Parents often are concerned with "lumps and bumps" that appear in the mouths of children. Pediatricians should be able to distinguish the normal clinical appearance of the intraoral tissues in children from gingivitis, periodontal abnormalities, and oral lesions. Recognizing early primary tooth mobility or early primary tooth loss is critical because these dental findings may be indicative of a severe underlying medical illness. Diagnostic criteria and treatment recommendations are reviewed for many commonly encountered oral conditions.

INTRAORAL SOFT-TISSUE ABNORMALITIES

Congenital Lesions

Ankyloglossia

Ankyloglossia, or "tongue-tied," is a common congenital condition characterized by an abnormally short lingual frenum and the inability to extend the tongue. The frenum may lengthen with growth to produce normal function. If the extent of the ankyloglossia is severe, speech may be affected, mandating speech therapy or surgical correction. If a child is able to extend his or her tongue sufficiently far to moisten the lower lip, then a frenectomy usually is not indicated (Fig. 1).

From Private Practice, Waldorf, Maryland (JED); and Department of Pediatrics, Division of Pediatric Dentistry, Duke Children's Hospital, Duke University Medical Center, Durham, North Carolina (MAK)
Developmental Lesions

Geographic Tongue

Benign migratory glossitis, or geographic tongue, is a common finding during routine clinical examination of children. It occurs most commonly on the dorsum and lateral borders of the anterior two thirds of the tongue. Geographic tongue appears as pink to red, round or irregular areas of dekeratinization and desquamation of filiform papillae with white or yellow elevated margins. Geographic tongue continuously changes patterns, creating a migratory appearance on the tongue. The lesions are usually asymptomatic but may be painful when inflamed. More common in girls, the condition has no known cause, although it has been associated with allergies in children. No treatment other than reassurance usually is indicated.
**Fissured Tongue**

Fissured tongue is a developmental anomaly that usually presents as one marked central fissure, anteroposteriorly, from which smaller fissures radiate laterally. The fissures may be shallow or deep. In deep fissures, food debris may become trapped, leading to inflammation or secondary fungal infections. Fissured tongue is less common in children than in adults; however, this is a common finding in children with Down syndrome (Fig. 2).

**Retrocuspid Papillae**

Present in most children, the retrocuspid (or retrocanine) papilla is an anatomic structure located on the attached gingiva lingual to the mandibular canines. Usually 2 mm to 3 mm in diameter, these lesions are pink to red. Frequently found bilaterally, these firm, round fibroepithelial papules are asymptomatic. They usually decrease in size and incidence with age; therefore, no treatment is necessary (Fig. 3).

**Gingival Overgrowth**

Gingival enlargement may be an inherited trait, may be induced by drugs, or may occur in patients with leukemia. Hereditary gingival

![Image](Image)

*Figure 2. A fissured tongue with a focal area of inflammation in a 4-year-old child.*
fibromatosis, or idiopathic hyperplasia, is a rare, progressive, fibrous enlargement of the gingiva beginning in early childhood. The thick, firm, and pink gingival enlargement affects buccal and lingual surfaces of both jaws. Failure or delay in the eruption of primary and permanent teeth is a common finding. Gingivectomy is the treatment of choice. Meticulous oral hygiene is recommended to help reduce recurrence.

A similar drug-induced gingival enlargement is seen in half of children taking phenytoin to control seizures, and other drugs, such as cyclosporin A, an immunosuppressive drug, and nifedipine, a calcium channel blocker (Fig. 4). Use of these drugs before tooth eruption may lead to delay or lack of eruption of the teeth into the oral cavity because
of the dense fibrous overgrowth. Taken after tooth eruption, these drugs produce overgrowth beginning at the interdental papillae. The gingival enlargement is exacerbated by the inflammatory response to dental plaque, with most prominent overgrowth on the labial aspects of anterior teeth. A combination of two or more of these drugs can lead to extreme rapid gingival proliferation, necessitating an alteration in the drug regimen for the patient. Gingival enlargement usually is not dose related; however, removal of the drug may resolve the overgrowth. For any child taking a drug with the potential for gingival enlargement, excellent oral hygiene is recommended. A gingivectomy is recommended for severe cases.

