29 - 38 Oral Manifestations of Disease

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Noninfectious causes of laryngitis include vocal trauma (e.g., due to yelling, screaming, or loud singing), inhalation injuries, allergies, gas troesophageal reflux disease (laryngopharyngeal reflux), asthma, and pollution. Immunosuppressed patients are at risk for infections with herpesvirus, HIV, and coxsackievirus. Smokers are at elevated risk for malignancy and other infections.

Laryngitis is characterized by a raspy, hoarse, or breathy voice, sometimes progressing to a complete loss of voice. Laryngitis can have associated dry cough and anterior throat pain; patients often feel a need to clear their throats. The physical examination in patients who may have laryngitis should focus on the head, neck, and lungs, but the diagnosis of laryngitis is generally based on history. If visualization of the vocal cords is necessary, indirect examination with a mirror or flexible laryngoscopy usually shows erythema and edema of the vocal cords and surrounding structures. PART 2 Cardinal Manifestations and Presentation of Diseases TREATMENT Laryngitis Laryngitis is generally self-limited, usually lasting 3–7 days, but may last up to 14 days. Vocal rest is crucial. Airway humidification and hydration should help. Patients likely to have laryngopharyngeal reflux should avoid gastroesophageal reflux–inducing foods and behaviors and should take antireflux medications. In randomized controlled trials, antibiotics were not effective in decreasing objec tive symptoms of laryngitis. Red flags for emergency evaluation and monitoring include short ness of breath, stridor, dysphagia, odynophagia, drooling, and pos turing that could indicate epiglottitis. Referral to an otolaryngologist should be considered for patients who rely on their voice for work, such as singers and teachers. A history of smoking or weight loss should raise suspicion of malignancy. Symptoms lasting >3 weeks should prompt referral to an otolaryngologist or speech specialist. ■ ■FURTHER READING Centor RM, Linder JA: Web exclusive. Annals on call—Fusobacterium pharyngitis debate. Ann Intern Med 171:OC1, 2019. Chua KP et al: Appropriateness of outpatient antibiotic prescribing among privately insured US patients: ICD-10-CM based cross sec tional study. BMJ 364:k5092, 2019. Lieberthal AS et al: Clinical practice guideline: The diagnosis and management of acute otitis media. Pediatrics 131:e964, 2013. Rowe TA, Linder JA: Novel approaches to decrease inappropriate ambulatory antibiotic use. Expert Rev Anti Infect Ther 17:511, 2019. Sanchez GV et al: Antibiotic stewardship in outpatient telemedicine: Adapting Centers for Disease Control and Prevention core elements to optimize antibiotic use. Telemed J E Health 30:951, 2024. Samuel C. Durso

Oral Manifestations

of Disease Internists are often asked to evaluate patients with disease of the oral soft tissues, teeth, and pharynx. Knowledge of the oral milieu and its unique structures is necessary to guide preventive services and rec ognize oral manifestations of local or systemic disease (Chap. A3). Furthermore, internists frequently collaborate with dentists in the care of patients who have a variety of medical conditions that affect oral

health or who undergo dental procedures that increase their risk of medical complications. ■ ■DISEASES OF THE TEETH AND PERIODONTAL STRUCTURES Tooth formation begins during the sixth week of embryonic life and continues through 17 years of age. Teeth start to develop in utero and continue to develop until after the tooth erupts. Normally, all 20 deciduous teeth have erupted by age 3 and have been shed by age 13. Permanent teeth, eventually totaling 32, begin to erupt by age 6 and have completely erupted by age 14, though third molars (“wisdom teeth”) may erupt later. The erupted tooth consists of the visible crown covered with enamel and the root submerged below the gum line and covered with bonelike cementum. Dentin, a material that is denser than bone and exquisitely sensitive to pain, forms the majority of the tooth substance, surround ing a core of myxomatous pulp containing the vascular and nerve supply. The tooth is held firmly in the alveolar socket by the peri odontium, supporting structures that consist of the gingivae, alveolar bone, cementum, and periodontal ligament. The periodontal ligament tenaciously binds the tooth’s cementum to the alveolar bone. Above this ligament is a collar of attached gingiva just below the crown. A few millimeters of unattached or free gingiva (1–3 mm) overlap the base of the crown, forming a shallow sulcus along the gum-tooth margin. Dental Caries, Pulpal and Periapical Disease, and Complica tions Dental caries usually begin asymptomatically as a destructive infectious process of the enamel. Bacteria—principally Streptococ cus mutans—colonize the organic buffering biofilm (plaque) on the tooth surface. If not removed by brushing or by the natural cleansing and antibacterial action of saliva, bacterial acids can demineralize the enamel. Fissures and pits on the occlusal surfaces are the most frequent sites of early decay. Surfaces between the teeth, adjacent to tooth restorations and exposed roots, are also vulnerable, particularly as individuals age. Over time, dental caries extend to the underlying dentin, leading to cavitation of the enamel. Without management, the caries will penetrate to the tooth pulp, producing acute pulpitis. At this stage, when the pulp infection is limited, the tooth may become sensi tive to percussion and to hot or cold, and pain resolves immediately when the irritating stimulus is removed. Should the infection spread throughout the pulp, irreversible pulpitis occurs, leading to pulp necro sis. At this later stage, pain can be severe and has a sharp or throbbing visceral quality that may be worse when the patient lies down. Once pulp necrosis is complete, pain may be constant or intermittent, but cold sensitivity is lost. Treatment of caries involves removing the softened and infected hard tissue and restoration of the tooth structure with silver amalgam, glass ionomer, composite resin, or gold. Once irreversible pulpitis occurs, root canal therapy becomes necessary; removal of the contents of the pulp chamber and root canal is followed by thorough clean ing and filling with an inert material. Alternatively, the tooth may be extracted. Pulpal infection leads to periapical abscess formation, which can produce pain on chewing. If the infection is mild and chronic, a peri apical granuloma or eventually a periapical cyst forms, either of which produces radiolucency at the root apex. When unchecked, a periapical abscess can erode into the alveolar bone, producing osteomyelitis; penetrate and drain through the gingivae, producing a parulis (gum boil); or track along deep fascial planes, producing virulent cellulitis (Ludwig’s angina) involving the submandibular space and floor of the mouth (Chap. 182). Elderly patients, patients with diabetes mellitus, and patients taking glucocorticoids may experience little or no pain or fever as these complications develop.

