A Review of Oral Histology and Physiology

HOW TO RECEIVE CREDIT

- Read the enclosed course.
- Complete the questions at the end of the course.
- Return your completed Answer Sheet to NetCE by mail or fax, or complete online at www.NetCE.com. Your postmark or facsimile date will be used as your completion date.
- Receive your Certificate(s) of Completion by mail, fax, or email.

Faculty
Mark J. Szarejko, DDS, FAGD, received his dental degree from the State University of New York at Buffalo in 1985. He received fellowship from the Academy of General Dentistry in 1994.

Faculty Disclosure
Contributing faculty, Mark J. Szarejko, DDS, FAGD, has disclosed no relevant financial relationship with any product manufacturer or service provider mentioned.

Director of Development and Academic Affairs
Sarah Campbell

Director Disclosure
The director has disclosed no relevant financial relationship with any product manufacturer or service provider mentioned.

Audience
This course is designed for all dental professionals.

Accreditations & Approvals
NetCE is an ADA CERP Recognized Provider.

ADA CERP is a service of the American Dental Association to assist dental professionals in identifying quality providers of continuing dental education. ADA CERP does not approve or endorse individual courses or instructors, nor does it imply acceptance of credit hours by boards of dentistry.

Concerns or complaints about a CE provider may be directed to the provider or to ADA CERP at www.ada.org/cerp.

NetCE
Nationally Approved PACE Program Provider for FAGD/MAGD credit.
Approval does not imply acceptance by any regulatory authority or AGD endorsement.
10/1/2021 to 9/30/2027
Provider ID #217994.

NetCE is a Registered Provider with the Dental Board of California. Provider number RP3841. Completion of this course does not constitute authorization for the attendee to perform any services that he or she is not legally authorized to perform based on his or her permit type.

NetCE is approved as a provider of continuing education by the Florida Board of Dentistry, Provider #50-2405.

Designations of Credit
NetCE designates this activity for 5 continuing education credits.

AGD Subject Code 010.

This course meets the Dental Board of California's requirements for 5 units of continuing education.

Dental Board of California course #05-3841-00378.

A complete Works Cited list begins on page 19.

Mention of commercial products does not indicate endorsement.

About the Sponsor

The purpose of NetCE is to provide challenging curricula to assist healthcare professionals to raise their levels of expertise while fulfilling their continuing education requirements, thereby improving the quality of healthcare.

Our contributing faculty members have taken care to ensure that the information and recommendations are accurate and compatible with the standards generally accepted at the time of publication. The publisher disclaims any liability, loss or damage incurred as a consequence, directly or indirectly, of the use and application of any of the contents. Participants are cautioned about the potential risk of using limited knowledge when integrating new techniques into practice.

Disclosure Statement

It is the policy of NetCE not to accept commercial support. Furthermore, commercial interests are prohibited from distributing or providing access to this activity to learners.

Course Objective

The purpose of this course is to provide clinicians with a better understanding of the cellular components of the oral hard and soft tissues, supplying a scientific basis upon which clinical decisions for dental treatment can be made.

Learning Objectives

Upon completion of this course, you should be able to:

1. Outline the basic components of the enamel, dentine, and pulp.
2. Review the histology of the constituents of the periodontium.
3. Identify the varying divisions and functions of the oral mucosa.
4. Differentiate between the histology and salivary composition of the major and minor salivary glands.
5. Describe the manner by which disease and radiation therapy affect salivary glands.

INTRODUCTION

General dentists and dental specialists, dental hygienists, and dental assistants all treat patients with dental caries, periodontal disease, edentulism, odontogenic infections, and oral and maxillofacial trauma. The unique oral environment is comprised of vastly different hard and soft tissues, with each component an essential link to appropriate oral and systemic health. The cellular constitution or histology of these structures is the basis from which the morphology originates and from which all physiologic actions commence. Form and function are inseparable.

This course will discuss the basic histologic components of the teeth, oral mucosa, salivary glands, and periodontium. The manner in which these structures are affected by varied oral pathologic processes, systemic diseases, and medications will be highlighted as related to their histologic origins and macroscopic appearance. The relationship between the normal histologic manifestations of these structures and their responses to inflammation, diseases, infection, and dental treatment will be outlined. An appreciation for the complexity of these tissues and their interrelationships with each other will instill dental clinicians with a renewed sense of respect for the teeth, bone, mucosa, and gingival tissues upon which they perform treatment on a daily basis.

TEETH

In the wild, most animals require their teeth for survival. Humans will suffer a decreased quality of life if natural teeth are not replaced by dentures, but edentulism alone is not a terminal condition. Beyond occlusion, teeth are critical to appropriate facial form and cosmetics, phonation and speech, and social acceptance. While some animals (e.g., sharks) regenerate new teeth to replace worn or fractured teeth, humans receive one set of deciduous (baby) teeth and one set of permanent teeth. (Rarely, supernumerary teeth may occur.) Teeth that are lost to caries, periodontal disease, or trauma can only be replaced by prosthetic means. Teeth are considered a triumph of engineering, as years of occlusion and masticatory forces, an oral environment that teams with complex microbial organisms, and parafunctional habits all contribute to challenge the integrity of teeth. This section will discuss the basic components of teeth, their response to common pathologic processes, and unique aspects of their clinical relevance.

ENAMEL

Enamel is the external layer of the tooth and is composed of 96% mineral deposits, with water and organic substances completing the remaining matrix [1]. It surpasses bone as the hardest and most mineralized substance in the body. A crystallized form of calcium phosphate known as hydroxylapatite is the main mineral component of enamel [2]. This compound accounts for the strength of enamel but also for its brittleness. Unlike the underlying dentin, enamel is without collagen. Enamel can exceed a thickness of 2 millimeters on the cusps of posterior teeth, but it exists as a thin veneer at the dentinoenamel junction.

Density and hardness are also highest where the enamel is thickest, such as the cusps of molars and the incisal edges of anterior teeth. These physical properties allow attrition to progress gradually on the surfaces of teeth that are subject to the repetitious forces of occlusion and mastication. Surface enamel is harder, denser, and less porous then the underlying layers of enamel. Enamel has a low tensile strength, which accounts for its brittle nature. However, its high modulus of elasticity, combined with support from the underlying dentin, decrease the incidence of enamel fracture [3].

Ameloblasts are the cells from which enamel is formed. The primary organizational units of enamel are the enamel rods (prisms) and interrod enamel (interprismatic substance). Enamel prisms originate at the dentinoenamel junction and extend to the surface of the enamel. Each enamel rod is approximately 5–6 micrometers in diameter and is as long as the enamel is thick [4]. Enamel is without a neural or vascular supply; therefore, teeth fractures strictly limited to enamel are painless and bloodless.

Although enamel is the hardest substance in the body, its lifelong exposure to the oral environment presents numerous challenges to its integrity and maintenance. When enamel is lost, there is no capacity for the production of new enamel. The loss of enamel through tooth-to-tooth contact is known as attrition. This is a gradual process that occurs throughout life but can be accelerated when parafunctional habits such as bruxing or clenching occur. The functional aspects of the teeth, such as the incisal edges of anterior teeth and the occlusal surfaces of posterior teeth, are most frequently involved, although the interproximal surfaces of teeth may also be affected. This loss of enamel can dull the incisal edges and flatten the occlusal surfaces of teeth, causing a decrease in the vertical dimension of occlusion and decreasing the penetrating ability of the involved surfaces during mastication. Sensitivity usually does not occur until the enamel has worn away to the point of exposing the underlying dentin. The loss of enamel supporting dental restorations can undermine their support and cause the loss of the restoration.