Gingival enlargement also may occur in patients with leukemia, especially the monocytic type, because of infiltration of the gingival tissues by malignant white blood cells. The gingival overgrowth in this case is edematous and hemorrhagic versus dense and fibrotic. Because of the bleeding tendency, patients may not practice good oral hygiene. Resulting inflammation may provide the stimulus for further connective tissue hyperplasia.14

**Benign Tumors**

**Hemangioma**

Hemangiomas are benign, enlarged, vascular hamartomas that may occur in any soft-tissue location, most commonly on the lip, dorsum of the tongue, gingiva, and buccal mucosa. Clinically, hemangiomas typically present as red or bluish red, slightly raised lesions and are moderately firm to palpation (Fig. 5). They occur early in life and are more common in girls than in boys. Hemangiomas may enlarge rapidly or progressively as the patient grows. They are usually painless but may ulcerate and possibly hemorrhage if traumatized. The hemangioma is a tumor of mesenchymal origin, which is characterized by the formation of vascular tubes of endothelial cells. Treatment consists of laser therapy or surgical resection of the lesion. The potential for severe hemorrhage caused by the vascular nature of the lesion must be considered.10

**Lymphangioma**

Lymphangiomas are benign tumors of lymphatic vessels that are present at birth or develop early in life. The tongue is the most common intraoral site, followed by the lips and buccal mucosa. Superficial tumors are soft and compressible and may be pink to reddish-blue. Deeper lesions may show no surface changes. Cystic hygroma is a large lymphangioma involving the floor of the mouth and neck. Any lymphangiomas creating a functional or aesthetic concern usually are surgically removed. Recurrences are common and caused by their lack of encapsulation.

**Mucocele**

The mucous extravasation phenomenon is commonly known as *mucocele*. This retention of mucus in subepithelial tissues most commonly occurs in children and adolescents, on the lower lip midway between the midline and commissure (Fig. 6). The cause is mechanical trauma to a minor salivary gland, with pooling of mucus in an obstructed and

Figure 6. A mucocele on the lower lip of a 6-month-old infant.
dilated excretory duct. The lesions are usually painless and smooth surfaced and are bluish or translucent. Most mucoceles are less than 1 cm in diameter. The treatment of choice is surgical excision, with removal of associated minor salivary glands to prevent recurrence.

**Fibroma**

One of the most common benign lesions of the oral cavity, the fibroma results from reactive connective tissue hyperplasia caused by a chronic irritant. Fibromas may be found on any area of the oral mucosa, but primarily on the palate, tongue, cheek, and lip (Fig. 7). Most fibromas are less than 1 cm in diameter, pale pink, smooth, and firm and have a sessile or pedunculated base. Treatment involves surgical excision and removal of the source of the irritation. Recurrence is rare.

**Odontogenic Cysts**

**Parulis**

The parulis or "gum boil" appears as a soft solitary, reddish papule located facial and apical to a chronically abscessed tooth. (See article by Caufield and Griffen earlier in this issue for additional discussion and figure.) It occurs at the endpoint of a draining dental sinus tract. Acute swelling and pain may result if the sinus tract is obstructed, but symptoms temporarily are relieved when pus escapes to the surface. Treatment of a parulis in a child consists of diagnosing which tooth is abscessed with clinical examination and a periapical radiograph and extracting the tooth or performing complete pulpectomy. The treatment of choice depends on the prognosis of successful pulpal therapy and the strategic importance of the tooth. Regression of the parulis usually

**Figure 7.** A 5-year-old child who has a habit of sucking her lip through her teeth resulting in a circumscribed fibroma on her lower lip.
occurs shortly thereafter. If the offending tooth is left untreated, the parulis may persist for years or mature into a pink fibroma (Fig. 8).

**Eruption Cyst and Hematoma**

Eruption cysts are associated with erupting primary and permanent teeth. They present as dome-shaped soft-tissue lesions overlying any erupting tooth in children. The eruption cyst results from fluid accumulation within the follicular space of an erupting tooth. When the fluid in the cyst is mixed with blood, the cyst is referred to as an eruption hematoma. No treatment is needed because the tooth erupts through the lesion, which disappears spontaneously.

