers to the following questions: What is wrong? Can it be fixed? How much will it cost? The practitioner should keep these questions in mind and plan case presentations accordingly. Many new-to-practice dentists overtalk during case presentations, confusing parents and

John L. Doe, D.D.S.
222 Ash Street
Greenville, NC 27834
(919) 752-5183

NEW PATIENT TELEPHONE INTERVIEW

Date of call _____ Appointment _____
Taken by _____ Call for earlier appointment _____
New patient packet mailed _____

Patient's name _____ Age (if minor) _____
Parent's name (if minor) _____
Phone (home) _____ (work) _____
Address _____ City _____ Zip _____
Other family members who are patients: _____
Referral _____
First dental visit: Yes No
If no, are there current radiographs to be requested from another office? Yes No
If yes, name of dentist _____
Address _____ City _____ Zip _____
Phone _____ Date called _____ Date received _____
Special dental concerns _____
Special medical concerns _____

Emergency (circle)

Lost filling
Bumped tooth
Fractured tooth
Knocked-out tooth
Toothache, how long? _____

Other symptoms:
Sensitive to hot/cold/sweet
Bleeding
Constant pain
Swelling
Apprehension
Awake at night
Other _____

Dental insurance Yes No or other coverage: (circle) Medicaid
Special health services

_____ Patient informed that fee for first examination is due at time of the examination.
_____ Patient informed we will be happy to file insurance for subsequent visits.
Comments _____

FIG. **29-3.** New patient telephone interview form.

patients with excessive clinical terminology. The dentist may wish to rehearse case presentations with staff members, seeking their critique and suggestions.

At some point during the first appointment, an assistant or hygienist should give home care instructions. The patient and parent are then escorted to a checkout area. The next appointment is usually scheduled before the fee is collected. After checkout, a staff member should express thanks to the parents for choosing the office and remind them that the practice appreciates

Explanation to Parents about Patient Safety and Privacy

For your comfort both parents, or **one** adult, are welcome but not required to accompany your child to the operatory. However, for the safety and privacy of other patients **all others,** including children who are not scheduled at this appointment, are asked to remain in the reception room. Young children in the reception room will need a supervisory adult.

In addition, the use of cell phones is prohibited in the operatory. The conversations carried on by others present in the clinical area can be most distracting to children, preventing us from close, careful communication with each young patient.

Thank you for your understanding. Your cooperation in these matters helps us to serve your child better.

Ili **29-4.** Explanation to parents about patient safety and privacy.

referrals. The child and parents should leave the office feeling that the dentist and staff are extraordinarily caring, thorough, skilled, and efficient. The first appointment should make parents eager to recommend the practice to other family members and friends.

PATIENT FLOW

For appointments subsequent to the initial examination visit, visualizing the overall pattern by which patients move through an office can make each appointment more productive and valuable for the patient and practice. All appointments have the following phases: Stage 1 is telephone contact, usually initiated by the parent or guardian. Stage 2 includes arrival, check-in and greeting, and escorting of the patient and parents to the conference room or operatory. During stage 3, the patient is seated in the operatory, with instruments and supplies placed for efficient treatment delivery. Stage 4 includes clinical treatment and performance of procedures. During stage 5, dismissal, the parents make the next appointment. If this is not possible, the patient's treatment needs must be entered into the computer before the chart is refiled. If treatment plans are entered consistently following each examination, new patient or recare treatment needs for the patient who refuses to schedule an appointment are never lost.

Stage 6 includes collection of the fee. Staff members must receive training in collection procedures so that collection efforts are handled properly and professionally.

During stage 7, the staff member thanks the parents and patient. This farewell should leave a lasting impression. Stage 8 involves recontact, including the recare system, special correspondence with the patient and parents, and other forms of ongoing communication.

The dentist and team should discuss all eight phases, set standards and procedures for each, plan ways to welcome patients and parents, provide service that will be perceived as extraordinary, ensure reappointment, and encourage referrals. In a practice committed to total quality of patient care, such concepts are worked and improved continuously.

PREVENTIVE PROGRAM

Offering preventive information and health counseling is an important part of dental service, appealing to an increasingly sophisticated health care consumer. Intraoffice educational efforts can include topics such as home care, thumb sucking, diet, oral health during pregnancy, the dangers of smokeless tobacco, and infant oral health. Information should be shared with individuals at their levels of need and comprehension. A well-planned and -executed preventive education program can be a dynamic marketing tool.

An excellent way to offer educational and preventive services is through a dental health educator–marketing coordinator. The educator may be developed from among current staff members, or a new person may be employed. An invaluable resource, the educator must be given proper training and sufficient time to perform intraoffice teaching responsibilities, patient-relations tasks, and community-wide educational activities.

MARKETING AND PRACTICE GROWTH

Professional marketing goes beyond just efforts to bring more patients into the practice. Through marketing, parents, patients, and others in the community (i.e., prospective patients) can be educated about the value of dental health and ways that it can be achieved.

There are two aspects to the marketing of a dental practice. Internal marketing includes activities within an office that affect current patients. New-patient packets mailed before the first appointment, special correspondence to patients, giveaways, and improved office ambiance are all forms of internal marketing.

External marketing activities are targeted at current and prospective patients. A web site, a telephone on-hold message system, schoolchildren tours of the office, lectures to educational, civic, and social organizations about disease prevention and the latest dental techniques, and career counseling with youngsters interested in learning more about the dental profession are forms of external marketing.

Internal and external marketing efforts must be well planned, coordinated among staff, included in the budget, reviewed periodically, and evaluated for effectiveness. Successful programs should result in

improved oral health habits among patients and in the attraction of many new patients.

Although opinions vary among management consultants concerning appropriate numbers of new patients monthly for a well-established general practice, 20 to 40 seems to be an accepted range. However, most experts agree that, after 5 to 7 years in practice, a pediatric dentist should set a goal of 60 to 75 new patients monthly. Typically, this number is needed to compensate for attrition as children grow older and transfer to a general dentist, move away, or leave the pediatric practice for some other reason.

COMMUNICATIONS WITH PARENTS AND PATIENTS

A sense of trust must be developed between parents and patients and the dental team. This rapport comes from skillful communications. Like communication among office team members, communication with parents and patients includes listening, speaking, writing, reading, and using nonverbal language.

Listening with empathy and patience to the questions and concerns of parents and patients is a must. Although the dentist must spend sufficient time with parents to fulfill professional responsibilities, ensure that informed consent is given, and establish trust, a well-trained auxiliary can deepen the parent- and patient-practice relationship. A staff member can listen to problems, interpret concerns, give home care instructions, reiterate answers to parents' and patients' questions, and help parents and patients understand office procedures.

Dentists and auxiliary personnel should speak in layperson's terms so that parents and patients can understand the diagnosis and recommended treatment. Informed consent may require that forms and instructions be read aloud to parents who cannot read or comprehend adequately.

Written health history forms, health status updates, patient instructions, preventive literature, and other paperwork must look impeccable and be easily read and understood. Completed forms (including parents' comments) must be read and understood by the dentist and team members. Diagnoses previously recorded on the patient's chart and treatment notes from the last appointment should be read by the dentist and auxiliary personnel before beginning treatment at each appointment.

Unspoken messages from parents such as facial gestures and other body language must be interpreted with sensitivity. Parents judge a practice, accept treatment, and refer others based more on good feelings than on the quality of dentistry received. Skillful communications and office ambiance help generate these good feelings.

OPERATIONAL SYSTEMS

SCHEDULING

Scheduling controls the activity and flow of the dental office, increases productivity and efficiency, and allows the dentist to work at a comfortable pace. Efficient appointment scheduling makes the office pleasant, whereas poor scheduling creates a hectic, stressful environment for patients and staff members.

The telephone links patients to the practice, and it must be answered in a cheerful and genial manner. The attitude of the appointment secretary can give a positive impression, making scheduling a pleasant experience, or create a negative one, resulting in an appointment's not being made. The appointment secretary should listen carefully to each parent or patient and try to be accommodating while offering two choices of appointment times. If neither time is convenient for the parents, the secretary offers two more until one is accepted. It is inefficient to ask when the patient would like to come because treatment often dictates the length of an appointment and the time of day it can be scheduled.

Because of the complex nature of appointment scheduling, it is essential for a dentist to develop an ideal schedule, a plan toward which the appointment secretary can work. In most situations, an office functions more efficiently when block scheduling is used. With block scheduling, patients with similar treatments are given appointments in a predetermined period. This can work well in any practice, although it is particularly effective in an office that treats large numbers of children. For example, more difficult and lengthy procedures, such as pulpotomies, crowns, composite restorations, orthodontic bandings, and procedures for preschool children 5 years of age and younger, are scheduled early, when the dentist, auxiliary personnel, and patients are fresh. Less difficult procedures such as simple operative treatments, orthodontic adjustments, and hygiene appointments for older children may be scheduled in the afternoons. Appointments just before lunch and in late afternoon should be reserved for the simplest procedures such as suture removals, simple extractions, eruption checks, and posttrauma examinations.

Sedated and difficult patients and those needing hospitalization require the dentist's undivided attention, so their treatment should be scheduled weekly at a particular time. Once a month or as the need arises, a period should be set aside for oral health appointments for infants. Infants can be checked for evidence of nursing caries or developmental problems while parents receive instructions about preventive care and diet. Teenagers' appointments may be scheduled together, perhaps several afternoons monthly. Many adolescents transfer

to another dentist if they find that their appointments are repeatedly scheduled at the same time as those of small children.

As part of the checkout process, the next appointment for treatment or hygiene should be made. Although it may seem efficient to schedule a series of visits for the patient who needs several restorative appointments, it is preferable to schedule only one appointment at a time. Scheduling several appointments may cause some parents or patients to disregard keeping one or more in the series. Also, scheduling serial appointments can fill the appointment book for weeks and months, and thus allow little time for booking new patients or patients with emergencies.

Despite the best efforts of the appointment secretary, a few individuals will refuse to keep appointments for treatment or hygiene. So that these patients are not lost, their names must be recorded in the computer. These patients should be contacted periodically and invited to reschedule. As mentioned previously, entering a treatment plan for every patient at every visit or, if no treatment is needed, entering the next hygiene appointment, assures that patients are not lost.

RECARE SYSTEM

The foundation of any successful dental practice is the recare system. The recare system provides a constant source of patients in a structured maintenance program that safeguards their oral health and stresses the fact that the dentist and staff care a great deal about them. The term *recare is* preferable to the frequently used word *recall. Recare* connotes ongoing, continual attention, whereas *recall* suggests an occasional check for problems (e.g., the recall of an automobile for repair or replacement of faulty parts).

The most effective and efficient recare systems that retain the most patients are those in which the next hygiene appointment is scheduled 6 months in advance at the conclusion of the present appointment. A reminder postcard may then be mailed 3 to 4 weeks before the appointment. Recare systems in which a postcard is sent to the parents requesting them to call the office to schedule an appointment are often ineffective. People forget to respond, and the postcards are frequently lost. Systems that rely on the appointment secretary's calling all recare patients can be productive but are inefficient because it can take many hours to reach all patients.

