4. ORAL PATHOLOGY

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DEVELOPMENTAL CONDITIONS

Tooth-related Problems

1. Describe the different types of dentinogenesis imperfecta.

Dentinogenesis imperfecta (Dl) causes the teeth to be opalescent and affects both the primary and permanent dentition.

Type I	Dl with osteogenesis imperfecta
Type II	DI without osteogenesis imperfecta
Type III	Brandywine type, which also occurs in the absence of osteogenesis imperfecta but is clustered within a racial isolate in Maryland. In addition to classic findings of DI, radiographs may exhibit multiple periapical radiolucencies, and large pulp chambers may lead to multiple pulp exposures.

2. What is the difference between fusion and concrescence? Twinning and gemination?

Fusion is a more complete process than concrescence and may involve either (1) fusion of the entire length of two teeth (enamel, dentin, and cementum) to form one large tooth, with one less tooth in the arch, or (2) fusion of the root only (dentin and cementum) with the maintenance of two clinical crowns. Concrescence involves fusion of cementum only.

Twinning is more complete than gemination and results in the formation of two separate teeth from one tooth bud (one extra tooth in the arch). In gemination, separation is attempted, but the two teeth share the same root canal.

3. What is a Turner's tooth?

A Turner's tooth is a solitary, usually permanent tooth with signs of enamel hypoplasia or hypocalcification. This phenomenon is caused by trauma or infection in the overlying deciduous tooth that damages the ameloblasts of the underlying tooth bud and thus leads to localized enamel hypoplasia or hypocalcification.

4. What are "bull teeth"?

Bull teeth, also known as taurodonts, have long anatomic crowns, large pulp chambers, and short roots, resembling teeth found in bulls. They are most dramatic in permanent molars but may affect teeth in either dentition. They occur more frequently in certain syndromes, such as Klinefelter syndrome.

5. What is the difference between dens evaginatus and dens invaginatus?

Dens evaginatus occurs primarily in persons of mongoloid descent and affects the premolars. **Evagination** of the layers of the tooth germ results in the formation of a tubercle that arises from the occlusal surface and consists of enamel, dentin, and pulp tissue. This tubercle tends to break when it occludes with the opposing dentition and may result in pulp exposure and subsequent pulp necrosis. Dens invaginatus occurs mainly in maxillary lateral incisors and ranges in severity from an accentuated lingual pit to a "dens in dente." This phenomenon is caused by **invagination** of the layers of the tooth germ. Food becomes trapped in the pit, and caries begin early.

6. What are the causes of generalized intrinsic discoloration of teeth?

Amelogenesis imperfecta	Fluorosis	Porphyria
Dentinogenesis imperfecta	Rh incompatibility	Biliary atresia
Tetracycline staining		

7. Why do teeth discolor from ingestion of tetracycline during odontogenesis?

Tetracycline binds with the calcium component of bones and teeth and is deposited at sites of active mineralization, causing a yellow-brown endogenous pigmentation of the hard tissues. Because teeth do not turn over like some bone tissues, this stain becomes a permanent "label" that fluoresces under ultraviolet light.

8. Which teeth are most commonly missing congenitally?

Third molars, maxillary lateral incisors, and second premolars.

9. What conditions are associated with multiplesupernumerary teeth?

Gardner's syndrome and cleidocranial dysplasia.

10. What are the most common sites for supernumerary teeth?

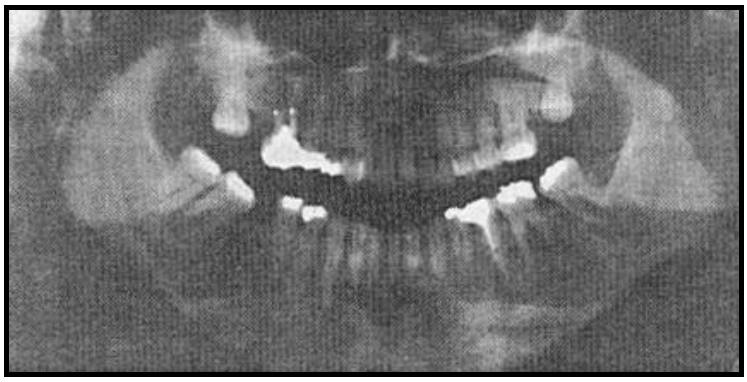
Midline of the maxilla (mesiodens), posterior maxilla (fourth molar or paramolar), and mandibular bicuspid areas.

Intrabony Lesions

11. A 40-year-old African-American woman presents with multiple radiolucencies and radiopacities. What is the diagnosis?

The African-American population is prone to developing benign fibroosseous lesions of various kinds. They range from localized lesions, such as periapical cemental dysplasia involving one tooth (usually mandibular anterior), to florid cementoosseous dysplasia, involving all four quadrants. The second condition also

has been referred to as familial gigantiform cementoma, multiple enostoses, and sclerotic cemental masses.



Florid cementoosseous dysplasia affecting at least three quadrants.

12. Are fibrous dysplasias of bone premalignant lesions?

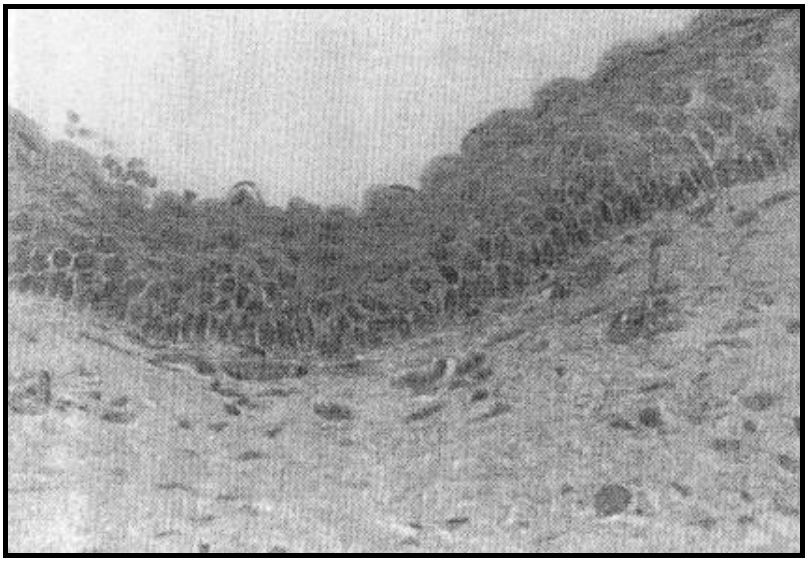
Fibrous dysplasia, a developmental malformation of bone, is of unknown etiology and is not premalignant. The monostotic form often affects the maxilla unilaterally. The polyostotic form is associated with various other abnormalities, such as skin pigmentations and endocrine dysfunction (Albright and Jaffe-Lichtenstein syndromes). Cherubism, which used to be termed familial fibrous dysplasia, is probably not a form of fibrous dysplasia. In the past, fibrous dysplasia was treated with radiation, which sometimes caused the development of osteosarcoma.

13. The globulomaxillary cyst is a fissural cyst. True or false?

False. Historically, the globulomaxillary cyst was classified as a nonodontogenic or fissural cyst thought to result from enclavement of epithelial rests along the line of fusion between the lateral maxillary and nasomedial processes. Current thinking puts it in the category of odontogenic cysts, probably of developmental origin and possibly related to the development of the lateral incisor or canine. The two embryonic processes mentioned above do not fuse. The fold between them fills in and becomes erased by mesodermal invasion so that there is no opportunity for trapping of epithelial rests. This cyst occurs between the roots of the maxillary lateral incisor and cuspid, both of which are vital.

14. The median palatal cyst is a true fissural cyst. True or false?

True. The epithelium of this intrabony cyst arises from proliferation of entrapped epithelium when the right and left palatal shelves fuse in the midline. The soft tissue counterpart, which also occurs in the midline of the palate and is known as the palatal cyst of the newborn (Epstein's pearl), is congenital and exteriorizes on its own. The histology is similar to that of dental lamina cysts of the newborn (see below).



Classic parakeratinized odontogenic keratocyst.

15. A neonate presents with a few white nodules on the mandibular alveolar ridge. What are they?

They are most likely dental lamina cysts of the newborn (Bohn's nodules). The epithelium of these cysts arises from remnants of dental lamina on the alveolar ridge after odontogenesis. Dental lamina cysts of the newborn tend to involute and do not require treatment.

16. A boy presents to the dental clinic with multiple jaw cysts and a history of jaw cysts in other family members. What syndrome does he most likely have?

The boy most likely has the bifid rib-basal cell nevus syndrome, which is inherited as an autosomal dominant trait. The cysts are odontogenic keratocysts, which have a higher incidence of recurrence than other odontogenic cysts. Other findings include palmar pitting, palmar and plantar keratosis, calcification of the falx cerebri, hypertelorism, ovarian tumors, and neurologic manifestations such as mental retardation and medulloblastomas.

17. Are all jaw cysts that produce keratin considered odontogenic keratocysts?

Yes and no. The odontogenic keratocyst is a specific histologic entity. The epithelial lining exhibits corrugated parakeratosis, uniform thinness (unless altered by inflammation), and palisading of the basal cell nuclei. The recurrence rate is high, and the condition is associated with the basal cell-bifid rib nevus syndrome. Odontogenic cysts that produce orthokeratin do not show the basal cell nuclei changes, do not have the same tendency to recur, and are not associated with the syndrome. However, some pathologists use the term "orthokeratinized variant" after odontogenic keratocyst to denote the difference, whereas others use the term "orthokeratinizing odontogenic cyst." The clinical differences are important.

18. What neoplasms may arise in a dentigerous cyst?

Ameloblastoma, mucoepidermoid carcinoma, and squamous cell carcinoma may arise in a dentigerous cyst. Odontogenic tumors that may arise in a dentigerous relationship, although not within a dentigerous cyst, include adenomatoid odontogenic tumor and calcifying epithelial odontogenic tumor (Pindborg tumor).

