Paparella: Volume III: Head and Neck

Section 2: Disorders of the Head and Neck

Part 2: The Oral Cavity

Chapter 14: Common Disorders of the Teeth and Gingiva

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Development and Growth of Teeth

Tooth development is first seen during the sixth week of embryonic life as a thickening of the epithelium in the region of the future dental arches. At certain points along this band of epithelium, known as the dental lamina, budlike proliferations of basal epithelial cells will press into the subjacent mesenchyme to form the early enamel organ of a deciduous tooth. This early development of the tooth bud is induced by ectomesenchyme of neural crest origin, which lies beneath the dental lamina and which will also ultimately form the dental papilla as well as the dental sac. Under the influence of the developing enamel organ, the ectomesenchyme then proliferates and condenses to form the dental papilla from which the dentin and dental pulp of the tooth will ultimately be derived. Concomitantly, proliferation and condensation of ectomesenchyme, which surrounds both the enamel organ and the dental papilla, will form the dental sac from which the periodontal ligament, which surrounds the tooth and secures it to alveolar bone, will form. With further development of the enamel organ, the band of dental lamina that connects it to the oral ectoderm will break up, leaving rests of odontogenic epithelium that remain and are found in the bone and attached gingiva throughout life.

As formation of the enamel and dentin reaches what will become the cementoenamel junction, the clinical dividing line between the crown and root of the tooth, Hertwig's epithelial root sheath is formed from the enamel organ that will ultimately shape the roots of the teeth. With root formation, this epithelial sheath breaks down to form persistent rests of cells within the periodontal ligament known as the epithelial rests of Malassez.

Epithelium derived from remnants of the dental lamina and Hertwig's epithelial root sheath, along with the dental follicle surrounding unerupted or impacted teeth, is the primary source of the odontogenic cysts and odontogenic tumors that may be found in later life.

With root development, and as Hertwig's epithelial root sheath breaks up, cementoblasts differentiate from previously undifferentiated mesenchymal cells to form cementum, the mineralized layer that covers the tooth root and to which the fibers of the periodontal ligament attach, binding it to the alveolar bone.

Tooth Eruption

The normal human dentition is composed of 20 teeth in the deciduous dentition and 32 teeth in the permanent dentition. Eruption of the deciduous teeth usually begins at approximately 6 months of age, with eruption of the mandibular central incisors. Eruption of the deciduous dentition is usually completed by 24 months of age, with eruption of the maxillary second molars. Eruption of the permanent teeth usually begins between 6 and 7 years of age with eruption of the mandibular central incisor teeth and will usually be complete by 21 years of age with eruption of the third molars. The primary dentition is composed of two maxillary and mandibular central and lateral incisors, two canine or cuspid teeth, and two first and second molars. The permanent dentition is composed of two maxillary and mandibular central and lateral incisors, two canine or cuspid teeth, two first and two second premolars or bicupsids, and two first, second, and third molar teeth.

Occlusion

Occlusion refers to the interrelationship of the teeth of the upper and lower jaws in contact. The subject of occlusion is a complex one with a number of different classifications having been introduced. The classification introduced by Angle in 1899 is the most universally used today. Angle used the position of the molar teeth as the key to his classification, placing the patient into one of three categories: class I (neutrocclusion, normocclusion), class II (distocclusion), or class III (mesiocclusion).

Class I Occlusion

A class I occlusion is one in which the arches are in normal mesiodistal relationships with the mesiobuccal cusp tip of the maxillary first molar aligned with the buccal groove of the mandibular first molar. Patients with this molar relationship will usually demonstrate fairly normal facial profiles.

Class II Occlusion

In a class II occlusion, the mandibular arch is distal to its normal relationship with the maxillary arch so that the mesiobuccal cusp tip of the maxillary first molar is positioned anterior to the buccal groove of the mandibular first molar. This type of occlusion often gives the middle face a more prominent appearance with respect to the lower jaw, which may appear recessive.

