369 Dental Caries

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EPIDEMIOLOGY

Dental caries, more commonly known as *tooth decay* or *cavities*, is an infectious, transmissible, diet-mediated oral disease that is largely preventable. It is the most common chronic disease among US children—5 times more common than asthma and 7 times more common than hay fever. In the United States, approximately 1 in 4 children who are 2 to 5 years of age have experienced dental caries. Caries prevalence increases with age, such that approximately 1 in 2 children aged 6 to 8 years have experienced caries in their primary teeth. In permanent teeth, approximately 1 in 5 children aged 6 to 11 years, 1 in 2 adolescents aged 12 to 15 years, and 2 in 3 adolescents aged 16 to 19 years have experienced caries. Children of racial and ethnic minorities and low-income families have higher rates of dental caries and untreated dental caries compared to their higher-income, non-Hispanic white counterparts. Since young children see their pediatrician for preventive visits more often (up to 10 times before age 2 years) than they visit their dentist, it is important that pediatricians and other primary care providers are knowledgeable about addressing dental caries by assessing risk and intervening with various strategies to prevent dental caries and promote optimal oral health in children.

ETIOLOGY AND PATHOGENESIS

As dental caries is a disease process that may be established in infancy, understanding its pathogenesis enables the pediatrician to conduct a dental caries risk assessment; provide anticipatory guidance and preventive counseling; and, in collaboration with their dental colleagues, ensure the establishment of a dental home by age 1 year, or as soon as possible. The concept of the dental home is derived from the American Academy of Pediatrics' definition of a medical home. The dental home is the ongoing relationship between the dentist and the patient, inclusive of all aspects of oral health care delivered in a comprehensive, continuously accessible, coordinated, and family-centered way.

Dental caries is the pathological disease process that leads to the loss of tooth mineral and eventually to cavitation of the tooth surface ("cavity"). The etiology of dental caries is multifactorial. The primary components required for dental caries activity to become established are 1 or more susceptible tooth surfaces, cariogenic (decay-causing) bacteria, fermentable carbohydrates (particularly sucrose), and time. Dental caries results from an overgrowth of specific oral bacteria that are present in dental plaque (the sticky white or yellow biofilm that forms on the tooth surface). The cariogenic bacteria are *acidogenic*, which means they produce acids by metabolizing fermentable carbohydrates. These acids lead to the loss of minerals (*demineralization*) such as calcium, phosphate, and carbonate from the tooth. The first sign of demineralization is a thin white line, typically along the gingival margin (Fig. 369-1). At this initial stage, the caries process is reversible through the process of *remineralization*. During remineralization, there is an uptake of calcium and phosphate from the saliva into the tooth enamel, which is facilitated by the presence of fluoride. Fluoride becomes incorporated into the remineralized enamel as fluorapatite, which renders the teeth more resistant to future acidic challenges. If demineralization is not stopped or reversed, the caries process continues and the result is tooth cavitation (Fig. 369-1).

A cavity, or hole in the tooth, represents a very late stage in the dental caries process. Thus, dental caries is a dynamic process, and under normal conditions, there is a balance between demineralization and remineralization. Pathological factors include cariogenic bacteria, altered salivary flow or function, and poor dietary habits. *Streptococcus mutans* (SM) are the cariogenic bacteria most strongly associated with dental caries, although *Streptococcus sobrinus* and *Lactobacillus* species have also been implicated. Sucrose is the most cariogenic carbohydrate. However, glucose, fructose, and cooked starches are also metabolized by plaque bacteria to produce acid. Protective factors include salivary flow and components, antibacterials, and fluoride. Successful rebalancing of risk factors in favor of protective factors may slow down the progression or completely arrest early caries.

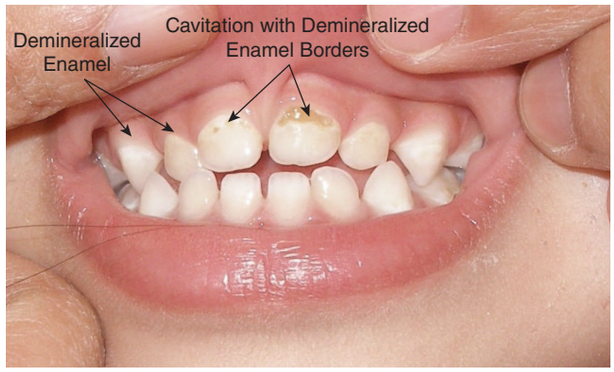


FIGURE 369-1 Demineralization and early cavitation.

EARLY CHILDHOOD CARIES

Early childhood caries (ECC), previously termed *nursing caries* or *baby-bottle tooth decay*, is a particularly virulent form of tooth decay that affects the primary teeth of infants and preschool children. Caries often begins on the smooth surfaces of the maxillary incisors and then spreads to the maxillary and mandibular molars. Caries may manifest solely on the biting surfaces of the molars if they have deep pits and grooves or developmental defects, or in the in-between surfaces of teeth that are in contact. The severity of ECC is based on the child's age, the number of teeth affected, and the tooth surface involved. ECC is considered severe if there is any sign of smooth-surface decay in a child younger than 3 years of age.

Studies demonstrate that children with ECC have SM counts that exceed 30% of the cultivable plaque flora. Conversely, in children with negligible to no caries activity, SM comprises less than 1% of total cultivable flora. SM may be acquired through vertical transmission from the primary caregiver or horizontal transmission among siblings or children in daycare. The timing of SM transmission is important, as acquiring SM before 2 years of age is a significant risk factor for developing ECC and future dental caries.

CONSEQUENCES OF DENTAL CARIES

Children with a history of ECC are more likely to experience future caries in both their primary and permanent teeth. The consequences of dental caries go beyond teeth. Dental caries can negatively impact growth and development and quality of life, interfering with learning and restricting activity, and resulting in missed days from school. Pain associated with dental caries can interfere with speaking, eating, and sleeping. Decay that progresses to the pulp causes intense pain and pulpal necrosis, and may result in a dental infection or abscess that may become life threatening. When children present with a severe dental abscess or facial cellulitis, treatment may require hospital admission with an intravenous (IV) course of antibiotics and extraction of the offending tooth. Due to the age and cooperative ability of the child, treatment may need to be rendered under general anesthesia, thus increasing the cost of care and potential morbidity.

CARIES PREVENTION AND MANAGEMENT STRATEGIES

When possible, a dental home should be established for a child by the first birthday. During this first visit, the dentist can provide an assessment for oral diseases and conditions, perform a caries risk

assessment, determine the current oral health behaviors of the child and family, and develop a customized risk-based plan to anticipate, prevent, or control caries activity. These oral health activities may be provided in the child's medical home. It is more efficacious and cost effective to prevent dental caries before the first signs of disease.

CARIES RISK ASSESSMENT

Assessing each child's risk of dental caries and customizing preventive strategies to address specific risk factors are most important to controlling disease and improving oral health. Risk factors are not constant, and an individual's risk of caries changes with time. Risk factors for caries may be physical, biological, behavioral, or lifestyle related. There is no single test or validated tool that takes into account all risk factors and accurately predicts an individual's risk for caries. However, the pediatrician can perform an assessment of caries risk by focusing on key risk factors that are associated with increased caries risk in children (Table 369-1).

In children, the best predictor of caries is a past history of caries, followed by parents' education and socioeconomic status. Family history of caries, particularly in the mother, is also an important predictor of caries in children. Children who were born premature or with low birthweight may require a special diet or may have developmental enamel defects or disabilities that increase their caries risk. Sugar consumption is an important risk factor in caries development; consumption of sugars at high frequency (eg, sugary beverages via use of and/or sleeping with bottle/sippy cup; frequent intake of sugared medications) or in a sticky and retentive form (eg, raisins and granola bars) increases the risk of caries. Protective factors include systemic and topical fluoride, sugar substitutes, and tooth brushing with fluoridated toothpaste.

The American Academy of Pediatrics recommends that every child begin to receive oral health risk assessments by 6 months of age by a qualified pediatric healthcare professional. Children who belong to high-risk groups in particular should be referred to a dentist by 12 months of age to establish a dental home.

NUTRITIONAL COUNSELING

Parents and caregivers should receive counseling on the importance of limiting the frequency of or avoiding prolonged exposures to obvious and hidden sugars in foods and drinks. Cariogenic foods, including sugary foods and drinks, should be restricted to meal times. The American Academy of Pediatrics has recommended children 1 through 6 years of age consume no more than 4 to 6 ounces of 100% fruit juice per day from a cup (not a bottle or covered cup) and as part of a meal or snack. Carbonated, sugared beverages and juice drinks that are not 100% juice should be avoided. Children should be discouraged from taking a bottle or sippy cup with milk or juice to bed. After eruption of the first primary teeth, infants may be at increased risk for caries if they breastfeed on demand or throughout the night. During sleep, the flow of saliva is decreased, resulting in reduced clearance of the sugary liquid from the oral cavity.

TABLE 369-1 RISK FACTORS FOR DENTAL CARIES

Past caries experience
High caries experience in mother or primary caregiver
Medical conditions/medications that decrease salivary flow
Medical conditions (eg, cerebral palsy) that impede good oral hygiene
Presence of plaque
Presence of demineralized enamel surfaces
Abnormal tooth structure (eg, defective enamel)
Inadequate exposure to fluoride
Frequent consumption of fermentable carbohydrates
Ad-lib/nighttime use of bottle/sippy cup containing a fermentable carbohydrate
Braces/orthodontic appliances
Low socioeconomic status

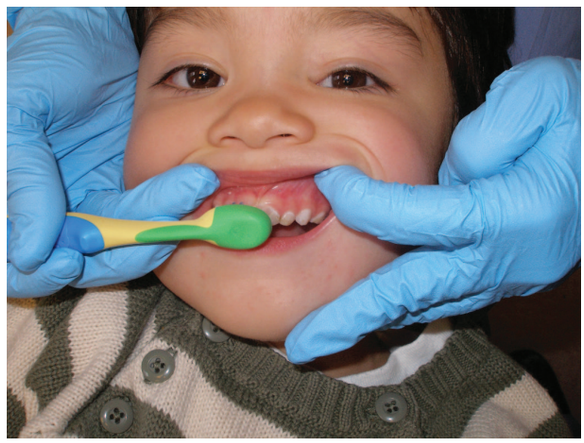


FIGURE 369-2 Tooth brushing a young child: Retracting lips and cheeks to brush at the gum line.

ORAL HYGIENE

Mechanical removal of plaque from the gingival margins and from the grooves of the teeth, through simple tooth brushing, is one of the most effective preventive measures that can reduce the risk of caries. Oral hygiene should begin no later than the time of emergence of the first tooth. Before teeth erupt, an adult caregiver may clean an infant's gums with a clean, damp washcloth. After the eruption of teeth, tooth brushing should be performed by an adult caregiver twice daily by using a soft toothbrush of age-appropriate size with the appropriate amount of fluoride toothpaste (see "Fluorides," below, for details). In very young children, the presence of plaque on the labial or facial surfaces of maxillary primary incisors is the best predictor of future development of ECC.

Since the quality of cleaning is most important, young children require assistance with tooth brushing from an adult caregiver. Resistance and a lack of cooperation are normal reactions to oral hygiene measures in infants, toddlers, and preschoolers. With correct positioning (such as using a knee-to-knee position with 2 adults or having the adult approach brushing from behind the child while supporting the head) and retraction of the lips and cheeks, it should take no more than 1 to 2 minutes to brush a young child's teeth (Fig. 369-2).

Older children who have adequate manual dexterity (typically by 6 to 8 years of age) may require only parental supervision. Flossing is required when teeth develop contacts (usually after 3 to 4 years of age for posterior teeth) and proximal surfaces cannot be reached with a brush. Brushing and flossing before bedtime is of paramount importance, as caries activity is higher at nighttime, when salivary flow decreases. Brushing more frequently with fluoride toothpaste can provide added protection from caries. The practice of brushing twice a day has become a social norm that is convenient for most daily routines, and it is the basic tenet for preventing caries.

FLUORIDES

Use of fluorides is the most effective way to prevent caries. In the ongoing dynamic between demineralization and remineralization at the enamel surface of teeth, fluoride demonstrates its greatest effects through topical mechanisms by promoting remineralization and inhibiting bacterial metabolism. Therefore, therapeutic use of fluoride for children should focus on maximizing topical contact and using lower dose, higher frequency approaches.

Water fluoridation is considered the most cost-effective, convenient, and reliable method of providing optimal fluoride benefits, because it does not depend on individual compliance. Caries rate decreases of 18% to 40% are attributable to water fluoridation. For children who do not have access to optimally fluoridated drinking water, systemically administered fluoride supplements may be recommended.

TABLE 369-2 DIETARY FLUORIDE SUPPLEMENT SCHEDULE

	Fluoride Ion Level in Drinking Water (ppm)[a]		
Age	**Less than 0.3 ppm**	**0.3–0.6 ppm**	**Greater than 0.6 ppm**
Birth–6 months	None	None	None
6 months–3 years	0.25 mg/d[b]	None	None
3–6 years	0.50 mg/d	0.25 mg/d	None
6–16 years	1.0 mg/d	0.50 mg/d	None

[a]1 part per million (ppm) = 1 milligram/liter (mg/L).

[b]2.2 mg sodium fluoride contains 1 mg fluoride ion.

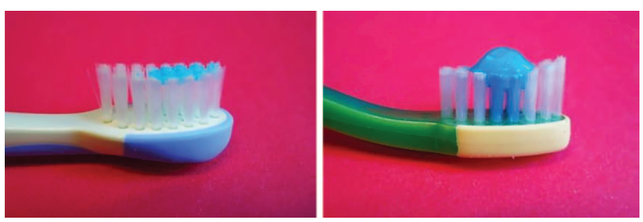
Reproduced with permission from the Centers for Disease Control and Prevention (CDC).

The most recent dietary fluoride supplementation schedule (Table 369-2), originally introduced in 1994 by the American Dental Association (ADA) and approved by the American Academy of Pediatrics and the American Academy of Pediatric Dentistry (AAPD), emphasizes initial supplementation at 6 months of age and continuing to age 16. This schedule assumes the regular use of fluoridated toothpaste and was develwith the intention of minimizing fluorosis while maximizing the topical cariostatic effects after teeth erupt. Fluorosis is hypomineralization of tooth enamel that develops from excessive ingestion of fluoride during tooth formation. Teeth with fluorosis usually manifest with mild discoloration and are more resistant to caries. In 2010, the ADA reaffirmed the supplementation schedule but concluded that dietary fluoride supplements should be prescribed only for children who are at high risk of developing caries and whose primary source of drinking water is deficient in fluoride. Due to concerns regarding known increases in fluoride exposure from multiple sources and the increased in prevalence of fluorosis, the ADA and the AAPD currently recommend fluoride supplementation only for children at high risk of developing caries.

Before recommending fluoride supplementation, the fluoride content of water should be tested. Testing of private wells is available through local and state public health departments and through some private laboratories. For bottled water, if the fluoride concentration is not listed on the label, the bottler can be contacted directly to obtain the information. If multiple water sources are used, determining the total fluoride exposure becomes difficult. Instead of purchasing bottled water, filtering tap water may be considered. However, some reverse osmosis systems reduce the fluoride content of water.

For self-administered care, fluoride toothpaste is the most powerful intervention for caries prevention, because it has high clinical effectiveness and social acceptability. The current recommendation is to brush using a fluoridated toothpaste beginning with the first tooth at least 2 times per day. Children younger than 3 years of age should limit the amount to a small smear applied onto a soft toothbrush by an adult caregiver, while children age 3 to 6 years should use a pea-size amount (Fig. 369-3). To maximize the beneficial effect of fluoride in toothpaste, it is now recommended to avoid or minimize rinsing after brushing.

Children who are at increased risk for caries may be prescribed topical fluoride in the form of gels or rinses to be used at home. For example, in high caries–risk children who are able to rinse and expectorate, 0.2% or 0.05% sodium fluoride rinse may be used.



A B

FIGURE 369-3 Amount of fluoridated toothpaste on toothbrush for children. **A:** Smear or grain-sized amount of fluoridated toothpaste for children less than 3 years of age. **B:** Pea-sized amount for children 3 to 6 years of age.

Professional applications of fluoride every 6 months or more also is recommended. These may be in the form of gels, foams, and varnishes. Fluoride varnish is a professionally applied, sticky resin of highly concentrated fluoride that hardens quickly on contact with saliva. Fluoride varnish is well accepted by young children. In children younger than 6 years of age, fluoride varnish is the only recommended method of professional fluoride application.

In 2014, the US Preventive Services Task Force recommended that primary care clinicians apply fluoride varnish to the primary teeth of all infants and children starting at the age of primary tooth eruption (B recommendation). Two or more applications of fluoride varnish per year are effective in preventing caries in high-risk children of all ages.

PERINATAL ORAL HEALTH

Early acquisition of SM from the child's mother is a major risk factor for ECC and future caries experience. Ideally, oral health education should begin with prenatal education. Thus, prevention, diagnosis, and treatment of oral disease are recommended during pregnancy with no additional risk to the fetus or to the expectant mother, compared to not receiving oral health care. Maternal oral health is also important after the delivery of the infant because decreasing maternal SM levels can reduce infant colonization and the child's subsequent caries risk.

SEALANTS

The pits and grooves (fissures) on the chewing surfaces of teeth are most susceptible to caries. A sealant is a plastic material that is usually applied to the chewing surfaces of the posterior teeth, the molars, and premolars. This plastic resin bonds to the tooth, acting as a barrier and providing protection from plaque and acids. Both primary and permanent teeth that are judged at risk for caries would benefit from sealants.

XYLITOL AND ANTIMICROBIALS

Xylitol is a sugar substitute that is part of the polyol family that includes sorbitol, mannitol, and maltitol. Xylitol is approved by the US Food and Drug Administration and safe to use in children. Sugar alcohols have been shown to be noncariogenic, and xylitol exhibits protective effects from dental caries. Some studies indicate that xylitol can reduce SM in plaque and saliva, which can reduce dental caries in young children and their mothers and can decrease the vertical transmission of SM from mother to child.

Xylitol use is attractive because, as a sugar substitute, it can reduce the overall consumption of other sugars in the diet. Xylitol is commonly available in chewing gum; however, a range of 6 to 10 g divided into at least 3 consumption periods per day is necessary for xylitol to be effective with chewing gum as the delivery system. Unfortunately, xylitol is relatively expensive and chewing gum is not a suitable product for toddlers and preschoolers. Presently, there is no safe xylitol substitute available for this age group.

Antimicrobial rinses can reduce the number of cariogenic bacteria and can be useful in high caries–risk individuals. Presently, the most effective antibacterial rinse is 0.12% chlorhexidine gluconate. To avoid the possibility of swallowing, these rinses are recommended only for children who can rinse and spit.

Although repairing damaged tooth structure improves stability, aesthetics, and function, it does not address the underlying disease process. Instead, since dental caries is a chronic bio-behavioral disease, a comprehensive approach to preventing and managing the disease modeled after the medical management of chronic conditions is necessary while repairing the damaged tooth structure. Effective disease prevention and management of dental caries must focus on engaging children, parents, and families to self-manage their disease, by mitigating high-risk behaviors and enhancing protective factors.

Young children who are apprehensive or uncooperative and children with special health care needs may require nitrous oxide/oxygen analgesia, sedation, or general anesthesia as adjunctive care to surgical dental treatment (see Chapter 375).

CONCLUSION

Oral health is an integral part of overall health. It is important that pediatricians be knowledgeable about dental caries and the interventions available to prevent and manage the disease.

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370 Dental Emergency Care

Howard L. Needleman and Zameera Fida

INTRODUCTION

It is important for healthcare professionals to properly triage children who present with dental trauma or pain for problems originating from the oral cavity. This chapter will provide clinicians with information allowing them to assess whether emergent care can be provided onsite or if the care needs to involve dental professions outside of the presenting venue.

OROFACIAL TRAUMA

PATHOGENESIS AND EPIDEMIOLOGY

Trauma to children's teeth is a very common event, affecting approximately 2% of children annually. The prevalence of these injuries varies depending on the population studied and the types of injuries reported; as many as 46% of children sustain traumatic injuries to their primary or permanent teeth during childhood. Males are more likely to sustain injuries, while the frequency and type of injuries vary with age. The greatest incidence of trauma to the primary dentition occurs at 2 to 3 years of age, when motor coordination is developing. The incidence peaks again between the ages of 8 and 10 years, which poses a risk to the permanent maxillary anterior teeth.

The majority of injuries involve the maxillary incisors due to their prominence in the dentition. Displacement injuries are more common in the primary dentition, because supporting bone in younger children is more flexible and pliable, while fractures are more common in the permanent dentition. Children who are very active, such as those with attention-deficit/hyperactivity disorder (ADHD), or those with poor motor coordination, such as those with cerebral palsy, have greater risk of trauma. Socioeconomic status can also affect the risk of trauma to the dentition; the risk is increased in those without a nuclear family (ie, without 2 parents). Children who are overweight and those with protrusive maxillary incisors are at greater risk of trauma. Individuals who have undergone general anesthesia with endotracheal intubation can experience "silent trauma" to their incisors (fractured or traumatized incisors during intubation). Failure to treat fractured teeth can impact a child's daily performance, specifically in smiling, laughing, and showing teeth without embarrassment.

The most common injuries to permanent teeth occur secondary to falls, violence, traffic accidents, and sports. Most sporting activities have an associated risk of orofacial injuries due to falls, collisions, and contact with hard surfaces or other players. There are numerous preventive measures to decrease these risks, such as wearing protective intraoral mouthguards and helmets during many activities. The Centers for Disease Control and Prevention estimates that universal use of helmets would prevent 45,000 craniofacial injuries and 55,000 maxillary and mandibular injuries annually. Depending on the stage of the child's dentition, 2 types of mouthguards are recommended. The "boil and bite" mouthguard (Fig. 370-1), which is inexpensive

FIGURE 370-1 "Boil and bite" intraoral mouthguard.

and can be adapted to the child's mouth by the parent, is especially helpful during mixed dentition when primary teeth are exfoliating and permanent teeth are erupting. The custom-made mouthguard (Fig. 370-2) is preferred due to its excellent fit; however, it is more expensive since it requires dental impressions and incurs laboratory fees for fabrication. Early orthodontic intervention to reduce a severe protrusion of the maxillary incisors also has been shown to decrease trauma to these teeth.

CLINICAL MANIFESTATIONS, DIAGNOSIS, AND MANAGEMENT

In all instances of trauma and infection in an otherwise stable patient, a careful medical history, including tetanus vaccination status, should be obtained. The skull, facial bones, and mandible should be palpated to assess for any areas of ecchymosis, paresthesia, crepitus, or pain, which may point to a concomitant facial bone fracture. In addition, concussion must be ruled out and an assessment of cranial nerve function test should be performed. Finally, to rule out aspiration in cases of a tooth fracture or avulsion, where the tooth or fragment cannot be located, immediate chest and abdominal radiographs should be obtained.

Orofacial trauma, including trauma to the dentition, is common in cases of child abuse and neglect. Dental trauma may be an important marker for child abuse, because craniofacial, head, face, and neck injuries occur in approximately 65% of recorded cases of child abuse. This can be attributed to the fact that the mouth is the source of yelling or crying and is readily accessible, and the face represents "the self." Trauma in children is often caused by an adult violently striking the child's lips and teeth with a hand. If the hand or associated object is directed forcefully and upward, the maxillary frenum may be torn and the lips and teeth may be damaged. Injuries that have typical shapes and patterns, human bite marks, adult handprints, or bilateral injuries indicate possible abuse.

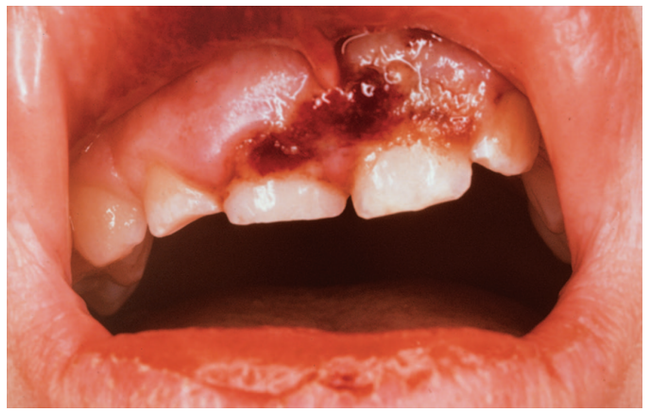


FIGURE 370-3 Tear of the maxillary labial frenum.

Soft Tissue Trauma

Typical oral injuries include lacerations, especially of the maxillary labial frenum (Fig. 370-3); fractured, luxated, or avulsed teeth; jaw and facial fractures; oral burns; oral and facial bruises; or tissue scarring from previous trauma. Teeth may also be discolored or abscessed from previous trauma. Sexually transmitted oral lesions such as condyloma acuminata (Fig. 370-4) in prepubertal children should raise suspicion of sexual abuse. Oral trauma from sexual abuse can also result in petechiae or bruising at the junction of the hard and soft palate. Burns and bruising on the lips may also be present and could be the possible sequelae of forced feeding.