**Infectious Diseases**

**Herpesvirus Infection**

**Primary Herpetic Gingivostomatitis.** The initial infection with herpes simplex virus (HSV) occurs in young children after contact with an infected child or adult. The manifestations of primary herpetic infection may be flulike symptoms or subclinical. In primary gingivostomatitis, after an incubation period of approximately 1 week, the patient complains of fever, malaise, and irritability. Marginal gingivae become fiery red, edematous, and may bleed easily. Clusters of small vesicles erupt throughout the mouth, then burst to form yellow vesicles surrounded by a red halo. The vesicles coalesce to form large ulcers of the oral and
perioral tissues. The lesions are accompanied by pain, fever, arthralgia, headache, and cervical lymphadenopathy.

Primary herpetic gingivostomatitis usually regresses spontaneously within 2 weeks without scarring. Treatment is supportive. Fluids should be encouraged because the child may become dehydrated. Antipyretics, analgesics, and oral anesthetic rinses may make the child more comfortable. Diphenhydramine (Benadryl) and attapulgite (Kaopectate), mixed equally, may be used as a topical anesthetic to control discomfort at meal times. Antibiotics may be given to prevent a secondary bacterial infection in the oral cavity. Caution should be exercised to prevent autoinoculation of the child's eyes, genitalia, or fingernail beds because additional HSV infection could result. The child should be isolated from other children.

Interestingly, studies have associated undiagnosed primary herpetic infection with the symptoms of teething difficulty; reports of fever, irritability, sleeplessness, and difficulty with eating are remarkably similar. King et al examined infants presenting with a parental chief complaint of teething difficulty and found positive HSV cultures in 9 of 20 infants.

Herpes Labialis and Recurrent Herpetic Stomatitis. Following the initial infection, HSV migrates to the trigeminal ganglion, where it remains in a latent state until reactivation following exposure to sunlight, cold, trauma, stress, or immunosuppression. Also known as fever blister or cold sore, herpes labialis is characterized by a vesicular eruption on the skin adjacent to the vermilion border of the lip. Usually, prodromal symptoms of tingling, burning, or pain are present at the site. The vesicles rupture to form ulcers and crusts, which heal without scarring within 2 weeks. When reactivation of HSV appears intraorally in recurrent herpetic stomatitis, lesions are almost always on the hard palate or attached gingiva. Treatment of recurrence of HSV infections includes acyclovir in severe cases or when the patient is immuno-suppressed. Sunscreen is recommended to prevent recurrences of lip lesions.

Coxsackievirus Infections

Herpangina. Herpangina is a disease of the oral cavity that occurs in infants and young children in the summer or early fall. This acute viral infection is caused by group A coxsackieviruses. It is highly contagious and has an incubation period of 4 days, after which the patient develops a fever, malaise, headache, vomiting, abdominal pain, and intraoral lesions. Vesicles appear on the anterior pillars of the tonsillar fauces, soft palate, uvula, and tonsils. The gray, pinhead-size vesicles rupture to form larger ulcers covered by fibrin. Treatment is palliative because the symptoms are usually mild and lesions heal spontaneously after 3 to 5 days.

Hand-Foot-and-Mouth Disease. Caused by several coxsackie A and B viruses, hand-foot-and-mouth disease is a highly contagious viral infection predominantly affecting children less than 5 years of age.
Typically occurring during the spring and summer, the patient's chief complaint is usually pain from oral lesions. Oral lesions begin as vesicles that rupture to form ulcers. Lesions form anywhere in the mouth, but especially on the palate, tongue, and buccal mucosa. Also, multiple maculopapular lesions appear on the feet, toes, hands, and fingers soon after the oral lesions appear. The cutaneous vesicles ulcerate and crust. Treatment is palliative; lesions heal spontaneously within 2 weeks. Milk products are helpful in coating the ulcerative surfaces and reducing pain.

Candidiasis

Acute pseudomembranous candidiasis, or thrush, is the opportunistic overgrowth of the fungus *Candida albicans*. Candidiasis commonly occurs in infants after exposure to the microorganisms in the vaginal tract. In children, candidiasis may be a sequela of oral broad-spectrum antibiotics or may reflect other systemic alterations, such as immunodeficiency. Clinically, oral lesions are characterized by creamy or curdy white plaques that can be wiped off, leaving a red, raw, and painful surface. Any mucosal surface in the oral cavity can be affected; the buccal mucosa, tongue, and soft palate typically are involved. Treatment with a topical or systemic antifungal agent usually resolves the infection. Caution must be exercised with the use of antifungal elixirs because many of the products contain sucrose and carry the risk for dental caries.