There are two types of preappointed recare systems: *preselected* and *preassigned.* When implementing either system, the appointment secretary should emphasize to parents and patients that the preappointed recare system is for their convenience and is more likely to allow an appointment to be made 6 months hence at the time that they prefer.

In *preselected* systems, at checkout, the parent chooses the next recare appointment. In 6 months, a notification with the day, date, and time is generated and mailed.

In *preassigned* systems, the patient may be told at checkout that a notice will be mailed 3 to 4 weeks before the next appointment with the day, date, and time included. The appointment may be at approximately the same time as the present visit or it may alternate to be during or after school. The parents are encouraged to call if they have questions when they receive the recare appointment, but the staff member should avoid mentioning a change of appointment times because that terminology invites them to make changes. *After* the patient leaves, the next appointment is assigned, with the time needed for prophylaxis, behavioral management, medical complications, and time of the current appointment taken into consideration. This method is especially helpful in a busy practice because it speeds the checkout process and allows more scheduling flexibility.

The appointment date is entered in the computer, and recare cards can then be generated just before mailing. Approximately 3 to 4 weeks before an appointment, recare reminders are printed and mailed. If the recare appointment must be changed for the dentist's convenience, the change can be made before the notification is mailed. If the time was preassigned, no call is necessary; the appointment time is simply corrected by staff. If the parent selected the recare appointment time, rescheduling should be done by telephone.

On receipt of the recare card, parents who find the time inconvenient usually call to reschedule. A confirmation telephone call should be made 1 or 2 days before the appointment. The staff person calling to confirm should be prepared to reschedule appointments if changes are requested.

The wording of the recare message must emphasize the importance of regular hygiene appointments. Box 29-1 shows an example of an appropriate message for a recare card in a pediatric dental office.

Flexibility to accommodate appointment changes initiated by the office can be built into the preappointed system by leaving 1 or 2 days unscheduled toward the end of each month. If the time is not needed for these changed appointments, it may be booked 1 or 2 weeks ahead for regular restorative appointments, new examinations, and rescheduled appointments initiated by patients.

CHECKING OF RECARE EFFECTIVENESS

The recare program is the life-support system for any practice. The recognized minimum goal is for at least 8 of 10 active patients to return regularly at the prescribed frequency for hygiene appointments (i.e.,

00X29-1 Example of Recare Postcard Message

> At this time, we will thoroughly examine all hard and soft tissue for dental disease, check for proper growth and development, clean teeth and apply special fluoride. Remember, if sealants are in place, regular 6-month examinations are necessary for proper maintenance.
>
> Changed appointments affect many people. If you cannot keep this appointment, please call at least 24 hours in advance so that this time may be given to another patient. We look forward to seeing you and your child on the above date.

80% effectiveness). The effectiveness of the recare system should be checked at least quarterly.

Accuracy depends on a close count of charts of patients who have been seen in the previous 18 months (active patients), except those whose treatments could be classified as single-visit emergencies (Clinical Dental Terminology [CDT] code No. D0140) who are not enrolled as regular patients.

The following example illustrates a quick, easy way to check the effectiveness of a recare system:

4000 active patients - 6 months (recare frequency) = 667 recare appointments per month for 100% effective system

Actual average for 3 months = 300 recare appointments per month
300 actual - 667 potential = 45% effective system

Goal = 80% or more of active patients returning for regular recare appointments
4000 active patients x 0.80 = 3200 patients

3200 patients = 6 months (recare frequency) = 533 recare appointments per month as 80% goal

533 recares per month as 80% goal
-300 recares per month actual average

233 additional recares per month

233 x 5$ average recare fee x 12 = thousands of dollars monthly and annually in increased income generated from current patients—plus better care and service to patients

After the effectiveness and the monthly minimum goal are determined, the dentist and staff can work toward the goal of 80% of active patients returning regularly for recare. Problems with the system should be listed and analyzed, and ways to improve the system must be planned and implemented. Many practitioners find that 60% or more of restorative and orthodontic treatment needs are diagnosed during hygiene examinations, which reinforces the importance of an effective recare system in any office.

BROKEN APPOINTMENTS AND PURGING OF PATIENT CHARTS

A phrase frequently used in dental offices is *"patients who drop between the cracks."* This phrase describes patients whose treatment is diagnosed and presented but who leave the office without scheduling and fail to call back for an appointment. It can also apply to patients whose treatment is not rescheduled after a broken or canceled appointment. Patient care suffers, and a great deal of money may be lost annually in a practice that lacks a viable system for contacting these patients.

If a patient fails to keep an appointment, a staff member should telephone the parents within 10 to 15 minutes, certainly within 24 hours. If the parents cannot be reached by telephone, a message should be sent asking them to contact the office. If the appointment cannot be rescheduled, the patient must be listed or flagged in the computer. Later, a broken appointment list can be printed so that patients with failed appointments can be recontacted.

A patient who has not completed treatment or who has an appliance or prosthesis should receive a letter explaining the dental consequences of not completing treatment or not having periodic adjustment and examination of a device. Such letters should be mailed to the patient's last known address after obtaining a Certificate of Mailing (form 3817) from the post office. A copy of the letter should be kept in the patient's chart with the Certificate of Mailing attached, which provides proof that the letter was mailed and that professional responsibility was fulfilled. A Certificate of Mailing adds nominal cost to first class postage.

It is important to purge patients' charts regularly, at least once a year, preferably in September or October. This is a relatively simple matter if the charts are marked with a yearly date sticker indicating that the patient had a recare appointment within that year. The appointment secretary can pull the charts of patients who were not seen during that year and telephone the parents to stress the importance of regular hygiene appointments. Parents of children with incomplete treatment should be informed of the treatment yet to be rendered and the importance of completing it. Parents with dental insurance should be reminded that benefits for that year will be lost unless they schedule an appointment for their child before year's end. Many people will schedule an appointment to avoid losing insurance benefits. Individuals who refuse to reschedule should be contacted once more before their charts are placed in the inactive file.

Active files, maintained in the accessible office chart shelves, are charts of patients who have received care within the previous 18 months. Inactive files consist of charts of patients seen before the previous 18 months who have been contacted but refuse to reschedule.

Some state laws specify a minimum period after the last treatment date that dental records must be maintained. Regardless of the statutory requirement, it is best to maintain records for as long as possible but certainly no less than 10 years from the last treatment date for adults and up to age 28 for patients treated when they were children. Charts should be kept as evidence against malpractice claims and to answer medical or dental questions that may arise in later years. If storage becomes a problem, records can be microfilmed. Microfilmed records are admissible in most state courts.

PRODUCTION AND COLLECTIONS

A dental practice is a small business, and like any business, it must be profitable. For the practice to operate profitably, high-quality dental treatment must be provided in an efficient manner, and the office must be administered according to recognized sound business principles and procedures.

Every successful business establishes goals. A production goal is one of the most important. It determines the amount of money that must be charged at the current collection rate to reach the break-even point (BEP) and enjoy a profit above and beyond that point.

The best way to calculate a production goal is to determine the BEP, the amount of money needed each year to pay all office expenses, the dentist's compensation (salary or draw plus benefits and retirement funding), and a profit (return on investment) for the owner-dentist. The dentist in an incorporated practice takes a "salary" as an employee of the corporation; in a sole proprietorship (unincorporated practice), the dentist takes a "draw." An example of the calculation of the BEP is as follows:

BEP = Total fixed costs = (1.0 — % variable costs are of gross collections)

Fixed costs include office overhead (e.g., staff salaries and benefits, occupancy costs, administrative costs, taxes, and insurance), the dentist's salary or draw and benefits, and repayment of money borrowed to finance the practice startup or purchase. Variable costs usually include laboratory fees and dental supplies (costs that vary according to patient load) and are typically 8% to 13% of gross collections in pediatric dental practice and 15% to 20% in general practice.

If total fixed costs are $550,000 and variable costs are 9%, then BEP is as follows:

BEP = $550,000 = (1 — 0.09) = $550,000 = 0.91 = $604,396 per year

In this example, to pay fixed costs of $550,000 and to pay variable costs of 9%, the office must collect $604,396 for the year. Assuming that the collection rate is 97% of production (97i of every $1 charged is collected), the practice would have to bill $623,089 to collect $604,396. If the dentist wishes a profit of $50,000 above and beyond salary or draw, the practice would need to produce $674,635 to collect $654,396 and thereby provide $50,000 profit over the BEP of $604,396.

To establish an accurate production goal, the dentist must know the BEP plus desired profit, the number of days worked per year, the collection percentage rate (the percentage of production charges collected), and the show rate (the percentage of patients who keep appointments as scheduled).

For example, if the BEP plus profit is $655,495 per year, the dentist works 190 days per year, the collection rate is 97%, and the show rate is 85% then

$655,495 =190 days = $3450 per day collection goal

$3450 = 0.97 collection rate = $3557 per day production to collect $3450 per day

$3557 = 0.85 show rate = $4185 per day scheduled to produce $3557 per day to collect $3450 per day

Many dentists are more concerned with production than with collections; however, collections control cash flow. One can spend only collected dollars. A very productive practice can have serious financial difficulties if collections do not provide sufficient cash flow to cover the expenses and generate a profit. Collections therefore must be monitored constantly. Most offices find that a 97% collection rate is the minimum acceptable level for private-pay and insurance patients. Of these collections, the over-the-counter portion should be a minimum of 35% and preferably as much as 50% to maintain an adequate cash flow.

PAYMENT POLICY

A clearly stated payment policy presented to parents in a professional manner facilitates collections. Although all team members should be familiar with payment procedures, one staff person should be designated as the patient account coordinator. The patient account coordinator should clarify payment methods with parents, process insurance forms and other third-party payments, and handle billing and delinquent accounts.

The most effective way to familiarize parents with payment procedures is to review methods of payment with them, preferably after the initial examination. Having a written policy ensures that there are no misunderstandings. Box 29-2 is an example of a payment policy.

DENTAL INSURANCE AND OTHER THIRD-PARTY PLANS

The new-to-practice dentist should realize that the parents' possession of dental insurance coverage does not mean that the parents will necessarily seek regular dental care for their children or accept all recommended

29-2 Payment Procedures

We appreciate your allowing us to provide dental care for your child. Because we value our relationship with you and believe that the best relationships are based on understanding, we offer these explanations of payment for services.