Trauma to the soft tissues of the oral cavity can be caused by physical, chemical, or electrical insults. Soft tissue injuries consist of abrasions, lacerations, contusions, ecchymoses, hematomas, and burns. Oral lacerations should be examined carefully for the presence of foreign bodies, especially in the presence of fractured teeth. A radiograph

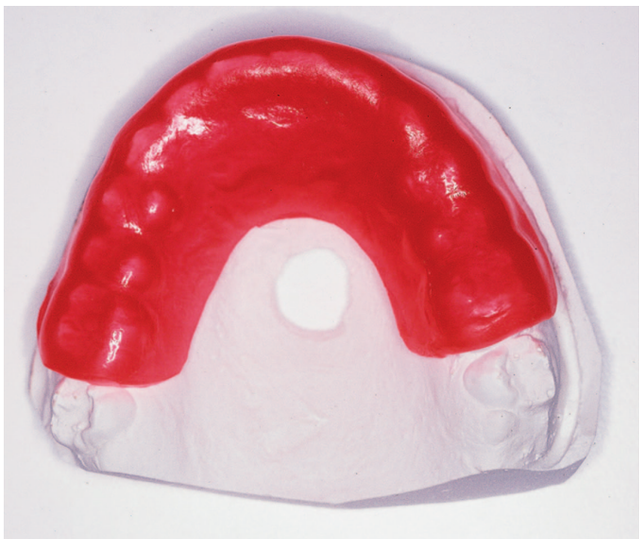


FIGURE 370-2 Custom-made intraoral mouthguard.

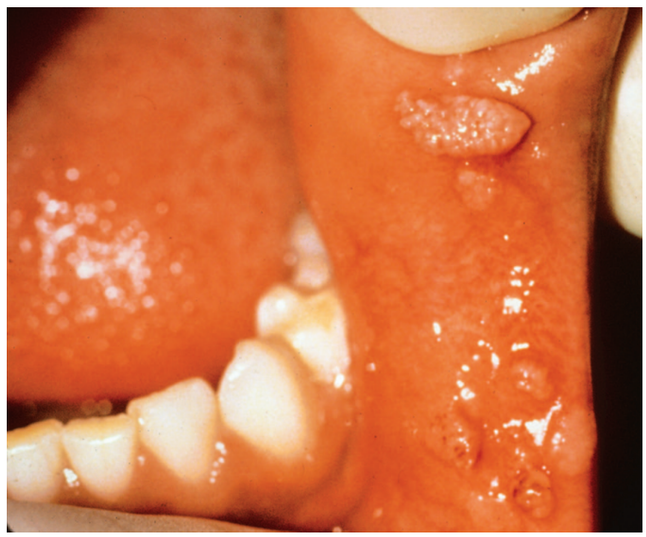


FIGURE 370-4 Oral condyloma acuminata.

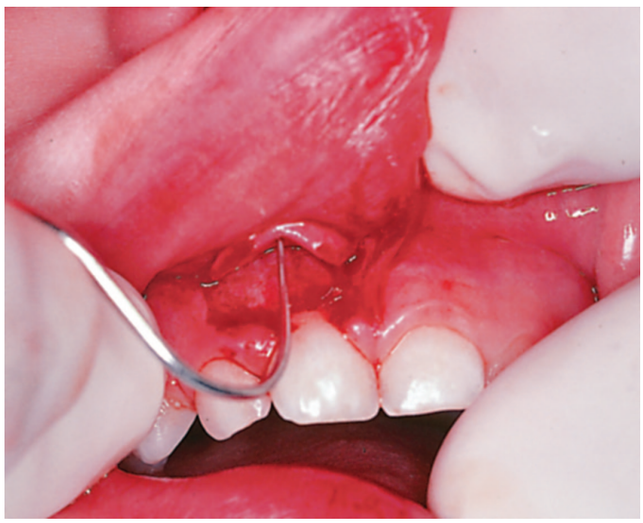


FIGURE 370-5 Degloving of the maxillary labial gingiva.

of the lesion should be obtained to rule out foreign bodies, as visual inspection and palpation alone are usually not sufficient.

Lip lacerations require careful management to provide an aesthetic closure, especially if the laceration is deep or extends beyond the vermilion border. Full-thickness lacerations require suturing in layers. Careful attention to anatomic alignment of the vermilion border is important. Through-and-through lacerations result in communication between the skin and oral environment and are frequently contaminated. Suturing of the intraoral laceration should precede skin suturing, and the patient should be placed on a course of antibiotics effective against staphylococcal organisms.

Tongue lacerations are commonly seen in children and usually result from a fall or blow to the chin. The tongue has a profuse blood supply, and injury can result in copious bleeding. Most tongue lacerations with approximating borders will heal without suturing; however, tears that leave unapproximated borders, such as at the tip or along the lateral borders, require suturing.

Gingival degloving occurs when both the gingival tissue and periosteum are pulled away from its normal position around the tooth, exposing underlying bone (Fig. 370-5). These injuries require careful repositioning of the gingival tissue and stabilization with sutures.

Electrical Burns

Electrical burns of the mouth are an infrequent but serious event with varied interdisciplinary treatment approaches. Approximately 3.5% of referred burns involve the mouth and commissure. These injuries, which affect children mostly under the age of 3, typically occur when a child sucks on the end of an extension cord or bites through the insulation of a live wire. The best way to avoid these accidents is to take preventive measures against them by using safety caps on all electrical outlets and assuring intact, solid insulation on all electrical cords. Immediate treatment should address systemic complications such as shock and hemorrhage. The wound requires careful daily debridement of the necrotic tissue, approximation of the wound edges with adhesive straps, and topical antibiotics. Complications such as bacterial infection, disfiguration, microstomia due to scarring, and discoloration of teeth may result from electrical burns of the oral commissure. Labial artery bleeding is a late complication of oral commissure burns. If the oral commissure is involved, the use of an intraoral splint to prevent labial adhesions and to limit the oral opening can decrease the need for commissuroplasty.

Tooth Injuries

Injuries to teeth can be divided into fractures and displacements. The International Association of Dental Traumatology has established guidelines for their evaluation and management. Fractures of teeth are classified by the extent of the lost tooth structure. Most of these

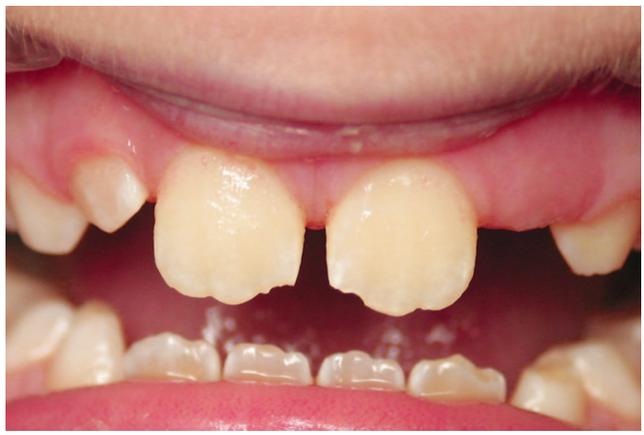


FIGURE 370-6 Enamel fracture of maxillary central permanent incisors.

injuries require a thorough extraoral and intraoral examination as well as dental radiographs. Both intraoral and extraoral radiographic evaluation is important to fully evaluate the extent of the injury. In addition, a baseline dental radiograph is essential for comparison to later radiographs to evaluate healing and the status of the periapical tissues, should symptoms of pulpal necrosis arise.

Fractures Uncomplicated fractures are limited to the enamel and dentin of the tooth's crown. If only the enamel is affected, the fractured edge appears white (Fig. 370-6). However, if both the enamel and dentin are involved, the fractured edge appears yellow in the center (Fig. 370-7). These relatively superficial fractures are nonurgent. Treatment consists of either smoothing the rough edges if limited to the enamel or restoring the fracture with a tooth-colored resin if cosmetics are an issue. When the dentin is exposed, the tooth may be sensitive to thermal changes. The exposed dentin needs to be covered within a few days with a temporary resin "bandage" to diminish the irritation of the pulpal tissues, which can lead to pulpal necrosis if left untreated. The tooth can be restored completely at a later time by bonding a complete resin restoration to the fracture site. If the fractured segment is available, bonding of the 2 segments is possible and can result in excellent aesthetics.

Complicated fractures extend beyond the dentin and need immediate referral to a dentist. These fractures extend into the pulpal tissues and/or root of the tooth, as evidenced by a red or bleeding area visible near the center of the fracture (Fig. 370-8). Pulpal/endodontic therapy (partial or total root canal treatment) should be instituted to avoid necrosis of the pulp and eventual alveolar abscess formation. The final restoration of the fracture should be accomplished at a later time. Root fractures may not be clinically obvious but can be diagnosed by excessive mobility of the tooth with radiographic signs of a fracture

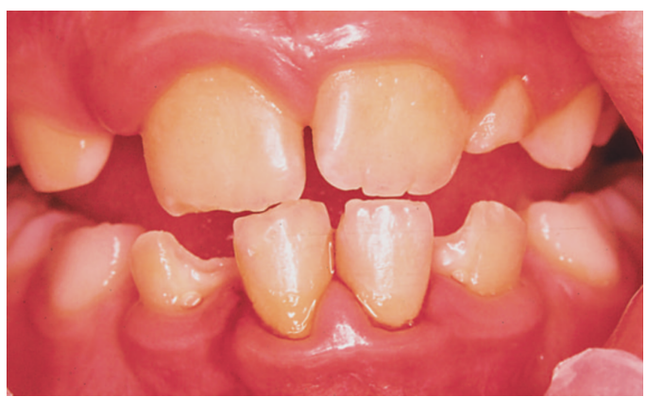


FIGURE 370-7 Enamel-dentin fractures of the mandibular permanent lateral incisors.

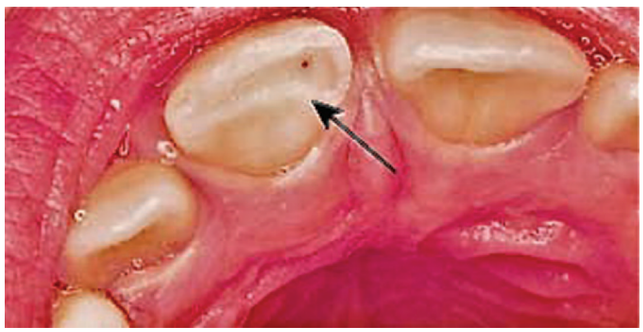


FIGURE 370-8 Fracture into the pulp of a maxillary right permanent central incisor showing red area (*arrow*) visible near the center of the fracture.

anywhere along the root. The fractured segments must be stabilized with a splint fabricated by attaching the involved tooth to the adjacent uninvolved teeth using resin and orthodontic wire. Splints should remain in place for at least 4 weeks, and root canal therapy may be necessary if there is necrosis of the pulp.

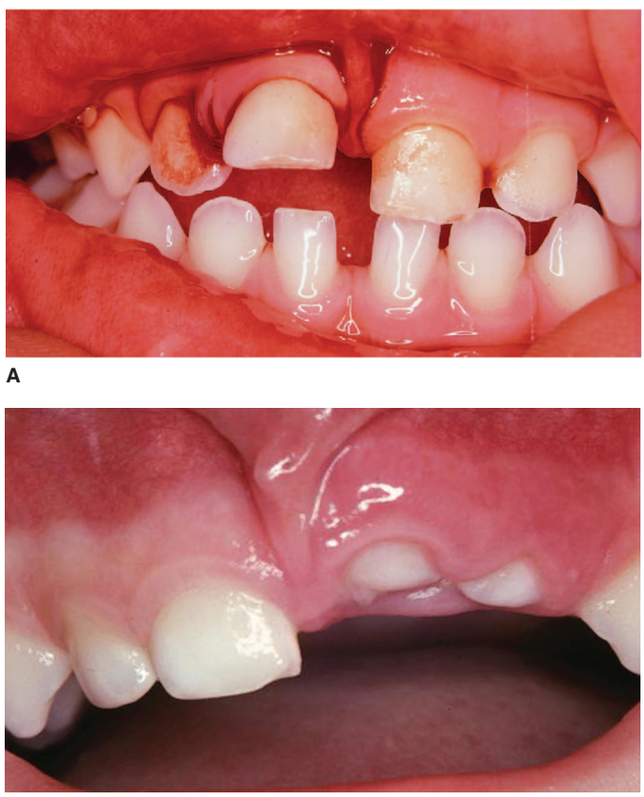
Displacements Displacement injuries to teeth include (1) concussion, in which the tooth is tender to percussion because of edema or inflammation of the periodontal ligament; (2) subluxation, in which the tooth is not displaced but is mobile within its socket; (3) luxation, in which the tooth is displaced from its alveolar socket (intruded, extruded, or laterally displaced) (Fig. 370-9); and (4) avulsion, in which the tooth is totally displaced from its socket. The goal of treatment for displacement injuries is normal reattachment of the periodontal ligament to both the tooth and the alveolar bone. This is best accomplished by reducing the displacement and stabilizing the tooth for 2 to 4 weeks, depending on the type of displacement; maintaining good oral hygiene of the surrounding soft tissues; using chlorhexidine oral rinses; and minimizing chewing with the involved tooth or teeth during this healing period. Normal reattachment is usually complete at 3 weeks, and the prognosis for pulpal survival can be made 3 months after trauma. Pulpal survival varies in likelihood, depending on the severity of displacement. Intrusions and avulsions have the highest likelihood of needing root canal therapy. The ultimate prognosis of displacement injuries is good if treatment is both prompt and appropriate, with close follow-up for at least 1 year. Alveolar fractures involve the displacement of multiple teeth along with a bony segment and need reduction with local anesthesia and splinting for at least 4 weeks. The risk of pulpal necrosis of the involved teeth is extremely high with the need for root canal therapy almost guaranteed.

Permanent teeth that are avulsed should be reimplanted immediately. Radiographic examination is vital to ensure that the missing tooth is not totally intruded rather than avulsed or that part of the tooth still remains in the socket. Avulsed primary teeth should not be reimplanted because of the risk of infection to both the primary tooth and the developing permanent dentition. In addition, the primary incisors are not critical to the developing dental arch and the complex treatment that is required is contraindicated in the very young child.

The protocol for immediate on-site reimplantation of avulsed permanent teeth is as follows:

1. The tooth should always be held by the crown and not the root to avoid damage to the periodontal ligament.
2. If uncontaminated, the tooth is gently but firmly placed back into the socket with digital pressure. If contaminated, the tooth should be rinsed with running water or saline before reimplantation. If resistance is met in reimplanting the tooth or if the child will not cooperate, the tooth should be stored in appropriate storage media such as Hank's balanced salt solution (Save-A-Tooth Kit, Phoenix-Lazerus, Pottstown, PA; www.save-a-tooth.com) or milk until seen by a dentist. Alternative storage media include saline or saliva. If the patient is able, he or she can keep the tooth in their oral vestibule.
3. The replanted tooth should be stabilized by having the patient bite on gauze or another readily available material until seen by a dentist.
4. The patient should be transferred either to a hospital emergency room with dental staff coverage or a dental office for immediate splinting.
5. Antibiotics should be prescribed and tetanus immunization status should be reviewed.

The prognosis for the reimplanted permanent tooth is based on the extra-alveolar period since the cells of the periodontal ligament usually are unable to survive for more than 10 to 15 minutes out of a physiological solution. Immediate reimplantation at the site of the injury is critical to minimize this extra-alveolar period. Once reimplantation is complete, stabilization and root canal therapy are required. Prognosis for the avulsed tooth is favorable if the reimplantation has been completed within 30 minutes of the avulsion or if the tooth has been stored in the appropriate medium before reimplantation. If the extra-alveolar period is greater than 30 minutes and the periodontal ligament is allowed to dry, the prognosis is extremely poor. More than 90% of these teeth become irreversibly ankylosed (fused to the bone) and eventually require extraction due to continued growth of the surrounding teeth with relative submergence of the affected tooth.



A

B

FIGURE 370-9 **A:** Labial displacement of a maxillary right primary central incisor. **B:** Intrusive displacement of maxillary left primary central and lateral incisors.

COMPLICATIONS AND LATE EFFECTS

The survival of an injured tooth depends on the normal reattachment of its periodontal ligament to the surrounding bone and on the preservation of the vitality of its pulpal tissues. Abnormal reattachments of a tooth to its alveolar bone include resorption of the root surface, which results in increased tooth mobility and ankylosis of the root surface, which results in decreased tooth mobility. Some of these abnormal reattachments are often irreversible and can result in loss of the tooth. Devitalization and infection of the pulp may be manifested clinically

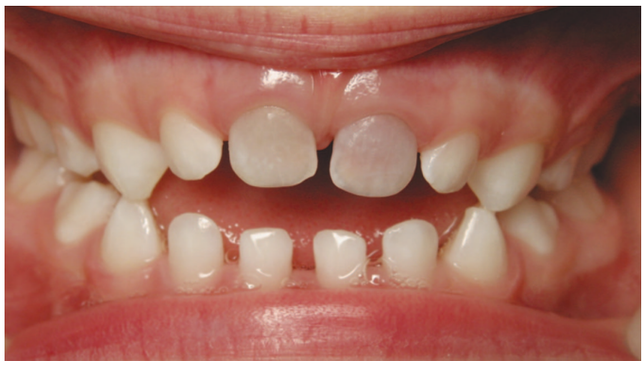


FIGURE 370-10 Discolored maxillary primary central incisors secondary to trauma.

as discoloration of the crown (Fig. 370-10), pain, excessive mobility, and/or alveolar abscess formation (Fig. 370-11). If the dental pulp becomes nonvital, root canal therapy is the treatment of choice and has an excellent prognosis.

Sequelae of trauma to primary teeth can result in a myriad of clinical presentations. The primary tooth's crown can change color: Gray indicates either extravasation of blood into the crown and/or necrosis of the pulpal tissues, yellow-opaque indicates calcification of the pulpal contents, and pink indicates internal resorption. Periapical infections can result in premature root resorption and excessive mobility of the tooth. Conversely, the tooth may become ankylosed and lose its normal mobility.

The traumatic incident itself or post-trauma sequelae can result in damage to the primary tooth's succedaneous tooth. These defects include hypoplasia of the crown (Turner's hypoplasia), root deformities, and/or disturbances of eruption (accelerated, delayed, and/or ectopic).

OROFACIAL PAIN AND INFECTION

PATHOGENESIS AND EPIDEMIOLOGY

Dental caries (see Chapter 369) is the most common cause of pain from the oral cavity. As untreated caries progresses, it can cause pain and infection of the pulp, which can then spread to the supporting tissues and the jaws. This may culminate in an advanced disease condition that is painful and can have significant systemic implications. Additionally, socioeconomic status is a significant risk factor for these conditions. Children from low-income families have nearly 12 times more restricted-activity days because of dental-related illness than children from higher-income families. Pain and suffering due to untreated tooth decay can lead to problems with eating, speaking, and learning.

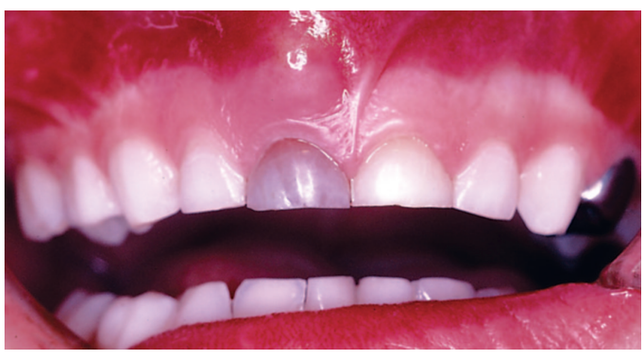


FIGURE 370-11 Discolored maxillary primary central incisor secondary to trauma with labial gingival swelling secondary to abscess formation.

Nondental sources should always be considered in any differential diagnosis of pain from the oral cavity. These may include referred pain from the middle ear, temporomandibular joint (see Chapter 374), and nasal tissues; sinus infection; intraoral viral infections; oral ulcers; and neuralgias (see Chapter 373).

CLINICAL MANIFESTATIONS AND DIAGNOSIS

It is important to note that progressive dental decay leading to infection can begin at a very early age as sequelae of poor hygiene and/or poor feeding practices. Severe tooth decay in young children is referred to as *early childhood caries* (ECC), also known as *nursing caries* or *baby bottle tooth decay*. It has long been recognized as a clinical syndrome that leads to pain and infection (see Chapter 369).

The first step in evaluating oral pain should be to determine the etiology. Children usually refer to pain in their mouths as "toothaches," or parents interpret complaints originating from the mouth as such. Pain may also be secondary to a dental procedure such as an extraction. This may manifest as swelling, pain, bleeding, and alveolar osteitis (dry socket). Alveolar osteitis is rarely seen in children, but may occur after permanent molar extraction in teenagers and adolescents. Smoking, spitting, drinking with a straw, playing a brass or wind musical instrument, or other activities that change the pressure inside the mouth can promote osteitis.

Occasionally, painful intraoral bacterial infections may also be attributed to food or debris impaction under the flap of tissue that still is covering the distal portion of an erupting tooth. The flap of tissue appears edematous and inflamed. Lymph nodes in the surrounding area may also be swollen and painful. This disorder, referred to as pericoronitis (Fig. 370-12), most commonly manifests in the 16- to 22-year-old age groups when wisdom teeth (third molars) are erupting, but can occur with any erupting posterior tooth.

An incipient carious lesion can result in dentinal pain. This pain is contingent upon a local stimulus such as cold temperatures or sweet foods or drink. The pain is alleviated quickly once the offending stimulus is removed. More significant pain does not occur until the decay reaches deeper into the dentin and approaches the dental pulp (blood vessels, nervous and connective tissue).

Inflammation and pain of the pulpal tissue can be characterized as reversible pulpitis, irreversible pulpitis, or necrosis. Reversible pulpitis is mild inflammation that is reversible if the etiology is treated. The pain is temperature or sweet specific. In these cases, the pain is not spontaneous, lasts for a few seconds, and subsides only when the stimulus is withdrawn. If the source such as dental caries is not

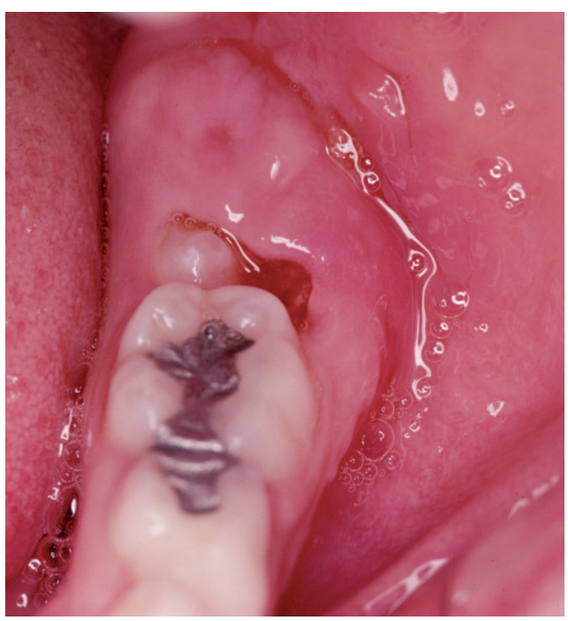


FIGURE 370-12 Pericoronitis of erupting molar.

treated, it will progress to an irreversible state of pulpitis. In this situation, pain can become severe and spontaneous and it may wake the child from sleeping. Necrosis of the pulpal tissue occurs when a long-standing irreversible pulpitis is not treated. Pain associated with a necrotic tooth that has progressed to an acute abscess is often severe, spontaneous, and persistent. However, unlike the pain of an irreversible pulpitis, it usually can be localized to a specific tooth. If this is the case, the tooth will be painful to percussion and the patient may have regional lymphadenopathy. A chronic alveolar abscess associated with a purulent drainage usually from the buccal tissues is not painful.

TREATMENT

Definitive treatment for reversible and irreversible pulpitis requires management by a dentist. Reversible pulpitis will require removal of the irritant and placement of a restorative filling material. Irreversible pulpitis is treated by either root canal therapy or extraction. The pain should be managed with appropriate analgesics such as a nonsteroidal anti-inflammatory drug or acetaminophen until the patient can be treated by a dentist.

Without proper treatment, irreversible pulpitis will progress to pulpal necrosis and can eventually lead to apical periodontitis, which can be either acute or chronic. Necrosis and subsequent apical periodontitis can lead to 2 emergent sequelae: acute abscess formation (Fig. 370-13) or orofacial cellulitis (Fig. 370-14). Both are common presentations in emergency room visits and are treated differently. Definitive treatment is root canal therapy or extraction. Referral to a dentist or oral maxillofacial surgeon must be made immediately to minimize the possibility that this will develop into a widespread facial cellulitis with ocular or extensive submandibular involvement. In the event of an abscess, immediate treatment may include incision and drainage. Antibiotic coverage should be considered and is essential if the localized abscess has progressed to a cellulitis.

The examination of a patient with cellulitis should focus on whether the infection remains localized or has spread regionally. Patients with localized cellulitis who are deemed appropriate for outpatient therapy should be treated by the physician with antistreptococcal oral antibiotics such as penicillin or amoxicillin, or with clindamycin if the patient is allergic to penicillin. The patient should then be referred to a dentist or oral maxillofacial surgeon for local management such as extraction or incision and drainage. Odontogenic infections with cellulitis that do not respond well to the first course of antibiotics or orofacial cellulitis of nonodontogenic origin are best managed with antibiotics that are effective against penicillin-resistant organisms, such as amoxicillin/potassium clavulanate (Augmentin). It is important to note that orofacial cellulitis usually presents with fever and may demonstrate respiratory compromise and orbital involvement.

Treatment of pericoronitis includes debridement with copious irrigation of a saline solution to remove any accumulated food debris under the flap of tissue, which will alleviate the problem in most cases.