Periodontal Disease Periodontal disease and dental caries are the primary causes of tooth loss. Like dental caries, chronic infection of the gingiva and anchoring structures of the tooth begins with formation of bacterial plaque. The process begins at the gum line. Plaque and calcu lus (calcified plaque) are preventable by appropriate daily oral hygiene, including periodic professional cleaning. Left undisturbed, chronic

inflammation can ensue and produce hyperemia of the free and attached gingivae (gingivitis), which then typically bleed with brush ing. If this issue is ignored, severe periodontitis can develop, leading to deepening of the physiologic sulcus and destruction of the periodontal ligament. Gingival pockets develop around the teeth. As the periodon tium (including the supporting bone) is destroyed, the teeth loosen. A role for chronic inflammation due to chronic periodontal disease in promoting coronary heart disease and stroke has been proposed. Epidemiologic studies have demonstrated a moderate but significant association between chronic periodontal inflammation and atherogen esis, though a causal role remains unproven. Acute and aggressive forms of periodontal disease are less common than the chronic forms described above. However, if the host is stressed or exposed to a new pathogen, rapidly progressive and destructive disease of the periodontal tissue can occur. A virulent example is acute necrotizing ulcerative gingivitis. The presentation includes sud den gingival inflammation, ulceration, bleeding, interdental gingival necrosis, and fetid halitosis. Localized juvenile periodontitis, which is seen in adolescents, is particularly destructive and appears to be asso ciated with impaired neutrophil chemotaxis. AIDS-related periodontitis resembles acute necrotizing ulcerative gingivitis in some patients and a more destructive form of adult chronic periodontitis in others. It may also produce a gangrene-like destructive process of the oral soft tissues and bone that resembles noma, an infectious condition seen in severely malnourished children in developing nations. Prevention of Tooth Decay and Periodontal Infection Despite the reduced prevalences of dental caries and periodontal disease in the United States (due in large part to water fluoridation and improved dental care, respectively), both diseases constitute a major public health problem worldwide. The internist should promote preventive dental care and hygiene as part of health maintenance. Populations at high risk for dental caries and periodontal disease include those with hyposalivation and/or xerostomia, diabetics, alcoholics, tobacco users, persons with Down syndrome, and those with gingival hyperplasia. Furthermore, patients lacking access to dental care (e.g., due to low socioeconomic status) and patients with a reduced ability to provide self-care (e.g., individuals with disabilities, nursing home residents, and persons with dementia or upper-extremity disability) suffer dispropor tionately. It is important to provide counseling (to patients and/or their caregivers) regarding regular dental hygiene and professional cleaning, use of fluoride-containing toothpaste, professional fluoride treatments, and (for patients with limited dexterity) use of electric toothbrushes. Cost, fear of dental care, and differences in language and culture cre ate barriers that prevent some people from seeking preventive dental services. Developmental and Systemic Disease Affecting the Teeth and Periodontium In addition to posing cosmetic issues, maloc clusion, the most common developmental oral problem, can interfere with mastication unless corrected through orthodontic and surgical techniques. Impacted third molars are common and can become infected or erupt into an insufficient space. Acquired prognathism due to acromegaly may also lead to malocclusion, as may deformity of the maxilla and mandible due to Paget’s disease of the bone. Delayed tooth eruption, a receding chin, and a protruding tongue are occasional features of cretinism and hypopituitarism. Congenital syphilis produces tapering, notched (Hutchinson’s) incisors and finely nodular (mulberry) molar crowns. Enamel hypoplasia results in crown defects

ranging from pits to deep fissures of primary or permanent teeth. Intrauterine infection (syphilis, rubella), vitamin deficiency (A, C, or D), disorders of calcium metabolism (malabsorption, vitamin D–resistant rickets, hypoparathyroidism), prematurity, high fever, and rare inherited defects (amelogenesis imperfecta) are all causes. Tetracycline, given in sufficiently high doses during the first 8 years of life, may produce enamel hypoplasia and discoloration. Doxycycline does not cause permanent tooth staining in children despite warnings included for all tetracycline-class antibiotics. Worn enamel is seen with age, bruxism, or excessive acid exposure (e.g., chronic gastric reflux or bulimia).

Celiac disease is associated with nonspecific enamel defects in children but not in adults.