19. What is the difference between a lateral radicular cyst and a lateral periodontal cyst?

A lateral radicular cyst is an **inflammatory** cyst in which the epithelium is derived from rests of Malassez (like a periapical or apical radicular cyst). It is-in a lateral rather than an apical location because the inflammatory stimulus is emanating from a lateral canal. The associated tooth is always nonvital. The lateral periondontal cyst is a **developmental** cyst in which the epithelium probably is derived from rests of dental lamina. It is usually located between the mandibular premolars, which are vital.

20. What is the incidence of cleft lip and/or cleft palate?

Cleft lip and cleft palate should be considered as two entities: (1) cleft palate alone and (2) cleft lip with or without cleft palate. The former is more common in females and the latter in males. The incidence of cleft palate alone is 1 in 2,000—3,000 births, whereas the incidence of cleft lip with or without cleft palate is 1 in 700—1,000 births. Of all cases, 25% are cleft palate alone and 75% are cleft lip with or without cleft palate.

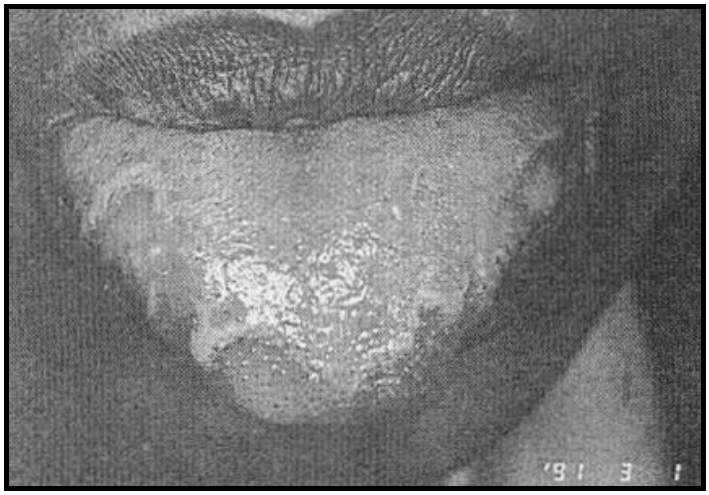
Soft Tissue Conditions

21. Name the organism that colonizes lesions of median rhomboid glossitis.

Candida sp. colonizes the lesions but probably is not the cause because in many instances, even with elimination of candidal organisms, the area of papillary atrophy persists. Some investigators have reverted to the original hypothesis that median rhomboid glossitis is a developmental malformation, possibly caused by failure of the tuberculum impar to retract completely.

22. Is benign migratory glossitis ("geographic tongue") associated with any systemic conditions?

Most cases of benign migratory glossitis occur in the absence of a systemic condition, although some cases have been associated with fissured tongue. However, patients with psoriasis, especially generalized pustular psoriasis, have a higher incidence of benign migratory glossitis.



Benign migratory glossitis.

23. What predisposes to the formation of a hairy tongue?

Hyposalivation, broad-spectrum antibiotics, systemic steroids, and oxygenating mouth rinses predispose to the formation of a hairy tongue. The "hairs" are filiform papillae with multiple layers of keratin that fail to shed adequately. The papillae are putatively colonized by chromogenic bacteria, so that the tongue may appear black, brown, or even green.

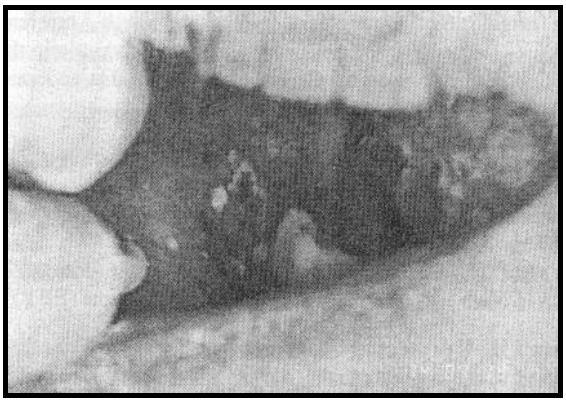
INFECTIONS

Fungal Infection

24. How many clinical forms of candidiasis are there?

Acute forms: pseudomembranous candidiasis (the typical type with curdy white patches) and atrophic candidiasis (angular cheilitis, often seen in HIV infection).

Chronic forms: hyperplastic candidiasis (leukoplakia-like patches that do not wipe off easily), atrophic candidiasis (denture sore mouth), mucocutaneous candidiasis (associated with skin candidiasis and an underlying systemic condition such as an endocrinopathy).



Acute pseudomembranous candidiasis.

25. What factors predispose to candidal infection?

Predisposing factors include (1) poor immune function, which may be due to age (very young and very old), malignancies, immunomodulating drugs, endocrine dysfunction, or HIV infection; (2) malnutrition; (3) antibiotics that upset the normal balance of flora; and (4) dental prostheses, especially dentures; and (5) alteration in saliva flow and constituents.

26. A culture performed on an oral ulcer grows *Candida sp.* Does this mean that the patient has candidiasis?

No. Approximately one-half of the adult population harbors *Candida sp.* in the mouth. These persons grow the organisms on culture in the complete absence of a candidal infection.

27. How do you make a diagnosis of candidiasis?

1. **Good clinical judgment.** Pseudomembranous plaques of candidiasis wipe off, leaving a raw, bleeding surface.
2. **Potassium hydroxide (KOH) preparation.** The plaque is scraped, and the scrapings are put onto a glass microscopic slide. A few drops of KOH are added, the slide is.warrned over an alcohol flame for a few seconds, and a coverslip is placed over the slide. The hyphae, if present, can be seen with a microscope.
3. **Biopsy** to show hyphae penetrating the tissues (too invasive for routine use).
4. **Cultures.** Although cultures are not the ideal way to diagnose candidiasis, the quantity of candidal organisms that grow on culture correlates somewhat with clinical candidiasis.

28. What are common antifungal agents for treating oral candidiasis?

- Polyenes: nystatin (topical), amphotericin (topical, systemic)
- Imidazoles: chlortrimazole, ketoconazole
- Triazoles: fluconazole

29. Actinomycosis represents a fungal infection. True or false?

False. Actinomycetes is a gram-positive bacteria. Do not be fooled by the suffix *mycosis.*

30. What are sulphur granules?

These yellowish granules (hence the name) are seen within the pus of lesions of actinomycosis. They represent aggregates of Actinomyces israelii, which are invariably surrounded by neutrophils.

31. Name two opportunistic fungal diseases that often present in the orofacial region.

Aspergillosis and zygomycosis tend to infect immunocompromised hosts; the latter causes rhinocerebral infections in patients with diabetes mellitus.

32. Name two deep fungal infections that are en in North America.

Histoplasmosis (caused by *Histoplasma capsulatum*) is endemic in the Ohio—Mississippi basin, and coccidioidomycosis (caused by *Coccidioides immitis*) is endemic in the San Joaquin Valley in California.

Viral Infection

33. Name the four most common viruses of the Herpesviridae family that are pathogenic in humans.

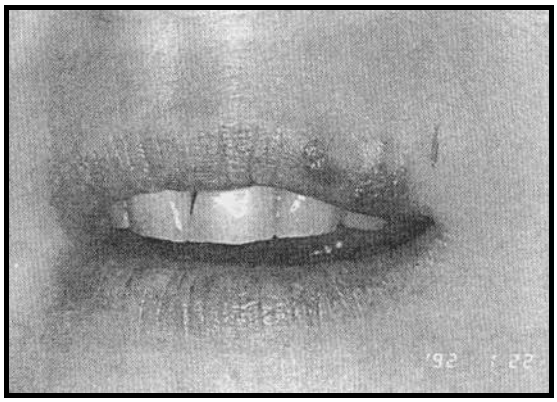
Herpes simplex virus (HSV 1 and 2)
Varicella zoster virus (VZV)
Cytomegalovirus (CMV)
Epstein-Barr virus (EBV)

34. Antibodies against HSV protect against further outbreaks of the disease. True or false?

False. The herpes viruses are unique in that they exhibit latency. Once one has been infected by HSV 1, the virus remains latent within the trigeminal ganglion for life. When conditions are favorable (for the virus, not the patient), HSV travels along nerve fibers and causes a mucocutaneous lesion at a peripheral site, such as a cold sore on the lip. A positive antibody titer (IgG) indicates that the patient has been previously exposed, and at the time of reactivation the titer may rise.

35. How do you differentiate between recurrent aphthous ulcers and recurrent herpetic ulcers?

Clinically, recurrent aphthous ulcers (minor) occur only on the nonkeratinized mucosae of the labial mucosa, buccal mucosa, sulci, ventral tongue, soft palate, and faucial pillars. Recurrent herpetic ulcers occur on the vermilion border of the lips (cold sores or fever blisters) and on the keratinized mucosae of the palate and attached gingiva. A culture confirms the presence of virus. In immunocompromised hosts, however, recurrent herpetic lesions may occur on both the keratinized and nonkeratinized mucosae.



Recurrent herpes labialis (cold sores or fever blisters).

36. An elderly patient with long-standing rheumatoid arthritis presents with a history of upper respiratory tract infection, ulcers of the right hard palate, right facial weakness, and vertigo. What does he have?

Herpes zoster infection, which typically is unilateral. The patient also has Ramsay-Hunt syndrome, which is caused by infection of cranial nerves VII and VIII with herpes zoster, leading to facial paralysis, tinnitus, deafness, and vertigo.

37. What lesions associated with the Epstein.Barr virus may present in the orofacial region?

Infectious mononucleosis
Burkitt's lymphoma (African type)
Nasopharyngeal carcinoma
Hairy leukoplakia

38. How does infectious mononucleosis present in the mouth?

Infectious mononucleosis usually presents as multiple, painful, punctate ulcers of the posterior hard palate and soft palate in young adults or adolescents. It is often associated with regional lymphadenopathy and constitutional signs of a viral illness.

39. What oral lesions have been associated with infection by human papillomavirus (HPV)?

- Focal epithelial hyperplasia (Heck's disease)
- Oral condylomas
- Verruca vulgaris
- Squamous papilloma
- Some squamous cell and verrucous carcinomas

The benign conditions are usually associated with HPV 6 and 11; the malignant ones with HPV 16 and 18.