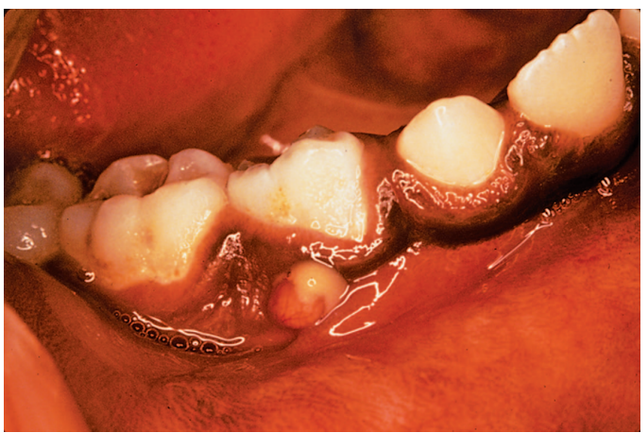


FIGURE 370-13 Chronic alveolar abscess with purulent drainage associated with carious tooth.

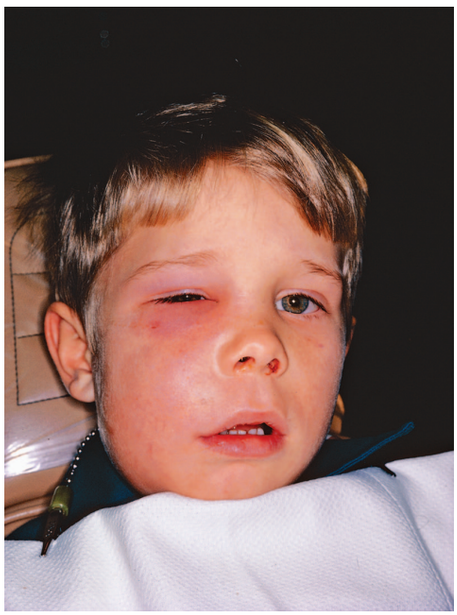


FIGURE 370-14 Facial cellulitis.

However, immediate referral to a dentist is recommended to determine the cause of the problem. Trismus, fever, and lymphadenopathy can occur in advanced cases of pericoronitis, which requires antibiotic therapy along with an immediate referral to a dentist or oral maxillofacial surgeon.

COMPLICATIONS AND LATE EFFECTS

Inappropriately treated cellulitis can be associated with serious outcomes such as vision impairment, cavernous sinus thrombosis, and even death. Patients with widespread cellulitis, dehydration due to inadequate oral intake, fever, or signs of airway compromise should be hospitalized to allow for adequate hydration, nutrition, and management of the infection.

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371 Management of the Developing Dentition and Occlusion

Richard Bruun and Sivabalan Vasudavan

INTRODUCTION

Dental occlusion is the term used to describe the relationship of the maxillary and mandibular teeth to each other when in contact and during function, as well as within each jaw. The American Academy of Pediatric Dentistry "recognizes the importance of managing the developing dentition and occlusion and its effect on the well-being of children, adolescents, and adults." Such management requires appropriate and timely diagnosis of any developing malocclusion and the ability to either provide the proper treatment or refer the patient to the appropriate specialist for treatment, with the ultimate goal of obtaining a stable, functional, and aesthetically pleasing occlusion in the permanent dentition.

The pediatrician or family medical practitioner is uniquely positioned to clinically detect many malocclusions in the course of medical practice. Although a definitive diagnosis and treatment can be formulated only after a dentist acquires and evaluates the appropriate records, the primary healthcare provider should feel comfortable evaluating the occlusion and referring the child to a dentist when appropriate.

This chapter covers the stages of dental development, the development of a normal occlusion, and provides basic knowledge regarding the most common types of malocclusions seen in children.

DEVELOPMENT OF THE DENTITION

The normal sequence of tooth formation is outlined in **Table 371-1**. The earliest sign is seen at about the sixth week of embryonic life. The tooth buds of the primary teeth develop at 10 specific sites in each of the developing maxilla and mandible. The odontogenic epithelium that develops into tooth buds forms on the lateral aspect of the medial nasal process, the inferior border of the maxillary process, and the superior border of the mandibular process at approximately the same time that the first 2 embryonic processes fuse into the upper lip and the alveolar process. When this fusion fails or breaks down, a cleft lip and/or alveolus results, accompanied by various defects and differences in the developing teeth. The 20 succedaneous teeth develop beneath the primary teeth while the permanent molars develop distally (farther from the dental midline) in sequential order. Calcification of the primary teeth begins at about 4 months in utero, and the enamel of all primary tooth crowns is completely calcified by 10 months after birth. The permanent teeth begin to calcify with the first molar around the time of birth, and the process is complete for all the crowns of the teeth, with the exception of the third molars, by the seventh to eighth year of life.

In both the primary and permanent dentitions, the process of tooth eruption correlates with root development. When the crown emerges through the gingiva, the root usually comprises one-half to two-thirds of its final length. Eruption continues until the antagonist tooth or teeth in the opposing jaw are contacted in occlusion. As dental attrition occurs throughout life, eruption continues but at a much reduced rate, thus keeping the teeth in occlusion. The primary stimulus for tooth eruption has not been definitively identified.

Exfoliation of the primary teeth is a normal physiological process that takes place as root development occurs in the permanent successors erupting beneath them. The sac-like structure surrounding the crown of each erupting tooth, known as the dental follicle, stimulates the formation of osteoclasts that result in the resorption of alveolar bone, resorption of the roots of the primary teeth, and eventually their exfoliation. Girls generally precede boys in the eruption of their permanent teeth, while Native Americans generally precede African Americans, who precede white Americans.

DEVELOPMENT OF THE DENTAL ARCHES AND THE OCCLUSION

The Primary Dentition

The primary (deciduous) dentition is comprised of 20 teeth that begin to erupt at approximately 6 months of age and usually complete their eruption before the age of 3 years (**Fig. 371-1A**). It is helpful to remember that there is significant individual variation in the ages of eruption. It is not necessarily a cause for concern if a child does not have any erupted teeth by the age of 1 year. In the absence of any medical issues, unnecessary imaging should be deferred in most of these cases.

In each quadrant from anterior (mesial) to posterior (distal), there is a central incisor, a lateral incisor, a canine, and first and second molars. Although the timing of eruption varies considerably from child to child, the sequence of eruption is commonly as follows: central incisor, lateral incisor, first molar, canine, and second molar, with the mandibular teeth erupting somewhat earlier than the maxillary teeth and bilateral symmetry usually the case. Ideally, there should be spacing present between all of the teeth, although any space present between the posterior teeth usually closes prior to the eruption of the first permanent molar. Absence of spacing in the complete primary dentition suggests a greater than 50% probability of crowding in later stages of the dentition.

A normal occlusion in the primary dentition is characterized by maxillary and mandibular teeth that are related properly to each other in the sagittal, transverse, and vertical dimensions. The maxillary incisors and canines are positioned slightly forward (labially) of the mandibular incisors (normal overjet with no anterior crossbite), and the maxillary molars are positioned so that their buccal cusps occlude slightly to the outside (buccally) of the mandibular molars and their palatal cusps occlude onto the center of the biting surface of the mandibular molars (normal buccal overjet with no posterior crossbite). There should be only a mild to moderate amount of vertical overlap of the maxillary and mandibular incisors (normal overbite). The midlines of the maxillary and mandibular dentitions should be approximately coincident with each other and with the facial midline (as approximated by the center of the philtrum), and there should be no significant (more than 1 mm) shifting of the mandible laterally or anteriorly as the teeth come into contact (**Fig. 371-2**).

The relative position of the jaws and therefore the occlusion is reflected in the profile. The facial soft tissue profile may also be affected by the position of the anterior teeth and by the gingival and overlying soft tissues themselves. It is normal for the profile to be convex during the primary dentition stage of dental development with this convexity usually decreasing during the subsequent stages

TABLE 371-1 CHRONOLOGY OF THE HUMAN DENTITION

Tooth	Hard Tissue Formation Begins	Amount of Enamel Formed at Birth	Enamel Completed	Eruption	Root Completed
Primary dentition					
Maxillary					
Central incisor	4 mo in utero	5/6	1.5 mo	7.5 mo	1.5 y
Lateral incisor	4.5 mo in utero	2/3	2.5 mo	9 mo	2 y
Canine	5 mo in utero	1/3	9 mo	18 mo	3.25 y
First molar	5 mo in utero	Cusps united	6 mo	14 mo	2.5 y
Second molar	6 mo in utero	Cusp tips still isolated	11 mo	24 mo	3 y
Mandibular					
Central incisor	4.5 mo in utero	3/5	2.5 mo	6 mo	1.5 y
Lateral incisor	4.5 mo in utero	3/5	3 mo	7 mo	1.5 y
Canine	5 mo in utero	1/3	9 mo	16 mo	3.25 y
First molar	5 mo in utero	Cusps united	5.5 mo	12 mo	2.25 y
Second molar	6 mo in utero	Cusp tips still isolated	10 mo	20 mo	3 y
Permanent dentition					
Maxillary					
Central incisor	3–4 mo	—	4–5 y	7–8 y	10 y
Lateral incisor	10–12 mo	—	4–5 y	8–9 y	11 y
Canine	4–5 mo	—	6–7 y	11–12 y	13–15 y
First premolar	1.5–1.75 y	—	5–6 y	10–11 y	12–13 y
Second premolar	2–2.25 y	—	6–7 y	10–12 y	12–14 y
First molar	At birth	Trace amount	2.5–3 y	6–7 y	9–10 y
Second molar	2.5–3 y	—	7–8 y	12–13 y	14–16 y
Mandibular					
Central incisor	3–4 mo	—	4–5 y	6–7 y	9 y
Lateral incisor	3–4 mo	—	4–5 y	7–8 y	10 y
Canine	4–5 mo	—	6–7 y	9–10 y	12–14 y
First premolar	1.75–2 y	—	5–6 y	10–12 y	12–13 y
Second premolar	2.25–2.5 y	—	6–7 y	11–12 y	13–14 y
First molar	At birth	Trace amount	2.5–3 y	6–7 y	9–10 y
Second molar	2.5–3 y	—	7–8 y	11–13 y	14–15 y

Reproduced with permission from Logan WH, Kronfeld R: Development of the human jaws and surrounding structures from birth to the age of fifteen years, *J Am Dent Assoc* 20:379-427, 1933.

as a result of greater forward growth of the mandible when compared with the maxilla (consistent with the overall cephalocaudal growth gradient) as well as continued development of the chin. This will be discussed in more detail below.

The Mixed Dentition

The mixed dentition is characterized by the presence of primary and permanent teeth beginning with the eruption of the permanent incisors and first molars usually between 6 and 7 years of age (Fig. 371-1B). The central incisors should erupt prior to the lateral incisors, with the mandibular teeth usually erupting before the maxillary teeth. In some children, eruption is precocious and in others it's delayed; there is a wide range of normal. However, if a tooth has erupted normally and the contralateral tooth is delayed in eruption by greater than 6 months' time, the patient should be referred for evaluation. Delayed tooth eruption is the most commonly encountered deviation from normal eruption time and may indicate a systemic problem such as an endocrine disorder or Down syndrome. Delayed tooth eruption is more frequently the result of local factors such as the presence of a supernumerary tooth or ectopic eruption of the delayed tooth (Table 371-2). Another issue that may be observed is ectopic eruption of the first permanent molar, which may cause the premature loss of the adjacent second primary molar. This occurs in 2% to 6% of children, more frequently in the maxilla than in the mandible. Timely diagnosis and treatment of ectopic molar eruption are important in preventing space loss and the malocclusion that might follow.

Once the permanent incisors and molars have completely erupted, there is usually a period of several years when no exfoliation of primary teeth or eruption of permanent teeth is observed. The transition to the permanent dentition usually begins between 9 and 11 years of age and continues to approximately age 14 years. The sequence of eruption is different in the mandible and the maxilla. In the mandible, the expected pattern is canine, first premolar, second premolar (premolars, also called bicuspids, replace the primary molars), and second molar. In the maxilla, the sequence is usually first premolar, canine, second premolar, and second molar. It is during this stage of development that 1 or both maxillary canine teeth may encounter difficulty in erupting. Ectopic maxillary canines occur in 1% to 3% of the population and can result in impaction (failure to erupt) of the canine itself and/or cause resorption of the roots of the neighboring permanent teeth (usually the lateral incisors), possibly even resulting in the complete loss of a tooth or teeth. A dentist should investigate significant delay in the eruption of 1 or both maxillary canines.

The spaces between the anterior teeth that may have been present in the primary dentition are largely consumed when the larger permanent incisors erupt ("incisor liability"). Space may remain between the maxillary central incisors secondary to a thick labial frenum (Fig. 371-3) that can prevent its normal closure. However, 83% of the patients with a maxillary midline diastema at 9 years of age do not have one at age 16, so most diastemas observed at this age do not require treatment. Importantly, the space available to accommodate the teeth from first permanent molar to first permanent molar (arch circumference) increases only marginally in the maxilla and actually decreases in the mandible during the mixed and permanent dentitions. Skeletal growth does not result in more space for the

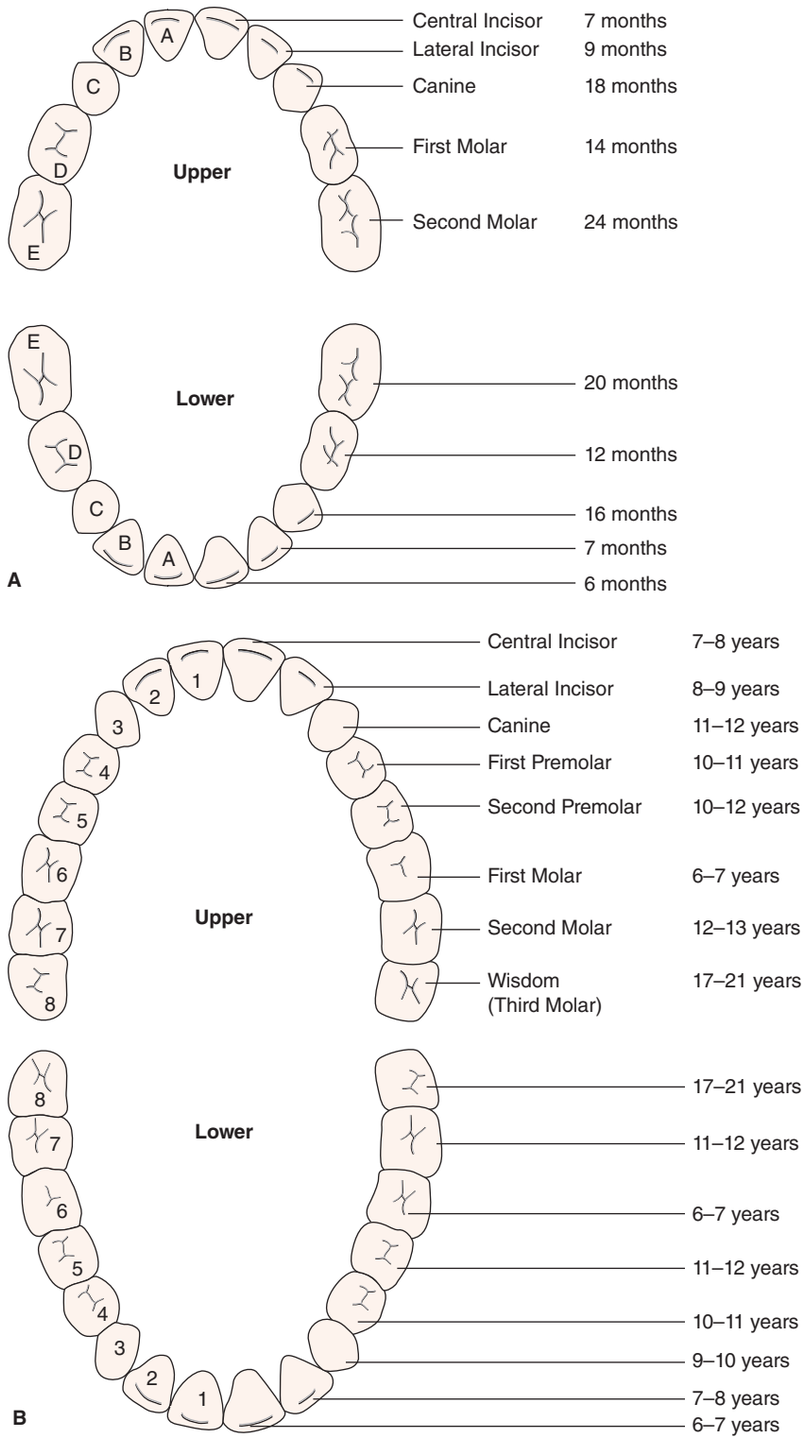

FIGURE 371-1 **A:** Schematic of the primary dentition with eruption times. **B:** Schematic of the permanent dentition with eruption times.

successional teeth in this region but does help to accommodate the teeth posterior to the first permanent molars (accessional teeth). The permanent canines and premolars are usually slightly smaller in size than the primary teeth that they replace ("leeway space"). Managing this space in the late mixed dentition may be important in the treatment of crowding and the ability to treat such crowding without the need to extract permanent teeth.

Normal occlusion in the mixed dentition is similar to that found in the primary dentition. A notable difference is in the incisor regions where crowding may develop, which occurs commonly if the interdental spacing that was present in the primary dentition is not adequate to accommodate the larger permanent incisors. The eventual nature of the final occlusion certainly becomes more defined as the mixed dentition develops.

Skeletal relationships in the mixed dentition stage are similar to those found in the primary dentition stage; however, the profile might become noticeably straighter as the forward growth of the mandible exceeds that of the maxilla.

The Adolescent and Permanent Dentitions

The adolescent or early permanent dentition begins when the last primary tooth is lost, usually between 10 and 13 years of age. As this happens, the second permanent molars erupt behind the first molars, resulting in subtle changes in the molar occlusion. Both the occlusion

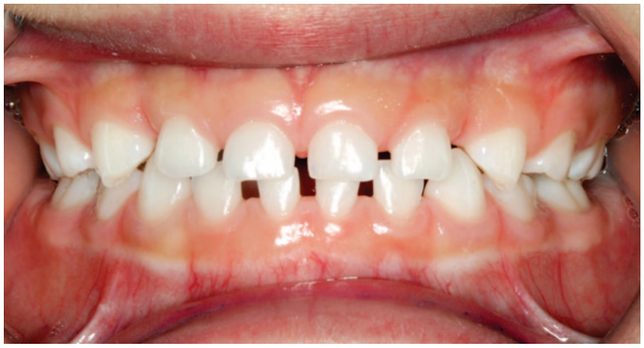

FIGURE 371-2 Normal primary dentition.

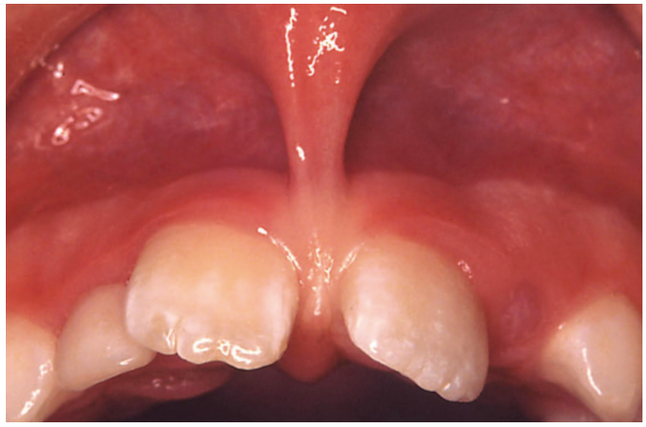

FIGURE 371-3 Thick maxillary labial frenum in the mixed dentition.

and the profile may continue to change as skeletal (particularly late mandibular) growth in the horizontal and vertical sagittal planes may alter the relationship of the jaws to each other. This may reveal or exacerbate late-developing problems such as skeletally based class III (often referred to with the lay term "underbite") or open-bite malocclusions, respectively. This growth may continue even after all other skeletal growth has been completed. The eruption of third molars (wisdom teeth) is typically expected in late adolescence but may not occur until well into the third decade of life. Third molar teeth are often implicated in the development of mandibular dental crowding, although it is universally accepted that late mandibular growth is central to the development of such crowding, which may develop even in the absence of third molar development.

FACIAL ANALYSIS

Facial analysis is indispensable in the evaluation of a child's occlusion. It provides important insight into the relationship among the bones of the facial skeleton and helps to determine the presence and nature of any anteroposterior discrepancies, vertical dysplasias, functional shifts, or craniofacial growth problems. The pediatrician can easily accomplish this during a routine clinical exam by using soft tissue landmarks that estimate the anterior extent of the cranial base, the maxilla, and the mandible. These landmarks are the bridge of the nose, the base of the nose, and the tip of the chin, respectively. Two lines can be mentally constructed between these points to describe the facial profile as convex, straight, or concave (Fig. 371-4). Vertical problems can be assessed using similar landmarks by determining if the portion of the face from the base of the nose to the bottom of the chin (lower face height) is properly related to the total face height (bridge of the nose to the bottom of the chin). Normal lower face height is approximately 55% of the total face height. Facial symmetry is best assessed from a posterosuperior view by determining if the chin point is approximately coincident with the facial midline. Mild asymmetry of the chin or nose (up to several mm) is considered to be within normal limits. Both profile and facial symmetry must be assessed when the mandible is in its normal position, not while it is artificially postured as sometimes happens when children are examined.

TABLE 371-2 COMMON FACTORS AND CONDITIONS ASSOCIATED WITH DELAYED TOOTH ERUPTION

Local	Systemic
Crowding	Vitamin D–resistant rickets
Ectopic teeth	Endocrine disorders
Supernumerary teeth	Radiation or chemotherapy
Early loss of primary teeth	Nutritional deficiency
Odontoma	Down syndrome
Gingival hyperplasia	Apert syndrome
Primary tooth ankylosis	Cleidocranial dysplasia

MALOCCLUSION

The American Association of Orthodontists (AAO) recommends that an orthodontist examine all children no later than 7 years of age, although a pediatric dentist would likely recognize any developing malocclusion prior to such an age. By the age of 7 years, the posterior occlusion is established as the first molar teeth erupt, the incisor teeth have begun to erupt, and problems such as crowding, anterior crossbites, posterior crossbites, open bites, oral habits, maxillary protrusion, and some developing facial asymmetries (Table 371-3) can be detected. A timely evaluation will lead to significant treatment benefits for some or reassurance that the child's dental growth and development are normal. Recognition and basic management of these disorders are summarized briefly in the sections that follow.

For those patients who have clear indications for early intervention, early treatment presents an opportunity to

- influence jaw growth in a positive manner;
- harmonize widths of the dental arches;
- improve eruption patterns;
- lower the risk of trauma to protruded upper incisors;
- correct harmful oral habits;
- improve aesthetics and self-esteem;
- simplify and/or shorten treatment time for later corrective orthodontics;
- reduce the likelihood of impacted permanent teeth;
- improve some speech problems; and
- preserve or gain space for erupting permanent teeth.

FIGURE 371-4 Variations in profile can be seen using 3 soft tissue landmarks. This is an example of a concave profile.

TABLE 371-3 COMMON ORTHODONTIC PROBLEMS DETECTABLE IN 7-YEAR-OLD

Anterior crossbite
Posterior crossbite
Dental crowding or arch-length deficiencies
Open bite
Excessive protrusion of teeth
Ectopic eruption
Skeletal imbalances between the maxilla and mandible
Diastemas or excessive spacing between teeth
Oral habits associated with malocclusions

DENTAL CROWDING

Dental crowding, or the lack of space for teeth in the dental arches, is one of the most commonly presenting malocclusions and is typically a primary reason for an individual to seek orthodontic treatment.

PATHOGENESIS AND EPIDEMIOLOGY

Crowding may affect as many as 40% of children aged 6 to 11 years and 85% of children aged 12 to 17 years. The etiology of crowding is multifactorial and includes tooth-size/jaw-size discrepancies (genetic), loss of arch length (due to interproximal caries or the premature loss of primary teeth), ectopic eruption of teeth, supernumerary teeth, genetic influences, jaw growth disharmonies, oral habits, and serious oral conditions.

CLINICAL MANIFESTATIONS AND DIAGNOSIS

Crowding often manifests itself clinically as poorly aligned teeth and can be associated with delayed eruption of teeth, over-retained primary teeth, ectopic eruption of teeth (which may result in early loss of primary teeth), and impaction of teeth. Difficulty in maintaining adequate hygiene in areas of crowding accompanied by ectopic position of teeth and possibly, traumatic occlusion may lead to periodontal problems. Diagnosing crowding involves an examination and analysis of the space (using physical or virtual dental casts) required to align the secondary teeth, the space currently available within each of the dental arches, the occlusion, the profile, the periodontal health, and the oral hygiene status. Analysis must also consider future growth and development of the jaws, the teeth, and the dental arches.

The premature loss of primary teeth, particularly molars, may require the use of fixed or removable space maintainers to prevent the development of crowding or the exacerbation of existing crowding. Failure to do so may result in the need for extraction of permanent teeth, challenges in maintaining adequate oral hygiene, and/or the need for more complicated and lengthy orthodontic treatment (Fig. 371-5). A healthy and complete primary dentition plays an essential role in maintaining the space needed for the permanent teeth. A patient with prematurely lost, missing, or decayed teeth should be referred to a pediatric/family dentist or orthodontist for management as soon as the problem is detected.

Crowding in the Mixed Dentition Treatment modalities range from space maintenance to comprehensive orthodontic treatment that may involve the removal of several permanent teeth. Pediatricians should encourage their patients with crowded dentitions to be evaluated while in the mixed dentition stage in order to preserve the choice between extraction and nonextraction treatment.