Total or partial tooth loss resulting from periodontitis is seen with cyclic neutropenia, Papillon-Lefévre syndrome, Chédiak-Higashi syn drome, and leukemia. Rapid focal tooth loosening is most often due to infection, but rarer causes include Langerhans cell histiocytosis, Ewing's sarcoma, osteosarcoma, and Burkitt's lymphoma. Early loss of primary teeth is a feature of hypophosphatasia, a rare congenital error of metabolism. Pregnancy may produce gingivitis and localized pyogenic granu lomas. Severe periodontal disease occurs in uncontrolled diabetes mellitus. Drug-induced gingival overgrowth may be caused by anti convulsants, calcium channel blockers, and immunosuppressants, although excellent daily oral care can prevent or reduce its occurrence. Idiopathic familial gingival fibromatosis and several syndrome-related disorders cause similar conditions. Discontinuation of the medication may reverse the drug-induced form, although surgery may be needed to control both of the latter entities. Linear gingival erythema is variably seen in patients with advanced HIV infection and probably represents immune deficiency and decreased neutrophil activity. Diffuse or focal gingival swelling may be a feature of early or late acute myelomono cytic leukemia as well as of other lymphoproliferative disorders. A rare but pathognomonic sign of granulomatosis with polyangiitis is a redpurplish, granular gingivitis (strawberry gums). Oral Manifestations of Disease CHAPTER 38 ■ ■DISEASES OF THE ORAL MUCOSA Infections Most oral mucosal diseases involve microorganisms (Table 38-1). Pigmented Lesions See Table 38-2. Dermatologic Diseases See Tables 38-1, 38-2, and 38-3 and Chaps. 59–64. Diseases of the Tongue See Table 38-4. HIV Disease and AIDS See Tables 38-1, 38-2, 38-3, and 38-5; Chap. 208. Ulcers Ulceration is the most common oral mucosal lesion. Although there are many causes, the host and the pattern of lesions, including the presence of organ system features, narrow the differential diagnosis (Table 38-1). Most acute ulcers are painful and self-limited. Recurrent aphthous ulcers and herpes simplex account for the majority. Persistent and deep aphthous ulcers can be idiopathic or can accompany HIV/ AIDS. Aphthous lesions are often the presenting symptom in Behçet's syndrome (Chap. 376). Similar-appearing, though less painful, lesions may occur in reactive arthritis, and aphthous ulcers are occasionally present during phases of discoid or systemic lupus erythematosus (Chap. 372). Aphthous-like ulcers are seen in Crohn's disease (Chap. 337), but, unlike the common aphthous variety, they may exhibit granuloma tous inflammation on histologic examination. Recurrent aphthae are more prevalent in patients with celiac disease and have been reported to remit with elimination of gluten. Of major concern are chronic, relatively painless ulcers and mixed red/white patches (erythroplakia and leukoplakia) of >2 weeks' dura tion. Squamous cell carcinoma and premalignant dysplasia should be considered early and a diagnostic biopsy performed. This awareness and this procedure are critically important because early-stage malig nancy is vastly more treatable than late-stage disease. High-risk sites include the lower lip, floor of the mouth, ventral and lateral tongue, and soft palate–tonsillar pillar

complex. Significant risk factors for oral cancer in Western countries include sun exposure (lower lip), tobacco and alcohol use, and human papillomavirus infection. In India and some other Asian countries, smokeless tobacco mixed with betel nut, slaked lime, and spices is a common cause of oral cancer. Rarer causes of chronic oral ulcer, such as tuberculosis, fungal infection, granuloma tosis with polyangiitis, and midline granuloma, may look identical to carcinoma. Making the correct diagnosis depends on recognizing other clinical features and performing a biopsy of the lesion. The syphilitic

TABLE 38-1 Vesicular, Bullous, or Ulcerative Lesions of the Oral Mucosa CONDITION USUAL LOCATION CLINICAL FEATURES COURSE Viral Diseases Primary acute herpetic gingivostomatitis (HSV type 1; rarely type 2) Lip and oral mucosa (buccal, gingival, lingual mucosa) Labial vesicles that rupture and crust, and intraoral vesicles that quickly ulcerate; extremely painful; acute gingivitis, fever, malaise, foul odor, and cervical lymphadenopathy; occurs primarily in infants, children, and young adults Recurrent herpes labialis Mucocutaneous junction of lip, perioral skin Eruption of groups of vesicles that may coalesce, then rupture and crust; painful to pressure or spicy foods Recurrent intraoral herpes simplex Palate and gingiva Small vesicles on keratinized epithelium that rupture and coalesce; painful PART 2 Cardinal Manifestations and Presentation of Diseases Chickenpox (VZV) Gingiva and oral mucosa Skin lesions may be accompanied by small vesicles on oral mucosa that rupture to form shallow ulcers; may coalesce to form large bullous lesions that ulcerate; mucosa may have generalized erythema Herpes zoster (VZV reactivation) Cheek, tongue, gingiva, or palate Unilateral vesicular eruptions and ulceration in linear pattern following sensory distribution of trigeminal nerve or one of its branches Infectious mononucleosis (Epstein-Barr virus) Oral mucosa Fatigue, sore throat, malaise, fever, and cervical lymphadenopathy; numerous small ulcers usually appear several days before lymphadenopathy; gingival bleeding and multiple petechiae at junction of hard and soft palates Herpangina (coxsackievirus A; also possibly coxsackievirus B and echovirus) Oral mucosa, pharynx, tongue Sudden onset of fever, sore throat, and oropharyngeal vesicles, usually in children <4 years old, during summer months; diffuse pharyngeal congestion and vesicles (1–2 mm), grayish-white surrounded by red areola; vesicles enlarge and ulcerate Hand-foot-and-mouth disease (most commonly coxsackievirus A16) Oral mucosa, pharynx, palms, and soles Fever, malaise, headache with oropharyngeal vesicles that become painful, shallow ulcers; highly infectious; usually affects children under age 10 Primary HIV infection Gingiva, palate, and pharynx Acute gingivitis and oropharyngeal ulceration, associated with febrile illness resembling mononucleosis and including lymphadenopathy Bacterial or Fungal Diseases Acute necrotizing ulcerative gingivitis (“trench mouth”) Gingiva Painful, bleeding gingiva characterized by necrosis and ulceration of gingival papillae and margins plus lymphadenopathy and foul breath Prenatal (congenital) syphilis Palate, jaws, tongue, and teeth Gummatous involvement of palate, jaws, and facial bones; Hutchinson’s incisors, mulberry molars, glossitis, mucous patches, and fissures at corner of mouth Primary syphilis (chancre) Lesion appearing where organism enters body; may occur on lips, tongue, or tonsillar area Small papule developing rapidly into a large, painless ulcer with indurated border; unilateral lymphadenopathy; chancre and lymph nodes containing spirochetes; serologic tests positive by third to fourth weeks Secondary syphilis Oral mucosa frequently involved with mucous patches, which occur primarily on palate and also at commissures of mouth Maculopapular lesions of oral mucosa, 5–10 mm in diameter with central ulceration covered by grayish membrane; eruptions occurring on various mucosal surfaces and skin, accompanied by fever, malaise, and sore throat Tertiary syphilis Palate and tongue Gummatous infiltration of palate or tongue followed by