Abrasion involves the loss of tooth structure secondary to an external source of friction, such as excessive and overly aggressive tooth brushing. It is not limited to enamel, as root surface exposure via gingival recession can subject the cementum and underlying dentin to abrasive forces. Toothbrush abrasion usually occurs in the cervical area of the labial surfaces of anterior teeth and the buccal surfaces of posterior teeth. Sensitivity to thermal stimuli and sweets can occur upon exposure of the underlying dentin. The use of a soft-bristled toothbrush and a thorough, but gentle, cleaning motion during tooth brushing will decrease the incidence and severity of abrasion. In one study, loss of enamel was influenced mainly by the abrasivity of the toothpaste slurry, although this was modified by toothbrush filament stiffness [32]. So, type of toothpaste selected is also a consideration when attempting to limit erosion.

Erosion refers to the loss of tooth structures such as enamel via acidic substances, often extrinsic sources such as carbonated beverages or citrus fruits/juices. The primary intrinsic source of erosion is gastric acid introduced into the oral cavity during repeated episodes of regurgitation or vomiting. Patients who suffer from bulimia, anorexia, alcoholism, and gastroesophageal reflux disease are most frequently affected. Pregnant patients who experience prolonged bouts of morning sickness can also develop erosive defects that characteristically occur on the lingual surfaces of the maxillary anterior teeth and the occlusal surfaces of posterior teeth. Exposure of the underlying dentin can eventually occur and cause increased sensitivity and increased risk of caries and damage to existing restorations. Restorative efforts may be futile if the underlying cause of erosion is not diagnosed and treated.

Dental caries are the most frequent pathologic entity to adversely affect teeth. Despite advances in prevention and in oral hygiene, dental caries remain the most common chronic disease among children in the United States, with a prevalence five times greater than childhood asthma [5]. Two species of cariogenic bacteria, *Streptococcus mutans* and *Lactobacillus*, have been implicated in the initiation of the carious process [1]. Initial demineralization of the enamel left unchecked can extend to the underlying dentin and dental pulp to cause pain, abscesses, local and regional infections, and even sepsis.

An end-product of the fermentation of carbohydrates (e.g., glucose, sucrose, fructose) by these cariogenic bacteria is acid, which causes demineralization of the enamel [7]. However, enamel is the only component of the tooth with the capacity for remineralization via externally supplied calcium and phosphate ions. These minerals are deposited into the crystal voids of demineralized enamel and can replace the mineral content lost in incipient carious lesions [8]. When this equilibrium between demineralization and remineralization favors the former, the carious process progresses through the enamel, into the dentin, and ultimately the pulp. Carious lesions may be asymptomatic until advanced stages, at which time the extent and cost of treatment are greater. Any factor that increases the amount and duration of dental plaque on the teeth will increase the risk of caries. This includes decreased salivary flow, which is a side effect of many medications, radiation therapy for head and neck cancer, and autoimmune diseases such as Sjögren syndrome.

Prevention of dental caries has been associated with excellent oral hygiene and the use of fluoride-containing toothpastes, gels, mouth rinses, and supplements. Fluoride makes enamel more resistant to dental caries by encouraging remineralization and by decreasing the production of acid by cariogenic bacteria [10]. According to the Centers for Disease Control and Prevention, laboratory and epidemiologic research indicates that fluoride acts directly as a result of topical application on the enamel after eruption of the tooth [9]. This is in contrast to the long-held belief that ingested fluoride was the primary means by which enamel was strengthened.

DENTIN

Dentin is the substance that underlies the enamel of the clinical crown of the tooth and the cementum of the root. Its composition differs from enamel, consisting of 70% inorganic components, 20% organic components, and 10% water [1]. The significantly lower mineral content makes dentin softer than enamel and permits a more rapid progression of decay. It is formed by cells called odontoblasts and comprises the bulk of the tooth structure supporting the enamel.

Most of the organic matrix of dentin is type I collagen, which confers a property of resiliency such that dentin can deform under the forces of occlusion and support the more brittle layer of enamel [12]. Dentin is avascular, like enamel, and the substructure of dentin is a series of minute channels called dentinal tubules that contain cytoplasmic extensions of the progenitor odontoblasts and traverse much of the thickness of dentin. These odontoblastic processes are the main component within the dentinal tubules, and some may extend to the dentinoenamel junction. Cellular organelles such as mitochondria and secretory granules are associated with the cell bodies of the odontoblastic processes. Enamel is completely devoid of these live cellular elements.

The dentinal tubules are usually covered by enamel or cementum and do not make direct contact with the external oral environment. However, pathologic events such as attrition, abrasion, and erosion can deplete the surface layer of enamel and cause exposure of the underlying dentin. Gingival recession causes exposure of the tooth root and its protective cementum layer, the abrasion of which will also cause exposure of the supportive dentin. Thermal stimuli can cause changes in the hydraulic pressure within the exposed dentinal tubules and exert a force upon the odontoblastic processes, which is painful. Desensitizing toothpastes, gels, and mouth rinses contain ingredients that occlude the open and exposed dentinal tubules and can alleviate the pain. Components of other pastes have a direct effect on the odontoblastic processes.

Bacterial egress into exposed dentinal tubules can extend toward the pulp and stimulate an inflammatory response and even cause pulpal necrosis, which would require endodontic therapy, although this risk is remote. Gram-positive bacteria are the predominant micro-organisms found in the dentinal tubules and can be associated with carious and non-carious dentin [13].

After a tooth erupts, the enamel-forming ameloblast cells are lost and with them the capacity to produce any more enamel. However, dentin is capable of growth and repair and exists in several forms. Primary dentin is present at the time of the eruption of the tooth and is the predominant form of dentin. It is located between the enamel and the pulp chamber with the layer adjacent to the enamel known as mantle dentin, a less mineralized type than the remaining dentin. Secondary dentin develops after the root formation is complete and surrounds the pulp. All dentin develops from the dentinoenamel junction and progresses inwards toward the pulp. This progressive layering decreases the size of the pulp chamber and manifests as a clinical entity known as pulpal recession. This is clinically significant in the restoration of carious teeth. Younger patients with less secondary dentin and a subsequently large dental pulp will have a higher risk of pulpal involvement from dental caries than older patients with more secondary dentin and a receded pulp. Tertiary dentin, also called reparative or sclerotic dentin, forms in response to a stimulus such as dental caries, restorative procedures, or leakage around deficient restorations. It is deposited adjacent to the pulpal surface and is formed in response to these varied stimuli by previously inactive odontoblastic cells; it is not merely the formation of more secondary dentin.

Because dentin is less mineralized and softer than enamel, the occlusal surfaces of posterior teeth and the incisal edges of anterior teeth, where exposure is more likely, are more subject to attrition, fracture, and caries. Such deficiencies should be treated promptly to prevent the progressive destruction of tooth structure.

THE DENTAL PULP

Commonly called the "nerve" of the tooth, the dental pulp is a complex configuration of tissues. Blood vessels, nerve fibers, fibroblasts, macrophages, lymph vessels, lymph cells, and T-lymphocytes are all contained within this soft tissue matrix. The outer layer of the pulp consists of the cell bodies of the odontoblasts, cells that produce dentin. The tissue in the clinical crown of the tooth is the coronal pulp and is contained within the pulp chamber. Pulp within the tooth root is the radicular pulp or root canal, which ends at the apical foramen of the root and is continuous with the periodontal ligament. Nerve fibers (myelinated and unmyelinated) and blood vessels enter through the apical foramen and are closely associated throughout the entire pulp. The main sensory neurons within the dental pulp are pain fibers, some of which may extend into the dentinal tubules and may account for the sharp pain felt when thermal stimuli or acidic substances contact exposed dentin.