COMPLICATIONS AND LATE EFFECTS

Delayed evaluation by an orthodontist or pediatric dentist may lead to the need for the removal of permanent teeth to resolve arch-length/tooth-size discrepancy. Untreated dental caries involving the interproximal surfaces of teeth may result in space loss and subsequently lead to the development of crowding.

ANTERIOR CROSSBITE

An anterior crossbite is defined as the lingual (inset) position of 1 or more maxillary anterior teeth in relationship with the corresponding mandibular anterior teeth (Fig. 371-6).

PATHOGENESIS AND EPIDEMIOLOGY

Anterior crossbites may be differentiated into dental, functional, and skeletal crossbites with any combination possible. "Dental" implies that malposition of teeth within the alveolar bone is the cause of the crossbite. "Skeletal" implies that the jaws themselves are misaligned and responsible for the crossbite. "Functional" indicates a forward shift of the mandible upon closing due to the premature contact of opposing teeth. The prevalence of skeletal class III malocclusions, where the maxilla is retrusive as compared to the anteroposterior position of the mandible, is estimated at 3% to 5% in the white population and up to 14% in the Chinese and Japanese population. Despite the relatively low incidence of this skeletal discrepancy in white patients, it has been reported that approximately one-third of orthognathic surgery patients present with this type of malocclusion.

CLINICAL MANIFESTATIONS AND DIAGNOSIS

Dental crossbites are diagnosed by ruling out both functional and skeletal contributions through clinical and possibly radiographic (eg, lateral cephalometric analysis) examination of the patient. Diagnosis of a skeletal or functional anterior crossbite may be aided by viewing the patient in profile. The child will usually present with a concave facial profile with the base of the nose deficient in relation to the chin point. If a skeletal crossbite results from maxillary retrusion, an associated malar deficiency will often be noticeable.

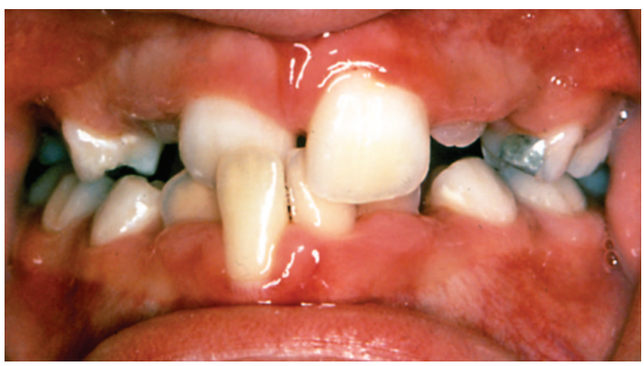

FIGURE 371-5 Crowding in the mixed dentition.

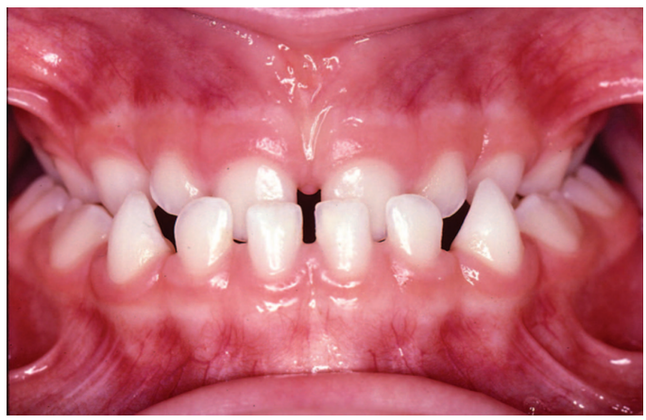

FIGURE 371-6 Anterior crossbite.

TREATMENT

The modality of treatment depends upon what stage the dentition is in, the involvement of a single tooth or several teeth, and the causative factors. A child presenting in the primary dentition (usually age 5 years or younger) may have a single incisor in dental crossbite which may also contact prematurely when biting (traumatic occlusion). Treatment options include simple reassurance, periodic monitoring, occlusal adjustment of the tooth responsible for the interference, appropriately timed extraction of the offending tooth (if it is close to exfoliation), and use of a removable or a fixed appliance, depending on the child's level of maturity and compliance.

A pseudo-class III malocclusion occurs when a child protrudes his/her mandible into an anterior crossbite (anterior functional shift) to avoid occlusal interferences. Treatment as simple as tilting or proclining the maxillary incisors or trimming down the interfering tooth is often all that is required to eliminate interferences and the crossbite. A crossbite of 2 or more incisors can generally be corrected with an upper removable appliance fastened to the first permanent molars. An anterior bite platform of acrylic will reduce the vertical overlap (overbite) of the teeth in crossbite and allow free passage as the tooth is proclined.

Treatment regimens for the child with mixed dentition (usually 6–11 years of age) include removable or fixed appliances and orthopedic devices. Appliance therapy may include expansion of the maxillary arch that creates room for alignment of a palatally displaced tooth. Maxillary retrognathia in preadolescent children may be successfully addressed with protraction facemask therapy with the aim of distracting the midface forward by applying pressure to separate the circummaxillary sutures. This treatment has limitations and risks and should be entered into only under the supervision of a specialist.

COMPLICATIONS AND LATE EFFECTS

Failure to correct anterior crossbites in a timely manner may lead to untoward growth of the skeletal and dentoalveolar components of the craniofacial complex, attrition of the incisors and canines, periodontal problems, and functional posterior crossbites. Treatment may also prevent habits such as bruxism as well as reestablish proper muscle balance.

The timing of treatment for a skeletal anterior crossbite malocclusion is critical when efforts to redress the problem involve a dentofacial orthopedic approach aimed at manipulation of the midfacial sutures. If treatment is undertaken prior to the age of 10 years, interceptive orthodontic treatment can be effective in correcting this malocclusion in as many as 75% of cases. Once the child and the midfacial sutures mature, the treatment options become more limited, and complicated. A recent development involves the use of so-called "Bollard" plates. These skeletal fixation plates, with transcutaneous hooks passing through the gingivae, are placed in the canine region of the mandible and the zygomatic buttress region of the maxilla of a patient in the late mixed or early adolescent dentition. The application of interarch elastic traction to these hooks may provide the orthodontic specialist another opportunity to influence jaw positions, although adequate supporting evidence has not yet been published. Definitive correction of an anterior crossbite in the physically mature individual may involve surgical correction at growth completion, or camouflage treatment involving the removal of permanent teeth and corresponding reductions in arch circumference. It must be understood that some of these malocclusions cannot be successfully treated without a combined orthodontic and surgical approach and that careful diagnosis and treatment planning is essential in all cases.

POSTERIOR CROSSBITE

A posterior crossbite malocclusion is defined as the abnormal buccolingual relationship between 2 or more maxillary and mandibular posterior teeth.

PATHOGENESIS AND EPIDEMIOLOGY

The prevalence of a posterior crossbite is estimated to be approximately 7.7% to 17.6% of preadolescent children. The etiology of transverse maxillary deficiency is considered multifactorial and includes genetic, environmental, traumatic, and iatrogenic causes. Many constricted maxillae may be the result of abnormal function or habits such as persistent thumb sucking. Researchers have been successful in their efforts to iatrogenically create narrow maxillary dental arches in rhesus monkeys by converting them from a nasal to obligatory oral respiration. Another example of an iatrogenic cause is the surgical repair of a young infant who presents with a cleft palate; the surgical scarring may restrict not only the subsequent anteroposterior but also the transverse growth of the maxilla.

CLINICAL MANIFESTATIONS AND DIAGNOSIS

Diagnosis is made primarily by clinical examination, perhaps with the aid of radiographic (eg, frontal cephalometric analysis) study, and an evaluation of functional shifting as previously described for anterior crossbites. The most common type of posterior crossbite occurs when the buccal cusps of the maxillary molars occlude palatally (inset) to the buccal cusps of the mandibular molars, unilaterally or bilaterally (Fig. 371-7). Much less common is the buccal, or "Brodie," crossbite, which occurs when the palatal cusps of the maxillary molars occlude to the outside of the buccal cusps of the mandibular molars. Posterior crossbites in children often appear to be unilateral, but on close examination are usually found to result from bilateral constriction of the maxillary arch and a shift of the mandible to one side on closure.

Dentoalveolar crossbites are usually due to insufficient arch length or the prolonged retention of deciduous teeth, both of which can cause ectopic eruption of a tooth or teeth into crossbite. A skeletal crossbite is related to discrepancies between the transverse dimensions of the maxilla and mandible and could be due to a narrow maxilla, a wide mandible, or a combination of both. Determining the dental or skeletal component of posterior crossbite represents the first step in treatment planning.

Posterior crossbites accompanied by a functional shift of the mandible are the most common type of unilateral crossbites found in the growing child, and it is important that they should be treated as early as possible. The pediatrician may determine if there is a lateral functional shift of the mandible by assessing the position of the chin in the posterosuperior view when the teeth are in occlusion and when they are not. When this condition is treated at the appropriate time, compensatory growth of the mandible may eliminate any positional and skeletal asymmetries, thereby creating optimum conditions for normal craniofacial growth and development.

TREATMENT

Treatment differs substantially depending on the underlying cause of the malocclusion. Skeletal crossbites, which usually result from a narrow maxilla but occasionally from an excessively wide mandible, are generally treated by heavy forces to open the midpalatal suture

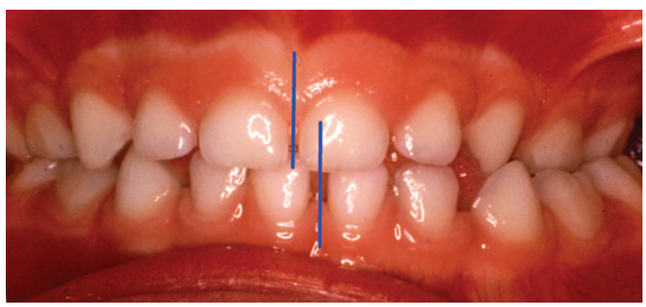

FIGURE 371-7 Posterior crossbite resulting from a maxillary constriction with a functional shift of the mandible to the left. Note noncoincident dental midlines.

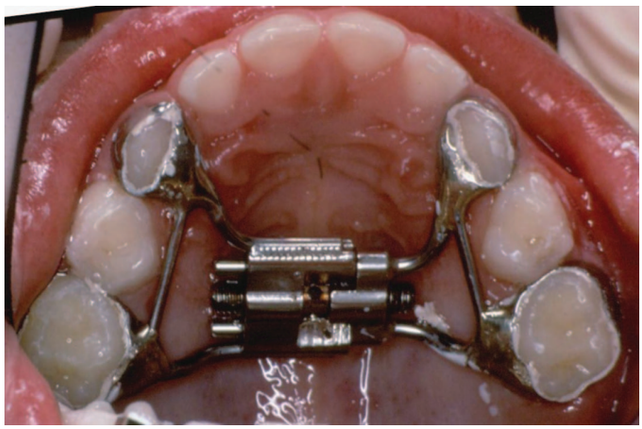

FIGURE 371-8 Fixed maxillary expander in the primary dentition.

and make the maxilla wider. Dental crossbites are treated by moving the teeth with lighter forces. Although this is a correct concept for children in the late mixed to early permanent dentition, the lesser interdigitation of the midpalatal suture in the late primary and early mixed dentitions means that even modest forces will cause both skeletal and dental changes while increased interdigitation at older ages may result in untoward movement of teeth without the planned widening of the maxilla (Fig. 371-8).

COMPLICATIONS AND LATE EFFECTS

The injudicious use of orthopedic expansion appliances may lead to pathological effects such as root resorption, alveolar dehiscence, and root fenestration.

OPEN BITE AND ORAL HABITS

An open bite has been defined as an occlusal characteristic in which upper and lower teeth are not in contact and vertical overlap does not exist. Although this type of malocclusion can occur unilaterally or bilaterally in the buccal segments, it most often occurs in the anterior segment. Dental open bites are most common, while skeletal open bites characterized by problems with the relative positions of the maxilla and the mandible are less frequent.

PATHOGENESIS AND EPIDEMIOLOGY

Open bites are more common in the younger, mixed dentition age group. They are often found in conjunction with horizontal sagittal dysplasias of the jaws, such as "underbite" types of malocclusions, but may also present in isolation. Some open bites correct themselves, especially during puberty, lending support to the theory that open bite, at least in part, is a dynamic condition subject to change.

There may be a racial predilection in the incidence of open bite. African Americans have a higher incidence (6.6%) than do whites (2.9%) or Hispanics (2.1%). The highest reported incidence of anterior open bite malocclusion is found in children with developmental delays and in children with Down syndrome.

Although the etiology of open bite is often difficult to confirm, many factors have been implicated, including genetic factors and non-inherited factors, such as trauma to the condyles, muscle dysfunction, or pathological disturbances of dental development (eg, supernumerary teeth, ankylosis, root dilacerations, cysts, or prolonged retention of deciduous teeth). Chronic oral habits, such as non-nutritive sucking, tongue thrusting, and mouth breathing, may be causative or contributing factors in the majority of open bite malocclusions.

During the primary dentition and early mixed dentition years, many children engage in finger and pacifier sucking. Although a prolonged and intense habit may deform the alveolus and dental arch form during the primary dentition years, much of the effect is on eruption of the permanent anterior teeth. The effect of such a habit on the hard and soft tissues depends on its frequency (hours per day), intensity, and duration (months vs years). While most children will discontinue sucking habits by the age of 3 or 4 years, some may continue. If the finger sucking habit ceases prior to the eruption of the permanent incisors, any dental changes resulting from the habit may resolve spontaneously. However, persistent sucking habits extending into the mixed and permanent dentition age groups may result in an anterior open bite malocclusion that requires intervention to resolve.

The etiological relationship of tongue thrusting and open bite malocclusion is not completely clear. While tongue thrusting may be considered by some as a direct cause of anterior open bite, the prevailing view is that the tongue thrust is an adaptation used to achieve a good oral seal during swallowing, with the open bite actually caused by a sucking habit or the result of skeletal factors. Respiratory patterns and airway obstructions have also been implicated in the etiology of open bite malocclusions, although experimental studies have failed to demonstrate a significant clinical correlation between airway obstruction and the development of open bite malocclusions.

CLINICAL MANIFESTATIONS AND DIAGNOSIS

An open bite malocclusion, most commonly found in the incisor region (Fig. 371-9), may also be between the maxillary and mandibular posterior teeth in the case of a posterior or lateral open bite. Dental open bites are restricted to malposition of the teeth (proclined incisors, infraocclusion) and deformation of the surrounding alveolar bone. Skeletal open bites are characterized by problems with the underlying growth patterns leading to improper relative positions of the maxilla and/or the mandible. A patient presenting with a dental open bite malocclusion will have relatively normal skeletal components, whereas in skeletal open bite malocclusions, a discrepancy in the cranial base, maxilla, or mandible may be observed.

TREATMENT

The management and retention of the results brought about by treatment of the malocclusion in the vertical dimension are challenging because the etiology is multifactorial, involving hereditary, skeletal, dental, respiratory, and habitual factors. The stability of treatment in the growing and nongrowing patients with hyperdivergent facial patterns depends on the clinician's ability to address the cause of the malocclusion.

Open bite malocclusion therapy in the mixed and early permanent dentition is directed toward addressing obvious etiological factors, eg, removing pathological obstructions of eruption of the anterior teeth and controlling oral habits. Skeletal discrepancies should be treated through intervention with ongoing growth, focusing on reducing or redirecting the vertical skeletal growth and controlling vertical dentoalveolar development by impeding molar eruption or extruding anterior teeth.

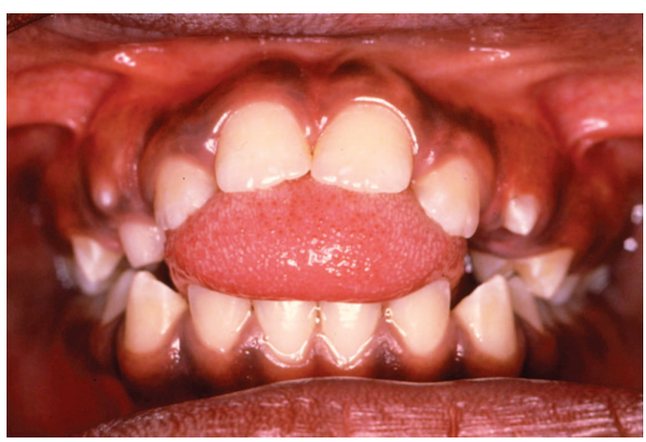

FIGURE 371-9 Anterior open bite.

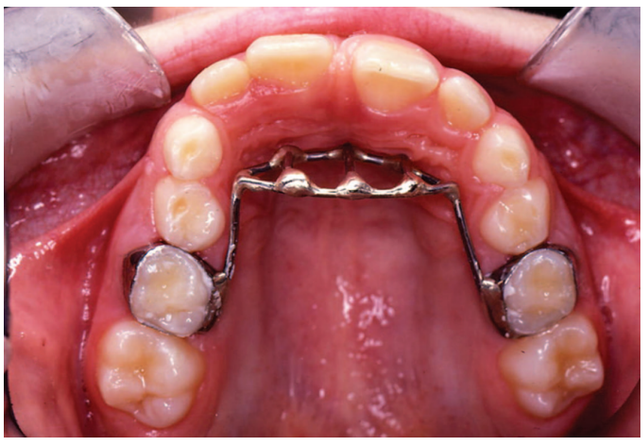

FIGURE 371-10 Palatal crib.

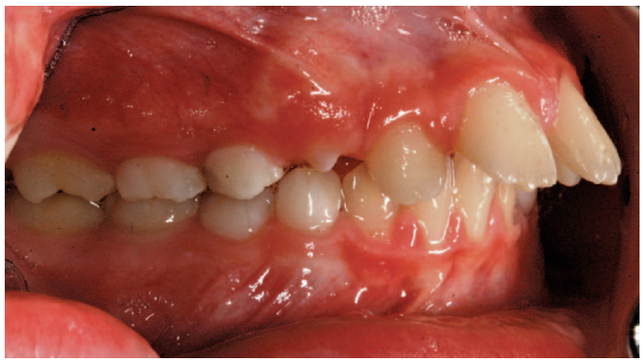

FIGURE 371-11 Maxillary protrusion.

The timing of surgical intervention, when necessary, is critical in a young adolescent because the subsequent effects of orthognathic surgery on the dentofacial complex are poorly understood and controversial.

The treatment of open bites resulting from ongoing oral habits involves addressing the habit itself. Most oral habits are simple, learned behaviors and not symptoms of emotional disturbance. Treating a habit causing a malocclusion in a child who is mature enough to understand the process will not result in any serious symptom substitution. A straightforward discussion with the parent, child, and dentist and counseling the child on the detrimental effects of the habit should be the first attempt at treatment. A reward system that encourages the child to cease the habit with daily accountability measures and reminders may also prove to be effective. Beyond this, physical reminders such as a bandage on the offending finger are the next level of intervention to be considered.

Interceptive therapy using removable or fixed orthodontic appliances such as a palatal crib are the next line of measures available. With the use of any appliance it is imperative that the child be informed that the appliance is meant only as a reminder to assist the child in breaking the habit, not as a punitive measure. A fixed palatal crib (Fig. 371-10) is an effective measure in arresting a persistent digit-sucking habit and serves to restrain the tongue, therefore allowing for some degree of self-correction of the open bite to occur.

COMPLICATIONS AND LATE EFFECTS

An uncontrolled habit of sufficient frequency, duration, and intensity may maintain or exacerbate the open bite malocclusion and significantly worsen the prognosis for successful orthodontic treatment. Failure to address the underlying etiology of an open bite will directly affect the correction achieved and typically result in regress of the original condition. If an open bite is left unresolved, an individual's speech and masticatory function will be compromised, in addition to the undermined facial and dental aesthetics.

MAXILLARY INCISOR PROTRUSION

Overjet is defined as the horizontal overlap of the upper incisors beyond the lower incisors. Normally, the upper incisors are ahead of the lower by the thickness of the incisal edges, which is approximately 2 to 3 mm. Maxillary incisor protrusion, defined as an excessive overjet (≥5 mm) may be accompanied by lip incompetence.

PATHOGENESIS AND EPIDEMIOLOGY

Maxillary incisor protrusion occurs in approximately 23% of children, 15% of adolescents, and 13% of adults (Fig. 371-11). The most common causes of increased maxillary incisor protrusion are the presence of oral habits (such as digit sucking or tongue thrusting); a predominant oral method of respiration (mouth breathing); abnormal soft tissue function, including a retained infantile swallowing habit, anterior tongue posture or thrust, and a hypotonic upper lip; dental crowding, including the maxillary lateral incisors being blocked out palatally; skeletal discrepancies between the positions of the upper and lower jaw; and miscellaneous causes including ectopic eruption, pathology, and supernumerary teeth.

CLINICAL MANIFESTATIONS AND DIAGNOSIS

The diagnosis of maxillary protrusion is again made primarily by clinical examination aided, when necessary, by lateral cephalometric analysis to determine the degree to which jaw positions contribute to the malocclusion. Maxillary protrusion may represent a normal developmental period as seen during the transition from the primary to the mixed dentition or from the mixed to the permanent dentition, but may also be related to an underlying dental or skeletal malocclusion.

TREATMENT

The rationale for treatment of protruding incisors are to (1) protect against imminent dental trauma, (2) to alleviate psychological trauma, (3) to obtain an adequate lip seal between the upper and lower lips and encourage proper development of lip and cheek function, and (4) to improve the appearance.

The approach to treating maxillary protrusion in children and adolescents is largely determined by the underlying etiology of the malocclusion. If a skeletal discrepancy exists, treatment options include extraoral traction (headgear), the use of functional appliances, or orthognathic surgery, if severe. Therapeutic techniques used to address maxillary protrusion of dental etiology include braces (fixed appliances), removable appliances, the distalization of the maxillary teeth, or camouflage treatment involving the removal of permanent teeth.

COMPLICATIONS AND LATE EFFECTS

Children with maxillary protrusion are at increased risk for injuries to their maxillary anterior teeth. Early treatment of excessive overjet should be considered for this reason alone, but may also serve to improve the child's self-image and their perception by peers and adults. Studies indicate, however, that young children treated in this manner will most often require a second phase of orthodontic treatment and that 1-stage treatment at a later time may provide a similar final outcome in most cases. Overjet is the leading reason why parents seek orthodontic treatment for their children. Additionally, addressing an increased overjet can serve to maintain the periodontal health of the anterior gingiva and prevent further development of abnormal soft tissue function and underlying skeletal discrepancies.

CONCLUSION

Managing the developing occlusion requires an in-depth understanding of dental/craniofacial growth and development, the effect of outside influences on this natural course, and the opportunities that exist

to intervene and improve the outcome. Successful "treatment" may involve knowing when not to act, as well as when and how to provide therapy that has the highest chance for a successful outcome. Treatment planning in orthodontics is based on accurate diagnosis and should be done by a specialist. The primary role of the pediatrician is to make sure that the child has an appropriate dental home as well as to assess for evidence of malocclusion and make timely referrals to a pediatric dentist or orthodontist.

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372 Periodontal and Gingival Health and Diseases

Nadeem Y. Karimbux, Irina F. Dragan, and David M. Kim

INTRODUCTION

The normal periodontium consists of gingiva, connective tissue, the periodontal ligament, cementum, and the surrounding alveolar bone. Clinicians commonly describe the healthy gingiva as being scalloped, firm, and knife-edged. The lack of bleeding on probing and the lack of exudates are also taken as clinical signs of health. In children, similar characteristics are observed and recorded (although there are more spaces between primary teeth resulting in a flatter, less scalloped appearance). Several different forms of gingival and periodontal disease in children and adolescents that can change the appearance (eg, erythema), contour (eg, swelling), size (eg, hyperplasia or overgrowth), and shape (eg, blunted papillae) of the gingiva range from reversible conditions such as inflammation of gingival tissues (ie, gingivitis) to those characterized by the destruction of the periodontal connective tissue attachment and alveolar bone (ie, periodontitis). If these conditions are left untreated, the deciduous or permanent dentitions may be jeopardized. Thus, fundamental principles concerning the etiology and the contributing factors of periodontal diseases need to be understood in order to identify and manage them as well to minimize or prevent complications.

According to the American Academy of Periodontology, periodontal disease in children and adolescents can be classified into 5 distinct periodontal disease categories:

1. Dental plaque–induced gingival diseases
2. Chronic periodontitis
3. Aggressive periodontitis
4. Periodontitis as a manifestation of systemic diseases
5. Necrotizing periodontal diseases

In 2014, the American Academy of Periodontology proposed an update that focuses on guidelines for determining the severity of the disease, taking into consideration the following parameters: probing depth, bleeding on probing, radiographic bone loss, and clinical attachment loss. The 3 categories for the severity remained the same: slight (mild), moderate, and severe (advanced). Further guidelines were presented in order to differentiate between chronic and aggressive forms of periodontitis. Aggressive periodontitis is diagnosed when there is onset of clinical attachment and radiographic bone loss (detected on clinical and radiographic examination) before the age of 25. An updated comprehensive classification is planned for development by 2017 will include peri-implant diseases and risk assessment factors for periodontal diseases.

EPIDEMIOLOGY

Gingivitis of varying severity is nearly universal in children and adolescents; however, the prevalence of destructive forms of periodontal disease is lower in children and adolescents than in adults. The prevalence rate of periodontitis in children ages 5 to 11 years is up to 9% and increases to up to 46% for those 12 to 15 years of age. Thus, children and adolescents should receive a periodic periodontal evaluation as a component of routine dental visits, because periodontitis is usually preceded by gingivitis.