ulceration and fibrosis; atrophy of tongue papillae produces characteristic bald tongue and glossitis Gonorrhea Lesions may occur in mouth at site of inoculation or secondarily by hematogenous spread from a primary focus Most pharyngeal infection is asymptomatic; may produce burning or itching sensation; oropharynx and tonsils may be ulcerated and erythematous; saliva viscous and fetid Tuberculosis Tongue, tonsillar area, soft palate Painless, solitary, 1- to 5-cm, irregular ulcer covered with persistent exudate; ulcer has firm undermined border Cervicofacial actinomycosis Swellings in region of face, neck, and floor of mouth Infection may be associated with extraction, jaw fracture, or eruption of molar tooth; in acute form, resembles acute pyogenic abscess, but contains yellow “sulfur granules” (gram-positive mycelia and their hyphae)

Heals spontaneously in 10–14 days; unless secondarily infected, lesions lasting >3 weeks are not due to primary HSV infection Lasts ~1 week, but condition may be prolonged if secondarily infected; if severe, topical or oral antiviral treatment may reduce healing time Heals spontaneously in ~1 week; if severe, topical or oral antiviral treatment may reduce healing time Lesions heal spontaneously within 2 weeks Gradual healing without scarring unless secondarily infected; postherpetic neuralgia is common; oral acyclovir, famciclovir, or valacyclovir reduces healing time and postherpetic neuralgia Oral lesions disappear during convalescence; no treatment is given, though glucocorticoids are indicated if tonsillar swelling compromises the airway Incubation period of 2–9 days; fever for

1–4 days; recovery uneventful Incubation period 2–18 days; lesions heal spontaneously in 2–4 weeks Followed by HIV seroconversion, asymptomatic HIV infection, and usually ultimately by HIV disease Debridement and diluted (1:3) peroxide lavage provide relief within 24 h; antibiotics in acutely ill patients; relapse may occur Tooth deformities in permanent dentition irreversible Healing of chancre in 1–2 months, followed by secondary syphilis in 6–8 weeks Lesions may persist from several weeks to a year Gumma may destroy palate, causing complete perforation More difficult to eradicate than urogenital infection, though pharyngitis usually resolves with appropriate antimicrobial treatment Autoinoculation from pulmonary infection is usual; lesions resolve with appropriate antimicrobial therapy Typically, swelling is hard and grows painlessly; multiple abscesses with draining tracts develop; penicillin first choice; surgery usually necessary (Continued)

TABLE 38-1 Vesicular, Bullous, or Ulcerative Lesions of the Oral Mucosa CONDITION USUAL LOCATION CLINICAL FEATURES COURSE Bacterial or Fungal Diseases (Continued) Histoplasmosis Any area of the mouth, particularly tongue, gingiva, or palate Nodular, verrucous, or granulomatous lesions; ulcers are indurated and painful; usual source hematogenous or pulmonary, but may be primary Candidiasisa Dermatologic Diseases Mucous membrane pemphigoid Typically produces marked gingival erythema and ulceration; other areas of oral cavity, esophagus, and vagina may be affected Painful, grayish-white collapsed vesicles or bullae of fullthickness epithelium with peripheral erythematous zone; gingival lesions desquamate, leaving ulcerated area EM minor and EM major (Stevens-Johnson syndrome) Primarily oral mucosa and skin of hands and feet Intraoral ruptured bullae surrounded by inflammatory area; lips may show hemorrhagic crusts; “iris” or “target” lesion on skin is pathognomonic; patient may have severe signs of toxicity Pemphigus vulgaris Oral mucosa and skin; sites of mechanical trauma (soft/hard palate, frenulum, lips, buccal mucosa) Usually (>70%) presents with oral lesions; fragile, ruptured bullae and ulcerated oral areas; mostly in older adults Lichen planus Oral mucosa and skin