HIV Infection

Children most commonly acquire HIV infection during pregnancy or at birth from an infected mother. Blood products, transfusion, and breast milk are other sources of pediatric HIV infection. The clinical oral manifestations of HIV infection are somewhat different in young children than in adults. The fungal disease most commonly seen in children with HIV is oral candidiasis, which may progress to esophageal candidiasis. Topical or systemic antifungal medications are used to treat candidiasis. Generalized or localized HIV gingivitis may occur in young children. HIV gingivitis is characterized by a linear erythema of the facial and interproximal gingival margins and is unresponsive to improved oral hygiene. HSV infection is the most common viral infection in children with HIV infection. Primary herpetic gingivostomatitis and chronic recurrent lesions may require hospitalization and the use of the antiviral drug acyclovir. Of unknown cause, parotid swelling is more common in children than in adults with HIV.

Hematologic Diseases

Leukemias

Oral manifestations commonly occur in patients with acute leukemia of the monocytic and myelogenous subtypes. Leukemic gingival
enlargement is characterized by a shiny, red, edematous, and boggy appearance, with extensive bleeding. Gingival enlargement occurs as malignant leukocytes infiltrate the gingival soft tissues. Spontaneous bleeding of the gingival sulcus eventually occurs. Oral complications of leukemia treatment include mucositis, which occurs as a result of chemotherapy (Fig. 9). Opportunistic infections of the oral cavity are primarily caused by *Candida* spp., especially in patients undergoing chemotherapy and antibiotic treatment. Oral viral infections include HSV, varicella-zoster virus, and cytomegalovirus. Gingival inflammation may result from poor oral hygiene. Spontaneous gingival bleeding may be caused by the underlying condition or treatment. Oral complications may be prevented or diminished in severity by developing preventive and treatment strategies, such as rinsing with chlorhexidine. Children

![Figure 9. A, Early mucositis presenting on the ventral surface of the tongue following induction chemotherapy for a bone marrow transplantation in a 5-year-old child. B, Severe mucositis during bone marrow transplantation in a 13-year-old.](image)
who have undergone bone marrow transplantation and developed chronic graft-versus-host disease must be monitored closely for the occurrence of gingival squamous cell carcinoma.¹²

**Ulcerations**

*Traumatic Ulcer*

The most common oral ulcer found in young children, traumatic ulcers are caused by mechanical, chemical, or thermal injuries to the oral tissues. Located on the peripheral borders of the tongue, buccal mucosa, lips, or palate, lesions vary in appearance depending on the source and intensity of the trauma. Burns on the anterior palate may occur after eating foods such as hot pizza or drinking liquids that are too hot. Infants and toddlers may develop traumatic ulcers on the soft palate from pacifier or digit sucking. Nervous fingernail scratching of the gingiva may result in a factitial injury (Fig. 10). In most cases, following removal of the source of the trauma, ulcers heal within 2 weeks.

Children with oral self-abusive conditions, such as Lesch-Nyhan syndrome, may intentionally bite their lips. They may require a more comprehensive treatment plan, including sedation, bite guards, lip bumpers, or extractions (Fig. 11).

*Recurrent Aphthous Ulceration*

Commonly known as *chancre sore*, recurrent aphthous ulceration is most common in young adults, but children also may be affected.

---

*Figure 10.* Gingival ulceration resulting from a nervous habit of gingival scratching in an 8-year-old child.
Characteristically appearing on vestibular and buccal mucosa, tongue, soft palate, fauces, and the floor of the mouth, minor aphthous ulcers are usually singular, painful, and less than 1 cm in diameter (Fig. 12). The cause is unknown but probably is an immunologic alteration. Local trauma, food allergies, stress, viral illness, or hormonal changes may trigger these recurrent lesions, which heal within 10 days. Treat-
ment, if needed, may include topical anesthetics; topical or systemic steroids; and antimicrobial rinses, such as chlorhexidine.