Pulpal tissues that are irreversibly inflamed or necrotic due to caries, trauma, or large restorations in close proximity to the pulp can be treated by endodontic (root canal) therapy. Occasionally, odontoblasts detach from the pulp chamber walls and cause the development of small conglomerations of dentin within the pulp, called pulp stones or denticles. These can be an obstruction to root canal therapy.

THE PERIODONTIUM

The structures that support, protect, and nourish the teeth externally are collectively referred to as the periodontium. This includes the free and attached gingiva, the cementum, the alveolar bone (process) of the maxilla and mandible, and the periodontal ligament. Each structure plays an important role in maintaining the health and stability of the teeth.

GINGIVAL TISSUES

The gingival tissues begin at the cervical area of the tooth, extend apically to cover the alveolar bone, and end at the intersection of the attached gingiva and the alveolar mucosa, referred to as the muco-gingival junction. In health, the gingiva is coral pink in color and does not bleed upon periodontal probing. It can be divided into three zones: the free or marginal gingiva, the attached gingiva, and the interdental gingiva.

Free (Marginal) Gingiva

The free gingiva is composed of stratified squamous epithelial tissue supported by a matrix of dense connective tissue. This tissue surrounds the tooth in a collar-like fashion and is not attached to the underlying alveolar bone. In some patients, there is a minute linear demarcation, called the free gingival groove, between the free and attached gingiva. The space between the tooth and inner aspect of the free gingiva is the gingival sulcus. Bacteria and plaque that are deposited on and not removed from the gingival sulcus initiate an inflammatory response known as gingivitis. There is no loss of alveolar bone at this point, but the continued assault of bacterial toxins and the host inflammatory response may cause a progressive deepening of the sulcus beyond the 3 mm or less depth associated with ideal gingival health. The development of a periodontal pocket indicates progressive inflammation that will continue to deepen without treatment and will eventually cause resorption of the alveolar bone supporting the tooth and tooth loss. The epithelium of the free gingiva facing the gingival sulcus is called the sulcular epithelium. These cells are generally not keratinized (although some keratinization may extend into the sulcus) and are thinner than the external epithelial surface of the free gingiva (which is keratinized). At the depth of the gingival sulcus, the attachment between the tooth and the gingiva is known as the junctional epithelium. Important in this attachment is the presence of the basal lamina, a noncellular adhesive supporting sheet comprised of glycoproteins secreted by epithelial cells [14]. The epithelial attachment is the part of the junctional epithelium where the epithelial cells adhere to the tooth surface. The gingival sulcus contains gingival (crevicular) fluid that originates from the adjacent gingival connective tissue. This fluid contains substances with antimicrobial properties, antibodies, and plasma proteins that may improve the adhesion of the epithelial attachment [15]. The amount of this filtration product from the adjacent and highly vascular gingival connective tissue increases during gingival inflammation.

Two basal laminas are involved with the connection of the gingiva to the tooth. An internal basal lamina approximates the surface of the tooth, while an external basal lamina approximates the connective tissue. Most new cells of the junctional epithelium are derived from the germinating cell layer near the external basal lamina. When the lesions of periodontal disease become established, the connective tissue adjacent to the junctional epithelium is damaged. Inflammation associated with periodontal disease also increases the rate of epithelial turnover, which in turn inhibits the development of a healthy stratum of cells and induces migration of cells, resulting in thinned tissue. The damaged proximate connective tissue cannot properly support the junction epithelium against the tooth surface. The unsupported junctional epithelium proliferates in an apical direction, with a consequent deepening of the periodontal pocket [16]. Without the appropriate therapy, this process will involve and destroy the alveolar bone with a consequent loss of teeth.

Attached Gingiva

The keratinized attached gingiva is continuous with the free gingiva and extends to the facial alveolar mucosa of both the mandibular and maxillary arches. Lingually, it is continuous with the loose alveolar mucosa of the mandibular arch and blends imperceptibly with the firmly bound palatal mucosa of the maxillary arch. Its width can vary from 1–9 mm [17]. Unlike the free gingiva, the attached gingiva is tightly bound to the underlying alveolar bone by periosteal gingival fibers [18]. The exterior layer of the attached gingiva is composed of stratified squamous epithelium supported by a connective tissue matrix. There are few elastic fibers in its connective tissue matrix, as the attached gingiva is designed for minimal movement. This histologic consideration allows the attached gingiva to afford the periodontium protection against external injury, stabilize the gingival margin, and absorb the forces of the alveolar mucosa and the frenal attachments [1]. The surface of the attached gingiva has a stippled, "orange peel" texture that is absent from the gingival margin. The microscopic basis for stippling is alternating protuberances and depressions in the underlying tissue. Inflammation and edema from periodontal disease or odontogenic infections that extend into the attached gingiva will reduce or eliminate the stippling; the texture will return after the restoration of tissue health.

A decreasing width of attached gingiva can compromise both periodontal health and the ability to achieve favorable restorative outcomes for fixed prosthetics and implants. Periodontal surgical procedures, such as a free gingival graft or a subepithelial connective tissue graft, are used to restore deficiencies in the zone of attached gingiva.

Interdental Gingiva

The gingival tissues that occupy the gingival embrasure, the interproximal space beneath the contact area of the adjacent teeth, are referred to as the interdental gingiva. It is composed of both free and attached gingiva, and its shape reflects the anatomy of the bordering teeth. In the anterior teeth, for which the interproximal space is narrow and slit-like, the buccal and lingual gingival tissues (known as a gingival papilla) meet in a pinpoint fashion. In the posterior teeth, for which the contact area is much broader, a slight depression exists between the buccal and lingual papillae; this is known as the interdental col. Removing debris from this area requires floss, as tight contact between adjacent teeth usually precludes access to the bristles of a toothbrush. Conditions such as interproximal decay, lost or deficient restorations, and drifted teeth with poor interproximal contacts can cause the impaction of food and complicate oral hygiene, causing inflammation in the interdental gingiva.

CEMENTUM

The calcified tissue covering the roots of the teeth is known as cementum, and it is formed by cells called cementoblasts. By weight, its composition is about 50% inorganic material and 50% organic material and water. Cementum is thinnest at the cementoenamel junction (CEJ), the point of union of the coronal enamel and the cementum of the root [1]. At the CEJ and for the coronal two-thirds of the root, the cementum lacks cellular components and is known as acellular cementum. The more permeable cellular cementum is present in the apical one-third of the tooth [1].

The primary function of the cementum is to provide a surface into which the periodontal ligaments can insert and anchor the tooth to the alveolar bone. There are no blood vessels or nerves within the cementum, and it is not seen intra-orally unless gingival recession and periodontal disease occurs. In periodontal disease, the apical migration of the gingival tissue due to alveolar bone loss exposes the previously protected cementum, which is then susceptible to abrasion and caries. Root planing and scaling procedures upon cementum will not illicit a painful response, as this substance is without innervation. However, sensitivity can occur if exposed cementum is abraded and the previously protected dentinal tubules are subjected to thermal stimuli, acidic substances, or sweets. Likewise, if the cementum is removed during planing, exposure of the underlying dentin may result in pain. Unlike enamel, cementum is formed throughout life, which allows for the continuous reattachment of the fibers of the periodontal ligament.