White striae in mouth; purplish nodules on skin at sites of friction; occasionally causes oral mucosal ulcers and erosive gingivitis Other Conditions Recurrent aphthous ulcers Usually on nonkeratinized oral mucosa (buccal and labial mucosa, floor of mouth, soft palate, lateral and ventral tongue) Single or clustered painful ulcers with surrounding erythematous border; lesions may be 1–2 mm in diameter in crops (herpetiform), 1–5 mm (minor), or 5–15 mm (major) Behçet's syndrome Oral mucosa, eyes, genitalia, gut, and CNS Multiple aphthous ulcers in mouth; inflammatory ocular changes, ulcerative lesions on genitalia; inflammatory bowel disease and CNS disease Traumatic ulcers Anywhere on oral mucosa; dentures frequently responsible for ulcers in vestibule Localized, discrete ulcerated lesions with red border; produced by accidental biting of mucosa, penetration by foreign object, or chronic irritation by dentures Squamous cell carcinoma Any area of mouth, most commonly on lower lip, lateral borders of tongue, and floor of mouth Red, white, or red and white ulcer with elevated or indurated border; failure to heal; pain not prominent in early lesions Acute myeloid leukemia (usually monocytic) Gingiva Gingival swelling and superficial ulceration followed by hyperplasia of gingiva with extensive necrosis and hemorrhage; deep ulcers may occur elsewhere on mucosa, complicated by secondary infection Lymphoma Gingiva, tongue, palate, and tonsillar area Elevated, ulcerated area that may proliferate rapidly, giving appearance of traumatic inflammation Chemical or thermal burns Any area in mouth White slough due to contact with corrosive agents (e.g., aspirin, hot cheese) applied locally; removal of slough leaves raw, painful surface aSee Table 38-3. Abbreviations: CNS, central nervous system; EM, erythema multiforme; HSV, herpes simplex virus; VZV, varicella-zoster virus. chancre is typically painless and therefore easily missed. Regional lymphadenopathy is invariably present. Disorders of mucosal fragility often produce painful oral ulcers that fail to heal within 2 weeks. Mucous membrane pemphigoid and pemphigus vulgaris are the major acquired disorders. While their clinical features are often distinctive, a biopsy or immunohistochemi cal examination should be performed to diagnose these entities and to distinguish them from lichen planus and drug reactions. Hematologic and Nutritional Disease Internists are more likely to encounter patients with acquired, rather than congenital, bleeding

(Continued) Systemic antifungal therapy necessary Protracted course with remissions and exacerbations; involvement of different sites develops slowly; glucocorticoids may temporarily reduce symptoms but do not control disease Oral Manifestations of Disease CHAPTER 38 Onset very rapid; usually idiopathic, but may be associated with trigger such as drug reaction; condition may last 3–6 weeks; mortality rate for untreated EM major is 5–15% With repeated occurrence of bullae, toxicity may lead to cachexia, infection, and death within 2 years; often controllable with oral glucocorticoids White striae alone usually asymptomatic; erosive lesions often difficult to treat, but may respond to glucocorticoids Lesions heal in 1–2 weeks but may recur monthly or several times a year; protective barrier with benzocaine and topical glucocorticoids relieve symptoms; systemic glucocorticoids may be needed in severe cases Oral lesions often first manifestation; persist several weeks and heal without scarring Lesions usually heal in 7–10 days when irritant is removed, unless secondarily infected Invades and destroys underlying tissues; frequently metastasizes to regional lymph nodes Usually responds to systemic treatment of leukemia; occasionally requires local irradiation Fatal if untreated; may indicate underlying HIV infection Lesion heals in several weeks if not secondarily infected disorders. Bleeding should stop 15 min after minor trauma and within an hour after tooth extraction if local pressure is applied. More pro longed bleeding, if not due to continued injury or rupture of a large vessel, should lead to investigation for a clotting abnormality. In addi tion to bleeding, petechiae and ecchymoses are

prone to occur at the vibrating line between the soft and hard palates in patients with platelet dysfunction or thrombocytopenia. All forms of leukemia, but particularly acute myelomonocytic leuke mia, can produce gingival bleeding, ulcers, and gingival enlargement. Oral ulcers are a feature of agranulocytosis, and ulcers and mucositis are often severe complications of chemotherapy and radiation therapy

TABLE 38-2 Pigmented Lesions of the Oral Mucosa CONDITION USUAL LOCATION CLINICAL FEATURES COURSE Oral melanotic macule Any area of mouth Discrete or diffuse, localized, brown to black macule Remains indefinitely; no growth Diffuse melanin pigmentation Any area of mouth Diffuse pale to dark-brown pigmentation; may be physiologic (“racial”) or due to smoking Nevi Any area of mouth Discrete, localized, brown to black pigmentation Remains indefinitely Malignant melanoma Any area of mouth Can be flat and diffuse, painless, brown to black; or can be raised and nodular Addison’s disease Any area of mouth, but mostly buccal mucosa Blotches or spots of bluish-black to dark-brown pigmentation occurring early in disease, accompanied by diffuse pigmentation of skin; other symptoms of adrenal insufficiency PART 2 Cardinal Manifestations and Presentation of Diseases Peutz-Jeghers syndrome Any area of mouth Dark-brown spots on lips, buccal mucosa, with characteristic distribution of pigment around lips, nose, and eyes and on hands; concomitant intestinal polyposis Drug ingestion (neuroleptics, oral contraceptives, minocycline, zidovudine, quinine derivatives) Any area of mouth Brown, black, or gray areas of pigmentation Gradually disappears following cessation of drug intake Amalgam tattoo Gingiva and alveolar mucosa Small blue-black pigmented areas associated with embedded amalgam particles in soft tissues; may show up on radiographs as radiopaque particles in some cases Heavy metal pigmentation (bismuth, mercury, lead) Gingival margin Thin blue-black pigmented line along gingival margin; rarely seen except in children exposed to lead-based paint Black hairy tongue Dorsum of tongue Elongation of filiform papillae of tongue, which become stained by coffee, tea, tobacco, or pigmented bacteria Fordyce spots Buccal and labial mucosa Numerous small yellowish spots just beneath mucosal surface; no symptoms; due to hyperplasia of sebaceous glands Kaposi’s sarcoma Palate most common, but may occur at any other site Red or blue plaques of variable size and shape; often enlarge, become nodular, and may ulcerate Mucous retention cysts Buccal and labial mucosa Bluish, clear fluid–filled cyst due to extravasated mucus from injured minor salivary gland TABLE 38-3 White Lesions of Oral Mucosa CONDITION USUAL LOCATION CLINICAL FEATURES COURSE Lichen planus Buccal mucosa, tongue, gingiva, and lips; skin Striae, white plaques, red areas, ulcers in mouth; purplish papules on skin; may be asymptomatic, sore, or painful; lichenoid drug reactions may look similar White sponge nevus Oral mucosa, vagina, anal mucosa Painless white thickening of epithelium; adolescence/early adulthood onset; familial Smoker’s leukoplakia and smokeless tobacco lesions Any area of oral mucosa, sometimes related to location of habit White patch that may become firm, rough, or red-fissured and ulcerated; may become sore and painful but is usually painless Erythroplakia with or without white patches Floor of mouth commonly affected in men; tongue and buccal mucosa in women Velvety, reddish plaque; occasionally mixed with white patches or smooth red areas Candidiasis Any area in mouth Pseudomembranous type (“thrush”): creamy white curdlike patches that reveal a raw, bleeding surface when scraped; found in sick infants, debilitated elderly patients receiving high-dose glucocorticoids or broad-spectrum antibiotics, and patients with AIDS Erythematous type: flat, red, sometimes sore areas in same groups of patients Candidal leukoplakia: nonremovable white thickening of epithelium due to Candida Angular cheilitis: sore fissures at corner of mouth Responds to topical antifungal therapy Hairy leukoplakia Usually on lateral tongue, rarely