- We ask that you pay the cost of the initial examination on the day of that appointment. Because most insurance policies have an annual deductible amount, it is likely that the fee for the examination will not be covered. We will promptly refund your payment if your insurance carrier pays.
- Payment in full by cash, check, or charge card at each appointment as service is rendered is requested. We accept_____ _____ , and_____ charge cards.
- After receipt of a predetermination of benefits from your insurance carrier, we will accept payment for treatment in excess of $ directly from the company. However, if we do not receive payment from the company within 5 weeks after the submission of a claim, you will be expected to pay for all dental services. In the event of duplicate payment, you will be reimbursed. We ask that fees totaling less than $ be paid as treatment is rendered. Your insurance carrier may then reimburse you directly.
- Even if you have insurance coverage, *you* are responsible for payment of your account. We will be happy to help you receive the maximum benefits available under your policy; however, please realize that the relationship is between you, the insured, and your insurance company.
- Payment plans may be set up for our patients without dental insurance. If more than $ of is required, payment may be made in four parts:
  - One fourth at the initial appointment.
  - One fourth 30 days after the initial payment
  - One fourth 60 days after the initial payment
  - One fourth 90 days after the initial payment
- If a payment plan is agreed on, you will be asked to sign a financial agreement. Payments are due when statements are received. A % service charge will be added monthly to the unpaid balance. Computer-prepared statements are mailed monthly, usually by the 25th of the month. If there is a balance on your account, you will receive a statement regardless of the date on which treatment was rendered.
- If insurance is pending, you will receive an interim statement to let you know that the account has not been paid. A service charge will not be made until payment has been received from your insurance carrier.
- If you require credit for longer than 90 days, personal arrangements should be made with your bank or credit union.
- Half the cost of an appliance or prosthesis must be paid when impressions are taken. This procedure is necessary because our office must pay a portion of laboratory costs when appliances are ordered. The remainder of the cost of the appliance is due when the appliance is placed.
- Please be aware that the parent bringing the child to our office is legally responsible for payment of all charges.

We look forward to years of close association with you as we work together to maintain your child's oral health.

Thank you!

treatment. Parents and patients tend to accept services they have been taught to value from practitioners they trust. Dentists and team members must work with all parents and patients to build rapport and to educate them about the value of good oral health beginning in infancy. Only in this way can comprehensive patient care be given and a practice grow to full potential. Insurance can also benefit dental practices because it allows many patients to accept treatment when they could not otherwise afford it.

The dentist and staff members should refer to dental insurance as "assistance" and should never allude to " full benefit coverage." In this way, parents are taught to think of dental insurance as a partial payment method, a help with their payment for care, never meant to pay all fees in full. A written explanation is an excellent way to clarify how dental insurance works to help parents understand their coverage. The information can be enclosed with the new-patient packet mailed prior to the first examination to parents who have dental insurance. Box 29-3 is an example of an explanation of dental insurance.

The dentist should not discount or disregard the portion of fees not paid by an insurance company (i.e., the patient copayment). Doing so may constitute insurance fraud because the dentist has misrepresented actual charges. For example, if a fee for a procedure is $100 and an insurance company pays only $80, do not submit a claim for the full $100 fee if $80 is accepted as payment in full. Submit the $100 claim if the parent is expected to pay the $20 that is not covered.

INSURANCE ASSIGNMENT

A basic decision to be made concerning collections is whether the office will wait for payment of fees from insurance carriers or ask that fees be paid as treatment progresses. An office that waits on payment from

00X29-3 For our Patients with Dental Insurance

The best dental service is based on friendly, mutual understanding. Therefore we invite you to discuss any aspects of your child's dental care or your dental insurance.

Your insurance is a contract between your employer and the insurance company. We cannot influence how much of our fees your insurance will cover. Your benefits are determined by the policy your employer purchased.

You will need to bring the insurance claim form, with your portion of the form completed and signed, to the first visit. We will file the insurance for you. If you have any questions about completing your portions of the claim form, please call our office.

We will appreciate your bringing your insurance benefits booklet also. By reading the booklet together, we can understand your dental benefits (i.e., what your insurance will cover).

We ask that the cost of the initial examination be paid on the day of that visit. Because most insurance companies have an annual deductible amount per family or insured person, the cost of the examination probably will not be covered.

Because there are certain fees or procedures most dental insurance policies do not allow, please expect to pay the portion of the fee not covered at each visit. We will refund any duplicate payment your insurance company happens to make.

Please sign the space provided on your insurance form for payment to be made directly to our office. Otherwise, we will not receive payment, and you will be billed for the treatment.

One copy of the insurance form is a receipt for your records and tax purposes. Please keep copies of all office correspondence for reference with insurance claims.

Please be aware that the parent bringing the child for dental care is legally responsible for payment of all charges, Your obligations are:

- To certify to the insurance company that your treatment was received
- To understand that *you,* and not your insurance company, are responsible to our office for the services rendered

We appreciate your selecting us to provide your child's dental care.

Thank you!

insurance carriers is said to "accept insurance assignment." An office that asks for fees to be paid as treatment is delivered is a "cash practice."

Many new dentists decide between accepting insurance or requesting full payment by determining the prevailing method in their communities. However, several other factors should also be considered when making the decision. For example, a mature pediatric dental practice with a patient load of 40 to 50 or more patients daily may have to process 175 or more insurance claims weekly. This amount of paperwork may necessitate hiring an additional staff person. Furthermore, accepting insurance assignment may delay cash receipt for 6 to 8 weeks when paper claims are filed and 2 to 3 weeks when electronic claims are submitted. The beginning practitioner who decides to wait on assignment of benefits must therefore plan for another source of operating cash while waiting for initial insurance claims to be paid.

On the other hand, many dentists and management experts agree that the acceptance of assignment can be a good marketing tool because parents seem to be more likely to accept recommended treatment for their child if they have to pay only the difference between the actual fee and the amount their dental insurance covers. In addition, parents who are pleased that their insurance benefits substitute for cash are more likely to tell others about this service and the office.

Some offices work with a hybrid system, expecting parents to pay full fees for treatments up to $300 and willing to accept insurance assignment with the parent paying the difference for treatments over $300. In this case, a predetermination should be obtained from the insurance carrier so the office knows how much copayment to collect from the parents as treatment progresses.

INSURANCE REFERENCE GUIDE

An insurance reference guide, a brief reference showing deductible amounts and fees not covered, is a tremendous aid to patients and staff members in offices that accept insurance assignment. Using such a guide allows the patient account coordinator to quickly determine at each visit the portion of the fee to be paid by the parents. Fees not covered and deductibles can then be collected as treatment is rendered. Collecting fees on the day of service increases cash flow and saves preparing and mailing statements for unpaid account balances after insurance payment has been received.

Many dental computer systems have software features that can record insurance payment schedules for numerous carriers. Whether a guide is maintained manually or as part of the computer software, business staff must obtain and update at least annually information about the schedule of benefits provided by the insurance carriers most frequently used.

Information about benefits can be gleaned from insurance booklets that parents are asked to bring to the initial visit. The most important information is the insurance company's name, telephone number, and plan number; the employer's name, telephone number, and contact person; deductible requirements and amount; maximum annual and lifetime benefits; pre-estimate requirements; percent or scheduled amount paid on various procedures, including preventive and orthodontic treatment; and any exclusions.

PREDETERMINATION

Many insurance carriers request the submission of a treatment plan for procedures that will cost $300 to $350 or more. Usually the choice of whether to file a predetermination and the task of doing it rests with the dental office. However, some parents may choose to submit the predetermination based on estimated costs, which allows the dentist to begin treatment before the predetermination is returned by the insurance company. The insurance company determines the amount to be paid according to the contract with the parent's employer. Before the beginning of treatment, the parent can be informed of the amount that the insurance will pay. The form approved by the American Dental Association (ADA), ADA Dental Claim Form Version 2000, also known as a *universal claim form,* is useful in submitting predeterminations and regular claims. These forms may be ordered through the ADA.

A copy of the predetermination form should be kept in a tickler file so that the patient's treatment can be scheduled immediately when clarification of benefits is received from the insurance company. The file must be checked monthly by a staff member. Any predeterminations submitted but not returned by an insurance company should be pursued, because the patient will be waiting for treatment to begin.

FILING CLAIMS

Third-party claims are filed manually using a paper claim form or electronically using the in-office computer. Although electronic claim filing is not allowed by a few third-party payers, electronic claims are widely accepted, are easier, faster, and less expensive to file than paper claims, and usually result in a significantly shorter turnaround time. The faster a claim is paid, the better the cash flow for the dental office. Details of electronic filing should be available from any computer vendor whose system allows such filing.

The team member who handles insurance and other third-party claims must be trained in filing and follow-up because requirements and procedures vary among third-party payers. This person should attend seminars about dental benefits, read management journal articles on the topic, and schedule meetings with insurance companies and other third-party claim processors to better understand how to handle claims.

Filing claims as treatment progresses, rather than waiting to file the total claim after a series of appointments, improves cash flow. In this way, the dentist receives payment as work is performed. Obviously, claims should be filed only for treatment delivered.

A master copy of the insurance form should be signed by the parents and kept in the patient's chart. Many insurance companies require that a new claim form be signed yearly by the insured. Therefore parents with dental insurance should bring a new claim form when they come for the first visit each year or when they change insurance carriers. When a signed claim form is on file, insurance claims can be filed by using the universal claim form stamped "signature on file" and can be mailed immediately or filed electronically. A few insurance companies require signatures on each claim and will not accept stamps. Maintaining a roster of such companies will save much time and prevent misunderstandings.

Some state welfare programs demand that claims be filed on their own claim forms. In these cases, parents must bring a new claim form to each appointment, or a supply of claim forms may be kept in the office. Extra forms are usually available from the appropriate state or federal office (e.g., Medicaid or Title XIX forms are currently available from the EDS Federal Corp.).

HEALTH INSURANCE PORTABILITY AND ACCOUNTABILITY ACT

Historically, the protection of patient privacy has been expected and accepted as an ethical standard in the health care professions. Effective April 2003, it is a legal requirement. The United States Department of Health and Human Services has issued comprehensive privacy regulations (see appendix on p. 750) that apply to all health care providers who transmit (or authorize a third party to transmit on their behalf) protected health information electronically (such as electronic claim forms). The health care provider may use and disclose a patient's protected health information only as the patient permits or as allowed under the privacy rules. In addition, the privacy rules give patients certain rights, such as the right to amend their protected health information. Further details on this act are available on the Department of Health and Human Services web site, http://www. os. dhhs.gov, or by telephone at 877-696-6775.

PENDING INSURANCE CLAIMS

Toward the end of each month, an outstanding insurance claims report should be generated and each overdue claim followed up. If time permits, the patient account coordinator may call the insurance company to inquire about payment. Alternatively, the parent may be called

to pursue the overdue claim. Typically, parents are informed as treatment begins that they will be expected to pay all noncovered fees as well as claims not paid by 5 to 6 weeks after treatment.

DIRECT REIMBURSEMENT

Direct reimbursement, a benefit gaining support among employers and employees nationwide, is an excellent alternative to dental insurance. Under direct reimbursement, the dentist receives payment from the parents as services are rendered. The parent then submits a paid statement directly to his or her employer and is reimbursed for a portion of the fee. The amount of reimbursement is decided by the employer.

MANAGED CARE

Dental benefit organizations fit under the umbrella term *managed care.* Managed care, loosely defined, is an insurance industry term for a system that curbs expenditures in benefit plans by cutting reimbursement levels and transferring financial risk to those providing care.

In managed care it is the payment methods rather than the actual dental treatment modalities that are different. Each managed care system has a specific mechanism of payment for treatment, usually accompanied by some form of utilization review. Dentists who participate in managed care organizations surrender control of their fees for that part of their patient population enrolled in the managed care organization.