Periodontitis is a multifactorial disease, and there are several local and systemic factors that are associated with it. Local contributing factors such as caries, inadequate restoration, and subgingival calculus may contribute to a bacterial accumulation resulting in a host-mediated inflammatory response (Fig. 372-1). Patients with an impaired host response from underlying conditions such as acquired immunodeficiency syndrome (AIDS), agranulocytosis, neutropenia, or neutrophil dysfunction are at a higher risk of developing periodontitis. Ethnic and socioeconomic factors have also been associated with the incidence and prevalence of periodontitis. For example, black children have a higher prevalence of localized aggressive periodontitis than white children.

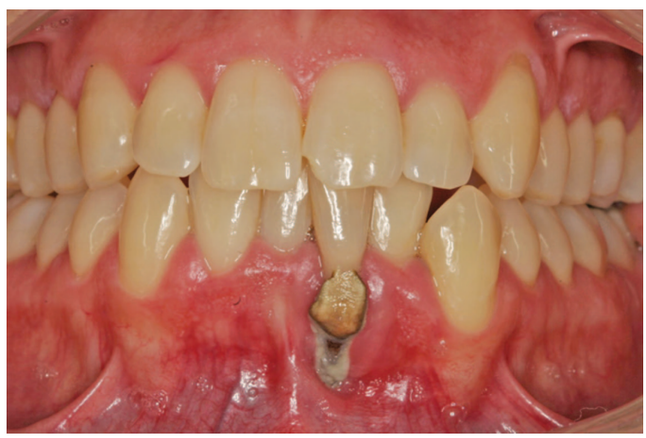

FIGURE 372-1 Clinical evidence of calculus buildup on mandibular central incisor causing recession and inflammation of the affected gingiva.

ASSOCIATED DISORDERS

The periodontal classification system for children and young adults has evolved over time. The most recent classification system has taken into account the latest information of pathophysiology, diagnosis, host defense, and microbiology. The 5 groups of gingival and periodontal diseases that occur most often in children and adolescents are listed below.

Dental Plaque–Induced Gingival Diseases

Dental plaque–induced gingival diseases are the most prevalent forms of periodontal disease in children and adolescents and are typically caused by nonspecific bacterial infections. They include the following:

- Gingivitis associated with dental plaque only (with or without local contributing factors)
- Gingival diseases modified by systemic factors
- Gingival disease modified by medications
- Gingival diseases modified by nutrition

Chronic Periodontitis

Chronic periodontitis, which may be localized or generalized, is more prevalent than other forms of periodontitis in young people. Similar to chronic adult periodontitis, this is a more localized disease process with less severe periodontal tissue loss. Local factors and environmental risk factors appear to play a major role in the initiation and progression of this disease, with a smaller role for genetic predisposition. Poor oral hygiene, local plaque-retaining factors, and smoking are important causative factors.

Aggressive Periodontitis

Aggressive periodontitis, which may also be localized or generalized, is characterized by severe and rapid loss of periodontal tissues that commences at or after the circumpubertal age. It may be caused by multiple factors; local factors appear to play a considerable role in its pathogenesis and there is a significant genetic predisposition. Aggressive periodontitis is more frequent in older age groups and in certain ethnic groups, particularly those of African or Hispanic descent.

Periodontitis as a Manifestation of Systemic Diseases

Periodontitis as a manifestation of systemic disease usually commences before puberty and may affect the deciduous and permanent teeth. Many of these diseases have a clear genetic etiology, although local factors may be responsible for initiating the periodontal inflammation.

Necrotizing Periodontal Diseases

Necrotizing periodontal diseases, which include necrotizing ulcerative gingivitis (NUG) and necrotizing ulcerative periodontitis (NUP), are associated with a diminished host resistance to bacterial infection of periodontal tissues. The immunosuppression predisposing to necrotizing periodontal disease may result from environmental factors such as malnutrition, psychological and/or physical stress, poor oral hygiene, alcohol use, or smoking. Necrotizing periodontal diseases are more common in patients from low socioeconomic backgrounds.

PATHOPHYSIOLOGY

Bacterial accumulation, resulting in a cascade of host-mediated inflammatory response, is the pathway to periodontal soft and hard tissue destruction. The predominance of a Gram-negative bacterial flora in combination with the cellular and cytokine profiles of the lesions indicates the likelihood that bacterial lipopolysaccharide activation of monocytes and subsequent production of tissue-destructive cytokines is likely to be a major pathway of destruction. Functional abnormalities (eg, increased adherence, abnormal signal transduction, or depressed chemotaxis) in neutrophils and monocytes taken from localized aggressive periodontitis patients have been reported.

Aggregatibacter actinomycetemcomitans, *Prevotella intermedia*, *Porphyromonas gingivalis*, *Tannerella forsythensis*, and *Treponema denticola* are Gram-negative organisms that have been found in children and young adults with periodontal attachment loss. Results of several studies suggest that *A actinomycetemcomitans* is a major etiologic factor in the pathogenesis of aggressive periodontitis. Spirochetes, Gram-positive cocci, β-hemolytic streptococci, *Borrelia* species, *P gingivalis*, *P intermedia*, and *Candida albicans* may also be present in patients with necrotizing periodontal disease. However, initiation of the tissue destruction in chronic periodontitis may be the result of the accumulation of subgroups of microorganisms and their bacterial products.

Gingivitis is histologically characterized by ulceration of the sulcular epithelium and inflammatory cell infiltration of the underlying connective tissue. Periodontal diseases, which are more destructive, are characterized by inflammation of the periodontal tissue, leading to an apical migration of the epithelial attachment and loss of periodontal soft and hard tissues.

The genetic component of periodontal disease can impact the initiation, path of progression, and response to treatment. In addition, it has long been recognized that severe attachment loss often accompanies certain genetic disorders such as Papillon-Lefèvre syndrome, Down syndrome, congenital neutropenia, leukocyte adhesion deficiency, Chédiak-Higashi syndrome, and other heritable syndromes.

CLINICAL MANIFESTATIONS

Dental Plaque–Induced Gingival Diseases

Typical features of gingivitis may include gingival redness, swelling, marginal bleeding, and pseudopockets in the absence of bone loss, which might be reversible following appropriate therapy (Fig. 372-2). Mild and moderate forms of gingival inflammation are an almost universal finding in young people. Normal and abnormal fluctuation in hormone levels, including changes in gonadotrophic hormone levels during the onset of puberty, can enhance the gingival inflammatory response to dental plaque.

Chronic Periodontitis

Chronic periodontitis can be classified based on disease severity and extent of involvement. Disease severity can be categorized as mild (1–2 mm clinical attachment loss), moderate (3–4 mm clinical attachment loss), or severe (≥ 5 mm clinical attachment loss). In addition, the disease can be localized (less than 30% of the dentition affected) or generalized (greater than 30% of the dentition affected) and is characterized by a slow to moderate rate of progression that may include periods of rapid destruction.

The key features of chronic periodontitis are apical migration of junctional epithelium, loss of periodontal connective tissue attachment, alveolar bone loss, and pocket formation. Studies of the role microorganisms play in the pathogenesis of chronic periodontitis concluded that the amount of bacteria and bacterial products that accumulate locally in the periodontal tissue is of prime importance resulting in attachment loss (Fig. 372-3).

Aggressive Periodontitis

Aggressive periodontitis can be subdivided into localized or generalized forms based on the extent, severity, and type of teeth affected. Localized aggressive periodontitis is characterized by severe

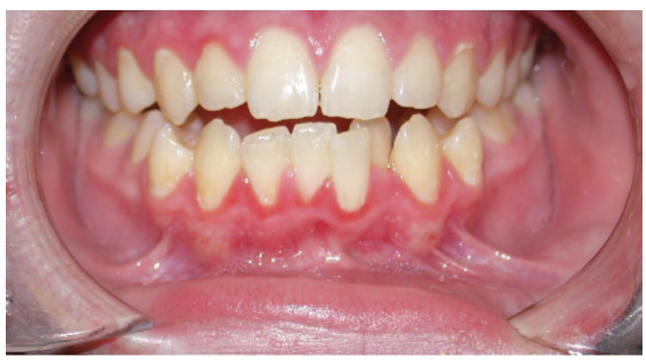

FIGURE 372-2 Clinical evidence of dental plaque–induced gingival disease, especially on lower anterior teeth.

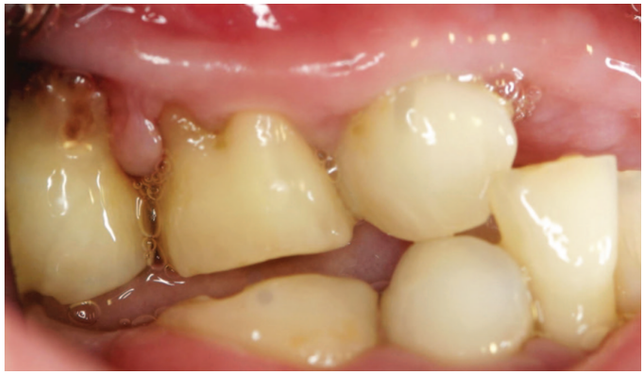

FIGURE 372-3 Clinical evidence of childhood chronic periodontitis, especially of upper posterior molars.

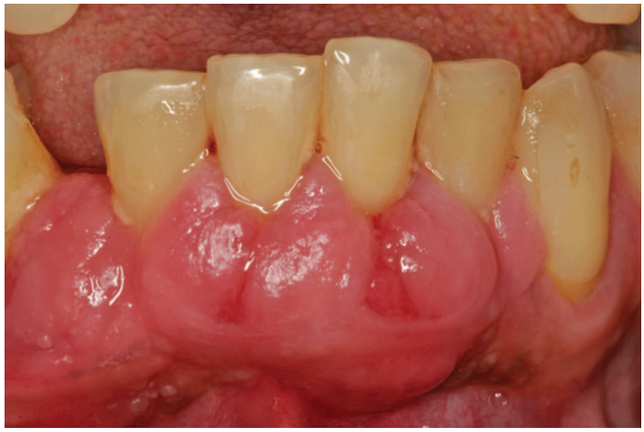

FIGURE 372-4 Clinical evidence of periodontal abscess on mandibular incisor areas.

periodontal tissue loss at the first molars and incisors and an age of onset between the circumpubertal age and late adolescence. Generalized aggressive periodontitis is also characterized by a rapid and severe periodontal tissue loss at the first molars and incisors, although the disease is not limited to these teeth and involves more than 3 other teeth. Both the localized and generalized forms of aggressive periodontitis show a rapid progression of disease. In the absence of early detection and treatment, the affected teeth usually show increased mobility, due to the severe loss of supporting tissue, and may eventually be lost.

Periodontitis as a Manifestation of Systemic Diseases

This category of periodontitis can occur in localized and generalized forms. In the localized form, affected sites exhibit rapid bone loss and minimal gingival inflammation. In the generalized form, there is a rapid bone loss around nearly all teeth and marked gingival inflammation. Both primary and permanent dentitions can be affected. Gingival enlargement or gingival overgrowth can be associated with drugs such as anticonvulsants (eg, phenytoin), calcium channel blockers (eg, nifedipine), and immunosuppressants (eg, cyclosporine), more or less related to the dosage and the amount of plaque buildup. As noted above in "Epidemiology," a variety of systemic diseases, particularly those that impact neutrophil function, place individuals at increased risk of periodontal disease.

Necrotizing Periodontal Disease

Necrotizing periodontal diseases are caused by abnormal overgrowth of normal oral bacteria as a consequence of poor oral hygiene. These periodontal diseases manifest as NUG and NUP. Necrotizing ulcerative gingivitis is a distinctive periodontal condition characterized by a rapid onset of intense gingival pain, spontaneous bleeding, necrotic ulceration of the gingival tissue, a fetid odor, and destruction and necrosis of 1 or more interdental papillae. Severe cases may show the presence of a pseudomembrane, swelling of lymph nodes, fever, and malaise. If untreated, the disease may lead to loss of periodontal tissue and may develop into NUP. Necrotizing ulcerative periodontitis may be a sequel of a single or recurrent episode of NUG, or it may occur as a result of necrotizing disease at a site previously affected by perichondritis. The differential diagnosis for NUG includes primary herpetic gingivostomatitis, caused by primary contact to herpes simplex virus type 1.

PERIODONTAL ABSCESS

Periodontal abscess is an acute suppurative infection of the deeper periodontal tissues that may lead to the destruction of periodontal ligaments and alveolar bone (Fig. 372-4). Patients may complain of localized pain and swelling with progression over hours or days, heat sensitivity, fever, and gingival bleeding. Clinical features may include a smooth, shiny swelling of the gingiva; pain, with the area of swelling tender to touch; a purulent exudate; or increase in pocket depth. In addition, the tooth may be mobile or sensitive to percussion. More severe infections may lead to trismus, dysphagia, respiratory difficulty, face or neck swelling, and regional lymph node involvement. Treatments include irrigation and mechanical debridement of the pocket, drainage and debridement of the lesion, and antibiotics.

LINEAR GINGIVAL ERYTHEMA

Linear gingival erythema is a manifestation of human immunodeficiency virus (HIV) infection. This condition is distinguished by a marginal band of 2 to 3 mm of intense erythema in the free gingiva, which may extend into the attached gingiva as a focal or diffuse erythema and may extend beyond the mucogingival line into the alveolar mucosa. Treatment includes improving oral hygiene, scaling and root planing, chlorhexidine rinses, and frequent dental cleaning.

DIAGNOSTIC EVALUATION

A definitive diagnosis or gingival or periodontal disease requires a detailed medical and dental examination. The dental assessment includes a clinical examination, periodontal charting, and dental radiographs. A referral to a pediatric dentist for further evaluation should be made if there is heavy plaque and calculus accumulation, edema, redness, bleeding, recession of the gingiva, or tooth mobility (Fig. 372-5). Microbiologic, genetic, biochemical, or other diagnostic tests may also be useful, on an individual basis, for assessing the periodontal status of selected patients or sites.

TREATMENT

In general, the therapeutic goals of periodontal treatment are to alter or eliminate the microbial etiology and contributing risk factors for periodontal disease, thereby arresting the progression and preserving

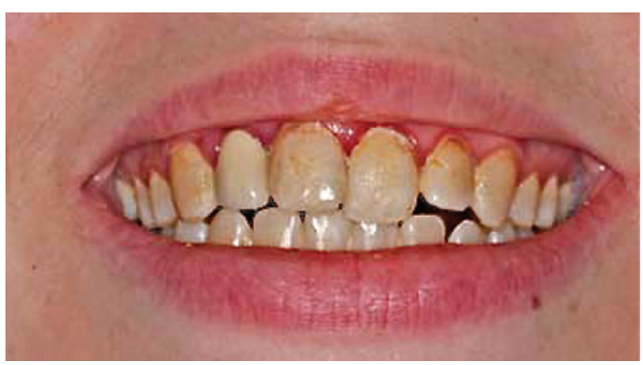

FIGURE 372-5 Dental referrals should be made if there is heavy plaque buildup and/or clinical signs of inflammation.

dentition. In addition, appropriate supportive periodontal maintenance that includes personal and professional care is important in preventing a recurrence of inflammation. Since contributing systemic risk factors may affect treatment and therapeutic outcomes, elimination, alteration, or control of these risk factors should be attempted, and consultation between the patient's pediatrician and the dentist is usually indicated to coordinate medical care with periodontal therapy. Specific treatment for periodontal disease is determined by the patient's age, overall health, medical history, and extent of the disease.

The clinical findings together with a diagnosis and prognosis should be used to develop a logical treatment plan in order to eliminate or alleviate the signs and symptoms of periodontal diseases and thereby arrest or slow further disease progression. A broad range of therapies exist in periodontics, and no single treatment approach can provide the only means of treating any or all periodontal diseases. Periodontal treatment should be completed in 3 phases: initial therapy, surgical corrective therapy, and maintenance therapy.

Initial therapy includes patient education to eliminate or control plaque accumulation and the debridement of tooth surfaces to remove supragingival and subgingival plaque and calculus deposits. Tooth brushing twice daily with fluoridated toothpaste is recommended. A power toothbrush may be efficacious if the patient is unable to brush effectively. Although certain mouthwashes containing various pharmaceutical agents (3% hydrogen peroxide mixed with equal parts water or 0.12% chlorhexidine) have some adjunctive antiplaque effect in adults, mouthwashes are not generally indicated in young children due to their inability to expectorate. Antibiotic treatments, such as doxycycline, metronidazole, or metronidazole/amoxicillin combination, may be utilized in the initial therapy. For example, amoxicillin and metronidazole plus scaling and root planing have been shown to be effective in the treatment of localized aggressive periodontitis.

Surgical corrective therapy might be indicated to provide access for plaque removal and to restore function and aesthetics for patients who not respond to initial therapy. Examples of surgical therapies are resective therapy (eg, flaps with or without osseous surgery or gingivectomy), regenerative therapy (eg, bone replacement grafts, guided tissue regeneration, or combined regenerative techniques), and gingival augmentation therapy.

Maintenance therapy is performed to prevent disease recurrence and progression and includes follow-up care. The interval between the maintenance therapy visits depends on the patient's response to treatment, plaque control, and the initial diagnosis. Recall intervals of no more than 4 months are appropriate for most patients who have been successfully treated.

INTERDISCIPLINARY CARE

Apart from managing the inflammatory and medical components of gingival and periodontal diseases in this population, there are times when treatment planning and care have to be coordinated between the pediatric dentist and other dental specialists.

MALOCCLUSION AND PERIODONTAL CONDITIONS

During orthodontic therapy, periodontal conditions and local anatomy must be taken into consideration prior to initiating tooth movement. In most cases, any gingivitis and periodontitis needs to be treated prior to initiating orthodontic treatment. Periodontal therapy prior to orthodontic therapy might include initial phase treatment and surgical treatment. In certain instances, localized treatments to correct mucogingival defects and other abnormalities need to be planned prior to orthodontic treatment. Such treatments can include augmenting keratinized tissue, performing root coverage procedures, and exposing impacted/unerupted teeth. Periodontal care may also need to be coordinated during orthodontic treatment.

It is not uncommon for patients to have gingivitis or hyperplastic tissue during orthodontic treatment. In these cases, frequent recall may be necessary or gingivectomies (Fig. 372-6) may need to be performed. On occasion, orthodontic treatment can also result in attachment loss, mucogingival defects, and blunted/shortened roots. The providers should monitor the patient's periodontal status frequently during orthodontic treatment and refer for periodontal care and maintenance as needed. A full periodontal examination (clinical and radiographic) should be completed at the completion of orthodontic therapy.

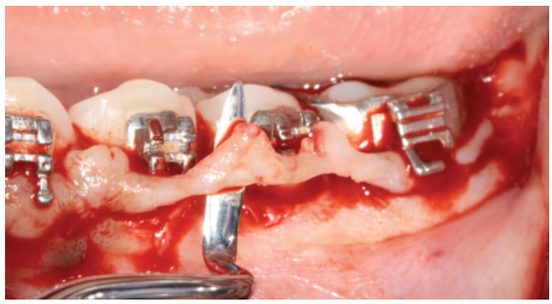

FIGURE 372-6 Gingivectomy of gingival enlargement associated with orthodontic appliances.

IMPLANT THERAPY AND RESTORATIVE PROCEDURES

Dental implants in general should not be placed until all skeletal growth is completed, as they can get submerged or displaced into the sinus if growth of the mandible/maxilla is still taking place. In most cases, if permanent teeth are missing/lost in younger patients, providers should try to conserve soft and hard tissues so that dental implants can be treatment-planned and placed in later years. A restorative dentist should also be consulted in these cases to plan for space maintenance until definitive restorative treatment can be delivered.

COMPLICATIONS

The results of periodontal treatment may be adversely affected by many external factors including systemic diseases; unknown or undeterminable etiologic factors that current therapy has not controlled; pulpal-periodontal problems; inability or failure of the patient to follow the suggested treatment or maintenance program; adverse health factors such as smoking, stress, or occlusal dysfunction; and uncorrectable anatomical, structural, or iatrogenic factors.

In patients for whom the periodontal condition does not resolve, treatment may include additional sessions of oral hygiene instruction and education, additional or alternative methods or devices for plaque removal, medical/dental consultations, additional tooth debridement, increased frequency of dental prophylaxis, microbial assessment, and continuous monitoring and evaluation to determine further periodontal treatment needs.

A satisfactory response to therapy should result in a significant reduction in clinical signs of gingival inflammation, stability of clinical attachment levels, and reduction of clinically detectable plaque to a level compatible with gingival health. Long-term outcomes in children with periodontal diseases depend on patient compliance with treatment regimens and the delivery of professional periodontal maintenance care at appropriate intervals.

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373 Oral Pathology

Andrew L. Sonis

INTRODUCTION

While dental caries and its consequences represent the most common oral pathologies encountered in children, both localized and systemic conditions of noncariogenic origin may manifest themselves in the mouth and surrounding tissues. This chapter will review some of the common and uncommon pathologies observed in or around the oral cavity of children.

SOFT TISSUE PATHOLOGY

ANGIOEDEMA

Angioedema is an allergic reaction often involving the oral soft tissues (see Chapter 190). Antigens precipitating this reaction include food and latex allergies, vaccines, insect bites, and medications. Typically, facial edema follows exposure to the antigen and may include swelling of intraoral soft tissues, potentially resulting in airway embarrassment. Initial management is with antihistamines. Administration of corticosteroids and/or epinephrine may be required in those with severe symptoms.

There are also several rare hereditary forms of angioedema (HAE), all inherited as an autosomal dominant trait, as well as an acquired form. Common to the hereditary forms is a deficiency or dysfunction with C1 esterase inhibitor (C1-INH). The classic manifestations of the disease typically include a history of recurring episodes of swelling without urticaria and may follow dental manipulation. Management of an acute attack includes intravenous administration of a C1-INH concentrate.

ANKYLOGLOSSIA

Ankyloglossia (Fig. 373-1) is a congenital anomaly of the tongue characterized by a short and sometimes anteriorly inserted lingual frenulum. Although attempts have been made to develop objective means of characterizing this condition, the diagnosis remains for the most part clinical.

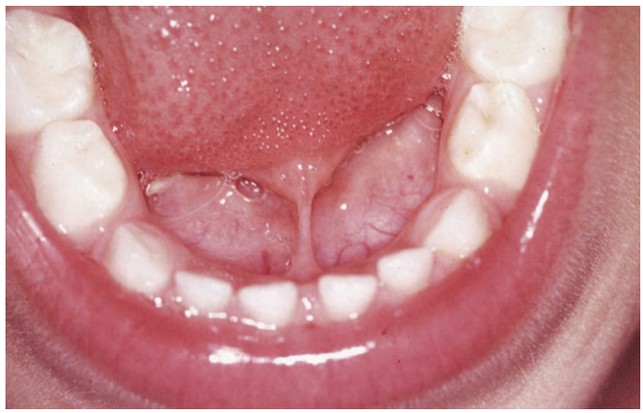

FIGURE 373-1 Ankyloglossia (tongue tie). A short lingual frenulum may interfere with feeding or speech and may necessitate surgical intervention.

Ankyloglossia reportedly occurs in approximately 2% to 5% of newborn infants with a male-to-female ratio of 2.6:1. While generally presenting as an isolated anomaly, it can be associated with other craniofacial abnormalities, ie, X-linked cleft palate syndrome, Kindler syndrome, van der Woude syndrome, and Opitz syndrome.

Tongue mobility may be restricted and consequently associated with functional limitations in breastfeeding; swallowing; articulation; orthodontic problems including malocclusion, open bite, separation of lower incisors; mechanical problems related to oral clearance; periodontal problems; or psychological stress to infant and mother. The tip of the tongue may appear notched or heart shaped. The frenum is typically observed to insert near the tip of the tongue. The clinical significance of ankyloglossia is a matter of controversy, particularly as it relates to breastfeeding difficulties such as sore nipples, poor infant weight gain, neonatal dehydration, and shortened breastfeeding duration.

Diagnosis of ankyloglossia typically involves observation of tongue function and tongue tip position, and visual inspection and/or palpation of the frenulum. More complex multiscale classification systems, such as the Hazelbaker Assessment Tool for Lingual Frenulum Function (HATLFF), allow for a more objective evaluation. The HATLFF was designed to assess frenulum form and function in infants younger than 3 months of age.

Frenotomy and frenuloplasty are the 2 main surgical procedures used in the treatment of ankyloglossia. Frenotomy, or clipping of the frenulum, is the procedure of choice in infants. It is a relatively simple procedure, performed without analgesia or anesthesia, with few reported complications. The use of a laser to perform the frenotomy, which is reported to decrease the likelihood of postoperative bleeding, is becoming more frequent.

Frenuloplasty, an operation that lengthens the frenulum via submucosal dissection and muscle release, is the preferred procedure for patients over 1 year of age. A more complex procedure than frenotomy, this surgical technique typically requires local or general anesthesia.

While the treatment of ankyloglossia rarely results in complications or late effects, lack of treatment may contribute to speech, dental, or social problems. Although rare, children with untreated ankyloglossia may experience articulation problems, periodontal and occlusion problems, and difficulties with kissing and playing woodwind instruments. A consultation with the appropriate specialist (eg, speech pathologist, dentist) is indicated.

Current research supporting a relationship between ankyloglossia and breastfeeding problems is weak. Consequently, the diagnosis of ankyloglossia, as well as the recommendation for frenotomy as a therapeutic intervention, should be made with the help of a lactation specialist.