**PEDIATRIC PERIODONTAL PROBLEMS**

**Normal Findings**

The gingival tissues of children differ from those of adults in their clinical appearance and resistance to the development of gingivitis and periodontitis. The color of the gingiva in children may be more reddish because of an increased vascularity and a thinner, less keratinized, more translucent epithelium. The gingival surface may be smooth or slightly stippled, and the tissues have a firm, resilient consistency. The contour of the marginal gingiva demonstrates a rounded or "rolled" shape because of the cervical constriction of primary teeth. The width of the attached gingiva is wider in the maxilla than in the mandible; this width increases in the transition from the primary to the permanent dentition. As the permanent teeth erupt, the surrounding gingiva may appear inflamed as the gingiva readapts to the each new tooth. Radiographically, the alveolar bone surrounding primary teeth demonstrates fewer trabeculae, larger marrow spaces, and less calcification. The periodontal ligament is wider than in adults.

**Gingivitis**

Worldwide studies demonstrate that marginal gingivitis, the most common form of periodontal disease, begins in early childhood. Using differing indices to measure inflammation, investigators have found gingivitis in 2% to 34% of 2-year-old children and 18% to 38% of 3-year-old children. Gingivitis in young children is typically less severe than in adults with similar plaque levels. Despite generalized plaque on the teeth of preschool children, inflammation may be localized to the primary molars. Gingivitis rarely progresses to periodontitis in children.

Immunologic and microbiologic factors may explain the decreased tendency of young children's gingivitis to progress to periodontitis. Compared with adults, chronic gingivitis in children seems to stop at the early lesion stage, in which T lymphocytes histologically infiltrate the connective tissue. In adults, established gingivitis appears histologically rich in plasma cells and B lymphocytes. The colonization of children's teeth with periodontally important bacteria occurs as the primary teeth erupt. Nonhuman primate studies demonstrate mother-to-infant transmission of periodontopathogens during infancy, with predictable acquisition of aerobic bacteria, facultative anaerobes, then anaerobic species. A mature periodontal microbiota occurs soon after the complete eruption of the primary dentition. Development of antibodies to these bacteria follows their colonization as the body recognizes the pathogens.
Figure 13. A, A 20-year-old mother with severe periodontal disease and heavy calculus (tartar) build-up on all of her teeth. B, The 6-month-old infant of the mother in A with a localized periodontal infection between her top front teeth resulting from bacteria identical to her mother's periodontal pathogens.

Thus, clinical signs of the body’s response to colonization of periodontal plaque are delayed (Fig. 13).

The goal of recognizing and treating gingivitis at this age is to instill in young patients the importance of periodontal health. Although most children have little tendency toward progression of gingivitis to periodontitis during the primary dentition, establishment of excellent oral hygiene habits may carry over into adulthood, when the risk for disease progression is greater.

Periodontitis, Tooth Mobility, and Tooth Loss

Historically, clinicians and investigators rarely found periodontal disease in young children because of the trend of waiting to examine a child’s dentition until they were sufficiently old to cooperate in the dentist’s office. Only the dramatic loss of a primary tooth before age 6 years would have triggered an assessment for periodontal disease. With
the advent of more pediatric dentists and the focus on early detection and prevention of dental disease, more children with periodontitis are being identified before the irreversible loss of a primary tooth.

Periodontitis can be detected clinically in young children by checking tooth mobility. The presence of early tooth mobility is a clinical sign of loss of periodontal support. A late sign of advanced periodontal disease is the premature loss of a primary tooth. In most cases, early periodontitis in children is not obvious clinically, except by probing for attachment loss, checking for tooth mobility, and obtaining radiographs to assess the bone levels around the teeth. Gingival recession, gingival erythema, and gingival edema usually are not found, except in patients with neutrophil defects. Periodontal bone loss can be localized or generalized and may be horizontal or vertical.

The diagnosis of periodontal disease in a child suggests the need for further investigation into a possible underlying systemic condition. Although many patients are healthy except for subtle abnormalities in host defense, some patients may be diagnosed with a specific neutrophil defect, such as cyclic neutropenia or leukocyte adhesion deficiency disorder; Papillon-Lefèvre syndrome; metabolic disorders, such as diabetes or hypophosphatasia; or a neoplasia, such as Langerhans' cell histiocytosis X.