The two most widely used types of dental managed care are the preferred provider organization (PPO) and capitation contract dentistry (CCD). A PPO is a network of private practitioners who contract with a plan to provide specific services to a particular patient population at reduced fees. Patients enrolled in a certain PPO must choose a dentist from the organization's list of providers. In CCD plans, the dentist contracts with a plan to provide specific services to enrollees in return for a monthly capitation amount whether or not the enrollees-patients actually make and keep appointments. In other words, the dental office receives a monthly lump capitation payment based on the enrolled number of patients eligible for treatment in that office.

Dentists must weigh many issues, options, and fiscal matters regarding managed care. The ADA has a comprehensive array of resources to help dentists understand and analyze the positive and negative aspects of managed care. Among the services offered is contract analysis, which is available from the ADA through individual state dental societies. Any dentist who is considering signing a contract with a dental benefit organization is well advised to consult with the ADA and his or her own attorney, financial advisor, and management consultant.

DENTAL MANAGEMENT COMPANIES

In addition to the traditional model of solo or group practice, in which the dentist-owner manages the practice, several entrepreneurial dental management models began to come into use within the last 10 to 15 years. Among these are non-dentist—owned practices, franchised practices, and dental practice management companies that often move into publicly owned entities. As with other types of businesses, the specific ownership and management design varies from individual control to group control. Any one of these alternative management systems can have a profound short-term and long-term impact on a dentist's career and lifelong income.

The dentist considering participation in managed care programs that pay reduced fees or pay on a capitated basis or the dentist pondering selling to or working with an entrepreneurial management program that charges a management fee must analyze the financial impact on his or her practice (Table 29-1).

In Table 29-2 the "fee cut" can occur when third-party providers or managed care organizations establish fees rather than the dentist setting his or her own fees. In addition, the management fee charged by entrepreneurial management companies can affect practice income similarly to a fee cut.

FISCAL MANAGEMENT

Fiscal management concerns the way in which the finances of a business are controlled. Fiscal aspects of a dental practice include accounts receivable, fee structure, accounts payable, budgeting, and monitoring methods. In a well-managed practice, the dentist carefully administers each of these aspects.

BILLING AND ACCOUNTS RECEIVABLE

The dentist and team members should establish a regular routine for handling billing; receipt of mail

TABLE 29-1 Impact of Fee Increase on Profitability

CURRENT OVERHEAD %	% FEE INCREASE	INCREASE IN NET INCOME %
65	5	14.3
65	10	28.6
65	20	57.2
60	5	12.5
60	10	25
60	20	50
55	5	11.1
55	10	22.2
55	20	44.4

Courtesy Dr. Charles W. Blair.

TABLE 29-2 Impact of Fee Cut on Production Required to Maintain Same Net Income

CURRENT OVERHEAD %	FEE CUT %	% OF PRODUCTION INCREASE REQUIRED TO MAINTAIN SAME NET
65	5	16
65	10	40
65	20	233
60	5	14
60	10	33
60	20	200
55	5	13
55	10	29
55	20	80

Courtesy Dr. Charles W. Blair.

payments from individuals, insurance companies, and other third-party organizations; and pursuit of delinquent accounts. Consistency in following these procedures is extremely important to ensure effective collection of amounts owed.

When patients do not pay on the day of treatment, a statement with an envelope addressed to the office should be presented at checkout. Regular monthly statements should be mailed by the twenty-fifth of the month to be received before the first, when many people pay bills. Statements should be sent to every person, including those waiting on insurance payments, regardless of when the service was rendered. Receipt of a statement reminds parents that they have outstanding balances. Some dentists prefer to have a fourth of the statements mailed each week to better manage the cash flow. With this method, patients whose last names begin with A through F receive statements the first week of the month, those whose last names begin with G through L receive them the second week, and so on.

Accounts receivable are fees billed but not collected (i.e., all monies owed to a practice by patients). In offices that accept assignment of insurance or allow patients to pay dental fees on a payment plan, 1 to $1^1/_2$ months' gross production is acceptable as the total amount of accounts receivable. When the accounts receivable total is higher than $1^1/_2$ months' production, cash flow suffers, and many accounts become uncollectible.

Many practices suffer from excessive accounts receivable because there is no system for determining the length of arrears and processing overdue (delinquent) accounts. These accounts must be separated into categories of over 30, 60, 90, or 120 days late in payment to organize and simplify the collection of fees and to determine which accounts need priority collection action.

Each dentist must determine when accounts are considered overdue or delinquent. An account on which no payment has been made for 30 to 60 days may be considered overdue. Concentrated collection efforts should begin if no payment has been received after 60 days, the often-used cutoff for delinquency of an account. The staff member acting as patient account coordinator must pursue payment using telephone calls and letters. If there is no response or payment by 90 to 120 days, the account may be forwarded for outside collection action (i.e., sent to a collection agency or bureau, an attorney, or small claims court).

If the patient's treatment is incomplete or the patient has an appliance or prosthetic device, the dentist should seek legal counsel before designating the patient's chart inactive and sending the account for outside collection action. An attorney can best advise on patient abandonment issues, so that potential legal problems can be avoided.

Another excellent source of information concerning patient chart inactivation is the ADA publication *Terminating the Dentist-Patient Relationship: Questions and Anszoers.*[4] Written by the ADA's Division of Legal Affairs, the booklet, publication L204, is available from the ADA, telephone 800-621-8099.

FEES

The following factors must be considered when setting proper fees:

1. The dentist's and assistant's time required to do the procedure or the hygienist's time for hygiene procedures
2. Cost of operations per hour (overhead), including salaries, benefits, rent, supplies, laboratory costs, insurance, taxes, continuing education, infection control, equipment purchases, maintenance, and repairs
3. Prevailing professional fees in the area
4. Profit after debt service

Fees must be reviewed at least annually and, if necessary, raised more than the inflation rate to continue to generate an adequate margin of profit. Most patients expect, accept, and may not even notice small adjustments in fees. Problems may arise, however, when a dentist who is reluctant to increase fees does not do so for several years and then must implement a significant increase of 20% or higher. A hiatus in raising fees while costs of operations continue to increase means lower profits. If this pattern is allowed to continue, a practice can have significant financial difficulties that force a fee increase of 20% to 30% or more. Many patients notice and complain about such a large increase. Therefore it is better to raise fees

gradually year to year to meet inflation and increases in expenditures.

Fee increases should not be announced to patients. An increase simply is made effective on a certain date. The dentist and staff who have pride in the quality of their care and service and therefore in fees will seldom feel challenged about costs. People are willing to pay for care they value and for service that exceeds the norm.

Staff members need some information about why regular fee increases are necessary. Without any idea of overhead costs, the staff may assume that all fee increases go directly into the dentist's pocket, which is far from true. Sharing information about expenses such as laboratory fees, clinical and office supplies, utilities, and taxes allows team members to understand the necessity of regular fee increases.

ACCOUNTS PAYABLE

Accounts payable is the total amount of money owed by the practice for goods and services received. One staff member or the dentist should be responsible for checking supplies received against invoices included with the shipments.

Whereas an invoice is an itemized list of goods received with each supply, a statement is a summary of all invoices for that month. Paying accounts regularly from statements rather than from invoices can help in avoiding mistakes, such as paying the same bill twice, and ensure that the practice maintains an excellent credit rating. Bills should be paid no more frequently than twice a month after statements are checked against invoices.

BUDGET SETTING

Sound fiscal management includes making certain that the business is profitable. As Dunlap and Wagner wrote in *Knowing the Numbers,*

> Business and profits are not dirty words. They are legitimate terms to describe activities directed towards the attainment of a livelihood. Quality cannot exist in the absence of profit. The attaining of profits is an essential part of a dentist's professional responsibility.[5]

There are two ways to increase profitability in any practice: earn more or spend less. Control of expenses (savings) is one of the most underutilized yet valuable tools to increase profitability. Dollars not spent can go directly to the bottom line in the form of profit.

The most effective way to save is to control spending through budgeting. A budget is the translation of the future plans of a business into dollars or the financial portion of the business plan. Basically, it is a plan for the income and expense of the practice.

Budgeting begins when the annual gross production goal is established. The BEP should be calculated, and the desired profit added to determine the total annual production-collection goal.

Remember that the collection percentage rate must be considered when forecasting annual production-collection goals. If, for example, the practice collection rate is 95%, almost $736,842 must be produced to collect $700,000. As previously mentioned, the minimum collection goal should be 97% (i.e., for every $1 of dentistry produced, 97 is collected).

Next, each expense should be scrutinized. Increases or decreases (savings) must be projected based on inflation and increases in practice productivity. Other expense considerations include staffing needs and equipment additions or upgrades.

After income and expenses are calculated, annual totals should be divided by 12 months to determine the monthly production-collection goal and the allowance for each expense category, including staff wages, occupancy costs, laboratory fees, costs of office and clinical supplies, taxes, insurance, and other costs.

In summary, a budget should project income, propose maximums for each category of expense, and forecast the year's profit. Figures should be monitored monthly, quarterly, and annually.

A monthly journal of receipts (collections) and expenses should show each category of expense as a percentage of collections. Box 29-4 gives average expenses commonly seen in pediatric dental practices. Obviously, expenses vary from practice to practice; therefore this list is only a guideline.

PRACTICE MONITORS

Well-managed businesses use numbers as management tools; decisions are based on facts and figures, not guesswork. Well-managed dental practices should use the same criteria. Vital statistics needed for good management include the following:

- Production: per day, week, month, and year to date compared with the same period last year
  - Goal and actual production per hour, day, month, and year
  - Dentist's production
  - Hygienist's production
- Collections: per day, week, month, and year to date compared with the same period last year
  - Goal of 97% or better of production compared with actual collections
  - Collection percentage rate (collections for a period divided by production for the same period)
- Number of hours and days worked per month and year
- Number of active patients seen within past 18 months, not including those seen for single-visit emergencies

BOX 29-4 Categories of Expenses in a Pediatric Dental Practice

Personnel

Salaries—21% to 26% including payroll taxes
Benefits—approximately 2% to 4%

Occupancy—5% to 10%

Lease or note payment
Repairs and maintenance to facility
Insurance on building and contents
Utilities (except telephone)
Janitor/grounds/security

Administrative—5% to 10%

Laundry
Accounting, legal—under 2%
Licenses/permits
Advertising—for team members
Meals for business
Answering machine or service
Office supplies, printing—under 2%
Collection costs/bank charges
Postage
Computer expenses
Taxes
Continuing education—staff
Telephone—1% to 2%
Dues and subscriptions
Miscellaneous
Insurance
Malpractice
Business
Disability

Equipment, furnishings, contingency—4% to 6%

Lease or note for purchase

Clinical supplies—4% to 7%

Off-site laboratory—under 2%

Marketing—2% to 5%

Advertising, yellow pages
Practice brochures and other literature
Giveaways
Web site
% of dental health educator/marketing coordinator's salary

Note: Depreciations and amortization do not directly affect cash flow and are, therefore, not listed. If the dentist prefers, depreciation on the office building can be monitored as an "occupancy" expense; depreciation on equipment and amortization for software can be monitored as part of the "equipment, furnishings, contingency" expense.