elsewhere on oral mucosa White areas ranging from small and flat to extensive accentuation of vertical folds; found in HIV carriers (all risk groups for AIDS) Warts (human papillomavirus) Anywhere on skin and oral mucosa Single or multiple papillary lesions with thick, white, keratinized surfaces containing many pointed projections; cauliflower lesions covered with normal-colored mucosa or multiple pink or pale bumps (focal epithelial hyperplasia)

Remains indefinitely Expands and invades early; metastasis leads to death Condition controlled by adrenal steroid replacement Oral pigmented lesions remain indefinitely; gastrointestinal polyps may become malignant Remains indefinitely Indicative of systemic absorption; no significance for oral health Improves within 1–2 weeks with gentle brushing of tongue or (if due to bacterial overgrowth) discontinuation of antibiotic Benign; remains without apparent change Usually indicative of HIV infection or nonHodgkin's lymphoma; rarely fatal, but may require treatment for comfort or cosmesis Benign; painless unless traumatized; may be removed surgically Protracted; responds to topical glucocorticoids Benign and permanent May or may not resolve with cessation of habit; 2% of patients develop squamous cell carcinoma; early biopsy essential High risk of squamous cell cancer; early biopsy essential Responds favorably to antifungal therapy and correction of predisposing causes where possible Course same as for pseudomembranous type Responds to prolonged antifungal therapy Due to Epstein-Barr virus; responds to high-dose acyclovir but recurs; rarely causes discomfort unless secondarily infected with Candida Lesions grow rapidly and spread; squamous cell carcinoma must be ruled out with biopsy; excision or laser therapy; may regress in HIV-infected patients receiving antiretroviral therapy

TABLE 38-4 Alterations of the Tongue TYPE OF CHANGE CLINICAL FEATURES Size or Morphology Macroglossia Enlarged tongue that may be part of a syndrome found in developmental conditions such as Down syndrome, Simpson-Golabi-Behmel syndrome, or Beckwith-Wiedemann syndrome; may be due to tumor (hemangioma or lymphangioma), metabolic disease (e.g., primary amyloidosis), or endocrine disturbance (e.g., acromegaly or cretinism); may occur when all teeth are removed Fissured ("scrotal") tongue Dorsal surface and sides of tongue covered by painless shallow or deep fissures that may collect debris and become irritated Median rhomboid glossitis Congenital abnormality with ovoid, denuded area in median posterior portion of tongue; may be associated with candidiasis and may respond to antifungal treatment Color "Geographic" tongue (benign migratory glossitis) Asymptomatic inflammatory condition of tongue, with rapid loss and regrowth of filiform papillae leading to appearance of denuded red patches "wandering" across surface Hairy tongue Elongation of filiform papillae of medial dorsal surface area due to failure of keratin layer of papillae to desquamate normally; brownish-black coloration may be due to staining by tobacco, food, or chromogenic organisms "Strawberry" and "raspberry" tongue Appearance of tongue during scarlet fever due to hypertrophy of fungiform papillae as well as changes in filiform papillae "Bald" tongue Atrophy may be associated with xerostomia, pernicious anemia, iron-deficiency anemia, pellagra, or syphilis; may be accompanied by painful burning sensation; may be an expression of erythematous candidiasis and respond to antifungal treatment for hematologic and other malignancies. Plummer-Vinson syndrome (iron deficiency, angular stomatitis, glossitis, and dysphagia) raises the risk of oral squamous cell cancer and esophageal cancer at the postcricoidal tissue web. Atrophic papillae and a red, burning tongue may occur with pernicious anemia. Deficiencies in B-group vitamins produce many of these same symptoms, as well as oral ulceration and cheilosis. Consequences of scurvy include swollen, bleeding gums; ulcers; and loosening of the teeth. NONDENTAL CAUSES OF ORAL PAIN Most, but not all, oral pain emanates

from inflamed or injured tooth pulp or periodontal tissues. Nonodontogenic causes are often over looked. In most instances, toothache is predictable and proportional to the stimulus applied, and an identifiable condition (e.g., caries, abscess) is found. Local anesthesia eliminates pain originating from dental or periodontal structures, but not referred pains. The most common nondental source of pain is myofascial pain referred from muscles of mastication, which become tender and ache with increased use. Many sufferers exhibit bruxism (grinding of the teeth) secondary to stress and anxiety. Temporomandibular joint disorder is closely related. Features include pain, limited mandibular movement, and temporomandibular joint sounds. The etiologies are complex; malocclusion does not play the primary role once attributed to it. Osteoarthritis is a common cause of masticatory pain. Anti-inflammatory medication, jaw rest, soft foods, and heat provide relief. The temporomandibular joint is involved in 50% of patients with rheumatoid arthritis and is usually a late feature of severe disease. Migrainous neuralgia may be localized to the mouth. Episodes of pain and remission without an identifiable cause and a lack of relief with local anesthesia are important clues. Trigeminal neuralgia (tic douloureux) can involve the entire branch or part of the mandibular