40. What oral conditions does coxsackievirus cause?

Herpangina and hand-foot-mouth disease are caused by the type A coxsackievirus and generally affect children, who then develop oral ulcers associated with an upper respiratory tract viral prodrome.

41. What are Koplik spots?

Koplik spots are early manifestations of measles or rubeola (hence they also are called herald spots). They are 1—2-mm, yellow-white, necrotic ulcers with surrounding erythema that occur on the buccal mucosa, usually a few days before the body rash of measles is seen. Koplik spots are not seen in German measles.

Other Infections

42. What are the organisms responsible for noma?

Noma, which is a gangrenous stomatitis resulting in severe destruction of the orofacial tissues, is usually encountered in areas where malnutrition is

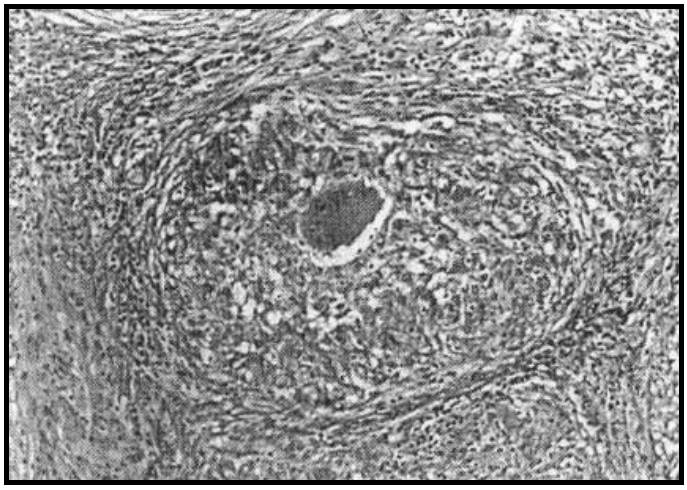
rampant. The bacteria are similar to those associated with acute necrotizing ulcerative gingivitis, namely, spirochetes and fusiform bacteria.

43. What are the oral findings in syphilis?

Primary: oral chancre
Secondary: mucous patches, condyloma lata
Tertiary: gumma, glossitis
Congenital: enamel hypoplasia, mulberry molars, notched incisors

44. What is a granuloma?

Strictly speaking, a granuloma is a collection of epithelioid histiocytes that often is associated with multinucleated giant cells like the Langhans-type giant cells seen in granulomas of tuberculosis. Many infectious agents, including fungi (such as histoplasmosis) and those causing tertiary syphilis and cat-scratch disease, can produce granulomatous reactions. Foreign body reactions are often granulomatous. Some granulomatous diseases, such as cheilitis granulomatosa, Crohn's disease, and sarcoidosis, have no known etiology.



Tuberculous granuloma with Langhans giant cell.

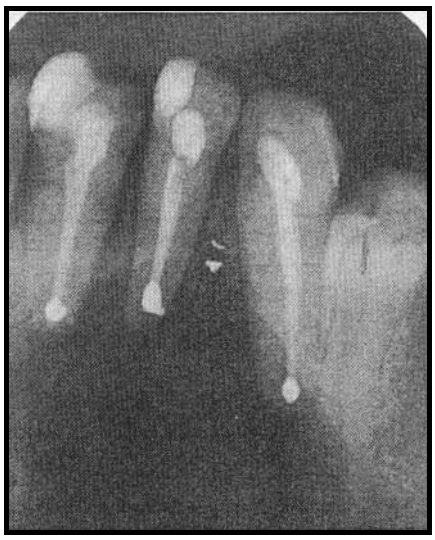
45. What are Langhans cells?

Langhans cells are multinucleated giant cells seen in granulomas, usually those caused by Mycobacterium tuberculosis. Their nuclei have a characteristic horseshoe distribution. Do not confuse them with Langerhans cells, which are antigen-processing cells.

REACTIVE, HYPERSENSITIVITY, AND AIJTOIMMUNE CONDITIONS
Intrabony and Dental Tissues

46. The periapical granuloma is composed of a collection of histiocytes, that is, a true granuloma. True or false?

False. The periapical granuloma is a tumorlike (-oma) proliferation of granulation tissue found around the apex of a nonvital tooth. It is associated with chronic inflammation from pulp devitalization. The inflammation can stimulate proliferation of the epithelial rests of Malassez to form a cyst, either apical radicular or periapical.



Apical radicular cyst

47. What is condensing osteitis?

Condensing osteitis, a relatively common condition, manifests as an area of radiopacity in the bone, usually adjacent to a tooth that has a large restoration or a root canal, although occasionally it may lie adjacent to what appears to be a sound tooth. It is asymptomatic. Histologically, condensing osteitis consists of dense bone with little or no inflammation. It probably arises as a bony reaction to a low-grade inflammatory stimulus from the adjacent tooth. It also has been referred to as idiopathic osteosclerosis, bone scar, and focal scierosing osteomyelitis. Idiopathic osteosclerosis/bone scar are similar lesions unassociated with teeth.

48. What are the etiologic differences among the wearing down of teeth caused by attrition, abrasion, and erosion?

Attrition: tooth-to-tooth contact

Abrasion: a foreign object-to-tooth contact, e.g., toothbrush bristles, bobby pins, nails

Erosion: a chemical agent-to-tooth contact, e.g., lemon juice, gastric juices

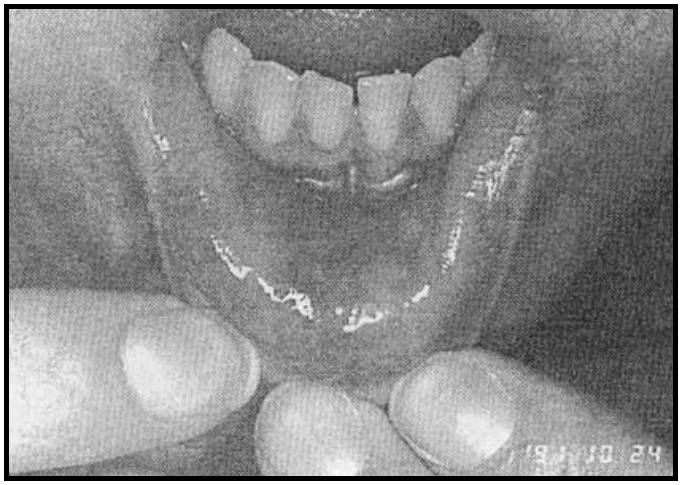
Soft Tissue Conditions

49. Aphthous ulcers may be associated with certain systemic conditions. Name them.

- Iron, folate or vitamin B 12 deficiency
- Inflammatory bowel disease
- Behçet's disease
- Reiter's disease
- HIV infection
- Conditions predisposing to neutropenia

50. An aphthous ulcer is the same as a traumatic ulcer. True or false?

False but with reservations. A traumatic ulcer is the most common form of oral ulcer and, as its name suggests, occurs at the site of trauma such as the buccal mucosa, lateral tongue, lower labial mucosa, or sulci. It follows a history of trauma such as mastication or toothbrush injury. An aphthous ulcer may occur at the same sites, but often with no history of trauma. However, patients prone to developing aphthae tend to do so after episodes of minor trauma.



Recurrent aphthous ulcer (minor) of lower labial mucosa.

51. A child returns one day after a visit to the dentist at which several amalgam restorations were placed. He now has ulcers of the lateral tongue and buccal mucosa on the same side as the amalgams. What is your diagnosis?

Factitial injury. Children may inadvertently chew their tongues and buccal mucosae while tissues are numb from local anesthesia, because the tissues feel strange to the child. Children and parents should be advised to be on the look-out for such behavior.

52. Is the mucocele a true cyst?

It depends. The term mucocele refers loosely to a cystlike lesion that contains mucus and usually occurs on the lower lip or floor of the mouth. However, it may occur wherever mucus glands are present. In most cases, it is not a true cyst because it is not lined by epithelium. It is caused by escape of mucus into the connective tissue when an excretory salivary duct is traumatized. Therefore, the mucocele is lined by fibrous and granulation tissue. In a small number of cases, it is caused by distention of the excretory duct due to a distal

obstruction. In such a case, the mucocele is a true cyst, because the lining is the epithelium of the duct.

53. What is the etiology of necrotizing sialometaplasia?

This painless ulcer usually develops on the hard palate but may occur wherever salivary glands are present. It represents vascular compromise and subsequent infarction of the salivary gland tissue, with reactive squamous metaplasia of the salivary duct epithelium that may mimic squamous cell carcinoma. The lesion resolves on its own.

54. Name the major denture-related findings in the oral cavity.

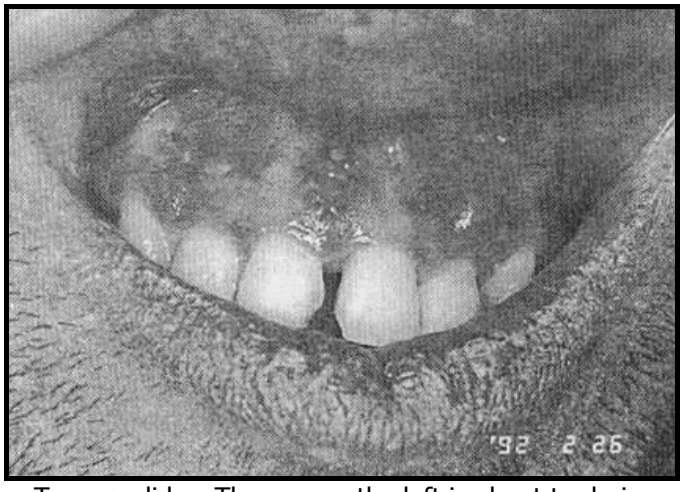
- Chronic atrophic candidiasis, especially of the palate (denture sore mouth)
- Papillary hyperplasia of the palatal mucosa
- Fibrous hyperplasia of the sulcus where the denture flange impinges (epulis fissuratum)
- Traumatic ulcers from overextension of flanges
- Angular cheilitis from overclosure
- Denture-base hypersensitivity reactions

55. A patient is suspected of having an allergy to denture materials. What do you recommend?

The patient should be patch-tested by an allergist or dermatologist to a panel of denture-base materials, which include both metals and products of acrylic polymerization. Usually, the lesions resolve with topical steroids.