Number of hygiene patients seen per month and year
- Goal: 80% or more of active patients enrolled in the recare system

Number of patient appointments *scheduled* in a day, week, and month
Number of patient appointments *kept* in the same day, week, and month
Show rate (percentage of kept appointments, calculated by dividing the number of patients who kept appointments as made divided by the number scheduled for that same period times 100)
- Show rate goal: 85% or better in pediatric dental offices

- Accounts receivable each month
  - Total accounts receivable and increase or decrease since previous month
  - At 30 days
  - At 60 days
  - At 90 days
  - At 120 days and over
  - Bad-debt write off
  - Welfare and other third-party adjustments
  - Professional-courtesy adjustments
- Overhead: per month, per year (average: 56% to 60%, not including compensation for doctors in pediatric dental practices); in addition, the percentage of collections spent for each of the following categories of expense:
  - Personnel costs
  - Occupancy costs
  - Administrative costs
  - Clinical supplies
  - Laboratory fees
  - Marketing
  - Equipment, furnishings, and contingency fund
  - Total compensation for dentist in actual dollars and as a percentage of collections

DAILY STATISTICS

A short form completed daily by the business staff keeps the dentist informed of daily activities. Fig. 29-5 illustrates the information to be monitored daily.

MONTHLY STATISTICS

Monthly statistics can be collected in a single-sheet format. Even if a practice is computerized, assembly of these data on a single sheet allows quick evaluation and comparison with previous similar time periods. Fig. 29-6 is an example of a monthly monitoring form.

AVERAGES, MEDIANS, AND GOALS IN A PEDIATRIC DENTAL PRACTICE

The following statistics are gleaned from scores of private pediatric dental practices. The practices range in duration from a few years to 30 years or more. Regional differences are seen; the following figures, as the title of this section suggests, represent averages and goals most typical of the specialty in most, but not all, regions of the country.

Production

- Goal: Dentist plus hygiene department per-hour charges—$500 to $550 per hour or more without full banded orthodontics; $750 to $800 per hour with orthodontics
- Average production per day—$4000 to $5000
- Average production per year—$800,000 to $900,000

Daily Activities

Date:_____

	Restorative	Orthodontic
No. patients scheduled	_____	_____
No. patients seen	_____	_____
% patients seen (show rate)	_____	_____
Combined total no. scheduled for restorative and for orthodontic		_____
Combined total no. seen for restorative and for orthodontic		_____
Combined % seen, not including emergencies (show rate)		_____
No. broken appointments	_____	_____
No. rescheduled	_____	_____

	Hygiene Activity
No. patients scheduled	_____
No. patients seen	_____
% patients seen (show rate)	_____
No. broken appointments	_____
No. rescheduled	_____

Production	$_____
Daily goal adjustment	$_____
Collection/deposit	$_____

f I G 29-5. Daily activities form.

- Gross production = every dollar charged at full fees
- Net production = gross production minus amount adjusted off for welfare, managed care, professional courtesy, etc.

Collections

- Minimum goal: 97% (collections for a period divided by production for that same period) of net production
- Over-the-counter goal: 35% to 50% of charges made each day or month; includes mail payments
- At $850,000 annual production, every 1% that collections is increased = $8500
- Gross collections = every dollar collected
- Net collections = gross collections minus amount adjusted off for returned checks due to nonsufficient funds (NSF), patient refunds, etc.

Accounts Receivable

- Goal: not more than 1 to $1^1/_2$ months' gross production
- Current—50% to 60% of total
- 30 days—15% to 20% of total
- 60 days—10% to 15% of total
- 90 days—under 10% of total
- 120+ days—to outside collection
- Do not enter full orthodontics case charges on day of banding, enter down payment only. Charging full fee inflates accounts receivable, and those fees are not yet earned.

Appointments

- Average patients per day = 55 to 65
- Average patients per month = 1000 to 1100
- Average new patients per month = 60 to 70 (count comprehensive initial examination [D0150]); do not count DO140 (emergency treatment for patients who do not return for a comprehensive examination).
- Average recare patients per month = 400 to 450

Average Work Times Per Week, Month, and Year

- Office hours—phone covered by staff 36 to 38 hours per week
- Dentist's hours with patients—32 to 36 hours per week

Monthly Practice Monitor for _____

Monthly statistics collected in single-sheet format allow quick evaluation and comparison to previous similar time periods.

Pediatric Dentistry Statistics		This Mo.	Last Mo.	Year to Date	Same Mo. Last Year	Last Year to Date
Days worked						
Hours worked						
Dentist production plus hygiene production						
Private and insurance production						
Welfare and other reduced fee production						
Welfare production to total production ratio (%)						
Production per day						
Production per hour						
Total collections						
Private and insurance collections						
Welfare and other reduced fee collections						
Collection to production ratio (%)– private and insurance						
Collection to production ratio (%)– welfare and reduced fee						
No. and date statements mailed						
Accounts receivable (A/R) total						
A/R aging $	Current					
	30 days					
	60 days					
	90 days					
	120 days					
	In collections					
Total adjustments (write-offs)						
New patient appointments						
Recare patient appointments						
Total patient appointments						
Show rate (% of kept appointments)						
Total patients leaving practice						
If full-banded orthodontics is part of the practice, complete this section also.						
Orthodontic production						
Orthodontic collections						
Contracts receivable C/R total						
C/R aging $	Current					
	30 days					
	60 days					
	90 days					
	120 days					
	In collections					
No. new patient exams						
No. recall for revaluation						
No. records taken						
No. bands and bonds						
No. periodic adjustments						
No. debandings						
No. leaving incomplete						

f **I G 29-6.** Monthly practice monitor form.

- Days per month—17 days
- Hours per month—130 to 138 hours
- Days per year—190 to 195 days

Treatment and Hygiene Minimum Goals

- Show rate: 85% or higher (number of patients who come *as scheduled* divided by number of patients scheduled for that period, *not* counting last-minute fill-ins or emergencies)
- Goal: case acceptance/case presentations = 80% to 90% (treatment delivered divided by treatment proposed)
- Goal: treatment completion/treatment scheduled = 88% to 93% (treatment completed divided by treatment scheduled)
- Goal: 70% to 80% of active patients enrolled in recare system

Example *of* Check *of* Recare System Effectiveness

- 4000 active patients = 6 months = 667 recares per month for 100% effective system
- Actually averaging 350 recares per month
- 350 = 667 = 52% effective system
- Goal: 80% effective system

- 4000 × 0.80 = 3200 active patients
- 3200 ÷ 6 months = 533 recares per month for 80% effective system

Effects of 80% Effective System on Practice Income

$$\frac{\begin{array}{r}533 \text{ recares per month—80\% goal}\\ -350 \text{ actual recares per month}\end{array}}{183 \text{ more recares per month if goal met}}$$

183 × $90 average recare fee = $16,470 per month increased production

$16,470 per month production × 12 months = $197,640 per year increased production

TYPICAL RATIOS IN A PEDIATRIC DENTAL PRACTICE

Overhead. Fifty percent to 60% of collections, not including dentist's compensation. Count an associate's compensation as part of dentist's compensation, *not* as part of staff compensation.

Hygiene Department Production. Typically 40% to 60% of total practice production.

Example:

$$\frac{\text{Hygiene dept. production}}{\text{Total practice production}} = \frac{\$340{,}000}{\$850{,}000} = 40\%$$

Note: The hygiene department production usually includes the hygiene examination and all procedures performed by the hygienist (and hygiene assistant if one is used), such as prophylactic treatment, fluoride application, radiography, and sealant application. A case can be made that the dentist performs the examination and reads the radiographs and therefore should get full or partial production "credit" for those procedures. More typically, however, these are considered hygiene production.

If an associate dentist is employed, compensated on a percentage of his or her collections on production, the examination is usually credited to the dentist's production. Whether radiograph production is credited to the dentist or to the hygiene department is negotiable.

Hygiene Compensation

$4 to $6 hygienist's production to $1 hygienist's compensation.

$$\frac{\text{Hygiene production}}{\text{Hygiene compensation}} = \frac{\$318{,}000}{\$53{,}000} = \frac{6}{1}$$

6% of total practice production-collections

Note: If the ratio falls below $1 compensation to $4 production, it usually indicates poor hygiene department productivity, a poor recare system, too many hygienists or assistants allowed to perform prophylactic procedures, overly long hygiene appointments, or hygiene fees that are too low.

Chairside Assistants' Compensation

$8 to $9 total practice production to $1 chairside assistant compensation.

Example:

$$\frac{\text{Assistants' compensation}}{\text{Total practice production}} = \frac{\$100{,}000}{\$850{,}000} = \frac{\$1.00}{\$8.50}$$

= 11.8% of total practice production-collections

Business Staff Compensation

$9 to $10 total practice production to $1 business staff compensation.

Example:

$$\frac{\text{Business staff compensation}}{\text{Total practice production}} = \frac{\$93{,}000}{\$850{,}000} = \frac{\$1.00}{\$9.14}$$

= 10.9% of total practice production-collections

Number of Business Staff. Typically a practice needs one full-time business staff member for each $15,000 to $20,000 of monthly practice production.

Example: $850,000 production ÷ 12 months = $70,833 per month
Production needs 3 to 4 business staff depending on whether insurance is accepted and how much bookkeeping is done by business staff

Staff Compensation Including Wages, Payroll Taxes, and Benefits. Pediatric dental practices need more staff members per dollar of production than do general practices because of the much heavier volume of patients.

For the example given earlier, annual staff compensation breaks down as follows:

Hygienist	$53,000
Assistants	$100,000
Business staff	$93,000
	$246,000 staff compensation

$$\frac{\$246{,}000 \text{ staff compensation}}{\$850{,}000 \text{ collections}} = 28.9\%$$

Total staff compensation in pediatric dentistry averages 25% to 30% or 31% of collections. Compensation includes wages, payroll taxes, and benefits. Pediatric dentists spend in staff compensation what is saved in laboratory fees; general practitioners spend in laboratory fees what is saved in staff compensation.

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CHAPTER

30

Community Oral Health

KAREN M. YODER

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CHAPTER OUTLINE

GUIDANCE FOR COMMUNITY-BASED PROGRAMS
- Healthy People 2010 National Health Objectives
- Oral Health in America: A Report of the Surgeon General
- Oral Health Program Guidelines

COMMUNAL WATER FLUORIDATION AND NEW RECOMMENDATIONS FOR THE USE OF FLUORIDE FOR CARIES PREVENTION

PROMOTION OF THE USE OF DENTAL SEALANTS IN COMMUNITY-BASED PROGRAMS

ASSESSMENT OF COMMUNITY ORAL HEALTH NEEDS

UTILIZATION OF DENTAL SERVICES AND ACCESS TO CARE
- Behavior Risk Factor Surveillance System
- Public Funding of Dental Care for Underserved Children
- Medicaid
- State Children's Health Insurance Program
- Children's Special Health Care Services

ACCESS TO INFORMATION ABOUT CHILDREN'S ORAL HEALTH

DEVELOPMENT OF COMMUNITY PARTNERSHIPS

SUMMARY

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he task of promoting optimal oral health for communities is a rewarding and daunting challenge for dental professionals, whether involved in pediatric dentistry, general practice, or dental public health. Unlike dentists in private practice, who provide services for patients one by one, dentists engaged in community oral health improvement adopt new techniques and build community partnerships to strengthen the possibilities for success for all members of the community. The rewards are great. Those who years ago fought to introduce water fluoridation can now look with pride at the dramatic difference that has occurred in oral health status. Those who are now developing innovative ways to take dental services to underserved children in rural and inner city locations will undoubtedly feel the satisfaction of having accomplished a meaningful goal.