In response to orthodontic forces, cementum has less of a tendency to be resorbed compared to the alveolar bone. However, small areas of cementum resorption can occur, the deficiencies of which are repaired with material similar to cellular cementum. Stimuli such as chronic periapical inflammation can cause the excessive production of cementum, known as hypercementosis, which is a generalized thickening of the cementum especially prevalent at the root apices. This is a benign condition, but globular masses at the root apex can complicate the ability to extract affected teeth.

Cementomas are benign cementum-producing tumors usually discovered upon routine radiographic examination. These lesions have a predilection for the mandibular incisor region but can involve any tooth. They are generally asymptomatic but can increase in size to a degree at which they are clinically palpable. While cementomas appear in the apical region, they may or may not be attached to the apex of the tooth.

The early development of these lesions features a fibrous composition that is radiolucent (dark) in radiographic appearance. As cementum deposition increases, a progressively more radiopaque (light) appearance will develop. Early lesions can present similar in radiographic appearance to the apical lesions associated with pulpal necrosis. The presence of pulpal caries or deep restorations and vitality testing allow for distinguishing between the lesions. Early radiolucent cementomas may be indistinguishable from initial malignancies of the mandible or maxilla. Referral to an oral surgeon should be considered so a differential diagnosis can be made and an appropriate treatment plan developed.

THE PERIODONTAL LIGAMENT

Ligaments are usually associated with connecting bones together at the joints. The periodontal ligament (PDL) connects the cementum of the tooth to the alveolar bone and has width ranging from 0.15–0.38 mm [4]. Radiographically, it is seen as a narrow black line surrounding the tooth. When widening of the PDL is seen on conventional periapical films, pulpal pathology or systemic medical conditions are usually the etiologic factor. Beyond its support and attachment of the tooth to the bone, it protects the underlying tissue from bacterial invasion. The PDL also has neural input that responds to the sensations of pain, touch, and pressure.

The PDL is composed of an interconnecting meshwork of fibers between the alveolar bone and cementum. Type I collagen fibrils are the principal constituent of the periodontal ligament [23]. Collagen-producing fibroblasts are bountiful in the periodontal ligament, with other cells such as macrophages, osteoblasts, and cementoblasts also present [24].

Individual fibers do not traverse this span in a cable-like fashion [25]. There is diversity among the fibers of the periodontal ligament, and the groups of fibers are classified according to their anatomic location. Beginning in the apical region of the tooth are the apical fibers, which extend from the cementum to the bone at the base of the socket and resist vertical force. Moving in a coronal direction, oblique fibers, the largest fiber group of the PDL, extend from the cementum to the alveolar bone in the apical one-third of the tooth. These fibers transmit much of the vertical masticatory stress to the alveolar bone. Horizontal fibers extend at right angles to the long axis of the tooth between the cementum and alveolar bone. These fibers resist lateral movement and primarily function to retain the tooth within the socket. The alveolar crest fibers extend from the cementum just below the junctional epithelium to the alveolar crest of bone and function in a manner similar to the horizontal fibers. Inter-radicular fibers, which are only present on multi-rooted teeth, extend perpendicularly from the root surface to the alveolar bone, acting to resist vertical and lateral movement [12]. These five fiber groups comprise the principal dentoalveolar fibers, the function of which is to attach the tooth to the alveolar bone. The ends of these fibers, referred to as Sharpey fibers, terminate in either cementum or alveolar bone. The matrix of the PDL also contains capillaries, which are essential for the metabolic needs of the PDL and lymph cells and vessels, which are important to the immunocompetence of the PDL. The resilience of the PDL is afforded by the elastic fibers present throughout the matrix. Oxytalan fibers within the PDL span the long axis of the tooth and provide structural support [1].

Collagen is an essential component of the PDL and requires vitamin C, which the human body cannot synthesize, for its production. A sustained deficiency of vitamin C, resulting in scurvy, adversely affects collagen synthesis and therefore oral health. Without the replenishment of this essential nutrient, the support of the tooth provided by the PDL decreases and tooth loss can occur.

Poorly controlled type 1 or type 2 diabetes can impair the ability of the immune system to provide an adequate defense against pathogenic bacteria, and patients with diabetes are at an increased risk for periodontal disease. In these patients, the direct effect of bacterial toxins and the host inflammatory response can cause realignment and the destruction of the principal fibers of the PDL, the progression of which will lead to the loss of teeth.

ALVEOLAR BONE

The portion of the maxillary or mandibular arch that supports and protects the teeth is known as the alveolar bone. A boundary at the level of the root apices separates the alveolar process from the basal bone of the mandible or the maxilla. The terms alveolar bone and alveolar process are often used interchangeably; for the purpose of this course, the bone that surrounds and supports the teeth in either the mandibular or maxillary arch is considered the alveolar process. Anatomically, the alveolar process can be divided into separate areas, but in support and retention of the teeth, it is one functional unit. It is composed of a thin inner socket wall of compact (cortical) bone called the alveolar bone proper and the supporting alveolar bone, which consists of facial (outer) and lingual (inner) plates of compact bone, interior to which is the spongy cancellous bone. Radiographically, the alveolar bone proper is known as the lamina dura and appears as a radiopaque band that surrounds the tooth root. It is the alveolar bone proper to which the fibers of the periodontal ligament attach. When radiographs reveal loss of density in the lamina dura, it can indicate resorption of this bone as a symptom of inflammation or infection of the periodontal tissues.

When teeth are extracted, the resorption of the alveolar process can significantly decrease the height of the residual ridge, and when all teeth have been extracted, the cumulative shrinkage can present a challenge for the placement of implants or the use of complete dentures. The combined loss of teeth and the alveolar process will decrease the support of the soft tissues of the face and detract from the cosmetic appearance.

The functional unit of compact bone is the osteon, also called the Haversian system, in the center of which is a Haversian canal. This canal provides the neural and vascular supply to the surrounding concentric lamellae in which mature bone-producing cells, osteocytes, reside in lacunae. Microscopic canals called canaliculi interconnect the osteocytes with the neural and vascular supply of the Haversian canal. The lattice-like structures or trabeculae that compose the spongy bone are nourished by diffusion through the canaliculi from capillaries of the endosteum that surround the trabeculae. Interspersed within the trabeculae are deposits of red and yellow bone marrow, with the latter predominating.

The thickness of the outer cortical plates of bone is variable. It is the thinnest in the maxillary arch and the thickest over the mandibular molars and premolars. This is evident in the manner in which local anesthesia of the teeth is obtained. Teeth of the maxillary arch can be anesthetized by the infiltration and subsequent diffusion of a local anesthetic over the bone and roots of the desired teeth. This technique will be unsuccessful for mandibular molars, over which the outer plate of cortical bone is much thicker. In these cases, anesthetizing the inferior alveolar nerve by a regional block technique is required.

Alveolar bone may appear to be a static tissue, but it is subject to remodeling and can undergo change as a result of local and systemic pathologic processes. The dynamic forces of the formation of new bone by osteoblasts and the resorption of bone by osteoclasts are subtle but ever-present. This is the basis for orthodontic treatment. The movement of teeth during orthodontic therapy occurs as osteoclasts are activated and cause the resorption of the alveolar process in the front of the moving teeth while osteoblast-stimulated bone deposition occurs behind the moving teeth. The ideal situation is a balance between these processes, but problems can occasionally occur when osteoclastic activity exceeds osteoblastic activity, resulting in a net loss of the alveolar process and tooth loss.