56. What is a gum boil (parulis)?

A gum boil is an erythematous nodule usually located on the attached gingiva. It may have a yellowish center that drains pus and may be asymptomatic. The nodule consists of granulation tissue and a sinus tract that usually can be traced to the root of the tooth beneath with a thin gutta percha point. It indicates an infection of either pulpal or periodontal origin.



Two parulides. The one on the left is about to drain.

57. What is plasma cell gingivitis?

Plasma cell gingivitis, reported in the 1970s, presented as an intensely erythematous gingivitis and was likely due to an allergic reaction to a component of chewing gum or other allergen.

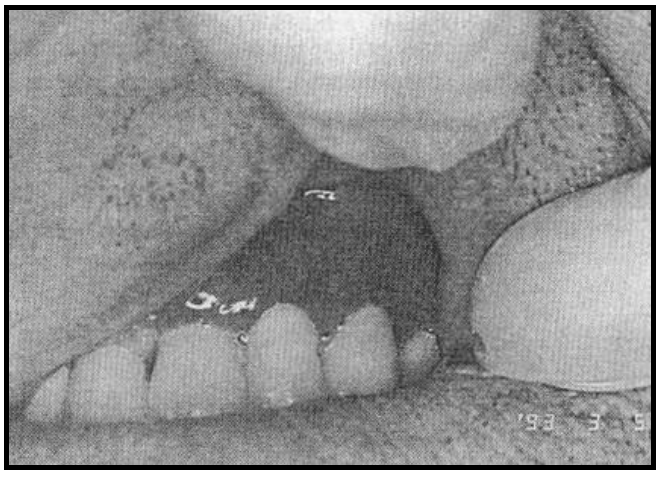
58. Some patients have a reaction to tartar-control toothpaste. What is the offending ingredient?

The offending ingredient is cinnamaldehyde. Susceptible patients develop burning of the mucosa and sometimes bright red gingivitis, akin to plasma cell gingivitis, after using the product. They often also have a reaction to chewing gum that contains cinnamon.

59. What is the differential diagnosis for desquamative gingivitis? What special handling procedures are necessary if you obtain a biopsy?

Desquamative gingivitis, which usually affects middle-aged women, is characterized by red, eroded, and denuded areas of the gingiva. Definitive diagnosis requires immunoreactive studies of the gingiva with various commerically available antibodies directed against autoantibodies, usually with direct immunofluorescence techniques. To preserve the integrity of immune reactants, the biopsy specimen should be split: one-half should be submitted in formalin for routine histopathology and the other half in Michel's solution or fresh on ice.

The immunofluorescence patterns show that 50% of lesions are cicatricial pemphigoid, 25% are lichenoid reactions or lichen planus, 20% have nonspecific immunoreactivity, and 5% are bullous pemphigoid and pemphigus vulgaris. Occasionally, other conditions, such as lupus erythematosus, linear IgA disease, and epidermolysis bullosa acquisita, may present as desquama tive gingivitis.



Desquamative gingivitis.

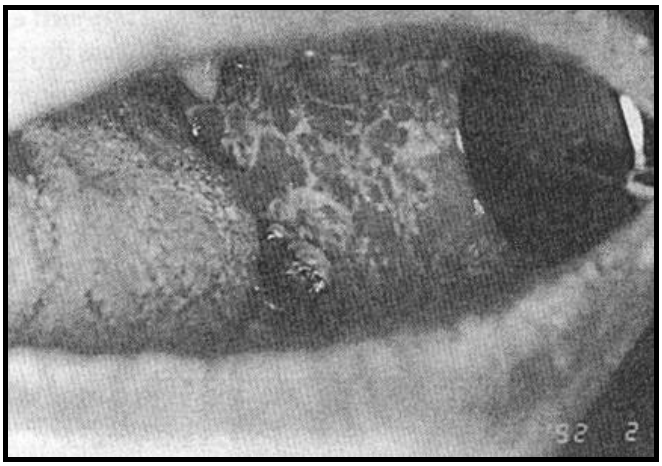
60. What is the Grinspan syndrome?

As reported by Grinspan, this syndrome consists of hypertension, diabetes mellitus, and lichen planus. Current thinking suggests that the lichen planus is

caused by medications that the patients take for hypertension (especially hydrochlorothiazides) and diabetes mellitus.

61. What drugs can give a lichen planus-like (lichenoid) mucosal reaction?

- Drugs for treating hypertension, such as hydrochlorothiazide, captopril, and methyldopa
- Hypoglycemic agents, such as chlorpropamide and tolazamide
- Antiarthritic agents, such as penicillamine
- Antigout agents, such as allopurinol
- Nonsteroidal antiinflammatory drugs.



Desquamative gingivitis.

62. Name the drugs that can be used to treat symptomatic lichen planus.

Most of the drugs involved are immunomodulating agents. The most commonly used are corticosteroids applied topically, injected intralesionally, or taken systemically. Dapsone, azathioprine, and cyclosporine A have been used with some success. More recently, retinoids also have been prescribed with limited success.

63. What is galvanism?

Galvanism is the processs by which different metals in contact with each other (as in amalgam) set up "cells" and "currents." In susceptible people, it may lead to electrogalvanically induced keratoses and lichenoid lesions of the mucosa in contact with amalgam restorations.

64. What are the typical skin lesions of erythema mulitforme called?

Target, iris, or "bull's eye" lesions. Erythema multiforme is an acute mucocutafleous inflammatory process that may recur periodically in chronic form. It may be idiopathic but also may occur after ingestion of drugs or after a herpes simplex virus infection.

65. Name the most common factors responsible for recurrent erythema multiforme.

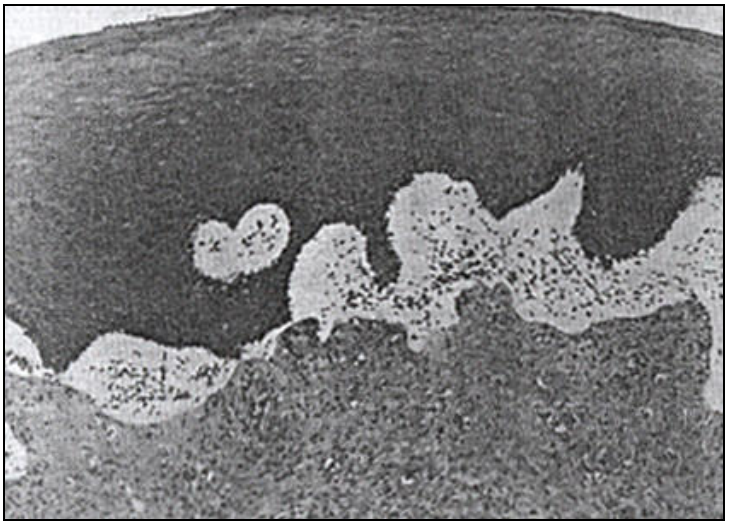
Herpes simplex virus reactivation and hypersensitivity to certain foods, such as benzoates. Do not expect to be able to culture herpes simplex virus from the lesions of recurrent erythema multiforme, which is a hypersensitivity reaction to some component of the virus. Usually the viral infection precedes the lesions of erythema multiforme.

66. What is Stevens-Johnson syndrome?

Stevens-Johnson syndrome is a severe form of erythema multiforme with extensive involvement of the mucous membranes of the oral cavity, eyes, genitalia, and occasionally the upper gastrointestinal and respiratory tracts. Desquamation and ulceration of the lips, with crusting, is usually dramatic. Typical target lesions may be seen on the skin.

67. What is the difference between pemphigus and pemphigoid?

Both are autoimmune, vesiculobullous diseases. In pemphigus (usually vulganis), autoantibodies attack desmosomal plaques of the epithelial cells, leading to acantholysis and formatiofl of an intraepithelial bulla. In pemphigoid (usually cicatricial), autoantibodies attack the junction between the epithelium and connective tissue, leading to the formation of a subepithelial bulla.



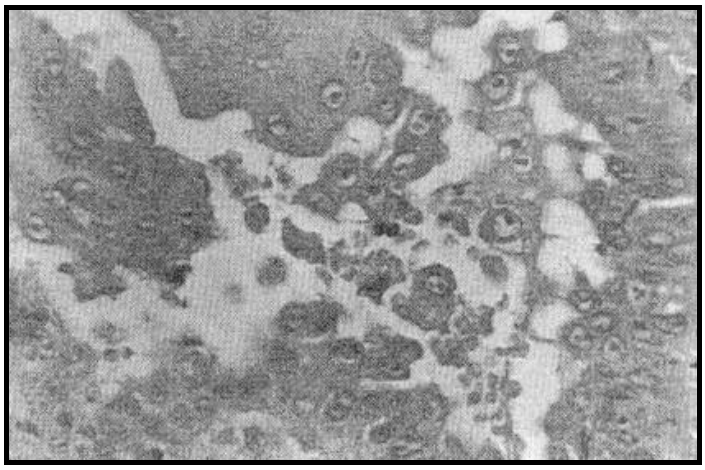
Subepithelial bulla formation in cicatricial pemphigoid.

68. What two forms of pemphigoid involve the oral cavity?

Cicatricial pemphigoid (formerly known as mucous membrane pemphigoid) and bullous pemphigoid. These autoimmune vesiculobullous diseases have antigens located in the lamina lucida of the basement membrane. Cicatricial pemphigoid presents primarily with oral mucosal and ocular lesions and occasionally with skin lesions, whereas bullous pemphigoid presents primanly with skin lesions and occasionally with mucosal lesions.

69. Differentiate between a Tzanck test and a Tzanck cell.

The **Tzanck test** entails direct examination of cells that may indicate a herpes simplex virus infection. The test is done by scraping the lesion (which may be a vesicle, ulcer, or crust) and smearing the debris on a slide. The slide is then stained and examined under a microscope for virally infected cells, which show multinucleatjon and "ground-glass" nuclei. **Tzanck cells** are acantholytic cells seen within the bulla of lesions of pemphigus vulgaris. Tzanck (acantholytic) cells of pemphigus vulganis.