Pediatric dentists and general dentists have the capability of enhancing the oral health of groups of people in the communities in which they practice. To fully understand dentists' role in influencing healthful public policy, developing oral health programs, and contributing to the understanding of the oral health needs of specific population groups, one must have back-ground information on the resources, systems, and information currently available. Collaboration with dental public health professionals is a constructive way to begin a community oral health program. Under-standing the role and skills of these professionals is basic to success.

Community oral health is a component of the larger field of public health. The core functions of public health at all levels of government are assessment, policy development, and assurance.' The *assessment* role of public health requires systematic collection and analysis of information about the health of communities. The information to be assessed includes statistics on health status and health needs, and epidemiologic data on health problems. The second core function, *policy development,* ensures healthful public policy by promoting the use of scientific knowledge in decision making about health issues and by conveying science-based information to decision makers. *Assurance,* the third core function of public health, strives to provide the services necessary to maintain the health of the community. These health services can be provided by the private or the public sector or by a coalition of sources, and should include subsidized or direct provision of necessary personal health services to those unable to pay for them.

The art and science of dental public health enables community-based approaches to oral health promotion and fosters skills necessary for the task. Dental public health is defined by the American Board of Dental Public Health as the science and art of preventing and controlling dental diseases and promoting dental health through organized community efforts. It is that form of dental practice that serves the community as a patient rather than the individual. It is concerned with the dental education of the public, with applied dental research, and with the administration of group dental care programs as well as with the prevention and control of dental diseases on a community basis.'

Specialists in dental public health are leaders in organizing programs to enhance community oral health and can be a resource for private practitioners interested in developing oral health programs for their communities. The fundamental attributes of the dental public health specialist include the following:

- Training as a dentist
- Demonstration of public health values, which essentially means viewing health issues as they affect a population rather than an individual and putting particular emphasis on prevention, the environment in its broadest sense, and service to the community
- Leadership characteristics, such as the ability to influence health policies and practice through research, education, and advocacy; to help articulate a vision for the profession (American Association of Public Health Dentistry); to negotiate and resolve conflicts; etc.
- Subscription to the code of ethics set down by the American Association of Public Health Dentistry

The skills that dental public health specialists can bring to program planning, implementation, and evaluation include the ability to:

- Plan oral health programs for populations
- Select interventions and strategies for the prevention and control of oral disease and promotion of oral health
- Develop resources and implement and manage oral health programs for populations
- Incorporate ethical standards into oral health programs and activities
- Evaluate and monitor dental care delivery systems
- Design and understand the use of surveillance systems to monitor oral health
- Communicate and collaborate with groups and individuals on oral health issues
- Advocate for, implement, and evaluate public health policy, legislation, and regulations to protect and promote the public's oral health
- Critique and synthesize scientific literature
- Design and conduct population-based studies to answer oral and public health questions

GUIDANCE FOR COMMUNITY-BASED PROGRAMS

HEALTHY PEOPLE 2010 NATIONAL HEALTH OBJECTIVES

For those making decisions related to community-based programs to improve oral health, *Healthy People 2010,*[3] developed by the Centers for Disease Control and Prevention, Health Resources and Services Administration, National Institutes of Health, and Indian Health Service, provides guidance in selecting which oral health issues to address. *Healthy People 2010* provides a set of measurable oral health objectives and baseline data against which program outcomes can be measured. The oral health objectives are part of a much larger document setting forth objectives for all areas of health. *Healthy People 2010* was designed to be a roadmap for improving the health of all people in the United States. Many other countries have national health objectives specific to their populations. *Healthy People 2010* targets the goals of increasing quality and years of healthy life and eliminating health disparities. It identifies 10 leading health indicators and 29 focus areas. Oral Health is focus area 21. Each component is stated in general terms and is followed by baseline data for various racial and ethnic groups. Several objectives contain subsections that specifically address children's oral health issues. The following are the oral health objectives set forth in focus area 21 of *Healthy People 2010:*

21-1. Reduce the proportion of children and adolescents who experience dental caries in their primary or permanent teeth. Target: 11%.

21-2. Reduce the proportion of children, adolescents, and adults with untreated dental decay. Targets vary by age category.

21-3. Increase the proportion of adults who have never had a permanent tooth extracted because of dental caries or periodontal disease. Target: 42%.

21-4. Reduce the proportion of older adults who have had all their natural teeth extracted. Target: 20%.

21-5. Reduce periodontal disease. Targets: gingivitis, 41%; destructive periodontal disease, 14%.

21-6. Increase the proportion of oral and pharyngeal cancers detected at the earliest stage. Target: 50%.

21-7. Increase the proportion of adults who report having had an examination to detect oral and pharyngeal cancer in the previous 12 months. Target: 35%.

21-8. Increase the proportion of children who have received dental sealants on their molar teeth. Target: 50%.

21-9. Increase the proportion of the U.S. population served by community water systems with optimally fluoridated water. Target: 75%.

21-10. Increase the proportion of children and adults who use the oral health care system each year. Target: 83%.

21-11. Increase the proportion of residents of long-term-care facilities who use the oral health care system each year. Target: 25%.

21-12. Increase the proportion of children and adolescents under age 19 years with a family income at or below 200% of the federal poverty level who have received preventive dental services during the previous year. Target: 57%.

21-13. Increase the proportion of school-based health centers that have an oral health component. No target established.

21-14. Increase the proportion of local health departments and community-based health centers, including community centers and those serving migrants and the homeless, that have an oral health component. Target: 75%.

21-15. Increase the number of states (including the District of Columbia) that have a system for recording and referring infants and children with cleft lips, cleft palates, and other craniofacial anomalies to craniofacial anomaly rehabilitative teams. Target: all states and the District of Columbia.

21-16. Increase the number of states (including the District of Columbia) that have an oral and craniofacial health surveillance system. Target: all states and the District of Columbia.

21-17. Increase the number of tribal, state (including the District of Columbia), and local health agencies that serve jurisdictions of 250,000 or more persons that have in place an effective public dental health program directed by a dental professional with public health training. No target established.

Additional objectives related to oral health are found in other focus areas of *Healthy People 2010.* The following are selected focus areas that include objectives related to oral health: Access to Quality Health Services (focus area 1), Cancer (focus area 3), Diabetes (focus area 5), Educational and Community-Based Programs (focus area 7), Health Communication (focus area 11), Heart Disease and Stroke (focus area 12), Immunization and Infectious Diseases (focus area 14), Injury and Violence Prevention (focus area 15), Maternal, Infant, and Child Health (focus area 16), Nutrition and Overweight (focus area 19), Public Health Infrastructure (focus area 23), and Tobacco Use (focus area 27).

*ORAL HEALTH IN **AMERICA:** A REPORT OF THE **SURGEON GENERAL***

Understanding which populations to target in community oral health programs is basic to achieving measurable outcomes. Recent publications have given precise direction to community oral health initiatives. *Oral Health in America: A Report of the Surgeon General,*[1] released in May 2000, described the impact of oral disease on people and communities and the interrelationship of general and oral health. The report also pointed out disparities in access to oral health care among specific populations. These are the major findings of the Surgeon General's report:

- Oral diseases and disorders in and of themselves affect health and well-being throughout life.
- Safe and effective measures exist to prevent the most common dental diseases—dental caries and periodontal diseases.
- Lifestyle behaviors that affect general health such as tobacco use, excessive alcohol use, and poor dietary choices affect oral and craniofacial health as well.
- Profound and consequential oral health disparities exist within the U.S. population.
- More information is needed to improve America's oral health and eliminate health disparities.
- The mouth reflects general health and well-being.
- Oral diseases and conditions are associated with other health problems.
- Scientific research is key to achieving further reductions in the burden of diseases and disorders that affect the face, mouth, and teeth.

A 2000 report of the U.S. General Accounting Office[5] provided documentation for the Surgeon General's report on oral health and described the extent to which dental disease is a chronic problem among many low-income and vulnerable populations. Poor children have five times more untreated dental caries than children in higher-income families, and poor adults are much more likely to have lost six or more teeth to decay and gum disease than higher-income adults. Dental problems result in pain, infection, and millions of lost school days each year. National survey data showed that fewer low-income children visit the dentist on an annual basis; about 36% of 6- to 18-year-olds living at or below the federal poverty level had visited a dentist in the preceding year compared with about 71% living in families with incomes higher than 400% of the federal poverty level. Another indicator of the use of dental care by children is the prevalence of dental sealant application. The General Accounting Office document reported that the poorest children were furthest away from the national health objectives as defined in a *Healthy People 2010*[3] goal calling for 50% of children to have sealants on permanent molars. Only 12% of children aged 6 to 14 years living at or below the federal poverty level had sealant on at least one tooth, roughly one-third the prevalence among children in higher-income families.

The report of the Surgeon General inspired the beginning of a Partnership Network Group dedicated to developing a National Oral Health Call to Action.[1] The two major goals of the call to action are (1) to eliminate oral health disparities and (2) to improve quality of life. The proposed vision is to promote general health and well-being by promoting oral health through the formation of critical partnerships at all levels. The key action elements of the national oral health call to action build on the five major components described in the Surgeon General's report on oral health and include two additional elements identified at the 2001 Partnership Network conference:

- To change perceptions regarding oral health and disease so that oral health becomes an accepted component of general health
- To accelerate the building of the scientific and evidence base and apply science effectively to improve oral health
- To build an effective health infrastructure at the local, state, and national levels that meets the oral health needs of all Americans and integrates oral health effectively into overall health
- To strengthen and expand oral health research and education capacity
- To ensure the development of a responsive, competent, diverse, and flexible workforce
- To remove known barriers between people and oral health services
- To use public-private partnerships and build on common goals to improve the oral health of those who still are affected disproportionately by oral diseases

ORAL HEALTH PROGRAM GUIDELINES

Guidance for developing community-based programs that specifically address the general and oral health needs of infants, children, and adolescents can be found in the Bright Futures publications. The Bright Futures guidelines were developed by the Maternal and Child Health Bureau of the Health Resources and Services Administration with additional program support from the Medicaid Bureau of the Health Care Financing Administration. Bright Futures publications attempt to improve health outcomes of children by enhancing health professionals' knowledge, skills, and practice of developmentally appropriate health care within the context of family and community. The guidelines

are designed to foster partnerships between families, health professionals, and communities, and to increase family knowledge, skills, and participation in health promotion and prevention activities.