**Neutropenias**

Neutrophils comprise the body's major initial cellular defense responses to bacterial infection. Without the continuous protection of neutrophils within the gingival sulcus (i.e., "gum gutter"), pathogenic dental plaque can lead to rapid and severe periodontal breakdown.

**Congenital neutropenia** is characterized by a marked decrease in or lack of circulating neutrophils in young children with no prior history of drug intake. The neutropenia is persistent. Clinically, the child experiences severe, recurrent, and often fatal infections. Hospitalizations and antibiotic use are common. Oral ulcers, severe gingivitis, alveolar bone loss, gingival recession, tooth mobility, and early tooth loss are common oral signs of congenital neutropenia (Fig. 14). Current medical therapies include administration of granulocyte colony-stimulating factor.

**In autoimmune neutropenia of infancy** mild but recurrent infections develop in young children. These children produce antibodies against neutrophils, resulting in cell destruction. Recovery from the disease usually occurs before 4 years of age. Neutropenia is severe until then, but absolute neutrophil count may increase temporarily in response to acute infection. Unfortunately, periodontal diseases are chronic infections; these children do not mount a first-line neutrophil defense. Without preventive measures, severe gingivitis and periodontitis may result before neutrophil recovery.

**Cyclic neutropenia** is characterized by the disappearance of neutrophils from the blood and bone marrow at regular intervals of approxi-
mately 3 weeks. Serial blood studies are needed to confirm the diagnosis of cyclic neutropenia. During the neutropenic phase of each cycle, patients may have clinical symptoms of fever, malaise, chills, anorexia, and ulcers of the oral mucous membrane. Soft-tissue manifestations of cyclic neutropenia are severe gingival inflammation, edema, and recession. Alveolar bone support often is lost in preschool-aged children, resulting in premature tooth mobility and tooth loss (Fig. 15).

**Leukocyte adhesion deficiency disorder** begins intraorally with
the eruption of primary teeth. It is characterized by severe gingival inflammation, rapid bone loss around nearly all teeth, mobility, and tooth loss. Abnormalities in polymorphonuclear and mononuclear leukocyte adherence have been detected in children with generalized severe periodontitis. Because the cell-surface glycoprotein that mediates adherence of leukocytes to surfaces is abnormal, these cells cannot exit the blood vessels. Thus, these children are deprived of their primary defense against infection. Patients are subject to otitis media, skin infections, upper respiratory tract infections, and even life-threatening infections.

Treatment of periodontitis in children with neutropenia ideally begins with aggressive measures to prevent periodontal bone loss, which requires identification of affected children by physicians and early referral to a dentist. Although the condition predisposes children to gingivitis and periodontitis, progression of bone loss is caused by the body’s response to pathogenic subgingival plaque; therefore, scrupulous oral
hygiene measures, antimicrobial rinses, frequent professional teeth cleaning, and targeted antibiotic therapy can delay or halt periodontal bone loss. Highly motivated parents are the key to successful treatment of preschool children. Unfortunately, many children present to the dentist after severe periodontal destruction has occurred.

The antibiotic of choice for treating children with periodontitis is determined by a gingival sulcus microbial sampling. Typical bacteria found in the subgingival crevice of children with periodontitis include high levels of *Prevotella intermedia*, *Actinobacillus actinomycetemcomitans*, *Eikenella corrodens*, and *Capnocytophaga sputigena*. Eradication or control of these periodontopathogens with appropriate antibiotic therapy is important in the treatment of periodontitis. Frequent monitoring of results is important; periodic repetition of the antibiotic therapy may be necessary. Treatment is generally more successful in children with localized, rather than generalized, periodontitis, which may be refractory to antibiotic therapy without correction of the underlying neutrophil defect.