The progression of gingivitis to periodontal disease is associated with a progressive loss of tissue attachment, deterioration of the periodontal ligament, and loss of the alveolar process. The complex interactions between pathogenic bacteria and the host response that can combine to cause the detachment of the periodontal ligament, the loss of the bone of the alveolar process, and the loss of teeth are many, and it is beyond the scope of this course to address each. However, it is a common problem among adults and a leading cause of tooth loss. Preventive maintenance, good oral hygiene, and early intervention when periodontal disease develops remain the foundation for a healthy periodontium and for the retention of teeth.

Diseases and Conditions Affecting the Alveolar Bone

The basal bone of the maxilla and mandible can be adversely affected by pathologic processes, which subsequently undermines support for the alveolar process. Radiation therapy for oral and pharyngeal malignancies that irradiates bone will cause hyalinization of the associated osseous blood vessels. The decreased perfusion of blood can cause ischemia and ultimately necrosis of the bone. Known as osteoradionecrosis, this condition can occur any time after the completion of radiotherapy and can involve small or large segments of bone that no longer function to support the alveolar process.

Osteoporosis can cause a significant decrease in bone density and a structural deterioration of the osseous architecture. Many studies suggest that patients with osteoporosis have decreased oral bone density and an increased rate of loss of the bone of the alveolar process [28]. Beyond the loss of support for the teeth, bone affected by osteoporosis has an increased risk of pathologic fracture.

THE ORAL MUCOSA

Within the restricted confines of the oral cavity, the variation of the composition of the oral mucosa reflects its multiple functions. An intact oral mucosa protects the underlying tissues from desiccation, physical trauma, chemical and mechanical irritation, and the penetration of pathogenic micro-organisms from the oral cavity. The integrity of the oral mucosa can be compromised due to mechanical trauma, various oral lesions, and ulcerative mucosal lesions that arise from chemotherapy for systemic malignancies or radiotherapy used to treat oral and pharyngeal malignancies. In such cases, bacterial and fungal organisms have access to deeper tissue layers and can cause local, regional, or systemic infections of varying severity. Speaking, chewing, and swallowing all depend upon harmony among the varied components of the oral mucosa. This section will review how the functions of the oral mucosa reflect the underlying histology.

KERATINIZED (MASTICATORY) MUCOSA

All of the oral mucosal tissues have an outer layer of stratified squamous epithelial cells, referred to as the mucosa proper, and an underlying connective tissue layer, known as the lamina propria. The latter layer contains numerous small loops of blood vessels, nerve fibers, and a dense network of collagen fibers that provide support for the mucosa proper. Keratinized tissue incorporates the protein keratin into the surface layers of the mucosa proper. Keratinization increases the tissue's strength and resistance to mechanical forces, but it decreases the flexibility.

Tissues of the oral mucosa that are keratinized include the mucosa that overlies the hard palate, the attached gingiva, and the external surface of the free gingiva. The internal epithelial surface of the free gingiva that constitutes the crevicular (sulcular) epithelium and the junctional epithelium are not keratinized. The dorsal surface of the tongue has keratinized and non-keratinized areas and is considered specialized mucosa. These tissues will be discussed later in this section.

Masticatory mucosa is designed to withstand compressive and shearing forces during mastication and the abrasive nature of food against soft tissue. In order for the masticatory mucosa to perform these functions and retain its original shape, the tissue must be firmly secured. The junction between the epithelial cells of the mucosa proper and the underlying lamina propria is a convoluted border that extends the available surface area for attachment and allows for a stronger union between the layers. In the free and attached gingiva, fingerlike extensions called rete pegs extend from the epithelial layer into the lamina propria and provide a more secure mechanical attachment [29]. The epithelial attachment of the junctional epithelium secures the attached gingiva to the tooth. Because there are few elastic fibers in the connective tissue, movement and flexibility in the gingiva is ideal in order to withstand masticatory forces and protect the underlying tissue [17].

Collagen fibers in the lamina propria (which collectively form the gingival ligament) aid in securing the free and attached gingiva to the cementum and the bone of the alveolar process, but they are not strictly considered to be a part of the periodontal ligament. The dentogingival fibers are the most numerous and extend from the cementum at the cervical aspect of the root to the lamina propria of both the free and attached gingiva. The alveologingival fibers connect the bone of the alveolar crest with the lamina propria. A group of fibers encircle the tooth in the cervical area and band other proximate fiber groups together to attach the free gingiva to the tooth. The two remaining fiber groups, the dentoperiosteal fibers and the transeptal fibers, do not have attachments with the free or attached gingival [4].

The free and attached gingiva do not have a distinct submucosal layer. The attached gingiva is tightly bound via dense collagen fibers to the periosteum, a double layer of connective tissue that overlies and nourishes the bone of the alveolar process. The juncture of the mucosa proper and lamina propria with the periosteum is called the mucoperiosteum. The concerted effort of these varied collagenous fiber connections provides the gingival tissue with the support and resilience needed to consistently function as masticatory mucosa.

The mucosa that overlies the hard palate is also considered masticatory mucosa, as the tongue thrusting a bolus of food against the hard palate aids in the breakdown of food. In addition, complete or partial dentures use the unyielding hard palate as a sturdy foundation to which masticatory stresses can be transferred. The palatal mucosa is bound tightly to the bone of the hard palate by dense connective tissue. Pronounced ridges of epithelial tissue called rugae extend horizontally across the anterior region of the hard palate and also assist with food breakdown.

The demarcation of the midline of the hard palate is known as the median raphe, under which no submucosa exists. The lateral regions of the anterior hard palate have a submucosa that contains adipose tissue. Beneath the surface of the posterior and lateral region of the hard palate are minor salivary glands that produce only mucous secretions. Local anesthetic injections are more painful on the hard palate and surgical flaps are more difficult to create because the mucosa is bound so tightly to the underlying palatal bone.

LINING MUCOSA

Most of the mucosa present in the oral cavity is categorized as lining mucosa, which is flexible and distensible. This includes the mucosa of the inside of the lips (labial) and cheeks (buccal), the ventral surface of the tongue and the floor of the mouth beneath it, and the alveolar mucosa beginning at the attached gingiva and blending with the labial or buccal mucosa. Lining mucosa consists of stratified squamous epithelium that, unlike the masticatory mucosa, is not keratinized. This tissue is designed for movement during function, and the histology reflects this function.

The collagen fibers are thinner and less organized than those present in the masticatory mucosa. Elastic fibers, which are scarce in the tightly bound masticatory mucosa, are more plentiful in the lining mucosa. This is because this tissue is designed to be stretched and moved but must have the elasticity and resilience to return to its original shape. The submucosal layer of the lining mucosa is also loosely organized, which further facilitates its freedom of movement. Lining mucosa has a well-developed capillary network, resulting in its red hue. Lining mucosa has a glossy and smooth appearance. The rete pegs that extend from the epithelium into the lamina propria are shorter than those seen in the masticatory mucosa, which also confers a higher degree of tissue mobility. The thinness of the tissue makes it more prone to injury compared to the thicker and more secured masticatory mucosa; however, its cells have a fast turnover rate, so healing occurs quickly.