Bright Futures health supervision guidelines are unique and especially useful for the development of community oral health programs because they view health from a developmental perspective and describe a continuum of care as children progress through childhood milestones. Bright Futures guidelines provide practical tools and materials for technical assistance and training. The guidelines were updated and revised in 2000 to incorporate current scientific knowledge in health practice. *Bright Futures in Practice: Oral Health'* provides information on oral health supervision for infants, children, and adolescents; risk assessment; outcomes measurement; and improvement in the accessibility of oral health supervision. It also includes helpful information about the essential components of oral health.

COMMUNAL WATER FLUORIDATION AND NEW RECOMMENDATIONS FOR THE USE OF FLUORIDE FOR CARIES PREVENTION

The single most influential dental public health measure to date has been the fluoridation of communal water supplies. Children's and adults' oral health has improved dramatically since the introduction of water fluoridation more than 50 years ago and the use of fluoridated dentifrice. Early studies of community water fluoridation showed caries reductions of 50% to 60%. More recent estimates are lower: 18% to 40%. This decrease is probably caused by exposure to fluoride from other sources such as fluoride dentifrice. Food and beverages processed in fluoridated areas but consumed in nonfluoridated areas spread some of the benefit of fluoridation to nonfluoridated communities through a "halo" effect. Therefore the dental caries differential between fluoridated and nonfluoridated communities has decreased. Water fluoridation also reduces the disparities in caries experience among poor and nonpoor children.'

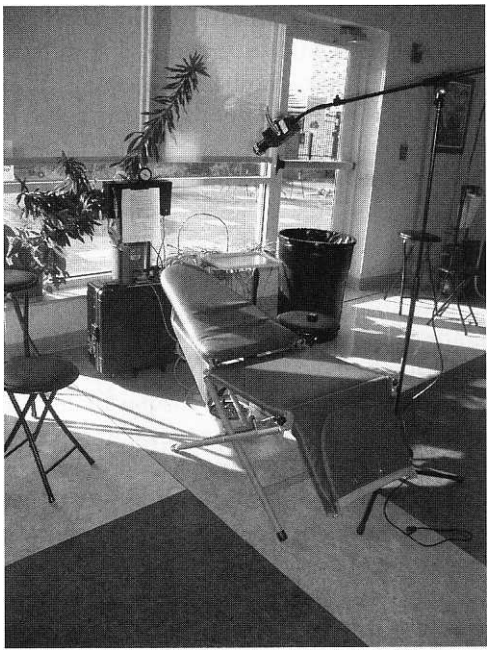
The Centers for Disease Control and Prevention's " Recommendations for Using Fluoride to Prevent and Control Dental Caries in the United States," published in August 2001, made it clear that the beneficial effect of fluoride is predominantly posteruptive and topical, and that, to achieve this effect, fluoride must be available in the right amount in the right place at the right time. This is a reversal of previously held beliefs. The message is slowly reaching practitioners and the public that fluoride works primarily after teeth have erupted rather than by strengthening enamel through incorporation of fluoride into the developing tooth structure. Fluoride is especially effective in preventing dental caries when low concentrations of fluoride are constantly present in the mouth in plaque and saliva. This new information refutes the traditional belief that water fluoridation is primarily beneficial for children and supports the view that water fluoridation has posteruptive benefits for adults as well. The Centers for Disease Control and Prevention strongly encourage that water fluoridation be continued and extended to more communities.

Additional Centers for Disease Control and Prevention recommendations for the use of fluoride that specifically affect children include the following:

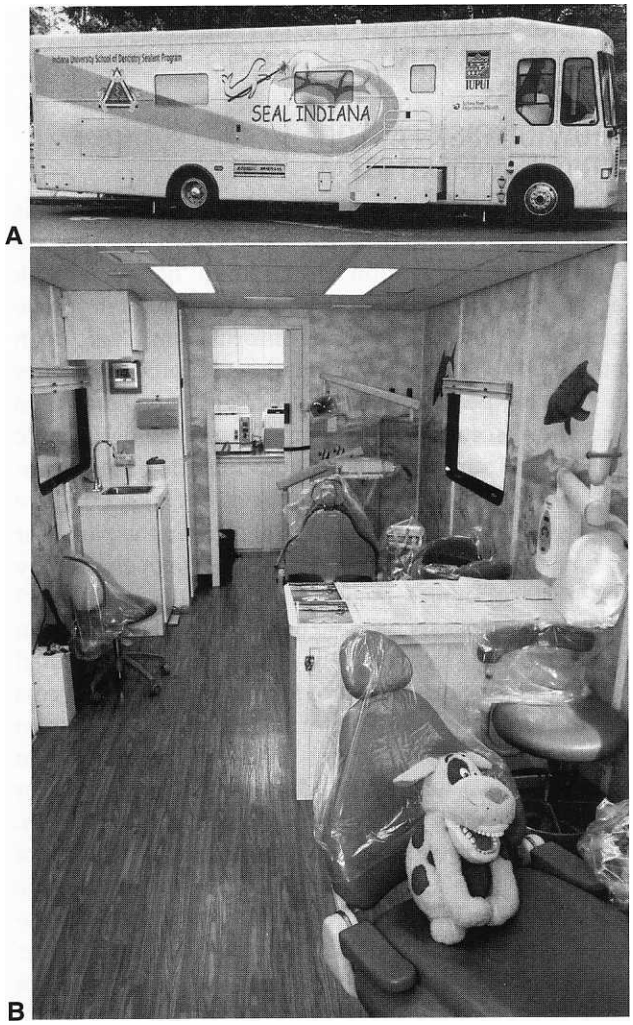
- Parents and caregivers should be counseled regarding the use of fluoride toothpaste by young children, especially those younger than 2 years of age.
- Fluoride supplements should be prescribed judiciously (this recommendation is especially pertinent in light of the new understanding that fluoride's predominant effect is posteruptive).
- The use of fluoride toothpaste should be supervised in children younger than 6 years of age.
- An alternative source of water should be obtained for children 8 years of age or younger whose primary drinking water contains more than 2 ppm fluoride.
- The use of small amounts of fluoride toothpaste by children younger than 6 years of age should be promoted.
- A low-fluoride toothpaste should be developed for children younger than 6 years of age.
- Biomarkers of fluoride should be identified.

PROMOTION OF THE USE OF DENTAL SEALANTS IN COMMUNITY BASED PROGRAMS

Most dental sealants are placed in private-practice dental offices, but the children at greatest risk for problems resulting from dental caries are those least likely to receive dental care in a private setting." Only 18.5% of children and adolescents have at least one sealed permanent tooth." In an attempt to provide sealant application for impoverished children, states and communities have initiated school-based sealant programs that use portable dental equipment (Fig. 30-1) or mobile dental vans (Fig. 30-2). The programs typically move from school to school and often target Title I schools that enroll the highest percentage of children who qualify for free and reduced-price lunches, an indicator of low income. A study conducted in Ohio among 11,191 third-graders who were eligible for the sealant program found that the use of appropriately targeted school-based programs increased the



f **G 30-1.** Portable equipment can be taken into schools and other community sites to create an instant dental clinic for application of dental sealants.



H G . **30-2.** Mobile dental clinics allow dental sealant treatment to be taken to locations that serve children with limited access to care. **A,** Exterior view of mobile van. **B,** Interior view of mobile van.

prevalence of dental sealant use among children from low-income families and reduced the racial and income disparities in sealant prevalence among elementary school students." Appropriate targeting for sealant programs includes identifying schools with a high percentage of children who are least likely to receive regular dental care and are at high risk for dental caries. A cost-effectiveness analysis of a school-based dental sealant program for children of low socioeconomic status in New York determined that, during a 5-year follow-up period, caries incidence was 6.8 among control children in another school and 2.2 in the group receiving sealant application."

ASSESSMENT OF COMMUNITY ORAL HEALTH NEEDS

The completion of large-scale oral health surveys conducted by qualified dental examiners presents logistic and financial challenges; therefore, the Association of State and Territorial Dental Directors developed materials to simplify information gathering. The system materials, *Basic Screening Surveys: An Approach to Monitoring Community Oral Health,* are available through the Association of State and Territorial Dental Directors web site.' The information collected through this system is not intended to be used for clinical diagnosis but rather is used to monitor the specific oral health components listed in the national health objectives in *Healthy People 2010.* Screeners with or without dental backgrounds can employ the protocol. The use of nondental professionals is advocated because, in many communities, no dental public health professionals are available but other public health professionals, such as nurses, have access to dentally underserved populations.

Useful items provided in the training materials include color photographic reference guides for screening preschool children, schoolchildren and adults; sample cover letter; sample consent form; screening criteria; advice on sampling; data-recording form; format for data entry; advice on human subjects clearance; and screener training information. This manual and accompanying videotape and computer disk are a valuable tool for communities interested in assessing the oral health status of their members.

UTILIZATION OF DENTAL SERVICES AND ACCESS TO CARE

BEHAVIOR RISK FACTOR SURVEILLANCE SYSTEM

Utilization of dental services in the United States has been tracked since 1995 by the Centers for Disease Control and Prevention through its Behavior Risk Factor Surveillance System, a national telephone survey.[15] Although useful information has been gathered, the survey has limitations because it reaches only those people who have telephones and thus eliminates some people from lower socioeconomic groups. The survey includes questions that are indicators of access to oral health services, such as: (1) Have you visited a dentist or dental clinic within the past year for any reason? (2) Have you had your teeth cleaned by a dentist or dental hygienist within the past year? (3) Have you lost six or more teeth due to decay or gum disease? Asking people if they had their teeth cleaned during the previous year provides an indicator of access to preventive services. An affirmative answer to the first question regarding dental visits for any reason identifies those who accessed dental professionals for preventive, restorative, surgical, or emergency services. It is apparent from Table 30-1 that annual utilization of dental services is clearly linked to socioeconomic factors. Race, educational level, and income are consistently strong predictors of access to oral health care: people who are white, have higher incomes, and have more education are more likely to have had a dental visit and to have received preventive services during the previous year. In 2001, 14 states included in the telephone survey a question related to dental insurance: Do you have any kind of insurance coverage that pays for some or all of your routine dental care, including dental insurance, a prepaid plan through a health maintenance organization, or a government plan such as Medicaid? Of those who responded in these 14 states, 58.77% answered yes.

PUBLIC FUNDING OF DENTAL CARE FOR UNDERSERVED CHILDREN

Medicaid. Medicaid is a program established in 1965 to provide health insurance to low-income populations. It is jointly funded by the federal and state governments, includes dental services for children, and covers one in every four children in the United States. Medicaid's Early and Periodic Screening Diagnostic, and Treatment program requires comprehensive children's health and dental services to be provided by all states. Because Medicaid is an entitlement program, enrollment must be offered to eligible children without regard to the fiscal impact on the state. Many barriers exist that deter children from receiving dental services through Medicaid, including low reimbursement rates to dentists, cumbersome claims processing, and real or perceived poor health behaviors by recipients.