BACTERIAL INFECTIONS

Dental caries, the most common bacterial infection in the mouth, may progress to abscess and cellulitis if left untreated (see Chapter 369). Non–tooth-related intraoral bacterial infections are relatively uncommon and are typically the result of salivary gland infections or compromise of the mucosal barrier resulting in secondary infections. Extraoral bacterial infections involving the face and perioral region (ie, impetigo [see Chapter 362], erysipelas, and infections resulting from animal bites to the face) tend to be far more common. Additionally, several systemic conditions that have bacterial etiologies may present with intraoral manifestations.

Bacterial infections involving β-hemolytic streptococci, which are responsible for scarlet fever and streptococcal pharyngitis (see Chapter 280), may present with unique intraoral lesions. The tonsils, soft palate, and pharynx become erythematous, and petechiae may be present on the soft palate. During the first 48 hours, the tongue's dorsal surface has a white coating referred to as *white strawberry tongue*. A few days later, the white coating desquamates to reveal an erythematous dorsal surface with hyperplastic fungiform papillae referred to as *red strawberry tongue*. Antibiotic therapy typically results in an excellent prognosis.

Congenital syphilis (see Chapter 283), produced by *Treponema pallidum*, may affect the developing dentition, resulting in an alteration of the normal anatomic appearance of the involved teeth. The incisal edges of the anterior permanent teeth present with a characteristic central hypoplastic notch (Hutchinson incisors) and multicuspid permanent molars (mulberry molars). The classic pathognomonic diagnostic features of congenital syphilis are described as the *Hutchinson triad*, which includes Hutchinson incisors, interstitial keratitis, and sensory neural deafness. Congenital syphilis can also result in craniofacial manifestations including frontal bossing, saddle nose, short maxilla, and protuberant mandible. Perforation of the palate secondary to gummatous destruction has been frequently reported in the literature and is considered pathognomonic for congenital syphilis. Patients are typically treated with penicillin.

Several other systemic conditions that have bacterial etiologies can present with oral lesions, including diphtheria, tuberculosis, cat scratch disease, actinomycosis, and gonorrhea.

BITE INJURIES

Accidental self-inflicted bite injuries are quite common in children, especially after having received intraoral local anesthesia. Not infrequently, children who have received a mandibular nerve block for dental treatment will unknowingly chew their lip, buccal mucosa, or inner lip, resulting in a significant area of epithelial necrosis accompanied by edema. Sites most frequently involved are the lateral border and tip of the tongue, buccal mucosa, and lower lip. The injury usually presents as a macerated, irregular lesion with painless white necrotic areas of epithelium surrounding the ulcerated surface. These children may complain of discomfort, particularly when exposed to foods high in salt or citric acid. Treatment, if necessary, is supportive and palliative since these areas of trauma rarely, if ever, become secondarily infected.

The clinician must always be aware that bite marks may not be accidental, but rather may be a manifestation of child abuse. Child abuse always needs to be ruled out and the clinician must report suspected cases to the proper agencies, allowing for appropriate investigation and possibly forensic analysis.

CHEILITIS

Cheilitis (Fig. 373-2), a common inflammatory disorder in children, typically arises from the chronic habit of licking the lips and perioral skin. Less frequently, cheilitis or angular cheilosis (perlèche) may occur from mechanical irritation, malocclusion, atopic dermatitis, fungal infection (typically candidiasis), bacterial infection, or nutritional deficiencies. Allergic cheilitis occurs far more frequently in females, typically from a sensitivity to lipstick or lip balm. Toothpaste flavoring agents such as spearmint oil are also a commonly identified allergen. The clinical features of cheilitis are erythema, mild edema, and formation of a thin layer of scaling of the lips and perioral areas. When due to chronic lip licking, treatment should be directed at breaking the habit. Empirically, this may be accomplished with lip balms with undesirable tastes. In severe cases, topical steroids, with or without fungicidal and bactericidal activity, may be beneficial. Obviously, in cases where a nutritional deficiency is thought to be the underlying etiology, nutritional supplements are indicated.

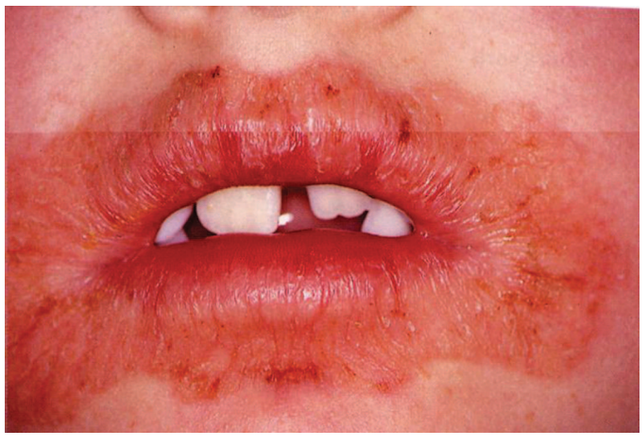

FIGURE 373-2 Cheilitis. The habit of chronic lip licking can result in significant inflammation of the perioral tissues. Severe cases typically respond well to topical steroids.

CONGENITAL EPULIS

Congenital epulis (Fig. 373-3), or gingival granular cell tumor of the newborn, is a relatively rare lesion typically arising from the maxillary anterior alveolus. Far more common in females (8:1), it presents as a smooth, firm, pedunculated soft-tissue lesion attached to the alveolus by a broad base.

These benign neoplasms typically arise from the anterior maxilla and can range in size from several millimeters to several centimeters, with larger lesions frequently being detected in utero. Excision of smaller lesions not interfering with function may be deferred several months to minimize anesthetic risk, whereas larger lesions require immediate surgical removal due to risks of obstruction.

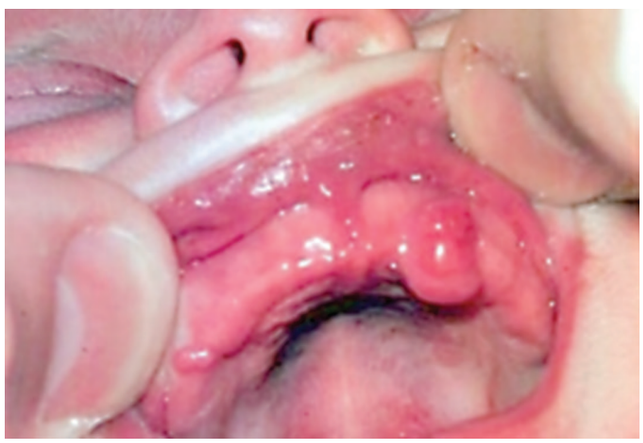

FIGURE 373-3 Congenital epulis. These benign neoplasms typically arise from the anterior maxilla and can range in size from several millimeters to several centimeters. Excision of smaller lesions not interfering with function may be deferred several months to minimize anesthetic risk.

RIGA-FEDE SYNDROME

Although frequently referred to as Riga-Fede syndrome or disease, this condition is in fact neither. It presents as an ulceration at the tip of the tongue secondary to irritation from newly erupted primary mandibular incisors. It is often painful and interferes with nursing or eating. Management may include smoothing the incisal edges of the offending incisors with resolution of ulceration taking 7 to 10 days.

ERUPTION HEMATOMA

An eruption hematoma (Fig. 373-4) is a soft tissue variant of the dentigerous cyst that develops around the crown of an erupting primary or permanent tooth. It is quite common and can occur in association with any erupting tooth. The lesion typically resolves on its own with the eruption of the underlying tooth. Rarely is it necessary to excise the roof of the cyst to allow for eruption of the involved root.

FIBROMA

The irritation fibroma is considered the most common childhood reactive lesion of the oral cavity. In the vast majority of cases, the lesion arises as a reaction to a local irritant. While the lesion may involve any oral anatomic site, the buccal mucosa and interdental gingiva are the most common sites in children. The lesion usually presents as an asymptomatic pedunculated or sessile growth secondary to irritation. Treatment is surgical excision with the elimination of any associated source of local irritation. Failure to remove the source of irritation will likely result in recurrence.

CANDIDIASIS

Candidiasis, or candidosis, is an infection involving the yeastlike fungal organism *Candida*, the most common being *C albicans*. It is a common microbe of the oral cavity, found in 20% to 70% of healthy individuals. It is the most common oral fungal infection in humans and may manifest in a variety of clinical presentations, which may make appropriate diagnosis difficult. Immunocompromised children and adults who are receiving a prolonged course of antibiotic therapy are particularly susceptible to such infections.

Erythematous or *chronic atrophic candidiasis* may also be caused by immunosuppression, xerostomia, or chronic exposure to broad-spectrum antibiotic therapy. Patients often complain of a burning sensation on their tongue associated with a loss of filiform papillae and a denuded appearance of the tongue's dorsal surface. Unlike the acute form characterized by sloughing white lesions, the erythematous form results in patchy lesions. Some forms of chronic candidiasis, including central papillary atrophy, may be asymptomatic. Some authors have suggested that median rhomboid glossitis may also represent a chronic candidal infection. In addition, angular cheilitis is a particular form of erythematous candidiasis that affects the commissures and is characterized by fissuring, erythema, and scalelike lesions. The treatment regimen is similar to that of acute candidiasis. These lesions may be caused by *C albicans* alone or by *Candida* in association with *Staphylococcus* bacterial infection.

Pseudomembranous candidiasis (acute), the most common form of oral candidiasis, is usually seen on the buccal mucosa, dorsum of the tongue, and soft palate. It is commonly known as *thrush* and appears as white cottage cheese–like lesions that can be removed by scraping. It is the most common manifestation of human immunodeficiency virus (HIV) infection in children. Topical antifungal medications are often the drug of choice for localized candidiasis. While compliance with the "swish and swallow" routine with these medications is readily accomplished by older patients, in the infected infant, appropriate delivery of the topical medication is often problematic. A foam-headed device (Toothette) is a useful aid in applying the medication to the infected oral mucosa. When this condition is seen, it is imperative to rule out a depressed immune system, which may be contributing to the candidiasis. Appropriate pharmacotherapy includes antifungal therapy with nystatin oral suspension, clotrimazole troches, or systemic antifungal therapy with agents such as fluconazole. (In 2013, the US Food and Drug Administration limited the use of the oral tablet formulation of ketoconazole, warning that it can cause severe liver injuries and adrenal gland problems and advising that it can lead to harmful drug interactions with other medications.)

GEOGRAPHIC TONGUE (ERYTHEMA MIGRANS)

Geographic tongue (Fig. 373-5) is a harmless common disorder of unknown etiology that primarily affects the dorsal surface of the tongue, although it may occasionally involve other mucosal surfaces, including the buccal and sublingual mucosa. Several studies have suggested an increased prevalence among patients with fissured tongue, allergies, seborrheic dermatitis, and psoriasis. Clinically, geographic tongue is characterized by an erythematous, well-circumscribed area of atrophy of the filiform papillae on the dorsum of the tongue, ranging in size from several millimeters to several centimeters. The erythematous area is most frequently surrounded by a slightly raised whitish margin. The involved area typically changes or "migrates" over several weeks or months, thus giving rise to the disorder's name. Onset tends to be fairly rapid, with lesions typically persisting for

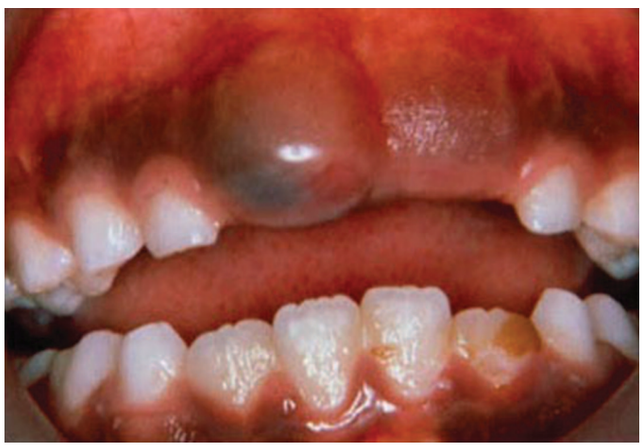

FIGURE 373-4 Eruption hematoma. Often associated with an erupting primary tooth, these benign lesions are the result of hemorrhage into the cystic cavity surrounding the crown of the involved tooth.

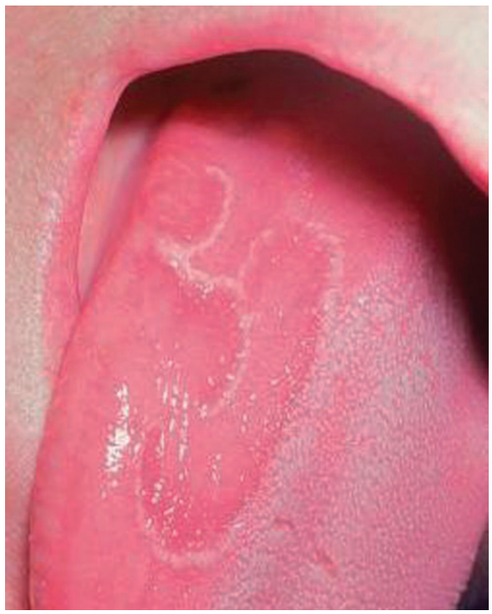

FIGURE 373-5 Geographic tongue (erythema migrans). Commonly appearing on the dorsal surface of the tongue, the lesion is characterized by localized atrophy of the filiform papillae. Less commonly, other oral mucosal surfaces may be involved.

several weeks, although occasionally they may last for several years. Patients are generally asymptomatic but may rarely complain of a "burning" sensation. No treatment is necessary; however, patients experiencing burning may benefit from palliative treatment with topical anesthetics.

HAIRY TONGUE

An accumulation of keratin and elongation and hypertrophy of the filiform papillae of the tongue results in the clinical appearance of hairy tongue. While the etiology is unknown, it is correlated with several predisposing factors, including poor oral hygiene, use of mouth rinses containing sodium perborate, long-term antibiotics or steroid therapy, head and neck irradiation, and *C albicans* infection. Clinically, the elongated filiform papillae can range in color from white to brown to black. Although patients are typically asymptomatic, they may occasionally complain of a bad taste or halitosis. Treatment involves removing the precipitating factors and improving oral hygiene, including brushing the tongue. A unique form of hairy tongue is associated with HIV and is referred to as *hairy leukoplakia*. Although this condition is usually observed in adults with HIV, there have been several reports of its occurrence in HIV-infected children. The lateral border of the tongue is most commonly affected bilaterally, and the condition is thought to be associated with Epstein-Barr virus (EBV) infection.

MELANOTIC NEUROECTODERMAL TUMOR OF INFANCY

Melanotic neuroectodermal tumor of infancy, believed to arise from the neural crest, is a rare condition that could potentially be confused with congenital epulis. Similarities to congenital epulis include its appearance in infancy, generally between 1 and 3 months, and its involvement of the anterior maxilla. However, unlike congenital epulis, the melanotic neuroectodermal tumor of infancy can be a locally aggressive lesion capable of causing significant tissue damage. The most common manifestation of the tumor is a soft tissue mass with an intact mucosal surface. The lesion may contain blue or black areas of pigmentation and may grow large enough to obliterate the labial buccal vestibule with resultant displacement and elevation of the lip. Pain is rarely associated with this tumor, whose growth rate is variable. Surgical excision or enucleation is the recommended treatment. Recurrence is relatively common, with rates as high as 20%.

PYOGENIC GRANULOMA

The pyogenic granuloma is a relatively common benign vascular lesion of the skin and mucosa that arises from an exaggerated tissue response to local irritation. Typically found on the gingiva in the oral cavity, it also can be found on the tongue, lips, and buccal mucosa. The granuloma appears as a painless, nodular, erythematous mass that is typically either pedunculated or sessile. The color ranges from bluish to purple, and it may resemble a hemangioma. The surface may be smooth or lobulated and is frequently ulcerated and covered with a whitish-yellow pseudomembrane. Probing or palpating this soft lesion will frequently result in bleeding. It grows rapidly and ranges in size from 5 to 10 mm. Since these lesions arise from an excessive tissue response to a local irritant, treatment involves both surgical excision and removal of the source of irritation. When on the gingiva, the irritant is typically calcified plaque (calculus), which must be removed from the tooth surface to prevent recurrence.

RECURRENT APHTHOUS STOMATITIS

Recurrent aphthae, or canker sores, are the most common forms of oral ulcerations in both children and adults. Epidemiological studies indicate that the prevalence of recurrent aphthous stomatitis in the general population is between 2% and 50%, with onset typically occurring in childhood. These episodic ulcerations generally continue past childhood and adolescence and occur throughout the patient's life span, with no age-, sex-, race-, or geographic-related predilection.

There are 3 forms of the disorder: minor, major (Sutton disease), and herpetiform. Minor aphthae are typically round, small (less than 10 mm in diameter), well-circumscribed ulcerations surrounded by an erythematous halo. These usually occur on movable or unbound (nonkeratinized) mucosa. Several may be present simultaneously. Healing is spontaneous within 7 to 10 days, usually without scarring.

Major aphthae are similar but are larger in size (> 1 cm in diameter), are generally much deeper, and may be debilitating. Healing may take 10 to 30 days and may result in scarring.

Herpetiform ulcers are very small (1 mm in diameter) and often occur in clusters. There is a tendency for adjacent ulcers to coalesce and form a larger affected area. Healing takes place usually within 7 to 10 days. The name stems from the clinical manifestation resembling a recurrent intraoral herpetic infection.

All 3 clinical types are associated with varying degrees of pain and discomfort, the extent of which depends on the location, size, and depth of the ulcers. The etiology of recurrent aphthous stomatitis is unknown, although an altered local immune response is the suspect. Precipitating factors include stress, endocrine alterations, allergies, trauma, and food hypersensitivity. Patients with frequent recurrences should be screened for potential underlying diseases such as anemia, nutritional deficiencies (eg, iron, folate, vitamin B_{12}), diabetes mellitus, immunosuppression, and inflammatory bowel disease (**Table 373-1**). Treatment is primarily aimed at pain relief and accelerating the healing process. All therapies are palliative, and none have resulted in permanent remission. Although the ulcer's etiology is not clearly understood, some treatment regimens focus on either preventing or at least reducing the recurrence rate. A variety of topical and systemic therapies have been tried, but so far, no studies have shown clear evidence of effectiveness, either as preventive or pain-relieving agents. Empirically, effective treatment includes topical anesthetics, corticosteroids (topical and systemic), antimicrobial mouth rinses, topical anti-inflammatory agents, and topical barriers. Severe, persistent, recurrent aphthous ulcers may be treated with medications such as systemic corticosteroids, pentoxifylline, colchicine, and thalidomide, but these should not be routinely used, as they have potential adverse side effects. Since several systemic disorders are associated with chronic aphthous ulcer formation, potential underlying conditions (eg, periodic fever, aphthous stomatitis, pharyngitis, and cervical adenitis [PFAPA]; inflammatory bowel disease; Behçet syndrome; Reiter syndrome; and cyclic neutropenia) must be ruled out (see **Table 373-2**). Recurrent aphthous stomatitis represents a highly common mucosal disorder, the etiology remains poorly understood, and the efficacy of therapies remains undefined.

VIRAL INFECTIONS

A variety of viral infections manifest with oral lesions (Table 373-2).

CONDYLOMA ACUMINATUM

Condyloma acuminatum is a benign papillary lesion most commonly associated with human papillomavirus (HPV) 16 and 18. It is usually sexually transmitted, with oral condyloma acuminatum resulting from genital-oral sexual transmission or hand-to-mouth autoinoculation. The presence of condylomata in children may be secondary to child abuse. The most common location for oral condyloma acuminatum in children is the palate, which differs from adults, who usually have lesions on the lower lip and tongue. The course of treatment and recurrence rate are similar to that for other types of HPV.

EPSTEIN-BARR VIRUS

Epstein-Barr virus causes several diseases that may have oral or maxillofacial manifestations, such as infectious mononucleosis, hairy leukoplakia, Burkitt lymphoma, and nasopharyngeal carcinoma (see Chapter 306). Infectious mononucleosis commonly has oral manifestations including pharyngitis and tonsillitis that extend into the soft palate in children and young adults. The involved mucosa is erythematous with erosions and small characteristic petechiae. The area is often coated with a grayish exudate and is associated with tender

TABLE 373-1 GLOSSITIS: CAUSES, ETIOLOGY, MANAGEMENT, AND TREATMENT

Causes of Glossitis	Etiology	Clinical Appearance	Management
Allergic	Tartar-control toothpastes, cinnamon flavoring in chewing gums/candy, nickel-containing dental/orthodontic appliances	Areas of erythema Other intraoral sites may be involved including gingiva, buccal mucosa	Remove/eliminate allergen Mild: palliation Moderate: topical steroids Severe: systemic steroids
Bacterial/fungal/other			
• Scarlet fever	β-Hemolytic streptococci	"Strawberry" tongue caused by prominent papillae and erythema	Antibiotic therapy
• Toxic shock syndrome	*Staphylococcus aureus*		Antibiotic therapy
• Kawasaki disease (mucocutaneous lymph node syndrome)	Unknown		Intravenous immune globulin and acetylsalicylic acid
Acute atrophic candidiasis	Overgrowth of *Candida albicans* secondary to broad-spectrum antibiotics, inhaled steroids, or immunocompromise	Atrophy of papillae on dorsum of the tongue; tongue is smooth, erythematous, and often painful	Antifungal agents
Graft-versus-host disease	Allogeneic hematopoietic stem cell transplantation	White hyperkeratotic reticulations and plaques, erythematous changes, and ulcerations	Topical steroids, antifungal agents, sialogogues, and salivary stimulation
Viral			
• Herpetic gingivostomatitis	Herpes simplex virus type 1	Vesicular lesions on the tongue may occur; typically break down and coalesce	Supportive and palliative
• Oral hairy leukoplakia	Epstein-Barr virus; associated with immunocompromise, human immunodeficiency virus infection	White filiform lesions arising from lateral border of tongue	Antiviral agents
Mechanical	Trauma-induced inflammation secondary to orthodontic appliances, fractured tooth/teeth, etc	Ulceration with irregular border and/or white plaque (hyperkeratosis) arising from chronic irritation, resistant to wiping off	Remove/correct source of irritation
Chemical	Sensitivity to sodium lauryl sulfate (a common detergent and surfactant present in toothpaste); alcohol-containing mouth rinses	Nonspecific ulceration and/or desquamation of mucous membrane	Discontinue use of these products
	Oral tobacco products	Areas of leukoplakia	Discontinue use of these products and monitor resolution; may take several weeks to resolve; if no change, consider biopsy
Thermal	Microwaved foods Electrical cords	Nonspecific ulceration, often with white necrotic slough	Supportive and palliative; typically heal in 7–10 days
Nutritional • Iron deficiency • Folic acid deficiency • Vitamin B_{12} • Riboflavin deficiency • Niacin deficiency	Malnutrition, chronic disease	Surface of tongue is smooth and glossy with pink or erythematous coloration	Treat nutritional deficiency

cervical lymphadenopathy. Symptoms will usually resolve in a month but may persist for longer in some cases.

Other conditions associated with EBV that may impact the oral cavity, but less commonly in the United States, are nasopharyngeal carcinoma (Asia) and Burkitt lymphoma (Africa). Lastly, hairy leukoplakia is an EBV-induced epithelial hyperplasia that causes hypertrophic corrugated white lesions on the lateral border of the tongue. Hairy leukoplakia can occur in the absence of HIV infection in organ transplant patients who are immunosuppressed. Unlike acute candidiasis, this lesion cannot be wiped off and is entirely asymptomatic.

HAND-FOOT-AND-MOUTH DISEASE

Hand-foot-and-mouth disease (HFMD), a common enteroviral infection caused by coxsackie virus type A-16, is another common cause of intraoral vesicular lesions (see Chapter 301). It has also been linked to coxsackie A-4 to A-7, A-9, A-10, B-1 to B-3, and 5 serotypes. It has a distinctly different presentation from the lesions seen in herpangina, which is also caused by the coxsackie virus. These are both common viral illnesses of infants and children. Infection usually occurs by the fecal-oral route, leading to viremia and invasion of the mucosa and skin. The infection begins with a mild fever, poor appetite, malaise, and frequently a sore throat. One or 2 days after the fever begins, painful sores develop in the mouth. They begin as small red spots that blister and then often become ulcers between 1 and 2 cm in diameter. The associated lesions of the hands and feet are usually painless and fade in 1 to 2 weeks without any apparent scarring. It is important to note that outbreaks occur more commonly in summer and autumn and can be transmitted from child to child or from child to adult.

No specific treatment is available for this or other enterovirus infections. Symptomatic treatment is given to provide relief from fever, aches, or pain from the mouth ulcers. The lesions regress and heal within 7 to 10 days. If an outbreak occurs in the childcare setting, it is imperative that meticulous personal hygiene measures be enforced. It may also be beneficial to thoroughly wash and disinfect contaminated items and surfaces using a diluted solution of chlorine-containing bleach.