All lining mucosa has a submucosal layer, but its attachment to the underlying tissues varies by region. The submucosa of the labial and buccal mucosa has a firm attachment to the underlying muscle, while the tissue in the floor of the mouth is loosely attached to the underlying muscles. The submucosa of the alveolar mucosa maintains a loose attachment with the underlying periosteum of the maxilla or mandible. The lining mucosa of the ventral surface of the tongue is thin, with a submucosal layer that is not distinct but is attached to the underlying muscles [3]. Most malignant oral lesions initially present on lining mucosa, as the thinness facilitates the infiltration of malignant cells. In particular, intraoral squamous cell carcinomas have a predilection for the floor of the mouth and the ventral surface of the tongue.

SPECIALIZED MUCOSA OF THE TONGUE

The mucosa on the superior surface or dorsum of the tongue is an entirely different entity in structure and function compared to the ventral (inferior) surface of the tongue. This tissue contains specialized elements to facilitate the sensation of taste and the function of mastication.

The tongue is divided into the anterior two-thirds (or body) and the posterior one-third (or base) and each has different embryologic origins. The dorsum of the tongue is masticatory mucosa, the epithelium of which contains numerous projections called papillae, most of which are filiform papillae. These flame-like projections have a surface layer of keratinized stratified squamous epithelium suited for the masticatory function of compressing a bolus of food against the hard palate. The mucosa between the filiform papilla is not keratinized. There are no taste buds present in the filiform papillae.

Some patients may develop benign migratory glossitis (or geographic tongue), a condition that involves the filiform papillae. With this condition, the temporary loss or desquamation of the filiform papillae leaves irregular and erythematous areas, although the location of these areas can change daily. The papillae will regenerate, but the condition often recurs. The etiology of these lesions is unknown, although genetic, autoimmune, and environmental factors have been implicated. There is no common or universally effective treatment, and no cure is available. The exposed mucosal surfaces can be sensitive to thermal, acidic, or mechanical stimuli, so treatment is usually palliative. Although the lesions resolve spontaneously, they should be monitored and any lesions that do not heal should be biopsied.

Scattered among the filiform papillae on the dorsum of the tongue are the mushroom-shaped fungiform papillae that contain taste buds on their superior surface. The fungiform papillae have a thin epithelial surface that may or may not be keratinized and a vascular connective tissue core, which gives the structures a red appearance. The fungiform papillae are fewer in number but larger than the filiform papillae.

Foliate papillae are found in grooves along the posterior and lateral surface of the tongue. These papillae are larger than the fungiform papillae but are fewer in number. The epithelial surface is not keratinized. There are several taste buds present in the lateral borders of the foliate papillae bilaterally.

The circumvallate papillae are located at the junction of the body and base of the tongue. They are large, round structures, each surrounded by a trough. The superior surface of the circumvallate papillae features a keratinized connective tissue core. However, the epithelium of the lateral walls is not keratinized, and numerous taste buds are present on this surface. Minor salivary glands, the glands of von Ebner, secrete serous fluid into the base of the trough that surrounds these papillae, which is a self-cleansing mechanism and allows for taste renewal.

Effects of Radiation on Taste Buds

Taste buds are very sensitive to the ionizing radiation used postsurgically for the treatment of oral, head, and neck malignancies. Cumulative doses of as little as 6,000 centigray (cGy) of radiation can cause irreversible damage to the taste buds, and this level is reached and even exceeded for most patients receiving radiation [33]. The location of the lesion being irradiated and the total cumulative dose of radiation will determine which taste buds are damaged and to what extent. Radiation-induced damage of the salivary glands can compound this problem, as food particles not dissolved by saliva will not come into contact with the taste buds.

THE VERMILION ZONE OF THE LIPS

The vermilion zone is the margin of the lips between the skin on the exterior surface and the labial mucosa on the interior surface. This thin epithelial layer is keratinized, with capillary loops in its connective tissue core that allow a copious surface perfusion of blood and account for the vermilion (red) color of the lips. The tissue of the vermilion zone is considered a specialized mucosa. It has a dense submucosal layer with a firm attachment to the underlying musculature [34].

There are no mucous glands in the vermilion zone, so saliva is necessary to keep the area moist. Excessive desiccation of the vermilion zone due to extremes of temperature or excessive exposure to the wind leads to the condition known as "chapped lips." Commercial lip balms can palliate the symptoms associated with this problem.

This area may also show evidence of sun damage. The vermilion zone of the lower lip has more exposure to solar radiation compared to the slightly invaginated upper lip and is thus more prone to the development of squamous cell carcinoma.

THE SALIVARY GLANDS

Before the major and minor salivary glands and their unique histology can be discussed, it is essential to review the main components of saliva and their contribution to oral health. Saliva is a unique substance that functions as a tissue lubricant, as a medium for the remineralization of enamel, as an antimicrobial agent, and as a critical component in the self-cleansing mechanism for the teeth. Adequate salivary flow is essential for good oral health, as the decreased flow of saliva increases retention of plaque on the teeth and the mucosa and thus increases the risk of caries and periodontal disease. It is 99.5% water, with an assortment of 0.5% solutes accounting for the remainder [35].

Mucous secretions in saliva provide tissue lubrication, which is especially significant for patients who wear partial or complete dentures. Inadequate lubrication of the tissues supporting these prostheses can cause ulcerations and preclude their use. Immunoglobulin A (IgA), which can inhibit the adherence of some strains of streptococci bacteria to the oral mucosa, is secreted in saliva from the parotid salivary glands and the minor salivary glands [36]. Lysozyme, a hydrolytic enzyme secreted by the major and minor salivary glands, disrupts the structural integrity of the cell walls of certain bacteria [22]. Amylase is an enzyme in saliva that begins the breakdown of carbohydrates contained in food while it is in the oral cavity. Remineralization of small areas of enamel is mediated through an exchange of calcium and phosphate ions present in the saliva. There are trace amounts of several other organic and inorganic solutes in saliva that contribute to the overall health of the oral and maxillofacial complex.

Saliva originates in one of the three paired major salivary glands (parotid, sublingual, and submandibular) and numerous minor salivary glands. The basic secreting units of each gland are the acini. Serous acini produce a watery secretion, and mucous acini produce a thick, viscous secretion. The parotid gland produces serous secretions only, while the sublingual and submandibular glands produce both mucous and serous secretions. The minor salivary glands are widely distributed in the mucosa and submucosa of the oral cavity and produce predominantly mucous secretions. The major salivary glands are considered tubuloacinar, as their secretions are produced by acini and the initial portion of the duct system that transports these secretions into the oral cavity [34]. They are also considered exocrine glands, because their secretions must be brought to the surface by a series of ducts and tubes. This is in contrast to endocrine glands, such as the pituitary and adrenal glands, whose hormones enter the bloodstream directly. The production of saliva is an involuntary activity regulated by the sympathetic and parasympathetic divisions of the autonomic nervous system.

THE PAROTID GLAND

The parotid salivary glands are probably the best known among the major salivary glands. They are the largest among the major salivary glands, but they produce only about 20% of the total saliva [27]. Each parotid gland is wrapped in a subcutaneous connective tissue capsule located anterior and inferior to the ear and lies to the exterior of both the masseter and buccinators muscles. The parotid (Stensen) duct pierces the buccinator muscle and enters the oral cavity in a papilla on the buccal mucosa opposite the maxillary first or second molar area. As noted, the secretions of the parotid gland are purely serous in nature. The acinar cells that produce the secretions appear pyramidal and have an abundance of secretory vesicles called zymogen granules within the cytoplasm. Alpha amylase, a substance that begins the digestion of starches, is produced by the serous acini [1]. The ducts in the parotid gland are more than a conduit for saliva to the oral cavity; they are also involved in the secretion and absorption of varied substances.