Tzanck (acantholytic) cells of pemphigus vulganis.

70. What is the difference between systemic lupus erythematosus (SLE) and discoid lupus erythematosus (DLE)?

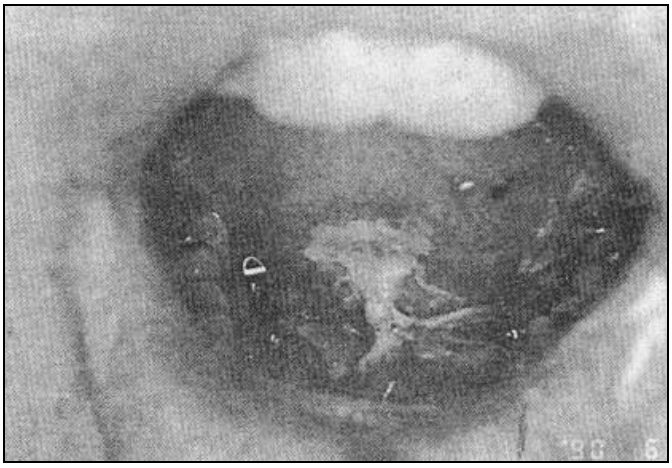
SLE is the prototypical multisystem autoimmune disease characterized by circulating antinuclear antibodies; the principal sites of injury are skin, joints, and kidneys. The oral mucosa is often involved, and the lesions may appear lichenoid, with white striae, and atrophic or erythematous. DLE is the limited form of the disease; most manifestations are localized to the skin and mucous membranes with no systemic involvement. DLE does not usually progress to SLE, although certain phases of SLE are clinically indistinguishable from DLE. The oral findings are similar in both.

71. What is the midline lethal granuloma?

This term describes a destructive, ulcerative process, usually located in the midline of the hard palate, that may lead to palatal perforation. Although the clinical picture is dramatic and ominous, the histologic picture may be somewhat nonspecific, showing only inflammation and occasionally vasculitis. Some authorities believe that midline lethal granuloma may be a localized form of an inflammatory condition known as Wegener's granulomatosis. Other conditions that may present in a similar fashion include fungal infections, syphilitic gummas, and malignant neoplasms such as lymphomas.

72. What are the common oral manifestations in patients who have undergone chemo therapy?

Chemotherapy can produce direct stomatotoxicity by acting on mitotically active cells in the basal cell layer of the epithelium. The mucosa becomes atrophic and, when traumatized, ulcerates. The chemotherapeutic agents also act on other rapidly dividing cells in the body, such as hematopoietic tissues. The results are neutropenia, anemia, and thrombocytopenia. Neutropenia may have an indirect stomatotoxic effect by allowing oral bacteria to colonize the ulcers. Usually, these ulcers develop in the period of profound neutropenia and resolve when neutrophils reappear in the blood circulation. In addition, patients are at increased risk for developing oral candidiasis, oral herpetic lesions, and deep fungal infections. Thrombocytopenia may cause oral petechiae, ecchymoses, and hematomas, especially at sites of trauma.



Chemotherapy-associated oral ulcerative mucositis.

73. A patient who underwent cancer chemotherapy now has recurrent intraoral herpetic lesions but no history of cold sores or fever blisters. Is this likely?

Yes. Many people have been exposed to herpes simplex virus without their knowledge and are completely asymptomatic. The virus becomes latent within sensory ganglia and reactivates to give rise to recurrent or recrudescent herpetic lesions. The prevalence of people who have been exposed to HSV increases with age.

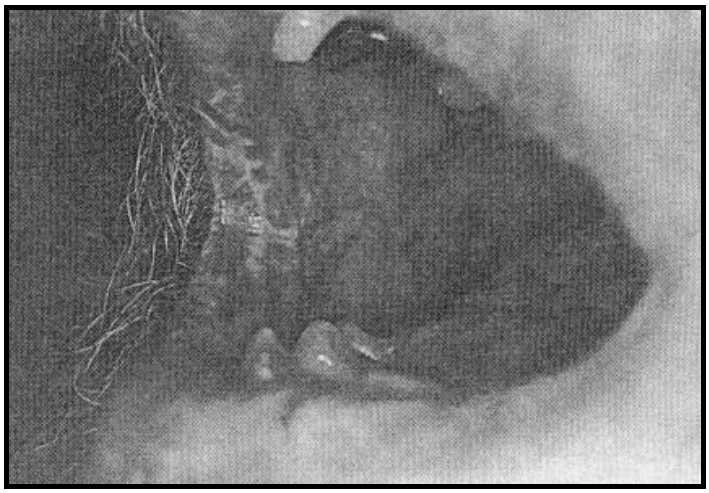
74. What are the complications of leukemia in the oral cavity, aside from those associated with chemotherapy?

Leukemic infiltration of the bone marrow leads to reduced production of functional components of the marrow. Granulocytopenia results in more frequent and more aggressive odontogenic infections; thrombocytopenia results in petechiae, ecchymoses, and hematomas in the oral cavity, which is subject to

trauma from functional activities. The patient may have a more than adequate white cell count, but many of the white cells are malignant and do not necessarily function like normal white cells. In addition, some leukemias, especially acute monocytic leukemia, have a propensity to infiltrate the gingiva, causing localized or diffuse gingival enlargement.

75. A patient underwent a matched allogenic bone marrow transplantation for the treatment of leukemia. Three months later he has erosive and lichenoid lesions in his mouth .What is your diagnosis?

The likely diagnosis is chronic oral graft-vs-host disease. The allogenic bone marrow transplant or graft contains immunocompetent cells that recognize the host cells as foreign and attack them. The oral lesions of chronic graft-vs.-host disease resemble the lesions of lichen planus.



Chronic oral graft-vs-host disease of buccal mucosa.

76. What are the effects of radiation on the oral cavity?

Short-term: oral erythema and ulcers, candidiasis, dysgeusia, parotitis, acute sialadenitis

Long-term: xerostomia, dental caries, osteoradionecrosis, epithelial atrophy and fibrosis

77. What factors predispose to osteoradionecrosis?

This necrotic process affects bone that has been in the radiation field. Predisposing factors include high total dose of radiation (especially if> 6,500 cGy), presence of odontogenic infection (such as periapical pathosis and periodontal disease), trauma (such as extractions), and site (the mandible is less vascular and more susceptible than the maxilla).

78. What is the basic cause of osteoradionecrosis?

The breakdown of hypocellular, hypovascular, and hypoxic tissue readily results in a chronic, nonhealing ulcer that can be secondarily infected. Some repo show that the infection is for the most part superficial.

79. What are the common oral manifestations of HIV infection?

Soft tissue: candidiasis, recurrent herpetic infections, deep fungal infections, aphthous ulcers, hairy leukoplakia, viral warts

Periodontium: nonspecific gingivitis, acute necrotizing ulcerative gingivitis, severe and rapidly destructive periodontal disease, often with unusual pathogens

Tumors: Kaposi's sarcoma, B-cell lymphoma, squamous cell carcinoma

80. A patient who tested positive for HIV antibodies presents with a CD4 count of 150 but has never had an opportunistic infection or been symptomatic. Does he have AIDS?

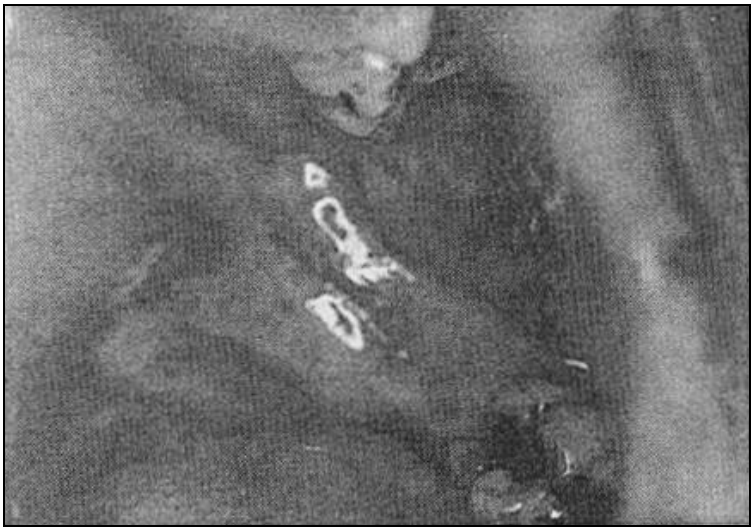
Yes. By the CDC definition (February 1993), patients with CD4 counts below 200 are considered to have AIDS.

81. Like other leukoplakias, hairy leukoplakia has a tendency t progress to malignancy. True or false?

False. Hairy leukoplakia is associated with EBV infection and usually a superimposed hyperplastic candidiasis. HPV also has been associated with hairy leukoplakia, which is not a premalignant condition. However, patients infected with HIV are more susceptible to oral cancer in general.

82. Are HIV-associated aphthous ulcers similar to recurrent major aphthae?

Yes. They tend to be greater than 1 cm, persist for long periods (weeks to months), and are difficult to treat.



HIV-associated aphthous ulcers of the soft palate and oropharynx.

83. Should HIV-associated aphthous ulcers be routinely cultured?

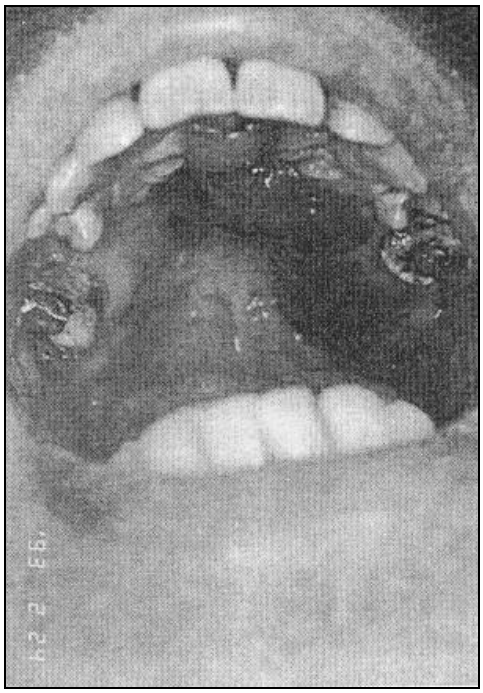
Yes. Often the culture is positive for HSV or even CMV, and the patient needs to be treated appropriately.