State Children's Health Insurance Program. The Balanced Budget Act of 1997 amended the Social Security Act to add a new program, the State Children's Health Insurance Program, which provides access to medical care for children through age 18 who live in households with incomes of up to 200% of the federal poverty guidelines. Twenty-four billion dollars of federal funds was allocated to cofund State Children's Health Insurance Program coverage through state programs for the working poor. The program provides states with a higher federal match rate than Medicaid and allows the states to design the program. The inclusion of dental care was optional for the State Children's Health Insurance Program in each state; however, all but two states included children's dental services. States were required to involve the public in the design and implementation of the plan, as well as to ensure ongoing public participation. Many uninsured Americans are already eligible for free or low-cost public coverage through Medicaid or the State Children's Health Insurance Program but do not enroll in that coverage. Several factors that influence enrollment include size of program benefits, convenience of the enrollment process, possibility of stigma or social sense of disrespect associated with program participation, availability of information about the program, and immigration status.'

Children's Special Health Care Services. The Children's Special Health Care Services program helps families of children with serious chronic medical conditions obtain treatment related to their child's condition. This program provides coverage for dental services. The program is federally and state funded, and features care coordination services that help children and their families get medical and dental care. To participate in the program, the individual must be between 0 and 21 years of age and meet certain medical and financial eligibility criteria. Families with an income no greater than 250% of the federal poverty level guidelines are eligible. To meet medical eligibility criteria, the individual must have a condition that is expected to last longer than 2 years and require more health care services than would usually be needed by a child of the applicant's age. The condition must be severe enough to have caused, or be expected to cause, disability, disfigurement, or limitation of function, or to require a special diet or dependence on an assistive device. Also, in the absence of treatment, the disorder must lead to a chronic, disabling physical condition.

TABLE 30-1 2002 Behavioral Risk Factor Surveillance: Utilization of Oral Health Services National Center for Chronic Disease Prevention and Health Promotion Centers for Disease Control and Prevention

HAD TEETH CLEANED BY THE DENTIST OR DENTAL HYGIENIST IN THE PAST YEAR						VISITED THE DENTIST OR DENTAL CLINIC WITHIN THE PAST YEAR FOR ANY REASON					
RACE	MEDIAN % YES	ANNUAL INCOME	MEDIAN % YES	EDUCATION	MEDIAN % YES	RACE	MEDIAN % YES	ANNUAL INCOME	MEDIAN % YES	EDUCATION	MEDIAN % YES
ALL 69.2%						ALL 69.2%					
White	72.1	<$15,000	48.6	<High school	47.2	White	71.8	<$15,000	49.8	<High school	47.2
Black	62.3	$15,000-24,999	56.1	High school or equivalent	64.7	Black	66.0	$15,000-24,999	56.5	High school or G.E.D.	65.1
Hispanic	65.4	$25,000-34,999	64.6	Some post-high school	72.4	Hispanic	66.4	$25,000-34,999	66.0	Some post-high school	72.8
Other	64.1	$35,000-49,999	72.3	College graduate	79.2	Other	64.3	$35,000-49,999	73.2	College graduate	80.5
Multiracial	56.2	$50,000	80.6			Multiracial	58.0	$50,000	81.6		

ACCESS TO INFORMATION ABOUT CHILDREN'S ORAL HEALTH

Information about the oral health of children and their access to dental services has never been as readily available as now. In addition to numerous dental professional journals and texts, extensive databases and electronic publications are available through the World Wide Web. One noteworthy compilation of information is found in the Maternal and Child Health Library.' This site, which includes a knowledge path about oral health in children and adolescents, offers recent, high-quality resources and links and is aimed at health professionals, policy makers, program administrators, and researchers./'

DEVELOPMENT OF COMMUNITY PARTNERSHIPS

Communities play a fundamental role in their own well-being, and the power of shared ideas, resources, and experiences aids in the development of programs that are more effective than those planned by health professionals alone. Collaborative solutions that bring communities, health professionals, and institutions together as equal partners build upon the assets and strengths of each; therefore, dental professionals should seek out partnerships within the communities they serve. When the goal is to establish programs to improve children's oral health, these partnerships are likely to include other health professionals and policy makers as well as agencies and institutions that provide services for children. When possible, dental and dental hygiene students should be included in community-based programs to provide a service-learning experience; this creates the opportunity for these students to learn about the structure and function of social service agencies and the role of these agencies in community oral health promotion.

SUMMARY

Applying recently published recommendations and goals for dentistry and drawing on the resources of dental public health professionals will allow dental professionals to appropriately target, plan, and implement educational and clinical programs that can make significant contributions to community oral health. Promoting communal water fluoridation and assuring appropriate preeruptive and posteruptive fluoride exposure for caries prevention will continue to reduce the prevalence of dental caries and dental fluorosis. Taking pit and fissure sealant programs to schools and community locations that serve children from low-income families will increase the use of sealants in this population group and allow more of these children to experience the preventive benefits of sealants. Effective use of resources for financing dental services can assure access to care for children from low-income families. All of these actions can be achieved by forging partnerships among general practice, pediatric, and public health dentists; other health professionals; dental and dental hygiene students; policy makers; and community agencies, especially those serving children from low-income families. Through these efforts great strides can be made in improving children's oral health.

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APPENDIX

HEALTH INSURANCE PORTABILITY AND ACCOUNTABILITY ACT (HIPAA) OF 1996

The Health Insurance Portability and Accountability Act (HIPAA) of 1996 was signed into law by President Bill Clinton on August 21, 1996. Conclusive regulations were issued on August 17, 2000, to be instated by October 16, 2002. HIPAA requires that the transactions of all patient health care information be formatted in a standardized electronic style. In addition to protecting the privacy and security of patient information, HIPAA includes legislation on the formation of medical savings accounts, the authorization of a fraud and abuse control program, the easy transport of health insurance coverage, and the simplification of administrative terms and conditions.

HIPAA encompasses three primary areas, and its privacy requirements can be broken down into three types—privacy standards, patients' rights, and administrative requirements.

1. Privacy Standards. A central concern of HIPAA is the careful use and disclosure of protected health information (PHI), which generally is electronically controlled health information that is able to be distinguished individually. PHI also refers to verbal communication although the HIPAA Privacy Rule is not intended to hinder necessary verbal communication. The U.S. Department of Health and Human Services (USDHHS) does not require restructuring, such as soundproofing, architectural changes, and so forth, but some caution is necessary when exchanging health information by conversation.

An Acknowledgment of Receipt Notice of Privacy Practices, which allows patient information to be used or divulged for treatment, payment, or health care operations (TPO), should be procured from each patient. A detailed and time-sensitive authorization can also be issued, which allows the dentist to release information in special circumstances other than TPOs. *A written consent is* also an option. Dentists can disclose PHI *without* acknowledgement, consent, or authorization in very special situations, for example, perceived child abuse, public health supervision, fraud investigation, or law enforcement with valid permission (i.e., a warrant). When divulging PHI, a dentist must try to disclose only the *minimum necessary* information, to help safeguard the patient's information as much as possible.

It is important that dental professionals adhere to HIPAA standards because health care providers (as well as health care clearinghouses and health care plans) who convey *electronically* formatted health information via an outside billing service or merchant are considered *covered entities*. Covered entities may be dealt serious civil and criminal penalties for violation of HIPAA legislation. Failure to comply with HIPAA privacy requirements may result in civil penalties of up to $100 per offense with an annual maximum of $25,000 for repeated failure to comply with the same requirement. Criminal penalties resulting from the illegal mishandling of private health information can range from $50, 000 and/or 1 year in prison to $250,000 and/or 10 years in prison.

2. Patients' Rights. HIPAA allows patients, authorized representatives, and parents of minors, as well as minors, to become more aware of the health information privacy to which they are entitled. These rights include, but are not limited to, the right to view and copy their health information, the right to dispute alleged breaches of policies and regulations, and the right to request alternative forms of communicating with their dentist. If any health information is released for any reason other than TPO, the patient is entitled to an account of the transaction. Therefore, it is important for dentists to keep accurate records of such information and to provide them when necessary.

The HIPAA Privacy Rule determines that the parents of a minor have access to their child's health information. This privilege may be overruled, for example, in cases where there is suspected child abuse or the parent consents to a term of confidentiality between the dentist and the minor. The parents' rights to access their child's PHI also may be restricted in situations when a legal entity, such as a court, intervenes and when a law does not require a parent's consent. For a full list of patient rights provided by HIPAA, be sure to acquire a copy of the law and to understand it well.

3. Administrative Requirements. Complying with HIPAA legislation may seem like a chore, but it does not need to be so. It is recommended that you become appropriately familiar with the law, organize the requirements into simpler tasks, begin compliance early, and document your progress in compliance. An important first step is to evaluate the current information and practices of your office.

Dentists will need to write a *privacy policy* for their office, a document for their patients detailing the office's practices concerning PHI. The ADA's *HIPAA Privacy Kit* includes forms that you (the dentist) can use to customize your privacy policy. It is useful to try to understand the role of health care information for your patients and the ways in which they deal with the information while they are visiting your office. Train your staff; make sure they are familiar with the terms of HIPAA and your office's privacy policy and related forms. HIPAA requires that you designate *a privacy officer,* a person in your office who will be responsible for applying the new policies in your office, fielding complaints, and making choices involving the minimum necessary

requirements. Another person with the role of *contact person* will process complaints.

A Notice of Privacy Practices—a document detailing the patient's rights and the dental office's obligations concerning PHI—also must be drawn up. Further, any role of a third party with access to PHI must be clearly documented. This third party is known as *a business associate* (BA) and is defined as any entity who, on behalf of the dentist, takes part in any activity that involves exposure of PHI. The *HIPAA Privacy* Kit provides a copy of the USDHHS "Business Associate Contract Terms," which provides a concrete format for detailing BA interactions.

The main HIPAA privacy compliance date, including all staff training, was April 14, 2003, although many covered entities who submitted a request and a compliance plan by October 15, 2002, were granted one-year extensions. Contact your local branch of the ADA for details. It is recommended that dentists prepare their offices ahead of time for all deadlines, which include preparing privacy polices and forms, business associate contracts, and employee training sessions.

For a comprehensive discussion of all of these terms and requirements, a complete list of HIPAA policies and procedures, and a full collection of HIPAA privacy forms, contact the American Dental Association for a *HIPAA Privacy Kit*. The relevant ADA Web site is www.ada.org/goto/hipaa. Other Web sites that may contain useful information about HIPAA are:

- USDHHS Office of Civil Rights www.hhs. gov/ocr/ hipaa
- Work Group on Electronic Data Interchange www. wedi.org/SNIP
- Phoenix Health www.hipaadvisory.com
- USDHHS Office of the Assistant Secretary for Planning and Evaluation http://aspe.os.dhhs.gov/ admnsimp/

Data from *HIPAA Privacy* Kit; and http://www.ada.org/prof/ prac/issues/topics/hipaa/index.html.
(From Mosby's Dental Dictionary, St Louis, Mosby, 2004.)

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