HERPANGINA

Herpangina has a similar presentation to that of HFMD, but the vesicular lesions are solely seen in the region of the soft palate (see also Chapter 304). Herpangina is usually caused by the coxsackie

TABLE 373-2 DIFFERENTIAL DIAGNOSIS OF INTRAORAL ULCERS

Diagnosis	Potential Differentiating Features
Viral	
Herpesvirus	Vesicular lesions progressing to ulceration; Tzanck prep positive for inclusion-bearing giant cells
Herpetic gingivostomatitis	Typical viral prodrome of fever, malaise, pain; marked involvement of gingiva, lips; vesicular lesions typically rupture, leaving ulcerated surface
Secondary	History of recurrent intraoral ulcers often precipitated by oral trauma, dental manipulation
Recurrent	History of recurrent extraoral vesicles typically involving the lips; often precipitated by exposure to sunlight
Cytomegalovirus	Immunocompromised patient; biopsy positive for multinucleated giant cells
Varicella zoster	Characteristic skin lesions accompany intraoral ulcers
Coxsackievirus	
• Hand-foot-and-mouth disease	Typical viral prodrome, more common in summer months; often occurring in epidemics; lesions on hands, feet, and intraorally; oral lesions typically involve buccal mucosa, gingiva, tongue, pharynx
• Herpangina	Typical viral prodrome; ulcerations most commonly found on soft palate, tonsillar pillars and tonsils, uvula, posterior pharynx
Human immunodeficiency virus (HIV)	Intraoral candidiasis: most common oral infection in children; aphthous stomatitis more common in infected adults
Unknown	
Aphthous	
• Minor	History of recurrent intraoral ulcers with onset in childhood; small, well-circumscribed ulcers with erythematous halo involving any unbound mucosa; typically heals in 7–10 days
• Major	Large intraoral ulcers (> 1 cm) causing significant discomfort; often lasts 10–30 days and may result in scarring
• Herpetiform	Multiple small, pinpoint lesions that may coalesce; typically heals in 7–10 days
• PFAPA (periodic fever, aphthous stomatitis, pharyngitis, cervical adenitis)	History of aphthous ulcers, recurrent fever, pharyngitis, lymphadenopathy
Autoimmune	
Behçet syndrome	Genital ulceration, uveitis, retinitis; intraoral ulcers can appear identical to aphthous ulcers
Reiter syndrome	Urethritis, conjunctivitis, arthritis; more common in men
Inflammatory bowel disease	Bloody diarrhea, other gastrointestinal ulcerations, labial or facial swelling
Hematologic	
Cyclic neutropenia	Recurrent fevers, neutropenia
Allergic	
Cinnamaldehyde-containing products	History of use of cinnamon-flavored toothpaste, chewing gum
Sodium lauryl sulfate–containing toothpaste	History of sodium lauryl sulfate–containing toothpaste

virus but may also be caused by an echovirus. It is relatively common in children between the ages of 1 and 4 years and is seen more often in summer and autumn. Herpangina is associated with headache, quick-onset fever, dysphagia, sore throat, pain, drooling, and a sudden decrease in appetite. The incubation period is 2 to 9 days, and fever usually lasts from 1 to 4 days. As in any viral infection, antibiotics are ineffective and treatment is supportive and palliative, consisting of hydration and acetaminophen for any associated fever. The lesions usually resolve in 7 to 10 days.

HUMAN IMMUNODEFICIENCY VIRUS

Human immunodeficiency virus infection predisposes infected children to a myriad of characteristic oral lesions and manifestations. Candidiasis has been documented in several studies as the most frequently occurring oral manifestation in HIV-infected children, with a prevalence as high as 70%. There is a significant relationship between oral mycotic infections and declining CD4 T-cell and neutrophil counts in HIV-infected children. Other lesions in these immunosuppressed children include parotid enlargement, angular cheilitis, herpetic stomatitis, oral hairy leukoplakia, petechiae, aphthous stomatitis, linear gingival erythema, and cervical lymphadenopathy. It is important to emphasize that parotitis, which may occur in up to 30% of children with HIV, does not require specific treatment and has been associated with a positive prognosis and long-term survival. Therefore, it has been suggested that both candidal infection and parotitis be included in prognostic indices and decisions regarding therapy for these patients. Undoubtedly, pediatric dentists and pediatricians can both play a role in the early detection of HIV-related oral lesions.

PRIMARY HERPETIC GINGIVOSTOMATITIS

Primary oral herpetic gingivostomatitis is caused by herpes simplex virus type 1 (HSV-1) (see also Chapter 304). Most individuals experience a subclinical infection with HSV-1, while a small percentage of the population develops the full manifestations of the infection. It is characterized by vesicular lesions on the gingival tissue, lips, tongue, buccal mucosa, and soft palate. These lesions will often appear in clusters and may frequently coalesce, creating large areas of ulcerations that resemble aphthous ulcerations. The tissue is fiery red in appearance and is painful. Herpes simplex is easily transmitted when the shedding herpesvirus comes in direct contact with mucous membranes or broken skin. Most primary infections are contracted from others who are shedding HSV-1 but who are free of obvious lesions. Childcare facilities and/or primary school classrooms are ideal grounds for transmission. Primary herpes simplex infection peaks between the ages of 2 and 3 but may present in children between 6 months and 5 years of age or older.

Symptoms manifest themselves after a brief prodromal period. The child may become febrile, have associated lymphadenopathy, and may not be able to eat properly. There is an associated risk of dehydration, so care must be taken to assure adequate hydration. Intravenous hydration therapy might be necessary in severe cases. The viral infection typically lasts between 10 to 14 days, with the most severe ulcerations appearing between days 3 and 7. Autoinoculation of the hand and/or fingers can result in herpetic whitlow.

Prior to the development of acyclovir, treatment was limited to palliative care consisting of administration of fluids to prevent dehydration, topical and systemic analgesics to relieve pain, and antipyretics to combat elevated temperature. The use of oral acyclovir is thought to be most effective if used during the prodromal period. The suggested oral dosage for HSV-1 infections is 15 mg/kg, 5 times daily, for a period of 7 days. Palliation may be provided with topical agents, including kaopectate and diphenhydramine elixir, or dyclonine. Over-the-counter topical anesthetics in gel form contain 20% benzocaine and may be helpful for those with a minimal number of lesions. Parents may have concerns about their child having a "herpes infection," since herpes is so often linked to sexual transmission. It should be explained that HSV-1 (rather than HSV-2) is considered the infecting organism for their child's condition.

RECURRENT HERPES LABIALIS

After the initial infection ameliorates, herpes simplex virus persists in the dorsal root ganglion and causes recurrent disease in approximately 15% of the population. The recurrent herpes labialis or "cold

sore" lesions occur on the lips and perioral tissues and are most often seen unilaterally. The infection is thought to be reactivated by exposure to stress, sun, illness, fever, or a depressed immune system. Since sunlight seems to be the most common precipitating agent, daily application of a lip balm containing sunblock may be a beneficial preventative in the susceptible individual. The viral infection has a course of 7 to 10 days, progressing from a vesicular stage to crusting. Therapy is generally palliative, as topical and systemic antiviral agents, while shown to be effective in the immunocompromised patient, have shown to be of limited efficacy in the immunocompetent individual. It must be stressed that patients keep their hands and fingers clean and away from the nidus of infection, as autoinoculation of the contralateral side of the mouth, eye, or other mucous membrane can occur readily.

RUBEOLA

The oral manifestations of the measles virus, which has a prodromal period of 7 to 14 days, include pathognomic lesions known as *Koplik spots*. These intraoral lesions appear on day 2 or 3, prior to the rash on the skin, and regress at or around day 6. They are blue-white spots the size of salt grains, surrounded by a reddish halo. They are located on the buccal mucosa opposite the premolar teeth.

SQUAMOUS PAPILLOMA

The human papillomavirus is a benign group of DNA-based viruses that can infect the skin and mucous membranes. Human papillomavirus infection can result in numerous exophytic white or pink finger-like projections that resemble cauliflower or warts. These lesions are pedunculated and represent a proliferation of stratified squamous epithelium. The average age of occurrence for a squamous papilloma is in the 4th decade of life, but nearly 20% of cases occur before 20 years of age. Over 50% of oral lesions are linked to HPV-6 and HPV-11 subtypes. The oral lesions are most commonly seen on the lips and on the palate, uvula, and dorsum of the tongue. They are transmitted through direct contact, and the only definitive treatment is excision, cryotherapy, or electrosurgery. Recurrence is rare.

The relationship of HPV (specifically types 16 and 18) and oral cancer is well established. Although the overall incidence of cancer has recently declined, the incidence of HPV-positive oropharyngeal cancers has increased in sexually active young nonsmokers and nondrinkers. Recent data suggest that the HPV vaccination may reduce the risk of HPV-related oropharyngeal cancer.

NEONATAL CYSTS

Neonatal cysts (Fig. 373-6) are benign developmental lesions. Previously, they were differentiated based upon locations. Epstein pearls are remnants of epithelial tissue trapped during the palatal fusion that occurs during the 8th to 10th gestational week. Bohn nodules are remnants of mucous gland tissue found on the buccal or lingual aspects of the dental ridges. Dental lamina cysts are found along the crest of the ridges. These terms, however, are frequently confused and used synonymously. Currently, the terms *palatal cysts* (designating entrapped epithelial-derived cysts that persist at the site of fusion of the palatal shelves) and *alveolar cysts* (remnants of degenerating dental lamina on the buccal, lingual, or crest portion of the alveolar ridge) are used in descriptions to avoid confusion. Alveolar cysts have also been referred to as *gingival cysts* or *inclusion cysts* in some texts. The clinical description of palatal and alveolar cysts varies in color from white to gray to yellow nodules, in size from a pinhead to 3 mm, and in numbers from 1 to 6. The frequency of inclusion cysts is high in newborns, but they are rarely seen after 3 months of age. The prevalence in term infants varies from 65% to 85% for palatal cysts and, depending on location, 9% for mandibular alveolar cysts to 36% for maxillary alveolar cysts. Prevalence is lower in premature infants. White infants are more likely to have palatal and alveolar cysts than African-American infants. No gender difference has been reported.

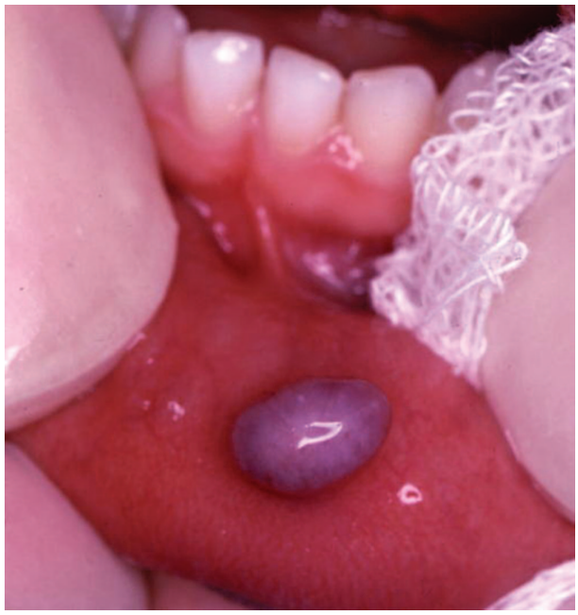

FIGURE 373-6 Mucocele. Trauma to the lower lip may result in occlusion of a minor salivary gland duct with extravasation of saliva into the surround soft tissues. Mucoceles are typically treated with surgical excision.

SALIVARY GLAND CONDITIONS

The major salivary glands play an important role in both oral and general health. Saliva contributes to protection against wound infections and caries, as well as providing lubrication for ease of eating, swallowing, and speech. Consequently, any conditions that compromise salivary flow and/or composition may negatively impact oral health. For example, medicines used for oral asthma therapy, such as β_2 agonists and cortisone, may reduce salivation. Similarly, protein-deficient diets or chemotherapy and/or radiotherapy (particularly if major salivary glands are in the field of radiation) for cancer during childhood may lead to a persistent functional impairment of the salivary glands, which may have lasting effects on the immune defenses and be associated with significantly greater pathological conditions of the teeth and associated soft tissues.

Apart from conditions that affect the saliva composition (eg, sticky saliva in Prader-Willi syndrome) or cause an inability to swallow and lead to drooling, hypoplasia or aplasia of the salivary glands is a rare occurrence that can affect just 1, several, or even all of the salivary glands. Aplasia of the salivary glands may occur in isolation or be associated with other malformations of the first branchial arch. These include hemifacial microsomy, mandibulofacial dysostosis (Treacher Collins syndrome), and other congenital malformations of the face.

MUCOCELE

Mucoceles (Fig. 373-7) are the most common salivary gland problem of childhood. They typically arise in the mucosa of the lower lip as a result of trauma to a minor salivary gland, although they may occur less frequently on the buccal mucosa, soft palate, or upper lip. The extravasation of saliva into the surrounding soft tissue causes the formation of a pseudocyst. This lesion clinically appears as a painless, raised, dome-shaped lesion and may be clear, pink to red, or bluish in color, or it may be pale and fibrotic. Size is typically less than a centimeter in diameter. A common sign is a periodic reduction in swelling due to rupture and partial release of fluid, followed by recurrence due to new accumulations of fluid. While mucoceles will occasionally resolve spontaneously, most require surgical excision.

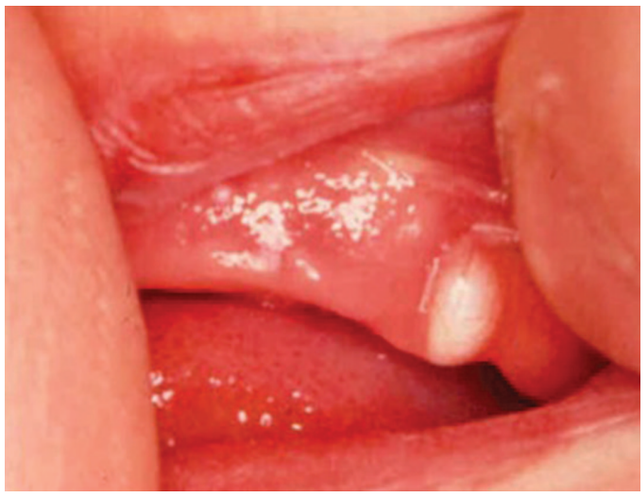

FIGURE 373-7 Neonatal cysts. These painless cysts are extremely common findings in infants. There are 3 forms: Bohn nodules (seen here), which typically present as multiple lesions on the buccal and lingual aspects of the alveolus; dental lamina cysts, which appear on the crest of the alveolus; and Epstein pearls, which present on the midline of the palate. None require any intervention and resolve spontaneously.

RANULA

A ranula is a mucous retention cyst that occurs on the floor of the mouth in association with the sublingual gland or, less frequently, the submandibular gland. Athough they may be congenital, most arise from trauma. The ranula presents as a smooth, dome-shaped, painless, fluctuant swelling. The color is typically bluish, but deep lesions may appear as normal mucosa. Smaller ranulas are typically excised, while larger lesions may require marsupialization. A plunging ranula is the extension of the lesion into the neck. This occurs when the submandibular gland or mucous retention cyst extends down through the mylohyoid. Clinically, these patients present with swelling of the neck. Treatment of the plunging ranula involves complete surgical removal of the sublingual gland.

SALIVARY GLAND INFECTIONS

Salivary gland infections may result from viruses or bacteria. The mumps virus, a member of the paramyxovirus family (see Chapter 313), is the classic example of parotitis. Although vaccination has decreased the prevalence of this disease, outbreaks still occur with some regularity. The cardinal symptoms of parotitis with fever are seen in 60% to 70% of all infections and in 95% of symptomatic patients. Bilateral involvement is seen in 90% of patients, although the swelling may not be seen at the same time on both sides; 10% of patients will also have submandibular and sublingual gland involvement. About one-third of mumps infections are asymptomatic. Sialectasis with further recurrent swelling is a rare complication. A variety of other viruses may be associated with salivary gland involvement including cytomegalovirus (CMV), EBV, HIV, and coxsackievirus.

Bacterial infections of the salivary glands that manifest as suppurative parotitis of parotid abscesses are uncommon in children and adolescents. Patients typically present with painful unilateral swelling of the parotid glands and purulent exudate from the parotid duct (Stenson duct), although infections can be bilateral. Not infrequently, patients require hospitalization for severe dehydration accompanied with high fever. Comorbidities are also common, including cystic fibrosis, leukemia, HIV, diabetes, and infantile cerebral palsy. The majority of these infections are caused by staphylococci and streptococci. Treatment of acute suppurative parotitis consists of gland massage, sialagogues, and antibiotic therapy to cover the suspected pathogens. The most common complication is abscess formation.

Neonatal suppurative parotitis, a form of acute sialadenitis occurring immediately after birth, is characterized by erythema and edema around the affected gland. Generally unilateral, purulent exudate can typically be expressed from the parotid duct. Reports suggest that fever and leukocytosis are not universal findings, and the most common organism isolated is *Staphylococcus aureus*. It has been suggested that the infection may be the result of retrograde flow of bacteria-laden saliva secondary to dehydration. Premature birth is also considered a risk factor. Patients treated with antibiotics generally have good outcomes.

OBSTRUCTIVE SIALADENITIS

Obstructive sialadenitis occurs as the result of a sialolith or foreign body blocking the normal flow of saliva, most commonly from the submandibular gland. Patients often complain of a painful swelling related to meals. The swelling generally dissipates over several hours. Symptoms may include pain, erythema of the overlying skin, fever, and an elevated white blood cell count. Treatment includes antibiotics, salivary flow stimulation, and heat. Surgery may be indicated to remove the sialolith and to establish patency of the duct. In recurrent cases, excision of the submandibular gland may be necessary.

HARD TISSUE PATHOLOGY

Intraoral hard tissue pathologies refer to conditions afflicting the skeletal components of the masticatory system, namely the maxilla, mandible, and alveolar bone, or the dentition. Both the skeletal conditions and anomalies of tooth number, size, shape, and color may manifest as isolated occurrences or may reflect an underlying systemic disturbance. Intraoral tumors and cysts involving the facial skeleton are relatively uncommon in children. An analysis of oral and maxillofacial pathology over a 30-year period revealed that only 8% of the specimens came from children. The vast majority of oral pathology in children is related to the dentition and oral soft tissues, as demonstrated by several large biopsy surveys. Dental or tooth-related pathology, salivary gland disease, and mucosal pathology accounted for the vast majority of biopsies in these studies. And while most intraosseous pathology is discovered as a result of a routine dental radiograph, enlargement of these tissues occasionally will be clinically detectable.

Flaitz and Coleman suggest that bony enlargements of the maxilla and mandible be divided into 3 categories: (1) inflammatory lesions, (2) benign cystic and neoplastic lesions, and (3) aggressive and malignant lesions. The inflammatory lesions are the most frequently encountered, followed closely by the benign cystic and neoplastic lesions. Aggressive and malignant lesions are rare in children, with a reported prevalence of less than 1% of biopsied lesions.

AGGRESSIVE AND MALIGNANT NEOPLASMS OF THE JAWS

Aggressive and malignant jaw neoplasms and tumors are rare in children. These include the melanotic neuroectodermal tumor of infancy (described elsewhere), Langerhans cell histiocytosis, Ewing sarcoma, primitive neuroectodermal tumor, osteosarcoma, and desmoplastic fibroma of bone.

Approximately 10% of Langerhans cell histiocytosis cases have oral involvement. Localized destruction of the tooth-supporting bone frequently results in mobile teeth, usually in the mandible. Typically, these hard tissue lesions are accompanied by soft tissue involvement characterized by localized ulceration and necrosis (see Chapter 459).

Ewing sarcoma and primitive neuroectodermal tumors involve the jaws in fewer than 3% of the cases. Typical symptoms are jaw enlargement frequently accompanied by pain. Approximately 6% to 7% of all osteogenic sarcomas occur in the maxillofacial region. The most common clinical manifestation is rapid enlargement of the affected bone that may or may not be accompanied with pain, which could mimic the symptoms of an odontogenic infection. Ulceration and paresthesia are commonly reported symptoms.

BENIGN CYSTIC AND NEOPLASTIC LESIONS OF THE JAWS

Intraosseous benign cysts and tumors are typically classified as either odontogenic, meaning arising from tooth-forming tissues, or nonodontogenic. Most odontogenic cysts in children are associated with

impacted teeth and are most frequently discovered from dental radiographs. Occasionally, these cysts will cause localized expansion that may be evident clinically. Of particular concern are odontogenic keratocysts, which typically occur in multiples when associated with basal cell nevus syndrome. These cysts have a relatively high recurrence following excision. The nonodontogenic cysts and tumors that may present with facial enlargement include the aneurysmal bone cyst, fibro-osseous lesions, fibrous dysplasia, cherubism, and central giant cell granuloma. Biopsy is necessary to establish a definitive diagnosis.

ATTRITION AND EROSION

After the initial mineralization process, tooth structure may be lost from a myriad of factors besides caries and traumatic fracture. These mechanisms include attrition, abrasion, and dental erosion.

Attrition is loss of tooth structure caused by tooth-to-tooth contact during mastication and occlusion. When the amount of tooth loss is extensive, it may begin affecting the appearance and function of the primary or permanent dentition. This wear may present in the form of habitual bruxism or involuntary grinding of the teeth. *Bruxism* is defined as an interruption of the normal rest position of the mandible during rest or sleep resulting from the forceful, rhythmic contractions of the masticatory musculature. It can be potentially harmful to the health of the oral tissues and restorative work. The prevalence of bruxism in children noted in the literature varies from 7% to 88%. This has been reported to occur in association with parasomnial activity and various dental, mental, neurological, and psychological risk factors. It has also been noted as a factor of other dental conditions such as malocclusion, eruption, or localized conditions, including mobile teeth, cuspal interferences, high restorations, and occlusal disharmony.

Dental erosion is an irreversible dental hard tissue loss due to acid exposure in the absence of bacterial involvement. Erosion may be caused by intrinsic factors, including recurrent vomiting (as in bulemia nervosa), regurgitation, gastroesophageal reflux (GER), or rumination. Extrinsic sources of acid, such as diets composed of acidic foods and drinks, carbonated beverages, and alcohol, are also possible causes of dental erosion. Continuous ingestion of acidic medications like chewable vitamin C and aspirin are also known causes of dental erosion.

Erosion is a relatively common finding in the dentition of children. Studies have suggested that up to two-thirds of children will present with some degree of dental erosion, with a 3-fold greater prevalence of dental erosion in primary teeth than permanent teeth. Erosion in primary dentition may be more severe than in the permanent dentition, which may be due to (1) the pattern of mineralization being different in primary teeth, or (2) thinner primary enamel. Dental erosion can result in pain, malocclusion, and even temporomandibular joint dysfunction (as the hard tissue wears, the bite deepens, causing changes in the temporomandibular joint).

Gastroesophageal reflux has been associated with varying degrees of dental erosion of both the primary and permanent dentition. The reported prevalence of dental erosive lesions in pediatric patients with GER disease ranges from 14% to 87%.

DISCOLORATION AND STAINING

The color of normal teeth varies and can be affected by a variety of factors, such as hygiene practices, diet, habits, bacteria, and underlying medical conditions. This discoloration can be classified as either extrinsic or intrinsic in nature. Extrinsic stains, or superficial pigmentation of the enamel, are caused by such things as bacterial stains, tobacco, foods, restorative materials, and medications like iron supplements. For example, chromogenic bacteria can produce stains that vary from orange to black-brown, and extensive use of tobacco, tea, and coffee often results in significant brown discolorations on the enamel's surface. However, the most common cause of such extrinsic stains is poor oral hygiene. Generally, extrinsic stains can be removed with a thorough dental prophylaxis.

Intrinsic stains are secondary to endogenous factors, resulting in discoloration of the dentin. These may be secondary to hereditary conditions such as amelogenesis or dentinogenesis imperfecta. They may also be attributed to dental fluorosis, erythropoietic porphyria, hyperbilirubinemia, and medications. Historically, tetracycline use in pregnant women and young children was 1 of the more common etiologies of intrinsic staining. Its ability to cross the placental barrier and chelate calcium results in staining of both teeth and bone in the developing fetus and child. Consequently, package warnings regarding this complication became commonplace, with tooth staining a rarity. An exception to this phenomenon is minocycline, which can result in abnormal pigmentation of the skin, thyroid gland, nails, bone, sclera, and conjunctivae in adults. Unlike tetracycline, minocycline has been reported to cause generalized intrinsic tooth staining posteruption. The mechanism of this staining is unclear but is likely related to the dynamic nature of the tooth dentin.

FUSION AND GEMINATION

Fusion and *gemination* refer to 2 distinct conditions resulting in a similar clinical appearance of "twinned" or "double tooth." They represent disturbances in the morphodifferentiation stage of tooth development.

Gemination is an attempt of a single tooth bud to divide, resulting in an incomplete formation of both the teeth and their clinical crowns. This may result in crowns that are partially or totally separate but with a single root canal. The total tooth count is normal. Fusion, however, is a complete joining of enamel or dentin of 2 distinct tooth buds. Usually there is 1 fewer tooth, unless this fusion occurs with an extra or supernumerary tooth. Fusion and germination are seen more commonly in the primary than in the permanent dentition. Fusion or gemination is found relatively frequently, occurring in 0.5% to 2.5% of the population studied.

HYPODONTIA

Congenitally missing teeth (tooth agenesis) is 1 of the most common dental diagnoses in the pediatric population. The congenital absence of teeth results from disturbances during the initial stages of tooth formation, initiation, and proliferation. Missing teeth can occur in isolation or may be associated with an underlying syndrome or medical condition and can range from complete anodontia (all teeth) to hypodontia (1 or more teeth). Additionally, these dental anomalies have been reported in children who have cleft lip, cleft palate, ectodermal dysplasia, Down syndrome, and Hallermann-Streiff (mandibulo-oculofacial) syndrome.

Isolated missing teeth in the permanent dentition are not rare. The most common missing permanent teeth are third molars, with prevalence rates reported at 9% to 37%. This is followed by mandibular second premolars and maxillary lateral incisors, depending on the population studied. It is important to note that it is possible for any tooth in the dental arch to be congenitally absent. Referral to a pediatric dentist may be necessary to determine whether the tooth is, indeed, congenitally absent or has failed to erupt.