84. Kaposi's sarcoma (KS) is seen equally in the different population risk groups. True or false?

False. Over 90% of the epidemic cases of KS are diagnosed in homosexual or bisexual men. KS is an AIDS-defining lesion that is seen much less frequently in the other risk groups. It is associated with the presence of a new virus—Kaposi's sarcoma-associated human herpesvirus 8.

85. What management issues other than infection control and diagnosis of oral lesions should you keep in mind when treating patients with AIDS?

Hematologic dysfunction is common. HIV infection is associated with autoimmune thrombocytopenic purpura granulocytopenia and anemia. In addition, antiretroviral agents such as zidovudine are myelosuppressive, as are drugs used as prophylaxis against *Pneumocystis carinii* pneumonia, such as trimethoprim-sulfamethoxazole. The patient's blood picture should be known before treatment, especially surgical procedures, begins.



HIV-related Kaposi's sarcoma of the palate.

86. How do you treat intraoral Kaposi's sarcoma?

Surgical excision, intralesional injections of ymca alkaloids, radiation, and possibly interferon.

87. Name the benign odontogenic tumors that are purely epithelial.

- Ameloblastoma
- Calcifying epithelial odontogenic tumor (Pindborg tumor)
- Adenomatoid odontogenic tumor
- Solid variant of the calcifying odontogenic cyst
- Squamous odontogenic tumor
- Clear-cell odontogenic tumor (rare)

88. Which odontogenic tumor is associated with amyloid production? With ghost cells?

Calcifying epithelial odontogenic tumor (Pindborg tumor) is associated with amyloid production; calcifying epithelial odontogenic cyst (Gorlin cyst) is associated with ghost cells.

89. Which two lesions, one in the long bones and one in the cranium, resèThble the ameloblastoma?

In the long bones, adamantinoma; in the cranium, craniopharyngioma.

90. All forms of ameloblastoma behave aggressively and tend to recur. True or false?

False. One form of ameloblastoma, which occurs in adolescents and young adults, behaves less aggressively and has a lower tendency to recur. It is is called unicystic ameloblastoma.

91. Because ameloblastoma is so aggressive, it can be considered a malignancy. True or false.

False. Ameloblastoma is a locally destructive lesion that has no tendency to metastasize. However, it has two malignant counterparts: ameloblastic carcinoma and malignant ameloblastoma.

92. To which teeth are cementoblastomas usually attached?

The mandibular permanent molars.

93. Name two odontogenic tumors that produce primarily mesenchymal tissues.

Odontogenic fibroma and odontogenic myxoma.

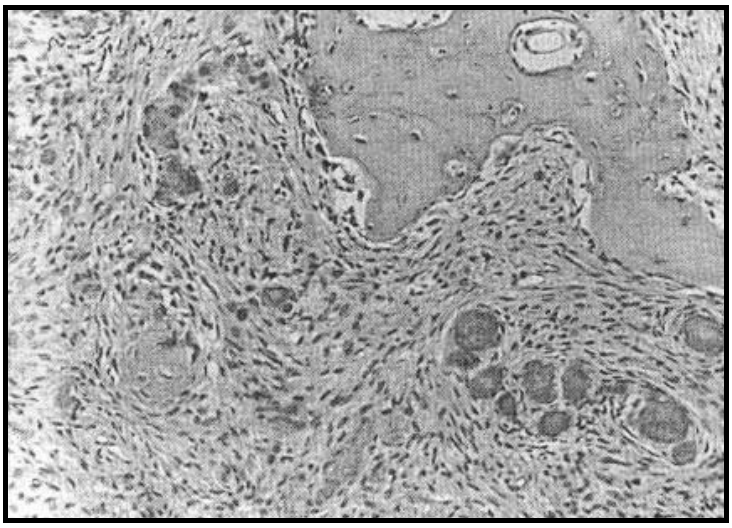
94. An adolescent presents with a mandibular radiolucency with areas that histologically resemble ameloblastoma as well as dental papilla. What is your diagnosis?

The diagnosis is ameloblastic fibroma, one of the rare odontogenic tumors that has both a neoplastic epithelial and mesenchymal component.

Fibroosseous Tumors

95. Ossifying fibromas arise from bone-producing cells, and cementifying fibromas are odontogenic in origin. True or false?

In real life and real pathology, the line of demarcation between the two is not so clear. They are clinically indistinguishable. Histologically, although pure ossifying and pure cementifying fibromas exist, it is much more common to see a mixture of bone/osteoid and cementum in any given lesion, with either predominating or in equal proportions. Many pathologists use the term cementoossifying fibroma as a unifying concept. The cell of origin is likely to be a mesenchymal cell in the periodontal ligament that is capable of producing either bone or cementum, therefore duplicating the two anchoring sites for Sharpey's fibers. From that point of view, both are odontogenic in origin.



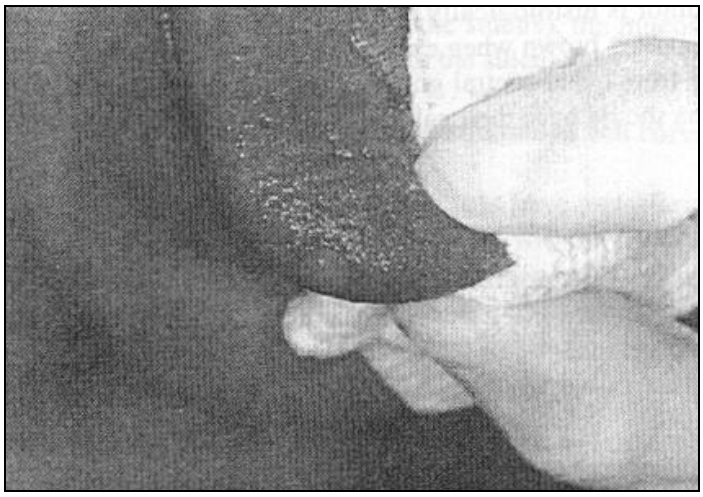
Central cementoossifying fibroma with round globules of cementum and trabeculae of osteoid.

96. Is it possible to distinguish histologically between fibrous dysplasia and central ossifying tThroma?

No. The clinical and radiographic findings are the most important for differentiating between the two. Fibrous dysplasia tends to occur in the maxilla of young people and presents as a poorly defined radiolucent or radiopaque area that is nonencapsulated. The radiographic appearance has been described as "ground glass." The central ossifying fibroma is a well-demarcated radiolucency, often with a distinct border, and may contain areas of radiopacity within the lesion. It is more common in the mandible.

97. Fibroma of the oral cavity is a true neoplasm. True or false?

It depends on your definition of neoplasm. As its name suggests, fibroma of the oral cavity is a tumor ("-oma") composed of fibrous tissue. It tends to occur as a result of trauma and therefore usually presents on the buccal mucosa, lower labial mucosa, and lateral tongue. It is nonencapsulated and grows as long as the inciting factor, such as trauma, is present. By Willis's definition of neoplasm ("new growth"), the growth, once established, continues in an excessive manner even after cessation of the stimuli that first evoked the change. Some pathologists, therefore, prefer the term fibrous hyperplasia rather than fibroma because it more accurately reflects its nature. The pathogenesis is similar to that of fibrous hyperplasias caused by poorly fitting dentures.



Fibroma of tongue

98. What are verocay bodies?

Verocay bodies consist of amorphous-looking, eosinophilic material that forms between parallel groups of nuclei in the schwannoma. They actually represent duplicated basement membrane produced by Schwann cells and are an important component of Antoni A tissue.

99. What is the cell of origin of the granular cell tumor? How is it different from the cell of origin of the congenital epulis of the newborn?

The cell of origin of the granular cell tumor is probably a neural cell, such as the Schwann cell. This tumor used to be called the granular cell myoblastoma because it was believed that the cell of origin was a myocyte. The cell appears granular because it contains many lysosomes. By light microscopy, these cells resemble cells of the congenital epulis of the newborn. Whereas the granular cell tumor stains for S-lOO protein, a marker for neural tissues, among others, the congenital epulis does not.

100. A patient presents with multiple neuromas of the lips and tongue. What do you suspect?

The patient probably has multiple endocrine neoplasia type III, which is inherited as an autosomal dominant condition. Patients also have pheochromocytomas, café-au-lait macules, neurofibromas of the skin, and medullary carcinoma of the thyroid. Recognition of the oral findings may lead to early diagnosis of the thyroid carcinoma.

101. What are venous lakes?

Venous lakes are purplish-blue nodules or papules, often present on the lips of older people, that represent dilated venules or varices.

102. What is the most common benign salivary gland tumor?

Pleomorphic adenoma.

103. Why is pleomorphic adenoma sometimes called the benign "mixed tumor"?

Pleomorphic adenoma is called a "mixed tumor" because histologically it may have a mixture of both epithelial and connective tissue components, although in fact it is an epithelially derived tumor. The connective tissue components may be prominent because one of the cells responsible for the tumor is the myoepithelial cell, which, as its name suggests, has properties of both epithelial and connective tissue. This cell is responsible for the areas of cartilage and bone formation as well as for the myxoid nature of many "mixed tumors." In addition, there are areas of epithelial cell proliferation in the form of ducts, islands, and sheets of cells.