The ducts originating in the secreting acini are narrow and intercalated and absorb chloride from and secrete bicarbonate ion into the acinar secretion. These ducts are lined with cuboidal or columnar cells. Striated (secretory) ducts continue from the intercalated ducts and resorb sodium and secrete potassium. A series of smaller excretory ducts merge to form a larger excretory duct that will discharge the saliva into the oral cavity. Myoepithelial cells with actin-containing contractile filaments lie adjacent to the acinar secretory cells and the intercalated ducts. The contraction of these myoepithelial cells helps to propel the contents of the secreting acinar cells into the lumen of the ducts and beyond. Bicarbonate secretion from the parotid gland is an important component of the buffering capacity of the oral cavity. A decrease in the bicarbonate production causes an increase in the acidity (lower pH) of the oral environment, which favors the proliferation of cariogenic bacteria and the resident fungal organism *Candida albicans*.

THE SUBMANDIBULAR GLAND

The submandibular gland is considered a mixed gland because its secretions are both serous and mucous, though serous secretions predominate. These bilateral glands are located inferior and medial to the posterior aspect of the mandible and superior to the digastric muscles. They enter the oral cavity via Wharton ducts. Small mucosal papilla (called sublingual caruncles) near the anterior midline on either side of the lingual frenum in the floor of the mouth mark the entrance of Wharton ducts into the oral cavity. Submandibular glands are smaller in size than the parotid glands, but they account for approximately 70% of salivary production [1]. The duct system and myoepithelial cells associated with the serous acini of the parotid gland remain unchanged in the submandibular gland [3]. However, mucous acini are also present and are responsible for the production of mucus, a glycoprotein-laden, viscous secretion whose primary functions are lubrication and protection of the tissue [31]. Each mucous cell contains numerous membrane-bound secretory granules, and the collective amount of mucus within these granules compresses the nucleus within each cell toward the cell membrane. After these granules have discharged their contents into the lumen of each acinar unit, the compression of the nucleus against the cell membrane is relaxed. Microscopically, the mucous acini have a pale appearance compared to the darker-staining granules of the serous acini.

THE SUBLINGUAL GLAND

The sublingual gland is also a mixed gland, with the mucous secretions predominating over the serous secretions. It is the smallest of the major salivary glands and produces about 5% of the salivary volume [27]. The sublingual gland is anterior to the submandibular gland and lies just beneath the mucosa of the floor of the mouth, between the mandible and the genioglossus muscle. Its inferior border is the mylohyoid muscle [30]. Unlike the parotid and submandibular glands, the sublingual gland does not have a connective tissue capsule sur-

rounding it nor is it a single unit. It is comprised of a posterior portion (the greater sublingual gland) and an anterior portion (the lesser sublingual gland) with a duct system for each portion. The greater sublingual gland drains into a main duct (Bartholin duct), while the lesser sublingual gland drains into smaller multiple ducts (the ducts of Rivinus) [23]. Bartholin duct may unite with Wharton duct or may open in the floor of the mouth with individual sublingual papilla. The basic histology of the mucous and serous units is consistent with that of the parotid and submandibular glands.

MINOR SALIVARY GLANDS

As noted, the minor salivary glands are scattered throughout the oral cavity. Their highest concentration exists in the buccal, labial, palatal, and lingual mucosa. There is one duct per secreting unit rather than a main duct, like that present in the major salivary glands. The minor salivary glands may be serous, mucous, or mixed, and together they secrete about 5% of all the saliva produced. Mucus is the primary secretion of the minor salivary glands. Approximately 70% of all the mucus secreted originates from the minor salivary glands [12]. Like the sublingual glands, the minor glands have a low but continuous level of salivary production.

SALIVARY GLAND PATHOLOGY

The multiple functions of saliva are often unappreciated until their reduction is of sufficient magnitude to adversely affect oral health and overall quality of life. This section will highlight a few among the numerous pathologic entities that directly target the salivary glands and can cause an interference with their appropriate functions.

Radiation-Induced Xerostomia

Radiation therapy administered for oral or head and neck malignancies may damage the salivary glands, even if efforts are made to shield them from collateral damage. Common cumulative doses of radiation for oral malignancies (most often squamous cell carcinomas) fall into the range of 5,000–7,000 cGy. The serous acini of the major and minor salivary glands can experience a 50% decrease in their secretory activity when a cumulative dose of 1,000 cGy of radiation has been reached [26]. When a dose of 4,000 cGy of radiation has been achieved, a permanent reduction in salivary production from irradiated glands will occur [21].

The resulting xerostomia (dry mouth) is irreversible. For these patients, even the basic functions of life, such as eating, chewing, speaking, and swallowing, can become challenging. A decrease in the self-cleansing action of saliva and the antibacterial actions of lysozyme and IgA can result in a greater incidence of caries and periodontal disease. A decrease in the lubricating function in saliva can make the use of partial or complete dentures difficult or impossible.

Sjögren Syndrome

Sjögren syndrome is an autoimmune disease affecting the moisture-producing glands of the body. It features keratoconjunctivitis sicca (dry eyes) and xerostomia (dry mouth) as two of the classic presentations. The third presentation is a systemic connective tissue disease similar to lupus or rheumatoid arthritis. Approximately 4 million Americans have this disease, about 90% of whom are women [20]. The pathophysiology of Sjögren syndrome is characterized by a progressive infiltration of CD4+ and T-cell lymphocytes and plasma cells into the supporting tissue and acinar units of the salivary glands.

Fibrosis of the affected salivary gland tissue ensues, followed by a permanent decrease in the production of saliva [19]. These patients often experience the same oral health and quality of life issues seen in patients with oral cancer after radiation therapy. Patients with Sjögren syndrome may also have compromised dexterity, which can decrease their ability to brush and floss properly. Impaired ability to perform oral hygiene coupled with increased plaque retention due to hyposalivation can seriously undermine the oral health of these patients.

Medication-Induced Xerostomia

Many medications can decrease the flow of saliva as a known side effect. In some cases, alternative medications may provide the equivalent pharmacologic efficacy, but this is not always an option. Long-term or lifetime use of medications that cause xerostomia requires patients to maintain excellent oral hygiene. Periodic recall appointments should be scheduled on the basis of the patient's ability to maintain oral hygiene.

Sialolithiasis

Sialolithiasis is a benign condition in which calcium-laden crystalized minerals known as sialoliths, salivary calculi, or salivary stones become deposited in a salivary gland or a salivary duct. It is the most common disease affecting the salivary glands. More than 80% of cases occur in the submandibular gland or its duct, 6% involve the parotid gland, and only about 2% involve the sublingual and minor salivary glands [11]. The exact etiology of sialoliths is not known.

Symptoms of sialolithiasis can include pain, swelling, and even suppuration in the affected area. Palpation, occlusal radiographs, and imaging studies aid in the diagnosis. Smaller sialoliths may be flushed out if the patient increases fluid intake and sialogogues are taken to stimulate salivary production. Warm, moist heat coupled with massage of the affected duct or gland may also be helpful [6]. Surgical removal is usually required for larger sialoliths that do not respond to more conservative means of treatment.

CONCLUSION

The oral environment is a unique conglomeration of diverse hard and soft tissues with varied cellular compositions. These tissues are coordinated together in a manner that allows us to speak, masticate, swallow, and enjoy a positive quality of life. The functions of the teeth, mucosa, and salivary glands are a reflection of the underlying cellular composition.