SUPERNUMERARY TEETH/HYPERDONTIA

Supernumerary teeth and conditions of hyperdontia occur when there is more than the normal complement of primary or permanent teeth. Overall, the prevalence of supernumerary teeth ranges from 1.5% to 3.9%. Single-tooth hyperdontia is most commonly found in the anterior maxillary region in the form of a mesiodens. A mesiodens is a supernumerary tooth (or teeth) present in the maxillary midline between the permanent central incisors and is often an incidental finding on dental radiographs. It is usually located palatally to the permanent incisors and may interfere with eruption or, if the incisors erupt, may result in a malposition. In the absence of timely eruption of the maxillary permanent incisors, referral to a pediatric dentist is appropriate, and removal may be necessary. Occasionally, supernumerary teeth may be found in the primary dentition and may signal the presence of extra permanent teeth as well.

Multiple supernumerary teeth may be a feature of several syndromes, including Gardner syndrome, cleidocranial dysplasia (CCD), cleft lip and palate, Hallermann-Streiff syndrome, Down

syndrome, and chondroectodermal dysplasia. Overall, supernumerary teeth are 5 times more common in the maxilla, with a male-to-female ratio of 2:1. They are more commonly associated with permanent dentition. The etiology of supernumerary teeth is not completely understood.

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374 Temporomandibular Joint Disorders

Cory M. Resnick and Bonnie L. Padwa

INTRODUCTION

Temporomandibular joint (TMJ) disorders in children are relatively rare. They can be divided into extra-articular and intra-articular types, which can occur alone or together.

PATHOGENESIS, EPIDEMIOLOGY, AND CLINICAL MANIFESTATIONS

Myofascial Pain Dysfunction (MPD)

The most common problem affecting the TMJ is myofascial pain dysfunction (MPD), which is extra-articular in origin. As many as 25% of adults may experience signs of MPD at some point in their lives, and 7% of patients between ages 12 and 18 years will seek treatment for a TMJ-related problem. Patients with MPD often experience pain and decreased jaw function. Related symptoms include headache, ear pain, joint noises, closed or open lock, difficulty chewing, and limited mouth opening.

MPD is characterized by increased tension and spasm of the muscles of mastication for any reason. Risk factors associated with MPD include gender (female), parafunctional habits, psychiatric conditions, unstable dental occlusion, history of facial trauma, and hypermobility syndromes. MPD may be caused by hyperfunction or parafunction, such as nocturnal jaw clenching and teeth grinding (bruxism), or may be a manifestation of psychological stress. The TMJs are initially normal in patients with primary MPD, though longstanding MPD may create intra-articular joint problems. Primary intra-articular TMJ dysfunction also frequently leads to MPD.

Intra-articular Disorders

Intra-articular TMJ disorders result from an abnormal relationship between the mandibular condyle and the cartilaginous intra-articular disk. Intra-articular disorders may present as painless TMJ noises or limited mouth opening. Intra-articular dysfunction is closely tied to MPD, however, as spasm of the lateral pterygoid muscle, which inserts into the disk, can displace the disk anterior and medial to the condyle. Therefore, patients with intra-articular disorders often present with signs and symptoms similar to those with primary MPD.

An anteriorly displaced disk may or may not impair normal mandibular opening depending on the ability for the disk to return to its normal position as the mouth opens. Reduction of the disk is usually associated with a midopening click, which can be audible and/or palpable. Anterior displacement without reduction is often the result of chronic intra-articular dysfunction resulting in tearing and scarring of the retrodiscal tissues. When this occurs, the mandibular condyle can only rotate within its fossa and is unable to translate down the slope of the articular eminence. This results in a limitation of mouth opening to the 25-mm range measured between the incisal edges of the maxillary and mandibular central incisor teeth. Longstanding displacement may result in a change in shape or tearing of the disk.

Inflammatory Arthritis

The TMJs are affected in 39% to 75% of children with juvenile idiopathic arthritis (Chapter 198). It is unusual for TMJ pain and inflammation to be the first presentation of undiagnosed juvenile idiopathic arthritis, but within 5 years of diagnosis, 50% of children develop some TMJ symptoms. In the acute setting, there may be swelling and tenderness of the joint with a variable amount of pain and limited mandibular movement. Chronic inflammation may result in erosion of the condyle leading to anterior open bite and mandibular retrognathia.

Idiopathic Condylar Resorption

Idiopathic condylar resorption (ICR), a diagnosis of exclusion, is an acquired TMJ disorder that manifests as a progressive decrease in condylar size and shape and is not associated with TMJ ankylosis or orofacial anomalies. Patients exhibit a decrease in posterior face height, retrognathia, and an anterior open bite as a result of clockwise rotation of the mandible. This condition is usually bilateral and has a predilection for females between 15 and 35 years of age who often have preexisting TMJ dysfunction and a high mandibular plane angle.

Other TMJ Abnormalities

Some of the other abnormalities of the TMJ seen in children include post-traumatic deformities, condylar hyperplasia, and tumors. Unilateral and bilateral intracapsular and subcondylar fractures are the most common mandibular fractures in children and can result in ankylosis, overgrowth or undergrowth of the injured condyle with resultant facial asymmetry. Condylar hyperplasia is due to an abnormal hypermetabolic growth center within 1 condyle that leads to a malocclusion and progressive facial asymmetry. Tumors of the TMJ are rare, and the most common include fibro-osseous lesions and giant cell tumors.

DIAGNOSIS

The diagnosis of TMJ disorders requires a careful history and physical examination. Differentiating primary MPD from internal derangement may be difficult, and, as the 2 often occur concomitantly, it may be necessary to differentiate the primary versus the secondary problem.

Tenderness to palpation of the muscles of mastication, limitation in mouth opening, and reproduction of pain by jaw clenching and palpation of trigger points are consistent with MPD. Detection of an audible or palpable click at the TMJ points to an internal derangement. Progressive mandibular asymmetry and malocclusion may be associated with conditions affecting the size and shape of the condyle, including resorption from inflammatory arthritis or condylar hyperplasia. Symmetric retrognathism can be caused by bilateral inflammatory arthritis or ICR. In order to make a diagnosis of ICR, all other causes of condylar resorption must be ruled out.

The diagnosis of TMJ disorders may be aided by imaging tests, which include x-rays such as panoramic radiographs, computed tomography (CT) or cone-beam computed tomography (CBCT) scans, and/or magnetic resonance imaging (MRI). Computed tomography or CBCT are best for evaluation of the mandibular condyle and ramus and the articular surface of the glenoid fossa. Therefore, CT is the imaging exam of choice when assessing for condylar erosion, fracture, or tumor. Magnetic resonance imaging is most sensitive for viewing the disk and surrounding soft tissue structures. When an internal derangement is suspected, MRI without contrast agent can be obtained with both closed mouth and opened mouth series so that the position and configuration of the disk can be observed throughout the opening cycle. When an inflammatory arthritis is suspected, the MRI should be obtained with contrast so that synovial enhancement, a surrogate marker for synovitis, can be observed. Nuclear medicine tests such as technetium-99 bone scans may play a role in management of overgrowth and resorptive conditions such as condylar hyperplasia and ICR.

The Wilkes staging system for internal derangements, which is comprised of 5 stages, can be used to aid in treatment, decision-making, and prognostication. Stage I, characterized by painless clicking, indicates a displaced disk that reduces early in the opening cycle. In stage II, characterized by occasional painful clicking, there is a displaced disk that reduces late in the opening cycle. In stage III, there is an acute or subacute nonreducing disk, whereas in stage IV, there is a chronic nonreducing disk. Stage V is characterized by a chronic nonreducing disk and findings of secondary osteoarthritic joint changes.

TREATMENT

Treatment of MPD is focused on diminishing muscle hyperactivity. Goals of therapy include eliminating behavior that is noxious to the muscles and joints; decreasing the frequency, duration, and intensity of pain episodes; providing support and counseling about stress reduction or other psychologic concerns; and establishing a functional and adequate jaw opening. Most patients with MPD respond well to nonoperative treatments.

Nonoperative treatment modalities include a soft diet with elimination of hard, chewy, and crunchy foods, thereby reducing the loading forces on the joint and decreasing muscle activity. Physical therapy to increase range of joint motion and muscle relaxation, massage of the muscles of mastication, and cold or hot compresses applied before and after exercises may also be helpful. Ultrasound can deliver heat to a depth of about 5 cm, and when used in combination with stretching, can alter the elastic properties of the underlying connective tissue.

Splint therapy with the use of a flat plane acrylic appliance that fits onto the dentition and disoccludes the teeth and jaws diminishes the loading forces on the disk. Additionally, the splint distracts the condyle slightly out of its fossa and reduces pressure on the intracapsular tissues. Interrupting the normal proprioception of the masticatory system reduces muscle activity. Splints are frequently worn at night by patients with bruxism. However, patients with chronic myofascial pain may need to wear the splint for up to 24 hours a day for a period of time to sustain symptomatic relief. The patient should subsequently be weaned from the splint.

Psychological stress is thought to play an important role in myofascial pain. Pain and limitation of TMJ movement that results from stress-induced muscle contraction can be treated with a variety of stress-reduction techniques and biofeedback. It is important to communicate the role of stress to the patient and to obtain a history of recent stressors (school or work pressures, family troubles). Counseling may be indicated.

A variety of pharmacologic agents have been used to treat MPD. Nonsteroidal anti-inflammatory agents are the most commonly administered. Anxiolytics reduce anxiety and muscle tension. Muscle relaxants invoke relaxation of skeletal muscle by a depressant effect on the central nervous system, albeit with side effects. Analgesics are occasionally used for treating acute pain episodes. Antidepressants are often useful in treating myofascial pain because psychiatric illness is common among chronic TMJ patients. Because of the close relationship between chronic pain and depression, treatment for chronic pain, even without depression, sometimes yields a response to antidepressants.

Additional nonoperative therapies include trigger point injections of a local anesthetic into areas of muscle that are palpable foci of hypersensitivity. Injection of botulinum toxin to paralyze hyperactive muscles may also decrease pain.

The myofascial pain components of internal derangement disorders are often treated successfully with nonoperative measures. However, arthrocentesis and/or arthroscopy of the upper joint space with lavage, lysis of adhesions, and injection of steroids and/or sodium hyaluronate can be helpful for those who do not fully respond to these treatments or for those in whom the primary pathology is within the joint. Arthroscopy has an added advantage over arthrocentesis in providing visualization of the superior joint space, which allows improved diagnosis, targeted lysis of adhesions, and the ability to perform additional procedures such as disk repositioning.

Refractory cases of internal derangement, especially Wilkes stages IV and V, and certain other TMJ pathologies may require open joint surgery for correction. Access is obtained via a preauricular incision, allowing both problems with the disk (repositioning, removal, replacement) or with the osseous structures (removal of tumor, repositioning or replacement of the condyle, removal of the articular eminence) to be addressed. In severe TMJ deformities, the components of the joint may require removal and replacement with either autologous grafts (eg, costochondral grafts) or alloplastic materials (eg, total alloplastic joint replacement).

Treatment for inflammatory arthritis of the TMJ depends on the severity of the condition, but in general is similar to treatment of other joints: relieve pain, improve function, and prevent destruction and deformity. In the acute stages, treatment options include systemic medication and/or intra-articular steroid injections. For treatment of late-stage mandibular deformities, reconstruction of eroded mandibular condyles, often in conjunction with orthodontic treatment, may be necessary.

Treatment for nonarthritic resorptive and overgrowth conditions, such as ICR and condylar hyperplasia, involves an operation to restore the normal facial profile. For ICR, treatment should be delayed until after documentation that the process has ceased. For condylar hyperplasia, management with a high condylectomy (removing the abnormal growth center) can be pursued during the active growth phase, or treatment with orthognathic surgery in conjunction with orthodontic treatment to align the teeth and reposition the jaw can be delayed until after condylar growth has become quiescent and skeletal maturity has been reached. For treatment of tumors affecting the TMJs, resection of the tumor and reconstruction of the ramus-condyle unit are usually indicated.

COMPLICATIONS AND LATE EFFECTS

MPD and internal derangement of the TMJ often become chronic problems with signs and symptoms waxing and waning throughout life. Findings may progress, sometimes with associated osteoarthritic joint destruction. This may lead to the need for increasingly aggressive treatment.

Longstanding or aggressive resorptive or overgrowth conditions can lead to significant facial deformity and malocclusion. If not corrected, patients may experience pain, limited ability to chew foods, and negative impact on psychosocial development.

The most dreaded complication of TMJ disorders is ankylosis, which involves formation of fibrous and/or osseous connections between the mandibular condyle and the glenoid fossa, thereby

limiting mobility. Ankylosis most commonly occurs as a result of trauma followed by a period of hypomobility, but any TMJ problem can theoretically lead to ankylosis. Ankylosis requires operative intervention for correction, often recurs, and is associated with significant morbidity.

CONCLUSIONS

TMJ disorders are relatively rare in children, and MPD is the most common type. MPD typically follows a waxing and waning course and responds to nonoperative treatments. Internal derangements, resorptive and overgrowth conditions affecting the mandibular condyles, and other TMJ disorders may require operative interventions for management.

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375 Behavioral Treatment Options in Pediatric Dentistry

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INTRODUCTION

This chapter discusses various behavioral treatment options for comprehensive dental treatment in infants, young children, adolescents, and individuals with special healthcare needs.

INFLUENCES ON CHILD'S BEHAVIOR AND CHANGES IN CHILD REARING

The task of the pediatric dentist has not changed in the last 60 years: to perform precise surgical procedures, measured in millimeters, on children whose behavior may range from cooperative to apprehensive to defiant. Additionally, these very precise surgical procedures require the child's full attention and commitment for varying lengths of time. In a survey of the American Academy of Pediatric Dentistry members on the use of behavior management techniques, the great majority of pediatric dental specialists (88%) believe that changes in parenting styles during their years of practice may have contributed to an increase in behavior management problems in the dental setting. In a recent survey, 50% of parents bringing their children to a children's hospital emergency department for after-hours emergency care expected that their child would be sedated for dental treatment.

Today's pediatric dental environment is designed to be child friendly. Consumer electronics, 21st century aesthetic dental materials, leading-edge digital dental technology, and well-trained dental auxiliaries add to the child-friendly environment of today's pediatric dental office. Decisions regarding behavior management of the pediatric dental patient must be made in concert with the parent and the dentist. Parents who call to request sedation or general anesthesia for appointments to clean their child's teeth because the child "cried at the last visit" often do not understand the indications, risks, benefits, limitations, or costs of anesthesia or sedation. Similarly, the pediatric dentist must evaluate as part of the overall treatment plan the child's potential for cooperation in light of age, individual growth and development, prior overall health and dental experiences, and the family's attitude toward dental health. The pediatric dentist must then provide safe, competent, comprehensive, and relatively pain-free care in an affordable manner without creating fear.

The American Academy of Pediatric Dentistry has a long history of developing guidelines for behavior management. The most recent guidelines state that behavior guidance techniques must be tailored to the individual patient and practitioner. Successful behavior guidance enables the oral health team to deliver treatment safely and efficiently and to nurture a positive dental attitude in the child. The goals of behavior guidance are to establish communication, alleviate fear and anxiety, deliver quality dental care, build a trusting relationship between the dentist and child, and promote the child's positive attitude toward oral/dental health care. Communication between the child and pediatric dentist is developed through an ongoing process of dialogue, both verbal and nonverbal, that involves facial expressions, body language, and voice tone, always taking into consideration the cognitive level of the child. This is a comprehensive, continuous method that must be individualized for each child.

All behavior guidance techniques, other than communicative techniques, utilized by the practitioner must involve the consent of the parent/guardian and, if appropriate, the child. The parent must be informed of the nature, risks, and benefits of the technique to give informed consent. Communicative techniques, because they are basic to any dialogue with the child, require no informed consent.

NONPHARMACOLOGIC BEHAVIOR MANAGEMENT

Parental presence during treatment is common and generally expected in our society. Parents must be educated so that they do not hover above or smother their child nor indicate their own discomfort, if applicable, through words or body language. Most behavior guidance techniques used in pediatric dentistry are "behavior shaping" communicative procedures, such as tell-show-do or the similar ask-tell-ask (Table 375-1).

The tell-show-do technique involves verbal explanations of procedures in phrases appropriate to the developmental level of the patient (tell); demonstration for the patient of the visual, auditory, olfactory, and tactile aspects of the procedures in a carefully defined, nonthreatening setting (show); and then, without elapsed time or deviation from the explanation and demonstration, completion of the procedure (do). The tell-show-do technique is used with continuous communication skills (verbal and nonverbal) and positive reinforcement. The ask-tell-ask technique, while similar to tell-show-do, assumes more of a dialogue, where children are asked if they know why they are in the office and how they feel about it; with the response in mind, the upcoming procedure is further explained (tell); and children asked again if they understand better what is to be done and what is expected of them,

TABLE 375-1 BEHAVIORAL GUIDANCE OPTIONS IN ORDER OF DECREASING PATIENT INTERACTION

- Parental presence
- Tell-show-do/ask-tell-ask
- Positive reinforcement
- Distraction
- Contingent distraction
- Voice control
- Modeling
- Live modeling
- Time out
- Contingent time out
- Mouth prop (intraoral stabilization)
- Parental restraint or wraps (protective stabilization)
- Pharmacologic adjuncts
- Minimal sedation using nitrous oxide/oxygen
- Moderate and deep sedation
- General anesthesia

and feel more comfortable. Communications are in terms that the children can understand.

Once beyond the tell-show-do explanation, the pediatric dentist may use positive reinforcement, role modeling, voice control, and time out, perhaps contingent on some accomplishment. "Attending" is a reinforcement technique that calls attention to discrete desirable behavior and doing what is asked, and, therefore, helps to shape a positive relationship and appropriate responses. It is the obvious choice in positive reinforcement, calling attention to what to do, rather than what not to do. Positive reinforcement is also a common and effective technique whereby the desired behaviors are rewarded in an effort to strengthen those behaviors and eliminate undesirable responses. The technique includes positive voice modulation, verbal praise, and small tokens such as stickers (which are never withheld). Voice control is a controlled alteration of voice volume, tone, or pace to influence and direct the patient's behavior. By necessity, it may be the initial way to gain the child's attention and compliance so that tell-show-do may be utilized. Parents who are unfamiliar with the technique should have an explanation of the procedure prior to its use to prevent misunderstanding.

Time-out or contingent escape gives children a sense of control over the dental procedure and trust in the dentist. By being able to raise a hand to stop the dental procedure when they perceive discomfort or need a break, children feel an element of control over their environment, and the technique encourages communication through cooperative behavior. This approach provides immediate feedback to teach children more adaptive coping behaviors. Distraction is the technique of diverting the patient's attention away from an unpleasant procedure. Giving the child a mirror to hold during the procedure or allowing the child to wear headphones or watch a movie are all effective distraction techniques. Role modeling may be found in many pediatric dental offices where children can observe behavior and treatment of other children who share a similar experience and who will help them feel more at ease by example.

Nonverbal communication, with appropriate eye contact, posture, facial expression, and body language to reinforce and enhance communicative behavior guidance, is used throughout the procedure. The presence of the parent, particularly with very young children, can sometimes be used to gain a child's cooperation for the procedure. While there remains disagreement among practitioners regarding parental presence or absence during pediatric dental treatment, there are clearly more parents present in the dental operatory today. While authoritarian methods of guidance are less acceptable, restraint in the form of mouth props (to avoid inadvertently biting on the instruments) or protective (parental) stabilization may be utilized when treatment is medically necessary and the child is uncooperative.

PHARMACOLOGIC AGENTS FOR SEDATION

Once beyond communicative efforts and minimal levels of restraint, dentists may turn to the use of pharmacologic agents. Guidelines developed by the American Academy of Pediatric Dentistry and the American Academy of Pediatrics divide sedation into 3 definable levels: minimal, moderate, and deep.

Minimal Sedation: Use of Nitrous Oxide

Nitrous oxide administration is the most common minimal sedation technique used by pediatric dentists to reduce anxiety. Its use requires a nasal hood and depends on the child's understanding and willingness to breathe through the nose. When children are able to cooperate in the inhalation process, nitrous oxide/oxygen is an excellent, safe, and effective technique to reduce anxiety and enhance effective communication in children with anxiety. Its onset and recovery are rapid, and the effects are easily titrated and reversible.

Nitrous oxide analgesia has a predictable effect among the majority of children. It raises the pain threshold, increasing the tolerance for and duration of procedures; reduces the gag reflex; and potentiates the effect of other sedatives (if utilized). Nevertheless, the use of nitrous oxide in children who are severely disruptive may prove futile, as precooperative or noncooperative children (or adults with disabilities) often breathe through their mouth, thus negating its effects. Children with compulsive personalities or "take-charge" children may not like the feeling of loss of control associated with nitrous oxide, and they may unilaterally terminate the procedure. In addition, nitrous oxide/oxygen inhalation has a very low success rate in children with upper respiratory tract or other acute respiratory infections. Any respiratory problem that prevents the use of the nasal inhaler as the route of entry for anxiolytic gases (eg, common cold, acute or chronic sinusitis, chronic mouth breathing, enlarged tonsils and/or adenoids, allergic rhinitis, tuberculosis, bronchitis, cough, chronic obstructive pulmonary disease [COPD], emphysema) should be avoided.

It should also be noted that air spaces in the maxillary sinus, vestibulocochlear complex, and bowel can be displaced by nitrous oxide. These nonrigid spaces can enlarge and cause discomfort as a result of high concentration and prolonged use of nitrous oxide/oxygen analgesia. The avoidance of hypoxia is especially important in children with congenital methemoglobinemia. The use of nitrous oxide/oxygen analgesia is also best avoided in the pregnant patient during the first trimester to avoid increasing the possibility of spontaneous abortion. Medical consultation between the dentist and obstetrician is always advisable for the pregnant patient. The psychogenic effects of nitrous oxide/oxygen analgesia should also be considered prior to its administration in children with mental health concerns.

Moderate to Deep Sedation and General Anesthesia

Although most children can be treated effectively with communicative measures or minimal sedation using nitrous oxide, some do not accept the nasal hood and require more advanced (less interactive) methods of behavioral guidance. The techniques commonly are used when children present with significant dental disease and behavioral considerations that cannot be controlled in the usual manner (eg, lack of psychological, emotional, or chronological maturity; mental, physical, or medical disability; or young age with overwhelming caries).

Children who are classified as American Society of Anesthesiologists Physical Status Classification System (ASA) III (severe systemic disease) or IV (severe life-threatening disease), who have special needs, and who have anatomic airway abnormalities or extreme tonsillar hypertrophy may have airway issues that require additional and individual consideration, particularly for moderate and deep sedation. Pediatric dentists must consult with appropriate subspecialists and/or anesthesiologists if there is an increased risk of experiencing adverse sedation events because of a patient's underlying medical or surgical condition. Additionally, in addition to performing the procedure in an appropriate environment, with appropriate providers, the dentist and staff must have training and skills necessary to appropriately manage any adverse response resulting from sedation. It is important

to emphasize that pediatric dental care under general anesthesia represents the behavior treatment modality of last resort after careful consideration of the requisite treatment and the risk-benefit ratio for the individual child.

INDIVIDUALS WITH SPECIAL HEALTHCARE NEEDS

Individuals with special healthcare needs may be at an increased risk for oral diseases throughout their lifetime. Such children may have aversions to consistency, textures, taste, or food temperature, making their diets more limited and challenging. Tongue coordination difficulties and oral tactile sensitivities may result in an inability to chew or swallow effectively or in pouching of food. Esophageal reflux can lower the intraoral pH which may lead to dental erosion and an increased risk of dental caries (see Chapter 369). Unmet dental needs of children with special healthcare needs and subsequent higher expenditures than for typical children emphasize the need for a dental home that seamlessly communicates with the patient's medical home and caregiver circle that may include the parents, family, pediatricians, specialists, community, teachers, therapists, and psychiatrists. With the support of the caregiver circle, most patients with physical and mental disabilities can be treated in the dental office with the traditional behavior management techniques discussed previously. However, special accommodations (eg, a sign language interpreter or picture boards) may be required for the dental visit. Other accommodations may be physical, such as keeping the child in his/her own reclined wheelchair or providing for protective stabilization, when traditional behavior management techniques are not adequate. If the medical findings, behaviors, or dental needs make dental care in the office not feasible, then either sedation or general anesthesia becomes necessary for providing optimal dental care.

The need for flexibility in the healthcare provider is paramount. Sometimes there is a special person in the patient's life whose presence in the dental office may ease the anxiety. If the individual won't sit in the dental chair, but is willing to lay on the floor, sit in the caregiver's chair or stand for dental treatment, this may become the initial venue for preventive visits, until trust is achieved and the person is willing to sit in the dental chair and be reclined. Familiarization through repetition of tasks is the hallmark of applied behavior analysis (ABA) training and can be applied to the dental environment. With frequent dental visits, there is a shared learning curve for both the pediatric dentist and the patient. For children with special healthcare needs David Tesini, DMD, says it best: "It becomes less about managing behavior and more about understanding it."

CONCLUSION

Oral health is an essential component of general health, and thus it is important that all children receive regular dental care. Parental involvement and an understanding of its importance are key to optimum oral health. Dentists and pediatricians should have a firm understanding of the psychosocial development of the child and, together with the menu of available techniques for behavioral guidance, should be capable of providing dental preventive and restorative care in a manner that will be acceptable, reproducible, and effective for infants, young children, and adolescents in the most cost-effective setting.

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