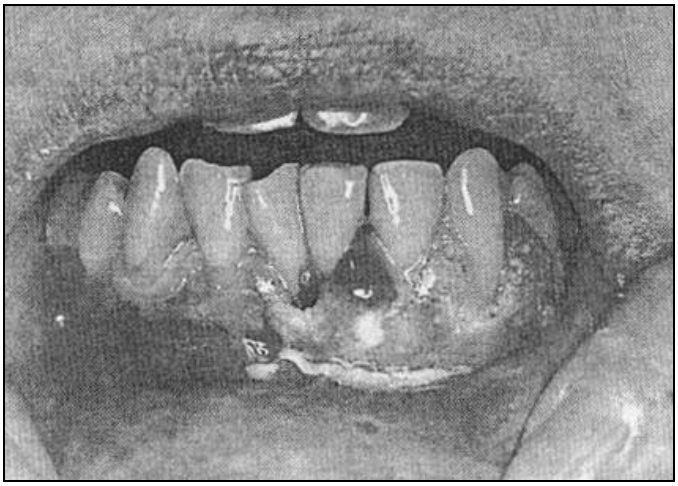
104. What is the brown tumor?

The brown tumor is histologically a central giant-cell granuloma associated with hyperparathyroidism. It appears brown when excised because it is a highly vascular lesion. Because it is indistinguishable from banal central giant-cell granuloma, all patients diagnosed with central giant-cell granuloma should have their calcium levels checked.

MALIGNANT NEOPLASMS

105. What percentage of the population has leukoplakia? What percentage of leukoplakias have dysplasia or carcinoma when first biopsied compared with erythroplakias?

Leukoplakia occurs in 3—4% of the population, and 15—20% of leukoplakias have dysplasia or carcinoma at the time of biopsy, whereas 90% of erythroplakias show such changes at the time of biopsy.



Squamous cell carcinoma presenting as leukoplakia with erythematous and verrucous areas.

106. What is proliferative verrucous leukoplakia?

It is a clinically aggressive and progressive form of leukoplakia with a higher rate of malignant transformation than banal leukoplakia.

107. What is the prevalence of oral cancer in the United States? Which country in the world has the highest prevalence of oral cancer?

Oral cancer accounts for 3—5% of all cancers in the United States if one includes oropharyngeal lesions. India has the highest prevalence of oral cancer, which is the most common cancer in that country and is related to the use of betel nut and tobacco products.

108. What are the risk factors for oral cancer?

- Tobacco products
- Alcohol (especially in conjunction with smoking)
- Betel nut products (especially in East Indians and some Southeast Asian cultures)
- Sunlight (especially for cancer of the lip in men)
- History of syphilitic glossitis
- History of submucous fibrosis
- Immunosuppression
- History of oral cancer or other cancer
- Preexisting oral mucosal dysplasia
- Age

109. What do snuff-associated lesions look like?

At the site where the snuff is placed (usually the sulcus), the mucosa is whitened with a translucent hue, and linear white ridges run parallel to the sulcus.

110. What is the difference in prognosis between a squamous cell carcinoma and a verrucous carcinoma?

Approximately one-half of squamous cell carcinomas have metastasized at the time of diagnosis. The larger they are, the more likely that metastases will develop. Verrucous carcinomas do not tend to metastasize despite the rather large size of some lesions. They are locally aggressive lesions. Whereas many squamous cell carcinomas are radiosensitive, verrucous carcinomas have been reported to become extremely aggressive and histologically anaplastic when treated with radiation

111. What is a "rodent ulcer"?

A rodent ulcer refers to a basal cell carcinoma that, despite its low tendency to metastasize, erodes through adjacent tissues like the gnawing of a rodent and through persistence may cause destruction of the facial complex.

112. What are the three most common intraoral malignant salivary gland tumors?

Mucoepidermoid carcinoma, polymorphous low-grade adenocarcinoma, and adenoid cystic carcinoma. The polymorphous low-grade adenocarcinoma also has been reported under the names of terminal duct carcinoma and lobular carcinoma.

113. Which two salivary gland tumors often show perinuclear invasion (neurotropism)?

Adenoid cystic carcinoma and polymorphous low-grade adenocarcinoma. However, any malignancy (particularly carcinomas) can show perinuclear invasion that may represent invasion of the lymphatics around a nerve.

114. The benign lymphoepithelial lesion of Sjögren's syndrome is an innocuous autoimmune sialadenitis. True or false?

False. The "benign" lymphoepithelial lesion is not so benign. Many experts believe that

these lesions are premalignant. Affected patients have a higher incidence of lymphoma than the

general population.

115. A patient with Sjogren's syndrome is referred for a labial salivary gland biopsy to identify a benign lymphoepithelial lesion. Does this sound right?

No. The benign lymphoepithelial lesion of Sjogren's syndrome is fou.ud in the major glands, mainly the parotid, especially if parotid enlargement is present. A labial salivary gland biopsy will show an autoimmune sialadenitis characterized by lymphocytic infiltrates that form foci. The more foci, the more likely the diagnosis of an autoimmune sialadenitis; foci are less specific than the lymphoepithelial lesion.

116. Do lymphomas of the oral cavity occur outside Waldeyer's ring?

Yes. Oral lymphomas are most common in Waldeyer's ring, but they may occur in the palate (a condition formerly described as lymphoproliferative disease of the palate), buccal mucosa, tongue, floor of the mouth, and retromolar areas. Not infrequently they are also primary lesions in the jaw bones.

117. What does a monoclonal plasma cell proliferation mean?

Plasma cells produce immunoglobulin that contains heavy and light chains. Each plasma cell and its progeny produce either kappa or lambda light chains. A group of plasma cells that produces only kappa or lambda light chains but not both is most likely due to a proliferation of a single malignant clone of plasma cells, such as a plasmacytoma or multiple myeloma. The presence of both light chains in a plasma cell proliferation is more in keeping with a polyclonal proliferation, which characterizes inflammatory lesions.

118. Name the different epidemiologic forms of Kaposi's sarcoma.

1. Classic r European form: usually Eastern European men (often Jewish); multiple red papules on the lo extremities, with rare visceral involvement and a more indolent course.

2. Endemic or African form: young men or children in equatorial Africa; frequent visceral involvement that may be fulminant.

3. Epidemic form: HIV-associated; may be widely disseminated to mucocutaneous and visceral sites; variable course.

4. Renal transplant-associated form: patients who have undergone renal transplantation with immunosuppressive therapy; lesions usually regress when immunosuppressive therapy is discontinued.

119. A patient has a suspected metastatic tumor to the mandible. What are the likely primary tumors?

- Lung
- Prostate
- Gastointestinal tract
- Thyroid
- Breast
- Kidney
- Skin

120. Osteosarcoma of the jaws occurs in younger patients more often than osteosarcoma of the long bones. True or false?

False. Patients with osteosarcoma of the jaws are 1—2 decades older than patients with osteosarcoma of the long bones.

121. What conditions predispose to osteosarcoma?

Many cases of osteosarcoma in young adults occur de novo. However, there are well-documented cases of osteosarcoma in association with Paget's disease, chronic osteomyelitis, a history of retinoblastoma, and prior radiation to the bone for fibrous dysplasia.

NONVASCULAR PIGMENTED LESIONS

122. What drugs can cause mucosal pigmentation?

- Oral contraceptives
- Antimalarial agents (e.g., plaquenil)
- Minocycline
- Zidovudine (possible)

123. Why does heavy metal poisoning primarily cause staining of the gingiva?

Heavy metals such as lead, bismuth, and silver may cause a grayish-black line to appear on the gingival margins, especially in patients with poor oral hygiene. Plaque bacteria can produce hydrogen sulfide, which combines with the heavy metals to form heavy metal sulfides that are usually black.

124. What can cause mucosal melanosis?

Benign: physiologic pigmentation, postinflammatory hyperpigmentation (especially in dark-skinned people), oral melanotic macule, smoking, mucosal nevus, melanoacanthosis

Malignant: melanoma

Systemic conditions: Peutz-Jegher's syndrome, Albright's syndrome, Addison's disease neurofibromatosis

125. What are the different forms of oral melanocytic nevi?

Intramucosal nevus: tends to be elevated. papular or nodular
Junctional nevus: tends to be macular
Compound nevus: tends to be papular
Blue nevus: tends to be macular

126. What is the most common site for oral melanoma?

Hard palate.

127. What is the difference between a melanocyte and a melanophage?

A melanocyte is a neuroectodermally derived dendritic cell that contains the intracellular apparatus to manufacture melanin. A melanophage is a macrophage that has phagocytosed melanin pigment and therefore can look like a melanocyte because it contains melanin. However, it lacks the enzymes to produce melanin.

METABOLIC LESIONS ASSOCIATED WITH SYSTEMIC DISEASE

128. What are the three presentations of Langerhans cell disease (histiocytosis X)?

Chronic localized disease: eosinophilic granuloma; usually in adults.

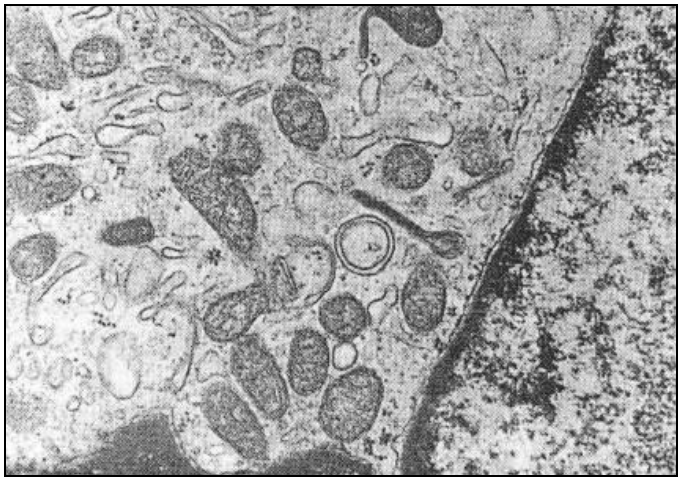
Chronic disseminated disease: limited to a few organ systems in adults. Hand-Schuller-Christian disease is a well-recognized form, characterized by

exophthalmos; diabetes insipidus and bony lesions; sometimes with skin and visceral involvement.

Acute disseminated disease: Letterer-Siwe disease in children; widespread involvement of multiple organ systems, especially skin; usually runs a rapidly progressive, often fatal course; considered a malignancy for the most part.