This course has provided a brief overview of the histology and physiology of the structures treated by members of the dental team each day. The complexity of the cellular composition and physiology of structures discussed should instill a sense of respect and a renewed commitment to treat the teeth and the oral mucosa in a manner that promotes optimal oral and overall health and the best possible quality of life.

Works Cited

1. Scheid RC, Weiss G. *Woelfel's Dental Anatomy*. 9th ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2016.
2. Boskey AL. Mineralization of bones and teeth. *Elements*. 2007;3:387-393.
3. Berkowitz BKB, Holland GR, Moxham BJ. *Oral Anatomy, Embryology and Histology*. 5th ed. revised. Edinburgh: Mosby; 2017.
4. Antonio N. *Ten Cate's Oral Histology: Development, Structure and Function*. 9th ed. St. Louis, MO: Elsevier; 2017.
5. U.S. Department of Health and Human Services. Healthy People 2030: Oral Conditions. Available at https://health.gov/healthypeople/objectives-and-data/browse-objectives/oral-conditions. Last accessed January 17, 2023.
6. Pietz DM, Bach DE. Submandibular sialolithiasis. *Gen Dent*. 1987;35:494-496.
7. Rogers AH (ed). *Molecular Oral Microbiology*. Norfolk: Caister Academic Press; 2008.
8. Cochrane NJ, Cai F, Huq NL, Burrow MF, Reynolds EC. New approaches to enhanced remineralization of tooth enamel. *J Dent Res*. 2010;89(11):1187-1197.
9. U.S. Department of Health and Human Services Federal Panel on Community Water Fluoridation. U.S. Public Health Service recommendation for fluoride concentration in drinking water for the prevention of dental caries. *Public Health Rep*. 2015;130(4):318-331.
10. Buzalaf MA, Pessan JP, Honório HM, ten Cate JM. Mechanisms of action of fluoride for caries control. *Monogr Oral Sci*. 2011;22:97-114.
11. Siddiqui SJ. Sialolithiasis: an unusually large submandibular salivary stone. *Br Dent J*. 2002;193:89-91.
12. Avery JK, Steele PF, Avery N (eds). *Oral Development and Histology*. 3rd ed. New York, NY: Thieme Medical Publishers; 2002.
13. Love RM, Jenkinson HF. Invasion of dentinal tubules by oral bacteria. *Crit Rev Oral Biol Med*. 2002;13(2):171-183.
14. Marieb EN, Hoehn K. *Human Anatomy and Physiology*. 11th ed. San Francisco, CA: Pearson; 2018.
15. Beltrami D. Gingival Crevicular Fluid. Available at http://flipper.diff.org/app/items/info/379. Last accessed January 17, 2023.
16. Kinane DF, Mombelli A. *Periodontal Disease*. Basel: Karger; 2012.
17. Oh SL. Attached gingiva: histology and surgical augmentation. *Gen Dent*. 2009;57(4):381-385.
18. Reddy S. *Essentials of Clinical Periodontology and Periodontics*. 5th ed. New Delhi: Jaycee Bros. Medical Publishers; 2017.
19. Kerr AR, Miller CS, Rhodus NL, Stoopler ET, Treister NS (eds). *Little and Falace's Dental Management of the Medically Compromised Patient*. 10th ed. St. Louis, MO: Mosby; 2023.
20. Sjögren's Syndrome Foundation. Understanding Sjögren's. Available at https://sjogrens.org/understanding-sjogrens. Last accessed January 17, 2023.
21. Barker BF, Barker GJ. Oral management of the patient with cancer in the head and neck region. *J Calif Dent Assoc*. 2001;29(8): 619-623.
22. Salama R. Biochemistry of Saliva. Available at https://www.slideshare.net/drwalidsalem/composition-of-saliva-2010. Last accessed January 17, 2023.
23. Smith SK, Karst NS. *Head and Neck Histology and Anatomy: A Self-Instructional Program*. Stamford, CT: Prentice Hall; 1999.
24. Sadatullah S. Periodontal Ligament. Available at https://www.slideshare.net/syedsadatullah/periodontal-ligament-6056478. Last accessed January 17, 2023.
25. Torabi S, Soni A. *Histology, Periodontium*. Treasure Island, FL: StatPearls Publishing; 2022.
26. Baharudin A, Khairuddin A, Nizam A, Samsuddin AR. Evaluation of irradiated salivary gland function in patients with head and neck tumors treated with radiotherapy. *J Laryngol Otol*. 2009;123(1):108-113.
27. Quizlet. Flashcards: DHYG Glands/Ducts. Available at https://quizlet.com/3417308/dhyg-glandsducts-flash-cards. Last accessed January 17, 2023.
28. Sidiropoulou-Chatzigiannis S, Kourtidou M, Tsalikis L. The effect of osteoporosis on periodontal status, alveolar bone and orthodontic tooth movement: a literature review. *J Int Acad Periodontal*. 2007;9(3):77-84.
29. Fiorellini JP, Kim D, Chang Y-C. Anatomy, structure, and function of the periodontium. In: Newman MG, Takei HH, Klokkevold PR, Carranza FA (eds). *Newman and Carranza's Clinical Periodontology*. 13th ed. Philadelphia, PA: Saunders; 2018: 19-49.
30. Rosen FS. Anatomy and Physiology of the Salivary Glands. Available at https://liveutmb.sharepoint.com/sites/collaboration/webfiles/Shared%20Documents/Forms/AllItems.aspx?ga=1&id=%2Fsites%2Fcollaboration%2Fwebfiles%2FShared%20Documents%2FOtolaryngology%20Department%2FWebsite%20Documents%2FQuinn%27s%20Website%20Documents%2FGrand%20Rounds%2F2001%2F1%2E24%2E2001%20Anatomy%20of%20the%20Salivary%20Gland%20Text%2EPDF&parent=%2Fsites%2Fcollaboration%2Fwebfiles%2FShared%20Documents%2FOtolaryngology%20Department%2FWebsite%20Documents%2FQuinn%27s%20Website%20Documents%2FGrand%20Rounds%2F2001. Last accessed January 17, 2023.
31. Histology at Southern Illinois University School of Medicine. Glands of the Gastrointestinal System. Available at http://www.siumed.edu/~dking2/erg/glands.htm. Last accessed January 17, 2023.
32. Wiegand A, Schwerzmann M, Sener B, et al. Impact of toothpaste slurry abrasivity and toothbrush filament stiffness on abrasion of eroded enamel: an in vitro study. *Acta Odontol Scand*. 2008;66(4):231-235.

33. Gomes Carvalho C, Medeiros-Filho JB, Coelho Ferreira M. Guide for health professionals addressing oral care for individuals in oncological treatment based on scientific evidence. *Support Care Cancer*. 2018;26(8):2651-2661.

34. University of Western Australia. Blue Histology: Oral Cavity and Oesophagus. Available at http://lecannabiculteur.free.fr/SITES/UNIV%20W.AUSTRALIA/mb140/CorePages/Oral/oral2.htm. Last accessed January 17, 2023.

35. de Almeida Pdel V, Grégio AM, Machado MA, de Lima AA, Azevedo LR. Saliva composition and functions: a comprehensive review. *J Contemp Dent Pract*. 2008;9(3):72-80.

36. Nieves A, Fitzgerel-Blue W. Saliva: Of Emerging Importance in the Medical and Dental Worlds. Available at https://www.rdhmag.com/pathology/temporomandibular-joint-dysfunction/article/16407983/saliva-of-emerging-importance-in-the-medical-and-dental-worlds. Last accessed January 17, 2023.