TABLE 38-5 Oral Lesions Associated with HIV Infection LESION MORPHOLOGY ETIOLOGIES Papules, nodules, plaques Candidiasis (hyperplastic and pseudomembranous)a Condyloma acuminatum (human papillomavirus infection) Squamous cell carcinoma (preinvasive and invasive) Non-Hodgkin’s lymphomaa Hairy leukoplakiaa Ulcers Recurrent aphthous ulcersa Oral Manifestations of Disease CHAPTER 38 Angular cheilitis Squamous cell carcinoma Acute necrotizing ulcerative gingivitisa Necrotizing ulcerative periodontitisa Necrotizing ulcerative stomatitis Non-Hodgkin’s lymphomaa Viral infection (herpes simplex, herpes zoster, cytomegalovirus infection) Infection caused by Mycobacterium tuberculosis or Mycobacterium avium-intracellulare Fungal infection (histoplasmosis, cryptococcosis, candidiasis, geotrichosis, aspergillosis) Bacterial infection (Escherichia coli, Enterobacter cloacae, Klebsiella pneumoniae, Pseudomonas aeruginosa) Drug reactions (single or multiple ulcers) Pigmented lesions Kaposi’s sarcomaa Bacillary angiomatosis (skin and visceral lesions more common than oral) Zidovudine pigmentation (skin, nails, and occasionally oral mucosa) Addison’s disease Miscellaneous Linear gingival erythemaa aStrongly associated with HIV infection. or maxillary branch of the fifth cranial nerve and can produce pain in one or a few teeth. Pain may occur spontaneously or may be trig gered by touching the lip or gingiva, brushing the teeth, or chewing. Glossopharyngeal neuralgia produces similar acute neuropathic symptoms in the distribution of the ninth cranial nerve. Swallowing, sneezing, coughing, or pressure on the tragus of the ear triggers pain that is felt in the base of the tongue, pharynx, and soft palate and may be referred to the temporomandibular joint. Neuritis involv ing the maxillary and mandibular divisions of the trigeminal nerve (e.g., maxillary sinusitis, neuroma, and leukemic infiltrate) is distin guished from ordinary toothache by the neuropathic quality of the pain. Occasionally, phantom pain follows tooth extraction. Pain and hyperalgesia behind the ear and on the side of the face in the day or so before facial weakness develops often constitute the earliest symp tom of Bell’s palsy. Likewise, similar symptoms may precede visible lesions of herpes zoster infecting the seventh nerve (Ramsy-Hunt syndrome) or trigeminal nerve. Postherpetic neuralgia may follow either condition. Coronary ischemia may produce pain exclusively in the face and jaw; as in typical angina pectoris, this pain is usually reproducible with increased myocardial demand. Aching in several upper molar or premolar teeth that is unrelieved by anesthetizing the teeth may point to maxillary sinusitis. Giant cell arteritis is notorious for producing headache, but it may also produce facial pain or sore throat without headache. Jaw and tongue claudication with

chewing or talking is relatively common. Tongue infarction is rare. Patients with subacute thyroiditis often experience pain referred to the face or jaw before the tenderness of the thyroid gland and transient hyperthyroidism are appreciated. “Burning mouth syndrome” (glossodynia) occurs in the absence of an identifiable cause (e.g., vitamin B12 deficiency, iron deficiency, dia betes mellitus, low-grade Candida infection, food sensitivity, or subtle

xerostomia). The etiology may be neuropathic. Clonazepam, α-lipoic acid, and cognitive-behavioral therapy benefit some patients. Some cases associated with an angiotensin-converting enzyme inhibitor have remitted when the drug was discontinued.

■ ■DISEASES OF THE SALIVARY GLANDS Saliva is essential to oral health. Its absence leads to dental caries, periodontal disease, and difficulties in wearing dental prostheses, masticating, and speaking. Its major components, water and mucin, serve as a cleansing solvent and lubricating fluid. In addition, saliva contains antimicrobial factors (e.g., lysozyme, lactoperoxidase, secre tory IgA), epidermal growth factor, minerals, and buffering sys tems. The major salivary glands secrete intermittently in response to autonomic stimulation, which is high during a meal. Hundreds of minor glands in the lips and cheeks secrete mucus continuously throughout the day and night. Consequently, oral function becomes impaired when salivary function is reduced. The sensation of a dry mouth (xerostomia) is perceived when salivary flow is reduced by 50%. The most common etiology is medication, especially drugs with anticholinergic properties but also alpha and beta blockers, calcium channel blockers, and diuretics. Other causes include Sjögren’s syn drome, chronic parotitis, salivary duct obstruction, diabetes mellitus, HIV/AIDS, and radiation therapy that includes the salivary glands in the field. Management involves eliminating or limiting drying medications, preventive dental care, and supplementation with oral liquid or salivary substitutes. Sugarless mints or chewing gum may stimulate salivary secretion if dysfunction is mild. When sufficient exocrine tissue remains, pilocarpine or cevimeline can increase secre tions. Commercial saliva substitutes or gels relieve dryness. Fluoride supplementation is critical to prevent caries. PART 2 Cardinal Manifestations and Presentation of Diseases Sialolithiasis presents most often as painful swelling but in some instances as only swelling or only pain. Conservative therapy consists of local heat, massage, and hydration. Promotion of salivary secretion with mints or lemon drops may flush out small stones. Antibiotic treat ment is necessary when bacterial infection is suspected. In adults, acute bacterial parotitis is typically unilateral and most commonly affects postoperative, dehydrated, and debilitated patients. Staphylococcus aureus (including methicillin-resistant strains) and anaerobic bacteria are the most common pathogens. Chronic bacterial sialadenitis results from lowered salivary secretion and recurrent bacterial infection. When suspected bacterial infection is not responsive to therapy, the differential diagnosis should expand to include benign and malignant neoplasms, lymphoproliferative disorders, Sjögren’s syndrome, sar coidosis, tuberculosis, lymphadenitis, actinomycosis, and granuloma tosis with polyangiitis. Bilateral nontender parotid enlargement occurs with diabetes mellitus, cirrhosis, bulimia, HIV/AIDS, and drugs (e.g., iodide, propylthiouracil). Pleomorphic adenoma composes two-thirds of all salivary neo plasms. The parotid is the principal salivary gland affected, and the tumor presents as a firm, slow-growing mass. Although this tumor is benign, its recurrence is common if resection is incomplete. Malignant tumors such as mucoepidermoid carcinoma, adenoid cystic carcinoma, and adenocarcinoma tend to grow relatively fast, depending upon grade. They may ulcerate and invade nerves, producing numbness and facial paralysis. Surgical resection is the primary treatment. Radiation therapy (particularly neutron-beam therapy) is used when surgery is not feasible and