129. What are Birbeck granules?

Birbeck granules are racket-shaped cytoplasmic inclusions seen in Langerhans cells of histiocytosis X.



Racket-shaped Birbeck granule of Langerhans cell histiocytosis.

130. What are the oral changes associated with pregnancy?

Gingivitis and pyogenic granuloma (epulis gravidarum).

131. An elderly man complains that his jaw seems to be getting too big for his dentures and that his hat does not fit him anymore. What do you suspect?

Paget's disease (ostejtis deformans), a metabolic bone disease in which initial bone resorption is followed by haphazard bone repair, with resulting marked sclerosis. This condition may lead to narrowing of skull base foramina and neurologic deficits. The maxilla is often affected; a "cotton-wool" appearance has been described on radiographs.

132. What oral lesions are associated with gastrointestinal disease?

The most common gastrointestinal disease associated with oral signs is inflammatory bowel disease, especially Crohn's disease. Patients may manifest cobblestoning of the mucosa and papulous growths, which represent granulomatous inflammation similar to what is seen in the gastrointestinal tract. Occasionally, patients also develop a pyostomatitis vegetans. In addition, they may have aphthouslike ulcers as well as symptoms of glossitis associated with vitamin B 12 deficiency if part of the ileum has been resected for the disease.

Patients with gluten-sensitive enteropathies also may present with aphthouslike ulcers.

133. what is primary and secondary Sjögren's syndrome?

Primary Sjogren's syndrome, which used to be called the sicca syndrome, consists of dry eyes (keratoconjunctivitis sicca) and dry mouth (xerostomia) in the absence of other systemic conditions. Secondary Sjogren's syndrome consists of primary Sjogren's syndrome plus a connectivetissue disorder such as rheumatoid arthritis, systemic lupus erythematosus, progressive systemic sclerosis, or polymyositis. Most patients with Sjogren's syndrome have circulating autoantibodies.

134. What is the dental significance of the Sturge-Weber syndrome?

This syndrome is characterized by vascular malformations of the leptomeninges, facial skin innervated by the fifth nerve (nevus flammeus), and the corresponding ipsilateral areas in the oral mucosa and bone. Bleeding is therefore an important consideration in dental treatment. Patients also may exhibit mental retardation and seizure disorders. Treatment may include phenytoin.

DIFFERENTIAL DIAGNOSES AND GENERAL CONSIDERATIONS

Intrabony Lesions

135. What are pseudocysts of the jaw bones? Give examples.

These conditions appear cystlike on radiograph but are not true cysts. Examples include:

- Traumatic (simple) bone cyst: empty at surgery
- Aneurysmal bone cyst: giant cells and blood-filled spaces
- Static bone cyst (Stalne bone cavity): salivary gland depression
- Hematopoietic marrow defect: hematopoietic marrow

136. What is the differential diagnosis for a multiloculated radiolucency?

- Dentigerous cyst
- Odontogenic keratocyst
- Ameloblastoma
- Vascular malformations, such as hemangiomas
- Odontogenic myxoma
- Intraosseous salivary gland tumors
- Lesions that contain giant cells, such as aneurysmal bone cyst, central giant cell granuloma. and cherubism

137. What is the differential diagnosis for an upper lip nodule?

Salivary gland lesion: sialolith, benign salivary gland tumor (especially pleomorphic adenoma and canalicular adenoma), malignant salivary gland tumor

Vascular lesion: hemangioma, lymphangioma, other vascular anomaly

Neural lesion: neurofibroma, schwannoma, neuroma

Skin appendage tumors

138. What may cause diffuse swelling of the lips?

- Vascular malformations, such as lymphangiomas and hemangiomas
- Angioneurotic edema
- Hypersensitivity reactions
- Cheilitis glandularis
- Cheilitis granulomatosa (e.g., Melkersson-Rosenthal syndrome)
- Crohn's disease

139. What is the differential diagnosis for a solitary gingival nodule?

The most common diagnoses are fibroma or fibrous hyperplasia, pyogenic granuloma (especially in a pregnant patient), peripheral giant cell granuloma, and peripheral ossifying fibroma (essentially a fibrous hyperplasia with metaplastic bone formation). Other less common conditions include benign and malignant tumors, especially of odontogenic origin, and (in elderly patients) metastatic tumors.

140. What may cause generalized overgrowth of gingival tissues?

Common causes include plaque accumulation; drugs such as phenytoin, cyclosporine A, sodium valproate, diltiazem, and nifedipine (the last two are calcium channel blockers); fibromatosis gingivae; and leukemic infiltrate.

141. A labial salivary gland biopsy is useful for diagnosis of certain systemic conditions.What are they?

- Sjogren's syndrome
- Autoimmune sialadenitis associated with connective-tissue disease
- Graft-vs.-host disease
- Amyloidosis
- Sarcoidosis

142. What may cause chronic xerostomia?

Common causes include many anticholinergic drugs, autoimmune sialadenitis (such as Sjogren's syndrome and graft-vs.-host disease), aging (although many experts believe this to be drug-related), radiation to the gland, primary neurologic dysfunction, and nutritional deficiencies (e.g., vitamin A, vitamin B, and iron).

143. Name possible causes of bilateral parotid swelling.

- Mumps
- Sjögren's syndrome
- Radiation-induced acute parotitis
- Diabetes mellitus
- Malnutrition
- Alcoholism
- Bulimia
- Warthin 's tumor

144. What may cause depapillation of the tongue?

- Vitamin B deficiency
- Iron deficiency
- Folate deficiency
- Benign migratory glossitis (focally)
- Median rhomboid glossitis (focally)
- Syphilis
- Plummer-Vinson syndrome

145. What may cause diffuse enlargement of the tongue?

- Congenital macroglossia
- Lymphangioma
- Hemangioma
- Neurofibromatosis
- Hyperpituitarisni
- Cretinism
- Acromegaly
- Trisomy 21
- Amyloidosis
- Hypothyroidism

146. What is the differential diagnosis of midline swellings of the floor of the mouth?

- Ranula (mucocele)
- Epidermoid cyst
- Derrriojd cyst
- Benign lymphoepithelial cyst

147. What may cause diffuse white plaques in the oral cavity?

- Lichen planus (especially plaquetype)
- Cannon's white sponge nevus
- Leukedema
- Hereditary benign intraepithelial dyskeratosis
- Pachyonychia congenita
- Dyskeratosis congenita
- Extensive leukoplakia (especially proliferative verrucous leukoplakia)
- Candidiasis

148. Name the conditions that may give rise to papillary lesions of the oral cavity.

Possible underlying conditions include papilloma, verruca vulgaris, condyloma, papillary hyperplasia of the palatal mucosa (denture injury), Heck's disease, oral florid papillomatosis, venucous carcinoma, papillary squamous cell carcinoma, pyostomatitis vegetans (associated with inflammatory bowel disease), and verruciform xanthoma.

149. What lesions may occur in the oral cavity of neonates?

Lesions in the oral cavity of neonates include neuroectodermal tumor of infancy, congenital epulis of the newborn, gingival cyst of the newborn, palatal

cyst of the newborn (Bohn's nodules and Epstein's pearls), lymphangiomas of the alveolar ridge, and natal teeth.

150. What may cause "burning mouth" syndrome?

This sensation usually results from mucosa that is atrophic or inflamed, which, in turn, may be caused by candidiasis (especially atrophic candidiasis of the tongue or of the palate caused by dentures), xerostomia, allergies (especially to denture materials), and specific inflammatory mucosal lesions, such as lichen planus and migratory glossitis. Sometimes a psychological component may be involved.

151. What may cause oral paresthesia?

Oral paresthesia may be caused by manipulation or inflammation of a nerve or tissues around a nerve, direct damage to a nerve or tissues around a nerve, tumor impinging on or invading a nerve, pnmary neural tumor, and central nervous system tumor.

152. Why do lesions appear white in the oral cavity?

Lesions appear white because the epithelium has been changed, usually thickened, causing the underlying blood vessels to be deeper, as in hyperkeratosis, epithelial hyperplasia (acanthosis), and swelling of the epithelial cells (Cannon's nevus, leukedema). Lesions may appear white if exudate or necrosis is present in the epithelium (candidiasis, ulcers) or if there are fewer vessels in the connective tissue (scar). Finally, a change in the intrinsic nature of the epithelial cell, such as epithelial dysplasia, may cause the mucosa to appear white (leukoplakia).

153. Why do lesions appear red in the oral cavity?

Lesions appear red because the epithelium is thinned and the underlying vessels are now closer to the surface, as in epithelial atrophy, desquamative conditions, healing ulcers, and loss of the keratin layer. Redness also may be caused by an increase in the number or dilatation of blood vessels in the connective tissue, as in inflammation. Finally, a change in the intrinsic nature of the epithelial cell, such as epithelial dysplasia, may cause the mucosa to look red (erythroplakia).

154. Distinguish macules, papules, and plaque.

A macule is a localized lesion that is not raised and is better seen than felt. It is often used to describe localized pigmented lesions, such as amalgam tattoos and melanotic macules. Both papules and plaque are raised lesions; the papule is <5 mm, and the plaque is larger.

155. What is the difference between a bulla and vesicle?

The bulla is usually >5 mm in size; the vesicle is <5 mm.

156. Differentiate between a hamartoma and a choristoma.

A **hamartoma** is a tumorlike growth consisting of an overgrowth of tissues that histologically appear mature and are native to the area (e.g., hemangioma, odontoma). A **choristoma** is a tumorlike growth consisting of an overgrowth of tissues that histologically appear mature but are not native to the area (e.g., cartilaginous choristoma or bony choristoma of the tongue). A hamartoma of the skin and mucosa is sometimes called a nevus (e.g., vascular, epidermal, or melanocytic nevus).

157. What are oncocytes?

Oncocytes are eosinophilic, swollen cells found in many salivary gland tumors, such as oncocytomas and Warthin's tumor, and in oncocytic metaplasia of salivary ducts. They are swollen because they contain many mitochondria.

158. What are Russell bodies?

Russell bodies are round, eosinophilic bodies found in reactive lesions and represent globules of immunoglobulin within plasma cells.

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