**Papillon-Lefèvre Syndrome**

Children with Papillon-Lefèvre syndrome have a condition characterized by hyperkeratosis of the palms and soles and premature loss of the primary and permanent dentitions. Hyperkeratotic lesions of the elbows and knees also may be found. Periodontal inflammation begins soon after the primary teeth erupt. Bone loss is severe, so that primary teeth are lost by 5 years of age. Historically, permanent teeth also were lost within a few years of eruption (Fig. 16). Patients with this syndrome have been diagnosed with abnormalities in neutrophil function. The neutrophil chemotactic or phagocytic response to bacteria may be depressed. Bacterial cultures from the gingival sulcus demonstrate periodontal pathogens, such as *A. actinomycetemcomitans*, *Capnocytophaga sp.*, *Fusobacterium nucleatum*, and *E. corrodens*. Attempts at conventional therapy with oral hygiene instruction, professional cleanings, frequent recalls, and antibiotics have failed to prevent tooth loss in patients with Papillon-Lefèvre syndrome; however, Tinanoff et al. successfully treated a 9-year-old patient by extracting all erupted permanent teeth and prescribing antibiotics, so that periodontal pathogens could be eradicated long enough to prevent infection of the patient's 16 unerupted teeth. Follow-up until the patient was 16 years old showed no further periodontal bone loss or tooth loss. Similar treatment of the primary dentition in a 4-year-old child was also successful. The preschooler was edentulous for 9 months before eruption of his first permanent tooth. Periodontitis of the permanent dentition was thus prevented by combined antibiotics and early extraction of primary teeth. Recommended periodontal treatment of young children with Papillon-Lefèvre syndrome thus includes identification of specific periodontal pathogens, antibiotic therapy appropriate to these microorganisms, and potentially dental extractions for severely periodontally com-
promised teeth. Kressin et al.8 have had success with the use of retinoids in conjunction with comprehensive periodontal therapy.

**Langerhans’ Cell Histiocytosis X**

Langerhans' cell histiocytosis X affects infants, children, and young adults. In patients with this disease, granulomatous lesions develop, with histiocytic proliferation.9 The clinical manifestations of this disease range from solitary or multiple bone lesions to disseminated visceral, skin, and bone lesions. The skull, mandible, ribs, vertebrae, and long bones commonly are involved. Lesions in the oral cavity may be the first presentation of the disease as pain, swelling, or premature eruption of the teeth in the area of affected alveolar bone occurs (Fig. 17). The gingival tissues adjacent to the affected bone may be inflamed, and root surfaces may be exposed. Radiographically, teeth appear to be “floating
in space” because of the radiolucent alveolar bone lesion. Common sites for alveolar bone lesions and premature eruption of primary teeth are in the molar regions. A gingival biopsy from the affected area can lead to a definitive diagnosis. Current treatment of disseminated forms of this disease in young children includes the use of chemotherapeutic agents, surgical curettage, or low-dose radiation therapy.

Metabolic Disorders

*Insulin-Dependent Diabetes*

Children with poorly controlled diabetes may be susceptible to periodontitis because these children exhibit altered neutrophil chemotaxis. Diabetic children in poor control display an increased plaque index and more severe gingivitis than in healthy children. Successful
treatment includes excellent oral hygiene and control of blood glucose levels.

**Hypophosphatasia**

Hypophosphatasia is a rare genetic metabolic disease characterized by abnormal mineralization of bone and dental tissues. Approximately 75% of children with hypophosphatasia experience premature exfoliation of primary teeth at as early as 1.5 years of age. Loss of primary incisors, without local inflammation, is commonly the first clinical symptom of the disease. The symptoms can range from mild, with only dental findings, to severe. Bone pain with spontaneous fractures and vitamin D-resistant rickets-like bone lesions also are found in children who are affected more severely. Patients usually have low serum levels of alkaline phosphatase and increased urine excretion of phosphoethanolamine. Dentally, partial or complete absence of the cementum covering the root structure occurs. Consequently, the fibers of the periodontal ligament are not anchored normally; thus, this lack of periodontal attachment leads to premature tooth mobility and finally tooth loss. No successful treatment is known; however, the mild form of the disease can be self-limiting and affect only the primary teeth, leaving the permanent dentition intact.

**SUMMARY**

Pediatric patients can present with various intraoral lesions that require accurate diagnosis, treatment or reassurance, and possible referral for a dental evaluation. Periodic review of oral soft-tissue pathology can help the medical team to easily recognize common and rare abnormalities affecting children. Recent years have brought new insights into the causes and treatment of periodontal diseases of children, making prevention or treatment of many formerly untreatable conditions possible. Early detection of these oral conditions may be life saving.

**References**


Address reprint requests to
Martha Ann Keels, DDS, PhD
Duke Children’s Hospital
Duke University Medical Center
Box 3120
Durham, NC 27710

e-mail: keels001@mc.duke.edu