Class III Occlusion

In a class III occlusion, the mandibular arch is mesial in its relationship to the maxillary arch, with the mesiobuccal cusp tip of the maxillary first permanent molar being posterior to the buccal groove of the mandibular first permanent molar. This type of occlusion is most commonly caused by excessive growth of the mandible and often leaves the patient with a concave facial profile.

Dental Caries

Dental caries is a disease of the calcified tissues of the teeth resulting from microbial action that is characterized by demineralization of the inorganic portion of the tooth with subsequent destruction of the organic portion. Dental decay occurs in one of two patterns: (1) caries in the pits or fissures of the teeth or (2) caries on the smooth surfaces of the teeth. Dental caries may also be classified according to cause, such as nursing bottle caries, which arise from prolonged contact of the teeth with milk, milk formula, or fruit juice from a nursing bottle, or radiation caries, whose pattern is unusual because of its occurrence on the smooth surfaces of the teeth, particularly at the gingival margin as well as occasionally on the incisal edges and cusp tips of the teeth.

Left unchecked, dental caries will progress to involve the pulp of the tooth, leading to death of the pulp and dental infection. The result of dental infection of the pulp may be a dental abscess or low-grade chronic inflammation, such as may be seen within a periapical granuloma or apical periodontal cyst arising from cystic degeneration of the rests of Malassez within the periodontal ligament surrounding the tooth.

Gingival Infections

Gingival infections may be acute or chronic in nature and may be bacterial, viral, or mycotic in origin. Only commonly encountered gingival infections will be discussed here. Two common types of gingival infections, primary and secondary herpesvirus infections as well as oral candidiasis, are discussed in Chapter 15 and will not be included in this discussion.

Acute Necrotizing Ulcerative Gingivitis (Vincent's Infection, Trench Mouth, Acute Ulceromembranous Gingivitis)

Acute necrotizing ulcerative gingivitis (ANUG) is an acute necrotizing inflammatory process primarily involving the marginal gingiva and interdental papillae. The interdental papillae and marginal gingiva present as punched-out or craterlike areas of tissue necrosis that are covered by a yellow-gray pseudomembrane surrounded by a pronounced linear erythema. The pseudomembrane is easily rubbed off, leaving an intensely red area of surface ulceration. The gingiva is quite painful and hyperemic. Gingival hemorrhage may be spontaneous or easily provoked by the slightest stimulation.

Other characteristic signs are a fetid odor to the breath, increased amounts of thick saliva, and a foul metallic taste. Constitutional signs and symptoms, such as fever, malaise, loss of appetite, gastrointestinal upset, regional lymphadenopathy, and in some cases severe leukocytosis may be noted.

ANUG is most common in young adults and has been associated with inadequate rest, overwork, change in living habits, extreme emotional stress, and poor nutrition. In most cases, long-standing chronic marginal gingivitis has been present for some time as a predisposing factor.

It is caused by aerobic fusospirochetal organisms that may become pathogenic during periods of decreased tissue resistance or following a debilitating infection, such as an upper respiratory tract infection. ANUG may also be a complicating factor in immunosuppressed patients.

The first step in management of ANUG is to remember that it usually occurs in individuals with decreased tissue resistance to infection, and the possibility of systemic disease should be ruled out. Once this is done, steps should be taken to improve the overall health of the patient and alleviate the factors (stress, overwork, and so on) that may have predisposed the patient to this infection. An oxidizing mouth rinse, such as equal parts of hydrogen peroxide (USP 3 per cent) and warm water, should be used every few hours initially and decreased to four times a day over a period of 4 to 5 days. A dentist should be consulted, who will initially cleanse the lesion with cotton swabs for the first 2 days or so until dental prophylaxis and dental scaling can be performed. In severe cases, antibiotic therapy, consisting of 250 to 500 mg of oral penicillin V four times daily for at least 5 to 7 days, should be instituted.