after resection for certain histologic types with a high risk of recurrence. Malignant salivary gland tumors have a 5-year sur vival rate of 94% when the stage is local, 70% with regional spread, and 43% when distant. Dental Care for Medically Complex Patients Routine dental care (e.g., uncomplicated extraction, scaling and cleaning, tooth resto ration, and root canal) is remarkably safe. The most common concerns regarding care of dental patients with medical disease are excessive bleeding for patients taking anticoagulants, infection of the heart valves and prosthetic devices from hematogenous seeding by the oral flora, and cardiovascular complications resulting from vasopressors used

with local anesthetics during dental treatment, although the risk of any of these complications is very low. Patients undergoing tooth extraction or alveolar and gingival surgery rarely experience uncontrolled bleeding when warfarin anticoagulation is maintained within the therapeutic range currently recommended for prevention of venous thrombosis, atrial fibrilla tion, or mechanical heart valve. Embolic complications and death, however, have been reported during subtherapeutic anticoagulation. Therapeutic anticoagulation should be confirmed before and contin ued through the procedure. Likewise, low-dose aspirin (e.g., 81–325 mg)

can safely be continued. For patients taking aspirin and another antiplatelet medication (e.g., clopidogrel), continuation of the second antiplatelet medication should be based on individual consideration of the risks of thrombosis and bleeding. Target-specific oral antico agulants (dabigatran, apixaban, rivaroxaban, and edoxaban) are in increasingly common use. Simple extractions of one to three teeth, periodontal surgery, abscess drainage, and implant positioning do not typically require interruption of therapy. More extensive surgery may necessitate delaying or holding a dose of the anticoagulant or more elaborate measures to manage the risk of thrombosis and bleeding. Patients at risk for bacterial endocarditis (Chap. 133) should main tain optimal oral hygiene, including flossing, and regular professional cleanings. Currently, guidelines recommend that prophylactic antibiot ics be restricted to patients at high risk for bacterial endocarditis who undergo dental and oral procedures involving significant manipulation of gingival or periapical tissue or penetration of the oral mucosa. If unexpected bleeding occurs, antibiotics given within 2 h after the pro cedure provide effective prophylaxis. Hematogenous bacterial seeding from oral infection can produce late prosthetic-joint infection and therefore requires removal of the infected tissue (e.g., drainage, extraction, root canal) and appropriate antibiotic therapy. However, evidence that late prosthetic-joint infec tion follows routine dental procedures is lacking. For this reason, anti biotic prophylaxis is generally not recommended before oral surgery or oral mucosal manipulation for patients who have undergone joint replacement surgery. Exceptions to this may be considered for patients who have experienced joint replacement complications. Concern often arises regarding the use of vasoconstrictors to treat patients with hypertension and heart disease. Vasoconstrictors enhance the depth and duration of local anesthesia, thus reducing the anesthetic dose and potential toxicity. If intravascular injection is avoided, 2% lidocaine with 1:100,000 epinephrine (limited to a total of 0.036 mg of epinephrine) can be used safely in patients with controlled hypertension and stable coronary heart disease, arrhythmia, or con gestive heart failure. Precautions should be taken with patients taking tricyclic antidepressants and nonselective beta blockers because these drugs may potentiate the effect of epinephrine. Elective dental treatments should be postponed for at least 1 month and preferably for 6 months after myocardial infarction, after which the risk of reinfarction is low provided the patient is medically stable (e.g., stable rhythm, stable angina, and no heart failure). Patients who have suffered a stroke should have elective dental care deferred for 9 months. In both situations,

effective stress reduction requires good pain control, including the use of the minimal amount of vasoconstrictor necessary to provide good hemostasis and local anesthesia. Bisphosphonate therapy is associated with osteonecrosis of the jaw. However, the risk with oral bisphosphonate therapy is very low. Most patients affected have received high-dose aminobisphosphonate ther apy for multiple myeloma or metastatic breast cancer and have under gone tooth extraction or dental surgery. Intraoral lesions, of which two-thirds are painful, appear as exposed yellow-white hard bone involving the mandible or maxilla. Screening tests for determining risk of osteonecrosis are unreliable. Patients slated for aminobisphospho nate therapy should receive preventive dental care that reduces the risk of infection and the need for future dentoalveolar surgery. Halitosis Halitosis typically emanates from the oral cavity or nasal passages. Bacterial decay of food and cellular debris account for the

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