Gingival Abscess

A gingival abscess may arise from one of two sources. It may be periodontal in nature or of dental origin, arising from infection and necrosis of the dental pulp. A periodontal abscess will develop when the purulent by-products of an acute infection within a periodontal pocket are walled off, first forming a cellulitis that may localize to form a draining fistula. A gingival abscess of dental origin develops when the purulent by-product of a necrotic dental pulp has formed a tract from the tooth through the alveolar bone, following the path of least resistance, to produce a gingival abscess and finally a draining fistulous tract.

A periodontal abscess is treated by debriding the infected periodontal pocket. A dental abscess will continue to recur unless the remnants of the necrotic dental pulp are removed from the root canal of the tooth and endodontic therapy is performed or the tooth is extracted. Antibiotic therapy, usually consisting of a course of 5 to 7 days of penicillin V, may be indicated.

Periodontal Disease (Periodontitis)

Periodontal disease is a collective term for the various inflammatory disease processes that affect the gingiva, periodontal tissues, and alveolar bone that support the teeth. Gingivitis, which affects nearly the entire population to some extent, is an inflammatory response to the accumulation of bacterial plaque. At some point, possibly with the overgrowth or addition of a pathogenic bacterial population, this inflammatory process extends into the supporting periodontal tissues, leading to progressive tissue destruction and ultimately tooth loss if not appropriately treated. Preventing or removing this bacterial plaque will avoid destruction of the supporting connective tissue and bone, thereby preventing this form of tooth loss.

At least four different forms of periodontal disease are known to occur: prepubertal, juvenile, rapidly progressive, and adult periodontitis. The last is by far the most common form of the disease and is the major cause of tooth loss in this age group. This form of periodontal

disease may be noted as early as the second decade of life and demonstrates a steady increase in prevalence and severity with age.

The clinical appearance varies from individual to individual and is to a large extent dependent upon the patient's current level of home care and overall extent of disease. It will vary from a localized marginal gingivitis with inflammation and swelling of the marginal gingiva and interdental papillae to a generalized diffuse gingivitis in which the entire gingiva and adjacent alveolar mucosa may be bright red, swollen, and hemorrhagic in appearance. The clinical appearance of the gingiva and its height of attachment on the tooth may, however, belie the presence of deep periodontal pocketing and marked bone loss. The pattern of bone loss is typically horizontal in nature and may be localized or generalized, often being most prominent in the molar regions, especially around the second molar teeth. Dental radiographs are useful in showing the pattern and depth of bone loss; however, they do not adequately show the level of attachment of the gingival tissue at the base of the pocket on the surface of the tooth. This pocket depth is the distance between the base of the pocket and the marginal gingiva and is measured clinically with a calibrated periodontal probe.

In addition to scaling and root planing of the teeth, systemic antibiotics, such as tetracyclines, metronidazole, and clindamycin, show promise as adjunctive agents in controlling periodontal disease. Tetracyclines have also been applied locally (subgingivally in the periodontal pocket) to reduce pathogenic subgingival microflora. Antibacterial agents, such as chlorhexidine gluconate 0.12 per cent (Peridex) and any commercial mouthwash, may enhance the control of supragingival plaque.

Three other less common, but clinically distinct, forms of periodontitis are currently recognized. Juvenile periodontitis (periodontosis) is characterized by rapid bone destruction in young individuals without known local or systemic predisposing factors commensurate with the severity of disease activity. There is a characteristic pattern of vertical bone destruction involving the incisor and first molar teeth in most patients. A generalized form of the disease with involvement of all teeth may be noted. Juvenile periodontitis is further differentiated from other forms of periodontitis by its development in young individuals, often with onset of the disease at puberty. Etiologically, it appears to be characterized by a unique gram-negative anaerobic bacterial flora along with specific defects in host immune mechanisms. There is evidence for a selective cell-mediated immunodeficiency.

Prepubertal periodontitis affects the deciduous teeth in young children, occurring in a localized and generalized form. Its onset appears to occur at around 4 years of age or before and may be accompanied by otitis media and upper respiratory tract infections, which though not life-threatening in the localized form of the disease may be life-threatening in the more severe generalized form. Patients with this disorder show abnormalities in peripheral blood leukocyte chemotaxis. In the localized form of the disease, there are defects in cell adherence in either neutrophils or monocytes, but not in both cell types; in the generalized form, there are defects in both cell types. Prepubertal periodontitis may be followed by a normal permanent dentition or by severe periodontitis of the permanent teeth.

A fourth clinical variant of periodontitis is rapidly progressive periodontitis, which is now recognized as a distinct clinical condition. This form of periodontitis is seen most frequently in young individuals in their 20s but may be found in postpubertal individuals up to approximately age 35 years. This form of periodontitis is characterized by a rapid, generalized destruction of alveolar bone that may occur over a period of a few weeks to months and may be accompanied by general malaise, weight loss, and depression. It may continue to progress relentlessly or it may go into clinical remission without therapy. Although a small number of patients will not respond to any form of therapy, including antibiotics, patients with this disorder usually respond favorably to dental scaling and root planing, especially with concomitant antibiotic therapy.

Reactive and Neoplastic Gingival Disease

Gingival enlargement may be localized or generalized, varying from reactive to neoplastic in nature. Reactive gingival fibrous hyperplasia is by far the most common cause of gingival enlargement. It results from some prolonged, chronic inflammatory process involving the gingiva and results in overgrowth of the fibrous connective tissue. One example is a fibroma, which is characteristically a dome-shaped lesion with a sessile base and smooth surface that is usually the color of the surrounding mucosa. The fibroma is the most common benign soft tissue tumor found in the oral cavity. It may represent a localized type of reactive fibrous hyperplasia or the end result of fibrosis of a healed pyogenic granuloma. Another example is the redundant fibrous tissue found overlying an edentulous region on the alveolar ridge at which point there has been irritation from an ill-fitting dental prosthesis, such as a fixed or removable partial denture. This common lesion is often called epulis fissuratum, best termed simply *inflammatory fibrous gingival hyperplasia.*

Localized or generalized gingival hyperplasia, which is inflammatory in nature, may also be seen at puberty, at which time it is termed *pubertal gingivitis,* and during pregnancy, when it is termed *pregnancy gingivitis.* In both cases it is associated with gingival inflammation, which is felt to be a predisposing factor.

There is an idiopathic form of fibrous gingival hyperplasia, termed *fibromatosis gingivae,* which is characterized by a diffuse gingival enlargement that may cover the teeth or may impede tooth eruption in the developing dentition. Drug-induced gingival fibrous hyperplasia may be noted after the use of the anticonvulsant drug Dilantin sodium as well as the immunosuppressant agent cyclosporine. Regardless of the cause, the therapy of choice is surgical excision.

Both benign and malignant neoplasms may involve the gingiva. The papilloma is a relatively common benign neoplasm of unknown origin that arises from the surface epithelium. It is typically an exophytic lesion with fingerlike projections arising from a pedunculated base. Oral verruca vulgaris, or oral warts, and condyloma accuminatum also occur on the gingiva and may be indistinguishable clinically from oral squamous cell papillomas. As oral squamous cell papillomas and oral verruca vulgaris have an identical clinical appearance and similar histomorphologic features, it is entirely possible that many oral papillomas have a viral cause.

Squamous cell carcinoma is well known to occur on the gingiva in both dentulous and edentulous areas and may mimic inflammatory disorders, such as periodontal disease or reactive growths, such as pyogenic granulomas. Although they are relatively uncommon lesions in the oral cavity, when malignant melanomas occur in this location, they are most frequently found on the maxillary anterior alveolar ridge and palate.

Disseminated neoplastic disorders may also involve the gingiva and range from metastatic epithelial and mesenchymal soft tissue neoplasms, which often present as exophytic lesions, to a leukemic infiltrate presenting as generalized gingival hyperplasia.